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Duchenne, Charcot and Babinski, three neurologists of La Salpetrière Hospital, and their contribution to concepts of the central organization of motor synergy

François Clarac [a,*], Jean Massion [b], Allan M. Smith [c]

[a] P3M, CNRS, 31 Chemin Joseph Aiguier, 13402 Marseille Cedex 20, France
[b] Les Micocouliers, 16, Route de Rognes, 13410 Lambesc, France
[c] Département de Physiologie, Pavillon Paul-G Desmarais, Université de Montréal, Montréal, Québec, Canada H3C 3J7

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ABSTRACT

Many currently accepted notions of motor control originate from a few seminal concepts developed in the latter half of the 19th century (see Bennett and Hacker, 2002). The goal of this review is to retrace some current ideas about motor control back to the thought of three French neurologists of Hospital of the Salpetrière hospital in Paris during the latter half of the 19th century and early 20th century (Fig. 1): Guillaume Duchenne de Boulogne (1806–1875), Jean-Martin Charcot (1825–1893), and Joseph Babinski (1857–1932). A common theoretical and methodological thread unites these three men as Charcot was taught neurology by Duchenne, and Babinski was trained by Charcot. The influential concepts developed by these pioneering French neurologists have been neglected for nearly a century and only rediscovered recently. We intend to highlight how these astute clinicians used their meticulous clinical observations of patients to reveal novel and original perspectives of motor co-ordination. Between 1850 and 1930, all three men played a major role in developing and shaping the entire field of normal and pathological motor control in addition to making important contributions to three major scientific issues; the centralist view of muscle sense, the emerging concept of muscle synergy in voluntary movements and in locomotion and finally the specific role of the cerebellum in muscle synergy. The important contributions of these men will be considered in the context of other significant schools of neurology from other countries. Finally, the concept of cerebellar asynergy as proposed by Babinski anticipated the development of the internal models which much later were able to provide a theoretical basis for understanding the mechanism of learned motor co-ordination involving the cerebellum.

1. Introduction

During the XIXth century, one of the major neurological issues concerned the mechanism of motor control and its consequence on posture and movements. It was a time when different cortical areas had been identified (see Broca, 1861, Fritsch and Hitzig, 1870), and a variety of neuro-pathologies were described and properly identified (see Parkinson, 1817, Charcot and Joffroy, 1869). Among the many interesting groups of scientists and clinicians involved in European centers dedicated to the analysis of motor control, the neurological center at La Salpetrière in Paris merits special attention because of three neurologists who contributed several key notions about motor co-ordination. Duchenne, Charcot and Babinski created a set of new concepts which were so complementary to each other that they seemed to express the thinking of a single person. Most of their ideas persist today despite the fact that the identity of the original contributors seems almost forgotten.

Although the three neurologists were not contemporaries, the knowledge passed down from one to the other formed a closely coherent ensemble. Charcot invited Duchenne to work in his hospital and he was strongly influenced by his clinical observations. Babinski, who was considered to be Charcot's favourite student, always respected Charcot, who he referred to as the master of La Salpetrière, as his mentor. Duchenne also indirectly influenced Babinski's clinical reasoning through Charcot despite the fact that Duchenne and Babinski were two generations apart.

Curiously the three neurologists are best remembered for some of some other clinical observations. For example, apart from the muscular dystrophy bearing his name, Duchenne is remembered for his essay on emotion-driven facial movements, Charcot for his interest in hysteria and Babinski for his "sign of the toes". In this account we intend to draw attention to their specific

* Corresponding author. Fax: +33 0491775084.
E-mail address: clarac@dpm.cnrs-mrs.fr (F. Clarac).

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contributions to our understanding of the initiation and co-ordination of voluntary movement, the field in which their contribution was so impressive across generations, although not always well recognized. Duchenne, Charcot and Babinski in total practiced for more than eight decades, from 1850 to 1930, at the most famous hospital in Paris. At that time La Salpetrière included among its 3000–5000 patients a vast variety of mental illnesses and neurological disorders, the etiologies and classifications of which were poorly understood. This heterogenous and diverse patient population no doubt contributed to sharpening the clinical observational skills of all three neurologists.

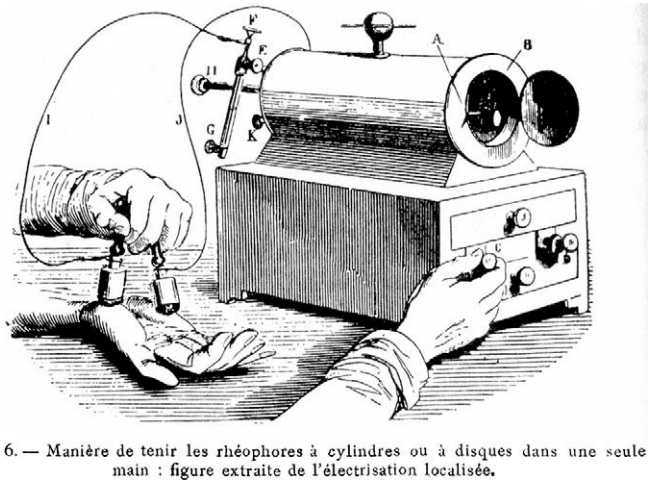
2. Duchenne de Boulogne, Charcot and Babinski and their clinical diagnostic practice at "La Salpetrière

Initially, the three neurologists had nothing in common since they came from divergent social backgrounds, and in contrast to their profession, they led distinctly different personal lives.

2.1. Duchenne de Boulogne, "the buccaneer" of neurology

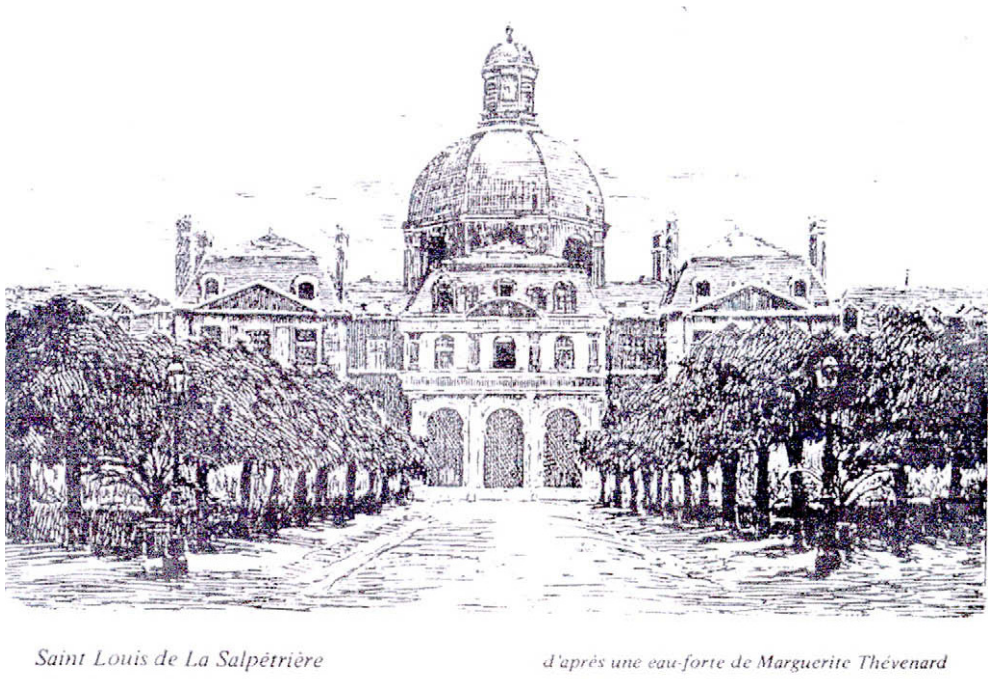
Duchenne de Boulogne (1806–1875), was the oldest of the three, and a native of the northern seaport of Boulogne. His father was one of the last of the "privateers" who raided colonies and ships along the American coast at the end of the 18th and early 19th century during the Napoleonic period. Rondot (2005), referred to Duchenne as a "buccaneer of neurology", alluding to his father's unusual occupation. Duchenne studied medicine in Paris under teachers including René T.H. Laennec (1781–1826), Guillaume Dupuytren (1777–1835), François Magendie (1783–1855) and Jean Cruveilher (1791–1874). He presented his thesis titled "*An Essay on Burns*" and graduated in medicine in 1831. He did not, however, initially pursue an academic career and returned to Boulogne where he practised medicine for a decade. A family feud made his life untenable in Boulogne and ultimately he was forced to return to Paris in 1842.

Upon his return to Paris he developed a particular interest in the physiology of "localized electrisation", using an induction coil to produce faradic current, he began to stimulate various muscles in some of his patients. He employed the apparatus proposed by the physician Jean Baptiste Sarlandière (1787–1838) who had used it to treat rheumatisms. From a position of total obscurity and without any support from the academic medical community of Paris, Duchenne began to study his patients by applying current to muscles with his faradic stimulating machine. This experience provided a unique insight into the *in vivo* functional anatomy of skeletal muscles. Prior to this time no one had studied the relationship between muscle weakness or paresis and muscle atrophy with such an objective and reproducible technique. In time Duchenne became a renowned expert on muscle physiology based on his technique of electrical stimulation and with his two "humid rheophores" (Fig. 2) and with his induction apparatus he was able to identify and clarify the distinct mechanical actions of many individual muscles. As a result of his extensive experience Duchenne was



6. — Manière de tenir les rhéophores à cylindres ou à disques dans une seule main : figure extraite de l'électrisation localisée.

Fig. 2. The Duchenne's electrical apparatus with his two humid electrodes ("rheophores"). Duchenne de Boulogne was able to stimulate very specifically some human muscles (in Duchenne de Boulogne, 1855, Fig. 24).



Saint Louis de La Salpétrière *d'après une eau-forte de Marguerite Thévenard*

Fig. 1. The Salpetrière Hospital and Church. Etching from Marguerite Thevenard (from UPMC/BIUSJ-SCDM/Bibliothèque Charcot).

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convinced that movements are the resultant action of muscle groups acting in synergy. In 1850 he wrote "*Galvanic stimulation of muscles can be called upon to establish the exact manner in which a large number of muscles are used. We know that under the influence of volition few muscles are contracted individually. On the contrary, most of them only contribute to a portion of the entire movement.*"[1]

From further observations he was able to discover that isolated muscles often play a different role than was previously deduced from the anatomical insertions and origins. This systematic description led naturally to the study of localized muscular atrophies and hypertrophies (described in his book, Duchenne de Boulogne, 1855 *De l'électrisation localisée et de son application à la physiologie, à la pathologie et à la thérapeutique*). Duchenne went on to demonstrate the impact of paralysis of an antagonist muscle on the mechanical action of the agonist. The muscle faradisation soon became an indispensable tool to his clinical examination (Guilly, 1977).

