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Internal anal sphincter: Clinical perspective

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ABSTRACT

Objective: To summarise current knowledge of Internal anal sphincter.
Background: The internal anal sphincter (IAS) is the involuntary ring of smooth muscle in the anal canal and is the major contributor to the resting pressure in the anus. Structural injury or functional weakness of the muscle results in passive incontinence of faeces and flatus. With advent of new assessment and treatment modalities IAS has become an important topic for surgeons. This review was undertaken to summarise our current knowledge of internal anal sphincter and highlight the areas that need further research.
Method: The PubMed database was used to identify relevant studies relating to internal anal sphincter.
Results: The available evidence has been summarised and advantages and limitations highlighted for the different diagnostic and therapeutic techniques.
Conclusion: Our understanding of the physiology and pharmacology of IAS has increased greatly in the last three decades. Additionally, there has been a rise in diagnostic and therapeutic techniques specifically targeting the IAS. Although these are promising, future research is required before these can be incorporated into the management algorithm.

Introduction

Faecal incontinence is a debilitating condition which is often under-reported. Although it is difficult to accurately determine the incidence of faecal incontinence due to under-reporting, a range of 2–24%, depending on the population studied, has been reported.[1] Despite a disagreement about prevalence rates of FI, there is a widespread acceptance of its increased prevalence in the older population.[1]

Maintenance of continence is a complex process requiring interplay between colon, rectum, the internal and external anal sphincter complex, pelvic floor and inter-related motor and sensory neural pathways, all governed by higher centres. Out of many factors controlling the continence, the anal canal, being the final determining step, with its sphincter complex is undoubtedly critical.

The anal canal is composed of two adjacent muscles – external and internal anal sphincter. This review will focus on the characteristics, pathology and therapeutic potential of the smooth muscle internal anal sphincter (IAS).

Anatomy

The internal anal sphincter is a ring of smooth muscle formed by the continuation of the involuntary circular muscle of rectum into the distal anal canal (Fig. 1). As such it is not under

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voluntary control. Caudally the IAS extends just proximal to the lowest part of the external anal sphincter (EAS).[2] It is about 2.5 cm long and 2–5 mm thick in size. It increases in thickness with advancing age, a phenomenon more pronounced in females after the age of fifty.[3] Histological studies have shown this rise in bulk to be due to an increase in the amount of connective tissue,[4] a finding which correlates well with the manometry findings of decreased resting pressure with ageing.

No correlation has been found between IAS size and body weight or parity.[5]

Physiology

Innervation of IAS

The IAS is dually innervated by the autonomic and the enteric nervous system. The parasympathetic and sympathetic nervous systems are responsible for exerting both excitatory and inhibitory influences on the smooth muscle whereas the enteric system influences the tone.[6].

In general, IAS is believed to be parasympathetically innervated by the first, second and third sacral nerves via pelvic plexus and sympathetically from both the thoracolumbar outflow and the hypogastric nerves.[7]

The enteric nervous system is organised into an interconnected network of neurons and glial cells that are grouped into ganglia located in two major plexuses: the myenteric (Auerbach's) plexus and the submucosal (Meissner's) plexus. Its components form entire reflex pathways that control peristaltic contractions independent of extrinsic innervation from the autonomic nervous system.

Effects of parasympathetic innervation on IAS

The parasympathetic fibres have been shown to exert an inhibitory effect on the tone of IAS thereby causing its relaxation. *In vitro* studies such as those by Burleigh et al. and O'Kelly et al. provided important insight into muscarinic receptor stimulation. The former study revealed that stimulation of muscarinic receptor by acetylcholine leads to relaxation of IAS[8] and the latter demonstrated that muscarinic receptor stimulation is mediated indirectly via nitric Nitric oxide (NO) release.[9] The property of IAS relaxation, on stimulation of muscarinic receptors via acetylcholine, led to a trial of botulinum toxin, which acts on cholinergic neurones, for treating anal fissures.

Effects of sympathetic innervation on IAS

The effects of sympathetic system are known to be mediated via the alpha and beta adrenoceptors, both of which are present on the IAS. Depending upon which adrenoceptor subtype is stimulated, the sympathetic innervation exerts excitatory or inhibitory influence on the IAS.

Stimulation of alpha-adrenoceptors is known to have an excitatory effect on the smooth muscles of IAS.[10] Of the two known subtypes, $\alpha 1$ and $\alpha 2$, the former has been shown to cause IAS contraction without affecting the rectoanal inhibitory reflex (RAIR) induced IAS relaxation whereas the latter inhibits RAIR-induced IAS relaxation but does not increase the IAS contractility.[11] This IAS contraction inducing property of $\alpha 1$ adrenoceptors has been clinically used in the form of phenylephrine and methoxamine, both being potent $\alpha 1$ adrenoceptor agonist, to treat FI. However, trials for both the drugs failed to demonstrate any significant clinical improvement in patients with FI.

Beta-adrenoceptors, have been shown to mediate the inhibitory effects of sympathetic nerve stimulation in the IAS[12] and other parts of the GI tract.[13] In IAS, presence of all the three subtypes of β adrenoceptors (β1, β2 and β3) was established by a recent study, which found varying level of inhibitory effect exerted on the IAS by each of the three receptor subtypes.[12] β1-agonists were found to be the most superior inhibitors of IAS as compared to β2 or β3-agonists, producing about 70% IAS relaxation vs 40% and 30% by β2 and

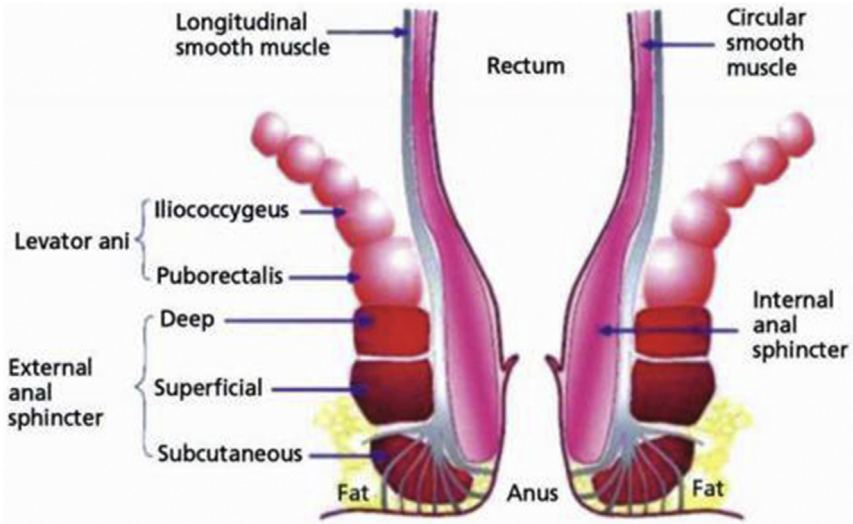

Fig. 1 – Anatomy of anal canal; Reprinted by permission from John Wiley and Sons: The Obstetrician & Gynaecologist, Management of obstetric anal sphincter injury, Volume 5, Issue 2, 72–8 (2003).

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