Introduction

nal incontinence is defined as the loss of voluntary control of feces (liquid or solid) from the bowel. The condition is socially embarrassing and the prevalence varies between 1% and 21% in different studies. This prevalence increases with age: 0.5% to 1% in people younger than 65 years and 3% to 8% in people older than 65 years. Multiple factors may contribute to the development of AI, including obstetric, traumatic, or neurologic damage, spinal injuries, mental disorders, or problems with loose stool consistency. Obstetric injury is the most common cause (*Mellgrene*, 2010 a).

Patients with AI complain of urgency and urge incontinence, often indicating EAS weakness or damage, or passive soiling secondary to IAS disruption or atrophy. Both symptoms can be present in the same individual (*Norton*, 2008).

The first step of evaluation of patients with AI begins with full history, and then examination is the second step. DRE can reveal masses or a fecal impaction. It also provides a gross assessment of both resting tone and voluntary squeeze. Lastly, Anoscopy and Proctosigmoidoscopy can be used. Investigations of anal incontinence include, EAUS, ARM, PNTML, EMG, Defecography and Colonoscopy (Sands and Madsen, 2011).

Endo-anal MRI is accurate in detecting EAS defects potentially suitable for repair in patients presenting with AI

when compared with the traditional gold standards of EAUS and surgery (*Dobben et al.*, 2007).

Management options include biofeedback, anal sphincteroplasty, stimulated graciloplasty, artificial anal sphincter, sacral nerve stimulation& injectable biomaterials (*Mellgren*, 2010 a).

Biofeedback educates patients regarding pelvic floor coordination, recognition of sensory thresholds, and conditioning of the pelvic musculature, and it helps develop improved pelvic floor habits (*Norton et al.*, 2006).

Anal sphincteroplasty, patients with significant preoperative symptoms are better candidates than patients with mild AI symptoms (*Trowbridge et al.*, 2006).

Stimulated Graciloplasty, mobilization of the Gracilis muscle from the medial leg, and then tunneling around the existing sphincter complex. An electronic pulse generator applies continuous low-grade current to aid tonic contraction of the muscle (*Chapman et al.*, 2002).

Artificial anal sphincter remains one of only a few available surgical treatment modalities for patients who have end-stage anal incontinence who otherwise would require a permanent stoma (*Mellgrene*, 2010 a).

Recently SNS has evolved to become a clinical efficient therapy applicable across a wide spectrum of causes of AI with reproducible results. SNS can be considered to be an essential part of the current surgical treatment algorithm for AI (*Matzel, 2011*).

Injectable biomaterials, Injection of bulk-enhancing agents into the anal canal area, usually intersphincterically or submucosally (*Portilla et al.*, 2008).

AI is a debilitating and socially embarrassing condition. Significant advances in the evaluation and treatment of this condition have been made in recent years and several new treatment modalities will be made available to affected patients. Surgeons who care for these patients should have a thorough understanding of the medical, non-operative, and operative components and the various studies available to properly select a treatment plan and optimize outcomes (*Mellgrin*, 2010 a).

Aim of the Work

The aim of this essay is to review the recent updates in literature concerning anal incontinence regarding its etiology, clinical picture, investigations, surgical management, results & prognosis.

Anatomy of the Rectum, Anal Canal and the Pelvic Floor

Embryology

The anorectal region derives from four separate embryological structures: the hindgut, the cloacae, the proctodeum, and the anal tubercles. The hindgut forms the distal third of the transverse colon, the descending colon, the sigmoid, the rectum, and the upper part of the anal canal to the level of the anal valves. The end of the hindgut enters into the cloaca. The cloaca is initially a single tube that is subsequently separated by caudal migration of the urorectal septum into anterior urogenital and posterior intestinal passages (*Cook and Mortenson, 2002*).

In the 10th week, the external anal sphincter is formed from the posterior cloacae after complete descent of the urogenital septum. By the 12th week, the internal anal sphincter is formed from a thickened extension of rectal circular muscle. The proctodeal portion of the cloacal membrane disintegrates to form the anal tubercles that join posteriorly and migrate ventrally to encircle a depression, known as the anal dimple or proctoderm (*Lau and Caty*, 2006).

