Intensive Care of Acute Guillain-Barré Syndrome

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Abstract: Guillain-Barré syndrome causes, in addition to paralysis and respiratory failure, many general medical problems that have great bearing on outcome. The main features of daily care that require attention are: respiratory and urinary tract infections, gastrointestinal dysfunction, hyponatremia, pain control, and the potential for pulmonary embolism. Dysautonomic problems that arise specifically in the intensive care unit include: hyper- and hypotension, cardiac arrhythmias, and ileus. Throughout the illness, certain psychological aberrations and communication problems must be addressed. The experience reviewed herein provides a practical approach to these problems.

Résumé: Syndrome de Guillain-Barré aux soins intensifs. Le syndrome de Guillain-Barré cause plusieurs problèmes médicaux généraux, en plus de la paralysie et de l'insuffisance respiratoire, qui ont une influence importante sur l'issue de la maladie. Les principaux aspects des soins quotidiens qui demandent une attention particulière sont: les infections des voies respiratoires et urinaires, la dysfonction gastrointestinale, l'hyponatrémie, le contrôle de la douleur et le risque d'embolie pulmonaire. Les problèmes dus à la dysautonomie, qui se présentent spécifiquement à l'unité de soins intensifs, incluent: l'hypertension et l'hypotension, les arythmies cardiaques et l'occlusion intestinale. Pendant toute la maladie, on doit tenir compte de certaines aberrations psychologiques et de problèmes de communication. L'expérience présentée propose une approche pratique de ces problèmes.

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The management of GBS requires special expertise in medical and intensive care, and the patience to be sensitive to the small daily needs of these patients and their families. The neurologist who plans to deal comprehensively with these patients must be familiar with therapy for infections, nutrition, fluid management, and selected aspects of pulmonary medicine as well as the indications for and complications of plasma exchange and gammaglobulin infusion. Because GBS is largely self-limited, these skills contribute as much, or more, to the overall outcome of an individual patient as do specific immune therapies. Nursing and psychological features also constitute a considerable portion of the care required when these patients become seriously ill. Most of our experience is derived from a "retrospective series" and a "prospective series" of 120 consecutive patients from 1979 to 1988. I have seen an additional 141 patients from 1989 to the present and some comments below are the result of that experience. Several texts[1,2] and recent reviews[3] have specifically addressed the critical care problems encountered in GBS and may be referred to for alternative views.

General Medical Management

Summary general guidelines for the intensive care treatment of these patients are given in Tables 1 and 2.

Infection and the Surveillance Approach

GBS patients in an ICU are at particularly high risk of acquiring nosocomial infections. The most common infections in my experience have included respiratory infections (pneumonia and tracheobronchitis), urinary tract infections, and less commonly, line sepsis or generalized sepsis. Urinary infections tend to occur later, on average, than pulmonary infections. Over half of nosocomial infections are caused by organisms that can be cultured from the patient at the time of admission to the intensive care unit. The most important measures that reduce the frequency of these infections are conscientious and frequent handwashing, proper management of urinary catheterization, and disinfection and sterilization of ventilatory equipment, especially nebulizers.

Respiratory Infections

Sputum can be collected weekly or more often for culture and gram stain, but the results must be interpreted with caution, since most patients have colonized their oropharynx with gram negative bacteria or staphylococcus aureus after 3-5 days in the ICU. Prophylactic antibiotics are not thought to prevent pneumonia and only increase the emergence of resistant organisms. The distinction between colonization and nosocomial pneumonia or tracheobronchitis is often difficult. The clinician must be guided by the appearance of an infiltrate on chest radiographs, purulent secretions, fever, leukocytosis, and the presence of intracellular organisms on Gram's stains in tracheal aspirates.

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Chest physiotherapy and intermittent positive pressure breathing have little effect on the resolution of pneumonia in intubated patients, but may make the patient more comfortable.

Nasally intubated patients are at increased risk of sinusitis since these cavities may not drain freely. Paranasal sinusitis can rarely be a source of sepsis. Some intensivists advocate suctioning of the respiratory tract only when needed to reduce substantial secretions, rather than routinely, in order to minimize damage to the tracheal mucosa. A tracheostomy is also a major risk factor for serious pseudomonas and enteric gram negative pneumonias and requires strict adherence to sterile techniques in subsequent tracheostomy care. In prospective studies the most common problems with tracheostomy have been stomal infection, stomal bleeding, excessive cuff pressure requirements, subcutaneous emphysema or pneumomediastinum, and tracheal stenosis, all of which occur in ventilated GBS patients.