Duchenne was also probably the first person to use muscle biopsy to obtain tissue from living patients for microscopic examination. His extraordinary skill in analysing clinical problems earned him a certain eminence in the medical community. Even today Duchenne is widely remembered in medicine for the first description of an inherited disorder in the 1860s that today is known as Duchenne type muscular distrophy. His rather aggressive and heated disagreements with Robert Remak (1815–1865), about whether galvanic or faradic current should be used for stimulation and about the optimal site for muscle stimulation further enhanced his reputation as an authority on the subject. Duchenne believed that the muscle could be directly stimulated by the current applied to its surface and not merely through the stimulation of motor nerves.

As his reputation grew, Duchenne became recognized as a muscle expert at several Parisian hospitals and in particular at "La Salpetrière" with Charcot, who offered Duchenne access to his patients. Many of Charcot's scientific approaches were influenced by Duchenne, and the young Charcot considered him as his "mentor". Duchenne also became interested in early photography and he recorded the effects of facial muscle faradisation as well as the most typical pathological cases he observed. In 1862 he published a collection of photographs of live patients (Duchenne de Boulogne, 1862; Parent, 2005). After an analysis of glossolabiolaryngeal paralysis, the origin of which is in the brain stem, he initiated the production of serial micrographs of that region and presented them like an atlas to the Academy of Medicine in 1864. Duchenne de Boulogne (1850) also studied the contraction of the facial muscles and related these expressions to the induction of emotions (see Walter et al., 2006). He explained for example that he obtained smiling by an electrical stimulation of the *zygomaticus major* while spontaneously the *orbicularis oculi* is also contracted simultaneously (Ekman et al., 1990). Although this work was widely criticized, Charles Darwin (1809–1882) was quite intrigued by his different facial expressions as well as by his splendid photographic records, and subsequently used some of them in his book on emotion in animals and men (see Darwin, 1872). Duchenne died in September 1875 in the company of Charcot who remained at his side during his final days before the end.

2.2. Charcot, professor of diseases of the nervous system

Charcot began to practice at La Salpetrière in 1862 with his fellow medical student, Alfred Vulpian (1826–1887), who first held the chair of Pathological Anatomy, that Charcot was to inherit a decade later. Considered as the founder of modern neurology, Charcot was the first professor to occupy a Chair of diseases of the Nervous system created for him in 1882. A tireless worker, and a captivating teacher, Charcot employed a number of theatrical techniques during his lecture-demonstrations (Fig. 3). On Tuesday mornings he held his famous and sometimes even spectacular clinical demonstrations for a large and varied audience and, on Fridays, he delivered his well-prepared and rehearsed lectures for a scientific public with a particular interest in clinical cases (Brais, 1990; Goetz et al., 1995). These lectures earned him an enviable reputation throughout the medical scientific community and even a certain popular celebrity as a person of "*le Tout Paris*"! Thanks to a marriage to a wealthy Parisian widow, Charcot moved into in a prestigious house called the "Hotel de Varangeville" built in 1704 at "N°217 boulevard Saint Germain," (note: It is now "*La Maison d'Amérique Latine*". Except for a "plaque" that refers to the presence of Charcot, nothing remains to recall this period). As a result of his influence, the majority of Charcot's hospital interns like Bouchard, Cornil, Joffroy, Debove, Strauss, Hanot, Raymond, Brissaud, Ballet, and Marie eventually became professors at the Faculty of Medicine (Brais, 1990; Goetz et al., 1995). He also attracted a large number of foreign neurologists to Paris such as Betcherew, Darkschewitch, Marinesco, Christiansen, Freud, Starr, and Sachs. I.S. Wechsler is quoted in Haymaker and Schiller (1953) as saying "*There was much of the artist in him. Few possessed more éclat*".

The name of Charcot is associated with at least 15 medical eponyms. During his immensely productive period between 1862 and 1870 he gave a series of masterly clinical descriptions, most of which were on movement disorders. For example, Charcot described the clinical spectrum of the "*paralysis agitans*" in detail, and in acknowledging its first description, Charcot suggested that the syndrome should be called Parkinson's disease. Charcot also contributed much to correctly distinguishing between bradykinesia and rigidity. He described a great number of disturbances originating from spinal cord defects, linking them to a variety of motor dysfunctions. Charcot identified "*Amyotrophic lateral sclerosis*" (ALS, *Sclérose latérale amyotrophique*" SLA, Charcot and Joffroy, 1869). Charcot's student Gombault (1844–1904) studied this pathology in nine cases and his thesis (1877) proved the first valid description of grey matter involvement and white matter

Fig. 3. Charcot during a lesson on the Basedow pathology drawing from Paul Richer (1849–1933) reproduced from "*La leçon de charcot, voyage dans une toile*" Catalogue-exposition. AP-HP Museum (from UPMC/BIUSJ-SCDM/ Bibliothèque Charcot).

[1] *La galvanisation musculaire est appelée à établir d'une manière exacte les usages d'un grand nombre de muscles. On sait qu'il est très peu qui peuvent se contracter individuellement sous l'influence de la volonté. La plupart d'entre eux, au contraire, ne font que concourir à des mouvements d'ensemble.*

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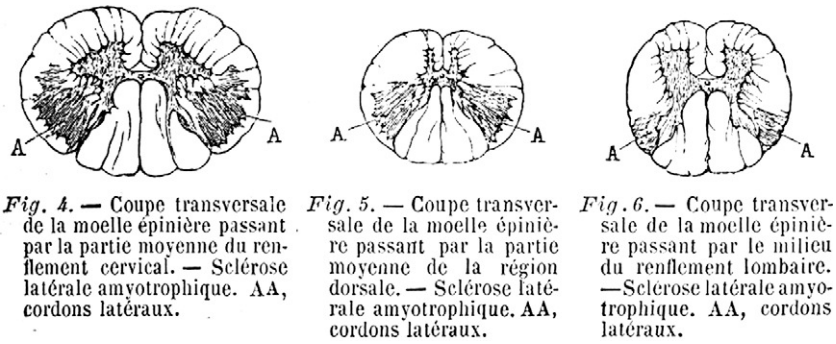

Fig. 4. — Coupe transversale de la moelle épinière passant par la partie moyenne du renflement cervical. — Sclérose latérale amyotrophique. AA, cordons latéraux.

Fig. 5. — Coupe transversale de la moelle épinière passant par la partie moyenne de la région dorsale. — Sclérose latérale amyotrophique. AA, cordons latéraux.

Fig. 6. — Coupe transversale de la moelle épinière passant par le milieu du renflement lombaire. —Sclérose latérale amyotrophique. AA, cordons latéraux.

Fig. 4. Drawing from Albert Gombault's Thesis. The spinal cord of an ALS patient is represented at three different levels. Progrès médical 1877.

degeneration (see Fig. 4). Charcot also entered the great argument of the period over cerebral localization with a celebrated debate with Brown-Sequard at the "*Societé de Biologie*" (1875/1876). Charcot supported the localisationist theory, which he reasoned was supported by the clinical observation that identical cortical lesions were always followed by the same symptoms. In contrast, Brown-Sequard rejected the localization theory claiming that interconnected networks extending throughout the whole hemisphere were responsible for the organization of movement (see comments in Wiesendanger, 2006). Charcot's theory of voluntary movement had two parts; "a fundamental apparatus" composed of a muscle-centered efferent representation of movements, and a second component corresponding to different afferent sensory representations of movements, signaled by kinesthesia and vision (Fig. 5, Gasser, 1995). He was convinced that Paul Broca (1824–1880) was right on aphasia and he fully supported Broca's data. He introduced Wernicke, Kussmaul and Exner and the contributions of the German school into France in 1874. Charcot was also quite influenced by the English "Associationists" (see A. Bain, 1818–1903), and he summarized his ideas on language in "*the bell schema*" (*Le schéma de la cloche,* 1884, see in Gasser, 1995) that combined all of these different interpretations.

Later Charcot's interests turned to hysteria where he advanced the concept that the affected persons had diseases of the brain that were more functional than structural. The famous 1887 painting of André Brouillet depicted the "*Caesar of the Salpetrière*" examining Blanche Wittmann, an hysterical patient made famous by Charcot. The patient is supported by Charcot's "chef de clinique", Joseph Babinski, aptly described by Khalil (1979) as the "bearded colossus", before an audience of students, colleagues and other literary persons. Today Charcot is best remembered and even criticized for his work on hysteria, whereas his major contributions to the motor pathologies of the nervous system seem to be all but forgotten. Charcot died unexpectedly of a heart attack in September 1893.

2.3. Babinski and his sign

Born of Polish parents, Joseph Babinski (1857–1932) was the youngest and most brilliant pupil of Vulpian and Cornil. He graduated in medicine from the University of Paris in 1884 with a thesis on multiple sclerosis. On the strength of a particularly strong recommendation from Vulpian, Babinski was chosen to become Charcot's chief resident at La Salpetrière from 1885 to 1887. Charcot quickly recognized Babinski's talent as a highly astute clinical observer. During the early 1890s both Charcot's health and his influence in the medical faculty were waning and after his sudden death in 1893, Babinski's academic career fell victim to an intrigue among certain professors of medicine who felt the time had come to diminish the great power of Charcot at the Salpetrière. As a result, Babinski was never able to obtain a promotion to the rank of "*associate professor*" and this spelled the end of his academic career.[2] He eventually became the head of the neurological clinic at the neighbouring "Hospice de la Pitié" (Philippon and Poirier, 2008). He was a masterly clinician, and considerably less dependant on neuropathological examinations and laboratory tests than most of his contemporary colleagues. "*In examining a patient ... he was a genius in searching for defects, a man of inexorable logic*" (Wartenberg, p. 399 in Haymaker and Schiller, 1953). Babinski also took an interest in the pathogenesis of hysteria and he was the first to present an acceptable differential diagnostic criteria for separating hysteria from organic neurological diseases. In 1900, he demonstrated that hysterical patients, unlike most neurological patients, had no reflex abnormalities. In later articles, he emphasized the power of suggestion emanating from the people surrounding the hysteric patient and he defined a sort of psychologically suggestable state that he called "*le Pythiatisme*" (Babinski, 1909).

According to Khalil (1979), Babinski's first attention to the reflex of the toes occurred during a chance observation of the contrasting responses between two patients; one a hysteric and the other a hemiplegic. First published description of his famous "sign" was in 1896 at a meeting of the Société de Biologie in a communication of merely 28 lines: the pathological extensor plantar response when the sole of the foot is stimulated whereas in normal subjects, the same stimulation produces a general flexion. This first publication was nearly ignored and Babinski felt compelled to give fuller accounts of the reaction in 1898 and 1903 calling it "*the toe phenomenon*" (*le phenomène des orteils)* emphasizing its invariable association with pyramidal tract lesions whatever their duration, intensity or extent. In its simplicity and physiological implications, Babinski's sign has hardly any equal in medicine (see van Gijn, 1996). Perhaps this phenomenon had been noted by others earlier, but Babinski's description was so precise that it became a sign of fundamental importance in neurological examination. Another lesser known observation by Babinski was the tendency for hemiplegics to pronate the contralateral hand. This test, currently called "*pronator drift*", was recently validated using modern brain imaging methods by Teitelbaum et al. (2002).

