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Question 1

Correct

1.00 points out of 1.00

Comment

A 13-year-old is admitted to the intensive care unit (ICU) after a transsphenoidal resection of a craniopharyngioma performed by neurosurgery. The patient tolerated the procedure well and was admitted to the ICU hemodynamically stable in room air. The bedside nurse informs the clinician of high urine output of 5 ml/kg/hr. Laboratory test results are shown:

Laboratory Test	Result
Sodium	155 mEq/L (155 mmol/L)
Potassium	4.4 mEq/L (4.40 mmol/L)
Chloride	105 mEq/L (105 mmol/L)
Carbon dioxide	32 mEq/L (32 mmol/L)
Urea nitrogen	30 mg/dL (3.0 g/L)
Creatinine	0.72 mg/dL (63.65 µmol/L)
Glucose	100 mg/dL (5.5 mmol/L)
Urine specific gravity	<1.005
Calcium	7.6 mg/dL (190.0 mmol/L)
Phosphorous	2.8 mg/dL (0.9 mmol/L)
Magnesium	2.6 mg/dL (1.07 mmol/L)
Serum osmolality	326 mOsm/kg (326 mmol/kg)
Urine osmolality	298 mOsm/kg (298 mmol/kg)

Of the following, the MOST likely cause of the patients increased urine output is

A. decreased absorption of sodium in the renal tubules

B. decreased distal tubule response to endogenous antidiuretic hormone (arginine vasopressin)

C. decreased secretion of antidiuretic hormone (arginine vasopressin)

D. osmotic diuresis secondary to intraoperative administration of mannitol

Your answer is correct.

PREP Pearl(s)

- Central diabetes insipidus can be a postsurgical complication of craniopharyngioma resections and other neurosurgical procedures.
- One should have a high suspicion for diabetes insipidus in the presence of polyuria with inappropriately dilute urine.

Critique

The patient in the vignette has evidence of central diabetes insipidus (DI), also known as arginine vasopressin deficiency. Diabetes insipidus can be broadly defined as the inability to concentrate urine secondary to a complete or partial failure of antidiuretic hormone secretion (classified as central diabetes insipidus), or, a complete or partial failure of the renal response to antidiuretic hormone (classified as nephrogenic diabetes insipidus); both result in variable degrees of polyuria. Whether or not the patient develops hypernatremia depends on the patient's hydration status and whether or not the patient has an intact thirst mechanism to counter the excessive urine output.

Lack of antidiuretic hormone (ADH) can be caused by disorders that affect one or more of the sites involved with ADH secretion and synthesis: the hypothalamic osmoreceptors, the supraoptic and paraventricular nuclei, and the superior portion of the supraoptico hypophyseal tract. In order to understand the development of central DI it is important to review the production and secretion of ADH. Antidiuretic hormone is first synthesized in the supraoptic and paraventricular nuclei of the hypothalamus. It is then streamed down the axons of the supraoptico hypophyseal tract and stored in and secreted by the posterior lobe of the pituitary gland. The clinical disorders of central DI are caused by disruption anywhere in this pathway.

The most common etiology of central DI is idiopathic, accounting for approximately 75% of all cases. These cases are most likely of autoimmune, antibody-mediated origin and/or associated with some rare genetic/hereditary forms. The remaining causes of central DI are acquired and are usually secondary to injury to the hypothalamic pituitary axis from head trauma, hypoxic ischemic encephalopathy, neurosurgery, and primary or secondary malignancies with a specific association with Langerhans cell histiocytosis. The clinical presentation of the patient is dependent on where in the pituitary axis the damage occurs. If the damage is below the hypothalamus, specifically below the median eminence, the clinical presentation of DI is usually transient, as ADH can also be synthesized and secreted via the portal capillaries surrounding the hypothalamus.

The patient in the vignette has evidence of possible neurosurgical induced central DI. The development of DI post neurosurgery is usually associated with treatment of a craniopharyngioma or other transsphenoidal surgeries, as there is a higher risk of actual damage to tracts during these procedures. While the incidence of neurosurgical-induced central DI outside of craniopharyngioma/transsphenoidal surgeries is not well-reported; there is a risk of developing central DI with any form of intradural neurosurgery, head trauma, or hypoxic ischemic encephalopathy. The etiology is not completely understood, but can be explained by the pathway that ADH travels in the hypothalamic tract. As ADH travels down the pathway to the posterior pituitary, it is also secreted and stored in the portal capillaries of the median eminence of the hypothalamus and in the cerebrospinal fluid of the third ventricle which is near the hypothalamus. Therefore, anything that changes or disrupts the balance of pressures in the ventricular system (eg, hydrocephalus, edema) can cause disruption in this hypothalamic pituitary axis.

