Luciani's work on the cerebellum a century later

Ermanno Manni and Laura Petrosini

In 1891, Luigi Luciani published his famous monograph on the cerebellum and formulated his triad of the cerebellar symptoms: atonia, asthenia and astasia, which explained all troubles provoked by cerebellar lesions; later he added a fourth sign, dysmetria. In spite of the fact that it was advanced in a pre-electrophysiological period, Luciani's interpretation of the cerebellar role in many motor functions survives more than a century later and his terminology has entered the routine of the neurological examination. With the modern knowledge of cerebellar circuitries, we can state that Luciani rightly pointed out the role of the cerebellum in regulating postural tone and muscular force, and that conversely he was wrong in denying cerebellar influence in co-ordination of multi-joint movements and the somatotopic localizations in the cerebellar cortex and nuclei. In spite of this, Luciani's work represents a milestone in cerebellar physiology.

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IN 1891, Luigi Luciani published his celebrated monograph, 'Il cervelletto: nuovi studi di fisiologia normale e patologica'[1] ('The cerebellum: new studies of normal and pathological physiology') (Fig. 1), which was translated into German in 1893. This study really represents a landmark in physiological studies, not only because it describes new scientific discoveries founded on incontestable experimental observations and on documented and logical interpretations of facts, but also because it can be taken as a template for other physiological research on different topics.

Luciani's life

Luigi Luciani was born in Ascoli Piceno on 23 November 1840. After graduating in medicine from the University of Bologna he entered the Physiological Institute as assistant professor[2].

From March to November 1873 he spent a period in Leipzig, working under Professor Ludwig, one of the most outstanding European physiologists, who first introduced the graphical method into physiology. Here he was quite impressed by the bulk of scientific instruments in Ludwig's institute, especially compared to the poverty of Italian universities, and performed a series of experiments on the isolated heart of the frog[2].

In 1875, Luciani was appointed Professor of General Pathology at the University of Parma. Sometime later, Luciani, working with Tamburini, carried out his first important neurophysiological investigations concerning cerebral localizations, demonstrating also the cortical origin of epileptic seizures[3]. After two years spent as Professor of Physiology in Siena, Luciani moved to Florence where he had the opportunity of working in an Italian institute well equipped with scientific instruments. During this period, Luciani selected the best pupils in his lab, and succeeded in fanning their enthusiasm for experimental investigation. His research on cerebellar physiology was published in the monograph 'The cerebellum'[1] in 1891.

In 1893, he was appointed Professor of Physiology at the University of Rome. Here, because of the lack of scientific instruments and his poor health, Luciani stopped experimental research. He took advantage of this forced rest by writing his monumental handbook of physiology[4], to which he dedicated the last 25 years of his life. The success of the handbook was so striking that five Italian editions were published, followed later by Spanish, German and English ones. This success was due to the peculiar nature of this handbook, as it was not a mere sequence of independent and incoherent facts, but an entirety of phenomena and concepts, all linked together harmoniously to provide a synthetic framework simultaneously with precise details.

Luciani was appointed Rector of the University of Rome (1898–1899), then Senator of the Reign (1905). He died in Rome on 23 June 1919, following a long illness.

The monograph 'The cerebellum'

In his monograph on the physiology of the cerebellum[1] Luciani analyses all facets of experimental research on this topic. He describes the surgical methodology for performing cerebellar lesions, the observations of signs present in different periods following lesions, their interpretation, the single protocols of any operated animal accompanied by detailed expositions of surgery, objective examinations and histological results, the effects of simultaneous cerebellar and cerebral ablations, clinical reports of cerebellar patients, and finally some cases of cerebellar agenesias. This precious volume ends by advancing a new unitary hypothesis on cerebellar physiology which, through an analysis of the preceding literature, encompasses or rejects earlier theoretical positions.

The experimental part describes experiments carried out in dogs (*Canis familiaris*) and primates (*Macaca cynemolgus*). Surgical approaches include splitting the cerebellum along the vermis (hemicerebellectomies), total cerebellectomies and unilateral sections of the cerebellar peduncles. Cerebellar and neocortical lesions were also combined in the same animals.

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0166 - 2236/97/$17.00 PII: S0166-2236(96)10077-1

Luciani analysed very carefully the symptomatology induced by cerebellar lesions in the operated animals, and separated the phenomena observable in the first postoperative days from those observed successively. In particular, he divided the cerebellar symptoms into three periods: dynamic phenomena, deficiency phenomena and compensation.