Babinski was also quite attracted by the nascent field of neurosurgery on the spinal cord and he referred patients to Victor Horsley (1857–1916) in England, who at the time, was uniquely able to do such surgery having performed his first tumor extirpation on June 9th 1887. Later Babinski convinced two of his students Thierry de Martel (1875–1940) and Clovis Vincent (1879–1947) to undertake the first successful spinal operation in France earning him the title of the father of French Neurosurgery (Fulton, 1933).

[2] It is worth noting that although Babinski received his training in clinical neurology at La Salpetrière, political intrigue prevented him from practicing there and instead he was relegated to the less prestigious, but nearby, La Pitié Hospice.

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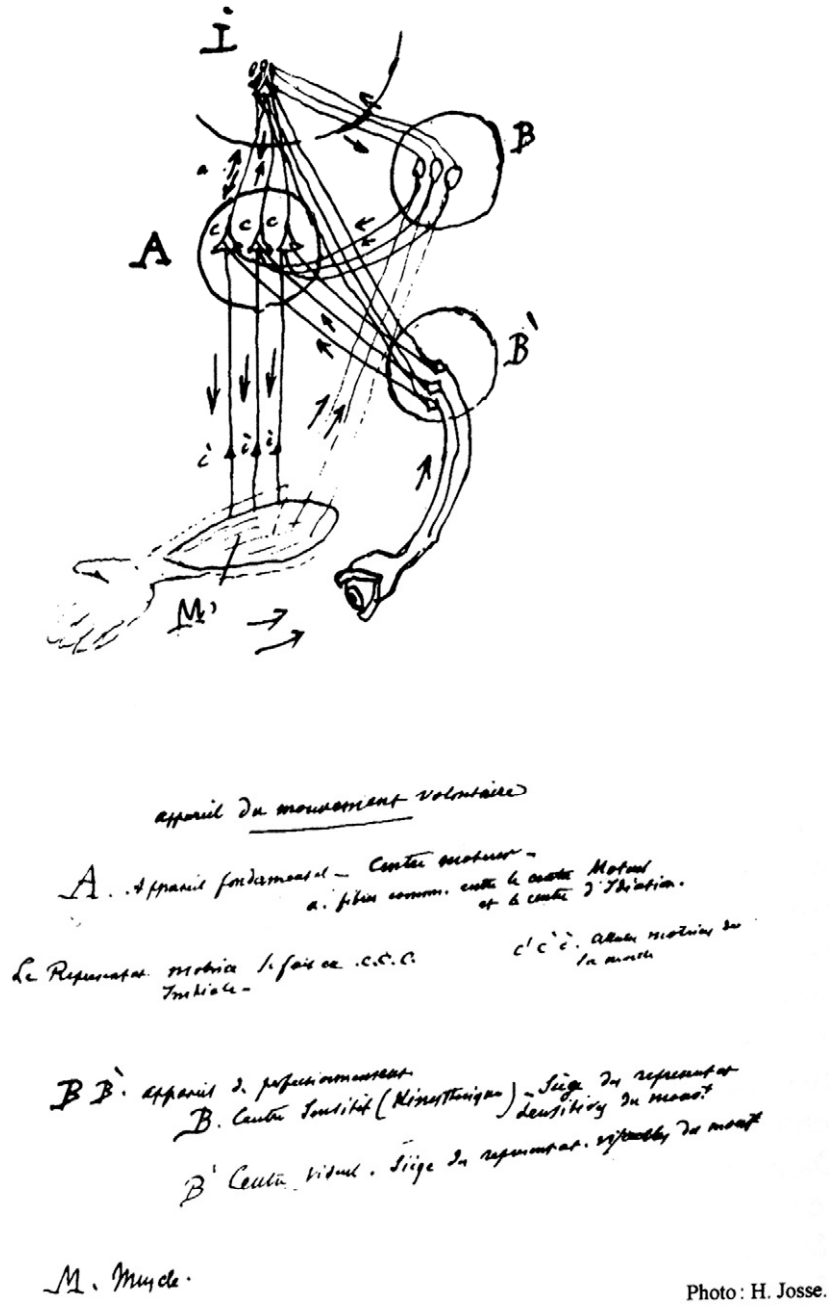

Fig. 5. Schema of a voluntary movement drawn by Charcot. A represents the functional apparatus that precedes the execution of the voluntary movement. I corresponds to the "idea center" where the sense of innervation and the two peripheral components converge (Kinaesthetic and visual center). See text for further explanation. UPMC/Bibliothèque Charcot. Observations. MA VIII.

Joseph Babinski's father, Alexandre was a civil engineer who after the birth of his two children spent five years working in Peru to support his family. During the father's absence Henri (1855–1931), Joseph's elder brother assumed a protective role for his younger sibling. Both Joseph and Henri Babinski remained bachelors, living first with their parents, then alone at 170 bis boulevard Haussmann from 1890 until their deaths in 1931 and 1932. Although Henri, like his father, was trained as an engineer, he was eventually to become one of the most celebrated culinary experts in France. His famous cookbook, *Gastronomie Pratique*; *Études Culinaires*, was published under the pseudonym of Ali Bab, and it is still widely cited today. The brothers were very close and their diner invitations were offered only to a few close acquaintances. These lucky individuals were treated to a unique gastronomic experience, the menus of which can be found in the *Gastronomie Pratique*. In later years Joseph Babinski was severely afflicted with Parkinson's disease and he survived his older brother's death by less than a year.

2.4. The anatomo-clinical method

Of our three pioneering neurologists, Charcot was perhaps the most famous and had the most important official position (Goetz et al., 1995). Testimony to his international renown was expressed at the International Medical Congress of London in 1881 where a special fireworks display was arranged for him and two famous neurosurgeons, the Englishman Sir James Paget (1814–1916) and the German Bernhard van Langenbeck (1810–1887). Charcot was

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an ardent anti-vivisectionist despite the fact that he was an active member of the influential *Société de Biologie*. Nevertheless, he was intimately familiar with the entire anatomical and physiological literature on the central nervous system (CNS). He energetically promoted his clinic at La Salpetrière to both the medical community and to an interested general public. It was at this clinic that he developed and definitively established the clinical anatomical method that correlated patients' symptoms with the lesions later discovered at the time of autopsy.

In fact, all three of our groundbreaking neurologists accorded the utmost importance to physical examination, and the fastidious observation of the patient were considered an essential part of their consultation. Of the three, Babinski was perhaps the most impressive in his examination of patients which, accompanied by his assistant, was always done with "great ceremony". The patient was presented totally naked and instructed to pay strict attention to Babinski's instructions while the master conducted his examination in complete silence. He would ask patients to perform particular movements or to walk about the room in order to analyse the specific contractions of the various muscles. He was a highly skillful user of the neurologist's hammer to induce various reflexes to better understand and localize dysfunction in the nervous system. Reflex testing had been introduced into the neurological examination several decades earlier, in 1875, by two German neurologists: Westphal (1833–1890) and Erb (1840–1921). Babinski's examinations often lasted several hours, as he carefully observed the spontaneous abnormalities in a patient's behaviour. He then proceeded to systematically evaluate the reflexes and reactions with infinite care. If Babinski was not completely satisfied with the reflexes he observed, his testing would be continued on another day! The culmination of this method was the autopsy, with a complete scrutiny of the brain, spinal cord and peripheral nerves. When J.M. Charcot initiated this combined clinical and anatomo-pathological approach it was considered an unconventional medical practice in France. He specifically engaged one of his pupils, Victor Cornil (1837–1908) to go to the laboratory of Rudolf L.K. Virchow (1821–1902) in Berlin to learn the different histological methods to be applied at autopsy. Later, he organized a room with several microscopes for histological study at La Salpetrière.

Charcot worked on different motor diseases during the first part of his career, from 1862 to 1875/1880, and it is quite obvious that most of his contributions were made in a direct line from Duchenne. For example, around 1850, Duchenne and Aran described "progressive muscular atrophy". This description concerned a major impairment of movement. When Cruveilher autopsied the circus performer Lecomte in 1853, he was able to demonstrate that its origin was due to severe atrophy of the anterior spinal roots. This work was in fact a first approximation of the description of amyotrophic lateral sclerosis (ALS). Moreover when Duchenne analysed a glossolabio-pharyngeal paralysis, Charcot considered that this was also present in ALS when the bulbar motor nuclei were lesioned.

3. The muscle sense: central versus peripheral origins

During the 19th century, the existence of a "muscle sense" was quite controversial, and Duchenne as well as Charcot were major protagonists in these controversies. In fact, it had been debated since Aristotle (384–322 BC) identified the classic five senses, suggesting that muscle sensation and in particular the sense of movements was associated with touch which, while not wrong, was certainly misleading. Galen, who followed Aristotle, described the muscles in detail and stressed the unity of the receptive and the active function of touch. During the XVIII century there emerged a new concept of muscle sensations, related to the operations of the body and particularly of the locomotor apparatus (Jones, 1972). This notion appears to have originated with the French "sensationist" philosopher, Etienne B de Condillac (1715–1780), who himself was influenced by John Locke (1632–1704). Condillac referred to a mechanism of "active touch" whereby we appreciate extent, weight and resistance by sensation allied with movement (Jones, 1972). This was later evoked in 1820, by the Scottish philosopher Thomas Brown (1778–1820) who spoke of an awareness of muscular contraction (Brown, 1846). However, according to Scheerer (1987), it was Johann J. Engel who was the first to consider the concept of a sense of "muscle force" in 1802, suggesting that muscles are organs by which ideas are acquired about external objects, and he was possibly the first to conceive an object based internal model. For Engel, active touch included not only sensation from the skin but also information derived from muscles as well (the combination of both corresponding to today's concept of haptic sense), and he was of the opinion that muscle sense involved two components; one "*efferent* (sense of effort)" and one "*afferent* (perceived muscle force)". Marc Jeannerod (1983) considered these two aspects calling them the "efferent" and "afferent" hypotheses. More recently Jeannerod (1996 and 2006), suggested that the former was promoted by the "*centralists*" and the latter by the "*peripheralists*".