The development of neurosurgical-induced central DI is usually associated with a triphasic response as described below, particularly if there is complete damage to the hypothalamus and tract:

Initial polyuric phase: typically begins 24 to 48 hrs after surgery and lasts 4 to 5 days, with a hallmark of high urine output and the development of hypernatremia. This is caused by the initial inhibition of ADH release due to hypothalamic dysfunction.

Antidiuretic phase: typically begins 6 to 10 days after the initial procedure, when previously synthesized ADH that has been stored in the posterior pituitary is released, with a hallmark of decrease urine output clinically mimicking the syndrome of inappropriate release of ADH (SIADH).

Third phase: return of high urine output and hypernatremia/permanent phase of central DI which develops once the stores of ADH in the pituitary are completely depleted.

In most cases of neurosurgical-induced central DI, the hypothalamic tract is not completely damaged; the patient will therefore develop a transient form usually involving phase 1 and 2 with resolution once the injury has healed. The development of neurosurgical induced central DI can be a diagnostic dilemma as initial polyuria post surgery can be secondary to appropriate excretion of excess fluid administered during the surgery, or from an osmotic diuresis secondary to mannitol administered during the surgery. This will usually develop early postoperatively and is not associated with hypernatremia, and would have an osmolar gap. One should have a high suspicion for DI in the presence of polyuria with inappropriately dilute urine (ie, when urine osmolality is lower than serum osmolality). A decreased response to endogenous ADH in the distal tubules of the kidney is a description of nephrogenic DI, and is not as likely in the patient in this vignette, as is the development of central DI. Decreased sodium reabsorption in the renal tubules is a description of cerebral salt wasting, which could be associated with high urine output postoperatively; however, this would be associated with hyponatremia and not hypernatremia, as seen in the patient in the vignette.

Suggested Reading(s)

- Nigro N, Grossmann M, Chiang C, Inder WJ. Polyuria-polydipsia syndrome: a diagnostic challenge. *Intern Med J.* 2018;48(3):244-253. doi:10.1111/imj.13627
- Wise-Faberowski L, Soriano SG, et al. Perioperative management of diabetes insipidus in children. *J Neurosurg Anesthesiol.* 2004;16(3):220-5. doi:10.1097/00008506-200407000-00006

Content Domain

- Endocrinology, Diabetes Insipidus

Learning Objectives

- Understand central versus nephrogenic diabetes insipidus
- Articulate why central diabetes insipidus is associated with neurosurgical procedures and cerebral injury

The correct answer is: decreased secretion of antidiuretic hormone (arginine vasopressin)

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Question 2

Correct

1.00 points out of 1.00

Comment

An 8-year-old child is admitted and requires intubation and mechanical ventilation after a severe episode of pulmonary hemorrhage. Oxygen saturation is 94% with a positive end expiratory pressure of 10 cm H_2O and FiO_2 of 0.6. A chest radiograph shows diffuse opacities, and chest computed tomography scan shows bilateral ground glass infiltrates with multiple pulmonary nodules. Further history reveals recurrent sinusitis and chronic otitis media in addition to her presenting symptoms of hemoptysis. The remainder of her laboratory studies are notable for a hemoglobin of 7.6 g/dL, a urea nitrogen of 49 mg/dL, creatinine of 1.6 mg/dL. Urinalysis is positive for 3+ protein and 3+ red blood cells. Her urine output is 1.5 mL/kg/hr. Treatment is initiated with intravenous methylprednisolone.

Of the following, in addition to pulse corticosteroid therapy, the MOST important treatment to begin is

A. extracorporeal membrane oxygenation

B. intravenous cyclophosphamide ✓

C. intravenous immunoglobulin

D. renal replacement therapy

Your answer is correct.

PREP Pearl(s)

- Granulomatosis with polyangiitis is a rare pediatric vasculitis. The majority of children have a history of upper respiratory tract manifestations prior to more severe systemic symptoms.
- Evidence for therapy is scarce, but consensus treatment plans recommend intravenous cyclophosphamide in conjunction with high-dose glucocorticoids as first-line induction therapy.

Critique

This child has a typical presentation for granulomatosis with polyangiitis (GPA, formerly Wegener granulomatosis), as evidenced by her imaging finding and involvement of both her renal and respiratory systems. Granulomatosis with polyangiitis is a systemic necrotizing vasculitis affecting small and medium blood vessels; it is one of the 3 main diseases in the group of anti-neutrophilic cytoplasmic antibodies (ANCA)-positive vasculitis, along with eosinophilic granulomatosis with polyangiitis (formerly Churg-Strauss syndrome) and microscopic polyangiitis. Other causes of potential pulmonary-renal vasculitis that should be considered include Goodpasture syndrome (which involves anti-glomerular basement membrane antibodies) and more rarely, systemic lupus erythematosus.