According to Luciani, the dynamic phenomena, which characterize the initial period following a cerebellar lesion, represent a kind of functional exaltation of the adjacent nervous structures induced by postoperative oedema and irritation. When this period fades away, the actual deficiency phenomena can be observed; subsequently, the compensation by the spared hemicerebellum or by other nervous structures occurs and the symptomatology becomes progressively milder.

Unilateral cerebellar ablations

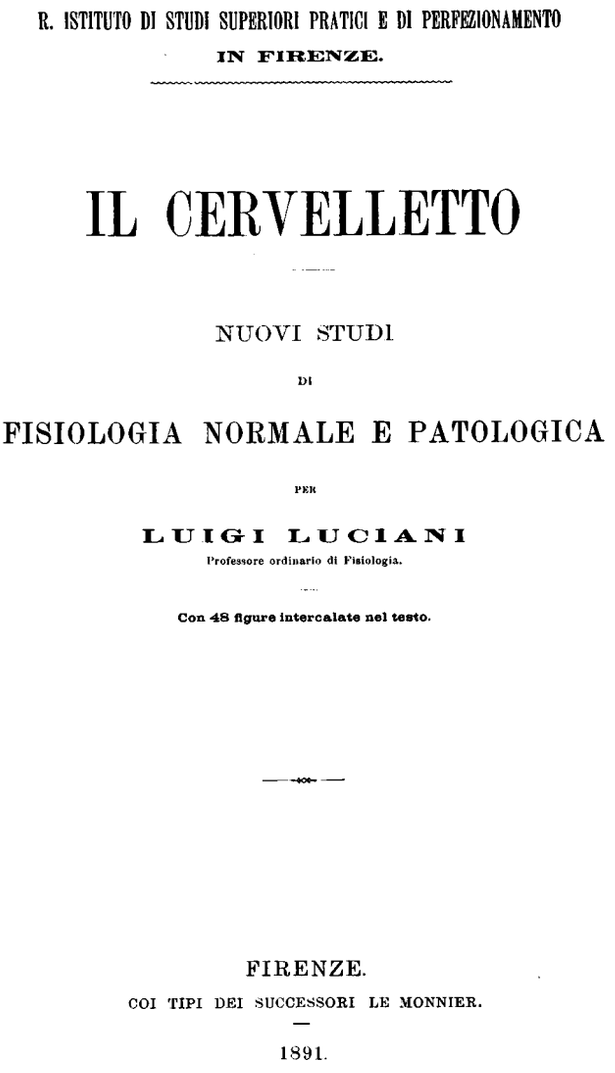
Luciani's theory was founded by considering in particular the phenomena exhibited by hemicerebellectomized animals in the deficiency period. He started from the conviction that a comparison between the symptoms of both sides, ipsilateral and contralateral to the operated side, was the same as comparing 'two animals of the same breed, age and constitution, one characterized by an almost normal cerebellar innervation and the other almost deprived of it'. This statement explains the discrepant results obtained by Luciani and other researchers, who developed their theories by analysing totally cerebellectomized animals in the compensation period.

According to Luciani's description, immediately after the lesion the hemicerebellectomized dog is unable to stand up and walk. It exhibits dynamic phenomena, such as head and trunk curvature toward the operated side (pleurothotonos), tonic extension of the ipsilateral forelimb, clonic jerks of the remaining limbs, head and trunk rotation around the longitudinal axis from the side operated on to the healthy side and eye nystagmus to the intact side. When attempting to move, the dog crawls on the buttock of the operated side and the main effort to move forward is carried out by the muscles of the intact side. When put into water, the dog is able to swim quite correctly, even if its trunk is slightly tilted towards the operated side.

Subsequently, the animal begins to stand up; however, the limbs of the operated side are so weak that the ipsilateral hindlimb seems paralysed. After 3–4 weeks, the extensor hypotonia decreases and the animal is able to stand up and walk awkwardly, although it often falls on the operated side. The deficiency period usually lasts several weeks.

Then, the compensation period begins. In hemicerebellectomized animals kept alive for more than one year, the intensity of cerebellar symptomatology decreases progressively, although it never disappears completely. In particular, an abduction of the ipsilateral limbs, which ensures enlarged support for body weight, is observed. Gait is clumsy and hesitant, characterized by wide-based support, with exaggerated movements of extension during the support phase and hyperflexion during the swing phase.

In experiments performed on primates, Luciani's results were similar to those obtained in the dog, the only difference being that the dynamic phenomena were much less relevant.



R. ISTITUTO DI STUDI SUPERIORI PRATICI E DI PERFEZIONAMENTO
IN FIRENZE.

IL CERVELLETTO

NUOVI STUDI
DI
FISIOLOGIA NORMALE E PATOLOGICA
PER
LUIGI LUCIANI
Professore ordinario di Fisiologia.

