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History of Neurology

From hysteria to gait dementia: History of the concept of astasia-abasia

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ABSTRACT

Paul Blocq (1860–1896) and his teacher Jean-Martin Charcot (1825–1893) introduced the expression "astasia-abasia" into medical terminology in 1888 to designate a pathology they believed to be caused by hysteria. This condition makes it impossible to remain erect and to walk, whereas the ability to move the legs while lying down remains normal. At the turn of the 20th century, and now almost exclusively, this motor disturbance is recognised as a syndrome with multiple possible organic causes, and now described as "higher-level gait disorder". After briefly mentioning earlier descriptions by other authors, I will review Charcot's Tuesday lessons in 1889 that covered astasia-abasia and elucidated the beginnings of the breakdown into organic aetiologies: medial-frontal and corpus callosum tumors, damage to the cerebellar vermis, lacunar state as described by Pierre Marie (1853–1940), Parkinson's disease, and Parkinson-plus syndrome. The long history of astasia-abasia reveals a cluster of neurologists, often emerging from oblivion herein and all of whom, through the precision of their clinical examinations and their pathophysiological findings, helped advance the understanding of the mechanisms by which human beings are the only erect, constantly bipedal mammals, whether immobile or walking.

1. Introduction

For the physician, and especially the neurologist, watching a patient's gait as they enter the consulting room still offers a wealth of fundamental semiological information. Longer life spans have amplified the socio-economic and medical problems of the "elderly who fall". After rheumatic-orthopaedic causes, poor nutrition, sarcopenia, and visual deficits have been eliminated, it is time to seek a neurological aetiology.

Controlling gait and balance involves the primary motor cortex; the motor, premotor, and supplementary areas; and the basal ganglia, notably the mesencephalic locomotor region, including the pedunculopontine and cuneiform nuclei. This is the functional region that initiates and modulates autonomic locomotion. In addition to this motor control, the vestibular system and the cerebellum play a role in controlling posture in dynamic equilibrium; as John Russell Napier (1917–1987) put it: "Man's bipedal mode of walking seems potentially catastrophic because only the rhythmic forward movement of first one leg and then the other keeps him from falling flat on his face" [1].

The study of gait and walking goes back to antiquity and Hippocrates (460–356 BC) [2]. Countless authors over the centuries have sought to elucidate the causes of falling. For example, in 1733, Philippe Hecquet (1661–1737) suggested that

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''when there is a lack in the quantity of animal spirits, their power of movement [that of the muscles] is idle'' [3].

It was not until the 19th century that the concept of astasia-abasia was formally and initially recognised as a deficiency of hysterical origin. But how did the pathology of astasia-abasia, considered functional and affecting children, become an organic gerontological pathology?

2. Paul Richer and Jean-Martin Charcot

In 1884, Jean-Martin Charcot (1825–1893) chose the first edition of a new Italian review, *La Medina contemporanea*, to publish an article, as requested by his friend Mariano Semmola (1831–1896), who had founded the publication. The article, written with his former resident who had become the head of his laboratory, Paul Richer (1849–1933), described ''a defect of coordination while standing and walking'' [4]; that is, ''a particular form of motor impotence in the lower limbs due to a lack of coordination'' (''*di una forma speciale d'impotenza motrice degli arti inferiori per diffetto di coordinazione*''). Domenico Miliotti (1857–?), a young Italian who spent two years at La Salpêtrière hospital, returned to the described cases in 1885, recording a lesson presented by Charcot on 7 March 1884, but not included in his complete works [5,6]. Charcot then referred to this subject in two Tuesday lessons presented successively on 5 and 12 March 1889 [7]. He used the term ''abasia'' to refer to ''motor incoordination or motor impotence relative to walking'' and paired it with ''astasia'', or the inability to remain standing.

In reality, it was Paul Blocq (1860–1896), a brilliant resident who worked under Charcot in 1887 but died very young, who introduced the expression ''astasia-abasia'' into medical terminology, based on advice from the linguist Jules Augustin Girard (1825–1902). Blocq defined this condition as follows: ''This term designates a morbid state in which the impossibility of standing upright and walking normally contrasts with the integrity of sensation, muscular force, and the coordination of other movements of the lower limbs'' [8]. He coined this neologism, still in use today, based on the Greek *astasia* or *astatos*, meaning ''instability'', and *basis*, meaning ''the action of walking''. The definition given by Raymond Garcin (1897–1971) in 1970 remains close to Charcot's definition: ''It entails the inability to remain upright and to walk despite muscular force and coordination of the limbs remaining entirely normal'' [9].

Richer had already presented a study of this supposedly hysterical disorder in a text originally written as his submission for the residents' gold metal competition in 1879 (Charcot then dissuaded him from entering the academic competition). This was in addition to his famous thesis defended on 9 April 1879: *Étude descriptive de la grande attaque hystérique ou attaque hystéro-épileptique et de ses principales variétés* (Descriptive study of *grande attaque hystérique*, or hystero-epileptic attack, and its main varieties) [10]. He submitted it in 1883 for the Bernard de Civrieux Prize of the French Academy of Medicine, which named him the winner on 19 May 1884. The commercial edition was published in 1892, which notes, ''This sort of paralysis is often found in childhood, among boys and girls, and can be considered a form of infantile hysteria'' [11]

Fig. 2. — Astasie. — Abasie (d'après un croquis de M. Charcot).

