

# **A Neurophysiological Evaluation Of Idiopathic Anorectal Incontinence and Rectal Prolapse**

A Thesis submitted for partial fulfillment of the Doctor Degree in  
Physical Medicine

By

**Hanan Ahmed Fahmy**

MB, Bch, Msc Physical Medicine, Ain Shams University

Under supervision of

**Prof. Dr. Nadia Abdel Salam El Kadery**

Professor and Head of Physical Medicine Department,  
Ain Shams University

**Dr. Nagla Ali Gadallah**

Assistant Professor of Physical Medicine,  
Ain Shams University

**Dr. Medhat Mahmoud Assem**

Assistant Professor of General Surgery, Cairo University

**Dr. Ahmed Farag Ahmed**

Assistant Professor of General Surgery, Cairo University

**Dr. Nahed Monir Sherif**

Lecturer of Physical Medicine, Ain Shams University

Faculty Of Medicine, Ain Shams University  
Cairo-Egypt



بِسْمِ اللَّهِ الرَّحْمَنِ الرَّحِيمِ

قَالَ اسْمُكَ لِلْعَالَمِ لَنَا إِلَّا مَا عَلَّمْنَا بِكَ أَنْتَ  
وَالْعَالِمُ وَالْحَكِيمُ



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## **LIST OF ABBREVIATIONS**

<b>FI:</b>	<b>Fecal Incontinence</b>
<b>IFI:</b>	<b>Idiopathic Fecal Incontinence</b>
<b>RP:</b>	<b>Rectal Prolapse</b>
<b>PD:</b>	<b>Perineal Descent</b>
<b>PN:</b>	<b>Pudendal Nerve</b>
<b>PL:</b>	<b>Pudendal Latency</b>
<b>PNTML:</b>	<b>Pudendal Nerve Terminal Motor Latency</b>
<b>SEP:</b>	<b>Somatosensory Evoked Potentials</b>
<b>EAS:</b>	<b>External Anal Sphincter</b>
<b>LA:</b>	<b>Levator Ani muscle</b>
<b>PR:</b>	<b>Puborectalis muscle</b>
<b>SQ:</b>	<b>Squeeze action</b>
<b>ST:</b>	<b>Strain action</b>
<b>MUAP:</b>	<b>Motor Unit Action Potential</b>

## **AIM OF THE WORK**

- 1- Review the literature about idiopathic fecal incontinence and rectal prolapse.
- 2- Conduct a clinical study on patients with idiopathic fecal incontinence and/or rectal prolapse, and do an electrophysiological study of the pelvic floor muscles in order to try to find out the cause of idiopathic fecal incontinence and/or rectal prolapse, which may be helpful in choosing the proper line of management



# Chapter I

## INTRODUCTION

Fecal incontinence is not a diagnosis but a symptom. For the first time clinicians are beginning to realize that incontinence of feces is common (Kiff, 1992). Many patients do not seek medical advice about fecal incontinence and thus suffer social alienation and serious psychiatric isolation (Snooks et al, 1985a). As a consequence, reliable epidemiological data on incidence and prevalence of fecal incontinence is unavailable (Enck et al, 1991).

Rectal prolapse is a distressing condition that is associated with fecal incontinence in 50-70% of patients (Andrews and Jones, 1992). Incontinence is particularly prevalent in elderly patients. The prolapse itself is socially embarrassing. The prolapse specially if it descends during normal activities (Keighley, 1993 b).

The pudendal nerve which arises from the anterior primary rami of S2,3 and 4 is involved intimately in the bowel, bladder and sexual function. Traction neuropathy of this nerve is supposed to be the cause of idiopathic fecal incontinence and rectal prolapse (Henry et al, 1982).

The pudendal nerve has sensory fibers which receives sensory information from the anus, pelvic floor muscles, urethra, and penis; and motor fibers which supply the pelvic floor muscles, external anal sphincter, external urethral sphincter as well as the bulbocavernous muscle (Meckel, 1979). Specific functions in which the pudendal nerve has physiologic role include defecation and stool continence, voiding and urinary continence as well as penile erection and ejaculation (Haldeman et al, 1982).

Rectal prolapse is, in effect, an intussusception of the rectum, a condition in which the full thickness of the rectum descends through the anus. It is a disorder of the pelvic floor, and while much of its etiology remains obscure, certain facts have emerged.

