Spinal cord injury

Spinal cord injury

SCI often results in significant LUT dysfunction with a high risk of UTI/sepsis, renal function deterioration, renal and bladder calculi and autonomic dysreflexia. Urinary tract complications and renal failure were the leading causes of death in this population, but thorough evaluation and early definitive management have considerably improved urinary tract outcomes in patients with SCI. Immediately after SCI, a period of ‘spinal shock’ occurs in which there is a marked reduction in all spinal reflex activ ity below the level of the lesion. This results in an areflexic and acontractile b ladder; urinary retention lasts 6–12 weeks in complete SCI but may be shorter in incomplete lesions. Patients are managed with indwelling catheterisation or CISC during this phase . Return of function is characterised by spas ticity (detrusor overactivity and DSD) and should be managed as described under Urinary incontinence, Treatment Autonomic dysreflexia Autonomic dysreflexia is a sudden and exaggerated autonomic (primarily sympathetic) response to various stimuli in patients with SCI or spinal dysfunction above the cord level of T6–8 (the sympathetic outflow). Stimuli below the level of the lesion (commonly a distended bladder or rectum) lead to symptoms of headache, hypertension (varying from mild headache to seizures or cerebral haemorrhage) and flushing or sweating of the face and body above the level of the lesion. Autonomic dysreflexia is thought to occur as a result of an unopposed sympathetic response to noxious stimuli. Noci ceptive a ff erents elicit refle x sympathetic outflow , resulting in piloerection, sweating and arteriolar vasoconstriction, leading to hypertension. The peripheral vasoconstriction activates nating in the central nervous system to counteract the sympa - thetic outflow . The resulting vagal outflow causes bradycardia, but this parasympathetic signal is unable to transmit below the level of the spinal cord lesion. This leads to brad ycardia, vaso - dilatation and flushing above the level of the lesion, but hyper - tension and pale, cold skin due to ongoing vasoconstriction below the level of the lesion. Initial treatment involves sitting the patient upright, remov - ing any constricting clothing and identifying and removing the source of stimulation (urinary retention, blocked catheter, loaded rectum). Regular blood pressure monitoring should be performed; if the systolic blood pressure remains elevated (>150 /uni00A0 mmHg), patients should be treated with fast-acting anti - hypertensives such as sublingual nifedipine or glyceryl trini - trate. -

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