Urinary Tract Infections

Thirty percent of all hospital-acquired infections involve the urinary tract. Despite the use of aseptic closed methods for urinary drainage, urinary tract infections are common in GBS, primarily because of the use of Foley catheters in persistently bed-bound patients. In my series, 14% to 22% of patients had urinary tract infections at some time during hospitalization, most often during the third week. After 10 days of catheterization the incidence of bacteriuria may exceed 50%. Meatal colonization is a major risk for catheter-associated bacteriuria, but use of antibacterial lubricants and cleansing of the meatus have not lowered infection rates and therefore are not routinely recommended. Antibiotic irrigation with a neomycin polymycin irrigant similarly has not affected the infection rate in a randomized controlled trial and may ultimately increase the incidence of pseudomonas infections. Although no controlled trial has been carried out, recent experience suggests that intermittent catheterization, even if done in a non-sterile fashion, may be an improvement over a chronic indwelling catheter. The most important preventive measure is strict maintenance of a closed system and handwashing by personnel handling the system.

Table 1. Guidelines in the General Intensive Care Treatment of GBS.

I. Measure vital capacity as indicated by rapidity of deterioration
II. VC 10-12 ml/kg: intubate; VC 12-15 ml/kg: intubate if bulbar paralysis
III. Incentive spirometry to prevent atelectasis; bronchial clearing and assisted coughing
IV. Chest x-ray bi-weekly, or more often; bi-weekly serum albumin, sodium, BUN, calcium measurements; urinalysis bi-weekly
V. Pulmonary embolism prophylaxis: 5000 U heparin every 12 hours subcutaneously;
VI. Check peristalsis GI bleeding prophylaxis: magnesium containing antacid or sucralfate
VII. Decubitus prophylaxis: frequent position changes; air-floating bed or water mattress and skin care
VIII. No antibiotic prophylaxis; urine and pulmonary infections treated with antibiotics after bacterial sensitivities available (unless septic)
IX. Tube feeding when swallowing is impaired (start with continously administered fiber enriched mixtures)
X. Inquire daily concerning pain, sleep, and hallucinosis; adequate pain treatment; limit antecubital phlebotomy if plasma exchange anticipated

Gastrointestinal Problems

Stress ulcers and acute erosive gastritis are potential complications in the immobilized GBS patient. Gastrointestinal bleeding, usually minor, occurred in 8% of patients in our retrospective series and in 2% of the prospective series.[1] The presence of a nasogastric tube attached to suction (rarely necessary in the GBS patient) has the potential for inducing superficial mucosal lesions of the gastric fundus or duodenum, but an increased risk of major gastric hemorrhage has not been demonstrated convincingly. Prophylaxis for gastric bleeding in patients who are not being fed regularly may be achieved with 30-100 ml of a magnesium containing antacid hourly, to maintain the luminal gastric pH above 3.5. Potential complications of this regimen are diarrhea, hypermagnesemia and possibly, aspiration from regurgitation. Intravenous cimetidine may not protect ICU patients as well as antacids from acute gastrointestial tract bleeding. Preliminary work, however, has suggested that the de-acidification of the stomach is associated with an increased rate of nosocomial pneumonia in ventilated ICU patients by a presumed mechanism of microaspiration of bacteria that have colonized the unprotected gastric mucosa. Sucralfate (carafate), an agent that preserves the natural gastric acid barrier, is a good alternative to antacids and H-2 blockers in the prevention of bleeding. It does not significantly lower gastric pH, and is associated with fewer serious aspiration pneumonias, but sucralfate aspiration can be disastrous. Sucralfate has few associated side effects but it often clogs feeding tubes.

Table 2. Summary of Approach to Treatment in the Acute Stages of GBS.

Severity of Illness	Surveillance	Treatment
Mild (walks, VC > 20ml/kg, no dysautonaomia)	– VC and MIF daily – weekly infection surveillance – baseline arterial blood gas	– observe; exchange or IVIG. – active physical therapy
Moderate – Move to ICU if deteriorating	– VC every 6-12 hours – biweekly infection surveillance	– begin plasma exchange or IVIG – incentive spirometry – passive physical therapy
Severe (bedbound; VC < 18 ml/kg)	– VC q 3-6 h if not intubated – alternate day infection surveillance	– exchange if hemodynamically stable – intubation (VC < 12 ml/kg) – passive physical therapy

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Because prolonged immobility, narcotic analgesics, and antacids predispose patients to constipation and ileus, the abdomen should be checked regularly for distention. Although ileus is rare in GBS (2% in my experience) it can be dramatic and has led to bowel perforation. Fecal impaction should be avoided, since, in addition to the mechanical risks, a distended cecum and sigmoid may precipitate a vagal response. The imparied airway function (and perhaps sensation) in GBS may lead to inadvertent pulmonary placement of nasogastric tubes.

