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Feedback	If you have satisfied the completion requirements for this assessment, you may proceed to claim your CME credit via the AAP Transcript tool.

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Question 1

Correct

1.00 points out of 1.00

Comment

A 10-year-old child with septic shock, acute respiratory distress syndrome, and fluid overload is receiving continuous venovenous hemodiafiltration (CVVHDF) through a double-lumen 10 French catheter placed in the right internal jugular vein, with a blood-flow rate of 200 ml/min. They are receiving regional citrate anticoagulation with post-filter ionized calcium levels maintained between 0.25 and 0.35 mmol/L. After initial improvement, the child develops worsening hypotension, increasing liver dysfunction, and a rise in lactate to 6 mmol/L. There is an increased anion gap and hypercalcemia (serum calcium 16 mg/dL) with normal systemic ionized calcium (1.2 mmol/L [4.81 mg/dL]). The calcium infusion rate has been increased to maintain normal systemic ionized calcium.

Of the following, the BEST next step in adjusting CVVHDF therapy in this patient is

- A. decrease the blood-flow rate ✓
- B. decrease the dialysate rate
- C. increase the citrate-infusion rate
- D. stop CVVHDF

Your answer is correct.

PREP Pearl(s)

- Regional citrate anticoagulation requires close monitoring of post-filter ionized calcium, systemic total and ionized calcium, and calculation of total ÷ ionized calcium.
- Citrate accumulation is suspected with an increase in calcium administration, elevated anion gap, increase in total calcium, and a total ÷ ionized calcium ratio > 2.5.
- Citrate accumulation is treated by reducing blood-flow rate, increasing dialysate or filtration rate, and decreasing citrate-infusion rate.

Critique

The child described in the vignette has severe liver dysfunction and lactic acidosis, suggesting limited capacity to metabolize citrate and a predisposition to developing citrate accumulation. Hypercalcemia with low ionized calcium and an elevated total:ionized calcium level (16 mg/dL ÷ by 4.81 mg/dL = 3.33) supports this. When citrate accumulation is suspected, the net citrate load must rapidly be decreased by any of the following:

- decreasing the blood-flow rate and corresponding citrate-infusion rate, thereby reducing the amount of citrate administered
- increasing the dialysate rate (in continuous venovenous hemodialysis) or the filtration rate (in continuous venovenous hemofiltration), thereby increasing the removal rate
- reducing the targeted citrate concentration within the filter by adjusting the targeted post-filter ionized calcium levels

- changing to another form of anticoagulation

Continuous renal replacement therapy should be continued to enable clearance of citrate-calcium complexes. Decreasing the blood-flow rate is the next best step for the patient in the vignette; reducing the blood-flow rate is coupled with decreasing the citrate-infusion rate. Decreasing the dialysate rate will result in reduced citrate clearance. Increasing the citrate-infusion rate will increase the citrate load worsening citrate accumulation. Stopping continuous venovenous hemodiafiltration will lead to reduced clearance of calcium citrate complexes.

Various anticoagulants have been used in continuous renal replacement therapy, with heparin, citrate, and prostacyclin being most common. Regional citrate anticoagulation has an advantage over systemic anticoagulation as it prolongs filter-life without the risk of bleeding.

Citrate, an organic acid, is used as an anticoagulant as either acid citrate dextrose or trisodium citrate. It has a high affinity for the divalent calcium ion and, when added to blood, results in the formation of citrate-calcium complexes, reducing the level of ionized free calcium. Calcium is required as a cofactor for enzymes of the coagulation cascade and citrate-mediated chelation of calcium leads to low ionized calcium levels resulting in effective anticoagulation.

There are various protocols used for regional citrate anticoagulation. All protocols require pre-filter administration of citrate. The dose of citrate infusion is coupled with the blood-flow rate. Optimal regional anticoagulation occurs when the circuit (or post-filter) ionized calcium is less than 0.35 mmol/L which corresponds to a citrate level of 3 to 4 mmol/L of blood. Regional anticoagulation is adjusted by titrating the citrate-infusion rate to maintain low post-filter ionized calcium levels or adjusting the citrate and blood-flow rate to achieve a circuit citrate concentration of 3 to 4 mmol/L. Some of the citrate-calcium complexes are filtered, and the rest enters the systemic circulation where citrate is metabolized to bicarbonate. To prevent systemic hypocalcemia, calcium is infused into the systemic circulation and the calcium infusion rate is titrated to maintain ionized calcium within normal range. Post-filter, systemic, and total calcium levels (with total ÷ ionized ratio calculation) are monitored at regular intervals.

