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Autonomic dysfunction in Guillain-Barré syndrome

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SUMMARY The following tests of autonomic function were performed on seven patients with the Guillain-Barré syndrome and compared with controls: (1) measurement of heart rate and blood pressure in the supine and erect positions, (2) measurement of baroreflex sensitivity, (3) Valsalva's manoeuvre, (4) sweat test. In two patients the heart rates were fixed and greater than 100/min and in three there was postural hypotension. The baroflex sensitivity of four patients was abnormal and heart rate response to Valsalva's manoeuvre was impaired in two of the three patients who were able to perform the manoeuvre. Areas of anhidrosis were found in all seven patients. These abnormalities probably reflect pathological alterations of the sympathetic and parasympathetic components of the autonomic nervous system of patients with Guillain-Barré syndrome. The severity of autonomic involvement is not related to the degree of sensory and motor disturbance which is consistent with the patchy distribution of lesions throughout the peripheral and autonomic nervous systems.

Autonomic dysfunction may occur in the Guillain-Barré syndrome and account for some of the fatalities in the condition. Manifestations of autonomic involvement are disturbances of sweating,[1] heart rate and rhythm,[2 3] blood pressure control,[3-6] and sphincter, visceral and pupillary function.[1 6]

The baroreceptor-heart rate reflex has not previously been studied in a group of patients with Guillain-Barré syndrome, as it has been in patients with peripheral neuropathy due to diabetes, alcohol and porphyria.[7-9] In view of the potentially fatal disturbances of autonomic function that can occur in the Guillain-Barré syndrome, it seemed appropriate to investigate the abnormalities of sweating and baroflex control of heart rate and blood pressure in patients with this condition. A brief account of this study has already been published.[10]

Materials and methods

Tests of autonomic function were performed on five healthy subjects and seven patients with Guillain-Barré syndrome after informed consent had been obtained.

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Control subjects

The control subjects were five healthy males whose ages ranged from 20 to 49 years (mean 38; SD 8). They were free of hypertension, heart disease and neurological disease and were taking no medication.

Patients

The ages of seven patients (five males, two females) with Guillain-Barré syndrome ranged from 33 to 55 years (mean 43; SD 9). None suffered from hypertension or heart disease. The interval between the onset of weakness and the time of maximum disability ranged from 12 hours to three weeks. Six patients had a presumed viral illness at an interval ranging from three days to seven weeks before the onset of neurological symptons. The patients disability at the time of testing was graded on a scale from 0 to 5 (table 1). None of the patients required assisted ventilation. In five patients, the CSF protein concentration was greater than 0·4 g/l. Abnormalities of peripheral nerve conduction were demonstrated in all patients. Serum B_{12}, serum folate and glucose tolerance

Table 1 *Disability status of patients with GBS at time of autonomic testing*

Status	*Criteria*
0	Normal
1	Symptoms but no signs
2	Mild motor and sensory symptoms and signs
3	Moderate disability
4	Assistance needed with walking
5	Unable to walk

tests were normal and urinary porphyrins were not detected.

The interval between the onset of weakness and the autonomic tests ranged from one to nine weeks. Three patients were being treated with oral corticosteroids at the time of the investigations; one patient (Case 5) was receiving amitriptyline 75 mg at night and diazepam 15 mg/day, but no other patient was being treated with drugs which affected autonomic function.

Measurement of heart rate and heart period
Heart rate and heart period (R-R interval) were measured from the patients' electrocardiogram with a Grass EKG Tachograph preamplifier (Model 7P4D) and recorded using a Grass (Model 7B) pen recorder.

Measurement of arterial blood pressure
The arterial blood pressure was measured through a cannula inserted into the left brachial artery using a pressure transducer (Statham Model P23AC) placed at the approximate level of the aortic valve, a Grass (Model 7P1B) preamplifier and Model 7DAE driver amplifier and was recorded on another channel of the pen recorder.

