

Conversive gait disorder: you cannot miss this diagnosis

Distúrbio conversivo da marcha: você não pode deixar de fazer esse diagnóstico

Péricles Maranhão-Filho^{1,2}, Carlos Eduardo da Rocha e Silva³, Maurice Borges Vincent¹

ABSTRACT

Bizarre, purposeless movements and inconsistent findings are typical of conversive gaits. The objective of the present paper is to review some phenomenological aspects of twenty-five consecutive conversive gait disorder patients. Some variants are typical – knees give way-and-recover presentation, monoparetic, tremulous, and slow motion – allowing clinical diagnosis with high precision.

Keywords: somatic symptom, conversive gait, neurological examination.

RESUMO

Movimentos bizarros, sem finalidade e inconsistentes são típicos das marchas conversivas. O objetivo deste artigo é descrever os aspectos fenomenológicos de vinte e cinco pacientes com distúrbio conversivo da marcha, salientando que algumas variantes são tão típicas – dobrando os joelhos e recuperando, monoparética, trêmula e em câmara lenta – que praticamente não possuem diagnóstico diferencial.

Palavras-chave: sintoma somático, marcha conversiva, exame neurológico.

The neurological examination as we know today, emerged by the end of the 19th century, when signs that would trustfully discriminate weakness due to structural damage from hysteria became crucial¹. Conversion disorder, which may affect 11-300/100,000 individuals, remains largely underdiagnosed, partially because its mechanisms are still unknown^{2,3}. Conversive gait disorders correspond to approximately 3% (0-7%) of the movements' disorders in specialized centres^{4,5}.

Expressions such as psychogenic, functional or medically unexplained symptoms have long been used to tentatively replace the term hysteria. For sake of clarity, we chose the term 'conversion disorder' as currently defined in Diagnostic and Statistical Manual of Mental Disorders (DSM-V)⁶ and the International Statistical Classification of Diseases and Related Health Problems 10th Revision (ICD-10)⁷.

According to the last version of the DSM-V⁶, the conversive disorders – paralysis, seizures, sensory loss, and gait disorders, now named Functional Neurological Symptom Disorder – belong to the “somatic symptom and related

disorders”, which replaced the previous so-called “somatoform disorders”⁹.

The cases presented herein suggest that objective landmarks do provide the neurologist with sturdy evidence for a trustworthy conversive gait diagnosis.

METHOD

In the course of the last 17 years, twenty-five consecutive conversive gait patients (72% women), age 44.4±17.5 (18-83) (mean ± SD range) with disease duration of 18.2±43.8 months (1 day-17 years) were examined at the Department of Neurology, *Hospital Clementino Fraga Filho, Universidade Federal do Rio de Janeiro* (n=12), *Instituto Nacional de Câncer do Rio de Janeiro* (n=8), and private practice (M-FP, n=5). The gait disorder was the chief complain in all individuals. All patients (except one) were examined by the same neurologist and present normal head and spinal cord imaging (MRI or CT). The clinical evaluation

¹Departamento de Neurologia, Hospital Clementino Fraga Filho, Universidade Federal do Rio de Janeiro, Rio de Janeiro RJ, Brazil;

²Departamento de Neurocirurgia, Instituto Nacional de Câncer do Rio de Janeiro, Rio de Janeiro RJ, Brazil;

³Departamento de Psicologia Médica, Hospital Clementino Fraga Filho, Universidade Federal do Rio de Janeiro, Rio de Janeiro RJ, Brazil.

Correspondence: Péricles Maranhão-Filho; Av. Prefeito Dulcídio Cardoso, 1680/1802; 22620-311 Rio de Janeiro RJ, Brasil; E-mail: pmaranhaofilho@gmail.com

Conflict of interest: There is no conflict of interest to declare.

Received 26 October 2013; Received in final form 17 January 2014; Accepted 06 February 2014.

