Myocarditis is defined as inflammation of the myocardium followed by necrosis and/or degeneration of myocytes. The inflammation can be diffuse or focal and is usually due to an infection.

Although myocarditis severe enough to be recognized is rare, it is the most common cause of heart failure in otherwise healthy children. In both children and adults, most cases are subclinical; thus, the true incidence of myocarditis in children is unknown. Unfortunately, the clinical features of myocarditis can vary widely, and often no cardiac signs or symptoms occur, complicating recognition. For children in whom the diagnosis is suspected or cardiovascular compromise is severe enough to require admission to the pediatric intensive care unit (PICU), critical care nurses are an essential component in determining management, care, and outcomes. In this article, I describe the etiology of viral myocarditis in children, potential insidious clinical features, pathophysiology of the disease, and critical care management.

Case 1

A.J., a previously healthy 3-year-old boy, was brought to the emergency department because his body temperature was 40.5°C. During the previous week, he had had some nasal discharge and a mild, nonproductive cough. He had no history of increased work of breathing, rashes, or diarrhea. One day before the visit to the emergency department, he had been vomiting and had been unable to tolerate anything by mouth.

On arrival in the emergency department, A.J. appeared ill but was alert and in no marked distress. Vital signs were heart rate, 162/min; respirations, 26/min; and oxygen saturation, determined by pulse oximetry, 99% on room air. No murmur or gallop was noted; capillary refill was brisk with 2+ peripheral pulses and warm extremities. Breath sounds were clear bilaterally. He appeared mildly dehydrated with tachycardia, mildly sunken eyes, and tacky mucous membranes.

Routine blood tests were done. Electrolyte levels were normal except for a carbon dioxide level of 18 mEq/L, an anion gap of 23 mEq/L, and a serum urea nitrogen level of 21 mg/dL (to convert to millimoles per liter, multiply by 0.357), consistent with dehydration. A complete blood cell count revealed a white blood cell count of 26,900/μL, a hemoglobin level of 12 g/dL, a hematocrit of 35.9%, and a platelet count of 321,000/μL. A differential count was not completed.

Approximately 15 minutes after his initial examination, A.J. received two 20 mL/kg intravenous boluses of normal saline and was given something to eat, which he tolerated well. Because his parents were comfortable with observing the child at home and expressed full understanding of the signs of dehydration, A.J. was readied for discharge.
While the discharge papers were being completed, A.J. suddenly became unresponsive and apneic. Bag-valve-mask ventilation was begun, and subsequently he was pulseless. Cardiopulmonary resuscitation was initiated. After a prolonged attempt at resuscitation, he was pronounced dead. Postmortem examination revealed myocarditis of probable viral origin.

Case 2
K.M., a 5-year-old boy with a history of asthma, was brought to the emergency department because he had fainted. After the syncopal episode, he had shortness of breath and tachypnea. Approximately 4 days before this visit to the emergency department, he had had gastroenteritis with vomiting and diarrhea that resolved. Findings on a physical examination were unremarkable except for bilateral wheezing and diaphragm that resolved. Findings on a physical examination were unremarkable except for bilateral wheezing and diarrhea that resolved. Findings on a physical examination were unremarkable except for bilateral wheezing and diarrhea that resolved.

On arrival in the PICU, his respiratory status continued to deteriorate. He was markedly tachypneic. Oxygen saturations were 86% to 91% on a 100% nonrebreather mask. Continuous albuterol was begun, but his pulmonary status did not change. Auscultation revealed diminished breath sounds at the bases bilaterally, no wheezing, and no murmur but a gallop. A repeat chest radiograph revealed pulmonary edema and cardiomegaly. After the repeat radiograph and continuing respiratory deterioration, he was intubated and mechanical ventilation was started. Within minutes of intubation, clinically significant unifocal premature ventricular contractions and hypotension (50-60/20-30 mm Hg) developed. He was given a lidocaine bolus, and continuous infusions of both lidocaine and epinephrine were started. Laboratory studies revealed a white blood cell count of 7300/μL, a hemoglobin level of 11.1 g/dL, a platelet count of 103 000/μL, and unremarkable electrolyte levels. An echocardiogram showed markedly decreased left ventricular function with an ejection fraction of 10% to 14% and diminished right ventricular function. Neither structural abnormalities nor pericardial effusion was seen. A 12-lead electrocardiogram showed a normal sinus rhythm with frequent premature ventricular contractions and nonspecific T-wave abnormalities (Figure 1). The diagnosis was acute myocarditis with severe cardiomyopathy. Because of the continued deterioration in K.M.’s condition, inability to oxygenate, and worsening cardiac function, extracorporeal membrane oxygenation (ECMO) was started.

ECMO was discontinued on hospital day 13. K.M. made steady improvement, although he continued to require vasoactive support and mechanical ventilation for another 6 days. He was extubated on hospital day 19. At that time, his ejection fraction was approximately 35%. He was subsequently transferred to the pediatric rehabilitation unit, from which he eventually was discharged to home; the diagnosis was severe dilated cardiomyopathy.

Incidence and Prevalence
Myocarditis appears to be more common in children than in adults. Its true incidence, however, is unknown; it is thought that subclinical cases (“silent” myocarditis) occur much more often than do severe cases. Many cases are unrecognized because of the wide range of signs and symptoms and, in some patients, the complete lack of clinical findings. In a postmortem study of children who died without a history suggestive of myocarditis, researchers found evidence of active or healed myocarditis in 17 of 138 cases (12.3%). Of the 17 cases, 15 occurred in children who died suddenly. In postmortem studies in adults, myocardial inflammation occurred in 1% to 9%.

Etiology
The most common form of myocarditis, endemic in both rural Central and South America, is Chagas’ disease, caused by the parasitic protozoan Trypanosoma cruzi. In North America, viral infections cause most cases of myocarditis. Enteroviruses, most importantly coxsackievirus B, are the most frequently reported cause of epidemics of viral myocarditis in children. However, more recent reports indicated that adenoviral infection is as common a cause as enteroviral infection is, if not more so. Rarely, bacteria, fungi, protozoa, parasites, and rickettsiae are causative agents. Myocarditis has also been associated with immune-mediated diseases, collagen vascular diseases, and toxins (Table 1).

Epidemiology
Occurrence of myocarditis can be affected by viral epidemics. An
outbreak of coxsackievirus B in Europe in 1965 correlated with cardiac dysfunction in 5% of infected patients. Incidences were as high as 12% that same year in Scotland, Finland, and Austria. Seasonal viral distributions have been recognized for decades (influenza prevalent during winter months; poliovirus and coxsackievirus A and B typically isolated during summer and fall). Myocarditis has been a prominent finding during epidemics of influenza; thus, occurrence may be seasonal.

Age plays a marked role in prevalence. During the neonatal period, myocarditis is usually abrupt, severe, and often fatal, with mortality as high as 75%. Infants infected with coxsackievirus B during the first year of life have a high incidence of myocarditis. Myocarditis has been linked to sudden infant death syndrome, because inflammatory infiltrates have been found on autopsies of some victims. Incidence increases again during late childhood and adolescence; the myocarditis usually has a delayed onset and patients recover.

Male predominance has been noted with coxsackievirus B heart disease, particularly in adolescents and adults. In these age groups, two-thirds to three-quarters of patients with myocarditis are male. Male predominance has also been reported with coxsackievirus A myocarditis and poliomyelitis. Whether or not differences between the sexes occur in other viral infections is unknown.

Pathophysiology

Although viral infection is the most common initiator of acute myocarditis, the subsequent autoimmune response plays a lead role in myocyte injury. The principal mechanism of myocardial damage is not just viral replication; it includes cell-mediated immunological reactions.

The pathophysiology of myocarditis has been studied in mice infected with a cardiotropic virus, such as coxsackievirus B. After systemic infection, the virus enters the myocyte, where it replicates in the cytoplasm of the cell. Some replicated viruses then enter the interstitium and are phagocytized by activated macrophages. Macrophage activation is due to both viral particles and are phagocytized by activated macrophages. Macrophage activation is due to both viral particles and are phagocytized by activated macrophages.

Figure 1 K.M.'s electrocardiogram on admission to the pediatric intensive care unit shows sinus tachycardia, frequent unifocal premature ventricular contractions, and nonspecific T-wave abnormality.
necrosis factor α). When activated by interleukin 2, NK cells eliminate virally infected myocytes and inhibit virus replication11,18,19 (Figure 2). The significance of the action of NK cells in the pathogenesis of the disease is well established; in animals depleted of NK cells before infection, a more severe myocarditis develops.8 As noted earlier, myocarditis after infection with coxsackievirus B occurs more often in men. This propensity may be related to differences between the sexes in the activation of NK cells. After infection, myocarditis that develops in female mice is less severe than that in male mice. In one study,11 after the same infection, male mice were markedly less efficient than female mice in activating NK cells. This decrease in NK-cell activation presumably results in decreased viral clearance with a resultant increase in illness severity.11 This difference in NK-cell activation may explain the male predominance for the development of clinically significant myocarditis.

