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Ελλήνων Ειδικευομένων Ουρολόγων
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Αθήνα, Ξενοδοχείο Metropolitan

Παθοφυσιολογία ΚΥΠ & συσχέτιση συμπτωμάτων με ΟΑΒ

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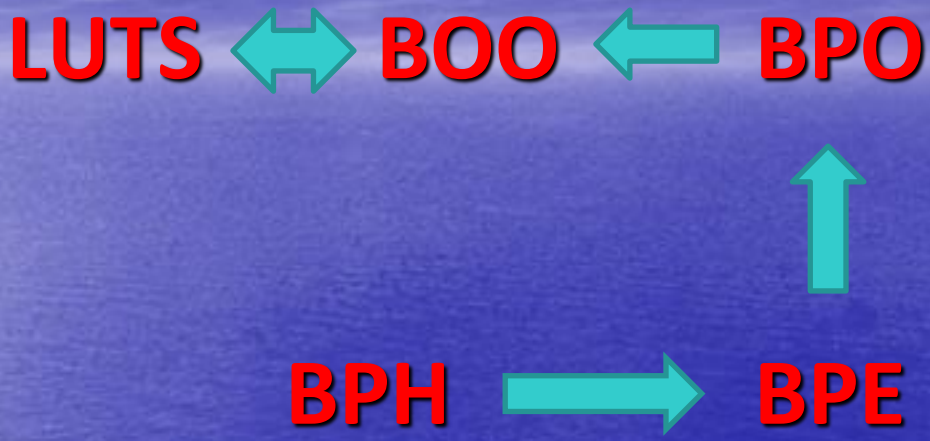
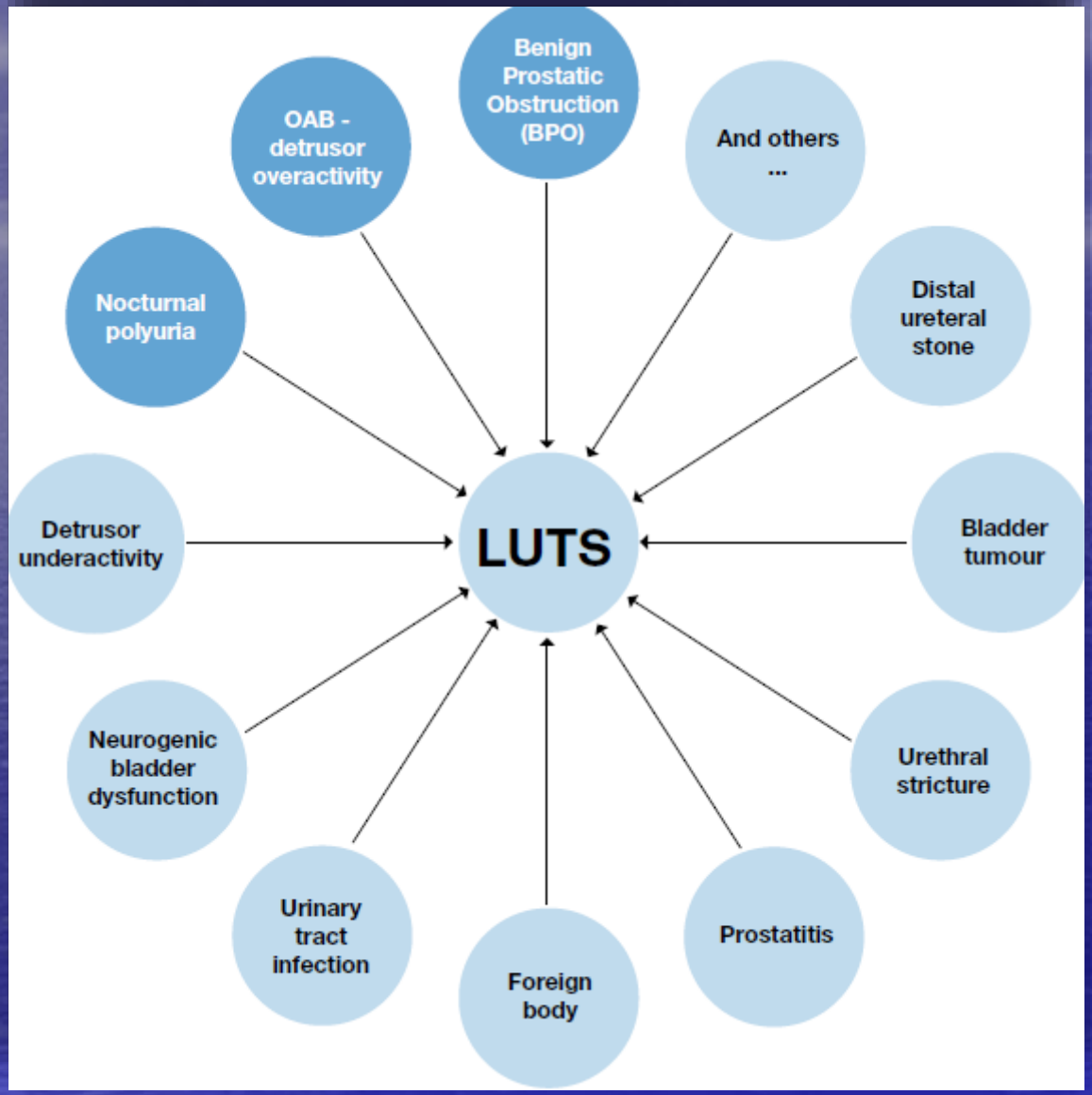
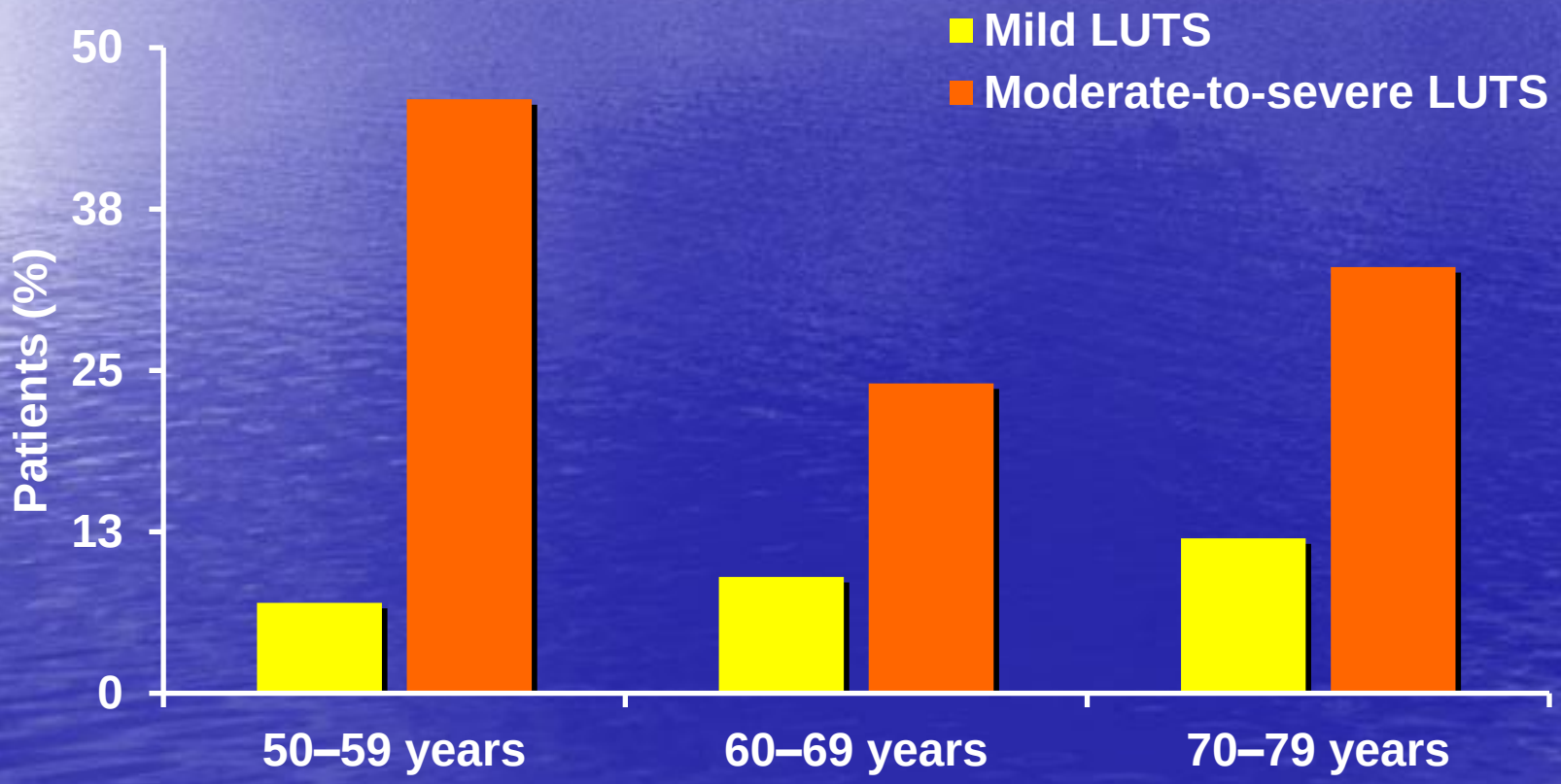
Επίκουρος Καθηγητής Ουρολογίας ΕΚΠΑ


