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Changing spinal cord circuts excitability can lead to regain cord function below the level of the lesion

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Abstract: *The spinal cord injury is characterized by loss or degradation of motor and sensory and autonomic functions and necessities life long treatment and care. The present study was conducted in the Imam Zain Alabiden hospital during 2018. Around 9 patients (6 males and 3 females) with more than 2 years of SCI history were enrolled in the present study. Informed consents were taken from the patients (n=9) before operative procedure. About 5 patients were ASIA grade A and remaining were ASIA grade B.. Male (79%) showed maximum SCI as compared to females (21%). Spinal cord injuries (SCI) other caused by trauma includes motor vehicle accident, acts of violence, fall from heigh and sports. Motor vehicle accidents showed maximum cause of traumatic SCI occurrence, followed by acts of violence and fall from height. The stimulation procedure was done successfully for all patients. Patients reported to have noxious feeling of electricity felt during stimulation. About 6 patients had a feeling of fatigability after stimulation. The motor lower extremity for LT and RT were found to be zero before and after stimulation. Sensory score (T10-S5, score) was found to be zero before stimulation except one patient. However, after stimulation all patients showed significant improvement in the sensory score (Table 1). Anal sensation was absent in the all the patients except two patients. The post injury period was ranging from 2 to 4 years. After surgical procedure, almost all patients showed improvement in the activities. They started sitting without support; twisting and rolling in bed without support. Only one patient showed sensation of errection and orgasm. After completion of the procedure the motor and sensory score is increased in all patients with significant clinical findings regained.*

Keywords: *Spinal cord injury, Sensory score, Surgery, Motar neurons, Stimulation*

Introduction

The spinal cord injury (SCI) is characterized by loss or degradation of motor, sensory and autonomic functions (Alizadeh et al., 2019). It is an enfeebling neurological condition with incredible socioeconomic impact on affected individuals and the health care system (Hachem et al., 2017). SCI requires a lifelong treatment and care costs besides loss of income, and social and psychological problems (Tator, 1998; Dumont et al., 2001). Around more than 50% SCI patients cannot return back to their normal life (Li et al., 2000). A report published by National Spinal Cord Injury Statistical Center showed around 12,500 new SCI cases were reported each year in the North America (Hachem et al., 2017). In Iraq, about 61.2% patients were admitted for SCI in 2012. Out of this round 84.4% patients were males and 15.6% were female patients. More than 90% SCI cases were due to violence, traffic accidents, falls or sports (WHO, 2013). SCI occurs in the adult than the children and male-to-female ratio was reported to 2:1 (WHO, 2013). Generally, males were affected during the age of 30-80 years while females were affected during the 15-19 years or after 70 years age (Middleton et al., 2012; Stein et al., 2015). Adults more than 60 years age showed the worst condition during SCI (Stein et al., 2015).

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A traumatic impact on the spine with dislocates or fractures in the vertebrae are referred to as SCI. At the time of injury, early mechanical forces delivered to the spinal cord are known as 'primary injury'. It can be characterized by disc materials, displaced bone fragments, ligaments tear or bruise of the spinal cord tissue, etc. (Dumont et al., 2001; Sekhon and Fehlings, 2001; Oyinbo, 2011). However, the SCI has characteristic mechanisms of primary injury, *i.e.* impact with transient compression, impact plus persistent compression, laceration/transection and distraction (Dumont et al., 2001; Rowland et al., 2008; Choo et al., 2009; Fehlings et al., 2012).

The "completeness" is a measure of the injury severity. It is further divided into two catagories *viz*. complete completeness (sensory and motor function are lost) and incomplete completeness (some motor or sensory function are observed). The International Standards for Neurological Classification of Spinal Cord Injury (ISNCSCI) is a universal classification tool for Spinal Cord Injury based on a standardized sensory and motor assessment and developed by American Spinal Injury Association (ASIA). These scale involves both sensory and motor examination to determine neurological injury level and / or completeness (Burns et al., 2011). SCI trauma includes sports and leisure injuries, road traffic accident and / or work-related accidents, beaten and falls at home (Tator, 1998). All these situations are more common in the men than the females. Especially, it was most prevalence in male adults and in young adults. Many authors reported about the SCI occurred during the war (Frances et al., 2009; Schoenfeld et al., 2013).

