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CASE REPORT

Epidural Stimulation of the Lumbosacral Spinal Cord After Basal Ganglia Haemorrhage: A Case Study

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ABSTRACT

Lumbar epidural spinal cord stimulation (SCS) was used to improve motor function in a 50-yr old patient who suffered hemiparesis due to a basal ganglia haemorrhagic stroke. Spinal cord stimulation targeted at the dorsal root afferent fibres at the conus improved the tonic control of the muscles at the knee and ankle joints. This allowed the patient better left knee and foot motor control. The improvement was documented initially during ambulation on a treadmill using decreasing body weight support and subsequently when using walking aids. Our observation is consistent with previous human data suggesting that in humans with brain lesions, the stimulation of preserved neural circuitry can increase spontaneous muscle tone in affected muscles and improve locomotion.

Introduction

Intracerebral haemorrhage (ICH) accounts for 20-30% of stroke incidence in the Asian population.[1-4] Functional outcome in survivors is often poor with less than 20% achieving independence at six months.[5] Compared with haemorrhage in other parts of the brain, ICH in the basal ganglia or internal capsule is associated with a higher survival rate.[1] Good functional outcome is usually associated with a hematoma volume of less than 30ml .

The basal ganglia have an essential role in the organization (coordination, timing, and sequencing) of normal motor output and motor learning processes. One of the most common impairments caused by stroke is hemiparesis.[6,7] Notably, the failure to recover motor deficits within the early months after a stroke reduces the effectiveness of rehabilitative therapy and subsequent physical independence.[2,3] The insult to the CNS is followed by neuronal reorganisation which then halts motor recovery. Nevertheless, rehabilitative treatment applying various sensory stimuli has shown benefits in improving early post-stroke lower-extremity impairment and late motor function recovery.[7-10] The mechanism involved is still unclear. Increased cerebral blood flow following cervical epidural stimulation[11] and spinal cord stimulation (SCS)-related inhibition of pathological movement patterns have been suggested.[10]

We report on the beneficial effect of lumbar spinal cord stimulation on the lower limb motor function in a hemiparetic patient following a basal ganglia haemorrhagic stroke.

Clinical case description

Clinical characteristics prior to spinal cord stimulation

A 50-year old female with history of hypertension sustained a large right sided basal ganglia haemorrhage in the external capsule (see Figure 1). She developed left hemiplegia with marked hemisensory loss to pinprick. The motor deficit improved marginally over the course of 6 months but hemisensory loss remained unchanged.

Prior to spinal cord treatment (at 16 months post-haemorrhage), her neurological deficit was 4 on the modified Rankin scale (mRS)[12]. She had weak motor function of left side but anal sphincter and bladder contraction were intact. Sensation was abnormal below C5 on the left with areas of patchy sensory loss and dysesthesia, especially in the left chest, trunk and abdominal regions. Sensory loss to pinprick in her left leg was dense up to T12 dermatome. There was complete loss of proprioception in both her left upper and lower limbs.