Prophylaxis of Venous Thrombosis and Pulmonary Embolism

Pulmonary embolus has occurred as a complication of immobilization in up to 5% of bedbound patients with GBS. In the GBS Study Group trial, 2 of the 7 deaths were from embolism. Reports that up to one-third of GBS patients have embolism were based on clinical criteria alone are probably excessive.[4] There were 2 patients with angiographically documented pulmonary embolus in our prospective series, and 6 in the retrospective series.[1] Most immobilized patients in the prospective series received prophylactic subcutaneous heparin. Pulmonary embolus should be suspected in a patient who has been bedbound for over 2 weeks and has a sudden deterioration in PaO2, or pleuritic chest pain with dyspnea. Rapid arterial oxygen desaturation within the first week or two is generally due to airway plugging rather than to pulmonary embolus. The differential diagnosis includes plugging, excessive pulmonary secretions, pneumonia, pneumothorax on positive pressure ventilation, or endotracheal cuff leak. Radionuclide ventilation and perfusion scans have usually been performed before angiography in all but the most convincing cases. Treatment is usually with a transvenously inserted caval interruption device. Most often there is no associated calf phlebitis, but impedance plethysmography testing of the legs may provide circumstantial evidence of more proximal deep venous thrombosis.

Prophylaxis with subcutaneous heparin has been effective in preventing pulmonary embolus in postoperative patients. The risk of bleeding complications is low and independent of the frequency of treatment; 5000 U every 12 hours appears to be effective in preventing deep-vein thrombosis and is more convenient than an 8 hour regimen. Thrombocytopenia develops in a few patients, and if severe, may require discontinuation of heparin. After several weeks the heparin dose may need to be increased because of tachyphylaxis. In those few patients who will be bedbound for many months, however, subcutaneous injections become painful and impractical and warfarin has been used, attempting to keep the prothrombin time 1.5 times control. One death in the Mayo Clinic ICU series of GBS patients was attributed to the combination of heparin and aspirin.[5]

Intermittent calf pneumatic compression ("air boots") are a potential alternative to subcutaneous heparin in an attempt to reduce the incidence of venous thrombosis, but many patients find these devices uncomfortable, often enough so to deprive them of sleep. My anecdotal impression had been that the early use of air boots left many patients with residual peroneal nerve palsies, but the newer long boots that come to the upper thigh and have a cutout for the knee and lateral fibular area, seem safe and I have used them. Patients who will be completely immobilized for long periods should probably be switched to coumadin.

Other Aspects of General ICU Care

Intravenous suxamethonium should be avoided (e.g., during tracheostomy and other procedures) in patients with severe and prolonged weakness and immobilization since it may lead to the release of large amounts of potassium, causing serious ventricular arrhythmias.[6] Phlebotomy for blood drawing must be minimized since all severely ill patients sooner or later become anemic and antecubital veins should be spared if the patient is to receive plasma exchange. Anemia virtually never results from plasma exchange but mild thrombocytopenia is common and of little consequence. Despite differing results given in a recent review, my experience has been that the main complications of exchange treatment are due to bleeding or pneumothorax from vascular access. Immobilization hypercalcemia has been mentioned in several well documented case reports and has occurred in two of my patients.[7] This complication becomes evident approximately four months after the onset of paralysis, as in other states of immobilization that favor resorption of calcium. Anorexia, nausea and vomiting were the sole clinical features but it may be asymptomatic. Subcutaneous calcitonin combined with oral etidronate disodium has been found to be useful if sodium infusions fail.[7]

Hyponatremia (< 132 mEg/L) is usually attributable to inappropriate antidiuretic hormone and occurs more often in patients on a ventilator. Seldom severe, it responds to fluid restriction. It appears on an average of 10 days after intubation. Several patients have appeared to have a natriuresis and volume depletion with hyponatremia. They may have an atrial natriuretic hormone spike, ususally with dysatuonomia, and the appropriate treatment is with NaCl infusion. Some patients have had a rapid decline to below 115 mEq/l and required hypertonic saline infusion.

