J Neurol (2010) 257:38–43
DOI 10.1007/s00415-009-5260-x

ORIGINAL COMMUNICATION

The occurrence of the Babinski sign in complete spinal cord injury

Jens A. Petersen · Martin Schubert · Volker Dietz

Received: 13 January 2009 / Revised: 23 June 2009 / Accepted: 8 July 2009 / Published online: 25 August 2009

Abstract The purpose of the present study was to explore factors that influence the occurrence of the Babinski sign (BS) in complete spinal cord injury patients. At Balgrist University Hospital, Zurich, Switzerland, thirty-five subjects suffering from a complete traumatic spinal cord injury (ASIA A) were examined for the occurrence of the BS, tendon reflex excitability and spastic muscle tone (Modified Ashworth Scale). Five subjects were acute/subacute (1–6 months after spinal cord injury (SCI)), 30 were chronic (SCI > 1 year). In one subject, the measures were examined before and after injection of intrathecal Baclofen. Subjects with a negative BS were investigated electrophysiologically for possible peripheral nerve damage. In 17 subjects (49%), the BS was present, while it was absent in 18 subjects (51%). The occurrence of the BS did not depend on the level of lesion. Most patients with a positive BS also presented a high spastic muscle tone, while those with a negative BS showed low level or absent spastic muscle tone. In 11 SCI subjects, absence of the BS was associated with peripheral nerve damage. In one patient, the BS along with spastic signs disappeared after intrathecal injection of Baclofen. In complete SCI subjects, the occurrence of the BS is connected with spastic muscle tone. The absence of the BS is frequently due to associated peripheral nerve damage.

Keywords Babinski sign · Reflex excitability · Spinal cord injury · Spasticity · Baclofen

J. A. Petersen (✉) · M. Schubert · V. Dietz
Spinal Cord Injury Center, University Hospital Balgrist, Forchstrasse 340, 8008 Zürich, Switzerland
e-mail: jens.petersen@balgrist.ch

Introduction

The Babinski sign (BS) was first described in 1896 [2]. It has been widely used in clinical neurology as a diagnostic sign indicating a pyramidal lesion. However, its pathophysiology has been a matter of debate, and it remains unclear whether its occurrence solely depends on the presence of a pyramidal lesion. The BS is thought to be part of the general withdrawal reflex synergy released by a lesion of supraspinal pathways that project onto the interneuronal zone of the lumbosacral cord [18]. At the same time, its presence also requires a lesion or functional deficit of those fibres projecting directly onto the motoneurons of the effector muscles [8, 13]. The effector organ of the reflex is the extensor hallucis longus (EHL) [8].

In pyramidal tract syndrome, only impairment of skilled foot movements is closely connected with occurrence of the BS. Therefore the latter is assumed to indicate a disturbance of the closely related pyramidal tract fibres [18]. If the BS is absent in the case of an upper motor neuron lesion this is thought to be due to a dysfunction of the segmental reflex pathways, normally caused by a pressure palsy of the peroneal nerve, or by an inexcitability of spinal motoneurons due to spinal shock [19].

Babinski himself assumed that a dysfunction of the pyramidal system is necessary but not sufficient to produce a BS [1]. Conversely, some post-mortem examinations have shown that an absence of the BS is compatible with severe lesions of the cortico-spinal tract and that in some cases the cortico-spinal tract appears to be intact while the BS is positive [17]. The conclusion from these studies would be that there is no particular relationship between the anatomical state of the cortico-spinal tracts and the appearance of the BS. In fact, our empirical impression from regular clinical examinations of complete tetra- and

paraplegic patients was that not all patients had a positive BS.

The aim of this study was to quantify this observation and to delineate factors that influence the inconsistent occurrence of the BS in complete spinal cord injury (SCI) after spinal shock has resolved.

