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Cardiovascular complications associated with spinal cord injury

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ABSTRACT

Spinal cord injury can lead to important functional, psychological and social sequelae. Despite the progress in medicine and greater understanding of the pathophysiological events associated with a traumatic spinal cord injury, spinal cord injury is still associated with a high morbidity and mortality. The involvement of the autonomic nervous system has implications in acute and chronic stages of the injured spinal cord patients. The most frequent cardiovascular complications in the acute phase of the traumatic spinal lesions are bradyarrhythmia, hypotension, increased vasovagal reflexes, ventricular and supraventricular ectopic beats, venous stasis, and vasodilation. In the chronic phase, we find orthostatic hypotension, alteration of the arterial pressure and the regulation of the body temperature as well as alteration of the blood volume. The knowledge of the cardiovascular alterations is of vital importance for the management and rehabilitation of the patients with spinal cord injury. In this article, we present a critical review of medical literature.

1. Introduction

Spinal lesions are of great importance worldwide because the onset mechanism is usually traumatic triggering multiple motor, sensory and autonomic disorders that require a multidisciplinary grouping composed of neurosurgeons, internists, intensive care physicians, general practitioners, nutritionists, psychologists, physiotherapists, nursing staff among others, which makes it a high-cost pathology[1–3]. It affects between 12 000 and 20 000 new people per year in the United States and usually has profound neurological sequelae[4,5]. Within its complications, whether in its acute or chronic phase, we are led to morbidity and mortality in cardiovascular complications, with a prevalence of up to 30% in spinal cord trauma deaths[4]. The management of these complications is particularly challenging in the acute phase of spinal cord injury because this is usually accompanied by other conditions that may influence the patient's hemodynamic stability such as hypovolemia[6]. or other trauma mechanisms, not to mention that these patients present predisposition to other complications such as deep venous thrombosis, pulmonary thromboembolism, acute renal injury, which we will mention later[7,8]. The main intention of this text is to review the main cardiovascular complications in relation to spinal trauma in its acute and chronic phases, a general review of

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the neuroanatomy and to understand the underlying pathophysiology that will guide us to an optimal treatment.

2. Anatomy of autonomic nervous system

Parasympathetic preganglionic neurons are found in the nuclei of the cranial nerves common motor (III), facial (VII), glossopharyngeal (IX) and vagus (X) in the brain stem. Most of the internal organs have parasympathetic innervation, especially given by the vagus nerve, except for the bladder, genitals, distal intestine, and anus which receive parasympathetic innervation by the sacral plexus S2-S4[8] (Figure 1). On the other hand, the preganglionic sympathetic neurons are found in the intermediate-lateral horn in the gray matter at the level of T1-L2[9]. The parasympathetic nervous system is responsible for decreasing heart rate and contractility while the sympathetic nervous system has stimulating effects. Homeostasis or balance between these two systems is regulated by chemoreceptors and baroreceptors located in the aortic arch, carotid sinus and coronary arteries[6] (Table 1). Depending on the level of spinal cord trauma, the organs that are innervated by the sympathetic nervous system will be affected, resulting in an abnormal adrenergic response below the level of the lesion. The parasympathetic innervation organs mentioned above, such as the bladder, colon and sexual organs, will also be affected, as they will obviously also lose supra-spinal control. Since the parasympathetic preganglionic neurons that innervate the heart are found in the brain stem, the parasympathetic tone will be intact in the spinal cord lesions[10].

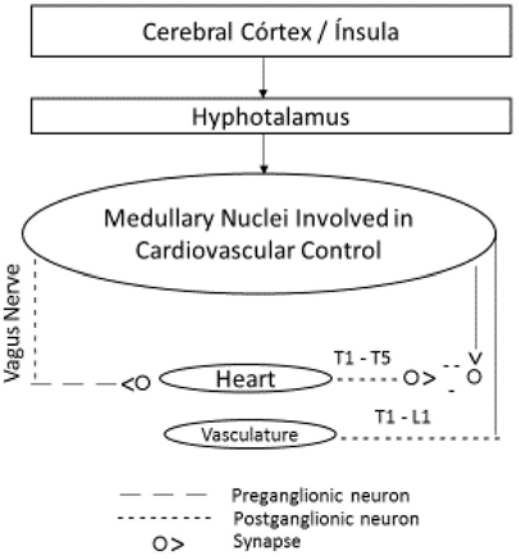

Figure 1. Diagram of autonomic cardiovascular control.