Alteration of posture
The subjects lay quietly on a tilt table until the pulse and blood pressure remained steady. The subjects and patients were then tilted until either they were vertical or their systolic blood pressure fell below 90 mmHg. Except when postural hypotension was severe, subjects were tilted for five minutes before being returned to the horizontal position. Heart rate and blood pressure during tilting were recorded continuously.

Measurement of baroreceptor—heart-rate reflex sensitivity
The sensitivity of the baroreceptor-heart rate reflex was measured by comparing changes in the heart period with changes in systolic blood pressure. Transient elevations of arterial blood pressure were produced with intravenous injections of phenylephrine, an alpha-adrenergic agent that raises blood pressure by causing peripheral vasoconstriction while having little direct effect on heart rate. The drug was given intraveneously over 10-30 seconds in doses ranging from 25-300 μg. Up to six injections were given to each subject, at least two minutes being allowed between each injection. The baroflex sensitivity was calculated according to the method described by Smyth *et al.*[11] Each heart period was plotted against the systolic blood pressure of the preceding heart beat from the start of the rise in blood pressure to the peak, and the slope of the linear regression of these points so obtained was used as an estimate of baroreflex sensitivity (ms/mmHg).

Valsava's manoeuvre
The manoeuvre was performed with the subjects on the tilt table elevated to an angle of 30°. The subjects were asked to maintain a column of mercury in a manometer at a height of 40 mm for ten seconds using a mouthpiece which consisted of a syringe barrel. After several trial attempts the heart period and blood pressure were recorded before, during and 30 seconds after three successive manoeuvres. Approximately two minutes were allowed between each. The Valsalva ratio was calculated by dividing the longest heart period occurring after the manoeuvre by the shortest during the manoeuvre.[12] The highest ratio from the three attempts was recorded.[7]

The sweat test
Sweating was assessed by mapping the areas of skin which sweated in response to body heating. Each subject lay supine while a powder (alizarine red 35 G, rice starch 60 G and sodium carbonate 20 G) that changed colour from pale pink to deep purple when wet was dusted over all of the visible skin surface except the face. A heating cradle was placed over the subject's chest and abdomen and heat was applied until oral temperature had risen 1°C or until sweating began to occur on the face when the areas over which sweating occurred were recorded.

Statistical methods
Means are expressed with standard deviations and were compared using the two-tailed Student's t test.[13] The sample variances were compared using the Variance Ratio Test (Snedecor's 'F' test). If there was a significant difference between variances ($p < 0.05$) the means were compared using t', instead of t.[13] Data which differed by more than two standard deviations from the control mean were regarded as abnormal. The data relating heart period and systolic blood pressure were stored on paper tape. The linear regressions of the heart period and blood pressure were calculated with the aid of a PDP 11/40 computer using the method of least squares.[13] The significance of the regression coefficient was calculated.[13]

Results

The disability status of the seven patients with Guillain-Barré syndrome and the individual pooled results of autonomic function tests performed on the five control subjects and the seven patients are shown in Table 2.

HEART RATE
The resting heart rates of five control subjects ranged from 57 to 68 beats/min (mean 64 ± 4). Heart rates of the seven patients ranged from 73 to 118 beats/min (mean 92 ± 16). The differences are significant ($p < 0.02$). In two patients (Cases 1 and 5) the resting pulse rates were greater than 100/minute.

BLOOD PRESSURE
The resting arterial blood pressures of the five control subjects ranged from 115/65 to 160/95 mmHg (mean 132/77 ± 19/12). The resting arterial pressures in the seven patients, none of whom were known to be hypertensive prior to their illness, ranged from 122/66 to 175/92 (mean 153/87 ± 21/14). There was no significant difference in the mean systolic or diastolic blood pressures in the two groups, although in Cases 1 and 7 systolic blood pressures were

Table 2 *Summary of results of autonomic tests performed on five healthy subjects and seven patients with GBS*