Unlike NK cells, T cells may contribute to damage in both infected and noninfected myocytes.5,11,20 Activation of T cells results in accumulation of macrophages within the myocyte and production of cell-mediated cytotoxic effects.2,8 Although T cells can lyse virus-infected myocytes, the accumulation of macrophages and the concomitant cytotoxic effects create a fine balance between viral clearance and myocyte damage.8 Adding to myocyte injury, lysis by T cells is indiscriminate; it occurs in both infected and noninfected cells.2,11 The result is necrosis of healthy as well as infected myocytes. Therefore, a percentage of myocardial damage comes from “friendly fire,” specifically the patient’s own immune response.

Permanent myocardial damage can be the end result of myocarditis. Such damage is thought to be due to an overaggressive immunological activation in which persistent T-cell infiltration leads to long-term tissue destruction and subsequent dilated cardiomyopathy.8,20 Infection with enteroviruses plays a major role in chronic forms of dilated cardiomyopathy.21,22

Clinical Manifestations and Diagnosis

Myocarditis is classified as fulminant, acute, or chronic. Fulminant myocarditis is preceded by a viral prodrome that is followed by sudden onset of severe hemodynamic compromise. Acute myocarditis has a less distinct onset and, initially, less severe compromise but is followed by a worse outcome than fulminant myocarditis. McCarthy et al23 found that adults with fulminant myocarditis had excellent long-term survival, whereas patients with acute myocarditis had progressive failure that led to death or the need for a heart transplant. Chronic myocarditis can be defined as persistent (lasting >3 months), recurrent, and latent.23

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Table 1  Causes of myocarditis

<table>
<thead>
<tr>
<th>Viruses</th>
<th>Immune-mediated diseases</th>
<th>Drugs</th>
</tr>
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<tbody>
<tr>
<td>Coxsackievirus A</td>
<td>Rheumatoid arthritis</td>
<td>Sulfonamides</td>
</tr>
<tr>
<td>Coxsackievirus B</td>
<td>Rheumatic fever</td>
<td>Cyclophosphamide</td>
</tr>
<tr>
<td>Echovirus</td>
<td>Ulcerative colitis</td>
<td>Acetzolamide</td>
</tr>
<tr>
<td>Adenovirus</td>
<td>Systemic ulcerative colitis</td>
<td>Amphotericin B</td>
</tr>
<tr>
<td>Rubella virus</td>
<td></td>
<td>Indomethacin</td>
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<tr>
<td>Measles virus</td>
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<td>Tetracycline</td>
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<td>Varicella virus</td>
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<td>Phenytoin</td>
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<td>Poliovirus</td>
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<td>Penicillin</td>
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<td>Mumps virus</td>
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<td>Herpesvirus</td>
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<tr>
<td>Epstein-Barr virus</td>
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<tr>
<td>Cytomegalovirus</td>
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<td>Arbovirus</td>
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<tr>
<td>Influenza virus</td>
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<tr>
<td>Human immunodeficiency virus</td>
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</tbody>
</table>

Bacteria

| Meningococci                  |                            |                            |
| Borrelia burgdorferi          |                            |                            |
| (Lyme disease)                |                            |                            |
| Salmonellae                   |                            |                            |
| Mycobacteria                  |                            |                            |
| Streptococci                  |                            |                            |

Drugs

| Sulfonamides                  |                            |                            |
| Cyclophosphamide              |                            |                            |
| Acetzolamide                  |                            |                            |
| Amphotericin B                |                            |                            |
| Indomethacin                  |                            |                            |
| Tetracycline                  |                            |                            |
| Phenytoin                     |                            |                            |
| Penicillin                    |                            |                            |

Fungi and yeast

| Aspergillus                   |                            |                            |
| Candida                       |                            |                            |

Protozoa

| Trypanosoma cruzi            |                            |                            |
| Toxoplasma gondii            |                            |                            |

Parasites

| Ascaris                       |                            |                            |
| Schistosoma                   |                            |                            |
| Trichinella spiralis          |                            |                            |

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Drugs leading to myocarditis include sulfonamides, cyclophosphamide, acetzolamide, amphotericin B, indomethacin, tetracycline, phenytoin, and penicillin. Infection with enteroviruses plays a major role in chronic forms of dilated cardiomyopathy.21,22
Diagnosing myocarditis in children can be challenging. Not only can children have a wide range of nonspecific signs and symptoms, but depending on cognitive development, they may not be able to communicate their symptoms. Despite a variety of invasive and noninvasive studies, myocarditis is a presumptive diagnosis based on history and clinical features.

**History**

Clinical features of viral myocarditis are affected by age, sex, and the child’s baseline health status. These variables affect the balance between pathogen clearance and degree of inflammation. Older children may have a history of upper respiratory infection; infants may have anorexia, vomiting, and lethargy. Often, signs and symptoms are nonspecific or may resemble the signs and symptoms of relatively common diagnoses in children, including bronchiolitis, pneumonia, failure to thrive, and gastroenteritis.

Both cases described earlier had the potentially insidious manifestations of viral myocarditis. In the first case, the signs and symptoms suggested benign gastroenteritis with slight dehydration, a situation that is not unusual in toddlers and preschool children. K.M., who had a history of gastroenteritis, also had syncope, wheezing, and increased work of breathing. His history of asthma confounded his clinical features. Consequently, he initially received treatment for an exacerbation of asthma.

Older children may have chest pain (the sole symptom in some patients). Some have abdominal pain. Atypical manifestations such as syncope (K.M.), seizures, and sudden death also have been reported. A more focused cardiac examination when no signs of congestive heart failure occur may be prompted by patients’ reports of chest pain, dyspnea, exercise intolerance, or fatigue. Tachycardia of unknown origin in an otherwise healthy child may be an ominous sign. Newborns and infants, unlike older children, are more likely to have circulatory shock.

When myocarditis is suspected, a thorough history is imperative for
identifying possible causes. Parents should be asked about exposures to potential infectious agents, pharmacological agents that can cause myocardial inflammation, and toxins, including illicit drugs (eg, cocaine). Immunization status should be obtained because several infectious diseases of childhood (diphtheria, poliomyelitis) are potential causes.

**Physical Examination**

During episodes of cardiac compromise, neurohormonal responses activate the sympathetic nervous system and the renin-angiotensin-aldosterone system, resulting in improved contractility, increase in heart rate, vasoconstriction, and fluid and sodium retention. Tachycardia out of proportion for age is common in children with myocardial compromise as the heart attempts to compensate for inadequate oxygen delivery to the tissues. Vasoconstriction occurs to improve preload and maintain blood pressure. This decrease in peripheral perfusion is manifested as cool extremities, described by level of coolness (eg, cool to midcalf, cool to knee), quality of pulses, capillary refill time (compromise considered at >3 seconds with the extremity at the level of the heart), decreased urine output (<1 mL/kg per hour), and changes in mental status.25 Pulse quality is characterized by using the 0 to 4 pulse intensity scale (0 = no pulses detected, 4 = bounding).26 Extremities may appear mottled or pale. Despite these alterations in perfusion, it must be emphasized that vasoconstriction in children will maintain a blood pressure within a normal range for age even when tissue perfusion is inadequate. Hypotension is considered not only a late finding of cardiac failure but also an ominous one.27

Often, an S₃ (ventricular gallop) occurs because of rapid filling of a non-compliant, poorly contracting left ventricle. Murmurs are less common, although a soft systolic murmur may be heard if mitral or tricuspid insufficiency is present.12 Heart sounds may be muffled if concomitant pericarditis is present.11 Rhythm irregularities may be detected, especially supraventricular tachycardia or ventricular ectopic beats as with K.M. Tachypnea is a common sign of myocardial failure in children. Tachypnea is the result of pulmonary edema due to left ventricular failure.24 Clinical findings can include wheezing, a cough, grunting, nasal flaring, and intercostal retraction. Older children may report orthopnea or inability to catch their breath. Cyanosis is rare. Rales are typically a late sign of pulmonary congestion and may not occur at all in infants.26 Hepatomegaly, an indication of venous congestion, may or may not occur. Liver tenderness or absence of a firm edge on palpation below the right costal margin may indicate liver engorgement. Clinical findings of viral myocarditis are summarized in Table 2.

**Diagnostic Evaluation**

The diagnostic approach for a child with suspected myocarditis includes strategies to both aid in establishing the diagnosis and rule out disease processes that may mimic myocarditis (eg, a structural cardiac defect or pericardial effusion). In addition, many of these interventions provide an estimate of myocardial function and can

<table>
<thead>
<tr>
<th>Subjective</th>
<th>Tachycardia</th>
<th>Tachypnea</th>
<th>Wheezing, cough, increased work of breathing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Objective</td>
<td>Alteration in peripheral perfusion</td>
<td>Decrease in pulse quality</td>
<td>Extremity coolness</td>
</tr>
<tr>
<td></td>
<td>Prolonged capillary refill</td>
<td>Paleness, mottling</td>
<td>Paleness, mottling</td>
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<tr>
<td></td>
<td>Decreased urine output</td>
<td>Murmur</td>
<td>Gallop</td>
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<tr>
<td></td>
<td>Murmur</td>
<td>Gallop</td>
<td>Rhythm irregularities</td>
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<td></td>
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<td></td>
<td>Ventricular ectopy</td>
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<td></td>
<td></td>
<td></td>
<td>Atrioventricular block</td>
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<td></td>
<td></td>
<td></td>
<td>Sinus tachycardia or supraventricular tachycardia</td>
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<td></td>
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<td></td>
<td>Hepatomegaly</td>
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<td></td>
<td></td>
<td></td>
<td>Rales</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>Hypotension</td>
</tr>
</tbody>
</table>

* May or may not be present.