Citrate accumulation can occur because of excessive citrate administration, low clearance in the hemofilter, or saturation of citrate clearance by the body (ie, “citrate lock”). Patients with acute liver failure or acute-on-chronic liver failure have decreased citrate metabolizing capacity. Patients with circulatory shock have decreased oxygen delivery to cells resulting in reduced citrate metabolizing capacity. Citrate accumulation is suspected when the total ÷ ionized calcium ratio is elevated (with a value > 2.5 indicative of significant accumulation); an increase in this ratio demonstrates an increase in anion-bound calcium that is seen with circulating citrate-calcium complexes. An increase in the rate of systemic calcium infusion may also be seen; this occurs due to the reduction or absence of calcium released from citrate-calcium complexes. High anion gap metabolic acidosis and increased lactate may occur. This is likely due to the process impairing the tricarboxylic acid cycle and reducing citrate metabolism, thereby limiting pyruvate metabolism and increasing lactate generation; it is not directly related to citrate accumulation. The accumulation of citrate-calcium complexes contributes to the elevated anion gap.

Suggested Reading(s)

- Brophy P, Khan I, Deep A. Anticoagulation in CRRT. In: Deep A, Goldstein SL, eds. *Critical Care Nephrology and Renal Replacement Therapy in Children.* Springer, 2018: 251-269.
- John JC, Taha S, Bunchman TE. Basics of continuous renal replacement therapy in pediatrics. *Kidney Res Clin Pract.* 2019;38(4):455-461. doi:10.23876/j.krcp.19.060
- Schneider AG, Journois DR. Complications of regional citrate anticoagulation: accumulation or overload? Crit Care. 2017;21(281). doi:10.1186/s13054-017-1880-1

Content Domain

- Critical Care, medical procedures (advanced)

Learning Objectives

- Identify and manage problems associated with continuous renal replacement therapy

The correct answer is: decrease the blood-flow rate

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Question 2

Correct

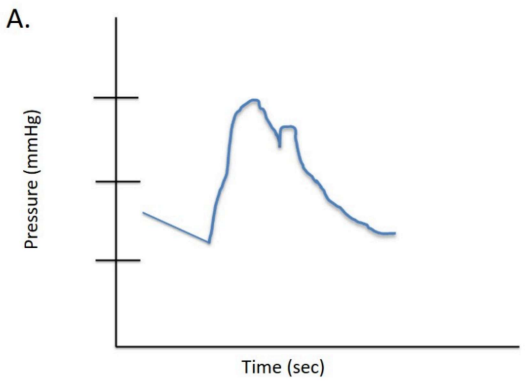
1.00 points out of 1.00

Comment

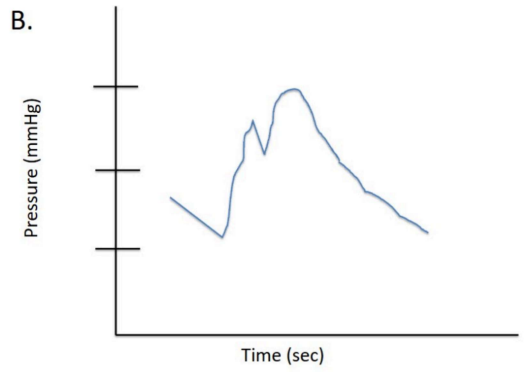
A 17-year-old adolescent is admitted to the pediatric intensive care unit with fever, rash, and emesis. On admission, her vital signs and laboratory findings are concerning for toxic shock syndrome. She is intubated due to altered mental status, and both a right internal jugular central venous line and right radial arterial line are placed. She is started on norepinephrine for persistent hypotension.

Of the following, the waveform MOST likely to be observed in the patient's arterial line tracing, if this medication is discontinued prematurely, is

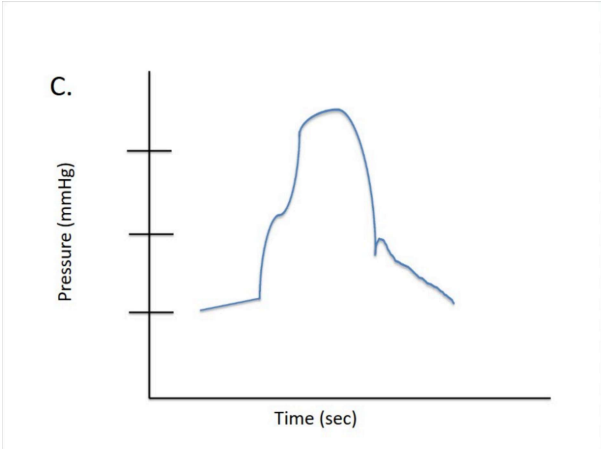
A. Response Choice A. waveform A.



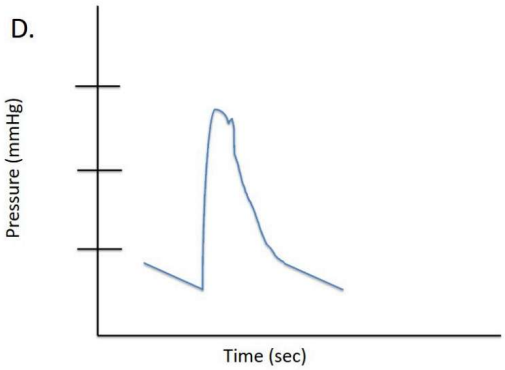
B. Response Choice B. waveform B.



