The role of weakness of triceps surae muscles in astasia without abasia

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Abstract

Objective—To investigate the role of weakness of the bilateral triceps surae muscles—the gastrocnemius and soleus muscles—in astasia without abasia and whether compensating for muscle weakness with ankle-foot orthoses improves this disability.

Design—Case-control study of clinical findings and before and after trial of ankle-foot orthoses.

Setting—Clinics of the departments of rehabilitation medicine of two university hospitals.

Patients—A stilt group consisting of 23 patients with astasia without abasia, and a non-stilts group without this phenomenon consisting of 12 patients with hereditary motor sensory neuropathy, 15 patients with lumbosacral spondylotic radiculopathy or spondylolisthesis, and 20 healthy volunteers.

Main outcome measure—Clinical findings of the stilts and non-stilts groups were compared and the sensitivity and specificity of each clinical finding was calculated. The length of the centre of foot pressure (COP) while standing was measured in a bilateral below knee amputee and 16 consecutive patients in the stilts group with and without ankle-foot orthoses.

Results—Weakness of the triceps surae muscles was the only finding that differed significantly between the two groups and was both sensitive and specific. The amputee was unable to stand in place without dorsiflexion bumpers, which functioned similarly to the triceps surae muscle. Bilateral ankle-foot orthoses improved the COPs of 14 out of 16 patients.

Conclusion—The main cause of astasia without abasia is weakness of the triceps surae muscles, and this disability is improved by bilateral ankle-foot orthoses.

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Keywords: astasia without abasia; disturbance of stance; stilts phenomenon; peripheral neuropathy

Although it is generally thought that a person who is able to walk is able to stand, we have encountered patients who are able to walk but unable to stand in one place. This curious disability can occur in patients with muscular

weakness and sensory disturbance of the lower limbs caused by peripheral neuropathies, spinal root or spinal cord lesions, and myopathies. The symptom was named "astasia without abasia" by Hirayama *et al*^{1,2} and is defined as an inability to maintain a stationary position to assume and keep an upright posture, although the legs are under control in walking. No other reports of the disability have been found. As a patient with astasia without abasia resembles a child on stilts who walks steadily but continuously steps forward and backward while trying to stand in place, it is also called the "stilts phenomenon."^{1,2} Hirayama *et al*² suggested that astasia without abasia is caused by a combination of three conditions: (1) loss of the ankle pattern of postural movements because of decreased muscular strength and tone about the ankle joints; (2) lack of compensatory postural movements by the hip pattern, which is dependent on sufficient strength about the hip joints; and (3) impairment of somatosensory feedback from the legs for postural stabilisation.

We have noticed that a patient who had weakness of the bilateral triceps surae muscles without sensory disturbances had astasia without abasia that improved with ankle-foot orthoses. Therefore, we examined patients who had and others who did not have astasia without abasia to determine what part weakness of the triceps surae muscle—which is composed of gastrocnemius and soleus muscles—plays in astasia without abasia and whether compensating for the muscle weakness with ankle-foot orthoses reduces this disability.

Patients and methods

This investigation consisted of three studies: statistical analysis of physical findings of patients with astasia without abasia; measurement of the stability of a bilateral below-knee amputee while standing; and evaluation of the efficacy of ankle-foot orthoses.

STUDY 1

Clinical criteria for astasia without abasia were devised by two of us (KH and MY) as follows: (1) a patient can keep the body more stable while walking than while standing and (2) a patient takes a step or must be supported within one minute after beginning to stand in place. Astasia without abasia was considered mild when a patient fulfilled only the first criterion and was considered severe when a patient fulfilled both criteria. Twenty three consecu-

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tive patients with astasia without abasia (stilts group) were identified from among patients who were referred to clinics at the Department of Rehabilitation Medicine, University of Occupational and Environmental Health Hospital, and at Kumamoto University Hospital for further rehabilitation from 1988 to the end of 1995. Clinical diagnoses of patients were previously established at the departments of neurology or orthopaedic surgery as follows: hereditary motor sensory neuropathy (five patients); spinal progressive muscular atrophy (two); acute inflammatory demyelinating polyradiculoneuropathy (two); familial amyloid polyneuropathy (two); hereditary sensory autonomic neuropathy (one); vasculitic neuropathy (one); beriberi (one); peripheral neuropathy of unknown aetiology (two); distal myopathy with rimmed vacuoles (one); myotonic dystrophy (one); cauda equina injury (two); lumbosacral spondylotic radiculopathy (one); spondylolisthesis (one); and myelomeningocele (one).

