Postural Rearrangement in IDDM Patients With Peripheral Neuropathy

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OBJECTIVE — To evaluate the influence of diabetic peripheral neuropathy on postural strategy.

RESEARCH DESIGN AND METHODS — Static posturography and nerve conduction velocity were performed in the following age-matched subjects: 10 IDDM patients with peripheral neuropathy, 23 IDDM patients without peripheral neuropathy, and 21 control subjects. All subjects with signs or symptoms of postural instability were excluded from the study. The following posturographic parameters were drawn: *1*) velocity of body sway, expressed as mean velocity and average of the SDs, 2) VFY, the parameter derived from the velocity variance and the anteroposterior mean position of the body (this parameter monitors the postural strategy pursued by the subject), and 3) fast Fourier transformation on the *x* (FFTX) and *y* (FFTY) planes, spectral analysis of the frequencies of body oscillation on frontal (*x*) and anteroposterior (*y*) planes.

RESULTS — Mean velocity and its SD were higher in IDDM patients with peripheral neuropathy than in control subjects and IDDM patients without peripheral neuropathy ($P < 0.001$). VFY was increased in IDDM patients with peripheral neuropathy versus control subjects and IDDM patients without peripheral neuropathy ($P < 0.01$). A direct relationship was found between parameters of posturography and some parameters of nerve conduction tests.

CONCLUSIONS — Diabetic patients with peripheral neuropathy demonstrate a shift from physiological ankle control to hip postural control as monitored by specific posturography analysis.

Body position in space results from several sensory inputs to the central nervous system and related motor outputs (1). Alterations of these inputs/outputs can give rise to postural reorganization (2). In a previous report from our group, abnormalities of posture have been documented in IDDM patients with peripheral neuropathy, suggesting a variation of posture-maintaining strategies (3).

The variation of postural pattern from ankle to hip strategy has been linked to various physiological and pathological conditions (4,5). These variations can be monitored by specific posturographic evaluations such as velocity mean and its SD, fast Fourier transformation (FFT) on *x* and *y* planes, and variance of velocity as a function of the body center of gravity on the anteroposterior axis (VFY) (6).

The aim of this study is to evaluate posturography in IDDM patients to demonstrate the suspected shift of postural control from ankle to hip strategy in neuropathic subjects.

RESEARCH DESIGN AND METHODS

— The same 54 subjects evaluated in our previous study (3) were enrolled. They were free from any interfering neurological or labyrinthine disorders, without clinical evidence of postural instability or postural hypotension and with correct visual acuity (10/10). Of the subjects, 33 were affected by IDDM, 23 without peripheral neuropathy and 10 with peripheral neuropathy, according to San Antonio Consensus Conference guidelines, namely the presence of both signs and symptoms of peripheral neuropathy and pathological results of nerve conduction velocity (7). The remaining 21 were normal age-matched control subjects. Table 1 shows the clinical profile of the study population.

Static posturography was performed on a standard platform (S.Ve.P. Amplaid), according to the method previously published (8). The center of gravity is monitored during the test performed with the patient's eyes open and with them closed.

The following parameters were drawn.

1. Velocity of center of gravity sway: this was recorded at 10 Hz frequency of acquisition, expressed as mean velocity, which expresses the body sway related to time, and average SD, which quantifies the variations of velocity. This is a more predictable test for the evaluation of the control of postural muscles (9).
2. VFY: in young normal adults, the stance is held by a contraction of posterior leg muscles that counterbalances the natural tendency of the center of gravity to shift forward on the anteroposterior axis. The close correlation between the mean center of gravity position on anteroposterior axis (mean *y*) and velocity variance has been demonstrated experimentally (6) and is expressed by a mathematical relationship: variance (V) = *f*(mean *y*). This function monitors short length–high velocity compensating movements used to maintain the upright position. The VFY parameter evaluates by the distance of the point representative of the subject from the normal curve (9).
3. Fast Fourier transformation on *x* and *y* (FFTX, FFTY) (3).