C. Response Choice C. waveform C.



D. Response Choice D. waveform D.



Your answer is correct.

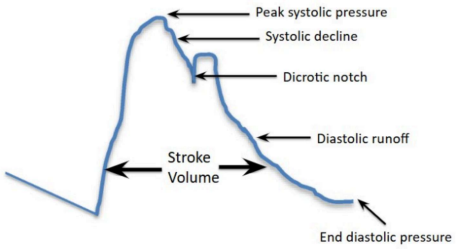
PREP Pearl(s)

- Toxic shock syndrome is often characterized by low systemic vascular resistance due to toxin-mediated cytokine release causing capillary leak and vasodilatation.
- Arterial line waveforms provide important information both on ventricular contractility and systemic vascular resistance.
- Low systemic vascular resistance on an arterial waveform can be observed by a steep diastolic downstroke and a low dicrotic notch.

Critique

Arterial cannulation is a routine and important procedure in the pediatric intensive care unit. Indications for arterial cannulation include the need for continuous blood pressure monitoring, frequent laboratory checks, as well as need for arterial blood sampling (as opposed to venous or capillary). The ability to obtain an arterial waveform is another advantage of arterial cannulation. A classic radial arterial waveform is shown:

Figure. Classic radial arterial waveform.



Courtesy of M. Rowin

A steep systolic upstroke indicates good contractility; the area under the systolic waveform is representative of stroke volume. Low systemic vascular resistance is represented by a steeper diastolic downstroke. The dicrotic notch represents the closing of the aortic valve.

The patient described in the vignette requires arterial pressure monitoring due to hypotension and need for vasoactive support. This patient has toxic shock syndrome. Toxic shock syndrome (TSS) is most commonly caused by either *Staphylococcus aureus* or *Streptococcus pyogenes.* Release of bacterial toxins causes the sequela of the disease; major toxins being TSST-1 and enterotoxins A, B, and C. These toxins cause release of proinflammatory cytokines leading to capillary leak, vasodilatation, and subsequent shock and multiorgan failure.

Norepinephrine is an α-agonist that increases systemic vascular resistance and was the vasoactive medication chosen for this patient. If norepinephrine was discontinued prematurely before the patient's vasodilatory shock resolved, the patient's systemic vascular resistance and stroke volume would likely decrease. This would be observed in the arterial pressure waveform as a decreased area under the systolic curve and a steeper diastolic downstroke (Waveform D).

Waveform A represents a normal finding. Waveform B shows an elevated diastolic component as might be seen in aortic stenosis. Waveform C shows an elevated systolic wave which would be seen in a hypertensive state.

Suggested Reading(s)

- Bronicki RA and Spenceley NC. Hemodynamic monitoring. In: Nichols DG, Shaffner DH, eds. *Rogers' Textbook of Pediatric Intensive Care.* 5th ed. Wolters Kluwer; 2016:1120-1136.
- Chambers D, Huang C, and Matthews G. Arterial pressure waveforms. In: *Basic Physiology for Anaesthetists*. 2nd ed. Cambridge University Press; 2019:155-157.
- Singhi SC, Jayashree M, Straumanis JP and Kotloff KL. Toxin-related diseases. In: Nichols DG, Shaffner DH, eds. *Rogers' Textbook of Pediatric Intensive Care*. 5th ed. Wolters Kluwer; 2016:1547-1571.

Content Domain

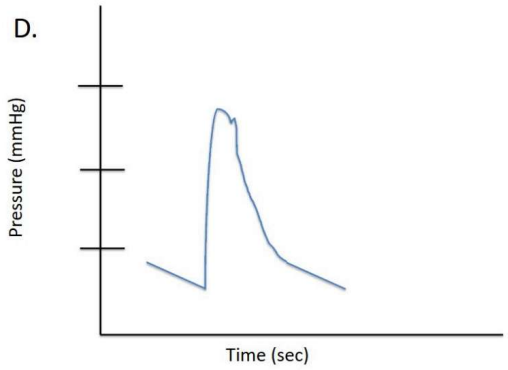
- Critical Care, Monitoring

Learning Objectives

- Demonstrate understanding of arterial pressure monitoring concepts as they relate to specific disease processes

The correct answer is:

Response Choice D. waveform D.



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Question 3

Correct

1.00 points out of 1.00

Comment

A 12-year-old child with systemic juvenile idiopathic arthritis is admitted to the intensive care unit with persistent fevers up to 40 °C for a week prior to admission. Initial blood pressure is 84/48 mm Hg. After fluid resuscitation, her blood pressure stabilizes to 105/65 mm Hg, but her heart rate remains 100 beats/min. On physical examination, she appears tired, and although she is oriented, she seems slow to respond to questions. The remainder of the examination is notable for hepatosplenomegaly and bilateral knee pain with mild swelling.

