The Challenges of Managing and Treating Guillain-Barré Syndrome During the Acute Phase

Stephanie B. Atkinson, RN, MS, ANP-C; Rebecca Lamb Carr, PhD, APRN, BC; Patricia Maybee, EdD, FNP, FAANP; Donna Haynes, PhD, APRN, BC, FNP

Guillain-Barré syndrome (GBS) is a randomly acquired inflammatory disease that affects approximately 2 persons in 100,000 annually. There have been no discriminating risk factors identified including age, sex, or race. The syndrome results in the demyelination of peripheral nerves, which leads to progressive motor weakness and paralysis. The critical care nurse should gain from this article an overview of Guillain-Barré syndrome during the acute phase. Included is the pathophysiology of the syndrome, clinical presentation, acute phase nursing assessment and management, and currently available treatment options.

Keywords: Guillain-Barré syndrome, Neurologic disease, Paralysis, Muscle weakness, Central nervous system diseases

Guillain-Barré syndrome (GBS) is a randomly acquired inflammatory disease that affects 2 persons in 100,000 annually. It results in the demyelination of peripheral nerves, which leads to progressive motor weakness and sensory abnormalities. Symptoms typically start in the legs and ascend symmetrically to the upper body. It can strike anyone regardless of age, sex, or race. Two populations, men and people ages 50 to 74 years, have experienced a slightly higher incidence.

A definitive cause of GBS has not been identified. In two thirds of patients affected, an acute illness, either viral or bacterial, precedes the neuropathy. The antecedent event usually occurs 1 to 3 weeks before the onset of symptoms. Currently, the treatment options are aimed at decreasing the duration and severity of the disease. These options include intravenous steroids, immunotherapy, plasma exchange, and cerebrospinal fluid (CSF) filtration. Research does not indicate which treatment option is most efficacious. The lack of a definitive cause and most effective treatment makes caring for the GBS patient a challenge. This article focuses on the care provided by critical care nurses during the acute phase of the disease. An overview of GBS, pathophysiology of the disease, treatment options, and nursing care will be discussed.

PATHOPHYSIOLOGY OF GBS
Guillain-Barré syndrome is an acute idiopathic polyneuritis that usually occurs symmetrically and results in ascending paralysis. The disorder causes demyelination, which is the destruction of the myelin sheath. The function of the myelin sheath is to speed up the transmission of nerve signals and allow them to travel long distances.
An autoimmune reaction directed at the peripheral nerve myelin and Schwann cells is thought to be the cause of the neurologic dysfunctions in GBS. Schwann cells are responsible for producing the myelin, a lipoprotein that insulates the axon.

Schwann cells are responsible for producing the myelin, a lipoprotein that insulates the axon.

The cause of the autoimmune attack on the peripheral nerves is idiopathic. Two thirds of the patients with GBS are known to have experienced an acute infectious illness that precipitates the onset of symptoms. Campylobacter jejuni, a bacteria commonly associated with gastroenteritis, has been identified as the most frequent antecedent pathogen of GBS. Other common pathogens include cytomegalovirus, Epstein-Barr virus, and Mycoplasma pneumoniae. A small number of case series and case reports have suggested a possible link between GBS and vaccinations, including oral poliovirus, influenza, measles, and diphtheria-tetanus-pertussis, but to date, no causal relationship has been established.

There are 3 theories supporting a viral link as the precipitating factor in the pathophysiology of GBS. The first theory is that the viral-like illness creates an autoimmune response that interferes with the T-suppressor cell circuits. The T-suppressor cell interference leads to the body's inability to suppress the inappropriate autoimmune response. This results in the demyelination of the peripheral nerves. The second theory is that the original antigen from the illness has similar cell surface markers as the myelin, which leads the body to mistakenly attack itself. The third and least popular theory is that a direct viral invasion of the spinal and cranial nerves leads to GBS.