Forty eight subjects who did not fulfil the criteria for astasia without abasia (non-stilts group) consisted of 12 patients with hereditary motor sensory neuropathy (NS-HMSN), 15 patients with lumbosacral spondylotic radiculopathy or spondylolisthesis (NS-LSR), and 20 healthy control subjects (NS-C). The NS-HMSN group consisted of consecutive patients referred to the clinic at the Department of Rehabilitation Medicine for gait training. The NS-LSR group consisted of consecutive patients referred to the clinic for muscle strengthening and gait training after surgery of the lumbar spine, and had weakness in the unilateral tibialis anterior or triceps surae muscles or both. The NS-C group consisted of volunteers who had never had neuromuscular disorders.

Neuromuscular testing was performed with standard techniques in all subjects. Muscle strength of the iliopsoas, gluteus maximus, quadriceps femoris, hamstrings, tibialis anterior, and triceps surae muscles was manually

evaluated as 5, 4, 3, 2, 1, or 0³; muscle weakness was defined as muscle strength of 2 or less. Range of motion of the ankle joint was measured with a goniometer,⁴ and limitation of the range of motion was defined as a range of motion of 5° or less. Muscle stretch reflexes of the knee and ankle were elicited with a hammer, and absent or diminished reflexes of the knee and absent reflexes of the ankle were defined as diminution of the muscle stretch reflex.⁵ Touch and pain on the dorsum of the foot and position and vibration sense of the great toe were rated as "severely disturbed," "moderately disturbed," "slightly disturbed," or "normal",⁵ and a sensation of less than normal was defined as sensory disturbance. Coordination of the lower limbs was rated as "disturbed," "slightly disturbed" or "normal",⁵ and coordination of the less than normal was defined as ataxia. Each result of the tests was converted into "no disturbance," "unilateral disturbance," or "bilateral disturbance" to allow use of the χ^2 test.

For the five patients in the stilts group in whom sensory disturbances were not found during physical examination, sural nerve conduction velocities,⁶ somatosensory evoked potentials (SEPs) to tibial nerve stimulation,⁷ and cutaneous vibration and thermal cooling thresholds⁸ were evaluated to confirm the presence or absence of sensory abnormalities.

STUDY 2

To study whether compensating for weak triceps surae muscles improves stability while standing, the length of the centre of foot pressure (COP) was measured in a patient who had had both legs amputated below the knee five years earlier because of frostbite. The patient had normal muscular strength in the lower limbs; had no disturbances of touch, pain, position, or vibration sense of the stumps; and was able to walk and run with prostheses.⁹ Results of SEP studies were normal. The COP was taken as the projection of the centre of gravity on to the force plates (Kistler, Switzerland), on which the amputee stood for 30 seconds with the artificial feet (Superankle©, LAPOC, Japan) maintained at 40° between the feet and the eyes open. Each measurement was repeated five times before and after dorsiflexion bumpers, which prevented excessive dorsiflexion of the feet, were attached to the feet (fig 1). The COPs were expressed in cm/30 s, and the average was calculated with a personal computer (PC9801BA, NEC, Japan). When the amputee took a step or was supported by members of the medical staff during the measurement at least once per five trials, the COP was defined as unmeasurable.

STUDY 3

Ankle-foot orthoses¹⁰ were fitted for 22 patients in the stilts group to improve stability while standing; a foot orthosis¹⁰ was made for one patient because of ulcers on the bottom of the feet. Patients subjectively evaluated stability while standing with orthoses as worse, unchanged, improved, and much improved.

