Neuro-urological Basis of Nocturia

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The principal physiological effect of neurological disease relevant to the symptom of nocturia, arising at night because of the desire to pass urine, is due to detrusor overactivity and diminished bladder capacity. Loss of neurons in the cerebral cortex after stroke which normally modulate impulses to void signaled in the pontine center for micturition lead to functionally small bladder, nocturia and incontinence. Similar pathophysiology may result from Parkinson's Disease, post traumatic brain injury and Multiple Sclerosis. Spinal disease such as trauma, MS, myelodysplasia and incomplete lesions such as anterior cord or Brown-Sequard Syndrome may cause upper or lower motor neuron disease and concomitant effects on bladder function, overactive or underactive, respectively. Less obvious correlates of neurologic disease may pertain to cardiovascular and renal disease which may cause nocturia due to nocturnal polyuria, overproduction of urine during sleep hours. Regardless of etiology, evaluation of the patient with nocturia commences with history and physical examination including the neurourological exam (cognition, gait, DTRs, perianal sensation, sphincter tone and function, bulbocavernosus reflex), urinalysis and 24 hour voiding diary. The latter may be analyzed to deliver categories of nocturnal polyuria (CHF, glycosuria, sleep apnea, third spacing, non-dipping nocturnal hypertension, idiopathic); diminished capacity (neurogenic bladder, lower urinary tract obstruction, malignancy); global polyuria (glycosuria, diabetes insipidus, primary polydipsia). Levels of evidence for epidemiology of nocturia and nocturnal polyuria is generally low. What is certain is that nocturia is frequent among Parkinsonians. Less well established is prevalence of detrusor overactivity in PD, MS and CVA patients as well as abnormal circadian patterns of blood pressure (non-dipping), solute (Na, urea) and water clearance in spinal cord injury patients. Level 1 evidence exists to support the use of desmopressin in MS patients in addition to pelvic floor muscle therapy in MS patients with overactive bladder. While not established specifically in the neurogenic bladder population, patients with nocturia due to decreased bladder capacity tend to improve with treatments designed to decrease nocturnal urine production even in the absence of baseline nocturnal polyuria.