Fig. 1 – Drawing by Paul Richer [11] (OW Collection).

occurring after an emotional shock. ''The organic memory of walking is lost.'' He noted that abasia can exist alone, without astasia: ''The condition is cured suddenly due to a moral impression or with no appreciable cause''. He did not fail to conclude his description with a fine drawing which he himself had produced (Fig. 1). Richer pointed out that Charcot distinguished between paralytic or paretic astasia-abasia, and ataxic astasia-abasia with movement incoordination, the latter form being further divided into ''choreic'' and ''trembling'' forms.

Richer continued his studies of gait throughout his medical and artistic career [12], helping Georges Gilles de la Tourette (1857–1904) illustrate his thesis [13]. As for the study of gait kinematics, it was initiated in 1883 by Étienne-Jules Marey (1830–1904). Around the same time, the Americans Francis Xavier Dercum (1856–1931) [14] and Eadweard Muybridge (1830–1904) [15] filmed normal and pathological gaits, from 1883 to 1901.

3. Before Paul Blocq

Sigismond Jaccoud (1830–1913) is classically considered to have first described in 1864 astasia-abasia in his book *Les*

Table 1 – Before Paul Blocq, descriptions evoking astasia-abasia

Before Paul Blocq	Descriptions evoking astasia-abasia
Ernest Mesnet (1825–1898) Thesis 1852	Hysterical paraplegia: ''Sometimes they have to remain in bed because their legs buckle when they stand'', whereas these women ''freely use their lower limbs'' in their beds [17]
Jean-Baptiste Séraphin Barnier (1827–1886) *Agrégation* thesis, 1857	''Paralysis without appreciable organic lesions''; ''it is not rare to run across patients who can no longer walk, even though they can move their legs as asked if lying down. This proves that the nervous influx is diminished rather than abolished'' (p. 24) [18]
Edmé Cothenet (1829–1878) Thesis, 1858	Diagnosing paraplegia: ''We often see patients who cannot walk, but who can effectively move their legs in the horizontal position as asked'' [19]
Paul-Alfred Lebreton (1837–1882) Thesis, 1868	Hysterical paralysis: ''Paraplegia is rarely complete. More often, a weakening of the lower limbs is observed, which ranges from a simple disturbance in the upright position and while walking, to a near impossibility to execute movement. Often the patient can move their lower limbs when recumbent. This could be taken as laziness or simulation, but if the patient is asked to take a few steps, at first the progression is slow yet all right, but their legs soon weaken, buckling under their body's weight. It would seem, then, that at some point, the patient's nervous influx is exhausted'' (p. 100) [20]
Guillaume Duchenne de Boulogne (1806–1875) 1872	''Localised electrification'': Observation CXXXVIII, p. 716 [21], observation of hysterical paraplegia evoking astasia
Silas Weir-Mitchell (1829–1914) 1885	''While in bed, Miss B. moves all her limbs somewhat slowly, but with a great deal of power; the lift of the leg was done in jerks, as by distinct orders of will, but she showed none of the tremor and twitching of face and the tearful look so common in hysteric girls called on for an usual effort. When held up on her knees, she swayed to and fro, always falling if not assisted'' [22] (p. 44)
Adolf–1877) 1885	A description evoking trembling abasia accompanied by cramps [23] (p. 308)
Eduard Heinrich Henoch (1820–1900) 1885	Paediatrics treatise: In the chapter on infantile hysteria, a case of astasia-abasia is described [24]
Serafino Romei 1885	*Paraplegia infantile nel solo atto della ambulazione*, (Infantile paraplegia occurring only during the act of walking), an observation like those of Charcot and Richer [25]
José-Dantas de Souza Leite (1859–1925) 1888	Two observations which he likened to those Blocq put forward in Paris [26], but his text is not entirely convincing as to whether these cases were the same pathology

paraplégies et ataxie du mouvement (Paraplegia and movement ataxia) [16]. He referred to the condition as ''functional ataxia'' or ''ataxia due to deficient automatic coordination'' and described it as follows: ''Movements are normal when executed in a lying or sitting position. They only become ataxic in the standing position and during walking, at which time involuntary contractions interfere with balance or disrupt the harmony of the functional act, every time the sole of the foot touches the ground; that is, when the centripetal forces resulting from contact engage the morbid hyperkinesia of the spinal cord''. Jaccoud used the term ''akinesis'' for gait ataxia, which did not remain in common usage. The following Table 1 summarises a number of similar previous examples described in France and abroad.

