PHYSICAL SIGNS

The Babinski sign

J W Lance

J Neurol Neurosurg Psychiatry 2002;73:360–362

Babinski's life and the story of the Babinski sign are summarised. The physiological basis of the sign is discussed.

THE NAME

Of all neurologists whose name is commemorated in daily usage, that of Babinski may not exceed Romberg in frequency but overshadows all in its dramatic impact and clinical implication. Wartenberg was said to evoke his name in rejecting compromise with an emotive "By the great Babinski, no!".[1] It seems to matter little that Remak first described the extensor plantar response in a patient with transverse myelitis in 1893[2]:

> "One is able, through stroking of the distal half of the plantar aspect of the metatarsus primus, to evoke a fairly isolated reflex of the extensor hallucis longus."

The sign is generally attributed to a lesion of the pyramidal tract. It is of interest to look back on the diagnosis of pyramidal lesions before Remak and Babinski.

THE TRACT

In about 150 AD, Aretaeus of Cappadocia made the following perceptive observations[3]:

> "If, therefore, the commencement of the affection be below the head, such as the membrane of the spinal marrow, the parts which are homonymous and connected with it are paralysed; the right on the right side and the left on the left side. But if the head be primarily affected on the right side, the left side of the body will be paralysed; and the right, if on the left side. The cause of this is the interchange in the origin of the nerves, for they do not pass along on the same side, the right on the right side, until their terminations; but each of them passes over to the other side from that of its origin, decussating each other in the form of the letter X."

And there the matter rested for some 1700 years.

van Gijn, in his definitive monograph[4] and article,[5] summarised the current knowledge of the pyramidal syndrome before the recognition of the extensor plantar response. Knee and ankle jerks, clonus, withdrawal of the lower limb in response to pain and diminished cutaneous reflexes on the affected side in hemiplegia had been recognised. In the second (1893) edition of his textbook, Gowers[6] describes "rigidity" of the limbs after a lesion of the pyramidal tracts with increased tendon jerks and clonus on the affected side. In the third edition of *Diseases of the Nervous System* in 1899 (three years after Babinski's report), Gowers[7] mentions "clasp-knife rigidity," but not the extensor plantar response.

THE MAN

Joseph Felix Francois Babinski was born in Paris of Polish parents in 1857, two years after his brother Henri, with whom he was destined to spend the greater part of his life.[4] In 1879 he was appointed to a general medical position as "interne des hôpitaux," during which time he published anatomical studies on the muscle spindle and the pathology of multiple sclerosis. In 1885 he became "chef de clinique" to Jean-Martin Charcot who had become the first professor of neurology in France in 1882 at La Salpetrière, a gunpowder factory in the 17th century that evolved into an asylum and then a hospital, becoming one of the world's great centres for the study of neurological disease.

In 1890 Babinski passed the examination for "Médecin des Hôpitaux" and the way appeared clear for a career in academic neurology. The next step would be an associate professorship (professeur agrégé) but this was not to be because of what appears to have been an act of professional jealousy by Charles Bouchard. Bouchard was trained by Charcot and their names are linked together as "Charcot-Bouchard aneurysms" preceding cerebral haemorrhage in hypertensive patients. After Bouchard became a professor of pathology in 1879, his relationship with Charcot deteriorated.[8] It may have been coincidental that Bouchard was presiding over the examination for professeur agrégé when Babinski was an unsuccessful candidate, whereas three of the five who passed were pupils of Bouchard.[8]

Babinski never attempted the examination again. In 1895 he became chief of service at the Hôpital de la Pitié which adjoins the Salpetrière, and remained in that post until he retired in 1922 at the age of 65. He wrote on a wide variety of topics and his fame attracted neurologists from overseas including S A K Wilson, C G Chaddock, and Robert Wartenberg.[8] He provided a stimulus to neurosurgery, particularly in reporting the successful removal of intracerebral tumours and the localisation of spinal cord tumours on clinical grounds. He encouraged some of his pupils, including Clovis Vincent, to become neurosurgeons.[9]

