Short-term variability of blood pressure and heart rate in Guillain-Barré syndrome without respiratory failure

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ABSTRACT

The effect of Guillain-Barré syndrome (GBS) on the short-term variability of blood pressure and heart rate was evaluated in six patients presenting with a moderate form of the syndrome, i.e. unable to stand up unaided and without respiratory failure, at the height of the disease and during recovery. The patients were compared with six age-matched healthy volunteers. During the acute phase of the syndrome, GBS patients exhibited a significant heart rate elevation (+26 beats/min compared with healthy subjects), but the acceleratory response to atropine, or to 60° head-up tilt, was maintained. Resting plasma noradrenaline levels were high in acute GBS, but the secretory response to tilt was preserved. Desensitization to noradrenaline was observed in acute GBS with a reduced pressor action of this α-adrenoceptor agonist. Blood pressure levels were normal and head-up tilt did not induce orthostatic hypotension in this moderate form of GBS. Power spectral analysis demonstrated marked alterations in cardiovascular variability. The overall heart period variability was markedly reduced with the reduction predominantly in the high-frequency (respiratory) range (−73%). The low-frequency component of heart period variability was also reduced (−54%). This cardiovascular profile of moderate GBS at the height of the disease could result from a demyelination of the reflex loop controlling respiratory oscillations in heart rate and from a desensitization of the arterial tree to an elevated plasma noradrenaline. Sympathetic nervous activation may contribute to the high resting heart rate in acute GBS.

INTRODUCTION

The Guillain-Barré syndrome (GBS) is the most frequent cause of acute generalized paralysis [1]. This syndrome is an inflammatory demyelinating disorder of the peripheral nervous system. Autonomic neuropathy is a complication of GBS and may affect the cardiovascular, sudomotor and gastrointestinal systems. Cardiovascular dysfunction may include sinus tachycardia, vagally mediated arrhythmias, pronounced blood pressure fluctuations and orthostatic hypotension [2–4]. By using quantitative tests of autonomic function, a very high proportion of subclinical involvement, affecting both parasympathetic and sympathetic arms of the autonomic nervous system [4], has been observed in GBS. With the aim of achieving a more specific and early diagnosis of autonomic dysfunction in this condition, six patients displaying a moderate form of GBS were enrolled in this study. These patients were compared with healthy volunteers to test whether non-invasive continuous blood pressure and heart rate recordings in the supine position and during head-up tilting could provide indices of autonomic

Key words: atropine, Guillain-Barré syndrome, noradrenaline, spectral analysis, tilt.
Abbreviation: GBS, Guillain-Barré syndrome.
Correspondence: Professor Jean-Luc Elghozi.
disturbances in moderate GBS. Patients were recorded during the acute phase of disease development and after recovery.

SUBJECTS AND METHODS

Subjects
The study population comprised six patients with GBS. Diagnosis was made according to the criteria of the National Institute of Neurological and Communicative Disorders and Stroke as modified by Asbury and Cornblath [5]. These patients were enrolled in our department as part of the French Cooperative Group on Plasma Exchange in Guillain-Barré Syndrome multicentre trial. Patients were considered moderately affected by their condition in that they could not stand up unaided but did not require mechanical ventilation. Their arterial blood gases at the height of the disease were within the normal range (pH 7.40 ± 0.1, PaCO₂ 5.15 ± 0.09 kPa, PaO₂ 11.32 ± 0.40 kPa, SaO₂ 96.4 ± 0.5%, HCO₃⁻ 26.83 ± 0.48 mmol/l, n = 6). They were randomized to receive four plasma exchanges [6]. No patient received exogenous catecholamines or sedative drugs that could interfere with the autonomic function. Volume expansion with 1 litre of water and 6 g of NaCl, given enterally using a gastric tube, was performed within 24 h of the last plasma exchange to counterbalance any plasmapheresis-induced hypovolaemia [7]. Experimental recordings at the height of the disease were performed 1 day after volume expansion. Subsequently, recordings were carried out during the early remission phase, 7 days after patients started recovering the ability to walk. The recovery period was generally between 5 and 6 weeks long. The GBS group comprised three females and three males aged 42.3 ± 3.9 years and weighing 66.0 ± 5.1 kg. Mean height was 169.3 ± 1.9 cm. The group of healthy subjects comprised four females and two males aged 40.8 ± 4.2 years and weighing 66.7 ± 5.1 kg. Their mean height was 168.2 ± 1.5 cm. None of the subjects had received any cardiovascular drug treatment before the study. Informed consent was obtained, and the study was approved by the ethics committee of the Société de Réanimation de Langue Francaise.

