



Heart Rate Beat to Beat Variability of Trauma Patient in Neurogenic Shock State: Time to Introduce New Symptoms

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The autonomic nervous system (ANS) is consisted of several centers in the central nervous system (CNS) and the connections with the end organs through plexus and peripheral nerves which controls the hemodynamics [1]. It has been well demonstrated that patients with spinal cord injury (SCI) at T6 and above levels experience several hemodynamic abnormalities such as baseline low systemic blood pressure, orthostatic hypotension and episodes of sudden increase in blood pressure probably due to autonomic dysfunction [2]. In addition to hemodynamic abnormalities, SCI can lead to cardiac arrhythmia including sinus bradycardia, supraventricular arrhythmia, ventricular arrhythmia and cardiac arrest [3–6]. Although these cardiac arrhythmias are relatively well recognized in the subacute and chronic stages after SCI, there is a paucity of studies focused on abnormal cardiac electrical activity during the early stages following SCI.

Recently Furlan *et al.*, [7] described electrocardiogram abnormalities such as R-R interval change within the first 72 h following

acute traumatic complete, cervical or high-thoracic above T6, Spinal Cord Injury (SCI). Hemodynamic changes of spinal cord injuries consist of two general parts. Vascular dilatation caused by vascular changes secondary to spinal sympathetic cord nerve injury and cardiac change when the heart sympathetic strings have lost their connection with the central nervous system. Vascular changes in spinal cord injuries happen at every level that the higher the level of injury are associated with more vessel dilatation and more reducing blood pressure [8]. In this case, usually, the lower blood pressure is compensated with fluid replacement and increase stroke volume and heart rate if the cardiac sympathetic strings are healthy and it is not necessary to further actions [1]. But if the higher the level of spinal cord injury occur, the probability of cardiac sympathetic nerves damage and a number of arterial dilation increases. The cardiac sympathetic nerves disconnection with the central nervous system that often occurs when a higher level of spinal cord injury happens, the heart rate and stroke volume of the heart in response to the pressure drop caused by the dilation does not

increase and despite low pressure does not increase heart rate [1]. In the presence of spinal cord injury, always, there is a vascular component of neurogenic shock but as is often the primary resuscitation fluid be replaced and also the vessels and the heart are activated to compensate so the pressure drop is not significant. Of course, if there is another source of bleeding, Blood pressure drop may be more tangible. In this way it can be expected that if the higher the level of spinal cord injury occur, the probability of more serious neurological shock is high and vasoactive and inotrope drugs also more likely to need. When the cardiac sympathetic fibers cut, the heart just will be affected by parasympathetic system and is faced with a reduction in heart rate and heart rate chaotic changes that result from the interaction of sympathetic and parasympathetic systems there will be not immortalized in sinoatrial node and as a result the beat to beat heart rate variability changes reduced and it can be followed on the cardiac monitor through the patient's heart rate number [9]. The heart rate per minute monitoring is calculated based on the R-R interval when due to lack of sympathetic fiber, vanguard node activities are more relaxing, the R-R interval changes decrease, significantly and physicians will find the relative stability of the number of cardiac beats. In the acute phase of multiple trauma, patients have significant fluctuations in the number of heart rate that commonly change

the number displayed on the cardiac monitor is more than 3 times in 10 seconds. The heart rate changes less than 3 times within consecutive 10 seconds on the cardiac monitor could be suggestive of the possibility of cervical cord injury, although can be seen in condition before death when all compensatory mechanisms fail, too. Another bidder condition of cervical spinal cord injury and cardiac sympathetic nerves damage is a lack of heart rate response or a significant delay response to painful stimuli. Usually, healthy cardiac sympathetic nerves increase the heart rate of more than ten percent of the base within 3 to 5 seconds with painful stimuli. There are two above-mentioned symptoms in patients with spinal cord injury, suggesting the conditions where it is the very high risk of neurogenic shock and the possibility of requiring the use of inotrope and vasoconstrictive drugs is also increasing. In patients with neurogenic shock that there are changes in heart rate and with painful stimuli are seeing an increase in heart rate, if you need cardiovascular drugs to increase the pressure, perhaps norepinephrine, which affects only the vessels, be a better choice but if there is no evidence for cardiac sympathetic activity, may be using drug which has both cardiac stimulants and vasoconstrictors property such as dopamine are better choice for increasing patient pressure.

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