THE PLANTAR REFLEX

a historical, clinical and electromyographic study

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A HISTORICAL, CLINICAL AND ELECTROMYOGRAPHIC STUDY

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Aan mijn ouders
Aan Carien, Maarten en Willem

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GENERAL INTRODUCTION

The plantar reflex is one of the most important physical signs in medicine. Few patients undergoing a full medical examination can avoid having their soles stroked, because an upgoing great toe is regarded as a reliable sign of dysfunction of corticospinal nerve fibres. So far, there is little to justify a new study.

One problem is, however, that it can be difficult to determine the direction of the reflex response: the movements of the great toe may be slight, wavering, inconstant, or masked by voluntary activity. Soon after the introduction of the reflex the comment had already been made that '... the average ward clerk's notes are quite worthless on the subject of the plantar reflex, though he may make fair notes on the knee jerk and the pupil reflex to light' (Harris, 1903). Anyone who is a regular participant in medical ward rounds knows that controversies about plantar reflexes still abound today. Decisions in such cases are ususally guided by a mixture of seniority and ill-defined intuition. This is because the toe response is widely regarded as an oracle which often speaks in riddles, rather than as a definite reflex phenomenon, subject to the rules of physiology. But to give the plantar reflex its proper value, the neurologist must be aware of what is happening in the spinal cord. Therefore the first leading theme of this study was to ascertain the connections between the toe responses and other spinal reflexes, and to apply these physiological relationships to the interpretation of equivocal plantar responses.

A review of previous clinical studies about the plantar reflex precedes the actual experiments. Since Babinski discovered the toe response in 1896, he has been awarded a prominent place on the neurological Olympus, and his papers on the subject have been canonized (Wilkins and Brody, 1967). However, to get full insight into the meaning of Babinski's words, we must connect these with pre-existing concepts, with the subsequent development of Babinski's own ideas, and with additional clinical and physiological observations of others (Chapter I).

A preliminary question is the extent of the problem of equivocal plantar responses. How far can the clinician differ with his colleagues, or even with himself? And does he always acknowledge that a given plantar response is ambiguous, or may doubts be discarded because 'we shut our eyes to observations which do not agree with the conclusions we wish to reach' (Asher, 1972)? If so, there would be even more reason to look for rigid clinical criteria. The variation between and within observers, as well as the bias effect of previous clinical information has been studied by means of filmed plantar reflexes (Chapter II).

Electromyographic recording from muscles that are active in the plantar reflex could be of help in equivocal cases. Unfortunately, there is no consensus on which muscle mediates the Babinski sign: the extensor hallucis longus (Landau and Clare, 1959, in keeping with older notions), or the extensor hallucis brevis (Kugelberg et al., 1960; Grimby, 1963 a). In the latter studies electrical stimuli were used, and the boundary between the presence and absence of the Babinski sign was electromyographically indistinct. Reviewers avow that the plantar response is 'not a simple reflex' (Basmajian, 1974) and 'a manifold phenomenon' (Brodal, 1969). Could not this complexity be caused by the use of electrical stimuli? This question has been re-examined by recording from the toe muscles during stroking of the sole, i.e. while the great toe is actually going up or down. This done, we can go on to see whether electrical stimuli give the same result. If not, studies in which electrical stimuli are used should be interpreted with caution (Chapter III).

Once the electromyographic patterns accompanying normal and pathological plantar responses are known, this technique can be applied to equivocal plantar reflexes. Are the electromyographic results consistent? This has been investigated by repetition and 'blind' interpretation, and the outcome in each patient has also been checked with the eventual neurological diagnosis. Then, clinical and electromyographic observations have been compared, and I have tried to deduce from the differences and similarities a set of rules (and pitfalls) for the bedside interpretation of plantar reflexes (Chapter IV).

The last main question in this study concerns the pathophysiology of the Babinski sign. On which descending fibres does it depend, which segmental pathways mediate it, and at what level in the lumbosacral cord do the descending and segmental fibre systems interact? After an appraisal of pathological studies, the termination of the descending fibres that are involved in the appearance of the Babinski sign has been investigated from a clinical angle: which other physical signs are most often associated with the Babinski response? More precisely, is the appearance of the Babinski sign linked to motor deficits, or rather to segmental release phenomena? If such correlations give insight into physiological and anatomical relationships, can we conversely explain an unexpectedly absent Babinski sign by the concurrent absence of some other pathological features? Or can a dysfunction of intraspinal pathways also play a part? Finally I have tried to consider whether the association patterns of various pyramidal signs justify the concept of *the* 'upper motor neurone' (Chapter V).

CHAPTER I

HISTORY OF THE PLANTAR REFLEX AS A CLINICAL SIGN

'Ich halte einen jeden Unterricht ohne historische Grundlage für eine Barbarei.'

(Virchow, 1865)

DISCOVERY

The plantar reflex before Babinski

Retraction of the leg caused by plantar stimulation has of course been observed since time immemorial. The modern theory of reflex action was founded by Descartes (1664); he postulated that certain actions resulted from 'reflection of spirits' within the brain, in which the will took no part. That the spinal cord, widely regarded as a bundle of cranial nerves, could also mediate automatic acts was implied by occasional experiments of 18th century scientists such as Hales and Whytt (see Clarke and O'Malley, 1968). The Czech physician Prochaska (1784) theorized that the reflection of external impressions from sensory nerves into motor nerves was mediated by a local 'sensorium commune'. Hall (1833) confirmed the existence of the 'spinal soul' by systematic experiments, and caused great excitement and controversy.

In the course of the 19th century, however, scientists and physicians became accustomed to these ideas. Withdrawal of the leg in man was particularly analogous with Hall's animal experiments, and continued to be regarded as a medullary automatism even when at one time the pathways of later discovered reflexes were in dispute. It was found in normal subjects as well as in disease. One way of evoking this reflex was by forceful passive flexion of the toes. Brown-Séquard (1868) relates how he first observed this manoeuvre in Paris, nine years before, performed by the valet of a young American suffering from paraplegia – it stopped extensor spasms and made dressing possible. Brown-Séquard introduced the procedure into both the French and the English school of neurology. Later it was rediscovered by Sinkler (1888) – his claims were voiced by Wartenberg (1947, 1951) – and again by Bechterew (1906).

It was more usual, however, to stimulate the sole of the foot in order to bring about reflex withdrawal of the leg, and depending on the stimulus intensity this resulted either in dorsiflexion of the foot alone, or in flexion of knee and hip as well. It is in this sense that we encounter the 'plantar reflex' as part of the neurological examination: in articles by Schwarz (1882) and Geigel (1892), and in the well-known textbooks by Wernicke (1881), Gowers (1892) and Blocq & Onanoff (1892). Most authors concurred that the plantar reflex was 'diminished' in hemiplegia.

Movements of the *toes* during reflex flexion of the leg were initially overlooked or mentioned only in passing. Oppenheim (1889) noted plantar flexion of the toes in a few normal subjects. On the other hand, Wernicke (1881) mentioned dorsiflexion of the toes as being the normal response, and this mistaken belief was widely shared, at least in Germany. This is testified by the many surprised reactions to Babinski's discovery,

FIGURE 1

BABINSKI (1857-1932)
This photograph was taken in 1904

viz. those of Strümpell (1899), Oppenheim (1899), Cohn (1899), Kalischer (1899), Remak (1899) and Specht (1902). In this light it is understandable that observations about reflex dorsiflexion of the great toe in patients (Strümpell, 1880; Remak, 1893) were not more than casual and passed unnoticed. To regard these – even in the authors' own eyes – insignificant notes as prior discoveries (Ritter, 1967:'the so-called Babinski sign') shows limited insight into history.

The toe phenomenon

It follows from the previous passage that the uniqueness of Babinski's observations was not so much the reflex extension of the toes itself, but the contrast of this movement with the reverse phenomenon of plantar flexion in normal subjects. In other words, dorsiflexion of the toes following plantar stimulation is not just a fortuitous reflex, it is a *sign of disease.*

Babinski (figure 1) first presented his findings before the Biological Society of Paris, on February the 22nd, 1896. The records of this meeting contain a very short communication (Babinski, 1896 a):

Sur le réflexe cutané plantaire dans certaines affections organiques du système nerveux central.	**On the cutaneous plantar reflex in certain organic affections of the central nervous system.**
J'ai observé dans un certain nombre de cas d'hémiplégie ou de monoplégie crurale liée à une affection organique du système nerveux central une perturbation dans le réflexe cutané plantaire dont voici en quelques mots la description.	I have observed that in a certain number of cases of hemiplegia or crural monoplegia, related to an organic affection of the central nervous system, there is a disturbance of the cutaneous plantar reflex which I shall describe in a few words.
Du côté sain la piqûre de la plante du pied provoque, comme cela a lieu d'habitude à l'état normal, une flexion de la cuisse sur le bassin, de la jambe sur la cuisse, du pied sur la jambe et des orteils sur le métatarse.	On the healthy side, pricking of the sole of the foot provokes, as usual in normal subjects, flexion of the thigh towards the pelvis, of the leg towards the thigh, of the foot towards the leg, and of the toes upon the metatarsus.
Du côté paralysé une excitation semblable donne lieu aussi à une flexion de la cuisse sur le bassin,	On the paralysed side a similar excitation also results in flexion of the thigh towards the pelvis, of

de la jambe sur la cuisse et du pied sur la jambe, *mais les orteils, au lieu de se fléchir, exécutent un mouvement d'extension sur le métatarse.* Il m'a été donné d'observer ce trouble dans des cas d'hémiplégie récente remontant à quelques jours seulement, ainsi que dans des cas d'hémiplégie spasmodique de plusieurs mois de durée; je l'ai constaté chez des malades qui étaient incapable de mouvoir volontairement les orteils, comme aussi sur des sujets qui pouvaient encore faire exécuter aux orteils des mouvements volontaires; mais je dois ajouter que ce trouble n'est pas constant.

J'ai aussi observé dans plusieurs cas de paraplégie crurale due à une lésion organique de la moelle un mouvement d'extension des orteils à la suite de la piqûre de la plante du pied, mais, comme en pareil cas, il n'y a pas chez le malade même de point de comparaison, la réalité d'un trouble est moins manifeste.

En résumé, le mouvement réflexe consécutif à la piqûre de la plante du pied peut subir dans les paralysies crurales reconnaissant pour cause une affection organique du système nerveux central non seulement, comme on le sait, une modification dans son intensité, mais aussi une perturbation dans sa forme.

the leg towards the thigh and of the foot towards the leg, *but the toes, instead of flexing, execute a movement of extension upon the metatarsus.* I have been able to observe this derangement in cases of recent hemiplegia, dating from only a few days before, as well as in cases of spastic hemiplegia of several months' duration; I have found it in patients who were incapable of moving their toes voluntarily, as well as in subjects who could still execute some voluntary movements in the toes; but I must add that this derangement is not constant.

I have also observed that, in several cases of paraplegia due to an organic lesion of the spinal cord, the toes show an extensor movement following pricking of the sole of the foot, but as there is no mode of comparison in the patient himself in such cases, the presence of a disturbance is less obvious.

In summary, the reflex movement following a prick on the sole of the foot may, in paralysis of the lower limb caused by an organic affection of the central nervous system, undergo not only, as one knows, a change in intensity, but also a disturbance in form.

In a footnote to a subsequent communication to the Biological Society Babinski again referred to the toe phenomenon ('phénomène des orteils'), as he had come to call it, but now mentioned *stroking* of the sole as a stimulus, along with pin-prick (Babinski, 1896 b).

These early reports did not reach many physicians outside Babinski's own circle. The first confirmatory report came from the Belgian neurologist van Gehuchten (1898 a); he surmised that the pathological reflex was related to a lesion of *pyramidal tract* fibres. In the meantime, however, Babinski (1897) had expressed similar views in a discussion at the International Congress of Neurology in Brussels, drawing a parallel between the pathological reflex and the occurrence of the toe phenomenon in healthy new-born *infants*. He wrote about this to van Gehuchten, who apologized gracefully and added that in Belgium the toe phenomenon was designated 'the Babinski reflex' (van Gehuchten, 1898 b). Another feature of the reflex was also noticed more or less independently by the two clinicians: the predominance of the *great toe* in the abnormal extensor movement (Babinski, 1897; van Gehuchten, 1898 a).

In 1898 Babinski devoted a more elaborate article to the toe phenomenon; it was drafted as a clinical demonstration, and published in a weekly medical journal. This is often referred to as his classical paper, and indeed it is of outstanding lucidity (an accessible English translation is that of Wilkins and Brody (1967)). Apart from reaching a wide audience, Babinski added some further observations:

- in normal subjects the toes can remain *immobile* after plantar excitation (but they never show an extensor movement)
- the response may vary according to the *part of the sole* that is stimulated, extension being more easily elicited from the outer side (and predominating in the first or first two toes), and flexion (especially of the outer toes) from the inner side
- he had encountered the toe phenomenon *during an epileptic seizure,* following a toxic dose of *strychnine* (to disappear afterwards), and in *meningitis*
- presence of the phenomenon *excludes hysteria.* Babinski also gave some advice to avoid misinterpretation of the toe reflex (see Chapter IV).

Five years later, while the upgoing toe phenomenon was already established as the sign of Babinski, its originator added a new feature: *abduction* of the toes after plantar stimulation, cleverly illustrated by a photograph showing the shadow of the toes against the other leg (Babinski, 1903 a; for an English version see again Wilkins and Brody (1967)). Babinski thought it could be of use in cases of doubtful toe extension, but started by saying that it had already been noted by others (he might have mentioned Collier (1899) or Walton and Paul (1900)), who attached little diagnostic value to it. Moreover, Babinski himself had occasionally found it in normal subjects. This feature is now known as the 'fan sign' ('signe de l'éventail'), a term coined by Dupré but with little appeal for Babinski (1903 b). In later years he rarely emphasized it again

(Babinski, 1907 b, 1924). Moreover, there is no objective study proving the diagnostic value of the fan sign, whereas its occurrence in normal subjects has been confirmed (Noica and Sakelaru, 1906; Barré and Morin, 1921). It is therefore misguided traditionalism to regard only toe extension plus abduction as a valid Babinski sign (a frequent textbook view); one might as well include fanning of the toes when the patient is rising from a couch (Babinski, 1903 b).

Joseph Babinski and his work

Babinski has had many biographers but few knew him intimately (Guillain, 1932; Vincent, 1932; Vaquez, 1932; Charpentier, 1937; Tournay, 1953). He was born of Polish parents in Paris, in 1857; his full Christian names were Joseph Félix François. His father, an engineer, had fled Poland after having taken part in an unsuccessful revolt against the Russian occupation in 1848. A few years later he married a compatriot, Henriette Weren. Life was not easy for the emigrants, and when Joseph was 5 years old (and his brother Henri about 7) Alexander Babinski had to earn his living in Peru, staying there for more than eight years. The boys went to a Polish school, while they acquired a taste for culture from their mother.

Later, when Henri had become a mining engineer, he went in turn also abroad in order to support his parents and Joseph's medical studies. These studies were concluded in 1885 with a thesis on the pathology of spinal plaques in multiple sclerosis, still a classic in the field.

Around this time Babinski's career took a decisive turn due to a stroke of luck. He became Charcot's 'chef de clinique' without ever having been his intern – by just failing to win the competition for the gold medal of the Paris Hospitals (it was given to Richardière), and Charcot had been advised to offer the post to the second best. At that time, Charcot was at the summit of his fame. Studying the countless handicapped inmates of the 'Salpêtrière' he had identified a major part of the chronic neurological diseases that we know today. In the eighties he was chiefly concerned with the study of hysteria, regarded by him as a localized, albeit functional, hereditary disease of the central nervous system. This elusive disease with its apparently unlimited manifestations was dramatically demonstrated by Charcot (one remembers Brouillet's famous painting of one of these 'Tuesday lectures' in which Babinski is supporting a woman showing an hysterical 'arch'), and hysteria was to be a leading theme of Babinski's scientific work.

Babinski was un-Gallic in his impressive stature and bearing as well as in his scrupulous but constructive doubt, taking nothing for granted, testing and re-testing. He was one of the first to perceive how many of

Charcot's concepts about hysteria were founded on iatrogenic induction, and he gradually realized that organic diseases could not truly be mimicked by hysteria. The boundary was 'not a ditch but an abyss', and he patiently looked for signs which could not be reproduced voluntarily, enabling him to distinguish the two at the bedside.

Babinski was in an even better position to pursue his own studies after he had been appointed to an independent consultant position ('médecin des Hôpitaux') at the Pitié Hospital, in 1890. He was to stay here for more than thirty years, although in 1892 he attempted to obtain an academic position via a highly competitive examination ('agrégation'). He was unsuccessful because the chairman, Bouchard, favoured his own pupils above those of Charcot (Satran, 1974); this was the more remarkable as Bouchard had himself been a former student of Charcot. Meanwhile, Babinski's financial situation had improved through Charcot's help, and Henri could return. The brothers stayed together after the death of both parents, near the end of the nineties (their father's last years were marred by parkinsonism). Neither seems to have considered marriage, although a famous actress is said to have died with Joseph's portrait in her hands. Henri served as a secretary and cook to his brother – in the latter capacity he was even a well-known author (Ali Bab, 1928).

The toe phenomenon was the most impressive, but by no means the only physical sign that Babinski discovered and used to distinguish organic from hysterical hemiplegia. In the arm he was able to demonstrate hypotonia objectively by increased flexibility (Babinski, 1896 b) or by exaggeration of passive movements (Babinski, 1909); he also noticed that the organically affected arm was apt to pronate when supported only at the extensor side of the wrist (Babinski, 1907 a). With regard to the face he pointed out that organic weakness of the mouth on one side should be accompanied by asymmetrical activity of the platysma, and in the same study he stressed that rising from a couch often resulted in hip flexion on the paralysed side (Babinski, 1900 a). Not only did he separate hysteria from structural disease, he also gave it a practical definition: a pathological state that could, in general, either be reproduced or dispelled by suggestion. In consequence he proposed another name: 'pithiatisme', from the Greek words for 'persuasion' and 'curable'. During the first world war he practised these ideas very successfully in the French military hospitals.

Babinski's work as a whole (published in 1934) was almost exclusively clinical – he preferred to study the living rather than the dead. Much of his work has been incorporated into growing concepts. Other findings are still applied unchanged: 'inversion' of the supinator jerk in lesions of the 5th cervical root, dysdiadochokinesia and hypermetria in cerebellar disorders, absence of the ankle jerk in sciatica – even when examined with the patient kneeling on a chair.

In 1922 he retired. His successor Vaquez created the opportunity for a weekly clinic, which he continued for another seven years. Babinski died in 1932, at the age of 75, and two years after Henri; he was buried at the Polish cemetery of Montmorency.

ACCEPTANCE

The pyramidal syndrome before the toe reflex

Most physical signs of supranuclear weakness that we use today had been known for some years before Babinski added his sign to the clinical armamentorium (Jendrassik, 1894; Verjaal, 1957, 1958). In the 1860's, Charcot had discovered ankle clonus ('épilepsie spinale'), with Vulpian, and also spasticity ('contracture tardive'), although it was only later that he published these phenomena in detail (Charcot, 1876). In 1875, Erb and Westphal independently described the most common tendon reflexes, and their exaggeration in diseases of the central nervous system. The cutaneous reflexes, as well as their disappearance on the affected side in hemiplegia, were discovered by Jastrowitz (1875; cremasteric reflex) and Rosenbach (1876; abdominal reflexes).

The response of the toes in the plantar reflex formed the key-stone of the pyramidal syndrome: it did not depend, as did all other abnormal features except clonus, on comparison with the other side – Charcot's opinion that cutaneous reflexes and tendon jerks were often affected in cases of hysterical weakness was popular, and remained so until much later (Dejerine, 1915). The toe phenomenon was proposed as an independent sign of a disturbance of the corticospinal system.

Confirmation

Babinski's publication of 1898 met with rapid approval from all over the world. Reports on large and small series of normal subjects and patients corroborated the conclusion that reflex extension of the great toe was found only in affections of the corticospinal system. These studies are listed in table I.

Although Vires and Calmettes (1900), and also Verger and Abadie (1900) concluded that clear reflex extension of the toes proved a disturbance of the pyramidal system, they considered the sign too inconstant and uncertain to be of great practical value. A similar opinion was expressed by Oppenheim (1899). Koenig (1899) was the only investigator to assert some sort of precedence – he declared that he had obtained the same

TABLE I
STUDIES CONFIRMING BABINSKI'S FINDINGS ABOUT THE 'TOE PHENOMENON'

Belgium	Van Gehuchten (1898 a)
	Glorieux (1898)
	Crocq (1901)
France	Cestan and le Sourd (1899)
	Vires and Calmettes (1900)
	Verger and Abadie (1900)
	Ardin-Delteil and Rouvière (1900)
	Charpentier (1900)
England	Buzzard (1899)
	Collier (1899)
	Ferrier (1900)
	Barnes (1904)
Germany	Kalischer (1899)
	Koenig (1899, 1900)
	de Pastrovich (1900)
	Schönborn (1901)
	Schneider (1901)
	Homburger (1901)
	Böttiger (1902)
	Specht (1902)
United States	Walton and Paul (1900)
	Fraenkel and Collins (1900)
	Prince (1901)
	Eskridge (1901)
Poland	Chodźko (1901)
Rumania	Marinesco (1903)
	Noica and Sakelaru (1906)
The Netherlands	van Valkenburg (1907)

results as Babinski in unpublished observations dating from 1891–1892, but he did not press these claims very hard. Moreover, he was joined by someone in his audience, Laehr, who also professed himself quite familiar with these reflexes.

The study of Collier (1899) is the most comprehensive; it deals with all reflex phenomena after plantar stimulation, aside from those in the toes, and with the plantar reflex in infants, as well as with sources of error in the interpretation – these findings will be discussed in the appropriate sections. Collier also introduced the now common expressions of 'extensor response' for Babinski's 'toe phenomenon', and 'flexor response' for the normal reflex.

As to nomenclature, this leads us on a short detour via The Netherlands.

A curious eponym in Holland

All over the world, the normal plantar flexion of the toes after stroking of the sole is known – *mutatis mutandis* – as flexor response, normal response, or downgoing toes. In the Netherlands, however, and only there, it is generally referred to as 'plantar reflex according to Strümpell' (see for instance the local medical dictionaries of De Haan and Dekker (1957), Pinkhof (1963) and Hoolboom-van Dijck (1974)). This is the more remarkable because nowhere in Strümpell's work is there an original observation on the toe reflex in normal subjects. Neither has Strümpell nor any of his compatriots ever claimed such a thing: one looks in vain for the expression in a whole array of German textbooks, old and new, including Strümpell's own (1912).

The unsuitable eponym can be traced back to 1902, when it was first used in print by Bouman in a case report from Amsterdam. One of the two professors of neurology in Amsterdam around that time also used the phrase a few years later (Wertheim Salomonson, 1905). In 1900, the latter had still mentioned the plantar reflex in its pre-Babinski sense ('the plantar reflex is remarkably weak'), although the toe phenomenon had been signalled in the Dutch medical press soon after Babinski's main article had appeared (Roebroeck, 1898). In fact, Dutch neurologists did not appreciate the diagnostic value of the toe responses until after a verbal report of Crocq (1901) in Amsterdam, at a congress of physicians working for life insurance companies. After the meeting the Belgian neurologist demonstrated Babinski's reflex in the ward of professor Wertheim Salomonson (Pinkhof, 1901).

It may well be that the delayed introduction of the toe phenomenon in the Netherlands resulted in extra attention to a study of Strümpell which was published in 1899. Here, as in 1880, Strümpell casually mentioned reflex dorsiflexion of the hallux, but a few lines further down he described plantar flexion of the toes in paraplegic patients (with a footnote quoting 'Babinsky' who found this response in normal subjects). This particular paragraph is likely to be the source of the misplaced tribute, double Dutch indeed, spreading from Amsterdam to the rest of the country.

False positive findings?

Babinski's thesis that the toe phenomenon was never met with in hysteria did not go quite unopposed: isolated cases to the contrary were cited by Cohn (1899), Giudiceandrea (1899), Roth (1900), Tumpowski (1901), Bickel (1902), von Kornilow (1903), Harris (1903) and Friedlaender (1904).

Even more numerous were reports about upgoing toes in normal subjects or at least patients in whom the nervous system was supposed to be normal (table II).

Surveying these studies, the first question that comes to mind is about the criteria for a Babinski sign, as not all upward movements of the great toe during plantar stimulation necessarily represent a reflex (see Chapter IV). Occasional comments are made on the difficulty of interpretation, but there is no clue as to how the authors overcame these problems. Davidson (1931) and Morgenthaler (1948) tried to mitigate their findings with the argument that an upgoing toe in normal subjects was never accompanied by fanning – curiously, as toe abduction is rather non-specific (see p. 24). Assuming, however, that some extensor responses in the table were genuine, its presence in a 'hysterical' or 'normal' subject would lead us, these many years later, to cast doubt on the diagnosis rather than on the sign. In some of the examples this is even obvious, such as in a boy described by Harris (1903) who suffered from 'functional hemiplegia' and also showed ankle clonus.

False negative findings

Babinski himself stated as early as 1896 that the pathological toe sign was not a constant feature in hemiplegia, and in 1898 he specified that, although the phenomenon was invariably associated with some disturbance of the pyramidal system, the reverse was not always true. He again emphasized this view in 1902. Some subsequent authors assumed that Babinski considered his sign obligatory in affections of the pyramidal tract, and of course they carried an easy victory (Cohn, 1899; Harris, 1903). Why the Babinski sign can be unexpectedly absent is a problem which I shall take up in Chapters IV and V.

TABLE II
ALLEGED FALSE POSITIVE BABINSKI SIGNS IN NORMAL SUBJECTS

author	*number or percentage of false positive extensor responses*	*subjects*
Schüler (1899)	6% (n?)	'healthy men and women'
Létienne and Mircouche (1899)	1/54 (by excessive treadling of sewing-machine?)	patients with diseases outside the nervous system
van Epps (1901)	7/500	165 patients 'presenting no nervous symptoms', 335 'insane patients'
	6/213	patients 'suffering from nervous disease' but without 'involvement of the lateral tracts'
Levi (1902)	6% (n?)	surgical and psychiatric patients
Rossolimo (1902)	11/79	patients without clinical signs of a pyramidal tract lesion
Munch-Petersen (1902)	27/250 (all walked with toes up; by wearing clogs?)	normal adults
Goldflam (1903)	3	2 cases with chronic rheumatoid arthritis (also showing foot clonus!), one case of osteitis tibiae
Richter (1903)	9/500	all sorts of patients, with exclusion of diseases affecting brain or cord
Pfeifer (1903)	3/200	various patients without disease of the nervous system
Clark (1913)	2	ankylosis of the knee joint
Bersot (1917-1919)	951/16000	repeated examination of 32 soldiers
Friedman (1920)	'common'	'in the neuropathic, in ductless gland disease and other degenerative conditions' (extension of the toes swifter than usual)
Critchley (1931)	'at times'	'old age, in the absence of obvious disease of the pyramidal pathway'
Davidson (1931)	12%	161 normal subjects
Savitsky and Madonick (1943)	4.3%	1000 persons with head injuries; 2500 patients admitted for general conditions
	1.15%	704 'inductees into the army'
Morgenthaler (1948)	2%	200 schoolboys

FLEXION AND EXTENSION SYNERGIES

The Babinski sign as part of a flexor synergy

We have seen how Babinski (1896 a, 1898) regarded the abnormal extensor movement of the toes as a disturbance of the normal synergy, in which the toes flexed together with all other limb segments. This view was at first generally accepted, along with the diagnostic value of the sign. Collier (1899) was no exception, and he even expanded the concept by pointing out some other change in the flexor synergy of the leg, in addition to reversal of toe movements and increased activity of other flexor muscles, viz. late contraction of the musculus tensor fasciae latae. This reflex was first described by Brissaud (1896) as the response which has the lowest threshold on plantar stimulation, in patients and normal subjects alike. Collier noted that it appeared much later in the sequence – i.e. at higher stimulus intensities – in patients with pyramidal disease, but these observations have not been confirmed.

However, van Gehuchten (1900 a,b), to some extent preceded by Mingazzini (1899), was the first proponent of a different point of view that was to receive increasing support over the years. In his opinion, the *normal plantar response* did not change, but *disappeared* when the corticospinal fibres were affected (just like the abdominal and cremasteric reflexes). The normal reflex was replaced by reflex *extension* of the toes as part of something basically different: an exaggerated medullary activity, a defensive reflex analogous to the classic experiments on the decapitated frog. These defensive movements might constitute a cutaneous reflex, but strictly speaking not a plantar reflex, because in some cases they could be evoked *by stimulation of any part of the lower limb.*

The fact that the receptive zone for reflex withdrawal of the leg, including dorsiflexion of the hallux, extended beyond the sole of the foot, although the reflex threshold was ususally at its lowest there, had also been pointed out by Strümpell (1899) and was later confirmed by many others including Babinski himself (1900 b). Van Gehuchten's opinion that the Babinski toe phenomenon was part of a primitive but complex medullary mechanism, whereas the normal response of the toes constituted a separate reflex with a limited receptive field, was subsequently shared by Schneider (1901) and Noica (1912). Finally, this was confirmed beyond doubt by Marie and Foix (1910, 1912, 1913, 1915) and Walshe (1914); I shall discuss these studies in some detail.

Marie and Foix (1910) added a further aspect to the Babinski sign by showing that forceful flexion of the little toes could also evoke it, together with retraction of the leg as a whole. It has been mentioned earlier (p. 19) that this was already a time-honoured manoeuvre, practised by Brown-

Séquard, Vulpian, Charcot and Hughlings Jackson; claims of precedence over Marie and Foix were therefore superfluous. In the same study, Marie and Foix described transverse pressure on the foot as an alternative procedure. The essence of these renewed observations was stressed in later studies (Marie and Foix, 1912, 1913, 1915): withdrawal of the leg was *not even an exclusively cutaneous reflex,* as stimulation of deep structures could also produce it. In this series of publications, Marie and Foix beautifully demonstrated the analogy between reflex synergies of the lower limbs in man, especially in diseases of the nervous system, and the pioneer experiments of Sherrington in spinal and decapitated animals (Sherrington, 1910). They showed that reflex withdrawal of the leg was also more complex in its effects than reflexes properly called cutaneous: apart from activation of muscles that shorten the limb, there is inhibition of muscles that extend it (including inhibition of foot clonus). Moreover, when skin stimuli are moved proximally, the flexion synergy often gradually reverses into extensor movements.

Marie and Foix confirmed and expanded van Gehuchten's opinion that the Babinski sign could be evoked by the same range of stimuli as withdrawal of the whole limb, and that the Babinski sign was perfectly synchronous with proximal withdrawal movements. They went even further and argued that *the anatomical toe extensors are, in a functional sense, shortening muscles or flexors – and vice versa.* This had been implied earlier by Strümpell (1899). When one is walking on tip-toe as most animals do, flexion of the toes (in the anatomical sense) causes the limb to lengthen (physiological extension), whereas (anatomical) extension of the toes shortens the limb (physiological flexion). Again, this conclusion of Marie and Foix was in perfect agreement with the findings of Sherrington in animals: among the muscles that always contracted in the flexion reflex was the 'extensor longus digitorum' (Sherrington, 1910).

Although these considerations relieve us of an apparent paradox (extension of the toes as part of a flexor synergy), it leaves the problem of terminology and possible confusion. Collier's (1899) widely adopted terms '*flexor response*' and '*extensor response*', however well defined in clinical practice, are especially misleading when used in a physiological context. Marie and Foix (1912) used 'shortening reflex' ('réflexe des raccourcisseurs') as a substitute for Sherrington's 'flexion reflex', but this term is not particularly feasible for describing toe movements separately. It is preferable, and this will be adhered to in the remainder of this study, to designate the *muscles* of the toes by their accepted anatomical names, but to describe toe *movements* as upward/downward or dorsiflexion/plantar flexion or as Babinski sign/normal response, rather than 'extension'/ 'flexion' of the toes.

The conclusions of Marie and Foix (1912, 1913) were almost fully confirmed by Walshe (1914). His is another classical study inspired by Sherrington, and it can now be recommended without the cautionary note 'not free from insular prejudices' (Jelliffe and White, 1923).

