Urinary Incontinence in the Elderly: Impact of Neurogenic bladder

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Neurogenic Bladder (NGB)

- Disorder of LUT function caused by an abnormality of innervation
- Not all patients with neurologic diseases & incontinence have NGB
- Patients without neurologic diseases may have incontinence due to subtle unrecognized neurologic abnormalities

Significance

- Neurogenic bladder plays an important role in our understanding of urinary incontinence in the elderly
- High prevalence of NGB in the elderly
- Underlying neurologic disorder can not only be the cause of incontinence, but also can pose a major obstacle to its treatment

11% → 25%	Prevalence	Incidence	Prevalence Incontinence
Alzheimer's Disease	5,400,000[19]		60-70%[20]
Stroke	4,500,000[1] (survivors)	795,000[8] (600,00 new strokes)	36%[12]
Parkinson's disease	1,000,000[2]	60,000[2]	27%[13]
Normal pressure hydrocephalus	750,000[3]	2 – 20 /million[9]	95%[14]
Multiple sclerosis	400,000[4]	10,000[4]	79%[15]
Spinal cord injury	276,000[5]	12,000[10]	81%[16] (bladder dysfunction)
Lumbar Spinal Stenosis	8,500,000[6]		50%[17] (bladder dysfunction)
Motor Neuron Disease	20,000[7]	5,600[11]	36-50%[18]

1. CDC; 2. Parkinson's Disease Foundation; 3. Ettinger; Overview of NPH. 2015; 4. Hersh; Multiple Sclerosis. 2014; 5. SCIMS; 6. Matt, Spine Lumbar; Spine U, 2009; 7. Mehta, Surveilence Summarries. 2014; 8. strokecenter.org; 9. Radford, NPH. 2016; 10. NSCIS; 11. UCSD; 12. Brittain, Stroke and Incontinence. 1998; 13. Siegl, Pflege Z. 2013; 14. Shprecher, Current Neurology and Neuroscience Reports. 2008; 15. Khalaf, Internt'l Journal of MS Care. 2015; 16. Ginsberg, AJMC. 2013; 17. Wyndaele, Neurologic Urinary and Feacal Incontinence; 18. Nubling, ALS Journal. 2014; 19. alz.org; 20. b&bf

Impact of Neurologic Disease on Incontinence

- **Reduced cognitive function:**
  - Decreased awareness & concern about bladder sensations & events
  - Loss of voluntary sphincter control
  - Difficulty complying with treatment strategies
- **Reduced mobility**
  - Getting to bathroom in time
- **Poor hand function**
  - Intermittent catheterization, appliances & pads

State-of-the-Art Knowledge

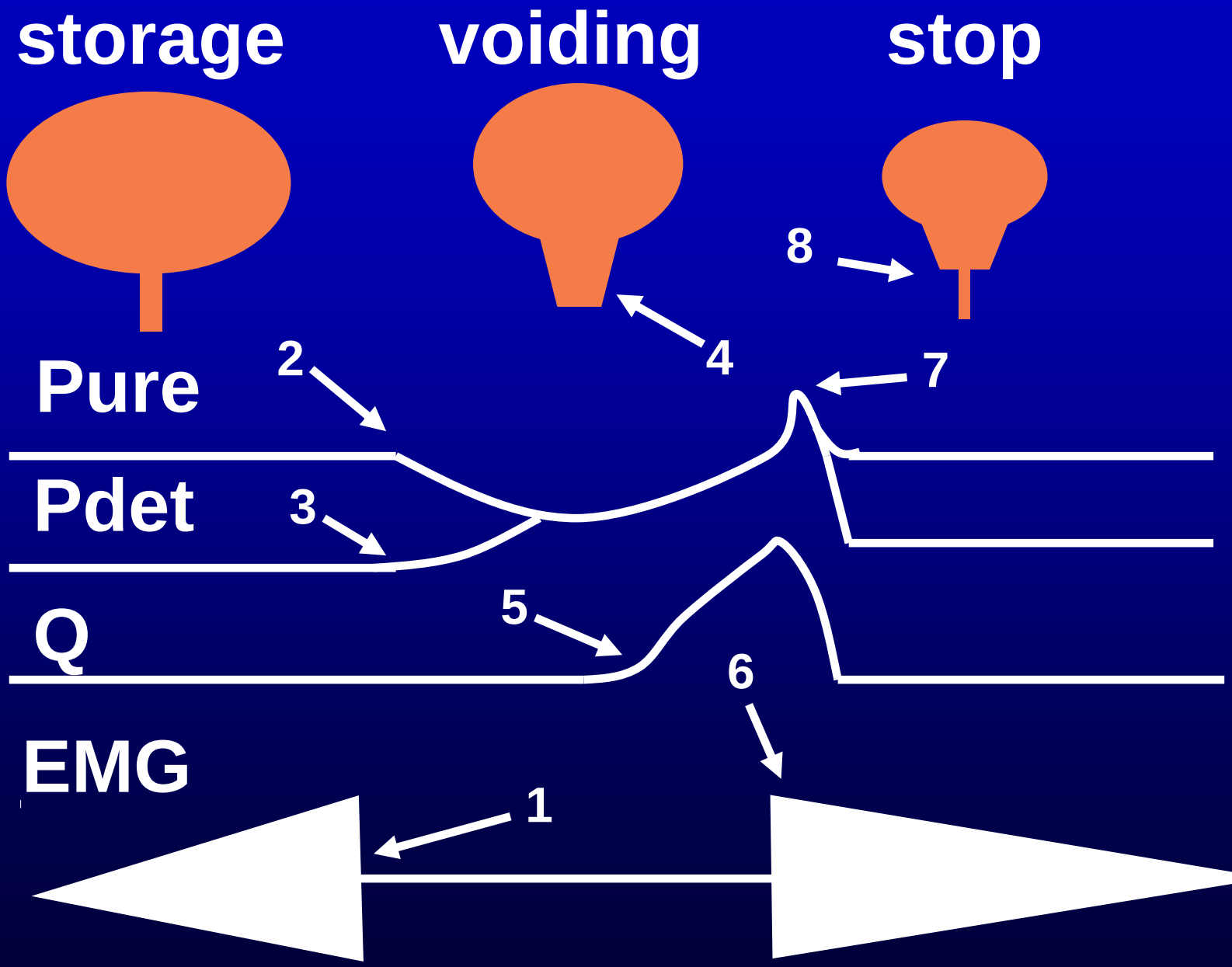
- Pathophysiology of incontinence
- Physiology of micturition
- Neurophysiology of micturition
- Efficacy of incontinence treatments