4. Blocq's astasia-abasia

In 1888, Blocq collected eleven observations including one from an article by Silas Weir-Mitchell (1829–1914) and another from an article by Adolf-Albrecht Erlenmeyer (1822–1877), not to mention Charcot's observations that Miliotti had recorded. ''The disorder only affects actions associated with the upright position and walking which are hindered to various degrees... When the patient sits or lies down, nothing appears to be abnormal... It is only when the patient is asked to stand and walk that the anomalies become apparent... However, in striking contrast, the same patient in genupectoral position can walk 'on all fours' without any difficulty... Standing upright, he produces abrupt flexion of the knees followed by very rapid extension; each of these flexions of the limbs corresponds to a flexion of the trunk at the hips followed immediately by extension of the body''. Blocq added that these movements were irregular and unrhythmical and that blocking eyesight could worsen the disturbance. Reflexes were normal. ''Other manners of moving forward, aside from normal walking, could persist, such as jumping, crawling, hopping on one foot, and walking on all fours.'' This deficiency could last three to fifteen months, followed by a nearly instantaneous recovery with resumption of normal gait. This explains why he thought only hysteria could be the cause, seeing nothing comparable in tabes or Sydenham's chorea.

Blocq attempted to put forward a pathophysiological explanation. He first compared astasia-abasia with pure agraphia; that is, normal use of the hand except for the act of writing. In a long discussion, he highlighted that ''standing and walking are learned over a long period. In this mechanism, groups of cortical and spinal cells come into play, but initially cortical groups predominate. All forces of attention and will are employed to establish, organise, and regulate this mechanism. But as the subject becomes increasingly practiced, these phenomena become more and more automatic and unconscious. The cortical role gradually falls away, and nearly everything takes place in spinal centres... Impulsion is always the initial phenomenon, but the mechanism organised for standing and walking can operate alone without the participation of the cortex, as it is regulated in advance of the moment it is put into action... It can be said that while the

cortical groups hold the memory of the type of impulsion needed to accomplish the mechanism of rising, descending, and walking (psychological memory), the spinal centres are in charge of so-called automatic, unconscious execution and thus hold the memory of the reactions necessary for responding to these various disorders of encephalic origin (organic memory).'' For Blocq, during astasia-abasia, either the initial impulsion does not take place, ''or the order given is not executed''. For him, hypnotism enabled experimental verification of this pathophysiology through an observation of a ''suggestion of motor impotence'', to use Charcot's terms. Fear, strong emotion, and trauma were among the triggering causes along with the ''phenomena of unconscious cerebration and ideation''.

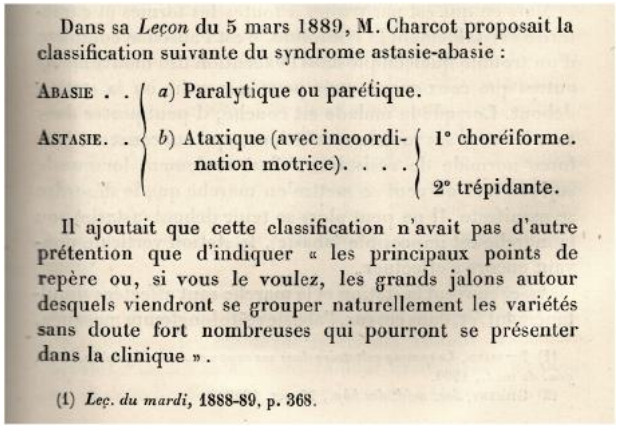
We should note that Blocq, in his observation III, described a young boy who recovered after a ''transfer session'' conducted by Joseph Babiński (1857–1932). Babiński would later remove this unscientific publication from his list of works; it dated from his time as senior resident under Charcot, from 1886 to 1887 [27].

5. Charcot's Tuesday lessons in March 1889

In his lesson on 05 March 1889, Charcot presented the case of a 41-year-old man whose ''symptom complex developed following asphyxia induced by coal steam and very probably as the result of this intoxication''. In bed and when standing, the patient perfectly executed all movements, and his reflexes were normal. Closing his eyes had no impact. The standing position was normal. But as soon as he walked ''with ordinary gait, except with a hurried step, his body bent forward, the lower limbs were stiff, in extension, so to speak, and could be described as stuck together. He moved forward on tiptoe, his feet seeming to glide against the ground. Progression involved a sort of a rapid tremor, like some cases of spasmodic paraplegia''. Charcot described his gait as trembling. Only walking was abnormal; the patient could jump on one or both feet and walk on all fours. Charcot referred to the writings of Jaccoud, Weir-Mitchell, Erlenmeyer, and Serafino Roméi.

He noted that abasia could exist alone whereas he had never encountered astasia by itself. He distinguished between various types: paralytic and paretic astasia-abasia (the patient falls down as soon as he is set upright), choreic abasia (abrupt, exaggerated bending movements of the lower limbs), and trembling abasia, where ''walking is hampered by contradictory movements of execution which stiffen the lower limbs and consist in a sort of walking in place''.

Charcot summarised the pathophysiological hypothesis exactly as Blocq had, indicating that, in fact, he had provided it to Blocq. For pedagogical reasons, he added a comparison between how the nervous system works and how a barrel organ works, which must have been more comprehensible for his audience in those days than for today's reader. Since Charcot's efforts to discover a cerebral lesion that caused hysteria had failed, he suggested at that time a ''dynamic lesion'' of psychic origin: ''In any case, it is hardly doubtful that in most circumstances, abasia results from a purely dynamic lesion... The various cellular groups that govern specific gait movements — walking, standing upright, hopping, etc. — make up several distinct centres in the central nervous system, and one can assume that each of these groups can be more or less seriously affected by an organic lesion'' (Fig. 2).