In one important aspect, however, Walshe's findings were different from those of his French colleagues. Marie and Foix regarded the upgoing toe as the minimal response of the shortening synergy. Accordingly, they stated that the Babinski sign could occur without other reflex phenomena in the leg. Walshe, on the other hand, found that contraction of the hamstring muscles (semitendinosus and semimembranosus) constituted the minimal response or 'motor focus' of the flexion reflex, and that the threshold for activation of the extensor hallucis longus was always higher. Walshe explains the difference by pointing out that early contraction of the hamstring muscles is often noticeable only by palpation of the tendons and may easily be overlooked; this was also observed by Munch-Petersen (1902). Visual information alone can be misleading, because the extensor hallucis longus muscle is almost unopposed by gravity; the same applies to the musculus tensor fasciae latae. The important inference was therefore that *the Babinski sign was never obtained without contraction of other limb flexors.* Forty-two years later, Walshe (1956) could still assert that he had never seen an isolated 'extensor response'! In contrast, a downward response of the toes may very well be the only reflex movement.

Opposition from Babinski and others

Babinski did not at first react favourably to the idea that the normal downward response of the toes had nothing to do with the flexion reflex of the leg, whereas the upgoing toe sign was part of this synergy – all this conflicted with his concept of the 'transformation' of the flexion reflex. He contested the 'simple' nature of the normal response with the argument that it could be evoked from the thigh or abdomen in some patients (Babinski, 1904 a, b), but it was later shown that this phenomenon is part of a pathological extensor synergy, including the (anatomical) toe flexors (see p. 36). Initially, Babinski had to oppose only van Gehuchten, a dialogue complicated by unrelated issues (van Gehuchten, 1904; Babinski, 1904 b), but when the other arguments were put forward by Marie and Foix (1912, 1913) and Walshe (1914), he became more or less reconciled to these ideas (Babinski, 1915 a, 1922). Babinski admitted in these studies that there were strong ties between 'his' sign and the flexion reflex of the lower limbs, but on the other hand he persistently maintained that these ties were not indissoluble. His arguments are summarized below, together with later objections.

a) Patients with pyramidal lesions in whom plantar flexion of the toes is found together with the flexion reflex ('réflexes de défense').

Babinski cited three such cases. The first was a patient he had described many years before (Babinski, 1899 b), a 50-year-old male who eventually died from an extracerebellar tumour. He suffered from severe headaches, troubled vision and 'weakness of motility', but when examined in bed power and reflexes were normal. In the terminal stage, the patient developed flexion contractures of the lower limbs; the plantar responses always remained normal. At autopsy, the corticospinal fibres were intact at all levels. Walshe (1956) rightly doubts the nature of this 'paraplegia' and comments that such a fetal posture was at that time commonly to be found among bedridden and demented patients.

The second example invoked by Babinski was the well-known case history of the 17-year-old acrobat Alonzo R., described by Dejerine and Lévy Valensi (1911). The patient, while on tour in the United States, fell from the top of a human pyramid, sustaining a complete transverse lesion of the lower cervical spinal cord. He survived the accident for eleven months. Plantar stimulation resulted in active 'defence reflexes', but with downward movements of the toes. The report also mentioned a downward position of the toes at rest, owing to a contracture of the plantar muscles. Walshe's comment (1914) that it is difficult to see how any other movement could occur was granted by Babinski (1922) to be 'not without value'. Pierre Marie expressed a somewhat similar view in the discussion of the pathological report (Dejerine and Long, 1912). Nevertheless, we shall see in Chapter V that after acute spinal lesions a downward toe response can be found together with a flexion reflex in proximal muscles, owing to the unequal distribution of reflex depression.

The third case that Babinski advanced as showing 'defence reflexes' without dorsiflexion of the hallux was a paraplegic patient described by Pastine (1913 b). Minimal excitation of the lateral plantar border gave rise to an isolated Babinski response, whereas more forceful stimuli, especially when applied more proximally, resulted in retraction of the whole limb together with plantar flexion of all toes. In retrospect, this reflex pattern probably represents a transitional form between flexion and extension synergies (Marie and Foix, 1912; Marshall, 1954; see also p. 36). At any rate, this example justifies the qualification that stimulation above the foot may no longer result in an upgoing toe sign, while other elements of the flexion reflex remain present.

b) *Dissociation of Babinski sign from flexion reflex by application of Esmarch's bandage*

Esmarch's bandage was a strip of India rubber, applied to a limb from the distal end towards the proximal part, to expel blood preceding an operation. Babinski (1911 b) investigated the influence of this procedure on pathological plantar reflexes. After some time, the toes ceased to react to stimulation of the sole. When the bandage was taken off, the plantar reflex reappeared, but in the shape of the normal response (usually only from the inner side of the sole, while stimulation of the lateral plantar surface gave an upward response). A few minutes later the reflex reverted to the original situation where dorsiflexion of the hallux could be obtained from all parts of the foot. Babinski concluded from these experiments that the normal plantar response had not disappeared at all, but was only mechanically overpowered by the antagonistic muscles. Moreover, the temporary depression of hallux dorsiflexion contrasted with the state of the 'defence reflexes', which were exaggerated after leg compression.

The dissociation between Babinski sign and flexion reflex is of course artificial here, owing to selective ischaemia of distal structures, of the long toe extensors in particular. Marie and Foix (1912) demonstrated this by showing that a ligature at the ankle did not result in disappearance of the Babinski sign, while it did abolish plantar flexion of the toes in normal subjects. Marinesco and Noica (1913) repeated Babinski's experiment in a healthy volunteer, and found selective loss of distal power and of the ankle jerk. The hyperexcitability of the flexion reflex after ischaemia – providing the efferents are not blocked – was later attributed to a specific effect on afferent nerve fibres from the skin, with a reverse change in tendon reflexes and tone (Gilliatt, 1952).

It is more difficult to explain why the reflex in the flexor muscles of the toes – provided this is still remnant in patients – recovers earlier from ischaemia than the response in its more proximal antagonists. Marie and Foix (1912), who confirmed this finding, assumed that the muscles of the sole had a better vascular supply. Landau and Clare (1959) implicated mechanical compression of the lateral popliteal nerve against the head of the fibula, but Babinski probably applied the bandage more proximally. Whatever its explanation, the difference in sensitivity to ischaemia between the normal and pathological plantar response does not detract from the identity of the Babinski sign with the flexion reflex. The dissociation only indicates that the normal response may still emerge under some circumstances.

c) *Upgoing toe without flexion reflex of the leg*

Whereas the two preceding categories were advanced by Babinski as instances of an exaggerated flexion reflex without dorsiflexion of the hallux, he also observed the reverse, if only by applying light stimuli. In this respect, Babinski was supported by Marie & Foix (1912, 1913), and not by them alone. Bing (1915) found that proximal reflex phenomena accompanied the upgoing toe sign in most patients with spinal lesions, but only exceptionally in cerebral disease. The occurrence of an isolated Babinski response was subsequently mentioned by Fulton and Keller (1932), Szapiro (1958) and also by Scholten (1964, 1965), who came to the surprising conclusion that only this constitutes the real Babinski sign, quite separate from the sign described by Babinski. Guillain and Dubois (1914) published the case history of a young woman with infantile hemiplegia in whom an upgoing toe phenomenon could be evoked from as far rostrally as the ipsilateral face, while 'the defence reflexes were nil'. In this last patient, the flexion reflex was probably masked by the concomitant spasticity of the extensor muscles in the leg – the extensor hallucis longus is the only physiological flexor muscle able to overpower its antagonists (Walshe, 1923).

This explanation, however, probably does not account for all reported instances of an isolated Babinski sign. Walshe (1914) contends that in such cases a barely palpable contraction of the hamstring muscles or a just visible twitch of the musculus tensor fasciae latae has been overlooked. Although this follows from one of the most detailed studies on the subject, at this stage it must remain an open question whether this explanation is universally applicable (it will be referred to again in Chapter IV). That the recruitment order of muscles taking part in the flexion reflex could vary from one patient to another was stressed by Bersot (1917 – 1919), and by Dimitrijević and Nathan (1968).

Some authors, apparently quite unaware of the physiological discussions about the toe muscles, continued to connect the upgoing toe phenomenon to extensor synergies of the lower limb (Meyers, 1920; Armenise, 1924; Tournay, 1926; Salomon, 1921), or the normal plantar response to the flexion reflex (Goldflam, 1908; Waggoner and Ferguson, 1930; Yakovlev and Farrell, 1941; Morgenthaler, 1948).

Ipsilateral limb extension with downgoing toes versus the normal plantar response

Proximal stimulation of the lower limb in patients with exaggerated exteroceptive reflexes can result in contraction of quadriceps and triceps surae, together with plantar flexion of the toes (physiological toe extension). This extensor synergy is the counterpart of the flexor movements that are evoked on distal excitation. It was first described by Remak (1893), in a 4-year-old boy with myelitis; he called it 'Femoral-reflex', after the receptive zone. Marie and Foix (1912, 1913) indicated, as in the flexion reflex, the analogy between these extensor movements in man ('réflexe des allongeurs') and similar phenomena observed in spinal animals (Sherrington, 1910). Walshe (1914) and Riddoch (1917) postulated that extension reflexes depended on supraspinal influences and were not met with in spinal man, but eventually this view had to be rectified (Kuhn, 1950; Guttmann, 1952).

Babinski (1904, a, b) suggested that the downward movements of the toes in this pathological synergy were identical with the plantar response in normal subjects – an idea recently exhumed by Bathien and Bourdarias (1972). However, it was argued by van Gehuchten (1900 b), and in more detail by Marie and Foix (1912, 1913) as well as by Walshe (1914), that *the normal plantar response is a skin reflex in a strict sense: a unisegmental reflex with a limited receptive field and a correspondingly limited effect, quite comparable to the abdominal and cremasteric reflexes.* Whereas plantar flexion of the toes as part of an extension synergy can be evoked only by stimuli outside the sole of the foot, the reverse is the case for the normal plantar reflex. Moreover, the response in healthy subjects is never accompanied by activity in other physiological extensor muscles. It is, on the contrary, often superimposed upon the usually moderate flexion reflex that follows plantar stimulation in normal circumstances. This co-existence in itself is no proof of synergistic linkage, as Landau and Clare (1959) imply – that would take us back to Babinski's original 'transformation'. Additional evidence for the 'simple' nature of the downward toe response is found in patients with spinal shock (Chapter V, p. 115).

Crossed toe responses

1. *Crossed plantar flexion,* usually in a foot showing a Babinski response when stimulated directly. This phenomenon was noticed, without further explanation, by Babinski (1898), Collier (1899) and Klippel et al. (1908). It was stressed by Steinberg (1908), Maas (1911) and Pastine (1913 a) that crossed plantar flexion of the toes could be observed in paretic limbs

without an ipsilateral upgoing toe sign. Conjectures about the mechanism of its appearance included segmental irradiation of the normal response from the stimulated side (Parhon and Goldstein, 1902; Bramwell, 1903). The latter retracted his theory in 1911, when he reported a patient with bilateral Babinski signs as well as crossed downward responses. Others have suggested short-circuiting of cortex-bound 'flexor' impulses to the contralateral side (Knapp, 1907), or diversion of these impulses via the uncrossed pyramidal tract (Sano, 1901; Fairbanks, 1911).

Speculations became superfluous when again Marie and Foix (1913, 1915), and Walshe (1914) pointed out that the contralateral downward movements of the toes were part of an extensor synergy, although often being the minimal response. A similar interpretation had been given earlier by Strümpell in 1899 (he produced it from the hemiplegic side, which is rare). Accurate description of this crossed extensor reflex as a regular accessory to the flexion reflex also stemmed from the work of Sherrington (1910) in spinal animals.

Plantar flexion of the opposite toes is not necessarily a pathological reflex – it has been found in normal subjects by Collier (1899) and Fairbanks (1911); Klippel and Weil (1908) and Pastine (1913 a) encountered it frequently in tuberculous patients.

2. *Crossed dorsiflexion of the hallux* is not met with in the absence of pyramidal lesions. Most reports relate to patients with bilateral corticospinal involvement (Collier, 1899; Walton and Paul, 1900; Parhon and Goldstein, 1902; Bramwell, 1911; Fairbanks, 1911; Kidd, 1911; Brain and Wilkinson, 1959). Only Maas (1911) and Pastine 1913 a) found it in some cases of hemiplegia, on stroking the sole of the non-paretic side.

A correlation with spinal synergies in animals is less obvious here than it is in the case of the flexion reflex, crossed extension reflex or ipsilateral limb extension. In human cases of long-standing paraplegia, however, the occurrence of simultaneous flexor spasms in the lower limbs is well-documented (Riddoch, 1917: 'mass reflex').

Tonic plantar flexion of the toes in hemiplegia

Of thirty-six patients with organic hemiplegia in whom the plantar reflexes were examined by Collier in 1899, three cases did not only show 'flexor responses' (in Collier's words) on both sides, but also the downward movement of the toes was even '. . . more marked on the paralysed side and . . . very atypical, consisting of vigorous flexion of all the toes'. A similar paradoxical downward toe response in cerebral lesions has been the subject of a number of heterogeneous reports.

One such category is formed by alert patients with long-standing hemiplegia who have gradually developed a dystonic posture on the paralysed side, whilst the Babinski sign has disappeared or can be evoked only from other areas than the sole of the foot. Involuntary clenching of the fingers is then often also present, together with activity in other flexor muscles of the arm. This clinical picture is described or implied by Barraquer (1921), Barraquer-Ferré (1930), Goldstein (1931), Landau and Clare (1966), and Dooling and Adams (1975). The report by Lomtadsé (1932) has a special flavour, not only because tonic plantar flexion of the toes is produced by sliding the thumb along the tibia, but also because of the 'balneological significance' attached to it: hot mud baths might precipitate a stroke in these patients.

A second type of cerebral disease in which tonic plantar flexion of the toes has been found is in diffuse dysfunction of the frontal lobes, also manifested by palmar grasping, sucking responses, and severe intellectual impairment. In these cases the downward response of the toes is often produced only by pressure or light touch on the sole, while stronger stimuli give a Babinski sign. Extensive damage of the frontal lobes was possibly present in the patient described by Schuster and Pinéas (1926 – 'forced grasping' of the foot). This can be stated more confidently for three of the four cases of Brain and Curran (1932 – 'grasp reflex of the foot') and for the seven patients reported by Goldstein in 1938 ('tonic foot response') – all these ten patients suffered from a cerebral space-occupying lesion.

A purely proprioceptive type of tonic activation of the toe 'flexors' was distinguished by Alajouanine et al. (1968); in such cases it is produced only by muscle stretch during walking or even standing. Distinction from the exteroceptive form is not always possible: Cohen and Iannone (1967) report a patient in whom all sorts of stimuli resulted in plantar flexion (only of the small toes and the foot, while the hallux went up). And Manfredi et al. (1975) give three case histories where only the combined effect of exteroceptive and proprioceptive stimulation during walking produced the 'tonic ambulatory foot response', whereas neither noxious stimulation nor passive stretch alone could bring it about.

A paradoxical tonic downward response of the toes may be found not only in patients with cerebral lesions, but also in patients with a recent transverse lesion of the spinal cord. This latter phenomenon will be discussed in Chapter V.

RIVAL SIGNS

Confusion

When Babinski gave a lecture to the Royal Society of Medicine, London, in 1922, he started with a story from Cervantes' 'Don Quichotte'. At one time this great knight had been travelling until dark, and he at last called at an inn. The keeper, before opening the door, cautiously enquired whom he had the honour of addressing. The nobleman proclaimed his names: Duque de Bejar, Marques de Gibraleon, Conde de Bañalcazar y Bañares, Visconde de la Puebla de Alcocer, Señor de las Villas de Capilla, Curiel y Burguillos. The publican replied that he could not lodge so many people, and thereby deprived himself of a guest who might have procured him great profit.

A similar misadventure, continued Babinski, lies ahead of the student who wishes to acquaint himself with the reflex phenomena of the lower limbs, but who fails to accommodate in his mind various names like 'defence reflexes', 'dorso-plantar flexion reflex of Bechterew', 'triple retraction of the lower limb', 'phénomène des raccourcisseurs', 'medullary automatism', 'mass reflex' – not knowing that all these terms relate to a single physiological phenomenon.

This story is also true for the Babinski sign. Many physicians lost sight of the fact that it was part of a complex movement, known long before anyone cared about toe reflexes. The many synonyms for the flexion reflex may be partly to blame. But the most important factor must have been the mystical power of a great toe which indicates whether there exists a disturbance of the corticospinal fibres: all eyes were fixed on the toe and no one dared to avert his eyes to knee or hip during the 'sacral' procedure. In this light it is understandable that alternative methods of addressing the oracle received much attention, although hardly surprising from a physiological point of view. Apart from Babinski himself only a few realized this from the beginning (van Gehuchten, 1900 a). The desire to survive eponymously has probably also contributed to the weed-like sprouting of rival signs – Wartenberg (1947) quotes Lewandowsky as saying how surprisingly ineffective stroking of the sole appeared to be in many of such reports.

Different sites of excitation

1. *Pinching of the Achilles tendon* (Schaefer, 1899 – 'Antagonistischer Reflex'). Babinski (1900 b) was quick to perceive that this reflex depended on local cutaneous stimulation, and that there was nothing

antagonistic about it. He received support from de Buck and de Moor (1900) and from Lasarew (1906). Verger and Abadie (1900) also admitted that the calf muscle was not involved, but reserved a role for afferents from the Achilles tendon. Levi (1902) found the reflex present in a large proportion of 'normal' subjects (but he also reported some positive findings in this group following plantar stimulation). Schaefer thought his reflex indicative of cerebral lesions only, which was refuted in most of the subsequent reports.

2. *Stroking the tibial surface* (Oppenheim, 1902 – 'Unterschenkelreflex') – a rather painful manoeuvre, but nevertheless practicable in 'even the most sensitive ladies at the private consulting-office'. Apart from the great toe, dorsiflexion of the foot is also considered pathological (later Babinski (1912, 1915 a) also stated that dorsiflexion of the foot is abnormal when evoked outside the sole). Pfeifer (1903, 1904) considered the signs of Babinski and Oppenheim complementary: cases where one was absent and the other positive were in balance. Hahn (1911) found the Oppenheim procedure less sensitive, and Walshe (1914) also commented that it rarely gave extra information.
3. *Compression of the calf muscle* (Gordon, 1904, a, b – 'paradoxic reflex'). Gordon took care that his sign was not forgotten (Gordon, 1907, 1911), and enriched us in 1914 with a crossed variety. His modification was supposed to be especially valuable in early or slight lesions of the motor tract, and it was even claimed that the sign could demonstrate involvement of the uncrossed pyramidal tract. Auerbach (1917) confirmed its exclusive presence in subtle changes of the spinal cord, such as caused by a gunshot wound at considerable distance, or by excesses 'in venere'! Bing (1918) was also inclined to acknowledge it as an index of slight pyramidal lesions.

   With regard to the presumed paradoxical nature of the reflex, Babinski's comments about Schaefer's so-called antagonistic reflex are equally valid in this case.
4. *Forceful plantar flexion of foot and toes* (Bechterew, 1906). Bechterew found it less often positive than the Babinski sign. We have already seen (p. 19) that this manoeuvre was practised decades before in Paris and London to induce the flexion reflex of the leg, and it is not surprising that it provokes the Babinski response at the same time.
5. *Downward massage of the calf* (Trömner, 1911 – 'Wadenphänomen').
6. *Stroking the skin beneath the lateral malleolus* (Chaddock, 1911 – external malleolar sign). It is interesting that Chaddock worked with Babinski, from 1897 to 1899 (Bailey, 1961).
7. *Stimulation of the thigh* (Austregesilo and Esposel, 1912). By way of exception, these authors claim no particular sensitivity or diagnostic importance.

8. *Stroking the anterior surface of the ankle* (Crafts, 1919).
9. *Stroking the lateral dorsum of the foot* (Roch and Crouzon, 1928). This site is a few centimetres more distal than in Chaddock's manoeuvre, and it should be stimulated with the thumb.
10. *Forceful abduction of the little toe* (Stransky, 1933).
11. *Forceful downward movement of (preferably) the fourth toe* (Grigorescu, 1940 – 'a new pyramidal sign'; Gonda, 1942 – 'a new tendon stretch reflex'). Allen (1945) advocated this variant in New Zealand. It is clear that this is not a stretch reflex at all, at least in the accepted sense; for further comments see nr. 4. Szapiro (1960) recommended plantar flexion of the small toes as an additional procedure to stimulation of the plantar surface.
12. *Stroking of the sole at the base of the toes* (Lenggenhager, 1945 – 'Fussballen-Streichreflex'). This method is indeed very effective, even in normal subjects (Verger and Abadie, 1900; Dosužkov, 1932).
13. *Pressure on the occiput,* where the lambdoid suture meets the temporal bone (Grünfelder, 1931 – 'Grünfelderscher Zehenreflex'). The upgoing toe was taken here to indicate not a pyramidal lesion but the presence of otitis media in children.

Considering the common physiological denominator of all these different methods, there is little justification for the use of eponyms.

Stretch reflexes of the toe muscles

Strictly speaking, pathological signs that are the result of muscle stretch are misplaced among the variants of an exteroceptive reflex. The few that follow, however, were clearly launched to surpass the Babinski sign, and their proprioceptive nature was not appreciated at the time.

1. *Snapping the great toe upwards* (Rossolimo, 1902). An absent hallux response was considered normal, dorsiflexion occurred in general hyperreflexia, and a downward response indicated a pyramidal lesion.
2. *Percussion of the dorsum of the foot* (Mendel, 1904 a, b; 1906 – 'Fussrückenreflex'; Bechterew, 1904 – tarsophalangeal reflex). Dorsiflexion of the little toes was the normal response, plantar flexion was found only in patients with organic disease of the central nervous system.
3. *Percussion of the dorsal metatarsophalangeal joint of the great toe,* resulting in its dorsiflexion (Throckmorton, 1911).
4. *Plantar flexion of the foot while the knee is bent* (Moniz, 1916). This relates to one case only, where it was the only manoeuvre that

produced an upgoing toe response; all other stimuli failed. The explanation of this singular phenomenon must remain in doubt.

Spontaneous or associated dorsiflexion of the great toe

The spell of the upgoing toe sign was such that some attached a similar importance to an upward position that was not a reflex at all.

Associated dorsiflexion of the hallux (and foot) on leg raising against resistance had already been observed before the time of the toe reflexes by Strümpell (1887); it was pronounced a pyramidal sign by de Pastrovich (1900) and Friedlaender (1904), while Lichtmann (1941) devoted a special communication to this 'modified Babinski reflex' or 'resistance reflex'. De Souza and de Castro (1913) found it present in normal subjects, and so did Brain and Wilkinson (1959). Klein (1928) considered associated dorsiflexion of the hallux an extrapyramidal feature when obtained by the Kernig manoeuvre.

An upgoing great toe on the side opposite the muscular efforts was also first noted by Strümpell (1887); Brain and Wilkinson (1959) found it a rather frequent but inconstant feature in patients showing a Babinski response. Hindfelt et al. (1976) advanced it as a new sign – in a few decades we may have to expect the next rediscovery.

Elevation of the hallux at rest was credited with diagnostic importance by Acchiote (1911) and Sicard (1911), but it is doubtful whether this position never occurs in healthy subjects.

Effects other than in the toes after plantar stimulation

Adduction of the foot after stroking the inner aspect of the sole was propounded as a pathological sign, comparable with the Babinski response, by Hirschberg and Rose (1904). Von Monakow (1909) made the same observation, and added the converse feature of foot eversion after lateral plantar stimulation. Jacobsohn and Caro (1912) reported a new reflex from the sole in a majority of patients, though not all with organic disease of the nervous system, consisting of a twitch in the lateral portion of the quadriceps muscle – this is no other than the response in the tensor fasciae latae, first described by Brissaud (1896). Puusepp (1923, 1924) considered abduction of the little toe pathognomonic for a lesion of the extrapyramidal system, but could not convince Babinski (1924).

Finally, it is understandable that neurologists have also looked for extension of the thumb after palmar stroking. Böttiger (1902) found it in a few patients with pyramidal lesions, but this has not been substantiated.

THE PLANTAR REFLEX IN INFANTS

Contradictory findings

Babinski (1897, 1898) found his upgoing toe phenomenon present in new-born infants and used this as an argument for its association with disturbances of the pyramidal tract, as this fibre system is not yet fully developed after birth. In subsequent publications, however, the hallux responses in this age-group were also reported downward or mixed, and this instigated new series of examinations – again conflicting or inconclusive. Rather than adding another set of observations to the thousands already on record, I shall try to analyse the reasons for the diverging results, displayed in table III.

Not included in the table are some observations which went back even beyond the normal neonatal period. Krabbe (1912) happened to be present at the birth of a 4-month-old fetus, and found to his surprise a downward response (of the small toes). Minkowski (1922, 1923) undertook a systematic study of the plantar reflex in the fetal period, and distinguished no less than five stages preceding the full-term period.

The grasp reflex of the foot

One important reason for the contradictory findings concerning the plantar response in babies is the interference of the grasp reflex, which has no counterpart in adults, except under specific pathological conditions (see p. 37). The grasp reflex of the toes can be elicited by a slight moving stimulus or even by pressure alone, and is generally present during the first year of life (Sittig, 1932; Brain and Curran, 1932).

The few studies which take account of this specific infantile downward response of the toes invariably mention dorsiflexion of the hallux and other digits after more vigorous stimulation: Lewy (1909 b), van Woerkom (1910, 1911), Minkowski (1923), and more recently Dietrich (1957), Willemse (1961) and Schoch (1967). In other words, it depends upon the intensity of the stimulus whether the toes go up or down.

The flexion reflex

Many authors comment on the difficulty of interpreting the plantar reflex in young children, caused by continual spontaneous, 'pseudo-athetoid' movements. As most studies dealt with reflex movements of the toes in isolation and did not use effects in other limb segments as a

TABLE III
STUDIES ABOUT THE PLANTAR REFLEX IN INFANTS AND YOUNG CHILDREN

(N = new-born infants, G = grasp reflex, L = longitudinal study)

Author(s)	*Year of publication*	*Number of children studied*	*early plantar response*			*age (in years) at which change to normal adult form occurs*
			(almost) invariably up	*(almost) invariably down*	*unpredictable: up, down or absent*	
Babinski	1897;'98	? N	x			–
Collier	1899	100	x			2-3
Cohn	1899	'a few' N	x			–
Schüler	1899	?			x	–
Passini	1900	100	x			$\frac{3}{4}-1$
Walton and Paul	1900	40 N			x	>1
Finizio	1900	500 N		x		–
Crocq	1901	'numerous'	x			2-10
Homburger	1901	? N			x	–
van Epps	1901	100			x	1-2
Morse	1901	254			x	>2
Cattaneo	1902	180			x	>2
Specht	1902	30 N	x			–
Munch-Petersen	1902	200	x			5-12
Pfeifer	1903	64			x	1-3
Marinesco	1903	? N	x			2
Léri	1903	155	x			1-3
Barnes	1904	?	x			2
Bertolotti	1904	?	x			–
Engstler	1905	1000	x			2
Laurent	1905	110	x			$\frac{1}{2}$-1$\frac{1}{4}$
Lewy	1909b	? N	x	(x G)		1-3
Fleischner	1910	500	x			1-5
van Woerkom	1910;'11	?	x	(x G)		–

Bersot	1920,'21	?			x	$1\frac{1}{2}$
Burr	1921	69			x	—
Rudolf	1922	101 N			x	—
Feldman	1922	500			x	1-3
Minkowski	1923	7 N	x	(x G)		—
de Angelis	1923	88 N			x	—
Lantuejoul and Hartmann	1923	160 N			x	—
Wolpert	1926	48			x	—
Schlesinger	1927	?	x			1-2
Zador	1927	80			x	$\frac{3}{4}$-1
de Bruin	1928	194			x	1->4
Waggoner and Ferguson	1930	182	x			$\frac{1}{2}$-1
Wolff	1930	60 L	x			7/12
Bendix	1931	184		x		—
Chaney and McGraw	1932	100 N	x			—
Dosužkov	1932	12	x			$\frac{1}{2}$ - 4
McGraw	1941	75 L	x			$\frac{1}{2}$-$2\frac{1}{2}$
Dietrich	1957	103 + L	x	(x G)		1-2
Roedenbeck	1958	560		x		0
Brain and Wilkinson	1959	35	x			$\frac{1}{2}$-2
Willemse	1961	138 N	x	(x G)		—
Schoch	1967	?	x	(x G)		—
Hogan and Milligan	1971	100 N		x		—
Rich et al.	1973	124 N		x		—
Touwen	1975	51 L	x			1-$1\frac{1}{2}$
Katiyar et al.	1976	561 + L	x			$\frac{1}{2}$-1

reference, it is not surprising that the distinction from spontaneous toe wriggling was problematical. Arbitrary restrictions such as counting only the very first movement (Hogan and Milligan, 1971) do not solve the problem (Landau, 1971). The neglect of the synergistic background of upward reflex movements of the toes is the second major factor which has contributed to the chaos about the infantile plantar reflex.

Although Babinski (1898) and some of his contemporaries (Collier, 1899; van Woerkom, 1910, 1911) clearly described dorsiflexion of the hallux in children after a vigorous stimulus as part of what we now call the flexion reflex, preoccupation with toe movements alone has been even more persistent in the pediatric literature than in adult neurology. For example, in a fairly recent manual for the neurological examination of the new-born infant (Prechtl and Beintema, 1964) the Babinski reflex appears under one heading and the withdrawal reflex under another. Since the plantar reflex became established in the pediatric examination, the identity of the upgoing hallux response with the withdrawal reflex has been stressed only by McGraw (1941), Brain and Wilkinson (1959), Willemse (1961) and Schoch (1967). Willemse peremptorily denies that this upward toe response of new-borns is similar to the Babinski sign in adults. He defines the latter as slow dorsiflexion of the hallux alone, with fanning of the other toes. The distinction is shared by Chaney and McGraw (1932) and Touwen (1975). But such a conclusion about adults can hardly follow from a study of infants, however detailed, and is refuted by a wealth of evidence which has been reviewed earlier in this chapter.

The normal plantar response and walking

It is rather obvious to suppose a connection between the change of the plantar response to the normal adult type and the beginning of walking, because these two events occur more or less at the same stage of normal development. Leaving all speculations aside (these are relegated to the next section), only observations in individual children will be taken into account here.

Collier (1899) and Barnes (1904) intimate that reversal of the plantar reflex is delayed in children who walk late. Some even draw a parallel between the direction of the reflex with the position of the great toe during locomotion (Munch-Petersen, 1902), and examples are cited of a persistent upward response in children wearing sandals (Schlesinger, 1927; Stolte, 1933).

On the other hand, Feldman (1922) and de Bruin (1928) failed to demonstrate a fixed coincidence of a changing toe response and the onset of walking in their series of normal children. Tournay (1922) records the curious case of one healthy infant (probably his own) in whom the Babinski signs disappeared at the age of about six months – apparently not related to walking, but with a time lag between right and left similar to that in the previous 'discovery' of the hands. Conversely, a normal plantar response in rachitic children who were yet unable to support themselves on their legs was observed by Passini (1900) and Engstler (1905). Sehestedt (1933) examined sixty-two older children who had been bedridden for months or years from 'surgical tuberculosis' and found a Babinski sign in ten. These children may not have been free from involvement of the brain or spinal cord, and persistent upgoing toe signs and delayed walking can of course both result from brain damage. There seem to be no distinguishing features between the physiological and pathological infantile response (Holt, 1961).

We can conclude that, although there is probably some physiological connection between the onset of walking and the appearance of the adult plantar response, as both changes depend on the functional development of descending pathways, a direct relationship has not been borne out by observations in individual children.

TELEOLOGICAL SPECULATIONS

Science or fiction?

Whether teleological considerations have a place in scientific thinking depends upon the definition of science one adheres to. Many contemporary biologists regard any discussion of 'design' as adverse to the objectivity of Nature and therefore as outside the realm of science (Monod, 1970). On the other hand it is a basic principle that, in general, all beings (it is difficult to avoid the word 'creature' here) strive towards survival and reproduction. It is clear that many biological features are 'useful' here. When one thus reverses cause and effect and substitutes 'use' for 'purpose', it becomes much more acceptable to ascribe a function to any particular biological structure or phenomenon – provided the survival value is evident (Lorenz, 1963). All other explanations are unfalsifiable, and, in consequence, incompatible with an operational definition of science. Now it is self-explanatory that animals or humans without legs, eyes or pain are at risk in the process of natural selection, but it is difficult to see how absence of the plantar toe reflex (or of the knee jerk, for that matter) interferes with fulfilment of primary biological aims.

During the first half of this century, guesswork about the purpose of reflexes was more respectable than it is today, although it was often veiled under terms like 'physiological significance'. The following survey is given mainly for historical reasons.