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FFT, fast Fourier transformation; VFY, parameter derived from velocity variance and anteroposterior mean position of the body.

Table 1—*Study subjects*

	Control subjects	IDDM patients	IDDM patients with neuropathy
n	21	23	10
Sex (M/F)	11/10	11/12	6/4
Age (years)	31 ± 0.9	31 ± 1.1	35 ± 1.9
Disease duration (years)	—	9.9 ± 0.84	16.5 ± 1.5*

*$P < 0.01$ vs. IDDM patients.

Table 2—*Posturography*

	Control subjects	IDDM patients	IDDM patients with neuropathy
Sway velocity (mm/sec)			
Eyes open	4.65 ± 0.7	7.6 ± 2.2	12.08 ± 3.7*†
Eyes closed	6.15 ± 0.7	11.2 ± 4.08	20.7 ± 7.9*†
Velocity SD			
Eyes open	5.07 ± 0.3	6.1 ± 0.7	8.2 ± 0.9*†
Eyes closed	7.9 ± 0.6	8.9 ± 2.3	16.5 ± 2.7*†
FFTY (Hz)			
Eyes open	0.02 ± 0.01	0.05 ± 0.04	0.05 ± 0.03
Eyes closed	0.03 ± 0.02	0.11 ± 0.25	0.10 ± 0.14
FFTX (Hz)			
Eyes open	0.05 ± 0.06	0.10 ± 0.14	0.06 ± 0.05
Eyes closed	0.03 ± 0.01	0.16 ± 0.22	0.09 ± 0.09
VFY	−0.44 ± 0.29	0.91 ± 0.39	3.5 ± 1.3*†

Data are means ± SD. Posturography results are divided by group. *$P < 0.01$ vs. control subjects; †$P < 0.01$ vs. IDDM patients.

The presence of peripheral neuropathy was quantitatively assessed by nerve conduction velocity, amplitude and latency of sensory (sural) and motor (peroneal) nerves, and sensory conduction velocity, assessed orthodromically.

Statistical analysis (Statview-Macintosh Apple) was performed by variance, regression, and multivariate analyses. $P < 0.05$ was considered statistically significant.

RESULTS — Mean velocity of sway, velocity SD, and VFY were higher in IDDM patients with peripheral neuropathy than in control subjects and IDDM patients without peripheral neuropathy. FFTX and FFTY were similar in all groups both when the test was performed with patients' eyes open and when it was performed with their eyes closed (Table 2).

Electrophysiological testing showed an impairment of both sensory and motor fibers in the group of IDDM patients with peripheral neuropathy (Table 3). Table 4 shows the correlations between posturography and nerve function tests.

Among posturography parameters, a significant correlation with age was found with velocity (closed eyes) ($r = 0.53$, $P = 0.0005$) and open eyes ($r = 0.41$, $P = 0.009$). In a model of multiple correlation adjusted for age and neuropathy, a significant correlation with duration of disease was found for velocity SD ($r = 0.30$, $P = 0.03$) and FFTY (open eyes) ($r = 0.4$, $P = 0.03$).

CONCLUSIONS — Normally, young subjects keep their orthostatic rest position by an anterior-posterior (*y* plane in posturography *x-y* reference planes) sway pattern that resembles an inverted pendulum: the fulcrum is the ankle, and the head is the opposite end of the pendulum. This is the so-called ankle strategy. The strategy is maintained by the contraction of posterior leg muscles (9) that generate low velocity–long distance balance sways. This results from torque about the ankle joints to resist the gravity and rotate the body as a relatively rigid mass about the ankles.

In case of jeopardized stability, the ankle pattern is shifted to the hip pattern. The latter is a double inverted pendulum movement on the anteroposterior plane in which the fulcra are located in both hip and ankle joints. This strategy uses rapid rotational accelerations about the hips to generate transient horizontal shear forces against a support surface (5). High velocity–short distance body sways result from this postural pattern. This kind of body sway tends to relax posterior leg muscles and contract anterior leg muscles.