Table. Conversive gait disorder (n=25).

n	Gender	Age (Years)	Phenotype	Duration
1	M	36	trembling, moon walking and camptocormia	5 y
2	F	36	trembling and dragging	1 w
3	F	26	monoparetic dragging	2 y
4	M	57	knee give way and recover	2 m
5	M	40	slow motion	2.5 m
6	M	38	trembling	1 d
7	F	40	knee give way and recover	3 m
8	F	78	trembling	3 w
9	F	39	monoparetica spastic	1.5 y
10	M	39	walking on ice	4 m
11	F	37	monoparetic spastic	8 y
12	F	25	knee give way and recover	2 m
13	F	43	knee give way and recover	1 y
14	F	52	knee give way and recover	17 y
15	F	83	slow motion	3 w
16	F	32	dystonic feet (scissor gait)	2 m
17	M	18	knee give way and recover and dystonic chorea	4 m
18	F	36	chorea	1.5 y
19	F	45	short steps, cephalic trembling and biting in the chest	3 w
20	F	30	knee give way and recover	2 w
21	F	73	ataxic	1 m
22	M	60	slow motion	2 w
23	F	46	monoparetic scarvant unilateral	7 m
24	F	26	knee give way and recover	2 w
25	F	76	shaking and hesitant	1 y

M: male; F: female; d: days; w: weeks; m: months; y: years.

included video recording for detailed movement analysis. The diagnostic criteria included both: a) a gait pattern incompatible with any known organic neurological or

systemic disease, and b) no proven disorder putatively related to the gait abnormality by means of appropriate subsidiary investigation.

RESULTS

The clinical data are summarized in the table. Four conversive gait types present in 23 (92%) patients. Successive knee give way movements presented as sudden buckling of the knees without falls (Figure 1) was the most common gait variant: 8 individuals (32%), followed by monoparesis in six (24%), subdivided in dragging (2), stiffness (3) (Figure 2), and foot drop (1). Tremulous gait occurred in five patients (20%), four show slow motion gait (16%); and one presented typical “moon walking” (Figure 3). Dystonic gait was evident in three patients: one walked with inverted feet (scissor feet); another flexed one leg while placing the opposite foot behind the knee (Figure 4); and a third walked with bizarre multiple contortion movements without falling. After one-year treatment patient could improve substantially her gait by sustaining a glass of water while walking (Figure 5). Ataxic and camptocormic (Figure 6) gaits were observed in one patient each. An old lady presented with a very bizarre walk characterized by short steps alternate, gorilla-like arms betting against the thorax and head bobbing. When stopped she would move all her body many times like a pendulum back and forth sway amplitudes in a crescendo-decrescendo way. Another woman moved quickly up and down while standing (vertical tremor). While sited she modified the movement pattern performing thighs adduction-abduction movements.



Figure 1. Knee give way and recovers. The patient was a 25 year-old woman presenting for 2 months gait characterized by suddenly flexing the knees in every step without falling. Images extracted from a movie.

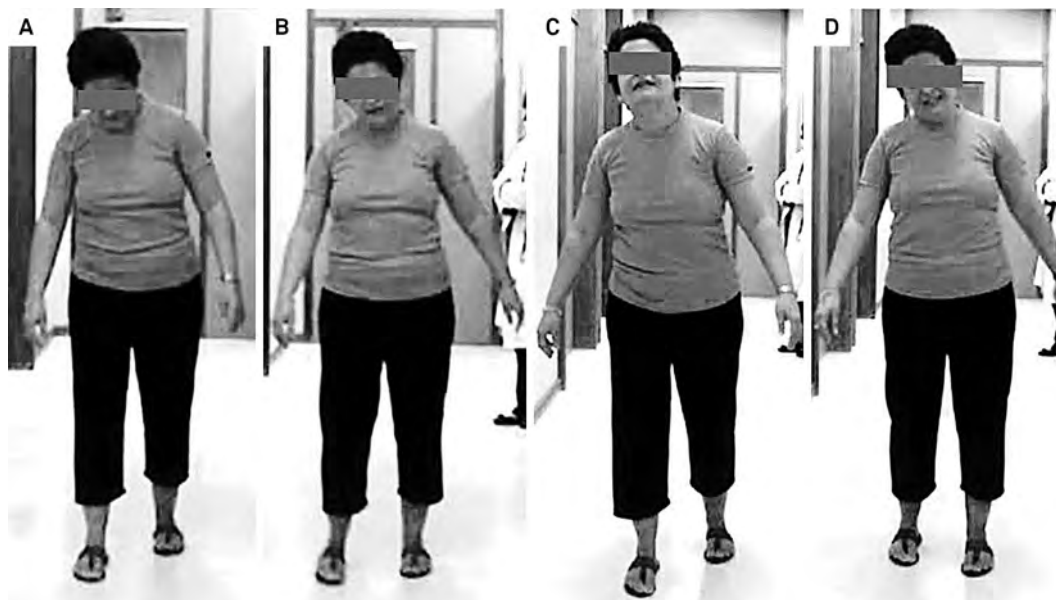


Figure 2. A 39 year-old woman walking with monoparetic stiffness at the left leg. Great effort, moans and grimacing at each stride are noticed. Images extracted from a movie.