The Babinski sign

1. *'Defence reflex', 'flight reflex'* or *'withdrawal reflex'*. Most authors who use one of these expressions include the activity of all other physiological flexor muscles of the leg: van Gehuchten (1900 a), van Woerkom (1910, 1911), Noica (1912), Walshe (1914), Wertheim Salomonson (1918, 1920), Babinski himself (1922), Kugelberg (1962), Schoch (1967). To regard the flexion reflex of the lower limb as a protective mechanism is compatible with the biological principles set out above; in any case Sherrington (1906) held such a view. It is more risky and even superfluous to assume a special, be it abnormal, defensive function for the upgoing toe alone (Goldstein, 1941). Davidson (1931) tried to reject all protective connotations with the indirect argument that painful stimuli could also produce downgoing toes.
2. *'Grasp reflex'* (with references to our simian ancestors): Hahn (1911), Astwazaturow (1923), and also Rudolf (1922), who examined the plantar reflex of almost all vertebrate animals in the Regent Park Zoo. He found an upward response in primates, but Fulton and Keller (1932) could confirm this in no way.
3. *'Locomotor reflex':* Marie and Foix (1912, 1915), van Woerkom (1912), Meyers (1920), Rabiner and Keschner (1926), Yakovlev and Farrell (1941), Droogleever Fortuyn (1958), Callens (1959), Scholten (1965).
4. *'Atavistic climbing reflex':* Friedman (1920), Wartenberg (1947).
5. *'Swimming or hopping reflex',* as in amphibians (Fay, 1955).
6. *'To withdraw from wet or sticky substances between the toes'* (Hoff and Breckenridge, 1956).

The normal plantar response

1. *'Grasp reflex':* Strümpell (1899), Lewy (1909 b), van Woerkom (1911), Babinski (1922), Goldstein (1941), Schoch (1967).
2. *'Protective reflex':* Collier (1899), van Woerkom (1912), Yakovlev and Farrell (1941).
3. *'Locomotor reflex':* Kalischer (1899), Munch-Petersen (1902), Noica (1912), Wertheim Salomonson (1918, 1920). Apart from theoretical considerations, it has been set out in the previous section that reversal of the toe reflex in children is not directly related to walking.

CONCLUSIONS

1. The term 'plantar reflex' was known long before Babinski studied the toe responses, and stood for flexion in ankle, knee and hip following stimulation of the sole.

2. Toe movements were occasionally noted as part of the plantar reflex, but these received little attention and it was a common belief that dorsiflexion was found in health as well as in disease.
   Babinski discovered that plantar flexion of the toes was the normal response, and that the reverse phenomenon, of the great toe in particular, occurred only in patients with affections of the pyramidal tract (as opposed to hysterical weakness) and in infants.

3. Abduction of the small toes following stimulation of the sole was one of the many accessory pyramidal signs noticed by Babinski. He did not regard it as indispensable for a pathological plantar reflex, nor does this follow from any objective study, and it is misguided traditionalism to think so.

4. In the Netherlands the normal plantar flexion of the toes is erroneously linked with the name of von Strümpell.

5. The upgoing toe sign is intimately related to the flexion synergy of the lower limb; that the (anatomical) toe extensors are flexor muscles in a functional sense (and vice versa) is corroborated by Sherrington's animal experiments.

6. A Babinski sign without concomitant reflex effects in more proximal muscles appears to be rare; the reverse is common.

7. The normal plantar response is a unisegmental reflex, comparable to abdominal and cremasteric reflexes.

8. The numerous alternative methods of producing an upgoing toe sign, often advanced as competitive signs, all stem from a basic misunderstanding of its intimate relationship with the flexion reflex; that this entire synergy could be evoked by various superficial and deep stimuli had been known for a long time.

(cont. next page)

9. Infants under one year of age generally show a brisk upgoing toe sign with a complete flexion reflex, at least after sufficient stimulation. Statements that infants do not show a Babinski sign arise either from misinterpretation of the plantar grasp reflex or from the unwarranted assumption that the Babinski response in adults is an isolated phenomenon.

10. Most speculations about the 'purpose' or even 'use' of plantar reflexes defy scientific scrutiny, but it is not unreasonable to assume that the flexion synergy of the lower limb (which includes the Babinski sign) serves a protective function.

CHAPTER II

INTERPRETATION OF PLANTAR REFLEXES: VARIATION AND BIAS

'Anyone who fancies that this applies only to his colleagues should be urged to participate in an observer-error test as a chastening experience.'

(Editorial, Lancet 1954)

INTRODUCTION

Observer error: examination or interpretation?

'Most of us assume that, except in occasional borderline cases, the signs we observe are present and those we do not observe are absent', wrote Fletcher in 1952, and went on to show a vast disagreement between different observers about the physical signs of emphysema. For plantar reflexes, McCance et al. (1968) also found a low reproducibility of the initial response, comparing the results of two physicians as well as those of the same observer on two different occasions (with an interval of one week). The diverging conclusions about the presence or absence of these physical signs are, in principle, the sum of differences in technique of elicitation and differences of interpretation, although variations in the patient cannot always be excluded.

Variability of interpretation alone can be studied when the material is presented in some recorded form: X-ray pictures (Garland, 1959 – review; Bull et al., 1960 – atherosclerosis on cerebral angiograms), electrocardiograms (Davies, 1956), isotope scans of the brain (Abbassioun et al., 1966), and echo-encephalograms (White et al., 1969). The list is not meant to be complete – for a recent review see Koran (1975). All these studies showed an appreciable inter-observer variation and, when considered, intra-observer error as well. Presentation of identical material to study interpreter error is not necessarily restricted to technical investigations where permanent records are usual – most physical signs can be recorded in some way. For plantar reflexes, where film or television is the obvious way, this method was chosen for two reasons (apart from the practical advantage). First, many textbooks and other sources rather emphasize the technique of stimulation, implying that this is the main problem in plantar reflexes. It is therefore interesting to investigate how much variation remains when this factor is eliminated. Furthermore, the use of uniform pictures was mandatory for the second question in this part of the study: the influence of circumstantial information.

Bias

Most people are or have been familiar with puzzle pictures in which one can recognize various shapes such as faces or animals, but only after having been guided by the text (Johnson, 1955). Another example is the animal head shown in figure 2. Wittgenstein (1953) used it as an example in a discussion about perception, and commented that if this shape is seen surrounded by pictures of ducks, and a similar one is shown amidst rabbits,

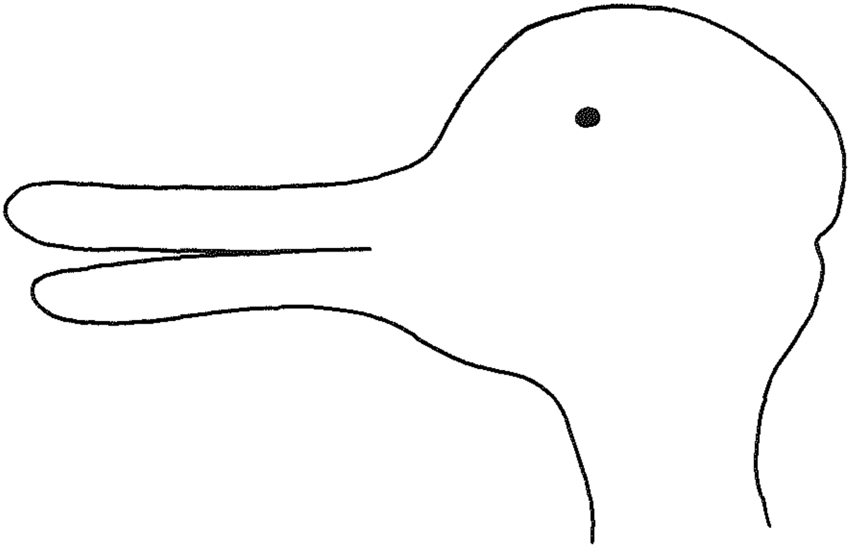

FIGURE 2

The 'duck-rabbit'

the congruence may pass unnoticed (it is appropriate to our subject that the two animals look to opposite sides).

Thinking along this line, it is not unreasonable to entertain the possibility that physicians may be guided by extraneous influences when asked to express an opinion on a doubtful sign or diagnosis. Bias in clinicians has already been suspected in some specific respects. Sexism is one, in particular the tendency to regard 'women's troubles' as psychogenic (Lennane and Lennane, 1973; Kelly, 1973). The aptitude to confirm the findings of seniors is another: Gross (1971) called attention to the ubiquitous occurrence of the 'Emperor's clothes syndrome' in teaching hospitals and claimed it was even epidemic in cardiology and neurology departments. A similar readiness to 'play safe' might bias the interpretation of equivocal results from physical examination or technical investigations towards more pertinent evidence from other sources.

Nevertheless, proof of these phenomena is scarce. White et al. (1969) demonstrated a powerful influence of suggestion on the reading of echo-encephalograms: the frequency of reported midline shifts decreased following a suggestion that most recordings with validated shifts had been removed from the series, while in fact only normal echograms were withdrawn. No such studies are known about physical signs.

The possible role of prejudice in the clinical interpretation of plantar reflexes was included in this study because its presence would require more objective methods. Thus the question was whether the idea that a Babinski sign ought to be there or not, on the strength of information about other signs and symptoms, biased the answer 'up' or 'down'.

METHODS

Subjects

Thirty neurologists from a Dutch university hospital rated a number of plantar reflexes on film, without being given any additional facts ('no information' group). Most of them had a few years experience in clinical neurology, some more, others less. The variability of interpretation was studied in this group, and these subjects also served as a control group for the neurologists who saw the films after some information about other signs and symptoms.

The 'information' group consisted of twenty other neurologists from two other university hospitals. For practical reasons they were examined in three sub-groups, but the instructions remained identical. It was explained that this was a study about the interpretation of plantar reflexes and that some of these reflexes were to be shown on film, accompanied by some other data in order to approach the clinical situation as well as possible.

Films

Nineteen short films, each showing a right foot from the medial side, formed the basis of the stimulus material. The films were separated by title-numbers. Except for one subgroup (six subjects) from the 'information' group, the films were shown on television. Plantar stimulation could be seen and was performed three times for every foot. Within the sequence of 19 films, two films were repeated: nr. 4 was identical with nr. 15 (film A), nr. 7 with nr. 18 (film B). These two duplicated films were the actual object of study ('test films'). All three 'reflexes' in either test film were simulated equivocal responses: slight up-and-down movements of the great toe. The other 15 films only served to cover up the two repetitions ('cover films') and consisted of downward, upward, equivocal and absent hallux movements in about equal proportions (true or simulated reflexes).

Accompanying information

A slide with a fictitious abstract of history and physical signs (minus the plantar reflex) introduced every film fragment for the 'information' group. The 19 abstracts were chosen from a stock of 36; three separate neurologists arranged these in nine groups of four, according to their 'Babinski-value'. The two films which occurred twice were on one occasion preceded by 'high-value-information' (I_{up}) and at the other presentation by 'low-value-information' (I_{down}), from the opposite end of the scale (see table IV). The order was reversed for the two test films, to control time effects: A (I_{up}), B (I_{down}), A (I_{down}), B (I_{up}). The remaining 15 abstracts were chosen from the whole range of 'values' and were assigned more or less at random to the cover films. I avoided mis-matches between the appearance of the foot and the age or sex given on the slide.

Recording of interpretations

All subjects were given a list of the patients' numbers, each number being followed by five possible choices: unmistakably upward, possibly upward, neither upward nor downward, possibly downward and unmistakably downward. They marked only one answer for each foot, according to their own opinion. At the end of every sitting the participants were asked to write down any comments they might have.

RESULTS

Cover films

The subjects' marked choices for all films were converted into numerical scores. With regard to the fifteen single films, showing various types of reflexes, the 'information' group and the 'no information' group showed no significant difference in mean ratings (Mann-Whitney, $z = 0.15$, not significant).

In the remainder of this chapter we shall only deal with the two duplicated *test films,* showing equivocal plantar reflexes.

Variation within and between observers ('no information' group)

Table V shows how the subjects in the 'no information' group interpreted the two test films on either occasion. The numbers within the diagonals

TABLE IV
INFORMATION PRECEDING THE TEST FILMS
(the Roman numerals indicate the 'Babinski-value' accorded by each of the three judges on a nine-point scale)

first presentation	second presentation
Film A: information suggesting Babinski sign (VIII, IX, IX) **widow, 42 years** *history* - hypertension for 10 years (treated with moderate success) - 1 day prior to consultation weakness of R arm after waking (noticed while combing); later that day dragging of R leg *examination* - slight obtundation - R-sided weakness of face, arm and leg; no hemianopia - tendon jerks rather weak on L side; on R slightly brisker?	Film A: information suggesting normal plantar response (I, I, II) **housewife, 22 years** *history* - for 6 months attacks of absent-mindedness, lasting a few minutes: perceives conversation as very remote – attacks can be aborted by shaking head - occasionally impaired vision, as if through a tunnel *examination:* normal
Film B: information suggesting normal plantar response (I, I, II) **office clerk, 36 years** *history* - 3 days earlier, after waking, suddenly unable to close L eye and to move mouth on L side - faint ache above the eyes for last few weeks *examination* - BP 180/105 - subtotal paralysis of all facial muscles	Film B: information suggesting Babinski sign (IX, IX, IX) **welder, 25 years** *history* - numbness and heaviness in R leg for 5 weeks; 4 years ago similar complaints – improved when he stopped playing soccer - while attending technical school two episodes of failing vision in L eye, for few weeks; ophthalmologist could find no cause *examination* - L eye: pale disc, acuity 8/10 - symmetrical horizontal gaze nystagmus - abdominal reflexes absent - R leg: moderate weakness of flexors, spasticity of quadriceps; marked hyperreflexia

TABLE V
RATINGS OF 'NO INFORMATION' GROUP

FILM A

		second presentation					
		↑	⇡	—	⇣	↓	
first presentation	↑						0
	⇡		2		3	1	6
	—			1	2		3
	⇣		2	2	7	4	15
	↓			2	3	1	6
		0	4	5	15	6	30

FILM B

		second presentation					
		↑	⇡	—	⇣	↓	
first presentation	↑		1		1		2
	⇡	1	13	2	3		19
	—			3	1		4
	⇣	1	1	1	2		5
	↓						0
		2	15	6	7	0	30

TABLE VI
RATINGS OF 'INFORMATION' GROUP

FILM A

Second presentation

BIAS →

first presentation

BIAS

	↑	⇡	—	⇣	↓	
↑				1	1	2
⇡				4	2	6
—		1		1	1	3
⇣		1		4	2	7
↓				1	1	2
	0	2	0	11	7	20

FILM B

second presentation

← BIAS

BIAS

first presentation

	↑	⇡	—	⇣	↓	
↑						0
⇡	4	9	1			14
—	1		1			2
⇣		2				2
↓		1		1		2
	5	12	2	1	0	20

indicate consistent ratings. It is apparent that many observers fell short of this ideal line. Cohen (1960, 1968) developed a statistical method to measure observer agreement – a method which takes into account the agreement to be expected by chance, and which also weighs the extent of possible disagreements. If we apply this measurement to the intra-observer variation between the first and the second presentation, displayed in table V, we find that Cohen's factor 'weighted kappa' (ranging from 0 for chance agreement and 1 for full agreement) is 0.09 for film A and 0.28 for film B. These very low kappa values indicate that the usual clinical interpretation of plantar reflexes can be meaningless in some cases.

The variation between different observers for the two films is indicated in table V by the totals in the margin. Because individual inconsistencies compensate each other to a large extent, the difference between ratings on the first and the second presentation is much less marked for the group as a whole. But the range of opinions is considerable, and the frequency distributions for both films have some characteristics in common:

1. All ratings were distributed among four of the five choices offered: one extreme of the scale was not opted for (unmistakably upward for film A, unmistakably downward for film B).
2. About one half of the observers agreed about one particular choice (possibly downward for film A, possibly upward for film B), while the other half were divided among the three other categories.
3. The category 'neither upward nor downward' is underrepresented in comparison with a symmetrical distribution.

This variation between observers is superimposed upon the intra-observer disagreement which has already been proved to be extreme for these films. The combined effect can only make the reproducibility of interpretation even lower.

Effects of preceding information

The results for the 'information' group are shown in table VI. Like the 'no information' group, only a minority of the observers rated consistently (within the diagonals). An important difference is, however, that here the inconsistent ratings are not scattered symmetrically on both sides of the diagonal, but are skewed towards one side, according to the combined direction of bias. To investigate this further, all relevant differences between the various mean ratings – shown in figure 3 – were subjected to the t-test. For both films, I_{up} resulted in significantly more pathological markings than I_{down} ($t_A = 3.10$, degrees of freedom (d.f.) = 19, $p<0.01$ and $t_B = 2.94$, d.f. = 19, $p<0.01$). Compared with I_0 (control group) at corresponding presentations, I_{up} also gave a significant deviation to the

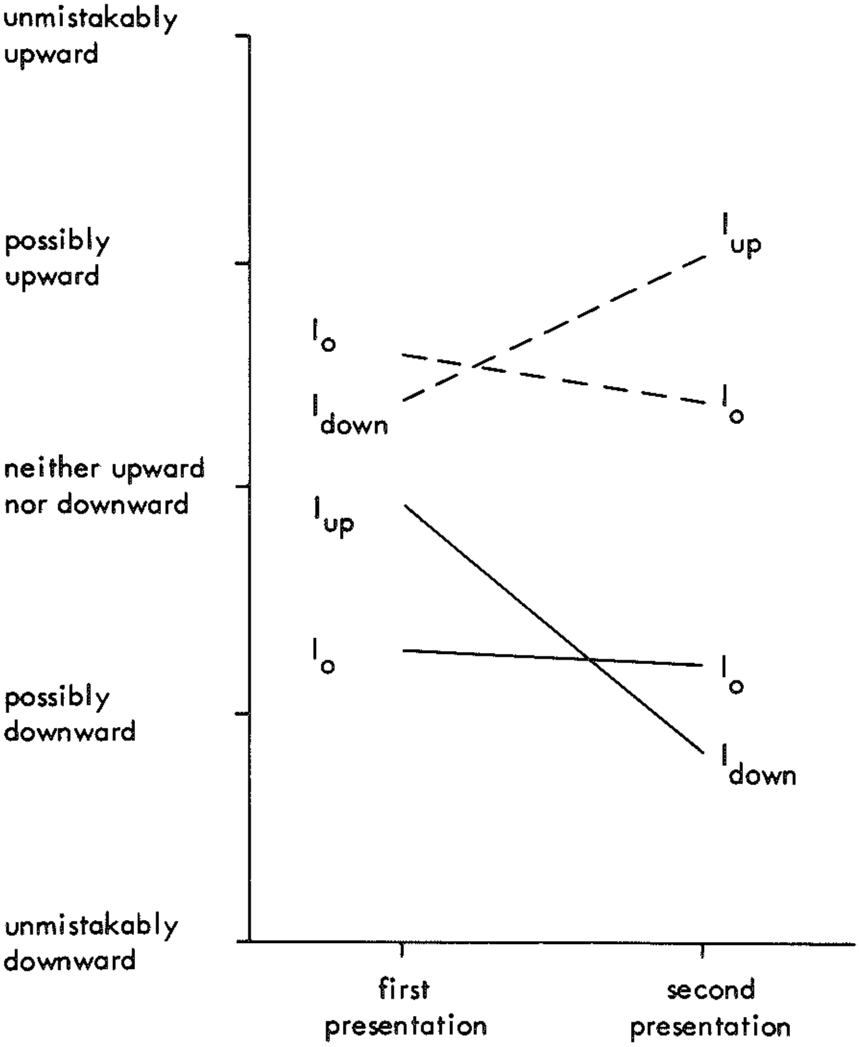

FIGURE 3

MEAN RATINGS FOR THE TEST FILMS

——— = *film A*

- - - - - - = *film B*

I_o = *no information*

I_{up} = *information suggesting Babinski sign*

I_{down} = *information suggesting normal plantar response* pathological side ($t_A = 2.03$, d.f. = 48, $p<0.05$ and $t_B = 2.59$, d.f. = 48, $p<0.01$). On the other hand, no significant differences were found between presentations with I_{down} and I_0, although I_{down} tended to give more normal scores ($t_A = 1.46$, $t_B = 0.74$, not significant). The differences between first and second presentation for I_0 did not reach significance ($t_A = 0.28$, $t_B = 0.95$).

Deviation against the tendency of bias was rare: it occurred in three observers for film A, and one of these showed the same paradoxical change for film B.

DISCUSSION

Disagreement within and between observers

Whatever the role of different techniques of stimulation may be, the interpretation of some plantar reflexes proves to be a tremendous problem in itself. This is at least true for the two sets of equivocal toe movements chosen for this study: in the group of thirty unbiased subjects, there were not only considerable differences between observers, but, to start with, the observers agreed with themselves scarcely better than was to be expected by chance alone.

Of course, there are reflexes about which almost everyone agrees, and this was confirmed by the ratings on some of the cover films. Such reflexes may form a majority in day-to-day practice, but doubt or controversy about plantar responses is by no means rare. This study illustrates the extent of the problem in such cases.

Bias

The results in the 'information' group show that many neurologists have, to some extent, already made up their minds whether the great toe will go up or not, before they have even started to examine the plantar reflex. This is facilitated by the circumstance that undetermined answers appear to be unpopular as long as the toes move at all.

Differences between biased and unbiased subjects were much smaller when the preceding information suggested no organic disease (see figure 3). It is apparently easier to convince oneself that an equivocal plantar response represents the expected Babinski sign than to disregard out-of-place upward movements of the hallux. However, as table VI shows, there were no confident ratings of a Babinski sign when the preceding information did not suggest this.

Clinical experience appeared to be no safeguard against bias: four of the twenty subjects had been practising neurology for five years or more and in them the effect of foreknowledge was similar to that in the others, although their number was too small for conclusions about relative probabilities. At the other end of the hierarchy were a few subjects with only some months' experience, and among these were two of the three observers whose change of opinion went against the leading information; it is tempting to speculate that they were 'protected' by a relative ingenuousness. The third observer who showed a paradoxic variation later commented that he had actively resisted the influence of the preceding data.

Prejudice has also marred studies about technique of plantar stimulation. This is most clearly shown by the originators of rival signs (see Chapter I), who report almost unanimously disappointing results with Babinski's method. Bias may also have affected the conclusion of a recent report (Dohrmann and Nowack, 1973) that slow 'hockey-stick' stimulation (lateral plantar border and plantar arch) is the 'best' method to produce an upgoing toe sign: the authors were their own measuring instruments and did not state what they expected to find.

Need for objective criteria

As interpretation of equivocal plantar reflexes is so much subject to the combined effect of inconsistency and preconceived ideas, this is not only a reason for considerable modesty at the bedside, but at the same time it creates a need for better criteria. The arguments whether or not a doubtful upward toe movement 'counts' ought to be defined, not intuitive: a neurologist should not only be able to say that he thinks a particular reflex is pathological, but also *why,* in order to make discussion possible.

Previous investigations, cited in the first chapter, stress the intimate relationship between the Babinski sign and the flexion reflex. It is striking that the impact of these studies on everyday interpretation of plantar reflexes has apparently been negligible, as not more than one of the fifty participants regretted that the films did not show the leg. A fair number complained that the angle under which the foot was filmed did not enable them to see a possible 'fan sign' of the toes, but it has been emphasized earlier (p. 24) that there is little reason to regard this as a sign of disease.

It can be concluded, at least for the large sample of neurologists in this study, that existing practical rules for interpretation of equivocal plantar reflexes are insufficient and at best controversial. In the next two chapters I will attempt to delineate adequate criteria.

CONCLUSIONS

1. Interpretation of plantar reflexes can be a hazardous procedure, even when variation in technique is eliminated: aside from variation between observers, agreement of observers with themselves is in some instances hardly better than may be accounted for by chance alone.

2. Previous information about other signs and symptoms can, in some cases, determine the interpretation of plantar reflexes even before they have been examined.

3. As interpretation of equivocal plantar reflexes is so much a matter of hazard and prejudice, better clinical criteria are needed.

CHAPTER III

BABINSKI SIGN: STIMULUS AND EFFECTOR

'If one wishes to study the Babinski reflex, one is well advised to study the reflex of Babinski.'

(Landau, 1975)

INTRODUCTION

Recording the plantar reflex

An important step towards defining the pathological plantar response clinically is to make use of permanent records in some form, rather than relying on inspection of fleeting movements. Such a method might be used first in reference groups of unequivocally upward and downward toe responses, and might subsequently be applied to dubious cases.

Before the advent of techniques that could measure muscular activity itself, mechanical registration of displacement was the only method of obtaining a graphic representation of motor phenomena. Verger and Abadie (1902, 1904) were the first to apply this to reflex movements of the great toe. Their tracings showed that the Babinski sign as well as the normal response occurred simultaneously with contraction of tibialis anterior and tensor fasciae latae. Walshe (1914) confirmed the temporal relationship between the Babinski response and the flexion reflex, but Meyers (1920) found the Babinski sign to be synchronous with activity in 'other' extensor muscles, i.e. quadriceps and gluteus. Apart from being an indirect technique, mechanical recording has other pitfalls such as superposition of movements and delay of transmission: latencies for the Babinski sign varied from 0.1–0.14 seconds (Verger and Abadie, 1902) to 0.2–0.52 seconds (Herzog, 1918).

Electromyography and the Babinski sign: the extensor hallucis longus

When recording of electrical activity from muscles became feasible, it was necessary to choose a specific muscle as the effector of the Babinski sign: either the extensor hallucis longus in the lower leg or the extensor hallucis brevis on the dorsum of the foot. The choice was not particularly difficult, as in fact no one had ever considered anything else than the extensor hallucis longus (table VII).

The string galvanometer which had made electrocardiography possible was used by Wertheim Salomonson (1918, 1920) to record from the extensor hallucis longus muscle in patients with a Babinski sign. However, he found that only the beginning of the upgoing toe sign was accompanied by alternating currents, and most of the movement took place under electrical silence. A similar sequence of events was found for voluntary activity. Wertheim Salomonson concluded that the initial response ('tetanus') represented the Babinski reflex proper, while the ensuing movement ('tonus contraction') was secondary to it and might even take place in a separate part of the muscle. Today we know that these theoretical

TABLE VII
CLINICAL COMMENTS ON THE EFFECTOR OF THE BABINSKI SIGN, BEFORE THE FIRST ELECTROMYOGRAPHIC STUDIES

musculus extensor hallucis longus	*musculus extensor hallucis brevis*
Collier (1899) Kalischer (1899) Strümpell (1899) Oppenheim (1902) Specht (1902) Goldflam (1903) Friedlaender (1904) Noica and Sakelaru (1906) Marie and Foix (1912) van Woerkom (1911, 1912) Walshe (1914) Bersot (1920) Salomon (1921) Astwazaturow (1923)	no one

conclusions have proved to be incorrect, since they were determined by the inability of the string galvanometer to follow the 'interference pattern' of muscle unit potentials; these can be demonstrated only by electronic amplifiers and cathode ray oscilloscopes. At the same time, however, this example should induce us to keep an eye open for the snares of present techniques.

Electrical stimulation of the extensor hallucis longus, although not a recording method, must be mentioned in this connection, as the Babinski sign has been 'explained' by an increased chronaxy of this muscle (Bourguignon, 1925, 1927; Claude et al., 1927; Gidro-Frank and Bowersbuch, 1948). Leaving to one side the difficulty of seeing how muscle excitability can be altered by a lesion within the central nervous system, this explanation is another instance of how the concepts of physiology can be strait-jacketed by the limitations of a given technique.

Electromyographic recording of the extensor hallucis longus, however sophisticated, is reliable only when there is no interference from activity in neighbouring muscles. The use of surface electrodes, with their widespread pick-up (Basmajian, 1974) is therefore precluded, the extensor hallucis longus being covered by the tibialis anterior or extensor digitorum longus in almost its entire course (figure 4). In some recent studies, surface electrodes were nonetheless used for this purpose (Bathien et al., 1970; Mahoudeau et al., 1971; Bathien and Bourdarias, 1972), and it is scarcely surprising to learn that the results were similar in patients with and without a Babinski response.

Recording with needle electrodes from the extensor hallucis longus muscle in patients with a Babinski sign was first applied by Kugelberg (1948), and subsequently by Landau and Clare (1959). Landau and Clare made a comprehensive study of the activity in various foot and leg muscles during mechanical stimulation of the sole. They showed that the extensor hallucis longus was silent in normal subjects, but that in patients with a Babinski response this muscle was recruited into the flexion reflex synergy, at the same threshold as the tibialis anterior and extensor digitorum longus.

. . . or the extensor hallucis brevis?

The studies confirming the role of the extensor hallucis longus as the effector of the Babinski response were contradicted by Kugelberg et al. (1960). They reported reflex activity of this muscle in both normal and pathological cases, following delivery of electrical stimuli to the plantar surface (pulse trains of 30–40 ms duration). In contrast, the extensor hallucis brevis was active in normal subjects only when the toes were stimulated, and this muscle was never activated by plantar stimulation.

Grimby (1963 a) employed the same technique of stimulation (short pulse trains, here 20 ms) and recording (needle electrodes) in a further study of normal subjects. Again, reflex discharges in the extensor hallucis longus were evoked from various parts of the plantar surface as well as from the pad of the hallux, but in the extensor hallucis brevis only from the latter site. It was inferred by Grimby that reflex action in the extensor hallucis longus had little value as an index of disturbed function, and subsequent studies (Grimby, 1963 b, 1965 a,b; Grimby et al., 1966) were based solely upon activity in the extensor hallucis brevis (and its antagonist). However, the results were complex, and no exact boundary could be drawn between the electromyographic reflex patterns of patients with and without a Babinski sign. To relate the electrically evoked discharges to the toe response after conventional stimulation, the tracings were differentiated not only into 'flexor' and 'extensor' patterns (Grimby, 1963 a), but also into early and late parts (Grimby, 1965 a), and into degrees of contrast between effects of hallux stimulation and of plantar stimulation (Grimby, 1965 b). Changes were not parallel in these respects, and the action of different supraspinal mechanisms had to be invoked (Grimby, 1963 b, 1965 b).

Normal plantar response: flexor hallucis brevis

The problem of which muscle mediates the normal downward response of the hallux is much less controversial than that regarding the Babinski sign. Early clinical speculations might have ranged from the adductor and abductor hallucis (Kalischer, 1899) and interossei (Goldflam, 1903) to flexor hallucis longus (Pfeifer, 1904) and flexor hallucis brevis (Noica and Sakelaru, 1906), but results from electromyographic studies were unequivocal. Landau and Clare (1959) as well as Kugelberg et al. (1960) identified the normal plantar response with activity in the flexor hallucis brevis muscle. In both studies the flexor hallucis longus hardly responded to plantar stimulation.

Equivalence of electrical and mechanical stimuli?

Landau and Clare (1959) had not only found that the extensor hallucis longus was active only in patients with a Babinski sign, but also that the extensor hallucis brevis responded in normal subjects and patients alike. To account for these results which were diametrically opposed to his own findings, Grimby (1963 a) argued that the type of electrodes used by Landau and Clare might have been at fault. He himself used paired needle electrodes, whereas Landau and Clare had employed concentric needle electrodes for the extensor hallucis longus (too selective?) and surface electrodes for the extensor hallucis brevis (not selective enough?).

However, another and perhaps more important difference between the two methods was not brought to light. That is, Landau and Clare (1959) stimulated the sole by simple stroking, whereas Kugelberg et al. (1960), and subsequently Grimby (1963 a etc.) applied brief electrical stimuli to one site. In principle, short electrical stimuli (10–100 ms) are attractive, because voluntary reactions can be excluded at latencies less than 150 ms (Grimby, 1963 a). Nonetheless, whatever the theoretical advantages, the validity of electrical stimulation as a substitute for stroking the sole should be tested first of all. So far it has only been assumed that electrical and mechanical stimuli are equivalent where the plantar reflex is concerned. And this assumption may be questioned in view of the puzzling fact that a reflex movement which can be simple to the eye – a toe going either up or down – had become not only complex but almost invisible in electromyograms after electrical stimulation.

The aims in this part of the study were twofold. In the first place, to reconsider from electromyographic records during mechanical stimulation of the sole which muscle is responsible for the Babinski sign: extensor hallucis longus, extensor hallucis brevis, or both. Secondly, to investigate whether electrically induced reflex activity of the muscles concerned is as useful in signalling dysfunction of descending pathways as the mechanically evoked reflex has proved to be in the past eighty years.

METHODS

Patients

The investigations were performed in 22 patients with a Babinski sign and 49 control subjects. The controls were out-patients and hospital employees. They were accepted on the basis of not showing a Babinski sign (toe responses downward or absent on both sides), and freedom from any other abnormal neurological signs. In addition, the control group of nine subjects in the first part of the study included results from the normal leg of two patients with a unilateral Babinski sign. In the Babinski group only patients with a definite response were accepted. In all members of this latter group there was evidence of disease of the central nervous system. They will be referred to as 'patients'.