Table 1
Types of receptors and location in sympathetic and parasympathetic nervous system.

Organ	Sympathetic		Parasympathetic	
	Receptor subtype	Effect	Receptor subtype	Effect
Heart	β_1, also β_2, also α and DA_1	↑ Heart rate	M_2	↓ Heart rate
		↑ Force of contraction		↓ Force of contraction
		↑ Conduction velocity		Slight ↓ conduction velocity
		↑ Automaticity (β_2)		
		↑ Excitability		
	α_1	↑ Force of contraction		
Arteries	β_1	Coronary vasodilatation	M^a	Vasodilatation in skin, skeletal muscle, pulmonary and coronary circulation
	β_2	Vasodilatation (Skeletal muscle)		
	α_1, α_2	Vasoconstriction (coronary, pulmonary, renal and splanchnic circulation, skin and skeletal muscle)		
	DA_1, β_2	Splanchnic and renal circulation		
Veins	α_1, also α_2	Vasoconstriction		
	β_2	Vasodilatation		
Lung	β_2	Bronchodilatation Inhibition of secretions	M_1, M_3	Bronchoconstriction Stimulation of secretions
	α_1	Bronchoconstriction		
GI tract	α_1, α_2, β_2	Decreased motility	M_2, M_3	Increased motility Relaxation sphincters Stimulation of secretions
	α_1, α_2	Contraction of sphincters Inhibition of secretions		
Pancreas	β_2	Increased insulin release		
	α_1, α_2	Decreased insulin release		
Kidney	β	Renin secretions		
Liver	β_2, α	Glycogenolysis	M	Glycogen synthesis

3. Effects on cardiovascular regulation

The severity of cardiovascular effects in spinal cord trauma will depend directly on the level of trauma and the degree of injury[11] (Figure 2). In the complete medullary section, the connection between the superior autonomic centers in the brain and the intermediate-lateral horn at the level of T1-L2 is completely lost[11]. Patients with cervical lesions are at increased risk of producing atrial arrhythmias, ventricular arrhythmias, cardiac arrest, and other complications that we will see later. This happens mainly in the first 4 to 5 weeks. We can also observe that immediately after the spinal cord trauma, there is a loss of the innervation of the smooth muscle of the arteries, generating vasodilatation and hypotension (medullary shock), which can be confused with hypovolemia given the mechanisms of trauma[12]. In the acute phase, the parasympathetic tone may be increased and being sensitive to the stimuli. Any stimuli such as hypoxia by hypoventilation, nasogastric tubes, endotracheal tubes can trigger a vagal response generating abnormal cardiovascular responses. Usually, this occurs in patients with complete/high lesions and can last a lifetime, on the contrary, in patients with incomplete/low lesions, it usually disappears after 4 to 5 weeks[13].

4. Cardiovascular complications

Next, we will describe the most common cardiovascular complications, according to the stage of spinal cord trauma[14–16].

Acute phase: Sinus bradycardia, loss of vascular tone, ventricular/supraventricular ectopics, hypotension, orthostatic hypotension, increased vagal reflex, vasodilatation and venous stasis.

Chronic phase: Orthostatic hypotension, decreased cardiovascular reflexes, decrease or absence of heart pain, autonomic dysreflexia (lesions above T6), loss of muscle mass of the left ventricle, pseudo myocardial infarction: Elevation of biomarkers with or without electrocardiographic abnormalities without cardiovascular disease.