Once the precipitating event has occurred, macrophages migrate into the areas adjacent to the nerve and attack the myelin surrounding the nerve fibers. This results in variable degrees of demyelination along the nerve segments. Consequently, the most heavily myelinated peripheral nerves, such as the motor and joint position sensory nerves, are more severely affected. The nerves that are unmyelinated or less myelinated, such as those involved in temperature control and pain, are less severely affected. Muscle weakness and paralysis are a result of the inappropriate immune response that leads to demyelination. The demyelination is self-limiting, and once it stops, the Schwann cells rebuild the lost insulation. Remyelination, or recovery, occurs in a reverse process when the nerves affected last recover and become functional.

**CLINICAL PRESENTATION**

There are 2 clinical features that must be present for a diagnosis of GBS. These features are progressive motor weakness of more than 1 limb and definitive hyporeflexia or areflexia (loss of tendon jerks). Several other signs and symptoms such as rapid, symmetrical progression of motor weakness and cranial nerve involvement are suggestive of the diagnosis. It can be difficult to differentiate GBS from other demyelinating disorders such as multiple sclerosis. In multiple sclerosis, neurologic deficits, such as impaired vision, vertigo, and short-term memory loss, are the initial symptoms. Symptoms usually remit, partially or completely, weeks after the onset of the episode. Multiple sclerosis is characterized by periods of relapse and remission, in comparison to a gradual worsening of symptoms and then plateau seen in GBS.

Four clinical variants of GBS exist. The first and most common variant is ascending GBS. Ascending GBS usually presents with muscle weakness, numbness, tingling, and leg pain that advances upward to the trunk and then the arms and/or cranial nerves. Deep tendon reflexes may be diminished or absent. Ascending is thought to be the most commonly seen variant because the distances the nerve impulses must travel are longest and, therefore, the most vulnerable to neurologic dysfunction. With this presentation, the symptoms are typically seen in the lower extremities first. The second clinical variant, pure motor GBS, involves the exacerbation of muscle weakness with no associated muscle pain. The third, descending GBS, is the least common and involves weakness starting at the cranial nerves and progressing downward to the respiratory muscles, trunk, and extremities. Normal reflexes are present. Common presenting symptoms of descending GBS are facial paralysis, dysphagia, and ophthalmoplegia. This variant of GBS is different from the common disorder of Bell palsy because it typically presents with unilateral paralysis of the facial nerve. A fourth form of GBS, the Miller-Fisher variant or encephalomyeloradiculopathy, results in weakness of eye movements and limb and gait ataxia with minimal or no muscle weakness and normal sensory function.

Guillain-Barré syndrome is divided into 3 phases. The acute phase begins with the onset of symptoms and continues with rapid progression of the disease until no further symptoms of deterioration occur. The acute phase can last up to 4 weeks, and symptoms include pain, muscle weakness with paralysis, and possible respiratory dysfunction. The second phase is the plateau phase when symptoms remain the same as the acute phase and do not worsen. This stage can last a few days to a few weeks.
The third phase, recovery phase, begins as the patient’s condition starts to improve until recovery. During this time, the patient will regain the use of the affected extremities, breathe spontaneously, and begin to function independently again. Patients may experience residual deficits such as weakness and fatigue for months to years afterward. This stage lasts a few weeks to 2 years. The overall recovery rate is highly variable. Literature reports an overall recovery of 48% to 90% at 1 year and 60% to 88% at 2 years. Predictors of a poor recovery include age older than 60, rapidly progressive disease, axonal loss, and prolonged mechanical ventilation.

**MEDICAL MANAGEMENT**

Presently, 4 therapies exist that are thought to lessen the severity of GBS and accelerate the recovery. However, research has not been able to demonstrate that one treatment is superior. Current treatments include corticosteroids, intravenous immunoglobulins, plasma exchange, and CSF filtration.

The oldest treatment option is the use of corticosteroids. The steroid's method of action on the pathophysiology of GBS is unknown. One theory is that steroids inhibit the inflammation associated with GBS. Steroids have been used in the treatment of GBS because they are beneficial in the treatment of other demyelinating diseases with features similar to those of GBS. The largest study examining the use of corticosteroids as an effective treatment option for GBS included 243 participants each randomly assigned to either a placebo group or the experimental group receiving intravenous methylprednisolone 500 mg daily for 5 days. The primary outcome measure selected was the improvement in disability grade 4 weeks after treatment. After taking into account initial disability grade, age, and sex, there was only a 0.06 grade difference between the placebo group and the experimental group receiving intravenous methylprednisolone.

