

The Babinski sign and the pyramidal syndrome

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SUMMARY The presence or absence of a Babinski sign can be puzzling, but in the light of existing pathological studies it is more fruitful to consider which pyramidal tract fibres release it than whether they release it. This was investigated clinically, by looking for correlations with other reflex changes and with motor deficits in the leg. A survey of 50 patients with a unilateral Babinski sign and six patients who lacked it in spite of other pyramidal tract signs was supplemented with follow-up of the patients who had acute lesions. Appearance of the Babinski sign proved to depend on the interaction of two factors: (1) activity (not necessarily hyperactivity) in the segmental pathways of the flexion synergy; (2) a motor deficit of the foot, in some cases consisting only in an impairment of rapid foot movements, and probably representing a disturbance of direct pyramidal tract projections to distal motoneurons.

In 1896 Babinski described the upgoing toe sign in patients with "organic affections of the central nervous system", and one year later he linked it more specifically to disturbances of the pyramidal tract. Since then, the Babinski sign has become firmly established as the cornerstone of the clinician's "pyramidal syndrome." Although it has been objected that the sign could appear in many temporary derangements of brain function (Lassek, 1944), or after partial cordotomies without subsequent degeneration of corticospinal fibres (Nathan and Smith, 1955), the absence of secondary degeneration does not necessarily imply functional integrity (Walshe, 1956). This is rather obvious in the case of an epileptic seizure or intoxication. But it applies equally to the cordotomy studies, where the lesions bordered upon the pyramidal tract and may well have caused loss of function by focal demyelination (Babinski, 1885; McDonald and Sears, 1970). Babinski's original view has received new support from two recent case reports which mention the appearance of a Babinski response after an almost pure lesion (infarction) of the contralateral pyramid (Chokroverty *et al.*, 1975; Leestma and Noronha, 1976). Moreover, there is no other spinal cord pathway in man that is crossed and descends low enough in the spinal cord.

It remains to be explained why the Babinski sign can be absent in cases of proven pyramidal tract degeneration (Nathan and Smith, 1955). If

the lesion is complete, we must assume interruption of the reflex arc. If not, we should keep in mind that corticospinal fibres do not only originate in different parts of the precentral and postcentral cortex, but also have different *terminations*. The restriction that a Babinski response can be expected only when "leg fibres" of the pyramidal tract are involved (Potts and Weisenburg, 1910) is still crude: the motoneurons of the leg muscles are grouped into separate columns within the anterior horn of the cord, depending on whether they supply proximal or distal muscles, flexor or extensor muscles (Sharrard, 1955; Kuypers, 1973). In addition, there are important pyramidal tract projections to the intermediate (interneuronal) zone, and even to cells in the dorsal horn of the cord (Kuypers, 1973).

This divergence of pyramidal tract projections allows two explanations of the Babinski sign.

(a) The Babinski sign might be released by dysfunction of pyramidal tract fibres that project on the *interneuronal zone*, at least on those interneurons that subserve the flexion reflex synergy, of which the Babinski sign is part (Marie and Foix, 1912; Walshe, 1914). As these interneurons are necessarily interconnected across the segments of the lumbosacral spinal cord, a Babinski sign would always be accompanied by hyperactivity in other, more proximal, flexor muscles; and in this case a Babinski response would not necessarily imply a disturbance of voluntary leg movements relayed by projections to motoneurons.

(b) Alternatively, the Babinski sign might result from interference with pyramidal fibres projecting directly on *motoneurons*. In that case the effector of the sign, the extensor hallucis longus (Landau and Clare, 1959; van Gijn, 1975), would at the same time be less responsive to descending impulses. We should expect impairment of voluntary dorsiflexion of the great toe, perhaps also of dorsiflexion and eversion of the foot, mediated by neighbouring motoneurons. Pyramidal tract control of distal muscles subserves not only muscle force, but also the capacity to perform fractionated and rapid movements (Tower, 1940; Kuypers, 1973). Therefore the motor deficit accompanying a Babinski sign might manifest itself not only by weakness, but also by loss of skill. The release of the flexion reflex would not necessarily involve proximal muscles.