* Late finding.

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Table 2  Myocarditis in children: clinical findings

Subjective  Objective

| Chest pain | Tachycardia |
| Abdominal pain | Tachypnea |
| Dyspnea/orthopnea | Wheezing, cough, increased work of breathing |
| Exercise intolerance | Alteration in peripheral perfusion |
| Fatigue | Prolonged capillary refill |
| | Decrease in pulse quality |
| | Extremity coolness |
| | Paleness, mottling |
| | Decreased urine output |

Hepatomegaly, an indication of venous congestion, may or may not occur. Liver tenderness or absence of a firm edge on palpation below the right costal margin may indicate liver engorgement. Clinical findings of viral myocarditis are summarized in Table 2.
help in establishing clinical interventions. Diagnostic findings are summarized in Table 3.

**Chest Radiography**
Evidence of cardiomegaly on a chest radiograph is an important clinical finding in myocarditis; however, the degree of heart enlargement depends on the stage of the disease. Subacute or chronic myocarditis is characterized by cardiomegaly; cardiomegaly may or may not occur in children with fulminant myocarditis. In fact, the possibility of myocarditis is often not considered until late, when cardiomegaly is evident on chest radiographs. Myocarditis should not be ruled out in infants or children with marked cardiovascular compromise or collapse of unknown cause who do not have evidence of heart enlargement on radiographs.

**Electrocardiography**
Although not diagnostic, findings on an electrocardiogram are rarely normal in patients with myocarditis. Some children have such mild illness that a conduction disturbance on an electrocardiogram is the only abnormal finding. Sinus tachycardia is a common finding with myocarditis. Low-voltage QRS complexes, ST-T wave abnormalities, or prolonged QT interval may be apparent. Left ventricular hypertrophy with repolarization changes (strain) is typical. Complete atrioventricular block has also been described. Occasionally, evidence of myocardial infarction is seen.

**Echocardiography**
Findings on echocardiograms are rarely normal in patients with myocarditis. As with K.M., impaired left ventricular function with dilatation of 1 or more chambers is typical. K.M.’s echocardiogram showed poor left ventricular systolic function, with dilatation and slightly diminished right ventricular function. In the absence of any structural abnormalities, these findings help establish the diagnosis. Generally, right ventricular function is less compromised than is left ventricular function; atrioventricular valve regurgitation may occur. Occasionally, left ventricular thrombi are found.

**Laboratory Studies**
Historically, despite its limited sensitivity and specificity and its inherent risks, endomyocardial biopsy has been the reference standard for diagnosing viral myocarditis. Nevertheless, for several reasons, endomyocardial biopsy is used less often in children than in adults. First, biopsy can be associated with significant sampling error. Several tissue specimens (5 or more) are needed because of the focal nature of the disease; many clinicians think that biopsy leads to underestimation of the presence of the disease. Second, borderline myocarditis can result in little to no evidence of myocyte destruction. Third, expert interpretation can vary, including variance with other markers of viral infection. Because of the possibility of false-negative results, lack of an endomyocardial biopsy positive for myocarditis does not rule out this disease.

<p>| Table 3 Myocarditis in children: diagnostic findings |
|-----------------|--------------------------|</p>
<table>
<thead>
<tr>
<th><strong>Assessment</strong></th>
<th><strong>Finding</strong></th>
</tr>
</thead>
<tbody>
<tr>
<td>Chest radiography</td>
<td>Cardiomegaly&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Electrocardiography</td>
<td>Tachycardia: sinus or supraventricular</td>
</tr>
<tr>
<td></td>
<td>Atrioventricular block&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Low-voltage QRS complexes</td>
</tr>
<tr>
<td></td>
<td>ST-T wave abnormalities (elevation or depression)</td>
</tr>
<tr>
<td></td>
<td>Prolonged QT interval</td>
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<tr>
<td></td>
<td>Ventricular ectopy</td>
</tr>
<tr>
<td>Echocardiography</td>
<td>Impaired ventricular function (left &gt; right)</td>
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<tr>
<td></td>
<td>Ventricular dilatation</td>
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<tr>
<td></td>
<td>Atrioventricular valve regurgitation&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Left ventricular thrombi&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Laboratory studies</td>
<td>Serological tests</td>
</tr>
<tr>
<td></td>
<td>Increased levels of creatine kinase and its MB isoenzyme</td>
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<tr>
<td></td>
<td>Increased level of cardiac troponin C</td>
</tr>
<tr>
<td></td>
<td>Elevated sedimentation rate&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Increased level of C reactive protein&lt;sup&gt;a&lt;/sup&gt;</td>
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<tr>
<td></td>
<td>Increased level of white blood cells, with lymphocytes predominating</td>
</tr>
<tr>
<td></td>
<td>Viral titers&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Increased level of immunoglobulin G&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td></td>
<td>Presence of virus&lt;sup&gt;a&lt;/sup&gt;</td>
</tr>
<tr>
<td>Myocardial biopsy</td>
<td>Myocyte destruction, fibrosis, inflammatory cells, lymphocytic infiltrates</td>
</tr>
</tbody>
</table>

<sup>a</sup> May or may not be present.
For K.M., findings on endomyocardial biopsy on hospital day 8 confirmed the diagnosis. Tissue specimens from the right ventricle had intact myocytes with small foci of lymphocytic infiltrates and extensive areas of myocyte destruction with organizing fibrosis and infiltrates of inflammatory cells most consistent with myocarditis.

Recently, use of less invasive means of diagnosing myocarditis in children has been emphasized. Use of polymerase chain reaction (PCR) and in situ hybridization has increased the sensitivity for diagnosing viral myocarditis. Tracheal aspirates for PCR analysis in intubated children are useful for detecting viral genomes in children with or without myocarditis. Akhtar et al reported that PCR of tracheal aspirates had a sensitivity of 100% for predicting the results of PCR of endomyocardial biopsy specimens, albeit for a small (n = 10) sample size. Although a tracheal aspirate positive for virus is not diagnostic for myocarditis itself, PCR of tracheal aspirates may provide a safer means of identifying a viral cause for illness in children with cardiac decompensation of unknown origin.

Serological studies may reveal an elevation in myocardial enzyme levels (creatine kinase, normal value 25-190 IU/L [to convert to microkatal, multiply by 0.0167]; MB isoenzyme of creatine kinase, normal value <12% of creatine kinase). In children, the level of cardiac troponin T is a more sensitive indicator of myocardial damage than are myocardial enzyme levels. In healthy children, cardiac troponin levels are typically less than the detection level (<0.02 ng/mL). Lauer et al reported that 35% of patients with suspected myocarditis also had elevated levels of cardiac troponin; among those, 98% had viral myocarditis confirmed histologically.

Other nonspecific laboratory studies may aid in the diagnosis of viral myocarditis. Elevations in sedimentation rate (>20 mm/h) and level of C-reactive protein (>0.05 mg/dL; to convert to nanomoles per liter, multiply by 9.524) may occur. A normal sedimentation rate, however, does not rule out myocarditis. Viral studies may reveal enteroviral or adenoviral antibodies, Epstei-Barr virus, cytomegalovirus, or a 4-fold increase in the level of immunoglobulin G but do not definitively establish causation. Levels of immunoglobulin G antibodies to Epstein-Barr virus nuclear antigen were elevated in K.M. PCR may also be used to detect viral genomes.

Elevation in white blood cell count with predominance of lymphocytes may suggest viral causes of myocarditis. Cultures of blood, stool, cerebrospinal fluid, or nasopharyngeal secretions are less specific than cultures of tracheal aspirates; Martin et al found that cultures showed growth in only 27% of children with viral myocarditis.

**Critical Care Management**

Care of children with clinical features consistent with myocarditis depends on the severity of myocardial dysfunction. Management is designed around supportive measures rather than being aimed at a causative agent. Maintenance of cardiac output with aggressive support of cardiac function is essential. Such support includes initiation and monitoring of inotropic therapy, afterload reduction, arrhythmia management, and adequate tissue oxygenation. Respiratory support may require intubation and mechanical ventilation. Fluid management may include both administration of fluid boluses and diuretic therapy. Monitoring of and interventions to prevent complications should be ongoing.