Study design
The session started at 07.00 hours in a quiet room with subjects resting on a tilt table maintained in the horizontal position. An initial venous blood sample was taken at 08.00 hours to measure resting plasma renin activity by a radioimmunoassay and plasma noradrenaline by HPLC with electrochemical detection. Finger blood pressure was measured using a Finapres® device (model 2300, Ohmeda, Trappes, France). This non-invasive procedure has been shown to provide a reliable assessment of frequency- and time-domain components of blood pressure and heart rate variability [8]. The cuffed finger was maintained in the mid-axillary position at heart level. Each recording session lasted 5 min. Supine blood pressure was measured at 09.00 hours. Another recording was started 15 min after the initiation of head-up tilt at 60 °. At the completion of the blood pressure recording a second blood sample was taken to measure plasma renin activity and noradrenaline levels during tilt. The table was then returned to the horizontal position and supine blood pressure recordings were obtained, while atropine sulphate was gradually administered intravenously up to a maximal dose of 2 mg. This 2-mg dose was not reached when the effect peaked before reaching this dose. At 14.00 hours blood pressure recordings were repeated. At this time the heart rate effects of atropine administered earlier were minimal. A concentration–response curve to exogenous noradrenaline was obtained using an intravenous infusion of a graded concentration of noradrenaline tartrate (Pharmacie Centrale des Hôpitaux, Paris, France). The initial infusion rate of 0.01 µg·min⁻¹·kg⁻¹ was increased in increments of 0.01 µg·min⁻¹·kg⁻¹ every 5 min in order to reach a plateau level. The final concentration of noradrenaline was 0.10 µg·min⁻¹·kg⁻¹.

Data acquisition and processing
The details of data acquisition and analysis have been described previously [9]. An evenly spaced (equidistant) sampling of systolic blood pressure, diastolic blood pressure, pulse interval (taken as a surrogate for heart period) and pulse rate (reciprocal of pulse interval, taken as a surrogate for heart rate) allowed a direct spectral analysis of each 256-s segment using a Fast Fourier Transform algorithm with the computer program Anapres 3.0® (Notocord Systems, Croissy sur Seine, France). The frequency of oscillation scale (abscissa) was analysed up to 0.5 Hz. Power of the systolic blood pressure and heart period spectrum (ordinates) shown with the recordings had units of mmHg² or ms⁴. The modulus (square root of the power in mmHg² or ms⁴) was used for calculations and is presented in the tables. The integration of the values of consecutive bands was computed to estimate the different components of variability. The high-frequency (respiratory) oscillation corresponded to the highest peak detected within the 0.2–0.5 Hz range and we summed the value of this band and those of the three consecutive values of higher and smaller frequencies (i.e. seven values corresponding to a total band width of 0.027 Hz) to integrate the high-frequency component of the systolic blood pressure and heart period spectra. The high-frequency oscillation of the heart rhythm corresponds to the respiratory sinus arrhythmia. The low-frequency component was obtained by integration of the values of consecutive bands from 0.068 to 0.127 Hz of the systolic blood pressure or heart rhythm spectrum, in order to include the 10-s
Table 1  Effect of GBS (acute phase and recovery) on blood pressure and heart rhythm levels in the supine position before and after atropine administration

<table>
<thead>
<tr>
<th></th>
<th>GBS patients</th>
<th>Healthy volunteers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Acute phase</td>
<td>Recovery</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>116.4 ± 8.8</td>
<td>130.6 ± 9.8**</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>65.4 ± 9.5</td>
<td>80.5 ± 9.9**</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>92.0 ± 9.4†</td>
<td>122.3 ± 10.0***</td>
</tr>
<tr>
<td>Heart period (ms)</td>
<td>691 ± 77</td>
<td>512 ± 52**</td>
</tr>
</tbody>
</table>