LUTS
BOO
BPO
BPH
BPE


Benign Prostatic Obstruction (BPO)
OAB - detrusor overactivity
And others ...
Nocturnal polyuria
Distal ureteral stone
Detrusor underactivity
LUTS
Bladder tumour
Neurogenic bladder dysfunction
Urethral stricture
Urinary tract infection
Foreign body
Prostatitis

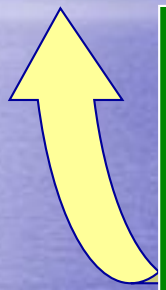
Επιδημιολογία LUTS



Roehrborn CG et al. Prostate Cancer Prostatic Dis 2006;9:30–34

LUTS

More bothersome



Storage

- Daytime urinary frequency
- Nocturia
- Urgency
- Urinary incontinence
- Stress incontinence
- Urge incontinence

Voiding

- Slow stream
- Splitting/spraying
- Intermittency
- Hesitancy
- Straining

Postmicturition

- Feeling of incomplete emptying
- Dribble

More common

Abrams et al. Urology 2003; 61: 37–49; Chapple and Roehrborn Eur Urol 2006; 49: 651–659

Επιδημιολογία BPH

- **Συχνότερη καλοήθης εξεργασία στον άντρα,** 40% θα υποβληθούν σε θεραπεία. Σημασία & η γήρανση.
- Μελέτες νεκροτομικού υλικού έδειξαν BPH στο **50%** στην 6η δεκαετία και στο 90% στην 9η δεκαετία.
- Όλοι αναπτύσσουν ιστολογικές αλλοιώσεις BPH, αλλά **μόνο 50%** αναπτύσσουν κλινικά έκδηλη νόσο.
- **IPSS > 7, Qmax < 15 ml/sec, όγκος > 30 ml** (17% 5η δεκαετία, 27% 6η δεκαετία, 35% 7η δεκαετία).
- Αύξηση επεισοδίων AUR.

Παράγοντες κινδύνου

Non-modifiable	Modifiable
Age	Hormones
Genetics	Testosterone
Geography	Dihydrotestosterone
	Estrogen
	Metabolic syndrome
	Obesity
	Diabetes
	Diet
	Physical activity
	Inflammation

LUTS=Lower urinary tract symptoms, BPH=Benign prostatic hyperplasia, DHT=Dihydrotestosterone

Παράγοντες κινδύνου

- **Ηλικία:** σαφής συσχέτιση με BPH (νεκροτομική διάγνωση 10%, 50%, 80% 4η / 6η / 9η δεκαετία), αύξηση μεγέθους 2-2.5% /y (Baltimore study of age).
- **Γεωγραφία:** Νοτιοανατολική Ασία μικρότερο μέγεθος
- **Γενετική προδιάθεση:** σε ασθενείς που χειρουργούνται για BPH σε ηλικία <60 (στο 50%, 4-6 φορές συχνότερο σε συγγενείς 1ου βαθμού). Μεγαλύτερο μέγεθος σε μικρότερη ηλικία. Μονοζυγώτες δίδυμοι 65% (LUTS)-25% (BPH). Μεταλλάξεις σε ένζυμα αντοχής στο οξειδωτικό stress (GSTE), πολυμορφισμοί AR.
- **Ανδρόπαυση:** μείωση τεστοστερόνης ορού

Παράγοντες κινδύνου

- **ΜΕΤΑΒΟΛΙΚΟ ΣΥΝΔΡΟΜΟ:** παχυσαρκία, υπέρταση, υπερλιπιδαιμία (80% LUTS).
- Αύξηση BMI 1kg/m2 = αύξηση 0.4 cc. BMI >35, 3.5 κίνδυνος για BPH vs <25.
- Αύξηση πιθανότητας Χ/Ο και μειωμένης αποτελεσματικότητας φαρμακοθεραπείας.
- Αυξημένα επίπεδα Ins, IGF-1, IGF bp3.
- **Άσκηση:** μείωση επίπτωσης κατά 25%.
- **Διατροφή:** Vit E, σελήνιο, λυκοπένιο, carotene.
- **Φλεγμονή:** (οξειδωτικό stress, proliferation), αυξημένος κίνδυνος για χειρουργείο