His manner was austere and his clinical practice was weird by present day standards. Patients entered into his consulting room naked

Correspondence to:
Professor J W Lance,
Wales Medical Centre,
66 High Street, Randwick,
NSW 2031, Australia;
jimlance@bigpond.com

Received 27 February 2002
Accepted 3 May 2002

Figure 1 Botticelli's Virgin and Angels. (From Lance JW, McLeod JG. *A physiological approach to clinical neurology*, 3rd ed. London: Butterworths, 1981:143.)

and, after a skimpy history, were subjected to physical examination. van Gijn[4] recounts the story that a male patient, when he was dismissed after his head had been examined with the aid of a galvanometer, pointed at his penis and asked plaintively "you don't have anything to make it work again?".

Babinski lived with his brother Henri, a mining engineer and an inspired chef whose *Gastronomie Pratique* ran to nine editions and whose skill doubtless contributed to the impressive bulk of both men. Babinski continued to attend the hospital for consultations after his retirement.

He died in 1932, a year after Henri.[4]

THE SIGN

In 1896 Babinski presented a brief paper to the Biological Society of Paris, translated as "On the cutaneous plantar reflex in certain organic disorders of the nervous system".[4] He had observed that pricking of the sole on the healthy side of a patient with hemiplegia or lower limb monoplegia caused withdrawal of the lower limb with flexion of the toes on the metatarsal bones. In contrast, the same stimulus applied to the sole on the affected side caused extension of the toes at the metatarso-phalangeal joints, even in patients who were unable to move their toes voluntarily. He later referred to "*stroking*" of the sole rather than pricking as the adequate stimulus, a point that should not be lost on today's registrars or residents, many of whom warn their patients that they are about to *scrape* their feet or use some other potentially intimidating term.

Babinski's definitive description appeared in 1898.[10] An English translation is included in van Gijn's monograph.[4] He stated that the "phenomenon of the toes" is most easily elicited from the lateral aspect of the sole and that the reaction of the anatomical extensor is most conspicuous in the first or the first two toes. He demonstrated the extensor response in hemiplegic and paraparetic patients and stated that he had observed it in Friedreich's ataxia. He attributed the sign to dysfunction of the pyramidal tract and pointed out that it was usually associated with exaggeration of tendon reflexes and clonic movements but that "this relation is far from indissoluble." He concluded by drawing attention to the presence of the sign in the newborn, an association that had not escaped the attention of a renaissance artist (fig 1). The reversion of the plantar response to flexor occurs at variable times from the age of seven months to a year or more. The results of many publications have been tabulated by van Gijn,[4] who concluded that the relation of this change to the onset of walking was probably indirect. Confusion may arise in infants because the grasp reflex, which is most easily elicited by stimulation of the ball of the foot, involves flexion of the toes.[11] It usually disappears between six and 12 months of age and appears to be related to the age of standing.[11]

Babinski had noted that the sign appeared transiently on the affected side in a man during a Jacksonian fit and bilaterally in another patient suffering from strychnine poisoning. I had the opportunity to observe such a brief alteration in a child of mine subject to night terrors. As I was attempting to comfort her I ran my thumb lightly against the lateral aspect of her sole as one does and observed a definite Babinski response. As soon as the paroxysm was over the response reverted to flexor.

In 1903, Babinski remarked during a case presentation that abduction of the toes might accompany extension of the toes in a pyramidal lesion but was by no means constant.[12] This was later known as "le signe de l'éventail" (the fan sign).