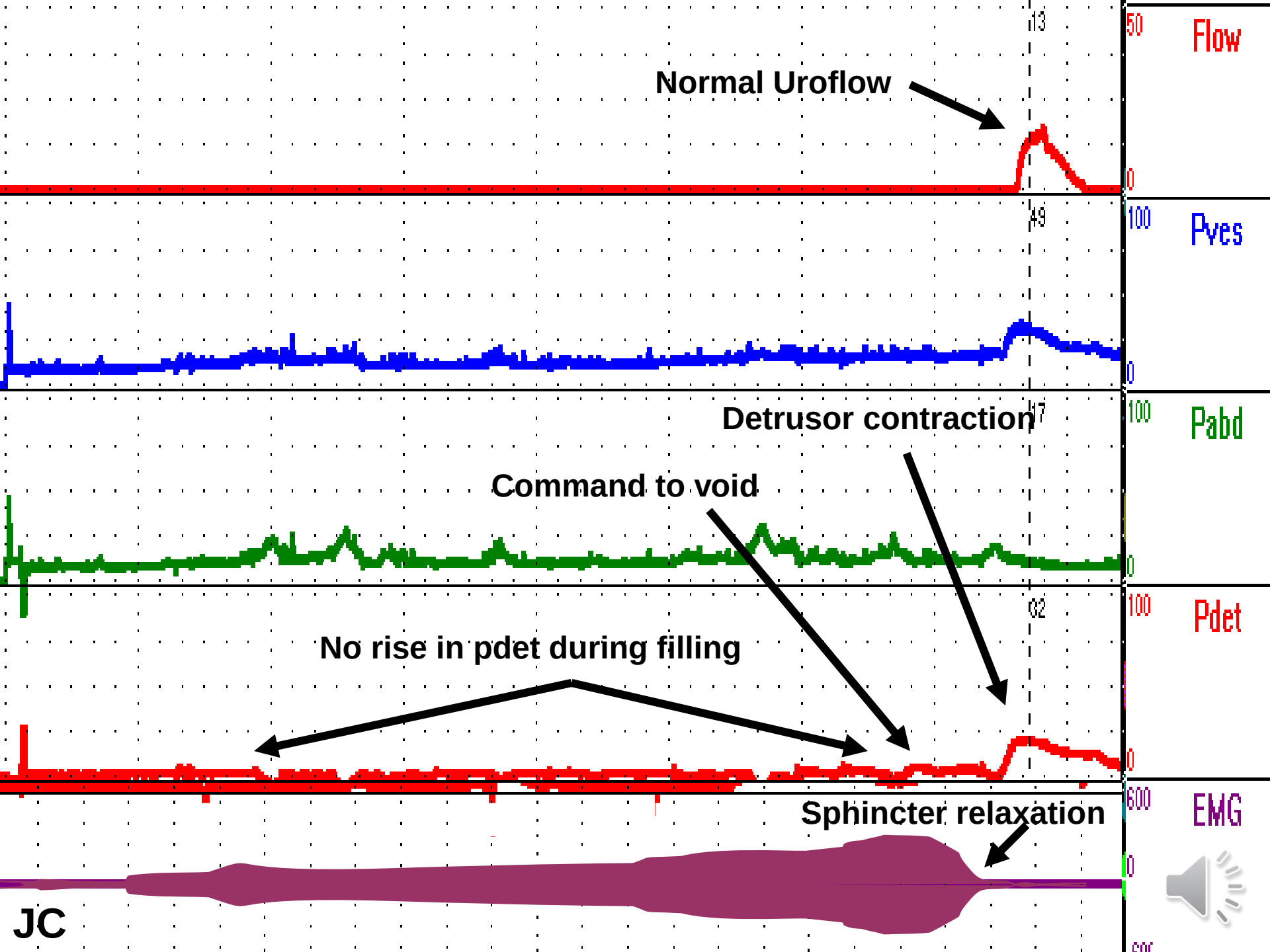
Causes of Incontinence

- **Bladder (common)**
  - **Involuntary detrusor contractions (IDO, NDO)***
  - **Cognitive or sensory abnormalities > uncontrollable voiding due to lack of awareness or concern**
  - **UTI**
- **Sphincter (rare)**
  - **Neurogenic (e.g. thoraco-lumbar lesions)***
  - **Non-neurogenic (e.g. ISD*, urethral hypermobility)**
- **Extra-urethral (very rare) e.g. fistula, ectopic ureter**

*neurogenic etiology

Physiology of Micturition




Normal Uroflow
Detrusor contraction
Command to void
No rise in pdet during filling
Sphincter relaxation
Flow
Pves
Pabd
Pdet
EMG
50
0
100
0
100
0
100
0
600
0
JC