Granulomatosis with polyangiitis is very rare in the pediatric population. A 2018 review found an incidence of 1.8 cases per 1 million person-years, although data were limited to severe cases requiring hospitalization. Nonetheless, GPA carries significant mortality and morbidity. Granulomatosis with polyangiitis primarily affects the upper and lower respiratory tract and the kidneys. A majority of children with GPA have upper respiratory tract manifestations that may include oral/nasal ulcers, recurrent epistaxis, sinusitis, and

subglottic stenosis. Many also have renal involvement with glomerulonephritis that may progress to kidney failure. Skin lesions like palpable purpura, livedo reticularis, and nonspecific rash can be present. Up to one-third of patients may present with nonspecific musculoskeletal complaints (arthralgia, myalgia). Less commonly, there could be neurologic and cardiovascular manifestations with seizures and myocarditis.

While ANCA-positivity is common, the absence of an ANCA antibody does not rule out GPA. The ARChiVe cohort study reported that PR3-ANCA and/or c-ANCA were positive in 67% of the GPA patients, whereas MPO-ANCA and/or p-ANCA were found only in up to 55% of GPA patients. In 2022, the American College of Rheumatology/European Alliance of Associations for Rheumatology published the newest classification criteria for GPA diagnostics (Robson, 2022).

Treatment for these vasculitides can be generalized into induction and maintenance phases. As these conditions remain rare in children, pediatric rheumatologists relied on adult clinical trials as a guide to management. In 2022, the Childhood Arthritis and Rheumatology Research Alliance (CARRA) published consensus treatment plans for severe pediatric antibody-associated vasculitides. The pediatric-specific recommendations to induce remission are high-dose glucocorticoids, in combination with IV cyclophosphamide. In severe adult antibody-induced vasculitides, rituximab has also been utilized as an alternative to cyclophosphamide. Use of rituximab can be considered in children in critical organ or life-threatening disease when they have not responded to standard therapy, or when there are concerns about cyclophosphamide-associated infertility.

For maintenance therapy, the recommended first-line therapy are azathioprine, methotrexate, mycophenolate mofetil, or rituximab combined with low-dose glucocorticoids. Adjunctive therapy has included plasmapheresis, although evidence for this in adult therapy has been controversial. More recently, the largest trial of plasmapheresis in adults (PEXIVAS) showed plasmapheresis to have benefits in 2 endpoints, which were death from any cause or end-stage renal disease. Most pediatric rheumatologists would recommend use of plasmapheresis in rapidly progressive renal disease or severe pulmonary hemorrhage.

The patient in the vignette does not meet criteria for extracorporeal membrane oxygenation. Intravenous immunoglobulin is not an accepted first-line treatment to induce remission in this initial presentation of vasculitides. Finally, while some patients with severe renal failure due to GPA may need dialysis, this child also does not meet the criteria for renal replacement at this point of her course.

Suggested Reading(s)

- de Graeff N, Groot N, Brogan P, et al. European consensus-based recommendations for the diagnosis and treatment of rare paediatric vasculitides - the SHARE initiative. *Rheumatol (Oxford).* 2019;58(4):656-671. doi:10.1093/rheumatology/key322
- Morishita KA, Wagner-Weiner L, Yen EY, et al; Childhood Arthritis and Rheumatology Research Alliance (CARRA) Antineutrophil Cytoplasmic Antibody-Associated Vasculitis Workgroup. Consensus treatment plans for severe pediatric antineutrophil cytoplasmic antibody-associated vasculitis. *Arthritis Care Res (Hoboken).* 2022;74(9):1550-1558. doi:10.1002/acr.24590
- Robson JC, Grayson PC, Ponte C, et al; DCVAS Investigators. 2022 American College of Rheumatology/European Alliance of Associations for Rheumatology classification criteria for granulomatosis with polyangiitis. *Ann Rheum Dis.* 2022;81(3):315-320. doi:10.1136/annrheumdis-2021-221794

Content Domain

- Rheumatology, Vasculitis

Learning Objectives

- Recognize the presentation of pediatric vasculitis
- Summarize common initial treatments for vasculitis

The correct answer is: intravenous cyclophosphamide

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Question 3

Correct

1.00 points out of 1.00

Comment

A 9-month-old infant with trisomy 21 presented to an outside facility with respiratory failure due to viral bronchiolitis and was subsequently intubated on a third attempt prior to transport. The child has been intubated and mechanically ventilated for 6 days with a cuffed oral endotracheal tube. The child has been receiving a continuous infusion of dexmedetomidine at 1 µg/kg/min with intermittent dosing of fentanyl and midazolam. Ventilator support has been weaned, and the patient is considered ready for extubation in the next 12 to 24 hours.