Stimulation

Mechanical stimuli were administered by the smooth, blunt handle of a patella hammer. The lateral plantar border and plantar arch were slowly stroked. It is generally accepted that stimulation of the medial plantar border is less effective in eliciting a Babinski sign, probably because the flexor hallucis brevis is directly activated in this way. Electrical stimuli were applied through a pair of needle electrodes, placed intracutaneously in the middle of the lateral plantar border, at about 1 cm distance. Each stimulus consisted of square wave pulses of 1.2 ms width and a frequency of 500 Hz, with a train duration of 10 ms.

Recording

The following muscles were studied:
- extensor hallucis brevis (EHB), only during the first part of the experiments
- extensor hallucis longus (EHL)
- tibialis anterior (TA)
- flexor hallucis brevis (FHB)

1. m. extensor hallucis longus
2. m. tibialis anterior
3. m. extensor digitorum longus

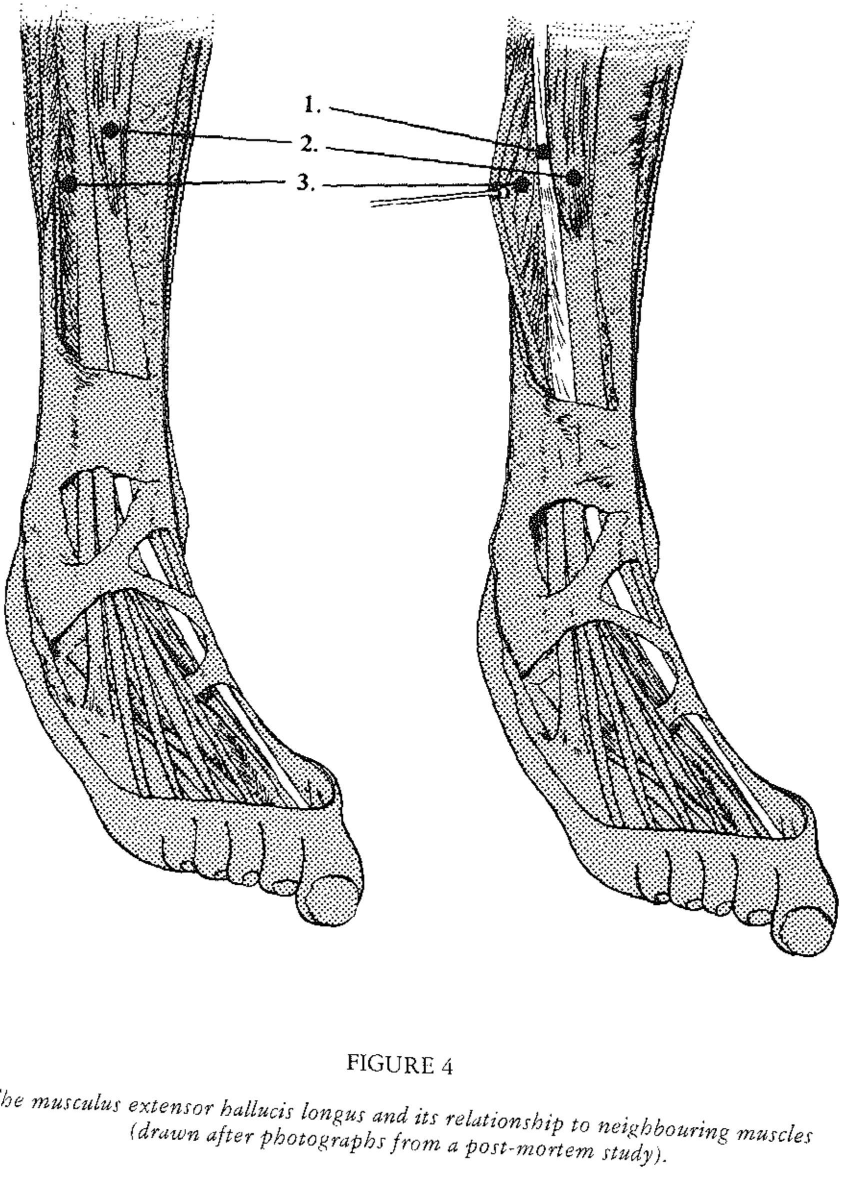

FIGURE 4

The musculus extensor hallucis longus and its relationship to neighbouring muscles (drawn after photographs from a post-mortem study).

Concentric needle electrodes were used in all muscles. The flexor hallucis longus was studied in a few patients, but this muscle did not show reflex activity. As these findings were in accordance with other work cited above, recording from this muscle was abandoned. The EHB, a small muscle, was first located by surface stimulation. The FHB electrode was introduced on the plantar side of the first metatarsal bone. The TA electrode was placed proximally. Needle position in these three muscles was checked by asking the subject to make the appropriate movement, and the electrodes were adjusted until an interference pattern of potentials was seen on the oscilloscope.

Correct positioning of the EHL electrode is of critical importance but proved to be most difficult. The needle was introduced laterally above the ankle, at a point dividing the middle and distal third of a line between lateral malleolus and caput fibulae (this point proved to be most convenient to reach the bulk of the EHL muscle in a post-mortem specimen; see figure 4). Placement of the tip of the EHL electrode was checked by passive movement of the hallux, by the pattern of action potentials on voluntary dorsiflexion, and by electrical stimulation through the recording electrode. These control measures were repeated during and after each experiment. Outward or inward movement of the electrode was limited by an adhesive strap around the needle itself and by another one securing its head to the skin.

Before each stimulus, care was taken that the muscles concerned were completely relaxed. Potentials were amplified and displayed on a two channel oscilloscope. Measurements were made from film.

RESULTS

Effector of Babinski sign

The reflex activity in EHL and EHB after mechanical stimulation was studied in seven patients and nine control subjects. A reflex was considered to be present when recruitment of motoneurones was seen on more than one occasion: large motor units are activated late in the reflex and disappear early (Henneman et al., 1974), so that all potentials together form a spindle shape on the oscilloscope screen. The results are shown in table VIII. EHL reflexes were present in all patients and in none of the control subjects. In contrast, EHB activity appeared in one third of the controls (not including the normal legs of the two patients) and was absent in more than half the patients. Figure 5 shows two illustrative examples. It can be safely concluded that the Babinski response is mediated by the EHL, and not by the EHB.

TABLE VIII
OCCURRENCE OF REFLEX ACTIVITY IN EHL AND EHB AFTER MECHANICAL STIMULATION

	EHL	*EHB*
Controls (n=9)	0/9	3/9
Patients (n=7)	7/7	3/7

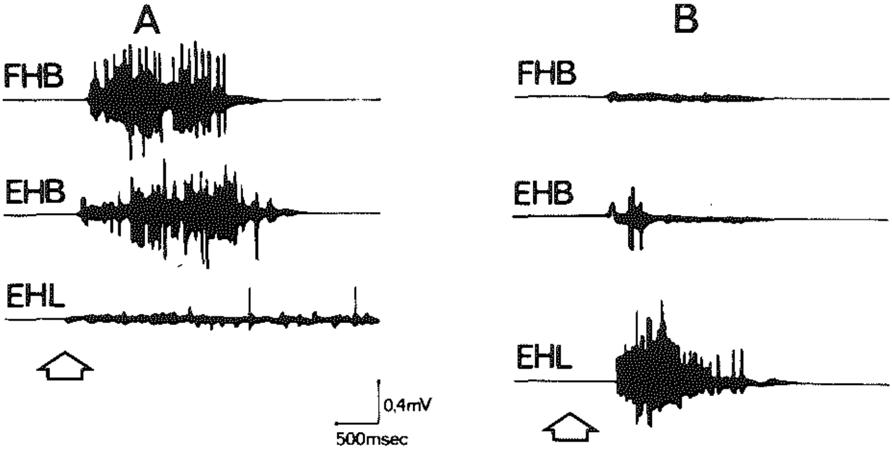

FIGURE 5

Reflex activity in FHB, EHB, and EHL after mechanical stimulation (arrows) *of the plantar surface in a normal subject* (A) *and in a patient with a Babinski sign* (B).

Mechanical stimulation: effects in EHL and FHB

The second half of this study concerns 15 patients and 40 control subjects. Recording from the EHB was abandoned, as activity in this muscle proved irrelevant to the occurrence of the Babinski sign. Attention was focused on activity in the two 'reins' of the plantar reflex, EHL and FHB. The effects of mechanical stimulation were studied first.

In general, reflex activity appeared within 0.5 to 1 second after the beginning of stimulation. No efforts were made to determine exact reflex latencies after mechanical stimulation (Magladery et al., 1958; Drobný et al., 1974); such measurements are impeded by a variable delay between the moment the skin is touched and the moment the stimulus becomes maximal. When stimulation had ended, the activity dwindled within a few hundreds of milliseconds. If potentials continued to appear, this could be attributed to voluntary contraction: repetition of the procedure when the subject was sufficiently relaxed then no longer showed the prolonged activity. This rule evidently does not apply if flexor spasms are present, but these can be recognized clinically.

There was a strong tendency towards reciprocal reflex activity: when the FHB was active (as in most controls) the EHL was not, and vice versa. Recruitment of FHB motor units was observed in only one of the 15 patients, a 46-year-old male with traumatic brain injury.

In view of the findings in the initial experiments, it was not surprising that recruitment of the EHL was found in all patients with a Babinski sign (table IX). EHL reflexes were also synchronous with activity in the TA in all patients (table X).

TABLE IX
OCCURRENCE OF REFLEX ACTIVITY IN EHL AFTER MECHANICAL AND ELECTRICAL STIMULATION

	Mechanical	*Electrical*
Controls (n=40)	(1)*/40	14/40
Patients (n=15)	15/15	10/15

* this result was equivocal (see text)

TABLE X
OCCURRENCE OF REFLEX ACTIVITY IN EHL AND TA AFTER MECHANICAL STIMULATION

	TA and EHL	*EHL alone*	*TA alone*
Controls (n=40)	0/40	(1)/40	3/40
Patients (n=15)	15/15	0/15	0/15

In three control subjects, reflex activity of the TA was found to occur unaccompanied by contraction of the EHL. In one control subject recruitment of EHL motor units was repeatedly recorded. This 49-year-old man, complaining of non-specific headaches, had bilateral pes cavus and was apt to show voluntary upward movement of the big toe. The potentials in the EHL continued for seconds after stroking, FHB potentials were also recruited and even interrupted by EHL activity, and there was no co-activation of the TA. These features suggest that persistently insufficient relaxation was the cause of the EHL activity, rather than a pathological reflex mechanism.

Activity of the FHB was found in all control subjects with a downward toe response on inspection, although the density of the electromyographic pattern was not always proportional to the briskness of the clinical response. These relative differences may be due to shortcomings of the recording technique or to individual anatomical variations. Nine subjects in the control group were judged to have absent plantar reflexes on clinical grounds; three of these showed only minimal FHB activity, six none at all. This means, at least for the subjects in this study, that clinically absent plantar reflexes are genuinely absent and do not represent an undecided tug of war between EHL and FHB.

Electrical stimulation: effects in EHL and FHB

Brief electrical stimuli to the plantar surface were applied in the same 15 patients and 40 controls, and the reflex effects are shown in Table IX together with those after mechanical stimulation. A reflex was considered to be present when potentials appeared repeatedly within 150 ms of stimulation, showing an interference pattern that lasted for 50 ms at least. These criteria are more or less arbitrary; the consequences of defining them otherwise will be discussed below.

In five of the 15 patients with a Babinski sign, electrical stimuli failed to activate the EHL. In one of them this failure was confirmed in three subsequent experiments. He was a 56-year-old man with spastic paresis of the right leg following haemorrhage from an arteriovenous malformation in the left parietal parasagittal region. In another patient, a 50-year-old man with a paraparesis probably due to multiple sclerosis, there were very brisk flexor reflexes of both legs, which could be elicited even by stroking the plantar surface with a finger pad. But electrical shocks were insufficient to evoke a response even at maximal intensity (Figure 6, B). Of the three remaining 'false-negative' patients, one had sustained a severe cerebral concussion, both the others were suffering from paraparesis of unknown origin.

In the control group, electrical stimuli evoked EHL reflexes in about one out of every three subjects. One example (a healthy neurologist!) is shown in figure 6,A. It could be possible that in these control subjects the stimulus strength exceeded the intensities required for activating the EHL in patients, and this was investigated further. The stimulus strength (mean values when inconstant) was expressed in multiples of the tactile threshold

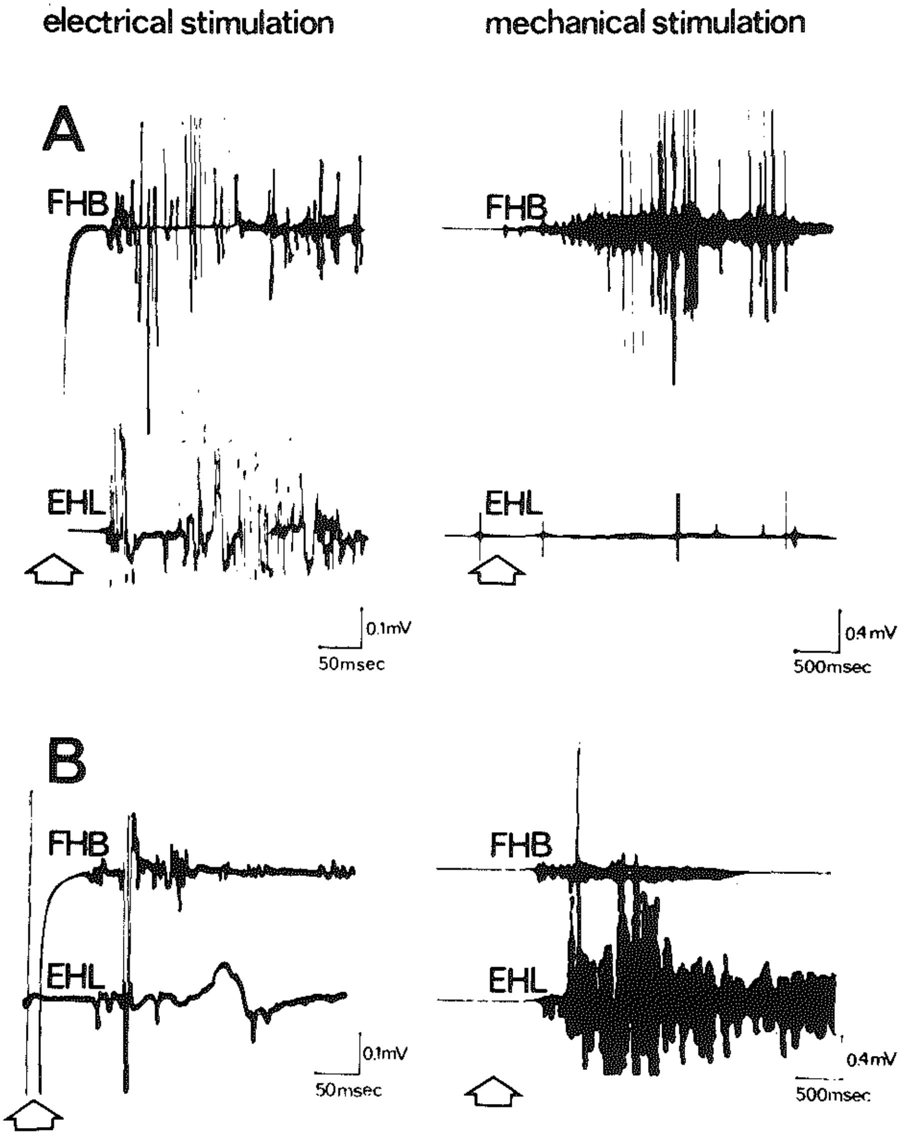

FIGURE 6

Reflex activity in FHB and EHL after electrical and mechanical stimulation (arrows) *of the plantar surface in a normal subject* (A) *and in a patient with a Babinski sign* (B).

value (the minimal stimulus intensity that could be felt at all by the subject in three trials, starting from both subliminal and supraliminal values). There are obvious objections to the estimation of the threshold for a spinal reflex by a sensory judgement, but this seemed a better method, especially for the control series, than estimating the distribution of current through the skin.

The range of threshold values in both groups is shown in table XI. One exceedingly high threshold for tactile sensation (1.60) in a patient with a spinal lesion was substituted by the mean threshold of the other patients. It is apparent that thresholds for EHL reflexes overlap considerably in both groups, and cannot be used to discriminate the 'false-positive' control subjects from patients with a Babinski sign.

TABLE XI
THRESHOLDS FOR EHL REFLEXES, IN MULTIPLES OF INDIVIDUAL TACTILE THRESHOLDS

	tactile threshold (L) in arbitrary stimulator units	*threshold for EHL reflexes*
controls (n=14/40)	0.55–1.30 (mean 0.77)	4L–15L (mean 8.6L)
patients (n=10/15)	0.55–1.25 (mean 0.81)	1.5L–10L (mean 6.3L)

The distinction between the effects of electrical stimulation in patients and controls can neither be improved by a change in the somewhat arbitrary criteria used to decide whether or not there is reflex activity at all (and some criteria are necessary since a single spike from a single motor unit can obviously not be counted as positive). If, for instance, one required shorter latency or longer duration of reflex discharge in the EHL, diminution of 'false-positive' controls would be nullified by an increase in number of 'false-negative' patients, and vice versa. Similarly, it is unlikely that altering the duration of the electrical stimulus will provide a clearer demarcation between EHL reflexes in patients and controls.

EHL reflexes evoked by electrical stimuli were often accompanied by simultaneous activation of FHB motoneurones: it occurred in all 14 control subjects with EHL reflexes (see figure 6,A), and in four of the ten patients. This synchronous activation of antagonists strongly contrasts with the reciprocal reflex patterns after mechanical stimulation of the skin.

DISCUSSION

Effector of Babinski sign

Recording from the extensor hallucis longus and brevis during mechanical stimulation of the sole has made it clear that the Babinski sign is mediated by the extensor hallucis longus. Activity in the extensor hallucis brevis is not essential, and it is also found in normal subjects. As we saw earlier, a similar conclusion was expressed by Landau and Clare (1959), but contradicted by Grimby (1963 a) on the basis of experiments involving electrical stimulation of the plantar surface. The results of the present study re-emphasize the principle – almost a truism – that the effector of any motor phenomenon can be determined with confidence only when the phenomenon is actually taking place, in this case when the great toe is going up after conventional mechanical stimulation.

The role of the extensor hallucis longus as mediator of the Babinski sign would have been at least probable from simpler observations than electromyographic recording, had it not become a matter of controversy. First, when a Babinski sign appears, the tendon of the extensor hallucis longus can be seen or at least felt to contract on the dorsum of the foot and hallux (figure 7). Secondly, it is hard to attribute the vigorous upward

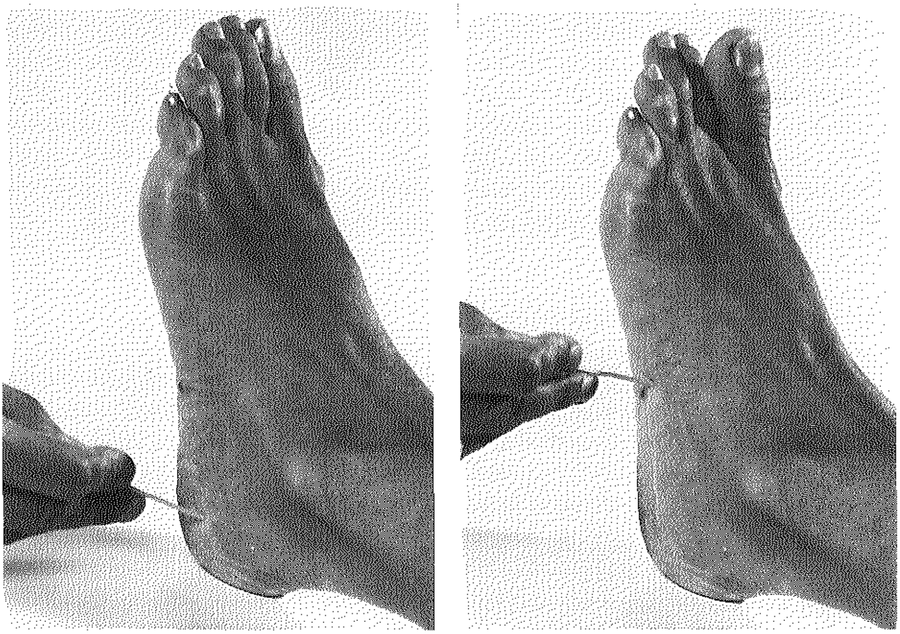

FIGURE 7. *Babinski sign – note the protrusion of the EHL tendon.*

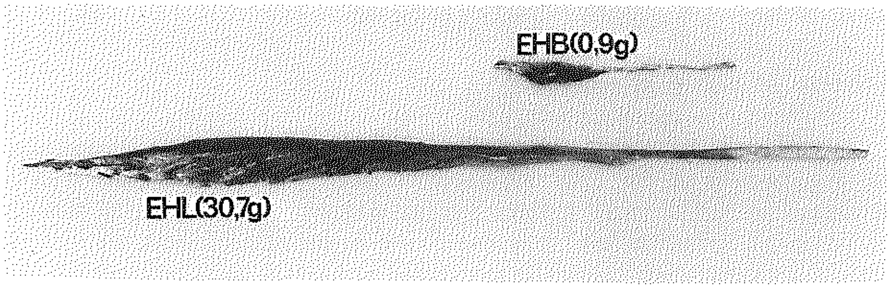

FIGURE 8

Relative dimensions and weights (after removal of tendons) of extensor hallucis longus and extensor hallucis brevis.

movement of the great toe that can be seen even in aged patients to the tiny extensor hallucis brevis muscle. In a post-mortem study of one human subject (adult of unknown age) the extensor hallucis longus weighed 30.7 grams, the extensor hallucis brevis 0.9 gram (figure 8).

Activity in the tibialis anterior always accompanied the Babinski sign. This not only confirms that the Babinski sign is part of the flexion reflex, it also implies that in patients the thresholds for both muscles are comparable, in keeping with the findings of Landau and Clare (1959). Concomitant action of the tibialis anterior might be a valuable criterion when plantar responses are equivocal. It is well-known that reflex dorsiflexion of the foot is not pathological on its own, and in this study the tibialis anterior was activated in three of the 40 control subjects.

Mechanical and electrical stimuli

Equivalence of electrical and mechanical stimuli with regard to the capacity to elicit normal or pathological activity in the toe muscles has been assumed not only by Kugelberg et al. (1960) and Grimby (1963 a), but much earlier by Lewy (1909 a) and Yoshimura (1909), who applied faradic currents to the lateral plantar border. Although Lewy (1909 a) reported a few 'unexplained' upgoing toe signs with this method of prolonged electrical stimulation, accordance with the results after mechanical stimulation was satisfactory. This parallelism cannot be extended to the brief electrical stimuli employed in the more recent studies: the extensor hallucis longus is activated in many control subjects (one third in the present series). This has in turn led to the erroneous supposition that the Babinski sign is mediated by the extensor hallucis brevis (Grimby, 1963 a). The complex and overlapping results in patients with and without a Babinski sign (Grimby, 1963 b, 1965 a, b) are easily explained by realizing that these represent activity in an irrelevant muscle, evoked by inappropriate stimuli.

In addition to the 'false-positive' controls, the present study also showed failure of electrical stimuli to activate the extensor hallucis longus in five of the 15 patients; in one of them this was observed on four separate occasions in the course of a few months. In this case both spatial and temporal summation of afferent impulses were necessary for activation of the extensor hallucis longus, as neither prolonged electrical stimulation at one site, nor simultaneous application of short pulse trains at several sites on the lateral plantar border were effective. Only when combined with subliminal mechanical stimulation a single pulse train could evoke a reflex in the extensor hallucis longus.

Another anomalous feature of electrical stimuli when compared with stroking the sole was the frequent co-activation of extensor hallucis longus and flexor hallucis brevis; this occurred in all controls with electrically induced reflexes in the extensor hallucis longus muscle, and in some of the patients. Mechanical stimulation did not cause synchronous activity in the two antagonist muscles in any of these subjects (18 in all). Co-activation of extensor hallucis longus and flexor hallucis brevis after mechanical stimulation was also rare in the entire group of patients and controls (2/55), but it is found more often in equivocal cases (see next chapter).

For the cases on hand it is obvious that the organization of reciprocal reflex patterns requires a stimulus of longer duration than a short train of pulses. This stands in some contrast to the reciprocal activity which Hagbarth (1960) found in various leg muscles of normal subjects after brief electrical stimuli at different skin sites: physiological flexor muscles were excited and their antagonists inhibited from most skin areas, except from those covering extensor muscles. Intrinsic foot muscles were not included in Hagbarth's study, but at any rate activation of the extensor hallucis longus from the plantar surface is a frequent exception to his rule. Dimitrijević and Nathan (1968) applied electrical stimuli to legs of paraplegic patients and recorded responses from all groups of leg muscles. They ascribed this absence of reciprocal innervation to lack of supraspinal control, but in the present study electrical activation of the extensor hallucis longus without concomitant activity in the flexor hallucis brevis occurred in patients only.

Generally speaking, I think that the results of this study justify caution in the interpretation of any effect evoked by electrical skin stimuli lasting only a few tens of milliseconds. Examples are:

- the two separate bursts of electromyographic activity which often appear (Kugelberg, 1948; Pedersen, 1954; Hagbarth and Finer, 1963; Shahani and Young, 1971);
- the determination of reflex thresholds, whether in spinal shock (McCouch et al., 1974) or as a 'pyramidal test' (Mahoudeau et al., 1972);
- the influence of skin stimuli on the recovery curve of the H-reflex (Gassel, 1970), the latter phenomenon being questionable in itself (Granit and Burke, 1973).

Even the theoretical advantage of electrical stimulation, viz. the possibility of distinguishing spinal reflexes from voluntary reactions by latency measurement, was not always valid: in legs deprived of voluntary innervation, latencies of 200 ms or more after electrical stimulation could be observed. Similar findings have been reported by Dimitrijević and Nathan (1968). A more reliable indication of the reflex character of activity after mechanical stimulation proved to be its cessation after the end of the stimulus; voluntary contraction tends to linger on for a few seconds afterwards. Flexor spasms do not conform to this rule, but can be recognized clinically.

Independently from my study, Nakanishi et al. (1974) have also found different effects of electrical and mechanical (in their case punctate) stimuli for plantar reflexes: electrical stimuli activated the extensor hallucis longus more easily, and could give rise to concomitant discharges in the short hallux flexor.

Punctate and moving stimuli

The finding that the clinician's stroke across the sole cannot be substituted by a short train of electrical pulses does not imply putting the blame on electricity as such – it is much more the application of a short stimulus at one site only that is 'unphysiological'. This is illustrated by the study of Nakanishi et al. (1974), who administered short mechanical stimuli to the plantar surface: one to ten pin pricks with a vibrator of 50 Hz (i.e. a stimulus duration of 20-200 ms). Muscular activity was recorded in the extensor hallucis longus and flexor hallucis brevis, as was done in the present study. The responses from three different parts of the sole were taken into account, but even then there remained an overlap between the results in patients and normal volunteers. Both groups had shown unequivocally opposite responses on conventional stimulation.

A probable reason why punctate electrical stimuli have been so uncritically used instead of the ordinary method is that the flexion reflex, including the Babinski sign, is supposed to serve a protective function (see

Chapter I), and that stimuli which are 'definitely noxious' (Kugelberg et al., 1960) should by implication be appropriate. This is as incorrect as the notion that the flexion reflex can be evoked only by stimuli which are 'noxious', i.e. painful in the average subject (Scholten, 1964, 1965). Anyone who has dealt with paraplegic patients knows that even a piece of cotton-wool can sometimes evoke a full flexion reflex (Walshe, 1914; Szapiro, 1958). Because of this, it should be stressed that association of the Babinski sign with activity in small myelinated fibres (A-delta) and unmyelinated fibres (C), as derived from latency measurements in conjunction with blocking techniques (Kugelberg, 1948; Ashby, 1949; Landau and Clare, 1959), does not necessarily implicate harmful stimuli in every case.

Of course stimulus intensity is not irrelevant, but most important of all is the combined spatial and temporal summation produced by the traditional moving stimulus. This summation of afferent impulses in the spinal cord determines the emergence of the characteristic reflex effects: in normal subjects usually activation of the flexor hallucis brevis, as opposed to recruitment of the extensor hallucis longus in certain supranuclear disorders.

Babinski empirically reached a similar conclusion much earlier. In his first publication of 1896 he mentioned 'piqûre' (pricking) of the plantar surface as the stimulus used, to be replaced in the main paper by stroking ('chatouillement').

CONCLUSIONS

1. The muscle which mediates the Babinski sign is the extensor hallucis longus, and not the extensor hallucis brevis.

2. The Babinski sign is invariably accompanied by reflex activity in the tibialis anterior; the reverse is not always true.

3. Electrical stimulation of the sole may activate the extensor hallucis longus in control subjects, and fail to do so in patients with a Babinski sign.

4. Electrical stimulation of the sole often evokes simultaneous activity in the antagonists for the plantar reflex (extensor hallucis longus and flexor hallucis brevis), whereas this co-activation is exceptional after mechanical stimulation.

(cont. next page)

5. In general, reflex effects produced by short pulse trains on the skin should be interpreted with caution.

6. Painful stimulation as such is not necessarily sufficient to evoke a possible Babinski sign, nor is a stimulus which causes a Babinski sign always painful in an average subject.

7. The reason why the effects of conventional stroking of the sole distinguish much better between patients and normal subjects than the effects of electrical stimulation is not that stroking is 'natural', but that it generates spatial and temporal summation of afferent impulses.

CHAPTER IV

EQUIVOCAL PLANTAR RESPONSES

'. . . le réflexe plantaire peut revêtir des caractères en partie pathologiques, en partie physiologiques.'

(Babinski, 1898)

INTRODUCTION

When is an upgoing toe a Babinski sign?

Quivering hallux responses after plantar stimulation are by no means exceptional. Most physicians react to an uncertain plantar reflex by stimulating again, or differently, according to their own favourite method – eponymous or not. The results from Chapter II have taught us, however, that the process of interpretation is heavily influenced by what the answer ought to be, rather than by what there is to see. Stimulation is of course important, but it should not be over-emphasized: re-testing the reflex may only provide time for bias to become effective, or may even be subordinate to this purpose. Rare indeed is the physician who goes on stroking the sole until he has proved that his first ideas were wrong.

A remedy against a 'blind eye' is to reinforce the visual information. This can be done by the introduction of criteria for the Babinski sign that are added to a mere upward movement of the great toe. Despite the prevailing concern with stimulation methods (see Chapter I: 'rival signs'), some advice has also been offered in the past on what to look for, although with little rationale:

1. *'The movement should occur at the metatarsophalangeal joint'* (Barnes, 1904; Bickerstaff, 1968). This criterion is meant to exclude passive movement by dorsiflexion of the foot. It is related to action of the extensor hallucis longus muscle, which proved to be vital in the preceding chapter. However, it is often less sensitive than inspection of the tendon of this muscle: the tendon has its insertion at the last phalanx and can cause movement in the distal joint only, or even tighten without any displacement at all.
2. *'The movement should be the first'* (Barnes, 1904; Bickerstaff, 1968; Hogan and Milligan, 1971). It is difficult to see why a patient cannot make a voluntary movement before the onset of reflex action.
3. *'The movement should be slow'* (Scholten, 1965; Mumenthaler, 1976). What is the limit of slowness? Moreover, reports of brisk flexion reflexes and Babinski signs abound (e.g. Walshe, 1914; Szapiro, 1960).

The flexion reflex

The fact that the Babinski sign forms an integral part of the flexion reflex in the lower limb has been discussed in detail in Chapter I. Later it was specified that the Babinski sign consisted in essence of recruitment of the extensor hallucis longus into synergy with the tibialis anterior (Landau and Clare, 1959; confirmed by the findings described in Chapter III).

Nonetheless, observation of activity in other leg flexors has rarely been applied as a tool in the interpretation of equivocal toe responses. At least this was true for all but one of a sample of fifty neurologists, who did not object to rating plantar reflexes from films which showed only the foot (see Chapter II). In published work there is also a paucity of allusions to the practical use of the flexion reflex in this respect. Some early writers even declare that any upward movement of the hallux should be discounted if it is not an isolated phenomenon (Homburger, 1901; Crocq, 1901; Levi, 1902; de Wilde, 1911). They heralded the present-day preoccupation with toe movements alone.