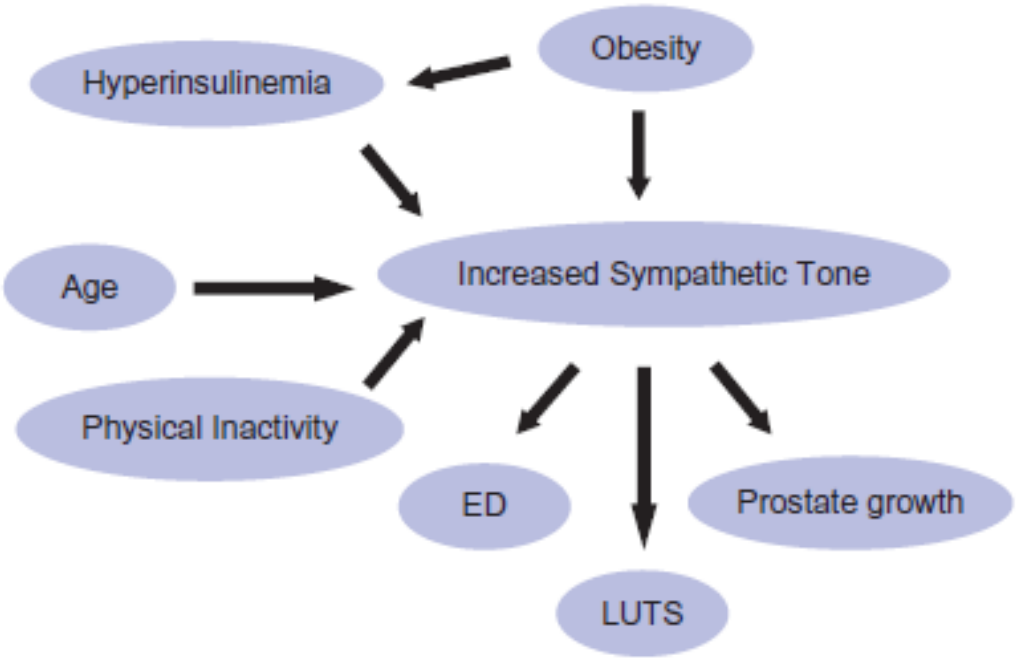
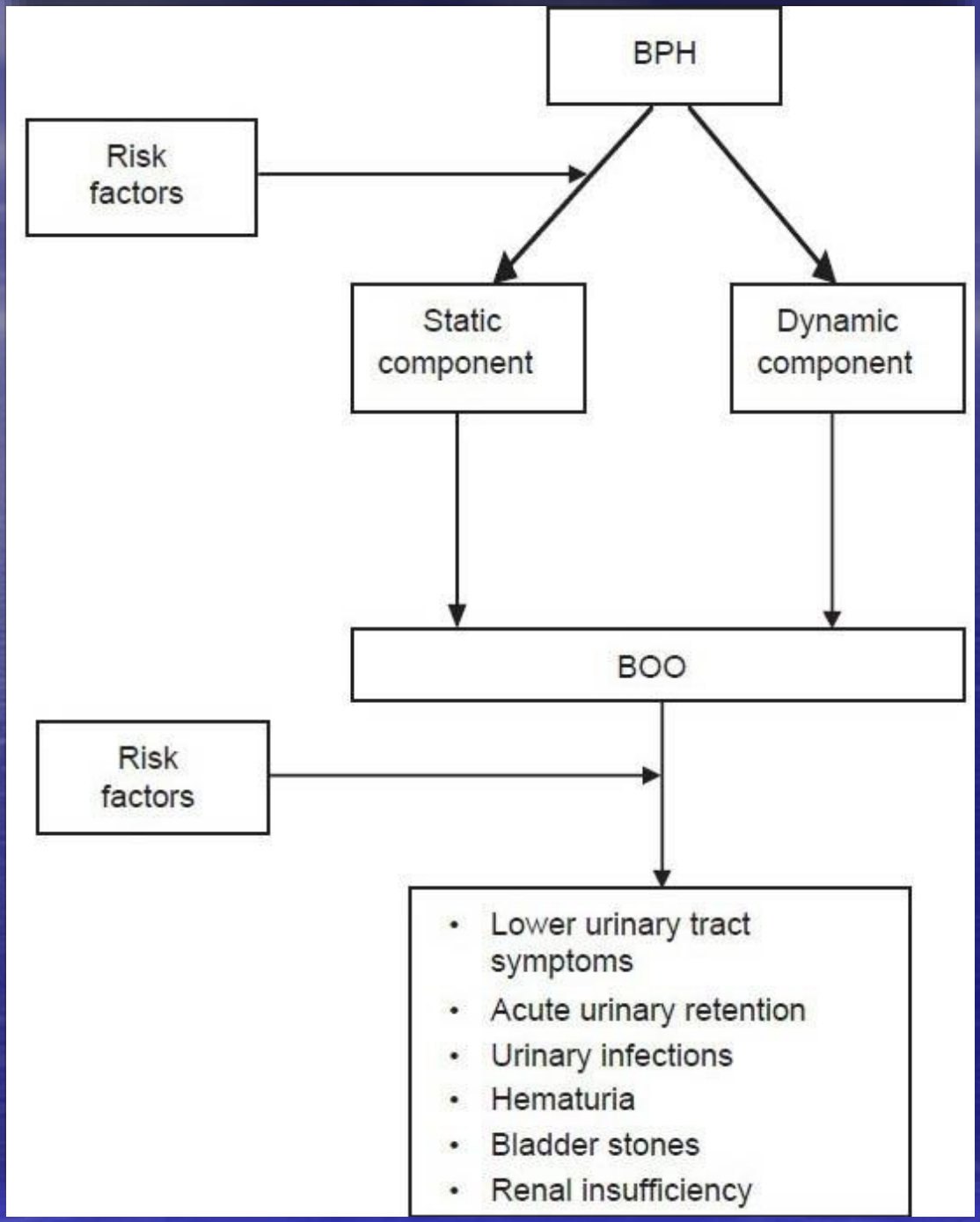


FIG. 3.
The autonomic hyperactivity and metabolic syndrome theory. Increased body mass index, hyperinsulinaemia, increasing age and physical inactivity result in autonomic hyperactivity, which leads to BPH growth, LUTS and ED. Adapted from Persson et al. [32].

Εξέλιξη BPH

- Φάσεις εξέλιξης BPH: 1η με αύξηση αριθμού οζιδίων και 2η με αύξηση μεγέθους οζιδίων (McNeal)
- Μικροοζίδια στη μεταβατική ζώνη (κυρίως αδενικά) και περιουρηθρική ζώνη (κυρίως στρώματος)
- Τα μικροοζίδια αυξάνουν σε αριθμό και μέγεθος και προκαλούν παραμόρφωση ουρήθρας και αποφρακτικά συμπτώματα.
- Εξέλιξη **BOO: στατική** (αύξηση μεγέθους προστάτη), **δυναμική** (αύξηση τόνου λείων μυϊκών ινών προστατικής ουρήθρας)

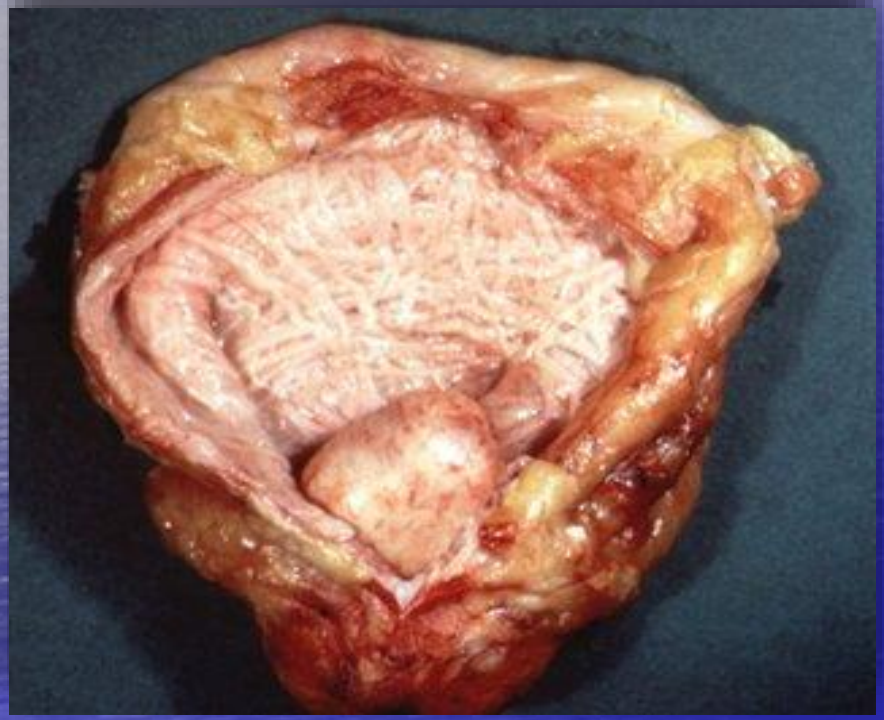

BPH
Risk factors
Static component
Dynamic component
BOO
Risk factors
• Lower urinary tract symptoms
• Acute urinary retention
• Urinary infections
• Hematuria
• Bladder stones
• Renal insufficiency