Corticosteroids have also been studied in combination with other therapies. One group of researchers compared oral prednisolone or prednisone tablets with a placebo. The oral dosage regimen varied in the studies but both consisted of the equivalent of at least 40 mg of steroid daily for 2 weeks. The conclusions of these studies did not support corticosteroids as an effective treatment.

The dosage of intravenous immunoglobulin is usually 1 to 2 mg per kilogram of body weight in divided doses over 3 to 5 days. The infusion is started slowly and is increased based on the patient’s tolerance and the total dose. During administration, patients must be monitored for anaphylaxis, chills, headache, hypotension, skin reactions, neutropenia, and fluid overload. Most research has focused on comparing IVlg with plasma exchange. Two trials compared IVlg with supportive care only. The sample sizes of each (N = 18 and N = 20) were too small to reach any conclusions. Immunoglobulin G has been used as a treatment choice for treating GBS in the acute phase.

A third treatment option is plasma exchange. Plasma exchange is a technique that separates plasma from cells using membrane filtration or centrifugation. The patient’s
plasma is separated selectively from whole blood, and its abnormal constituents are washed out, or the plasma is exchanged with normal plasma or a colloidal substitute such as albumin. Plasma exchange is performed over a 10- to 15-day period. The plasma volume is 40 mL per kilogram of body weight per treatment. Plasma exchange should begin days or less after the onset of symptoms. However, patients may benefit from plasma exchange up to 30 days after disease onset. Plasma exchange is often reserved for more severe cases of GBS for several reasons. It is more expensive in comparison to other treatments. Plasma exchange requires special equipment and personnel to perform that may not be readily available at all hospitals. There is also an increased risk for infection because of the need for venous access for exchange.

Review of the research over the past 20 years supports plasma exchange use in the treatment of GBS versus supportive care alone. In one study, 91 acute phase GBS patients who were able to stand up alone or walk with assistance (disability grade 1 to 3) were randomized to receive either 2 sessions of plasma exchange in 3 days or supportive care only. In the treatment group, the median time of onset of motor recovery was significantly shortened compared with the control group (8 vs 4 days). The number of patients with 1 or more grades of improvement at 1 month was significantly more, 26 out of 45 in the treated group compared with 13 out of 46 in the control group. In yet another study, the researchers concluded that in mild GBS cases, 2 sessions of plasma exchange are superior to supportive care only. Plasma exchange has shown to accelerate recovery in comparison to supportive care alone. Plasma exchange, like immunoglobulin G, is being used as a first-line therapy for treating GBS.

Several comparison studies exist between IVIg and plasma exchange. In 1997, the Plasma Exchange/Sandoglobulin GBS Trial group examined 379 patients, of which 130 patients received IVIg alone, 121 received plasma exchange alone, and 128 received both treatments. The primary outcome measure was the disability grade after 4 weeks. Statistically, there were no significant differences in mean grade change between the groups (0.8 in the IVIg group and 0.9 in the plasma exchange group). A small but insignificant difference was found in the combination group over each of the single treatments (1.1). The findings of the trial signify that the 2 groups did not differ significantly with regard to outcome measures. Another study included 23 subjects who were randomized to the IVIg group and 24 to the plasma exchange group. No significant differences were found between the groups in any of the measured outcomes, which included (1) proportion of patients improved by 1 or more disability grades after 4 weeks, (2) change in disability grade after 4 weeks, (3) time until improvement by 1 disability grade, and (4) time until improvement by 2 disability grades. Using the disability grades as an outcome measure showed that there was no significant difference in the outcome between plasma exchange and IVIg.