In the literature, some evidence has been reported to demonstrate the negative influence of diabetic neuropathy on postural control (3,10,11). In particular, we have observed in neuropathic diabetic patients evaluated by static posturography an increased trace surface, trace length, and velocity (3). Among the postural parameters evaluated in this study,

Table 3—*Nerve conduction parameters*

	Control subjects	IDDM patients	IDDM patients with neuropathy
Sural			
Latency (m/sec)	3.1 ± 1.2	3.48 ± 0.96	4.02 ± 1.1†‡
Amplitude (μV)	18.9 ± 1	15.1 ± 0.93	9 ± 1.2†§
Velocity (m/sec)	48 ± 0.5	47.54 ± 0.89	39.9 ± 0.9†§
Peroneal			
Latency (m/sec)	3.77 ± 0.16	4.08 ± 0.14	6.6 ± 0.78†‡
Amplitude (μV)	5.1 ± 0.9	5 ± 0.58	2.06 ± 0.414†§
Velocity (m/sec)	48.3 ± 0.91	46.2 ± 0.5	34.34 ± 1.4†§

Data are means ± SD. Neuroelectrophysiologic parameters were recorded in both sides. The sural nerve was explorable in only 6 out of 20 nerves in the group of IDDM patients with neuropathy. †$P < 0.01$ vs. control subjects; ‡$P < 0.05$ vs. IDDM patients; §$P < 0.01$ vs. IDDM patients.

Table 4—*Posturography versus neurophysiology*

	Velocity (eyes open/closed)	Velocity SD (eyes open/closed)	FFTY (eyes open/closed)	VFY
Sural				
Latency	0.4/0.5 (0.03/0.001)	NS/NS	NS/NS	NS
Amplitude	0.6/0.7 (0.0004/0.0001)	0.5/0.45 (0.001/0.005)	NS/NS	NS
Velocity	0.4/0.6 (0.02/0.0005)	NS/0.34 (NS/0.03)	NS/NS	0.4 (0.02)
Peroneal				
Latency	0.3/0.6 (0.002/0.005)	0.36/0.44 (0.02/0.006)	0.46/0.3 (0.001/0.03	NS
Amplitude	NS/0.3 (NS/0.05)	0.3/0.33 (0.05/0.04)	NS/NS	0.5 (0.007)
Velocity	0.5/0.7 (0.002/0.0001)	0.34/0.45 (0.03/0.04)	NS/NS	0.5 (0.004)

Data are *r* (*P*). Linear regression between some posturography parameters and neurophysiological results.

the sway velocity and its SD and the VFY were affected by the presence of neuropathy.

The increased trace surface previously reported (3) in diabetic neuropathic patients is a sign of decreased ability to stabilize the body. The increase of velocity and its SD found in this study is due to a compensating mechanism achieved to maintain posture at the expense of the major energy involved. The increase of VFY indicates that neuropathic patients show a compensatory abnormal pattern of posture (6).

Our young subjects tend to compensate their neural impairment by shifting from the physiological ankle postural control to the hip control typical of the elderly (12). In normal young subjects (<40 years of age), hip control is used only for correction of center of gravity when it reaches the limits of stability (5) or after sensory input disruption induced by ischemia (4,5). It is otherwise well known that somatosensory inputs from the feet are the dominant inputs to maintain posture under normal support surface conditions (5).

Postural data matched to neurophysiological results show that the impaired postural performances of diabetic-neuropathic subjects are related to the reduction of peripheral, sensory and motor, neural conduction. These impairments seem to be partially compensated by shifting from the physiological ankle control to hip postural control. The presence of hip control in diabetic neuropathic patients is of relevance, since this control pattern may lead to decreased stability of the body (13).

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