Table 2  Effect of GBS (acute phase and recovery) on the modulus of the two spectral components (low and high frequency) of systolic blood pressure and heart period time series in the supine position before and after atropine administration

<table>
<thead>
<tr>
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<th>GBS patients</th>
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</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Acute phase</td>
<td>Recovery</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>1.7 ± 0.3</td>
<td>2.2 ± 0.4</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>0.6 ± 0.1</td>
<td>0.3 ± 0.1*</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>7.9 ± 0.7</td>
<td>2.0 ± 0.3***†‡</td>
</tr>
<tr>
<td>Heart period (ms)</td>
<td>0.29 ± 0.04</td>
<td>0.30 ± 0.03</td>
</tr>
</tbody>
</table>

Statistical analysis

Data are presented as means ± S.E.M. Comparisons between supine values before and after atropine, and between supine and tilt in each condition (acute GBS, recovery, healthy controls), were performed using Student’s paired t-test. The effects of the three conditions on the resting levels, post-atropine levels or tilted levels were analysed using a one-way ANOVA, followed by Tukey’s post-hoc test for multiple comparisons. The effect of tilt on noradrenaline levels or plasma renin activity was evaluated using a paired t-test. Comparisons between acute GBS and healthy controls for the effects of tilt on noradrenaline levels and plasma renin activity were performed using Student’s unpaired t-test. The same test was applied to analyse the baseline levels of noradrenaline and plasma renin activity in acute GBS and healthy control subjects. The effects of noradrenaline infusion on mean arterial pressure levels obtained in the three states (acute GBS, recovery and healthy controls) were analysed using a two-way ANOVA. Log transformation was applied when the variance ratio of the variables was significant (F test). Differences were considered significant when P < 0.05.

RESULTS

Cardiovascular variables in GBS patients at rest and after atropine

Resting blood pressure levels were similar in the GBS patients (acute phase and recovery) and the control group.
Figure 1  Examples of 256-s heart period recordings from one healthy volunteer and one acute GBS patient obtained in the supine position at rest or after atropine.

The corresponding heart period power spectra (frequency scale in Hz, power spectral units in ms$^2$) are represented above each recording (insets).
Table 3  Effect of GBS (acute phase and recovery) on blood pressure and heart rhythm levels in the supine position and during a 60° head-up tilt

Values are means ± S.E.M. Differences between supine and tilt: *P < 0.05, **P < 0.01, ***P < 0.001.

<table>
<thead>
<tr>
<th>GBS patients</th>
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<th>Recovery</th>
<th>Healthy volunteers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Supine</td>
<td>Tilt</td>
<td>Supine</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>104 ± 3.7</td>
<td>113.9 ± 4.5</td>
<td>106.4 ± 7.9</td>
</tr>
<tr>
<td>Diastolic blood pressure (mmHg)</td>
<td>57.3 ± 3.0</td>
<td>68.9 ± 5.7*</td>
<td>58.6 ± 4.9</td>
</tr>
<tr>
<td>Heart rate (beats/min)</td>
<td>85.5 ± 5.7</td>
<td>97.4 ± 8.3*</td>
<td>77.0 ± 4.0</td>
</tr>
<tr>
<td>Heart period (ms)</td>
<td>720 ± 51</td>
<td>642 ± 60*</td>
<td>793 ± 46</td>
</tr>
</tbody>
</table>

Table 4  Effect of GBS (acute phase and recovery) on the modulus of the two spectral components (low and high frequency) of systolic blood pressure and heart period time series in the supine position before and during a 60° head-up tilt

The breathing frequency is also shown. Values are means ± S.E.M. Differences between supine and tilt: *P < 0.05, **P < 0.01, ***P < 0.001. Differences between the tilted values in acute GBS and healthy volunteers: †P < 0.05.