Con 48 figure intercalate nel testo.

FIRENZE.
COI TIPI DEI SUCCESSORI LE MONNIER.
1891.

Fig. 1. *Title page of the monograph on the cerebellum by L. Luciani (1891).*

Luciani's triad

On the basis of the results obtained during the deficiency period, Luciani advanced the existence of a triad of cerebellar symptoms, later known as Luciani's triad, composed of atonia, asthenia and astasia.

Atonia is characterized by a decrease in muscle tone in the limbs ipsilateral to the lesioned side. Because of atonia, the muscles of the ipsilateral limbs show reduced resistance to passive displacement. Sometimes, when standing up, the animal suddenly tends to fall on the operated side. Asthenia is expressed by weakness of the limbs on the operated side compared to the contralateral ones, provoking frequent falls during walking. While atonia is the expression of muscle weakness in postural tone, asthenia is the corresponding deficiency during voluntary movements. Astasia is characterized by head and trunk oscillations and tremors during movement. Some years later, Luciani[4], considering that movements of the hemicerebellectomized animal lost their normal measure and harmony, added a fourth symptom to the triad, dysmetria, that is, errors in the metric of movement: thus the ataxia results.

Total cerebellar ablations

Analysing the phenomena elicited by a total cerebellectomy, Luciani reports the presence of opisthotonos

in the dynamic phenomena period, that is a compulsive dorsal extension of the head, accompanied by extension of both forelimbs and clonic jerks in the hindlimbs. Eye nystagmus is also present. The animal is not able to stand up and walk. The successive deficiency period is characterized by marked weakness of limb muscles, provoking frequent falls on both sides. The animal begins to assume a compensatory wide-based standing position and exhibits marked astasia, characterized by a series of oscillations, with rhythmic bobbing of head and body. Both totally cerebellectomized animals and hemicerebellectomized ones retain their ability to swim.

When compensation occurs, to preserve balance in walking, lesioned animals exhibit an exaggerated abduction of the four limbs with a lowered centre of gravity; this causes them to collapse on their bellies, and they often cross their legs.

Compensatory phenomena

Luciani recognized two kinds of compensation: one exerted by the spared cerebellar structures, obviously present only in the hemicerebellectomized animal (the so-called organic compensation) and another represented by the bulk of voluntary movements carried out by the animal to overcome deficiency symptoms (functional compensation).

In this compensation he attributed a key role to the cerebral cortex, in particular, to the motor area. He was the first to observe that, in a hemicerebellectomized dog, deficiency symptoms can reappear when the contralateral motor areas of the cerebral cortex are removed. Luciani also noted that no compensation of the cerebellar symptoms occurs in an animal in which the cortical motor area has been ablated some months before cerebellectomy. Considering all observations together, Luciani reached the conclusion that the cerebellum has a 'tonic facilitating influence' on the motor cortico–spinal system.

What was right in Luciani's theory?

Luciani's investigations on the cerebellar functions attracted a large audience. The mere fact that Luciani was the first person able to keep cerebellectomized animals alive was in itself an event recognized as remarkable by the entire scientific world.

First of all, Luciani's findings eliminated some wrong ideas about the role of the cerebellum. Gall[5] was the first to uphold that the cerebellum could be the centre for sexual activity. Luciani demonstrated that this statement was groundless by reporting that, in his laboratory, totally cerebellectomized bitches were fertilized by dogs and that, on the other hand, totally cerebellectomized dogs fertilized bitches. In all litters, puppies were perfectly normal.

Other researchers[6] upheld that the cerebellum was also concerned with the general sensory sphere, and, in particular, that it was the centre of the muscular sense. It was easy for Luciani to show that cerebellectomy does not affect sensation.

Luciani's triad plus dysmetria were later confirmed in human subjects suffering from cerebellar lesions of different aetiology (tumours, abscesses, vascular accidents, etc.). Gordon Holmes[7] had the opportunity to examine patients who had sustained gunshot wounds in the cerebellum in World War I. In them he recognized all the signs described by Luciani in primates and in dogs, and described them using the terminology proposed by Luciani. The cerebellar patient exhibits typical postural disturbances, with a tendency to fall on both sides, when standing with his feet close together. He displays a drunken sailor's gait, characterized by wide stance and unsteady balance. The patient shows a peculiar form of tremor called intentional tremor, most evident at the end of a movement, when the greatest precision is required. Other symptoms such as adiadochokinesia and asynergia are present only in humans (see below).

Luciani's observations on dogs and monkeys were later confirmed in other animals, such as rats[8–10].

