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Pelvi-perineal functional anatomy

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Abstract

The complex anatomy of the pelvi-perineal region is the basis of the interaction between organs, muscles and ligaments that are responsible for different functions. Proctology, urology and gynecology are involved. This paper aims to describe all the organs and muscular-ligament structures of pelvis and perineum and their physiology, before and after surgical interventions. The involvement of anatomical structures in the main functional diseases of the region is also described.

Key words

Functional anatomy, surgical anatomy, pelvic floor, bladder, uterus, vagina, rectum, and anus.

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1. PELVIC FUNCTIONAL ANATOMY & PROLAPSES

a) Anorectal embriology.

Much information on anorectal malformations comes from animal model studies and this explains why appropriate embryologic descriptions are still lacking. As a result, pediatric surgeons are often confused in their understanding of abnormal embryonic development and understanding of normal embryology stems from the interpretations of observed malformations than from proper embryologic observations. The cloaca, already present in the 15-day old embryo, is the terminal part of hindgut. Traditionally, the normal differentiation of the cloaca into the dorsal anorectum and the ventral urogenital tract is attributable to the proper process of septation by the so called urorectal septum. The study starting point, in a 12.5-day-old rat embryo (comparable with human embryos between the third and 7th week of gestation), shows all features of a typical “cloaca”: the hindgut enters the cloaca from dorsocranial side whereas the allantois can be identified as a cranioventral diverticulum. The dorsal part, forming part of the hindgut, will form the rectum and the anus. The ventral part is the allantois, which will become the urinary bladder. Between this diverticulum and the hindgut, the urorectal fold can be seen: this fold marks the cranial border of the undifferentiated hindgut, the so-called “cloaca” [1]. In the same moment, the future anal orifice is placed in the dorsal part of the cloacal membrane close to the tail groove. Therefore, it is not the result of the migration of the rectum that does not take place. Some investigators still assume that a shift of the rectum (“caudal migration”) or a shift of the caudal cloaca to the tail groove is necessary to establish the anorectal canal but this claim is replaced by the experimental findings. In this way the assumption that anal ectopia is the result of an abnormal migration of the rectum is overcome. At 6 weeks of gestation, the cloacal membrane is disrupted by the descent of the urorectal septum that replaces it, while an anal membrane develops in the primitive anal canal: anal membrane disappears at 8 weeks of gestation, evolving in the deep part of the anal canal [2]. The cutaneous part of the anal canal, located below the white line, is instead of ectodermal origin and derives from an external ectodermic folding named proctodeum. The rectum is the evolution of dorsal anorectum of cloaca. It is located behind the urogenital tract, makes contact first and then continues with the deep anal canal. Course of the rectum, its anatomical relationships with contiguous organs and structure of the wall are the result of the late evolution in the last three months of pregnancy. Their description goes beyond the scope of this chapter and therefore they will not be described. In concomitance with the first outlines of pelvic organs formation, two separate groups of muscles start to grow: tail muscle or pubo-caudal muscle and cloacal or Gegenbauer muscle; from the former the ischio-coccigeus muscle, levator ani muscle and pubo-sacral and sacro-spinal ligaments will descent, from the latter the sphincters [3]. After the genito-urinary septum will separate the rectum posteriorly from the urethra-vagina anteriorly, the Gegenbauer's muscle will divide itself into two sphincters, one for the urethra and one for the rectum; successively ischio-cavernous muscle, bulbo-cavernous muscle and superficial and deep transverse muscles are added.

b) Anorectal malformations.

1. Functional anatomy

Anorectal malformations (ARMs) are among the more frequent congenital anomalies encountered in pediatric surgery, with an estimated incidence ranging between 1 in 2000 and 1 in 5000 live births[4]. The need to classify anorectal malformations derives from the aim of offering useful elements for the treatment with the predictability of its outcome. For this reason, over time some classifications, different from each other, based on numerous factors have been detailed. The Wing spread conference classification (1984) was based on the position of the terminal bowel in relation to the levator ani or pelvic floor[5]. Peña proposed a classification in 1995 (Table 1) based on the presence and position of the fistula (if perineal, rectourethral or rectovesical) because of his experience with the posterior sagittal anorectoplasty[6].

Table 1. Anorectal malformations. Peña classification.

Males	Female
Perineal fistula	Perineal fistula
Rectourethral fistula	Vestibular fistula
Bulbar	
Prostatic	
Rectovesical fistula	Persistent cloaca
	<3 cm common channel
	>3 cm common channel
Imperforate anus without fistula	Imperforate anus without fistula
Rectal atresia	Rectal atresia

Finally, Krickenbeck group in 2005 gave a classification to standardize the methodology for evaluation of outcome of patients with ARM and included elements of Wing spread and Peña classifications[7]. It incorporated three distinct sections: a diagnostic category, a surgical procedure category, and a category documenting functional outcome.

Whichever classification has been selected, the anatomical elements that must be considered for choosing the surgical technique are:

1. *Pouch perineal distance*. The presence of a fistula is combined on the rectal side with a distal pouch. The upper edge of pouch is distant to varying degrees, depending on the specific case, from the perineal plane. This distance can be identified by ultrasonic examination via transperineal or infracoccygeal route.

2. *Levator ani muscle (pelvic diaphragm).* The biggest part of levator ani and ischio-coccigeus deal with organ support and delimits the funnel-shaped dome containing the pelvic organs. It is therefore crucial to know the relationship between the muscles of the pelvic diaphragm, the rectum and the vagina in the presence of a rectovaginal fistula.

3. *Sphincteric muscles*. External and internal anal sphincters surround the anal canal and both together are the major structural components for fecal continence. Anal ectopia

and anal atresia need a perfect reconstructive position of the “ex-novo” anal canal surrounded by muscular rings.

4. *Perineal muscles*. Ischio-cavernous muscle, bulbo-cavernous muscle and superficial and deep transverse muscles must be pre-operatively identified. The positioning of the anal canal must consider the anatomical-functional relationships of these muscles with the anal sphincter apparatus to finalize their coordinate correct functioning.

Diagnostic techniques, to be used to identify the type of anorectal malformation and its muscle-visceral relationship with the contiguous structures, are: pelvi-perineal MRI, transperineal sonography and intraoperative perineal EMG mapping. The last carries out the electromyographic perineal mapping in search of muscle fibers that are indicative of the presence of the external anal sphincter in the perineum of newborns with anal atresia.

2. Functional Anatomy after surgical treatment

Operative procedures that may be used in anorectal malformations are:

- Posterior sagittal anorectoplasty.
- Anterior sagittal approach.
- Sacroperineal approach.
- Perineal operation.
- Abdominosacroperineal pull-through.
- Abdominoperineal pull-through.
- Laparoscopic assisted pull-through

Understanding of anatomy and thus the principal of surgical repair of ARM has evolved continuously over the past century. Despite the vast number of procedure and their modification, the postoperative outcome of ARM remained dismal and variable throughout the world until Peña introduced the posterior sagittal approach (PSARP) for the repair of high anorectal malformations[6]. A complete exposure of the anorectal region by means of a median sagittal incision that runs from the sacrum to the anal dimple, cutting through all muscle structures behind the rectum, allows access to the rectum. The superficial and deep layers of the external sphincter are identified by electrostimulation and divided in the midline. Ileo- and pubococcygeal portions of the levator dorsally and then the striated muscle complex of the external sphincter, pubococcygeus and the presumed puborectalis are split ventrally to the urethra. In this way the posterior sagittal approach provides an excellent exposure for evaluation and mobilization of the terminal bowel. It enables one to construct an anal canal, suture the bowel wall to the striated musculature and the mucosa to the skin. Soon after its introduction, PSARP became the gold standard procedure worldwide for anorectal malformations, both with fistula and without fistula. This approach allowed surgeons to view the anatomy of these defects clearly, to repair them under direct vision, and to learn about the complex anatomic arrangement of the junction of the rectum and genitourinary tract.

There has been debate over single stage versus staged surgical repair of ARM. Single stage procedure has less morbidity, mortality and low cost. Moreover, easier dissection in the neonatal period, due to virgin tissue planes with no fibrosis induced by pouchitis, and no need

for bowel preparation and colostomy, are all together the other principal factors suggesting a single stage operation.

The success of a surgical procedure for ARM must be judged not only for the successful correction of the anatomic damage but also for the achievement of correct defecation and urine discharge. These functions may be evaluated to start with 1 year old baby and they will improve as the complete maturation of the continence factors is reached, maturation to be judged complete only after 4 years of age.

C) Pelvic floor

The Pelvic floor closes the pelvis and holds organs (uterus, vagina, rectum, urethra, bladder and prostate) inside the body. It has a deep layer (pelvic diaphragm and urogenital diaphragm) and a superficial layer (perineum).

DEEP LAYER

1. Pelvic diaphragm

Pelvic diaphragm is the deep layer of pelvic floor. Levator ani muscle and urogenital diaphragm make up pelvic diaphragm. Pelvic cup closure is insured by this muscular-fascial connection whose fibrous and muscular structure are rigidly fixed because attached to pelvic bones.

a) ***Levator ani***. This muscle includes fibers of ischiococcygeus, iliococcygeus, pubococcygeus and puborectalis muscles. Some authors consider the ischiococcygeus muscle as a muscle, given the well-marked and separated insertions from the other constituents of the levator ani. Ischiococcygeus, pubococcygeus and iliococcygeus muscles, with their insertions on ilium, ischium and pubis, deal with organ support and to the pelvic cup closure, while only puborectalis muscle has a visceral function because its fibers reach rectum, urethra and vagina[3]. The former is put between bone and bone and exert tone and isometric contraction; the latter has an isotonic contraction and does not insert itself directly and properly on organs but with its contraction it shortens and acts as a fulcrum followed by a movement of the organ or of a part of it. Levator ani contributes to circumscribe the hole through which prostate, urethra, vagina and rectum pass: this hole is named urogenital hiatus. This hiatus has a funnel conformation at anatomic dissection while nuclear magnetic resonance demonstrates the disposition of muscular bundles be horizontal, as a plate. This different view is due to the complete absence of muscles contraction in cadavers while in living a firm tone is constantly present, which expires only during micturition and defecation.

Ischiococcygeus muscle. The spatial arrangement of ischiococcygeus muscle fibers, directed from above their bony attachments on the ischial spines and sacrospinous ligaments to down the midline on the coccyx, explains the function of posterior support for the rectal ampulla. The rectum, in rest conditions, lies down on it: during the defecation act, the contraction of the ischio-coccygeus muscle pushes anteriorly the superior part of rectal ampulla, forward, contributing to the alignment movement of the rectum on the anal canal.

Iliococcygeus muscle. The muscle fibers are directed from the arcus tendinous of levator ani, a thickening of obturator internus muscle, to the coccyx posteriorly and to the perineal body medially. It contributes to the creation of anococcygeal ligament. The function of iliococcygeus muscle is like that of ischiococcygeus and concerns the rectum support, in a lower portion towards the anal canal. The important relationship with the arcus tendinous of the levator ani explains what happens during defecation in a squatting position. The internal obturator, rigidly fixed to the arcus tendinous, contracts to induce the external rotation of the femur, in turn stimulating at the same time the contraction of the iliococcygeal muscle and therefore the alignment of the rectum with the anal canal for anterior tilting of the inferior rectal ampulla.

Pubococcygeus muscle. Some authors divide the pubococcygeus muscle into three regions named: puboperineal, pubovaginal (in women) and puboanal[8]. These portions have different distal insertions in addition to the coccyx. The puboperineal has muscular fibers that are directed from pubic branch to the perineal body, fixing levator ani in the middle inferior of perineum. The pubovaginal closes the urogenital hiatus and surrounds the vagina crashing into its wall: it is the muscle involved by finger vaginal exploration during the pelvic floor muscle function test[9]. It modulates the vaginal penis receptivity and the intercourse feelings: its hyperactivity leads to introital dyspareunia. Finally, the puboanal fibers fuse posteriorly with those of deep portion of external anal sphincter: the contraction of these muscle fibers contributes to the closure of the anal canal in a posterior-anterior direction during maximal anal voluntary contraction.

Puborectalis muscle. It is the visceral part of levator ani[3]. The muscle fibers arise from the pubis near the symphysis; they go posteriorly and in the final part they surround the rectum at the level of the posterior wall of the bowel merging into each other. Thus, a sling is formed. This anatomic muscular arrangement, due to basic tonic contraction, causes the formation of an anorectal angle of approximately 90°. Thus, an angle of the anal canal is created on the terminal portion of the rectal ampulla. This angle is determinant for fecal continence in conditions of rest: any increase in intra-abdominal pressure further folds the rectum towards the anal canal ("flap valve theory"), also increased by the simultaneous reflex contraction of the puborectalis muscle, and urgent fecal continence occurs. Finally, fibers of the puborectalis muscle go on the one hand into the deep part of the external anal sphincter, fusing with it, and on the other into the longitudinal muscular layer of the rectal wall, forming the longitudinal joint muscle in an intersphincteric position. The contractile activity of the puborectalis muscle is particularly complex. Simultaneously sharing with the external anal sphincter, it contracts during voluntary contractile recruitment for emergency continence, causing the anorectal angle to become more acute thanks to the posterior-anterior direction of the force vectors. Conversely, the puborectalis voluntary relaxation, implemented at the beginning of the defecation act, makes the anorectal angle obtuse, contributing to the alignment of the rectum with the anal canal. In this way the rectum can empty without any mechanical obstacle to the transit of feces.