The crosstalk between the spinal interneuronal networks and their involvement in the movement were studied comprehensively in animals (Jankowska, 2001, 2016a,b; Kiehn, 2016; Côté et al., 2018). However, the fewer reports were available about the implementation of these neurophysiological findings in the human studies (Jankowska and Hammar, 2002; Côté et al., 2018). In the present study, we aim to prove that changing spinal cord circuts excitability can lead to regain cord function below the level of the lesion regardless the period and the pathology of the spinal cord injury.

Material and methods

The present study was conducted in the Imam Zain Alabiden hospital during 2018. Around 9 patients (6 males and 3 females) with more than 2 years of SCI history were enrolled in the present study. Informed consents were taken from the patients (n=9) before operative procedure. About 5 patients were ASIA grade A and remaining were ASIA grade B. The SCI cause was traumatic, shell injury and inflammatory in the 4, 4 and 1 patients, respectively. Eight patients were operated by fixation on site of the lesion.

Operative procedure

The patients were anesthetized locally before surgical operation. Under fluoroscopic guidance, the epidural stimulation catheter inserted through the caudal space and advanced up to the site of injury with steering of the catheter in most accessible entry point in the epidural space. Motor stimulation response was set to 2Hz stimulation for confirmation the desired level.

Stimulation procedure done with 5 bursts. Each burst was 5Hz with manual increment in voltage to the highest tolerable level. It was continued for 2 mints. About 1 min rest was taken between successive bursts. Another, 5 bursts of 200Hz were given to the patient. Each burst last for 1 second and voltage increased up to the highest tolerable level.

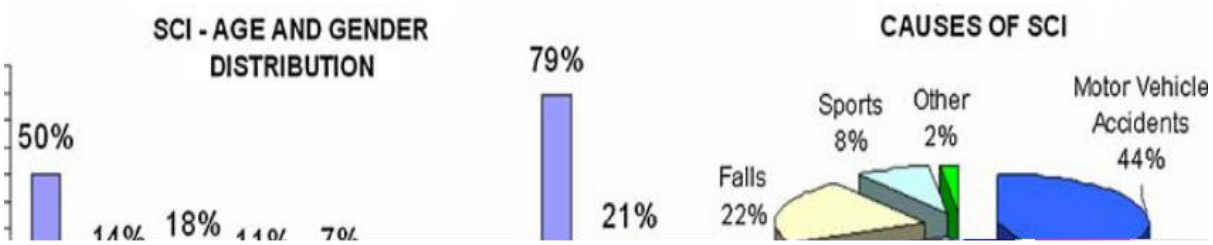
Study parameters

The patients were followup for further __six weeks after surgery. Sensory score (T10-S5, score); sitting without support ability; twisting and rolling in bed ability; sensation of errection and orgasm were evaluated.

Results

In the present study, spinal cord injuries were observed maximum in the 15-35 years age. However, 64 and above year age group showed minimum SCI. Male (79%) showed maximum SCI as compared to females (21%). Spinal cord injuries (SCI) other than trauma includes tumors spinal vascular diseases inflammatory diseases spinal stenosis surgical intervention, radiation myelopathy, neural tube defects and other causes. Motor vehicle accidents showed maximum cause of SCI occurrence, followed by acts of violence and fall from height. Age and gender vise SCI distribution and its causes are depicted in the Figure 1.

Figure 1. Spinal cord injuries (SCI) other than trauma include



The stimulation procedure was done successfully for all patients. Patients reported to have noxious feeling of electricity felt during stimulation. About 6 patients had a feeling of fatigability after stimulation.

The motor lower extremity for LT and RT were found to be zero before and after stimulation. Sensory score (T10-S5, score) was found to be zero before stimulation except one patient. However, after stimulation all patients showed significant improvement in the sensory score (Table 1). Anal sensation as absent in the all the patients except two patients. The post injury period was ranging from 2 to 4 years.