Laboratory test results are shown:

Laboratory Test	Result
White blood cell count	5,000/μL (0.5 x 10^9/L)
Hemoglobin	6.2 g/dL (62 g/L)
Platelet count	42 x 10^3/μL (42 x 10^9/L)
C-reactive protein	24 mg/L
Aspartate aminotransferase	89 U/L

Of the following, the laboratory test that would be MOST helpful in diagnosing her likely condition is

- A. anti–double-stranded DNA
- B. cystatin C
- C. ferritin ✓
- D. interleukin-12

Your answer is correct.

PREP Pearl(s)

- Macrophage activation syndrome is a rare but life-threatening complication of some rheumatologic diseases, such as systemic juvenile idiopathic arthritis.
- Early diagnosis and treatment of macrophage activation syndrome are crucial, necessitating a high index of suspicion.
- Hyperferritinemia is universal in all patients with hemophagocytic lymphohistiocytosis/macrophage activation syndrome with systemic disease.

Critique

The child in the vignette is experiencing macrophage activation syndrome (MAS), a serious and potentially life-threatening complication that can occur following certain viral infections, and which is often associated with immunologic and rheumatologic disorders. While most commonly associated with systemic juvenile idiopathic arthritis (sJIA), MAS has been reported in patients with systemic lupus erythematosus, juvenile dermatomyositis, and Kawasaki disease.

Macrophage activation syndrome is a hyperinflammatory condition related to hemophagocytic lymphohistiocytosis (HLH) with many overlapping features. It begins with the uncontrolled activation of T cells, as well as macrophages exhibiting phagocytic activity. Considered a secondary or acquired HLH, MAS occurs in the context of pre-existing disease, in contrast to primary HLH. While the pathogenesis of primary and secondary HLH may differ in some pathways, the end stage is an overwhelming release of pro-inflammatory mediators, such as interferon-ɣ, interleukin (IL)-1, IL-6, and tumor necrosis factor-α, which sometimes is referred to as the cytokine storm. Left untreated, MAS can lead swiftly to multiorgan dysfunction syndrome and death.

Diagnosing MAS can be challenging, as diagnostic criteria lack specificity, and clinical features can have significant overlap with other conditions, such as malignancy or infection. However, MAS should be suspected in patients with pre-existing rheumatologic/immunologic disease who present with unremitting fevers, cytopenias (at least 2 cell lines down), hepatosplenomegaly, hepatic dysfunction, and coagulopathies. Thrombotic microangiopathy is a common manifestation, as well as encephalopathy. Several criteria have been developed to identify patients with syndromes that fit either HLH/MAS, including HLH-2004, MAS-2016, and the HScore. Out of these, the MAS-2016 was developed specifically to classify MAS in patients with known or strongly suspected sJIA. The HLH-2004 guidelines have demonstrated low sensitivity for MAS in sJIA. The HScore was developed in adults with primarily malignancy or infection-associated HLH.

Ferritin is a sensitive test for both HLH/MAS, and there is broad consensus in the 2022 HLH/MAS task force that ferritin levels should be checked in all patients with new, ongoing, or high suspicion for MAS. Hyperferritinemia is a part of all existing HLH/MAS diagnostic criteria. In MAS-2016, a febrile patient with JIA is diagnosed with MAS if they have a serum ferritin level > 684 ng/mL, and at least two of the following criteria:

- Platelet counts < 181 x 10^9/L
- Aspartate aminotransferase > 48 U/L
- Triglyceride concentration > 156 mg/dL
- Fibrinogen concentration < 360 mg/dL

There are increasingly more specialized biomarkers available, although these generally require send out to reference laboratories and may not be immediately helpful or practical. These include measures for macrophages (CD163, neopterin), the interferon-ɣ pathway (IFN-ɣ CXCL9), activation of T cells (soluble IL-2 receptors-α/CD25), and inflammasomes (IL-18).

Glucocorticoids remain the mainstay of therapy for MAS, although anakinra (a recombinant IL-1 receptor antagonist) has also entered the current therapeutic protocols for treatment. Other alternative agents include etoposide, cyclosporine, ruxolitinib (JAK inhibitor pathway), emapalumab (IFN-ɣ neutralization), and rituximab (B-cell depletion).

A ferritin level is required for the diagnosis of the patient in the vignette. Anti–double-stranded DNA is used in the diagnosis of systemic lupus erythematosus, and IL-12 is not a laboratory value checked in MAS associated with sJIA. Cystatin C is a marker used to assess renal function.

Suggested Reading(s)

- Alongi A, Naddei R, De Miglio L, Natoli V, Ravelli A. Macrophage activation syndrome in pediatrics. *Pediatr Allergy Immunol*. 2020; 31(suppl_24): 13–15. doi:10.1111/pai.13158
- Crayne C, Cron RQ. Pediatric macrophage activation syndrome, recognizing the tip of the Iceberg. *Eur J Rheumatol.* 2020;7(suppl_1):S13-S20. doi:10.5152/eurjrheum.2019.19150
- Shakoory B, Geerlinks A, Wilejto M HLH/MAS task force*, et al*The 2022 EULAR/ACR points to consider at the early stages of diagnosis and management of suspected haemophagocytic lymphohistiocytosis/macrophage activation syndrome (HLH/MAS). *Ann Rheum Dis.* 2023;82:1271-1285. doi:10.1136/ard-2023-224123

Content Domain

- Infectious Diseases, Inflammatory diseases

Learning Objectives

- Recognize macrophage activation syndrome as a complication of childhood rheumatologic disease.
- Discuss the diagnostic criteria for macrophage activation syndrome.