	Number	Age	Sex	Disability status	Time from onset of GBS to date of tests (day)	Heart rate Supine (beats/minute)	Heart rate Erect (beats/minute)	Increase %	Arterial blood pressure Supine mmHg	Arterial blood pressure Erect mmHg	Arterial blood pressure Change %	Valsalva ratio	Baroreflex sensitivity ms/mmHg
Control subjects	1.	34	M	0	—	57	77	35	125/70	118/75	—5·6 / 7·1	2·4	15
	2.	45	M	0	—	63	71	13	160/95	155/98	−3·1 / 3·2	2·3	14
	3.	34	M	0	—	67	89	33	115/65	112/70	−2·6 / −7·7	2·29	9·1
	4.	49	M	0	—	65	73	12	118/73	110/68	−6·8 / −6·9	2·1	6·3
	5.	29	M	0	—	68	85	25	140/83	130/80	−7·1 / −3·6	2·7	11
	Mean	38	—	—	—	64	79	24	132/77	125/78	−5·0 / −1·6	2·5	*12 r = 0·84; n = 29
	SD	8	—	—	—	4	8	11	19/12	18/12	2·1 / 6·5	0·3	—
Patients	1.	48	F	5	30	118	120	1·7	175/92	153/90	−13 / 2·2	—	0·16
	2.	35	M	2	20	83	100	21	135/77	130/73	−3·7 / −5·2	2·9	17
	3.	44	M	5	32	88	112	27	122/66	112/66	−8·2 / 0	1·5	5·6
	4.	54	M	5	17	83	92	11	140/75	136/71	−2·9 / −5·3	1·7	8·4
	5.	55	F	5	63	110	112	1·8	168/98	100/65	−41 / −34	—	0·41
	6.	33	M	2	13	86	104	21	155/100	112/65	−28 / −35	—	3·0
	7.	32	M	5	7	73	95	30	173/100	121/80	−30 / −20	—	5·1
	Mean	43	—	—	—	92	105	16	153/87	123/73	−18 / −14	2·0	*5·7 r = 0·50; n = 42
	SD	10	—	—	—	16	10	12	21/14	18/9	15/15	—	—
	p	>0·05				<0·02		>0·05	>0·05 / >0·05		>0·05 / 0·05		<0·01

(*Slope of regression of pooled data)

abnormal (table 2). In one patient (Case 5), the blood pressure fluctuated during the course of the illness to levels as high as 230/120.

EFFECT OF POSTURE ON HEART RATE AND BLOOD PRESSURE

The heart rate was measured immediately before tilting and as soon as tilting was complete. The mean increase in control subjects was 24% (±11%) and in patients it was 16% (±12%). There was no significant difference in the two groups, although in Cases 1 and 5 the heart rate increased only 1·7% and 1·8% respectively, both of which are abnormal.

The systolic blood pressures of all control subjects fell slightly on tilting, the mean fall being 5% (±2·1%). The mean fall in the systolic blood pressure of patients was 18% (±15%) which did not differ significantly from the control mean. In all patients the systolic blood pressure fell on tilting and in Cases 1, 5, 6 and 7 the percentage fall was abnormal.

The diastolic blood pressures fell in three and increased in two of the control subjects when tilted, the mean change being −1·6% (±6·5%). The diastolic blood pressure fell on tilting in six of the patients and remained unchanged in the seventh. The mean change was −14% (±15%) which was not significantly different from the control mean but in Cases 5, 6 and 6 the fall in diastolic blood pressure was abnormal.

The changes in heart rate and the blood pressure responses to tilting in a control subject and in two patients are shown in fig 1.

BAROFLEX SENSITIVITY

The range of sensitivity of the baroreceptor-heart-rate reflex was 6·3 to 15 ms/mmHg in five control subjects (mean 12 ms/mmHg; r = 0·84; N = 29). In the seven patients the range of baroreflex sensitivity was 0·16 to 17 ms/mmHg; r = 0·50; n = 42). The difference was significant ($p < 0·01$) (figs 2 and 3). When the data for each patient were compared with the pooled control data, the baroreflex sensitivities of Cases 2, 3 and 4 did not differ significantly