Figure 1 The below-knee prosthesis. The lateral half of the artificial foot is removed to show the mechanism of the superankle. The dorsiflexion bumper supports the shaft and prevents excessive dorsiflexion while standing.

Comparison of clinical findings between the stilts and non-stilts groups

	Stilts group (n = 23)	Non-stilts group (n = 47)	P value	Sensitivity	Specificity
Muscular weakness:					
Bil gluteus maximus	5	1	0.006	0.83	0.72
Unil gluteus maximus	0	6	0.168	0.00	0.64
Bil tibialis anterior	18	4	< 0.001	0.82	0.90
Unil tibialis anterior	0	4	0.150	0.00	0.65
Bil triceps surae	22	1	< 0.001	0.96	0.98
Unil triceps surae	0	5	0.164	0.00	0.65
Limitation of range of motion:					
Bil ankle joints	2	6	0.615	0.25	0.66
Unil ankle joint	0	1	1.000	0.00	0.67
Diminution of muscle stretch reflex:					
Bil knee joints	15	13	0.003	0.54	0.81
Unil knee joint	0	1	1.000	0.00	0.67
Bil ankle joints	23	13	< 0.001	0.64	1.00
Unil ankle joint	0	14	0.003	0.00	0.59
Sensory disturbances:					
Touch on bil dorsi	16	16	0.005	0.50	0.82
Touch on unil dorsum	0	8	0.046	0.00	0.63
Pain on bil dorsi	16	16	0.005	0.50	0.82
Pain on unil dorsum	1	8	0.254	0.11	0.64
Position of bil toes	15	12	0.001	0.56	0.81
Position of unil toe	0	6	0.076	0.00	0.64
Vibration on bil toes	17	15	0.001	0.53	0.84
Vibration on unil toe	0	6	0.168	0.00	0.64

Muscular weakness of the iliopsoas, quadriceps, and hamstrings, and coordination are not shown because of low frequencies. bil = bilateral; unil = unilateral; dorsi = dorsi of the feet; dorsum = dorsum of the foot; toe(s) = big toe(s).

The COP was also measured in 16 consecutive patients in the stilts group.

STATISTICAL METHODS

Student's *t* test and one way analysis of variance (ANOVA) were used to examine the differences in age between the stilts and non-stilts groups and among the NS-HSMN, NS-LSR, and NS-C groups. The χ^2 test was used to detect any difference in sex ratio between the two groups. Each clinical finding between the two groups was compared using the χ^2 test and Fisher's exact test, if necessary. Sensitivity was the rate of patients with astasia without abasia among those having a finding, and specificity was the rate of patients without astasia without abasia among those not having a finding. Student's *t* test was used to compare COPs between trials with and without orthoses.

Figure 2 The COP values of 16 consecutive patients in the stilts group with and without ankle-foot orthoses. Each patient's COPs with and without ankle-foot orthoses were linked. The COPs decreased considerably in seven patients while wearing ankle-foot orthoses (Nos 3 to 9), decreased significantly in another seven (Nos 10 to 16), and were unmeasurable (patient took a step or was supported at least once per five trials) either with or without orthoses in two (Nos 1 and 2), although the patients claimed improvements. COP = average length of the centre of foot pressure (cm/30 seconds); ■ = without orthoses; □ = with orthoses; $P < 0.05$; Student's *t* test.

Results

STUDY 1

In the stilts group patients had peripheral neuropathies, spinal root lesions, or distal dominant myopathies, and astasia without abasia was mild in 15 patients and severe in eight. Differences in age were not significant either between the stilts (mean (SD) 45.9 (18.1) years,) and non-stilts groups (40.4 (12.3) years; $df = 68$, $t = 1.51$, $P = 0.136$) or among the three subgroups of the non-stilts group (NS-HSMN, 37.2 (13.6) years; NS-LSR, 39.7 (13.9) years; NS-C, 42.8 (10.4) years; $df = 46$, $F = 0.788$, $P = 0.461$). Sex ratios (male:female) did not differ significantly between the stilts (10:13) and non-stilts groups (28:19; $df = 1$, $\chi^2 = 1.612$, $P = 0.204$).