All the contractile activity of the muscle, basic tone, voluntary contraction and voluntary relaxation is regulated thanks to the activity of nerve branches of the pudendal nerve[10].

b) *Urogenital diaphragm*. Below the levator ani, the anterior urogenital diaphragm lies formed by perineal membrane (or midperineal aponeurosis) which spans the deep transverse perineal muscles. This strong membrane has a triangular shape strained between the two ischiopubic branches; its apex looks anteriorly toward the pubic symphysis and is bored by urethra and vagina, fills the space where in the anterior part of perineum the two levator ani do not join each other. The urethral

part, also called transverse perineal ligament, is strongly connected to ischiopubic branches and to obturator fascia; it has very dense structure so much to become so sharp to guillottine the urethra in the ischiopubic fractures dislocations. The retro-urethral part divides itself in two sheets, inferior, well thick and pressure-proof, and superior thin. The two sheets of perineal membrane are in continuation: the inferior one with the inferior perineal fascia covering the superficial transverse perineal muscle; the superior one with Denonvilliers fascia. Based on the anatomical arrangement and structure, the function of the urogenital diaphragm is to support the urethra and vagina, encapsulating them in a strong fibrous structure and fixing them to the perineum. A recent anatomical study defined the spatial arrangement of the rectourethral muscle, an anterior extension of the rectal longitudinal smooth muscle that connects the lower part of the front wall of the rectum with the rectum-belt of the Denonvilliers fascia[11]. It has the function of fixing and stabilizing the urethra at the rear.

SUPERFICIAL LAYER - PERINEUM

The perineum may be considered the superficial plane of the pelvic floor. It is the region containing fat and muscles below the pelvic diaphragm extending to the perineal skin. Topographically, the perineum has a lozenge shape and a theoretical line connecting the two ischial tuberosities of the pelvis divides the perineum into the posterior anal triangle containing anus and anterior urogenital triangle containing urethra and vagina in women and only urethra in men. The perineum lodges, in addition to superficial and deep (transverse) perineal muscles, striated urethral and anal sphincters and bulbo and ischio-cavernous muscles in both sexes.

2. **Superficial plane of perineum**. The superior perineal fascia limits deep the superficial plane of perineum, which contains perineal body and bilateral superficial muscles: some muscles are placed around the genital system (bulbocavernosus, ischiocavernosus, and the superficial transverse perinei muscles) and others around the anal canal (external anal sphincter). All these muscles, excluding the ischiocavernosus muscle, are anchored medially to the perineal body, a fibrous dense structure placed halfway between the anal verge and the posterior commissure of the labia major. Rectovaginal septum and levator ani muscle fibers also converge on it. The superficial plane of perineum has certain roles, both for the urogenital and the anal function. The ischiocavernosus muscle participates in the rigidity and erection of penis (or clitoris)[12]. Bulbocavernosus muscle propels the semen along the penile urethra[13]. Superficial transverse perinei muscles support the "straining-perineal reflex" with their contraction on straining[14]. This reflex, which results in perineal muscle contraction against the increased intra-abdominal pressure, resists the tendency of the perineum to descend. Its impairment is the first pathophysiological element of descending perineum syndrome[15].

3. **Perineal body.** As already mentioned, the perineal body, also referred to as the tendon center of the perineum, is the point of convergence of many muscular and fascial structures. It works as an anchor for all these structures and therefore helps to mechanically stabilize not only the pelvic floor, but also the pelvic viscera. It is easy to understand how in maternal birth trauma with posterior perineal tears, structure disruption may occur, with repercussions both on the statics and on the dynamics of

the pelvic viscera. Diagnosis of maternal birth lesions by translabial ultrasound imaging is mandatory[16]. Levator ani muscle avulsion, anal sphincter injury, puborectalis muscle and other perineal muscles defects may be detected, all of them variously combined with injuries to perineal body.

4. **Rectogenital septum**. Rectogenital septum exists in both women (rectovaginal septum, RVaS) and men (rectovesical septum, RVS)[17]: it begins under the rectogenital pouch. Rectovaginal septum has the shape of a curtain, placed between the posterior wall of the vagina and the anterior wall of the rectal ampulla. It is pulled in its upper corners by the bilateral anchorage on the tendon arch and, in the lower tract, by the fixation on the perineal body: it converges approximately midway between the pubis (3.75 cm) and the ischial spine (4.8 cm)[18]. The rectovaginal septum is formed of a network of collagen, elastic fibres, smooth muscle cells, nerve fibres emerging from the autonomic inferior hypogastric plexus, and variable numbers of small vessels[19]. The rectovesical septum is located between the seminal vesicles and the anterior wall of the rectum. It begins below the peritoneal reflection of the rectovesical space and continues as Denonvilliers' fascia up to the perineal body. It encloses along its layers several fibers of the neurovascular bundle that are important to spare when running radical prostatectomy[20].

The rectogenital septum is considered as sliding plane between the rectum and the urogenital system, necessary to allow the filling-emptying of the rectum (RVaS and RVS) and vaginal coitus (RVaS). Moreover, crucial is the RVaS role in rectocele because its trauma with tears causes the least resistance on the anterior wall of the rectum.

5. **Pelvic sub-peritoneal connective tissue**. Biomechanics of the pelvic floor are influenced by connective tissue disposed between muscular and visceral pelvic content. Fat and fibrous connective tissue are the support scaffold for all pelvic contents and actively participate in pelvic dynamics and statics. Subperitoneal fat, in addition to a widespread distribution, accumulates in some areas, which need a more accurate description. Three areas are well defined: ischiorectal pit, subperitoneal fat between the peritoneum and levator ani and, to a lesser extent, fat in the rectovesical space. Ischiorectal pit is the area between the levator ani superiorly, obturator internus muscle and ischial bone laterally, rectal ampulla and anal canal medially, perineal and perianal skin inferiorly. A transverse dense fibrous septum divides the ischiorectal fossa into a deep and a superficial part, while in the posterior area the anococcygeal raphe divides a deep postanal space from a superficial postanal space. The fat contained in the ischiorectal fossa has a shot-absorbing activity and allows the volumetric modification of the rectum during its expansion due to the presence of feces in the ampulla. It also contains important structures because the Alcock canals are positioned laterally, in contact with the fascia of the internal obturator muscle and the ischial tuberosity: they contain the pudendal vessels, artery and vein, and the pudendal nerve. The ischiorectal pit is the area where perianal horseshoe abscesses spread in the postanal spaces by the passage of pus below and/or above the anococcygeal raphe. The subperitoneal fat between peritoneum and levator ani muscles is a thin layer of fat and connective tissue, with a laminar arrangement, which entirely covers superiorly the levator ani muscle. It contains the ureters in their course before entering the bladder. The fat in the rectovesical space lies behind the Denonvilliers fascia; it forms the cleavage plane between the rectum and the urogenital component. This plan, surgically, allows the blunt separation between the rectum, on one side, and the vagina, in the female, or the prostate-bladder complex, in the male, on other side.

Mesorectum is below described in the paragraph relating to the rectum.

6. **Pelvic fascia**. The pelvic fascia is a variable density structure of connective tissue that covers, with a subperitoneal location, the pelvis both on its musculoskeletal wall, *parietal pelvic fascia*, and its visceral content, *visceral pelvic fascia*. The parietal pelvic fascia, continuation of the umbilical-prevesical fascia, is distributed bilaterally towards the pelvic walls and forms some thickenings in the anteroposterior direction: the arcuate ligament of the pubis, the tendon arch of the levator ani muscle, the medial pubovesical ligament and the lateral pubovesical ligament. The visceral fascial coverage of the upper surfaces of the bladder, uterus, and rectum, in women, contributes, after having covered them, to the formation of the bladder-uterus and uterus-rectum cavities. The visceral pelvic fascia delimits, posteriorly, the walls of pararectal space. Lateral rectal ligaments are constant dense connective bundles which are in either lateral side of the lower part of the rectum, run between rectal visceral fascia and pelvic parietal fascia above the levator ani, and covered by superior fascia of pelvic diaphragm. They are pathways of blood vessels and nerve fibers toward the rectum and lymphatic vessels from the lower rectum toward the iliac lymph nodes. The visceral pelvic fascia forms a series of ligaments which stabilize the pelvic viscera: the spatial distribution, schematically, reproduces a cross (Richard's cross) which presents, around the bladder, uterus and rectum placed in central median position, two anterior pillars, two lateral and two rear. On each side, one anterior pillar is respectively formed by the pubourethral and uteropelvic ligaments, the lateral pillar is the cardinal ligament, the posterior pillar is the uterosacral ligament, reinforced by loose connective tissue arranged around the vascular-nervous structures of the superior hypogastric plexus and the middle rectal artery. All these ligaments not only constitute the support scaffolding for the pelvic viscera but, also, they are landmarks in surgical anatomy because they include important vessels (venous, arterial and lymphatic) and nerve trunks. The cardinal ligaments contain the uterine vessels, in the lateral pubovesical ligaments are positioned the inferior bladder vessels and vaginal vessels, together with the ureter, the uterosacral ligaments are adjacent to the internal iliac vessels. The intrapelvic parts of the autonomic nervous system are the hypogastric nerves, which derive from the superior hypogastric plexus and carry the sympathetic signals to the internal urethral and anal sphincters as well as to the pelvic visceral proprioception; and the pelvic splanchnic nerves, which arise from S2 to S4 and carry nociceptive and parasympathetic signals to the bladder, rectum, and the sigmoid and left colons. The hypogastric and pelvic splanchnic nerves merge into the pararectal fossae of visceral pelvic fascia to form the inferior hypogastric plexus. This anatomical disposition is a fundamental landmark for hysterectomy and anterior rectal resection when performed with nerve sparing techniques.

7. **Blood supply and lymphatics**. The pelvic viscera are supplied by venous and arterial vessels coming from the internal iliac vessels: in their path they are accompanied by the lymphatics which, during their route, flow into some lymph node stations. The rectum and the anus are supplied by the rectal arteries. The superior rectal artery, terminal branch of the inferior mesenteric artery, is distributed in the wall of the upper third of the rectum: in the same walls there is the beginning rise of the superior rectal vein which ends in the inferior mesenteric vein. At the origin of the inferior mesenteric artery is fixed the lymphatic drainage station where the greatest amount of lymph, coming from the pelvic areas, converges. Also, bilateral rectal middle artery, not present in all people, comes out from internal iliac artery. Its destination is the wall of

the middle rectum, with terminal branches intersecting, in the superior and inferior territories, with those of the superior and inferior rectal arteries. Finally, the inferior rectal artery, bilaterally, arises from the internal pudendal artery before it enters the Alcock canal; its terminal branches are destined for the inferior rectum and the anal canal. The lymphatic drainage of the middle and lower rectum goes to lymph nodes located along the respective arterial vessels and from there to the internal iliac lymph nodes. Lymph from the anal canal and the neighboring rectal ampulla is drained to the inguinal lymph nodes. The blood supply to the uterus originates mainly from the uterine artery coming from internal iliac artery, but the uterine and ovarian arteries form anastomoses bilaterally. The vaginal artery is the main arterial vessel for vagina but there are also vaginal vessels from uterine artery. The veins follow the same path backwards next to the arterial vessels. Lymphatic drainage is mainly directed towards internal iliac nodes. The bladder has superior and inferior arteries coming bilaterally from internal iliac artery, which are accompanied in their route by venous vessels directed with inverse flow to internal iliac veins. Internal and external iliac nodes drain lymph coming from bladder.

D) Colon.

The colon is the viscera responsible for the formation, transit and storage of feces before their expulsion outside. The anus is considered separate due to its embryological deriving, structure and function.

Traditionally, colon is divided into 6 sections, all with a different caliber and diameter: cecum, ascending colon, transverse colon, descending colon, sigmoid colon and rectum, each of which has a slightly different function than the others. The colon, with an average length of 150-170 cm, begins at the ileocecal valve and ends at the level of the anal canal, occupies lateral and pelvic sections of the abdomen and contracts relationships with different organs during its long route. The structure of the wall has 4 layers, present throughout the colon: mucous, submucosal, muscular, serous. The mucous layer is lined with mucous cells and contains glandular crypts in its thickness. Inside glandular crypts there are many cellular components: numerous goblet cells, Paneth cells, few enteroendocrine cells and undifferentiated cells for the continuous replacement of the lining epithelium. The mucous layer is divided from the submucosal layer by muscularis mucosae. It is a thin smooth muscular layer which adjusts, with its contraction/relaxation, the local shortening and lengthening of the mucosa on the submucosa. The submucosal tunica consists of connective tissue and contains vascular structures, lymphatic structures and the nerve structures of Meissner's plexus. The muscular wall of the colon is structured in two overlapping layers of smooth muscle: the inner layer with circular muscle, covered by the outer layer of longitudinal muscle. The circular layer has smooth muscle fibers arranged in a radial conformation: their contraction circularly narrows the lumen of the bowel ("haustra"). It produces the formation of small chambers which reduce the speed of progression of the endoluminal content and at the same time favors its absorption. This type of contraction is responsible for the segmental activity of the viscus[21], which shows a cranio-caudal gradient, progressively increasing from the ascending colon in the distal direction and becoming of maximum intensity and duration at the sigmoid level (sphincter of O'Beyrne). The longitudinal musculature, grouped in some areas of thickening visible on the outside ("teniae"), is made up of muscle fibers whose contraction involves the shortening of the bowel and the propulsion of the intraluminal contents at a distance ("mass movements")[22]. The serous layer corresponds to the peritoneal lining which, in the colon, completely covers the bowel only in some sections. Appendix, caecum, transverse colon, and sigmoid colon are fully lined; ascending colon, descending colon are

retroperitoneal because only the anterior surface is lined by parietal peritoneum. The rectum, only in the upper 1/3, is intraperitoneal; about 8 cm from the anal margin, the rectum becomes subperitoneal due to the reflection fold of the layer of the parietal peritoneum which forms the Douglas split.