Thus a study of the coincidence of the Babinski response and other pathological signs in the lower limbs might give insight into physiological and anatomical relationships. Earlier workers have investigated the concurrence of various deficits and release phenomena in the lower limbs (Graeffner, 1906; Lassek, 1945; Dohrmann and Nowack, 1974). But none of these reports mentioned the clinical features that are most needed to elucidate the pathophysiology of the Babinski sign: first, possible asymmetry of the flexion reflex in proximal muscles, secondly the distribution of weakness or—in the absence of weakness—the capacity to perform skilled foot movements.

Patients and methods

Two groups of patients were studied.

1. Fifty patients (27 men and 23 women) with a strictly unilateral and unequivocal Babinski sign were found among inpatients and outpatients during a period of six months. To be selected for study they also had to be well enough to co-operate in the various tests, and they had to be free of unrelated motor disorders (diseases of cerebellum, basal ganglia, or peripheral nerves). A cerebral lesion was diagnosed in 42 of the patients: 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.

2. In the same time span six patients were found to show unilateral pyramidal tract signs without a

Babinski sign. Four of these suffered from cerebral infarction, one had motor neurone disease, and one had undergone surgery for an intracerebral haematoma.

All patients in these two groups who had acute lesions were re-examined every few days, until no further change occurred. Three patients in group 1 "lost" a unilateral Babinski response, while other pyramidal tract signs remained. Conversely, two patients in group 2 developed an up-going toe response only after an initial period in which it had been unexpectedly absent. These five patients will be considered separately.

EXAMINATION

Plantar reflex

All patients were examined in the supine position. Stimulation was performed with a small wooden stick (orange stick), at the lateral plantar border and plantar arch. If voluntary movements interfered, the stimulus was repeated on the lateral border of the dorsum of the foot.

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 was used 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

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-.

Skill

Four tests were used: dorsiflexion of the great toe, separately from the foot; wriggling of the little toes; rapid foot tapping against the examiner's hand (with the patient supine); rapid up-and-down movements of the great toe, with the foot 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 three-quarters of that on the control side, or when there were obvious qualitative differences, such as dysrhythmia or simultaneous movement of the great and little toes on one side only.

Associated dorsiflexion of the great toe

This was investigated in three ways: ipsilateral hip flexion against resistance; contralateral hip flexion against resistance; contralateral rapid toe movements.

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 -4 (absent) through zero (normal) up to +4 (clonus).

Tone

Passive resistance was separately examined at 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.

Abdominal reflexes

Two levels were tested on each side, above and below the umbilicus. In men, the cremasteric reflex was also included.

Wasting

The circumference of the thigh was measured 150 mm above the medial margin of the knee joint, the calf at its maximal girth. Wasting was assumed to be present when the difference was 20 mm or more at one of these levels.

Results

UNILATERAL BABINSKI SIGNS

Voluntary movements

The most frequent finding accompanying the up-going toe response was weakness of foot movements: 76% (Table 1). In almost half of this number (32%) the weakness was only slight. This always concerned dorsiflexion of the foot, dorsiflexion of the hallux, foot eversion, or a combination of these movements. In 10% the weakness was confined to the foot.

Loss of skill in the foot was always a relative judgment—that is, on comparison with the control side. If a difference was found, it almost always concerned the capacity to perform rapid foot or

Table 1 Weakness and loss of skill (%) on the side of unilateral Babinski response (50 patients)

Response	Percentage change	
Weakness		
Paralysis of foot	28	} weakness of foot: 76
Marked weakness	16	
Slight weakness of foot (> MRC 4)	32	} no weakness of foot: 24
Only proximal weakness	14	
No weakness	10	
	100	
Loss of skill		
With paralysis or marked weakness of foot (44)	44	} no weakness of foot but loss of skill: 16
With slight weakness of foot (32)	30	
With only proximal weakness (14)	12	
Without weakness	4	
No loss of skill	10	
	100	

toe movements. The ability to dorsiflex the great toe apart from the foot was found in almost every patient who could dorsiflex the great toe at all, at least after a few attempts. Asymmetry in wriggling the little toes was also rare in the absence of weakness. Comparing the rate and rhythm of rapid foot or toe movements appeared to be a much more sensitive test. The two procedures gave similar results in most patients, but occasionally one or other was more sensitive. In either test, normal values range from 20 to 40 taps in 10 seconds.