The anal tubercles join the urorectal septum and genital tubercles to form the perineal body, completing the separation between the rectum and the urogenital tract. The upper portion of the anal canal is derived from endoderm and is supplied by the inferior mesenteric artery, which supplies the hindgut. The lower third of the anal canal has ectodermal origins and is supplied by the rectal arteries, which are branches of the internal pudendal artery (*Bharucha and Blandon*, 2007).

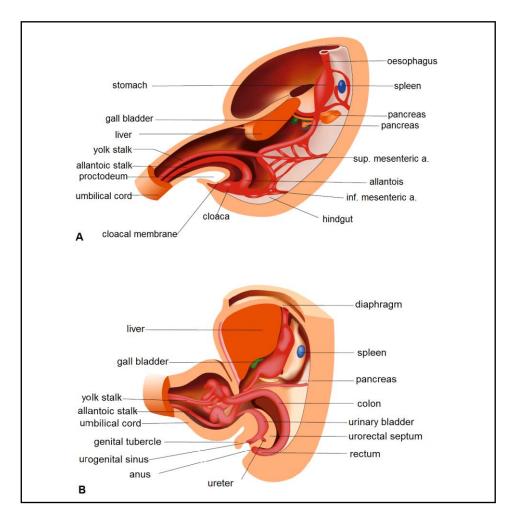


Figure (1): (A) GIT development at 5 weeks. (B) Development of the anorectum after 5 weeks, growth of the urorectum towards the cloacal membrane (*Thakar and Fenner, 2007*).

Anatomy

The Rectum

The rectum can be discriminated from the sigmoid colon by typical colonic properties, such as taenias, haustras, and omental appendices that diminish on the level of the third sacral vertebra. The rectum can be divided into three portions: the upper third extending from the rectosigmoid junction to the peritoneal reflection, the middle third terminating at the level of the puborectalis muscle sling, and the lower third passing over to the anal canal at the dentate line (*Aigner and Fritsch*, 2010).

Apart from a narrow segment of the ventral wall of the rectum, the terminal part of the hindgut lies totally extra peritoneally. This peritoneal coverage turns over to the apex and the back of the body of the bladder in the male and to the uterus and the posterior part of the vaginal fornix in the female, thus forming the rectovesical and the rectouterine pouch respectively (Douglas' pouch). The lateral border of this pouch is well demarcated by the rectouterine fold in the female, which contains the uterosacral ligament (*Stoker and Zbar*, 2008).

In the male the rectovesical pouch is bordered laterally by a peritoneal fold that covers the autonomic pelvic nerve plexus (the inferior hypogastric plexus). The rectum forms a slight curve to the left lateral side in an axial plane, resulting in a corresponding transverse fold of the rectum within the rectal lumen (the fold of Kohlrausch) (*Schuenke et al.*, 2010).

The extensible rectal ampulla enables the filling of the rectum which leads to urgency via the activation of rectal receptors. The rectum further takes an S-shaped course within the pelvic cavity, which can be seen when viewed in a sagittal plane: the sacral flexure, which adapts to the concavity of the sacrum, and the perineal flexure of the anorectal junction, which includes a 90° angle with the sacral portion of the rectum (anorectal angle). The perineal flexure is supported by the anterior and superior traction of the puborectalis sling. The anorectal angle increases during normal defecation (~130°) (Aigner and Fritsch, 2010).

The Anal Canal

The surgical anal canal is approximately 4 cm long and extends from the anal verge to the anorectal ring, which is defined as the proximal level of the levator–external anal sphincter complex. The embryological anal canal extends from the anal valves to the anal margin and is approximately 2 cm long (*Cook and Mortenson*, 2002).

The anal canal is attached posteriorly to the coccyx by the anococcygeal ligament, a midline fibromuscular structure, which runs between the posterior aspect of the EAS and the coccyx. The anus is surrounded laterally and posteriorly by loose adipose tissue within the ischioanal fossae, which is a pathway for spread of perianal sepsis from one side to the other. The pudendal nerves pass over the ischial spines at this point (*Standring*, 2005 a).

The perineal body separates the anal canal from the vagina. The anal canal is lined by an inner epithelium, a vascular subepithelium, IAS, EAS and fibromuscular supporting tissue. The lining of the anal canal varies along its length due to its embryologic derivation. The proximal anal canal is lined with rectal mucosa (columnar epithelium) and is arranged in vertical mucosal folds called the columns of Morgagni (*Cook and Mortenson*, 2002).