Scattered throughout the colonic circular muscle there are clusters of ganglion cells and interstitial cells of Cajal (ICC). Nervous connections between ganglion cells and ICC are overly complex: it is assumed that ICC generate spontaneous pacemaker currents which are conducted to smooth muscle cells causing rhythmic contractile patterns[23]. Regulation of colonic motility depends on the integrity of enteric inhibitory neurotransmission mediated by nitric oxide (NO), purine neurotransmitters, and neuropeptides. ICC and platelet-derived growth factor receptor-α-positive (PDGFRα+) cells engage in generating responses to NO and purine neurotransmitters, respectively[24] but the role of neurotransmitters and effector cells in the neural regulation of gastrointestinal motility has not been definitively provided. The presence of numerous nitric oxide synthase-positive neurons, all along the colon and at both plexuses, Meissner's and Auerbach's, supports the hypothesis that excessive production of nitric oxide may cause the persistent inhibition of contractions that may be detected in idiopathic chronic constipation[25].

In the following sections, the anatomical-functional consequences of colonic resections will be described. Shortening of the colon and therefore the reduction of colonic transit times with an increase in the transit speed of fecal substance will be the common consequences for all types of colonic resections, both open, laparoscopic and robotic.

Right hemicolectomy. Right hemicolectomy involves the removal of the entire right colon and the last ileal loop: ileocecal valve, appendix, cecum, ascending colon and right flexure will be removed, and the operating piece will be 15-20 cm long. The ileocolic resection is a partial resection, where the section line on the colon will fall on the ascending colon.

The reconstructive anastomosis of right hemicolectomy will be an ileal-transverse latero-lateral or end-to-end anastomosis: possible could be also the end-to-side anastomosis.

Both ileocolic resection and right hemicolectomy remove the ileocecal valve, changing the enteric liquid inflow into the colon. The sphincteric function of the ileocecal valve is lost. Uncontrolled and continuous flow of the enteric fluid passes into the colon: more than 1500 ml/day can enter the bowel downstream by overloading the residual colon with water and electrolytes. Ileocecal valve removal also allows easy back transition of colonic bacteria into the ileum: this phenomenon may induce small intestinal bacterial overgrowth (SIBO) syndrome that is characterized by diarrhea and weight loss[26]. Fast transit time, liquid overload and bacterial overgrowth, all together, explain the possible high frequency of bowel movements that may appear after ileocecal resections or right colectomies.

As mentioned above, the ileocecal valve affects the motility of the small intestine: by regulating the passage of chyme into the colon, it is part of the "ileal brake" which is the main inhibitory feedback mechanism that controls the transit of a meal through the gastrointestinal tract to optimize digestion and nutrient absorption[27]. The absence of the ileocecal valve can compromise the "ileal brake" and disarticulate the motor coordination of the intestinal loops. Postoperative ileus may be prolonged due to small bowel dysmotility: gastric stagnation begins and may last a few days, until gastric and small bowel motility resumes.

Transverse colectomy. Transverse colectomy may be performed for colonic lesions located between the hepatic flexure and the splenic flexure. Removal of transverse colon together with

both flexures may be used for tumors. The location of the transverse tumor determines the extent of surgery and whether the middle colic artery should be included in the resection. It has been shown that colectomies together with complete mesocolic excision (CME) have demonstrated increased lymph node collection, increased distance between the tumor and the central vascular linkage, and ultimately increased disease-free and overall survival[28]. Even if this surgery, for tumors treatment, is now codified, the evaluation of functional effects is lacking in scientific literature. There are only studies that compare laparoscopic versus open transverse colectomy. They showed that the time taken to dispel flatus and the time before oral feeding were significantly shorter in laparoscopic transverse colectomy than in open transverse colectomy[29].

Left colectomy – Sigmoid colectomy. Regardless of the type of surgery involving a sigmoid resection (left colectomy, extended left colectomy, sigmoid colectomy, total colectomy, anterior resection of the rectum and recanalization after Hartmann's procedure) the functional effects on colonic physiology will always be the same. The removal of the "rectosigmoid brake" involves, as mentioned above, in addition to the variable shortening of the intestine - depending on the type of surgery - a fast colonic transit with reduced colonic transit time. The result is a higher frequency of defecation compared to the preoperative situation with loose stools discharge. Colonic manometry may reveal reduced background segmental contractile activity, sometimes combined with an increased frequency of giant migratory waves[30]. This manometric finding suggests that the full propulsive activity of the colon, because increased, acts against a decreased resistance of segmental activity, vice versa reduced.

Anterior resection of the rectum.

Anterior resection of the rectum removes sigmoid colon, totally or partially, and rectum up to a different level of the rectal ampulla: high rectal resection, with colorectal anastomosis 8-10 from the anal verge, low rectal resection with extraperitoneal colorectal anastomosis below 8 cm from the anal margin.

Ultralow anterior resection, performed with an anastomosis under 5 cm from anal verge, may be supported by ultralow colorectal anastomosis, coloanal anastomosis or intersphincteric resection. The functional asset background for these surgical operations is a combination of a small neorectal capacity and relative high endo-neorectal pressures which together act against a weakened sphincter mechanism[31]. In addition, as consequences of the effects of sigmoid resection on colonic transit, they also can have negative influences on bowel movements. This functional background, which is often worsened after adjuvant or neo-adjuvant radiotherapy, sometimes results in a severe defecatory damage, known as low anterior resection syndrome (LARS)[32]. As mentioned above, negative influences on anal sphincter function may often take place. Anal sphincter damage may occur and it is due to injuries of the internal anal sphincter: up to 18 % of patients who undergo stapled low anterior resection have long-term evidence of internal anal sphincter injury[33]. Internal anal sphincter function may also be compromised if the internal anal sphincter nerves are damaged[34]. Finally, ultralow anterior resections may sometimes be associated with anal sensation defects, such as sometimes occur after intersphincteric resection[35]: excisions of anal mucosa, particularly in the anal transitional zone might destroy the typifying receptors that allow the patient to discriminate between flatus and liquid or solid stool. The effects, variously combined, on colonic function, neorectal capacity and, finally, on anal function are the main reasons that lead to the great variety of signs and symptoms in LARS: they include a mix of high bowel frequency/day with liquid stools, sometimes multiple evacuations within a limited time, urgency, and fecal incontinence.

Total colectomy – Restorative proctocolectomy.

Total colectomy with ileorectal anastomosis (IRA) is often performed for inflammatory bowel disease, more rarely for colic atony. The removal of the entire colon up to the borders of the upper rectum is followed by subsequent anastomosis between the terminal ileum and the residual rectal stump. Total proctocolectomy involves the complete removal of the colon and rectum and is performed for diffuse familial polyposis, ulcerative colitis, or other multicentric disease of the colon. Bowel function is restored with a definitive ileostomy or an ileo-anal anastomosis, either straight or through an ileal pouch-anal anastomosis (ILA) (total restorative proctocolectomy). In IRA or in restorative proctocolectomy intestinal continuity is restored by using the ileum. The ileum is an intestine with no tract for volumetric capacity, nor for storage of contents. Furthermore, there are two other crucial factors: firstly, the ileal contents are liquid or semiliquid, and, secondly, the transport of the intraluminal fluid is constant and continuous, without any temporal interruption, until it reaches the anastomotic site. Even if the rectum has been saved or a pouch has been created to implement volumetric capacity, ileal motility and the type of fluid contained are not greatly affected, with a continuous incoming ileal fluid that develops the conditions for high defecation frequency due to the great flow rate. Urgency and fecal incontinence may appear if malfunctioning of the neorectum and/or anus materializes.

Preservation of the rectum positively influences intestinal function. In patients affected by familiar adenomatous polyposis, IRA has been compared with ILA[36]. Bowel frequency, night defecation and use of incontinence pads were significantly less in the ileorectal group, although fecal urgency was reduced with the ileal pouch. This implies that rectal volumetric capacity and rectoanal coordination are determinant for an optimal defecation.

E) Rectum

The rectum begins at the point of the sacral promontory where the taenia coli fuse to form a continuous longitudinal muscle layer, approximately at S3 level: it is long about 13-15 centimeters. It descends along the curve of the sacral hollow to the level of the levator ani and then turns downwards and anteriorly through the anorectal ring where it becomes continuous with the anal canal. The rectum is anatomically separated into upper, middle, and lower thirds based on approximate distances between the lower, middle, and upper valves of Houston and the dentate line (DL). *Upper rectum* is considered from left upper Houston valve (HV) to right middle HV at 11 cm from the dentate line, *middle rectum* between right middle HV and left lower HV at 7 centimeters from DL, *lower rectum* from under left lower HV to the upper edge of anatomical anal canal. This distinction of the borders and then of the rectum sections is decisive for the surgical treatment strategies for rectal cancer. The rectum upper limit, radiologically identified at S3 as anatomical marker, has been recently used in the method for application to the rectum segmentation for geometrical modeling in MRI, and as contextual information source in virtual colonoscopies and computer aided detection systems[37].

The rectum and its fascia propria are connected to the pelvic parietal fascia by two lateral ligaments and one posterior ligament (called rectosacral fascia) serving as an anchor for the rectum to the pelvic walls. Lateral ligaments are constant dense connective bundles which are in either lateral side of the lower part of the rectum, run between rectal visceral fascia and pelvic parietal fascia above the levator ani, and covered by superior fascia of pelvic diaphragm. They are pathways of blood vessels and nerve fibers toward and from the rectum and of lymphatic vessels from the lower rectum toward the iliac internal lymph nodes[38]. Clamping, dividing and ligating these ligaments are essential procedures in surgical mobilization of the

rectum. The functional effects of this maneuver are controversial: on the one hand the section of the ligaments influences the transit of the colon with total and segmental colonic transit times doubled, on the other hand the effects on anorectal function are not significant and do not significantly influence postoperative functional outcome[39].

The mesorectum, the pararectal autonomic nerves and the nodes stations of lymphatic drainage are the surgical anatomical landmarks for cancer rectal operations.

The term *mesorectum* has been largely used by surgeons to indicate the perirectal adipose tissue contained within the lower rectal fascia[40]. The rectum does not have a true “meso”, that is a peritoneal fold linking a segment of the digestive tube to the wall and containing vessels and nerves. Posteriorly, the lower rectum lacks any peritoneal folds. Posterior to the rectal fascia, an avascular space (the retrorectal space) is present. This space is filled with adipose tissue and is situated between the rectal and the presacral fascia (the portion of parietal pelvic fascia which covers the sacral bone)[41]. The “so called mesorectum” contains vessels, nerves and lymphatics, all connected to rectal walls. A mesorectal fascia is juxtaposed and covers the adipose tissue: it forms the surgical cleavage plan to perform the total mesorectal excision (TME), specific surgical timing of low and ultralow anterior rectal resections for cancer of the rectum. TME, with its intact enveloping fascia, provides for complete removal of fat with the lymph nodes, and respects the hypogastric nerves. Complete strip TME, leading to a perfect lymphadenectomy, drastically reduces local recurrences to 1-4%.

The autonomic nerves of the pararectal spaces are the sympathetic hypogastric nerves and the parasympathetic pelvic plexus. The superior hypogastric plexus is the direct extension of the aortic plexus below the aortic bifurcation. It lies immediately behind the peritoneum and descends over the anterior surface of the fifth lumbar vertebra in the retroperitoneal tissue. The superior hypogastric plexus ends by bifurcating into the right and left hypogastric nerves[42]. The two hypogastric nerves diverge from each other at about the level of the sacral promontory and run down and forward along the walls of the pelvis in the lamina of the pelvic fascia closest to the peritoneum. The nerves of pelvic plexus (“erigentes nervi”) take origin from the second to fourth sacral ventral rami just after the sacral nerves have emerged from the pelvic sacral foramina[42]. They always form plexus at the lateral ligaments and are crossed by middle rectal arteries. The knowledge of the course of both groups of nerves, hypogastric and those of pelvic plexus, is essential to perform the nerve sparing technique that is adopted in anterior rectal resection for cancer. The dissection must be accurate. At the level of the sacral promontory, the superior hypogastric plexus gives rise to the right and left hypogastric nerves, which are found along the posterolateral pelvic brim just below the course of the ureters. The hypogastric nerves lie in the areolar tissue plane between the visceral mesorectal fascia and the endopelvic parietal fascia and are easily isolated and preserved. The erigentes nervi, in close anatomical relationship with the lateral ligaments, must be saved during the maneuver of dividing the lateral ligaments: this maneuver should be performed near the rectum, as close as possible, to achieve nerve preservation.

Rectal lymph nodes are dispersed across multiple lymph node stations: pararectal, leaning against the rectum, lateral pelvic lymph node in the lateral ligaments from the lower rectum toward the internal iliac lymph nodes, lymph nodes around inferior mesenteric artery and finally deep and superficial inguinal lymph nodes from lower anorectal wall. Lymphadenectomy is a fundamental step in the surgical treatment of rectal cancer but there is no one-size-fits-all behavior on how to perform it. The decision of ligation at the origin of the inferior mesenteric artery (IMA) or below the origin of the left colic artery has remained a dilemma for surgeons in colorectal cancer surgery. A meta-analysis of 3119 patients showed that high ligation of IMA

has a better survival benefit for the patients with IMA positive lymph nodes. It signifies that high ligation should be recommended for the advanced cases or with the suspected elevated risk of IMA lymphatic metastasis[43]. Lateral pelvic lymph-node metastases occur in 10-25% of patients with rectal cancer and are associated with higher local recurrence and reduced survival rates. A meta-analysis of 2577 patients was undertaken to assess the value of extended lateral pelvic lymphadenectomy in the operative management of rectal cancer. Extended lymphadenectomy did not seem to confer a significant overall cancer-specific advantage but did seem to be associated with increased urinary and sexual dysfunction[44].

