Babinski response: stimulus and effector

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SYNOPSIS This is an electromyographic study of the activity in flexor and extensor muscles of the big toe in 22 patients with a Babinski sign and 49 controls, after mechanical or electrical stimulation of the sole. The results indicate: (1) the Babinski sign is mediated by the extensor hallucis longus (EHL), and not by the extensor hallucis brevis; (2) electrical stimuli may fail to activate the EHL in these patients, and conversely may evoke EHL reflexes in control subjects; (3) in skin reflexes, electrical and mechanical stimuli are not freely interchangeable.

Before Babinski (1896) introduced his ‘phénomène des orteils’, the plantar reflex was known as contraction of several leg flexors on stimulation of the sole (Bloq and Onanoff, 1892; Gowers, 1892). This was considered a normal phenomenon. Babinski observed the contrast between the downward movement of the toes which accompanied this withdrawal mechanism in normal subjects and dorsiflexion of the toes in patients with hemiplegia. Later, various diseases of brain and spinal cord affecting the pyramidal tract appeared to be accompanied by reflex dorsiflexion of the toes, particularly the big toe (Babinski, 1898).

Dorsiflexion of the big toe after plantar stimulation was later shown to be an integral part of the withdrawal reflex of the leg (van Gehuchten, 1900; Marie and Foix, 1912; Walshe, 1914), as in the flexion reflex of the spinal dog (Sherrington, 1910). Thus, the toe muscles are paradoxically termed: the anatomical extensors act as physiological flexors, and vice versa.

Stimulation of skin can also evoke physiological extensor reflexes in the leg, particularly on areas overlying (physiological) extensor muscles. The normal ‘flexor response’ of the toes (plantar flexion or physiological extension) is assumed to be a similar reflex, mediated by the FHB1 (Kugelberg et al., 1960).

The alleged relation of Babinski’s sign with pyramidal tract lesions was never conclusively proved (Nathan and Smith, 1955) nor disproven (Walshe, 1956). Understandably, clinicians do not bother very much about this issue.

Of far greater practical importance is the problem that the plantar reflex can be equivocal where clinical doubt exists (Matthews, 1970). Interpretation can vary with observer or occasion (McCance et al., 1968), and with technique of stimulation: Dohrmann and Nowack (1973) found slow stroking of the lateral plantar border and the plantar arch to be the best way to elicit (anatomical) extension of the big toe in patients. But even with the appropriate techniques doubts may remain. Knowledge of which muscle is involved in the (anatomical) extensor response could, in these cases, lead to more precise inspection of toe movements or to recording of electromyograms. But there is no unanimity in this matter.

Walshe (1914), Wertheim Salomonson (1920), and Kugelberg (1948) assumed the EHL to be the effector of the (anatomical) extensor response, but the EHB was not investigated or discussed. Landau and Clare (1959) recorded from various foot and leg muscles in normal subjects and in patients with a Babinski sign. Stimulation consisted for the most part in stroking the lateral border of the sole (this will now be called mechanical stimulation). They not only found that the (anatomical) flexor response was due to contraction of FHB, but also that the EHB was


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active simultaneously, in controls and patients alike. After stronger mechanical stimuli, the TA muscle was also activated, together with EDL. The EHL was silent in normal subjects, but in patients showing a Babinski sign the EHL was recruited into the (physiological) flexion reflex pattern, together with TA and EDL.

In contrast, Kugelberg et al. (1960), applying electrical stimuli (pulse trains of 50 ms duration) to the plantar surface, reported reflex activity of EHL in both normal and pathological cases. The EHB muscle, however, was active in both normal and pathological patients. Grimby (1963a), employing subjects only from electromyographic (EMG) recordings of toe muscles which muscle is responsible for the (anatomical) extensor response after mechanical stimulation: EHL or EHB, 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 almost 80 years.

METHODS

PATIENTS The investigations were made in 22 patients with a Babinski sign and 49 control subjects. The controls were outpatients and medical employees. They were accepted on the basis of normal plantar reflexes (anatomical) flexor responses symmetrical or absent on both sides), and freedom from any central nervous abnormalities. In a group of nine controls which formed, together with seven patients, the first part of the study, investigation of the normal legs of two patients with a unilateral Babinski sign was included. In the Babinski group only patients with a definite (anatomical) extensor response were accepted: the upgoing toe could be seen to take part in the flexor reflex of the leg. In all members of this 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 (see Dohrmann and Nowack, 1973). It is generally accepted that stimulation of the medial plantar border is less effective in eliciting an (anatomical) extensor response, probably because the FHB is directly activated in this way. Electrical stimuli were effected through a pair of needle electrodes, placed intracutaneously at the middle of the lateral plantar border. 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: EHB (only in the first part of the experiments), EHL, TA, and FHB. The FHL was studied in a few patients, but this muscle did not show reflex activity. As this was in accordance with other work (Landau and Clare, 1959; Kugelberg et al., 1960), recording from FHL was abandoned. Concentric needle electrodes were used in all muscles. The EHB, a small muscle, was first located by surface stimulation. The FHB electrode was introduced at the plantar side of the first metatarsal bone. The TA electrode was placed proximally. Needle position in these three muscles
was then controlled by asking the subject to make the appropriate movement, and an interference pattern of potentials should then be 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 capit fibulae (this point proved to be most convenient to reach the bulk of the EHL muscle in postmortem material). Placement of the tip of the EHL electrode was checked by passive movement of the hallux, by the electrical response on voluntary dorsiflexion, and by electrical stimulation through the recording electrode. These control measures were repeated twice during the experiment.

