The Babinski sign

Introduction

Joseph Babinski (1857–1932), a French neurologist of Polish descent, was the first person to describe extension of the big toe following stimulation of the sole of the foot on 22 February 1896 (Babinski, 1896). He referred to the sign as 'phénomène des orteils' (toes phenomenon) but it is now usually referred to eponymously as the 'Babinski sign' or descriptively as the extensor plantar response.

Some authors refer to the Babinski sign as the most important in neurology (Purdy, 2010), while others argue it should not continue to be part of the neurological examination (Miller and Johnston, 2005). Regardless, its importance in neurology is reflected both in the proliferation in the literature of thirty related manoeuvres (Kakitani et al, 2010) and the ongoing use of the sign to facilitate urgent specialist review or scanning. This article outlines how to perform, judge and interpret the plantar reflex and briefly reviews studies of the reliability of the sign.

Performing the manoeuvre

In his monograph on the Babinski sign, van Gijn (1996) details the optimum method:

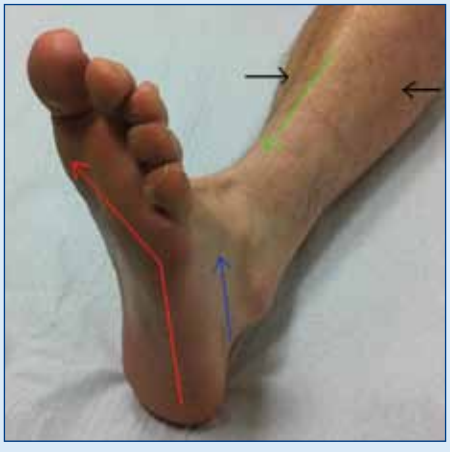
1. Lie the patient supine on couch or bed with the whole leg exposed
2. Warn the patient that you are going to scratch the sole of his/her foot, and ask him/her to remain as floppy as possible (preferable to the term relaxed which usually induces the opposite effect)
3. If available use the wooden end of an 'orange stick' (a thin stick with cotton wool on one end), although any instrument may be used if it is not too sharp and has not been used on other patients
4. Any part of the foot may be stimulated (or indeed lower leg, hence the development of numerous other eponymous signs – see below), but best results are achieved by stroking the lateral aspect of the sole of the foot (*Figure 1*).

While as many as thirty other techniques for eliciting the same response have been described in the literature, none have been shown to be superior (van Gijn, 1996). However, three variants have endured and may be encountered on neurology rounds:

- Oppenheimer sign – firmly stroking the medial tibial surface
- Gordon sign – firm compression of the lower calf muscle
- Chaddock sign – stroking the skin beneath the lateral malleolus.

These are different methods of stimulating the same reflex although, unless the sole is stimulated, flexion of the big toe in the healthy patient will not be seen as this is part of a separate localized cutaneous reflex. Some patients require a greater stimulus to elicit the abnormal response, while for those of a ticklish disposition, a lesser stimulus such as Chaddock sign may result in an easier to interpret response without excessive squirming. Regardless of what stimulus is used, the key aspect of the Babinski sign is in fact judging what is observed.

Figure 1. Various sites of eliciting the 'up-going toe'. Red arrow: Babinski sign, blue arrow: Chaddock sign, green arrow: Oppenheimer sign, black arrows: Gordon sign.



Judging the response

The normal and pathological responses to plantar stimulation are succinctly described by Babinski (1896) in his original communication:

> **'On the healthy side pricking of the sole provokes... flexion of the thigh on the pelvis, of the leg on the thigh, of the foot on the leg and of the toes upon the metatarsus. On the paralysed side a similar excitation also results in flexion of the thigh on the pelvis, of the leg on the thigh, of the foot on the leg, but the toes, instead of flexing, execute a movement of extension upon the metatarsus.'**

A slight diversion into the physiology of the Babinski sign is useful to aid understanding of these observations. When the sole of the foot is scratched sufficiently a 'flexion synergy' response is seen. This is the simultaneous contraction of muscles which shorten the lower limb, presumably to remove the limb from the noxious stimulus.