Rectum controls storage and gas/stool expulsion. It is empty during resting conditions and fills when feces have been carried by colonic mass movements. Defecation takes place due to the coordinated activity of multiple anatomical elements: an integrated somatic and visceral activity occurs thanks to the interaction of somatic components, such as the perineal pelvic striated muscles and the external anal sphincter, and visceral components, such as the sensorimotor activity of the rectum/colon and the internal anal sphincter. The sensorimotor activity of the rectum is a complex function that depends on rectal tonic parietal adaptation to its content and on its wall motor activity. Anorectal manometry measures this activity. Volumetric rectal sensations are the Conscious Rectal sensitivity Threshold (CRST), that is the minimal perception of fecal bolus, the Constant Sensation (CS), that is the constant perception with desire to defecate or call to stool and the Maximal Tolerated Volume (MTV) that is the unbearable distressing defecatory perception indicative, for convention, of the maximum rectal volumetric capacity[45]. The response of rectal wall adaptation to increasing endoluminal volumes is, on the contrary, evaluated by means of Rectal Compliance that is expressed by a curve built from $\Delta V/\Delta P$. A fully ambulatory system for prolonged pressure recording was developed and used to study the relationship between anal canal tone and rectal motor activity[46]. Rectal motor complexes, such as bursts of contractions seen in the rectum, were identified in all studied subjects. Importantly, a rectal motor complex was invariably accompanied by a rise in mean anal canal pressure and contractile activity such that pressure in the anal canal was always greater than pressure in the rectum. This temporal relationship could represent an important mechanism preserving fecal continence but the physiological role of the rectal motor complexes is still to be defined: it has been suggested that rectal motor complexes may serve as an intrinsic braking mechanism that prevents untimely flow of colonic contents, particularly during sleep[47].

F) Anal canal

The anal canal is the terminal segment of the alimentary tract and lies entirely below the level of the pelvic floor in the region termed the perineum. Where the rectum passes through the pelvic diaphragm, the puborectalis portion of the levator ani muscles fuses with the longitudinal muscle coat of the rectum and, together with the deepest portion of the external sphincter, forms a prominent fibromuscular ring that is called the ano-rectal ring[48]. The anal canal passes downwards and backwards from its beginning at the ano-rectal ring, forming a right-angle (Anorectal Angle: ARA, approximately 90°) with the termination of the rectum. The anal canal is classically presented as the anatomical anal canal or the surgical anal canal. The first, the anatomical anal canal, is confined between the anal verge and the dentate line and it is about 2.5-3 cm long. Its mucous layer is covered by a layered non-keratinized squamous epithelium which presents a smooth appearance. There is copious innervation with a variety of specialized sensory nerve endings: Meissner's corpuscles which record touch sensation, Krause endbulbs which respond to thermal stimuli, Golgi-Mazzoni bodies and Pacinian

corpuscles which support the answer to changes in tension and pressure, and genital corpuscles which are responsive to friction. In addition, there are large diameter free nerve endings sensitive to pain within the epithelium[49]. The surgical anal canal is long about 4-4.5 cm, placed between the anal verge and the apexes of Morgagni rectal columns: it corresponds to the functional length of the high resting pressure zone that is present in the anal canal and it represents the surface entity to be considered for sphincter-saving operations. The epithelium above the dentate line is like that of epithelial lining of the rectal mucosa and is made up of columnar cells, crypts and goblet cells. It is relatively insensitive to pain and no specific sensory receptors have been detected through histological examination of the rectum in humans[50].

The submucosal layer of anal canal contains hemorrhoidal tissue that is arranged on the whole circumference (360°) of the anal wall but is also assembled in three cushions respectively in the left lateral position (at 3.00 o'clock), the right anterior position (at 7.00) and the right posterior position (at 11.00). This spatial arrangement helps to close the lumen, sealing the anal canal, and determines 5-10% of anal resting pressure (ARP). The structure of hemorrhoidal tissue is made of connective tissue with elastic fibers that contains smooth muscular fibers (Treitz's muscle) and arterioles, venules and arteriovenous anastomoses which all take together a cavernous-like conformation. The Treitz's muscle fixes the hemorrhoidal tissue to the internal anal sphincter: destructive changes in this supporting connective tissue, within the anal hemorrhoidal cushions, is the paramount condition of hemorrhoidal prolapse. The muscular structures of surgical anal canal include the internal anal sphincter (IAS), the external anal sphincter (EAS), the joint longitudinal muscle and the puborectalis portion of the levator ani muscle.

1. ***Internal anal sphincter (IAS)***. The IAS is an extension of the inner circular smooth muscle layer of the rectum[50]. It is wrapped superiorly by the puborectalis portion of the levator ani muscle, then more distally by the superficial external sphincter muscle (an extension of the anococcygeal ligament), and subsequently by the subcutaneous external striated anal sphincter muscle. Radiologic studies by means of MRI showed that IAS is approximately 3-3,5 cm in length and continually closed by tonic contraction[51]. An anatomical study performed by Uz et al[52] demonstrated that the IAS is composed of flat rings of smooth muscle bundles stacked on top of each other. The mean number of ring lamellae observed was 26.33 +/- 2.93 (range = 20-30) and each was covered by its own band. The smooth fibers and fascia merged at three equally spaced points around the anal canal to form three columns extending distally into the lumen. At rest, the ring-like lamellae have a spatially organized structure as horizontal leaves within the columns and play a key role in closing the lumen of the anal canal, thus promoting anal continence. During defecation the three columns are pulled peripherally towards the fibromuscular fascia of the joint longitudinal muscle, the annular lamellae become vertical, opening the lumen and allowing stool to pass distally.

The IAS, as mentioned above, develops important tone for maintaining high anal pressure (70% of ARP) and continence. The mechanism underlying tone generation is controversial. The hypothesis that tone depends upon generation of electrical slow waves initiated in intramuscular interstitial cells of Cajal (ICC-IM) by activation of Ca2+ -activated Cl- channels and voltage-dependent L-type Ca2+ channels has been recently advanced[53]. The inhibition of IAS contractile activity (Recto Anal Inhibitory Reflex: RAIR) would be conversely activated by nitric –oxide (NO) release from non-cholinergic non-adrenergic nerve endings. Add to all this that the IAS also has an extrinsic autonomic nerve supply by means of internal anal sphincter nerves that emerge from the anterior-inferior edge of the pelvic plexus. They travel within the

neurovascular bundle along the inner surface of the levator ani muscle, antero-laterally to the rectum, to penetrate the longitudinal rectal muscle layer just at the fusion line with the pubococcygeal muscle at the anorectal junction to form the joint longitudinal muscle[34]. At this point they enter the intersphincteric space to reach the IAS. Their identification and precisely described topographical location provides a basis for nerve-sparing rectal resection procedures and help to prevent postoperative functional anorectal disorders due to internal anal sphincter nerves lesions[34]. Whenever possible, transection of the rectum, during anterior rectal resection with total mesorectum excision, should be done proximally to the conjunction of the pubococcygeal and longitudinal rectal muscle just at the lowermost end of the mesorectum. On the contrary, preservation of these nerves in intersphincteric resections is not usually possible as a rule, because the region of their penetration into the anorectal muscle tube is just a part of the resected specimen.

As mentioned above, IAS is involved in the resting anal pressure (ARP) and in its transient relaxation during the sampling reflex. In the first case it is an involuntary tonic contraction, in the second it follows the distension of the rectal ampulla from a volume of gas and/or feces. Both mechanisms are fundamental. ARP is one of the mechanisms that engage in regulating the anal continence. The sampling reflex is instead the reflex by which defecation starts thanks to the cortical perception of the contents of the rectal ampulla[54]. The volumetric distension of the rectum is perceived by distension receptors located in the levator ani and through a reflex sacral arc induces the relaxation of the IAS (RectoAnal Inhibitory Reflex: RAIR), mediated by locally NO release from non-cholinergic non-adrenergic nerve endings. Thanks to this reflex small amount of feces penetrate the anal canal, encountering typing receptors located in its mucosa. The receptors, in turn, send information to the parietal cortex, via afferent medullary pathways, on the content with which they have come into contact, whether fecal or gaseous ("*sampling reflex*"). In this way, the cortical typification of the ampullary content is achieved.

2. **Joint longitudinal muscle**. The joint longitudinal muscle (JLM) originates from the fusion of the striated muscle fibers of the puborectalis muscle with the smooth muscle fibers of the longitudinal rectal muscle, occupying the intersphincteric space between the internal anal sphincter and the external anal sphincter, finally ending up peeling into fibers anchored in the perianal subcutaneous tissue[55]. The path of the muscle has suggested that its contraction causes the wrinkling of the anus, for this reason the muscle is also called the "corrugator muscle of the anus", but it is very probable that its function is to assist during defecation by everting the anus. It has been hypothesized that it may participate in fecal continence by influencing resting anal pressure but its specific role in this function is not known. Presence of oblique fibers suggest that the JLM may represent the intermediate longitudinal course of small bridging muscle bundles going reciprocally from the striated EAS to the smooth IAS and vice versa. The spatial result is the helical course of striated and smooth muscle fibers between the EAS and IAS, which contribute not only to the narrowing but also to some shortening of the anal canal during sphincter contraction[56]. Being present in the intersphincteric plane, JLM is involved also during the intersphincteric resection of the rectum, pointing out the planes to be followed in surgical dissection that may be achievable to optimize resection results[57]. It must be borne in mind that the intersphincteric plane might be irregular and unpredictable due to an inter-individual variability and/or age-related atrophy. Furthermore, in some subjects the JLM is partly lacking and the area between the IAS and EAS is filled with adipose tissue, so there is not true intersphincteric space, as adipose tissue is present when parts of the JLM are lacking. The intersphincteric plane is a potential plane rather than an actual plane. Therefore, as the amount of adipose tissue between the IAS and EAS generally

increases towards the anal aperture, some surgeons may prefer to start an intersphincteric resection by perineal dissection[57].

3. **External anal sphincter**. The EAS forms the inferior external aspect of the anal sphincter and envelops the intersphincteric space almost completely 360°. The external sphincter is longer and wider than the internal sphincter, and the distal border of the EAS is normally at least 1 cm distal to that of the IAS. Between these two edges, the intersphincteric groove at the anal margin is relatively easy to palpate. Sex-related differences include a significantly shorter external sphincter in women than in men both laterally and anteriorly[58].

The external anal sphincter is conventionally described as consisting of three parts: a subcutaneous part, a superficial part and a deep part which all act synergistically together[50]. The *subcutaneous part* is included in the perianal subcutaneous tissue, in contact with the external hemorrhoidal plexus and is crossed by joint longitudinal muscle fibers. The *superficial part*, which constitutes the main portion of the muscle, originates from a narrow tendon band, the anococcygeal raphe, which extends from the tip of the coccyx to the posterior margin of the anus; it forms two flattened planes of muscle tissue, which surround the anus and meet anteriorly to insert at the central tendinous point of the perineum, joining with the superficial transverse perineal muscle, levator ani, and bulbocavernosus muscle. The *deep part* is fused with the puborectalis muscle behind the rectoanal junction, which is identified by touch as the anorectal ring. Shafik hypothesized that the synergistic activity of the EAS parts may occur by means of a triple loop system, consistent respectively with the three EAS parts, whereby the muscle spatial organization allows for sealing off or opening the anal canal[59].

EAS comprises predominantly slow-twitch muscle fibers: it is capable of prolonged contraction but with age there is a shift towards more type II (rapid) fibers[60]. His muscular activity shows a tonic asset at rest, responsible for about 20% of anal resting pressure. The contractile activity, synchronous with that of the puborectalis muscle, is related to 1) further voluntary muscular recruitment that provides contraction as an emergency continence mechanism, manometrically identified by maximal voluntary contraction (MVC) or to 2) a voluntary relaxation that opens the anal canal during defecation, a function detected by means of the manometric straining test during attempts to strain to defecate[61]. Finally, EAS is responsible for the Rectoanal Excitatory Reflex (RAER), integrated into the sampling reflex together with that of the RAIR: it prevents the leakage of typed material into the anal canal during IAS release[54].

EAS contractile activity is controlled by somatic nerves, the right and left inferior rectal nerves, each derived directly from the corresponding pudendal nerve (S2-S4).

G) Mechanisms of continence and defecation.

Fecal continence and defecation are managed by the differentiated interaction of the same factors, by means of synchronized interplay of multiple voluntary and involuntary mechanisms: pelvic floor muscles, anal sphincters, motor activity of the colon, sensory and motor activity of the rectum, sensory and motor neural pathways and fecal consistency are the main actors[62].

1. ***Fecal continence*** refers to the ability to contain fecal and/or gaseous material within the rectum, without any external leakage. Following the propagation of the endoluminal

material from the colon to the anus, the first factor to be involved in temporal order is the fecal consistency, the product of the reabsorption of liquids from the colonic lumen on the enteric material. About 1200 ml/day of enteric fluid enters the colon from the ileocecal valve. The reabsorption process, especially of water and electrolytes, involves the progressive concentration of the intraluminal material, up to the emission of 130-150 cc/day of feces. The more the fecal bolus is dehydrated, the greater its hardness and vice versa the greater the water content, the more the feces are soft or liquid. Bristol Stool Form Scale (BSFS) is the recommended tool for assessment of fecal consistency[63]. In a recent study[64] the stool water content was analyzed, where ≤68.5% water content was classified as hard stool (BSFS: grades 1-2), while ≥78% was classified as diarrhea (BSG: grades 7-8). For BSFS categories 1-2, 77% had water content ≤68.5%, whereas for BSFS categories 6-7, 52% had water content ≥78%. The consistency does not depend only on the water content in the fecal bolus, but also on other factors such as, for example, undigested fibers, fatty acids, inorganic matter (calcium and phosphates), mucus, desquamated intestinal cells and intestinal bacterial flora. In any case, liquid stools are associated with urgency and high bowel frequency, often present in 45% of patients with fecal incontinence[65], while hard stools, sometimes caprine-shaped, are typical of chronic constipation[66]. Therapeutic aims will be to solidify the stools in case of fecal incontinence and to make the stools more voluminous and softer in the case of chronic constipation. The motor activity of the colon is responsible for the transport of endoluminal contents. The aboral progression and the retropulsion of the content are mechanisms that have not yet been well defined in their temporal and spatial sequence. Suggestions come from many instrumental investigations, which also include the study of colonic transit times and colonic manometry, but what really happens in the interactions between wall cells/lumen content, colonic muscle contraction and intraluminal transport is not yet known. Some regulatory mechanisms were identified and retrograde contractions responsible of retropulsion movements are also added to the well-known anterograde types of motilities, segmenting activity and mass movements (see what is described above in the paragraphs relating to the description of the colon): retrograde propagating cyclic motor patterns in the sigmoid and rectum promote retrograde transit to prevent the continuous flow of content into the anal canal[62]. In any case it can be said with certainty that the segmenting activity slows down the progression of the content reaching the maximum effect at the level of the rectosigmoid junction with a sphincter-like activity[67,68] while the mass contractions promote its transport over long stretches. Precisely as, a consequence of a mass contraction, the fecal material, both solid and gaseous, is transferred from proximal colonic sections/sigmoid colon into the rectum and can trigger the defecation.

