increased vitamin B6 could inhibit firing activities in the nigrostriatal dopaminergic neurons, resulting in pain [10]. Another putative etiopathogenic mechanism could involve homocysteine, a known risk factor for idiopathic painful conditions such as migraine, recently identified as a glutamatergic-sensitizing factor of trigeminal neurons and satellite glial cells. Furthermore, homocysteine metabolism leads to production of metabolites (homocysteic acid and cysteine sulfinic acid) with known neurotoxic effects on dopaminergic neurons [11]. Other studies suggest that genetic singularities may alter the link between serum levels of homocysteine, vitamin B6, catecholaminergic, and noncatecholaminergic neurotransmitter metabolism, leading to depression, a frequent context of iBMS development.

Further investigations are needed to better understand the implications of vitamin B6 serum level increases in the physiopathology of iBMS. It should also be kept in mind that iBMS is not a homogeneous nosological entity and that vitamin B6 level alterations might therefore be involved only in a specific subgroup of iBMS patients.

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LETTER TO THE EDITOR

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Recovery of H-Reflex with Transforaminal Epidural Steroid Injection in S1 Radiculopathy

Dear Editor,

Transforaminal epidural steroid injection (TFESI) is a recognized nonsurgical management of radicular back pain through its effects on neural inflammation and edema [1]. Electrodiagnostic changes of the compressed nerve root such as Hoffmann reflex (H-reflex) are apparent within days following injury and persist throughout nerve compression [2], with sensitivity and specificity of 50% and 91%, respectively, making the detection of H-reflex abnormality an option to identify early radiculopathy [3]. Despite its prevalent diagnostic use, to our knowledge there is limited information in the current literature regarding changes of an abnormal H-reflex following treatment of radiculopathy. We have investigated the changes of the H-reflex profile following fluoroscopy-

Table 1 Clinical, functional, and H-reflex parameters of patients before and 1 h, 3 wk, and 3 mo after transforaminal epidural steroid injection

	Clinical parameters	Pre-procedure	1st h	3rd wk	3rd mo
Patient I (right side affected)	NRS	7	2	1	3
	ODI	34	–	20	26
	H-reflex amplitude (μV) (right)	1.1	–	1.2	2.3
Patient II (right side affected)	NRS	4	1	3	2
	ODI	20	–	12	6
	H-reflex amplitude (μv) (right)	0.8	–	2.5	15.9
Patient III (right side affected)	NRS	9	3	0	2
	ODI	38	–	28	26
	H-reflex amplitude (μv) (right)	3.5	–	8.3	5

NRS = numerical rating scale for pain (minimum score = 0; maximum score = 10); ODI = Oswestry Disability Index (minimum score = 0; maximum score = 100).