Only at the time of Babinski's discovery a few neurologists were able to make practical use of the connection between the toe responses and the more proximal effects which had until then constituted the plantar reflex. Collier (1899) stated: '... it is very important that all the muscles of the lower limb should be observed at the moment of stimulation'. And Koenig (1899, 1900) even employed a second observer, who '... focused his attention on the events which occurred higher up ...'. Finally, Babinski himself has always stressed the synergistic nature of his sign, although we have seen in Chapter I that he insisted on including the normal response in the flexion reflex. Babinski also perceived an important application: reflex movements of the great toe often coincide with contraction of the tensor fasciae latae, whereas this is never seen during voluntary retraction of the toes (Babinski, 1906 a, 1915 b, 1922). The same counsel was given by Specht (1902).

One of the aims of the study described in this chapter is to find out whether the present neglect of the flexion reflex as a whole in the interpretation of equivocal plantar responses is justified or not.

Procedures to 'enhance' the Babinski sign

Apart from stimulation itself, some indirect influences on the plantar reflex have been described in previous studies, often with the implication that these could be of use in dubious cases:

1. *Temperature of the foot:* little response in cold feet, reflex return on warming (Collier, 1899; Kalischer, 1899; Babinski and Froment, 1916; Noica and Radovici, 1919).
2. *Ischaemia of the leg:* immediately after restoration of normal blood flow, there is a transient enhancement of withdrawal reflexes (Babinski, 1911 b; Marinesco and Noica, 1913; Gilliatt, 1952).
3. *Tonic neck reflexes:* rotation of the subjects' head away from the

stimulated side diminishes extensor hypertonus and facilitates flexion reflex and Babinski sign (Walshe, 1923).

4. *Decubitus:* Babinski signs have been reported to disappear with the patient in a prone position (Guillain and Barré, 1916; Boveri, 1916). Some have attributed this change to proprioceptive effects of the knee flexion which is necessary to examine the plantar reflex after this fashion (Mankowsky and Beder, 1925; Zador, 1927; Kastein, 1937); a reverse phenomenon has been found on passive extension in the knee (Verwey and Kastein, 1937). Others upheld the influence of body position as such (Bychowsky, 1919; Katzenstein, 1931). In normal subjects these procedures seem to have no effect (Davidson, 1931).
5. *Walking:* it has been affirmed that a march provoked Babinski signs even in some normal subjects (Yakovlev and Farrell, 1941), on the other hand normal gait has been found to inhibit the flexion reflex (Lisin et al., 1973). And Eskridge (1901) blamed exertion in general for the high absence rate of the plantar reflex in 'healthy, strong and vigorous nurses'.

These various procedures, whatever their merits, have not been applied to patients in the present study – apart from the fact that all patients were examined in the supine position. The purpose was to study equivocal plantar responses in the same form as the referring clinician.

Electromyography

The results from the preceding chapter show that if the sole is stimulated in the usual way, the electromyographic activity recorded from the extensor hallucis longus and flexor hallucis brevis correlates excellently with inspection of undisputed toe movements. The same electromyographic technique was now applied to patients with equivocal plantar responses, because it might offer important advantages over independent clinical examination alone:

- muscle fibre potentials reflect the discharge pattern of motoneurones more exactly than a quick succession of movements, at least in theory, and the time sequence of these discharges can be studied on film;
- the results can be judged without any clues about patient or diagnosis, and repeating the examination as well as the interpretation allows objective assessment of the consistency of the electromyographic method;
- if an electromyographic outcome in a given patient is consistent and not at odds with the 'final' neurological diagnosis, it can be used as a

standard to measure the provisional opinion of the referring neurologist as well as the value of a detailed clinical examination of the flexion reflex.

METHODS

Patients

My colleagues from the department of neurology were asked to refer in-patients or out-patients with equivocal plantar responses; resident and specialist had to agree about the difficulty of interpretation. Thirty patients were proposed and examined in the course of six months.

Stimulation and recording

Stimulation consisted of slowly stroking the lateral plantar border and plantar arch with the smooth, blunt handle of a patella hammer. Recordings were made from the following muscles:

- extensor hallucis longus (EHL)
- flexor hallucis brevis (FHB)
- tibialis anterior (TA).

The technique of electrode placement was similar to that described in Chapter III.

Clinical examination and exclusion of bias

To ensure maximal objectivity of the EMG results, they were kept separate from the deliberations of the physicians in charge and from my own examination. How this was done is shown schematically in figure 9. First of all, I was not involved in the management of any of these patients. The resident and specialist who referred the patient did this on a form; they jointly rated the plantar reflex, choosing from five possibilities: upward, possibly upward, absent, possibly downward and downward (the two extremes were never chosen, which was in keeping with the purpose of the study). The form was sent to the secretary of the study. I was notified only of the patient's name and of the side where the clinicians considered the plantar reflex most equivocal. The inconvenience of the procedure to the patient did not permit investigation of both feet at the same time.

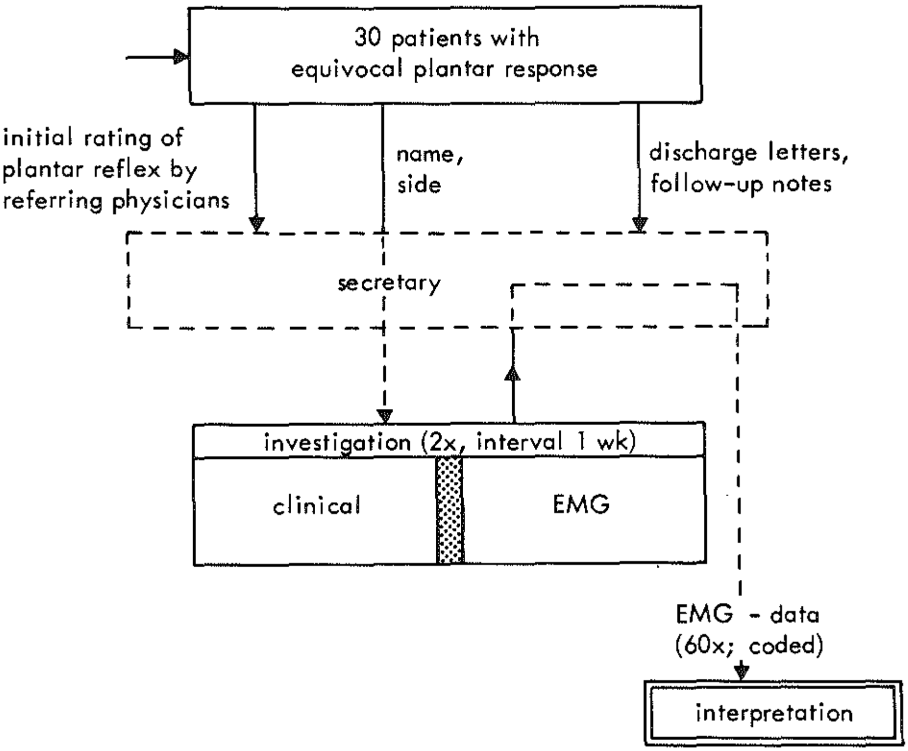

FIGURE 9

Design of study of equivocal plantar reflexes

The day before EMG, I examined the patient myself, and made detailed notes about toe responses and the flexion reflex in general. Although I was unaware of the suspected diagnosis, this examination might influence the subsequent recording. Therefore, electromyography was performed without visual or auditory feedback from the recording apparatus. Because a two-channel oscilloscope was used, pictures were taken from three subsequent reflexes in FHB and EHL, and from another three in TA and EHL. A reflex was rejected and repeated only when the tracing was technically inacceptable. The six pictures were mounted on a blank card and kept by the secretary, together with the protocol of the experiment and the notes of the day before. After one week, the clinical and electromyographic examinations were repeated in the same manner.

At the end of the study, the 60 cards, identified only through a code number given by the secretary, were interpreted five times, on separate occasions and without knowledge of previous ratings. Subsequently, discharge letters and follow-up notes were consulted, covering a period of up to two years after the investigation.

Electromyographic criteria

Guidelines as to what electromyographic patterns make up a pathological EHL reflex – representing a Babinski sign – were derived from Chapter III, which includes findings in 15 patients with a clear Babinski response and in 40 control subjects:

1. The EHL reflex should coincide with a reflex in the TA. Isolated potentials in the EHL could result from irritation by the tip of the recording electrode, especially when the needle is levered by voluntary or reflex activity of other muscles through which it was inserted.
2. Potentials should be dense enough not to be separately identifiable ('interference pattern' – that is, at 500 ms/cm time base). The potentials can be either continuous or in regular clusters (clonic reflex – of course not to be confused with clonic tendon jerks (Barré, 1926)).
3. Larger potentials should appear in the middle of the reflex, indicating recruitment of motoneurones of increasing size, so that, ideally, a spindle shape is formed. On the other hand it should be kept in mind that, besides motor unit size, electrode position is another factor determining spike amplitude.
4. The end of the reflex should be visible: voluntary withdrawal tends to linger on for a few seconds, and flexor spasms are hardly to be expected in this group of patients.
5. There should be no concomitant reflex in the FHB.

Starting from these criteria, the EMG patterns of each investigation were allocated to one of five possible categories. Relevant examples are shown in figure 10. If the qualifications for a pathological EHL reflex were partly met, a probable EHL reflex could be rated; this was minimally defined by the presence of the first two criteria in two out of the six photographs. When neither of these two pathological ratings was applicable, the result was considered normal, and was further specified as definite or probable FHB reflex (cf. criteria 2, 3 and 4 for the EHL) or no reflex activity at all. Strictly speaking, an absent reflex can be abnormal when a FHB response is found on the other side (Harris, 1903; Kino, 1927), but only one side was investigated.

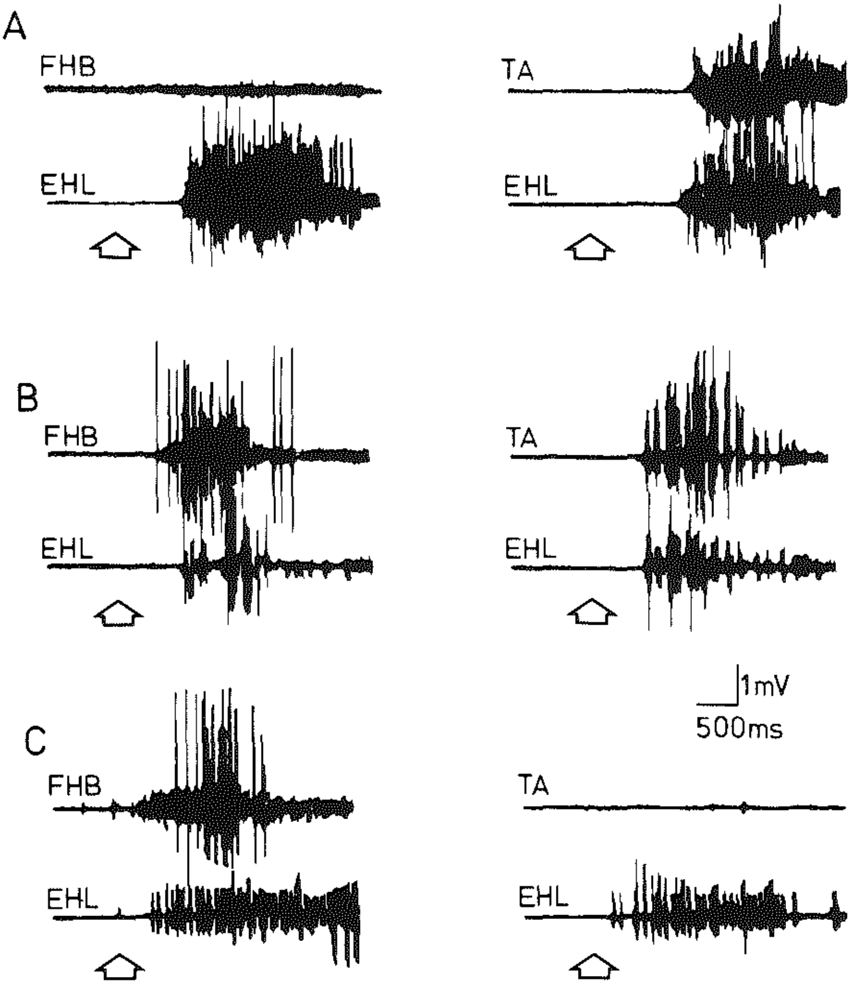

FIGURE 10

Patterns of *activity in EHL. Beginning of stimulation marked by arrows.*

A. Pathological EHL reflex: synchronous with TA, merged potentials, spindle-like, no FHB reflex.

B. Probably pathological EHL reflex: synchronous with TA, merged potentials in clusters, some recruitment, FHB reflex present.

C. No pathological EHL reflex: TA silent, no clear merging of potentials, no recruitment or decreasing of activity, FHB reflex present.

RESULTS

Observer consistency

The rating of EMG patterns scarcely varied throughout the five sittings. Intra-observer variation in the form of a contradiction 'pathological' and 'not pathological' occurred in five of the 60 sets of pictures. In four, only one of the five readings was at variance with the others. These exceptions were disregarded when both investigations in each patient were compared (EMG consistency), as they did not appear in the same pair, so that the chance of a contradictory outcome was one in ten in these four patients. In one further set, two readings were opposed to the other three.

Of the remaining 55 sets, 43 had received an identical classification on the five-point scale on each of the five occasions. In 12, there was some shift within the same group – that is, FHB reflex, probable FHB reflex or no reflex on the one side versus EHL reflex or probable EHL reflex on the other.

EMG consistency

After interpretation, the patient code was broken and the results of the two investigations for each patient were matched and compared. The repetition of the EMG examination led to a similar conclusion in 26 of the 30 patients (87%). Twenty pairs were both interpreted as normal, six both as pathological. When chance agreement is taken into account, as well as disagreements within the two main categories of 'pathological' and 'not pathological', the weighted agreement rate (kappa; Cohen, 1960, 1968) was 0.61.

Of the four mismatches, two plantar reflexes were rated normal once and possibly pathological the other time, in one the outcome went from absent to definitely pathological. These three cases with inconsistent EMG results included two patients who showed on clinical examination bilateral but exceptionally fast upgoing toes, with the additional problem that no adequate cause could be found for this; they will be discussed separately below. The fourth mismatch was caused by the set mentioned in the previous section, in which two readings contradicted the other three; the twin set showed a definite EHL reflex. True variation of the plantar reflex within one week is of course possible, but was not a factor here.

Inexperienced observer

An additional question was whether it is easy for an electromyographer not familiar with plantar reflexes to decide whether a pathological EHL reflex is present or not. A nurse with three years' experience as an EMG assistant was asked to interpret the photographs in the same manner as the author. The previous results in patients with a Babinski sign and in control subjects were briefly discussed beforehand. Compared with the author, about twice as many sets were rated pathological, including patterns that may be seen in control subjects. Seven pairs could not be matched because of observer variation (two ratings opposite to the other three in at least one of both sets); five more pairs did not fit. Clearly some experience with reference groups is necessary.

EMG result and initial rating of referring physicians

The EMG indicated a pathological reflex considerably less often than the opinion of the referring neurologists (this was never definitely upward or downward, so that in fact a choice was made between probably upward versus absent or probably downward). In 20 patients a Babinski sign was suspected: the EMG was pathological in four, normal in 13, and contradictory in three. In the remaining 10 patients, tentatively classified as normal by the neurologist, the distribution of EMG results was similar: two pathological, seven normal, one contradictory. The identical proportions of EMG categories in these two groups can be taken to illustrate that these plantar responses were truly equivocal.

Whether the EMG study gave the right answer must be substantiated further by clinical data concerning individual patients.

EMG result and 'final' neurological diagnosis

The diagnosis in the patients was not influenced by this study; otherwise, the argument would be circular. Even then, it is obviously impossible to prove the precision of the EMG outcome by referring to the neurological label: a pathological EHL response can be lacking despite other pyramidal signs and even the reverse situation is not unimaginable. On the other hand, follow-up was the only yardstick available, if we exclude the fact that interpretation was based on results from reference groups.

The most interesting category of patients is that in whom doubtful plantar reflexes are the only reason to suspect organic disease of the central nervous system; then the problem arises how far one should go with ancillary investigations. In other patients, with established supranuclear weakness, the matter is more theoretical. Thus, the 30 patients were subdivided according to the problem that arose as a result of the uncertain plantar responses.

1. *An unexpected Babinski response?* In eight patients there were possible Babinski signs, which did not fit in with the history and the rest of the neurological examination. The EMG was normal in all of them. In table XII, the EMG outcome is shown for each of the two investigations, together with the neurologist's opinion about the plantar reflex at the time of the study, with the final clinical diagnosis, and with special features which were noticed during the author's examination. In this and the next group of patients, a dubious upward response was the clinician's most common choice; those who rated possibly downward or absent plantar responses apparently retained some doubt, since the patient was referred for this study. It can be seen from the table that the eventual neurological diagnosis was still incompatible with a Babinski sign in all eight cases.
2. *An additional Babinski response?* In 10 patients (table XIII) some clues already implied disease of the central nervous system, but not necessarily involving descending pathways to the side in question, and the neurologist wondered if the equivocal plantar reflex indicated an unexpected localization. The EMG showed EHL reflexes in two patients, was normal in five, and contradictory in three.
3. *The missing flexor response.* Two patients showed unilateral absence of the plantar reflex, perhaps pathological, because for other reasons an intracranial lesion was suspected. EMG recording gave evidence of FHB activity on both occasions in these two patients, and subsequently no cerebral lesion could be substantiated in either of them.
4. *An expected Babinski response is equivocal.* Ten patients showed dubious plantar reflexes in the presence of other pyramidal signs on the affected side. In four an EHL reflex was shown by electromyography on both occasions (two probable, two definite), in one only the first time; the clinicians had not rated a downward response in these cases. Of the remaining five patients, an FHB reflex was found in four, and no reflex activity in one.

We can summarize the comparison of EMG results with the eventual clinical diagnosis as follows: the EMG never indicated a pathological plantar reflex when the central nervous system was after all supposed to be normal, whereas the criteria were wide enough to identify expected, but clinically unconvincing, Babinski signs.

TABLE XII

PATIENTS SHOWING EQUIVOCAL PLANTAR RESPONSE AS AN ISOLATED FINDING

patient	*sex*	*age (yr)*	*side*	*clinician's opinion*	*EMG reflex*	*final clinical diagnosis*	*comments*
1	F	71	R	absent?	1. FHB 2. FHB	none (complaint: 'heavy legs')	voluntary contraction of TA
2	M	51	L	upward?	1. FHB? 2. FHB?	sciatica L	jerky voluntary toe movements
3	M	22	R	upward?	1. FHB 2. FHB?	neuritis of brachial plexus	flexion reflex +++ bilaterally, and brisk downward response of toes; once all movements have ceased, hallux goes 'up' again
4	F	21	L	upward?	1. none 2. none	lumbago	hallux immobile, little toes down
5	F	43	R	absent?	1. FHB? 2. FHB?	hysteria	
6	M	46	L	upward?	1. none 2. none	polyneuropathy of unknown cause	toe movements just possible
7	M	28	L	upward?	1. none 2. none	polyneuropathy (post-infectious)	flexion reflex ++ bilaterally; abundant voluntary activity
8	F	55	R	upward?	1. FHB 2. FHB	non-specific oro-facial pain R	second toes amputated – shift of first metatarsal bone?

TABLE XIII

PATIENTS WITH SOME EVIDENCE OF CENTRAL NERVOUS SYSTEM DISEASE, IN WHOM EQUIVOCAL PLANTAR REFLEX MIGHT SUGGEST AN UNEXPECTED LESION

patient	*sex*	*age (yr)*	*side*	*clinician's opinion*	*EMG reflex*	*final clinical diagnosis*	*comments*
9	M	18	R	upward?	1. none 2. EHL	epileptic seizure two years before	flexion reflex +++ bilaterally, including great toe
10	F	29	R	upward?	1. EHL? 2. none	vertigo and paraesthesiae of L arm (cause unknown); CSF protein raised	flexion reflex ++ bilaterally, including great toe; bilateral pes cavus
11	F	58	R	upward?	1. EHL? 2. EHL	parkinsonism, dementia, depression	upgoing toe synchronous with flexion reflex
12	F	60	R	absent?	1. EHL? 2. EHL	episodic vertigo (brain stem?); generalized arterial disease	upgoing toe synchronous with flexion reflex
13	M	62	R	absent?	1. EHL? 2. FHB?	pyramidal weakness L leg (unknown cause)	extensive voluntary movements
14	M	40	R	upward?	1. FHB 2. FHB?	hemi-parkinsonism R	hallux immobile, also on electrical stimulation of FHB; voluntary flexion possible (long flexor?); initially diagnosed as R (pyramidal) hemiparesis
15	M	56	L	upward?	1. FHB? 2. FHB?	cerebral concussion	isolated fanning of toes
16	M	57	L	upward?	1. FHB 2. FHB	presenile dementia	jerky toe movements, out of phase with (weak) flexion reflex; originally suspected of cerebral tumour
17	M	53	R	upward?	1. none 2. none	pinealoma and hydrocephalus	flexion reflex prominent in TA
18	F	39	R	downward?	1. none 2. none	transient dysfunction 6th and 7th nerve R – multiple sclerosis?	wriggles toes sometimes

Practical use of the flexion reflex

Having accepted the EMG results (inconsistencies excluded), we can now compare them with the notes of the two clinical examinations which were performed in each patient. On these occasions special attention was paid to the flexion reflex as a whole, and to the time relations of this synergy to any toe movements which occurred. The action of proximal flexor muscles was particularly noted, i.e. of tensor fasciae latae, hamstring muscles, and hip flexors. This stands in some contrast to the emphasis on the tibialis anterior as a reference muscle in the EMG procedure. The reason for the shift of attention is the supporting hand on the foot during plantar stimulation. This position sometimes makes it difficult to see dorsiflexion of the foot, and the 'tug' felt by this hand is the compound action of all flexor muscles – which is very useful information as such!

In view of all previous work on the relationship between the Babinski sign and the flexion reflex, it is perhaps not surprising that in all six patients with electromyographically pathological plantar reflexes the upgoing toes were clinically found to be synchronous with activity in proximal flexors. This number is small, but I can add some results from the next chapter: of 50 patients with a unilateral Babinski sign, 48 showed activity in other (more proximal) flexor muscles.

In two cases (patients 9 and 10, table XIII), there was, on clinical examination, simultaneous activity of the extensor hallucis longus and flexors of knee and hip, but electromyography showed a pathological reflex on only one occasion in each (they account for half of the four EMG inconsistencies in the study). The referring clinicians doubted if the Babinski signs were 'real', because they did not fit easily into the clinical picture, and also because they were unusually fast – but so was the flexion reflex as a whole in these two patients. Failure of the EMG technique to confirm the Babinski signs on one occasion in these two patients must also be attributed to the high speed of movement, which did not always correspond with the chosen criteria. The reproducibility of the clinical phenomena was decisive in establishing the pathological nature of the upgoing toe movements.

The clinical features were even more vital in explaining why the referring physicians had had their doubts about plantar reflexes which proved to be normal electromyographically: often there were in fact upward movements of the great toe, but out of phase with the flexion reflex and in some cases not even caused by action of the extensor hallucis longus. Sometimes the confusing movements coincided accidentally with the flexion reflex and were not reproducible; fatigue of the Babinski sign (Bauer and Biach, 1910) is a negligible factor. Almost all these observations bear on patients in the first two diagnostic categories, in whom the possible Babinski signs were wholly or partially unexpected, and have been included in tables XII and XIII. The fallacies that were identified will be discussed more systematically below.

DISCUSSION

Use of electromyography for plantar reflexes

Recording from flexor hallucis brevis, extensor hallucis longus and tibialis anterior during mechanical plantar stimulation generally provides reproducible results. These electromyographic records make it possible to study patterns of action potentials from separate muscles rather than quick and superimposed movements, and to determine from photographs whether a pathological recruitment of the extensor hallucis longus into the flexion reflex (with the tibialis anterior) is present or not. For reliable interpretation some experience with reference groups should be gained. Initially, it appears difficult to ignore the activity of only a few motor units of the extensor hallucis longus. An important reason for this may be the unusual time base of 500 ms/cm, 10 times slower than is customary in routine electromyographic practice, where a similar picture represents a definite degree of contraction.

In this study, electromyography gave normal results in a great number of cases where equivocal plantar responses had brought the clinician into a diagnostic dilemma. So the impression that 'where clinical doubt exists, the plantar reflex is apt to be equivocal' (Matthews, 1975) proved to be true, but in the reverse sense. Of course this conclusion must be weighed against the fact that clinicians were invited to refer patients with difficult plantar reflexes, but it is in keeping with the finding from Chapter II that bias inclines more towards a Babinski sign than towards a normal response.

This study by no means advocates the routine use of electromyography to classify plantar reflexes; that would be absurd. Differences between the outcome of the electromyographic investigation and clinical judgments of plantar responses can often be traced back to fallacies of observation and interpretation or to peripheral anatomical factors. This feedback to the clinical neurologist seems to be the most important use of the technique.

False suspicions

Several factors were able to mimic a Babinski sign in cases where electromyography did not disclose a pathological reflex (patient numbers refer to tables XII and XIII):

1. *Contraction of the tibialis anterior,* voluntarily (patient 1) or as part of the flexion reflex (patient 17): the toes go upward passively, without contraction of the extensor hallucis longus. This is a classical pitfall (Oppenheim, 1899; Kalischer, 1899).
2. *A very active flexion reflex, combined with brisk plantar flexion of the toes:* once these bewildering movements have ceased, the big toe goes 'up' (back) to the original position (patient 3).
3. *Voluntary toe wriggling* (patients 2, 16, 18). The jerky extensor movements are inconstant and out of phase with the flexion reflex. Involuntary movements caused by disease of the basal ganglia may also confuse the examiner (C. and O. Vogt, 1920; Dosužkov, 1932).
4. *The great toe is immobile, while the other toes go gently down;* this may create an optical illusion (patient 4).
5. *Isolated fanning of toes* (patient 15). This 'signe de l'éventail' also occurs in normal subjects (Babinski, 1903 a; Noica and Sakelaru, 1906; Barré and Morin, 1921) and can lead to errors of both observation and interpretation. The reverence paid to this phenomenon is unnecessary from a historical as well as from a practical viewpoint.
6. *Anatomical variations which prevent toe flexion,* although the flexor hallucis brevis is active. In patient 8, both second (hammer) toes had been amputated long before, probably with subsequent shift of the first metatarsal bone and its tendons, as in hallux valgus (Iida and Basmajian, 1974). In patient 14, there seemed to be another abnormality of insertion of the flexor hallucis brevis, as only isometric contractions could be provoked by stimulating through the recording electrode; voluntary toe flexion was possible, probably mediated by the flexor hallucis longus.
7. *Peripheral lesions.* It is an old notion that a false upgoing toe response can occur by damage to the neuromuscular apparatus which mediates the normal plantar reflex (Babinski, 1898; the gist of the relevant passage is unfortunately inverted in the translation of Wilkins and Brody, 1967). Most subsequent reports cite, like Babinski himself, cases of poliomyelitis (Pfeifer, 1904; Salomon, 1921; Sicard and Seligman, 1925; Laignel-Lavastine, 1925; El Sherbini, 1971), although others have stressed that pyramidal features can occur in poliomyelitis (Fuchs, 1905; Tournay, 1924; Souques and Ducroquet, 1924; Sebeck and Wiener, 1926). Less often the phenomenon has been encountered in lesions of nerve roots or plexus with predominant affection of toe flexors (Sicard and Haguenau, 1919; Rouquier and Couretas, 1926), in polyneuropathy (Lortat-Jacob, 1902), and even in myopathy (Léri et al., 1923).

Despite the abundant literature on this 'peripheral pseudo-Babinski sign', its occurrence is much less logical than it seems at first sight. The Babinski sign is produced by pathological recruitment of extensor hallucis longus motoneurones, and it is difficult to see how this is any more likely to happen because impulses are unable to reach the short toe flexors. On the other hand, active muscular tension in the patient is of course restricted to intact muscles: even a few discharging extensor hallucis longus motor units might then cause upward quivering of the great toe. This could have been the case in patients 6 and 7, who suffered from polyneuropathy. The flexor hallucis brevis, being more distal, was probably most involved, and in fact both patients showed scattered extensor hallucis longus activity, versus even fewer and later potentials of flexor hallucis brevis in one patient and none at all in the other. This possible mechanism does not detract from the principle that one need not be aware of the peripheral lesion to disqualify these upward toe movements if they are unrelated to the flexion reflex.

8. *Pes cavus.* This deformity has also acquired some notoriety for producing false-positive Babinski signs (Mumenthaler, 1976). It is true that the primary upward position of the great toe mechanically favours further dorsiflexion by even the slightest activity of the extensor hallucis longus. This is the more so because pes cavus is often accompanied or even caused by atrophy of intrinsic foot muscles (Thomas, 1975; the hypothesis of Collier (1899) that increased tone of the extensor hallucis longus contributed to the deformity is untenable). But again, when the upward movement of the great toe is a genuine reflex (patient 10), it signifies abnormal processing of impulses in the spinal cord, irrespective of peripheral anatomy.

Lacking Babinski response

Finding an equivocal plantar response in the presence of other pyramidal signs is almost the reverse situation of finding it as an isolated phenomenon, and usually it presents fewer problems of clinical management. Close attention to toe movements in their relation to action of other leg muscles may also be helpful in this group, as shown above by the comparison of electromyographic results with clinical data. If a Babinski response is truly absent there are three possible reasons:

1. *Joint deformity.* This is not unusual, especially in the form of hallux valgus. Figure 11 illustrates occult Babinski signs in a patient who could not even move his great toes voluntarily because of hallux valgus.

2. *Peripheral nerve lesions.* Pressure palsy of the lateral popliteal nerve is (or at least was) a common complication of chronic paraplegia, and precludes a Babinski sign (Collier, 1899; Marie, 1912; Marie and Thiers, 1913; Guttmann, 1952; Grossiord and Kahn, 1957; Landau and Clare, 1959). The same problem can occur at another level in motor neurone

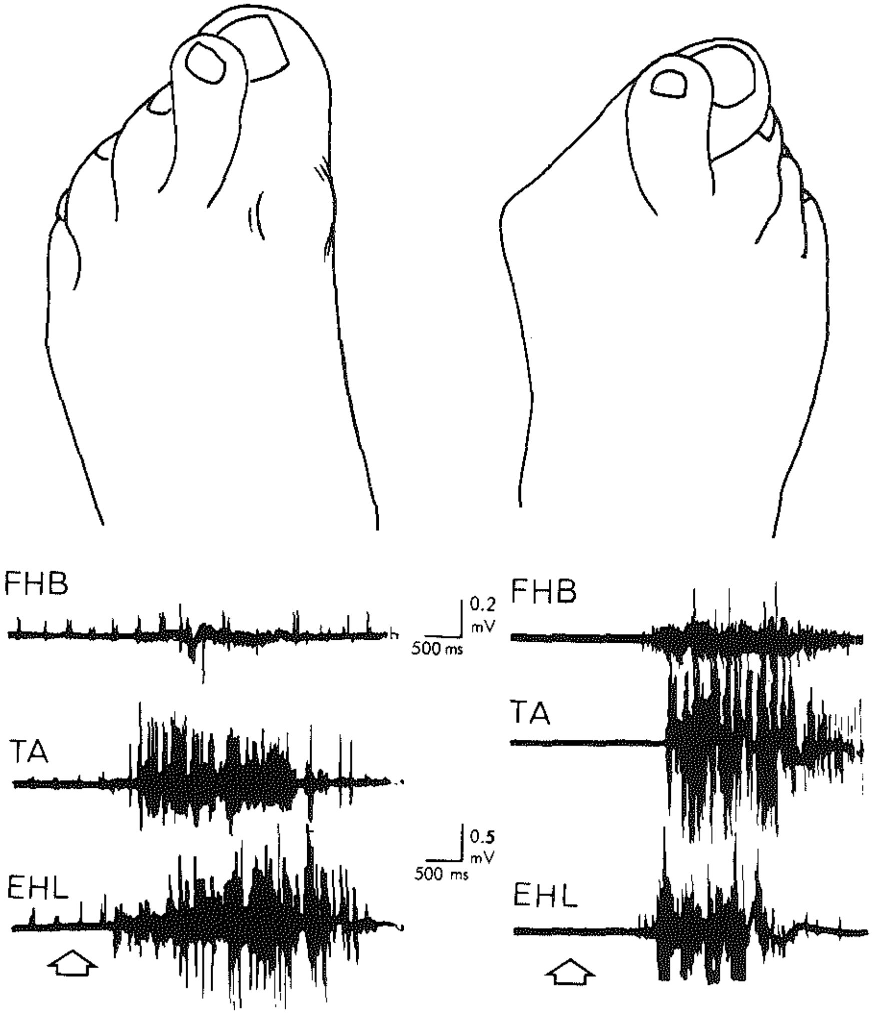

FIGURE 11

Occult Babinski signs in a 70-year-old male with cervical spondylotic myelopathy and hallux valgus.

disease (Homburger, 1901). Testing the peripheral apparatus by voluntary movements is usually not possible because of the primary supranuclear weakness. If there is no atrophy, only electromyography and conduction studies can reveal the secondary lesion.