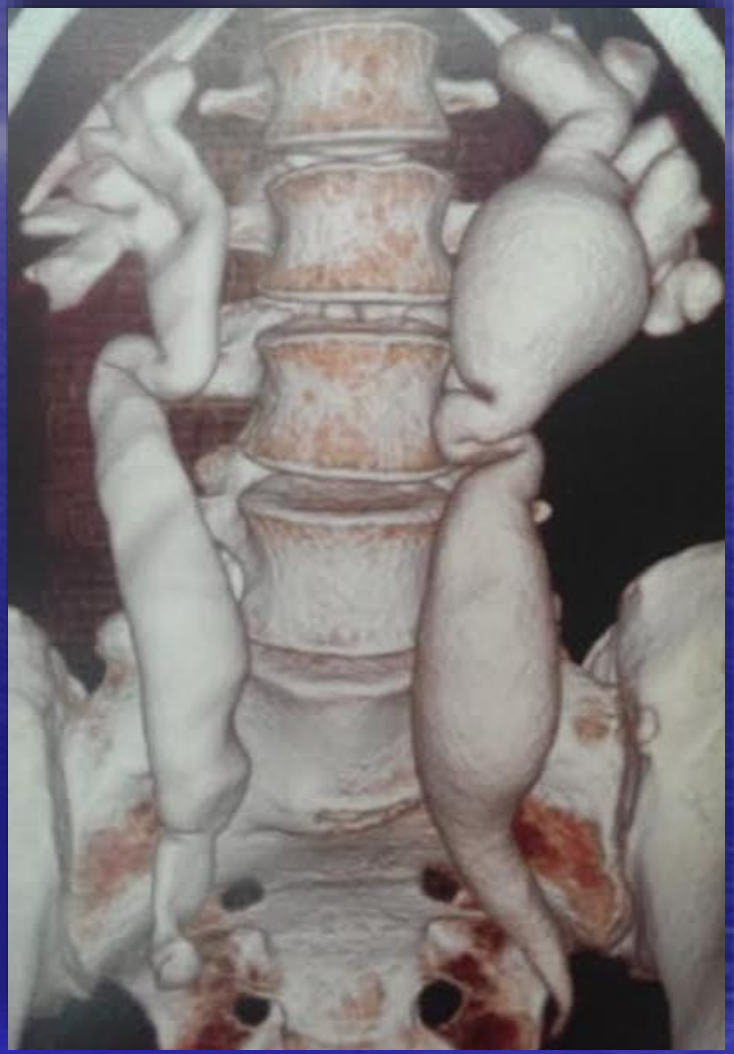
3-stage theory of “prostatism”

- 1ο: υπετροφία κύστης, δοκιδώσεις
- 2ο: εκκολπώματα
- 3ο: λέπτυνση τοιχώματος, υπολειτουργικός εξωστήρας, ακράτεια υπερπλήρωσης, ουρητηρο-υδρονέφρωση

- ***Στάδιο προσαρμογής*: υπερτροφία εξωστήρα για επίτευξη κένωσης κύστης επί αύξησης ουρηθρικής αντίστασης. Αύξηση κολλαγόνου**

- ***Στάδιο μη προσαρμογής*: λέπτυνση εξωστήρα, μειωμένη σύσπαση, υπόλειμμα ούρων, διάταση αποχετευτικής μοίρας**





Μοριακό επίπεδο: προστάτης

- Σχέση στρώματος-αδενικού επιθηλίου (αύξηση από 2:1 σε 4:1).
- Οι λείες μυϊκές ίνες (25-50% στρώματος) έχουν αδρενεργική νεύρωση.
- Σημασία έχει και το αδρενεργικό νευρικό σύστημα (κυρίως μέσω των α_{1A} υποδοχέων) και η δράση άλλων μεταβιβαστών.

Μοριακό επίπεδο: κύστη

- **Aging bladder:** μείωση διατασιμότητας, συσπαστικότητας, υπεραντανακλαστικότητα κύστης

- ***Σε κυτταρικό επίπεδο*:** υπερτροφία μυϊκών κυττάρων, αυξημένη εναπόθεση κολλαγόνου/ελαστίνης, μείωση λειτουργίας μιτοχονδρίων, αναερόβιος μεταβολισμός, μείωση καναλιών Κ, ενεργοποίηση μυοσίνης

Prog Urol 2002;12:569-74

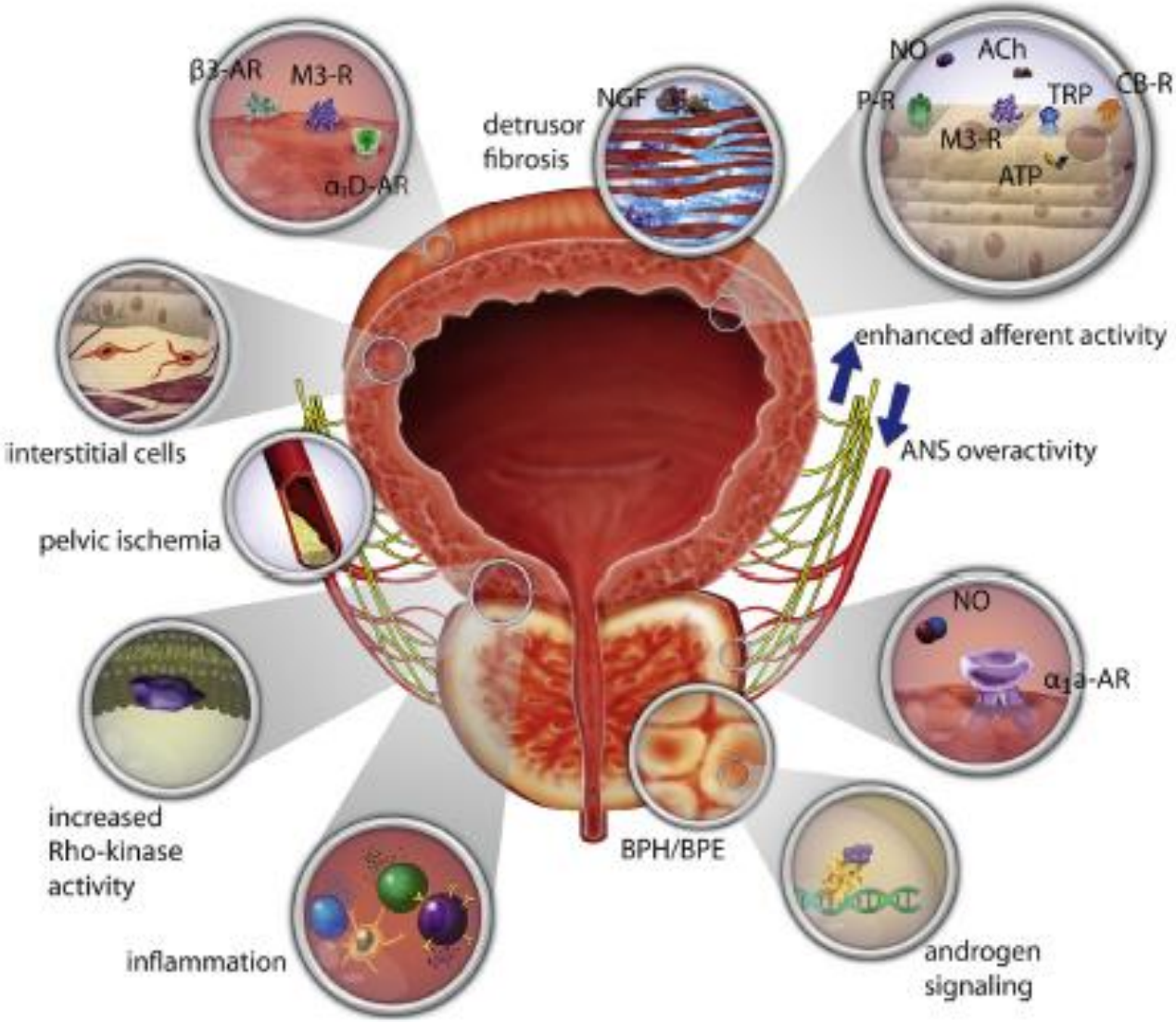


Fig. 1 – Pathophysiologic mechanisms and targets for pharmacotherapy for male lower urinary tract symptoms. Ach = acetylcholine; ANS = autonomic nervous system; ATP = adenosine triphosphate; CB-R = cannabinoid receptors; M3-R = M3 muscarinic receptor; NGF = nerve growth factor; NO = nitric oxide; P-R = purinergic receptors; TRP = transient receptor potential (channels); α_1a-AR = α_1A-adrenoreceptor; α_1D-AR = α_1D-adrenoreceptor; β3-AR = β3-adrenoreceptor.

Urgency, 'the complaint of a sudden compelling desire to pass urine' is the key, defining symptom of the overactive bladder syndrome (OAB).