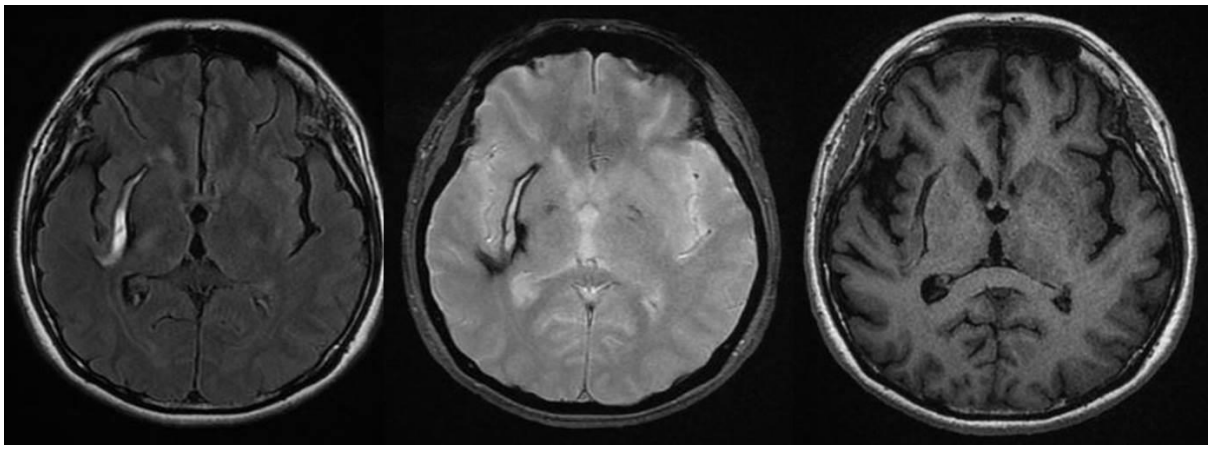

Figure 1. MRI Brain at 6 months post-haemorrhage

Prickling neuropathic pain started in the areas of paraesthesia from about 3-4 weeks after the haemorrhahe, and evolved to severe (7/10 on a numerical rating scale – NRS) burning pain in her left arm and leg. Severe paroxysmal episodes (NRS 8) resulted in difficulty sleeping at night. She was started on Gabapentin 600mg/day which had to be increased to 1500mg /day over the next 12 months resulting in marked drowsiness, yet poor pain control (NER 6/10) despite the addition of Tramadol 400 mg/day.

Motor weakness had improved significantly such that most muscle groups in her left leg were of power 4/5. Her quadriceps (L3-4) and peroneus (L5-S1) were however weak with power 3/5. This resulted in her knee buckling and ankle inverting during attempts to ambulate.

The patient received more than 50 locomotor training sessions over a period of 6 months using body weight support (25-50%) and robotic gait orthosis on a treadmill with manual facilitation, with no objective improvement in left leg control.

The patient signed informed consent for electrode implantation, on-table stimulation, and physiological monitoring studies at 16 months post-haemorrhage.

The patient was reviewed at 1, 3, 6, 8, 16, 24, 36 and 52-week intervals post-SCS treatment. Motor stimulation was only started after 6 weeks when implanted leads have stabilised in location.

Rehabilitation physiotherapy was resumed with motor epidural stimulation cycled at a frequency of 3 seconds "on" and 3 seconds "off".

Procedure and methods

Percutaneous electrodes and stimulator insertion

An epidural spinal cord stimulation system (RestoreSensor SureScan™, Medtronics) was used to stimulate the lumbosacral enlargement. Two percutaneous 8-contact electrodes (Vectris SureScan™, Medtronics) were implanted under fluoroscopic guidance (see Figure 2). Precise positioning of the electrodes was obtained using sensory mapping at 40-50Hz and EMG was recorded from left leg muscles at a frequency of 25 Hz. A lateral lead placement allowed the generation of paraesthesia in the locations of neuropathic pain while the more medial lead produced the EMG signals needed to activate the rectus femoris, the vastus lateralis, and the tibialis anterior. The medial lead was mapped with EMG to isolate the quadriceps (0+ 1- at 3.45V PW 180ms Frequency 25Hz) (see Figure 3) and the lateral calf muscles (5- 6+ at 4.0V PW 150ms Frequency 25Hz) (see Figure 4). Both leads were then tunneled to a subcutaneous left upper buttock pouch where the pulse generator was implanted.