Table 1. Clinical characteristics of patients before stimulation

Age (year)	Gender	Post injury	Neurological level	ASIA Grade	Anal sensation	Stimulation	Sensory score (T10-S5, score)			
							LT(rt)	PP(rt)	LT(lt)	PP(lt)
22	Male	2.2	D4	A	0	Before	0	0	0	0
						After	6	4	4	4
29	Male	3	D6	A	0	Before	0	0	0	0
						After	4	2	4	2
26	Male	2.5	D6	B	Altered	Before	12	8	15	10
						After	14	14	15	12
30	Male	2.6	D7	A	0	Before	0	0	0	0
						After	6	10	10	8
34	Male	4	D8	B	0	Before	10	9	6	3
						After	18	16	10	12
32	Male	3.2	D5	A	0	Before	0	0	0	0
						After	10	10	12	10
30	Female	2.4	D4	B	Altered	Before	12	7	10	7
						After	18	16	16	10
32	Female	2.9	D3	A	0	Before	0	0	0	0
						After	12	8	8	7
34	Female	2	D2	B	0	Before	8	8	10	10
						After	16	14	16	12

After surgical procedure, almost all patients showed improvement in the activity. They started sitting without support; twisting and rolling in bed without support. Only one patient showed sensation of errection and orgasm (Table 2).

Table 2. Significant clinical finding of patients after stimulation

Age (year)	Gender	Post injury	Sitting without support	Twisting & rolling in bed without support	Sensation of errection	Orgasm
22	Male	2.2	+ / -	+ / -	-	-
29	Male	3.0	+	+	-	-
26	Male	2.5	+	+	+	+
30	Male	2.6	+	+	-	-
34	Male	4.0	+	+	-	-
32	Male	3.2	+	+	-	-
30	Female	2.4	+	+	-	-
32	Female	2.9	+ / -	+	-	-
34	Female	2.0	+	+	-	-

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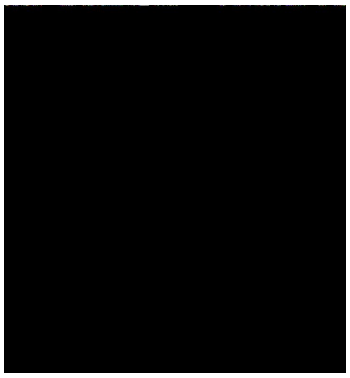
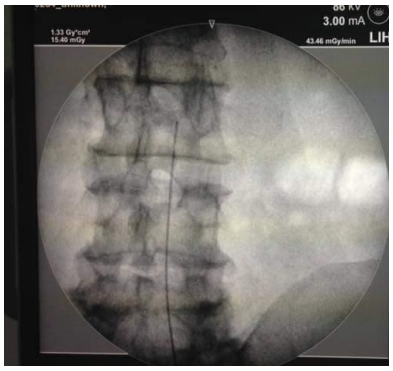
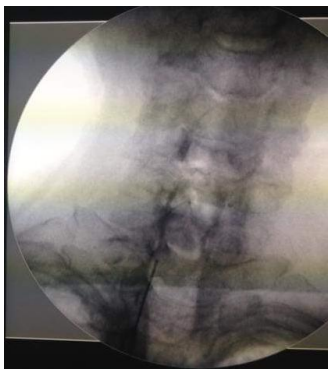
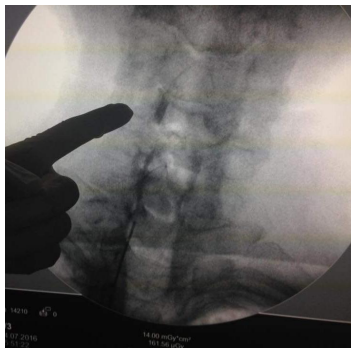
a.  b. 

c. 

d. 

a)Please write the specification about this figures (a)_epiduralcatheter in the the lumber and thorasic vertebrae

b) epidural stimulating catheter in the lumber vertebrae _____

(c) _contrast is ingected through epidural space in cervical vertebrae

(d) __ontrast in the epidural space in the cervical vertebrae_____

Discussion

Recently, spinal cord stimulation is becoming popular and choose of treatment for the spinal cord injury (Karacan et al., 2000; Pagliacci et al., 2003; Citterio et al., 2004). Electrical stimulation (ES) of the posterior spinal cord (dorsal column stimulation; DCS) is a crucial therapeutic tool for chronic pain conditions treatment. ES generate action potential which travels through both anterograde pathway (towards the neuromuscular junction) and retrograde pathway (towards the anterior horn cell). The single nerve processes react within minutes of exposure to an applied electric field. The growing nerve fibres respond immediately to the voltage gradient and tend to orient themselves parallel with the long axis of this gradient (Jaffe and Poo, 1979; Cosman and Cosman, 2005). The tissue near the active tip of an electrode get exposed to high electric fields (E-fields) due to radio frequency (Sluijter et al., 1998) leads to the strong ionic force generation and eventually electrolyte shifting (Jaffe and Poo, 1979; Cosman and Cosman, 2005)