Five patients had more than one distinctive gait pattern. No patient showed urinary incontinence, a symptom that strongly suggests an organic aetiology⁹.

DISCUSSION

Gait examination is the best single test of neurological function. Walking reflects motor, sensory, vestibular and

cerebellar capacity, including the attitude adopted towards the dysfunctions plus the strategies to offset movement ineffectiveness¹⁰. Conversive (psychogenic) motor symptoms are diagnosed based in congruity with any known neurologic ailment^{11,12}.

Two separate but interrelated skills are essential for a regular walk: balance and mobility¹³. For the first, straightening reflex, support reaction, anticipatory postural reflex, rescue and protective reaction are needed. For the second, gait ignition, shifting the centre of gravity, and modifying central and peripheral motor programs accordingly are required. In conversive gait disorder, a functional fail may occurs in any aspect of these skills.

The physician may evoke signs of conversive gait in few minutes by simply observing the spontaneous walk throughout a sufficient distance, in different velocities, while turning, walking backward, and by analyzing the pattern under tiptoe and heel walking. Not infrequently deficits expressed as walking bizarreness will emerge in only one of these situations, indicating that the abnormality is not compatible to any organic neurologic condition¹⁴.

Lempert et al.¹⁵ studied thirty-seven patients with psychogenic disorders of stance and gait and found six features that, alone or in combination, occur in 97% of patients with abnormal conversive stance and gait. Among conversive gait types they found predominantly excessive slowness, “walking on ice”, and sudden buckling. The most frequent associated features were momentary fluctuations, and uneconomic postures.

Among 228 consecutives functional patients, Keane¹⁰ studied 60 patients with functional gait presenting problem. Ataxic (24), hemiparetic (13), paraparetic (10), trembling (9),



Figure 3. A 36 year-old men present for five years walking with extremely slow movements (moon walking or slow motion). He modifies this pattern on request (see Figure 6). Images extracted from a movie.



Figure 4. A 18 year-old men who with a 4 months duration walking with knee give way-and-recover pattern and place his leg behind the support leg with each stride. Images extracted from a movie.

and knee give way-and-recover (5), were the most frequent in that study.

In our series four gait types clearly prevail, present in 23 patients (92%). Although there are several subtypes of conversive gait^{3,10,15} we consider that four variants – knee give-way-and recover, monoparetic, tremulous and slow motion gaits – are phenotypically typical enough to be considered as almost pathognomonic.

Although traditionally associated to conversive gait¹⁶, camp-tocormia was present in only one of our patients. Like in other conversive gait disorders series^{10,15} many of our patients

showed momentary fluctuation, excessive latency, grimace, groans, obvious disproportionate effort, and exaggerated swaying without falling as part of the gait phenotypes^{19,17}.

All twenty-five patients met “Clinically Defined” Fahn Williams criteria for psychogenic movement disorders²¹.

Treatment approach is beyond the scope of this article, but it is worth mentioning that no standard therapy for this problem is available. Treatment needs to be individualized and depend on the psychogenic factors involved⁹. Dramatic cure is the best diagnostic evidence of conversion^{9,10}. Offering a placebo medication or for instance applying a tuning fork to the affected body part often eliminates the symptoms¹⁸. However, the disorder is never a piece of cake. Anxiety and depression are the commonest psychological accompaniments of conversive gait disorders, sometimes requiring specific treatment. Some patients respond quickly to psychological management,



Figure 5. The patient, a 37 year-old woman, walks normally when sustaining a glass of water (A). After dropping it (B), sudden body distortion movements and dystonic walking immediately occur (C-G), ceasing only when she holds the glass again (H). Images extracted from a movie kindly provided by Doctor Adriana Fiszman.