Pain Management

At the onset of illness pain is often described as similar to the sensation that occurs hours or a day after strenuous exercise in the low back, large fleshy muscles, anterior or posterior aspects of the thighs, buttocks, and, less often, calves.[8] Low back pain has sometimes been accompanied by sciatica and misled the workup. Pain is generally worst between 10 PM and 4 AM, preventing sleep, and is reported most accurately by the night-shift nurses. It is often alleviated when the knees and hips are slightly flexed. Some patients have benefitted from cold or warm packs or gentle massage. Pain response to medication is variable; acetaminophen and aspirin may provide temporary, incomplete relief. Antidepressants or anticonvulsants have generally not been effective for dysesthetic distal pain in our experience. Sustained relief for a few days may occur with a single injection of 40-60 mg I.M. of methylprednisolone. Epidural morphine sulfate has proved successful in a patient with no response to other pain medication.[9] Narcotics (oxycodone with acetaminophen orally, meperidine HCI intramuscularly) have been effective fairly consistently and they should be administered liberally at night if necessary. If patients are so marginally compensated that narcotics for pain will cause respiratory failure, then they should be intubated; sleep would probably be disastrous in these patients. Narcotics, however, may contribute to ileus. Limited experience with transcutaneous electrical stimulation suggests that it may be helpful for some patients with back and buttock muscular pain or painful dysesthesias.[10] A number of my patients have had no relief with this modality.

Psychological Support and Communication

Several accounts by physicians of their own illnesses[11,12] are most helpful in understanding the experience of GBS. Rice's description of nighttime GBS pain is poignant and sensitizes physicians and nurses to the travail. These descriptions, and others, can be useful if given judiciously to patients and their families because they demonstrate that their fears and concerns are appropriate and typical for the illness, and reinforce that recovery is likely. The novel "Bed Number Ten", is perhaps better read after recovery, but it may be helpful to families of GBS patients during the illness.[13]

Communication with a virtually "locked-out" patient is often difficult, but preservation of slight jaw movement, even in the most severely paralyzed patients, can be used to push a button for signaling. A code system for communication must be started as soon as possible, and posted above the patient's bed. A very useful device has been a clear plastic spelling and signaling board. By standing on the opposite side from the patient, it is possible to rapidly appreciate the patient's eyes spelling out messages. A computer has been devised with a cursor that can be moved by the jaw or finger to spell out messages. Similar commercial and "homebrew" systems that use personal computers have been described. A simpler electro-oculographic switch has only two states but is correspondingly easier to calibrate and use.

Dysautonomia

A wide variety of autonomic nervous system disturbances occur in GBS, most without serious consequences. Dysaustonomia is more frequent in patients with severe motor deficits and respiratory failure than in those with less severe disease, but some autonomic disturbances occur in less severe, and otherwise commonplace cases. Few large unselected series provide enough details to estimate the overall incidence of the various types of autonomic dysfunction. Sixty-five percent of patients with typical GBS in our retrospective series had some autonomic dysfunction.[1] Severe dysautonomia may be more common in the axonal form of GBS. It is advisable to exclude secondary causes of autonomic changes including hypoxia, pulmonary embolus, gastrointestinal bleeding, or fluid electrolyte disturbances before assuming that they are due to GBS. Several cases of sudden death in GBS have been attributed to dysautonomia, particularly complete heart block, but this drastic complication is rare. Dysautonomia may have contributed to death in one case in our retrospective series, but we found none in the prospective series. Dysautonomia generally resolves as other features of GBS improve, but blood pressure lability may persist for several weeks or longer.

Blood Pressure The incidence of hypertension in GBS varies from 5% to 61% in various series, in part depending on the criteria for diagnosis. In our retrospective series, excluding patients with preexisting hypertension, 24% had intermittent or paroxysmal hypertension and 3% had sustained hypertension (diastolic pressures greater than 90 mm Hg and systolic pressures greater than 140 mm Hg on at least 5 occasions, or continuously). Paroxysmal hypertension is almost always associated with quadriplegia and respiratory failure. Hypertension in GBS has occasionally been so severe or paroxysmal that pheochromocytoma has been suspected. Extremes of blood pressure elevation have been tentatively associated with subarachnoid hemorrhage, seizures, increased intracranial pressure and papilledema, encephalopathy, and neurogenic pulmonary edema, all in single cases, some with tentative connections.