2. ***Defecation***. The arrival of a certain quantity of feces and/or gas in the rectal ampulla, usually empty, stimulates the distention receptors, hidden in the levator ani muscle. The stimulus perceived from the ampulla is exclusively volumetric and the sensory potentials, that depart from the distension receptors, after running across the sensory afferent pathways, finally stimulate the pre-central cortical cortex. Therefore, the rectal volumetric sensations (see the description in the paragraph relating to the rectum) will allow the quantitative perception of the fecal volume collected in the ampulla. Simultaneously, the same material starts the process of identifying the contents in the rectum ("sampling reflex": see above). The identification, in understanding if fecal and/or gaseous material, will be integrated with the volumetric perception of the content and, at that point, the subject will decide whether to defecate, with flatulence

or not, or to postpone defecation. If a decision to defecate is made, the subject should assume the correct squatting or sitting position. In the first case, activation of the internal obturator muscle with external rotation of the thighs will reflexly activate the levator ani muscle via its tendon arch, with the result of relaxation of the puborectalis muscle and contraction of the iliococcygeal and pubococcygeus muscles. In this way there will be alignment of the anal canal on the rectum, for the simultaneity of both the straightening of the anorectal angle and the rectal ampulla overturning in an anterior-posterior direction, consensual to the complete opening of the anal canal. In the sitting position, however, voluntary cortical activation of the entire sequence will be required, sequence which involves almost simultaneously the pelvis-perineal musculature, anal sphincter and rectum. In any case, in both defecatory positions, it is necessary that the anal canal opens completely by relaxing the anal sphincters, both internal and external. The former is released reflexively, with the persisting activation of the inhibitory rectoanal reflex throughout defecation, the latter through voluntary cortical activity. Some years ago, a study was conducted on healthy volunteers to compare squatting end sitting positions for defecation, by measuring abdominal pressure and the anorectal angle simultaneously[69]. The results suggested that the greater the hip flexion achieved by squatting, the straighter the rectoanal canal will be reached, and accordingly, less strain will be required for defecation.

A new Fecobionics® device was developed to imitate the defecation process and provide geometric mapping and manometric profiles in a single examination. The goal was to provide mechanistic understanding of defecation in health and defecatory disorders[70]. The device provided numerous innovations and improvements to current diagnostic technologies, including: (1) mechanical properties that mimic stool, (2) objective electronic measurement of the anorectal angle, and (3) integration of geometric data with multiple anal pressure measurements. By preliminary results, Fecobionics® device seems to give a simple and inexpensive assessment of a range of defecation biomarkers that will be important in research and clinical practice.

H) Pelvic organ prolapse

The clinical definition of Pelvic Organ Prolapse (POP), made by International Urogynecological Consultation in 2021[71], is: "*Anatomical prolapse with descent of at least one of the vaginal walls to or beyond the vaginal hymen with maximal Valsalva effort with the presence either of bothersome characteristic symptoms, most commonly the sensation of vaginal bulge, or of functional or medical compromise due to prolapse without symptom bother.*"

Cystocele, urethrocele, uterine prolapse, colpocele, rectocele and enterocele are considered as belonging to this category. All of them can be associated to pelvic floor dysfunction symptoms: lower urinary tract, bowel, sexual dysfunction and abdominopelvic pain[72]. The same scientific commission also outlined the pathophysiological profile[73], conservative treatment[74] and finally patients' perception of disease burden of pelvic organ prolapse[75].

Some different classifications are used to describe POP. The grading system Baden-Walker “half-way” vaginal profile is simple to use[76]. The most dependent position of the pelvic organs during maximum straining or standing is used and graded as normal or first-, second-, or third-degree prolapse. First-degree prolapse refers to vaginal segments that descend halfway (but not to) the hymen; second-degree is descent to the hymen; and third-degree is prolapse

beyond the hymen. A standardized Pelvic Organ Prolapse Quantification (POPQ) system was introduced to give a better guide[77]. In the POPQ system, the pelvic organ anatomy is described during physical examination of the external genitalia and vaginal canal. This descriptive system contains a series of site-specific measurements of the woman's pelvic organ support. Prolapse in each segment is evaluated and measured relative to the hymen (not introitus), which is a fixed anatomic landmark that can be identified consistently and precisely. The anatomic position of the six defined points for measurement should be in centimeters above or proximal to the hymen (negative number) or centimeters below or distal to the hymen (positive number), with the plane of the hymen being defined as zero. A final profile for quantifying prolapses provides a precise description of anatomy for individual patients.

1. ***Cystocele***. Cystocele is the bladder prolapse into the vagina. Three types of cystoceles are possible: apical, medial, and lateral or paravaginal; a combined mediolateral defect is also possible. A radiological study, performed by means of three-dimensional stress magnetic resonance imaging, reported that two vaginal attachment factors (apical support, paravaginal defects) and levator ani hiatus size are highly correlated and are strong predictors of cystocele presence and size[78]. Each cystocele type can be identified on clinical examination. A defect of anterior vaginal support structures engages in cystocele pathophysiology but different anatomic theories were proposed[78]. Anterior vaginal wall support, or perineal sling, plays a key role in maintaining bladder position. The main support structures involved are the anterior vaginal wall (AVW) plus the pubocervical fascia (PCF), arcus tendinous fasciae pelvis (ATFP), arcus tendinous levator ani (ATLA), endopelvic fascia (EF), and levator ani muscle (LAM). DeLancey's "*hammock theory*"[79] was based on anatomical models, confirmed by MRI studies, establishing, on a transverse plane, a three-level pathologic definition of prolapse. *Level 1* is described as supporting the vagina and cervix by posterior sagittal uterosacral ligaments suspension: here, paracolpium fibers are almost vertical, leading back toward the sacrum. *Level 2* comprises the vagina's lateral connections to the ATFP, supporting the mid-third of the vagina: the vagina is moored laterally to the ATFP and superior ATLA and maintained transversally between bladder and rectum. *Level 3* comprises connections between the perineal membrane and perineal body fascias, supporting the lower third of the vagina and playing an essential role in pelvic statics. The scheme of the ship moored between two piers and held in tension by two hawsers stretched on the bollards is particularly explanatory. The ship is the pelvic organ, the sea is the plane of the elevators, the hawsers are ligaments and fasciae. Defects in one or more of these elements amend to organ statics and may induce cystocele. Petros integral theory[80] proved interdependence between pelvic organ support systems, ligament-fascia injury linkage, and clinical expression. Laxity of vaginal-wall connective tissue and ligamentous–fascial structures are the main pathophysiological factors. The aggravation of degenerative changes in the connective tissue leads to the progression of POP[81]. The Petros theory supposes a sagittal ligamentous–fascial support extending from the posterior pubis in front to the S3/4 sacral concavity behind, comprising pubourethral ligaments and the pubocervical fascia and inserting into the cervical ring (peri-isthmic collagen condensation). This creates a vast sagittal hammock on which, from front to back, lie the urethra, bladder, uterus, and upper rectum between the two uterosacral ligaments. To this is added the pelvic muscular complex, a dynamic pelvic organ support forming the "wings" of the hammock. In Table 2 the structures involved in cystocele pathophysiology are reported according to DeLancey and Petros theories.

Table 2. Anatomic structures involved in the pathophysiology of cystocele according to the theories of DeLancey and Petros.

	Medial cystocele		Apical cystocele		Lateral cystocele	
Anatomic structures	**PCF**	**Endopelvic fascia**	**Cervical ring**	**Uterosacral ligament**	**ATFP**	**ATLA**
Petros	***Yes***	***No***	***Yes***	***No***	***Yes***	***No***
DeLancey	***Yes***	***Yes***	***Yes***	***Yes***	***Yes***	***Yes***

PCF: pubocervical fascia, **Cervical ring**: cardinal ligament insertion, **ATFP**: arcus tendinous fasciae pelvis, **ATLA**: arcus tendinous levator ani.

Therefore, Lamblin et al in 2016, combining the two theories, stated that apical cystocele is due to lesion of the posterosuperior pubocervical support structures; medial cystocele, due to isolated pubocervical fascia lesion; and lateral cystocele, due to transverse hammock failure[78].

Cystocele treatment may be conservative or surgical according to symptoms and prolapse degree.

There are two different surgical approaches: traditional repair and mesh repair[81,82]. Anterior colporrhaphy, performed in 1866 by Sim, was the standard practice for anterior compartment prolapse care but had a high rate of failure, as it is based on the use of women's weak or damaged tissue[83]. In 1990s the use of non-absorbable synthetic mesh began: the anatomical success was higher and there was a significant reduction in complications, post-operative pain, bladder injury, reoperations, perioperative blood loss and days in hospital. Regardless of the surgical technique chosen, the restoration of the anatomy is the goal to be achieved, but the functional results on discharge of urine are few. Really, it is possible that cystocele repair can lead to de novo stress urinary incontinence (SUI) or exacerbate pre-existing SUI. For this reason, when both stress urinary incontinence and cystocele are present, a concomitant anti-incontinence surgery may be combined with cystocele surgical repair[83]. The treatment of stress urinary incontinence in adult women may be performed by means of retropubic tension-free vaginal tape (TVT) or trans-obturator tape (TOT). Efficacy, complications and re-operations are similar in both treatments; both techniques have also good long-term objectives and subjective cure rates[84].

2. ***Uterine prolapse***.

Uterine prolapse is the herniation of the uterus into or beyond the vagina because of failure of the ligamentous and fascial supports[85]. It often coexists with prolapse of the vaginal walls, involving the bladder or rectum. The loss of integrity of the cardinal-uterosacral ligament complex and weakening of the pelvic diaphragm are the main pathophysiological anatomic landmarks. Multiple factors, common also to other POP, are involved: older age, increased body mass index, higher parity, vaginal delivery and chronic constipation are all confirmed risk factors[85]. Additional risk factors include connective tissue disorders such as Marfan syndrome and Ehler's Danlos syndrome[86].

The uterus itself plays no role in uterine prolapse[87]. Anatomic reports in uterine prolapse are related to ligament suspension system disruption. The parametrium, i.e., the subperitoneal connective tissue that surrounds the supravaginal part of the uterine neck and continues both with the connective tissue around the nearby organs and with the one at the base of the broad ligament, is of considerable importance. In the latter site, the parametrium is in continuity with the connective thickenings which accompany and surround the uterine and vesicovaginal vessels and which form, on each side, a robust transverse septum which connects the lateral margin of the uterus and the vagina to the lateral wall of the pelvis, the cardinal ligament. In it is possible to distinguish a medial and a lateral portion, the limit of which is represented by the point where the ureter is crossed by the uterine artery. The medial portion is more robust as it is made up of fibrous connective tissue which includes an intricate vascular tangle mainly made up of the uterovaginal venous plexus; the lateral portion, thinner, is formed by the connective tissue that accompanies the uterine arterial and venous trunks and the middle rectal venous trunk. A lower amount of collagen in the parametrium of women with uterine prolapse, both in menopause and in post-menopause, than in the parametrium of women without prolapse was observed[87]. Cardinal ligaments samples were studied in women affected by uterine prolapse and some structural changes were found: the alterations included loosely arranged connective tissue fibers and less dense extracellular matrix with sparsely distributed fibroblasts[88]. The study, conducted to determine elastin content and RNA expression of related enzymes of elastin synthesis in uterosacral ligament biopsies from women with severe prolapse, and controls with normal pelvic support, suggested that altered elastin metabolism was present in women with uterine prolapse[89].

The descent of the uterus, up to the exit of the bowel outside in the most serious degrees, obviously involves the dislocation of the organs connected with it. Vagina, fallopian tubes, ovaries, bladder and rectum can follow the uterus in its downward progression and undergo consistent anatomical changes. Uterus goes down the vaginal passage and vagina begins to introflect at the level of the vaginal arches which are structurally connected to the uterine walls. Progressive downward invagination of the vagina results in dragging of the bladder by the anterior vaginal wall and drawing of the rectum by the posterior vaginal wall. In this way both cystocele and rectocele may occur. Finally, the surgical anatomic landmark of the ureter, in its course near the uterus, is significant. It is located between the uterosacral ligament and the tendinous arch of the pelvic fascia, crossing and going below the cardinal ligament. DeLancey showed that for every 3 cm of cervical descent in uterine prolapse the ureters descend 1 cm, thereby widening the ureterocervical gap and permitting ligament shortening during vaginal hysterectomy[90].

The anatomical course of the uterine artery is not affected by the dislocation of the uterus: the uterine artery lengthens as the uterus descends but it remains in the usual pathway within the cardinal ligament. So, it can be easily identified and treated during surgical operation for

uterine prolapse, whatever is the operating theater whether during open, laparoscopic or robotic surgery.

