The pelvic floor muscles support the pelvic organs such as the bladder, prostate, uterus, and rectum. Besides providing support, they’re responsible for the complex relaxing & contracting that enables us to urinate and defecate properly. These muscles are always interacting with the pelvic organs. For example, urination involves relaxation of these muscles while the bladder contracts. Sometimes, these muscles either spasm or simply don’t interact as they should with the pelvic organs, so-called “pelvic floor dysfunction,” which may give: difficulty initiating the urine stream, pain with urination, frequent daytime voiding, constipation, increased discomfort when sitting for long intervals etc.
Most important pelvic floor muscles:

External anal sphincter
Levator ani:
  - Puborectalis
  - Iliococcygeus
  - Pubococcygeus
INNERRATION

Internal anal sphincter
Sympathetic nerve fibres from presacral plexus and parasympathetic fibres via pelvic nerves from SII-IV sacral segments.

External anal sphincter
Via the pudendal nerve from SII-IV sacral nerves.

levator ani
Direct nerve branches from SII-IV sacral nerves (the sacral motor nerve)

Indications for neurophysiological evaluation

Anal incontinence  Constipation  Botox treatment

Anal incontinence: Causes

- Traumatic sphincter lesion: Obstetric, post op. mm.
- Idiopathic (neurogenic): No anatomical anomaly
- Rectal invagination and prolaps
- Aging
- Congenital: Spina bifida, anorectal anomaly, Hirschprung disease, CF
- Other: Food allergy, abuse of laxantia, malabsorption, gastrointestinal fistula, inflammatory bowel disease, MS, diabetic neuropathy, dementia etc.
FACTORS FOR CONTINENCE

Consistency of bowel content
Transport of content to rectum
Rectal capacity
Collaboration
Ano-rectal sensation
Function in ext. anal sphincter
Muscles & nerves in all of pelvic floor

NEUROGENIC (IDIOPATHIC INCONTINENCE)

Motor dysfunction dominates
> "urgency" incontinence

Sensory dysfunction or insufficiency in inner anal sphincter
> "leakage" incontinence

Neurogenic incontinence was earlier thought of as a non-surgical disorder.
However, plastic surgery of the levator ani has proven a good therapy.
Also electrostimulation is a method used for therapy.

Grading of incontinence according to Miller

<table>
<thead>
<tr>
<th>Incontinent for</th>
<th>Gas</th>
<th>Loose stool</th>
<th>Formed stool</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incontinence &lt;once/month</td>
<td>1</td>
<td>4</td>
<td>7</td>
</tr>
<tr>
<td>Incontinence 1week/month</td>
<td>2</td>
<td>5</td>
<td>8</td>
</tr>
<tr>
<td>Incontinence &gt;once/week</td>
<td>3</td>
<td>6</td>
<td>9</td>
</tr>
</tbody>
</table>

0 p = total continence; 18 p = maximal incontinence

Miller et al., Br J Surg 1988;75:101-105
NEUROPHYSIOLOGICAL INVESTIGATION of anal incontinence

- EMG bilaterally in external anal sphincter & puborectalis m.
- Fibre density bilaterally in the external anal sphincter

(- Distal pudendal latency)

________________________________________________________________________

EMG in EAS och puborectalis muscle

Mean MUP amplitude around 350 uV
Somewhat increased polyphasia may be seen

________________________________________________________________________

EMG in EAS and puborectalis muscle

At maximal squeeze:
the interference pattern may often
show "central weakness"
Fibre density in external anal sphincter

<table>
<thead>
<tr>
<th>EAS analysis</th>
<th>Left Sphincter m/s</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Right and left side of EAS

Upper normal limit 1.8

At least 10 insertions, preferably 20

Distal pudendal latency

Normal value in ages 25-75 yrs: <= 2.5 ms

St Mark’s electrode; 50 mm between stimulation and recording

Normally 10mA is enough.

Several stimulations > reproducibility

The signals are phase reversed when stimulating left side (change-over switch can be used)

CONSTIPATION: CAUSES

Colonic:
- Slow transit (transit time men 2.8d, females 4.7)

Anorectal causes:
- Rectocele
- Rectal invagination and prolapse
- Paradoxal puborectalis contraction

Enterocoele

Neuropathological evaluation

Other causes:
- Diet and habit based
- Metabolic and endocrine
- Neurologic
- Psychiatric
EMPTYING OF THE BOWEL
a complex and not entirely mapped phenomenon

When there is large enough content in the sigmoid, it will be released into rectum.
Stretching of rectum: stimulates stretch receptors in the pelvic floor > further stretching leads to relaxation of the inner anal sphincter and contraction of the external (= recto-anal inhibition reflex; RAIR).
Emptying: increase of the intra-abdominal and intra-rectal pressures at strain. The inner anal sphincter and puborectalis muscle relaxate; this, plus the sitting position increase the ano-rectal angle, which leads to formation of a funnel at the top of the anal canal. The intra-abdominal pressure is directed to content in this funnel and a defecation takes place.
Closing reflex after emptying: EAS and the puborectalis muscle contract shortly, the ano-rectal angle is restored, and the inner sphincter closes the anal canal.