<table>
<thead>
<tr>
<th>GBS patients</th>
<th>Acute phase</th>
<th>Recovery</th>
<th>Healthy volunteers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Supine</td>
<td>Tilt</td>
<td>Supine</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>1.6 ± 0.2</td>
<td>1.7 ± 0.3</td>
<td>1.3 ± 0.2</td>
</tr>
<tr>
<td>Low frequency</td>
<td>0.6 ± 0.2</td>
<td>0.5 ± 0.2</td>
<td>0.9 ± 0.2</td>
</tr>
<tr>
<td>Heart period (ms)</td>
<td>9.8 ± 2.3</td>
<td>5.7 ± 1.0<em>i</em>†</td>
<td>12.2 ± 2.3</td>
</tr>
<tr>
<td>Low frequency</td>
<td>4.0 ± 0.6</td>
<td>2.4 ± 0.3**i*†</td>
<td>9.5 ± 4.1</td>
</tr>
<tr>
<td>Breathing frequency (Hz)</td>
<td>0.27 ± 0.02</td>
<td>0.32 ± 0.03</td>
<td>0.28 ± 0.03</td>
</tr>
</tbody>
</table>

Both low- and high-frequency components of heart period variability were reduced during acute GBS (P < 0.001 and P < 0.05 compared with healthy controls, unpaired Student’s t-test), as shown in Figure 1.

Atropine resulted in a profound reduction in the high- and low-frequency components in the supine position.

Effects of GBS on supine systolic blood pressure and heart period spectra before and after atropine

The blood pressure spectral components were unaffected by GBS (Table 2). In contrast, the overall variability of heart period was diminished in acute GBS as indicated by the standard deviation of the heart period distribution (45 ± 7 ms in controls versus 26 ± 4 ms in acute GBS, P < 0.05). Both low- and high-frequency components of heart period variability were reduced during acute GBS (P < 0.001 and P < 0.05 compared with healthy controls, unpaired Student’s t-test), as shown in Figure 1.

Atropine resulted in a profound reduction in the high- and low-frequency components in the supine position.

Effects of GBS on the response of cardiovascular variables to tilt

Tilt resulted in a blood pressure rise with a concomitant increase in heart rate. The rise in heart rate was quantitatively similar in the three groups although the resting heart rate was elevated in acute GBS (Table 3).

Effects of GBS on the spectral responses of systolic blood pressure and heart period to tilt

The high-frequency component of heart period variability during tilt was diminished in acute GBS as shown in Table 4. In addition, tilting increased the low-frequency/high-frequency ratio of heart period variability in healthy controls (+56%, P < 0.05). The effects
Figure 2  Examples of 256-s systolic blood pressure recordings from one healthy volunteer and one acute GBS patient obtained in the supine position at rest or after head-up tilt at 60°. The corresponding systolic blood pressure power spectra (frequency scale in Hz, power spectral units in mmHg²) are represented above each recording (insets).
Table 5  Effect of GBS (acute phase) on noradrenaline levels and plasma renin activity in the supine position and during a 60° head-up tilt

<table>
<thead>
<tr>
<th></th>
<th>GBS patients, acute phase</th>
<th>Healthy volunteers</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Supine</td>
<td>Tilt</td>
</tr>
<tr>
<td>Noradrenaline (pg/ml)</td>
<td>615 ± 60ttt</td>
<td>1798 ± 179***ttt</td>
</tr>
<tr>
<td>Plasma renin activity (pg/ml)</td>
<td>33.7 ± 10.3tttt</td>
<td>97.3 ± 29.5*</td>
</tr>
</tbody>
</table>

Values are means ± S.E.M. Differences between tilt and supine: *P < 0.05, **P < 0.01, ***P < 0.001. Differences between acute GBS patients and healthy volunteers in the supine or tilted position: †P < 0.05, ††P < 0.01, †††P < 0.001.

Figure 3  Mean blood pressure responses to exogenous noradrenaline in the GBS patients and control subjects

○, healthy volunteers; ■, acute GBS; □, recovery period.

of this manoeuvre on systolic blood pressure variability are shown in Table 4 and Figure 2.

Effects of GBS on noradrenaline levels and plasma renin activity in supine and tilted positions

Noradrenaline levels and plasma renin activity were elevated in acute GBS (Table 5). After head-up tilt the level of both parameters was increased in both groups (Table 5) but the increases were smaller in the GBS patients compared with those in the controls.

