

**Case report.**  
**Differentiating symptoms of panic  
from relapse of Guillain-Barré Syndrome**

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**Summary**

*The aim of the paper is to discuss the differentiation between symptoms of panic and a relapse of Guillain-Barré Syndrome (GBS). Longer-term sequelae of panic, sleeplessness and depression may cause patients who formerly had GBS to believe that they are suffering a relapse of their GBS. A case report is presented which highlights some of the neurological and psychiatric symptoms of GBS and illustrates how symptoms of panic may mimic those of GBS and other illnesses (i.e., cardiopulmonary diseases). The treatment of panic symptoms associated with the syndrome, as well as strategies for how patients can differentiate between anxiety and a relapse of GBS are discussed.*

**Introduction**

Guillain-Barré Syndrome (GBS), also known as acute inflammatory demyelinating polyradiculoneuropathy, is a postinfectious polyneuritis that affects myelin sheaths of peripheral nerves and nerve roots in the body. Its central features are symmetric progressive motor weakness, ataxia, and areflexia, often accompanied by paresthesias, dysautonomia, loss of tactile sensitivity, and respiratory insufficiency. The syndrome occurs at the rate of two cases per 100,000 [1] and is considered to be the most common cause of neuromuscular paralysis [2]. It varies in severity and is common at any age and in both sexes. Although neurologic symptoms are reversible with early intervention, the longer-term sequelae of panic, sleeplessness and depression may cause patients to believe that they are suffering a relapse of their GBS.

We provide a case report highlighting some of the neurological and psychiatric symptoms of GBS and illustrating how symptoms of panic may mimic those of GBS and other illnesses (i.e., cardiopulmonary diseases). Following the case report, we discuss the treatment of panic symptoms associated with the syndrome, as well as strategies for how patients can differentiate between anxiety and a relapse of GBS.

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### **Case report: the violinist**

André, a 41-year-old professional violinist from the Czech Republic, suffered a severe case of GBS shortly after his 40th birthday. Following a ten-city concert tour, he experienced a slight tingling in his fingertips. Having performed particularly hard during his last concert, he passed off the sensation as a result of fatigue — he had experienced previously on occasion. Over the next several days, however, the tingling sensations spread throughout his hands and arms and progressed to his legs. He eventually experienced ataxia, which affected his gait and made him feel as if he were “turning to stone.”

André sought consultation with his physician, who immediately referred him to a neurologist. Upon further examination by the neurologist, which included a nerve conduction study, a full blood profile, and a lumbar puncture, André was diagnosed with GBS. Following admission to the hospital, he became completely paralysed and was placed on a respirator. He was subsequently treated with plasmapheresis. (This technique consists of placing an intravenous line in one arm for withdrawing blood and a second line in the other arm for the return of blood following its purification.) By the time plasmapheresis was initiated, he could only blink his eyes.

After two months in the hospital, André was discharged to a rehabilitation facility, where he spent an additional two months. Within 8 months of the onset of his illness, he had recovered and returned to work. Approximately 18 months after his bout with GBS, André began experiencing a slight tingling in his hands and feet once again. He believed that he was experiencing a relapse of his illness and became distraught. Although the chance of relapse is less than 5%, André feared that he was one of the unfortunate few and was experiencing a reoccurrence of the horror that he had endured a year and a half earlier. He promptly sought consultation with his neurologist, who tested his reflexes and strength and pronounced him in good physical health. The neurologist suspected that André may be suffering from anxiety and conducted an electromyogram, which confirmed the absence of GBS.

Because André refused medication to treat his anxiety, he was referred to a clinical psychologist, who conducted a full history and educated him regarding autonomic activity of the nervous system and its relationship to the symptoms he had experienced during his neurological illness. The psychologist administered the Anxiety Disorders Interview Schedule for DSM-IV[3], a structured interview designed to assess a patient's condition comprehensively according to DSM-IV criteria. It also evaluates the presence of coexisting disorders and aids the clinician in differentiating among the subclasses of anxiety disorders. In addition, it assesses symptom severity, degree of impairment, and clinical history. This instrument is widely used by clinicians in research facilities that treat anxiety. The results of this assessment instrument indicated that André was suffering from limited symptoms of panic disorder.

The psychologist also administered the Body Sensations Questionnaire (BSQ),[4] an 18-item instrument designed to measure body sensations associated with panic and agoraphobia (see Figure 1).

Figure 1

**Body Sensations Questionnaire\***

1. Below is a list of body sensations that may occur when you are nervous or in a feared situation. Please mark down how afraid you are of these feelings. Use a five-point scale from not worried to extremely frightened. Please rate all items.

1. Not frightened or worried by this sensation
2. Somewhat frightened by this sensation
3. Moderately frightened by this sensation
4. Very frightened by this sensation
5. Extremely frightened by this sensation

2. Circle the three sensations that you find most difficult in your life. These feelings would be the frightening feelings that occur most frequently.