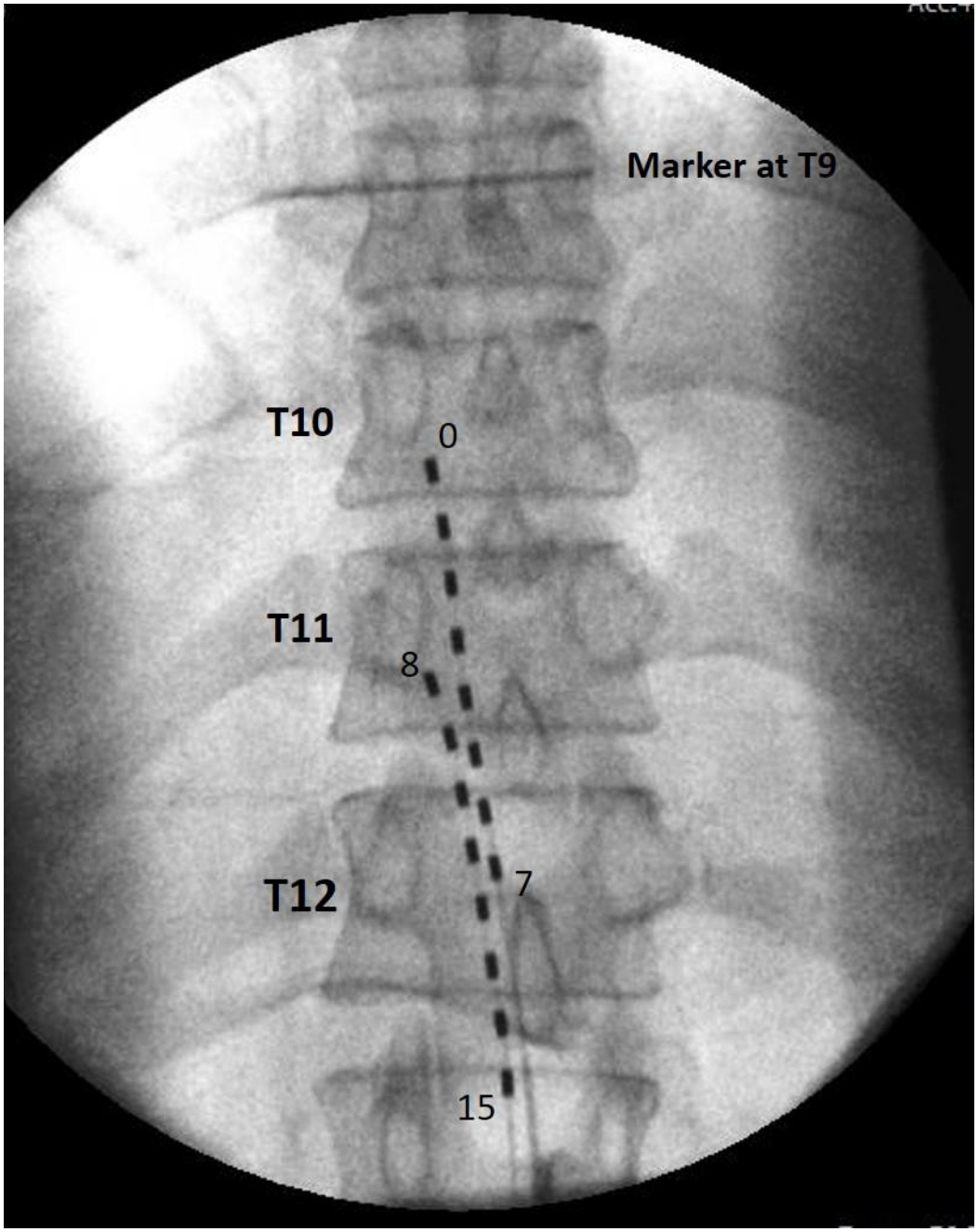

Figure 2. Fluoroscopic placement of left sided epidural site leads at T11-12

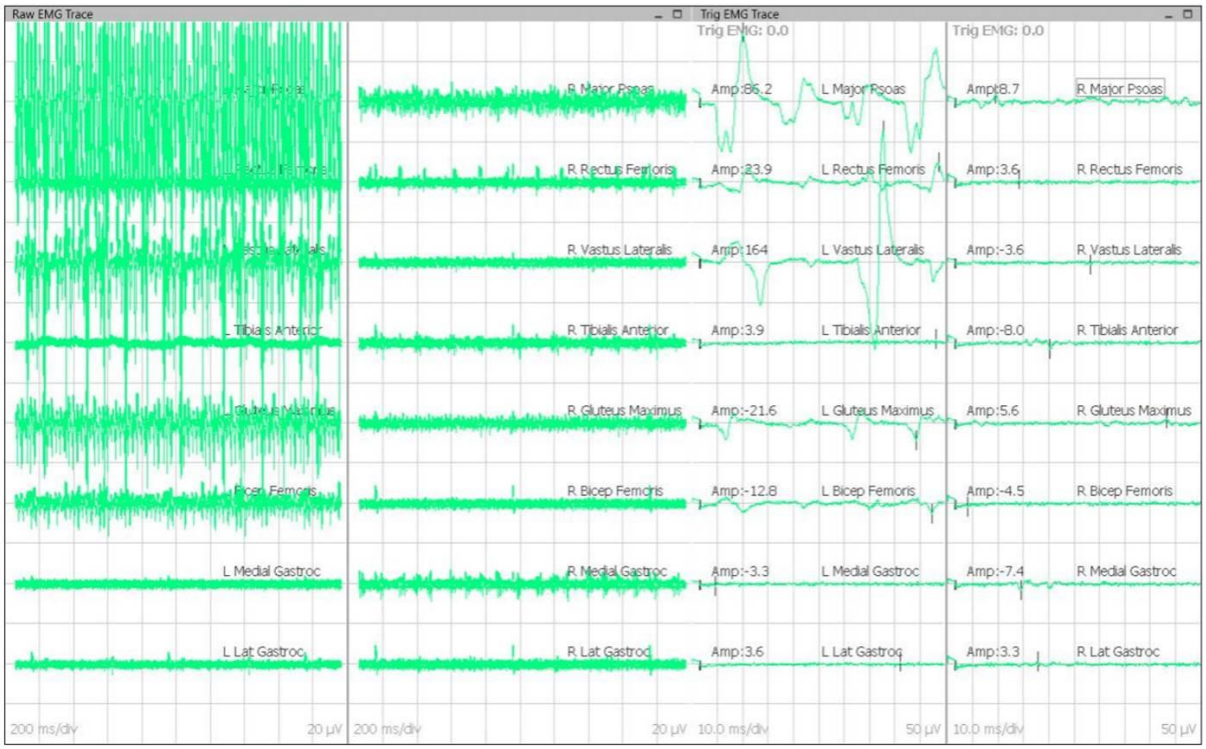

Figure 3. Intraoperative EMG mapping isolating the quadriceps (0+ 1- at 3.45V PW 180ms Frequency 25Hz)

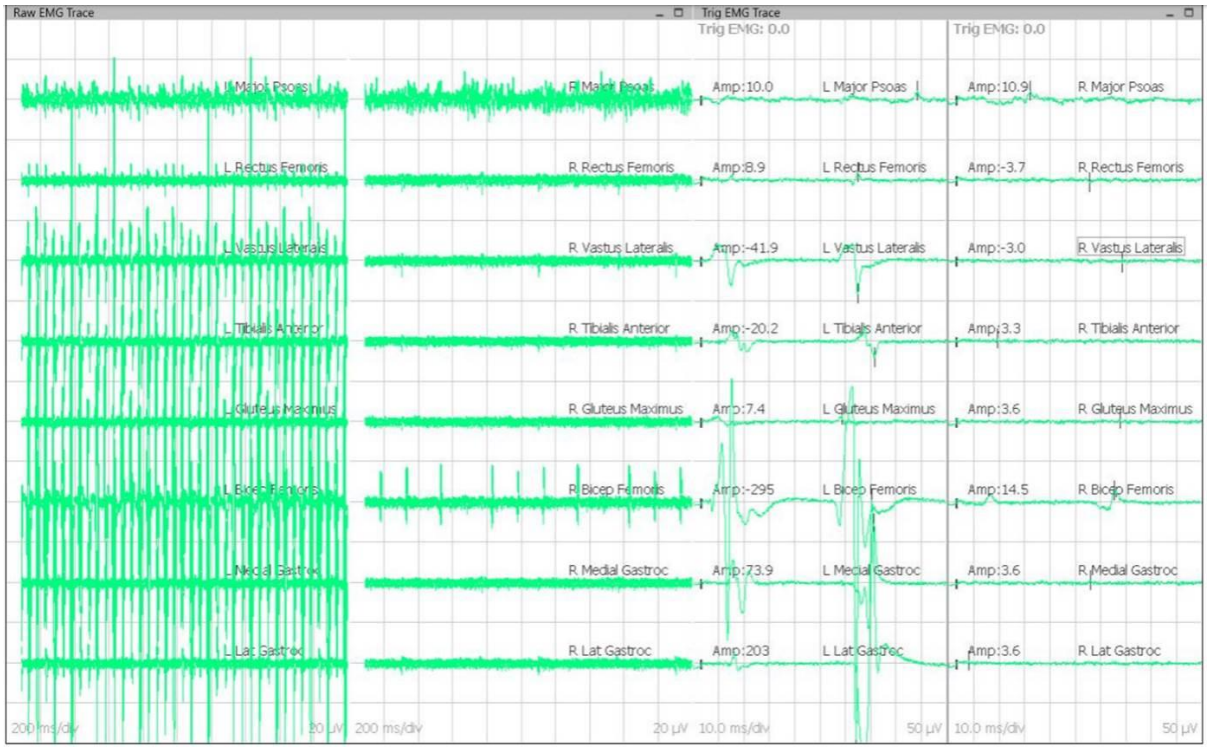

Figure 4. Intraoperative EMG mapping isolating the lateral calf muscles (5- 6+ at 4.0V PW 150ms Frequency 25Hz)

Data Acquisition

Bilateral EMG (NIM Eclipse® E4, Medtronic) from the medial and lateral gastrocnemius, tibialis anterior, biceps femoris, rectus femoris, vastus lateralis, and gluteus maximus muscles was recorded using pairs of intradermal needle electrodes inserted 2 cm apart.[13] Bilateral EMG from the iliopsoas was recorded with surface electrodes.