Of the 12 patients without evidence of foot weakness, loss of skill could be demonstrated in eight. Six of these eight patients had slight proximal weakness, two had normal power. Only four patients showed neither weakness nor loss of skill. On comparing the occurrence of other signs in these exceptional patients with their frequency in the entire group, the only statistically significant difference was to find in all four the relatively rare sign of hallux dorsiflexion on contralateral hip flexion against resistance (occurrence in the whole group 22%, the Fisher test gives P=0.001). However, this phenomenon was also found on the control side of other patients (Table 2), and thus it can hardly explain in itself why a Babinski sign occurs without a 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.

Reflex changes

The flexion reflex on the side of the Babinski sign almost invariably involved simultaneous activity in tensor fasciae latae, hamstrings, or iliopsoas; there were only two exceptions among the 50 patients. Exaggeration of the flexion reflex in these

Table 2 *Associated dorsiflexion of the great toe (%) on the side of unilateral Babinski response (50 patients)*

	<i>On ipsilateral hip flexion</i>	<i>On contralateral hip flexion</i>	<i>On contralateral rapid toe movements (mirror movements)</i>
Not possible	14	2	2
Positive	14	18	2
Bilaterally positive	20	4	2
On control side only	18	6	14
Absent	34	70	80
	100	100	100

proximal limb muscles in comparison with the control side was found in not more than 58%. This means that the Babinski sign is not dependent upon hyperactivity of the flexion reflex as a whole.

Increase of tendon jerks was found in three out of four patients with a Babinski sign (74%). Table 3 shows that asymmetry usually affected several reflexes together. The ankle jerk was rarely increased alone (10%), and not preferentially in cases with a purely distal motor deficit. The jerks elicited from the adductor and biceps femoris muscle rarely gave extra information. In other words, the release of tendon reflexes generally involves different lumbosacral segments, and flexor as well as extensor muscles. It is, therefore, striking to find that the presence of increased tendon jerks is not statistically correlated (Fisher test) with exaggeration of the flexion reflex, which is

Table 3 *Additional reflex changes (%) on the side of unilateral Babinski response (50 patients)*

<i>Reflex</i>	<i>Percentage change</i>	
Flexion reflex in proximal muscles		
Exaggerated	58	
Symmetrical	38	
Diminished	4	
Tendon reflexes		
Increase of ankle jerk only	10	} increase of one or more tendon reflexes 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	
Tone		
Increased in ankle (at least)	10	
Increased in knee only	6	
Symmetrical	62	
Decreased in knee only	8	
Decreased in ankle	14	
Abdominal reflexes		
Diminished	42	
Only cremasteric reflexes diminished	6	(11% of men)
Symmetrical	22	
Absent	26	
Decreased on control side	4	

also a multisegmental release phenomenon. The lack of correlation, positive or negative, between these two forms of hyperreflexia also holds true when "absolute" values on both sides are considered instead of asymmetry: every activity level of the flexion reflex was found together with a wide range of myotatic reflex activity, and vice versa.

Disorders of tone varied with time. In the 42 patients with cerebral lesions, hypotonia occurred significantly more often when the onset of disease had been less than four weeks before (10/19 versus 1/23; Fisher test gives $P=0.001$), and a reverse tendency was found for spasticity (1/19 versus 7/23; $P=0.05$). The presence of the other physical signs did not depend on the duration of disease.

Abdominal reflexes, represented much higher in the spinal cord, were reduced in 42%, but 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, infantile hemiplegia).