In the stilts group at least 18 patients (75%) showed weakness in the bilateral tibialis anterior and triceps surae muscles and diminution of muscle stretch reflexes of the bilateral ankles (table). Although 11 clinical findings were significantly different between the stilts and non-stilts groups (χ^2 test, $P < 0.05$), only one, with weakness of the bilateral triceps surae muscles, had both sensitivity and specificity that exceeded 0.95.

Five patients of the stilts group in whom sensory disturbances were not found during physical examination had mild astasia without abasia. Results of sural nerve conduction velocity and SEP testing were normal in all five patients, and cutaneous vibration and thermal cooling thresholds were normal in three.

STUDY 2

The bilateral below-knee amputee was able to stand in place while wearing prostheses and had a COP of 51.0 (4.3) cm/30 s. After the dorsiflexion bumpers were detached from the prostheses, he was unable to stand in place and took a step; therefore, the COP was unmeasurable.

STUDY 3

With ankle-foot orthoses, stability while standing was subjectively judged as much improved by 17 patients, and as improved by five patients. The COPs decreased greatly in seven of the 16 consecutive patients while wearing ankle-foot orthoses (Nos 3 to 9 in fig 2), decreased significantly in another seven patients (Nos 10 to 16), and were unmeasurable either with or without orthoses in two (Nos 1 and 2), although the patients claimed improvements. Astasia without abasia was severe in the two patients (Nos 1 and 2) who had severe sensory disturbances and was mild in three patients (Nos 11, 13, and 16) who had no sensory disturbances.

Discussion

Because astasia without abasia is encountered in various conditions, this study included patients with hereditary motor sensory neuropathy or spinal root lesions who did not show astasia without abasia and healthy volun-

teers. As there were no significant differences in age and sex ratio between the stilts and non-stilts groups, their clinical findings could be compared to investigate physical factors contributing to astasia without abasia.

Hirayama *et al*¹² reported that the cause of astasia without abasia was a combination of the loss of the ankle pattern, lack of compensatory movements of the hip, and impairment of somatosensory feedback from the legs. However, one patient with progressive spinal muscular atrophy and astasia without abasia had neither weakness of the hip extensor and flexor muscles nor sensory disturbance of the lower limbs.¹¹ Therefore, we hypothesise that the most important factor in the development of astasia without abasia is weakness of the triceps surae muscles although disturbance of compensatory postural movements and impairment of sensations are aggravating factors.

In this study the weakness of the bilateral triceps surae muscles was the only one of 28 physical findings that was both sensitive and specific for astasia without abasia. Moreover, as patients without sensory disturbances and without weakness of the iliopsoas and gluteus maximus muscles also had astasia without abasia, these findings also support our suggestion that neither impairment of somatosensory feedback nor disturbance of compensatory postural movements is essential for astasia without abasia.

Although we suggest that weakness of the triceps surae muscles causes astasia without abasia, proving that it does so regardless of compensatory postural movements of the hip and somatosensory feedback from the legs is difficult. The bilateral below-knee amputee is one of the best models for investigating the effect of the strength of the bilateral triceps surae muscles on stability while standing with normal compensatory postural movements of the hip and constant sensory input from the legs. Firstly, we selected an amputee who was thought to have no impairment of compensatory postural movements or somatosensory feedback. The amputee had normal muscular strength, sensations, and SEP results and did not have any disability of standing or walking while wearing prostheses. Secondly, to mimic weakness of the triceps surae muscles, the dorsiflexion bumpers were detached from the artificial feet. The bumpers continuously support the shafts of the prostheses and prevent excessive ankle dorsiflexion while standing. Because they function similarly to the triceps surae muscle, detaching the bumpers can mimic complete paralysis of the bilateral triceps surae muscles. As the interface between the socket and stump is unaltered before and after detaching the bumpers, transmission of tactile information from the prostheses to the stump is considered constant. Therefore, an inability to stand without the dorsiflexion bumpers indicates that the muscular strength of the bilateral triceps surae is essential to maintain a straight posture while standing. Results of this amputee study are consistent with Basmajian's belief that maintaining a standing position