Results

Pain control

Paraesthesia was perceived from the left hip to the thigh and down to the calf. This resulted in significant relief from her burning neuropathic

pain. By week 8 post-SCS, the medication was reduced to Gabapentin 300mg and Tramadol 37.5mg per day.

Motor stimulation

In contrast to her marked preoperative quadriceps and tibialis anterior weaknesses, the patient had increased tone in these muscles and was able to ambulate effectively 6 weeks post-SCS at her first ambulation training session. Using appropriate contact setting, pulse width and intensity, the motor stimulation was then set at 20Hz frequency cycled 3 seconds "on" and 3 seconds "off". This corresponds to a walking pace of 0.5 km/h. By the 3rd session 7 days later, no body weight support or lower limb orthosis was needed. The patient managed an ambulation duration of between 34- 42 minutes after the 3rd session. There was significantly increased muscle tone with little or no incidence of knee buckling or ankle inversion before 30min on the treadmill. However, when epidural motor stimulation was turned off or when sensory stimulation was turned on, the knee would still buckle and the left foot would still invert during ambulation. From week 12 onwards, the patient was ambulating with a quad-stick and at faster speeds. However, due to the absence of cutaneous and proprioceptive sensory inputs, she needs to look carefully before she takes each step.

Discussion

In patients with spinal cord injury, continuous epidural spinal cord stimulation enables static activity such as standing. However for effective walking, additional proprioceptive information from the hip, the knee and the ankle is required.[14,15] Dynamic changes during ambulation must be accompanied by motor patterns to coordinate joint positions.[16,17] Continuous epidural stimulation has been shown to only facilitate locomotion within a narrow range of stimulation parameters and is unable to provide meaningful locomotor improvements in humans without rehabilitation.[14,15] This might be because continuous epidural stimulation blocks the propagation of naturally generated proprioceptive signals to the brain and spinal cord.[14] With that in mind, we arbitrarily used the 3 seconds "on" and 3 seconds "off" cycling of the targeted motor stimulation parameters to aim at engaging motor pool activity as well as avoiding blocking proprioceptive signals to the sensorimotor cortex. As direct motor stimulation was not the intention, we suggest that epidural stimulation enabled ambulation by stimulating afferent fibres in the dorsal root and subsequently recruiting populations of interneurons that integrated ambulation related proprioceptive input to coordinate motor pool activity. This observation is consistent with the possibility that recruitment of proprioceptive pathways with cycling epidural stimulation modulates cortical excitability, facilitating locomotion in the process.[18]

There are still unanswered research questions such the variability of motor stimulation in conjunction with associated exercise protocols (passive versus active). The optimal motor stimulation dosage and specific clinical measurement parameters documenting specific deficits and treatment parameters focused on augmenting motor learning and motor function[7] are endpoints that will need better alignment in future prospective studies.

With conventional sensory epidural stimulation, the patient had near complete relief of her neuropathic pain. She also reported improvement in temperature regulation of her left leg. The relatively young age and the non-thalamic site of her stroke were good predictive factors of epidural stimulation providing effective relief from her central post-stroke pain.[19]

At 12 months post-implant, there was still no improvement in proprioception in this patient, even with motor stimulation turned on. Clinical studies reports suggest that preservation of proprioceptive information is key to recovery after neurotrauma[20], as it supposedly steers the reorganization of residual descending pathways to restoring ambulation.[14] It is not known if the continuous epidural stimulation targeted at the dorsal horn islets may in some way block the proprioceptive recovery achieved during motor stimulation and rehabilitative efforts.

Conclusion

Our observations support the proof of concept that humans with brain injuries may have conserved cortical circuitry as found in other mammals and previous published case series. Stimulation of this circuitry can lead to increased spontaneous muscle tone and improved locomotion.

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Competing interests

Nil

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