In a newborn infant, the full response can be seen with contraction of hip flexors including tensor fascia lata, hamstrings (knee flexion), tibialis anterior (ankle dorsiflexion) and extensor hallicus longus (big toe extension). However, by the age of 1 year, downward inhibition of the reflex from the upper motor neuron via the pyramidal tract has developed, particularly of the extensor hallicus longus component, such that big toe extension is replaced by a separate localized cutaneous toe flexion reflex (similar to the abdominal reflexes) which results in flexion of the big toe following stimulation of the sole.

Thus in healthy adults, with sufficient stimulus, while the other components of the flexion synergy response may still be seen, the upward movement of the big toe seen in infants is replaced by downward movement. In patients with upper motor neuron dysfunction there is reduction of the cutaneous toe flexion reflex (similar to reduced abdominal reflexes seen with upper motor neuron lesions) and release of inhibition of extensor hallicus longus activation such that the big toe moves up synchronous to the other components of

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the flexion synergy response, thus explaining the patterns originally described by Babinski (van Gijn, 1996).

Based on the physiological considerations above, three rules should be borne in mind when interpreting the movement of the big toe (*Figure 2*). These rules improve accuracy of the sign compared with clinical and electromyographic recordings (van Gijn, 1976) and when taught to medical students have been shown to improve their performance (Raijmakers et al, 1991).

The contraction of the extensor hallicus longus, seen through elevation of its tendon, is important to distinguish from upward movement of the big toe which is caused by dorsiflexion of the ankle or the illusion of upward motion if the big toe remains stationary while other toes flex, neither of which are pathological. Synchronous reflex activity in other flexion muscles ensures that the response seen in the big toe is part of the synergistic reflex and not voluntary. Observation of tensor fasciae lata is particularly useful in this regard as this muscle is activated with flexion synergy, but not during voluntary withdrawal. Finally, a true Babinski sign should be reproducible with repeated stimulation. There are many other pitfalls in interpreting the Babinski sign (*Table 1*) (van Gijn, 1996).

Figure 2. Three rules for interpretation of the Babinski sign.

1. Upward movement of the toe is pathological only if caused by contraction of the extensor hallicus longus muscle
2. Contraction of the extensor hallicus longus muscle is pathological only if it occurs synchronously with reflex activity in other flexor muscles
3. A true up-going toe sign is reproducible, unlike voluntary withdrawal

Interpreting the sign

Once recognized as being present, interpretation of the Babinski sign is straightforward: it implies upper motor neuron dysfunction in the brain or spinal cord. Clinically this information is assimilated with other upper motor neuron signs such as spasticity, hyper-reflexia and pyramidal distribution weakness. It is very uncommon for Babinski to be the only sign of pyramidal tract dysfunction.

Common conditions in which a Babinski sign is seen include cerebrovascular disease (stroke), multiple sclerosis and tumours. It is worth noting that dysfunction of the pyramidal tracts need not be structural: infection (e.g. meningitis), seizures and drug toxicity may all be associated with the Babinski sign. Likely causes of Babinski sign in a clinical examination setting such as PACES differ slightly to clinical practice (*Figure 3*).

Figure 3. Interpretation of the Babinski sign in the PACES exam.

Common causes in PACES	Vascular disease – sudden onset, stepwise progression	Stroke – together with signs confined to a single vascular territory
		Multi-infarct dementia – shuffling gait and bilateral Babinski sign
		Haemorrhage – including from vascular malformations which may be multiple
	Inflammation – sub-acute onset, relapsing	Transverse myelitis – if hemi-cord seen with dissociated sensory loss (Brown–Séquard syndrome)
		Multiple sclerosis – with signs of additional CNS involvement
	Benign tumours of brain or spinal cord – gradual onset and progression	
	Inherited – very gradual onset and progression	Friedreich's ataxia – with absent ankle reflexes and cerebellar signs
		Hereditary spastic paraplegia
Rare causes in PACES	Malignant tumours of the brain or spinal cord	
	Infection – meningitis	
	Post-ictal	
	Drug toxicity	
	Vitamin B_{12} deficiency – may have absent reflexes	
Considerations	Some causes such as malignant tumours are common in clinical practice, but unlikely to be encountered in PACES	
	Other causes such as Friedreich's ataxia are more likely to be encountered in PACES than clinical practice	
	The key to refining the differential diagnosis is the time course. There may be a clue to this in the stem given before the case	
	Always consider voluntary withdrawal, the most common cause of a false positive Babinski and apply the three rules to distinguish	
	Consider and comment on which other upper motor neuron signs are present: brisk reflexes, increased muscle tone, ankle clonus and weakness in a pyramidal distribution	