Cowley et al. (2010) reported about 27% success can be achieved with a locomotor command to the lumbrosacral spinal cord transmission in the absence of long direct transmission (corticospinal tract), this transmission done by propriospinal pathways through commissural projections. Our results are according to this report which means the stimulation may facilitate excitation of propriospinal neurons which supports propagation of the voluntary command to the lumbosacral spinal cord, beside that the anatomical presentation of extrapyramidal tracts and anterior corticospinal tract in antromedial side of the spinal cord may spare these tracts or at least less subjected to injury by the trauma or ischemia and this might be the cause for the significant clinical finding that we have regarding sitting or rolling in bed without support, and the balance and good control of the trunk .

Melzack and Wall (1965) proposed the gate control theory. He proposed that the activation of coarse fiber systems suppressed transmission of nociceptive information at the segmental level. Also, he predicted that all types of pain could be suppressed using this mechanism. The theory was supported by many researchers (Todd, 2010; Woolf 2011; Liang and Mendell, 2013; Liebano et al, 2013; Mendell, 2014). After spinal cord injury, anatomical connections may be still continued which was silent because of loss of conduction as a result of disruption of myelin or the ionic channels of the neurons (Waxman, 1989; Fehlings and Nashmi, 1996; Shi and Blight, 1997; Sinha et al., 2006; Coggan et al., 2011; Angeli et al., 2014). Therefore loss of voluntary control of movement, sensations , or autonomic function, may be attributed to not only a physical disruption of descending connections, but also to a physiological alteration of the central state of excitability of the spinal circuitry. (Edgerton et al., 1997; de Leon et al., 1999; Tillakaratne et al., 2002; Angeli et al., 2014). In the present study, after surgical procedure, almost all patients showed improvement in the activity. They started sitting without support; twisting and rolling in bed without support. Only one patient showed sensation of errection and orgasm.

Conclusion

In the present study, we found that the motor lower extremity for LT and RT were found to be zero before and after stimulation. Sensory score (T10-S5, score) was found to be zero before stimulation except one patient. However, after stimulation all patients showed significant improvement in the sensory score. Anal sensation as absent in the all the patients except two patients. The post injury period was ranging from 2 to 4 years. The study can be concluded as the change in the spinal cord circuits excitability can lead to regain cord function below the lesion level. After completion of the procedure the motor and sensory score is increased in all patient with significant clinical findings regained.

References

1. Edgerton VR, de Leon RD, Tillakaratne N, Recktenwald MR, Hodgson JA, Roy RR. Use-dependent plasticity in spinal stepping and standing. Adv Neurol. 1997; 72():233-47.