1. Heart palpitations.
2. Pressure or a heavy feeling in chest
3. Numbness in arms or legs
4. Tingling in the fingertips
5. Numbness in another part of your body
6. Feeling short of breath
7. Dizziness
8. Blurred or distorted vision
9. Nausea
10. Having butterflies in your stomach
11. Feeling a knot in your stomach
12. Having a lump in your throat
13. Wobbly or rubber legs
14. Sweating
15. A dry throat
16. Feeling disoriented and confused
17. Feeling disconnected from your body; only partly present
18. Other (please describe)

\*Reprinted with permission, from Chambless DL, Caputo GC, Bright P, Gallagher R. Assessment of fear in agoraphobics: the Body Sensations Questionnaire and the Agoraphobic Cognitions Questionnaire. *J Consult Clin Psychol* 1984;52:1090-7.

The specific items were generated from interviews with patients and therapists in an agoraphobia treatment program. The BSQ has very good internal consistency, with an R-coefficient of 0.87; it also has a good reliability with a 1-month test/retest correlation of 0.67, and a very good concurrent validity, correlating with other measures of psychopathology. The BSQ is scored by adding the individual item ratings and dividing the total by the number of items rated to provide an overall value.

André stated that experiencing the symptoms of his anxiety was very difficult for him because they closely resembled what he had felt at the onset of GBS — paresthesias, tightness in the chest, lightheadedness, and some numbness. Worry about the return of GBS always lurked in the back of his mind; he felt that he could not endure a second bout with this disorder. Consequently, the psychologist explained how his anticipatory anxiety about experiencing a recurrence of GBS had actually contributed

to undue stress and, in combination with his heavy work schedule, produced the very symptoms that he feared. André also needed to face the fact that he had returned to a very hectic work pace just 1 year after his illness, and stress was probably contributing to his problems.

Subsequently, André was also taught progressive muscle relaxation and deep breathing exercises, which was intended to reduce his level of anxiety. He was educated on the specific cognitions and behaviours that contributed to exacerbating his symptoms and shown the methods for reducing these symptoms via progressive muscle relaxation exercises. In addition, a specific list of early symptoms was drawn up with the help of André's neurologist, to help him differentiate between a relapse of his GBS and panic symptoms. This included duration of numbness and tingling in the extremities, as well as a clear delineation of ataxia and/or awkward motor movements. An agreement was devised that allowed André to decide to contact his neurologist, should his symptoms not abate within a certain time period with the use of relaxation exercises and cognitive coping strategies that he had learned. André was encouraged to replace his negative, catastrophic thoughts about body sensations with self-statements that were less alarming and adhered more to a process of weighing the evidence for and against a relapse of his GBS. He was asked to record these self-coping statements and carry them with him on an index card, particularly as he experienced any worrisome body sensations. He was also encouraged to discuss some of his emotional reactions to the trauma of experiencing GBS and his fears about the future of his health.

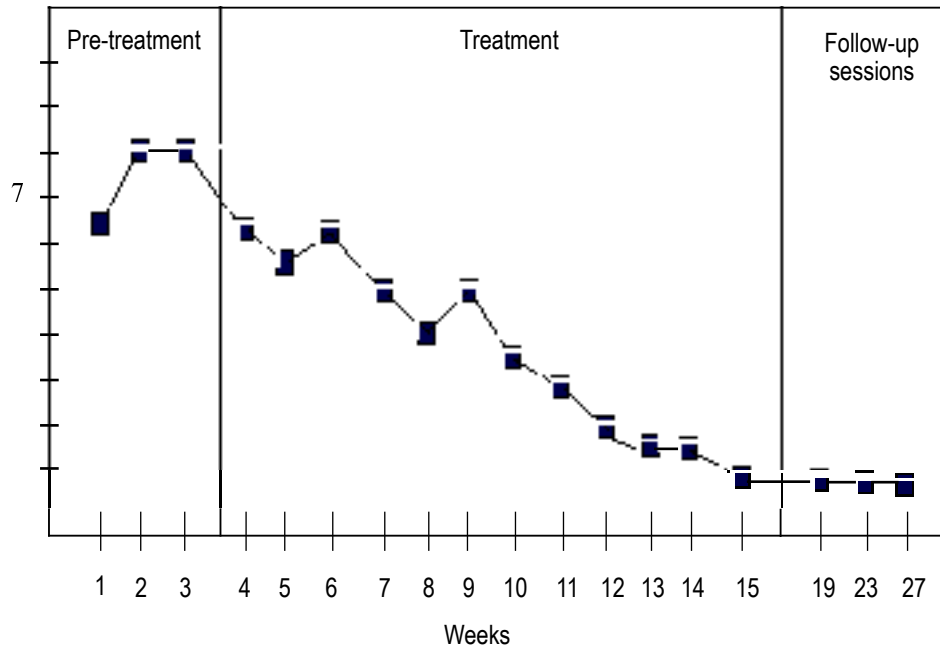


Figure 2. André's level of panic symptoms, from pretreatment through follow-up, as measured with the Body Sensations Questionnaire. The mean score was 3.05, with a standard deviation of 0.86

Figure 2 illustrates André's scores on the BSQ prior to treatment for anxiety, through treatment, and finally into a 3-month follow-up period.