Each column contains a terminal radical of the superior rectal artery and vein. The vessels are largest in the left-lateral, right- posterior and right-anterior quadrants of the wall of the anal canal where the sub-epithelial tissues expand into three anal cushions. These cushions seal the anal canal and help maintain continence of flatus and liquid stools. The columns are joined at their inferior margin by crescentic folds called anal valves (*Thakar and Fenner*, 2007).

About 2 cm from the anal verge, the anal valves create a demarcation called the dentate line. Anoderm covers the last 1–1.5 cm of the distal canal below the dentate line and consists of modified squamous epithelium (*Standring*, 2005 a).

As a result of tonic circumferential contraction of the sphincter, the skin is arranged in radiating folds around the anus and is called the anal margin. These folds appear to be flat or ironed out when there is underlying sphincter damage. The junction between the columnar and squamous epithelia is referred to as the anal transitional zone, which is variable in

height and position and often contains islands of squamous epithelium extending into columnar epithelium (*Kaiser and Ortega*, 2002).

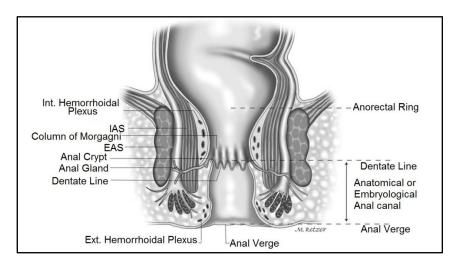


Figure (2): Anal canal (Jorge and Habr-Gama, 2011).

Anal Sphincter Complex

The anal sphincter complex consists of the EAS and IAS separated by the conjoint longitudinal coat. Although they form a single unit, they are distinct in structure and function (*Thakar and Fenner*, 2007).

Internal Anal Sphincter (IAS)

The IAS is a 2 to 3 mm thick circular band representing the distal 2.5 to 4.0 cm condensation of the yeircular muscle layer of the rectum and ends with a well defined rounded edge about 1.2 cm distal to the dentate line at the junction of the superficial and subcutaneous parts of the EAS. In contrast to the EAS, the IAS has a pale appearance to the naked eye (*Jorge and Habr-Gama*, 2011).

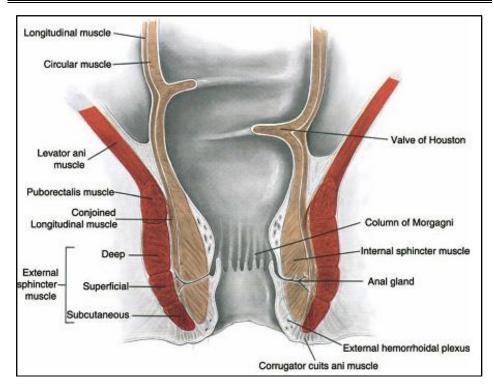


Figure (3): Anal canal (Nivatvongs and Gordon, 2007)

External Anal Sphincter (EAS)

EAS is subdivided into three parts: the subcutaneous, superficial and deep. In females, the EAS is shorter anteriorly. The deep EAS is related to the puborectalis muscle and does not have posterior attachments. The superficial EAS is attached posteriorly to the anococcygeal ligament, which is attached to the tip of the coccyx. The subcutaneous part is circular but may have attachments to the perineal body anteriorly and the anococcygeal ligament posteriorly. In females, the bulbospongiosus and the transverse perineii fuse with the EAS in the lower part of the perineum (*Thakar and Fenner, 2007*).

The Pelvic Floor (pelvic diaphragm)

The pelvic floor is a musculotendineous sheet that spans the pelvic outlet and consists mainly by two levator ani muscles. The fasciae investing the muscles are continuous with visceral pelvic fascia above, perineal fascia below and obturator fascia laterally. The pelvic floor supports the urogenital organs and the anorectum. The muscles of the levator ani maintain constant tone, except during voiding, defectaion and the valsalva manoeuvre; have the ability to contract quickly at the time of acute stress (cough or sneeze) to maintain continence; and distend during delivery (*Barber et al.*, 2002).

The levator ani muscle

It is a broad muscular sheet attached to the internal surface of the true pelvis and is subdivided into iliococcygeus, pubococcygeus and ischiococcygeus (*Standring*, 2005 b).