THE CAUSE

The normal plantar response to cutaneous stimuli of the sole can be considered a superficial reflex like the abdominal and cremasteric reflexes that are abolished by an upper motor neurone lesion.[4] It is then replaced by the Babinski response. The upgoing toe is regarded anatomically as extension of the great toe but physiologically it is part of a flexor reflex, apparently disinhibited by loss of upper motor neurone control, and its receptive field may extend in some instances to the leg or thigh.[13 14] This led to the description of many "reflexes" such as Chaddock's and Oppenheim's signs which were simply different ways of eliciting the Babinski sign.[15]

Although the sign usually accompanies spasticity, and has been described as being caused by infarction apparently limited to one medullary pyramid in three cases cited by van Gijn,[4] its causation by lesions of the pyramidal tract has been questioned. Nathan and Smith[16] studied patients before and after operations on the spinal cord (anterolateral cordotomy), correlating clinical findings with the extent of the surgical lesion. They found that destruction of the anterior half of the spinal cord may be associated with a Babinski response, whereas the sign could be absent with histologically verified lesions of the lateral corticospinal (pyramidal) tract. Later, Nathan[17] reported 44 patients subjected to cordotomy for relief of pain from cancer. The Babinski response was found in general to be present after lesions of the corticospinal tract and not with lesions elsewhere in the cord. Nevertheless, a transient Babinski response could be observed after anterior lesions and some patients with lesions of the tract retained normal plantar responses.

Landau and Clare[18] considered that the patients with pyramidal lesions that they studied who did not develop extensor responses had peripheral nerve damage or were recovering from shock. They felt that the correlation between the sign of Babinski and pyramidal tract dysfunction was significant but added that it would be "absurd to deny that lesions of non-pyramidal internuncial pathways may facilitate release phenomena at the spinal level." What evidence is there for this proposition?

There is an important difference in comparison with the decerebrate spinal cat that throws some light on the matter. In the decerebrate cat the stretch reflex of the quadriceps muscle becomes more active as the degree of stretch (that is, muscle length) is increased.[19] In contrast, in those chronic spinal animal preparations with increased muscle tone, the reflex response of the quadriceps becomes progressively less as the degree of stretch is increased, analogous to the clasp-knife response in human spasticity. As the reverse applies to flexor muscles (that is, increasing muscle stretch *enhances* the reflex response) it appears that receptors sensitive to stretch, such as group II afferent fibres, inhibit the stretch reflex of hind limb extensors and facilitate that of the flexors as long as the stretch is maintained. These flexor reflex afferents (FRA) are normally suppressed by the dorsal reticulospinal system which arises from the pontomedullary reticular formation and descends in the dorsolateral funiculus of the spinal cord.[20] Burke *et al* made discrete lesions in the reticular formation and upper quadrantic sections in the spinal cord of the decerebrate cat,[19] which transformed the length dependent facilitation of decerebrate rigidity into the length dependent inhibition of spinal spasticity. These changes observed experimentally can readily be applied to the human situation.

Using the H reflex as an indicator of motor neurone excitability in spastic patients, it was shown that stretch of the calf muscles *diminished* the amplitude of the H reflex recorded from the calf while stretch of the pretibial flexor muscles *augmented* the H reflex recorded from those muscles[21]—that is, the flexor reflex afferents have been released in spastic patients, as in the chronic spinal cat. This explains the clasp-knife phenomenon in human spasticity, in which the tonic stretch reflex in quadriceps is inhibited by increasing muscle length beyond the mid-point of knee flexion. It also explains the enhancement of the flexor protective response, including the Babinski sign. In cats the inhibitory reticulospinal pathway is directed from the motor cortex by parapyramidal fibres that descend in the medial part of the internal capsule and medial area of the midbrain dorsal to the cerebral peduncle.[22] If one can extrapolate to humans from these studies in the cat, any disturbance in function of this cortico-reticulospinal tract, which is closely applied to the pyramidal tract throughout its course from cortex to spinal segmental levels, would release flexor reflexes, including the Babinski sign.[23]

THE LEGACY

It thus appears that the Babinski sign is an indication of withdrawal of supraspinal control of flexor reflexes in the lower limbs. Clinically it can be equated with inactivation, transient or permanent, of the upper motor neurone, which term implicates corticoreticulospinal fibres as well as the pyramidal tract anywhere in its path from cerebral cortex to termination in the cord.

Flexor reflexes are prominent in the newborn and young infant. In order for the baby to stand, flexor reflexes in the lower limbs must be brought under control by the dorsal reticulospinal tract. For the baby to walk the flexor synergy must then be harnessed to the motor cortex as part of the walking pattern. With the maturation of upper motor neurone pathways the Babinski sign disappears and the great toe goes down on stimulation of the sole. If upper motor neurone control is temporarily suspended, as in an epileptic fit, or is abolished by disease the Babinski sign reappears.