Patients and methods

All subjects included in the study provided informed consent before participation. Ethical approval for the study was obtained from the local research ethics committee. The investigations were made in 10 tetraplegic and 25 paraplegic patients (five women and thirty men). The patients' ages ranged from 19 to 85 years (mean 45 years). All patients had sensory and motor complete (ASIA A) traumatic SCI according to the American Spinal Injury Association Impairment Scale. Levels of lesion were from C4 to T12. Subjects with conus, cauda and lumbar lesions were excluded. The time between the initial trauma and the date of examination was between four weeks (date of injury 2007) and 47 years (date of injury 1960). At the time of examination, 12 patients received spasmolytic medication (Baclofen tablets, Baclofen pump, benzodiazepines, cannabis, or Botox injections, Table 1).

Clinical examinations

The clinical examinations were performed based on a standard protocol when patients came to the paraplegia center for a regular check-up or at least one month after they had been hospitalized after trauma. Examinations were conducted over two years (2006–2008) by the same examiner. To test for the BS, mechanical stimuli were applied by the smooth, blunt handle of a reflex hammer (0 = absent, 1 = present). According to the recommendations of Dohrmann et al. [7] the lateral plantar surface was repeatedly stroked in a slow movement lasting 5–6 s from the heel to up to the middle metatarsophalangeal joint. Patella and Achilles tendon tap reflexes were taken for evaluation of lower limb reflex excitability in all patients (0 = no elicitable reflex, 1 = weak, 2 = normal, 3 = exaggerated reflex). In tetraplegic subjects, biceps reflex, triceps reflex and brachioradialis reflex were also tested. Muscle tone of upper (only tetraplegic patients) and lower limbs was assessed according to the Modified Ashworth Scale (MAS 0, flaccid muscle tone, to 4, limb rigid in flexion or extension). Reflex status and muscle tone were also repeatedly assessed in one tetraplegic subject who received three intrathecal injections with Baclofen (50, 75 and 100 μg) for therapeutic reasons.

Electrophysiological recordings

In all patients lacking a BS, electrophysiological examination of the common peroneal nerve was performed in order to demonstrate integrity of these motor units. Examinations were conducted over two years (2006–2008) by the same examiner. Compound muscle action potentials and nerve conduction velocity were measured. In subjects with abnormal neurography (cMAP obtained from peroneal nerve <1.0 mV), needle recordings (EMG) were performed in the M. extensor hallucis longus (Table 2). Ratings were as follows: no peripheral damage, acute denervation, chronic neuropathy, complete denervation, not testable.

For subjects with an injury date after the year 2000, completeness of SCI was additionally confirmed by a lack of tibial nerve evoked somatosensory potential (tSSEP) (pre-existing data). Before the year 2000, tSSEP was not part of the standard assessments of the conducting center.

Statistics

Mann–Whitney Test was used to relate the following clinical parameters: BS, level of lesion, subject age, duration of SCI, age at onset of SCI, spasmolytic medication, reflex excitability, and the maximum MAS score of each subject. Reflexes were assumed to be exaggerated if at least one reflex in arms and legs (cervical lesions) or in the legs was exaggerated (Table 1). A significant result was assumed if $P < 0.05$.

Results

A total of 17 (49%) out of the 35 traumatic complete SCI subjects examined presented with a positive BS while this was absent in 18 patients (51%). In three cases, the BS was only elicitible in one leg while in 13 it appeared bilaterally. In two patients (No. 10 and 22, Table 1) only one leg could be examined because the patients wore a leg cast. One of them presented with a positive BS, the other one with a negative sign. Eight patients with a positive BS and four patients with a negative BS were under some spasmolytic medication (Table 1). The neurographic and EMG examinations of the peroneal nerve were conducted in 14 out of 18 patients with a negative Babinksi sign (Table 2). Three patients showed no peripheral nerve lesion, while in 11 patients (22 legs) bilateral peripheral nerve damage could be demonstrated. Acute denervation was found in eight legs. Chronic neuropathy could be demonstrated in six legs. Complete denervation could be proven in eight legs. In four subjects, the electrophysiological examination could not be performed due to death (No. 15), unwillingness to participate (No. 3), move to a foreign country