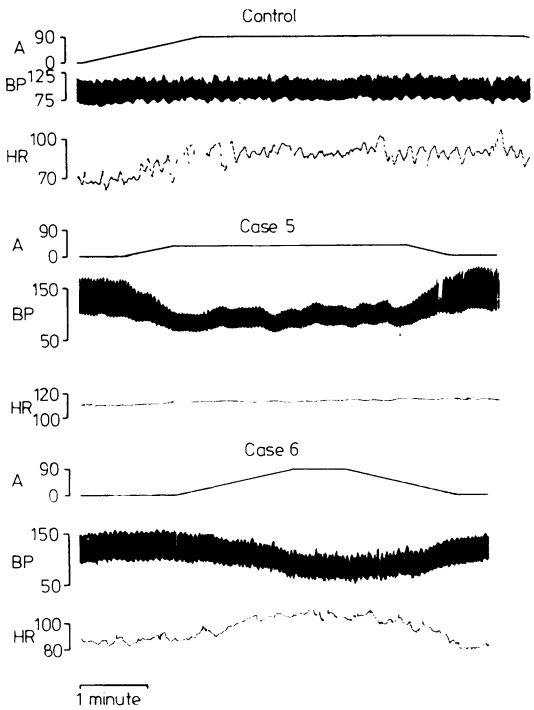

Fig 1 *Records obtained from one control subject and two patients with GBS (Cases 5, 6) showing the changes in heart-rate and blood pressure in response to tilting. Postural hypotension is present in both patients and in Case 5, the heart-rate is fixed at 110 per minute (A = angle of tilt; BP = arterial blood pressure, mmHg; HR = heart rate, beats/minute).*

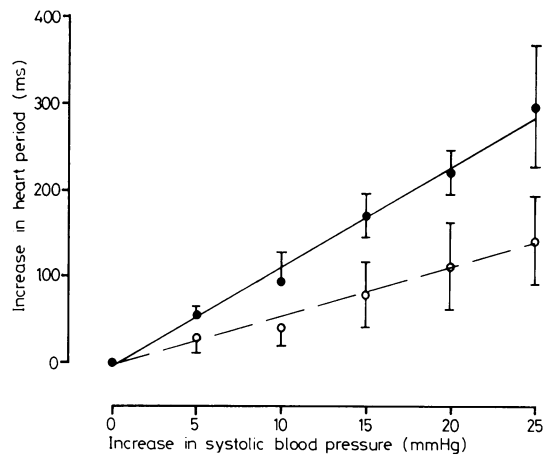

Fig 2 *The relationship between the increase in heart period and the increase in systolic blood pressure following intravenous injections of phenylephrine. The closed circles represent the means of the pooled data from the 5 control subjects and the open circles represent the means of the pooled data from the 7 patients with GBS. Vertical bars represent ± 1 SE. The slopes of the regression lines are 12 ms/mmHg for the control subjects ($r = 0{\cdot}84$, $n = 29$) and 5·7 (ms/mmHg) for the patients with GBS ($r = 0{\cdot}50$, $n = 42$). The difference between the slopes was significant ($p < 0{\cdot}01$).*

from the control sensitivity. The sensitivity was significantly less than the control value in Case 7 ($p < 0{\cdot}05$) and in Cases 1, 5 and 6 ($p < 0{\cdot}01$). Two of the patients (Cases 1 and 5) had baroreflex sensitivities of only 0·16 and 0·41 ms/mmHg, respectively (fig 3). Thus, their heart rates remained almost unchanged despite increases of up to 30 mmHg in systolic blood pressure. Both of these patients had fast resting heart-rates (118 and 110 beats/min respectively) and one (Case 5) had postural hypotension.

VALSALVA'S MANOEUVRE

When the five healthy subjects performed Valsalva's manoeuvre there was an initial transient increase in the arterial blood pressure (Phase I) which then fell to a level less than the resting value at which it remained until forced expiration ceased (Phase II). At that moment, there was a transient fall in arterial blood pressure (Phase III). The blood pressure then rapidly increased to a level above the resting value to which it returned over the subsequent 15-40 seconds (Phase IV). The mean ratio of the shortest R-R interval (Valsalva ratio) was 2·5 ± 0·3).