Figure 6. When asked, the subject changes his slow motion gait pattern (see Figure 3) and trembling gait (not show), to a camptocormia. Images extracted from a movie.

but persistence for more than one year – like in this series – is frequent. Despite some psychiatric authorities consider a true heresy to openly declare to the patient that their somatic symptom has a psychosocial origin¹⁹, others consider more recently that clarifying the conversive nature contributes to a better resolution of their condition¹².

Gait is a complex process that involves motor centres at the spine, brain stem, cerebellum, motor cortex and their connections. These structures rely on visual, vestibular and somatosensory input to implement effective movements²⁰. When any of these links fail, a particular gait phenotype emerges, each of them typically related to dysfunction in a specific constituent of the gait controlling system.

Conversive gaits do not fit with these provisos. They look particularly bizarre, frequently too complex to be true, apparently unstable but surprisingly leading to no fall, or expressing with too dramatic gestures. These features actually prove the integrity of the motor systems rather than indicate a lesion.

Acknowledgments

We are indebted to Pericles Maranhão Neto for technical assistance and to Doctor Henrik Maultasch for kindly providing us with one case.

References

1. Okum MS, Koehler PJ. Babinski's clinical differentiation of organic paralysis from hysterical paralysis. *Arch Neurol* 2004;61:778-783.
2. Nicholson TRJ, Stone J, Kanaan RAA. Conversion disorder: a problematic diagnosis. *J Neurol Neurosurg Psychiatry* 2011;82:1267-1273.
3. LaFrance W C Jr. Somatoform disorders CDROM AAN 2012 Course 8AC-006.
4. Vuilleumier P, Chicherio C, Assal F, et al. Functional neuroanatomical correlates of hysterical sensorimotor loss. *Brain* 2001;124:1077-1090.
5. Lang AE. Phenomenology of psychogenic movement disorders. In Hallett M, et al (Eds). *Psychogenic Movement Disorders*. UK, Cambridge University Press, 2011.
6. American Psychiatric Association (APA). *Diagnostic and statistical manual of mental disorders, 5th Edition (DSM-V)*. Washington, DC, American Associated Press, 2013.
7. World Health Organization. *ICD-10 Classification of mental and behavioural disorders*. Geneva, 1992.
8. Task Force on DSM-IV TR. *Diagnostic and statistical manual of mental disorders: DSM-IV-TR, 4th, text revision ed*. Washington, DC: American Psychiatric Association; 2000.
9. Sudarsky L. Psychogenic gait disorders. *Semi Neurol* 2006;26:351-356.
10. Keane JR. Hysterical gait disorders: 60 cases. *Neurology* 1989;39:586-589.
11. Maria R. Explaining the unexplained: understanding hysteria. *Brain* 2001;124:1065-1066.
12. Stone J, Edwards M. Trick or treat? Showing patients with functional (psychogenic) motor symptoms their physical signs. *Neurology* 2012;79:282-284.
13. Nutt JG, Marsden CD, Thompson PD. Human walking and higher-level gait disorders, particularly in the elderly. *Neurology* 1993;43:268-279.
14. Monrad-Krohn GH. *Exploración clínica del sistema nervioso*. 3^a Edición. Ed.Labor Ltda., Barcelona, 1967:263.
15. Lempert T, Brandt T, Dieterich M, Huppert D. How to identify psychogenic disorders of stance and gait. A video study in 37 patients. *J Neurol* 1991;238:140-146.
16. Azher SN, Jankovic J. Camptocormia Pathogenesis, classification, and response to therapy. *Neurology* 2005;65:355-359.
17. Fahn S, Jankovic J. *Principles and practice of movement disorders*. Elsevier. Philadelphia, 2007.
18. Shamy MCE. Treating psychogenic movement disorders with suggestion. In: Hallett M, Lang AE, Jankovic J, et al, (Eds). *Psychogenic movement disorders and other conversion disorders*; New York, Cambridge University Press. 2011:295-301.
19. Guggenheim FG. Somatoform Disorders. In: Sadock B, Sadock V, (Eds). *Kaplan & Sadock's Comprehensive Textbook of Psychiatry*. 7th ed. Philadelphia: Lippincott Williams & Wilkins, vol. 1, 2000:153-154.
20. Dickinson MH, Farlehman CT, Full RJ, et al. How animals move: an integrative view. *Science* 2000;5463:100-106.
21. Fahn S, Williams PJ. Psychogenic dystonia. *Adv Neurol* 1988;50:431-455.