The correct answer is: ferritin

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Question 4

Correct

1.00 points out of 1.00

Comment

A 9-year-old child is brought to the emergency department with status epilepticus. His seizure activity terminates with administration of intravenous lorazepam and levetiracetam. Evaluation includes a normal electrolyte profile, negative urine drug screen, normal lactate level, and normal blood gas analysis. His parents report that 6 months ago, he had an event where he developed difficulty walking and was evaluated by a local neurologist. Significant magnetic resonance imaging (MRI) findings from that event are shown (**Figure 1**). The patient gradually improved and was back to his neurologic baseline at the time of today's new onset of seizure activity. He is admitted to the pediatric intensive care unit, where another MRI is obtained (**Figure 2**).

Figure 1. Magnetic resonance imaging of brain with FLAIR imaging. Yellow arrow highlights area of interest.

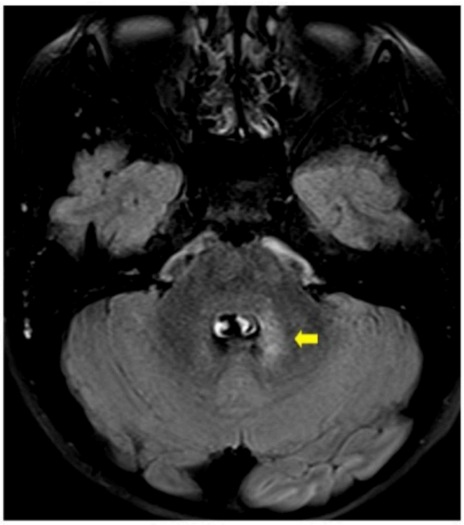
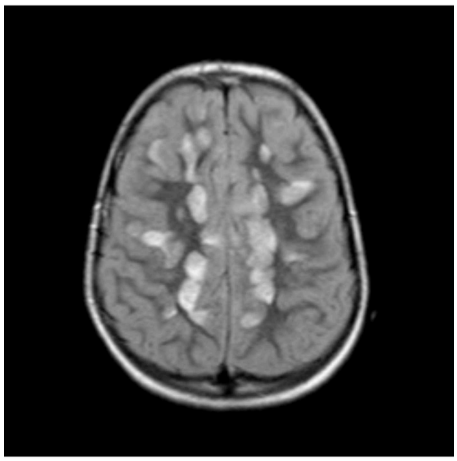


Figure 2. Magnetic resonance imaging of brain with FLAIR imaging.



Courtesy of M. Rowin

Of the following, the antibodies to central nervous system proteins MOST likely to be present are

A. ɣ-aminobutyric acid receptor type A

B. glutamic acid decarboxylase

C. myelin oligodendrocyte glycoprotein

D. N-methyl-D-aspartate receptor

Your answer is correct.

PREP Pearl(s)

- Acute disseminated encephalomyelitis is a postinfectious autoimmune encephalitis most commonly seen in pediatric patients.
- The demyelinating lesions seen on magnetic resonance imaging in acute disseminated encephalomyelitis patients can mimic those seen in multiple sclerosis.
- Antibodies to myelin oligodendrocyte glycoprotein are found in a significant portion of patients with acute disseminated encephalomyelitis, especially recurrent acute disseminated encephalomyelitis.

Critique

Autoimmune encephalitis is increasingly recognized in the pediatric population. Acute disseminated encephalomyelitis (ADEM) is an immune-mediated demyelinating central nervous system (CNS) disorder characterized by new-onset neurologic symptoms coupled with neuroimaging evidence of demyelination. A prodromal viral illness is identified in a majority of patients. Acute disseminated encephalomyelitis occurs most frequently in childhood, with a median age of onset of 6.5 years. The incidence of ADEM is estimated at 0.2-0.4/100,000 pediatric patients annually.

Neurologic symptoms of ADEM can begin as early as 1 to 2 weeks following a febrile, often viral, illness. Patients may experience headaches, malaise, vomiting, lethargy and low-grade fevers. Acute onset of encephalopathy with multifocal neurologic defects follows, typically within 2 to 5 days. Patients may exhibit ataxia, inability to walk, and slurred or decreased speech. Cranial neuropathies and abnormal reflexes (typically hyperreflexia) are common findings. Altered mental status, including agitation, delirium, somnolence, confusion and seizure activity is noted in up to 60% of patients.