Of the following, the BEST next step is

- A. assess for air leak ✓
- B. determine state behavioral score
- C. evaluate effectiveness of cough and ability to handle oral secretions
- D. transition to spontaneous mode of ventilation

Your answer is correct.

PREP Pearl(s)

- The development of clinical practice guidelines offers a conceptual framework with evidence-based recommendations, delineating best practices for the liberation of pediatric patients from mechanical ventilation.
- Assessment of air leak should be the first test performed in children with a cuffed endotracheal tube as part of the extubation-readiness test bundle to evaluate the risk for postextubation upper airway obstruction 12 to 24 hours prior to extubation-readiness testing.
- Extubation-readiness testing occurs after an air leak test; the readiness testing includes assessment of sedation and neurologic parameters, and should occur before performing a spontaneous breathing trial.

Critique

Liberating pediatric patients from mechanical ventilation presents a complex and multifaceted challenge due to the delicate balance required between ensuring adequate respiratory support and avoiding the risks associated with prolonged ventilation. Factors such as the variability in underlying conditions, the risk of respiratory muscle fatigue, and the potential for adverse events (eg, reintubation or respiratory failure) add to the intricacy of the weaning process. Historical data demonstrated that extubation-readiness tests (ERTs) were helpful to accurately predict successful extubation in children. One such study by Faustino 2017 demonstrated that in children with acute respiratory failure from lower respiratory tract disease, those that passed the first ERT had a 92% positive predictive value (PPV) of remaining extubated. Recently, Abu-Sultaneh 2023 embarked on creating the first international pediatric-specific ventilator liberation clinical practice guidelines, targeting acutely hospitalized children on invasive mechanical ventilation for more than 24 hours. These guidelines utilized the GRADE methodological approach (ie, grading of recommendations, assessment,

development, and evaluation) to generate 15 recommendations. Among the recommendations, the authors state that the first step for a patient who may be extubated in the next 12 to 24 hours is evaluation for presence of an air leak. The ERT recommends deflation of the cuffed endotracheal tube (ETT) as part of the assessment for risk for the development of postextubation upper airway obstruction (UAO). They further recommend administering corticosteroids 6 hours before extubation if the air leak pressure was >25 cm H_2O or those at high risk for UAO. Risk factors for UAO identified by the authors include multiple intubation attempts, traumatic intubation, use of large-for-age endotracheal tube, air leak pressure >25 cm H_2O for cuffed ETTs, and anatomic anomaly of the UAO airways. This recommendation is based on prior data which demonstrated that for children with cuffed ETTs, the presence of an air leak at the time of extubation was not correlated with extubation failure. However, these data did show that the absence of an air leak did have some diagnostic accuracy in predicting postextubation UAO airway obstruction. The guidelines note that the air leak test is unreliable for children with uncuffed ETTs. Of note, these recommendations were based on very low certainty of evidence, and the authors state that given the low sensitivity of the test, identifying patients who do not have an air leak could result in a delay in extubation to administer dexamethasone, which may prolong duration of mechanical ventilation.

The patient in the vignette has several risk factors for UAO airway obstruction; given the guidelines, the best next step in this case would be assessment of air leak. Evaluating the effectiveness of cough and ability to handle oral secretions, or determining the state behavioral score (SBS) would not be the best initial step, since they follow the initial assessment for an air leak. These components are evaluated after the air leak assessment, falling within the domain of the ERT safety screening. During this phase, clinicians assess several variables that may compromise extubation. These include the level of sedation using a validated tool, such as the SBS, the Richmond agitation-sedation scale, or the need for any potential invasive procedures. Physiologic parameters assessed include gas exchange, hemodynamic stability, and neurological function, including assessments of cough effectiveness and the patient's ability to manage oropharyngeal secretions. Lastly, if patients pass ERT safety screening, they are transitioned to a spontaneous mode of ventilation following initial assessment for air leak. The spontaneous breathing trial may be performed with pressure support or continuous positive airway pressure alone for 30 to 120 minutes based on patient-unique variables. During this phase of ERT, a measurement of maximal inspiratory pressure should be used during airway occlusion as an element of the ERT bundle for critically ill children at risk for muscle weakness, or at risk for extubation failure. In addition, cough, and oropharyngeal secretions are assessed throughout the trial.