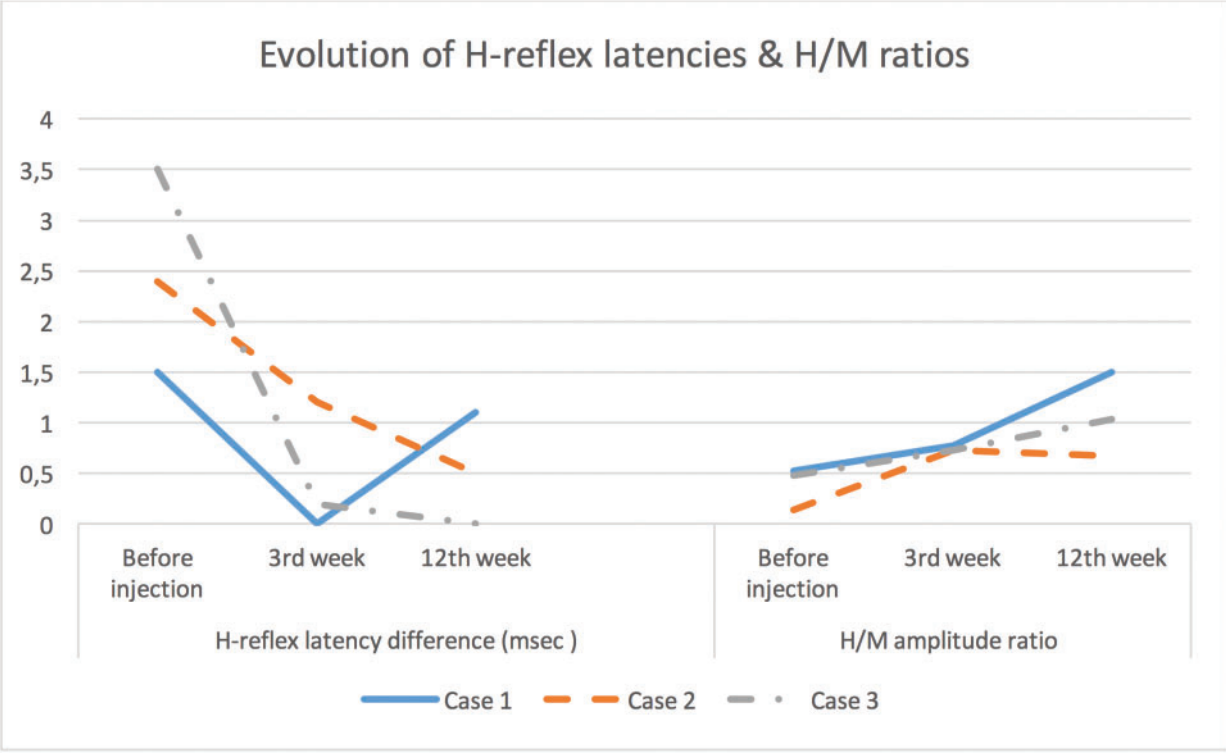

Figure 1 Evolution of the H reflex latencies and H/M ratios in subjects receiving S1 TFESI before, at 3rd week, and 3rd month after procedure.

guided epidural steroid injections in three patients with S1 radiculopathy secondary to disc herniation.

All our subjects presented with low back pain and lower limb radiation, abnormalities of at least one component of neurological assessment, positive herniation of intradiscal substance on lumbar MRI, H-reflex abnormalities in pre-TFESI electrodiagnostic test, and an unsuccessful trial of conservative management for their presenting symptoms. H-reflex recording was done from bilateral

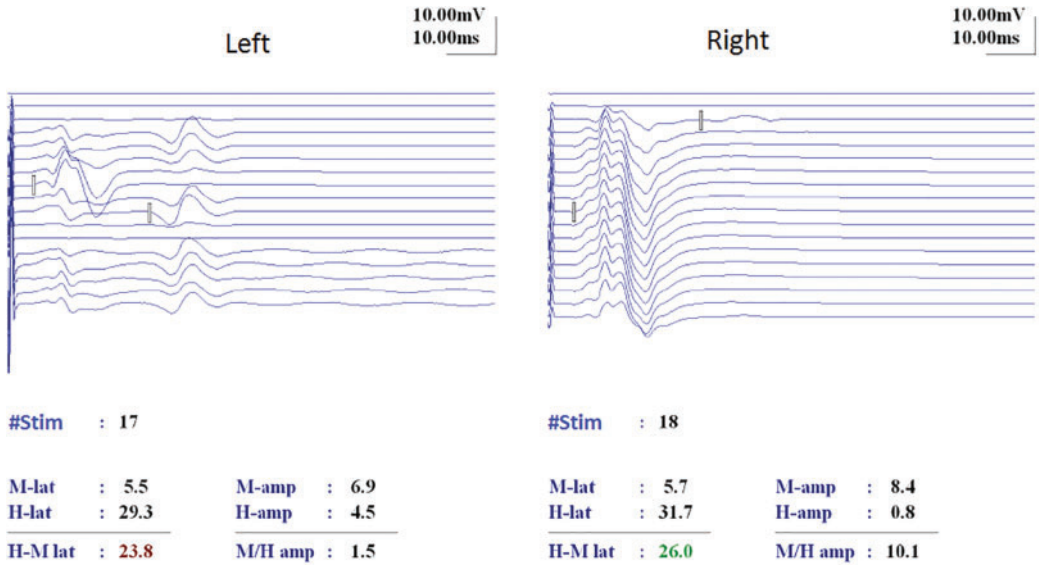

Figure 2 A sample of H-reflex recording for case 2 before TFESI procedure.