A fourth treatment option is the use of CSF filtration. During CSF filtration, CSF is removed via a spinal catheter and passed through a sterile filter system and reinfused into the patient. The filter is designed to eliminate cells, bacteria, endotoxins, immunoglobulins, and inflammatory mediators that may be present. Side effects were minimal, including headache and pleocytosis. One group of researchers conducted a prospective clinical trial with 37 acute GBS who were randomized to either received CSF filtration or plasma exchange. The results, though preliminary, showed the CSF filtration to be as effective as plasma exchange. However, further studies examining this new treatment are needed.

Based on the above research, there is no superior treatment of GBS. Research has found that corticosteroids are ineffective treatment and are reserved for use in treating other underlying problems. Both plasma exchange and IVIg have shown some improvement in the outcomes of GBS patients. Plasma exchange has been shown to be of benefit during the acute phase of GBS. The barriers to this treatment option are the risk for infection, cost, and length of treatment, usually 8 to 14 days to be of benefit. Intravenous immunoglobulin G has been shown to be as effective as plasma exchange. It is easy to administer, readily available, less invasive, and has fewer side effects than plasma exchange. Intravenous immunoglobulin G is usually well tolerated by the patient. The main limitation of IVIg is that it is expensive.

The treatment choice in GBS must be individualized, taking into account the severity of the syndrome, cost, and possible side effects coupled with the enormous cost of intensive care and rehabilitation. Continued research into these and other possible treatment options are warranted because a portion of GBS patients have residual effects from the illness. More intensive and effective treatments are needed to improve the prognosis of these patients.

**NURSING MANAGEMENT**

Patients with GBS require complex nursing care. The goals of management and treatment of GBS include accelerating recovery, decreasing complications during the acute phase, and decreasing the amount of long-term residual neurological deficits. Of the patients affected, 5% to 10% will die and the other 90% to 95% will eventually recover with treatment over a couple of months to years; some patients will have residual
symptoms, however.\textsuperscript{2,3} Mortality is related to complications such as adult respiratory distress syndrome, sepsis, pulmonary emboli, and cardiac arrest.\textsuperscript{4} Nursing care during the acute phase should focus on both the physical and psychosocial needs of the GBS patient. Physically, the patient is experiencing multiple system involvement and psychologically high levels of fear and anxiety exist. In this section, nursing management including implementing appropriate nursing interventions and monitoring for complications in the GBS patient during the acute phase will be discussed.

Pain is one of the most frequent and difficult symptoms of GBS to treat. Although the patient has extensive paralysis, there is no associated numbness. Types of pain the patient may experience are paresthesia, deep muscle aches, and muscle stiffness.\textsuperscript{2} Paresthesia includes numbness, prickly, tingling, and burning sensations, especially in the feet and hands. Hyperesthesia, or extreme sensitivity, can cause the touch of a hand or bed sheet to be extremely painful. Suitable analgesics for treatment of pain range from nonsteroidal anti-inflammatory agents to opioids.\textsuperscript{1} Neuropathic pain, which is the result of the alteration in nerve function, can be reduced with medications such as gabapentin or a tricyclic antidepressants.\textsuperscript{2}

There are several nonpharmacological interventions that have been shown to be beneficial. Nonpharmacological measures including passive range of motion, massage, and heat can also be used to decrease the patient’s pain.\textsuperscript{1} Complementary and alternative medicine may also help the patient’s pain control. Techniques may include hypnosis, imagery, relaxation techniques, and biofeedback.\textsuperscript{24} Uncontrolled pain in GBS can result in physical and mental effects, such as anxiety, depression, anger, and agitation. Expeditious treatment of the uncontrolled pain by the nurses is necessary to reduce these effects.\textsuperscript{1}