A large systematic review had aimed to answer the following clinical questions: what are the effects of non-surgical or surgical treatments in women with genital prolapse? Scientific evidence was found for each proposed procedure and the most appropriate treatment choices to adopt were therefore suggested. Please refer to the article to read the contents[91]. Uterine preservation strategies have been developed, especially if they are women who are still of childbearing age, to suit those patients that wish to maintain future fertility and desire to retain their uterus[92]. Another patient-centered lifestyle advantage includes a natural transition to menopause. Finally, patients who undergo uterine-sparing treatments require continued follow-up for surveillance of gynecological cancers; therefore, uterine preservation is contraindicated in patients that have a history of uterine or cervical oncologic pathology. Hysterectomy, usually, should not be the prime treatment, and fixing the cervix to strong ligament, such as sacrospinous ligament, could give a more successful result[93]. Other techniques, such as abdominal mesh hysteropexy or posterior intravaginal slingoplasty with conservation of the uterus, are alternative surgical options. Hysteropexy allows for decreased intraoperative blood loss, shorter operative time, and faster recovery compared to hysterectomy for prolapse repair. Hysterectomy, performed via a vaginal or transabdominal approach, is indicated in the treatment of grade ≥ 2 uterine prolapse. Vaginal approach is less invasive and offers the opportunity to repair pelvic floor defects. Additional procedures can be performed concomitantly to reduce the risk of prolapse of other pelvic organs. To underline the superiority of conservative surgical procedures, an observational 5-year follow-up study can be cited[93]. It showed significantly less anatomical recurrences of the apical compartment, with bothersome bulge symptoms, or less repeat surgery after sacrospinous hysteropexy when this technique was compared with vaginal hysterectomy with uterosacral ligament suspension.

3. ***Rectocele***

Rectocele is herniation of anterior wall of the rectum into the posterior wall of the vagina. Its progressive enlargement can lead the hernia to emerge on the hymen or, in the most serious cases, outside the vaginal ostium. Structural failure sites in rectocele, by comparing women with and those without posterior vaginal wall prolapse, were recently identified[94]. Lower perineal and lateral posterior vaginal location and enlarged genital hiatus size were strong predictors of rectocele. Based on the width, which is the most widely used parameter during barium defecography, rectoceles are regarded as small (<2 cm), medium (2–4 cm), or large (>4 cm)[94]. Among constipated patients, rectoceles may be associated with increased perineal descent ("*displacement rectocele*") or can be the result of pelvic floor dyssynergia ("*distension rectocele*")[95]. Common pathophysiologic tools are weakness of rectovaginal septum, structural deficit of perineal body, pelvic floor dysfunction; anal sphincter injury and hysterectomy are the most frequently cited risk factors[96]. Loss of integrity of rectovaginal septum can happen in a variety of ways, including childbirth, age-related connective tissue changes, and increased stress on the tissue through straining or obesity. It is easy to think how around least resistance of the rectovaginal septum, increases in pressure can cause the wall to yield, favoring anterior herniation towards the posterior wall of the vagina. The perineal body in patients with low rectocele is damaged or destroyed; live dissection demonstrated that the perineal body in rectocele patients was divided into two parts, joined by a stretched central part, anchored laterally by the deep transverse perineum muscle to the descending ramus of the pubic bone[97]. Abnormal function of the pelvic floor musculature corresponds to either increase activity (hypertonicity) or diminished activity (hypotonicity) or

inappropriate coordination of the pelvic floor muscles[98]. Levator ani defects may induce descending perineum syndrome (DPS): muscular hypotonicity, which is the main landmark, and the excessive straining, typically reported in DPS patients, brings to bear further pressure on the anterior rectal wall. This increased pressure results from the vector of forces, associated with straining, that starts from the abdomen wall contraction and diaphragmatic excursion to overcome the resistance triggered by the distal outlet obstruction, and is channeled towards the pelvis converging on the anterior wall of the rectum and making easy the appearance of a rectocele ("*displacement rectocele*"). The innervation of the rectovaginal septum in patients with displacement rectocele may be associated with increased nerve fiber immunofluorescence intensity, a sign suggestive of reinnervation significantly correlated to increased perineal descent[99]. Inappropriate coordination of pelvic floor muscle is the pathophysiological sign of pelvic floor dyssynergia (PFD): in PFD patients, during the evacuation phase of defecation, the anal canal is not aligned with the rectum due to the failure of the anorectal angle to open, dysfunction caused both by the paradoxical contraction of the puborectalis muscle and by the uncoordinated activity of the pubococcygeal and iliococcygeal muscles. In this way the feces find the mechanical obstacle of the anorectal angle which has become even more acute and the pressure exerted breaks through the anterior part of the rectum towards the vagina inducing the rectocele ("*distension rectocele*").

Rectocele can coexist with several organic causes of obstructed defecation: rectal and rectoanal intussusception, solitary rectal ulcer, descending perineum syndrome, enterocele, and sigmoidocele. Faced with multiple causes of obstructed defecation in the same patient, a series of problems arises related to which of them is the real cause, how much it is related to the symptoms and signs, which is the best therapy to adopt. These are questions that remain without a definitive answer and the best choice to adopt is to try to place the various coexisting organic alterations in a single physiopathological mechanism.

Surgical correction of posterior vaginal wall prolapse includes vaginal, trans-anal and abdominal approaches. Vaginal approaches include site specific repairs and traditional posterior colporrhaphy with levator ani plication. Graft augmentation has been described with both approaches to improve outcomes and decrease failure rates. Among the many trans-anal techniques, a minimal invasive trans-anal approach, known as Stapled Trans-Anal Rectal Resection (STARR), was proposed for treatment of rectocele and rectoanal intussusception, obtaining good outcomes[100]. Over the years, different circular staplers have been placed on the market and the techniques adopted have changed. The peculiarity of first operation involved the packaging of three separated half (180°) purse-strings including mucosa, submucosa and rectal muscle wall, inserted 1–2 cm above the hemorrhoidal apex to include the top of rectocele and internal rectal prolapse. A circular stapler was opened and the head placed above the three anterior half purse-strings; the stapler was then fired and gently withdrawn. A recent review included 24 studies and the total number of patients treated with STARR procedure was 4,464. The results proved that STARR procedure was safe and effective in treating symptoms of obstructed defecation and improving patients Quality of Life (QoL)[101]. Currently, surgical procedures with an abdominal approach have become predominant and many technical variants in laparoscopy, with the use of prostheses, have been described and used. Among them, the laparoscopic ventral rectopexy has better long-term functional outcome, less complications, and less recurrences when compared with stapled transanal rectal resection[102].

The effects of surgery on the pelvic-perineal anatomy are consistent. Surgery by the vaginal route, providing for the use of prosthetic material, strengthens the rectovaginal septum and the perineal plane and, sometimes, depending on the technical variant used, reconstructs the

plane of levator ani. Transanal techniques directly attack the rectocele and eliminate it, consequently reducing the volume of the rectum. The rectal ampulla undergoes volumetric cutting according to the technique adopted. For example, the sizes of surgical STARR specimens may have mean horizontal length of the resected tissue about 6.2 ± 1.6 cm anteriorly and 4.8 ± 1.4 cm posteriorly and mean vertical height about 5.7 ± 1.2 cm anteriorly and 4.8 ± 1.4 cm posteriorly[103]. Finally, the abdominal approaches use prostheses, which may be positioned differently to treat any simultaneous POPs and fix the rectum to the sacrum. It should be remembered that rectopexy, in any case, immobilizes the rectum and affects its motility by reducing its defecatory excursion.

4. ***Enterocele***

Enterocele is defined as a herniation of the peritoneal sac between the vagina and the rectum. Unlike other types of pelvic organ prolapse, enterocele is a true herniation with a peritoneal sac containing small bowel or sigmoid colon (defined also "*sigmoidocele*")[104]. A substantial number of enteroceles are found after hysterectomy or cystopexy. Others pelvic floor disorders are associated in 91% of enterocele: rectocele (25%), cystocele (42%), uterine prolapse (28%), rectal intussusception (52%), rectal prolapse (4%) and abnormal perineal descent (30%)[105]. This focus underlines a pathophysiological path that is common to other POP, but there is the anatomical peculiarity of the peritoneal depression from which enterocele begins. Rectouterine space or pouch of Douglas (cul-de-sac) represents the caudal extension of the peritoneal cavity and it is the area where the parietal peritoneum, as hernial sac, begins to deepen by opening its way into the rectovaginal space. The deepening of the hernial sac determines the progressive disconnection of the rectovaginal septum and the sac with its contents, usually intestinal loops, sits between vagina and rectum. This explains why enterocele can be diagnosed by bidigital examination, vaginal and rectal, appreciating hernial swelling, preferably with the patient in the standing position. The etiology of enterocele is multifactorial. Frequent and prolonged straining at stool, with intra-abdominal pressure force vectors channeled into the Douglas pouch, might result in stretching of peritoneal lining with lesion of the underlying continuity of the layer tissue between vagina and rectum. Failure of fusion of the anterior and posterior peritoneum in the pouch of Douglas, during late fetal development, could support the lesion of the underlying continuity of the tissue layer placed between the vagina and the rectum[106]. Pelvic surgical operations can induce lacerations in the Douglas pouch which, if not repaired, become of least resistance to intra-abdominal pressure. Hysterectomy, if not followed by correct suspension of the vaginal dome, is the typical example of this mechanism. Also, other factors common among POPs, as described above, may play a role: older age, increased body mass index, higher parity, vaginal delivery and chronic constipation are all involved.

Surgical correction aims to reposition the bowel loops in the abdomen, to resect the excess peritoneal sac and finally to seal the neo rectouterine space. There is, at present, no gold standard for the surgical treatment of enteroceles. The results are acceptable in the anatomical correction but sometimes they are questionable on the symptoms and on the defecatory and sexual functions. The founder of enterocele repair was Moschcowitz who, in 1912, was the first to describe an abdominal obliteration of the pouch of Douglas, even if included in the surgical treatment of rectal prolapse[107]. After some years, transvaginal repair was introduced: for many decades, thank also to some technical variants, this approach became extremely popular among gynecologists. The results showed good anatomical repair, but the appearance of "de novo" dyspareunia was an important drawback. Many trans-abdominal procedures were proposed. Open and laparoscopic techniques are now performed:

sac excision and vaginal vault suspension, sacral colpopexy, colporectosacropexy and laparoscopic ventral rectopexy are some of the techniques used.

2. ANAL AND FECAL INCONTINENCES

1. Functional anatomy of anal sphincter complex.

External anal sphincter, joint longitudinal muscle (JLM) and internal anal sphincter are described in the previous chapter on the anal canal. While the anatomy of the individual components of the sphincter complex is well known, the knowledge of the functional anatomy of the sphincter complex is quite different, due to the failing of knowledge of the interactions between them in the defecation mechanisms. Looking at the structure of the anal canal, the anatomical entity that could connect the two sphincters in their muscular activity could be the joint longitudinal muscle. Immunohistochemical staining showed that JLM consists of outer striated muscle fibers and smaller numbers of inner smooth muscle fibers, respectively coming from the levator ani muscle and from the longitudinal muscular layer of the rectum. As above stated, the oblique fibers suggest that the JLM may represent the intermediate longitudinal course of small bridging muscle bundles going reciprocally from the striated EAS to the smooth IAS and vice versa[56]. The spatial result is the helical course of striated and smooth muscle fibers between the EAS and IAS, which contribute not only to narrowing but also to some shortening of the anal canal during sphincter contraction. Thus, rather than being a boundary, the JLM gives anatomical evidence of a functional connection between two muscle systems with different structures and topography. But even if the structure that connects the mechanical activity of the two anal sphincters, i.e., the joint longitudinal muscle, has been codified, extraordinarily little is known about the coordination of the sphincter muscle complex. The question, which remains with partial answers, is: what regulates the coordinated activity of this muscle complex as a whole? The coordinated activity of the anal canal muscle complex can be partially understood from the analysis of defecatory mechanisms. The sampling of the rectal material, mediated by the sampling reflex, involves the relaxation of the internal anal sphincter and the simultaneous contraction of the external anal sphincter. Coordination can only be nervous with an integrated activity between the sympathetic and parasympathetic nervous systems, the pudendal nerve and pacemaker cells. In defecation, evacuative sensations offer a range of sensations (e.g., fullness, urgency, and pain) resulting from cortical conscious perception: the sensations induce a motor response at a later stage. In humans, the anterior insula is involved in conscious perception, while a set of brain circuits referred to as the emotional motor system mediates responses[108]. It is obvious to hypothesize that the cortical activity regulates the contraction and relaxation of the external anal sphincter. Aim of a study was to determine the cortical areas associated with voluntary EAS contraction. Seventeen asymptomatic adults were studied using functional magnetic resonance imaging (fMRI) to detect brain activity. EAS contraction was associated with multifocal fMRI activity in sensory/motor, anterior cingulate, prefrontal, parietal, occipital, and insular regions[109]. Even if multiple cortical regions were identified for EAS control, how their activity can be correlated with the regulation of activity of that of the smooth internal anal sphincter and of the smooth fibers of the longitudinal joint muscle, this is not currently identified in its modality. It therefore remains a mystery how, for example, there is the combined inhibition of the whole anal muscle complex of smooth and striated muscle to the passage of the fecal bolus during defecation.

2. Musculo-elastic anal properties and anal closure.

The repeated relaxations and contractions of the anal canal occur thanks to the mechanical properties of the anal tissues that adapt with their elasticity and compliance to the mechanical stresses induced by muscle contractions; without these, overall, it could give rise to hardening of the musculo-connective structures of the anal canal. These properties are essential to prevent muscle-connective stiffness of the anal wall that could impede volumetric changes of the anal canal. Therefore, the anorectal tissues and muscles mechanical properties (elasticity, compliance and stiffness) are considered as the main actors of anal function.