3.1. Centralist views of muscle sense

The French philosopher Maine de Biran (de Biran, 1766–1824, "dit" Maine de Biran) in expounding on a philosophical framework describing the "*will and the sense of effort*", postulated that motor actions start within the central nervous system, claiming that only voluntary (willed) muscular activity was accompanied by a subjective experience (1799). Maine de Biran was a close friend of the physicist Ampère (1775–1836), and corresponded with him on this point. In a letter around the end of 1805, Ampère replied: "*You confound the sense of effort with that of muscle sense. For me, the two things are absolutely different. When I move my arm, I attribute the muscle sensation to the arm. I feel effort in the brain and I report it as inside the head. It is this entirely internal and purely cerebral impression arising from the brain within the head or if you prefer reflected, and produced by the movement excited in the nervous fluid by a hyper-organic force and not by the brachial nerve, which constitutes the self.*" (Letter No. 263[3]).

This "centralist" position had a great influence and perhaps it owes some of its origin to Duchenne. Some time later, Freud's concept of the "ego" may well have been derived from this centralist notion with which he would have been familiar from his studies in Charcot's neurological unit. According to Scheerer (1987) the theoreticians of innervation sensations (sense of effort) share two common assumptions. The motor (or efferent) impulse originating in the brain (1) is available to consciousness, and (2) it somehow combines with afferent impulses to produce a given perceptual phenomenon.

3.2. "Muscular consciousness" of central origin in patients

An efferent sense based on "muscular consciousness" or a conscious efferent sensation of central origin was an idea that Duchenne fully elaborated and supported. He observed six patients, who had lost muscle sense, and were asked to perform voluntary

[3] *Vous confondez le sens de l'effort et la sensation musculaire; pour moi, ce sont deux choses absolument différentes. Quand je meus mon bras, je rapporte la sensation musculaire au bras ... Je sens l'effort dans le cerveau et je le rapporte ... à l'intérieur de la tête. C'est cette impression toute intérieure et purement cérébrale, ou si vous voulez réfléchie, produite par le mouvement excité dans le fluide nerveux par la force hyper-organique et non par le nerf brachial, qui constitue le moi.*

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limb movements (to open or close the hand, flex or extend the forearm). He noticed that these patients were unable to perform the instructed voluntary movement in the absence of vision, even though they felt that the movement had been performed. In his essay on co-ordination, Duchenne (1867, p. 779) spoke about the muscular consciousness: "*A motor ability independent of vision which is used to accomplish voluntary contractions. These abilities allow for the selection of which muscles to contract commanded by volition, reacting to encephalic drive so to speak. This capacity, which accomplishes voluntary movements and appears to precede the contraction, should not be confused with the sensation of weight or resistance etc., which was called muscle sense by Bell and which is the result of the contraction*".[4]

It is interesting to note that Charcot resumed Duchenne's ideas when he described cortical localisations and when he observed hysterical patients, explaining the respective roles of the nervous senses with a central origin, and of the muscular senses with a peripheral origin. He explained his theory of voluntary movement having two parts in a schema that he drew and named "the apparatus of the voluntary movement" (see Charcot's drawing, Fig. 5). In the first stage, there is a fundamental apparatus composed of the initial motor representation that necessarily precedes the execution of a voluntary movement. This takes place in the cortical motor centers which form the organic substrate and it is accomplished by motor control neurons within these centers. These centers are responsible for the "sense of innervation" derived from the "discharge of neurons". The second part of the schema corresponds to the "improving apparatus" (*l'appareil de perfectionnement*), with its two components, the sensitive or kinaesthetic center where data converges from the periphery (skin, muscles, aponeurosis, tendons, joint capsules), and the visual center, the seat of visual representations of movement.

Several reports from hemiplegic patients indicate that during recovery from a stroke, each movement required a greater effort than before the stroke. Ernst Mach (1938–1916), a physician who observed his own recovery from a hemiplegic stroke noticed that as he began to recover, each of his movements required a great effort, causing him to reflect on the sensation of motor innervation (Mach, 1886). Much more recently in 1973, Brodal (1910–1988), also a stroke victim, described a similar sensation of effort while recovering. Johannes Müller (1801–1858) suggested that we have a precise knowledge of the magnitude of the "nervous impulse" issued by the brain that is necessary to produce a movement (1838).

The fact that efferent motor impulses somehow combine with afferent impulses to produce a given perceptual phenomenon was illustrated by the reports on the sensation of effort during weight estimation. In 1846, Weber (1795–1878) compared the differential sensitivity for actively lifting an object and for similar weights being placed on a hand lying supported on a table (quoted by Scheerer, 1987). Active lifting resulted in a better discrimination, and this gave rise to the notion of a sense of force, derived from the sense of effort which could be localized in those parts of the brain which are subject to the action of the will. However, passive extension of muscles could also be used for the estimation of weight, suggesting an involvement of both efferent and muscular sensitivity in the sense of force.

To explain the stability of the visual world despite a moving retinal image by active eye movement, Purkinje (1787–1869) proposed the contribution of both efferent and afferent signals in the perception of movement as a result of his studies on vision (1825). From his experiments on gaze, he deduced the importance of motor commands in visual movement perception and concluded that signals representing active motor commands interact, in the central nervous system (CNS), with afferent signals. The same concept was further supported by Müller (1838) and von Helmholtz (1821–1894) who thought that apparent motion afterimages perceived during active eye (1867) movements were due to the perception of the internal motor signal called the "effort of will" (Scheerer, 1987). Like Purkinje, Helmholtz proposed that during normal vision the afferent retinal displacement signals and the effort of "will" cancel each other within the CNS. Wundt (1832–1920) also considered that active and the passive movements differed from each other with respect to their perceptual consequences (1863). There appears to have been a general consensus among all these scientists that the differences could be explained by the match or mismatch between the efferent impulses and the referent response.

The concept of the sense of innervation fell into disuse for a period of time until it was resurrected in 1928, by new support from von Uexküll (1909–1985) with his treatise on how the world of sensation integrates with the world of action. In the 1950's the concept of the sense of innervation was again reformulated when Sperry (1950) and von Holst and Mittelstaedt (1950) separately presented the idea that "outflow signals" (corollary discharge or efference copy) were especially important for the visuo–optomotor interactions. The core concept was that goal-directed motor commands interact with the afferent signal flow from the sense organs to form the perceptions within the CNS, which are the result of this interaction (see also Grüsser, 1986).

McCloskey et al. (1983) reexamined the sense of effort by the estimation of weight supported by the hand. He showed that the perception of weight was based on the sense of effort, derived from the centrally generated force supporting the weight. He also showed that proprioceptive afferents also took part in this evaluation. The perception of weight resulted from the addition or subtraction of the central effort for supporting the weight and the peripheral afferent impulses issued from the supporting muscle or from the antagonistic muscles. He also confirmed that in case of central lesion such as suffered by hemiplegic patients or cerebellar patients (see also Holmes, 1939, p. 8), or local paresis by local injection of curare, the sense of effort was increased, resulting in an evaluation of higher weight on the paralyzed or lesioned side.

Is the sense of effort alone able to provide a perceptual basis for force production? This interesting question was approached recently by Larfargue et al. (2003) in a patient deprived of proprioception, using a force matching task. The patient was asked to reproduce with one hand different levels of isometric forces achieved by the other hand. The results indicate that the patient successfully reproduced different levels of force exerted under initial visual control of the other hand in the absence of proprioception, on the basis of a perceived central effort related to force production in the other hand. Although the task of force reproduction was accurately performed, the patient did not refer to any perceived perceptual feeling that could be related to the muscle force level. The scaling of the force reproduction in the patient deprived of proprioception would appear to be unconscious.

Several investigations raised the question of the possible role of efferent signals in the sense of position. It was shown by Gandevia et al. (2006) in a wrist position matching task, that after exclusion of peripheral feed-back, and of any possibility of muscle contraction, realizing a sort of phantom limb, subjects when attempting to flex or extend the wrist, felt the wrist position changing by more than 20° in the direction of the attempted movement. These illusory position changes revealed that the sense of position after exclusion of all

[4] *Aptitude motrice indépendante de la vue, qui sert à l'accomplissement de la contraction volontaire. C'est elle qui, sans doute excitée par la volonté et réagissant à son tour sur l'encéphale, l'éclaire, pour ainsi dire, sur le choix des muscles dont il doit provoquer les contractions. Il ne faut pas confondre cette aptitude, qui dans l'accomplissement des mouvements volontaires, semble précéder et déterminer la contraction, ... avec la sensation qui donne le sentiment de la pesanteur, de la résistance etc., qui a été appelée sens musculaire par Bell ... qui est le résultat de la contraction.*

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sources of feed-back could be based on the sole central command addressed to the muscles. Surprisingly the 19th century views on "muscular consciousness" of central origin quoted by Duchenne (1867, p. 779) and by Charcot were very close to the sense of movement of central origin recently reported by Gandevia, using a much more accurate method for measuring this central effect.

3.3. Peripheral sensation of muscle sense

For several reasons by the end of the 19th century the theory of a central innervation sense was surprisingly neglected. The new psychologists were more preoccupied with a "peripheralistic determinism". William James (1842–1910), for example, thought that the principle of an innervation sensation was simply not required and he considered the central representation as superfluous and useless (1890). Müller (1850–1934) came to the same conclusion (1878). One of the arguments against the sense of effort in the case of lifting weights was that the perception was related not to the central sense of effort but to the expectation of weight. The memorized image in the brain of previous muscle sensations when lifting a weight was the source of reafferent expectation when the movement was to be performed.

Moreover, the discovery of the muscle spindles at this time gave further impetus to the peripheralistic view. Although muscle spindles had been mentioned by Wiessman and also observed by Khune, they were only well described by Ruffini (1864–1929) in 1889. Incidently, Babinski in 1886, independently discovered the muscle spindles and described them as curious structures in the muscles, and even though he did not specifically name them, he pointed out that they were a normal constituent of the muscle and not merely present in cases of amyotrophic lateral sclerosis.

The peripheral view was promoted in 1830 by Bell (1774–1842) who was the first to define the "sense of muscular action" as a sixth sense. Bell confirmed the importance of vision in motor pathologies (Bell, 1836). He recalled an army captain with locomotor ataxia "*who could feel the touch of a lady's petticoat on the calf of his leg*" but "*could not tell the position of his feet without looking at them.*" Landry (1826–1865), in 1852, called the afferent impulses from the muscles the sense of muscular activity ("*le sens de l'activité musculaire*") (Schiller, 1995), and several people supported the idea that the loss of co-ordination in progressive locomotor ataxia was due to the loss of muscle sense, or "*sentiment d'activité musculaire*" as proposed by Gercy and reported by Jaccoud (1864).

Duchenne rejected the idea that the muscle afferents could be the source of a muscle sense. He accepted the concept of a muscle sensitivity, but for Duchenne this was in no way comparable to a sense. Similarly, William James (1842–1910) did not believe that the muscle contraction by itself could provide the knowledge of the position or movement of the limbs (1890). This would depend on the joints which are sensitive and would inform on the direction of the movement and its amplitude. Goldscheider (1889) established that the afferents from the joint are the predominant if not exclusive source of sensations related to movement and relative position of the limbs. These reports were all against the peripheral concept of muscle sense as strictly depending on muscle afferents.