Fear and anxiety are 2 psychosocial components that the GBS patient experiences. Both components may be caused by knowledge deficit, pain, the inability to communicate effectively, a loss of control, and frequent thoughts of dying. Reducing fear and anxiety can be accomplished by starting to teach the patient and family immediately. Answering questions with clear answers and resolving any misconceptions they may have facilitates understanding and increases the sense of control. Reassurance and keeping the patient and family informed of the plan of care may reduce anxiety of both the patient and the family members. Routine visits from family to share feelings, hope, and information can also reduce fear and feelings of isolation.\textsuperscript{1} The patient should be allowed to make as many decisions as possible. Assigning a primary nurse to the client may give the patient and family more security in the knowledge that there is one person from whom they can get consistent information.\textsuperscript{18} Similar to pain, nonpharmacologic measures such as massage, biofeedback, and music therapy can be used to manage feelings of anxiety and fear.\textsuperscript{1,24} Many patients will become depressed and should be treated with an appropriate antidepressant.\textsuperscript{25}

Many GBS patients and family members experience anxiety related to the financial burden the disease places on them. The patient and family should meet with a social worker early in the illness to discuss concerns related to insurance coverage and loss of income related to disability. The social worker can discuss options related to community and governmental resources and assistance.\textsuperscript{1} Pastoral care and support groups may also be of benefit to the anxious and stressed families.\textsuperscript{2}

The inability of the GBS patient to communicate with family members and medical staff can also be a major source of fear and anxiety. Interventions such as lip reading, letter and picture boards, and nonverbal options such as eye blinking should be used when possible. A suitable call device such as one that requires minimal pressure to activate or a sip-and-puff system should be supplied to enable the patient to contact the nurse.\textsuperscript{1,2} Fear and feelings of insecurity can prompt the patient to demand more of the nurse’s attention. The nurse can reassure the patient with communication and letting the patient know specific times when the nurse will return to the patient’s room. The nurse should also consider allowing the family to spend more time with the patient.\textsuperscript{18} Family support has been shown to decrease the patient’s level of anxiety and fear. These interventions can provide comfort and reassurance to the patient thereby decreasing anxiety.

Sleep deprivation is very common in the intensive care unit setting and may exacerbate the fear and pain felt by the patient. The unfamiliar environment, chronic sleep deprivation, pain, and the feeling of being trapped in the patient’s own body can lead to intensive care unit psychosis. Nurses should monitor the patient’s sleep schedule and administer sedatives and hypnotics when needed. Procedures, assessments, therapy, and visiting times should be clustered together to provide periods of uninterrupted sleep for the patient.\textsuperscript{1} Turning off lights and providing a quiet environment can also promote sleep.\textsuperscript{18}

Patients with GBS may experience malnutrition related to immobility, dysphagia, decreased gastric motility, and depression. During the acute phase, enteric or parenteral nutrition may be required.\textsuperscript{1} Enteral feedings are the diet of choice because they maintain the integrity and immunologic role of the intestine, serve as prophylaxis against stress ulcers, are less expensive, and eliminate the risk of sepsis and pneumothorax associated with parenteral nutrition.\textsuperscript{26} High-protein, high-energy...
enteral formulas are preferred because they deliver the energy needed to maintain basal metabolic rate and prevent muscle wasting. Complications associated with enteral feeding include aspiration, delayed gastric emptying, paralytic ileus, diarrhea, and constipation. Aspiration is a complication that can be fatal. Elevating the head of the bed to 30 degrees, checking for residuals routinely, and stopping the feeding 30 minutes before positioning the patient flat for care or procedures are interventions that will reduce the risk of aspiration.

Important indicators of nutritional status are daily weights, serum albumin, and total protein counts. The nurse and registered dietician are responsible for routinely assessing the nutritional status of the GBS patient.

The potential for impaired skin integrity for the GBS patient also poses a challenge to the nurse because of the patient’s complete or partial paralysis and poor nutritional status. Skin integrity is directly related to the patient’s nutritional status because adequate energy and nutrients such as protein, vitamin C, potassium, zinc, and magnesium are needed to maintain skin integrity and repair damaged or broken skin such as pressure ulcers. Interventions to maintain skin integrity include (1) frequent turning and repositioning as frequently as every 30 minutes, (2) applying lotion on bony prominences, (3) massage, (4) range of motion exercises, and (5) keeping the skin dry and sheets unwrinkled. Air mattresses, specialty beds, and heel and elbow pads should be used to relieve pressure when indicated. Specialty beds may include kinetic beds that are in continuous motion and are able to rotate the patient. Pressure reducing air mattresses are also available. Skin assessments should be completed twice daily or more frequently during the acute phase of GBS to prevent complications such as ulceration and infection.