Suggested Reading(s)

- Abu-Sultaneh S, Iyer NP, Fernández A, et al. Executive summary: international clinical practice guidelines for pediatric ventilator liberation: a Pediatric Acute Lung Injury and Sepsis Investigators (PALISI) Network Document. *Am J Respir Crit Care Med*. 2023;207(1):17-28. doi:10.1164/rccm.202204-0795SO
- Faustino EV, Gedeit R, Schwarz AJ, Asaro LA, Wypij D, Curley M. Randomized evaluation of sedation titration for respiratory failure study investigators: accuracy of an extubation readiness test in predicting successful extubation in children with acute respiratory failure from lower respiratory tract disease. *Crit Care Med.* 2017;45(1):94-102. doi:10.1097/ccm.0000000000002024
- Khemani RG, Sekayan T, Hotz J, et al. Risk factors for pediatric extubation failure: the importance of respiratory muscle strength. *Crit Care Med.* 2017;45(8):e798-e805. doi:10.1097/ccm.0000000000002433
- Randolph AG, Wypij D, Venkataraman ST, et al; Pediatric Acute Lung Injury and Sepsis Investigators (PALISI) Network. Effect of mechanical ventilator weaning protocols on respiratory outcomes in infants and children: a randomized controlled trial. *JAMA*. 2002;288(20):2561-8. doi:10.1001/jama.288.20.2561

Content Domain

- Critical Care, Medical Procedures (advanced)

Learning Objectives

- Develop an understanding of the sequential steps in extubation-readiness testing
- Apply pediatric ventilator liberation guidelines to identify appropriate timing and interventions
- Recognize the role of air leak testing in predicting the risk of postextubation upper airway obstruction as part of the extubation-readiness test bundle in pediatric patients with a cuffed endotracheal tube

The correct answer is: assess for air leak

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Question 4

Correct

1.00 points out of 1.00

Comment

A previously healthy 18-month-old infant is brought to the emergency department via emergency medical services after being found at home drooling, not moving her extremities, and gasping for breath. She was intubated in the field prior to arrival, for which she received ketamine 2 mg/kg and rocuronium 1 mg/kg. A noncontrast head computed tomography scan was negative for intracranial hemorrhage, skull fracture, or midline shift.

Her physical examination approximately 1 hour after intubation is n FiO_2.otable for pinpoint pupils and flaccid paralysis of her upper and lower extremities. She has copious endotracheal tube secretions. There are no signs of trauma. Vital signs are a temperature of 37.5 °C, heart rate of 110 beats/min, respiratory rate of 20 breaths/min (mechanically ventilated), blood pressure of 80/45 mm Hg, oxygen saturation of 99% on 0.3 FiO_2.

Family history reveals that there are multiple over-the-counter medications in the home, and the patient's older sibling takes 2 medications for attention-deficit/hyperactivity disorder. The family has a lawn-care business and a small farm.

Of the following, the MOST likely type of substance ingested by the child is a(an)

- A. anticholinergic
- B. insecticide ✓
- C. psychostimulant
- D. rodenticide

Your answer is correct.

PREP Pearl(s)

- Organophosphates result in cholinergic excess and cause a toxidrome of muscarinic and nicotinic receptor-mediated symptoms.
- After decontamination and stabilization of airway, breathing, and circulation, antidote therapy for organophosphate poisoning should begin with atropine and then pralidoxime.
- Effects of cholinergic toxicity can be remembered using the mnemonic "DUMBELS": diarrhea/defecation, urination, miosis, bronchorrhea/bronchospasm, emesis, lacrimation, salivation as well as "SLUDGE/BBB": salivation, lacrimation, urination, defecation, gastric emesis, bronchorrhea, bronchospasm, bradycardia.

Critique

The patient in the vignette is likely suffering from organophosphate toxicity. Organophosphates are found in insecticides and pesticides as well as chemical agents used in warfare ("nerve agents.") Exposure to these agents may be transdermal, inhalational, or via ingestion. Examples of organophosphate pesticides/insecticides include: acephate, bensulide, chlorpyrifos, diazinon, dimethoate, ethophoros,

malathion, phorate, and phosmet. These are used throughout the world for agricultural use and on many crops, ornamental lawns, and plants. Children may be exposed by ingestion of these agents, and even relatively small amounts found as residue on foods such as greens, herbs, and fruits may be toxic. Organophosphate poisoning in children may be misdiagnosed as infectious diseases such as polio or botulism, post-infectious syndromes such as Guillain-Barré syndrome, or transverse myelitis; however, it can be distinguished from these by the acute nature of the presentation and presence of miosis and other muscarinic symptoms.

Organophosphates stimulate cholinergic excess in the parasympathetic nervous system. In the normal physiologic state, the neurotransmitter acetylcholine is degraded by the enzyme acetylcholinesterase, preventing excess accumulation of acetylcholine in plasma, red blood cells, and the neuromuscular junction. Organophosphate compounds covalently bind to and phosphorylate acetylcholinesterase, rendering it inactive and causing excess accumulation of acetylcholine. These include muscarinic and nicotinic receptor effects in the parasympathetic nervous system.