Duchenne (see Fig. 6), opposed the view that the muscle sense was the basis for locomotor co-ordination or that its loss accounted for locomotor ataxia. "*To write that voluntary motor coordination is subordinate to the integrity of muscle sense and by extension to the sense of touch is to profess the most regrettable heresy of physiology in its application to pathology*" (Duchenne, 1867, p. 773).[5] This

DUCHENNE, de Boulogne (1).

Fig. 6. Photograph of Guillaume Duchenne de Boulogne at the end of his life. UPMC. Bibliothèque Charcot.

was a criticism aimed at Jaccoud. "*Muscle sense or whatever other names we give it ... skin or joint sensitivity only assists in perfecting the faculty of coordination*" (Duchenne, 1867 p. 772).[6]

As by the end of the 19th century the theory of a central innervation sense was rejected, due to the dominance of the "peripheralistic determinism" in Psychology, it remained to reexamine the meaning of muscle sense. Bastian (1837–1915), provided the most complete description of the peripheral view (1888, see Jellinek, 2000). He coined the term of kinesthesis, which was thought to be of peripheral origin, and leaving central memory images within the kinesthetic (or visual) centres. According to this view movement initiation resulted from these unconscious images within the kinesthetic or visual centers. This was also the view of James (1890, Ch. XXVI): "*In perfectly simple voluntary acts, there is nothing else, in the mind but the kinesthaetic idea, thus defined, of what the act is to be*". What was the anatomical substrate for kinesthesis? In 1888, Bastian carefully defined kinesthesis in functional but not anatomical terms. Sherrington's experiments on the cat spinal cord (1900) helped to define proprioception with respect to exteroceptors and interoceptors, although his view of voluntary movement was based on a concept of integrated reflexes. In spite of the fact that his experiments with Grünbaum (Grünbaum and Sherrington, 1903) defined the exact location of the cortical motor center, Sherrington (1857–1952) consistently promoted the idea that the movement was the product of a chain of reflexes (Sherrington, 1910). Thus at the beginning of the XXth century, kinesthesis and proprioception had replaced the peripheral aspect of muscle sense. The anatomical background for kinesthesis and proprioception has been slowly clarified during the XXth century (see for an example Gandevia et al., 1983). Interestingly, in the early XXIth century, the

[5] *Ecrire que la coordination motrice est subordonnée, en tant qu'opération volontaire à l'intégrité du sens musculaire et accessoirement à l'intégrité du sens tactile c'est professer une hérésie physiologique on ne peut plus facheuse au point de vue de son application à la pathologie* (Duchenne, 1867, p. 773).

[6] *La sensibilité musculaire quelque nom qu'on lui donne ... la sensibilité de la peau et par dessus tout la sensibilité articulaire viennent en aide à la faculté coordinatrice; mais toutes espèces ou degrés de sensibilité ne font que perfectionner l'exercice de la faculté coordinatrice* (Duchenne, 1867, p. 772).

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hot discussions concerning the efferent (central) and afferent (peripheral) contributions to perception have been reactivated, and the present discussions are very similar to those one finds in the literature of the XIXth century.

4. Concept of synergy, from voluntary movements to locomotion

From his very precise analysis of the muscular contractions in locomotion, Duchenne deduced that co-ordination must be centrally controlled. From his study of the effects of single muscle faradisation, he concluded that a contracted muscle has little functional importance on its own. "*An isolated muscle contraction does not exist in nature*" he lectured (Duchenne, 1867) and that single muscle faradisation must be complemented by clinical observation in patients in order to understand how movements result from the compound action of several muscles. Duchenne's notion is strongly reminiscent of Hughlings-Jackson's enigmatic dictum: "*the central nervous system knows nothing of muscles, it only knows movements*" (Jackson quoted by Phillips, 1975), and is in direct opposition to the ancient view first promulgated by Galen. Duchenne argued for "*muscular associations*", the functional classification proposed by Jacobus B. Winslow (1669–1760), a French anatomist, who classified muscles according to their contribution to movement. Those muscles that effected the movements Duchenne called "*impulsive*" or "*principaux moteurs*" (prime movers according to Winslow, 1732), those that oppose the same movement he called "*modérateurs*" (moderators using the same term as Winslow). Finally, a third group, was identified as assisting the movement ("*les collateraux*" (collaterals) or "*muscles directeurs*" (directors according to Winslow)). This last group provided the direction of movement in some specific instances.

4.1. Voluntary movements

Duchenne considered that all voluntary movements, involved muscles with opposing actions. The "*impulsive*" and the "*moderators*", were activated together with the former producing the movement, the latter for moderating or arresting the movement. "*In all movements, muscles of the limb and trunk, which possess opposing actions contract simultaneously; the one to produce the movement, and the other to moderate its effect*".[7] As an example of the co-ordination between impulsive (agonists) muscles and moderator (antagonists) muscles, Duchenne cited the full extension of the hand and fingers. He showed that this extension was not due to the sole action of the finger extensors, but to a combination of muscle contractions including the finger flexors (acting on the first phalange), the interosseus muscles (which extends the second and third phalanges, but also flexes the first phalange[8]), and the palmar muscles which prevent dorsiflexion of the wrist due to the finger extensors (Duchenne, 1967, p. 764).

As we have seen, the claim by Duchenne (and others) that the agonists and antagonists could be co-activated during the same movement has been the subject of persistent controversy. In 1903, Beevor (1854–1908) in his third Cronian lecture agreed with Winslow's classification of "prime movers", "moderators" and "directors". He mentioned that Winslow who first proposed the simultaneous action of agonists and antagonists, had stipulated that the antagonists did not come into action when external forces (gravity or other forces) substituted for the antagonist muscle contraction. Georges Demeny (1850–1917), a collaborator of Etienne J. Marey (1830–1904) noticed that during natural elbow flexion or extension slow movements at uniform speed, there is co-contraction of antagonistic muscles (Demeny, 1924). In contrast, Sherrington (1906b) found that stimulating the motor cortex of monkeys always produced excitation of agonists and inhibition of antagonists introducing the idea of reciprocal innervation at the spinal level. In contrast, Tilney and Pike (1925), experimenting on anesthetized cats, and measuring the tension of antagonistic muscles around the ankle joint, observed that agonist and antagonist muscles form synergic units that are more often co-activated than reciprocally active when their tendons were freed from their insertions. Today most motor neurophysiologists would agree that the muscles synergies of reciprocal inhibition and co-contraction can both be observed depending on the movement. The predominance of one or other mode of muscle activation depends on the state of gravitational, inertial and visco-elastic forces which create the peripheral afferents interacting with central commands that together impinge on motoneurons.

4.2. Pathological analysis and locomotor synergy

From his observations on muscle atrophy or paralysis, Duchenne asserted, that muscle actions observed during voluntary limb movements were the result of combinations of coordinated activity similar to the more complex function of locomotion involving both a stance and a swing phase. "*There is a close analogy between the muscle synergies involved with vertical stance and those producing voluntary limb movements. The coordinated movements of the vertebral column, which precede and ensure standing derive from two principal phenomena; the first is the associated muscle activity that produces vertebral column extension, and the second, is the action of antagonist muscles that moderate and insure the normal attitude of the spine*".[9]

Duchenne, could not conceive that such a complex and precise function could emerge without the cooperation of a "coordinating faculty". In locomotion this co-ordination was for him the best example of a central organization which put into play ... "*the association of agonist and antagonist muscles*" (Duchenne, 1867, p. 759). He focused his attention on the leg oscillations during the second phase of gait and maintained, on the basis of his clinical observations that this phase was due to a synergy of hip, knee and ankle flexors. Interestingly, Duchenne contested the view expressed by Weber and Weber (1838), that the oscillation phase was due to a passive pendular movement of the leg. "*The leg could not possibly be able to oscillate under the cotyloid cavity when in extension; therefore, its three segments (thigh, leg and foot) are bent in relation to one another by the synergic contraction of those muscles which effect each of these movements and not according to the theory of MM Weber by the limb oscillating like a pendulum composed of segments of different lengths*" (Duchenne, 1867, p. 761).[10]

In his investigations of a pathological entity that he called " *Progressive locomotor ataxia*", which is better known as tabes

[7] ... *Tous ces mouvements des membres et du tronc résultent d'une double excitation nerveuse, en vertu de laquelle les deux ordres de muscles qui, par leur association, possèdent une action contraire ... sont mis simultanément en contraction, les uns pour produire ces mouvements, les autres pour les modérer* (Duchenne, 1867, p. 776).

[8] Duchenne refers to the muscles as interossei but his description clearly refers to the lumbricals.

[9] *Il existe une grande analogie entre les synergies musculaires mises en action, pendant la station verticale, et celles qui produisent les mouvements volontaires des membres. En effet, dans la coordination des mouvements de la colonne vertébro-crânienne, qui préside à la station verticale, on doit considérer deux ordres de phénomènes principaux : 1° L'association musculaire qui produit son extension; 2 L'harmonie des muscles antagonistes, qui modère et assure cette extension et l'attitude normale du rachis* (Duchenne, Physiologie des mouvements 1867, p. 768).

[10] *Ce membre ne pourrait osciller sous la cavité cotyloïde s'il était dans l'extension; c'est pourquoi ses trois segments (cuisse, jambe et pied) sont infléchis les uns sur les autres par la contraction synergique des muscles qui opèrent chacun de ces mouvements, et non par la seule action du membre oscillant considéré d'après la théorie de MM Weber, comme un pendule composé de segments de longueur différente* (Duchenne, 1867, p. 761).