Neurophysiology

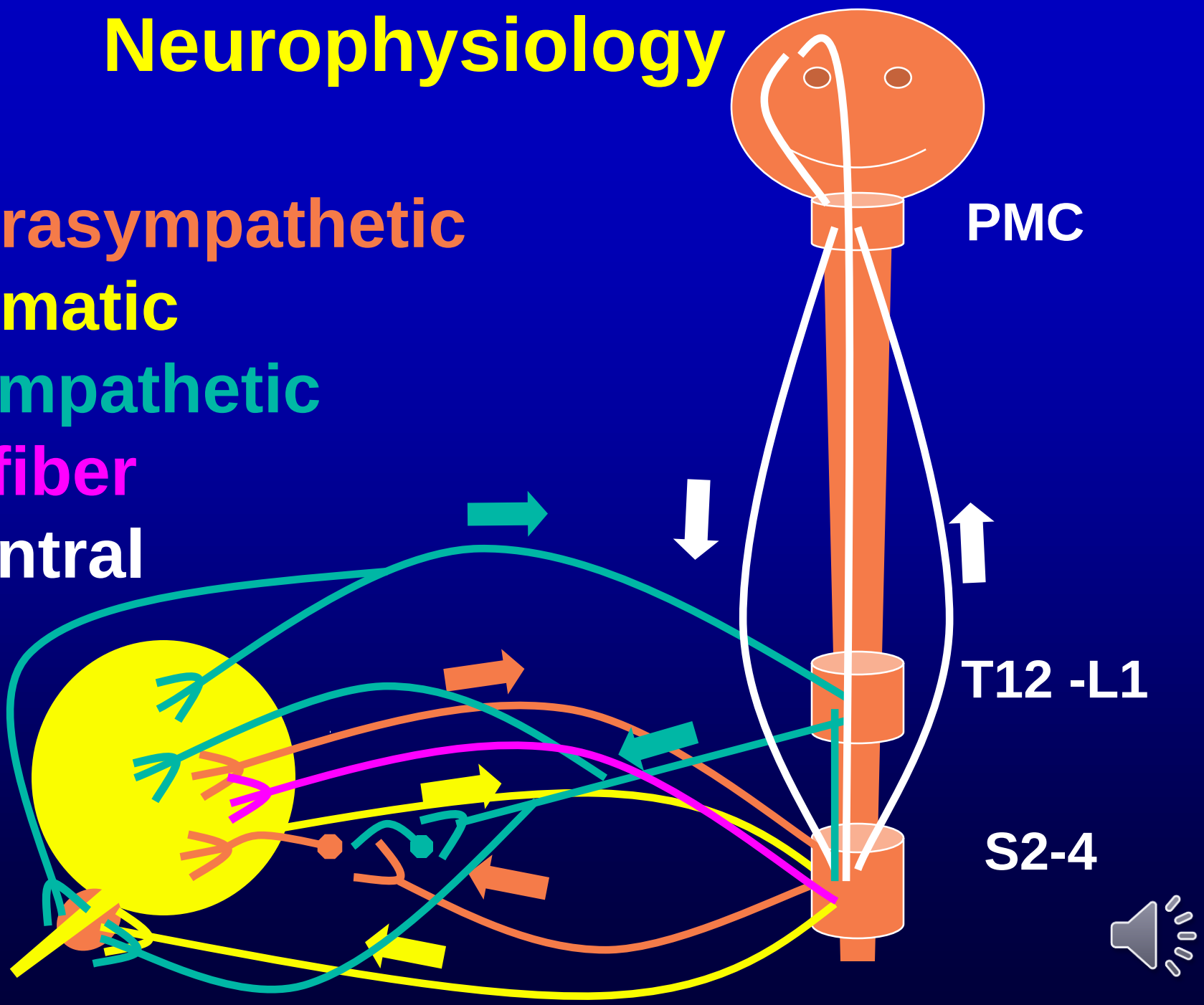
parasympathetic

somatic

sympathetic

C fiber

central



Sacral Micturition Center

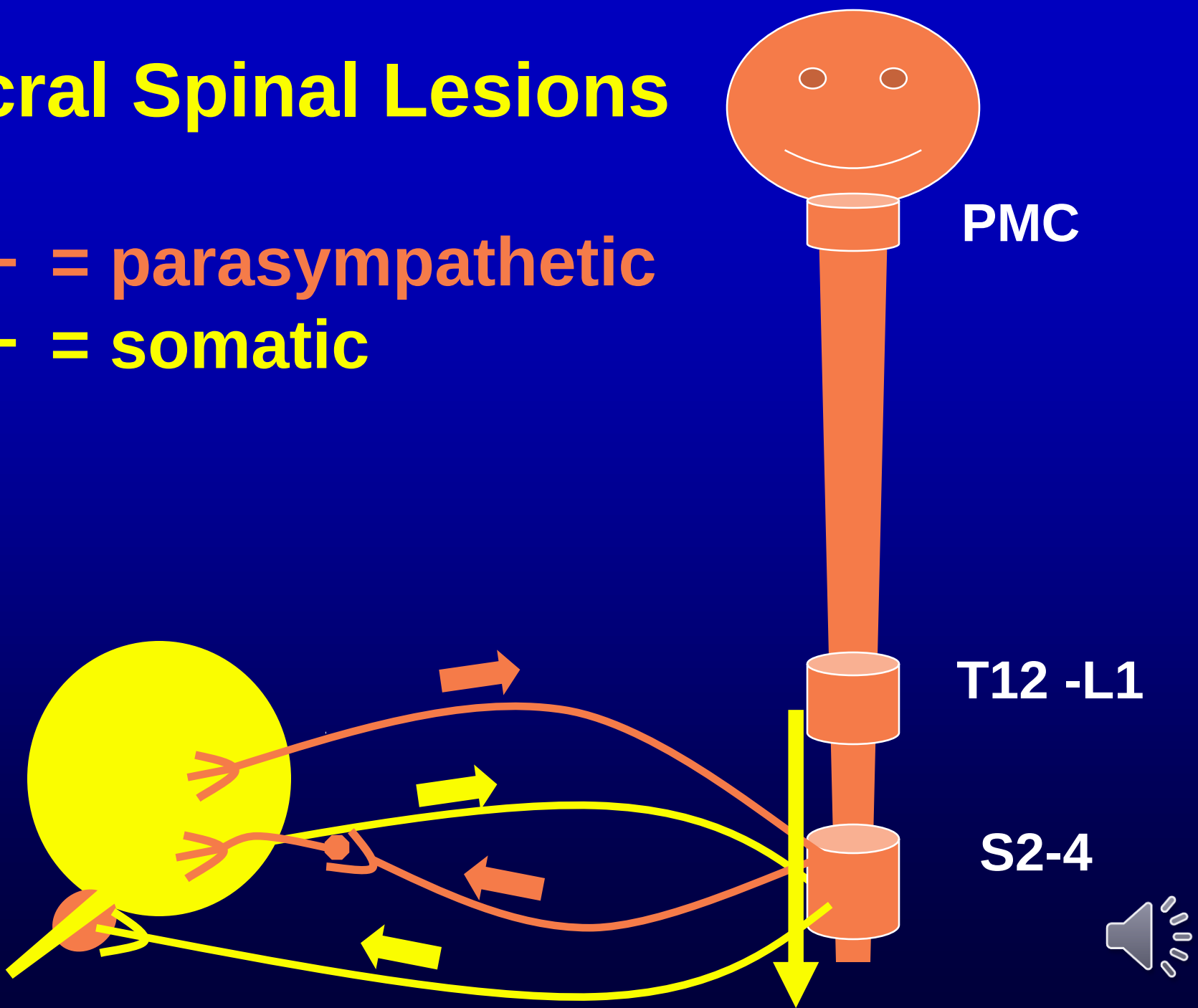
- Primitive micturition reflex center
- Parasympathetic (pelvic nerve)
  → detrusor contraction
- Somatic (pudendal nerve)
  → sphincter contraction