Muscarinic acetylcholine receptors are widely distributed throughout the central nervous system. There are 5 subtypes: M1 (cerebral cortex, stomach, salivary glands), M2 (smooth muscle and heart), M3 (smooth muscle, stomach, salivary glands), M4 and M5 (both in hippocampus and substantia nigra). Muscarinic receptors are postganglionic, and stimulation of muscarinic receptors causes acute cholinergic toxicity. These effects can be remembered using the mnemonic “DUMBELS”: diarrhea/defecation, urination, miosis, bronchorrhea/bronchospasm, emesis, lacrimation, salivation as well as “SLUDGE/BBB”: salivation, lacrimation, urination, defecation, gastric emesis, bronchorrhea, bronchospasm, bradycardia.

However, neither of these mnemonics describe the central nervous system effects of organophosphates which are due to nicotinic receptor stimulation by acetylcholine: fasciculations, muscle weakness and/or paralysis. In addition, there are also nicotinic and muscarinic receptors in the brain, and stimulation of these receptors with excess acetylcholine may cause central apnea or respiratory depression, lethargy, seizures, or loss of consciousness.

Initial therapy for organophosphate toxicity focuses on airway protection and resuscitation. Decontamination of the patient and protection of healthcare workers from contamination should also occur. The initial antidote therapy for organophosphate poisoning is atropine. Atropine is a selective muscarinic receptor blocker and reverses the muscarinic effects only. High doses of atropine (0.05 mg/kg) may be required to fully compete at the muscarinic receptors. Due to atropine’s short half-life, a continuous infusion is often required to maintain the antidote effect. Atropine is given first followed by pralidoxime.

For reversal of nicotinic effects of organophosphate poisoning, pralidoxime is given. Pralidoxime reactivates cholinesterase by displacing the enzyme from its receptor sites and removing the phosphoryl group from the active site of the inactivated cholinesterase enzyme. The loading dose for children is 20 to 50 mg/kg intravenously with a maximum dose of 2000 mg per dose. Continuous infusion dosing is 10 to 20 mg/kg/hr, not to exceed 500 mg/hr. It is crucial to begin treatment with pralidoxime as soon as possible. After a period of time, the bond between organophosphate substances and acetylcholinesterase undergoes an “aging” process, rendering the patient refractory to pralidoxime therapy and significantly prolonging recovery.

Anticholinergic is incorrect because it would cause anticholinergic symptoms, such as dry mucous membranes, dry flushed skin, mydriasis, and urinary retention rather than the opposite symptoms seen in cholinergic toxicity. Rodenticide is incorrect because these substances are typically anticoagulants such as warfarin. Symptoms consistent with ingesting anticoagulant rodenticides include bruising, petechiae, and bleeding (eg, hematuria, epistaxis). A psychostimulant is incorrect because this overdose would cause tachycardia, agitation and possibly mydriasis.

Suggested Reading(s)

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- Dodson J, Feng S, Rodriguez D. That's not his regular formula. *Pediatr Emerg Care.* 2021;37(4):e206-e209. doi:10.1097/pec.0000000000001836
- Howland MA. Antidotes in depth: pralidoxime. In: Nelson LS, Howland MA, Lewin NA, Smith SW. Goldfrank, Hoffman RS, eds. *Goldfrank's Toxicologic Emergencies*, 11th ed. McGraw-Hill; 2018:1503-1507.
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Content Domain

- Critical Care, Toxins

Learning Objectives

- Recognize the clinical presentation of organophosphate exposure

The correct answer is: insecticide

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Question 5

Correct

1.00 points out of 1.00

Comment

A previously healthy 17-year-old lacrosse player weighing 90 kg collapsed while playing in a tournament. Emergency medical services found them flushed, febrile, confused, and with a weak pulse. The adolescent is given 3 L normal saline en route to the hospital; they remain confused and unable to correctly answer questions. Vital signs on arrival are a temperature of 40 °C, heart rate of 124 beats/min, respiratory rate of 28 breaths/min, blood pressure of 115/68 mm Hg, and 99% SPO_2 on ambient air. A urine sample collected for urine toxicology is negative but noted to be dark brown.

Of the following, the BEST next step is (are)

- A. cooling measures ✓
- B. dantrolene
- C. mannitol
- D. midazolam

Your answer is correct.

PREP Pearl(s)

- Immediate cooling to 39 °C or lower is necessary to help prevent further organ damage in patients presenting with heat stroke.
- Hypocalcemia, hyperphosphatemia, and hyperkalemia are common electrolyte abnormalities in rhabdomyolysis.
- Early aggressive treatment decreases risk of renal failure and subsequent need for dialysis.