Dans sa *Leçon* du 5 mars 1889, M. Charcot proposait la classification suivante du syndrome astasie-abasie :

Abasie . . { *a*) Paralytique ou parétique.

Astasie. . { *b*) Ataxique (avec incoordination motrice). . . . { 1° choréiforme. 2° trépidante.

Il ajoutait que cette classification n'avait pas d'autre prétention que d'indiquer « les principaux points de repère ou, si vous le voulez, les grands jalons autour desquels viendront se grouper naturellement les variétés sans doute fort nombreuses qui pourront se présenter dans la clinique ».

(1) *Leç. du mardi*, 1888-89, p. 368.

Fig. 2 – G. Gilles de la Tourette, traité clinique et thérapeutique de l'hystérie, hystérie paroxystique, volume II, p. 139 (OW Collection).

To return to the case Charcot presented to his students, he suggested that the carbon monoxide poisoning, leading to a coma lasting three days, caused amnesia, initially global then partial. Given the patient's medical history, he settled on a diagnosis of trembling astasia-abasia of hysterical origin, with ''self-suggestion'' secondary to asphyxia and not an organic cerebral lesion from the intoxication, based on the thesis defended in 1843 by Hippolyte Bourdon (1814–1892), the first author to focus on paralysis following asphyxia [28]. During the following lesson, on Tuesday 12 March 1889, Charcot was happy to show his students the rapid recovery of nearly normal gait in this patient, which he saw as confirming the reality of the hysteria and the effectiveness of the persuasion used: ''You know that everything we said was done to persuade him that he would recover, and that he would recover rapidly''.

Charcot did not fail to point out that Joseph Grasset (1849–1918) in Montpellier had recently published a case of trembling astasia-abasia in a hysterical man that was in keeping with his lessons [29]. However, Grasset noted that non-hysterical cases of astasia-abasia existed. The following year, Albert Mathieu (1855–1917), a student of Maurice Debove (1845–1920), made a detailed review of Grasset's observation to train French physicians to recognise this new clinical picture [30]. Paul Berbez (1859–1928), Charcot's resident in 1886, aimed to disseminate this knowledge among family physicians in a summary published in 1888 in the *Gazette Hebdomadaire de Médecine et de Chirurgie* [31].

It is surprising that Charcot never referred to the work of his colleague Jules Luys (1828–1897) on cerebellar anatomy and physiology published in 1864. Luys discussed patients who ''had to continuously remain recumbent, [who] initially presented a series of disturbances in locomotive functions, which became increasingly incoherent and irregular'' [32]. Luys referred to asthenia as opposed to paralysis. In this long article, Charcot could have found a possible explanation for a non-hysterical form of astasia-abasia, the cerebellar form.

6. Later French and international publications

The non-resident Georges Cahen (1862–?), a childhood friend of Blocq, defended his thesis on astasia-abasia — *Contribution à l'étude de l'astasie-abasie* (Contribution to the study of astasia-abasia) — on 18 December 1890, before a jury presided by Charcot. Cahen grouped together thirty-seven observations, both personal and those found in the literature, to show a prognosis that was always favourable in the long term. His clinical descriptions were based on those of Charcot and Blocq, with no new elements. For Cahen, ''hysteria is a main cause of the abasia-astasia syndrome''. He drew on the work of the Italian Angelo Mosso (1846–1910) dealing with fear, describing this emotion as the cause of astasia-abasia [33]: ''A terrifying sensation together with an inability to walk'' was the cause of ''amnesia affecting the memory of how to walk''. This reference led him to devise a treatment based on the method developed by Jules Séglas (1856–1939) and Paul Sollier (1861–1933), when Sollier was a resident. This method ''consisted in awakening old amnestic images, or forming new ones, by amplifying their intensity through various processes (concomitant emotions), such as the focusing of attention and image association''. Sollier presented the clinical case, astasia-abasia during ''puerperal delirium'', at the Mental Medicine Conference in Rouen in August 1890 [34]. This demonstrates that Cahen was more innovative than his teacher Charcot. Sollier did not further develop his technique, the forerunner of cognitive-behavioural therapies, until after 1900 [35].

After attending Charcot's lesson, Paul Ladame (1842–1919) of Geneva submitted the description of a case of ''abasia in the form of attacks'' to *Les Archives de Neurologie* on 02 August 1889. The 54-year-old man, who had travelled extensively, including in Amazonia, was subject on several occasions to a sudden inability to walk lasting several hours, followed by a return to normal function. Ladame recognised that he showed no signs of hysteria but seemed to have attacks of anxiety. His neurological examination was normal. Ladame thus suggested adding a form simply arising from anxiety to the nosology of hysterical astasia-abasia [36].