Facial and respiratory weakness prevented four patients from performing Valsalva's manoeuvre. In the other three the blood pressure changes during and after the manoeuvre were normal. In Case 2 the Valsalva ratio was in the control range while in Cases 3 and 4 the ratios were 1·5 and 1·7 which were below the normal range.

SWEAT TEST

A sweat test was attempted on all seven patients. One patient (Case 5) was unable to tolerate the test but she had not demonstrated any signs of sweating after her oral temperature had risen from 35·8° to 36·4°C. The other six patients were all able to tolerate heating, and all had some areas of anhidrosis (fig 4). Two patients (Cases 3 and 7) had complete anhidrosis of the lower limbs and three patients (Cases 1, 3 and 6) had areas of anhidrosis on their abdominal walls.

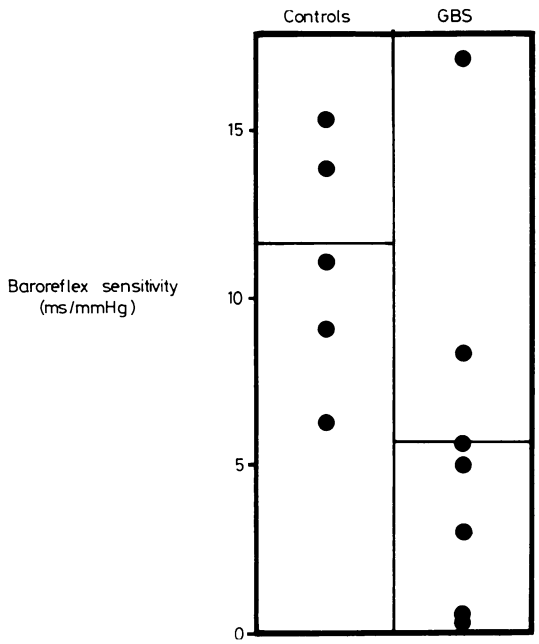

Fig 3 *The baroreflex sensitivities of 5 healthy subjects and 7 patients with GBS. The baroreflex sensitivity of the control group was 12 ms/mmHg ($r = 0{\cdot}84$, $n = 29$) and of the patient group was 5·7 ms/mmHg ($r = 0{\cdot}50$, $n = 42$). The difference between the two sensitivities was significant ($p < 0{\cdot}01$).*

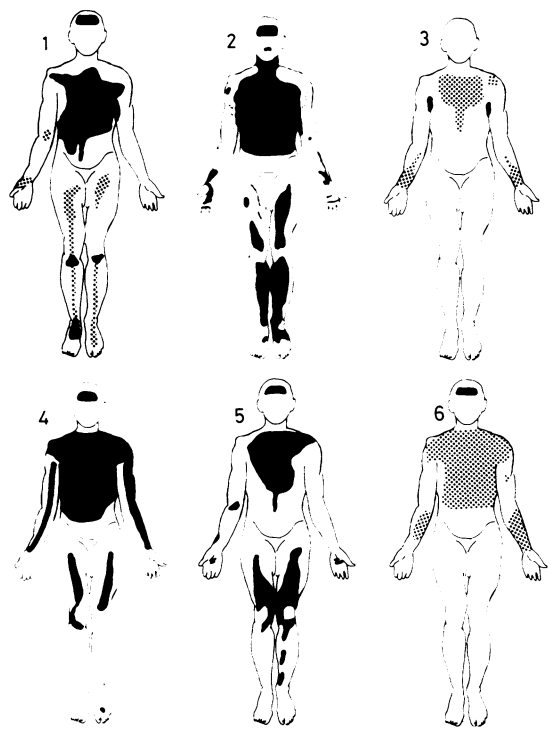

Fig 4 *Patterns of sweat production in 6 patients with GBS. Black areas indicate regions of normal sweat production while the spotted areas are those in which sweating was patchy.*

