

## Pathophysiology

When prevention efforts are not effective and conditions are favorable, the organisms proliferate and form potent exotoxins, one of which is tetanospasmin. Tetanospasmin affects the CNS to produce the clinical manifestations of the disease. The ideal conditions for the organisms' growth are devitalized tissues without access to air, such as wounds that have not been washed or kept clean and those that have crusted over, trapping pus beneath. The exotoxin appears to reach the CNS by way of either the neuron axons or the vascular system. The toxin becomes fixed on nerve cells of the anterior horn of the spinal cord and the brainstem. The toxin acts at the myoneural junction to produce muscular stiffness and lower the threshold for reflex excitability.

The incubation period for tetanus varies from 3 days to 3 weeks and averages 8 days; most cases occur within 14 days. In neonates, it is usually 5 to 14 days. Shorter incubation periods have been associated with more heavily contaminated wounds, more severe disease, and a worse prognosis (American Academy of Pediatrics, Committee on Infectious Diseases, and [Pickering, 2012](#)).

The manner of onset varies, but the initial symptoms are usually a progressive stiffness and tenderness of the muscles in the neck and jaw. Eventually, all voluntary muscles are affected ([Box 30-11](#)). As the child recovers from the disease, the paroxysms become less frequent and gradually subside. Survival beyond 4 days usually indicates recovery, but complete recovery may require weeks.

### **Box 30-11**

## **Clinical Manifestations of Tetanus**

### **Initial Symptoms**

Progressive stiffness and tenderness of muscles in neck and jaw

Characteristic difficulty in opening the mouth (trismus)

Risus sardonicus (sardonic smile) caused by facial muscle spasm

### **Progressive Involvement**