Critique

Exertional heat stroke is a medical emergency that requires immediate treatment to prevent organ injury and death. Immediate cooling measures are necessary to bring the core body rectal temperature down to < 39 °C, making cooling measures the best next step. The discolored urine in this vignette suggests myoglobinuria. Rapid cooling will decrease muscle breakdown and resultant rhabdomyolysis. At outdoor festivals and sporting events held on hot summer days, the best way to accomplish this is to have tubs with ice water available to immerse the overheated patient. The National Athletic Trainers Association have recommended that ice tubs or similar cooling measures be available during intense pre-season training sessions to rapidly lower the core body temperature of any athlete showing signs of heat exhaustion.

Rhabdomyolysis can result from both strenuous exercise and exertional heat stroke, both of which can result in muscle injury and necrosis. While the patient is being cooled via whatever measures are available, serial electrolyte, renal function panel, creatine kinase, and glucose levels need to be followed. The patient in the vignette received adequate initial resuscitation but will need ongoing fluid replacement to ensure adequate perfusion. Electrolyte abnormalities such as hypocalcemia, hyperphosphatemia, and hyperkalemia can occur in rhabdomyolysis. Hyperkalemia can be difficult to manage without hemodialysis if renal function is

compromised by myoglobinuria. Hypoglycemia is a common finding in rhabdomyolysis. Hepatic function can be impaired in up to 25% of severe cases with the release of intracellular myoglobin causing direct hepatic injury. These patients may need continuous infusions of dextrose to prevent hypoglycemia.

Methamphetamine, 3,4-methylenedioxymethamphetamine (MDMA), and cocaine intoxication can all result in sympathomimetic syndrome where the patient presents hot and confused, much like the patient above. These patients also need immediate cooling if their core temperature is elevated to prevent rhabdomyolysis and organ injury. No antidotes for these medications exist, but midazolam or other benzodiazepines are titrated as needed to decrease agitation and/or seizures resulting from intoxication.

Dantrolene is the treatment of choice in malignant hyperthermia triggered by anesthetic agents or succinylcholine. Dantrolene binds the ryanodine receptor in skeletal muscle and decreases the intracellular calcium concentration. These receptors modulate the release of calcium from sarcoplasmic reticulum which is part of the muscle contraction cycle. As a result, skeletal muscle contraction and the heat generated by this contraction are reduced. Although dantrolene is used for malignant hyperthermia, its use to treat rhabdomyolysis from other causes has not been supported by clinical evidence.

Mannitol is frequently used in rhabdomyolysis caused by crush injuries. The recommendation in these patients is to give sufficient saline to maintain urine output of at least 20 mL/hr followed by administration of mannitol. The use of mannitol in these patients has decreased the number of patients progressing to renal failure and subsequent need for dialysis. In patients with rhabdomyolysis from exertion or heat stroke, it has not been shown to provide any additional benefit.

Suggested Reading(s)

- Kruijt N, van den Bersselaar LR, Hopman MTE, et al. Exertional heat stroke and rhabdomyolysis: a medical record review and patient perspective on management and long-term symptoms. *Sports Med Open*. 2023;9(1):33. doi:10.1186/s40798-023-00570-y
- Monseau AJ, Hurlburt GA, Balcik BJ, Oppenlander KE, Chill NM, Martin PS. Status of US emergency medical service protocols regarding pre-transfer cooling for exertional heat stroke. *Cureus.* 2021;13(11):e19505. doi:10.7759/cureus.19505

Content Domain

- Sports Medicine, serious injury

Learning Objectives

- Recognize that rhabdomyolysis can cause significant organ injury if not promptly recognized and treated.

The correct answer is: cooling measures

View Peer Results

Question 6

Correct

1.00 points out of 1.00

Comment

A previously healthy 4-year-old child is brought to the emergency department by emergency medical services (EMS). The parents report that the child was playing when he slumped over and made jerking movements in his arms and legs. On arrival to the house, EMS personnel noted the presence of tonic-clonic seizures. They place a peripheral intravenous (IV) line and administer IV lorazepam 0.1 mg/kg, and give supplemental oxygen through a face mask. Parents had been supervising the child; they report no trauma or ingestion.

In the emergency department, the child continues to have tonic-clonic seizures. Vital signs are a temperature of 39.1 °C, heart rate of 164 beats/min, blood pressure of 104/64 mm Hg, respiratory rate of 22 breaths/min, and oxygen saturation of 95% on supplemental oxygen by face mask. He is given a second dose of IV lorazepam 0.1 mg/kg, followed by IV levetiracetam 60 mg/kg for continued seizures. He is intubated for airway protection and a head computed tomography scan demonstrates no evidence of trauma or intracranial hemorrhage. A dose of IV fosphenytoin is given, but despite these interventions the child continues to seize.