Detrusor external sphincter dyssynergia

Sacral Spinal Lesions

— = parasympathetic

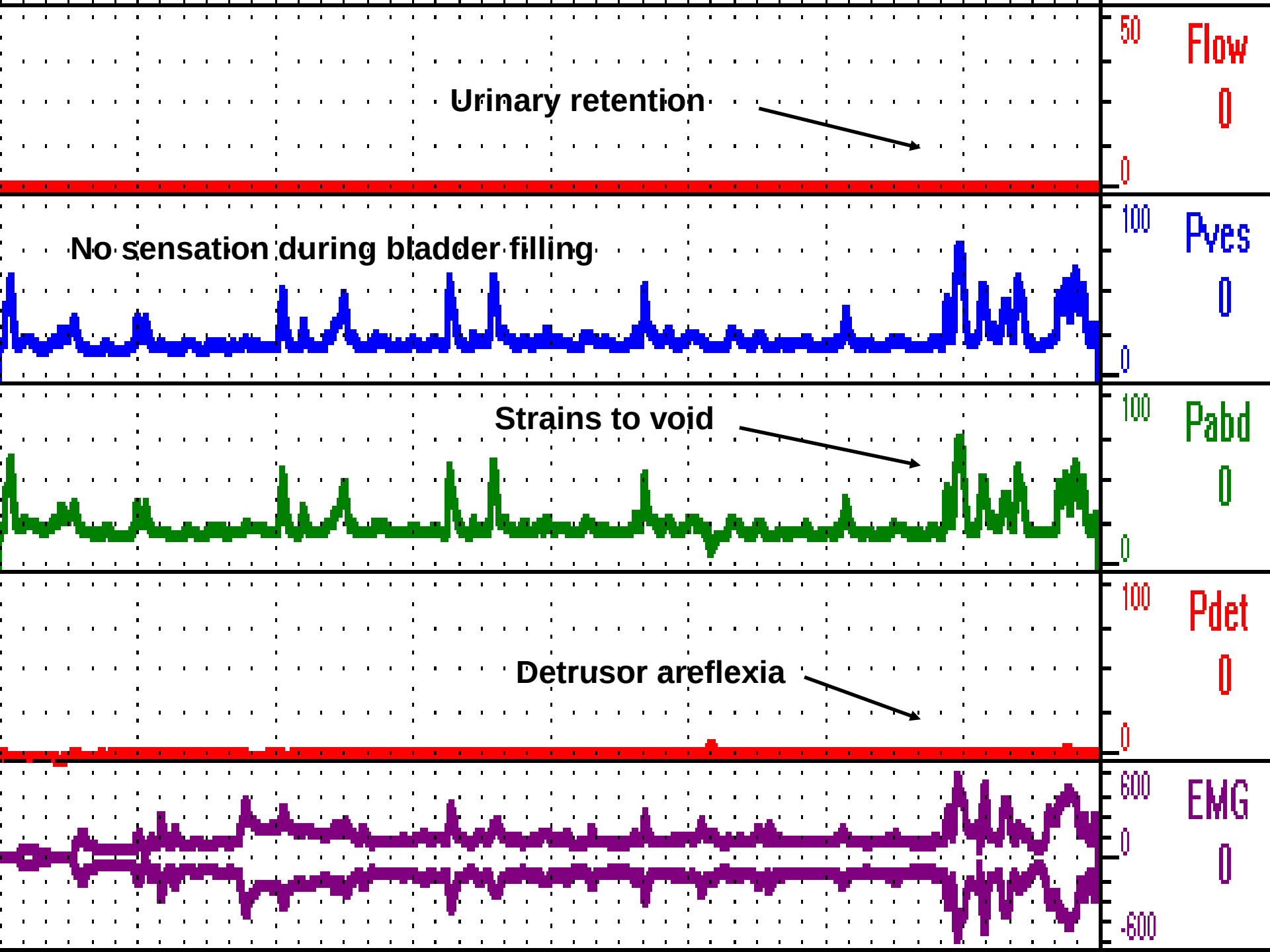
— = somatic



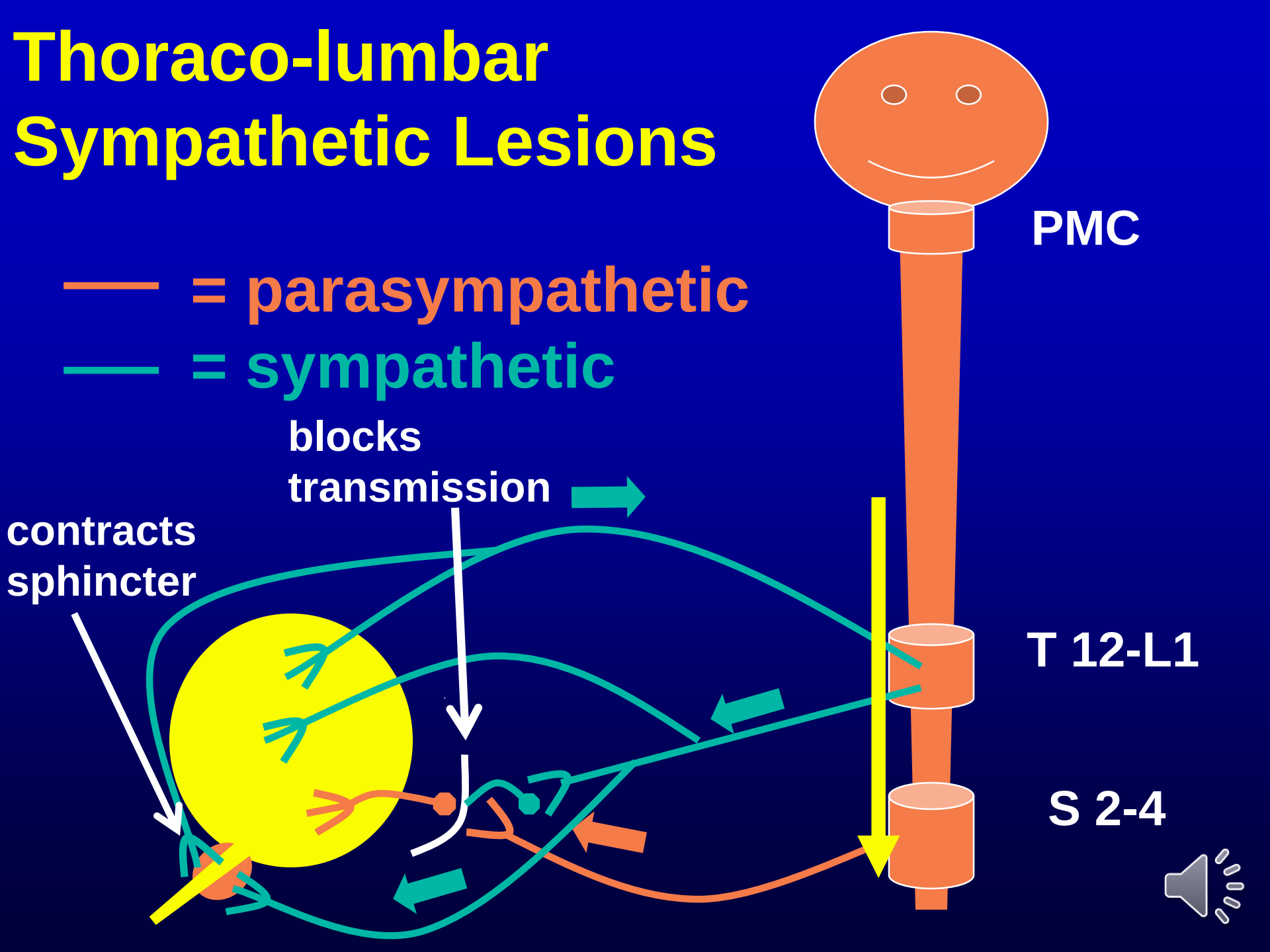
Sacral Spinal Lesions

- **Loss of micturition reflex**
- **Detrusor areflexia**
- **Smooth & striated muscles of the urethra keep the sphincter closed**

Urinary retention


Urinary retention
No sensation during bladder filling
Strains to void
Detrusor areflexia
50
Flow
0
0
100
Pves
0
0
100
Pabd
0
0
100
Pdet
0
0
600
EMG
0
-600

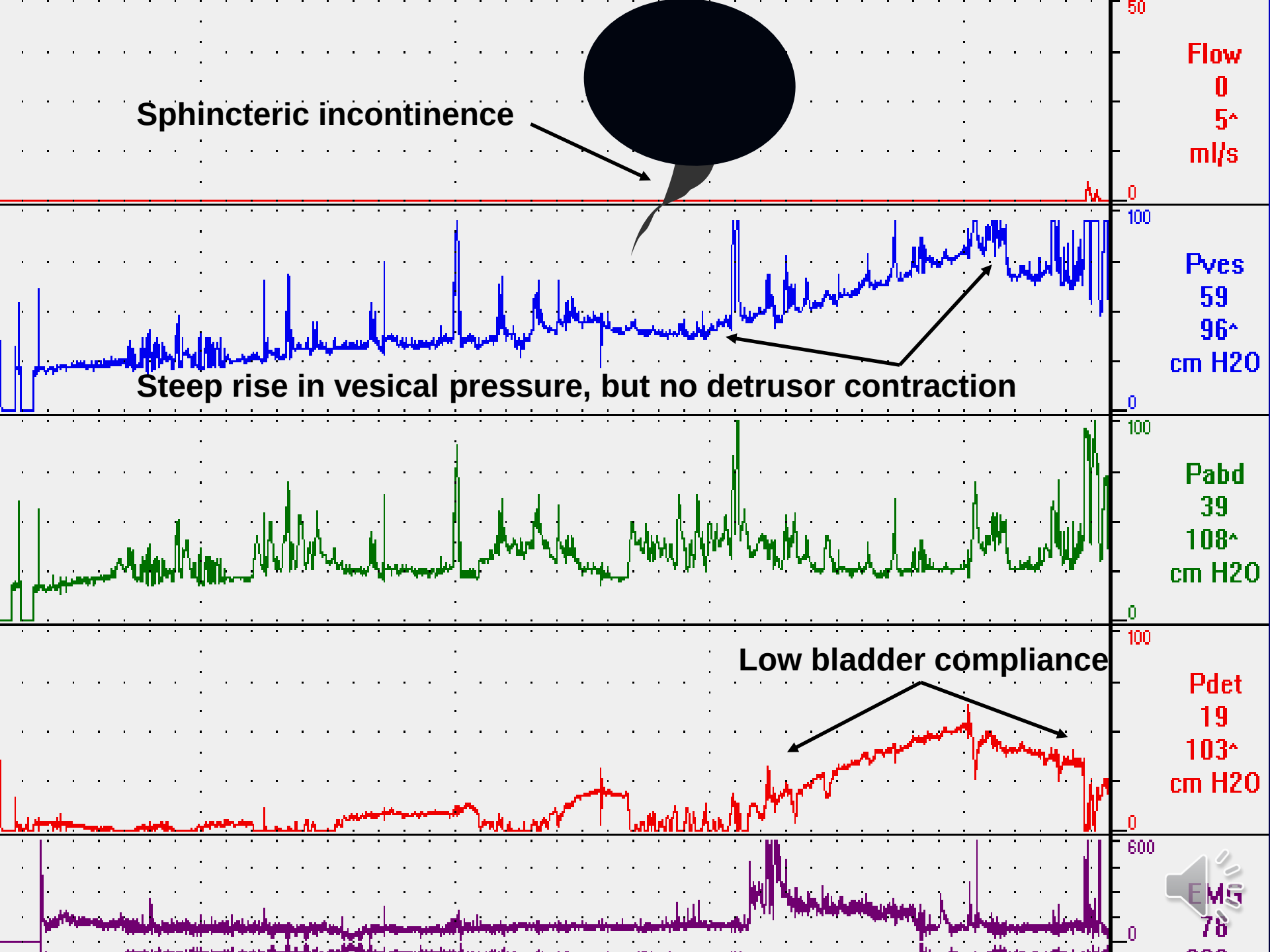
Thoraco-lumbar Sympathetic Lesions



Thoraco-lumbar Lesions

- **Loss of smooth muscle sphincteric function**
- **Loss of striated sphincter control**
- **Detrusor areflexia or overactivity**
- **+/- low bladder compliance**

Paradoxical urinary retention & sphincteric incontinence


Sphincteric incontinence
Steep rise in vesical pressure, but no detrusor contraction
Low bladder compliance
50
Flow
0
5^
ml/s
0
100
Pves
59
96^
cm H2O
0
100
Pabd
39
108^
cm H2O
0
100
Pdet
19
103^
cm H2O
0
600
EMG
78
0