Abrams P, et al. Neurourol Urodyn 2002; 21: 167–78

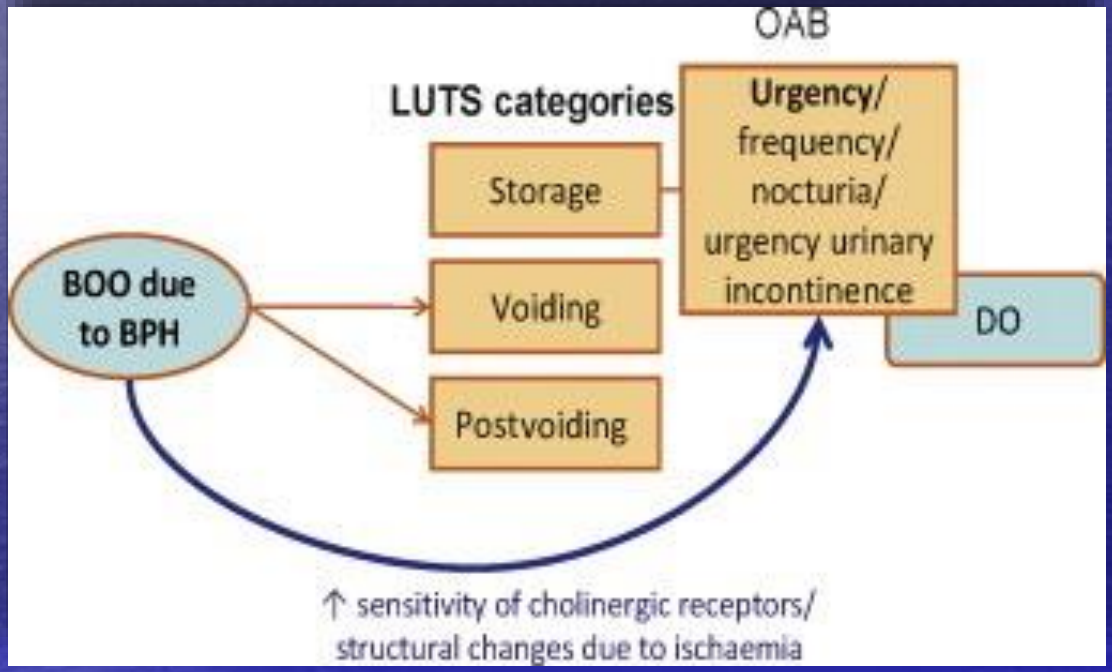
It is urgency itself that leads to frequency, nocturia and urgency incontinence.

The symptom of urgency has a greater impact on health-related quality of life than the other symptoms of OAB, including urgency urinary incontinence.

Chapple CR, BJU Int 2005; 95: 274-5

Chapple CR, BJU Int 2005; 95: 335-340

OAB is a syndrome of storage symptoms defined as urgency, with or without urgency incontinence, usually with increased daytime frequency and nocturia [1,2], and it is often associated with detrusor overactivity (DO) [1].



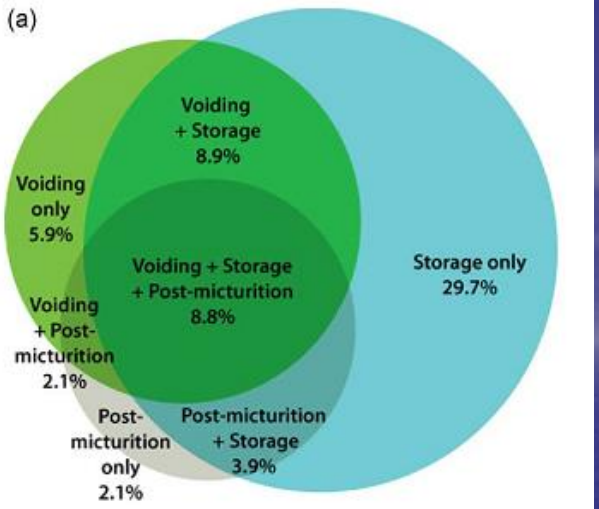
Prevalence, Severity, and Symptom Bother of Lower Urinary Tract Symptoms among Men in the EPIC Study: Impact of Overactive Bladder

Debra E. Irwin [a,*], *Ian Milsom* [b], *Zoe Kopp* [c], *Paul Abrams* [d], *Walter Artibani* [e], *Sender Herschorn* [f]

EUROPEAN UROLOGY 56 (2009) 14–20

Design, setting, and participants: A secondary analysis of data from EPIC, a multinational population-based survey of 19 165 adults, was performed. Current International Continence Society definitions were used for individual LUTS and OAB; OAB cases were defined as men reporting urgency.

Conclusions: Men with LUTS commonly experience coexisting storage, voiding, and post-micturition symptoms, emphasizing the need for comprehensive urologic assessments. Men with OAB symptoms reported more LUTS and greater severity than the general population. Symptom bother was related to number of LUTS and urgency severity.



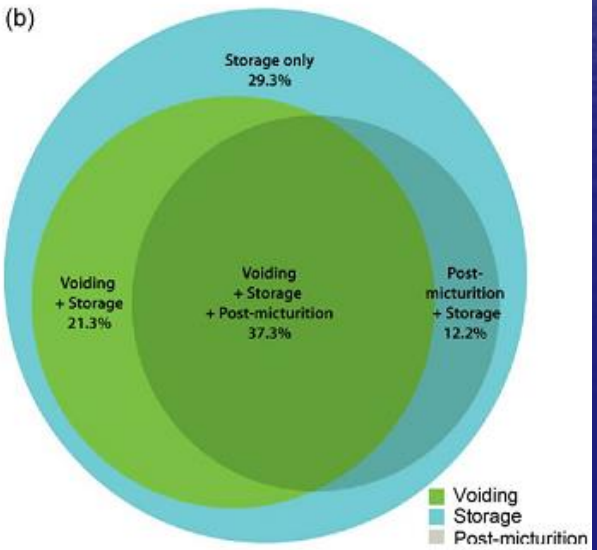


Fig. 1 – Distribution* (%) of lower urinary tract symptoms (LUTS) by type among (a) all men in the general population (n = 7210) and (b) men in the overactive bladder (OAB) population (n = 502). For all men in the general population (a), the total distribution for individual LUTS is indicated with overlapping or nonoverlapping symptoms indicated as the percent in each section. Storage LUTS include the following symptoms: micturition frequency, nocturia, urinary urgency, and urinary incontinence. Voiding LUTS include slow or weak stream, hesitancy, and terminal dribble. Postmicturition LUTS include sensation of incomplete emptying and postmicturition dribble.
***Weighted by age, household size, and country size.**