In 1890, Charles Féré (1852–1907), former resident and secretary under Charcot, reported an observation of an epileptic in his book on epilepsy [37]. A 39-year-old man presented typical astasia-abasia after his generalised epileptic seizures. Unfortunately, no additional information was provided, notably concerning the progression. The same year, Otto Binswanger (1852–1929) of Jena contested the pathophysiological theory proposed by Blocq and Charcot. For Binswanger, astasia-abasia was a phenomenon like hypochondria, where thinking was exclusively given over to the motor deficiency [38]. Paul Julius Möbius (1853–1907) shared Binswanger's view of an obsessional, differentiated state, involving the fear of falling in a patient suffering from obsession, as opposed to the inability to remain standing, or the desire not to, in a hysteric [39]. Pierre Boulloche (1864–1923) and Maurice Debove (1845–1920) returned to this concept in 1894 in a presentation to the Medical Society of the Paris hospitals. They distinguished basophobia, a term they added to the medical nomenclature, denoting the idea of an inability to walk; that is, obsessional abasia [40]. Another of Debove's students, Paul Delarue, made this the subject of his thesis in 1901, on ''stasobasophobia'', in which the fear was an anticipatory fear of falling rather than of walking [41].

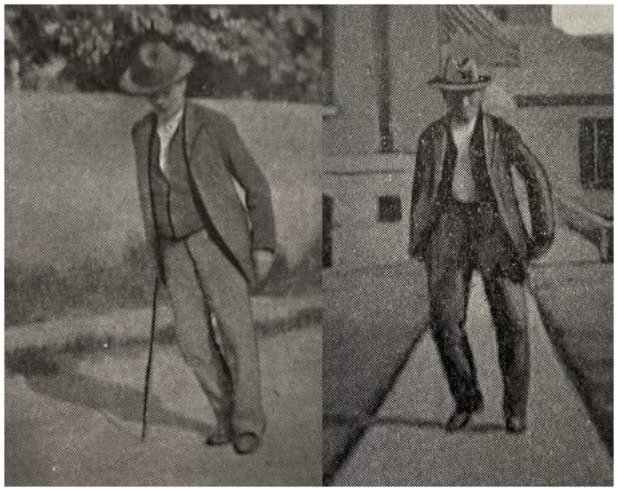


Fig. 3 – Patient of P.C. Knapp [45] (BIU santé, université Paris Cité).

In 1891, Albert Pitres (1848–1928) focused one of his lessons covering hysteria on hysterical paraplegia and reported three observations of adolescents with astasia-abasia, strictly keeping to the path laid out by his teacher Charcot, providing no personal input [42].

Also in 1891, *Les Archives de Neurologie* published a translation into French of a paper presented at the International Medical Conference in Berlin by the Dutch Eduard Hendrik Marie Thyssen (1856–1932), trained at La Salpêtrière [43]. He described the case of an 11-year-old girl with epilepsy, whose astasia-abasia temporarily abated after each generalised seizure [44], along with three other observations that provided no new information.

After his presentation at the 17th Conference of the American Neurological Association in Washington on 22 September 1891, Philip Coombs Knapp (1858–1920) published the first observation of ''trembling abasia'' in a patient with ''paralysis agitans'' [45]. He translated all Blocq's observations and noted that the symptomatology of his 58-year-old patient, essentially abasia, was significantly different from the cases described at the La Salpêtrière. He saw general slowing, stiffness in the lower limbs, especially initially, and a tendency toward retropulsion, etc. (Fig. 3). His description does not evoke Parkinson's, as he thought, but progressive supranuclear palsy, making it one of the earliest reports on this pathology, described in 1963 by the neurologists John Steele (1934–2022), John Clifford Richardson (1909–1986), and Jerzy Olszewski (1913–1964) [46].

On 21 July 1892, Paul Maigre (1863–1927) defended his thesis, *Quelques considérations sur l'astasie-abasie* (Some considerations regarding astasia-abasia) [47], before a jury presided by Debove, but his work was, in fact, directed by Fulgence Raymond (1844–1910), Charcot's successor, and owed much to the help of Jean Nageotte (1866–1948). Maigre added four new observations very similar to those of Blocq. He was able to cure the symptoms, which must have been hysterical in his

opinion, through hypnosis as taught by Nageotte, who would go on to become a famous neuropathologist.

In 1892, Edmond Weill (1858–1924), who went on to become a professor of paediatrics in Lyon, put forward the observation of a 28-year-old woman with ''choreic astasia'' which disappeared with pressure applied to her shoulders or the buttock region when she stood up, clearly a case of hysteria [48].

In 1914, in his monumental *Sémiologie des affections du système nerveux* (Semiology of nervous system affections) [49], Jules Dejerine (1849–1917) once again linked astasia-abasia to hysteria, ''to an ataxia due to deficient autonomic coordination''. However, he added a few lines on rapid forward gait and retropulsion during Parkinson's disease, which he seemed to consider a form of astasia-abasia. André Léri (1875–1930), a student of Pierre Marie (1853–1940) who wrote the ''Incoordination'' chapter of Marie's *Pratique neurologique* (Neurological practice) in 1911, was already of the same opinion [50].

In 1916, at a medical meeting of the 6th army, Georges Guillain (1876–1961) and Jean-Alexandre Barré (1880–1967) once again attributed to hysteria a case of astasia-abasia for which the description indeed casts doubt on an organic cause [51].