3. *The pyramidal syndrome is really incomplete.* This was the case in five of the ten patients in whom an expected Babinski sign was equivocal. The next chapter will deal with this problem.

Clinical criteria

Apart from specific errors enumerated above, the following general rules for interpreting equivocal plantar responses emerged from the comparison of clinical data with electromyographic results:

1. *Upward movement of the great toe can be pathological only when caused by contraction of the extensor hallucis longus muscle*
   - in most patients this can be reliably checked by inspection or at least palpation of the tendon on the dorsum of hallux and foot
2. *Contraction of the extensor hallucis longus muscle is pathological only if it occurs synchronously with activity in other flexor muscles*
   - in some subjects the flexion reflex is weak, but almost always tightening of the tensor fasciae latae or hamstring muscles can be seen and used as a reference
   - activity of the flexion reflex as a whole can also be felt, with the hand that supports the patient's foot
   - it should be kept in mind that the hallux is lighter than foot, leg, or thigh
   - activity in physiological flexor muscles other than the extensor hallucis longus is not pathological on its own, unless asymmetrical or spasmodic.
3. *Voluntary withdrawal may be confused with the flexion reflex, but can be prevented and recognized*
   - tell the patient what is going to happen; blindfolding (Crocq, 1901) is not helpful
   - adapt the stimulus – it is useless to complete faithfully a textbook manoeuvre while the patient is writhing on the couch; a change to the lateral dorsum of the foot may help; otherwise repeated stroking of only a few centimetres of skin will, in the case of a flexion reflex, produce each time an approximately similar reduction in the extent of the toe and leg movements, whereas voluntary retraction will then be abolished or inconstant
   - voluntary withdrawal does not involve the tensor fasciae latae, and may precede or outlast the stimulus (Babinski, 1906 a, 1915 b).

Finally the method of stimulation. In principle, any stimulus is legitimate when it produces an upgoing toe response which fulfils the above criteria, as long as it is moving (Chapter III), and as long as it is not applied more distally than the ball of the foot, as this may give false-positive results (Verger and Abadie, 1900; Dosužkov, 1932). As to effectiveness, it is generally accepted that the lateral plantar border is preferable to the medial side, which overlies the flexor hallucis brevis. We also saw (Chapter III) that temporal and spatial summation are important. This means that the ideal stimulus should be rather slow and should extend to the plantar arch. On the other hand, repeated interference of voluntary movements makes a shorter or different stimulus desirable. Stroking the lateral dorsum of the foot often helps in these cases, and is sometimes even more effective (Bing, 1915; Landau and Clare, 1966; Dohrmann and Nowack, 1973). This is also because irritation of the sole can give rise to voluntary clawing of the toes.

In short, the Babinski sign is a reflex, not a ritual. And how to scratch is less essential than how to watch.

CONCLUSIONS

1. Current rules for the interpretation of equivocal plantar reflexes are scarce and arbitrary, while relatively too much attention is paid to methods of stimulation.

2. Electromyographic investigation of equivocal plantar reflexes, based on findings in reference groups, can give a reproducible outcome in a large majority of cases.

3. In this study, the electromyogram confirmed a pathological reflex in only one third of the patients in whom this was suspected by the clinicians. Checked with the eventual diagnosis, false pathological results did not occur; conversely, the electromyographic criteria were wide enough to demonstrate expected, but clinically unconvincing, Babinski signs.

4. The main advantage of the electromyographic study of plantar reflexes is that it helps to delineate clinical criteria, but it may be the only feasible method in cases where skeletal or tendinous abnormalities restrain or distort hallux movements.

(cont. next page)

5. If dorsiflexion of the great toe is mechanically favoured (pes cavus, paralysis of short toe flexors), this only means that voluntary upward movements are more likely to interfere, and not that a possible Babinski reflex is false.

6. To decide whether an upward movement of the great toe is a Babinski sign, there are three step-wise criteria:
   (1) action of the extensor hallucis longus muscle,
   (2) simultaneous contraction of other (physiological) flexor muscles,
   (3) exclusion of voluntary movements.

CHAPTER V

THE BABINSKI SIGN AND THE PYRAMIDAL SYNDROME

'. . . se demander s'il n'y a que certaines parties du système pyramidal dont l'altération puisse produire le phénomène des orteils.'

(Babinski, 1898)

INTRODUCTION

In this chapter I shall try to deduce the pathophysiology of the Babinski sign from its relationships with other components of the clinician's pyramidal syndrome:

- is the Babinski response chiefly related to negative phenomena, in particular to disturbances of voluntary innervation? If so, is any motor deficit sufficient to release it, irrespective of its distribution?
- or is the Babinski sign linked more closely to segmental release phenomena, especially to release of the entire flexion reflex?

The various positive and negative features might depend on different descending fibres, and the presence or absence of clinical correlations might give insight into physiological and anatomical connections. In other words, the pyramidal syndrome is perhaps not a syndrome. Some workers have postulated that it is not even pyramidal. Therefore I shall gradually develop and specify the above questions in the light of previous studies.

Early theories: supraspinal reflex pathways?

We have seen in Chapter I how Babinski, having discovered the upgoing toe phenomenon in patients with affections of the central nervous system, linked this sign one year later to disturbances of the pyramidal tract (Babinski, 1897); van Gehuchten (1898 a) arrived independently at the same conclusion.

That the Babinski sign is caused by disturbances of the pyramidal tract soon became generally accepted by neurologists, but it long remained a matter for debate at what level in the nervous system the reflex reversal of the great toe was brought about. This was because the pathways of the normal and the pathological plantar reflex were not exactly known.

The various theories are displayed in table XIV. Most were based on the once wide-spread belief (Jendrassik, 1894) that normal cutaneous reflexes passed through the cortex and pyramidal tract because they showed a long latency and were diminished in hemiplegia, in contrast to tendon reflexes. Moreover, there was some evidence that cutaneous reflexes disappeared before tendon reflexes in chloroform anaesthesia (Laureys, 1900).

It can be seen from the table that within this line of thought (A), a majority assumed a spinal pathway for the Babinski response, in accordance with classic animal experiments about the flexion reflex as a whole (see Chapter I).

TABLE XIV
THEORIES INVOKING A SUPRASPINAL PATHWAY FOR ONE OR BOTH PLANTAR RESPONSES

A. *The normal plantar reflex has its pathway via the cerebral cortex and pyramidal tract; when this course is interrupted by disease, a Babinski sign appears because the reflex activity takes a 'lower' alternative route:*
   1. a different cortical pathway: Munch-Petersen (1902), Marinesco (1903)
   2. via the basal ganglia: Crocq (1901)
   3. thalamo-spinal or tecto-spinal tract: Homburger (1902), Heldenbergh (1903)
   4. bulbo-spinal pathway, cerebello-spinal pathway or via basal ganglia: Pfeifer (1904)
   5. mesencephalic pathway: Armenise (1924)
   6. 'extrapyramidal' pathway: Tournay (1926)
   7. spinal route: van Gehuchten (1900 a, b), Schneider (1901), Specht (1902), Goldflam (1903, 1908), Friedlaender (1904), Knapp (1907), Lewy (1909 b), Chaddock (1911), Fairbanks (1911), Herzog (1918), Wertheim Salomonson (1918, 1920), Russetzki (1930).

B. *The normal plantar reflex is conducted via the spinal cord, whereas the pathological plantar reflex takes a supraspinal course:*
   1. 'supra-medullary centres': Dejerine and Long (1912), Dejerine (1914), Dejerine et al. (1914, 1915)
   2. tecto-spinal pathway: Minkowski (1922, 1923)
   3. hypothalamus or globus pallidus: Zador (1927)

Conversely, some observations of downward toe responses in cases of complete transection of the spinal cord led others to believe that only this reflex was spinal, and that the Babinski sign was mediated by higher centres (Category B in table XIV). These observations included Dejerine's famous case (1912) of the unfortunate young acrobat (see Chapter I p. 34), and some early victims of the first world war studied by the Dejerines and Mouzon (1914, 1915). Later experiences with paraplegic patients were to show that upward toe responses are the rule, and that failure to elicit them can be attributed to initial reflex depression, pressure palsy of the lateral popliteal nerve or a terminal toxic-infectious state (Riddoch, 1917).

The modern concept that the spinal cord is the centre for both the normal and the pathological plantar reflex was predicted in general terms by Strümpell (1899) and exemplified by Walshe (1914). A few of the older notions survived for some time, as table XIV shows. Van Woerkom (1910, 1911) took an intermediate view by supposing that both plantar reflexes had their centre in the spinal cord, but with additional dependence of the normal response on a tonic influence from the posterior part of the thalamus.

In conclusion, the change from the normal plantar response to the Babinski sign must be the result of a defective influence of descending pathways on the *spinal cord*. Because the upgoing toe phenomenon is found contralaterally to focal lesions in the brain, often accompanied by weakness, implication of the pyramidal tract seems obvious. However, the relationship between the Babinski sign and disturbances of the pyramidal tract is not free from exceptions or even criticisms, and these will be considered in the following sections.

Babinski signs without pyramidal *lesion*

Although Babinski mentioned only organic affections of the central nervous system in his very first communication of 1896, his comprehensive paper (Babinski, 1898) refers to a temporary 'toe phenomenon' during an epileptic attack, and in a case of strychnine poisoning. One year later, he was to devote a separate note in the 'Revue neurologique' to the abnormal toe sign in epilepsy (Babinski, 1899 b).

In subsequent years the Babinski sign was reported in many varieties of functional disturbance of the central nervous system (table XV). The

TABLE XV
REPORTS OF BABINSKI RESPONSE IN 'FUNCTIONAL' CHANGES OF THE CENTRAL NERVOUS SYSTEM

1. *epileptic seizures:* Babinski (1898, 1899 b), Collier (1899), Crouzon (1900), de Pastrovich (1900), Schönborn (1901), Specht (1902), Barnes (1904).
2. *intoxications*
   - strychnine: Babinski (1898), Collier (1899)
   - chloroform: Bickel (1902)
   - morphine: Hawthorne (1914)
   - scopolamine: Volkmann (1903), Link (1906), Hahn (1911), Rosenfeld (1921, 1925)
   - barbiturates: Elliott and Walshe (1925), Piotrowski (1926)
   - insulin: Heiman (1939)
   - coal-gas: Elliott and Walshe (1925)
3. *infections*
   - meningitis: Babinski (1898), Walton and Paul (1900), Barnes (1904)
   - tetanus: Collier (1899)
   - typhoid fever: Lévi (1900)
   - pneumonia: Foerster (1913), Biach (1915)
   - pericarditis: Biach (1915)
4. *renal failure* (usually with convulsions): Walton and Paul (1900), Barnes (1904), Curschmann (1911), Biach (1915), Elliott and Walshe (1925)
5. *liver failure:* Hawthorne (1914), Biach (1915), Elliott and Walshe (1925), Piotrowski (1926)
6. *deep sleep*
   - in older children: Collier (1899), Goldflam (1903), Marinesco (1903), Tournay (1928)
   - in adults: Bickel (1902), Kleitman (1923), Batini et al. (1964), Fujiki et al. (1971).

change of the plantar reflex was often but not always accompanied by disturbances of consciousness: in some of the listed conditions the patients were stated to have remained alert while showing Babinski signs (Hahn, 1911; Curschmann, 1911; Rosenfeld, 1921; Heiman, 1939), and conversely normal plantar responses have been found in diabetic coma (Biach, 1915; Elliott and Walshe, 1925).

A separate and interesting category is formed by patients with periodic Babinski signs. Rennie (1912) cites the case of a 14-year-old boy in whom the plantar reflexes depended upon the volume of a frontal hydatid cyst. Tournay (1927) and Monier-Vinard (1931) report Babinski signs only during the apnoeic phase of periodic breathing. Lhermitte and Dupont (1929) describe a patient who showed upgoing toes only when in cardiac failure – eventually he was found to have a discrete pontine infarct (Lhermitte and Trelles, 1930). More bizarre are reports of pathological plantar responses in normal subjects after a march of 14 miles (Yakovlev and Farrell, 1941), or caused by hypnosis with 'age regression' beyond the level of five months (Gidro-Frank and Bowersbuch, 1948 – one may wonder whether the suggestion affected only the subjects here). Berggren (1933) advanced Ménière's syndrome as another cause of transient Babinski signs, but it is hardly surprising to find that the symptoms in at least two of his four cases suggest organic involvement of the central nervous system.

It is obvious that the distinction between organic and functional involvement of descending pathways is artificial from a physiological point of view. 'Perturbation' was the term used by Babinski from the start (1898) until later years (1922); of course he never committed himself to a verifiable anatomical lesion for every instance of a pathological toe sign. This is, however, what posterity often made of it, as Nathan and Smith (1955) point out. Crocq (1901) and Kornilow (1903) are early examples of this misconstruction, while Lassek (1944) went to great lengths in showing from the literature that Babinski signs were not necessarily indicative of destruction of the pyramidal tract. But gross anatomical integrity is no proof of normal function.

Babinski signs without *pyramidal* lesion?

More serious evidence against the association between the Babinski sign and affections of the pyramidal tract seems to come from pathological studies that failed to demonstrate degeneration of lateral tracts in patients who had shown persistent upgoing toe signs. Nathan and Smith (1955) found this disparity in five patients in whom a partial cordotomy had been performed for the relief of pain (their cases 52, 33, 19, 21 and 38; all had bilateral operations). Adding this to the less startling fact that normal plantar responses were found in other patients who eventually showed severe descending degeneration of the lateral tracts (I shall discuss this problem separately), the authors arrived at the iconoclastic conclusion: 'any lesion of the lateral and ventral columns of the cord may, or may not, cause the Babinski response'.

However, the question of Babinski signs in the absence of obvious corticospinal degeneration is only a pseudo-problem, as Walshe (1956) was quick to point out. The fallacy is to assume that a non-degenerated pyramidal tract is the same thing as a normal pyramidal tract, particularly for patients in whom a cordotomy lesion borders upon pyramidal fibres: focal demyelination can produce severe loss of function without Wallerian degeneration. The experimental demonstration of conduction block by focal demyelination in the central nervous system is only a recent milestone in neurology (McDonald and Sears, 1970).

From the clinico-pathological side, however, it had been noted long before that demyelinating lesions with preservation of axis cylinders could produce neurological deficits, and Babinski was also a pioneer in this particular field (Babinski, 1885), together with Holmes (1906). It is therefore hardly surprising that he described patients with the toe phenomenon in whom the pyramidal tracts were not degenerated (Babinski, 1898, 1899 a, 1911 a). Nathan and Smith (1955) comment that Babinski, by these lines, disproved his own supposition about the relation between pyramidal tract and upgoing toe sign; this interpretation is inappropriate, as Babinski was very much alive to the relative importance of absent secondary degeneration – some of his cases were paraplegic! Walshe (1956) also gives Babinski too little credit in this respect, although the latter was not always fully consistent in that he sometimes explained an absent toe phenomenon by referring to undegenerated lateral tracts (Babinski, 1906 b, 1911 a).

In summary, the evidence that Babinski signs can result from lesions outside the pyramidal tract rests on incorrect premises. But of course this is a negative conclusion which does not prove the reverse.

'Pure' pyramidal lesions

Anatomical evidence from lesions caused by disease is often ambiguous, not only because absence of secondary degeneration does not necessarily imply functional integrity, but also because most lesions interfere with more than one fibre system. For this reason a number of studies were wholly or partially devoted to the plantar reflex after *experimental lesions in monkeys and apes.*

An upgoing toe response in lower primates is not precluded by differences in peripheral anatomy: the extensor hallucis longus muscle is comparable with that in humans (Howell and Straus, 1961). Whether normal animals never show an upgoing hallux is somewhat controversial: incidental reports of Babinski signs in monkeys come from Collier (1899), Kalischer (1899), Rudolf (1922) and Tower (1940), whereas Fulton and Keller (1932) never found it in over one hundred monkeys and apes, comprising twelve species. Fulton and Keller also carried out a comprehensive study of changes of the plantar response after surgical lesions at different levels in the central nervous system. In macaques, the only operation capable of producing a unilateral upgoing toe response was hemisection of the thoracic spinal cord (later, Forster and Campbell (1942) found it after extensive and bilateral cerebral lesions, and Mettler (1944) after various brain stem coagulations). Fulton and Keller contrasted the extensive lesions which were necessary in macaques with the results in apes (chimpanzee, gibbon), in which ablation of the contralateral cortical 'leg area' was sufficient to cause a pathological plantar response; baboons occupied an intermediate position. They concluded that control of the plantar reflex moved increasingly cephalad as the primate scale was ascended, and that it had become a purely pyramidal function in apes.

Kennard and Fulton (1933) restricted the motor cortex associated with the Babinski sign in chimpanzees to area 4 (ablations of area 6 alone could be followed by a temporary fan sign – an unconfirmed finding).

However, even selective ablations of the motor cortex probably involve descending connections other than the corticospinal system (Kuypers, 1973). An exclusive lesion is possible only by medullary pyramidotomy. Sarah Tower (1940) performed this operation in chimpanzees and afterwards observed a Babinski sign on the contralateral side. We can safely conclude from this that, at least in apes, pure pyramidal tract lesions may cause a Babinski response.

In *man,* reports of 'isolated' lesions of the corticospinal system resulting in a contralateral Babinski sign were, until recently, confined to its supramedullary course: the motor cortex (Walshe, 1935), the internal capsule (Fisher and Curry, 1965; Englander et al., 1975), and the cerebral peduncle (Bucy et al., 1964). This evidence could not be regarded as conclusive, because the corticospinal fibres are, at these levels, closely related to cortical projections on brain stem nuclei. Some, like Walshe (1947), took a fatalistic attitude: 'disease and injury in man do not provide us with clean sections of the medullary pyramids'. This has proved to be a premature conclusion: of late, two case histories have been published that describe a Babinski sign after an almost pure lesion (infarction) of the contralateral pyramid (Chokroverty et al., 1975; Leestma and Noronha, 1976).

These new facts firmly support the relationship between the Babinski response and affections of the pyramidal tract. In theory, a role of other descending fibre systems cannot be excluded with certainty for all cases. Brodal (1969), for instance, takes such a broad view about the release of the Babinski sign. However, there is no suitable candidate to fill the place of an alternative pathway that is crossed and descends low enough in the spinal cord. In infra-human primates the rubrospinal tract is closely connected with the pyramidal system, at least in functional terms (Kuypers, 1973), but its extent in man is controversial (Stern, 1938; Sie Pek Giok, 1956).

Having found that there are insufficient reasons to attribute the presence of a Babinski sign to dysfunction of any system other than the pyramidal tract, we are left with the reverse problem – *why the relationship is not, in the words of Babinski (1898), a necessary one.* A condition where the upgoing toe sign is often conspicuously absent is the acute transverse lesion of the spinal cord.

A paradoxical downward toe response in spinal shock

Most patients discussed in this section are victims of war or, more recently, of traffic, and I should like to start by citing Babinski (1922), in that he was one of the few writers on the subject to call attention to the human misery lying behind what little scientific advancement was made.

The study of reflexes in patients with complete paraplegia had been impeded for some time by the tenacity of 'Bastian's law' (1890), which proclaimed that total transections of the spinal cord in man were followed by permanent areflexia. Previous case reports to the contrary were held in doubt as to the completeness of the lesion. On the Continent, Bruns (1893) was a strong advocate of this law. Nevertheless, Bastian himself (1890) mentioned 'very slight movements of the toes . . . when the soles of the feet are strongly tickled', and in 1899 Collier described the newly discovered Babinski sign in three cases of a complete cord lesion; he was to be joined by Goldflam (1903). At the International Congress of Medicine in 1900, Bruns defended only the absence of tendon reflexes, and in this form the law survived (Collier, 1904; Walshe, 1914; Dejerine and Mouzon, 1914; Holmes, 1915), until dispelled by improved nursing care (Riddoch, 1917). The reflex depression proved to be only temporary, although of longer duration – days or even weeks – than in animals with 'spinal shock' (Hall, 1841).

During this phase of reflex depression after spinal transection, some noted an unexpected downward response of the great toe. Apart from casual remarks of Harris (1903) and van Woerkom (1911) the first descriptions were those of the Dejerines and Mouzon (1914, 1915), who noticed no special features, and of Holmes (1915): '. . . a simple flexion of the great toe . . . differs from the normal flexor response in that it is slower and smaller in range . . .'. Subsequent early reports came from Collier (1916) and from Guillain and Barré (1917). These French authors also stressed the slowness of the response, but differed from Holmes' observations in remarking that the downward toe movement was often ample, and had a long latency and duration. Finally, Riddoch (1917) showed in a comprehensive study how this initial plantar flexion was gradually replaced by a Babinski sign in the course of a few weeks, as the flexion reflex of the leg emerged from spinal shock. This transition had also been noted by Holmes (1915), but he inferred – perhaps still influenced by Bastian's law – that the transverse lesion was probably not complete in patients with toe reflexes.

Quite to the contrary, in the course of time an initial downward response of the toes has come to indicate a poor prognosis for recovery (Editorial, 1960), although it has been reported in a functionally incomplete lesion (Minkowski, 1923). With the wide-spread introduction of assisted ventilation, the reflex has also been reported in brain death (Ivan, 1973). As far as spinal functions are concerned, primary brain death is comparable to a transection at C_1. In another series of such patients, toe responses were not found (Jørgensen, 1973).

Why does the downward toe response 'escape' from spinal shock? Holmes (1915) argued that it was a unisegmental reflex, as opposed to the complex intersegmental mechanism of the flexion reflex. Moreover, it was pointed out by Riddoch (1917) and Guttmann (1952, 1976) that sacral segments of the cord suffered less depression than rostral segments: reflex activity of the external sphincters disappeared only for a short time or not at all, and ankle jerks were also found in the earliest phase after injury. This gradient in spinal shock had been noted earlier in animals (Sherrington, 1906).

The mechanism of reflex depression has remained elusive, although it became clear long ago that Goltz's hypothesis (1876) of inhibition by irritation from the lesion was untenable (Sherrington, 1906). Most information stems from animal experiments. The Sherringtonian concept of a diminished central excitatory state (Liddell, 1934; Ruch 1942) has been confirmed by demonstration of hyperpolarisation of motoneurones (Barnes et al., 1962). This is compatible with loss of excitation as well as with release of postsynaptic inhibition (van Harreveld, 1940). The morphological findings of Illis (1967) – temporary disorganisation of the synaptic zone – favour a negative causation. In man, conclusions have ranged from depressed fusimotor activity (Weaver et al., 1963) or depressed fusimotor activity plus initial decrease of motoneurone excitability (Diamantopoulos and Olsen, 1967) to increased presynaptic inhibition (Ashby et al., 1974).

These experiments in humans, however, are all based on H-reflexes; whether the amplitude of this reflex is a valid measure of motoneurone excitability has been put in doubt (Granit and Burke, 1973), and the results are certainly not applicable to exteroceptive reflexes.

A final question is whether disconnection of one descending pathway from the spinal cord is more important in the production of spinal shock than that of another. In animals, Sherrington (1906) concluded from the much more severe character of the depression when the transection passed below the pons that there was 'an aborally directed influence from some nucleus of the pontine or midbrain system'. He also found that a second spinal transection, caudal to the first one, had little additional effect. This phenomenon was later also found in some degree when spinal transection in monkeys was preceded by cortical ablations (Fulton and McCouch, 1937): the side of the body contralateral to the earlier cerebral lesion showed a faster return of the flexion reflex. McCouch et al. (1966) investigated this 'protective effect' of various lesions against subsequent spinal shock; asymmetry of reflex return was greatest after hemisection of the spinal cord, a little less after pyramidotomy, still slighter after ablation of area 4, and least marked after postcentral lesions.

We can summarize the literature on this subject as follows. After acute disconnection of the lumbosacral cord from the brain the Babinski sign is often absent as a result of general reflex depression (spinal shock). This is in turn caused by sudden loss of background excitation of spinal neurones via descending pathways from the cortex and brain stem. Instead, a slow and tonic downward response of the toes may be found; when the reflex depression is slowly wearing off, this unisegmental reflex gradually gives way to the flexion reflex, including the Babinski response.

Pyramidal syndrome without Babinski sign

More numerous than patients with spinal shock are patients who show some elements of the clinician's pyramidal syndrome (weakness, increased tendon jerks, spasticity and diminished abdominal reflexes), but without a Babinski response. That this could occur was noted by Babinski himself in his very first communications on the toe sign (1896 a, 1898).

Landau and Clare (1959) re-emphasized the occurrence of peripheral nerve lesions in cases with an unexpectedly absent Babinski sign, especially in bedridden patients, but reserved a role for individual variation in the remaining exceptions. Nevertheless, this paradox deserves further scrutiny, for absence of a sign can be as informative as its presence.

A possible solution advocated by Bucy et al. (1964) was that the Babinski response might be a pyramidal sign, but that the other features of the 'pyramidal syndrome' were not. Their thesis rested on a case history with the reverse problem: an upgoing toe sign with little else after a subtotal unilateral pedunculotomy. One might object to this interpretation because 17% of the pyramidal fibres had not degenerated, and part of these may have been functioning; moreover, there was some distal motor impairment and hyperreflexia. Voorhoeve and de Jong (1975), in a review, attributed only spasticity and hyperreflexia to interruption of descending systems other than the pyramidal tract. Meanwhile, however, the two recently reported patients with almost pure infarction of one pyramid (Chokroverty et al., 1975; Leestma and Noronha, 1976) showed the complete pyramidal syndrome, except for the abdominal reflexes which received no mention in either case, and for spasticity which was present in only one patient. Thus, proof is lacking so far that interference with descending pathways other than the corticospinal tract is essential for the production of the 'pyramidal syndrome'. Moreover, it would not help to explain absent Babinski signs in cases of proven pyramidal degeneration (Nathan and Smith, 1955).

Others have tried to relate the Babinski response to the proportion of damaged pyramidal fibres (Barnes, 1904), or to the level of interference with the pyramidal tract: Bychowski (1916) and van der Scheer (1926) thought that superficial lesions of the motor cortex could cause weakness without a Babinski sign.

The most pertinent approach to this problem is, however, to consider the termination rather than the name of the descending fibres in the spinal cord. A hallmark in this direction is a case study by Potts and Weisenburg (1910).They concluded that a Babinski response could be expected only when pyramidal 'leg fibres' were implicated. This may now seem self-evident, but many neurologists used to regard it as an alarm signal for the pyramidal system as a whole, and some were surprised by its absence in brachial monoplegia (Bergmark, 1909), or in focal epilepsy involving mainly face and arm (Tournay, 1925). To take account of the termination of pyramidal fibres also puts an end to speculations about the role of the ventromedial ('uncrossed') corticospinal tract in producing an ipsilateral Babinski response (Goldflam, 1903; Hawthorne, 1915), as these fibres rarely descend below the thoracic cord (Brodal, 1969) and mainly subserve axial movements (Kuypers, 1973).

Heterogeneity of the pyramidal syndrome?

The need to go even further than the restriction that a Babinski sign can follow only from a disturbance of pyramidal 'leg fibres' may not seem obvious at first notice. But so far the distinction is still a crude one. In the craniocaudal direction 'leg fibres' comprise projections to neurones from the L_1 segment down to S_2. As the upgoing toe sign is a manifestation of pathological recruitment of the extensor hallucis longus muscle into the flexion reflex synergy (see Chapter III), and as the motoneurones of this muscle are located in the dorsolateral part of the ventral horn of the L_5 segment (Sharrard, 1955), why should we *a priori* expect it when there is, for instance, weakness of hip flexion (L_1–L_3) or an increased knee jerk (L_2-L_4)? In addition to divergence of pyramidal projections in a craniocaudal direction, the descending innervation within each segment is not restricted to the anterior horns, but includes important contributions to the intermediate zone and even the dorsal horn (Kuypers, 1973). In consequence, if we should find that the Babinski sign showed little segmental specificity, particularly in relation to motor deficits (projections to anterior horn cells), it might be possible to connect it with multisegmental release of the flexion reflex as a whole (projections to interneurones, which are heavily interconnected).

The question I shall attempt to answer in this chapter is now as follows. *Given that the Babinski sign is not always associated with all other components of the pyramidal syndrome, is it possible to relate it more closely to certain features of the pyramidal syndrome? Such correlations might, in turn, give insight into physiological and anatomical connections:*

- *if the Babinski sign is released by a disturbance of projections to motoneurones, we should expect it to be always accompanied by motor deficits in foot and toe muscles;*
- *if the Babinski sign is the result of general release of the interneuronal pool in the intermediate zone of the spinal gray matter, we should expect exaggeration of the entire flexion reflex in all cases.*

A few earlier studies have registered the concurrence of various deficits and release phenomena in the lower limbs. Graeffner (1906) examined 116 patients with hemiplegia and found an increased knee jerk more often (77%) than an upgoing toe sign (63%). Lassek (1945) assembled 1600 cases with one or two Babinski signs from the literature: motor deficits (97%) and increased patellar reflexes (66%) were at the top of the list. Dohrmann and Nowack (1974) looked for mutual correlations among various features on the side of a Babinski sign (61 patients, some bilateral). For the leg they found significant correlations only between hyperreflexia and clonus (the authors admit they were not surprised by this) and between weakness and hypertonus.

In view of the theoretical presumption that the upgoing toe sign might be preferentially associated either with distal motor deficits or with exaggeration of the flexion reflex as a whole, examination of the patients in the present study included not only the power of various foot and toe movements, but also *the capacity to perform discrete and rapid movements of the foot and great toe,* as well as *the activity of the flexion reflex in other muscles than the extensor hallucis longus muscle alone.* Neither aspect has, to my knowledge, ever been studied quantitatively with the object of establishing correlations with other pathological signs. Another addition to the usual neurological examination was to look for associated movements of the toes during voluntary efforts.

Four categories of patients were studied:

1) patients with a unilateral Babinski response (the main group)
2) patients who showed pyramidal signs but lacked a Babinski response
3) patients who changed from group 1 to 2 or vice versa
4) patients showing a paradoxical downward response of the great toe after spinal transection.

METHODS

Patients

Within six months, 50 patients with a strictly unilateral and unequivocal Babinski sign were found among in-patients and out-patients and were included in the study. A restriction was that they should be well enough to co-operate in the various tests and did not have unrelated motor deficits, e.g. diseases of cerebellum, basal ganglia or peripheral nerves. They were also followed up, when possible and relevant, and without consultation of previous notes. A cerebral lesion was verified or supposed in 42 of the patients. The diagnosis was infarction in 33, tumour in three, Mills' syndrome in two (Mills, 1900), trauma in two, angioma in one, and infantile hemiplegia in one; in 19 of these 42 patients the onset of disease had been less than four weeks before the examination. The other eight patients had a (presumably) spinal lesion: six were thought to have multiple sclerosis, one had an old traumatic lesion, and one a recent infarction of the thoracic spinal cord.

A second group was formed in the same time span and from the same population by six patients who unexpectedly lacked a Babinski sign, without peripheral lesions to account for this. Four of these suffered from cerebral infarction, one had motor neurone disease, and one had been operated upon one year before because of an intracerebral haematoma.

A third group of five patients emerged from a follow-up study of patients with an acute lesion from the two preceding cross-sectional groups and hence overlaps with these. It included three patients who 'lost' a unilateral Babinski response that had been present for some time (first group), while other pyramidal signs remained. The two other patients developed an upgoing toe response only after an initial period in which it had been unexpectedly absent (group two).

The fourth group consisted of seven patients showing a paradoxical downward response of the great toe after spinal transection (primarily spinal trauma in four, cerebral death in three). These patients were seen in the neurosurgical unit, in the course of two and a half years.

Examination

1. *Plantar reflex*

All patients were examined in the supine position. Stimulation was performed with an orange stick, at the lateral plantar border and plantar arch, and was, if necessary (see Chapter IV), repeated on the lateral dorsum of the foot.