5. Orthostatic hypotension

It is defined as a decrease of 20 mm Hg in systolic blood pressure or 10 mm Hg in diastolic blood pressure when performing an orthostatic maneuver, it is much more frequent in patients with cervical or upper thoracic spinal cord injuries[17]. Due to the lack of sympathetic innervation on the cardiovascular system, hypotension can be severe in the acute phase of the spinal cord injury leading to a spinal cord injury, which is a challenge for the treating staff, even more so if we are in the presence of hypovolemia secondary to the mechanism of trauma that led to the spinal cord. This is accompanied by blurred vision, dizziness, fatigue, dyspnea, the agitation which is attributed to cerebral hypoperfusion. It is important to note that the time since a spinal cord injury and decompression surgery may be associated with the occurrence of cardiovascular dysfunction[18]. It is recommended to maintain an average arterial pressure between 85-90 mm Hg for at least the first week since the onset of spinal cord trauma, it is believed that when the cord is severed, it loses its capacity for self-regulation, whereby hypotension causes hypoperfusion and tissue hypoxia which would be related to worse outcomes[19]. An optimal state of hydration should be maintained to keep the patient hypervolemic or slightly hypervolemic, and if vasoactive drugs are required. There is insufficient evidence to support the use of one vessel over another.

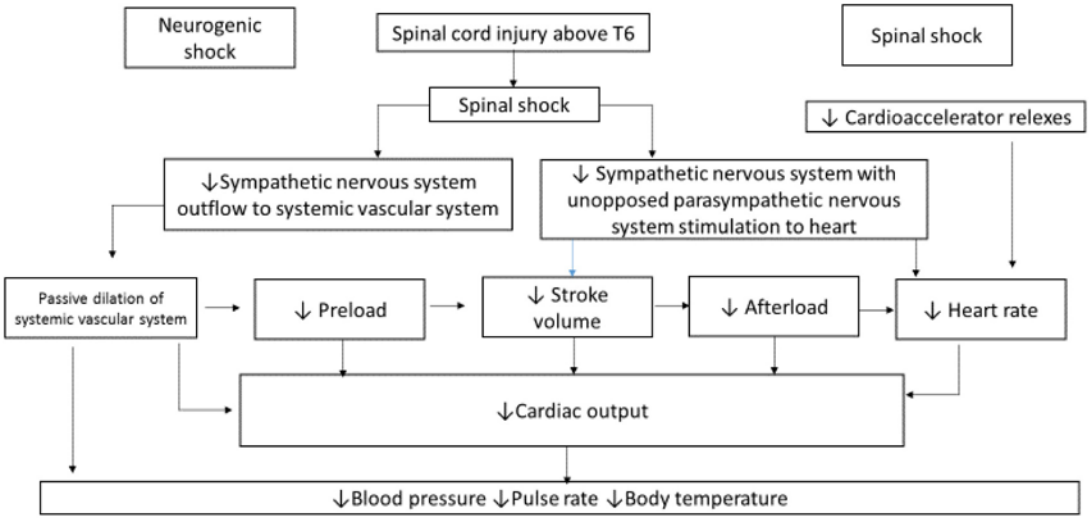

Figure 2. Initial cardiovascular effects of spinal cord injury.

6. Anatomy of autonomic nervous system

Patients with high thoracic (T6 or higher) or cervical trauma may experience sudden episodes of peripheral vasoconstriction accompanied by dysautonomia signs and/or symptoms in response to hyperstimulation below the level of the lesion. The most common stimuli are intestinal or bladder distention. We can define it as a 20% increase in basal blood pressure with increased heart rate and accompanied by signs and/or dysautonomia symptoms such as dizziness, nausea, vomiting, nasal congestion, headache, and others[20]. Management is initially conservative, eliminating the harmful stimulus and waiting for an adequate hemodynamic response, in the case of hypertension present refractoriness to conservative treatment, antihypertensive agents such as ACE inhibitors, calcium antagonists, alpha 1 blockers or intravenous nitrates could be used[13,14,21].

7. Cardiac arrhythmias

TThe risk of cardiac arrhythmias associated with spinal cord trauma is much higher in the acute phase and decreases as time passes, they tend to be riskier during the first few weeks, but there is also a risk of developing cardiac arrhythmias in the chronic phase of spinal cord injury[22]. Bradyarrhythmias are usually the most frequent and added to the hypotension commonly found in these patients can lead to hemodynamic instability. During the chronic phase, the incidence of bradyarrhythmia's may be frequent in patients with tetraplegia and rarely in patients with paraplegia. Tachyarrhythmias usually occur during episodes of dysautonomic dysreflexia and most require immediate pharmacological treatment to return to basal rate. It should be considered that patients with spinal cord trauma are at risk for all types of conduction disorders such as ST-segment elevation, atrial and/or ventricular extrasystoles, and branch blocks among others. Special care must be taken with these patients when performing maneuvers that increase vagal tone, as mentioned above, laryngoscopy, tracheal suction, especially in the presence of hypoxia since it also generates an increase in vagal tone. These maneuvers can easily trigger bradyarrhythmias or even asystole[13–15].

