Introduction

The term “sphincter atrophy” refers mostly to external anal sphincter (EAS) atrophy, as the EAS is the most important factor for maintaining fecal continence. EAS atrophy, often due to pudendal neuropathy caused by stretch injury during childbirth [1] or chronic constipation [2, 3], is an important cause of fecal incontinence. When a woman is fecally incontinent and there is a history of a difficult childbirth with prolonged labor or chronic constipation as well as a sphincter rupture, there is always a chance that, besides the rupture, some atrophy is present in the EAS.

The importance of differentiating between the contribution of a defect or neuropathy/atrophy to the fecal incontinence lies in the fact that only patients with a significant sphincter defect are offered a sphincter repair. There is some, although not unanimous, evidence that severe atrophy interferes with a good result after sphincter repair.

It has been suggested that patients with low anal pressures and poor innervation to the pelvic floor and elderly patients have less favorable results with postanal repair [4, 5] and anal sphincter repair [6–8]. However, prospective studies are lacking. Reviewing the recent literature, Gearhart et al. [9], Pinta et al. [10], and Engel et al. [11] could not find a relationship between pudendal nerve terminal motor latency (PNTML) and anal repair. Birnbaum et al. [12] found a relationship between PNTML and the results of rectopexy. Establishing (the amount of) atrophy, at least the extreme cases, seems of clinical importance when selecting patients for sphincter repair.

Diagnosing External Anal Sphincter Atrophy

Establishing atrophy of the anal sphincter complex has been evaluated with endoanal magnetic resonance imaging (MRI) [13–17]. Studies with endoanal MRI have demonstrated that severe atrophy of the EAS corresponded with a poor clinical outcome [15] and histopathology in biopsies taken from the EAS during surgery [13]. In general, atrophy can be established by measuring EAS thickness and surface area, and the subjective evaluation of the amount of fat. Another study found no relationship between fat content and anorectal function [18]. One study described the aspect of the EAS with anal endosonography in comparison with endoanal MRI, but without three-dimensional (3D) application and without transversal or longitudinal sphincter measurements [18]. With 3D anal endosonography (3D-AE), it is possible to measure EAS length on the lateral view and subsequently perform volume measurements. The high expectations of EAS volume measurements were not met, as no discrimination was found between healthy controls and patients with fecal incontinence [19]. In a subsequent study, using volume measurement was found to be unsuccessful in predicting EAS atrophy in patients with fecal incontinence. Another issue was that in all patients, MRI mentioned atrophy but no histology was performed [20].

A recent study in 18 women with fecal incontinence compared 3D endoanal ultrasound (EUS) and MRI to evaluate EAS atrophy [21]. Atrophy of the EAS with EUS was judged upon its reflection of the outer interface (border of the EAS and subadventitial fat), reflection pattern, and length. Atrophy was scored as none (clearly visible outer interface, mixed reflection pattern), moderate (partly visible outer interface, moderate shortening), and severe (hardly visible outer interface, hyperechogenic reflection pattern, severe shortening). These criteria were derived from Williams et al. [17]. Examples of normal and atrophic anal sphincters are shown in Figures 1–5.

EAS atrophy with MRI was defined as diffuse thinning of the EAS muscle or diffuse replacement of EAS muscle by fat. EAS atrophy was graded as none (no thinning of the EAS and no replacement of EAS muscle by fat), moderate (≥50% thinning of the EAS and/or replacement of EAS muscle by fat), or severe (>50% thinning of the EAS and/or replace-
ment of EAS muscle by fat). Three-dimensional AE and MRI did not significantly differ for the detection of EAS atrophy ($p=0.25$) and defects ($p=0.38$): 3D-AE demonstrated EAS atrophy in 16 patients; MRI detected EAS atrophy in 13 patients. Also, 3D-AE agreed with MRI in 15 of 18 patients in detecting EAS atrophy. Using the grading system, eight of the 18 patients scored the same grade. It was concluded that both endoanal techniques are comparable in detecting EAS atrophy and EAS defects, although there is a substantial difference in grading EAS atrophy. Exact thickness and length measurements do not really contribute to atrophy score. This indicates that this imaging technique can be added as a diagnostic tool for EAS atrophy in patients with fecal incontinence. Limitations of this study are the small number of patients and the absence of a gold standard. Further prospective studies should consist of more patients, healthy controls, and evaluation with surgery and histology.

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**Fig. 1a, b.** Endoanal ultrasonography (EAS). Normal anatomy of the anal sphincter and puborectalis muscle in three-dimensional (3D) imaging. 

- **a** Frontal view of puborectalis muscle.
- **b** Frontal view of the anal sphincters. 

PR puborectalis muscle, SM submucosa, IAS internal anal sphincter, EAS external anal sphincter

**Fig. 2.** Magnetic resonance imaging (MRI) (axial view). Normal anatomy of the internal and external anal sphincter. 

R rectum, IAS internal anal sphincter, EAS external anal sphincter
**Internal Anal Sphincter Atrophy**

Internal anal sphincter (IAS) atrophy will often occur combined with EAS atrophy. Although the IAS is innervated by autonomic nerves, often the same injuries can afflict both somatic and autonomic nerves. Generally, IAS problems will lead more to soiling (leakage) of fecal fluid or mucous. Several reports have emerged about rare causes of fecal incontinence, such as primary IAS degeneration in passive fecal incontinence [22] and IAS sclerosis in mixed connective tissue disease [23] and systemic sclerosis [24]. In these patients, there is diffuse thinning (<0.2 mm) of the IAS. Clinical consequence is small, as no causative therapy is available, and general measurements such as defecation regulation and local hygiene are the only options.

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**Fig. 3a, b.** Endoanal ultrasound (EUS) image of external anal sphincter atrophy. *a* Frontal and *b* lateral views. *R* rectum, *IAS* internal anal sphincter, *EAS* external anal sphincter

**Fig. 4a, b.** Endoanal ultrasound (EUS) image of external and internal anal sphincter atrophy. *a* Frontal and *b* lateral views. *R* rectum, *EAS* external anal sphincter. The IAS is hardly visible and is also atrophic (*arrow*)
References


Fig. 5a, b. Magnetic resonance imaging (MRI) (axial) of external anal sphincter atrophy in two different patients. R rectum, IAS internal anal sphincter, EAS external anal sphincter.