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

RESULTS

EFFECTOR OF BABBINSKI SIGN  Reflex activity of EHL and EHB was compared after mechanical stimulation in seven patients and nine control subjects (Table I). 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, so that all potentials together form a spindle shape on the oscilloscope screen. EHL reflexes were present in all patients and in none of the control subjects. In contrast, EHB activity appeared in one-third of controls (not including the normal legs of the two patients) and was absent in more than half the patients (Fig. 1). It can be concluded that the (anatomical) extensor response is mediated by the EHL, and not by the EHB.

MECHANICAL STIMULATION: EFFECTS IN EHL AND FHB  This was investigated in 15 patients and 40 controls. In general, reflex activity appeared within 0.5 to 1 s after stimulation and decreased within a few hundreds of ms when stimulation was ended. When potentials continued to appear, this could be interpreted as voluntary activity. Repetition of the procedure when the subject was sufficiently relaxed then no longer showed the continuing activity. This rule evidently does not apply if flexor spasms are present, but these can be recognized clinically.

There appeared to be 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 15 patients, a 46 year old male with severe cerebral concussion. In one control subject EHL activity was recorded more than once, together with potentials from FHB; this case will be discussed below.
It was not surprising that the EHL was recruited in all patients (Table 2). EHL reflexes were also synchronous with activity in TA in all patients (Table 3). In three control subjects, reflex activity of TA was found to occur unaccompanied by contraction of EHL. The same phenomenon was observed in the normal leg of a patient with infarction in a cerebral hemisphere (this experiment was not included in the control series). In one control subject recruitment of EHL motor units was repeatedly recorded. This 49 year old man, complaining of non-specific headaches, showed bilateral pes cavus, and was apt to show voluntary (anatomical) extension of the big toe. But the potentials in the EHL continued seconds after stroking, FHB potentials were also recruited and even interrupted by EHL activity, and there was no coactivation of TA. These features suggest that insufficient relaxation or functional variation, or both, were the cause of the EHL activity, rather than a pathological reflex mechanism.

**TABLE 3**

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**FIG. 2** Reflex activity in FHB and EHL after electrical and mechanical stimulation (arrows) of the plantar surface in a normal subject (A) and a patient with a Babinski sign (B).

**ELECTRICAL STIMULATION: EFFECTS IN EHL AND FHB** These studies were performed in the same 15 patients and 40 controls, and the reflex effects are shown in Table 2 together with those after mechanical stimulation. A reflex was considered to be present when potentials repeatedly appeared 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 15 patients with a Babinski sign, electrical stimuli failed to activate the EHL. In one patient, a 56 year old man with spastic paresis of the right leg after haemorrhage from an arteriovenous malformation in the left parietal parasagittal region, this was confirmed in three later experiments. 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 even be elicited by stroking the plantar surface with a finger pad. But electrical shocks were insufficient even at maximal intensity (Fig. 2, 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 in every three subjects. 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. Stimulus strength was expressed in multiples of
tactile threshold (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 from a sensory judgement, but this seemed a better method, especially for the control series, than estimating the distribution of current through the skin. In controls, tactile threshold varied from 0.55 to 1.30 (arbitrary stimulator units), with a mean of 0.77. Threshold for EHL reflexes in the 13 controls ranged from 4 times ($\times$) tactile threshold (in a healthy neurologist!, see Fig. 2, A) to 15 $\times$; the mean was 8.6 $\times$. In the group of patients, tactile thresholds were found from 0.55 to 1.25 (mean 0.81). The 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. The minimal stimulus intensities required to obtain EHL reflexes in patients, if found at all, varied from 1.5 $\times$ to 10 $\times$, with a mean value of 6.3 $\times$. Thus, thresholds for EHL reflexes in both groups overlap considerably, and cannot be used to discriminate the ‘false-positive’ control subjects from patients with a Babinski sign.

A change in the arbitrary criteria used to decide whether or not there is reflex activity after electrical stimulation (and criteria are necessary since a single spike from a single motor unit can obviously not be counted as positive) cannot improve the distinction between reflex effects in patients and controls. If, for instance, one required shorter latency or longer duration of reflex discharge in 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 stimulus will provide a clearer demarcation between EHL thresholds of patients and controls.