7. Non-hysterical astasia-abasia

Joseph Cénas (1858–?) published an initial case of symptomatic astasia-abasia in 1895 [52]. He drew on the work of the German Ludwig Bruns (1858–1916) who had described a similar case in 1892 [53]: a frontal tumour causing astasia-abasia. A 54-year-old man progressively developed difficulty moving their feet when they are in contact with the ground and who tend to fall backwards. His legs flexed without any other signs, pointing to a cerebellar or ataxic cause. Tendon reflexes were exaggerated. The autopsy found a tumour in the cerebral falx at the paracentral lobule. Similarly, Josef Gerstmann (1887–1969) and Paul Schilder (1886–1940) of Vienna, in addition to their work on the body schema, showed in 1926 that gait disorders, including astasia-abasia and the tendency to fall backwards, could be frontal in origin [54]. In 1907, Friedrich Hartmann (1871–1937) described, alongside other cases, a movement impulse disturbance and astasia-abasia in a patient with a tumour in the anterior commissure of the corpus callosum [55].

At the session of the Paris Neurological Society on 05 February 1920, Jean Lhermitte (1877–1959) and his student Maurice Quesnel (1890–1960) presented a paper on ''gait disturbances in the elderly'' [56]. Quesnel is also linked to Lhermitte and Victor Cornil (1837–1908) by the unused eponym Lhermitte-Cornil-Quesnel syndrome, or ''progressive pyramidopallidal degeneration syndrome'', a form of extrapyramidal syndrome [57]. They noted that trembling abasia had been described in 1901 by a student of Dejerine, the Swedish Karl Anders Petrén (1868–1927), who became a professor of neurology at Uppsala university then at Lund university [58]. Petrén distinguished paraplegia and its spasmodic phenomena from astasia-abasia, which he described as ''a flagrant disturbance, sometimes even involving complete obliteration of the ability to walk''. In cases of paraplegia,

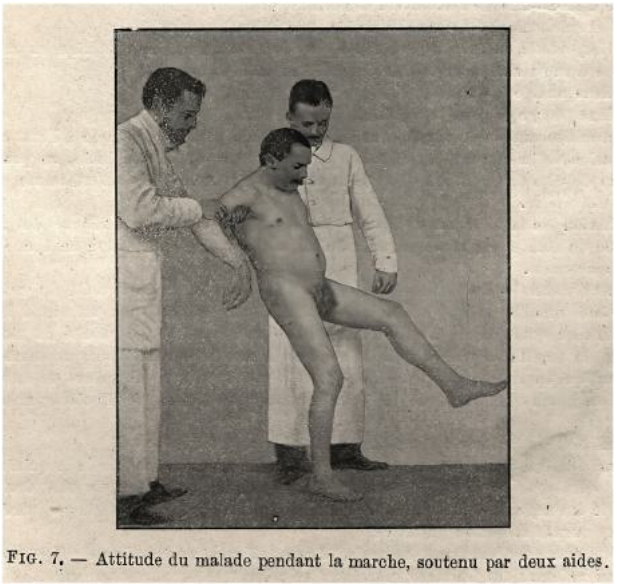


FIG. 7. — Attitude du malade pendant la marche, soutenu par deux aides.

Fig. 4 – Henri Mouninou (1864–1923), famous patient of J. Babiński [54] (OW Collection).

Lhermitte, like Petrén, considered hysteria as an exceptional aetiology, and an organic cause as the rule. Their description of astasia-abasia in the lacunar state, pseudobulbar of vascular origin, added an anxiety-phobic element to the psychological deterioration. This element was considered responsible for the gait disturbance, rather than Parkinson's disease. In the discussion that followed Lhermitte's presentation at the French Society of Neurology, Achille Souques (1860–1944) contested the interpretation of Lhermitte and Quesnel, considering astasia-abasia as only a symptom of Parkinson's.

At the session of the Paris Neurological Society on 09 March 1922, Lhermitte returned to astasia-abasia, presenting a case ''with a typical cerebellar, pseudo-drunken gait, with festination; the subject widens the horizontal region over which his centre of mass lies'' [59]. Babiński had described his region, or ''base de sustentation'', in 1899 (Fig. 4) [60]. After the patient died of pancreatic cancer, the autopsy did not reveal any vascular lesions but did show an exclusive cerebellar lesion involving atrophy in the ''superior and inferior cerebellar vermis and quadrilateral lobe'' (*du vermis supérieur et inférieur et du lobe quadrilatère*) (paleocerebellum). His student, Jean Arteaux (1897–?), made this type of cerebellar astasia-abasia, involving atrophy of the vermis, the subject of his thesis, defended in 1924 with Henri Claude (1869–1945) presiding over the jury [61]; at that time, the formal diagnosis could only be made at the point of autopsy. As for the patient, his gait started to zigzag after fifty years of age. Standing immobile, he tended to fall backward despite the enlarged horizontal support region. Arteaux agreed with the theory developed by André Thomas (1867–1963) in Dejerine's laboratory at La Salpêtrière: ''The vermis does indeed regulate balance and coordination of the movements in the lower limbs'', notably because it is anatomically linked to vestibular centres by the lower cerebellar peduncle. He saw in the vermis the involuntary, ''reflex'' part of the cerebellum. Thomas wrote, ''There is trembling, oscillations, and staggering that seem to depend on

an imperfect summation of elementary impulses on which the contraction depends. This category of phenomena constitutes astasia'' [62].

