Urinary incontinence

Common

May be:

Nocturnal or diurnal Intermittent or continuous Primary or secondary Organic or functional

Typically presents in one of three ways:

1. Purely night-time wetting

Primary monosymptomatic enuresis **always functional**. Secondary nocturnal enuresis usually functional; occasionally organic 2' to BOO or neuropathic bladder

- 2. Intermittent daytime wetting
 - Typically functional. Occasionally organic 2' BOO/neuropathy
- 3. Continuous daytime wetting

Always organic. Neurological or structural. Structural causes include epispadias, ectopic ureter, short urethra, urovaginal confluence.

NB. Any duration of dryness excludes ectopic ureter, female epispadias, urovaginal confluence, short urethra. Dribbling *between* voids may be associated with ectopic urethra or more commonly labial adhesions. Always ask about bowel function in addition to above features Physical examination

Abdominal	palpable bladder/kidneys
Male genital	epispadias, phimosis, meatal stricture
Female genital	epispadias, urovaginal confluence, imperforate
	hymen, labial adhesions
Spine	hairy patch, swelling, haemangiomata, sacral
	agenesis. UMN lesion (brisk reflex or clonus), calf
	wasting, lower limb neurology

Other examination

USS and urine dipstick in all patients Occasionally MRI and UDS for neuropaths

Organic daytime urinary incontinence

UTI	intermittent	
Bladder outflow obstruction*	intermittent	
Neuropathic bladder	intermittent	continuous
Hinman syndrome	intermittent	continuous
Exstrophy-epispadias		continuous
Ectopic ureter		continuous
Congenital short urethra**		continuous
Urovaginal confluence		continuous

* Bladder outflow obstruction Uncommon Boys > girls Boys

Posterior valves

Urethral stricture Meatal stenosis Pathological phimosis (rare) Constipation/tumour

Girls

Haematocolpos Hydrocolpos Constipation/tumour Incontinence typically 2' to obstructive detrusor overactivity rather than decompensation with overflow (except constipation/tumour) Investigation PMR Flow rate EUA and cystoscopy in selected cases

** Congenital short urethra

Continuous urethral leakage Diagnosis confirmed by cystourrethroscopy

Functional daytime urinary incontinence

Relatively common

3% girls and 2% boys of 7 years wet at least one day per week.

Of these 30% girls and 50% boys also wet at night

May be divided into dysfunctional storage or dysfunctional voiding

Dysfunctional storage (characterised by detrusor instability)

Urge syndrome

Diurnal frequency syndrome/sensory urgency Giggle incontinence

Dysfunctional voiding (often characterised by detrusor sphincter dyssynergia) Staccato voiding

Deferred voiding/lazy bladder syndrome

Occult neuropathic bladder (Hinman syndrome)

1. Urge syndrome

? persistence of transition phase to maturity voiding such that detrusor inhibition is volume related

End-filling detrusor instability typical; short duration from first sensation to incontinence. Absence of bladder overactivity = sensory urgency Re-inforcement manoeuvres common (Vincent's curtsey sign)

Urinary stream typically smooth (vs. staccato)

Natural history to resolution (2-3% persist into adulthood) USS/MSU (occasionally UDS)

Мx

Reassurance Pads Oxybutynin (60-70% effective: fewer side effects cf. adults) Bladder retraining Simple Timed fluid intake

Timed voiding

Biofeedback Pelvic floor relaxation etc Children over 8 yrs



2. Giggle incontinence

Inherited

Typically girls 9-12 yrs

Leakage may be significant

Often persists into adulthood

Modest improvement with anticholinergics: Ritalin (methylphenidate) most effective treatment but controlled drug

3. Dysfunctional voiding

Believed by some to represent an abnormal learned response to urge syndrome: sustained attempts to prevent leakage in response to OAB leads to failed sphincter relaxation or DSD Bladder emptying incomplete High intravesical pressures predispose to VUR +/- scarring Mx as for urge syndrome with ISC for high residuals

4. Lazy bladder syndrome

Long-standing dysfunctional voiding results in decompensation Increased compliance and decreased contractile efficiency leads to floppy bladder, chronic retention and UTI Rx with cognitive bladder retraining +/- ISC

5. Hinman syndrome

aka occult neuropathic bladder Children typically present with dysfunctional voiding leading to eventual decompression, poor compliance, overflow incontinence and upper tract changes.

Majority have bowel dysfunction (constipation, impaction)

Bladder displays all features of neuropathic bladder without an obvious cause

detrusor-sphincter dyssynergia 'fir-tree' bladder on cystography (sacculation) upper tract dilatation (> 60%; VUR in 50%)

occasionally obstructive uropathy Traditionally believed to be functional disorder 2' severe emotional upset (Hinman 1973): however similar features in Ochoa urofacial syndrome (Ochoa 1992) suggest subtle defect at brainstem level (facial LMN located close to pontine micturition centre)

bladder – Mx as for neuropaths (see notes)

Primary monosymptomatic nocturnal enuresis

Definition: wet at night over the age of five. Secondary enuresis = wet at night after 6 month symptom-free interval Natural history 85% of 5 year-olds are dry at night Approximately 10% 7 year olds wet bed > 3 x per week 10-15% per year resolve 7% of those who wet after 5 years old continue into adulthood Two thirds boys Positive FHx in 75% Features Impaired functional bladder capacity Impaired noctural secretion of vasopressin Impaired sleep arousal Management Supportive measures first Counselling Fluid restriction before bed Double voiding before bed Lifting may help Behavioural modification Star chart for young children Enuretic alarms Age 7 or above 70% success rate Low relapse rate Usually available from school nurse Requires no pants/nappy in bed Can malfunction/alarm if lead detached Medication Desmopressin desmospray 10-40ug, desmotabs 200-400 ug, desmomelt 125-250ug 70% success rate 30-50% relapse rate

useful for special occasions Anticholinergics may be useful in patients with a low bladder capacity. oxybutynin first line imipramine a/w cardiotoxicity and death in OD - avoid

Appendix

Voiding co-ordinated at pontine micturition centre in brainstem – not simple spinal reflex as previously believed

3 sets of peripheral nerves involved

- 1. thoracolumbar sympathetic (T10 L2)
 - via sympathetic chain and hypogastric nerves
- 2. sacral parasympathetic nerves (S2,3,4)
 - via pelvic nerves
- 3. sacral somatic outflow (S2,3,4)

via pudendal nerve

Evidence of innate influence of higher centres even in infants – infants only void during wakefulness (Ohel 1995): never when fully asleep. Voiding frequency vs. age

Foetal (third trimester)30 per 24 hoursFirst year20 per 24 hours2 – 3 years10 per 24 hours7 years5 per 24 hours (adult)

Reduction in voiding frequency due to growth-related increase in bladder capacity

Establishment of normal voluntary voiding dependent on 3 factors:

Age-related increase in bladder capacity

Development of voluntary control of striated urethral sphincter Direct control over bladder sphincteric unit

Attainment of complete control thought to pass through transitional phase where ability to inhibit voiding reflex is related to bladder volume.

Determination of expected bladder capacity in kids:

Infants (Holmdahl et al, 1996)

Bladder capacity (mL)= 38 + (2.5 × age (mo))

Children > 1 yr = Koff's formula (Koff, 1983)

Bladder capacity (mL)=[Age (yr)+ 2] \times 30

Post-micturition volume exceeding 10% of expected bladder capacity abnormal