In all control subjects with EHL reflexes and in four of the 10 patients in whom EHL reflexes could be evoked electrically there was synchronous excitation of FHB motoneurones (Fig. 2, A). This simultaneous activation of antagonists contrasts with the strong tendency to reciprocal activity after mechanical stimulation of the skin, and may be an argument against the physiological validity of short electrical skin stimuli.

**DISCUSSION**

**EFFECTOR OF BABINSKI SIGN** From the first part of this study it emerges that the (anatomical) extensor plantar reflex is mediated by the EHL and not by the EHB, as had been observed previously by Landau and Clare (1959). If it had not already become a matter of controversy in the literature, it would have been evident from simpler observations than electromyographic recording. Firstly, when a Babinski sign appears, the tendon of the EHL can be seen or at least felt to contract on the dorsum of the foot and of the big toe. Secondly, it is hard to relate the vigorous (anatomical) extension of the hallux that can be seen even in old people to the tiny EHB muscle. Moreover, the EHB is fully comparable with the EDB, which is subject to progressive atrophy with increasing age (Jennekens et al., 1972). In one human subject (adult of un-
known age) the EHL weighed 30.7 g, the EHB 0.9 g (Fig. 3).

Kugelberg et al. (1960) and Grimby (1963a) considered the EHB to be the specific muscle for the pathological plantar response. Grimby (1963a) assumed that the difference between their results and those of Landau and Clare (1959) could be attributed to technical factors but the main difference in methods was not discussed. Landau and Clare (1959) applied mechanical stimuli, a method that has proved to be reliable in decades of clinical practice, whereas the electrical pulse trains were only supposed to be so.

**MECHANICAL AND ELECTRICAL STIMULI**
The equivalence of mechanical and electrical stimuli seems unlikely in view of the second part of this study: activation of the EHL by electrical stimulation of the skin occurred in about a third of control subjects, at stimulus intensities not higher than those needed to evoke the effect in patients. Synchronous potentials in EHL and FHB appeared in all control subjects with electrically induced EHL reflexes and in some of the patients. It may be that the organization of reciprocal patterns requires a stimulus of longer duration than the short train of pulses. Recruitment of EHL and FHB together on mechanical stimulation was found in only one of 15 patients, and also once in 40 controls (the pes cavus case). In addition to the 'false-positive' controls, electrical stimuli failed to activate the EHL in five out of 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 was necessary for activation of the EHL, as neither prolonged electrical stimulation at one spot, nor simultaneous application of short pulse trains at several sites on the lateral plantar border were effective. After subliminal mechanical stimulation a single pulse train could, however, evoke a reflex in the EHL.

It can be understood now that Grimby (1963b) found no clear difference in EHB patterns after electrical stimuli between patients with supra-nuclear motor disorders who showed a Babinski sign and those who did not: neither the type of stimulus nor the action of EHB help to discriminate between the normal and the diseased state. Bathien and Bourdarias (1972) applied electrical stimuli to the sural nerve, below the lateral malleolus, and found a decreased threshold of the EHL in six of 15 patients with a Babinski sign; the results in the remaining patients are not stated. Moreover, muscle activity was recorded with surface electrodes in this study, and as the EHL is a deep muscle, covered by TA and EDL in its entire course, it is doubtful if the recorded potentials were related exclusively to EHL.

Even the theoretical advantage of electrical stimulation (the possibility of distinguishing spinal reflexes from voluntary reactions by latency measurement) was not always valid: in legs which cannot be moved voluntarily, latencies of 200 ms or more after electrical stimulation could be observed. Conversely, a fairly 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 hold on for a few seconds afterwards. Flexor spasms do not conform to this rule, but they can be recognized clinically.

It would therefore appear that, in man, descending pathways can switch the afferent activity after mechanical stimulation of skin to the spinal segmental systems subserving either flexor or extensor muscles. In the cat, it is well known that different descending pathways can switch the effects of stimulation of flexor reflex afferent nerve fibres to different spinal interneuronal systems (Lundberg 1966). Interruption of these pathways generally leads to bias in favour of (physiological) flexor reflexes. In the toes, (physiological) extension occurring in normal subjects after stroking of the sole may then be reversed to (physiological) flexion, which indicates a defect in the switching mechanism. Synchronous volleys in skin afferent fibres evoked by short electrical stimulation may fail to show this defect by not activating the EHL at all or by activating it together with its antagonist, and the latter can also happen in normal subjects. Thus, the reciprocal patterns of activity that would emerge on mechanical stimulation are obscured.

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
next paper (1898) by ‘chatouillement’ (stroking). The ‘unphysiological’ nature of electrical skin stimuli may also affect other skin reflexes than the plantar reflex.

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