Anal canal resting pressures and squeeze pressures are low in these patients, there is frequently descent of the perineum on straining (Parks et al, 1966). Conduction time in the nerves which supply the sphincter mechanism are prolonged, and the biopsy of the muscles shows evidence of denervation. Therefore, it is likely that rectal prolapse is due to traction neuropathy of the pudendal nerve resulting in progressive denervation of the pelvic diaphragm.

Idiopathic fecal incontinence (IFI) is thought to be due to weakness of the striated muscles of the pelvic floor and external anal sphincter as a result of entrapment of the pudendal nerve as it angulates around the ischial spine to enter the upper part of the pudendal canal during the stretch that occurs during perineal descent (Parks et al, 1977).

Swash (1985) suggested that prolonged straining during defecation or vaginal delivery may cause perineal descent which is a frequent finding in patients with IFI, thus descending perineum syndrome (DPS) has been regarded as a precursor of IFI.

Swash et al (1984) have studied the electromyography (EMG) of the pelvic floor and the pudendal nerve terminal motor latency and noticed decrease in the mean integrated activity during one second of maximum contraction in external anal sphincter and puborectalis muscle, also they noticed delay in the terminal motor latency of the pudendal nerve.

As IFI and rectal prolapse are not uncommon conditions which are both annoying and embarrassing to the patients and as their etiology is still obscure, the nerve pathways together with the muscles involved in their pathophysiology should be evaluated.

# Chapter II

## REVIEW OF LITERATURE

## **Anatomical Considerations**

### **1- Anatomy of the rectum:**

Although anatomists traditionally assign the origin of the rectum to the level of the third sacral vertebra, surgeons generally consider the rectum to begin at the level of the sacral promontory. It descends along the curvature of the sacrum and coccyx and ends by passing through the levator ani muscles at which level it abruptly turns downwards and backwards to become the anal canal. This anorectal angle which is maintained by the puborectalis sling has been regarded as an important mechanism in maintaining continence. It measures 12 to 15 cms (Goligher, 1984).

The rectum describes three lateral curves: the upper and lower curves are convex to the right, and the middle is convex to the left. On their inner aspect these foldings into the lumen are known as the valves of Houston (Abramson, 1978).

The upper third of the rectum is covered by peritoneum anteriorly and laterally, the middle third is covered only anteriorly, and the lower third is devoid of peritoneum.

Shafik (1982) stated that the rectum is the part of the hind gut which extends from the third sacral vertebra to the perineal skin. The narrowest and lowest part of the rectum which extends from the levator plate to the perineal skin has been named the "rectal neck". The "rectal neck inlet" occupies the junction between the rectum and its neck, whereas the "rectal neck outlet" denotes the anal orifice (Fig 1). The "rectal neck" in adults varied in length from 1.5 to 2 inches. It is directed downwards and slightly backwards being slinged to the symphysis pubis by the top

loop of the external sphincter (Shafik, 1982). Four muscles are related to the rectal neck so as to constitute an integral part of its wall: the internal sphincter, the longitudinal muscle, the external sphincter, and the levator ani muscle.

The angle between the rectum and its neck is termed the "rectal angle". It lies about 1.5 inches above the anal outlet. Two structures are related to the rectal neck; the top loop of the external sphincter and the anococcygeal raphe. The top loop lies in direct relation to the rectal angle, slings it forward to the symphysis pubis, the anorectal raphe is connected to the rectal angle by the hiatal ligament and fixes the angle backwards to the coccyx.

The rectal angle is a right angle created by the resting tone of the top loop of the external sphincter. The extension of the perineal skin into the rectum is called the intrarectal skin, while that surrounding the rectal outlet is the perirectal skin (Shafik, 1982).

The fascia of the rectum is formed of:

a) Lateral ligament: fibrous element in fibrofatty tissue form a triangular shaped ligament on either sides of the rectum with the base laterally towards pelvic side wall and the apex joining the side of the rectum. Inside these ligaments pass the middle rectal vessel and branches of the hypogastric plexus.

b) Fascia of Denonvieliers: a condensed layer of visceral pelvic fascia covers the extraperitoneal part of the rectum and continued below the superior fascia of the urogenital diaphragm, and laterally it becomes continued with the front of the lateral ligament. It intervenes between the rectum behind and the prostate and seminal vesicles or the vagina anteriorly (Fig 2).