**Inotropic Support**

When cardiac output is inadequate, a rapid acting inotropic agent is administered. Inotropic agents are administered cautiously because the myocardium is irritable and use of inotropic agents can instigate and/or worsen arrhythmias. One inotropic agent or a combination of such agents, including dopamine, dobutamine, and milrinone, can be administered (Table 4).

Dobutamine is often used when the primary cause of inadequate tissue perfusion is cardiac in origin. Dobutamine improves contractility and decreases systemic vascular resistance, providing afterload reduction in addition to improving cardiac output. A mild increase in heart rate can occur, although significant tachycardia is unusual. Although dobutamine increases myocardial oxygen demand in adults with congestive heart failure, in children with patent coronary arteries, coronary blood flow and oxygen supply to the heart actually increase.

Dopamine has both inotropic and vasopressor properties and is useful in patients who are in shock with cardiac dysfunction and associated mild to moderate hypotension. Dopamine is not routinely used to...
treat poor cardiac contractility if blood pressure is normal; in this instance, a purely inotropic agent is recommended.\textsuperscript{26} Milrinone, a phosphodiesterase inhibitor, improves myocardial diastolic function, resulting in improved ventricular relaxation and filling times. Although not specific to myocarditis, improvement in cardiac index and decreased systemic vascular resistance have been reported in both pediatric and neonatal populations with the use of milrinone. The PRIMACORP study, a large, randomized, placebo-controlled trial, showed the effectiveness of high-dose milrinone for preventing low cardiac output syndrome in children after cardiac surgery.\textsuperscript{23} Although postoperative cardiac dysfunction differs from dysfunction related to viral myocarditis, this trial\textsuperscript{23} is the only one in which researchers specifically looked at milrinone and improvement in cardiac function after myocardial injury; thus, its results may be relevant to children with myocarditis. Milrinone is also less arrhythmogenic than many other agents, making it the inotropic agent of choice in many centers.\textsuperscript{24}

Severe shock may require the addition of epinephrine, although excessive administration of catecholamines is discouraged. Epinephrine is highly arrhythmogenic and markedly increases myocardial oxygen consumption and workload.\textsuperscript{24} Low doses (0.05-0.2 μg/kg per minute) of epinephrine cause peripheral vasodilatation, increased heart rate, and improved contractility. With adequate intravascular volume, stroke volume and cardiac output increase. Higher doses result in stimulation of $\alpha_1$-adrenergic receptors, causing further increase in heart rate and an increase in systemic vascular resistance. Although initially blood pressure may increase, cardiac output will ultimately decrease. As heart rate increases, the myocardium loses filling time, and oxygen demand and consumption markedly increase. With increasing systemic vascular resistance, workload is amplified in an already failing myocardium. Epinephrine therefore is not routinely used as a first-line inotropic agent for myocarditis.\textsuperscript{33}

### Arrhythmia Management

Rhythm disturbances can be life threatening and must be treated aggressively. Ventricular ectopy, ventricular tachycardias, and heart

---

### Table 4 Continuous infusion inotropic support

<table>
<thead>
<tr>
<th>Drug</th>
<th>Classification</th>
<th>Dose, µg/kg per minute</th>
<th>Action\textsuperscript{a}</th>
<th>Adverse effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dopamine</td>
<td>Sympathomimetic</td>
<td>0.5-2</td>
<td>Dopaminergic: renal vasodilatation</td>
<td>Tachyarrhythmias</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Promotion of sodium excretion</td>
<td>Reduced renal blood flow at &gt;5 µg/kg per minute</td>
</tr>
<tr>
<td></td>
<td></td>
<td>5-10</td>
<td>$\beta_1$-adrenergic effects</td>
<td>Increased myocardial oxygen demand</td>
</tr>
<tr>
<td></td>
<td></td>
<td>10-15</td>
<td>Mixed $\alpha_1$, $\beta_1$-adrenergic effects</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>&gt;20</td>
<td>Predominantly $\alpha_1$-adrenergic effects</td>
<td>Ectopy</td>
</tr>
<tr>
<td>Dobutamine</td>
<td>Sympathomimetic</td>
<td>2-20</td>
<td>Pure $\beta_1$-adrenergic effects</td>
<td>Tachycardia</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>Sympathomimetic</td>
<td>0.1-1.0</td>
<td>Adrenergic agonist effects, predominately $\alpha_1$-adrenergic effects</td>
<td>Tachyarrhythmias</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Ventricular ectopy</td>
<td>Hypertension</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Myocardial ischemia</td>
<td></td>
</tr>
<tr>
<td>Milrinone</td>
<td>Phosphodiesterase enzyme inhibitor</td>
<td>May use loading dose of 50-75 µg/kg before infusion</td>
<td>Improves contractility Afterload reduction</td>
<td>Hypotension</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>Thrombocytopenia</td>
<td>Ventricular ectopy</td>
</tr>
</tbody>
</table>

\textsuperscript{a} $\alpha$-adrenergic: constriction of veins and arteries; $\beta_1$-adrenergic: increased heart rate, contractility; $\beta_2$-adrenergic: vasodilatation, bronchodilatation.
block can develop in children with myocarditis. Adding to the challenge of arrhythmia management is determining whether the rhythm disturbance is the result of myocardial inflammation, hypoxia, inotropic therapy, or a combination of those factors (Table 5).

Regardless of the cause, tachyarrhythmias must be controlled to prevent further deterioration of ventricular function. For patients with supraventricular tachycardia, adenosine is used for those whose hemodynamic status is stable; electrocardioversion is used for those whose hemodynamic status is unstable.

In children with recurring supraventricular tachycardia, amiodarone has shown clinical effectiveness and has improved survival rates after cardiac arrest due to ventricular tachyarrhythmias. Thus, the American Heart Association now recommends amiodarone as the first-line antiarrhythmic therapy in pulseless ventricular tachycardia. Amiodarone requires a loading dose; in some patients, the ectopy stops after the loading dose is administered. Other patients require a continuous infusion.

Ventricular ectopy, unifocal or multifocal, is common, as in K.M.’s case (see Figure 1). Ventricular ectopy is often treated with lidocaine, the most widely used class IB antiarrhythmic agent in pediatric critical care. As with amiodarone, a loading dose is required, followed by a continuous infusion. Dosing is determined by resolution of ectopy and achieving an adequate therapeutic serum concentration (1.5-5 μg/mL).

Although digoxin has led to improvement in patients with heart failure, it is not routinely used during the acute phase of myocarditis. Digoxin increases cytokine production as well as intracellular calcium loading, which, in patients with myocardial inflammation, can induce or worsen ventricular arrhythmias. If digoxin is used, the loading dose should be no more than 75% of the normal total loading dose (Table 6).

If complete atrioventricular block or second-degree block with inadequate perfusion develops, a temporary transvenous or epicardial

<table>
<thead>
<tr>
<th>Table 5 Intravenous antiarrhythmic agents</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Drug</strong></td>
</tr>
<tr>
<td>Adenosine</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Digoxin</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Amiodarone</td>
</tr>
<tr>
<td></td>
</tr>
<tr>
<td>Lidocaine</td>
</tr>
</tbody>
</table>

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pacemaker may be used. Insertion of a permanent pacemaker is indicated if heart block persists for more than 2 weeks.11,26

Fluid Management
The goal of fluid management is to maintain an intravascular volume sufficient to preserve preload and ventricular filling without overload.26 In acute or decompensated heart failure, diuretics are often administered because removal of excess intravascular volume may help improve cardiovascular function.24 Diuretics are administered cautiously because too rapid a removal of intravascular fluid can result in hypovolemia and hypotension. Tobias et al24 recommend initial dosing of furosemide at 0.1 to 0.25 mg/kg per dose to prevent an excessive reduction in cardiac output. Doses can be repeated every 6 to 12 hours up to 2 mg/kg per dose. Furosemide can also be administered as a continuous infusion at 0.1 to 0.4 mg/kg per hour, with the additional benefits of maintaining a constant serum level and minimizing the opportunity for sodium retention by the kidneys.24 Once the acute episode of failure is controlled, other diuretic agents, including thiazides (diuril) and spironolactone, are used.

Anticoagulation
Reports of the use of anticoagulants in patients with myocarditis are largely anecdotal, and such use is not supported by retrospective studies of adults with heart failure.33 Severely compromised ventricular function and stasis of blood flow, as can occur in depressed myocardial function, can increase the risk for thrombosis formation. Thrombi potentially can embolize to the cerebrovascular system (causing neurological sequelae) or to the pulmonary vasculature. Because of the increased risk for thrombus formation, use of anticoagulation is common, typically done as a continuous infusion of heparin in the critical care unit. Dosing varies depending on age, and the dose is titrated to maintain a predetermined anticoagulatory state.