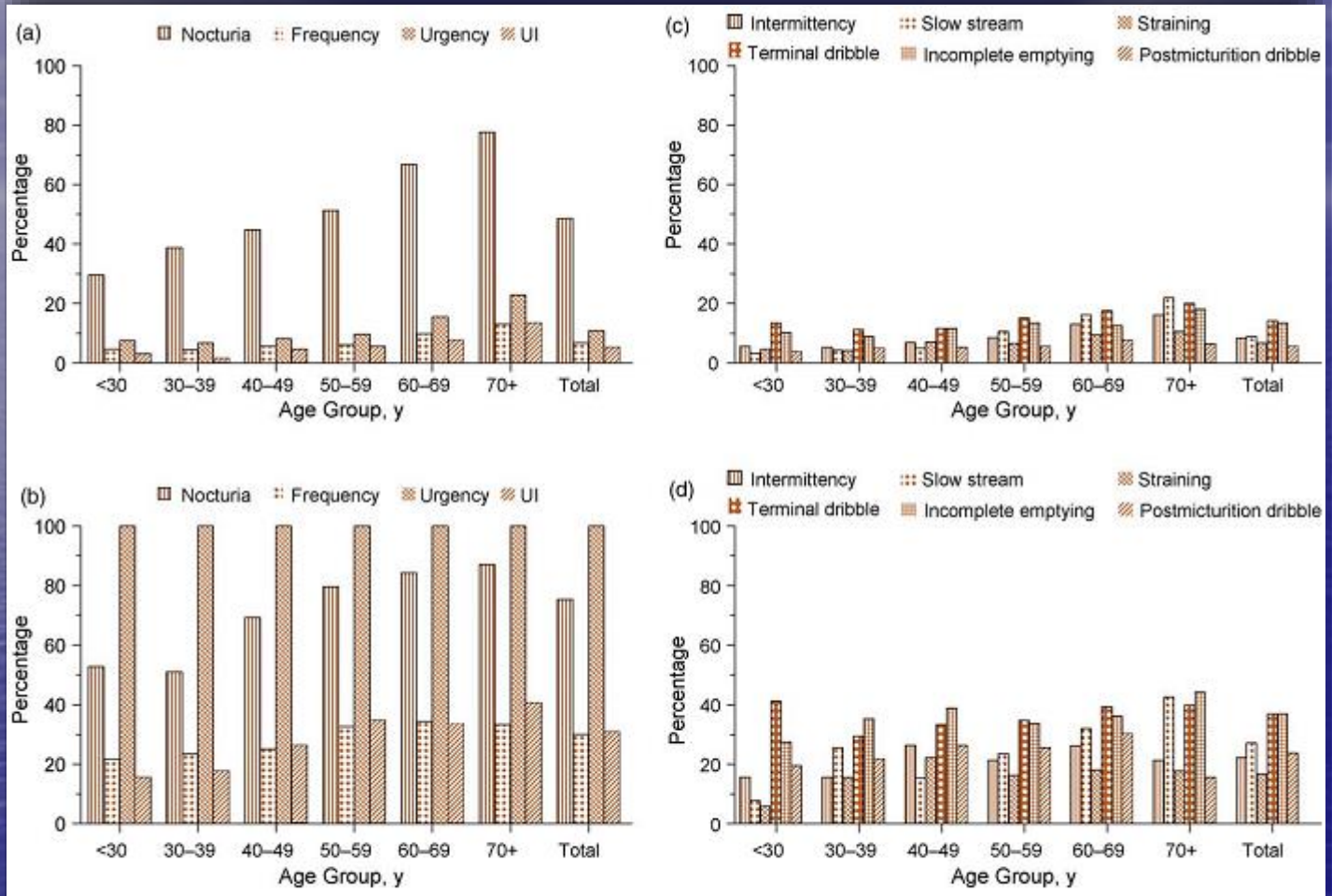


Fig. 2 – Distribution (%) of storage lower urinary tract symptoms (LUTS) by age group among (a) men in the general population ($n = 7210$) and (b) men in the overactive bladder (OAB) population ($n = 502$); distribution (%) of voiding and postmicturition LUTS by age group among (c) men in the general population ($n = 7210$) and (d) men in the OAB population ($n = 502$).
UI = urinary incontinence.

A Contemporary Assessment of Nocturia: Definition, Epidemiology, Pathophysiology, and Management—a Systematic Review and Meta-analysis

Jean-Nicolas Cornu[a,*], *Paul Abrams*[b], *Christopher R. Chapple*[c], *Roger R. Dmochowski*[d], *Gary E. Lemack*[e], *Martin C. Michel*[f], *Andrea Tubaro*[g], *Stephan Madersbacher*[h]

EUROPEAN UROLOGY 62 (2012) 877–890

Evidence synthesis: *Nocturia* is still defined as the symptom of wakening from sleep once or more often to void. The prevalence is high in both genders and increases with age. Frequency–volume charts, which are the pivotal tool of clinical assessment, detect 24-h polyuria, nocturnal polyuria (NP), or reduced nocturnal bladder capacity and help to target specific nonurologic etiologies. Nocturia is a morbid condition that significantly affects quality of life and increases mortality. Besides behavioral measures, validated treatment options include oral desmopressin, which is superior to placebo in treating NP. While the level of evidence for desmopressin is high, limited data support the use of α_1-blockers and antimuscarinics; however, only rarely has nocturia been a primary end point when studying these drug classes, and studies have not consistently controlled for the effect of NP.

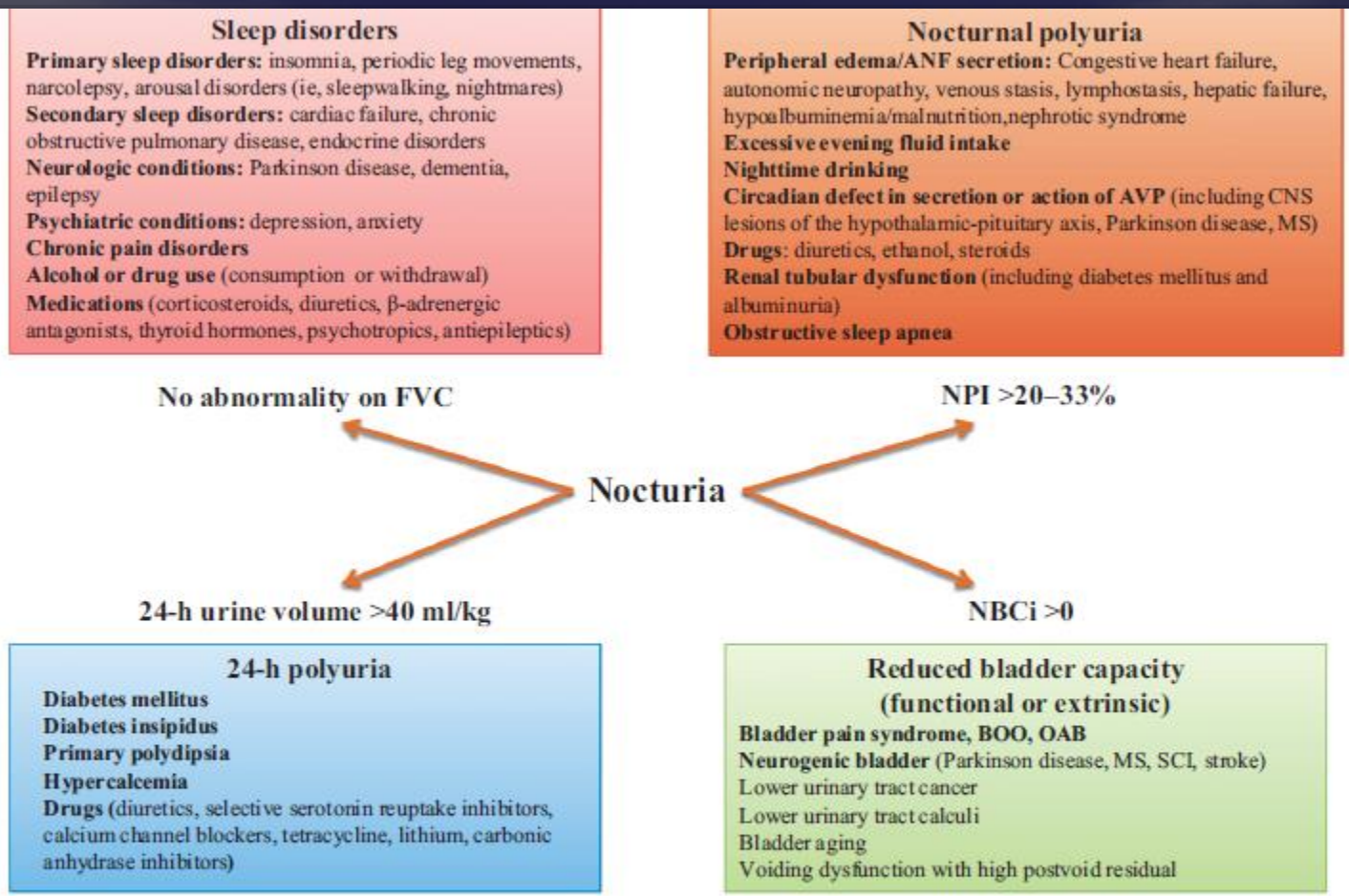


Fig. 2 – Etiologies of nocturia, classified according to the four definitions based on the frequency volume chart (FVC). Sleep disorders have been classified as a cause of nocturia, given the considerations exposed in the International Continence Society document [2].
ANF = atrial natriuretic factor; AVP = arginine vasopressin; BOO = benign outlet obstruction; CNS = central nervous system; MS = multiple sclerosis; NBCi = nocturnal bladder capacity index; NPI = nocturnal polyuria index; OAB = overactive bladder; SCI = spinal cord injury.

