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From trepidant abasia to motor network failure—gait disorders as a consequence of subcortical vascular encephalopathy (SVE) Review of historical and contemporary concepts

Hansjoerg Baezner*, Michael Hennerici

Department of Neurology, Universitätsklinikum Mannheim, University of Heidelberg, 68135 Mannheim, Germany

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Abstract

Gait disorders in progressive subcortical vascular encephalopathy (SVE) and their impact on the burden of disability in the growing elderly population are underrepresented in medical scientific literature. The absence of a clear framework for the diagnosis and classification for gait disorders on the basis of SVE has multiple reasons: (1) neither movement disorder specialists nor stroke specialists are truly familiar with this topic and feel responsible for its treatment, (2) the existing terminology lacks a clear concept and a consistent classification, and (3) only in recent years have large prospective trials started to address the natural course of SVE.

This article reviews the classical descriptions of gait disturbances with preferential view to our present concept of SVE, and comments on historical and current nosology of gait disorders aiming to propose for a new classification.

Keywords: Subcortical vascular encephalopathy; Gait; Gait disorders; White matter; Classification; Locomotion; Postural control

1. Introduction

Gait problems and falls are a major cause of morbidity and mortality in the elderly. An appreciable percentage of falls result in serious injuries such as hip fractures or head trauma. However, limited attention has been focused on early identification of fallers and prevention strategies for falls or injuries, although some retrospective studies discussed aspects such as musculoskeletal degenerative processes, obstructive vascular lesions causing cerebral hypoperfusive states, or autonomic failure with orthostatic hypotension in the elderly. In addition, interest was placed on processes leading to degeneration of the complex motor network that guarantees the integrity of central coordination of stance and gait [1]. However, only few studies focus on this topic and provide prospective data [2,3]. Recently, in an important study, Verghese et al. [4] showed that gait abnormalities may predict non-Alzheimer's dementia, i.e., mainly vascular dementia. Hence, recognition and early diagnosis of this devastating condition may be feasible by an early detection of gait abnormalities and offer preventive medical treatment. Unfortunately, diagnosis of gait and postural problems is incapacitated by the lack of a clear framework for classification [5]. This is amazing because extensively detailed descriptions have existed for more than 100 years. The terminology, however, is quite confusing due to the existence of more than a dozen terms for the same type of gait disturbance. Recent efforts to classify gait disturbances yielded rather unsuitable solutions for this important problem because they consist of a mixture of presumed anatomical location and of clinical phenomenology [6] or use the much debated notion of apraxia [7].

2. Disturbances of locomotion

2.1. Marche à petits pas (short-stepped gait)

The first descriptions of gait disorders associated with SVE date back more than 100 years. In 1895, Brissaud [8]

* Corresponding author. Tel.: +49 621 383 2885; fax: +49 621 3833807.

E-mail address: baezner@neuro.ma.uni-heidelberg.de (H. Baezner).

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presented a characteristic clinical variety of senile softening (old term meaning encephalomalacia) with short-stepping, hesitating, cautious gait and slow and uncertain movements in subjects with numerous small cerebral lesions of thrombotic origin. Pierre Marie and his pupils recognised the clinical significance of lacunar infarctions in excellent clinical and pathoanatomical studies [9,10]. Obviously, the clinical picture was common clinical practice in the "hospice de bicêtre" (Bicêtre Hospital), leading to the term "lacunaires" ("lacunar" patient) for the patients whose gait was typically a "marche à petits pas". Marie describes this gait as follows: "the patient advances with shuffling steps, not spastic but rather flaccid; each leg is moved forward not more than 10–15 centimetres during each step, the lower limbs are slightly flexed in their various joints, the trunk is slightly bent forward..." Interpreting this syndrome as a consequence of multiple lacunar infarcts, the "hémiplégie des vieillards" (hemiplegia of the elderly) was often accompanied by a pseudo-bulbar palsy with dysarthria, dysphagia, pathological crying, laughing, and dementia. The excellent work by the Italian neurologist Catola published in 1904 [11], also a pupil of Pierre Marie contains the detailed clinical descriptions of disturbances of stance and gait. According to Catola, the patient, when asked to start walking, either stays in place, rigid, unable to make regular steps, or, if able to walk, makes short steps, the body slightly bent forward, shuffling, the joints bent. Catola's study is based on multiple brain sections from the Bicêtre hospital, and he underlines the importance of lacunar infarctions in the thalami for the type of gait disturbance. Other protagonists of the French school coined terms like meïopragie cérébrale (cerebral "meiopragia") [12], which included aspects of subcortical dementia. Souques' excellent clinical study on parkinsonism [13] contains a classification of parkinsonian syndromes; two of the five categories potentially come within the scope of this paper: parkinsonian syndromes due to presenile lesions of the corpus striatum, and parkinsonian syndromes due to macroscopic focal lesions (e.g., lacunes). Some of Lhermitte's cases in "syndromes striées du vieillard (1922)" (striatal syndrome of the elderly) [14] and "paraplégies des vieillards (1907)" (paraplegia of the elderly) [15] belong to the category of patients with parkinsonian gait on the basis of subcortical vascular encephalopathy (SVE).