Immunosuppressive Therapy
Because immune dysfunction may play a primary role in many cases of myocarditis, immunosuppressive therapies might shorten the course and lessen the severity of illness. Intravenous immune globulin is beneficial in the treatment of children with acute myocarditis.10,17 Improvement in left ventricular function and 1-year survival rate in patients with biopsy-proven myocarditis has been demonstrated after treatment with a single, high dose of intravenous immune globulin (2 g/kg body weight).17 In children, Gagliardi et al36 reported an 83% survival rate at 13 years with high-dose cyclosporine (6-8 mg/kg per day; dose adjustment when therapeutic serum levels were reached) and prednisone (2 mg/kg per day, tapered off over 6 months). Ahdoot et al37 found significant improvement in left ventricular function in 5 patients with acute viral myocarditis after combination treatment with muromonab (OKT3), intravenous immune globulin, and high-dose methylprednisolone. In that study, 4 patients also received cyclosporine, 3 received azathioprine, and 1 received a 2-month course of methotrexate. Although Ahdoot et al found improvement in function with immunosuppressive therapy in a small sample, their study is skewed; 4 of 5 patients also received additional therapies.

Although these studies indicate that immunosuppression is safe in children with myocarditis, without large, randomized controlled studies, it cannot be determined if outcome is worse without the addition of immunosuppressive therapies. Currently no randomized controlled trials of immunosuppressive therapy in children with myocarditis have been done. Because children overall have higher recovery rates than adults for many diseases, the question is whether children with myocarditis, with or without immunosuppression, intrinsically have a higher recovery rate than adults do.38 Thus, the beneficial effects of immunosuppression in children with viral myocarditis have yet to be determined.

Rescue Therapies
In children with progressing low cardiac output syndrome, use of extracorporeal life support may be necessary. Clinical guidelines for the

### Table 6 Intravenous digoxin dosing

<table>
<thead>
<tr>
<th>Age</th>
<th>Digitalizing dose, µg/kg</th>
<th>Maintenance dose, µg/kg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Full-term neonate</td>
<td>20-30</td>
<td>5-8</td>
</tr>
<tr>
<td>1 month to 2 years</td>
<td>30-50</td>
<td>7.5-12</td>
</tr>
<tr>
<td>2-5 years</td>
<td>25-35</td>
<td>6-9</td>
</tr>
<tr>
<td>5-10 years</td>
<td>15-30</td>
<td>4-8</td>
</tr>
<tr>
<td>&gt;10 years</td>
<td>8-12</td>
<td>2-3</td>
</tr>
</tbody>
</table>

*Recommended dosing based on average patient response.* Recommended dosing for children with myocarditis is 75% of the usual dose, with digitalization accomplished over 24 to 36 hours.*
studies are limited to less than 2
assist devices has not been possible
obtained with the use of ventricular
Unfortunately, the long-term support
ered for infants and small children.
Because of the physical
limitations of ventricular assist
devices, ECMO should be consid-
management and as a bridge to trans-
plantation. Because of the physical
properties of ECMO, ECMO should be consid-
ered for infants and small children.
In children who weigh more
than 15 kg, ventricular assist devices
This device is particularly
European, pulsatile ventricular assist devices
(Berlin Heart EXCOR, Berlin, Ger-
many; MEDOS VAD System, Stoberg,
Germany) have shown promise in
children who weigh less than 15 kg
and are as young as 2 days old.41
In the United States, pulsatile devices
for infants and small children are
being developed.42,43

**Nursing Considerations**

Care of children with suspected
or proven viral myocarditis is chal-
enging. The responsibilities of pedi-
atrie critical care nurses are numerous
and include, but are not limited to,
airway management with adequate
oxygenation and ventilation, detec-
tion and management of arrhythmias,
ongoing monitoring of hemody-
namic parameters (including ade-
quacy of intravascular volume and
tissue oxygenation), pain manage-
ment/comfort (including assessing
therapeutic effects of medication
deliveries), prevention of complica-
tions, and family support (Table 7).

**Oxygenation, Ventilation, and
Airway Management**

Oxygen delivery should be
maximized. The method of delivery
depends on the child’s signs and
symptoms and severity of illness.
Most children will require some
form of positive pressure ventilation
via nasal cannula or face mask. Posi-
tive pressure ventilation has shown
benefit in patients with congestive
heart failure (improvement in gas
exchange, decreased work of breath-
ing, and afterload reduction of the
left ventricle).33 Positive pressure
ventilation can be noninvasive in
the form of continuous positive air-
way pressure or bilevel positive air-
way pressure. Both of these types of
positive pressure ventilation are
associated with improvement in car-
diac and respiratory functioning in
patients with acute heart failure and
can potentially postpone the need
for intubation. Long-term benefits
in children with viral myocarditis,
however, are unknown.32 More
often than not, children who arrive
in the PICU with respiratory com-
promise and low cardiac output
syndrome require intubation and
mechanical ventilation.

Nursing care of intubated children
is focused on careful monitoring of
oxygenation and ventilation and
prevention of complications. Arteri-
al blood gases are typically assessed
on a routine basis; however, contin-
uous monitoring of oxygen satu-
ration and end-tidal carbon dioxide
levels are excellent noninvasive
means for determining the effective-
ness of mechanical ventilation and
decrease the need for blood gas
analyses.42

Once correct placement of the
endotracheal tube has been estab-
lished, positioning and security of
the tube should be routinely assessed.
Such assessment can be done by
noting the centimeter mark where
the tube is taped at least once a
shift and ensuring that the tape is
secure. The depth of the endotra-
cheal tube is noted on daily chest
radiographs. The tip of the endotra-
cheal tube should be 1 to 2 cm
above the carina or at the level of
T3. All attempts should be made to
ensure neutral alignment of the
patient’s head and neck when chest
radiographs are obtained. Depth of
the endotracheal tube is altered
with neck flexion, neck extension,
and turning of the head.

Tracheal length varies markedly
with age. Infants and small children
have short tracheas. Oral to carina
lengths vary, from 12 to 14 cm in
newborns, 14 to 16 cm in 1- to 3-
year-olds, 17 to 18 cm in 8- to 10-
year-olds, and 20 to 22 cm in 16-
year-olds.43 Because of the short tra-
cheal length in small children, sim-
ple turning of the head can cause
malalignment of the endotracheal
tube or accidental extubation.
Preventing flexion or extension of
the head and neck is necessary not only
when obtaining a chest radiograph
but also when repositioning the
patient. Flexion of the neck short-
ens the airway between the mouth
carina, causing the endotracheal
tube to move downward, increasing
the possibility of migration of the
tube into the right main bronchus.
Findings would include an immedi-
ate increase in peak pressure (vol-
ume ventilation) or a decrease in
### Table 7  Myocarditis: nursing considerations

<table>
<thead>
<tr>
<th>Topic</th>
<th>Nursing considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Airway</strong></td>
<td><strong>Nursing considerations</strong></td>
</tr>
</tbody>
</table>
| Maintain patency of endotracheal tube| • Perform routine checks of tape security  
• Note depth on daily chest radiograph (1-2 cm above carina or at T3)  
• Maintain neutral head and neck alignment, particularly when repositioning and obtaining chest radiographs  
• Reassess breath sounds after every position change  
• Perform endotracheal suctioning on an as-needed basis only: increased airway pressure, decreased tidal volume, decreased oxygen saturations, secretions in endotracheal tube, rhonchi, coughing  
• Avoid instillation of normal saline with suctioning  
• Use neuromuscular blockade in conjunction with sedatives if necessary |
| Oxygenation and ventilation          | **Monitor and maintain gas exchange**  
• Continually monitor oxygen saturation, end-tidal carbon dioxide, arterial blood gases if applicable  
**Minimize oxygen demand/consumption**  
• Delay or cluster nursing interventions, depending on patient’s tolerance  
• Maintain normothermia  
• Minimize pain, anxiety  
  - Use nonsteriodals, opioids, anti-anxiolytics as needed  
  - Assess and eliminate any external, internal, or environmental factors contributing to anxiety  
  - Keep environment calm and quiet |
| Hemodynamic status                   | **Be alert for ectopy, tachyarrhythmias, bradyarrhythmias, atrioventricular blocks**  
• Differentiate between sinus tachycardia and supraventricular tachycardia (may need 12-lead electrocardiogram)  
• Have adenosine (hemodynamically stable supraventricular tachycardia) and defibrillator (hemodynamically unstable supraventricular tachycardia) available  
• Administer, evaluate, and titrate antiarrhythmics  
  - Amiodarone, lidocaine  
• Maintain pacemaker  
  - Transthoracic: ensure proper placement and adherence of electrode, change electrodes every 24 hours  
  - Transcutaneous: monitor for signs of cardiac tamponade, follow hospital’s policy for care of insertion site  
  - Keep extra battery at patient’s bedside  
**Monitor tissue perfusion**  
• Assess pulse quality, capillary refill, skin temperature and color, urine output, mental status if applicable  
**Continually monitor arterial blood pressure**  
• Be alert for fluctuations in blood pressure or hypotension  
• Administer, evaluate, and titrate inotropic and/or vasoactive infusions  
• Carefully assess extremity distal to arterial catheter for skin color, temperature, pulse, numbness, and tingling  
**Monitor central venous pressure**  
• Use distal port for monitoring if possible  
• Monitor at least hourly, continuously preferred  
• Assess for changes after delivery of fluid bolus and diuretic therapy  
• Assess in context with entire clinical picture  
  - Recognize variables that affect central venous pressure: positive pressure ventilation, positive end-expiratory pressure, conditions that increase peripheral vascular resistance, right ventricular dysfunction  
• Monitor trends, not necessarily actual numbers  
• Monitor for complications: bloodstream infection, thrombus, catheter occlusion |
| Prevention of complications          | **Monitor for hospital-acquired infection**  
• Monitor body temperature at least hourly  
• Assess white blood cell count, differential  
• Assess for changes in pulmonary secretions: color, viscosity, amount  
• Assess for changes in urine characteristics  
• Assess catheter insertion sites for redness, edema, drainage  
**Prevent alterations in skin integrity**  
• Keep skin free from moisture and secretions  
  - Use moisture-barrier products  
  - Apply topical moisturizers (free from alcohol or perfume)  
• Perform mouth care a minimum of every 8 hours  
  - Keep oral mucosa clean and moist  
• Apply artificial tears, eye lubricant as needed  
• Turn patient frequently and alleviate pressure points  
  - Use pillows, stuffed animals  
  - Use an air mattress or rotating bed  
• Alternate position of mechanical devices (oxygen saturation probes, blood pressure cuffs, electrodes) |
tidal volume (pressure ventilation), a decrease in oxygen saturation, and asymmetric chest rise. If the tube migrates into the right main bronchus, the child should immediately be placed back in the previous position and reassessed. Breath sounds should be assessed after any change in body position.