There is a tendency to abolish eponymous names for signs or clinical syndromes but they do serve to remind us of the debt we owe our forebears. Should we abandon the term Babinski sign in favour of "the upgoing toe" or "extensor plantar response"? I would side with Wartenberg in saying: "By the great Babinski, no!".

REFERENCES

1 **Aird RB**. Obituary: Robert Wartenberg. *Neurology* 1957;**7**:146–7.
2 **Pearce JMS**. Babinski or Remak? *J R Coll Physicians Lond* 1996;**30**:190.
3 **Adams F**. *The extant works of Aretaeus the Cappadocian.* London: Sydenham Society, 1856:306. (Reprint, Boston: Milford House, 1972.)
4 **van Gijn J**. *The Babinski sign – a century*. Utrecht: Universiteit Utrecht, 1996.
5 **van Gijn J**. The Babinski sign: the first hundred years. *J Neurol* 1996;**243**:675–83.
6 **Gowers CR**. *A manual of diseases of the nervous system*, 2nd ed. Philadelphia: Blakiston, Son and Co, 1893:83.
7 **Gowers WR**. *A manual of diseases of the nervous system*, 3rd ed, vol 1. London: Churchill, 1899:255–65.
8 **Iragui VJ**. The Charcot–Bouchard controversy. *Arch Neurol* 1986;**43**:290–5.
9 **Lanzino G**, di Pierro CG, Laws ER. One century after the description of the "sign": Joseph Babinski and his contribution to neurosurgery. *Neurosurgery* 1997;**40**:822–8.
10 **Babinski J**. Du phénomène des orteils et de sa valeur semiologique. *Semaine Médicale* 1898;**18**:321–2.
11 **Dietrich HF**. A longitudinal study of the Babinski and plantar grasp reflexes in infancy. *Arch Neurol Psychiatry* 1957;**94**:265–71.
12 **Babinski J**. De L'abduction des orteils. *Rev Neurol* 1903;**11**:728–9.
13 **Walshe F**. The Babinski plantar response, its forms and its physiological and pathological significance. *Brain* 1956;**79**:529–56.
14 **Estanol R**. Temporal course of the threshold and size of the receptive field of the Babinski sign. *J Neurol Neurosurg Psychiatry* 1983;**46**:1055–7.
15 **Wartenberg R**. The Babinski reflex after fifty years. *JAMA* 1947;**135**:763–7.
16 **Nathan PW**, Smith MC. The Babinski response: a review and new observations. *J Neurol Neurosurg Psychiatry* 1955;**18**:250–9.
17 **Nathan PW**. Effects on movement of surgical incisions into the human spinal cord. *Brain* 1994;**117**:337–46.
18 **Landau WM**, Clare MH. The plantar reflex in man, with special reference to some conditions where the extensor response is unexpectedly absent. *Brain* 1959;**82**:321–55.
19 **Burke D**, Knowles L, Andrews C, *et al*. Spasticity, decerebrate rigidity, and the clasp-knife phenomenon: an experimental study in the cat. *Brain* 1972;**95**:31–48.
20 **Lundberg A**. Control of spinal mechanisms from the brain. In: Tower DB, ed. *The nervous system*, vol 1. New York: Raven Press, 1975:253–65.
21 **Burke D**, Lance JW. Studies of the reflex effects of primary and secondary spindle endings in spasticity. In: Desmedt JE, ed. *New developments in electromyography and clinical neurophysiology*, vol 3. Basel: Karger, 1973:475–95.
22 **Ashby P**, Andrews C, Knowles L, *et al*. Pyramidal and extrapyramidal control of tonic mechanisms in the cat. *Brain* 1972;**95**:21–30.
23 **Lance JW**. The control of muscle tone, reflexes and movement: Robert Wartenberg Lecture. *Neurology* 1980;**30**:1303–13.