The diagnosis of ADEM requires magnetic resonance imaging (MRI). Abnormalities are most frequently identified on T2-weighted and fluid-attenuated inversion recovery (FLAIR) sequences. Acute disseminated encephalomyelitis lesions on MRI are often multiple and asymmetric, but can be solitary. Lesions tend to be of the same age and appearance. Acute disseminated encephalomyelitis lesions can be similar in appearance to those associated with multiple sclerosis, but subtle differences are reported. Magnetic resonance imaging findings in multiple sclerosis tend to show lesions that are periventricular in location, more heterogeneous in appearance, and of different ages.

Acute disseminated encephalomyelitis was originally believed to be a single event that resolves over the course of months. Recently, repeated separate episodes of ADEM have been reported. This has been termed recurrent, or, multiphasic disseminated encephalomyelitis (MDEM), and requires 2 separate clinical events consistent with ADEM, but separated by a period of at least 3 months. Patients with MDEM tend to be older (typically >10 years of age) and demonstrate more severe and prolonged local neurological symptoms.

The pathogenesis of ADEM is unclear; it is believed to be the result of an autoimmune process in the CNS triggered by an infectious or environmental exposure in a genetically susceptible individual. The concept of molecular mimicry is one of the most prevalent theories on the pathogenesis of ADEM. This suggests that certain amino acid sequence homologies and antigenic epitopes are shared between an invading pathogen and the host CNS protein. Myelin components, such as myelin basic protein, myelin proteolipid protein, and myelin oligodendrocytes glycoprotein share antigenic determinants with many pathogens. Genetics also

likely play a role. There is increasing evidence that individuals with the haplotype HLA-DRB1 have increased frequency of the disease. This haplotype appears to increase immunoreactivity to epitopes of myelin proteins.

Myelin oligodendrocyte glycoprotein (MOG) is expressed on oligodendrocytes and the surface of myelin sheaths. Antibodies to MOG were initially discovered in 2007, with widespread clinical testing now available. Numerous reports have identified the presence of antibodies to MOG in a number of acute CNS demyelinating syndromes in both adult and pediatric patients. MOG antibodies have been described in patients with ADEM, multiple sclerosis, optic neuritis, transverse myelitis, and N-methyl-D aspartate-receptor (NMDA) encephalitis. In a recent study (Mol, 2020), 17% of serum in pediatric patients with radiologic evidence of demyelinating CNS disease showed the presence of anti-MOG antibodies. In pediatric patients with MDEM, up to 33% of pediatric patients were MOG-IgG positive, making this the most common antibody discovered to date associated with ADEM, and the correct response in this vignette. Clinically, seizure activity was more often reported in patients with ADEM who demonstrate the presence of MOG antibodies.

In addition to MOG antibodies, other antibodies are suspected as the causative agent in other autoimmune encephalitides. The presence of NMDA-receptor antibodies is one of the most frequently reported autoimmune encephalitides. Patients tend to be older, and often present with psychosis, irritability, and a deterioration of speech and language skills. Neuroimaging in encephalitis patients with antibodies to NMDA receptors is often normal. Antibodies to glutamic acid decarboxylase (GAD) can cause an autoimmune encephalitis. Patients often present with headache, expressive aphasia, and auditory hallucinations. Findings on MRI can show edema in the temporal lobes. Endocrinopathies are often associated with anti-GAD antibody-associated encephalitis. Antibodies to γ-aminobutyric acid receptor type A are reported to cause encephalitis in both adults and children. Most patients present with difficult-to-control seizure activity. Findings on MRI show diffuse cortical and subcortical edema. Almost one-third will have an associated malignancy detected at time of diagnosis.

Suggested Reading(s)

- Cole J, Evans E, Mwangi M, et al. Acute disseminated encephalitis in children: an updated review based on current diagnostic criteria. *Pediatr Neurol.* 2019;100:26-34. doi:10.1016/j.pediatrneurol.2019.06.017
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- Gklinos P, Dobson R. Myelin oligodendrocytes glycoprotein-antibody associated disease: an updated review of the clinical spectrum, pathogen mechanisms and therapeutic management. *Antibodies.* 2024;13:43-60. doi:10.3390/antib13020043
- Hennes EM, Baumann M, Lechner C, et al. MOG spectrum disorders and role of MOG-antibodies in clinical practice. *Neuropediatrics.* 2018;49:3-11. doi:10.1055/s-0037-1604404

Content Domain

- Critical Care, Neurological disorders and support

Learning Objectives

- Review the pathophysiology of autoimmune encephalitis.

The correct answer is: myelin oligodendrocyte glycoprotein

View Peer Results

Question 5

Correct

1.00 points out of 1.00

Comment

A 17-month-old, 10-kg infant is admitted to the pediatric intensive care unit with severe malnutrition and growth faltering. The family reports that the infant has been sick on and off for a number of months and has not fed well, with ongoing anorexia, frequent emesis, and diarrhea. Due to concerns for refeeding syndrome, the child is ordered to receive nothing by mouth, and is maintained on an infusion of parenteral isotonic fluids containing 10% dextrose at a rate of 40 ml/hr. A nutritional assessment is performed, and it is determined that the child's goal caloric intake to address the current needs, including catch-up nutrition, approximates 1,000 kcal/day. The family raises concern that the child is not receiving any nutrition because the child is not being fed by mouth.

