Management of the Neurogenic Bladder (MS) in Community Nursing

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INTRODUCTION

Multiple Sclerosis (MS) is an autoimmune inflammatory brain and spinal cord disease (Hickey, 2014). It is characterised by unpredictable prognosis, influenced by the area(s) of demyelination within the central nervous system (CNS). Cited as the most distressing symptom (Fowler et al, 2009), bladder symptoms affect up to 75% of people with MS (De Ridder et al, 2013), often involving significant physical and psycho-social implications (Rushworth, 2009).

This poster explores the underpinning pathophysiology influencing neurogenic bladder symptoms in MS, to facilitate diagnostic reasoning and optimise clinical decision-making within community nursing in the context of National Institute for Clinical Excellence (NICE) guidance. Differential diagnosis is considered, identifying 'red flags' warranting comprehensive evaluation. Key recommendations for diagnostic reasoning specifically within the community setting are made.

PATHOPHYSIOLOGY

- Normal micturition comprises of two phases (Chapple, 2014) (Figure 1); that is storing (Figure 1A) and voiding (Figure 1B).
- In normal micturition, the detrusor muscle (bladder) is relaxed when empty.
- As the bladder fills, pressure is placed upon the internal sphincter.
- Alpha fibres transmit messages to the spinal cord via the pelvic (sacral level 2-4) and hypogastric nerves (T12-L2) (figure 1A) via afferent (sensory) nerves of the peripheral nervous system (PNS).
- Sensory excitability in the pelvic nerve alongside sympathetic (autonomic) activity of the hypogastric nerve inhibits detrusor contraction instigating voiding by external sphincter contraction.
- The pontine storage centre (PSC) inhibits this reflex if it is an inappropriate environment to void.

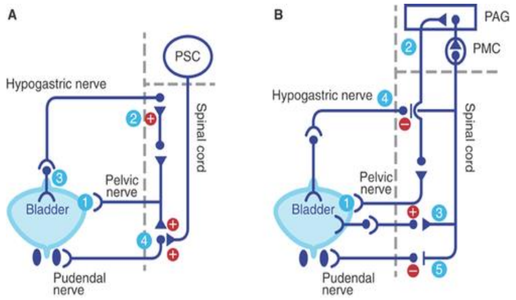

Figure 1: Neural control of Micturition (Chapple, 2014) Reproduced with permission from John Wiley and Sons Inc (licence 4371451321883) *

- Activation to void occurs through external sphincter relaxation via a CNS response (figure 1B) once the bladder reaches a specific threshold.
- In an intact CNS, messages are relayed via Alpha fibre cell bodies to the PSC and pontine micturition centre (PMC), where voiding signals are initiated.
- If appropriate, a spinobulbospinal reflex stimulates parasympathetic outflow to the bladder at s2-4, initiating contraction.
- Finally, the somatic nervous system enables voluntary control of micturition via activation of the pudendal nerve (S2-4).

PATHOPHYSIOLOGY IN MS

Normal bladder function is dependent upon intact neurons (Figure 2) enabling electrical transmission throughout PNS and CNS mechanisms. In MS, demyelination is confined to the CNS. However, Neurogenic bladder dysfunction is defined as "*any bladder disturbance … attributable to motor or sensory pathways in the central or peripheral nervous systems that have input into the bladder*" (Hickey, 2014, p230).

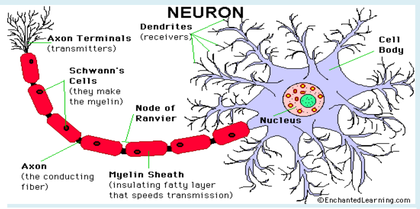

Figure 2: Neuron (Anon, 2018) licensed under Creative Commons Attribution Share-Alike 3.0 License.

In MS this predominantly arises from spinal cord inflammation (Fowler et al, 2009) and disparity between the pons and sacral element. For example, alpha cell bodies sit within the CNS in dorsal root ganglions (spinal cord), and demyelination may occur at this point. Furthermore, cortical lesions may inhibit the PMC leading to neurogenic detrusor overactivity (Hyperreflexia bladder) affecting around 62% of people with MS. Medullary lesions may lead to detrusor sphincter dyssynergia affecting around 25% (De Ridder et al, 2013) of people with MS. Neurogenic dysfunction (Hickey, 2014) issues are summarized in Table 1.

Table 1: Neurogenic bladder classification in MS (Modified from Table 11-6 (Hickey (2014) p231)

Classification	Location of Disruption	Clinical Symptoms
Uninhibited Neurogenic	• Lesions – PMC/ frontal lobe.	• Reduced initiation/ inhibition to void/↓ bladder capacity. • Urgency/frequency/nocturia.
Reflex neurogenic	• Lesion higher than T12/L1 (Spinal cord).	• Lesion may lead to sensory and motor innervation disruption if higher than S2-4. • Loss of control from higher centres (brain) - uninhibited/ involuntary contractions resulting in uncontrolled voiding. • External sphincter unable to relax as bladder contracts.
Sensory paralytic neurogenic	• Dorsal roots of sacral reflex centre (S2-4). • Cerebral cortex.	• Reduced/no feeling of bladder fullness. • Infrequent voiding but large output. • Increased bladder capacity (overflow incontinence).