Table 1 Clinical data of subjects included in the study

No.	Sex	Age, years	Level	Examination (months after SCI)	Medication	Babinski right	Babinski left	Maximum MAS	Exaggerated reflexes
1	M	41	C5	102		0	0	1	None
2	M	37	C6	201		0	0	1	AR ri, AR le
3	M	56	C7	276		0	0	0	AR ri, AR le
4	F	58	C4	13	Baclofen pump	1	0	3	None
5	M	19	C4	2	Baclofen 3 × 5 mg/day	1	1	2	None
6	F	65	C5	23		1	1	1	None
7	M	27	C6	62		1	1	2	PR ri, PR le, AR ri, AR le, BR li, BR re, TR ri, TR le, BRR ri, BRR le
8	M	36	C7	424	Clonazepam 2 × 2 mg/day	1	1	3	PR ri, PR le, AR ri, AR le, BR ri, BR le, TR ri, TR le, BRR ri, BRR le
9	M	29	C7	104		1	1	3	PR ri, PR le, AR ri, AR le, BR ri, BR le, TR ri, TR le, BRR ri, BRR le
10	M	67	C8	567		1	NT	0	None
11	M	29	T10	1		0	0	0	None
12	F	75	T11	471		0	0	0	None
13	M	27	T11	116		0	0	0	None
14	M	25	T11	91		0	0	0	None
15	M	55	T12	343		0	0	0	None
16	M	52	T2	396		0	0	0	None
17	M	40	T2	13		0	0	0	PR ri, PR le, AR ri, AR le,
18	M	41	T3	5		0	0	1	None
19	M	43	T4	222	Occasionally cannabis	0	0	3	None
20	M	85	T4	401	Baclofen 3 × 25 mg/day	0	0	1	None
21	M	56	T4	6	Baclofen 2 × 10 mg/day	0	0	1	None
22	M	59	T5	315		0	NT	0	None
23	M	45	T5	281	Baclofen 5 × 25 mg/day; Clonazepam 2 × 2 mg/day	0	0	0	PR ri, PR le
24	M	42	T6	182		0	0	3	None
25	M	44	T7	117		0	0	1	None
26	M	43	T10	235	Diazepam 1 × 10 mg/day	1	1	3	PR ri, PR le, AR ri, AR le
27	F	20	T10	83	Botox 300 IE Botox in lower extremities	1	1	1	None
28	F	83	T11	20		1	1	2	PR ri, PR le, AR ri, AR le
29	M	23	T11	13		1	1	2	None
30	M	44	T2	13	Baclofen 4 × 25 mg/day	1	1	3	PR ri, PR le, AR ri, AR le
31	M	37	T4	185		1	0	3	PR ri, PR le, AR ri, AR le
32	M	70	T5	29	Baclofen 1 × 50 mg/day	1	1	3	None
33	M	35	T7	170		1	1	3	PR ri, PR le
34	M	39	T7	3		0	1	2	PR ri, PR le
35	M	33	T9	88	Baclofen 7 × 25 mg/day	1	1	1	PR ri, PR le, AR ri

NT Not testable (leg cast), *PR* Patellar reflex, *AR* Achilles reflex, *BR* biceps reflex, *BRR* brachioradialis reflex, *TR* triceps reflex, *ri* right, *le* left

(No. 1) or because the patient did not respond. In 14 out of 35 subjects, clinically complete SCI had previously been confirmed by the lack of tibial somatosensory potentials.