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dorsalis, described in 1846 by Romberg (1795–1873), Duchenne fully expressed his opinion on the respective roles of central control as opposed to a peripherally based muscle sense in the co-ordination of limb movements during locomotion (Duchenne, 1872). Several times he described the symptoms (Duchenne, 1858) as the: "*progressive abolition of coordinated movements and apparent parises which is in contrast to the integrity of muscle force*".[11] He described patients who, when standing, presented difficulty in coordinating leg movements during walking, and disturbances of equilibrium during both stance and walking. Interestingly, these difficulties increased in darkness, indicating a compensatory influence of vision. Romberg, a German neurologist of the University of Berlin described tabes dorsalis in his "*Lehrbuch des Nervenkrankeiten des Menschen*", in 1846. This description was identical to the locomotor ataxia of Duchenne, with increasing leg and trunk oscillation in the absence of vision known as the Romberg sign (Romberg, 1846; Pearce, 2005), and was associated with a diminished "*muscle sense*" (see Schiller, 1995). Without a post-mortem pathology examination, Duchenne was unable to relate the locomotor ataxia to the degeneration of the dorsal column or dorsal root fibers, despite the fact that the English physician, Todd (1809–1860) had mentioned a lack of coordination of movements linked with the posterior column lesions as early as 1847 in "*the physiology of the nervous system*". However, after Charcot confirmed the dorsal root and dorsal column fibrosis in locomotor ataxia, Duchenne (1872, p. 660–661) accepted the idea that fibers in the dorsal columns could contribute to ataxia and that muscle and joint afferents play a role in coordinated locomotor function. However he noted that "*the deficits caused by locomotor ataxia are much greater when accompanied by a loss of vision than by a loss of muscle or joint sense. However, one would not suggest that locomotor coordination is dependent on vision. It is even less justified to say that locomotor coordination is dependent on muscle sense.*" (Duchenne, 1867, p. 777).[12]

From this premise arose the need to investigate the central origin of this coordinating faculty of locomotion. Initially, Duchenne thought that the cerebellum was the center for this coordinating faculty, because of the claim by Flourens (1824) that it was a center for motor co-ordination. However in 1864 he gave a short presentation of cerebellar nosology from which he concluded that "*Locomotor deficits caused by cerebellar lesions are similar to those of alcoholic intoxication ... They are characterized by a staggering dizzyness ... that is easily distinguishable from locomotor ataxia.*" (Duchenne, 1867, p. 788).[13] His observations on cerebellar patients were similar to those of patients with vestibular deficits, but no locomotor ataxia was seen. He concluded that the coordinating faculty, is not localized within the cerebellum. "*It is perfectly conceivable that a lesion of the dorsal columns and dorsal roots can disrupt coordinated locomotor function; but one must look higher in the nervous system, to the myelencephalon, for the real neural source of locomotor drive and coordination*" (Duchenne, 1867, p. 791).[14] Although Duchenne did not speak of a locomotor central pattern generator he assumed that a central organization was responsible for the multi-joint synergy which characterizes the flexion and extension phases of the limb.

Even though Charcot did not study human gait control himself, he devoted one of his famous Tuesday lessons (March 5, 1889), to an explanation of the organization of human locomotion (Fig. 3). For Charcot, this rhythmic behaviour was due to the co-ordination of two different central levels, one in the cortex and the other in the spinal cord. According to Charcot, the latter was the more complex, since it was able to coordinate the various different rhythmic mechanisms: "*The structures related to the execution of stance and locomotion each involve two centers or cell groups; one seated in the cerebral cortex and the other residing in the spinal cord... Without doubt, the spinal group is the more complex of the two, as it is responsible for the unconscious automatic execution of coordinated acts, whereas the relatively much simpler role of the cortical group consists of the voluntary issuing of commands to sometimes initiate, sometimes accelerate or decelerate and sometimes to completely arrest the activities of the spinal group. In other words, the psychological memories of casual acts needed to initiate or arrest locomotion reside in the former cerebral structure whereas the organic memory presiding over the details of the locomotor movement resides in the spinal cord*" (in Gasser, 1995, p. 99/100).[15] This fascinating presentation is the first scientific description of the central mechanisms able to induce the locomotor processes and the concept of two levels which is still very much present today.

4.3. Reflexes or central control of lomotion?

During the XIXth century, one major question was the role of sensory information in the control of movements. Since Jiri Prochazka's (1749–1820) demonstration that reflexes in the spinalized animal depended on sensory stimulation, the idea that voluntary movements were initiated by either actual or remembered sensory stimuli, dominated the neurological thinking during the 19th century. In his book "*The reflexes of the brain*", Yvan Sechenov (1829–1905) proposed that an actual or memorized sensory stimulation was at the origin of all movement (Clarac, 2005a). However, the effect of the sensory stimulus was thought to depend on the level of the brain on which the sensory stimulation was acting. This was proposed by J. Hughlings Jackson (1835–1911) who defined three levels in the central organization of the brain, a lower level, where automatic movements such as reflexes were organized (spinal cord and brain stem), a middle level including motor cortex at the origin of voluntary movements and a higher level corresponding to other cortical areas, responsible for higher functions such as speech. The middle and higher levels were more flexible than the lower automatic level, although they too could become automatic after repeated practice and learning (see Jackson, 1931).

At the Salpetrière, the position was not far from that of Jackson but while reflexes were a very useful tool used to analyse patient' pathologies, their role in movement control was considered more ancillary. Interestingly, Charcot's concept of how the brain controls

[11] *Abolition progressive de la coordination des mouvements et paralysie apparente, contrastant avec l'intégrité de la force musculaire.*

[12] *... les désordres occasionnés par l'ataxie locomotrice sont bien plus grands, par la perte de la vue que par la perte de la sensibilité musculaire et articulaire. Cependant il ne viendrait à l'esprit de personne de dire que la coordination locomotrice est subordonnée au sens de la vue. On était encore moins autorisé à dire que cette coordination locomotrice est subordonnée au sens musculaire* (Duchenne, 1867, p. 777).

[13] *Les troubles de la locomotion dans les affections cérébelleuses sont semblables à ceux de l'ivresse alcoolique ... Ils sont caractérisés par une titubation vertigineuse ... facile à distinguer de la titubation asynergique observée dans l'ataxie locomotrice* (Duchenne, 1867, p. 788)

[14] *On conçoit donc parfaitement que la lésion des cordons et des racines postérieures puisse troubler le fonctionnement de la coordination locomotrice; mais c'est plus haut qu'il faut aller rechercher la source de la force nerveuse locomotrice, le point du myélencéphale doué de la virtualité appelée faculté coordinatrice de la locomotion* (Duchenne, 1867, p. 791).

[15] *Les divers appareils relatifs à l'exécution des mouvements de la station, de la marche composent chacun deux centres ou groupes cellulaires différenciés dont l'un siège dans l'écorce cérébrale, tandis que l'autre réside dans la moelle épinière ... Le groupe spinal, le plus compliqué des deux, sans aucun doute, est chargé de l'exécution automatique, inconsciente des actes coordonnés pour l'accomplissement de chaque fonction ; tandis que le rôle relativement beaucoup plus simple du groupe cortical consiste dans l'émission volontaire des ordres prescrivant tantôt la mise en jeu, tantôt l'accélération ou le ralentissement tantôt enfin l'arrêt définitif des actes exécutés par le groupe spinal correspondant. Dans celui-ci, en d'autres termes, réside la mémoire psychologique des actes sommaires qu'il faut prescrire soit pour mettre en jeu l'appareil, soit pour en arrêter le fonctionnement, tandis que la mémoire organique, qui préside à l'exécution, dans tous leurs détails, des mouvements prescrits réside dans celui-là ...* (in Gasser, 1995, p. 99/100).

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locomotion was similar to Duchenne's in suggesting an organic memory that is in charge of the details of the movements analogous to the coordinating faculty of locomotion ("*faculté coordinatrice de la locomotion*"). Charcot made a further distinction between this coordinating processes located within the spinal cord, and the triggering system, "the volitional center" located at the cortical level and supervising the spinal coordinating process. However, in describing the role of this cortical region, Charcot's description seems closer to the mesencephalic locomotor region (MLR) defined by the Russian school in the mid 20th century (Shik and Orlovsky, 1976).

The early 20th century saw considerable controversy and discussion among physiologists about locomotor control. In 1905 Philippson (1877–1938) was the first to define the different phases of a single step: one flexion phase (F) and three extension phases (E1, E2, and E3), from his study of photographs of a dog walking, trotting and galloping taken at 100 frames/s by Marey. Considering the extensor and flexor reflexes involved in locomotor co-ordination, Philippson suggested that medullary centres were responsible for coordinating inter-segmental movements on the basis of a description of a spinal dog able to move its hind limbs spontaneously when lifted above the ground.

From his study of the step cycle in the cat, Sherrington, introduced a general theory of neurophysiology based on the integration of reflexes (1906b; 1910) that became the dominant view of the nervous system for much of the first half of the 20th century (Clarac, 2005b). Sherrington viewed reflexes as the basic units of neural function (Swazey, 1969; Stuart et al., 2001). He also believed (with perhaps a few reservations) that the reciprocal inhibition of antagonist muscles was the basis for almost all motor functions including Hughlings-Jackson's "voluntary" and "automatic" movements. Sherrington's reflexology was accorded a special reverence by the generation of neurologists that succeeded him. For example F.M.R. Walshe (1885–1973) said that in physiology *The Integrative Action of Nervous System* "holds a position similar to that of Newton's Principia in physics" (Gibson, 2001).

Still, this view was not without its opponents. Thomas Graham-Brown (1882–1965), a contemporary of Sherrington introduced an alternative to Sherrington's interpretation of cat locomotion based on his theory of "half centres", which postulated that the locomotor rhythmic activity was centrally organized (Graham Brown, 1911, 1914). As will be shown, the views of Duchenne and Charcot were close to those of Graham Brown. It required a full half century before Engberg and Lundberg (1969) demonstrated in intact cats that the onset of the extensor EMG activities invariably occured before the leg contacted the ground and therefore could not be the result of reflex action (Stuart and Hultborn, 2008). This critical observation helped to establish the role of central coordinating actions in the control of locomotion and subsequently Viala and Buser (1971) recorded spinal rhythmic activity without sensory feedback (fictive locomotion) in the rabbit. The Russian studies of treadmill walking in the decerebrate cat after the stimulation of the MLR combined with Grillner's experiments eliciting by stimulating a "central pattern generator" (CPG) in a spinalized and deafferented cat spelled the end of the reflex hypothesis of locomotion (Grillner, 1981). Today these CPGs are now considered as wiring diagrams for genetic programs that determine motor co-ordination (Clarac et al., 2004; Grillner and Wallen, 2004). Recently, Yuste et al. (2004) and Grillner et al. (2005) suggested that these assemblies of neurons are ubiquitous structures within the central nervous system (CNS) and that each such circuit has a particular function.

If the centralist view of the central pattern generator finally prevailed in locomotion, could the same conclusion be reached for the synergies associated with voluntary movements? The centralist view was supported by those utilizing movement analysis and EMG as a tool to explore the central mechanism for movement organization. Kurt Wachholder (1893–1961) was the first to show that in single wrist movements, the onset of movement was always preceded by an EMG activation or inhibition (1928). He thus showed that a central command always preceded the onset of movement. However, the EMG pattern during movement performance varied markedly according to the mechanical constraints associated with the movement, such as velocity, inertia etc. In order to explain the variability of the pattern, he claimed that the goal of the movement was central to the movement organization and to the co-ordination of its various central, reflex and mechanical components, which is an inherent property of the system involved in movement execution.