In 1922, in his remarkable thesis presided by Pierre Marie and covering the after-effects of encephalitis lethargica, Gabrielle Lévy (1896–1934) described gait with shuffling steps and festination in Parkinsonian syndrome ''that could lead to a case of trembling astasia-abasia'' (p. 61) [63].

At the 15 March 1925 session of the Paris Neurological Society, Barré published the first observation of a case of astasia-abasia of labyrinthine origin. A 54-year-old man had spells of dizziness and walked with the shuffling steps characteristic of astasia-abasia: ''This set of disturbances evokes the existence of partial vestibular syndrome not involving the peripheral or ascending central pathways but the descending pathways and, in particular, the left vestibular-spinal pathways''. He concluded, ''It is useful to look for, in so-called functional astasia-abasia, more or less latent disturbances in labyrinthine or vestibular nerve functions, and also syphilitic antecedents'' [64].

On 13 April 1933 at a conference held in Rabat for alienists and neurologists from France and francophone countries, Raymond Garcin (1877–1971) issued a report on ataxia. A brief chapter covered astasia-abasia, the origin of which he placed in the frontal lobe (tumour), or which he considered to indicate a lacunar state [65].

8. Astasia-abasia versus gait apraxia

The term gait apraxia is currently in common use by neurologists. Gait apraxia includes disturbances of trunk movements, standing, and walking that are not caused by orthopaedic abnormalities, muscle wasting, arteriosclerosis, pyramidal deficits, ataxia (cerebellar, vestibular, or proprioceptive), dystonia and dyskinesia, psychiatric disease, drug side effects, or ''cautious gait'' due to fear of falling (also known as ''abasie trépidante'', to use Charcot's term, or stasobaso-phobia). The term ''gait apraxia'' appeared for the first time in 1871 in a book by the German philologist Chaim Steinhal (1823–1899) [66]. But it was not until thirty years later that the German Hugo Liepmann (1863–1925) brought this term into neurological practice, within the meaning of ''a motor asymbolia'' [67]. He described melokinetic apraxia, or loss of kinetic memory of a limb; bilateral ideomotor apraxia, corresponding to an interruption between the representation of the act and motor control; and finally ideational apraxia, corresponding to damage or destruction of the ''movement formula''. For Liepmann, these forms of apraxia were the result of left hemispheric lesions.

In 1926, the Viennese, later American, neurologists Gerstmann and Schilder used the term ''apraxia of gait'' in a more specific sense. According to them, gait disorder resulted from bilateral ''gluing'' of the feet to the ground in patients with extensive bilateral frontal lobe damage but no elementary motor deficits [68]. In 1958, Derek Denny-Brown (1901–1981) characterised gait apraxia (or kinetic apraxia) as a mental incapacity to adapt gait to one's environment; that is, ideational apraxia causing loss of autonomy in ambulation [69]. In practice, patients initially walk with slow, irregular steps. They struggle to negotiate turning, stepping onto a stool, avoiding obstacles in their path, or lying down on and getting up from the examining Table 1. Then as the disease progresses, any ambulation becomes impossible, due solely to the persistence of primitive action routines not adapted to the subject's environment (disturbances in spatial thought). Some cases of Alzheimer's disease provide an example of this [70].

Are gait apraxia and astasia-abasia synonymous? Not exactly. The term ''gait apraxia'' encompasses, in addition to a clinical meaning, different pathophysiological concepts. On the contrary ''astasia'', to designate the motor incapacity to remain vertical, and ''abasia'', to designate the inability to initiate a step (gait initiation failure) or continue walking, illustrate the specificity of a clinical sign for clearly depicting a medical status.

The eponym ''Bruns apraxia'' appeared after a description by Bruns in 1892, as explain above [53]. The symptomatology suggested by Burns seems in some ways similar to that of chronic hydrocephalus at normal pressure, evoked by a triad: superior function disorders, gait disturbances, often astasia-abasia, and sphincteric problems, as described in 1964 by Salomón Hakim (1922–2011) in his thesis and validated by Charles-Miller Fisher (1913–2012) [71–73]. In fact, patients present various gait phenotypes according to the severity of the dilated cerebral ventricles, also influenced by comorbid neurological conditions such as vascular lesions. According to John G. Nutt, gait impairment is best characterised as ''a higher-level gait disorder'', which, in the absence of primary sensorimotor deficits, cerebellar dysfunction, or involuntary movements, involves difficulty in incorporating sensory information about the position of the body in its environment, including the effect of gravity and properly selecting and executing motor plans for gait or postural reflex. Findings include difficulty with transitional movements (sitting to standing or standing to sitting); gait initiation failure; shuffling and poor foot clearance; tripping, falling, or festination; unstable multistep turns; and retropulsion or anteropulsion [74]. Patients with risk factors may have secondary hydrocephalus; for example, those with a history of intracranial haemorrhage, trauma, infections in the central nervous system, or those who have undergone intracranial neurosurgery [75]. At the present time, astasia-abasia suggests the possibility of chronic hydrocephalus at normal pressure; i.e., a potentially treatable disorder, but still a severe disease in many patients. The only effective treatment is a cerebrospinal fluid (CSF) shunt, usually configured between the lateral ventricle and the abdomen (ventriculoperitoneal shunt) [75]. To assess the reversibility of gait impairment in this pathology, the CSF tap test, introduced in 1961 by Fisher, represents a widely used prognostic procedure [76]. The anatomical pathway of lower limb control just adjacent to the lateral ventricles is the most widely accepted mechanism today, and may explain cases of immediate improvement after the spinal tap test.