requires continuous muscular activity of the triceps surae muscle.¹²

Lack of compensatory postural movements of the hip has been postulated as one of the three conditions contributing to astasia without abasia.¹² The postural movements supporting the erect stance are organised by using one or a combination of two basic patterns: those of the ankle or the hip.¹³ The dominant frequencies of postural sway in the patient's upright posture are about 1 Hz,² which is identical to the mechanically limited frequencies of the centre of gravity sway when pure hip pattern postural movements are used.¹⁴ Considering that 18 patients who had astasia without abasia did not show any weakness of the gluteus maximus muscles (table) and results of the amputee study, the 1 Hz oscillation in the upright posture may not suggest a disturbance of postural movements but compensation of a process by the hip pattern.

Although our study found that an impairment of sensation is not required for this disability to develop, balance control requires continuous regulation and integration of sensory inputs.¹⁵ Because patients in the stilts group with severe sensory disturbances were very unstable while standing and patients without any sensory disturbance were slightly unstable, impairment of sensation likely worsens astasia without abasia. As a patient who had sensory disturbances and no muscular weakness was not included in this study, it is unclear whether sensory disturbance without muscle weakness can cause astasia without abasia. However, we think that such a patient would be unstable while both walking and standing in place—that is, sensory ataxia.

The treatment for astasia without abasia consists of treatment of the underlying condition, if possible, and compensatory therapy for the weakness of the triceps surae muscles to maintain an upright posture. We suggest that ankle-foot orthoses should be prescribed for patients with this disability to prevent inappropriate ankle dorsiflexion while standing and orthoses are the best method to improve standing balance.

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Octave Landry's ascending paralysis and the Landry-Guillain-Barré-Strohl syndrome

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But it was not until 1916 that the next major contribution materialised, as the celebrated paper by Guillain, Barré, and Strohl describing the illness in two French soldiers.⁹

"The syndrome is characterised by motor disorders, abolition of the tendon reflexes with preservation of the cutaneous reflexes, paraesthesias with slight disturbance of objective sensation, pain on pressure of the muscle masses, marked modifications in the electrical reactions of the nerves and muscles, and remarkable hyperalbuminosis of the cerebrospinal fluid with absence of cytological reaction (albuminocytological dissociation).

This syndrome seemed to us to depend on a concomitant injury of the spinal roots, the nerves, and the muscles, probably of infectious or toxic nature."

The soldiers described both showed gross elevation of cerebrospinal fluid protein (2.5 g/l in the first case) without a cellular reaction. Strohl, whose name is often overlooked, performed the electrophysiological tests ("myographic curve"). Osler's 1892 classic *The principles and practice of medicine* recognised the illness, calling it acute febrile polyneuritis, and Bradford, Holmes, and others regarded it as acute infective polyneuritis.

In 1949 Haymaker and Kernohan commended the

eponym Landry-Guillain-Barré syndrome.¹⁰ But Guillain was angered by the inclusion of Landry's name,¹¹ arguing that Landry's acute bulbar form was a separate condition, and since lumbar puncture was not practised until 1891 (Wynter and Quincke) before Landry's time, that the inclusion of his name was "une confusion nosographique absolue".

Barré, a fine clinician, became professor of neurology in Strasbourg, concentrating his research on vestibular disorders. He died in 1967.

Landry, living for a time in straitened circumstances, had made no further contributions to the subject. His wife, Claire Giustigniani, he described as: "d'une grande beauté, d'une distinction suprême, mais beaucoup plus riche de noblesse que d'argent". A gentle and modest man, he directed a hydrotherapy clinic for nervous diseases at Auteuil, which eventually brought its own success and financial reward. A gifted exponent of the violoncello, he was also an accomplished singer and dancer. He cut an elegant and popular figure in artistic salons. But he was no lounge lizard, occupying his leisure as alpinist, geologist, horseman, and huntsman. Sadly, in his 40th year, Landry attended the penniless, destitute victims of a cholera epidemic in Paris, contracted the illness himself, and died a few days later.

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