Muscle tone was increased (MAS 1–4) in 24 (67%) of all patients examined in this study. This was the case in all but one subject with a positive BS (16 of 17, 94%). Figure 1 shows that subjects with a positive BS tend to have high levels of MAS while in those without a BS muscle tone is rather low. Tendon tap reflexes were exaggerated in 14 subjects (40%). Ten of these subjects had a positive BS, while in four of the patients a BS was absent. The relationship between a positive BS and increased muscle tone (MAS) was highly significant ($Z = -3.669$; $P < 0.001$), while the relationship with tendon reflex

Table 2 Patients with a negative Babinski sign: electrophysiological data (peroneal nerve)

No.	Cmap ri (mV)	NCV ri (m/s)	Cmap le (mV)	NCV le (m/s)	sA EHL ri	sA EHL le	Needle potential EHL ri	Needle potential EHL le	Damage ri	Damage le
1	NT	NT	NT	NT	NT	NT	NT	NT		
2	0.00		1.70		4/10	4/10	2.00	2.60	Acute denervation	Acute denervation
3	NT	NT	NT	NT	NT	NT	NT	NT		
11	1.20		1.10		ND	ND	ND	ND	None	None
12	0.00	0.00	0.00	0.00	0/10	0/10	ND	ND	Chronic neuropathy	Chronic neuropathy
13	0.00		0.00		ND	3/10	0.00	0.00	Complete denervation	Complete denervation
14	NT	NT	NT	NT	NT	NT	NT	NT		
15	NT	NT	NT	NT	NT	NT	NT	NT		
16	0.00		0.00		0/10	4/10	3.90	1.60	Chronic neuropathy	Acute denervation
17	7.80	58.60	6.50	54.60	ND	ND	ND	ND	None	None
18	0.20		0.00		8/10	3/10	2.80	0.00	Acute denervation	Complete denervation
19	0.00		0.00		0/10	0/10	0.00	0.00	Complete denervation	Complete denervation
20	0.00		0.00		4/10	4/10	0.00	1.20	Complete denervation	Acute denervation
21	0.00		0.00		6/10	7/10	1.10	1.50	Acute denervation	Acute denervation
22	0.60		0.10		2/10	2/10	3.50	1.10	Chronic neuropathy	Chronic neuropathy
23	0.00		0.00		5/10	3/10	1.00	0.00	Acute denervation	Complete denervation
24	0.00		0.00		1/10	1/10	0.00	1.20	Complete denervation	Chronic neuropathy
25	2.90	42.50	3.90	49.30	ND	ND	ND	ND	None	None

NT Not testable, *ND* not done, *Cmap* compound muscle action potential, *NCV* nerve conduction velocity, *sA* spontaneous activity, *EHL* M. extensor hallucis longus, *ri* right, *le* left

excitability was at the border of significance ($Z = -2.177$; $P = 0.067$) (Table 1). Testing for an association between reflex exaggeration and MAS also yielded a trend that both were related ($Z = -2.010$; $P = 0.053$).

Levels of lesion ranged from C5 to T12 in the group with a negative BS and from C4 to T11 in the group with a positive BS with an almost symmetric distribution of lesion levels in both groups. Average duration of SCI was 9.3 (0.5–47.7) years for patients with a positive BS, and 16.9 (0–39.4) years for patients with a negative BS. The BS did not show a significant relationship with any of the following data: level of lesion, subject age, duration of SCI, age at onset of SCI, and spasmolytic medication.

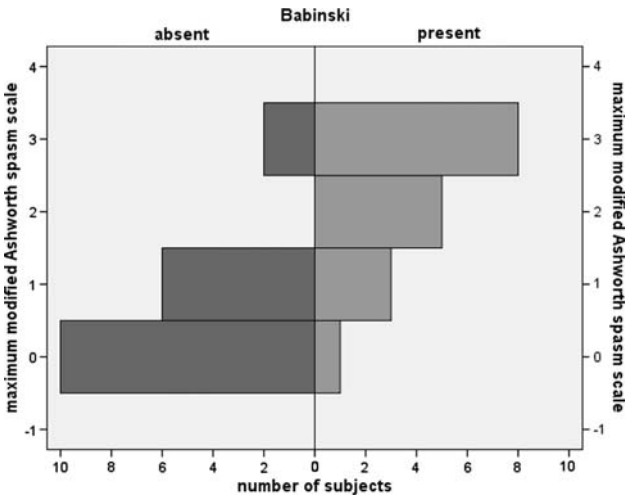

Fig. 1 Degree of muscle tone in lower extremities according to the Modified Ashworth Scale (MAS) in 35 SCI subjects with absent or present Babinski signs. Maximum MAS = highest muscle tone from upper and lower (tetraplegic) or lower (paraplegic) extremities according to the MAS