PYRAMIDAL TRACT SIGNS WITHOUT BABINSKI RESPONSE

Six patients lacked a Babinski response while other pyramidal tract signs were present. Two developed it at a later stage and will be discussed separately. In the remaining four patients, reflex changes on the side in question consisted in exaggerated tendon jerks (cases 1, 2, and 4) and reduced abdominal reflexes (cases 3 and 4). The flexion in proximal muscles was (symmetrically) present in all four patients, giving no reason to suppose a disturbance in the spinal pathways of the plantar reflex.

Because, in the first group of patients, the Babinski sign was so closely associated with disorders of voluntary innervation of the foot, it was important to find that of these four patients in whom the Babinski sign was unexpectedly absent, only one showed definite motor impairment of the foot (case 1, suffering from motor neurone disease, with weakness (MRC 4) of dorsiflexion and eversion, and with slowing down of foot movements). Case 2 showed equivocal weakness of foot dorsiflexion but no loss of skill, case 3 had only proximal weakness, also with symmetry of rapid foot movements, and case 4 had no motor deficit at all.

BABINSKI SIGNS APPEARING IN OR DISAPPEARING FROM THE PYRAMIDAL SYNDROME

Until now, the relationship between upgoing toe response and other pyramidal tract signs has been

studied by a cross-sectional survey. Obviously it is also of great interest to study temporal relationships: when a Babinski sign disappears while other signs remain, or, conversely, 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 5 (improvement of power and skill) A 60 year old driver with an infarction in the left cerebral hemisphere showed, on the ninth 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 upgoing, 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 jerks were still increased on the right.

Case 6 (improvement of skill) A 53 year old maintenance technician suffered a mild stroke in the territory of the left middle cerebral artery. On the tenth 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; the flexion reflex was weak on both sides. Three weeks later he had no longer any motor disability, nor a Babinski sign; only definite hyperreflexia remained.

Case 7 (waning of flexion reflexes) A 56 year old publican sustained an acute right sided hemiparesis. On the second day he was found to have moderate weakness of the right leg (all flexor movements with impaired skill and hyperreflexia. Flexion reflexes were bilaterally brisk (++) , with a Babinski sign on the right. On the tenth day the clinical picture was practically unchanged but for the right plantar response which was now downgoing, and for both flexion reflexes which were weak (±).

Late Babinski signs

Two patients initially lacked a Babinski sign in the presence of other pyramidal tract signs, but developed it later. Both had a dense hemiplegia.

Case 8 (initial absence of flexion reflex) A 58 year old nurse sustained a subarachnoid haemorrhage from one of two left sided berry aneurysms. On the fourth day she had a paralysed right foot, symmetrical tendon jerks, and no flexion reflex or Babinski sign on either side. On the thirteenth 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 paralysed, 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 9 (initial depression of flexion reflex) A 50 year old industrial manager suddenly developed a right hemiplegia, without impairment of consciousness. On the third day his right leg was still paralysed. No tendon reflexes could be obtained on either side (this had been noted before by insurance physicians). Plantar stimulation on the left gave a downgoing 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.

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 impairment of voluntary foot movements, the latter either in the form of weakness or only as a reduced capacity to perform rapid alternating movements. Secondly, the Babinski sign is not necessarily found in association with hyperactivity of the flexion reflex in other muscles than the extensor hallucis longus. Nevertheless, a Babinski sign can appear only if the intraspinal pathways of the flexion reflex synergy are operative, however severe the motor deficit in the foot. Both these factors, impaired corticospinal control and the activity of the flexion reflex, will be reviewed separately before considering how their interaction results in a Babinski sign.

IMPAIRED CORTICOSPINAL CONTROL OF THE FOOT

If a disturbance of the pyramidal tract affects projections to motoneurons 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 such tests proved to be equally rewarding. In the group of 50 patients with

a unilateral Babinski sign, normal power of foot movements was found in 24%, but reduced skill could still be demonstrated in two-thirds of these, leaving only 8% of Babinski signs unassociated with a motor deficit in the foot. In addition, 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 while other pathological signs persisted (cases 5 and 6). Lesions of the neuraxis which cause proximal weakness alone should not lead us to expect a Babinski sign. One patient who "lacked" it (case 3) showed weakness of hip and knee flexion (MRC 4) and reduced abdominal reflexes, but power and skill in the foot were normal.