Neck extension increases airway length, potentially causing unintentional extubation because the uncuffed tube can easily pass upward through the vocal cords. Immediate respiratory distress, decrease in oxygen saturation, marked decrease in or undetectable end-tidal carbon dioxide levels, grunting, or vocalization can indicate possible inadvertent extubation. If dislodgement of the endotracheal tube into the esophagus is suspected, the tube should be removed and bag-valve-mask ventilation performed until reintubation or effective spontaneous breathing occurs.

Although a common practice in intensive care units, endotracheal suctioning should not be performed routinely. Suctioning can be associated with complications, including hypoxemia, bradycardia, atelectasis, and dysrhythmias. Suctioning should be done on an as-needed basis rather than routinely. Indications for suctioning include increase in airway pressure, decrease in tidal volume, decrease in oxygen saturation, visualization of secretions in the tube, development of rhonchi, and coughing. Hyperoxygenation may or may not be required before suctioning, depending on the child’s arterial oxygenation and tolerance to the procedure. The catheter should not be deeper than the end of the endotracheal tube. Catheters inserted to the point of resistance (carina) cause tissue inflammation and damage. Instillation of normal saline for lavage should be avoided because that practice is not supported by research and may actually be harmful.

Routine bedside nursing interventions such as dressing changes, bathing, weighing, and repositioning all significantly increase tissue oxygen consumption. Delay in carrying out these procedures may be necessary. Conversely, some or all of these activities may be clustered if tolerated in order to allow longer periods of rest.

Fever increases oxygen consumption 10% for each 1°C elevation in body temperature. Although not all fever is “bad,” treatment of fever is recommended in children with cardiopulmonary disease. Attempts should be made to maintain normothermia. Unless contraindicated, antipyretics (acetaminophen, ibuprofen) should be administered. Fever reduction can also be attempted with external cooling, usually by sponging with tepid water.

Pain and anxiety significantly increase oxygen demand and consumption and cause considerable distress for children in the PICU. Assessment and management of pain and anxiety should be ongoing.

### Arrhythmias

Heart rate and rhythm require monitoring. Arrhythmias are a significant life-threatening complication of myocarditis. Development

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**Table 7 Continued**

<table>
<thead>
<tr>
<th>Topic</th>
<th>Nursing considerations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Comfort</td>
<td>Routinely assess pain and/or agitation by using hospital-approved scales</td>
</tr>
<tr>
<td></td>
<td>Provide comfort measures</td>
</tr>
<tr>
<td></td>
<td>- Repositioning, calm, quiet environment, parents at bedside, elimination of factors potentially contributing to agitation</td>
</tr>
<tr>
<td></td>
<td>- Monitor for effectiveness after administration</td>
</tr>
<tr>
<td>Family support</td>
<td>Recognize uniqueness of parental stress</td>
</tr>
<tr>
<td></td>
<td>- Alteration in parental role</td>
</tr>
<tr>
<td></td>
<td>- Loss of control</td>
</tr>
<tr>
<td></td>
<td>- Loss of ability to care for child</td>
</tr>
<tr>
<td></td>
<td>- Disruption of parent-child relationship</td>
</tr>
<tr>
<td></td>
<td>Use Nursing Mutual Participation Model of Care</td>
</tr>
<tr>
<td></td>
<td>- Individualize nursing interventions to fit parent and family</td>
</tr>
<tr>
<td></td>
<td>- Recognize and support parent-child connectedness</td>
</tr>
<tr>
<td></td>
<td>- Empower parents</td>
</tr>
<tr>
<td></td>
<td>- Deliver concise, consistent information</td>
</tr>
<tr>
<td></td>
<td>- Provide anticipatory guidance</td>
</tr>
<tr>
<td></td>
<td>- Act as a role model</td>
</tr>
<tr>
<td></td>
<td>- Encourage parental participation in care when appropriate</td>
</tr>
<tr>
<td></td>
<td>- Encourage active parental involvement in child’s daily plan of care</td>
</tr>
</tbody>
</table>

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of bradycardia or tachyarrhythmias is unlikely to be tolerated and requires immediate recognition and treatment.

Infants and small children depend on heart rate for adequate cardiac output because stroke volume is relatively fixed; in young children, bradycardia can rapidly diminish cardiac output. Because chronotropic drugs can induce ventricular tachycardia in patients with viral myocarditis, cardiac pacing is the preferred treatment for life-threatening bradycardias, including atrioventricular block.25

Transcutaneous or transthoracic pacing is a noninvasive procedure that can be done rapidly. Two electrodes are placed on the patient with the anterior electrode in the V2 to V5 position and the posterior electrode under the scapula to the left of the spine. Electrode adherence should be checked frequently, especially if the child is diaphoretic. If needed for prolonged periods, electrodes should be changed a minimum of every 24 hours to maintain effectiveness of the contact gel.

For ongoing bradyarrhythmias, invasive pacing should be considered. Transvenous leads are placed percutaneously via a large vessel into the right atrium or ventricle. Leads are stiff; nurses must be alert for indications of ventricular perforation, including cardiac tamponade. An extra battery should be kept at the bedside of any patient who is pacemaker dependent. Hospital policy should be followed when caring for the insertion site and catheter.

Conversely, an excessively rapid heart rate will decrease ventricular filling time, resulting in inadequate preload and subsequent cardiac output. Tachycardias also result in inadequate coronary artery filling time. Coronary arteries fill and perfuse the myocardium during diastole; the faster the heart rate, the shorter is diastole, leading to a decrease in myocardial oxygen supply during a time of increasing consumption.

Differentiation between sinus tachycardia and supraventricular tachycardia is critical. Typically, sinus tachycardia is heart rate greater than 140/min in children, greater than 160/min in infants, and varies from beat to beat. Heart rate generally remains less than 200/min. Sinus tachycardia can be the result of a variety of factors, including fever, anxiety, pain, intravascular dehydration, and marked vasodilatation. Sinus tachycardia will resolve, or partially resolve, with resolution of the inciting factor.

Supraventricular tachycardia is an extremely rapid heart rate (200-280/min); is unresponsive to resolution of fever, pain, hypovolemia, and so on; and has no beat-to-beat variability. Rates of sinus tachycardia and supraventricular tachycardia can overlap, making determination of the type of tachycardia difficult. A 12-lead electrocardiogram may be necessary to differentiate between the two. Once supraventricular tachycardia has been established, rapid treatment with adenosine in the patients with stable hemodynamic status or cardioversion in patients with unstable hemodynamic status is performed. A defibrillator should remain near any child who has myocarditis or recurring supraventricular tachycardia.

**Hemodynamic Monitoring**

With invasive hemodynamic monitoring, physiological variables specific to cardiovascular function can be monitored and, when coupled with clinical examination, the results provide data for sound clinical decision making.

**Arterial Pressure.** Continuous blood pressure monitoring via an arterial catheter is preferred in children with severe cardiac dysfunction, particularly those who require infusions of vasoactive agents. Noninvasive measurements may be inaccurate or impossible to obtain because of impaired peripheral perfusion and vasoconstriction. Arterial catheters can be placed peripherally (radial artery) or centrally (femoral artery) and provide a dynamic picture of systolic, diastolic, and mean blood pressures. Because evaluations of interventions and their effectiveness will be based partly on invasive measurement of blood pressure, care must be taken to ensure accuracy of readings. Routinely inspecting for proper transducer placement (phlebotatic axis), zeroing the transducer a minimum of once a shift, inspecting for and eliminating air bubbles in the system, and using noncompliant pressure tubing will help ensure accurate measurements.