8. Coronary heart disease

Coronary heart disease is found in about 20% of deaths from spinal cord trauma[23]. The risk of coronary heart disease increases due to physical inactivity, obesity, hyperlipidemia, insulin resistance and diabetes. In the chronic phase, there is usually an increase in LDL cholesterol and a decrease in HDL, the reason why this occurs is still under discussion, but is assumed to be due to lack of exercise, adrenergic dysfunction, and inappropriate diet[24].

9. Treatment of bradyarrhythmias

The first line drug is dopamine in the case of bradycardia, atropine and transcutaneous pacemakers are second-line if there is no response to dopamine. It may be necessary to use a transvenous pacemaker in severe and prolonged bradycardias. Keep in mind that the treatment should be preventive, to avoid mainly the maneuvers that generate an increase of the parasympathetic tone and in the case of requiring endotracheal aspirations to use atropine previously[6,16].

10. Treatment of dysautonomic dysreflexia

While the elimination of the stimulus that triggers dysautonomic dysreflexia improves symptomatology and tension figures we should not initiate pharmacological therapy but try to prevent the emergence of new harmful stimuli. As far as possible place the patient upright to redistribute the blood to the lower body[21]. Although many of the episodes of dysautonomic dysreflexia are easily resolved with non-pharmacological management, sometimes pharmacological therapy is needed, especially in patients with high or cervical thoracic lesions. Nitrates are top-line orally or topically. It also describes the use of intravenous sodium nitroprusside, which rapidly resolves hypertension, but we reserve this medication for very severe episodes. Dihydropyridine calcium antagonists are widely used in clinical practice, but their evidence is not very strong in favor because it could lead to a decrease in long-term blood pressure figures and generate hypoperfusion and medullary ischemia. Alpha-1 blockers, such as prazosin have less cardiovascular effects and have been described as prophylactic in the development of dysautonomic dysreflexia[25,26].

11. Prophylaxis of deep venous thrombosis

Pharmacological and non-pharmacological therapies are mandatory for this type of patients, including early limb mobilization, physical therapy, intermittent compression measures. Low molecular weight heparins are effective in preventing the formation of thrombi and do not significantly increase the risk of bleeding, especially within 24 hours of injury[27], mechanical prophylaxis has shown to be less effective[28]. If patients develop deep venous thrombosis (DVT), treatment with intravenous heparin should be started in 7 to 10 days, continuing with oral anticoagulants for at least 3 months, but some are considering to extend this recommendation up to 6 months due to high incidence of deep venous thrombosis chronic spinal cord injury[29]. In a case of deep vein thrombosis in patients with anticoagulation or who cannot receive anticoagulation due to the high risk of bleeding, a lower vena cava filter may be considered[30].

12. Treatment of cardiovascular disease and dyslipidemia

It is based on reducing exposure to risk factors such as smoking, obesity, physical inactivity, poor control of blood pressure figures, poor control of cholesterol levels among others, dyslipidemia in patients with spinal cord injury is as high as 77%[31]. Statins have been shown to be effective as secondary prevention for cardiovascular disease, but to also atorvastatin has shown neuroprotective effects[32], decreases pain at 3-6 months[33], and at the molecular level cause a significant decrease in IL-1 β , IL-6 and lipid peroxide[34]. Patients benefit from both the lipid-lowering activity and the pleiotropic effects of statins, such as the improvement of endothelial dysfunction, reduction of platelet activity, stabilization of atherosclerotic plaques, and others.