Discussion

The results of the present investigation have shown that in patients with Guillain-Barré syndrome there are abnormalities of control of heart rate, blood pressure and sweating. Although only five control subjects were studied in detail, the values obtained were similar to those of other workers for the mean resting heart rate[7 14] and change in heart rate[15] and blood pressure with tilt.[16] The increased mean resting heart rate in the seven patients, in two of whom (Cases 1 and 5) it remained relatively fixed throughout the study at greater than 100/minute, is consistent with the observations of previous investigators who noted tachycardia in this condition.[1–3] Increased resting heart rates have been observed in patients with diabetes mellitus[7] and acute intermittent porphyria.[17] In both these conditions the tachycardia has been attributed to lesions of the vagus or glossopharyngeal nerves or both.[17 18] A fixed tachycardia also occurs when the autonomic nerve supply to the heart is blocked pharmacologically.[15] Therefore, it is likely that the relatively fixed rapid heart rate of Cases 1 and 5 was a result of involvement of the cardiac autonomic nerve supply. In cases 1 and 7 supine systolic blood pressures were greater than two standard deviations above the control mean and in Case 5 the blood pressure fluctuated considerably during the course of the illness reaching levels as high as 230/130. Elevated or fluctuating arterial blood pressure has been documented previously in Guillain-Barré syndrome[1 2 5] and may be caused by lesions of the glossopharyngeal nerves which contain afferent fibres from the arterial baroreceptors. The hypothesis that lesions of the afferent nerves from arterial baroreceptors are responsible for hypertension, fluctuating arterial blood pressure and tachycardia is consistent with studies on experimental animals and human subjects with lesions of these nerves.[19–21] It is significant that the patient who had fluctuating arterial blood pressure and tachycardia (Case 5) had left-sided palatal weakness, dysphagia and dysphonia, clinical features that are consistent with a lesion of the left vagus nerve.

A significant fall in both the systolic and diastolic arterial blood pressures occurred on tilting three of the seven patients with Guillain-Barré syndrome.

Orthostatic hypotension has been found in patients with Guillain-Barré syndrome by other investigators[1 4 5] and is also a common finding in patients with diabetic autonomic neuropathy.[7] It does not normally occur in healthy subjects because of the control of arterial blood pressure by the baroreflexes. On assuming the upright position there is an increase in splanchnic vascular resistance which contributes substantially to the overall vascular resistance.[22] The decrease in splanchnic blood flow is attributed to arteriolar constriction which is mediated by the sympathetic nervous system.[23] It has been shown by direct intraneural recording of sympathetic nervous activity from muscle nerves in man that changing position from supine to sitting or standing increases muscle sympathetic nerve activity.[24] The veins may also play some part in maintaining arterial blood pressure in the upright position.[25] The distensibility of superficial veins decreases in normal subjects during the performance of Valsalva's manoeuvre[26] but in some patients with Guillain-Barré syndrome this response has been found to be absent presumably due to involvement of their sympathetic nerve supply.[27] The postural hypotension of patients in the present study was probably related to lesions of the sympathetic nervous supply to the arterioles and veins or of the afferent fibres from the arterial baroreceptors. Of the three patients in the present study with marked orthostatic hypotension, there was evidence in two (Cases 6 and 7) that the baroreceptor afferent fibres were partially intact because the changes in their heart rate with increases in blood pressure were qualitatively normal. The third patient with postural hypotension (Case 5) had a fixed heart rate as well as severe postural hypotension which may have been caused by lesions of the sympathetic and vagus nerves on the efferent side of the baroreflex arc and/or lesions of the afferent fibres from the baroreceptors. In this patient there was clinical evidence of involvement of one vagus nerve in which afferent fibres from the aortic arch baroreceptors and efferent fibres to the heart could also have been affected. However from the present study it is impossible to determine exactly the site of the autonomic lesions. The absence of postural hypotension in one (Case 1) of the two patients who had a resting tachycardia is evidence that the baroreceptor afferent and sympathetic efferent fibres were intact but that the vagal efferent fibres to the heart were affected.