And what was wrong?

As with all really innovative discoveries, Luciani's findings gave rise to criticism as well as praise. One criticism concerned the surgical approach used by Luciani for the hemicerebellectomy; that is, splitting the vermis along the midline and then removing one half of the cerebellum. This 'anatomical hemicerebellectomy' induces some degenerations in the contralateral cerebellar deep nuclei and cortex remaining *in situ*. Di Giorgio and Simonelli[11] proposed removing only one cerebellar hemisphere, without affecting the vermis. This approach, called 'functional hemicerebellectomy', elicits a milder symptomatology. Sectioning the three cerebellar peduncles does not strictly reproduce the anatomical and behavioural effects of the anatomical hemicerebellectomy either, because of crossing the ventral spinocerebellar tract and Russell's uncinate bundle.

Another criticism concerns the subdivision of cerebellar symptomatology. The dynamic phenomena, considered by Luciani as an expression of a generic irritation, were interpreted subsequently as true deficiency phenomena, particularly in the case of forelimb extension. Later investigations[12,13] demonstrated that this symptom depends on ablation of the anterior lobe. Thus, it seems correct[14] to consider a first period of unstabilized deficiency, encompassing the dynamic phenomena and deficiency periods, followed by a stabilized deficiency period, corresponding to Luciani's compensation period.

As already pointed out, following cerebellar lesions primates and humans exhibit marked asthenia and atonia. However, in cats and dogs some researchers[15,16] observed clear signs of hypertonia and hypersthenia, such as the presence of the magnet and positive supporting reactions, both interpreted as an exaggeration of postural reflexes, corresponding to the exaltation of tendon reflexes described by many researchers in man and animals following cerebellar lesions. Luciani also reported occasional fits of limb extension in totally cerebellectomized dogs. This discrepancy arises from the fact that Luciani referred to observations made in hemicerebellectomized dogs in the deficiency period, while his opponents carried out their observations on totally cerebellectomized animals in the compensation period. Localized lesions of deep cerebellar nuclei showed that in the cat destruction of one fastigial nucleus provokes ipsilateral atonia, and bilateral ablation of fastigial nuclei elicits bilateral hypertonia[17], thus raising the new question of the role played by cerebellar nuclei not only in controlling muscle tone, but in general cerebellar functions.

The importance of the cerebellum in maintaining muscle tone, first recognized by Luciani, was successively

demonstrated to be exerted through gamma motoneurone innervation[18–20]. Thus, cerebellar hypotonia was interpreted as due to the loss of linkage between gamma and alpha motor activities.

Luciani only occasionally carried out circumscribed cerebellar lesions. Wrongly, he did not believe in the existence of a functional localization within the cerebellum. The only subdivision he considered was that the vermis was related to the trunk, and the hemispheres controlled the limbs. Subsequently, some researchers were able to demonstrate a somatotopic localization relative to limb musculature in the cerebellar cortex[14,21–24] and nuclei[25], as well as a somatotopic organization of all spinal and cerebral afferent projections[26,27]. The anatomo-comparative investigations by Bolk[28], Comolli[29], Ingvar[30] and Larsell[31] also made a great contribution toward the recognition of the somatotopic organization of the cerebellar cortex. Recently this view has been complicated by results obtained with single-cell recordings (see references cited in Refs 14,32,33). These investigations revealed multiple representations of the same part of the body in different localizations, an arrangement referred to as fractured somatotopy, certainly much too advanced for Luciani's knowledge and available technology!

Luciani's theory is unacceptable today also with regard to the co-ordination of movements. Luciani advanced his theory on cerebellar function when the scientific world was divided between Rolando's[34] and Flourens'[35] supporters, the former ones considering the cerebellum as a motor organ, while the latter ones advancing the idea that the cerebellum could co-ordinate muscular activity. Luciani persisted in denying a cerebellar role in muscle co-ordination, considering that, because cerebellar-lesioned dogs swim correctly with perfect co-ordination, the triad was sufficient in explaining all observed cerebellar symptoms. Thus, it was not necessary to call into action the property of co-ordination, 'a fictitious entity, obscure, imperfect and unintelligible'.

Luciani's statement was taken up again by Holmes[7], who maintained that atonia, astasia and asthenia could explain all movement irregularities constituting cerebellar ataxia. It was necessary to wait for Babinski[36] to demonstrate the existence of decomposition of the movement in cerebellar patients, which he called asynergia.