Of the following, given the caloric goals established above, the percentage of the daily caloric goal currently approximated by the dextrose-containing parenteral fluid is

- A. 33% ✓
- B. 44%
- C. 55%
- D. 75%

Your answer is correct.

PREP Pearl(s)

- The primary sources of calories from parenteral nutrition include dextrose (3.4 kcal/g), lipid (10 kcal/g), and protein (4 kcal/g).
- A percent solution is defined as g of solute per 100 mL of solution; 10% dextrose has 10 g of dextrose per 100 mL of solution.
- Attention to the three nutritional components of parenteral nutrition is important to balance osmolarity and maintain appropriate carbon dioxide production.

Critique

The child in the vignette presents with growth faltering and malnutrition, and is maintained on a parenteral infusion of dextrose containing fluids. An understanding of the caloric components of parenteral and enteral intake is required to ensure adequate caloric intake in any pediatric patient, and is particularly important in those with critical illness to ensure caloric and nutritional needs are met. When prescribing total parenteral nutrition, clinicians must balance the different components while ensuring neither too few nor too many calories are prescribed.

The three primary sources of calories from parenteral nutrition include dextrose, lipid, and protein. Dextrose provides 3.4 kcal/g and represents the dominant immediate energy source for cellular metabolism. Lipid provides ~10 kcal/g and promotes protein-sparing when co-administered with sufficient protein. Protein

provides 4 kcal/g and is used to mitigate further catabolism of endogenous stores in critical illness. Also, the amino acids provided in commercial protein products provide substrate for synthesis of acute-phase proteins and other components of the acute stress response.

The child described in the vignette is receiving only dextrose as a caloric source and thus receives 3.4 kcal/g of dextrose. A 10% solution of dextrose provides 10 g of dextrose per dL of fluid. Thus, in a 24 hour period, at 40 ml/hr, the child above receives 960 ml (9.6 dL). This equates to 96 g of dextrose or 326 kcal (3.4 kcal/g x 96 g/day= 326 kcal/day). Thus, if the daily caloric goal is 1,000 kcal, the child is receiving 32.6% of the daily goal from the current dextrose infusion.

10% dextrose= 10 g dextrose/100 ml

At 40 ml/hr = 960 ml/day = 9.6 dL/day= 96 g dextrose/day

96 g dextrose/day x 3.4 kcal/g dextrose = 326 kcal/day

326 kcal/1000 kcal goal= 32.6% of goal calories

When prescribing parenteral nutrition, the individual needs of the patient should be considered, however, there are some general guidelines regarding the individual components prescribed. A balance between the three components is also important to balance admixture osmolarity, maintain appropriate carbon dioxide production and respiratory quotient. General recommendations include:

- Carbohydrates (dextrose): typically provide 40% to 60% of the total calories
- Fats (lipids): can supply 30% to 50% of the total calories
- Protein: 1.5-3 g per kg of body weight per day is recommended (depending on patient age and comorbidities)

Suggested Reading(s)

- Ben XM. Nutritional management of newborn infants: practical guidelines. *World J Gastroenterol.* 2008;14(40):6133-9. doi:10.3748/wjg.14.6133
- Mesotten D, Joosten K, van Kempen A, et al. ESPGHAN/ESPEN/ESPR/CSPEN guidelines on pediatric parenteral nutrition: carbohydrates. *Clin Nutr.* 2018;37(6):2337-43. doi:10.1016/j.clnu.2018.06.947
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Content Domain

- Gastroenterology, Nutrition

Learning Objectives

- Understand the caloric components of parenteral nutrition.
- Calculate caloric intake from parenteral nutrition.

The correct answer is: 33%

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Question 6

Correct

1.00 points out of 1.00

Comment

A bystander discovers a young child in a car-seat parked outside of a grocery store on a hot day. Through the window, he can tell the child is not moving and has flushed skin. He calls emergency medical services and asks the store to use their intercom to try to find the child's caregiver. A few minutes later, the child's mother opens the car door and an ambulance arrives. The 18-month-old child has irregular respirations and a pulse rate of 160 beats/min, and does not respond to painful stimuli. A rectal temperature measures 41 °C. The paramedics intubate the child and bring them to the local emergency department. On arrival, the nearest pediatric intensive care unit is called for a transfer. The child has had their clothing removed by emergency medical services personnel. The team maintains the patient's airway, provides mechanical ventilation, and gives a fluid bolus.

Of the following, the MOST important recommendation to the transferring emergency department is

- A. immerse the patient in ice water, leaving the head exposed
- B. place ice packs in the patient's groin and axilla areas
- C. provide chilled intravenous fluids as a bolus
- D. spray the patient with lukewarm water, and run fans to allow evaporative cooling ✓

Your answer is correct.