The prevalent role of the goal in movement co-ordination was also an important feature for Bernstein (1967), who insisted on the permanent role of sensory afferents, not as a source of reflexes, but as involved in a continuous updating of the central command according to internal or external messages occurring during the movement performance. In the introductory lecture to a Symposium on motor programming (1973), Fessard (1974) insisted on both aspects of movement organization, central and peripheral. "*The variety of influences that can initiate movement is certainly centrally determined, however, it is not immutable. Instead it is modified by the peripheral conditions available. We are dealing with a program elaborated as a function of necessity and not a sequence of actions imposed by rigid neuroanatomical circuits like a common reflex*".[16] To conclude this section both Duchenne and Charcot developed the concept of centrally organized muscles synergies exemplified by locomotion. However, they also recognized the role of sensory afferents, related to muscle sense and of vision for updating the performance.

5. The cerebellar asynergy

Let us now see how Babinski, in line with Duchenne's concept of motor co-ordination, developed the notion of "synergy" which he defined as "*the capacity to accomplish the variety of movements that constitute a single act*" (Babinski, 1899; Babinski, 1934, p. 197).[17] His definition extended beyond the simple muscle co-ordination in a given movement. Asynergia was the pathological counterpart of this capacity, and it was observed in cerebellar patients. His concept of asynergia was criticized and finally rejected by most neurological schools. It is only in recent years that a reexamination of Babinski's "asynergia" has begun, based on a series of new concepts in motor control. One was the necessity for the central command to accommodate the internal constraints of the musculo-skeletal system and the external forces such as gravity. This was first pointed out by Wachholder (1928) (see Wiesendanger, 1997) then later by Bernstein (1967) who insisted on the role of motor learning. Several theoretical models were proposed on how the controller could overcome the problem of internal and external constraints. As a result, the concept of asynergia, as proposed by Babinski in cerebellar patients, has been reconsidered as an important aspect of cerebellar pathology.

Given the 50-year difference in their ages it is unlikely that Duchenne and Babinski ever met. However, Duchene's concept of motor co-ordination (synergy) had a definite impact on Babinski, probably as a result of Charcot's influence. Although the term

[16] *La distribution des influx qui déclenchent le mouvement est bien centralement prédéterminée, mais n'est pas immuable et se modifie en fonction des conditions périphériques offertes au mouvement ». « Nous sommes bien en présence d'un programme élaboré en fonction du besoin et non de séquences d'action imposées par des liaisons neuroanatomiques rigides comme dans un réflexe ordinaire* (Fessard, 1974).

[17] *La faculté d'accomplir simultanément les divers mouvements qui constituent un acte* (Babinski, 1934, p. 197).

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'synergy' is still in use today, ideas about its meaning vary widely. About 30 years after the death of Duchenne, Babinski's observations on the movements of cerebellar damaged patients caused him to reassess how the brain achieves motor co-ordination. In spite of the medical training from Charcot and indirectly Duchenne, Babinski's description of "asynergia" in cerebellar patients was quite different from the concept of locomotor synergy first described by Duchenne. In fact, for Duchenne, locomotor synergy was a genetically fixed and prewired combination of muscle contractions for producing a given leg, hip, knee and ankle movement, for instance during the flexion phase of locomotion (see Paillard, 1960). Although the loss of co-ordination in locomotor ataxia (tabes dorsalis, first described by Romberg (1846) was Duchenne's prime example of asynergia, he did not feel this was in any way dependent on the cerebellum. Babinski's movement synergy deficits were related to cerebellar lesions and contrary to those described in locomotor ataxia, not influenced by vision (see Déjerine, 1914).

5.1. Babinski and cerebellar symptoms

Babinski was a highly observant and astute clinician, and perhaps his most brilliant clinical contribution, was in defining a set of signs, which are now considered the classical symptoms of the cerebellar syndrome (Fig. 7). His expertise in cerebellar pathology was recognized at the international congress of medicine in London, where his invited presentation outlining the signs of *dysmetria, asynergia* and *adiadochokinesis* received an enormous ovation (Babinski and Tournay, 1913). Unfortunately, Babinski's original and insightful observations on cerebellar patients were largely eclipsed by the vociferous, but not entirely well founded objections of Déjerine (1837–1915) and André-Thomas (1867–1963) in France, and Gordon Holmes (1876–1966) and Francis Walshe (1885–1973) in England.

Fig. 7. Photograph of Joseph Babinski. From Eugène Pirou. <www.whonamed-it.com/doctor.cfm/370.html>.

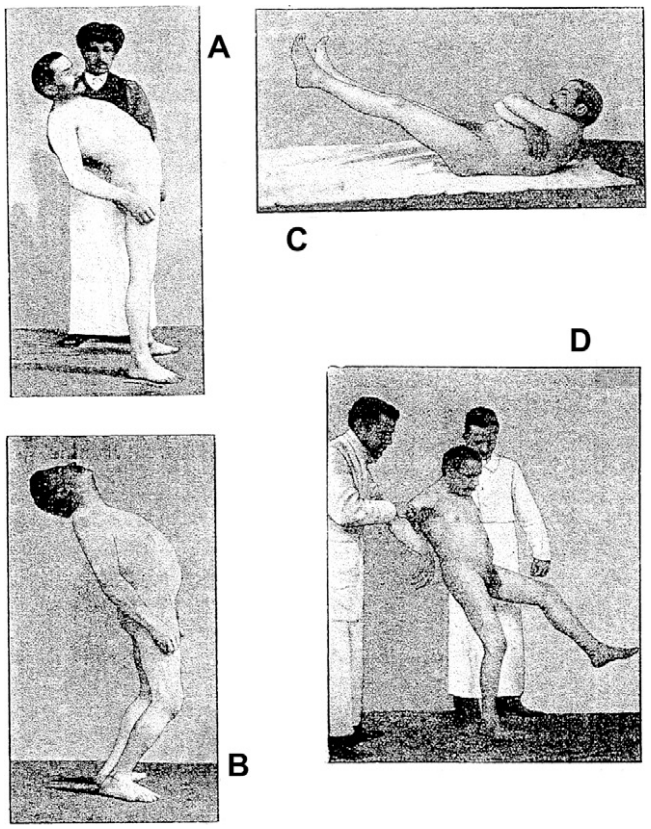

Fig. 8. Examples of asynergia presented by Babinski with the patient Henri Mounoulou. A: Postural asynergia compared with a normal patient (in B). C: The patient is unable to stand up. D: The patient needs to be supported by two nurses for walking from (Babinski, 1913, Figs. 15–18).

Babinski described *asynergia*, mainly based on two well-studied patients, the most frequently cited of which was Henri Mouninou. His post-mortem examination was reported at the April 23, 1925 session of the Academy of Medicine. The autopsy revealed "*a single lesion extending from the antero-dorsolateral pons to the medulla oblongata and penetrating deep into the white matter of the cerebellar hemisphere*" (Tournay, 1967, p. 78),[18] indicating a partially extracerebellar lesion. As reported by Déjerine (1913, 1926, p. 423): "*up to this point asynergia had been observed especially in patients with rather complex cerebellar and pontine lesions*".[19] Babinski (1899) illustrated asynergia with two examples shown in Fig. 8. The first was the failure of the trunk to lean forward at the onset of walking, which resulted in staggering or falling when the first step was initiated. A second example was the absence of a forward displacement of the hip and knee when the standing patient was asked to look upward by tilting the head and trunk backward. The absence of this postural adjustment also resulted in falling. These two synergies were evidently related to equilibrium control during movement and were illustrations of the anticipatory postural adjustments associated with movement described much later (see Massion, 1992; Horak and Macpherson, 1996). Interestingly, in contrast with the locomotor ataxia of Duchenne, where ataxia increases in the absence of vision, cerebellar asynergia was not exacerbated in the absence of vision. In his 1913 review paper on the cerebellum Babinski provided other examples of the lack of synergy (Babinski and Tournay, 1913). Based

[18] *foyer unique occupant la partie antéro-externe droite de la protubérance et du bulbe et s'enfonçant dans la substance blanche centrale de l'hémisphère cerebelleux correspondant.*

[19] *jusqu'ici l'asynergie a été surtout constatée chez des maladies atteints de lésions assez complexes du cervelet et de la protubérance.*

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on his previous observations of spastic hemiplegic patients, who presented a lack of synergy between leg extension and hip flexion on the hemiplegic side, when a supine patient was asked to sit up, he noticed that his cerebellar patients showed the same "asynergia" on both sides, and were unable to sit up from a supine position. The hip flexion was not associated with a leg extension (in this case the legs were raised above the bed). He also showed several examples illustrating the lack of simultaneous flexion of the hip and knee, for example when a supine subject was asked to place the heel next the buttock: the hip flexes first, and then the knee flexes with the heel striking the buttock, demonstrating the absence of a harmonious synergy involving the hip and knee flexors. As a result the main characteristic of asynergia is the dissociation of movements of different body segments. In his "Exposé de travaux scientifiques (1913)" Babinski explained in more detail the meaning of his term "*asynergie*" saying that it was not due to muscular weakness but rather to an absence of co-ordination in the successive muscular contractions. For example during locomotion: "*The immobility of the upper part of the body while the legs are moving forward cannot be attributed to muscle paralysis in which locomotion propels the trunk because the muscle force is maintained and the movement components are preserved*" (Babinski and Tournay, 1913 p. 197).[20] Babinski considered that *asynergia* and *adiadochokinesis* (*impairment in performing rapid successions of alternating movements such as pronation and supination of the hand*) respectively represented a breakdown in the spatial and temporal organization of movement synergies (Babinski, 1902).

5.2. Criticism of the concept of asynergy

The identification of asynergia as one of the main cerebellar symptoms was criticized by several authors. Déjerine, who ultimately became professor of neurology at the Salpetrière, and who was a close collaborator of André-Thomas, a well-known expert on the cerebellum, was one of the first to critique Babinski's concept of synergy. In his book on "Sémiologie des Affections du Système Nerveux", Déjerine (1914, 1926) noted that Babinski's description of the cerebellar syndrome was derived from a restricted number of patients "*none of whom had a lesion of the cerebellum proper but instead their lesions involved the medullary pontine region or tumours compressing the cerebellum*" (Dejerine, 1914; 1926, p. 416).[21] However in one comment that rather supported Babinski's view, André Thomas stated that "*the cerebellum assures body equilibrium by regularly associating the compensatory movements necessary to maintaining stability during movement execution*" (André Thomas, 1911, quoted by Dejerine, 1914; 1926, p. 412).[22] This is in agreement with the two observations made by Babinski in his paper of 1899, in which the absence of compensatory postural adjustments resulted in a loss of balance in cerebellar patients. However, Thomas suggested that the dysmetria (hypermetria) could be the cause of asynergia by disturbing equilibrium during movement performance. "*During walking the movements of the limbs, the trunk, the shoulders, neck and head must combine to maintain balance. If, due to a cerebellar lesion, each of the movements is executed in an unmeasured way, the patient is unable to coordinate them and because he is afraid, he will lose his balance, he walks slowly. As a result we see what role dysmetria plays in the loss of balance whether it is in walking or in stationary standing or whatever movement of the body. Asynergia can only be the consequence of dysmetria*" (André Thomas, 1911, quoted by Dejerine, 1914, 1926, p. 472).[23]

Gordon Holmes an English neurologist who treated many soldiers with traumatic head injuries in the battlefield hospitals in France during World War 1, made a systematic study of the effects of acute cerebellar injuries in 40 men. With Grainger Stewart, his chief resident, Holmes confirmed the existence of Babinski's adiadocokinesia, in which the patient was unable to realize a rapid succession of reciprocal hand movements like supination and pronation. In his original article in Brain in 1917, Holmes very succinctly reported finding Babinski's asynergia. However, later, he expressed some scepticism, perhaps after reading the comments of Déjerine and Thomas, so that in 1939, he stated that the term of "asynergie" in Babinski's original sense to signify a lack of co-ordination between wider groups of muscles, including those which should fix segments of a moving limb to be "*... unnecessary because it would include symptoms of different origin*".