As can be seen, there was a significant reduction in André's report of body sensations with the beginning of treatment. Use of cognitive-behavioural techniques, a renewed sense of self-reassurance, and an improved ability to cope with stress brought a steady reduction in his symptoms over the next several weeks.

### Discussion

Persons with a serious medical illness, including cancer and cardiovascular, endocrinological, and respiratory disorders, often experience panic symptoms subsequent to their illness [5]. In André's case, the symptoms of panic closely paralleled the symptoms of GBS, making it difficult for him to discern the difference.

It should be noted that there is no guarantee that panic symptoms and the symptoms of these medical disorders can always be differentiated by patients or their clinicians. However, some attempt should be made to aid individuals in coping with benign symptoms. Otherwise, patients are apt to respond to false alarms and make unnecessary visits to health-care professionals that may affect their potential to receive proper medical attention in the future. Cognitive-behavioural therapy (CBT) seems to have been quite effective in treating André's panic symptoms. It also helped him to differentiate panic symptoms from GBS and to address the emotional difficulties that he experienced in the wake of his severe medical illness.

Three French physicians, Georges Guillain, Jean-Alexandre Barré, and André Strohl, are credited with describing the entity now called GBS, even though it first appeared in medical writings in 1859 under the term "Landry's ascending paralysis." In 1916, these pioneering physicians identified the clinical syndrome of muscle paralysis and areflexia. They added to the description the characteristic anomaly of elevated proteins in the spinal fluid, which aids in differentiating the syndrome from other neurological disorders.

The cause of GBS is not clear, but it involves an autoimmune attack on the myelin of the peripheral nerves. Onset commonly occurs within 4 weeks of a viral respiratory tract infection or gastroenteritis. The illness may be seen following surgery or in association with hepatitis B, infectious mononucleosis, Lyme disease, toxoplasmosis, systemic lupus erythematosus, porphyria, or lymphoma. A recent study [6] has fortified the link between GBS and *Camphylobacter jejuni*, a gram-negative rod now determined to be a common cause of bacterial gastroenteritis. The authors found that 26% of their subjects with GBS (n = 96) experienced *Camphylobacter jejuni* infection prior to the onset of GBS as compared with 2% of household controls and 1% of hospital controls.

As a result of the autoimmune attack, the myelin sheath and sometimes even the axon of the neurone are damaged, causing nerve signals to be delayed or otherwise altered in transmission. The affected individual experiences abnormal sensations including malaise, fatigue, dysequilibrium, muscle pain in the back and neck, and paresthesias (which may be painful in some cases). The loss of tactile sensitivity is fibre-length-dependent; it begins in the lower extremities and ascends to other parts of the body.

Research [1, 9, 10] has indicated that it is not uncommon for patients who have had

GBS to experience varying degrees of post-traumatic stress disorder, as well as other anxiety and depression disorders. In rare cases, psychotic features may surface during the illness or the recovery phase of GBS. [9] (GBS is thus sometimes misdiagnosed as “metabolic encephalopathy” or “critical care psychosis.” [11]) Psychosis occurs only in a small proportion of patients with acute cases of GBS who experience complete loss of mobility and communication, and the cause is unknown [9].

A problem sometimes seen during recovery, particularly during the early phase of it, is the development of various symptoms of panic that overlap with the symptoms of the patient’s recent illness. In such cases, patients may become confused as to whether or not they are experiencing residual symptoms of illness or the anticipatory anxiety of having a relapse. This is compatible with the anxiety sensitivity theory proposed by Reiss, [12] which states that individuals vulnerable to panic are prone to becoming particularly concerned with physical symptoms or threats. Additional research [13] indicates that panic biases individuals toward selectively attending to threatening information.

Post-GBS anxiety is similar to postcardiac anxiety, in which patients misinterpret benign arrhythmias as a heart attack [11, 14, 15]. An aspect of GBS that renders it unique among medical disorders is the paresthesias, symmetric motor weakness, and unsteady feelings that may also be present during severe cases of panic disorder.

There are a number of reasons why individuals may experience anxiety following GBS. For one, they are often demoralised during the illness by profound anxiety and a sense of helplessness as they are faced with a life-threatening condition that progressively paralyzes muscle movement [16]. One theory [20] proposes that increased anxiety results from heightened activity in central serotonergic neurones which may be effected by the polyneuritis itself. The activation of different serotonergic receptors may lead to different, even opposing, functional effects [19, 21]. This area requires further study, since its implications for pharmacological treatment (and particularly for the use of selective serotonin-reuptake inhibitors) are significant [22].