Elastography-based imaging techniques have received substantial attention in recent years for non-invasive assessment of tissue mechanical properties[110]. These techniques take advantage of changed soft tissue elasticity in various pathologies to yield qualitative and quantitative information that can be used for diagnostic purposes. Measurements are acquired in specialized imaging modes that can also detect tissue stiffness in response to an applied mechanical force (compression or shear wave: SWE). SWE assessment of the levator ani and the external anal sphincter muscles, performed during pregnancy, provided original in vivo human data about the biomechanical changes of pregnant women's pelvic floor[111]. Shear wave elastography looks like a reliable tool also for assessing the elastic properties of the external anal sphincter in term pregnant women[112]. External anal sphincter, considering its location and the strain applied by the fetal head during its expulsion, is exposed to a significant strain too. It is likely that the ability of EAS to lengthen, and thus the woman's biomechanical intrinsic characteristics, could be associated with the risk of obstetric anal sphincter injury in childbirth. In this study, the probe was placed immediately above the anus in a transversal plane using a transperineal approach. The results showed that SWE may lead to an individualized risk assessment of perineal trauma at childbirth. SWE may be applied also in anal diseases. When applied in patients suffering from anal fissure, the results showed that tissues mechanical properties, measured by shear wave elastography, showed increased tissue stiffness[113]. To evaluate the internal anal sphincter elasticity, the real-time tissue elastography (RTE), available on conventional ultrasonography, has been developed[114]. It was used for the quantitation of IAS hardness for evaluating the influence of chemoradiotherapy (CRT) on anorectal function[115]. Representativity of elasticity measurements was demonstrated. It revealed a trend: IAS elasticity had a moderate inverse correlation with maximal anal resting pressure regardless of whether measurements were made before or after CRT. This result has led to the final consideration which, unfortunately, elasticity, that represents the physical hardness of the anal sphincter, it might not be correlated with the anorectal function, including the physiological contractile activity of IAS.

In conclusion, we are still in a preliminary phase of studying the mechanical properties of the anal canal wall. It is not possible to give decisive elements on the evaluation of the parietal elastic components both during sphincteric relaxation and anal closure. Future studies could focus on defecatory disorders, i.e., how much these properties can influence the framework of fecal incontinence or obstructed defecation.

3. Role of gut microbioma.

Large epidemiologic studies report that bowel symptoms related to gut motility and sensation, such as diarrhea and fecal urgency, are significant contributing factors to fecal incontinence. It is well known that loose or watery stools are associated with fecal incontinence[116] (values 6 – 7 of Bristol Stool Form Scale[117]:) and that the urge to defecate is a dramatic prelude to urge fecal incontinence phenomena. Stool consistency was studied in healthy Japanese subjects if it was related to gut microbiota differences[118]. Result showed that *Fusobacterium* in male subjects was significantly higher in the loose consistency group while *Serratia* was significantly higher in female subjects with loose stools. What is the meaning of these findings is not currently interpretable, nor has a clear pathophysiological mechanism been identified. The indication of the need to investigate the microbiota/fecal consistency relationship remains.

Stool metabolites, produced by the interaction of gut microbiota with the host, can modulate neuro-hormonal mechanisms implicated in gut motility and gut sensation. Several studies show that stool metabolites enhance contractile responses of intestinal smooth muscle and neuronal excitability of the enteric nervous system resulting in symptoms such as diarrhea, fecal urgency, and sense of incomplete emptying that are common in women with FI[119] but direct evidence on the role of stool metabolites and microbiota in women with FI is lacking. The brain can influence commensal organisms (enteric microbiota) indirectly, via changes in gastrointestinal motility and secretion, and intestinal permeability, or directly, via signaling molecules released into the gut lumen from cells in the lamina propria (enterochromaffin cells, neurons, immune cells). Communication from enteric microbiota to the host can occur via multiple mechanisms, including epithelial-cell, receptor-mediated signaling and, when intestinal permeability is increased, through direct stimulation of host cells in the lamina propria. Disruption of the bidirectional interactions between the enteric microbiota and the nervous system could, therefore, be involved in the pathophysiology of acute and chronic gastrointestinal disease states, including functional and inflammatory bowel disorders.

Fecal microbiota transplantation

Fecal microbiota transplantation (FMT) is the transfer of stool from a healthy donor into the colon of a patient whose disease is a result of an altered microbiome, with the goal of restoring the normal microbiota and thus curing the disease[120].

FMT is only strongly recommended for multiple recurrent C*lostridium difficile* infection[121]. There are emerging data on the usefulness of FMT in the treatment of inflammatory bowel disease: FMT might be effective for ulcerative colitis but not yet in patients with Chron's disease[122]. Also, there is not a specific scientific paper on FMT and obstructed defecation. A recent follow-up study evaluated the efficacy of washed microbiota transplantation (WMT) for therapeutic targets of refractory functional constipation and demonstrated that 55.6% (5/9) of patients with a sense of anorectal obstruction achieved symptom improvement at week 4 after the first WMT course[123]. Diarrhea and related fecal incontinence are characterized by gut microbiota dysbiosis. Reprograming gut microbiota communities by fecal bacteria transplantation could be introduced to treat or prevent diarrhea[124] and therefore fecal incontinence could improve. Results of FMT are promising (see Clostridium difficile infection and ulcerative colitis) but donor selection, ethics, operation method, and the follow-up should be standardized to guarantee the safe and effectiveness of FMT in clinical practice.

4. Functional anatomy after surgical treatment.

A calibrated therapeutic strategy is the aim of the therapeutic treatment of fecal incontinence. Surgical therapy, based on this concept, certainly enters as a therapeutic option only after the failure of the conservative medical-rehabilitative therapy. Different surgical techniques can be adopted and each of them will modify the pre-existing anatomy, but this does not automatically mean an improvement in function. Anorectal surgery for fecal incontinence is aimed at the anal sphincter/pelvic floor complex or the rectum. The first group includes sphincteroplasty, postanal repair, muscle transposition and artificial anal sphincter: the second group includes the several types of rectopexy.

Sphincteroplasty. Sphincteroplasty may be considered as a surgical procedure useful to restore the anatomical integrity of the injured sphincter. Overlapping sphincteroplasty is preferred to end-to-end direct sphincter repair and it is the treatment of choice for single EAS defects with or without concomitant IAS defect[120]. The anterior route represents the approach of choice for sphincteroplasty in obstetric tears. Several studies have shown the usefulness of preserving the scar to anchor the suture made for overlapping sphincteric flaps but, despite this technical expedient, dehiscence of the sutured stumps can occur, even after some time. The overlapping of the stumps can modify the normal spatial arrangement of the two anal sphincters; in fact, the window, which is physiological in the female anterior proximal external sphincter, can be partially or completely obliterated. Of course, the sphincteric thickness will also be affected by influencing the age-related thinning of the external sphincter. At last, given the anterior approach, the preparatory dissection of the two stumps will interrupt the relationship of sphincter continuity with perineal body and with the transverse muscles of the perineum. The new sphincter assembly might explain why, although sphincter continuity is restored, continence does not always improve: complete closure of the anal canal might not be achieved due to the asymmetry of the muscular walls, disassembled and not in axis just by the overlapping of the muscular stumps.

Postanal repair. Indications for postanal repair include, at present, patients with weak sphincter without demonstrable defects. The operation reported by Browning and others[121] and described by Parks[122] was the surgical treatment adopted in patients with fecal incontinence related to pelvic floor neuropathy and deficient anorectal angle. After a postanal transverse incision, by careful posterior retraction of the external sphincter, the inner fibers of the puborectalis muscle can be seen lying just above the sphincter. The intersphincteric plane is dissected and Waldeyer's fascia is incised to log into puboccygeus and ileococcygeus muscles. A series of sutures are then placed into the puborectalis and pubococcygeus using a mass closure technique. In this way the posterior repair is performed. If an anterior repair is to be added, three or more anterior sutures can be placed on the ischioccygeus muscle[123]. The success rate is low (35 percent) but the absence of any mortality and the low morbidity suggest that postanal repair may be a valid therapeutic approach. However, it should be offered only to selected patients with persistent, severe fecal incontinence despite an anatomically intact external anal sphincter who are not candidates for or refuse all other operative modalities[124]. There is some indication that failure may be due to an inadequate flap valve mechanism despite the repair. The failure to establish a sufficient anorectal angle and flap-valve by postanal repair could be due to continuing perineal descent, as suggested by post-operative defecography[125]. Postanal repair, by manipulating the levator ani muscles with a median suture, reduces the urogenital hiatus space. This new spatial arrangement, especially if an anterior repair is added, has positive effects on a descending perineum and

on pelvic organ prolapse: for the latter, as direct consequence, the volumetric reduction of the available space in the urogenital hiatus limits the pelvic organ descent. Finally, it is possible to hypothesize that the suture of the puboccygeus can induce dyspareunia with a constrictive mechanism on the vaginal walls.

Muscle transposition. Muscle transposition is a surgical technique used to physically replace the anal sphincter with *in vivo* muscle bulk. Gluteoplasty (GLP) and graciloplasty (GRP) are options that may be considered to replace the anal sphincter when extensive sphincter damage, muscle loss and pudendal neuropathy are involved[126]. The two muscles widely described in the literature for transposition are the gluteus maximus and gracilis muscles. These are useful because of their proximity to the anus, sizeable muscle bulk, and nerve locations which are amenable to preservation upon transposition.

Gluteoplasty. The gluteus maximus was thought to be an excellent choice for transposition given that involuntary gluteal contraction occurs with the strong urge to avoid involuntary defecation. Bilateral incisions over the gluteus are made and two tongues (one from each side) of the lower 10% of the muscle are raised with care taken to preserve the neurovascular bundles[127]. The mobilized muscle is then tunneled and delivered through separate bilateral curvilinear incisions around the anus. The contralateral mobilized segments are sutured together to create a ring of muscle. Gluteoplasty has a good success in restoring long-term fecal continence in some patients (45%) but has a high incidence of donor-site and perirectal complications ($\simeq$ 60%) even if without gait and/or defecatory complications[128, 129].

Graciloplasty. In a graciloplasty procedure, two or three incisions are made along the longitudinal access of the gracilis muscle on the chosen side to harvest the entire length of the gracilis. The neurovascular bundle is preserved through its identification during medial dissection. The muscle is released distally and tunneled medially. A perineal incision is made and the gracilis is wrapped circumferentially around the anus. In the dynamic graciloplasty technique, an electrode is placed in the gracilis muscle and an implantable device like that used for sacral nerve stimulation is implanted in the abdominal wall. Successful functional long-term outcomes for graciloplasty, dynamic or unstimulated, is consistently reported to be about 69%-71%[130]. Complications of the procedure are common, and include surgical site infections, pain, rectal injury, and erosion of the device in the case of dynamic graciloplasty. In addition, constipation due to obstructed defecation is commonly reported in as many as 50% of patients and colonic enemas may be needed.[131]

In both muscle transpositions, striated muscles are wrapped around the anal canal but GLP may induce a double curvation of the anal canal in contrast to the GRP, which enhanced the natural anorectal angle[132]. When the gluteus muscle contracts, the active muscular narrowing and the dorsolateral deviation of the anal canal act together with the remaining natural anorectal angle induced by puborectalis muscle. Therefore, this created double curvation of the anal canal may be added to the normal continence mechanism. The gluteus maximus is the most representative muscle of the gluteal region as well as the main hip extensor, allowing the upright position, and external rotator. The surgical technique involves the bilateral section of the lower third of gluteus maximus for transposition: a final close approximation between the muscles and the anal canal is reached. The danger is that part of the transposed muscle may undergo atrophy: experimental studies have in fact shown that, whether a neurostimulator is used, around 30-35% of the muscle undergoes atrophy[132]. Additional mass muscle reduction may be brought by the same electrostimulation: muscle electrostimulation experiments have demonstrated that a shift toward oxidative and type I fibers takes place. These histochemical changes are accompanied by muscle mass reduction. Therefore, the

anatomical-functional effects of gluteoplasty can be of two types: the first on body statics, negatively influencing the maintenance of the upright position, the second on fecal continence, showing a progressive long-term impairment. The gracilis muscle arises from the anterior side of the ischiopubic branch and terminates at the level of the medial point of the knee/tibia. Due to its biarticular position, it exerts a move of flexion/internal rotation of the leg and contributes to the stabilization of the pelvis during walking and running. It is not possible to think that its distal disconnection does not cause some type of alteration in walking. Also, for graciloplasty there have been studies on the morphology of the gracilis muscle after transposition. A study conducted with nuclear magnetic resonance found that gracilis diameter decreased from 12 (5 days after transposition) to 8 mm, 6 weeks later but that no correlations were detected between reduction of the gracilis muscle diameter and clinical outcome in terms of achieved continence and/or anal canal pressures[133]. In conclusion, dynamic GRP is currently the most studied and employed transposition procedure for fecal incontinence and, despite the complications and imperfect continence, patients with GRP report a significant improvement in quality of life[134].

Artificial anal sphincter. The implant of foreign material in the anorectal region is used when it could be a potential option to restore continence in patients with severe symptoms of fecal incontinence at end stage. Good functional results are reported in >50% of patients who tolerated the artificial anal sphincter (AAS). In a prospective, non-randomized, trial[135] the incontinence scores improved significantly in 67% of the patients with a functioning neo-anal sphincter at 12 months of follow-up. The device was removed in forty-one out of 112 patients and a revision was made in 46%. The Acticon Neosphincter, the only device certified by the US Food and Drug Administration, consists of three components integrated by kink-resistant tubing: an inflatable cuff placed around the anus, a manual control pump placed under the skin of the scrotum in males or labia in females, and a pressure-regulating balloon implanted extraperitoneally. The purpose of the device is to take over from the muscle to control the opening and closing of the anus. Explantation, surgical revisions, infection, erosion, device malfunction and obstructed defecation are the main system complications. Furthermore, the most critical issue of AAS is to maintain long-term biomechanical compatibility between the artificial sphincter and the surrounding tissue in clinical applications. The thickness of tissue around the implant changed dramatically by a stress shielding effect caused by the large mechanical stimulus. The stress shielding effect makes the force between the implant and its surrounding tissues unbalanced, causing the device to be ineffective and leading to ischemic necrosis and tissue atrophy[136]. it is therefore obvious that the local functional anatomy, conditioned by the alterations of the tissues surrounding the device, is different from patient to patient and therefore influences continence differently from case to case.