Validity and reliability of the patient's perception of intensity of urgency scale in overactive bladder

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Rufus Cartwright, Sushma Srikrishna, Linda Cardozo and Dudley Robinson
Department of Urogynaecology, King's College Hospital, London, UK

BJU INTERNATIONAL © 2010 BJU INTERNATIONAL | 107, 1612–1617 | doi:10.1111/j.1464-410X.2010.09684.x

Indevus Urgency Severity Scale	Sensation-Related Bladder Diary	Patient Perception of Intensity of Urgency Scale
0: NONE – no urgency	Grade 1 – no desire to void (convenience void)	0 – No urgency: I felt no need to empty my bladder but did so for other reasons
1: MILD – awareness of urgency, but it is easily tolerated and you can continue with your usual activity or tasks	Grade 2 – desire to void but voiding can be delayed for at least 30 min	1 – Mild urgency: I could postpone voiding for as long as necessary without fear of wetting myself
2: MODERATE – enough urgency discomfort that it interferes with or shortens your usual activity or tasks	Grade 3 – desire to void but voiding can not be delayed for more than 15 min	2 – Moderate urgency: I could postpone voiding for a short while without fear of wetting myself
3: SEVERE – extreme urgency discomfort that abruptly stops all activity or tasks	Grade 4 – desire to void but voiding can not be delayed for more than 5 min	3 – Severe urgency: I could not postpone voiding but had to rush to the toilet in order not to wet myself
		4 – Urge incontinence: I leaked before arriving at the toilet

	Rating of each void	Interpretation for analysis
0	No urgency: I felt no need to empty my bladder but did so for other reasons	Convenience voids
1	Mild urgency: I could postpone voiding for as long as necessary without fear of wetting myself	Voids with normal urge sensation
2	Moderate urgency: I could postpone voiding for a short while without fear of wetting myself	
3	Severe urgency: I could not postpone voiding but had to rush to the toilet in order not to wet myself	Voids with urgency
4	Urge incontinence: I leaked before arriving at the toilet	Voids with urge incontinence

Reviews

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Total urgency and frequency score as a measure of urgency and frequency in overactive bladder and storage lower urinary tract symptoms

Christopher R. Chapple, Marcus J. Drake*, Philip Van Kerrebroeck†, Linda Cardozo‡, Ted Drogendijk§, Monique Klaver§, Karin Van Charldorp§, Zalmai Hakimi¶ and Gerhard Compion¶

BJU Int 2014; **113**: 696–703
wileyonlinelibrary.com

BJU International © 2013 BJU International | doi:10.1111/bju.12555
Published by John Wiley & Sons Ltd. www.bjui.org

	IUSS	URS	PPIUS	Urgency perception scale	Urgency perception score
Tool overview	Four-point scale used with 7-day micturition diary	Five-point scale used with 7-day micturition diary	Five-point scale used with micturition diary	Three-point scale of responses when asked to describe their typical experience when they feel the desire to pass urine/	Four-point scale in response to one question: What is the reason that you usually urinate?
Used in drug development programme	Trospium chloride	Tolterodine extended-release Tolterodine and tamsulosin	Transdermal oxybutynin, mirabegron and solifenacin	Tolterodine, Solifenacin plus tamsulosin	None published
Validated	Patients with OAB		Volunteers Patients with OAB Patients with LUTS		Volunteers Patients with LUTS Patients with OAB
Responses	0: None – no urgency	1: No urgency	0: No urgency – I felt no need to empty my bladder, but did so for other reasons	1: I am usually not able to hold urine [urgency incontinence]	0: Out of convenience (no urge)
	1: Mild – awareness of urgency, but it is easily tolerated and you can continue with your usual activity or task	2: Mild urgency	1: Mild urgency – I could postpone voiding as long as necessary, without fear of wetting myself	2: I am usually able to hold urine until I reach the toilet if I go immediately [urgency]	1: Because I have a mild urge (but can delay urination for over an hour if I have to)
	2: Moderate – enough urgency discomfort that it interferes with or shortens your usual activity or tasks	3: Moderate urgency	2: Moderate urgency – I could postpone voiding for a short while, without fear of wetting myself	3: I am usually able to finish what I am doing before going to the toilet [first desire to void]	2: Because I have a moderate urge (but can delay urination for >10 but <60 min if I have to)
	3: Severe – extreme urgency discomfort that abruptly stops all activity or tasks	4: Severe urgency	3: Severe urgency – I could not postpone voiding, but had to rush to the toilet in order not to wet myself		3: Because I have a severe urge (but can delay urination for <10 min)
		5: Urgency incontinence	4: Urge incontinence – I leaked before arriving at the toilet		4: Because I have desperate urge (must stop what I am doing and go immediately)

COMBINATION TREATMENT WITH AN α-BLOCKER PLUS AN ANTICHOLINERGIC FOR BLADDER OUTLET OBSTRUCTION: A PROSPECTIVE, RANDOMIZED, CONTROLLED STUDY

A. ATHANASOPOULOS, K. GYFTOPOULOS, K. GIANNITSAS, J. FISFIS, P. PERIMENIS AND G. BARBALIAS

THE JOURNAL OF UROLOGY® Vol. 169, 2253–2256, June 2003

Materials and Methods: The study included 50 consecutive patients with urodynamically proven mild or moderate bladder outlet obstruction and concomitant detrusor instability. All patients were initially treated with 0.4 mg. tamsulosin orally once a day. A week later the patients were randomly allocated into group 1—25 who continued treatment with tamsulosin only and, group 2—25 who also received 2 mg. tolterodine orally twice daily. Reevaluation with a quality of life questionnaire and urodynamic study was performed after 3 months.

Conclusions: Combination treatment with an α-blocker (tamsulosin) plus an anticholinergic (tolterodine) improves quality of life in patients with bladder outlet obstruction and concomitant detrusor instability. Interestingly, no acute urinary retention was observed and tolterodine did not affect the quality of urine flow or residual urine volume. The proposed combination appears to be an effective and relatively safe treatment option in patients with bladder outlet obstruction and detrusor instability.

Future Direction in Pharmacotherapy for Non-neurogenic Male Lower Urinary Tract Symptoms