Baclofen injection

One subject received three therapeutic injections of 50, 75 and 100 μg of Baclofen on three consecutive days. Before each injection, it was clinically confirmed that the patient had increased muscle tone in the lower legs (up to MAS 3) and a positive BS bilaterally. Four hours after the first injection (50 μg), muscle tone was low (MAS 1) and the BS was present unilaterally. Four hours after the second injection (75 μg), muscle tone was low (MAS 0–1) and the BS was bilaterally absent. Four hours after the third injection (100 μg), muscle tone was flaccid (MAS 0) and the BS was absent bilaterally. Twenty-four hours later, the BS reappeared.

Discussion

Four principal features emerged from this case study on the occurrence of a BS in SCI. First, the Babinksi sign may or

may not be present in complete SCI subjects. Secondly, there seems to be a close association between the occurrence of the BS and increased muscle tone. Third, the BS is depressed and muscle tone reduced by intrathecal injection of Baclofen [16]. Therefore, muscle tone seems to be a requirement for the occurrence of the BS. Fourth, the absence of the BS is otherwise most likely due to peripheral nerve damage if not explained by low muscle tone.

In the present study it is unlikely that the co-occurrence of a lacking BS and reduced muscle tone is due to a protracted spinal shock, defined by flaccid muscle paresis and loss of tendon reflexes below the level of lesion [4]. Spinal shock has been reported to end 1–3 days [6] to a few weeks post-injury. The BS is usually present within 2 weeks after SCI [12] and its absence in the initial phase is probably due to a temporary suppression of interneuronal activity [15].

In our study, duration of SCI and presence of the BS were not interrelated, i.e. 30 of the tested subjects were in the chronic stage of SCI while five subjects were injured 1–6 months prior to examination. In these latter cases, the lack of the BS might be due to a persistence of spinal shock (patients number 5, 11, 18, 21, 34, Table 1). However, 15 of the 30 chronic patients were also lacking a BS and thus the lack cannot be attributed to spinal shock.

In most of our SCI subjects the absence of the BS was associated with a peripheral neuropathy. This is in line with earlier reports indicating that an absence of the BS is frequently due to damage to the segmental reflex pathway. [19] It is known that peripheral nerve function is often abnormal in the lower limb in patients with cervical lesions. Specifically, CMAP in the tibial and peroneal nerves are often reduced and motor conduction velocity is slow [11]. A possible explanation might be a transsynaptic peripheral degeneration of alpha motoneurons in the context of SCI [11, 14], which occurs when motoneurons are deprived of supraspinal trophic inputs. Additionally, peripheral nerve damage in chronic SCI subjects might be due to neuropathy or pressure palsy which in this population is more likely to occur due to immobility and wheelchair use. The latter possibility however seems rather unlikely in the subjects presented here since in all cases, peripheral nerve damage was observed bilaterally. Axonal motor neuropathy was also observed in conus or cauda damage [3]. However, conus and cauda lesions as well as lumbar lesions had been excluded from this study beforehand.

From the present data it cannot however be excluded that some of our chronic patients had a positive BS before developing a motor neuropathy or transsynaptic neuronal degeneration.

Interestingly, there seems to be only a weak relationship between the BS and tendon tap reflex excitability. Consequently the BS and exaggerated tendon reflexes are not necessarily conjoint signs of a pyramidal tract lesion [18]. Correspondingly, a stronger positive relationship between the occurrence of a BS and increased muscle tone is in line with the observation of a discrepancy between clinical signs of spasticity and spastic movement disorders [5]. This may be attributed to the different roles of reflexes in passive and active movement conditions. In line with this, it is known that recovery from spinal shock is characterised by mono- and poly-synaptic reflex activity reappearing at different time points after SCI [9, 12]. This indicates that different mechanisms are responsible for either phenomenon, while pathophysiologically, both reflex activity and muscle tone are part of the spastic syndrome [4].