Laboratory test results are shown:

Laboratory Test	Result
Sodium	142 mEq/L (142 mmol/L)
Potassium	3.8 mEq/L (3.8 mmol/L)
Chloride	110 mEq/L (110 mmol/L)
Urea nitrogen	12 mg/dL (428 mmol/L)
Creatinine	0.3 mg/dL (26.5 µmol/L)
Glucose	120 mg/dL (6.6 mmol/L)
Calcium	9.0 mg/dL (2.25 mmol/L)
White blood cell count	6,400/µL (6.4 x 10^9/L)
Hemoglobin	11.5 g/dL (115 g/L)
Platelet count	164 x 10^3/µL (164 x 10^9/L)

Of the following, the BEST next step in management of this patient would include

A. intravenous methylprednisolone

B. midazolam infusion ✓

C. repeat levetiracetam

D. valproic acid administration

Your answer is correct.

PREP Pearl(s)

- Status epilepticus is defined as a single seizure lasting more than 5 minutes or a cluster of seizures lasting longer than 5 minutes without return of consciousness between seizures.
- First-line treatment for status epilepticus involves administration of benzodiazepines (eg, midazolam, diazepam, lorazepam).
- Second-line treatment for status epilepticus include administration of levetiracetam, fosphenytoin, or valproic acid. Studies in pediatrics demonstrate equivalent efficacy of these agents.

Critique

The patient in this scenario has status epilepticus. Status epilepticus is defined as a single seizure lasting more than 5 minutes or a cluster of seizures lasting longer than 5 minutes without return of consciousness between seizures. Seizures lasting more than 5 minutes are unlikely to stop without medication administration.

The table depicts a supported treatment strategy for status epilepticus (Table). First-line treatment for status epilepticus involves administration of benzodiazepines (eg, midazolam, diazepam, lorazepam). A benzodiazepine dose can be repeated if the seizure does not stop in 5 minutes. Second-line agents should be considered if seizures continue following benzodiazepine administration, and ideally be given within 20 minutes of seizure onset. Studies have shown levetiracetam, fosphenytoin, and valproic acid to be equivalent in efficacy as second-line anticonvulsant agents. Levetiracetam is often utilized due to its low side-effect profile. Phenobarbital has also been utilized as a second line agent, most often in infants. Third-line agents for continued seizure include continuous infusions of midazolam, pentobarbital, or ketamine. Refractory status epilepticus may require therapies such as a magnesium sulfate infusion, ketogenic diet, additional antiseizure medications, immunomodulatory therapies (eg, methylprednisolone, intravenous immunoglobulin, plasma exchange) or inhaled anesthetics.

Medical management of seizures should focus on the airway, breathing, and circulation. Airway patency and oxygen saturation should be assessed. Intubation may be required for respiratory depression due to seizures or sedative effects of medications utilized for seizure management. Blood pressure may require support with fluid resuscitation or vasoactive medications. Electrolytes should be monitored, and hyponatremia, hypoglycemia, and hypocalcemia should be corrected. A thorough history should be obtained. Seizures may be triggered by trauma, toxic ingestions or envenomations, infection, metabolic, or autoimmune causes.

The patient in this scenario has failed treatment with first-and second-line therapies including lorazepam, levetiracetam and fosphenytoin. Therefore, the correct answer is a third-line therapy: midazolam infusion. A magnesium sulfate infusion or use of methylprednisolone or a ketogenic diet can be considered in some cases of refractory status epilepticus, but should not be utilized before initiating a third-line therapy.

Suggested Reading(s)

- Chamberlain JM, Kapur J, Shinnar S, et al; Pediatric Emergency Care Applied Research Network Investigators. Efficacy of levetiracetam, fosphenytoin, and valproate for established status epilepticus

by age group (ESETT): a double-blind, responsive-adaptive, randomised controlled trial. *Lancet.* 2020;395(10231):1217-1224. doi:10.1016/S0140-6736(20)30611-5

- Freedman DA, Roach ES. Status Epilepticus. *Pediatr Rev.* 2023;44(7):383-392. doi:10.1542/pir.2022-005632
- Glauser T, Shinnar S, Gloss D, et al. Evidence-based guideline: treatment of convulsive status epilepticus in children and adults: report of the guideline committee of the American Epilepsy Society. *Epilepsy Curr.* 2016;16(1):48-61. doi:10.5698/1535-7597-16.1.48
- Chamberlain JM, Kapur J, Shinnar S, et al; Pediatric Emergency Care Applied Research Network Investigators. Efficacy of levetiracetam, fosphenytoin, and valproate for established status epilepticus by age group (ESETT): a double-blind, responsive-adaptive, randomised controlled trial. *Lancet.* 2020;395(10231):1217-1224. doi:10.1016/S0140-6736(20)30611-5

Content Domain

- Critical Care, Neurological Disorders and Support

Learning Objectives

- Identify the treatment strategies for status epilepticus.

The correct answer is: midazolam infusion

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