Suprasacral Spinal Lesions

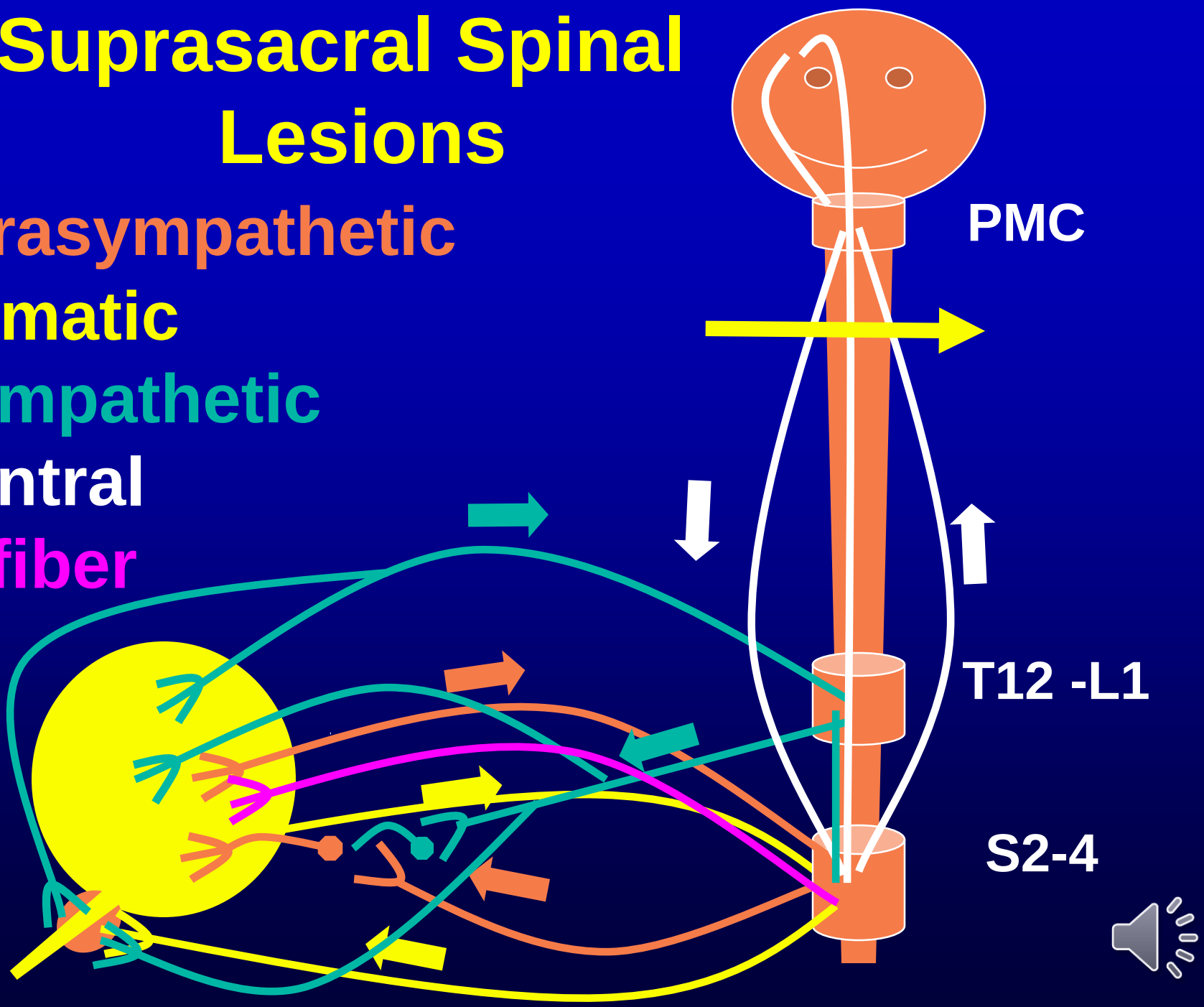
parasympathetic

somatic

sympathetic

central

C fiber



Detrusor External Sphincter Dyssynergia

- Involuntary sphincter contraction during involuntary detrusor contraction
- Due to a neurologic lesion between sacral & pontine micturition center
- Poses serious urologic risks – UTI, urosepsis, stones, hydronephrosis, renal failure

Obstruction due to sphincter contraction

30
Flow
0
0

100
Pves
54
0

Involuntary detrusor contraction

100
Pabd
38
0

100
Pdet
16
0

Involuntary sphincter contraction

600
EMG
168
0
-600

1000
VH2O
147
0

Cortical Centers

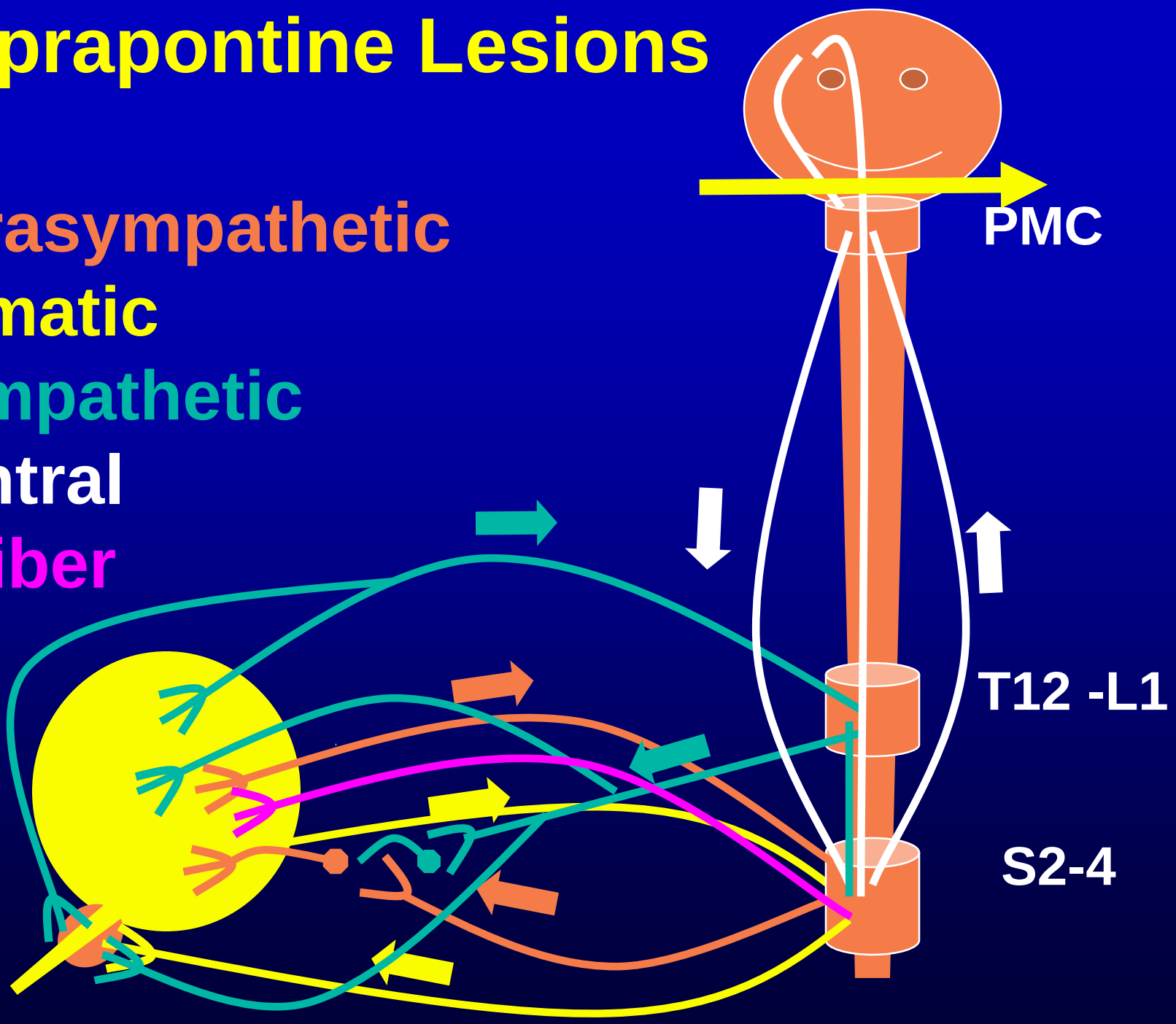
- Conscious awareness of bladder sensations & events
- Assess the propriety, social context & timing of micturition
- Voluntary control of micturition

Suprapontine Neurologic Lesions

- Micturition reflex usually intact
- When micturition is affected, there is usually loss of voluntary control
- +/- loss of awareness & concern
- +/- loss of voluntary sphincter control

Suprapontine Lesions

parasympathetic
somatic
sympathetic
central
C fiber



incontinent

Flow

50

0

Pves

100

0

Pabd

100

0

No awareness of bladder filling or urge to void

Pdet

100

0

EMG

600

0

-600

Involuntary detrusor contraction

RS

VH2O

1000

0

Suprapontine Neurologic Lesions

- The “neurogenic bladder” poses no threat to health unless there is an underlying urologic condition such as
  - Urethral obstruction (prostate, prolapse)
  - Prostate or bladder cancer
  - Incomplete bladder emptying
  - Recurrent UTI