The sensitivity of the heart-rate-baroreceptor reflex was measured in one patient with Guillain-Barré syndrome by Davidson and Jellinek[28] but the present study is the first in which changes in the sensitivity of the reflex have been documented in a group of such patients. The method used measures rapid changes in heart rate which are mediated largely by the parasympathetic nervous system in response to changes in arterial pressure.[11 14 23] The method has been used to demonstrate impairment of baroreflex sensitivity in patients with diabetic neuropathy.[29] It is simpler than that devised by Korner *et al*,[14] which measures the steady state characteristics of the baroreflex and which has been used previously in our laboratory.[7 8] Lesions in the afferent nerve fibres in the glossopharyngeal and vagus nerves from the carotid sinus and aortic arch baroreceptors and/or efferent fibres in the vagus nerves may reduce the sensitivity of the baroreflex which was found in four of the patients in the present study and in the patient of Davidson and Jellinek.[28] The glossopharyngeal and vagus nerves are known to be affected both clinically[6] and pathologically[30] in Guillain-Barré syndrome although in the present study only one (Case 5) of the four patients with an abnormal baroreceptor heart rate reflex had obvious clinical evidence of involvement of these cranial nerves.

Impaired function of the baroreflexes could be due to a lesion or lesions in the vasomotor centres of the medulla. This explanation is unlikely in a disease of the peripheral nervous system in which pathological changes within the brainstem are usually minimal or absent.[3 30] However, extensive changes in the brainstem involving the vagal nuclei have been described[31] and physiological studies[32] provide some evidence that central autonomic pathways maybe involved.

None of the patients in the present study suffered from heart disease and the only ECG abnormality was sinus tachycardia. However, pathological changes have been found in the hearts of some patients who died with Guillain-Barré syndrome[3] although Bredin[2] could find no abnormalities in the hearts of two patients who died apparently as a result of cardiac complications of the syndrome.

Abnormalities of the heart rate and blood pressure responses to Valsalva's manoeuvre have been reported in Guillain-Barré syndrome,[5 27] but the three patients in the present study who were able to perform the manoeuvre had normal responses, although in two, the Valsalva ratio was below the control range of the present study. None of the three patients had orthostatic hypotension or abnormal baroreflex sensitivities and it was concluded that they had minimal disturbance of reflex autonomic cardiovascular control. It is probable that the patients with postural hypotension and impaired baroreflex sensitivities would have had an abnormal Valsalva response if they had been able to perform the manoeuvre.

The sweat test was abnormal in six patients in whom it was performed satisfactorily. Absent or

impaired sweating was found on the abdominal wall of three patients—two of whom had postural hypotension (Cases 6 and 7). Since surgical resection of the lower thoracic sympathetic ganglia and splanchnic nerves cause postural hypotension[33] and anhidrosis of the abdominal wall,[34] it is suggested that the anhidrosis and postural hypotension in Cases 6 and 7 was due to involvement of the lower thoracic and upper lumbar segments of the sympathetic chains or splanchnic nerves or both. Postural hypotension in patients with diabetes mellitus has been attributed in part to pathological changes that occur in the greater splanchnic nerves.[7]

The present investigations have demonstrated varying degrees of impairment of autonomic nervous system function in all seven patients. The findings suggest that lesions may occur in the afferent fibres from the arterial baroreceptors in the vagus and glossopharyngeal nerves, in the efferent parasympathetic fibres in the vagus nerves which innervate the heart and in the sympathetic fibres which control sweating and vasomotor tone. Pathological studies have demonstrated demyelinating lesions in the glossopharyngeal and vagus nerves[30 31] and in the sympathetic chains and white rami.[35] The severity of involvement of the autonomic nervous system does not appear to be related to the degree of motor and sensory disturbance. The consequences of involvement of the autonomic nervous system in Guillain-Barré syndrome are negligible in many patients but on occasion they can be life threatening. Postural hypotension may lead to syncope and irreversible brain damage in a paralysed patient who is inadvertently left in a sitting position or who requires an anaesthetic.[36] Sudden cardiovascular collapse may be the terminal event. Although the mechanism is uncertain involvement of the autonomic nervous system has been implicated not only in Guillain-Barré syndromes[1 2 5 26] but also in diabetic autonomic neuropathy.[37]

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