13. Treatment of neurogenic shock

Neurogenic shock is defined as a systolic blood pressure <100 mm Hg and a heart rate <80 beats per minute in the context of a spinal cord injury after ruling out other diagnoses, occurring in lesions above T6[6]. Several pathophysiological mechanisms are involved in the spinal injury. Neurogenic shock describes the sudden loss of autonomic tone due to injury to the spinal cord. Alteration of the sympathetic descending pathways results in alteration of the vagal tone in the vascular smooth muscle, causing a decrease in systemic vascular resistance and vasodilation, other new symptoms have been introduced like changes in heart rate less than 3 times in 10 seconds, opposing multiple trauma patients pattern and the absence of an increase in heart rate of more than 10% of the base within 3 to 5 seconds of painful stimuli[35]. The management of neurogenic shock should be focused on prevention, since a not insignificant number of patients in the context of spinal cord injury presents this type of distributive shock and the management should initiate in the prehospital care with the appropriate identification of the context of the patient suggesting a possible spinal cord injury, in order to proceed to the appropriate immobilization with a rigid cervical collar and spinal table, with the prompt transfer to a proper care unit[36,37].

14. Conclusions

Spinal cord trauma is a devastating disease. Cardiovascular disorders are frequent and are causes of mortality in the acute and chronic phase. Bradycardia is the most frequent cardiac dysrhythmia and changes in heart rate response to painful stimuli should be reviewed as well. It is also important to mention that deconditioning may directly result in an increase in morbidity and mortality in patients with traumatic spinal cord injury since the decrease in exercise ability is dramatically reduced in this class of patients. The preparation of guidelines for the management of various cardiovascular disorders in patients with medullary traumatic injury is necessary and should be a task for the immediate future.

Conflict of interest statement

We declare that we have no conflict of interest.

References

[1] Ma VY, Chan L, Carruthers KJ. Incidence, prevalence, costs, and impact on disability of common conditions requiring rehabilitation in the united states: stroke, spinal cord injury, traumatic brain injury, multiple sclerosis, osteoarthritis, rheumatoid arthritis, limb loss, and back pain. *Arch Phys Med Rehabil* 2014; **95**: 986–995.e1. DOI: https://doi.org/10.1016/j.apmr.2013.10.032.

[2] Rouanet C, Reges D, Rocha E, Gagliardi V, Silva GS, Rouanet C, et al. Traumatic spinal cord injury: Current concepts and treatment update. *Arq Neuropsiquiatr* 2017; **75**: 387–393. DOI: https://doi.org/10.1590/0004-282x20170048.

[3] Lenehan B, Street J, Kwon BK, Noonan V, Zhang H, Fisher CG, et al. The epidemiology of traumatic spinal cord injury in British Columbia, Canada. *Spine (Phila Pa 1976)* 2012; 37: 321–329. doi: https://doi.org/10.1097/BRS.0b013e31822e5ff8.

[4] Garshick E, Kelley A, Cohen SA, Garrison A, Tun CG, Gagnon D, et al. A prospective assessment of mortality in chronic spinal cord injury. *Spinal Cord* 2005; **43**: 408–416. DOI: https://doi.org/10.1038/sj.sc.3101729.

[5] Ning G-Z, Wu Q, Li Y-L, Feng S-Q. Epidemiology of traumatic spinal cord injury in Asia: A systematic review. *J Spinal Cord Med* 2012; **35**: 229–239. DOI: https://doi.org/10.1179/2045772312Y.0000000021.

[6] Furlan JC, Fehlings MG. Cardiovascular complications after acute spinal cord injury: pathophysiology, diagnosis, and management. N*eurosurg Focus* 2008; **25**: E13. DOI: https://doi.org/10.3171/FOC.2008.25.11.E13.

[7] Grigorean VT, Sandu AM, Popescu M, Iacobini MA, Stoian R, Neascu C, et al. Cardiac dysfunctions following spinal cord injury. *J Med Life* 2009; **2**: 133–145.

[8] Lavanderos FJ, Muñoz GS, Vilches AL, Delgado MM, Cárcamo HK, Passalaqua HS, et al. Traumatismo raquimedular. *Cuad Cirugía* 2008; **22**: 82–90. DOI: https://doi.org/10.4206/cuad.cir.2008.v22n1-12.