Pathophysiology of NGB

- **Suprapontine lesions:**
  - Loss of voluntary control
- **Suprasacral lesions:**
  - Detrusor sphincter dyssynergia
- **Thoracolumbar lesions**
  - Neurogenic sphincteric incontinence
  - Detrusor areflexia or overactivity
- **Sacral lesions:**
  - Detrusor areflexia

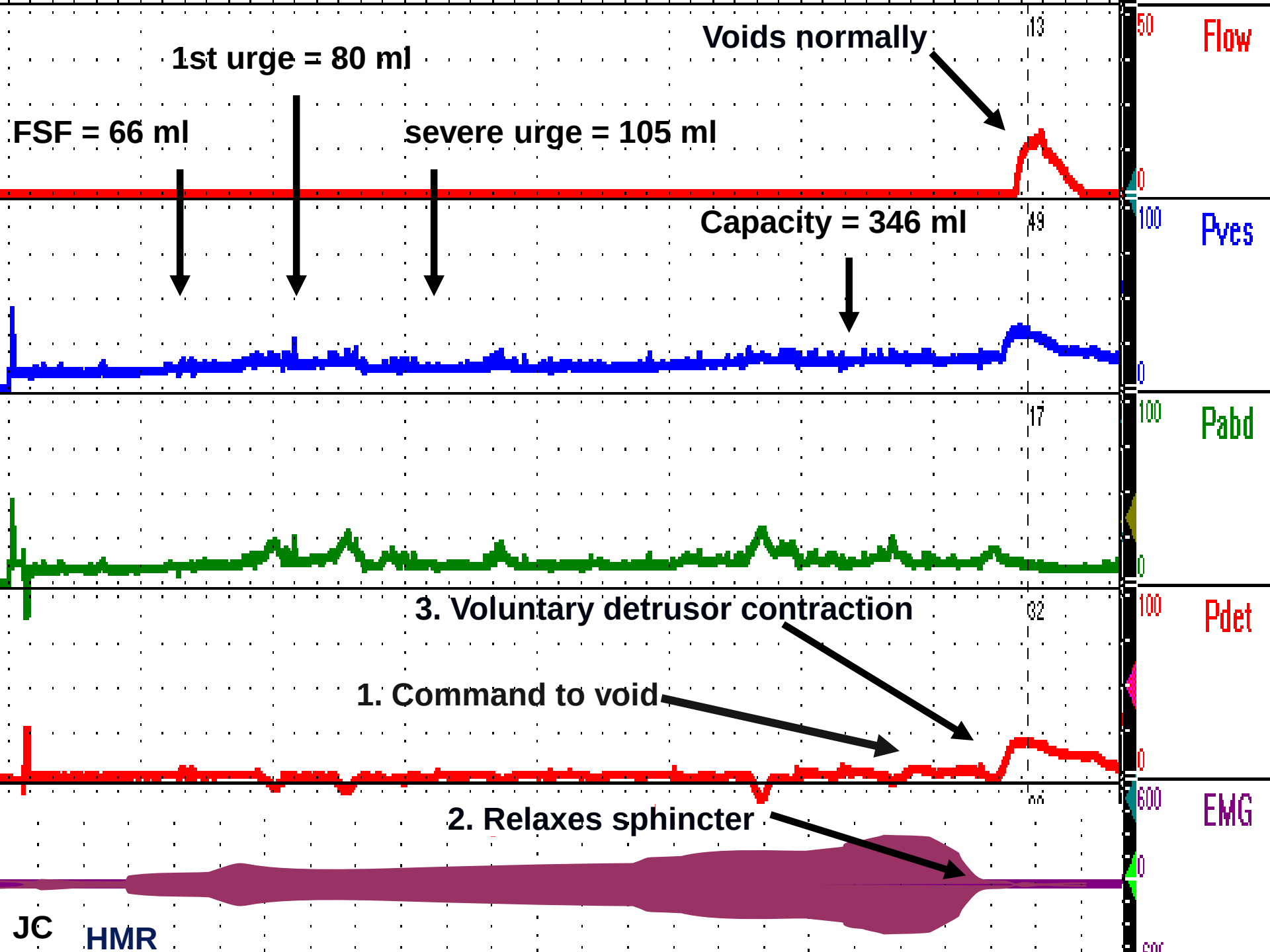
Blaivas JG, The Neurophysiology of Micturition. *J Urol* 127:958-3,1982

Knowledge Gaps & Research Opportunities

Understanding Urgency and Involuntary Detrusor Contractions

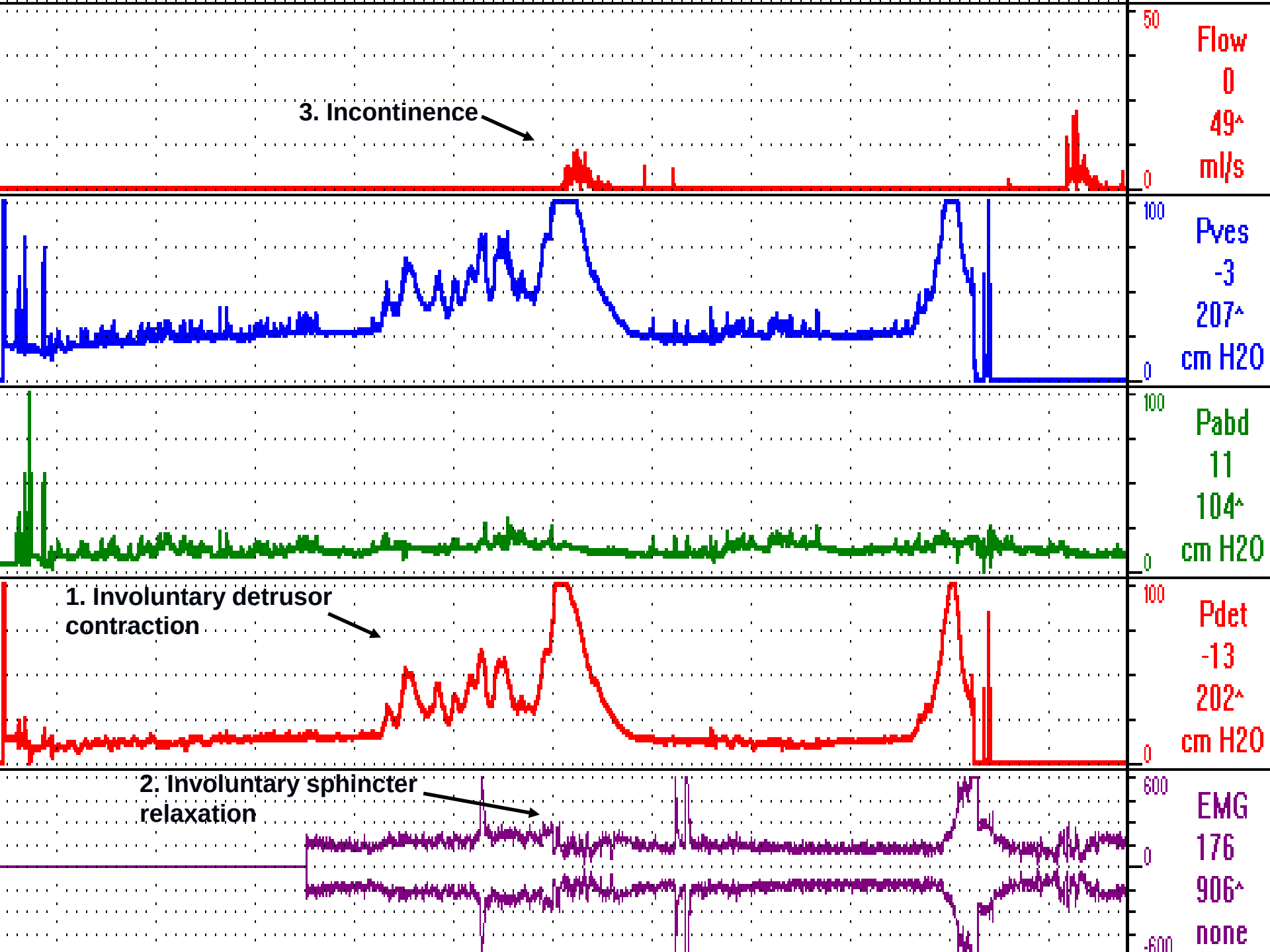
- **Type I: patient has urgency and/or urge incontinence, but no IDC at urodynamics**