After an extensive review of the earlier French literature on muscle activation and an intensive study of ankle antagonists in the cat, a well-known and highly respected American neurologist, Fredrick Tilney, proposed that muscles are functionally arranged in "*synergic units*" of antagonist pairs which were more frequently co-activated than they were activated reciprocally (Tilney and Pike, 1925). Furthermore, they suggested that the synergic activity of these units was controlled by the cerebellum although no mention of Babinski was ever made in this paper. At a joint meeting of the American Neurological Association and the neurology section of Royal Society of Medicine in 1927 Tilney and Pike's suggestion was vigorously opposed by both F.M.R. Walshe, and G. Holmes. Walshe objected that cerebellum could not be involved in the control of muscle synergies because Sherrington had clearly shown that this function was already performed by the spinal cord. Holmes felt that in his clinical opinion no such disturbance as *asynergia* of antagonist muscles existed in sufficient degree to account for cerebellar dysfunction. (Discussion reprinted in Brain, 50, pp. 377–390, 1927.) As a result, asynergia was not included in the listing of terms and definitions of cerebellar pathology reported by Walker and Botterell (1937, p. 330), nor was it mentioned by Dow in his chapter on the clinical symptomatology of cerebellar disorders (in Dow and Moruzzi, 1958). This failure to mention asynergia was again more recently noted by Fine et al. (2002).

In his review published in the "Revue Neurologique in 1958, on cerebellar syndrome, François Lhermitte (1921–1998) discussed Babinski's definition of asynergia as a specific cerebellar deficit. He developed the idea that the main symptom was the decomposition of movement, and that this resulted, as proposed by André-Thomas, from dysmetria and hyposthenia (weakness) of agonist muscles and hypersthenia (hyperexcitability) of antagonistic muscles. However, there is no evidence that either muscle group is in fact weak, but rather it is the onset of their activity that is delayed producing hypermetria and intention tremor.

"If we understand by the term synergy to be that neural organization which presides over a set of several muscles that accomplish an act, this function is certainly not included in the cerebellum.

[20] *L'immobilité de la partie supérieure du corps, pendant que le membre inférieur se porte en avant, ne saurait, en effet, être attribuée à une paralysie des muscles qui dans la marche impriment au tronc une propulsion puisque, la force musculaire étant conservée, les mouvements élémentaires peuvent s'accomplir* (Babinski and Tournay, 1913).

[21] *chez aucun des malades, le cervelet n'était altéré par une lésion destructive; il s'agissait de lésions bulbo-protubérentielles ou de tumeurs comprimant l'organe.*

[22] *le cervelet assure le maintien de l'équilibre en associant régulièrement les mouvements compensateurs, nécessaires à la stabilité pendant l'exécution des mouvements* (Déjerine, 1926, p. 412).

[23] *Pendant la marche, les mouvements des membres, du tronc, des épaules, du cou et de la tête doivent se combiner pour maintenir l'équilibre. Si du fait de la lésion cérébelleuse, chacun de ces mouvements n'est plus exécuté avec mesure, le malade n'est plus à même de les coordonner; il a peur de perdre l'équilibre, c'est pourquoi il marche lentement ... On voit donc quel rôle peut revenir à la dysmétrie dans la production de la déséquilibration, qu'il s'agisse de la marche, de la station debout ou d'un mouvement d'ensemble quelconque. L'asynergie peut n'en être que la conséquence* (André Thomas, 1911, quoted by Déjerine, 1913, p. 472).

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...however the cerebellum is indispensable to its correct execution" (Lhermitte, 1958, p. 447).[24]

5.3. Reassessment of Babinski's asynergia

Babinski's term asynergia fell into disuse until the concept re-emerged in the context of the particular role of cerebellum in motor learning (Ito, 1984, see also Kawato et al., 1987).

The renewed interest in Babinski's synergies was prompted by the idea that postural changes must "anticipate" the equilibrium disturbances in order to closely synchronize movements with their postural support. This was originally proposed by Walter Hess (1881–1973) who, in a theoretical note published in the Helvetica Physiologica Acta in 1943, proposed that two components were present in every voluntary movement: a teleokinetic or goal-oriented component and a postural or ereismatic component. He surmised that the postural component occurred in anticipation of the movement in order to allow the teleokinetic component to reach the goal (see also Stuart, 2005). Martin (1967) also suggested that posture adjustments were associated with movement in standing subjects. The anticipatory postural adjustments were actually first described by Gurfinkel and his coworkers in 1967 (Belinkiy et al., 1967). Gurfinkel was a pupil of Bernstein, and he developed the idea that during the execution of complex movements, such as raising the arm when standing, the mechanical interaction between segments caused perturbation of posture and equilibrium, which were compensated in advance by so-called learned anticipatory postural adjustments, characterized by leg muscle activation preceding the prime mover activation of deltoid (Belinkiy et al., 1967). Bouisset and Zatarra (1987) using accelerometers placed on the finger of the arm being raised and on several body axial and leg segments during a task of arm raising in standing subjects showed that the forward forces generated at the level of the center of gravity preceded the onset of arm raising and cancelled the backward forces generated at the shoulder level in reaction to the arm raising movement. When considering the first description of Babinski's (1899) asynergia during backward rotation of the head, the cerebellar patients, failed to make the necessary trunk inclination and forward hip displacement preceding the main movement. This demonstrated a lack of postural anticipation in movement performance comparable to the description of Belinkiy et al. (1967) and suggests a possible role of the cerebellum in storing these learned anticipations.

Second, Smith (1996) in a review paper, proposed the hypothesis that "cerebellum plays an important role in motor learning by forming and storing associated muscle activation patterns for the time dependent control of limb mechanics. By modulating the co-contraction of agonist–antagonist muscles ... the visco-elastic properties of joints can be regulated throughout movement..." Thach et al. (1992,1993) and Thach, 1996, reported "that cerebellar lesions impair compound movements more than simple, and suggested that the cardinal role of cerebellum is to combine through learning ... the elements of movements using the parallel fibers contacts on the long beam of Purkinje cells". Similarly, the concept of dynamic internal models of movements built up by learning was proposed by Wolpert et al. (1995). These models were used to simulate the dynamic disturbances caused by movement execution (direct dynamic models) and to anticipate the appropriate corrections (inverse dynamic models). Wolpert et al. (1995, 1998) suggested that these internal models might be stored in the cerebellum. Some confirmation is provided by the more recent investigations using fRMI by Imazu et al. (2003, 2004).

Of course, Gordon Holmes was correct in claiming that the term of synergy used by Babinski concerned functionally different actions. The trunk–limb synergy was aimed at preserving equilibrium during movement, the limb extension associated with hip flexion was aimed at providing a support allowing the supine subject to sit up, and the simultaneous flexion movement of several joints, such as hip and knee were used for the smooth performance of a multi-joint movement. This new view of the cerebellum as a center for storing the learned internal dynamic (and inverse dynamic) models used to anticipate the disturbances associated with movement performance gives new meaning to the concept of asynergia. The loss of "anticipatory" commands compensating in advance for disturbances of posture and equilibrium resulting from movements suggests a possible unifying mechanism for explaining the functional diversity of the various multi-joint synergies and their loss after cerebellar damage (see Massion et al., 2004). Babinski may be credited for noticing that a large variety of functionally different multi-joint movements were consistently disturbed in cerebellar patients. With his typical perspicacity for clinical observation Babinski showed that the anticipatory postural adjustments associated with movements were absent in cerebellar patients. To this specific cerebellar symptomatology, he bestowed the term asynergia. In contrast to the innate co-ordinations proposed by Duchenne de Boulogne, the synergies of Babinski were learned and stored in the cerebellum, and ultimately used to anticipate the perturbation of posture, equilibrium and movement trajectories associated with movement performance. In this sense, Babinski was really a prescient prophet for what could only be explained after a long empirical study of cerebellar function.

6. Conclusion

For three generations, Duchenne, Charcot and Babinski combined meticulous clinical examination, muscle stimulation and anatomo-pathological analysis to address three important issues. First, they grappled with the problem of how, in the absence of sensory feed-back, does the brain know what it is doing – what we refer to today as the phenomenon of corollary discharge. Second, they demonstrated the mechanisms of centrally organized muscle synergies in performing voluntary and locomotor movements. Finally they described the role of the cerebellum in organizing complex movement synergies.

Today the scientific discussions among the neurologists of the latter 19th century seem somewhat remote. Their arcane methods of investigation appear crude and simplistic compared to the elaborate technical methods used in contemporary neuroscience. However, the basic issues related to how voluntary movements are coordinated were fully appreciated by these historical figures. Moreover, these same issues are not that far removed from contemporary controversies about motor control. There is a natural tendency for the original seminal concepts to be neglected, but, despite extraordinary progress in neuroscience, recalling the origins of current notions provides a fresh perception of the basic issues unencumbered by current transient fashions. The views of these founding fathers of modern neurology and the manner in which they debated these scientific questions can be quite useful in highlighting contemporary issues that are otherwise obscure and difficult to analyse without the historical context of their earlier origins. An examination of the historical roots of current concepts of motor control should help redefine the basis on which contemporary hypotheses of motor control are based. We hope that by reviewing some of these early neurological contributions, modern concepts of motor control can be placed in an appropriate

[24] *Si l'on entend par synergie l'organisation nerveuse qui préside au jeu associé de plusieurs muscles en vue de l'accomplissement d'un acte, il est certain que cette fonction n'est pas incluse dans le cervelet. ... Le cervelet est indispensable à son exécution correcte* (Lhermitte, 1988, p. 447).

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historical context, and suitable attention can be paid to current fundamental issues. Interestingly, the description by Babinski of asynergia was rejected by most neurologists because the theoretical basis for understanding the mechanisms underlying the organization of cerebellar synergies had not yet been discovered. One had to wait for the discovery of the role of the cerebellum in motor learning and the development of biomechanical modeling of direct and inverse dynamics before giving a scientific basis to his concept.

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