It is currently common to speak of gait apraxia to describe a slowed gait, as seen in cases with bilateral frontal damage generally involving degeneration or resulting from chronic hydrocephalus. However, this description seems imprecise because it suggests an automatic-voluntary dissociation, which does not occur in reality. That is, corticobasal

connections are more involved in the pathogenic process than the cortex strictly speaking. The term ''astasia-abasia'' is more suited to describing this disturbance in standing upright since, aside from remaining upright and waking, the functional organisation of the axial musculature is affected in its entirety. As an alternative to ''astasia-abasia'', Henri Hécaen (1912–1983) coined the term ''troncopedal apraxia'' for cases that English-language authors now call ''higher-level gait disorders'' [77], which is approximative to some degree. Unlike the term ''astasia-abasia'', it denotes an anatomical and cerebral origin for gait disturbance and eliminates a peripheral cause (proprioceptive deficit in diabetic neuropathy, tabes, etc.), indicating possible damage to the white matter forming the circuits that control balance and locomotion. Imaging shows various types of damage at various possible levels, from the medial parts of the frontal lobes to the pathways that unite them with the basal ganglia and the mesencephalic locomotor region (subthalamic nuclei, pedunculopontine nuclei, substantia nigra) [78]. The causes are frontal parasagittal tumors (meningioma, glioma, metastasis), tumors in the corpus callosum, vascular damage in the territory of the anterior cerebral arteries, aneurysm of the anterior communicating artery, lacunar state as described by Pierre Marie, and neurodegenerative diseases such as Parkinson's or Parkinson-plus syndrome [78–80].

In 1993, Nutt, Marsden, and Thompson proposed a clinical approach to making an accurate diagnosis: ''Clinically, description of gait disorders will be facilitated by attention to each of the following key elements:

- rising from a chair (righting reflex);
- standing (supporting response);
- withstanding a push fore and aft and side-to-side (reactive postural responses);
- initiation of walking, presence of freezing, start hesitation, and turn hesitation;
- locomotion, with reference to width of the base, stride length, foot clearance, arm swing and cadence;
- negotiation of turns;
- presence of effective rescue and protective reactions if imbalance spontaneously occurs, or in response to a push'' [81].

9. Conclusion

From the last quarter of the nineteenth century until the middle of the twentieth century, the concept of astasia-abasia, as named by Blocq and Charcot, saw its semiological reality confirmed but its aetiology changed. The cases of functional astasia-abasia, described by Blocq in children, have nothing in common with current astasia-abasia in adults, often elderly and suffering from gait difficulties due to organic pathologies of the nervous system [80]. Contemporary neuroimaging tends to reveal selective decreases in the activity of frontal and subcortical circuits involved in motor control in the case of so-called ''conversive'' phenomena. This observation of a functional deficit may illustrate the ''dynamic lesion'' conceived of (dreamt?) by Charcot to provide a pathophysiological explanation for the hysterical astasia-abasia described with Blocq [80,82], thus bringing together historical functional disorders and the organic disorders observed today. The rise in the number of people leading longer lives seems valid explanation. But we need to add that three biases limited Charcot in his identification of the organic causes of astasia-abasia.

The first bias involves knowledge. Starting the second half of the 20th century, advances were made in the nosography of neurological pathologies and in the differentiation of pathophysiology enabled by brain imaging, neurobiology, and tractography. This enhanced the knowledge necessary for identifying the various organic causes of astasia-abasia.

The second bias relates to classification and attribution. The concepts of neurons, neurodegenerative pathology, and hydrocephalus at normal pressure did not exist in Charcot and Blocq's day. The semiology of cerebellar diseases was still in its infancy.

The third bias is that of selection. During the first twenty years of his practice at the hospice de vieillesse femmes de La Salpêtrière, both a nursing home and an asylum, Charcot only treated elderly patients, most often bedridden, and young women with epilepsy or hysteria. He asked for and obtained a weekly consultation time for patients from the city, and it was only from 1881 until his death in 1893 that he saw an increase in the recruitment of less seriously impaired patients, allowing him to observe non-geriatric pathologies, such as Gilles de la Tourette syndrome. This period only lasted some ten years, during which time he lacked enough of mostly very old patients of the city, for identifying cases of non-hysterical astasia-abasia, which present-day neurologists are able to observe [83,84].

In sum, the long history of astasia-abasia reveals a cluster of neurologists, all of whom, by the precision of their clinical examinations and their pathophysiological findings, helped advance the understanding of the mechanisms by which human beings are the only erect, constantly bipedal mammals, whether immobile or walking.

Statement of ethics

This work required no approval from an institutional review board and was prepared in accordance with the ethical guidelines of the journal *La Revue Neurologique*.

Disclosure of interest

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