2.2. Arteriosclerosis and arteriosclerotic rigidity

Collins [16] refers to the syndrome he describes in a detailed case study (1906) illustrated by several photographs showing elderly patients with broad based unsteady and short-stepped gait as "a definite clinical variety of cerebral arteriosclerosis" which in his series of 135 patients occurred 13 times.

Early German literature including Binswanger's and Alzheimer's contributions only incidentally mention gait disturbances as an important clinical feature of a disease they call arteriosclerotic brain atrophy, arteriosclerotic brain degeneration or "encephalitis subcorticalis chronica progressiva": While Alzheimer enumerates symptoms like leg weakness, increased deep tendon reflexes as early features [17–20], Binswanger, in his seminal paper in 1894, describes a patient with a "somewhat staggering gait" [21]. Marsden and Thompson [22] rediscussed Binswanger's work in 1987 and referred to his 1894 publication, when describing the gait disorder of subcortical arteriosclerotic encephalopathy which, in their article is used synonymously to Binswanger's disease. Their patients presented with difficult walking, representing elements mixed of both Parkinsonism and ataxia. As in the historical cases with "trepidant abasia" (see below), the difficulty in using their legs to walk was out of proportion to that of other movements of the lower limbs when lying or seated. Furthermore, upper limb mobility and facial expression were markedly preserved. As a possible mechanism for this gait disorder, the authors suggest damage to the afferent and efferent interconnections of the leg areas of the motor and supplementary motor areas of the cerebral cortex with the cerebellum and basal ganglia. Almost 100 years before, in a textbook edited by Binswanger in 1904, Wollenberg describes the gait of a patient with arteriosclerotic disease as unsteady and tripping [23]. Buchholz [24], in a paper published in 1905, gives a series of case reports with several excellent clinical descriptions and pathological studies which include multiple mentions of pathological gait (case 1: slow and broad-based gait, spastic-paretic, very unsteady, complaints of dizziness, later in the course unable to walk without assistance; case 2: staggering and shuffling gait, complaints of dizziness; case 5: stance very unsteady; case 6: unsteady and staggering gait. Foerster coined the term of arteriosclerotic rigidity in 1909 [25] when he described several patients with increased muscle tone which he interpreted as due to cerebrovascular disease. In a later paper, Nissl [26] reviews a case of arteriosclerotic dementia with the patients "walking with small stiff steps ", later "unable to walk". In the 1909 edition of Kraepelin's textbook [27], the gait of patients with arteriosclerotic madness is characterised as unsteady and slow.

2.3. Arteriosclerotic parkinsonism, lower-body parkinsonism, and vascular (pseudo)parkinsonism

Elements of parkinsonism lead to terms like arteriosclerotic parkinsonism [28], arteriosclerotic pseudo-parkinsonism [29], vascular pseudoparkinsonism [30], lower body parkinsonism [31], and lower half parkinsonism [32]. Critchley's excellent 1929 paper contains several most remarkable clinical descriptions of gait disturbances in SVE. The French school described the parkinsonian form of pseudo-bulbar palsy and the parkinsonian syndrome of the "lacunaires" ("lacunar" patients), both representing clinical varieties of disorders on the basis of