PREP Pearl(s)

- Heat stroke is differentiated from heat exhaustion or heat injury, as resulting in a core body temperature >40 °C (104° F), and showing central nervous system dysfunction and end-organ damage.
- For adults and children older than age 2 years, cold water immersion up to the neck is the preferred method of rapid cooling for heat exhaustion and heat stroke.
- Children younger than age 2 years with heat stroke should be cooled using evaporative methods after airway, breathing, and circulation are stabilized.

Critique

With temperatures rising worldwide, heat-related illnesses are increasing globally. While exertional heat-related injury is more common in older children and adolescents participating in sports activities or working outdoors, younger children and infants are more likely to die due to nonexertional heat injury, such as from being left in a hot car or getting into a car and being unable to let themselves out. In the United States, an average of 38 children die in hot cars each year. Eighty-eight percent of children who have died in a hot car are age 3 and younger. The average age of children left in cars was 16.3 months, and >90% of deaths occurred in children under age 5. While the majority of deaths occurred when the temperature was >90 °F, it is notable that 10% of cases occurred when the outdoor temperature was <80 °F. In addition, it is important to note that caregiver substance abuse was involved in approximately 10% of fatalities. Overall, the incidence of nonexertional heat injury related to children being left in cars has increased significantly over the last 10 years. Public health efforts to decrease these fatalities have centered on education of caregivers as well as

audible and visual reminders in new cars and car seats which alert drivers to look in the back seat for children. However, these are not mandated by law; older cars still do not have alerts, and technologies such as alarms in infant and child car seats are not currently universal in the United States or worldwide.

Heat-related illness encompasses a spectrum of diseases ranging from mild “heat rashes” (miliaria) to the most severe, heat stroke. In between these two extremes are heat edema (vasodilation) and heat exhaustion. The patient in this vignette has suffered the most severe form of heat-related illness: heat stroke. Heat exhaustion, also called heat injury, is a less severe form of heat-related illness. Heat exhaustion symptoms include thirst, weakness, headache, dizziness, and syncope and are marked by tachycardia, dehydration, and heavy sweating, as well as hypotension. Patients with heat exhaustion by definition have a core temperature between 37 °C to 40 °C (98.6 °F–104 °F), and there is no significant central nervous system (CNS) dysfunction or end-organ damage. In comparison, heat stroke results in a core temperature >40 °C and is associated with central nervous system dysfunction and end-organ damage.

The pathophysiology of heat stroke is complex. As the body’s core temperature increases, heart rate, stroke volume, and cardiac output increase. In order to produce sweat and allow evaporation of heat, vasodilation causes blood to be pushed peripherally. Hypotension results, and with peripheral vasodilation, central perfusion to the kidneys, intestines, and liver decreases. Over a relatively short period of time, with continued intense heat exposure, the body’s ability to compensate diminishes, and complete dysregulation of normal physiology ensues. The CNS symptoms may begin as mild confusion but become severe once core temperature exceeds >41 °C (105.8 °F). These symptoms progress from delirium to seizures, encephalopathy, and finally, coma.

Evaporative cooling rather than cold-water immersion is recommended in children younger than 2 years of age. This method involves spraying the patient with lukewarm water while using fans to maximize air circulation; this is the correct answer in this vignette. For adults and children older than 2 years of age, cold-water immersion is the most effective method of cooling for heat exhaustion or heat stroke. If possible, cooling should be initiated by emergency responders using whatever method is available. In cases of heat stroke, cooling should be started after airway, breathing, and circulation are addressed. In infants and younger children, there are limited data regarding immersive cooling, and concerns have been raised about safety due to risk of reflex bradycardia in this population. Placing ice packs in the patient’s groin and axilla areas is the treatment of last resort if no other methods are available, but placing ice packs in the axilla and groin is difficult in small children and is less effective in terms of the body surface area available for cooling. There is no evidence for using chilled intravenous fluids or chilled peritoneal or nasogastric lavage as first-line cooling methods. Likewise, dantrolene, a medication used to treat malignant hyperthermia, is also ineffective in lowering core temperature in environmental heat injury.

The goal of cooling is to rapidly lower the core temperature, while aiming to prevent overshoot hypothermia. The recommended threshold to stop cooling varies in the literature and ranges from 101.5 °F to 102 °F (38.6 °C–38.9 °C). Core temperature should be continuously monitored via a rectal or esophageal probe during cooling.

Suggested Reading(s)

- Hammett DL, Kennedy TM, Selbst SM, Rollins A, Fennell JE. Pediatric heatstroke fatalities caused by being left in motor vehicles. *Pediatr Emerg Care.* 2021;37(12):e1560-e1565. doi:10.1097/pec.0000000000002115
- Mangus CW, Canares TL. Heat-related illness in children in an era of extreme temperatures. *Pediatr Rev.* 2019;40(3):97-107. doi:10.1542/pir.2017-0322

Content Domain

- Emergency Medicine, Environmental Exposures

Learning Objectives

- Recognize levels of severity of environmental heat injury.
- Plan initial management of environmental heat injury

The correct answer is: spray the patient with lukewarm water, and run fans to allow evaporative cooling

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