Effects of GBS on the pressor response to exogenous noradrenaline

Figure 3 illustrates the reduced sensitivity to exogenous noradrenaline in the acute stages of GBS when compared with healthy volunteers (effect of noradrenaline: F = 8.5, P < 0.001; effect of the condition: F = 3.9, P < 0.05). The response to noradrenaline was restored during the early recovery phase.

DISCUSSION

The degree, in addition to the mechanism, of autonomic dysfunction in GBS is a matter of debate. In the early stages of the disease, transient impairment of autonomic function may be detected using standardized autonomic tests [4]. In this study the blood pressure and heart rate variability profiles in moderate GBS differed from the profiles of healthy controls at the height of the disease, with subsequent recovery paralleling the improving clinical course. In our study the patients were breathing spontaneously, there was no clinical evidence of respiratory failure, and their blood gases were within the normal range. The patients did not receive any cardiovascular or sedative drugs that could interfere with heart rate or blood pressure variability. In addition, they were studied 2 days after the last course of plasma exchange and after fluid filling to avoid effects of plasmapheresis on short-term variability [10].

During the acute developmental phase of GBS, our patients exhibited a marked rise in heart rate, a phenomenon commonly observed in GBS [2,3,11]. The administration of atropine clearly disfavoured a vagal inhibition as the cause since the heart rate change resulting from atropine administration was quantitatively similar in the patients and control subjects. The effect of atropine also supports the idea of a relative integrity of vagal efferent nerves in moderate GBS. The mechanism for this heart rate rise could well be β-adrenoceptor overstimulation of the heart. The heart rate observed after atropine was indeed higher in acute GBS compared with that in controls. This hypothesis is supported by the observation of elevated plasma noradrenaline levels in patients with GBS. Such a hypothesis should be validated using β-adrenoceptor blockade. In addition, since the tilt test revealed a normal acceleratory response, on top of the high resting heart rate, it is unlikely that cardiac sympathetic efferent nerves are altered. This cardiac response also supports the relative integrity of the baroreceptor–heart rate reflex loop in moderate GBS. No evidence of a postural-induced fall in blood pressure was observed during passive tilt. Together with the normal blood pressure levels recorded at rest, the blood pressure data indicate the relative integrity of vascular sympathetic tone and its activation in response to baroreceptor deactivation during tilt. This is not to deny that the integrity of the arterial baroreflex, leading to orthostatic hypotension, may be altered in more severe cases of GBS [1,3].
Our observations of elevated plasma noradrenaline levels in conjunction with increased plasma renin activity in acute GBS are difficult to interpret without additional experiments. The four most plausible mechanisms underlying our observations are: (i) alterations of the main characteristics of the baroreceptor and chemoreceptor reflexes, such as delay, gain and cut-off frequency, due to the demyelination process, which may affect the resting activity of the efferent sympathetic tone; (ii) a centrally mediated sympathetic activation associated with the expected anxiety resulting from an acute motor disability; (iii) a sympathetic and plasma renin activation compensating a relative hypovolaemia (although a volume load was administered on the day preceding the initial test, muscle paralysis may alter the venous return and lead to a cardiothoracic hypovolaemia); (iv) an effect of GBS on noradrenaline clearance (this possibility supports the validity of plasma noradrenaline measurements as a clinical index of overall sympathetic nervous activity [12]; in the absence of data concerning noradrenaline clearance, which is reduced after head-up tilt, it is difficult to comment on our reported rise in noradrenaline levels after tilt). The first hypothesis of a reduced inhibition of cardiovascular centres caused by lesions of arterial baroreflexes was favoured by Fagius and Wallin [13]. These authors recorded muscle sympathetic nerve discharges in patients with moderate to severe GBS who had transient hypertension and tachycardia during the illness [13]. A temporarily increased sympathetic outflow was observed during the acute phase of the disease. The authors discussed these findings in detail and concluded that damage to afferent pathways with diminished, but not entirely absent, baroreceptor inhibition might explain these data. The same group also described a similar clinical picture of baroreceptor deafferentation during the first minute of bilateral nerve block, i.e. before the block became complete [14].