2. *Flexion reflex*

Apart from the extensor hallucis longus, special attention was paid to activation of the tibialis anterior muscle and more proximal flexors: tensor fasciae latae, knee flexors and hip flexors. An arbitrary grading scale served to record the findings:

–	no visible activity
±	flicker of movement
+	slight knee or hip flexion
++	knee lifted from couch
+++	similarly, but even on slight stimulation with finger

3. *Power*

The following movements were tested:

- extensors: hip extension, knee extension, knee adduction, foot plantar flexion, toe plantar flexion
- flexors: hip flexion, knee flexion, dorsiflexion of foot (starting from a dorsiflexed as well as from a relaxed position), foot eversion, dorsiflexion of hallux.

Grading was according to the MRC scale (Medical Research Council, 1943); slight weakness was recorded as '4+' or even '5-'.

4. *Skill*

Four tests were used:

- dorsiflexion of the great toe, separately from the foot
- wriggling of the little toes

- rapid foot tapping on the couch, against the examiner's hand
- rapid up-and-down movements of the great toe, with the foot planted on the floor.

The results of the latter two tests were quantified by counting the number of taps in 10 seconds. Skill was considered to be impaired when this number was less than 3/4 of that on the control side, or when there were obvious qualitative differences, such as dysrhythmia, or associated movements of the little toes on one side only.

5. *Associated dorsiflexion of the great toe*

This was investigated in three ways:

- ipsilateral hip flexion } against resistance
- contralateral hip flexion }
- contralateral rapid toe movements

6. *Tone*

Passive resistance was separately examined in the ankle (by shaking the leg and foot with the patient supine), the knee (if possible also by comparing pendular movements with the patient sitting), and the hip.

7. *Tendon reflexes*

Examined were: ankle jerk (when necessary also in the kneeling position), knee jerk, adductor reflex and biceps femoris reflex. These were recorded on a nine-point scale (Mayo Clinic, 1963) ranging from minus four (absent) through zero (normal) up to plus four (clonus).

8. *Abdominal reflexes*

Two levels were tested on both sides, above and below the umbilicus. In men, the cremasteric reflex was also included (the first patient group consisted of 27 men and 23 women).

9. *Wasting*

The circumference of the thigh was measured 15 centimetres above the medial margin of the knee joint, the calf at its maximal girth. Wasting was assumed to be present when the difference was two centimetres or more on one of these measurements.

RESULTS

1. UNILATERAL BABINSKI SIGNS

Homogeneity of the group

The various findings in all 50 patients with a Babinski sign are shown in table XVI. The results for the muscles that move the foot are considered separately under the headings weakness, skill, tone and tendon jerks. For the eight patients with a (presumably) spinal lesion the profile on the side with the Babinski response did not appreciably deviate from that in the 42 patients with cerebral pathology, but this does not rule out some differences if more patients with spinal lesions could have been included. It was especially in the spinal group that motor impairment occurred on the control side (proximal weakness in two, paralysis in one). It must be emphasized in general that the contralateral side was not necessarily normal – it only did not show a Babinski response and was therefore interesting for comparison.

To investigate the effect of time in this sample, the 42 patients with cerebral lesions were divided into two groups, according to whether the illness had lasted less than four weeks (19 patients) or longer (23 patients). On comparison, hypotonia occurred significantly more often in the acute group (10/19 versus 1/23; Fisher test: $p < 0.001$), and a reverse tendency was found for spasticity (7/23 versus 1/19; $p = 0.05$). As this 'change' in time was not associated with any other difference between the two groups, they were considered as one, together with the spinal group.

Weakness and loss of skill in the foot

The most frequent finding accompanying the extensor plantar response was weakness of foot movements: 76%. In almost half of this number (32%) weakness was only slight. This always involved dorsiflexion of the foot, dorsiflexion of the hallux, foot eversion or a combination of these movements (see table XVII); weakness was never confined to physiological extensors (plantar flexion of foot or toes). In 10% the weakness was confined to the foot.

Loss of skill in the foot was always a relative judgment, i.e. on comparison with the control side. If a difference was found, it almost always concerned the capacity to perform rapid foot or toe movements. With regard to the other tests listed in the 'methods' section, it was found that every patient (with one exception) who could dorsiflex the great toe at all, was able to do this without moving the foot, at least after a few

TABLE XVI
FREQUENCY OF VARIOUS SIGNS ON THE SIDE OF A UNILATERAL BABINSKI RESPONSE IN 50 PATIENTS

1. *Flexion reflex*

– exaggerated	58%
– symmetrical	38%
– diminished	4%

2. *Weakness*

– paralysis of foot	28%	weakness of foot: 76%
– marked weakness (MRC 1–4) of foot	16%	
– slight weakness of foot	32%	
– only proximal weakness	14%	no weakness of foot: 24%
– no weakness	10%	

3. *Loss of skill*

– with paralysis or marked weakness of foot (44%)	44%	
– with slight weakness of foot (32%)	30%	
– with only proximal weakness (14%)	12%	no weakness of foot but loss of skill: 16%
– with no weakness (10%)	4%	
– no loss of skill	10%	

4. *Associated dorsiflexion of great toe*

	a) *on ipsilateral hip flexion*	b) *on contralateral hip flexion*	c) *on contralateral rapid toe movements*
– not possible	14%	2%	2%
– positive	14%	18%	2%
– bilaterally positive	20%	4%	2%
– positive on control side	18%	6%	14%
– negative	34%	70%	80%

5. *Tone*

– increased in ankle (at least)	10%
– increased in knee only	6%
– symmetrical	62%
– decreased in knee only	8%
– decreased in ankle	14%

6. *Tendon reflexes*

– increase of ankle jerk only	10%	74%
– increase of ankle jerk plus other reflexes	60%	
– only other reflexes increased	4%	
– symmetrical	22%	
– only other reflexes decreased	2%	
– ankle jerk decreased	2%	

(cont. next page)

7. *Abdominal reflexes*		
– diminished	42%	
– only cremasteric reflexes diminished	6%	(11% of men)
– symmetrical	22%	
– absent	26%	
– decreased on control side	4%	
8. *Wasting*		
– present	10%	
– absent	90%	

attempts. And examination of toe wriggling was even less rewarding, as it was difficult to estimate differences between the two sides, apart from obvious paralysis. Comparing the rate and rhythm of rapid foot or toe movements appeared to be a much more sensitive test. This ability may of course also suffer in diseases of the cerebellum or basal ganglia, which, however, were excluded from this study. The two procedures gave similar results in most patients, but occasionally one or the other was more sensitive. In either test, normal values range from 20–40 taps in 10 seconds.

All patients with foot weakness (76%) were also found to have impairment of skilled movements, with one exception. This patient, diagnosed as having multiple sclerosis, showed on both sides a slight weakness of the foot and a moderate performance of rapid movements, but a Babinski sign was only found on one side. Hence the problem in this case is rather why the second Babinski response was absent (see below) than why weakness was not accompanied by loss of skill.

TABLE XVII
DISTRIBUTION OF SLIGHT FOOT WEAKNESS (16/50 PATIENTS)
(between brackets: weakness confined to foot)

– dorsiflexion of hallux, dorsiflexion of foot and eversion of foot	7	(2)
– dorsiflexion of foot only	4	(1)
– dorsiflexion of hallux only	2	(1)
– dorsiflexion of hallux and eversion of foot	2	(1)
– eversion of foot only	1	
	16 (32%)	(5) (10%)

TABLE XVIII
PATIENTS WITH A BABINSKI SIGN AND NO WEAKNESS OR LOSS OF SKILL IN THE FOOT

(i.h.f. = ipsilateral hip flexion; c.h.f. = contralateral hip flexion; r.t.m. = rapid (contralateral) toe movements; c. = control side).

case nr.	*age*	*sex*	*diagnosis and duration of disease*	*side of Babinski sign*	*flexion reflex*	*weakness*	*associated movements of great toe*	*tone*	*tendon reflexes*	*abdominal reflexes*	*wasting*
1.	47	F	?spinal MS 4 months	R	symmetrical (+)	none	i.h.f. – c.h.f. + r.t.m. –	normal	↑	symmetrical	–
2.	60	F	R parasagittal meningeoma, operated about 10 yrs before	L	↑ (+/±)	none	i.h.f.+,c.+ c.h.f.+,c.+ r.t.m.–	normal	absent on both sides	↓	–
3.	63	F	cerebral infarction, 4 months before	R	↑ (+/±)	only knee flexion (4+)	i.h.f.+,c.+ c.h.f.+ r.t.m. c.+	↑	↑	absent	–
4.	34	M	infantile hemiparesis	R	↑ (++/+)	none	i.h.f.–,c.+ c.h.f.+ r.t.m.–	normal	↑	symmetrical	+

Of the 12 patients without evidence of foot weakness, impairment of skilled movements could still be demonstrated in eight. Six of these eight patients had discrete proximal weakness, two had no loss of power at all.

In the whole group of 50 patients with a unilateral upgoing toe sign, only four remained who showed neither weakness nor loss of skill. All relevant findings in these exceptional patients are listed in table XVIII. To find in all four the relatively rare sign of hallux dorsiflexion on contralateral hip flexion against resistance is statistically highly significant (occurrence in the whole group 22%, the Fisher test gives p = 0.001). Because this phenomenon was also found on the control side of other patients, (table XVI) it can hardly explain in itself why a Babinski sign occurs in the absence of demonstrable motor deficit in the foot.

In summary, 92% of the patients with a unilateral Babinski sign showed some motor deficit in the foot, 16% having a disturbance of rapid movements alone.

Increased tendon reflexes

Hyperactivity of tendon jerks came second in the list of motor abnormalities found in conjunction with a Babinski sign: 74%. There was no correlation (either positive or negative) with the presence of other pathological signs: table XIX, A, shows that the distribution of the other signs in patients with symmetrical tendon jerks is quite comparable to that in the group of 50 patients as a whole (Fisher test: differences not significant).

Table XVI shows that asymmetry usually involved various reflexes at the same time. The ankle jerk was rarely increased alone (10%), and not preferentially in cases with a purely distal motor deficit: of the seven patients with motor impairment in the foot only, all or most tendon reflexes were exaggerated in four, only the ankle jerk in one, and in two patients the tendon jerks were symmetrical. The jerks elicited from the adductor and biceps femoris muscles rarely gave extra information, and obtaining the latter was sometimes a little awkward. These findings imply that hyperreflexia usually involves all leg muscles, with little segmental specificity or differentiation between (physiological) flexor and extensor muscles.

Exaggerated flexion reflex in proximal muscles

By definition the flexion reflex is more active on the side of a unilateral Babinski response, as the extensor hallucis longus muscle is recruited into the synergy. One of the principal questions in this chapter was how often this also involved increased reflex activity in proximal flexor muscles.

TABLE XIX
LACK OF CORRELATION BETWEEN OTHER PYRAMIDAL SIGNS IN 50 PATIENTS WITH A UNILATERAL BABINSKI RESPONSE

	exaggeration of flexion reflex	*weakness or loss of skill in the foot*	*spasticity*	*increased tendon reflexes*	*decrease of abdominal or cremasteric reflexes*
All patients with a Babinski sign (n = 50)	58%	92%	16%	74%	48%
A. Patients without ipsilaterally increased tendon reflexes (n = 13)	6/13 (46%)	11/13 (85%)	0/13 (0%)	----	3/13 (23%)
B. Patients without ipsilaterally increased flexion reflex in proximal muscles (n = 21)	----	20/21 (95%)	2/21 (10%)	14/21 (67%)	8/21 (38%)

Leaving asymmetry aside for a moment, almost all legs with a Babinski sign showed at least some contraction in tensor fasciae latae, hamstrings or iliopsoas. There were only two exceptions among the 50 patients.

Exaggeration of the flexion in proximal limb muscles accompanied the Babinski response in 58% of the patients. In other words, *the Babinski sign is as often as not accompanied by hyperactivity of the flexion reflex as a whole.*

It is clear from table XIX, B, that release of the flexion reflex in proximal limb segments occurs independently of other signs, and in particular of an increase in tendon jerks. The lack of correlation between these two forms of hyperreflexia also holds true when 'absolute' values are considered instead of asymmetry: figure 12 shows that every activity level of the flexion reflex can be found together with a wide range in activity of tendon reflexes, and vice versa.

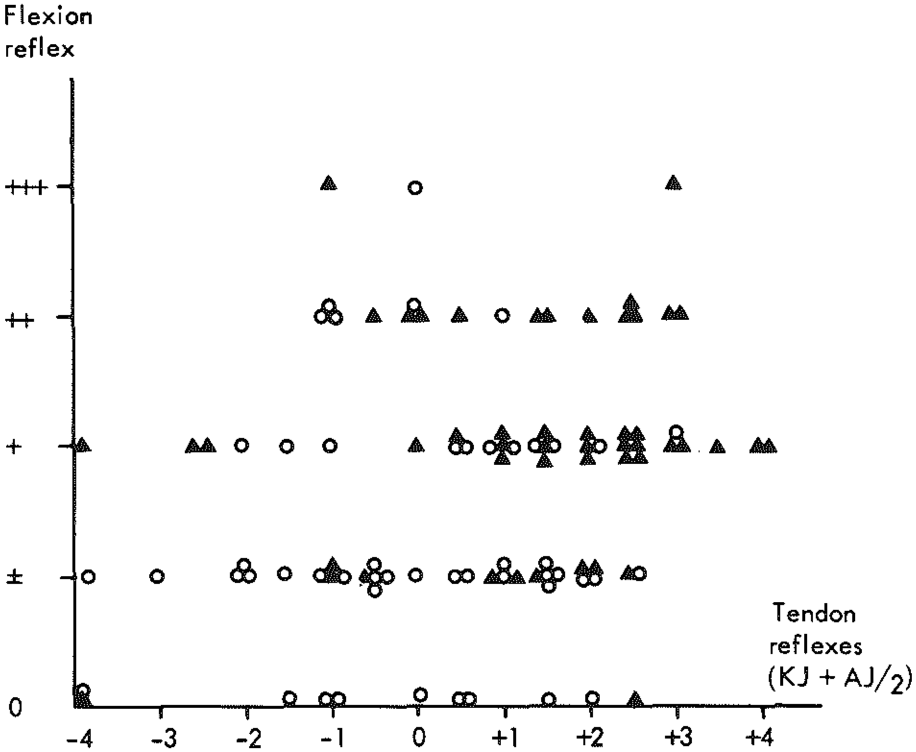

FIGURE 12. *Lack of correlation between 'absolute' values of flexion reflex and tendon reflexes in 50 patients with a unilateral Babinski sign (triangles = side of Babinski sign; circles = control side).*

TABLE XX
PATIENTS SHOWING PYRAMIDAL SIGNS WITHOUT A BABINSKI RESPONSE.

(i.h.f. = ipsilateral hip flexion; c.h.f. = contralateral hip flexion; r.t.m. = rapid (contralateral) toe movements; c. = control side).

case nr.	*age*	*sex*	*diagnosis and duration of disease*	*side*	*flexion reflex*	*weakness*	*skill*	*associated movements of great toe*	*tone*	*tendon reflexes*	*abdominal reflexes*
5.	59	M	motor neurone disease	R	symmetrical (+)	hip fl. 5.– foot ev. 4 EHL 4	impaired	i.h.f. – c.h.f. – r.t.m. –	bilat. ↑	bilat. clonus	symmetrical
6.	53	M	cerebral infarction (L), 2 years before	R	symm. (±)	foot dorsifl. 5^- ?	symm.	i.h.f.+,c.+ c.h.f.+,c.+ r.t.m.–	normal	↑	symm.
7.	47	M	cerebral infarction (R) after subarachnoid haemorrhage	L	symm. (+)	hip fl. 4 knee fl. 4	symm.	i.h.f. + c.h.f. + r.t.m. –	normal	symm.	↓
8.	68	M	intracerebral haematoma (L) operated 1 year before	R	symm. (±)	none	symm.	i.h.f. – c.h.f. – r.t.m. –	normal	↑	↓

Other signs

Associated dorsiflexion of the great toe on ipsilateral or contralateral hip flexion against resistance was relatively rare, and occurred on the side of the Babinski sign as well as on the control side (see table XVI). Mirror movements of the toes on the control side were a more reliable sign of contralateral motor impairment, but this was never found in the absence of contralateral weakness.

Reduced abdominal reflexes, represented much higher in the spinal cord, were present in 42% (the reverse occurred in two patients). Reliable though this finding is as an index of disease, the high absence rate of the reflex on both sides (26%) makes it difficult to estimate correlations. Wasting was rare (10%); in two instances it was found in syndromes known to be associated with 'supranuclear atrophy' (Mills' syndrome and infantile hemiplegia).

2. PYRAMIDAL SIGNS WITHOUT BABINSKI RESPONSE

Six patients lacked a Babinski response while other pyramidal signs were present. Two developed it at a later stage and will be discussed later. The other four are represented in table XX. None of them showed paralysis of the extensor hallucis longus muscle, so that peripheral damage is not a ready explanation for the absent upgoing toe response, even in the patient with motor neurone disease (case 5).

The last-mentioned patient was, however, the only case of these four in which definite motor impairment of the foot and toe was found. Case 6 showed equivocal weakness of foot dorsiflexion but no loss of skill, case 7 had only proximal weakness, with symmetrical skilled foot movements, and case 8 had no motor deficit at all. Other pyramidal signs were increased tendon jerks (cases 5, 6 and 8) and reduced abdominal reflexes (cases 7 and 8). The flexion reflex in proximal muscles was (symmetrically) present in all four, giving no reason to suppose a defect in the spinal pathways of the plantar reflex.

Having learnt that motor impairment of the foot is so highly associated with the Babinski sign in the first group of patients, and that it is not found in three of these four patients, one can only conclude that the absence of the Babinski response is not really so unexpected after all.

3. BABINSKI SIGNS APPEARING IN OR DISAPPEARING FROM THE PYRAMIDAL SYNDROME

In the two preceding groups of patients the relationship between the upgoing toe response and other pyramidal signs was studied by a cross-sectional survey of pyramidal features with and without a Babinski sign. Obviously it is also of great interest to study 'dynamic' relationships: when a Babinski sign disappears while other signs remain, or, inversely, appears only after some time, what other findings change with it?

Vanishing Babinski signs

Three patients in the first group of 50 patients 'lost' their Babinski response while other abnormal features remained.

Case 9 (improvement of power and skill).
A 60-year-old driver with an infarction in the left cerebral hemisphere showed on the 9th day of his illness a mild right-sided weakness of hip flexion (4+) and foot dorsiflexion (4+), with reduced skill and hyperreflexia. The right plantar response was upward, with a brisk flexion reflex (++; control side +). Six weeks later, power and skill were normal. Both plantar responses were absent, the flexion reflex + on the previously weak side and ± on the other. The tendon reflexes were still increased on the right.

Case 10 (improvement of skill).
A 53-year-old maintenance technician suffered a mild stroke in the territory of the left middle cerebral artery. On the 10th day his only deficit was some weakness of hip flexion (4+) and slowing of rapid foot and toe movements. All tendon reflexes of the right leg were increased, and there was a Babinski response on the same side (both flexion reflexes ±). Three weeks later he had no longer any motor disability, nor a Babinski sign; only a definite hyperreflexia remained.

Case 11 (waning of flexion reflexes).
A 56-year-old publican sustained an acute right-sided hemiparesis. On the 2nd day he was found to have moderate weakness of the right leg (all flexor movements 4+), with impaired skill and hyperreflexia. Flexion reflexes were bilaterally brisk (++), with a Babinski sign on the right. On day 10 the clinical picture was practically unchanged, but for the right plantar response which was now downward, and for both flexion reflexes which were weak (+).

Late Babinski signs

Two patients initially lacked a Babinski sign in the presence of other pyramidal signs, but developed it eventually. Both were hemiplegic.

Case 12 (initial absence of flexion reflex).

A 58-year-old nurse sustained a subarachnoid haemorrhage from one of two left-sided berry aneurysms. On the 4th day she had a paralysed right foot, symmetrical tendon jerks, and no flexion reflex or Babinski sign on either side. On the 13th day she developed a right hemiplegia. Examination on day 24 showed a paralysed right leg, increased tendon reflexes, and again no flexion reflexes. On day 39, while the right leg was still paralytic, plantar stimulation resulted on both sides in slight reflex dorsiflexion of the foot, on the right accompanied by weak contraction of the extensor hallucis longus.

Case 13 (initial depression of flexion reflex).

A 50-year-old industrial manager suddenly developed a right hemiplegia, without impairment of consciousness. On the 3rd day his right leg was paralytic. No tendon reflexes could be obtained on either side (this had been noted before by insurance physicians). Plantar stimulation on the left gave a downward response of the toes, together with contraction of tensor fasciae latae, tibialis anterior and biceps femoris (+). On stimulation of the right sole the toes remained immobile, and there was only weak action of the ipsilateral biceps femoris and adductor (±). Three months later, the patient had regained some power in the right leg, but not in the foot. At this time the right plantar reflex was clearly upward, with exaggeration of the flexion reflex (++) as compared with the other side (still +), and some weak tendon jerks could be elicited in the right leg.

4. PARADOXICAL DOWNWARD RESPONSE OF THE GREAT TOE AFTER SPINAL TRANSECTION.

The strange and almost impudent phenomenon of a downward toe response after disconnection of the lumbosacral spinal cord from the brain, first encountered in victims of the first world war (see p. 115), was found in seven patients. Four suffered primary injury of the spinal cord, the other three were cases of intractable cerebral lesions (diffuse traumatic damage in two, infarction of one hemisphere in the other) which caused progressive deterioration of brain stem function. Reflex plantar flexion of the toes was certainly not an invariable finding in such cases. The patients with spinal lesions were examined within one day of the injury, the patients

TABLE XXI

PATIENTS SHOWING A PARADOXICAL DOWNWARD RESPONSE OF THE GREAT TOES AFTER SPINAL TRANSECTION

case nr.	*age*	*sex*	*clinical level of spinal transection*	*special features of downward toe responses*	*flexion reflex*	*ankle jerk*	*knee jerk*	*abdominal reflexes*	*cremast. reflexes*	*comments*
S1	32	M	D 3	long latency, slow and prolonged	knee flexion R (±)	-3/-1	–/–	–/–	–/–	
S2	16	M	D 8	fast	–/–	–/–	–/–	–/– +/+	–/–	
S3	33	F	D 4	weak, rather slow	bilateral knee and hip flexion ±)	–/–	–/–	–/–	....	
S4	33	M	C 7	long latency, moderately slow; see figure 13	–/–	+1/+1	+2/+2	–/–	–/–	incomplete lesion: able to move R knee flexors, tib. ant., ext. hall. longus (MRC 4); some sensation left in L leg
S5	41	M	C 1 (brain death)	weak, prolonged	–/–	–/–	–/–	–/–	+/+ (tonic)	also present in earlier phase with extensor spasms
S6	22	F	C 1 (brain death)	'normal'	–/–	–/–	–/–	?	....	bilateral Babinski signs present when still breathing spontaneously
S7	25	M	C 1 (brain death)	fast	–/–	–/–	–/–	?	–/–	

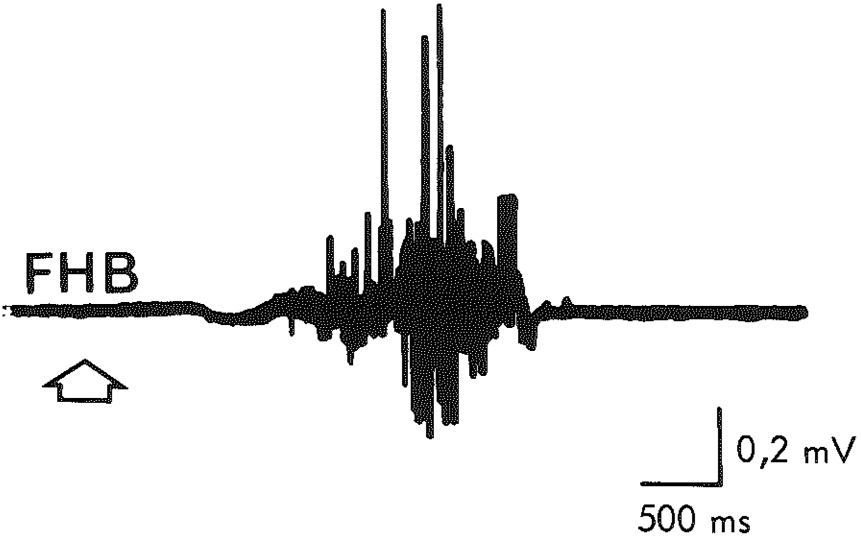

FIGURE 13

Electromyogram from the flexor hallucis brevis in a patient with a paradoxical downward response of the great toe after spinal transection (patient S4, table XXI).

with a clinical diagnosis of brain death within hours of cessation of spontaneous respiration. Follow-up of the four paraplegic patients was limited by transfer to other centres, but case S1 and S3 showed bilateral Babinski signs after one month.

The early findings in this group are listed in table XXI. The reflex plantar flexion was bilateral in all cases. It did not always show the distinguishing features of long latency, slow movement and prolonged duration; in two cases it was even unusually fast. In one case (S4) electromyography was possible, and the response was accompanied by electrical activity in the flexor hallucis brevis (figure 13). Although activation of the extensor hallucis longus muscle by plantar stimulation was conspicuously absent, weak contraction of knee or hip flexors occurred in two cases (S1 and S3). Finally, it must be stressed that the paradoxical downward response can appear without complete isolation of the lumbosacral cord: in case S5 (brain death) it was for some time observed together with extensor spasms (respiration had already ceased), and patient S4 (lesion at C_7) was able to perform some distal movements on one side, including dorsiflexion of the toes!

DISCUSSION

Two principal features emerge from the cross-sectional and longitudinal case studies presented above. First, there seems to be a close association between occurrence of the Babinski sign and impaired voluntary control of the foot and toes, the latter either in the form of weakness or as a reduced capacity to perform rapid alternating movements. Secondly, the Babinski sign is not necessarily found together with hyperactivity of the flexion reflex in other muscles than the extensor hallucis longus. On the other hand, whatever the degree of motor deficit in the foot, a Babinski sign can appear only if the intraspinal pathways of the flexion reflex synergy are operative. Both these factors, impaired pyramidal control and the activity of the flexion reflex, will be separately considered below. This is followed by a discussion of the probable interaction of the two pathways, as they converge upon the motoneurones of the extensor hallucis longus muscle. Finally, the concept of 'the upper motor neurone' as the substrate of the pyramidal syndrome will be scrutinized.

IMPAIRED PYRAMIDAL CONTROL OF THE FOOT

Linkage between Babinski sign and loss of skilled foot movements

If a disturbance of the pyramidal tract affects projections to motoneurones of distal muscles but is not more than slight, the only clinical manifestation may be impairment of rapid or fractionated movements. For the foot this is less well known than for the hand, but testing of this capacity proved to be equally rewarding. A delayed single reaction time in patients with pyramidal lesions has been found for the leg as well as for the arm by Jung and Dietz (1975). That the foot, too, has a high and therefore potentially vulnerable ability to perform fine movements is demonstrated by the well-known paintings of artists deprived of the use of their arms.

In the group of 50 patients with a unilateral Babinski sign impairment of rapid foot or toe movements was found in all subjects with foot weakness, with one exception (see p. 125). And of the remaining patients with normal power of foot movements (24%), reduced skill was found in two thirds. Thus, *loss of skill can be regarded as an additional expression of a central motor deficit, more sensitive than weakness.* Only 8% of Babinski signs was unassociated with any demonstrable motor deficit in the foot.

The association between the Babinski sign and impairment of voluntary foot movements is reinforced by the finding that two of the three patients in this group who were eventually found to have 'lost' their Babinski sign, at the same time regained full control of foot movements (cases 9 and 10,

p. 132). The fact that both abnormalities disappeared together in these cases also goes some way to dispel the possibility that the general population might harbour many Babinski signs without motor deficit, patients presenting at hospital being biased towards weakness. Moreover, in a recent survey of 808 elderly subjects at home, Broe et al. (1975) found a completed stroke in 7.3%, upgoing toe signs plus hyperreflexia without definite diagnosis in 1.6%, and isolated Babinski signs in only 1.0% (bilateral in half).

The next most frequent sign accompanying the Babinski response was increase of tendon reflexes (74%). However, hyperreflexia was not only absent in a quarter of the 50 patients with a unilateral Babinski sign, it also persisted in two cases where the upgoing toe sign and motor impairment of the foot disappeared simultaneously. In other words, the Babinski response and increase of tendon jerks occur regularly without one another. Hence one is not entitled to expect a Babinski sign on the strength of increased tendon jerks alone (cases 6 and 8, table XX). All other signs were found in half the patients at most and could be attributed even less to the same part of the lesion as the Babinski response.

Within the pyramidal syndrome, only impairment of skilled foot movements is so intimately related to occurrence of the Babinski sign that these two pathological features can be assumed to result from disturbance of identical or similar pyramidal fibres, i.e. fibres having the same termination.

Proximal versus distal motor deficit

One patient who 'lacked' a Babinski sign (case 7, table XX) showed weakness of hip and knee flexion (MRC 4) and reduced abdominal reflexes, but power and skill in the foot were normal. In retrospect the absence of the Babinski response is therefore not very surprising. It has even been questioned if the pyramidal system is concerned with proximal movements at all. For monkeys, Tower (1940) concluded from her experiments (medullary pyramidotomies) that the corticospinal system was almost exclusively concerned with distal motor functions. The work of Kuypers (1973) has modified this concept: there are two separate brain stem pathways for control of distal and of proximal and axial movements, both with a pyramidal contribution. For man, in the two recent case reports of unilateral infarction of the medullary pyramid (Chokroverty et al., 1975; Leestma and Noronha, 1976) some movements on the affected side were preserved, but these were not described in detail.

On the other hand, disease seldom produces lesions that exclusively affect descending projections to proximal muscles. This is illustrated by seven patients in the first group in whom weakness was confined to proximal muscles but who nevertheless showed a Babinski sign and also (except one) reduced skill in the foot. Similar clinical experiences led Walshe (1947) to postulate that the various defects in hemiplegia represent 'a change of degree, and not of kind', distal movements always suffering first. However often this view may be confirmed by everyday practice, its essence is refuted by occasional exceptions, by the experimental evidence cited above, and by theoretical considerations.

Strictly speaking, if a Babinski sign is expected only when pyramidal control fails at the level where the motoneurones of the extensor hallucis longus muscle are located, the only relevant factor in the examination of the motor system would seem to be the power and skill of this very muscle. Nevertheless it was thought wise not to rely on too few tests, as a clinical examination can overlook disturbances of delicate functions which are quite annoying to the patient himself (Brodal, 1973). It is also important to bear in mind that the motor system has a considerable 'reserve capacity', at least as regards power: Sharrard (1955) studied the spinal cord of patients who died from poliomyelitis and found that motor cell destruction was always much more severe than would have been expected. Individual differences in this 'reserve' may have contributed to the apparently random distribution of slight weakness of the foot in table XVII. As the motoneurones active in dorsiflexion and eversion of the foot are intimately related to those of the extensor hallucis longus, I took account of defects in power and rapidity of these movements as well. This was borne out by the occasional finding of defective 'toe-tapping' when only foot dorsiflexion seemed to be weak, and vice versa.

Involuntary dorsiflexion of the great toe

It is recounted in Chapter I how from early on attention has been drawn periodically to dorsiflexion of the hallux as an associated movement, accompanying ipsilateral or contralateral hip flexion against resistance. The consensus of previous work holds that it is an inconstant feature in pyramidal lesions, and the ipsilateral phenomenon has also been found in normal subjects (de Souza and de Castro, 1913; Brain and Wilkinson, 1959).

In the patients of this study, dorsiflexion of the hallux with these two procedures was infrequent, and it was found on the control side as well as on the side of the Babinski response, and also bilaterally (see table XVI). The mechanism of its appearance must be irradiation to extensor hallucis longus motoneurones of impulses that are directed to proximal flexor muscles; it is not clear whether this irradiation takes place in the cerebral cortex or in the interneuronal zone of the spinal cord, but in any case it has little or no value as a feature of pyramidal involvement. The recent claim of Hindfelt et al. (1976) that the 'crossed upgoing toe sign' can be regarded as a reliable and sometimes superior test is not confirmed by my study, nor by previous work.

In contrast, the occurrence of mirror movements when fast toe movements were attempted in a weak foot proved to be a much less equivocal sign, although it did not provide new information.