2. de Leon RD, Tamaki H, Hodgson JA, Roy RR, Edgerton VR. Hindlimb locomotor and postural training modulates glycinergic inhibition in the spinal cord of the adult spinal cat. J Neurophysiol. 1999 Jul; 82(1):359-69.
3. Tillakaratne NJ, de Leon RD, Hoang TX, Roy RR, Edgerton VR, Tobin AJ. Use-dependent modulation of inhibitory capacity in the feline lumbar spinal cord. J Neurosci. 2002 Apr 15; 22(8):3130-43.
4. Coggan JS, Ocker GK, Sejnowski TJ, Prescott SA. Explaining pathological changes in axonal excitability through dynamical analysis of conductance-based models. J Neural Eng. 2011 Dec; 8(6):065002.
5. Waxman SG. Demyelination in spinal cord injury. J Neurol Sci. 1989 Jun; 91(1-2):1-14.
6. Fehlings MG, Nashmi R. Changes in pharmacological sensitivity of the spinal cord to potassium channel blockers following acute spinal cord injury. Brain Res. 1996 Oct 14; 736(1-2):135-45.
7. Shi R, Blight AR. Differential effects of low and high concentrations of 4-aminopyridine on axonal conduction in normal and injured spinal cord. Neuroscience. 1997 Mar; 77(2):553-62.
8. Sinha K, Karimi-Abdolrezaee S, Velumian AA, Fehlings MG. Functional changes in genetically dysmyelinated spinal cord axons of shiverer mice: role of juxtaparanodal Kv1 family K+ channels. J Neurophysiol. 2006 Mar; 95(3):1683-95.
9. Angeli CA, Edgerton VR, Gerasimenko YP, Harkema SJ. Altering spinal cord excitability enables voluntary movements after chronic complete paralysis in humans. Brain. 2014;137(Pt 5):1394-409.
10. Alizadeh A, Dyck SM, Karimi-Abdolrezaee S. Traumatic Spinal Cord Injury: An Overview of Pathophysiology, Models and Acute Injury Mechanisms. Front Neurol. 2019;10:282.
11. Schoenfeld AJ1, Newcomb RL, Pallis MP, Cleveland AW 3rd, Serrano JA, Bader JO, Waterman BR, Belmont PJ Jr. Characterization of spinal injuries sustained by American service members killed in Iraq and Afghanistan: a study of 2,089 instances of spine trauma. J Trauma Acute Care Surg. 2013 Apr;74(4):1112-8.
12. Hachem LD, Ahuja CS, Fehlings MG. Assessment and management of acute spinal cord injury: From point of injury to rehabilitation. J Spinal Cord Med. 2017; 40(6):665-675.
13. WHO WHO | Spinal Cord Injury. WHO, Fact sheet N°384 (2013). Available online at: https://www.who.int/news-room/fact-sheets/detail/spinal-cord-injury [Ref list]
14. Middleton JW, Dayton A, Walsh J, Rutkowski SB, Leong G, Duong S. Life expectancy after spinal cord injury: a 50-year study. Spinal Cord. 2012 Nov; 50(11):803-11.
15. Stein DM, Pineda JA, Roddy V, Knight WA. Emergency Neurological Life Support: Traumatic Spine Injury. Neurocrit Care. 2015 Dec; 23 Suppl 2():S155-64.
16. Frances M. Weaver, Stephen P. Burns, Charlesnika T. Evans, Lauren M. Rapacki, Barry Goldstein, Margaret C. Hammond. Provider Perspectives on Soldiers With New Spinal Cord Injuries Returning From Iraq and Afghanistan. Archives of Physical Medicine and Rehabilitation VOLUME 90, ISSUE 3, P517-521, MARCH 01, 2009,
17. Tator CH. Biology of neurological recovery and functionalrestoration after spinal cord injury. Neurosurgery1998;42:696-707.
18. Dumont RJ, Verma S, Okonkwo DO, et al. Acute spinal cord injury, part II: contemporary pharmacotherapy. Clin Neuropharmacol 2001;24:265-279.
19. Li M, Ona VO, Chen M, et al. Friedlander RM. Functionalrole and therapeutic implications of neuronal caspase-1 and -3 in a mouse model of traumatic spinal cord injury. Neuroscience 2000;99:333-342
20. Burns S, Biering-Sørensen F, Donovan W, Graves D, Jha A, Johansen M, Jones L, Krassioukov A, Kirshblum, Mulcahey MJ, Schmidt Read M, Waring W. International Standards for Neurological Classification of Spinal Cord Injury, Revised 2011. Top Spinal Cord Inj Rehabil 2012;18(1):85-99.
21. Oyinbo CA. Secondary injury mechanisms in traumatic spinal cord injury: a nugget of this multiply cascade. Acta Neurobiol Exp. (2011) 71:281–99.
22. Dumont RJ, Okonkwo DO, Verma S, Hurlbert RJ, Boulos PT, Ellegala DB, et al. . Acute spinal cord injury, part I: pathophysiologic mechanisms. Clin Neuropharmacol. (2001) 24:254–64.
23. Sekhon L, Fehlings M. Epidemiology, demographics, and pathophysiology of acute spinal cord injury. Spine. (2001) 26:S2–12.
24. Rowland JW, Hawryluk GW, Kwon B, Fehlings MG. Current status of acute spinal cord injury pathophysiology and emerging therapies: promise on the horizon. Neurosurg Focus. (2008) 25:E2.
25. Choo AM, Liu J, Liu Z, Dvorak M, Tetzlaff W, Oxland TR. Modeling spinal cord contusion, dislocation, and distraction: characterization of vertebral clamps, injury severities, and node of Ranvier deformations. J Neurosci Methods. (2009) 181:6–17.