Recent work carried out by making separate lesions of cerebellar nuclei has clearly shown a loss of co-ordination in different muscle groups involved in multi-joint movements[37]. Definitively repudiating Luciani, Thach[37] and collaborators reached the conclusion that muscular co-ordination is an important function of the cerebellum, attributing a key role in it to the long extension of the parallel fibres: 'The parallel fibre appears optimally designed to combine the actions at several joints and to link the modes of adjacent nuclei into more complex co-ordinated acts', arguing that such a mechanism requires the adaptive capacity of the cerebellar cortex, specialized in combining simple elements of movement into more complex synergies.

What 'The cerebellum' and the cerebellum do not take care of

When Luciani wrote his monograph, a number of other functions were attributed to the cerebellum. In spite of often unacceptable language due to its aspecificity and occasional roughness, there are some hints in the literature of the possible contribution of cerebellar networks to what are today called cognitive functions. Luciani writes:

> 'Although the cases illustrated here do not allow considering the cerebellum as the site of any psychic function, it cannot be excluded that both irritative state and functional deficiency can modify the way of being and functioning of psychic centres, so that a noticeable change in character is elicited. The ultimate solution of such a delicate and ticklish topic will derive from clinical observation rather than from experimental research.'

This cautious position has been drawn upon from time to time, but no successive investigation has been definitely encompassed in the unitary theory of cerebellar functions. Only recently, a century after Luciani's work, has positive evidence been shown of a cerebellar contribution to cognitive functions (namely: language, timing, sequential behaviours, spatial analysis, attentive processes), in man as well as in animals, and the participation of the cerebellum in higher cortical functions has remained incontestable[38–41].

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Acknowledgements
This work has been supported by CNR and MURST grants.

Towards a European forum for the neurosciences

Wolf Singer

In 1994, *TINS* published a brief analysis of the problems faced by the European neuroscience community in its attempt to meet the challenges associated with the proclamation of the European 'Decade of the Brain'. Since then numerous initiatives have been taken by national and European neuroscience societies with the common goals of improving communication among European neuroscientists, increasing the visibility of the neurosciences as an autonomous and increasingly important domain of research in the appreciation of national and European granting bodies, and facilitating integration of colleagues from Eastern Europe into the international scientific community. These initiatives have led to major changes in the organization and self-apprehension of national and European neuroscience societies.

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SEVERAL YEARS AGO, the European Neuroscience Association (ENA), the owner of the *European Journal of Neuroscience* and the only multidisciplinary, supranational European society for neuroscience, offered its infrastructure to the European neuroscience community to be used to promote co-ordination (see Ref. 1). It was recognized that Europe needs a forum that is accessible for all neuroscientists both from within Europe and from other continents. The annual meeting in North America of the Society for Neuroscience has all of the scientific and political features that one expects to be fulfilled by a large interdisciplinary conference. However, a number of reasons argue in favour of an additional European forum. Not all European students can afford to attend the meeting in North America, European granting agencies are not represented at the meeting, and contacts required for the exploitation of the European job market are difficult to establish there. This latter point is becoming increasingly important because funding of neuroscience through the European Commission (EC) requires the establishment of European networks and partner projects that can be structured more easily on the basis of a European forum rather than a world congress. Finally, the rapid proliferation of national neuroscience societies and European oligodisciplinary associations calls for some co-ordination. To this end, the ENA approached the European neuroscience societies and proposed that the annual meeting of the ENA be transformed gradually into a truly multidisciplinary forum. After internal discussions within the respective societies this proposal has been approved by the representatives of European neuroscience societies at the meeting in Amsterdam in 1995, and concrete steps towards closer co-operation were reported at the Strasbourg meeting in 1996.

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ENA reorganization

In order to prepare the grounds for the European neuroscience forum, the ENA has introduced the following modifications in its organization. To broaden the scope of the scientific programme and to facilitate input from European societies a Programme Committee has been created that operates independently of the ENA Council and is responsible for the scientific organization of the meetings. Currently, the Programme Committee comprises 15 members: five delegates from national neuroscience societies, five delegates from the ENA Council whereby one represents the European Brain and Behaviour Society (EBBS), and four members nominated by the independent Chairman of the Programme Committee. The members of the present Programme Committee are listed in Table 1. This Committee is in charge of co-ordinating the scientific frame of the 3rd Meeting of European Neurosciences that will be held in Berlin, 27 June–1 July 1998. In order to ensure a broad representation of disciplines, members of the neuroscience community are now requested to put forward proposals for symposia, special interest sessions, European network conferences, technical workshops and satellite symposia that they, or their organizations, wish to organize at the Berlin meeting. Proposals should be sent to the present chairman of the Programme Committee, Professor Barry

  0166 - 2236/97/$17.00 PII: S0166-2236(96)01014-4