Paroxysmal hypertension often occurs in combination with orthostatic hypotension (12 of 40 patients in our retrospective series). One study has shown a slightly elevated peripheral vascular resistance, cardiac output, and stroke index during hypertension. CSF levels of 5-HIAA and HVA were high, suggesting that sympathetic overactivity led to an elevated peripheral vascular resistance.[14] In one of our patients, there were simultaneous marked elevations in systemic vascular resistance and cardiac output with minimal, if any, tachycardia during hypertension.[15] Baroreceptor sensitivity has also been shown to be reduced, probably on the basis of an afferent lesion. The precise mechanisms of disordered blood pressure are not known, but some evidence favors an afferent baroreflex abnormality as the cause of hypertension. A number of disparate physiological observations are compatible with disinhibition of central cardiopressor systems, due to reduction of afferent input from baroreceptors. If immediate blood pressure reduction is necessary, short-acting titratable agents such as nitroprusside should be used. We, and others, have used clonidine, dihydralazine, nitroprusside and nifedipine with varying results.

Supine hypotension may be profound and is the most troublesome aspect of severe cardiovascular dysautonomia. It may be paroxysmal and alternating with episodes of hypertension Trend monitoring often shows precipitous drops in mean BP, at times below 50 mmHg associated with a slight decrease in heart rate. Pulmonary artery pressures decline in parallel with BP but have usually followed hypotension. An important feature accompanying each episode of hypotension in Dalos's[16] and in our case[13] was a slight bradycardia relative to a usually fixed heart rate. Some hypotensive episodes are preceded by "vagotonic" stimuli such as intubation, tracheal suctioning, or medications as in Dalos's cases, but others have been spontaneous. Any of the stimuli capable of producing reflex vagal syncope such as gagging, tracheal stimulation, or distention of a hollow viscus may trigger vasodepressor episodes at some times, and not others. The inability to mute the vasodepressor response, and alternating episodes of severe hypertension, suggest that one component of the blood pressure swings was an afferent lesion from baroreceptors, as discussed above. Measurements of cardiac output and pulmonary artery diastolic pressure in our patients have suggested that changes in SVR were the major determinant of blood pressure, but others have proposed that alterations in central filling pressures are more important.[14]

Cardiac Arrhythmias Sustained sinus tachycardia is probably the most common, though usually harmless, abnormality of the autonomic nervous system in GBS. In our retrospective series, 37% had sustained sinus tachycardia (heart rate greater than 100/min) in the absence of fever, at some time during their illness. A persistent sinus tachycardia has been consistently more common in ICU and respirator patients. Several patients with post-surgical GBS who were hospitalized before the onset of GBS, had sinus tachycardia just prior to the onset of neurological symptoms. Lack of atropine-induced tachycardia is also common. Both are probably the result of loss of vagal efferent innervation. Treatment of sinus tachycardia is unnecessary unless it causes myocardial ischemia. Resolution of severe tachycardia has been reported with verapamil or pindolol administration. Lability of

heart rate, mainly transient episodes of sinus tachycardia, occurrs in occasional patients.

In contrast, vagally-mediated arrhythmias, reported in up to 30% of some selected series are among the most ominous cardiac complications in GBS. "Vagal spells" are episodes of bradycardia, sinus arrest or asystole that may occur with or without an obvious "vagotonic" stimulus. Atrio-ventricular block and other arrhythmias may also occur in response to a direct vagotonic maneuver (e.g., ventricular tachycardia precipitated by carotid sinus massage). In our retrospective series, 8% of patients had multiple vagal spells, typically sinus bradycardia or asystole, during tracheal suctioning or Valsalva-like maneuvers. Vagal spells generally appear during the period of peak motor disability but have been reported during convalescence. None of our patients died or required a cardiac pacemaker solely for intermittent bradyarrhythmias; however, numerous cases requiring pacemakers have been reported. Abnormal oculo-cardiac reflexes or pathologic carotid sinus massage responses may be present in GBS patients with vagal spells[17] and some have suggested that eyeball pressure be used as a provocative test to determine which patients are at risk of permanent heart block and may need cardiac pacemakers. Prophylactic pacemakers are probably not called for until a patient has demonstrated second or third degree heart block, particularly with new externally-applied devices that can sustain patients until the need for prolonged pacing is established. Atrio-ventricular block (but not other arrhythmias) was associated with evidence of other more generalized autonomic dysfunction in one correlative study.[18] Arrhythmias other than sinus tachycardia or "vagal spells" occur in only a small number of patients and most can be ascribed to pre-existing disease or superimposed stresses such as hypoxia although there are occassional cases of unexplained ventricular fibrillation.

In summary, neurologists should be aware of the potential complications of GBS and participate in their management. These details of daily ICU management are, in my view, the most crucial aspect of care, probably more important than the more often discussed immune modulating therapies.

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