26. Fehlings MG, Smith JS, Kopjar B, Arnold PM, Yoon ST, Vaccaro AR, et al. . Perioperative and delayed complications associated with the surgical treatment of cervical spondylotic myelopathy based on 302 patients from the AOSpine North America Cervical Spondylotic Myelopathy Study. J Neurosurg Spine. (2012) 16:425–32.
27. Pagliacci MC, Celani MG, Zampolini M, et al. An Italian survey of traumatic spinal cord injury. The Gruppo Italiano Studio Epidemiologico Mielolesioni study. Arch Phys Med Rehabil 2003;84:1266-1275.
28. Karacan I, Koyuncu H, Pekel O, et al. Traumatic spinal cord injuries in Turkey: a nation wide epidemiological study. Spinal cord 2000;38:697-701.
29. Citterio A, Franceschini M, Spizzichino L, et al. Nontraumatic spinal cord injury: an Italian survey. Arch Phys Med Rehabil 2004;85:1483-1487.
30. Sluijter ME, Cosman ER, Rittman WJ, Van Kleef M. The effects of pulsed radiofrequency fields applied to the dorsal root ganglion – a preliminary report. The Pain Clinic 1998; 11(2):109-118
31. Jaffe LF, Poo M-M (1979) Neurites grow faster towards the cathode than the anode in a steady field. J Exp Zool 209:115–127
32. Cosman ER Jr, Cosman ER Sr. Electric and thermal field effects in tissue around radiofrequency electrodes. Pain Medicine 2005;6(6):405-424.
33. Cowley et al. (2010)
34. Mendell LM. Constructing and deconstructing the gate theory of pain. Pain. 2014 Feb;155(2):210-6.
35. Liang L, Mendell LM. Bilateral transient changes in thalamic n. ventroposterior lateralis after thoracic hemisection in the rat. J Neurophysiol. 2013;110:942–951.
36. Liebano RE, Vance CG, Rakel BA, Lee JE, Cooper NA, Marchand S, Walsh DM, Sluka KA. Transcutaneous electrical nerve stimulation and conditioned pain modulation influence the perception of pain in humans. Eur J Pain. 2013;17:1539–1546.
37. Melzack R, Wall PD. Pain mechanisms: a new theory. Science. 1965 Nov 19; 150(3699):971-9.
38. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain. 2011;152:S2–S15.
39. Todd AJ. Neuronal circuitry for pain processing in the dorsal horn. Nat Rev Neurosci. 2010;11:823–836.
40. Tator CH. Biology of neurological recovery and functionalrestoration after spinal cord injury. Neurosurgery1998;42:696-707.
41. Côté MP, Murray LM, Knikou M. Spinal Control of Locomotion: Individual Neurons, Their Circuits and Functions. Frontiers in Physiology, 2018, 9:784
42. Jankowska, E. (2001). Spinal interneuronal systems: identification, multifunctional character and reconfigurations in mammals. J. Physiol. 533, 31–40.
43. Jankowska, E. (2016a). “Spinal interneurons”, in Neuroscience in the 21st Century, eds D. W. Pfaff and N. D. Volkow (New York, NY: Springer Science+Business Media), 1189–1224.
44. Jankowska, E. (2016b). “Spinal reflexes”, in Neuroscience in the 21st Century, eds D. W. Pfaff and N. D. Volkow (New York, NY: Springer Science+Business Media), 1599–1621.
45. Jankowska, E., and Hammar, I. (2002). Spinal interneurones; how can studies in animals contribute to the understanding of spinal interneuronal systems in man? Brain Res. Brain Res. Rev. 40, 19–28. doi: 10.1016/S0165-0173(02)00185-6
46. Kiehn, O. (2016). Decoding the organization of spinal circuits that control locomotion. Nat. Rev. Neurosci. 17, 224–238. doi: 10.1038/nrn.2016.9