[9] Krassioukov A, Biering-Sørensen F, Donovan W, Kennelly M, Kirshblum S, Krogh K, et al. International standards to document remaining autonomic function after spinal cord injury. *J Spinal Cord Med* 2012; **35**: 201–210. DOI: https://doi.org/10.1179/1079026812Z.00000000053.

[10]McCorry LK. Physiology of the autonomic nervous system. *Am J Pharm Educ* 2007; **71**: 78.

[11]Phillips WT, Kiratli BJ, Sarkarati M, Weraarchakul G, Myers J, Franklin BA, et al. Effect of spinal cord injury on the heart and cardiovascular fitness. *Curr Probl Cardiol* 1998; **23**: 641–716.

[12]Eldahan KC, Rabchevsky AG. Autonomic dysreflexia after spinal cord injury: Systemic pathophysiology and methods of management.

Auton Neurosci 2017; **209**: 59–70. DOI: https://doi.org/10.1016/j.autneu.2017.05.002.

[13]Ackery A, Tator C, Krassioukov A. A global perspective on spinal cord injury epidemiology. *J Neurotrauma* 2004; **21**: 1355–1370. DOI: https://doi.org/10.1089/neu.2004.21.1355.

[14]Driussi C, Ius A, Bizzarini E, Antonini-Canterin F, d'Andrea A, Bossone E, et al. Structural and functional left ventricular impairment in subjects with chronic spinal cord injury and no overt cardiovascular disease. *J Spinal Cord Med* 2014; **37**: 85–92. DOI: https://doi.org/10.1179/2045772313Y.0000000161.

[15]Bartholdy K, Biering-Sørensen T, Malmqvist L, Ballegaard M, Krassioukov A, Hansen B, et al. Cardiac arrhythmias the first month after acute traumatic spinal cord injury. *J Spinal Cord Med* 2014; **37**: 162–170. DOI: https://doi.org/10.1179/2045772313Y.0000000181.

[16]Evans DE, Kobrine AI, Rizzoli H V. Cardiac arrhythmias accompanying acute compression of the spinal cord. *J Neurosurg* 1980; **52**: 52–59. DOI: https://doi.org/10.3171/jns.1980.52.1.0052.

[17]Jeffrey B., Mote MB, Clay EC. Evaluation and management of orthostatic hypotension. *Am Fam Physician* 2011; **84**: 527–536.

[18]Wilson JR, Singh A, Craven C, Verrier MC, Drew B, Ahn H, et al. Early versus late surgery for traumatic spinal cord injury: The results of a prospective Canadian cohort study. Spinal Cord 2012; **50**: 840–843. DOI: https://doi.org/10.1038/sc.2012.59.

[19]Partida E, Mironets E, Hou S, Tom VJ. Cardiovascular dysfunction following spinal cord injury. *Neural Regen Res* 2016; **11**: 189–194. DOI: https://doi.org/10.4103/1673-5374.177707.

[20]Milligan J, Lee J, McMillan C, Klassen H. Autonomic dysreflexia: recognizing a common serious condition in patients with spinal cord injury. *Can Fam Physician* 2012; **58**: 831–835.

[21]Krassioukov A, Warburton DE, Teasell R, Eng JJ. Spinal Cord Injury Rehabilitation Evidence Research Team TSR. A systematic review of the management of autonomic dysreflexia after spinal cord injury. *Arch Phys Med Rehabil* 2009; **90**: 682–695. DOI: https://doi.org/10.1016/j.apmr.2008.10.017.

[22]Hector SM, Biering-Sørensen T, Krassioukov A, Biering-Sørensen F. Cardiac arrhythmias associated with spinal cord injury. *J Spinal Cord Med* 2013; **36**: 591–599. DOI: https://doi.org/10.1179/2045772313Y.0000000114.

[23]Bauman W, Spungen A. Risk assessment for coronary heart disease in a Veteran population with spinal cord injury. *Top Spinal Cord Inj Rehabil* 2007; **12**: 35–53. DOI: https://doi.org/10.1310/sci1204-35.

[24]Cragg JJ, Noonan VK, Krassioukov A, Borisoff J. Cardiovascular disease and spinal cord injury: results from a national population health survey. *Neurology* 2013; **81**: 723–728. DOI: https://doi.org/10.1212/WNL.0b013e3182a1aa68.