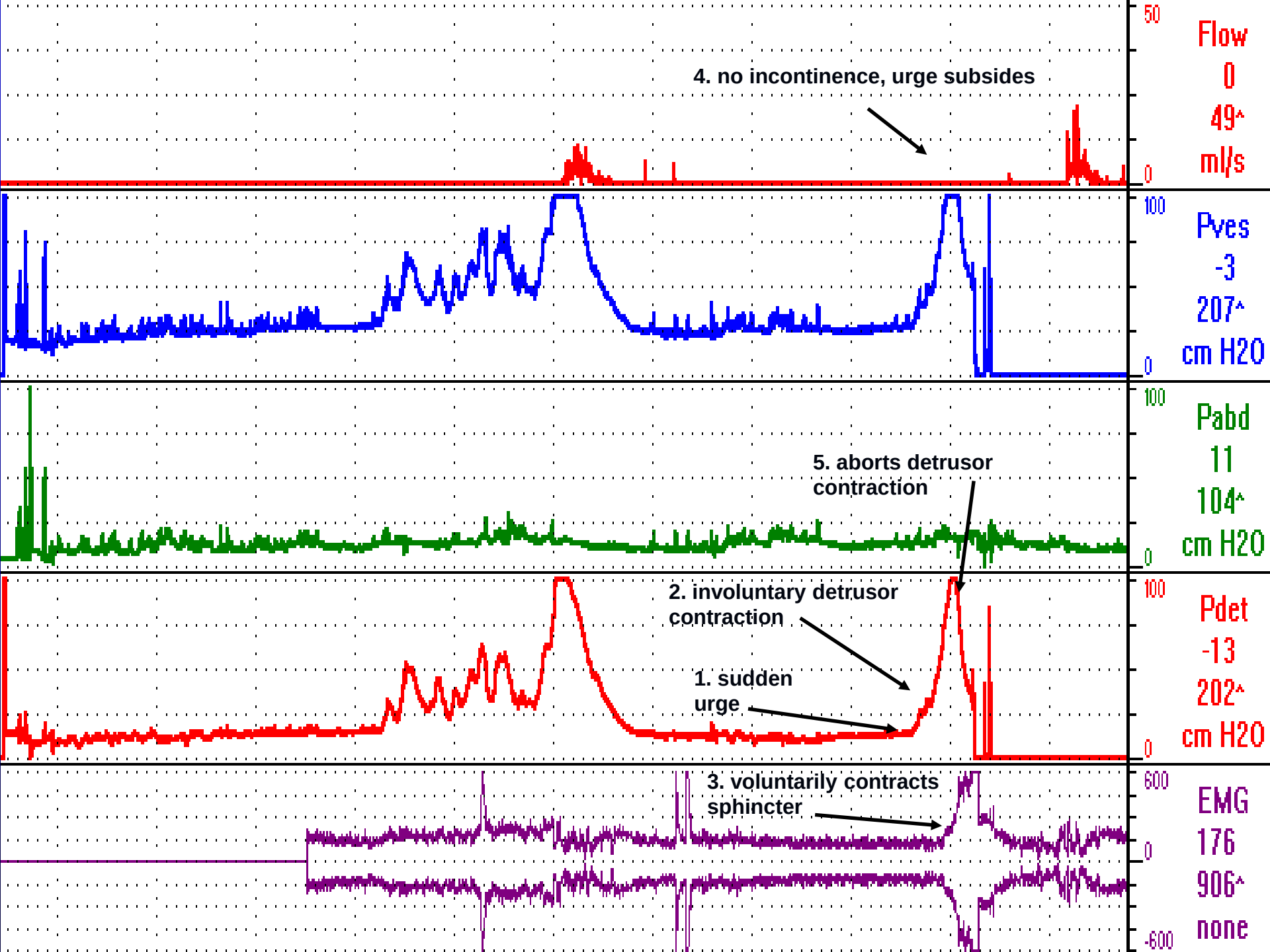
Flisser, J. Urol 169: 529-534, 2003


Voids normally
1st urge = 80 ml
FSF = 66 ml
severe urge = 105 ml
Capacity = 346 ml
3. Voluntary detrusor contraction
1. Command to void
2. Relaxes sphincter
Flow
Pves
Pabd
Pdet
EMG
JC
HMR

Understanding Urgency and Involuntary Detrusor Contractions

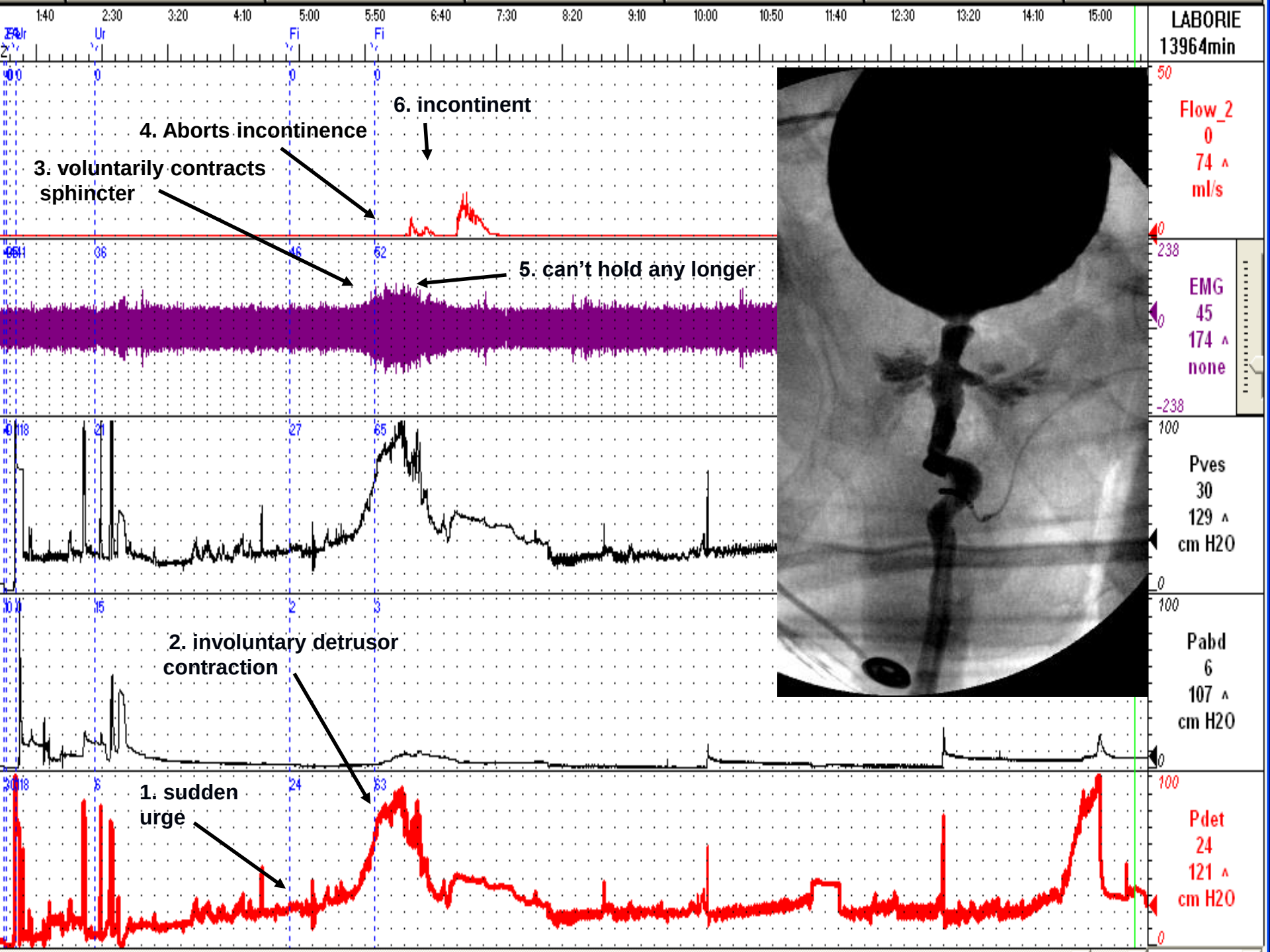
- **Type I:** symptoms of overactive bladder, no IDC at urodynamics
- **Type II:** IDC present; patient is aware, can contract his sphincter, abort the detrusor contraction and prevent incontinence