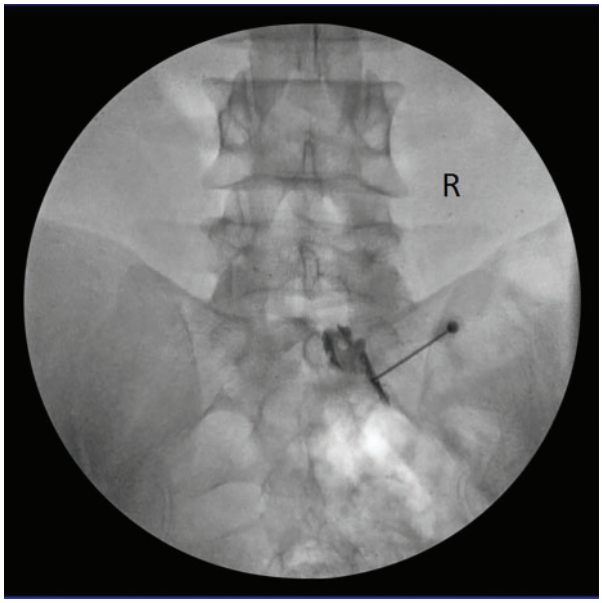

Figure 3 Transforaminal epidural injection of contrast along epidural S1 nerve root sleeve to confirm needle placement prior to epidural steroid and local anaesthetic injection.

soleus and gastrocnemius muscles using 1.0-msec uniphasic pulses to the tibial nerve applied through circular electrodes. Peak-to-peak amplitudes of the motor responses (maximum H response) measured in microvolts (μV) and latencies were recorded in milliseconds (msec). Latency differences of more than 1.5 msec between a lower limb pair [4] and an H-reflex amplitude of less than 1 mV were considered abnormal. We believe that the ratio between maximal H-reflex and maximum muscle (M) response (H/M ratio) is a more appropriate measurement given individual variations of normal H-reflex amplitude [5], and we postulate that it would be a more

reliable measurement of H-reflex change during postprocedure follow-up. S1 TFESI approached via the L5-S1 neural foramina with 80 mg methylprednisolone, 1 cc 0.5% bupivacaine, and saline were administered in a single injection under fluoroscopic guidance for symptomatic relief of pain due to unilateral S1 root compression. Subjects were subsequently reassessed clinically and functionally at one hour, three weeks, and three months postprocedure.

Table 1 summarizes the clinical and functional assessments prior to and following the TFESI. Pain score reduction of 50% or more and significant improvements of functional parameters were observed at three months post-TFESI in all subjects. Figure 1 illustrates the H-reflex evolution following TFESI. Interextremity H-reflex latency difference was $\geq$ 1.5 ms in all subjects before TFESI; all subjects demonstrated reduction of the latency difference and increase in H/M ratios after TFESI. The reduction in H-reflex latency and the increase in H amplitude at the affected side post-TFESI were consistent with improved clinical parameters and persisted at three-month follow-up.

In contrast to the general view asserting that H-reflex cannot recover once impaired, Stretanski has reported changes in H-reflex following treatment [4]. Chronic neural demyelination and axonal injury were considered the probable reason of prolonged H-reflex latency or marked interlimb H-reflex latencies differences as seen in our subjects [5]. Significant regression of increased latency difference and increasing bilateral H/M ratios parallel H-reflex amplitude increase after TFESI. They suggest that epidural steroid injection suppresses early and delayed inflammatory processes around the nerve root. This leads to regression of axonal compression and improvement in demyelination-induced conduction delay within the H-reflex spinal pathway [6]. A clear relationship between recovery of H-reflex and improvement in ODI was not observed.

In a study of H-reflex latency before and 10 minutes after interlaminar epidural steroid injection in 10 patients, Stretanski found changes in H-reflex postprocedure similar to our case series; however, there are some fundamental differences between the two studies [4]. First, the transforaminal approach is known to be more effective in treating radiculopathy. We believe a longer follow-up duration of three months increases the reliability of our results. In this case series, there are some limitations including lack of control group and unknown effect of spontaneous recovery.

In conclusion, this series discussed the H-reflex evolution following TFESI and the possible pathophysiology underpinning these changes. Our observations suggest that H-reflex is not only diagnostic, it may be an important treatment outcome measure. To our knowledge, there is no literature to date examining the pattern of H-reflex changes over an extended duration after TFESI. We acknowledge that there remain many unanswered questions relating to the therapeutic mechanisms of epidural steroid treatment; however, our study sheds new insights to the therapeutic uses of H-reflex, its changes following targeted treatment of neural inflammation, and H-reflex evolution as evidence of TFESI therapeutic effect in radiculopathy.

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