Table 1. Potential causes of an incorrectly interpreted positive Babinski sign

Contraction of tibialis anterior	This causes the toes to go up passively as a result of ankle movement without contraction of extensor hallicus longus
Very active flexion synergy	Following a brisk normal flexion synergy reflex including usual flexion of the toes, the observer sees the big toe return to its neutral position by going up, but this is caused by relaxation of the toe flexors rather than extensor hallicuslongus contraction
Voluntary toe wriggling	These movements are jerky, inconsistent and not synchronous to flexion synergy in the rest of the leg
Relative movement	The smaller toes go down while the big toe remains immobile creating the illusion of an up-going toe
Isolated fanning of the toes	Although suggested by some to be an important feature of the Babinski sign, in fact may be seen in healthy subjects and may not be seen in Babinski sign so is of limited use

The Babinski sign is very rare in healthy adults, but is normal in infants under the age of 1 year, and may be seen in adults during deep sleep (van Gijn, 1996). However, scratching the soles of your patient's feet while in a deep sleep is not recommended. There may be difficulty in interpreting clinical signs in conditions where upper and lower motor neuron pathology coexist, such as in motor neuron disease. Similarly, absent ankle reflexes with extensor plantar responses may be seen in Friedreich's ataxia and vitamin B_{12} deficiency. Situations where there may be pathology of the pyramidal tract without a Babinski sign evident are outlined in *Table 2*.

Reliability and reproducibility of the sign

Studies examining the reliability of the Babinski sign have reported inter-observer Kappa values ranging from 0.15 (Maher et al, 1992) to 0.57 (Singerman and Lee, 2008) with others in between (Sisk et al, 1970; Miller and Johnston, 2005). All studies were relatively small which, together with methodological variations, probably explain the range of Kappa values obtained.

One study directly compared the Babinski sign with the rival signs of Chaddock, Gordon and Oppenheimer and found that Babinski had the highest inter-observer reliability of the four (Singerman and Lee, 2008). Raijmakers et al (1991) showed improvement in performance and interpretation of the plantar response when an instructional videotape was shown to medical students incorporating the three key rules to interpreting the response (*Figure 2*) in addition to usual bedside teaching. Overall, these studies suggest that the most important aspect of ensuring reliability of the sign is careful observation and interpretation of the response, rather than the exact method of stimulation.

Conclusions

Of all eponymous signs in medicine, Babinski is probably the most well known which reflects that after more than 100 years it remains a valuable part of the clinical examination. Judging the response is more difficult than it first appears with numerous causes of erroneous interpretation and reduced reliability. However, if the up-going toe is caused by extensor hallicus longus contraction, synchronous with contraction of other muscles involved in the flexion synergy reflex, and reproducible on repeated stimulation, then it is a reliable indicator of upper motor neuron dysfunction. **BJHM**

Conflict of interest: none.

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Table 2. Potential causes of a negative Babinski sign despite upper motor neuron dysfunction

Joint deformity	Quite common, especially hallux valgus such that the joint can not go up, although extensor hallicus longus activation may be shown on electromyography of these patients
Lower motor neuron lesions	Any cause of lower motor neuron dysfunction may mask a Babinski sign which would otherwise be present. For example pressure palsy of the common peroneal nerve which can occur in chronic paraplegia can mask a Babinski sign
Incomplete pyramidal syndrome	If the fibres which innervate dorsiflexor muscles of the foot are not involved
Spinal shock	In acute spinal shock no response might be seen from plantar stimulation as a result of depressed activity of the segmental pathways which mediate the flexor synergy

KEY POINTS

- The Babinski sign is elicited by firmly stroking the lateral side of the sole of the foot with an orange stick or similar.
- Following this stimulus in healthy adults the big toe goes down as the result of a localized cutaneous reflex.
- In patients with an upper motor neuron lesion the big toe goes up as a result of both inhibition of normal flexion and release of toe extension which is part of the flexion synergy reflex.
- Correct judgement of the response can be improved by following the three rules in *Figure 2*.
- Inter-observer reliability of the Babinski sign is reasonable and is better than rival signs.