The next most frequent sign accompanying the Babinski response was increase of tendon reflexes (74%). However, apart from being absent in a quarter of the 50 patients with a unilateral Babinski sign, it also persisted in two cases where upgoing toe sign and motor impairment of the foot simultaneously disappeared. In other words, the Babinski response and hyperreflexia regularly occur without one another, and patients with increased tendon jerks alone are not "entitled" to a Babinski sign (cases 2 and 4). 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. Associated dorsiflexion of the hallux on ipsilateral or contralateral hip flexion was found infrequently, and on the control side as well as on the side of the Babinski sign (Table 2). A recent claim that the "crossed upgoing toe sign" can be regarded as a reliable and sometimes superior test (Hindfelt *et al.*, 1976) is not confirmed by my study, nor by earlier work (Strümpell, 1887; Brain and Wilkinson, 1959).

Within the pyramidal tract syndrome, only impairment of skilled foot movements is so intimately connected with occurrence of the Babinski sign that these two pathological features can be assumed to result from a disturbance of identical or closely related pyramidal tract fibres.

ACTIVITY OF THE FLEXION REFLEX

The Babinski sign is part of the flexion reflex synergy, and it is rarely the minimal response, as noted earlier (Walshe, 1914). Of 50 patients in whom the stimulus intensity was not higher than necessary to elicit a clear upgoing toe response, more proximal leg muscles were activated in 48. In cases where a Babinski sign is absent in a weak foot, we may, therefore, infer from an absent or feeble response in proximal leg flexors that there is insufficient activity in the reflex pathways con-

verging upon the extensor hallucis longus muscle. This inexcitability may be the result of individual variation (case 8) or of initial reflex depression (case 9), common after acute spinal transection (Guttmann, 1976). Also, a Babinski sign can disappear after the acute episode because the activity of the flexion reflex dwindles (case 7).

If a Babinski sign does appear, it cannot be explained simply by a pathological overactivity of the flexion reflex as a whole, because it was as often as not accompanied by an exaggerated response in proximal flexor muscles. Moreover, asymmetry of the flexion reflex can persist after disappearance of a Babinski sign (case 5). As release of the entire flexion reflex involves several segments, it is probably caused by dysfunction of projections to interneurons in the intermediate zone of the spinal grey matter.

The identity of the descending pathways concerned with control of the flexion reflex has been investigated only in animals. In the cat, interneurons mediating the flexion reflex are tonically inhibited by the medial portion of the reticular formation of the brainstem (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 man, an argument for at least partial control of the flexion reflex by brainstem centres is that flexor spasms are much more common after spinal lesions than in cerebral disease.

RELEASE OF THE BABINSKI SIGN

The results show that the Babinski response can be released separately from the flexion reflex as a whole, and that it is almost invariably associated with motor impairment of the foot. In some cases this motor impairment is manifested only by slowing down, dysrhythmia, or reduced fractionation of foot movements. The capacity for independent and fast movements is mediated by direct cortical projections to individual motoneurons (Kuypers, 1973). Because loss of this ability alone is apparently sufficient to permit release of the Babinski sign, the corticospinal "shielding" of extensor hallucis longus motoneurons from activity in the flexion reflex arc must have its impact at or near these motoneurons. In other words, an excitatory action (direct and selective innervation of distal motoneurons) 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 for the extensor hallucis longus, or consist in

separate, but intimately related pyramidal tract fibres. The proposed interaction between descending tracts and flexion reflex pathways is represented schematically in the Figure.

Nevertheless, there are exceptions. Some patients showed a Babinski sign among other pyramidal features but without demonstrable weakness or loss of skill; one other lacked it while having some motor deficit in the foot. There are two ways of explaining these inconsistencies. The first is that excitation of distal motoneurons and inhibition of impulses via flexion reflex afferent nerve fibres can be dissociated because they are mediated by different neurones, however closely linked. The alternative is to assume motor abnormalities that are too subtle for testing on the one hand, and a relatively poor influx of segmental impulses to motoneurons of the extensor hallucis longus on the other.