In conclusion, there exists an association of the BS and increased muscle tone, which may depend on a combination of altered spinal interneuronal activity and changes in muscle mechanics [5]. On the one hand, this can also be suggested since the BS disappears after the intrathecal application of Baclofen. By this route of administration, Baclofen concentrations at the spinal level reach four times the local concentration reached in the spinal cord after a typical oral dose [10]. On the other hand, the presence of peripheral neuropathy will also contribute to decreased muscle tone and thus can explain the interrelation of the occurrence of a BS and the level of muscle tone.

Our findings support the notion that there is no relationship between a particular anatomical lesion site in the cortico-spinal tract and the presence of a BS [17]. Roughly half of the patients with complete SCI in our study did not show a BS or increased muscle tone. This was the case even though a damaged pyramidal tract was assumed to exist in the SCI subjects according to clinical and electrophysiological data. Therefore, it is concluded that the presence of a BS in SCI subjects does not primarily depend on the damage of a specific spinal level or on the pathways projecting onto the interneuronal zone of the lumbosacral cord [18] or of direct cortico-spinal projections [8, 13]. Furthermore, it must be assumed that a minimal muscle tone is required to allow for the presence of a BS.

References

1. Babinski J (1898) Du phénomène des orteils et de sa valeur semiologique. Sem Med 18:321–322
2. Babinski J (1896) Sur le reflexe cutané plantaire dans certaines affections organiques du système nerveux central. R Soc Biol 48:207–208
3. Curt A, Dietz V (1999) Electrophysiological recordings in patients with spinal cord injury: significance for predicting outcome. Spinal Cord 37:157–165
4. Dietz V (2000) Spastic movement disorder. Spinal Cord 38: 389–393

5. Dietz V, Sinkjaer T (2007) Spastic movement disorder: impaired reflex function and altered muscle mechanics. Lancet Neurol 6:725–733
6. Ditunno JF, Little JW, Tessler A, Burns AS (2004) Spinal shock revisited: a four-phase model. Spinal Cord 42:383–395
7. Dohrmann GJ, Nowack WJ (1973) The upgoing great toe. Optimal method of elicitation. Lancet 1:339–341
8. Gijn JV (1975) Babinski response: stimulus and effector. J Neurol Neurosurg Psychiatry 38:180–186
9. Hiersemenzel LP, Curt A, Dietz V (2000) From spinal shock to spasticity: neuronal adaptations to a spinal cord injury. Neurology 54:1574–1582
10. Kirshblum S (1999) Treatment alternatives for spinal cord injury related spasticity. J Spinal Cord Med 22:199–217
11. Kirshblum S, Lim S, Garstang S, Millis S (2001) Electrodiagnostic changes of the lower limbs in subjects with chronic complete cervical spinal cord injury. Arch Phys Med Rehabil 82:604–607
12. Ko HY, Ditunno JF Jr, Graziani V, Little JW (1999) The pattern of reflex recovery during spinal shock. Spinal Cord 37:402–409
13. Landau WM, Clare MH (1959) The plantar reflex in man, with special reference to some conditions where the extensor response is unexpectedly absent. Brain 82:321–355
14. Lin CS, Macefield VG, Elam M, Wallin BG, Engel S, Kiernan MC (2007) Axonal changes in spinal cord injured patients distal to the site of injury. Brain 130:985–994
15. Lundberg A (1979) Multisensory control of spinal reflex pathways. Progress in brain research 50:11–28
16. Milanov IG (1992) Mechanisms of baclofen action on spasticity. Acta Neurol Scand 85:305–310
17. Nathan PW, Smith MC (1955) The Babinski response: a review and new observations. J Neurol Neurosurg Psychiatry 18:250–259
18. Van Gijn J (1978) The Babinski sign and the pyramidal syndrome. J Neurol Neurosurg Psychiatry 41:865–873
19. Van Gijn J (1996) The Babinski sign: the first hundred years. J Neurol 243:675–683