Normal blood pressure in children is dependent on age and size (see Sidebar). As previously noted, however, children with myocarditis
can maintain a blood pressure within normal limits for age until very late in the disease. No single clinical parameter should determine critical care interventions, and this rule is particularly true for blood pressure, unless hypotension is present. Adequacy of tissue perfusion is determined by analysis of clinical findings on bedside examination along with blood pressure.

The extremity distal to the arterial catheter should be assessed at least once a shift to monitor for any compromise in perfusion related to catheter placement. Extremity coolness, color changes, delayed capillary refill, and diminishing pulse quality can indicate inadequate perfusion. Numbness, tingling, and motor deficit can also be indicators of impaired circulation. Development or change in any one or more of these variables requires immediate attention and potentially removal of the catheter.

**Central Venous Pressure.** Central venous pressure (CVP) or right atrial monitoring provides a continual reflection of intravascular volume status and is a good determinant of right ventricular end-diastolic volume, or preload. CVP should be interpreted along with other measures of cardiac function (heart rate, peripheral perfusion, urine output). In critically ill children, the absolute value is not as important as serial measurements and changes in response to interventions.

CVP monitoring should be done via the distal lumen of the catheter. Monitoring can be continual or intermittent, depending on availability of intravenous access. If the distal lumen is being used for continuous intravenous administration of fluids and/or drugs, CVP should be assessed a minimum of once an hour and after any administration of fluid boluses or diuresis. When intermittent CVP monitoring is used, care must be taken to ensure that no cardiotonic medications are infused through the distal port. If such infusions are used, turning the stopcock for pressure readings causes a disruption in the delivery of medication and potential hemodynamic compromise. Ideally, infusions of inotropic or vasoactive agents should be given through the medial or proximal lumen only.

Normal CVP values can vary widely in infants and children, depending on age, disease, concomitant conditions, and critical care therapies. In healthy infants, normal values can range from -2 to
+4 mm Hg, from 4 to 8 mm Hg in those with congenital heart disease, and from 2 to 6 mm Hg in those with respiratory disease requiring mechanical ventilation. In this age group, measurements above 8 mm Hg are generally indicative of myocardial dysfunction or high intrathoracic pressure. In older children without underlying disease, a CVP range of 2 to 6 mm Hg is considered normal. Values can vary markedly and should be interpreted along with the child’s underlying disease state, concomitant conditions, and critical care therapies.

CVP measurements do not stand alone and must be considered in context of the entire clinical picture. Any changes in intrathoracic pressure will affect CVP values; spontaneous respirations decrease CVP, whereas increased intrathoracic pressure increases CVP. Values lower than expected may or may not indicate hypovolemia; high CVP values may or may not indicate hypervolemia. Elevated CVP values may also occur in patients receiving positive pressure ventilation and with the addition of positive end-expiratory pressure. Conditions that increase pulmonary vascular resistance (hypothermia, hypoxemia, acidosis, severe respiratory disease, underlying pulmonary hypertension) also increase CVP. High CVP values do not necessarily reflect cardiac disease but may be an indication of elevated right ventricular end-diastolic pressure, suggesting right ventricular dysfunction. Variables that affect interpretation of CVP values must be considered when therapies are determined.

Complications of central venous catheters include bloodstream infection (increased risk with multilumen catheters), venous thrombosis, thrombophlebitis, and catheter occlusion. Routine maintenance of the catheter and insertion site should be done per hospital policy.

Pulmonary Artery Pressure. Pulmonary artery catheters can provide valuable information in children with cardiopulmonary failure. The ability to measure pulmonary artery pressures, cardiac output/index, and systemic and pulmonary vascular resistance can provide important data for directing therapies. However, because of potential complications, pulmonary artery catheters tend to be used less often in children than in adults. Pulmonary artery catheters are therefore indicated only when the additional data
obtained will enhance clinical decision making.26

**Comfort**

Several pain intensity scales for children are available; determining which scale to use is based on the child’s age and/or ability to communicate. Self-reporting scales can be used with children who are able to express their level of pain. The faces scale is a visual scale that provides facial expressions varying from no pain (smiling) to worst pain (deep frown, crying). The child is asked to choose the facial expression that best reflects his or her pain. The pain intensity scale is a numerical scale from 1 to 10, with 1 = no pain and 10 = severe pain. It is used with older children who understand the concept of rank and order. For infants, scales designed for assessment of postoperative pain are available; these scales may or may not provide adequate assessment of other types of pain; thus subjective assessment by nurses within the context of the clinical situation is critical.

In children who are unconscious, are sedated, or require mechanical ventilation, observational scales are used. Unfortunately, these tools are not effective for assessing pain in children with chemically induced paralysis because the tools require scoring of alertness and physical movement. For children with neuromuscular blockade, bedside nurses must maintain a keen sense of awareness as to whether, given the severity of illness and the treatments in use, it is reasonable to expect that a child is experiencing pain and, if so, to treat the child accordingly.

For severe pain, opioids (morphine, fentanyl, dilaudid) remain the most commonly used class of analgesics. Children receiving opioids should be monitored not only for effectiveness of therapy but also for respiratory depression, particularly if they are dependent on spontaneous breathing, with or without mechanical ventilation. Hypotension can also occur after administration of opioids. Dosing can be adjusted accordingly, or regimens can be revised to minimize side effects. Acetaminophen, ketorolac, and ibuprofen (if not contraindicated) can be used for mild to moderate pain or as adjunct therapy in addition to opioids.

Anxiety in children is often expressed as agitation. Assessing agitation can be especially challenging. Agitation is a means of communicating physical and/or mental discomfort; many children in the PICU cannot communicate their needs. Nurses must systematically review and rule out any internal, external, or environmental factors that may be causing or contributing to agitation.

Anxiety and/or distress not relieved with comfort measures (repositioning the child, having a parent at the bedside, or quieting the environment) are typically treated with sedatives and anxiolytics (lorazepam, midazolam). These medications alleviate agitation and can provide tolerance of invasive procedures and intensive care therapies. Additionally, these medications provide a lack of awareness, and subsequent memory, of what can be an extremely frightening experience. The need for chemically induced paralysis must be assessed on an individual basis. Although some children maintain ventilator synchrony with sedation and analgesia alone, others may require neuromuscular blockade to achieve synchrony. Neuromuscular blocking agents can be given intermittently or as a continuous infusion. Before a chemical paralytic agent is administered, patency of the airway and adequacy of sedation and analgesia must be established. Neuromuscular blockade should never be used without concomitant administration of an opioid and/or an anxiolytic, whether the paralytic agent is given as a one-time dose or as a continuous infusion. Train-of-4 monitoring should be routinely assessed for all children receiving continuous infusions of chemical paralytics, and dosing should be titrated to the ordered level of neuromuscular blockade.

**Acquired Infection**

Hospital-acquired infections are a constant threat for any critically ill child in the PICU. Acquired nosocomial infection coupled with extreme severity of illness in a child with myocarditis significantly increases morbidity and mortality. Nurses’ diligence in preventing and monitoring for infection is essential.

Within hours of admission, hospital-associated bacteria can be detected on skin and in the respiratory and genitourinary tracts. Invasive devices, antibiotic use, and/or critical care procedures increase the risk of bacterial invasion. Some of the highest rates of hospital-acquired infection occur in PICUs; bloodstream infection and ventilator-associated pneumonia are the most common.45

Fever is often the first sign of infection in children; however, infants
often have decreases in body temperature because of the immaturity of the brain’s temperature-regulating center. Monitoring of temperature changes in this age group can be complicated by the use of radiant warmers, designed to maintain normothermia. Radiant warmer temperature is adjusted according to an infant’s skin temperature; consequently, decreases in body temperature related to infection may be masked. Although skin temperature is continuously monitored in infants under a radiant warmer, core body temperature should be monitored a minimum of once an hour, if not continuously, in all age groups.

White blood cell count with a differential count should be assessed closely. Leukocytosis (elevation) or leukopenia (decrease) develop in children with infection, depending on age. A normal total white blood cell count is 5000 to 10,000 cells/μL but varies depending on the age of the child; the younger the child, the higher the upper range limit. Monitoring trends is as important as getting absolute counts.

The differential count provides the percentage of each subset of white blood cells (eosinophils, neutrophils, basophils, monocytes, and lymphocytes) and can aid in differentiating the type of infection. In older infants and children, a “shift to the left,” or an excess of immature neutrophils (bands), usually indicates bacterial infection. An increase in the number or percentage of bands along with fever may warrant further workup (eg, blood cultures for detection of secondary bacterial infection). Coagulase-negative staphylococci are the most common cause of nosocomial bloodstream infections in the PICU. Unlike older children, young infants commonly have neutropenia when infected because of their small storage pools and inability to produce white blood cells at a fast rate.