3. Incontinence
1. Involuntary detrusor contraction
2. Involuntary sphincter relaxation
50
Flow
0
49^
ml/s
0
100
Pves
-3
207^
cm H2O
0
100
Pabd
11
104^
cm H2O
0
100
Pdet
-13
202^
cm H2O
0
600
EMG
176
906^
none
0
-600


4. no incontinence, urge subsides
Flow
0
49^
ml/s
50
0
Pves
-3
207^
cm H2O
100
0
5. aborts detrusor contraction
Pabd
11
104^
cm H2O
100
0
2. involuntary detrusor contraction
1. sudden urge
Pdet
-13
202^
cm H2O
100
0
3. voluntarily contracts sphincter
EMG
176
906^
none
600
0
-600

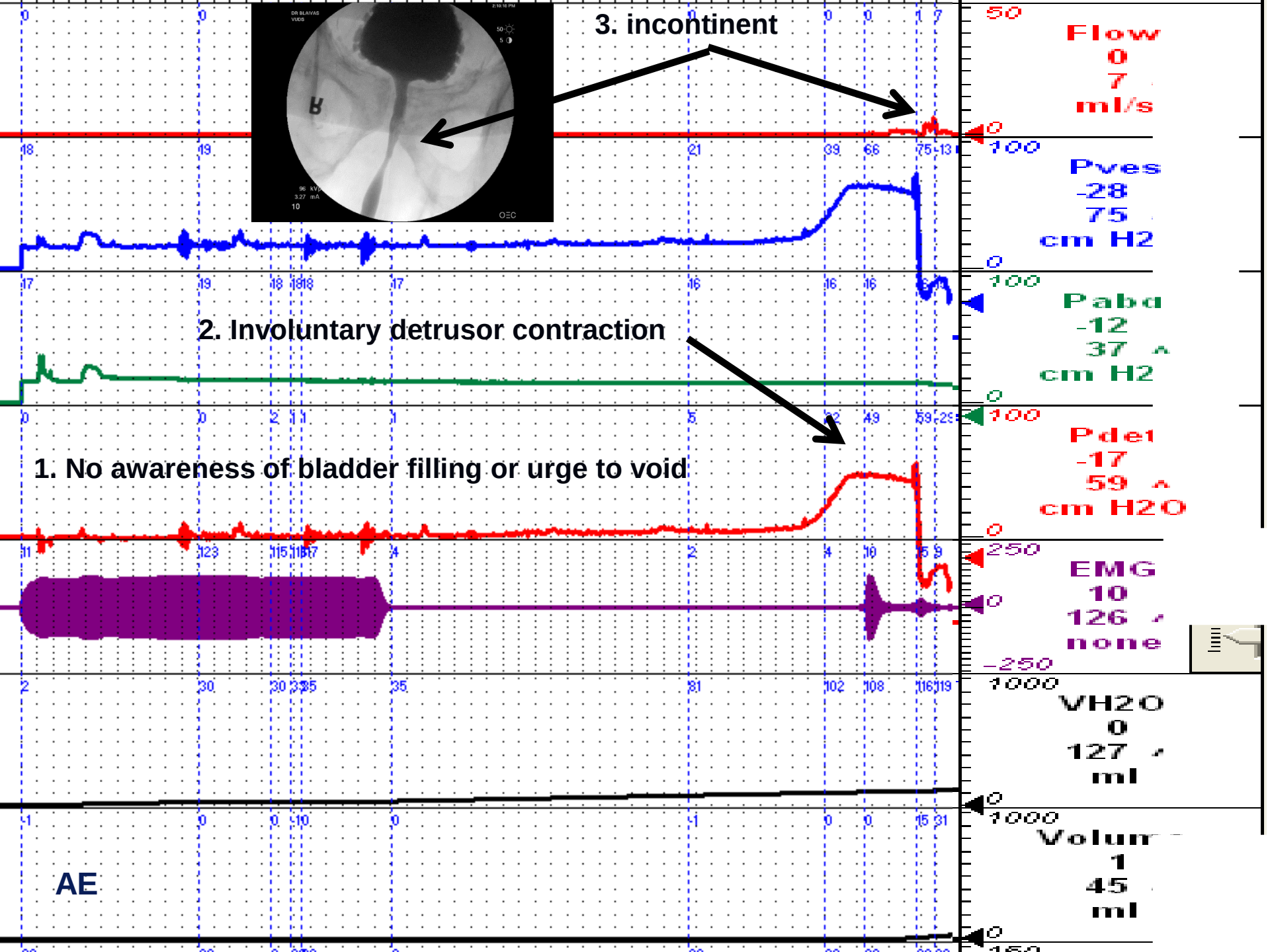
OAB Understanding Urgency and Involuntary Detrusor Contractions

- **Type I:** symptoms of overactive bladder, no IDC at urodynamics
- **Type II:** IDC present; patient is aware and can abort the IDC
- **Type III:** IDC patient aware, cannot abort but can temporarily maintain continence by contracting the sphincter; once the sphincter fatigues is incontinent.


1:40
2:30
3:20
4:10
5:00
5:50
6:40
7:30
8:20
9:10
10:00
10:50
11:40
12:30
13:20
14:10
15:00
LABORIE
13964min
Ur
Fi
Fi
6. incontinent
4. Aborts incontinence
3. voluntarily contracts sphincter
5. can't hold any longer
2. involuntary detrusor contraction
1. sudden urge
Flow_2
0
74
ml/s
EMG
45
174
none
Pves
30
129
cm H2O
Pabd
6
107
cm H2O
Pdet
24
121
cm H2O

Understanding Urgency and Involuntary Detrusor Contractions

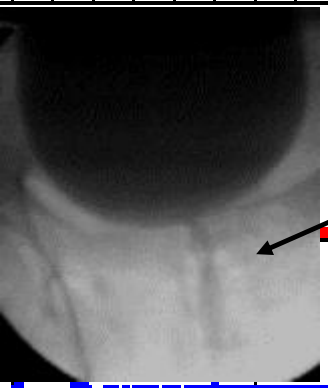
- **Type I:** symptoms of overactive bladder, no IDC at urodynamics
- **Type II:** IDC present; patient is aware and can abort the IDC
- **Type III:** IDC patient aware, cannot abort but can temporarily maintain continence by contracting the sphincter
- **Type IV: IDC, no awareness or control**


3. incontinent
2. Involuntary detrusor contraction
1. No awareness of bladder filling or urge to void
AE
Flow
0
7
ml/s
Pves
-28
75
cm H2
Pabd
-12
37
cm H2
Pdet
-17
59
cm H2O
EMG
10
126
none
VH2O
0
127
ml
Volum
1
45
ml

Detrusor hyperreflexia with Impaired Detrusor contractility (DHIC)

Type 3 OAB
Impaired Detrusor Contractility

Resnick & Yalla, Detrusor hyperactivity with impaired contractile function. An unrecognized but common cause of incontinence in elderly patients. JAMA (1987),12;257(22):3076



5.VOID: 5/180/570/4

4. Qmax = 5 mL/S

Flow

50

0

Pves

100

0

Pabd

100

0

3. pdet@Qmax = 28 cm H_20

Pdet

100

0

2. Involuntary detrusor contraction

1. urgency

EMG

600

0

-600

VH20

1000

0

AT

Knowledge Gaps & Research Opportunities

- What stimuli > urgency, IDO & NDO?
  - wall tension, pdet, volume?
  - constituents in urine, caffeine?
  - neurotransmitters, receptors?
- Is IDO just a subtle NDO?
- What neural pathways, transmitters & receptors are involved in aborting urgency & DO

Knowledge Gaps & Research Opportunities (cont'd)

- Etiology & Rx of impaired detrusor contractility & DHIC
- Better Rx for:
  - Urgency & DO
  - Detrusor sphincter dyssynergia