ACTIVITY OF THE FLEXION REFLEX

A prerequisite for the Babinski sign

As the Babinski sign is part of the flexion reflex synergy (see Chapter I), the flexion reflex pathways have to be activated in order to produce an upgoing toe. Recruitment of the extensor hallucis longus is only rarely the minimal response: in the 50 patients with a unilateral Babinski sign the stimulus intensity was no higher than necessary to elicit a clear hallux response, and in 48 of the 50 patients more proximal leg muscles were activated at the same time (figure 12). Although no special notes were kept about relative thresholds, and individual variations could occur, the tensor fasciae latae was often the first muscle to respond visibly (Brissaud, who described this part of the flexion reflex in 1896, found the same). On more intense stimulation this response is joined successively by tibialis anterior (with or without extensor hallucis longus), by knee flexors at comparable thresholds, and finally by hip flexors when the reflex is very active. Landau and Clare (1959) recorded electromyographically from several leg flexors and found activity of the tensor fasciae latae only late in the sequence, i.e. with strong stimuli, but on inspection contraction of this muscle is easy to note because of its superficial location. In patients with paraplegia, Walshe (1914) found the biceps femoris muscle to be the motor focus of the flexion reflex, whereas Dimitrijević and Nathan (1968) observed individual idiosyncratic responses.

With these qualifications in mind, we may assume that the motoneurones of the extensor hallucis longus muscle are usually not the first to be activated by the flexion reflex pathways in patients with a Babinski response. Hence when a Babinski sign is absent in a weak foot, we may infer from an absent or feeble response in proximal leg flexors that there is insufficient activity in the flexion reflex pathways that project upon the extensor hallucis longus muscle. This inexcitability may be the result of individual variation (case 12, p. 133) or of initial reflex depression (case 13, p. 133). Conversely, a Babinski sign can disappear because the activity of the flexion reflex dwindles after the acute episode (case 11, p. 132).

The most striking examples of Babinski signs that fail to appear for this reason are provided by cases of acute spinal transection, where reflex depression is generalized. Proximal flexor muscles are nevertheless activated in some of these patients (table XXI, case S1 and S3); the same has been noted by Guillain and Barré (1917), and by Guttmann (1976). This dissociation may reflect the preferential activation of proximal muscles by the flexion reflex pathways rather than deviation from the rule that spinal shock affects proximal more than distal segments of the spinal cord.

Supraspinal control

In not more than 58% of those patients in whom a Babinski sign indicated abnormal recruitment of the extensor hallucis longus muscle into the flexion reflex, there was also exaggeration of the flexion reflex in more proximal limb muscles. Conversely, asymmetry of the flexion reflex can even remain after disappearance of the Babinski sign (case 9). Therefore *the appearance of the Babinki response cannot be explained simply by a pathological overactivity of the flexion reflex as a whole:* the descending fibres which, when interfered with, release the flexion reflex in proximal muscles, are separate from the normal pyramidal control that isolates the motoneurones of the extensor hallucis longus musle from the flexion reflex synergy. Release of the entire flexion reflex, involving several segments, is probably the result of increased activity of the interneuronal pool in the intermediate zone of the spinal gray matter, whereas the Babinski sign seems to be associated with more specific projections. This is of course not to say that reflex activity of proximal flexor muscles has no relationship at all to recruitment of the extensor hallucis longus muscle, but only that the Babinski sign can appear without asymmetry of other components of the flexion reflex.

Investigation of which descending pathways are concerned with control of the flexion reflex as a whole has concerned only animals. In the cat, interneurones mediating the flexion reflex are tonically inhibited by the medial portion of the reticular formation of the brain stem (Eccles and Lundberg, 1959; Holmqvist and Lundberg, 1961) and they are facilitated by the pyramidal tract (Lundberg and Voorhoeve, 1962) and by the rubrospinal tract (Hongo et al., 1972). In decerebrated macaques, Chambers et al. (1970) found enhancement of skin reflexes after section of the dorsolateral funiculi of the spinal cord, but interruption of the ventrolateral funiculi suppressed cutaneous reflexes; the authors supposed that both effects were mediated by reticulospinal fibres.

As regards man, we are rather in the dark. Release of the flexion reflex as a whole shows as little segmental specificity as an increase in tendon jerks, which is a pyramidal feature (Chokroverty et al., 1975; Leestma and Noronha, 1976). However, exaggeration of the flexion reflex and of tendon jerks appeared to occur quite independently. A striking dissociation between exteroceptive and proprioceptive hyperreflexia has also been noted in, for instance, the Holmes-Adie syndrome (Swash and Earl, 1975). This does not exclude the possibility that release of the flexion reflex is a pyramidal sign, but one would still have to invoke different projections. An argument for at least partial control of the flexion reflex by brain stem centres is that flexor spasms are much more common after spinal lesions than in cerebral disease. The presence of separate descending fibres to control the segmental relay of myelinated and unmyelinated afferent fibres (Kugelberg, 1948) has not been substantiated.

The reverse side of the question is which of the descending fibre systems provide the normal facilitation of flexion reflex pathways that is disturbed in spinal shock and in the acute phase of extensive cerebral lesions (case 13). Some evidence is furnished by the patients in this study who suffered from acute spinal transection (table XXI). One patient (S4), with an incomplete spinal cord lesion at C_7, showed total inactivity of the flexion reflex (including the hallux, which was downgoing), although he was able to dorsiflex the great toe voluntarily. Hence we cannot easily ascribe depression of reflex activity in this patient to an interruption of the pyramidal tract. Another patient (S6), with progressive tentorial herniation, showed bilateral Babinski signs until respiratory arrest occurred (the blood pressure remained normal for a period). This suggests a 'permissive action' for the Babinski sign from the lower brain stem.

BABINSKI SIGN: INTERACTION BETWEEN FLEXION REFLEX AND DISTAL PYRAMIDAL CONTROL

Release at or near the motoneurone

We have seen that activation of flexion reflex pathways is necessary for the appearance of the Babinski response, but that the Babinski response can be released separately from the flexion reflex as a whole. We have also seen that occurrence of the Babinski sign is almost invariably associated with motor impairment of the foot, and that in some cases this motor impairment is manifested only by the slowing down, dysrhythmia or reduced fractionation of movements. The capacity for independent and fast movements is probably mediated by direct cortical projections to individual motoneurones (Kuypers, 1973). Because loss of this ability alone is apparently sufficient to release the Babinski sign, the pyramidal 'shielding' of extensor hallucis longus motoneurones from activity in the flexion reflex arc must have its impact at or near these motoneurones. In other words, an excitatory action (direct and selective innervation of distal motoneurones) is closely connected with an inhibitory action (counteracting the recruitment of the extensor hallucis longus via the flexion reflex pathways). The inhibitory neurone might be activated by collaterals of corticomotoneuronal fibres or consist of separate, but intimately related, pyramidal fibres. The proposed interaction between descending tracts and flexion reflex pathways is represented schematically in figure 14.

Nevertheless, there are exceptions. Some patients show a Babinski sign together with other pyramidal features but without demonstrable weakness or loss of skill (cases 1–4, table XVIII), others lack it whilst having some motor deficit in the foot (case 5, table XX). There are two ways of explaining these inconsistencies. The first is that direct excitation of distal motoneurones and inhibition of impulses via flexion reflex afferent nerve fibres can be dissociated because they are mediated by different neurones, however closely they are linked. The alternative is to invoke individual factors in exceptional patients: motor abnormalities that are too subtle for testing on the one hand, and an idiosyncratically poor influx of segmental impulses to motoneurones of the extensor hallucis longus on the other.

Bilateral Babinski signs without other pyramidal features

In normal subjects, even in those with rather brisk flexion reflexes, a normal pyramidal system keeps the extensor hallucis longus muscle out of the flexion reflex synergy, at least after infancy. This is of course the basis for the great practical value of the plantar reflex. However, one meets exceptional cases where extremely brisk flexion reflexes are found on both sides, in the absence of other abnormalities: mere touch activates all flexor muscles of the leg, including the extensor hallucis longus, and this can be reproduced indefinitely.

Two such patients were described in Chapter IV. One had sustained a (probably) epileptic seizure two years before and the other showed a raised CSF protein, but in neither were there abnormalities on neurological examination. Rather than to invoke an occult disturbance of distal pyramidal projections, it may be more reasonable to assume that in these patients the flexion reflex – either released from supraspinal control or being only at one extreme of normal variation – breaks through the normal descending control of extensor hallucis longus motoneurones. That such a view is not too heretical is illustrated by the fact that upgoing toes can be produced in many normal subjects by stimulating the base or pads of the toes (Verger and Abadie, 1900; Dosužkov, 1932). It is unnecessary to honour Babinski by postulating absolute impermeability of extensor

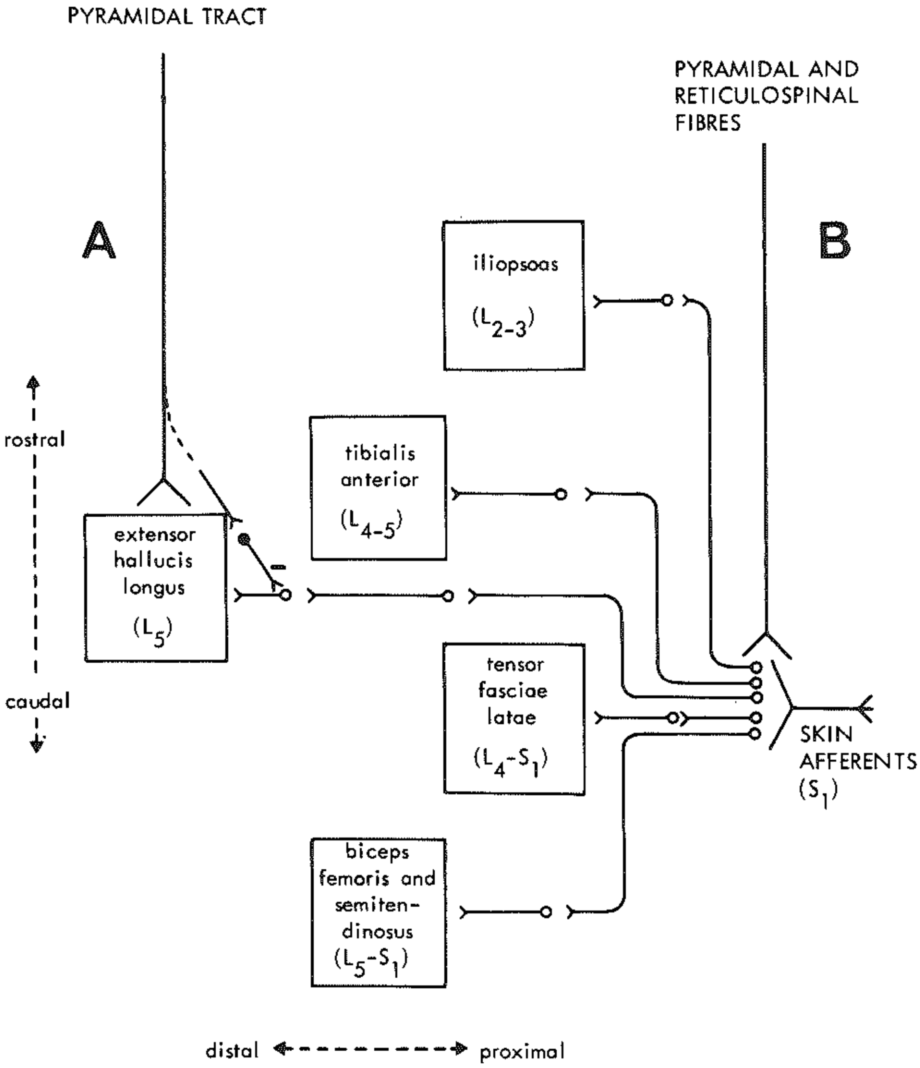

FIGURE 14

PROPOSED INTERACTION OF PYRAMIDAL TRACT AND FLEXION REFLEX PATHWAYS IN THE SPINAL CORD

A – direct pyramidal control of distal motoneurones, with local inhibition of flexion reflex pathways; dysfunction causes loss of skill and releases the Babinski sign.

B – descending control of interneurones which mediate the flexion reflex; dysfunction releases the flexion reflex as a whole, but this is not essential for the appearance of a Babinski sign.

hallucis longus motoneurones to segmental afferent activity in every normal subject.

THE 'UPPER MOTOR NEURONE'

Although in the present series of 50 patients who had a unilateral Babinski sign there were always other abnormalities as well, the classical pyramidal syndrome was usually incomplete. This common paradox had already prompted early clinicians to suppose that the descending fibres concerned with voluntary innervation were not identical with those releasing positive phenomena (Strümpell, 1899), and in time it has led to many anatomical hypotheses (mostly from animal experiments).

Lassek (1957) assumed that the pyramidal tract took its origin from many regions other than the central area of the cerebral cortex, but the degeneration studies on which he based this conclusion were invalidated by van Crevel and Verhaart (1963).

Others have invoked descending fibre systems outside the pyramidal tract. Tower (1940) failed to find spasticity and increased tendon jerks after medullary pyramidotomy in monkeys, while Kennard and Fulton (1933) had found these very signs after ablation of area 6 in chimpanzees. Bucy (1957) even went so far as to regard the motor system as a 'co-ordinated maze'. More recently Nyberg-Hansen and Rinvik (1963) and Brodal (1969) also assigned a role to lesions of structures other than the pyramidal tract in the production of major clinical phenomena. These suppositions are largely refuted, as far as man is concerned, by two unprecedented case histories of patients with almost pure infarction of one medullary pyramid (Chokroverty et al., 1975; Leestma and Noronha, 1976). The following signs were noted on the contralateral side: weakness, Babinski sign, and increased tendon jerks. Spasticity was present in one of the two patients. Some (proximal?) motility was preserved; abdominal reflexes were not mentioned, nor was asymmetry of the flexion reflex in proximal muscles.

In contrast to speculations about the origin of fibres concerned with various features of the pyramidal syndrome, Verhaart (1962) pointed out that many problems could be solved by realizing that the pyramidal tract consists only partly of 'upper motor neurones', i.e. direct (monosynaptic) corticomotoneuronal connections. The present study confirms this: only impairment of rapid foot movements and, closely linked with it, occurrence of the Babinski sign can be related to direct corticomotoneuronal connections. The capricious behaviour of the pyramidal syndrome as a whole must be due largely to divergence of corticospinal projections towards different segments of the lumbosacral cord as well as within antero-posterior divisions of the spinal gray matter.

CONCLUSIONS

From previous studies:

1. It is a common, but sometimes forgotten truth that a Babinski sign does not necessarily imply an anatomical lesion. The sign has been reported – also by Babinski himself – in many temporary disturbances of the central nervous system, with or without unconsciousness.

2. Statements that the Babinski sign can appear following lesions which do not involve the pyramidal tract are based on the incorrect notion that absence of secondary degeneration implies normal function.

3. The following abnormalities can occur after unilateral infarction of the medullary pyramid in man: weakness, Babinski sign, increased tendon jerks, and spasticity.

From this study:

4. The Babinski sign is strongly associated with motor impairment of the foot, either in the form of weakness or only as a reduced capacity to perform rapid and independent movements. It is not necessarily associated with release of the flexion reflex as a whole.

5. Increase of tendon jerks and exaggeration of the flexion reflex occur independently of one another.

6. Associated dorsiflexion of the great toe on ipsilateral or crossed hip flexion against resistance is inconstant and unreliable as a pathological sign.

7. Regardless of the degree of pyramidal weakness in the foot, the intraspinal pathways of the flexion reflex have to be operative in order to produce a Babinski sign.

8. Depression of spinal reflex pathways after spinal transection ('spinal shock') in man is at least partly due to interruption of facilitatory projections from the lower brain stem onto spinal interneurones.

(cont. next page)

9. The paradoxical downward toe response in acute transverse lesions of the spinal cord, caused by relative sparing of sacral segments from reflex depression, is not always slow or prolonged, nor is it always indicative of a complete transection.

10. Pyramidal projections onto individual motoneurones of muscles that move the foot and toes are identical to or at least closely connected with the fibres that 'suppress' the Babinski sign.

11. The unpredictable behaviour of the pyramidal syndrome as a whole is not the result of different origins of the fibres in question, but of their divergent terminations within the spinal gray matter.

12. Only patients who show loss of skilled foot movements are 'entitled' to a Babinski sign.

SUMMARY

This thesis describes a historical, clinical and electromyographic study of the plantar reflex, with emphasis on:

- physiological relationships between the toe responses and the other spinal reflexes that are evoked by plantar stimulation
- practical application of these associations to cases where the toe response is equivocal
- pathophysiology of the pathological plantar response.

Chapter I gives a review of previous clinical studies. The plantar reflex as such was not discovered by Babinski: many neurologists before his time had noted flexion in ankle, knee and hip after stimulation of the sole. What Babinski did discover was the diagnostic importance of the toe responses, which had only been casually reported by others (in the Netherlands the normal plantar reflex is erroneously connected with the name of von Strümpell). Babinski assumed that the upgoing toe sign as well as the normal downward response of the great toe were part of the flexion synergy of the leg, but later studies left no doubt that this relationship included only the pathological response: anatomical toe extensors are flexor muscles in a physiological sense, and vice versa. However, the synergistic background of the upgoing toe sign has become obscured by its diagnostic weight. This neglect of physiology has in turn led to undue emphasis on different methods of stimulation, and to exotic speculations about the 'purpose' of the plantar reflex.

The notion that only an upgoing great toe plus fanning of the little toes constitutes a full-fledged Babinski sign is proved to be a distortion of history. Another popular dictum is that normal infants do not show a Babinski sign – this is based either on misinterpretation of the plantar grasp reflex or on the unwarranted assumption that the Babinski response in adults is an isolated phenomenon.

The normal plantar response is a unisegmental reflex, not related to any synergy.

Chapter II is devoted to a study of the variation and bias that occur in the interpretation of equivocal plantar reflexes. Fifty neurologists from three Dutch university hospitals were asked to judge a number of plantar responses on film. The main part of the presentation only served to disguise the fact that two films, both showing equivocal toe movements, were presented twice at the same sitting. Variability was studied in thirty neurologists who saw the films without additional information: they

showed very little agreement with themselves with regard to the interpretation of the identical films (only slightly better than was to be expected by chance alone). Added to this there was a considerable disagreement between observers.

The effect of bias was investigated in the remaining twenty neurologists, who rated each film after seeing a slide with a fictitious abstract of history and examination (minus the plantar reflex); the films that occurred twice were on each occasion preceded by different information, with opposite bias as to the probability of a Babinski sign. The mean interpretation of these identical pictures differed significantly, conforming to the information given; the former group of neurologists, without prior information, showed no such systematical change of opinion.

It follows that current interpretation of plantar reflexes is, at least in some cases, a matter of hazard and prejudice, even when differences in technique are eliminated. Better criteria are obviously needed.

Chapter III describes an electromyographic study of the normal and pathological plantar reflex, as a reference for equivocal cases. In 22 patients with a definite Babinski sign and in 49 control subjects, the electrical activity was recorded from flexor and extensor muscles of the great toe and from the anterior tibial muscle. Mechanical and electrical stimulation of the sole was used in each. The Babinski sign was found to be mediated by the extensor hallucis longus, and was always accompanied by reflex activity in the tibialis anterior. The normal plantar response is the result of contraction of the flexor hallucis brevis muscle.

Electrical stimulation of the sole was found to activate the extensor hallucis longus in many control subjects – this had previously led to the mistaken idea that the extensor hallucis brevis was the effector of the Babinski sign. Conversely, electrical stimuli failed to recruit the extensor hallucis longus in some patients with a Babinski sign. In general, reflex effects produced by the application of short pulse trains to the skin should be interpreted with caution – not so much because electrical stimuli are 'unnatural', but because they are punctate, as opposed to moving stimuli which generate spatial and temporal summation of afferent impulses. The fact that a stimulus is painful is not in itself sufficient to evoke a possible Babinski sign; neither is a stimulus which causes a Babinski sign always painful in an average subject.

In *Chapter IV* the electromyographic criteria for normal and pathological plantar reflexes, developed in the preceding chapter, were applied to thirty patients in whom others found equivocal plantar responses. Bias in recording or interpretation was excluded. The study was repeated in each patient after one week, and the electromyographic outcome – considered in terms of 'pathological' or 'not pathological' – was reproducible in 26 patients (87%). Of these 26, a pathological reflex was suspected by the clinician in 17, but shown electromyographically in only six patients. Checked with the 'final' diagnosis reached by others, electromyography did not lead to false-pathological results; inversely, the electromyographic criteria were wide enough to demonstrate expected, but clinically unconvincing, Babinski signs.

Comparison of the electromyographic results with an independent clinical examination of the same patients allowed definition of pitfalls in the bedside interpretation of plantar reflexes, as well as delineation of step-wise clinical criteria, viz.:

1. action of the extensor hallucis longus muscle
2. simultaneous contraction of other (physiological) flexor muscles
3. exclusion of voluntary movements.

Electromyography may be the only feasible method to investigate the plantar reflex in cases where hallux movements are limited by skeletal or tendinous deformities.

Chapter V deals with the pathophysiology of the Babinski sign and its relationship to other components of the pyramidal syndrome. The pyramidal syndrome has come to be dismissed by some physiologists as mere neurological folklore, have little to do with the pyramidal tract. However, the following abnormalities have recently been described after almost 'pure' unilateral infarction of the medullary pyramid in man: weakness, Babinski sign, increased tendon jerks, and spasticity. A role for other descending fibre systems is not excluded by these reports, but positive proof is lacking or inconclusive.

Whether the Babinski sign does or does not accompany other pyramidal signs might then depend on the termination of the fibres involved. To test this hypothesis, 50 patients with a unilateral Babinski response were examined for the presence of other pathological signs in the lower limbs. Almost all of them (46) showed motor impairment of the foot, either in the form of weakness or merely as a reduced capacity to perform rapid and fractionated movements. In some cases where the Babinski sign disappeared, skilled movements of the foot became normal at the same time, whilst other pyramidal signs remained. Of four other patients who 'lacked' a Babinski sign, three showed no impairment of foot movements.

The Babinski sign was as often as not accompanied by exaggeration of the flexion reflex as a whole; this release is multisegmental and is probably caused by involvement of projections to spinal interneurones.

It appears that occurrence of the Babinski sign is closely linked with a disturbance of direct corticomotoneuronal connections which subserve differentiated movements of the foot and toes, and that the release of the Babinski sign takes place at or near the motoneurone level.

A second condition to be fulfilled before a Babinski sign can appear, is that the flexion reflex must be operative. Depression of the flexion reflex may therefore preclude an upgoing toe sign. Spinal transection is a well-known cause of reflex depression. In this situation the plantar reflex can even be 'normal'. Acute lesions of the cerebral hemispheres can also give rise to depression of the flexion reflex.

The capricious behaviour of the pyramidal syndrome in disease must be attributed to selective involvement of pyramidal fibres, which have diverging terminations within the spinal gray matter. Only patients who show loss of skilled foot movements are 'entitled' to a Babinski sign.

SAMENVATTING

Dit proefschrift behelst een historisch, klinisch en elektromyografisch onderzoek van de voetzoolreflex. De nadruk is hierbij gelegd op:

- de fysiologische verwantschap tussen de teenreflexen en andere spinale reflexen die door prikkeling van de voetzool worden opgewekt
- praktische toepassing van deze samenhang in gevallen waar de voetzoolreflex twijfelachtig is
- de ontstaanswijze van de pathologische voetzoolreflex.

Hoofdstuk I geeft een overzicht van voorgaande onderzoeken. De voetzoolreflex als zodanig werd niet door Babinski ontdekt: voordien hadden al vele neurologen opgemerkt dat prikkeling van de voetzool leidde tot buiging in enkel, knie en heup. De ontdekking van Babinski bestond uit het diagnostische belang van de teenreflexen hierbij; deze waren eerder alleen terloops door anderen vermeld (in Nederland wordt de normale voetzoolreflex ten onrechte naar Von Strümpell genoemd). Babinski veronderstelde dat zowel de pathologische als de normale teenreflex deel uitmaakten van de flexiesynergie van het been, maar later onderzoek liet geen twijfel dat deze samenhang alleen de pathologische teenreflex betreft: de anatomische teenextensoren zijn in fysiologisch opzicht flexoren, en omgekeerd. De ruimere betekenis van de pathologische teenreflex is echter in de loop der tijd overschaduwd door het diagnostische belang. Dit voorbijzien aan de fysiologie heeft op zijn beurt geleid tot overmatige nadruk op allerlei prikkelingsmethoden, en ook tot vreemdsoortige gissingen over het 'doel' van de voetzoolreflex.

De mening dat 'extensie' van de grote teen alleen een waarlijk teken van Babinski is wanneer dit gepaard gaat met spreiden van de kleine tenen berust op onjuiste uitleg van de oorspronkelijke beschrijvingen. Een andere veelgehoorde uitspraak is dat normale zuigelingen geen Babinski-reflex vertonen – deze komt voort òf uit het niet herkennen van de grijpreflex aan de voet, òf uit de verkeerde veronderstelling dat de Babinski-reflex bij volwassenen een op zichzelf staand verschijnsel is.

De normale teenreflex is een unisegmentaal mechanisme, dat los staat van synergieën in het been.

Hoofdstuk II is gewijd aan variatie en vooroordeel bij de beoordeling van dubieuze voetzoolreflexen. Aan vijftig neurologen (specialisten en assitenten), afkomstig uit drie Nederlandse universiteitsklinieken, werd gevraagd een aantal gefilmde voetzoolreflexen te beoordelen. Het merendeel van de films diende alleen om te verhullen dat twee andere films, beide met dubieuze voetzoolreflexen, later in de serie nog eens terugkwamen. Wat betreft de variatie: dertig neurologen die de films zonder voorafgaande informatie zagen, toonden een zeer geringe overeenstemming met zichzelf wat betreft de beoordeling van de identieke films (nauwelijks groter dan op grond van het toeval kon worden verwacht). Daarenboven liepen de waarnemers onderling sterk in oordeel uiteen.

Beïnvloeding van de beoordeling door andere gegevens werd onderzocht bij de overige twintig neurologen, die elke film pas beoordeelden na het zien van een dia met een gefingeerde samenvatting van anamnese en onderzoek (met weglating van de voetzoolreflex); de films die tweemaal voorkwamen werden bij elke aanbieding voorafgegaan door andere informatie, onderling tegengesteld wat betreft de waarschijnlijkheid van een Babinski-reflex. De gemiddelde beoordeling van de identieke beelden toonde significante verschillen, al naar de aard van de gegevens; de eerste groep neurologen, zonder voorinformatie, toonde geen verschuiving van de gemiddelde beoordeling.

Hieruit volgt dat de gangbare beoordeling van voetzoolreflexen in sommige gevallen voornamelijk bepaald wordt door toeval en vooroordeel, zelfs wanneer verschillen in techniek geen rol kunnen spelen. Betere maatstaven zijn daarom zeer gewenst.

Hoofdstuk III beschrijft een elektromyografisch onderzoek van de normale en pathologische voetzoolreflex, om als uitgangspunt te dienen voor twijfelachtige gevallen. Bij 22 patienten met een Babinski-reflex en bij 49 controle-personen werd de elektrische activiteit afgeleid uit flexoren en extensoren van de grote teen, en uit de tibialis anterior. De voetzool werd zowel mechanisch als elektrisch geprikkeld. Het bleek dat de Babinski-reflex tot stand komt door contractie van de extensor hallucis longus, en dat dit altijd samengaat met activiteit in de tibialis anterior. De normale voetzoolreflex berust op contractie van de flexor hallucis brevis.

Elektrische prikkeling van de voetzool bleek in vele normale proefpersonen activiteit in de extensor hallucis longus op te wekken – dit had eerder tot de onjuiste conclusie geleid dat de musculus extensor hallucis brevis als de effector van de Babinski-reflex beschouwd moet worden. Omgekeerd was het in sommige patienten met een Babinski-reflex niet mogelijk om de extensor hallucis longus door middel van elektrische prikkels te activeren. In het algemeen moeten reflex-effecten die het gevolg zijn van kortdurende stroomstootjes met reserve worden bezien – niet zozeer omdat elektrische prikkels 'onnatuurlijk' zijn, maar omdat deze puntvormig zijn, in tegenstelling tot strijken, wat gepaard gaat met spatiële en temporele summatie van afferente impulsen. Het is niet zo dat elke pijnlijke prikkel voldoende is om een eventuele Babinski-reflex op te wekken, en evenmin is een prikkel die een Babinski-reflex veroorzaakt altijd pijnlijk voor een gemiddeld individu.

In *Hoofdstuk IV* werden de elektromyografische criteria voor normale en pathologische voetzoolreflexen die in het voorgaande hoofdstuk zijn ontwikkeld, toegepast bij dertig patienten bij wie andere artsen de voetzoolreflex als twijfelachtig kenmerkten. Vooroordeel bij de registratie of de beoordeling werd uitgesloten. Het onderzoek werd bij elke patient een week later herhaald, en het elektromyografisch resultaat – gemeten naar de indeling 'pathologisch' of 'niet pathologisch' – bleek reproduceerbaar bij 26 patienten (87%). Van deze 26 patienten vermoedde de clinicus bij 17 een pathologische voetzoolreflex; elektromyografisch werd dit vermoeden slechts bij zes patienten bevestigd. Beoordeeld naar de 'uiteindelijke' diagnose die door anderen werd gesteld, gaf elektromyografisch onderzoek geen vals-positieve resultaten; omgekeerd waren de elektromyografische criteria ruim genoeg voor het aantonen van verwachte, maar klinisch onduidelijke Babinski-reflexen.

Vergelijking van de elektromyografische resultaten met een onafhankelijk neurologisch onderzoek van dezelfde patienten maakte het mogelijk om foutenbronnen aan te geven die een rol spelen bij de beoordeling van voetzoolreflexen aan het ziekbed, en ook om klinische maatstaven op te stellen, stapsgewijs gerangschikt:

1. activiteit van de musculus extensor hallucis longus
2. gelijktijdige contractie van andere (fysiologische) flexoren
3. uitsluiten van willekeurige bewegingen.

Elektromyografisch onderzoek kan de enige methode zijn om de voetzoolreflex te onderzoeken wanneer de bewegingen van de grote teen worden beperkt door afwijkingen van gewricht of pezen.

Hoofdstuk V behandelt de pathofysiologie van de Babinski-reflex, en de samenhang met de andere componenten van het piramidale syndroom. Het piramidale syndroom wordt door sommige fysiologen wel afgedaan als niet meer dan klinische overlevering, zonder werkelijk verband met de piramidebaan. Kortgeleden zijn echter de volgende afwijkingen beschreven na bijna 'zuivere' unilaterale infarcering van de piramis bij de mens: zwakte, Babinski-reflex, verhoogde peesreflexen, en spasticiteit. Een mogelijke invloed van andere afdalende vezelsystemen kan op grond hiervan niet geheel worden verworpen, maar een sluitende bewijsvoering daarvoor ontbreekt.

Of de Babinski-reflex al dan niet optreedt naast andere piramidale verschijnselen zou dan kunnen afhangen van de eindiging van de getroffen vezels. Om deze hypothese te toetsen werden 50 patienten met een eenzijdige Babinski-reflex onderzocht op de aanwezigheid van andere neurologische afwijkingen aan de benen. Bijna allen (46) toonden motorische uitval van de voet, hetzij als zwakte, hetzij alleen als een stoornis van snelle en onafhankelijke bewegingen. In sommige gevallen waarin de Babinski-reflex verdween, werd de 'voetvaardigheid' tezelfdertijd normaal, terwijl andere piramidale afwijkingen bleven bestaan. Van vier andere patienten die een Babinski-reflex 'misten', bleken er drie geen motorische uitval aan de voet te hebben.

Een pathologische voetzoolreflex bleek slechts in iets meer dan de helft van de gevallen gepaard te gaan met ontremming van de flexiereflex als geheel; deze ontremming omvat meerdere segmenten en wordt vermoedelijk veroorzaakt door een stoornis van afdalende vezels die eindigen in de interneuronale zone van het ruggemerg. Dit alles leidde tot de conclusie dat het optreden van de Babinski-reflex nauw verbonden is met een stoornis van de directe corticomotoneuronale verbindingen die in dienst staan van de fijne motoriek van voet en tenen, en dat de Babinski-reflex dicht bij de betrokken voorhoorncellen tot stand komt.

Een tweede voorwaarde waaraan moet zijn voldaan voordat een Babinski-reflex kan optreden, is dat de flexie-reflex werkzaam is. Remming van de flexie-reflex kan daarom een Babinski-reflex onmogelijk maken. Acute ruggemergsletsels zijn een bekende oorzaak van reflex-remming. In dergelijke gevallen kan de voetzoolreflex zelfs 'normaal' zijn. Acute laesies van de grote hersenen kunnen ook remming van de flexie-reflex veroorzaken.

Het onvoorspelbare optreden van het piramidale syndroom bij ziekten van het centrale zenuwstelsel moet toegeschreven worden aan selectieve stoornissen van corticospinale vezels, die verschillend eindigen binnen de grijze stof van het ruggemerg. Andere afwijkingen dan een stoornis van snelle voetbewegingen geven geen 'recht' op een Babinski-reflex.

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