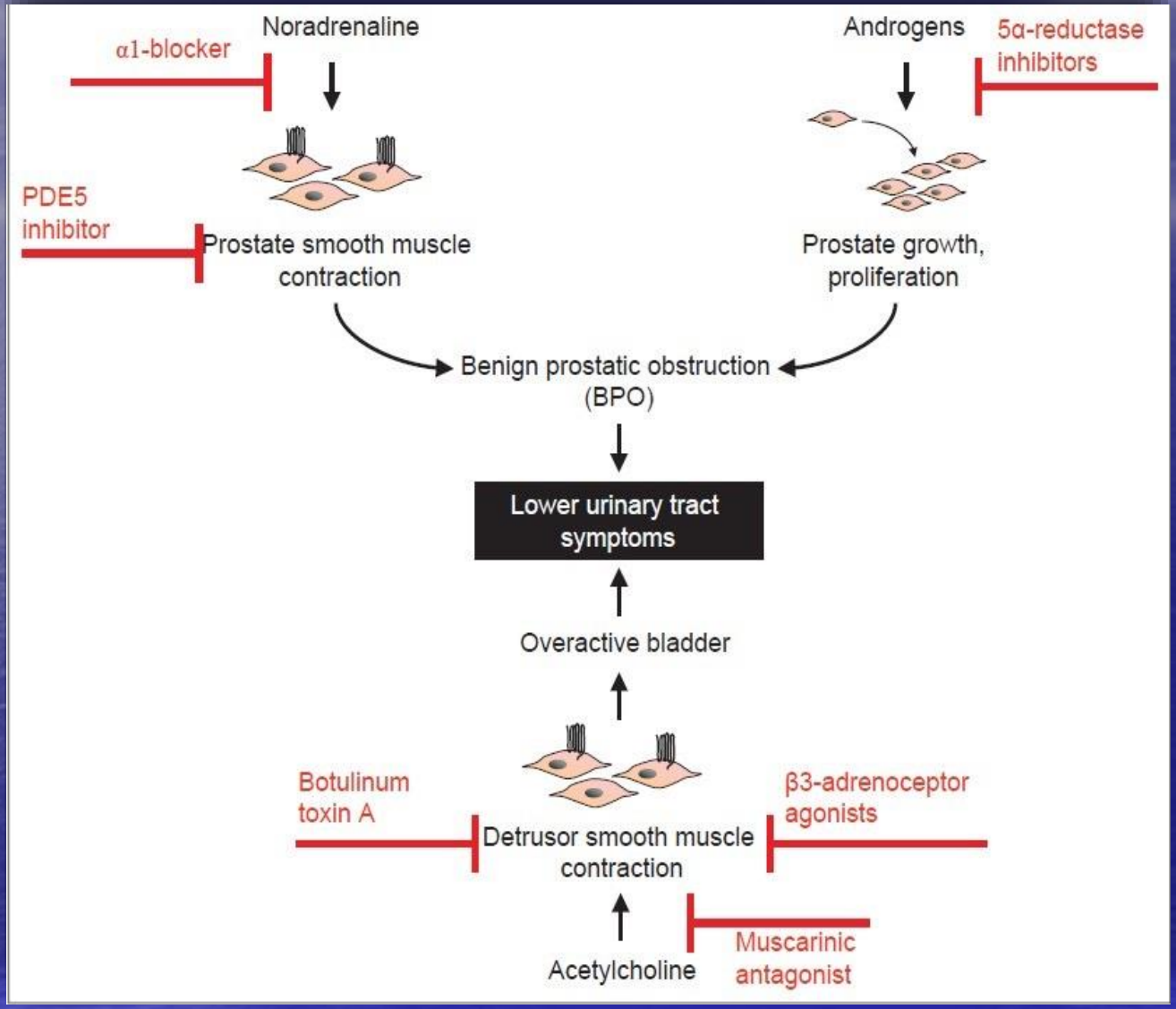
EUROPEAN UROLOGY 64 (2013) 610–621

Roberto Soler[a], *Karl-Erik Andersson*[b], *Michael B. Chancellor*[c], *Christopher R. Chapple*[d], *William C. de Groat*[e], *Marcus J. Drake*[f], *Christian Gratzke*[g], *Richard Lee*[h], *Francisco Cruz*[i,*]

Table 1 – Future pharmacotherapy for non-neurogenic male lower urinary tract symptoms, their respective targets

Class of drugs	Target	Phase of studies
Antimuscarinics	M3 muscarinic receptors	Clinical practice
Phosphodiesterase inhibitors	NO/cGMP	Clinical practice
	ROK	
	Autonomic nervous system	
	Afferent nerves	
	Blood perfusion	
	Inflammation	
β3-agonists	β3-adrenergic receptor	Phase 2 (completed)
		Clinical practice
Botulinum toxin	Acetylcholine release	Clinical practice
	Prostate tissue	Phase 3 (currently)
LHRH antagonists	Testosterone	Phase 3 (completed)
Cannabinoids	Cannabinoid receptors	Phase 3 (completed)
NX-1207	Prostate tissue	Phase 3 (currently)
PRX-302	Prostate tissue	Phase 2 (completed)
Vitamin D3 receptor analogs	Vitamin D3 receptors	Phase 2 (completed)
Anti-inflammatory drugs	Inflammation	Phase 1
Transient receptor potential channel blockers	Transient receptor potential channels	Experimental
Rho-kinase inhibitors	ROK	Experimental
Purinergic receptor blockers	Purinergic receptors	Experimental
Endothelin-converting enzyme inhibitors	Detrusor muscle	Experimental

cGMP = cyclic guanosine triphosphate; LHRH = luteinizing hormone-releasing hormone; NO = nitric oxide; ROK = RhoA/Rho kinase.


Noradrenaline
α1-blocker
Androgens
5α-reductase inhibitors
PDE5 inhibitor
Prostate smooth muscle contraction
Prostate growth, proliferation
Benign prostatic obstruction (BPO)
Lower urinary tract symptoms
Overactive bladder
Botulinum toxin A
β3-adrenoceptor agonists
Detrusor smooth muscle contraction
Muscarinic antagonist
Acetylcholine

LUTS can be divided into storage, voiding and post-micturition symptoms [2]. LUTS are prevalent, cause bother and impair QoL [5-8]. They are strongly associated with ageing [5, 6], so associated costs and burden are likely to continue to increase overall in the future [5, 9].

Most elderly men report having at least one LUTS [6]. However, clinically meaningful prevalences are lower as symptoms may be mild or not very bothersome [8]. LUTS progress dynamically: for some individuals LUTS persist and progress over long time periods, and for others they remit [5].

LUTS have traditionally been related to bladder outlet obstruction (BOO), which is often caused by benign prostatic enlargement (BPE) resulting from the histologic condition of benign prostatic hyperplasia (BPH) [2, 7]. Recent studies have shown, however, that LUTS are often unrelated to prostate [3, 6]. Bladder dysfunction may also cause LUTS, including detrusor overactivity/OAB, detrusor underactivity/underactive bladder, as well as other structural or functional abnormalities of the urinary tract [3]. In addition, many non-urological conditions also contribute to LUTS, especially nocturia

Guidelines on the Management of Non-Neurogenic Male Lower Urinary Tract Symptoms (LUTS), incl. Benign Prostatic Obstruction (BPO)

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Autonomic nervous system activity assessement by heart rate variability in experimental bladder outlet obstruction

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female rats, divided into two groups: BOO animals (n=15), with surgically induced BOO (by partial ligation of the proximal urethra) and control ones (n=15), which underwent sham procedure (without urethral ligation). Two weeks after the surgery, in both groups, ANS activity was estimated using time- and spectral analysis of the heart rate variability recordings. The bladder overactivity in BOO animals was confirmed using urodynamic recordings and bladder histological assessment, juxtaposed against the results of the control group. The key finding of our study was the development of autonomic disturbances in bladder outlet obstruction (BOO) rats. Our study revealed that BOO animals were characterised by diminished rMSSD and spectral HRV parameters: TP, LF and HF, in comparison with the control group. The normalised nLF and nHF parameters did not differ significantly in both groups, although slight changes in the nLF (increased) and nHF (decreased) were noted in BOO group. The absolute VLF value was almost the same in both studied populations, however, the percentage part of this component in the appropriate HRV spectrum differed considerably in both studied groups. In BOO animals, VLF percentage amounted to about 90%, whereas in control animals this parameter reached only about 53% of the total power spectrum.

Thus, to sum up, our findings suggest autonomic imbalance with decreased global autonomic tension and diminished parasympathetic activity with relatively sympathetic overactivity.

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THE PATHOPHYSIOLOGY UNDERLYING OVERACTIVE BLADDER SYNDROME POSSIBLY DUE TO BENIGN PROSTATIC HYPERPLASIA

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The pathophysiology in the development of overactive bladder syndrome (OAB) possibly due to benign prostatic hyperplasia (BPH) has not fully been understood. The clinical study in male outpatients aged over 50 years with lower urinary tract symptoms showed that the frequency of urgency was significantly associated with aging, bladder outlet obstruction (BOO) and benign prostatic enlargement (BPE). From the results of the experiments we did using rats, the mechanisms underlying the development of OAB were suggested as follows. The functional impairment of acetylcholine neuron in the central nervous system is induced by aging and decreases the bladder capacity. Non-voiding contractions of the bladder may have some bearing on OAB associated with BOO. The C-fiber in the urethra may be involved in the generation of the detrusor overactivity associated with BPE. These results showed that the pathophysiology of OAB related to BPH is quite complex, suggesting that a multidisciplinary approach is necessary for the treatment.

(Hinyokika Kiyo **54** : 449–452, 2008)