but the symptoms gradually subside over the first few months of life and disappear by 12 months old (Sarnat, 2016c).

Pathophysiology

GBS is an immune-mediated disease often associated with a number of viral or bacterial infections or the administration of certain vaccines. It has been associated with infectious mononucleosis, measles, mumps, *Campylobacter jejuni* (gastroenteritis), cytomegalovirus, *Borrelia burgdorferi* (Lyme disease), Epstein-Barr virus, *Helicobacter pylori,* and *Mycoplasma* and *Pneumocystis* infections. Onset of GBS symptoms usually occurs within 10 days of the primary infection. Pathologic changes in spinal and cranial nerves consist of inflammation and edema with rapid, segmented demyelination and compression of nerve roots within the dural sheath. Nerve conduction is impaired, producing ascending partial or complete paralysis of muscles innervated by the involved nerves. GBS has three phases:

1. **Acute:** Phase starts when symptoms begin and continues until new symptoms stop appearing or deterioration ceases; it may last as long as 4 weeks.

2. **Plateau:** Symptoms remain constant without further deterioration; it may last from days to weeks.

3. **Recovery:** Patient begins to improve and progress to optimal recovery; it usually lasts a few weeks to months depending on the deficits incurred by the illness.

Diagnostic Evaluation

The diagnosis of GBS is based on clinical manifestations (Box 30-10), CSF analysis, and EMG findings. CSF analysis reveals an abnormally elevated protein concentration, normal glucose, and fewer than 10 white blood cells (WBCs)/mm^3 (Sarnat, 2016c). EMG shows evidence of acute muscle denervation, but other laboratory studies are usually noncontributory. The symmetric nature of the paralysis helps differentiate this disorder from spinal paralytic poliomyelitis, which usually affects sporadic muscles.