When patients present with reoccurring symptoms that might appear to be due to GBS, the initial course of action is to conduct a neurological evaluation including an electromyography and nerve conduction studies. If the results of these studies are negative, then the diagnosis of anxiety can be considered, and the patient can subsequently be educated to the properties of anxiety and the effects on the autonomic nervous system. He or she can learn the criteria for differentiating symptoms of anxiety from those of GBS. The key criteria in making this differentiation are the presence of ataxia and the duration of parasthesias.

Clearly, one treatment of choice involves medications such as tricyclic antidepressants, selective serotonin-reuptake inhibitors, and high-potency benzodiazepines [23, 24]. Monamine oxidase inhibitors are sometimes used as second- or third-line agents when there is no response to other medications [25]. True symptoms of GBS do not improve with the use of these medications. Although such interventions may be effective in temporarily alleviating panic, they may not be enough to eradicate it over the long term; especially in the case of high-potency benzodiazepines. [26] and monamine oxidase inhibitors [27], they may produce untoward side effects. In addition, medication

does very little for helping the patient learn to discern the severity of their symptoms and rely on self-reassurance without the aid of chemical agents. Consequently, several adjuncts to psychopharmacotherapy may prove to be more effective in helping patients to differentiate the symptoms of anxiety from a true relapse of GBS.

Although the treatment of choice for anxiety disorders involves a combination of interventions [28, 29], CBT alone may offer some advantage beyond what is provided by medication [23]. By reducing symptoms, CBT allows the patient to evaluate the severity of symptoms and determine whether or not they are the result of panic. The use of such techniques in lieu of pharmacotherapy may spare the individual from the repeated need for medication.

The standard protocol for CBT in patients with anxiety typically involves various combinations of breathing retraining, along with the reinterpretation of interoceptive cues and the cognitive “decatastrophisation” of symptoms [30, 31]. The professional literature is replete with empirical studies underscoring the efficacy of CBT combined with pharmacotherapy. CBT has been demonstrated to be efficacious and to provide fast relief [28] and lasting benefits to most sufferers of panic [33]. It is noted for having a high success rate and a low dropout rate, with no side effects [33, 34]. The patients are able to reduce symptoms of panic, avoidance behavior, and secondary symptoms of depression. Therapeutic benefits have been documented for 2 years subsequent to treatment [35, 36].

CBT for panic disorder has four central components: preparation, skills training, exposure, and relapse prevention. These components are usually implemented in sequence, with the best results derived from having therapeutic focal points rather than distinct stages. Elements from one component are typically introduced while the treatment is still focused on another component. CBT is particularly useful in treating patients like André, whose anxiety is complicated by medical illness [14].

Using controlled or diaphragmatic breathing is one form of breathing retraining for counteracting parasthesias and, in some cases, numbness, since hyperventilation might also explain such symptoms [37]. Individuals are instructed to breathe normally through the nose and count the number of breaths while at rest; they are also directed to place both hands over the abdomen to feel the movement of the diaphragm. The goal is to maintain the respiration rate at nine to 16 times per minute, slowing it to that frequency when it is higher. Individuals are instructed to practice the exercise during both symptomatic and asymptomatic periods [38].

The reinterpretation of interoceptive cues is also an important aspect of treatment, since patients may jump to extreme conclusions about sensations, causing and/or maintaining a vicious cycle of autonomic activity. During this period of vulnerability, individuals tend to overestimate perceived danger and to underestimate their capacity for coping [15, 39]. Patients are encouraged to think about the specific symptoms they are experiencing and to record the automatic thoughts that occur simultaneously with them as well as the emotional reaction. They are provided with a list of criteria to help them decide whether they have reason to be concerned about their bodily sensations. In the case of parasthesias, individuals may be instructed to remain calm when experiencing the tingling in their extremities and utilise deep breathing and muscle

relaxation to reduce or eliminate unpleasant sensations. One of the benefits of breathing retraining is that it prevents hyperventilation and consequent hypocapnia-related anxiety symptoms [40].

Patients may also employ cognitive self-talk, decatastrophise their symptoms, and allow a certain amount of time to pass before determining the seriousness of their symptoms [41]. Such techniques allow them to wait to see whether their symptoms abate. This helps to reinforce the notion that these symptoms were generated by anxiety rather than a relapse of GBS. Patients are encouraged to use this type of thought correction whenever symptoms appear, until they can decide whether they need to seek medical help.

The authors wish to thank Lawrence Levitt, M.D., for his helpful comments on the final draft of this manuscript, as well as Eric Frey for his assistance with the literature review.

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