The ischiococcygeus part may be considered as a separate muscle. It is a narrow triangular sheet of muscular and tendinous fibers, its apex arising from the spine of the ischium and sacrospinous ligament, and its base inserting into the margin of the coccyx and into the side of the lowest piece of the sacrum. It assists the levator ani and piriformis in closing the back of the pelvic outlet. The iliococcygeus muscle is the lateral component of the levator ani muscle and arises from the ischial spine below and anterior to the attachment of the ischiococcygeus and to the obturator fascia as far forward as the obturator canal (*Thakar and Fenner*, 2007).

The most posterior fibers are attached to the coccyx and the sacrum, and join with fibers from the opposite side to form a raphe, which continuous with the anococcygeal ligament, and is attached to the coccyx and anococcygeal raphe. The pubococcygeus arises from the back of the pubis and from the anterior part of the obturator fascia, and is directed backward horizontally along the side of the anal canal to attached to be inserted into the coccyx and into the last one or two pieces of the sacrum. The pubococcygeus is subdivided into separate parts (i.e. pubourethralis and puborectalis in the male, pubovaginalis and puborectalis in the female) (*Bartolo and Macdonald*, 2002).

The most medial fibers of the pubococcygeus form a sling around the rectum named the puborectalis. The puborectalis is the most caudal component of the levator ani complex. It is situated cephalad to the deep component of the external anal sphincter, from which it is almost inseparable. Its U-shaped sling pulls the anorectal junction anteriorly to the posterior aspect of the pubis, resulting in an angulation between the rectum and anal canal called the anorectal angle that has a role in maintaining continence. Between the two arms of the puborectalis lies the levator hiatus, through which the rectum, vagina and urethra pass. In the female, the anterior fibres of the levator ani descend upon the side of the vagina to form the puboperineal muscle (*Kearney et al., 2004*).

Innervation of the Levator Ani

The levator ani is supplied on its superior surface by the sacral nerve roots (S2–S4) and on its inferior surface by the

perineal branch of the pudendal nerve, recent cadaveric dissections along with nerve staining studies have shown that the female levator ani is not innervated by the pudendal nerve, but rather by innervation that originates in the sacral nerve roots (S3–S5) and travels on the superior surface of the pelvic floor; levator ani nerve (*Barber et al.*, 2002).

The pubococcygeus is supplied by (S2-S3) spinal segments via the pudendal nerve, and the ischiococcygeus and iliococcygeus are supplied directly by (S3-S4) spinal segments (*Standring*, 2005b).

Vascular Supply

The levator ani is supplied by branches of the inferior gluteal artery, the inferior vesical artery and the pudendal artery (*Standring*, 2005 b).

The Rectogenital Septum (Denonvilliers' fascia)

It forms an incomplete ventrocranial partition between the rectum and the urogenital organs and is completed caudally by the perineal body. Its collagenous fibers and smooth muscle cells are integrated in front of the rectal wall. It is longitudinally oriented smooth muscle fibers that can be traced to the ventral rectal wall, level with the middle transverse fold of the rectum (fold of Kohlrausch), splitting into muscle fibers between the sphincter muscles, covering the anal sphincter muscles in the ventral position, and terminate in the perineal body (*Sebe et al.*, 2005).

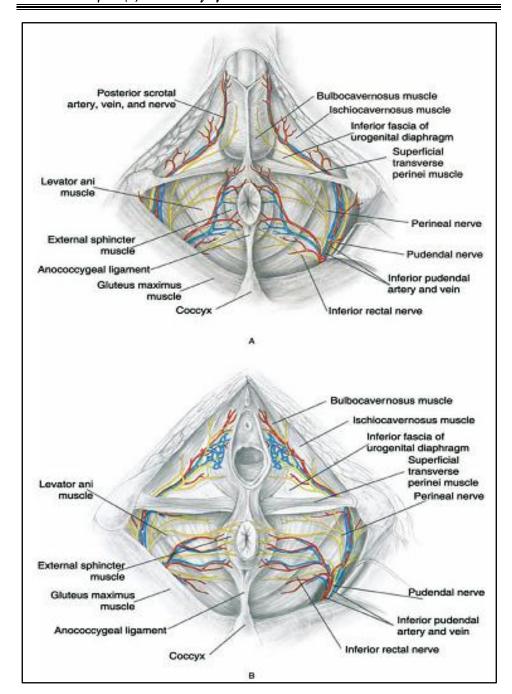


Figure (4): Pelvic Floor, (A) Male perineum. (B) Female perineum (Nivatvongs and Gordon, 2007)