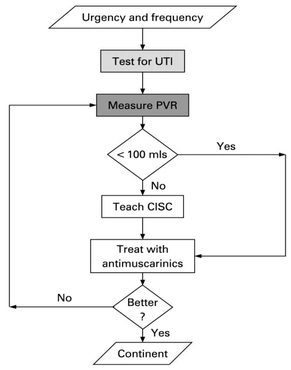

Figure 3: Algorithm of assessment for neurogenic bladder dysfunction in MS (Fowler et al, 2009). Reproduced with permission from BMJ Publishing Group Ltd (licence 4364880093676)

DIAGNOSTIC REASONING

Accurate diagnostic reasoning must be underpinned from an understanding of abnormal pathophysiology in neuro-urological function.

Based upon NICE guidelines (reviewed by NICE (2014a), Fowler et al's (2009) work (Figure 3) was pivotal in promoting consensus in diagnostic reasoning for neurogenic bladder assessment. Critical review of identified diagnostic interventions is evaluated in Table 2. Red flags are considered in Table 3.

Table 2: Review of diagnostic interventions

	Rationale and supporting evidence	Other considerations and differential diagnosis
Urinalysis (Reagent strips)	Reagent testing strips ascertain infection. Specificity/sensitivity (Fowler et al, 2009): and influences such as pre-analysis storage, or sampling procedure should be considered (Delanghe et al, 2014).	• Ongoing monitoring for infection recommended; may ↑MS symptoms. • Interstitial Cystitis. • Additional abnormalities unrelated may emerge (e.g. Diabetes Mellitus).
Bladder Scan (Post Void Residual Volume (PVR))	PVR should be undertaken prior to antimuscarnaric treatment. If significant PVR found, or >2 UTI/one year, PVR should be by Ultra-sound (USS) or CISC (Fowler et al, (2009).	• Caution in abnormal pelvic pathology (e.g. cyst) /surgery. • Referral if PVR >100-150ml, incontinence, ↓ quality of life , bypassing catheter (De Ridder et al, 2013).
Urodynamics	Urodynamic studies measure pressure and flow rate. They are not first-line procedure in the UK, (epidemiology of upper urinary tract problems are less common in MS compared to spinal cord injury), (Fowler et al, 2009). Urodynamics should be considered in context of non-maleficence. Fowler et al (2009) argues the benefit of intrusive studies is likely not to be outweighed by a change in management, although utilised routinely elsewhere.	• MS is more common in women (Hickey, 2014), and may have a number of factors impacting urological function (e.g. gynaecological aetiology) which may benefit from urodynamics. • As disability increases in MS, so does risk of urinary problems; people with MS are likely to be under the care of an urologist where additional studies are likely to be undertaken. • There is a dearth of recent evidence evaluating the reliability of urodynamics in relation to the neurogenic bladder.

Table 3: Red flags and cause for concern

- If haematuria present (NICE, 2015), refer for urgent investigation.
- People with neurogenic bladder symptoms are at risk of bladder cancer (NICE 2012), therefore consider early referral.
- Difficulty with voluntary control, may lead to renal complications. Sung et al (2016) identify risk factors for chronic kidney disease to be x3 higher in populations with neurogenic bladder issues.
- Prostate cancer is the most common cancer in males (NICE, 2014b); urgent referral required.

THERAPEUTIC CONSIDERATIONS

- First line treatments: adequate fluid, reduced caffeine (Fowler et al, 2009).
- Physiotherapy: pelvic floor exercises / Neuromuscular stimulation inhibit detrusor muscle (Fowler et al, 2009).
- Clean Intermittent Self Catheterisation (CISC) (impaired voiding).
- Antimuscarnaric medications are often combined with CISC (caution in patients with MS cognitive dysfunction).
- Rai et al (2012) describe combinations of treatments as best option.
- Often unlicensed, severe symptoms may benefit from other therapeutic or surgical options.

KEY RECOMMENDATIONS

- Fowler et al's (2009) work is pivotal to the detection of neurogenic bladder symptoms and easily transferable to the community.
- Resources enabling initial diagnostic reasoning within the community (e.g. bladder scanners) are accessible.
- The need for review of the validity of diagnostic reasoning in neurogenic bladder assessment, specifically bladder scanning /urodynamics is identified.
- Annual review for people with MS by an appropriate specialist (NICE 2014a) should include a review of urological, bowel and sexual symptoms (NICE, 2012).
- Open, prompt communication between primary and secondary care for timely referral in suspected differential diagnosis.
- Establishing a MS community database to measure outcome (e.g. reduced hospital admission/ improved patient outcomes).

CONCLUSION

The complexity of underpinning pathophysiology of the neurogenic bladder (MS), critiquing current diagnostic reasoning and its transferability to the community has been explored. It is suggested the degree of neurogenic dysfunction correlates with disability in MS (De Ridder, 2013) .Diagnostic reasoning must be underpinned by clinical history to optimise clinical outcome, ensuring principles of beneficence (Beauchamp and Childress, 2013) are upheld in therapeutic intervention.

As MS progresses, likelihood of neurogenic bladder symptoms increase. With emergency admissions for MS costing the NHS £43 million in 2013/2014 (National Health Interview Survey, 2015), it is imperative innovative, but cost-effective, diagnostic reasoning occurs in a timely and accessible location for optimal outcome.

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