Pulmonary secretions should be monitored for changes in viscosity, color, and amount. Changes in the character of the secretions along with evidence of new pulmonary infiltrates may indicate the development of ventilator-associated pneumonia (the second most common hospital-acquired infection in PICUs) in intubated patients. Although nosocomial urinary tract infections occur less often than do acquired bloodstream or pulmonary infections in children, urine should be routinely examined for color, odor, and changes in opacity, particularly if a child has a urinary catheter in place.

Any invasive catheter is a potential source of infection. Insertion sites should be routinely inspected for edema, redness, and drainage. Although central venous catheters are not routinely changed in children, signs of infection at the site may warrant removal of the catheter. Factors increasing the risk for bloodstream infection include multilumen catheters, repeated catheterizations, and certain types of dressing. In order to minimize risk of infection, catheters should be manipulated as little as possible and should be removed as soon as they are no longer needed.

Immobility and alteration in tissue perfusion increase the potential for skin breakdown. Skin should be kept free from exposure to moisture and secretions; moisture-barrier products can be applied to the perineal area. Application of topical moisturizers, with avoidance of products containing perfume or alcohol, aids in maintaining skin hydration. Mouth care, a minimum of every 8 hours, helps keep oral mucosa clean and moist. In children without a blink response (chemically paralyzed) or with incomplete lid closure, the eyes should be kept moist with artificial tears and/or lubricant as needed. Frequent turning (as tolerated), alleviation of pressure points on bony prominences (use of pillows, stuffed animals), and alternating pressure points related to mechanical devices (oxygen saturation probes, blood pressure cuffs, electrodes) should be performed on a routine basis. Air mattresses or rotating beds can also be used to prevent skin breakdown. Any evidence of altered skin integrity should be immediately addressed.

**Family Support**

As with all children admitted to the PICU, the bedside nurse has an essential role in providing ongoing support and education to the patient’s family. Emergent admission to the PICU creates feelings of overwhelming shock and disbelief associated with helplessness. The possibility of death is real and frightening. Adding to parental stress is alteration in the parental role, that is, loss of control and/or ability to care for their child. Alteration in parenting and disruption of the parent-child relationship are deemed the most stressful characteristics of a PICU admission.

Use of the Nursing Mutual Participation Model of Care can help nurture a trusting environment, establish effective communication patterns, and limit parental powerlessness.
The survival rate in children and adults with myocarditis is approximately 80%.46 Lee et al10 found that patients who died almost always did so within the first few hospital days. Survival after 72 hours in patients not requiring ECMO is as high as 97%.10 Some centers have had a 1-year survival rate of 80% in children who required mechanical circulatory support.3

Unfortunately, survival to discharge does not imply full recovery. Although Lee et al found that recovery of ventricular function without the need for ongoing cardiac medications or restrictions in physical activity was the norm, some evidence suggests that myocarditis can progress to dilated cardiomyopathy.30 Viral persistence in the myocardium has been associated with progressive impairment of left ventricular function and is predictive of adverse outcomes.46,49 Progression from myocarditis to dilated cardiomyopathy is further supported by the finding of a coxsackievirus B–adenovirus receptor gene present in larger amounts in the myocardium of patients with dilated cardiomyopathy than in healthy hearts.30 The discovery of this receptor also suggests a role in not only the viral origin of myocarditis but also the occasional “familial” case of myocarditis.15 Survival in children with dilated cardiomyopathy ranges from 60% to 70% at 1 year to 34% to 56% at 5 years. Gagliardi et al36 reported that in children with dilated cardiomyopathy, the first 2 years after diagnosis are the most crucial.36 Favorable events (nearly complete recovery) and worse outcomes (death or need for a transplant) occur during this period.

For children with residual myocardial dysfunction at discharge, diuretics and afterload reducers are considered first-line agents. Diuretics are used for congestive heart failure of any cause because they reduce respiratory signs and symptoms associated with heart failure.33 Thiazides (diuril, hydrochlorothiazide) are often used; the addition of spironolactone may obviate potassium supplementation. Angiotensin-converting enzyme inhibitors (captopril and enalapril) may also improve chronic signs and symptoms24 (Table 8).

The use of digoxin in patients with chronic congestive heart failure as a result of myocarditis remains controversial. The poor diastolic function associated with myocarditis is not improved with digoxin.33 Digoxin also increases the risk of ventricular arrhythmias if myocardial inflammation is still present. If digoxin is used, digitalization should be accomplished during a 24- to 36-hour period with 75% of the usual total loading dose to minimize the possibility of arrhythmia development.24

Beta-blockade may improve outcome in children with dilated cardiomyopathy. Carvedilol, a non-selective β-blocker used in adults, has been examined as an adjunct therapy for management of heart failure in children. Carvedilol may improve congestive heart failure through several mechanisms, including decreased heart rate leading to improved myocardial oxygen consumption and enhanced coronary artery blood flow, improvement in left ventricular ejection fraction, and reduction in neurohormonal vasoconstrictor systems. Carvedilol may also prevent ventricular arrhythmias and sudden death.
Use of carvedilol in children remains controversial. Bruns et al.\(^1\) studied the use of carvedilol in 46 children with heart failure in 6 different centers and found improvement in left ventricular function and overall signs and symptoms in 67%. Schwartz and Wessel\(^3\) also examined use of carvedilol in children with heart failure and found no statistically significant effect; left ventricular ejection fraction improved in both the carvedilol and the placebo groups. Although carvedilol is effective in reducing mortality in adults, more research into the use of carvedilol is needed in children with heart failure.

Transplantation is sometimes the only option for children with significant cardiac failure or dilated cardiomyopathy due to myocarditis. Preferably, children are not put on a list for transplantation until they have progressed to the chronic phase of the disease, because recovery is possible in even the most severe cases.\(^2\) Transplantation may, however, be considered an option for a child who requires ECMO for more than 10 days and has no clinical signs of improvement.\(^a\)

**Conclusion**

Viral myocarditis remains an uncommon but challenging illness. In children, the initial signs and symptoms are often those of common pediatric illnesses and without any obvious cardiac signs. Adding to the challenge is the potential inability of the child to convey precise symptoms or describe location or intensity of pain or discomfort. A history of asthma or congenital heart disease can skew the clinical picture.

Although few children actually have viral myocarditis diagnosed and require admission to the PICU, practitioners must maintain a heightened sense of awareness for the “zebra in the herd of horses.” Concomitantly, for any previously healthy child with sudden cardiogenic shock, myocarditis must be included in the differential diagnosis.

Most children with myocarditis recover without requiring admission to the PICU; only a small percentage progress to development of significant cardiopulmonary compromise that requires intensive cardiopulmonary support and monitoring. Critical care management is aimed at supportive rather than curative measures and depends on the severity of myocardial dysfunction. The most common techniques used in the PICU for children with cardiovascular compromise or collapse include invasive hemodynamic monitoring, infusions of inotropic agents, antiarrhythmics, afterload reducers, mechanical ventilation, prevention of complications, and, when a child is unresponsive to ordinary PICU therapies, extracorporeal life support.

**References**

16. Pres S, Lippkind RS. Acute myocarditis in

CE Test  Test ID C0812: Viral Myocarditis in Children

Learning objectives: 1. Identify which population is at most risk of death as the result of myocarditis  2. Identify the mechanism that results in morbidity and mortality in children with myocarditis  3. Discuss the signs and symptoms of myocarditis in children  4. Describe the treatment of patients with myocarditis

1. The mortality rate in infants with myocarditis can be as high as what percentage?  a. 25%  b. 50%  c. 75%  d. 95%

2. The incidence of myocarditis is more prevalent in which of the following?  a. Infants and adolescents  b. Infants and toddlers  c. Infants and young adults  d. Adolescents and young adults

3. Which of the following plays a lead role in myocyte injury in patients with myocarditis?  a. The fever associated with the infection  b. The autoimmune reaction  c. The exaggerated healing process forms scar tissue within the myocyte  d. None of the above


5. Which of the following types of myocarditis results in worse outcomes?  a. Chronic  b. Acute  c. Fulminant  d. Latent

6. Which of the following characterize the signs and symptoms of myocarditis in children?  a. Easy to recognize and easy confirm diagnose  b. Easy to recognize and hard to confirm diagnose  c. Hard to recognize and hard to confirm diagnosis  d. Hard to recognize and challenging to confirm diagnosis

7. Which of the following statements is true for children with myocarditis?  a. Hypotension is an early sign of myocarditis.  b. Hypotension is always present with myocarditis.  c. Hypotension is extremely rare with myocarditis.  d. Hypotension is a late and ominous sign.

8. Which of the following are common rhythm issues in children with myocarditis?  a. Supraventricular tachycardia and premature ventricular contractions  b. Bradycardia with AV blocks  c. Bradycardia with ventricular tachycardia  d. None of the above


12. Endotracheal suctioning should not be performed routinely due to which of the following?  a. The increased association with bradycardia and atelectasis  b. The increased incidence of accidental extubation  c. The increased association with dysrhythmias and tachycardia  d. None of the above—routine suctioning is a good thing

Test answers: Mark only one box for your answer to each question. You may photocopy this form.

1. 2. 3. 4. 5. 6. 7. 8. 9. 10. 11. 12.

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