[25]Bauman CA, Milligan JD, Lee FJ, Riva JJ. Autonomic dysreflexia in spinal cord injury patients: An overview. *J Can Chiropr Assoc* 2012; **56**: 247–250.

[26]Hagen E, Rekand T, Grønning M, Færestrand S. Kardiovaskulære følgetilstander etter ryggmargsskade. *Tidsskr Den Nor Legeforening* 2012; **132**: 1115–1120. DOI: https://doi.org/10.4045/tidsskr.11.0551.

[27]DiGiorgio AM, Tsolinas R, Alazzeh M, Haefeli J, Talbott JF, Ferguson AR, et al. Safety and effectiveness of early chemical deep venous thrombosis prophylaxis after spinal cord injury: pilot prospective data. *Neurosurg Focus* 2017; **43**: E21. DOI: https://doi.org/10.3171/2017.8.FOCUS17437.

[28]Chung S-B, Lee S-H, Kim ES, Eoh W. Incidence of deep vein thrombosis after spinal cord injury: A prospective study in 37 consecutive patients with traumatic or nontraumatic spinal cord injury treated by mechanical prophylaxis. *J Trauma Inj Infect Crit Care* 2011; **71**: 867–871. DOI: https://doi.org/10.1097/TA.0b013e31822dd3be.

[29]Mackiewicz-Milewska M, Jung S, Kroszczy ski AC, Mackiewicz-Nartowicz H, Serafin Z, Cisowska-Adamiak M, et al. Deep venous thrombosis in patients with chronic spinal cord injury. *J Spinal Cord Med* 2016; **39**: 400–404. DOI: https://doi.org/10.1179/2045772315Y.0000000032.

[30]Johns JS, Nguyen C, Sing RF. Vena cava filters in spinal cord injuries: Evolving technology. *J Spinal Cord Med* 2006; **29**: 183–190.

[31]Vichiansiri R, Saengsuwan J, Manimmanakorn N, Patpiya S, Preeda A, Samerduen K, et al. The prevalence of dyslipidemia in patients with spinal cord lesion in Thailand. *Cholesterol* 2012; **2012**: 1–6. DOI: https://doi.org/10.1155/2012/847462.

[32]Nazli Y, Colak N, Alpay MF, Uysal S, Uzunlar AK, Cakir O. Neuroprotective effect of atorvastatin in spinal cord ischemia-reperfusion injury. *Clinics (Sao Paulo)* 2015; **70**: 52–60. DOI: https://doi.org/10.6061/clinics/2015(01)10.

[33]Aghazadeh J, Samadi Motlagh P, Salehpour F, Meshkini A, Fatehi M, Mirzaei F, et al. Effects of atorvastatin in patients with acute spinal cord injury. *Asian Spine J* 2017; **11**: 903–907. DOI: https://doi.org/10.4184/asj.2017.11.6.903.

[34]Nacar OA, Eroglu H, Cetinalp NE, Menekse G, Yildirim AE, Uckun OM, et al. Systemic administration of atorvastatin improves locomotor functions and hyperacute-acute response after experimental spinal cord injury: an ultrastructural and biochemical analysis. *Turk Neurosurg* 2014; **24**: 337–343. DOI: https://doi.org/10.5137/1019-5149.JTN.8131-13.1.

[35]Paydar S, Karami MY, Khalili H, Dehghankhalili M, Sabetian G, Ghaffarpasand F. Heart rate beat to beat variability of trauma patient in neurogenic shock state: Time to introduce new symptoms. *Bull Emerg Trauma* 2017; **5**: 141–142.

[36]Galeiras Vázquez R, Ferreiro Velasco ME, Mourelo Fariña M, Montoto Marqués A, Salvador de la Barrera S. Actualización en lesión medular aguda postraumática. P*arte 1. Med Intensiva* 2017; **41**: 237–247. DOI: https://doi.org/10.1016/j.medin.2016.11.002.

[37]Taylor MP, Wrenn P, O'Donnell AD. Presentation of neurogenic shock within the emergency department. *Emerg Med J* 2017; **34**: 157–162. DOI: https://doi.org/10.1136/emermed-2016-205780.