Regular physical therapy including range of motion exercises will help to prevent joint contractures and reduce muscle strength loss in the GBS patient during the acute phase. Full range of motion of joints should be completed at least every 8 hours. Isometric and isotonic exercises of uninvolved or partially involved muscle groups should accompany range of motion exercises to prevent or correct joint contractures. Positioning the joint in the neutral position with splints may be indicated to prevent or correct joint contractures also. Padded footboards or bunny boots to prevent foot drop and supportive wrist splints to prevent hyperflexion should be used. Family members should be encouraged to participate in this aspect of the patient’s care to ease anxiety and help them with gaining sense of control. As the patient recovers, the family can take on a more active role in their care. Working together, the nurse, family, and physical therapist can reduce the amount of muscle strength and range of motion lost in the GBS patient.

Respiratory muscle dysfunction is a serious complication that places the GBS patient at high risk for complications such as pneumonia, atelectasis, and aspiration. The nurse should assess the patient’s respiratory function frequently and be prepared to intervene when necessary. Interventions may include increased suctioning, chest percussion to loosen secretions, and oxygen therapy. Cough and swallowing reflexes should be assessed routinely to prevent choking and aspiration. Signs and symptoms of respiratory tract infections, such as pleuritic-type chest pain, dyspnea, decreased oxygen saturation, and blood tinged or green sputum, should be monitored.

Twenty-five percent of the patients affected by GBS will require mechanical ventilation related to respiratory failure. Mechanical ventilation may have several negative effects on patients including increased risk for infection and increased morbidity. Once intubated, GBS patients may feel dependent on the ventilator for breathing and fear suffocation when there is no physiologic basis for this dependence. Both physical and mental readiness will determine the patient’s capability regarding the timing of mechanical ventilator weaning. Weaning criteria includes an appropriate vital capacity and negative inspiratory force when breathing unassisted by ventilator, resolution of pharyngeal paralysis, and a productive cough. If the patient cannot be extubated after 10 to 14 days of oral intubation, a tracheostomy may be indicated.

Disturbance in the autonomic nervous system is another potential complication of GBS. Autonomic dysfunction may include cardiac arrhythmias, labile blood pressure, abnormal hemodynamic responses to drugs, papillary dysfunction, gastrointestinal dysfunction, urinary retention, and sweating abnormalities. Serious cardiac arrhythmias observed in GBS patients include symptomatic bradycardia, tachycardia, complete heart block, and cardiac arrest. Treatment of autonomic dysfunction is preventive and symptomatic. A pulmonary artery catheter and vasopressant drugs may be indicated.
in the hypotensive patient. To reduce orthostatic hypotension, nurses should elevate the head of the bed at night to enhance renin secretion, retain sodium, and increase blood volume. To reduce the risk of severe vagal stimulation, which may lead to arrhythmias, the duration of tracheal suctioning should be limited. Patients may also experience bladder dysfunction related to motor weakness and paralysis.

Due to the prolonged immobility associated with the paralysis and muscle weakness, GBS patients are at risk for developing deep vein thrombosis. Several treatment options exist to prevent deep vein thrombosis. One option is the use of low-molecular weight heparin subcutaneously such as enoxaparin. A second option is the use of sequential compression devices, plexipulses, or thromboembolic deterrent stockings to the patient’s lower extremities to prevent venous stasis. Early mobilization has also been shown to reduce the incidence of deep vein thrombosis in this population. Several of the above treatments are frequently used in the patient. Deep vein thrombosis can be a life-threatening complication and prophylactic measures should be initiated early in the patient’s care.

Another potential complication of GBS is acute gastrointestinal bleeding and the development of stress ulcers. Decreased perfusion of the stomach mucosa is the main mechanism of ulcer development. Interventions should focus on reducing the acidity of the gastric secretions. This reduction in acid is accomplished by the use of the histamine (H2)-antagonistic drugs. Examples include ranitidine, cimetidine, and famotidine. Enteral feedings can also help reduce the development of ulcers by neutralizing the pH of the stomach.