NEUROPHYSIOLOGY IN CONSTIPATION

Index based on EMG findings at strain and squeeze
Strain amplitude / squeeze amplitude x 100
Index >50 is a sign of increased paradoxical puborectalis activation

The investigation
- Hook-electrodes bilateral in EAS and puborectalis muscle.
- Patient in left lateral position and in sitting
- Mean value of 3 squeezes and 3 strains in each muscle and position


EMG IN CONSTIPATION:
HOOK-ELECTRODES BILAT IN EAS OCH PUBORECTALIS MUSCLE

Electrodes in puborectalis applied 3-4 cm deep, lateral - upward
Electrodes in EAS applied 0.5-1cm deep at the anal verge

Keypoint EMG program for spontaneous activity since it contains amplitude cursors.
Sweep: 200ms/div; 0.1 mV (sometimes 0.2)
EAS and the puborectalis muscle act as a unity, there is a high correlation between the index values.

Simplified Evaluation of the Paradoxical Puborectalis Contraction with Surface Electrodes

Hans Axelsson, M.D., Ph.D. 1, Karin Edebol Eeg-Olofsson, M.D., Ph.D. 1
Department of Neuroscience, Section for Clinical Neurophysiology, University Hospital, Uppsala, Sweden

Submitted
BOTULINUM TOXIN TREATMENT: INDICATIONS

- Anal fissure
- Dyssynergia, sphincter spasm, anal pain
- Congenital conditions, ie. Hirschsprung disease

Uppsala 2008: 33 patients:
26 anal fissures (79%), 2 anal spasms, 2 Hirschsprung dia., 2 anal pain, 1 sacral malform.

Uppsala 2009 Jan-July: 28 patients:
20 anal fissures (71%), 4 anal spasms, 2 Hirschsprung dia., 2 vaginal spasms

The Botox injections are EMG guided to ensure optimal needle placement in the external anal sphincter (EAS). Bilateral injections.

Normal dosage to adults: 5-15 units BTA in right and left EAS, i.e. a total of 10-30 units BTA.

Referring physicians:
Surgeons, pediatric surgeons, gynecologists

Botox treatment in Hirschsprung disease

<table>
<thead>
<tr>
<th>Gender</th>
<th>Birth year</th>
<th>No. of treatments and total dose of Botulinum toxin type A per treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>M</td>
<td>1991</td>
<td>4 treatments in 10 weeks, starting in May 2004</td>
</tr>
<tr>
<td></td>
<td></td>
<td>40 units, 35 units, 50 units, 50 units, 50 units.</td>
</tr>
<tr>
<td>M</td>
<td>1992</td>
<td>4 treatments in 12 months, starting in August 2004</td>
</tr>
<tr>
<td></td>
<td></td>
<td>80 units, 80 units, 100 units, 100 units, 100 units.</td>
</tr>
<tr>
<td>M</td>
<td>1993</td>
<td>4 treatments in 12 months, starting in April 2004</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60 units, 60 units, 60 units, 60 units.</td>
</tr>
<tr>
<td>M</td>
<td>1994</td>
<td>4 treatments in 12 months, starting in March 2004</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60 units, 60 units, 60 units, 60 units.</td>
</tr>
<tr>
<td>M</td>
<td>1995</td>
<td>4 treatments in 12 months, starting in November 2002</td>
</tr>
<tr>
<td></td>
<td></td>
<td>30 units.</td>
</tr>
<tr>
<td>M</td>
<td>2000</td>
<td>2 treatments in 24 months, starting in April 2004</td>
</tr>
<tr>
<td></td>
<td></td>
<td>40 units, 60 units.</td>
</tr>
<tr>
<td>M</td>
<td>2003</td>
<td>2 treatments in 24 months, starting in September 2004</td>
</tr>
<tr>
<td></td>
<td></td>
<td>40 units, 40 units, 40 units.</td>
</tr>
</tbody>
</table>
Hirschsprung disease (HH 1887)

- **Prevalence**: 1/5000 living born babies
- **Ethiology**: Developmental disorder in the enteric nervous system with absence of ganglion cells distally in the bowel, in shorter or longer segment (congenital aganglionosis). Normally the ganglion cells have migrated to the small intestine during fetal week 7, and then reach rectum.
- **Leads to**: No peristalsis in the large intestine; the child develops severe constipation.
- **Treatment**: Surgery, enema, Botox