The most striking alterations in cardiovascular profiles were observed in the frequency domain analysis. Heart rate variability was markedly diminished in the acute stages of GBS. Although this reduction predominated in the high-frequency range it also affected the low-frequency components. A similar degree of heart rate variability reduction has been reported during aging and cardiac failure [15]. Interestingly, these changes occurred in conjunction with elevated rates of cardiac noradrenaline spillover [15]. However, the effect of atropine in healthy volunteers illustrated the vagal predominance in the genesis of this respiratory (high-frequency) peak, and the marked but non exclusive contribution of the vagus in the genesis of the low-frequency peak, since the high- and low-frequency peaks were reduced to 11% and 19% of the respective control values. The low-frequency peak is considered to be a mixture of parasympathetic and sympathetic activity [16]. Atropine also influenced the residual components of heart rate variability in GBS. This alteration in spontaneous heart rate variability was recently reported by Flachenecker et al. [11]. This author also observed a striking decrease in low- and high-frequency power in GBS patients presenting with tachycardia. R–R interval variation during deep breathing is also commonly diminished in patients with tachycardia [2]. The unit used for calculating heart rhythm may affect the observed differences [17]. In fact, in our study, the choice of the variable used for the calculation of the spectral indices (heart rate or heart period) did not influence the difference between acute GBS patients and controls, given that significant differences were also demonstrated when heart rate spectral values were used (results not shown). The moderate tachypnoea associated with acute GBS, although being a factor able to decrease the respiratory sinus arrhythmia, was not high enough to solely explain the altered heart rate variability [18]. Tidal volume was also shown to affect the respiratory sinus arrhythmia [18]. Changes in tidal volume in GBS patients were plausible but probably small in the absence of respiratory failure. Therefore, one can reasonably discuss the mechanisms of an altered vagal (mainly respiratory) modulation of heart rhythm. Since the efferent vagal activity responded normally to atropine, a peripheral alteration mainly on the afferent arc could be present and create, as postulated by Truax [2], a ‘phase-dependent block’ based on slowing due to demyelination of the reflex loop pathway. A longer delay for the reflex translating respiratory movements into heart rate changes could well reduce the amplitude of respiratory sinus arrhythmia.

Interestingly, in the GBS patients, blood pressure fluctuations were altered in the low-frequency range during tilt. The absence of a blood pressure fall during this manoeuvre, together with a normal rise in plasma noradrenaline, supports the view mentioned above of a normal function of baroreflexes during tilt. However, the high baseline level of noradrenaline may well be responsible for a desensitization of the arteries to the constrictor action of noradrenaline [19]. Graded doses of the α-adrenoceptor agonist determined less pressure rises in acute GBS compared with those seen in the healthy volunteers. Given that blood pressure oscillations in the low-frequency range (0.1 Hz, Mayer waves) correspond to the resonance frequency of the arterial baroreflex [20], and that this oscillation is amplified during tilt as a consequence of sympathetic activation [21], it is conceivable that the vascular desensitization in acute GBS reduced the blood pressure oscillatory response to tilt. Finally, the high-frequency fluctuations in systolic blood pressure were preserved in GBS patients. The respiratory fluctuation in systolic blood pressure, related to stroke volume, mainly depends on mechanical factors such as the respiratory pattern, the venous return to the thorax and the cardiac function [22]. The absence of a significant change in the high-frequency peak (central frequency and
amplitude) illustrates the preservation of respiratory function in these patients and argues against hypervolaemia [23].

In conclusion, the cardiovascular profile of moderate GBS at the height of the disease could result from a demyelination of the reflex loop (mainly on the afferent nerves) controlling the respiratory oscillations in heart rate. The amplitude of the respiratory sinus arrhythmia was markedly diminished. The reduction of the blood pressure response to tilt in the frequency domain, consisting of a reduced amplification of the 10-s fluctuations during tilt, may reflect a desensitization of the arterial tree to noradrenaline. Baseline plasma noradrenaline levels were elevated, and this index of sympathetic activation could underlie the resting tachycardia observed in the acute stages of moderate GBS. The characteristics of the patients reported here probably differ from the alterations reported in more severe GBS associated with respiratory failure, where symptoms of autonomic denervation are found [24].

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REFERENCES


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