**IMPLICATIONS FOR NURSING PRACTICE**

Critical care nurses are essential members of the multidisciplinary team needed to manage the complex care of the GBS patient. The nurse is responsible for assessing, monitoring changes in the patient’s condition and intervening appropriately. Complications such as respiratory muscle dysfunction, autonomic dysfunction, sepsis, and embolism must be recognized early and managed effectively to ensure the patient’s recovery. Other components of the patient’s care that require appropriate nursing interventions include pain management, psychosocial assessment, nutritional status assessment, skin integrity evaluation, and knowledge deficit appraisal. The nurse must also serve as a resource person for the patient and family concerning the disease process and course of the illness. Therefore, nurses need to be knowledgeable about the basic pathophysiology, phases of the disease, variants, and uncertain clinical course of GBS. The multidisciplinary team including the critical care nurse, physician, physical therapist, respiratory therapist, and dietician must work together to meet the goals of the GBS patient’s plan of care.

Nurses should recognize that gaps in knowledge about GBS still exist regarding both the cause of GBS and the most effective treatment. Two thirds of the patients with GBS have an antecedent acute infectious illness, most commonly an upper respiratory tract infection or gastroenteritis 1 to 3 weeks before the onset of neurological symptoms. More research is needed, however, to determine the definitive cause of GBS.

Nurses should be aware that the 2 most widely used treatments are IVIg and plasma exchange. Each of these treatments has risks and benefits as outlined earlier in this article. Intravenous immunoglobulin G has fewer side effects and is considered safer and easier to administer. Plasma exchange is often reserved for the most severe cases of GBS because it is considered more invasive and has more side effects. Research has concluded that the use of corticosteroids is of no benefit. The most recent research examines the use to CSF filtration. The recommendation regarding this treatment is still under review and research. There are no current guidelines regarding CSF filtration. The nurse may be responsible for the actual administration of the medication or assisting in the preparation of the patient for the treatment. The nurse should be cognizant of the potential side effects and their interventions before treatment. The nurse may also be responsible for educating the patient and family about the treatment and its possible side effects.

Future research on GBS has several barriers. One barrier is the small number of patients affected by the disease and their lack of willingness to participate in research studies. Another barrier is the lack of knowledge about the disease, including the exact cause of the syndrome. Once researchers know the specific cause of GBS, efforts could be made to prevent the disease, recognize those who are at the highest risk, and initiate treatment earlier. If a definitive cause is found, healthcare providers armed with that knowledge and diagnostic criteria could begin to treat the patient sooner and possibly reduce residual deficits. Specific diagnostic tests would also be of benefit. At present, nerve conduction studies and examination of CSF can aid but not definitively diagnose GBS.

As presented in this article, Guillain-Barré presents numerous challenges to healthcare providers. The critical care nurse is responsible for assessing, monitoring, and implementing interventions based on the findings. The nurse needs to have the critical thinking skills and knowledge about the disease to appropriately manage and treat the patient.
References

ABOUT THE AUTHORS
Stephanie B. Atkinson, RN, MS, ANP-C, is a Nurse Practitioner at the Gastroenterology Associates, Spartanburg, SC.
Rebecca Lamb Carr, PhD, APRN, BC, is Assistant Professor in Mary Black School of Nursing at the University of South Carolina Upstate, Greenville, SC. Dr Carr is a reviewer of manuscripts for DCCN.
Patricia Maybee, EdD, FNP, FAANP, is a Family Nurse Practitioner with Mountain View Family Practice, Greer, SC.
Donna Haynes, PhD, APRN, BC, FNP, is the Manager of Mobile Service in Joseph Sullivan Center at Clemson University, Clemson, SC. Address correspondence and reprint requests to: Rebecca Lamb Carr, PhD, APRN, BC, 229 Lark Circle, Clemson, SC 29631.