THE "UPPER MOTOR NEURONE"

In the present series of 50 patients with a unilateral Babinski sign there were always other abnormalities as well, but 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 the fibres that controlled the reflexes (Strümpell, 1899). The exact course of these different fibres has been the subject of several hypotheses, usually derived from animal experiments. One view has been that the pyramidal tract took its origin from many other regions than the central area of the cerebral cortex (Lassek *et al.*, 1957), but the degeneration studies underlying 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. Although nowadays few scientists regard the motor system as a "co-ordinated maze" (Bucy, 1957), many assign a role to lesions of structures other than the pyramidal tract in the production of major clinical phenomena (Nyberg-Hansen and Rinvik, 1963; Brodal, 1969; Voorhoeve and de Jong, 1975).

Such speculations are largely unnecessary, as far as man is concerned, in view of two unprecedented case histories of almost pure infarction of one

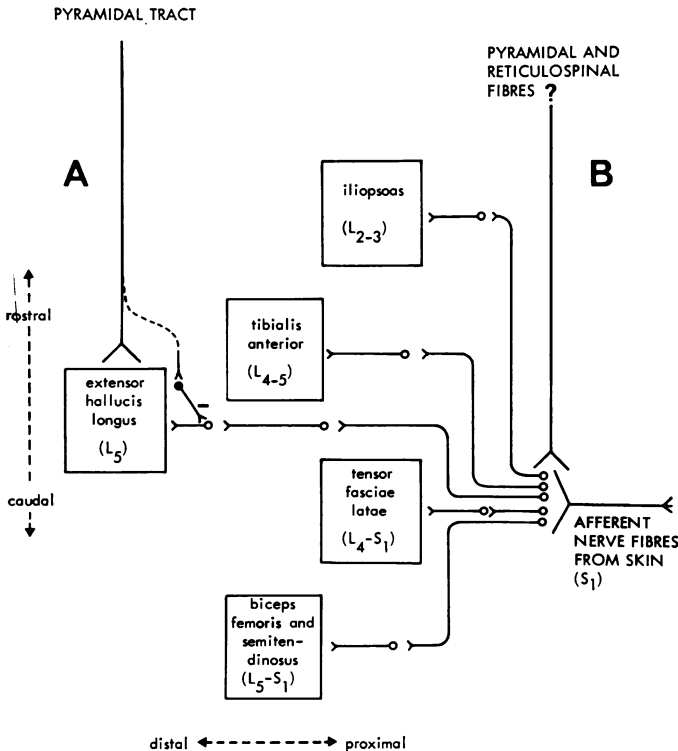


Figure Proposed interaction of pyramidal tract and flexion reflex pathways in the spinal cord. A—direct pyramidal tract control of distal motoneurons, with local inhibition of flexion reflex pathway; dysfunction causes loss of skill and releases the Babinski sign. B—descending control of interneurons that mediate the flexion reflex; dysfunction releases the flexion reflex as a whole, but this is not essential for the appearance of the Babinski sign.

medullary pyramid (Chokroverty *et al.*, 1975; Leestma and Noronha, 1976). The following signs were noted on the contralateral side: weakness, Babinski sign, increased tendon jerks, and—in only one patient—spasticity. These observations cannot exclude that interference with other descending fibre systems contributes to the appearance of clinical signs, and changes in abdominal and flexion reflexes were not recorded. However, it is even more important to realise that the fibres passing through the medullary pyramids are heterogeneous in terms of termination, regardless of origin (Kuypers, 1973). The tract consists only partly of “upper motor neurones” in the sense of direct corticomotoneuronal connections (Verhaart, 1962). Within the pyramidal syndrome of the leg, only an impairment of skilled foot movements and—closely linked to it—the Babinski sign, can be attributed to a disturbance of these direct connections. Other signs must depend on different projections, to the interneuronal zone in particular. Partial lesions of this diverging pyramidal tract cannot give a uniform clinical picture.

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