5. Functional anatomy after surgical treatment of rectal prolapse.

The rectal prolapse is only part of a massive sliding hernia in which the peritoneal sac is formed by the rectovesical pouch or by the pouch of Douglas. The rectum is abnormally mobile on a long mesentery and lack of fixation of the rectum to the sacrum allows its protrusion to the outside. These peculiar multiple pathological findings must be corrected with surgical therapy and this explains the numerous proposed techniques, none of which is fully satisfactory.

The surgeries for rectal prolapse can fall into two categories: perineal approach or abdominal approach (Table 3).

Table 3. Rectal prolapse. Surgical operations.

Perineal approach	Abdominal approach
Thiersch operation	Orr-Loygue rectopexy
Anal sling	Ripstein repair
Delorme procedure	Wells technique
Altemeier procedure	Frykman-Goldberg procedure
Perineal stapled prolapse resection	Laparoscopic ventral rectopexy

The expert panel of SICCR/AIGO Consensus Statement on fecal incontinence[126] recommend that 1) elderly and frail patients could be best suited for perineal surgery, even if no conclusive evidence is available; 2) the abdominal approach is traditionally believed to be superior in terms of lower recurrence rate[137] but this has been questioned by a randomized controlled trial[138]; 3) resection rectopexy results in a lower recurrence rate and is recommended in cases of constipation; 4) among the rectopexy techniques, ventral rectopexy is preferred nowadays but without evidence; 5) the laparoscopic approach should be preferred only because it is less invasive and more cosmetic.

A) Perineal approach: functional anatomy

The perineal approach causes changes on all the anatomical planes involved in the single procedure, including the rectum.

Anal cerclage, such as the ***Thiersch operation*** and ***anal sling***, are intended to narrow the anal canal with external compression. Conventionally, the Thiersch operation typically involved blind positioning of the sling, and sling tension is subjectively based on a rule-of-thumb estimate. The sling was placed outside of the external anal sphincter muscle and at the junction of the external anal sphincter muscle and puborectalis muscle. The local anatomical effects depend on the biological interaction between the material used and the anal tissues, and it can be assumed that the continuous mechanical compression exerted by the prosthetic material can induce a tissue fibrotic reaction up to atrophy. This could explain the progressive decline over time of the surgical procedure on continence.

The ***Delorme procedure*** may be used in case of short prolapse[139] and consists, after removal of the rectal mucosa, in the full thickness longitudinal muscular plication of the rectum for a vertical extension depending on the length of the rectal prolapse[140]. This produces a structural alteration of the wall and a decreased volumetric capacity of the rectum, both expressed by the manometric values of impaired rectal compliance with a decreased volume of first rectal sensation and maximum tolerated volume[141]. The pre-operative rectosacral separation seen at defecography seems to be a factor associated with Delorme failure[142]. However, improvement in fecal incontinence is reached in 46-54% of patients.

The ***Altemeier procedure*** is a perineal proctosigmoidectomy born for extensive rectal prolapse, exceeding 5 cm, in elderly and debilitated patients[143,144]. All layers of rectal wall are included in a circumferential incision that begins about 2-4 cm proximally to dentate line and then proceeds upwards. Once the extraperitoneal rectum is fully mobilized, the Douglas pouch

is opened allowing access into peritoneal cavity. At that point, viscera exteriorization continues, also including the sigma, and isolation of the colon must be interrupted when it cannot be drawn out of the pelvis without tension. After the complete resection of elongated colon, the exteriorized anastomosis is performed, manually or by means of a circular stapler, between the distal descending colon and the anorectum. The suture must include mucosa and muscular walls. Levatorplasty may be added: a posterior muscle plication and sometimes one or two anterior stitches narrow the hiatus of levator ani. Postoperative functional anatomy is like that of ultralow anterior resection with coloanal anastomosis but the peculiarity of the Altemeier procedure is that it addresses a particular and complex pathology involving the rectum, the pelvic floor with a descending perineum, chronic anal strain and a possible pudendal neuropathy. All these factors affect the functional outcomes of proctosigmoidectomy: a survey from six major colorectal centers of the Italian Society of Colorectal Surgery showed that fecal incontinence improved in 30% of patients and continence restoration remained unpredictable[145].

Perineal stapled prolapse resection (PSPR) was proposed in 2008 by Scherer et al. as easy, fast and safe procedure to use in the surgical treatment of the external rectal prolapse, suitable for elderly, high-risk patients for whom an abdominal approach under general anesthesia is not advisable[139, 146]. The prolapse is axially cut open with a linear stapler at three and nine o'clock until 2 cm from the dentate line; then, the two flaps of the prolapse are resected in counterclockwise direction with a curved circular stapler. The median length of resected bowel is about 7.5 cm (range 5-11 cm)[147]. The functional anatomy of this surgery is comparable to that of a partial proctectomy. The residual rectal stump is a few centimeters, practically halved compared to the normal volume of the rectum. The decreased volumetric capacity of the rectum explains the urge to defecate which can sometimes lead to fecal incontinence, especially in patients who pre-operatively presented anal sphincter lesions and/or pudendal neuropathy. A multicenter prospective study, performed in 27 patients treated by PSPR, found that median Wexner incontinence score improved from 10 pre-surgery to 5 after surgery ($P < 0.001$): this finding underlined that some symptom or sign, related to fecal incontinence, was still present postoperatively[148].

B) Abdominal approach: functional anatomy

Rectopexy, with or without mesh, open or laparoscopic either robotic, has regained general favor, mainly because of the hypothetical lower rate of recurrence associated with it. Indeed, some studies underlined that the reoperation rate for recurrence was similar after abdominal and perineal approaches[138, 149, 150]. Studies comparing laparoscopic with open techniques for treatment of rectal prolapse have shown similar morbidity, recurrence rates and functional results[151]. In 2014

a systematic review and meta-analysis demonstrated a similar recurrence rate and reoperation between robotic versus laparoscopic rectopexy[152]. The common element in the functional anatomy of the diverse types of rectopexy is the suspension of the rectum, prosthetic or else, to the sacrum. The suspension necessarily involves the fixation of the bowel to the bone surface, immobilizing it in its new position. The rectum loses its motility, remains anchored to a new anatomical relationship and sometimes the anchorage of the rectum becomes the folding pivot of the overlying sigmoid. Mechanical obstruction thus occurs, and this occurrence should be investigated when chronic postoperative constipation should become severe. It must also be emphasized that the correct position of the sutures on the sacral promontory, the preferential place for suspension, must be lateral to the hypogastric nerves and medial to the ureter[153].

Orr-Loygue rectopexy. To the procedure described by Orr in 1947[154], Loygue, from 1953 until 1982, added mobilization of the rectum prior to its suspension and eliminated the pouch of Douglas, using nylon strips for its suspension to sacral promontory[155]. Recurrent rectal prolapse was observed in 4.3 per cent of the patients. In 136 patients anal incontinence was associated with rectal prolapse. Normal continence was restored in 84.1 per cent of 107 patients with rectopexy alone and in 64.2 per cent of 14 patients who underwent rectopexy and anal sphincter repair. A retrospective study evaluated a laparoscopic modified Orr-Loygue procedure for recurrent complete rectal prolapse in pediatric age[156]. A prerectal non-absorbable mesh was used to suspend the rectum to the presacral fascia and promontory avoiding any tension on the rectal wall. This modified technique was an efficient surgical treatment: no postoperative constipation or recurrence was reported during the median follow-up period of 6 years.

Ripstein repair. Ripstein first described an anterior sling rectopexy from a graft of fascia lata in 1952[157]. The fundamental aspect was to restore the normal posterior curve of the rectum in the hollow of the sacrum, with its fixation to the sacral promontory. Once rectum was fully mobilized, a rectangular-shaped piece of graft is placed over the lower rectum and fixed to the sacrum. The graft envelops the rectum in a postero-anterior direction and the stitches between the two wings of the graft and the anterior wall of the rectum are positioned respectively on the right and left sides. The ultimate result is an anteriorly graft-wrapped rectum. The Ripstein procedure became a common procedure for treatment of rectal prolapse because of its low recurrence rate (< 10%), and the avoidance of bowel anastomosis. However, in the following years a synthetic mesh replaced the originally graft described and now the procedure has been supplanted by sigmoid resection and rectopexy or laparoscopic sutured rectopexy. Nowadays, it is primarily used for patients in whom an abdominal procedure is indicated, but resection is either not indicated (those patients with no constipation), or where an anastomosis may be at elevated risk. The Ripstein procedure may also be undertaken in patients with recurrent rectal prolapse after other repairs. Patients who have failed previous perineal approaches, especially the Altemeier approach, may also be good candidates for a Ripstein type repair. The biggest post-operative problem is chronic constipation. Colonic transit time is often prolonged and transit markers are distributed in the left colon and sigmoid area. Therefore, it is suggested that the angled stiffness of the rectum with respect to the sigma, especially if a redundant sigmoid colon kinking exists, may be an obstacle to the passage of stools[158]. On the contrary, continence improves after Ripstein rectopexy and this improvement is accompanied by increased anal resting pressure, indicating a recovery of internal anal sphincter function[159].

Wells technique. The rectopexy proposed by Wells makes use of the polyvinyl alcohol sponge (Ivalon) which suspends the rectum to the concavity of the sacrum[160]. A sheet of Ivalon is attached to the anterior surface of the sacrum between the promontory and the third or fourth segment, by three midline sutures of thread. The rectum is then drawn firmly upwards and the Ivalon folded around it to enclose all save the anterior fourth or fifth of its circumference. The substance becomes incorporated into the tissues in which it excites a vigorous fibrotic response, with the production of a firm, almost cartilaginous, mass. In the following years, Wells technique became particularly popular as a posterior mesh rectopexy and sacral mesh fixation with low mortality and recurrence rates, which were less than 5% overall[161]. Two anatomical defects are not corrected by this technique: the ‘gap’ in the levator ani muscle, which is not narrowed, and the patulous atonic anus, which is not strengthened[162]. Moreover, alternative mesh implants (Marlex, Gore-Tex, Teflon and absorbable mesh) were used to decrease the pelvic sepsis rate and laparoscopic rectopexy was performed with

excellent results[163]. In conclusion, the Wells procedure is safe and gives improvement of fecal incontinence (3-40%), but there is a reported incidence of some evacuation difficulty with increase in prevalence of constipation (≃ 25%)[164]. Distal lateral ligament division, local autonomic neuropathy and changes in rectal wall thickness were considered as possible causal factors.

Frykman-Goldberg procedure. The Frykman-Goldberg procedure is a rectopexy combined with sigmoid resection[165]. When the rectum is fully mobilized it is pulled up into the abdomen. The lateral rectal ligaments are attached to the periosteum of sacral promontory to hold the organ firmly and the cul-de-sac is obliterated by suturing the endopelvic fascia anterior to the rectum. The sigmoid colon is then prepared for resection and whole redundant viscus needs to be removed. An end-to-end anastomosis is then performed. In recent years, the introduction of laparoscopic and robotic surgery has offered a minimally invasive approach to this procedure, with comparable results to the open technique for safety and recurrence rate[166,167]. Functional scores for chronic constipation and fecal incontinence improve significantly ($P < 0.001$) and the improvements remain constant and significant over time[168]. Resection rectopexy results in a lower recurrence rate and is recommended in cases of constipation[126]. The Frykman-Goldberg procedure can be considered the first choice in young patients with a long-life expectancy and may be used also in old patients with both rectal prolapse and sigmoid diverticula.

Laparoscopic ventral rectopexy. D'Hoore et al. in 2004 published scientific data of a new laparoscopic nerve-sparing technique for the treatment of rectal prolapse that avoided mobilization of the rectum[169]. The dissection is restricted to the anterior aspect of the rectum and therefore limits the risk for autonomic nerve damage. Rectopexy is made by means of a mesh which is sutured to the ventral distal rectum and further fixed to the lateral seromuscular borders of the rectum proximal and distal to the incised pouch of Douglas. The mesh is then fixed upon the sacral promontory. The posterior vaginal fornix is elevated and sutured to the anterior aspect of the mesh; this allows closure of the rectovaginal septum and correction of a vaginal vault prolapse if present. Therefore, particular care is taken not to damage the right hypogastric nerve. The choice of a ventral position for the mesh is based on defecographic data that identified rectorectal intussusception as the leading mechanism for development of external rectal prolapse[169]. A recurrent rectal prolapse was seen in 4.7% of patients. Continence improved markedly: 51.6% of incontinent patients became fully continent and other five patients experienced minor grades of incontinence. Symptoms of difficult evacuation were resolved in 63.1% of patients and no severe constipation of new onset was observed. These functional results suggest that laparoscopic ventral rectopexy appears to be as effective as classical rectopexy in terms of the recurrence rate and preservation or restoration of continence.

3. OBSTRUCTED DEFECATION

Obstructed defecation (OD) is a subset of chronic constipation. Identified by Bartolo and Roe[170], is broadly defined as the inability to evacuate contents from the rectum[171], with symptoms of dyschezia and a subjective sensation of anal blockage during defecation. The functional pelviperineal anatomy in patients suffering from organic obstructed defecation depends on the existing anatomical alterations. In fact, the specific anatomopathological lesion shapes the appearance of an altered defecation, whose signs and symptoms, however, are non-specific because they are referable to a generic defecation disorder. The involved organic diseases of organic obstructed defecation are rectocele, rectal intussusception, descending perineum syndrome, solitary rectal ulcer syndrome, enterocele, sigmoidocele and megarectum. In selected cases, all of them could undergo surgery and therefore the functional anatomy would depend on the surgical technique. The functional anatomy of rectocele, enterocele and sigmoidocele were discussed in the chapter on pelvic organ prolapse. In the following paragraphs only the functional anatomy of the descending perineum syndrome, rectal intussusception, solitary rectal ulcer syndrome and megarectum will be described.

1. ***Descending perineum syndrome***. As originally described by Parks et al.[172], descending perineum syndrome (DPS) is characterized by the ballooning of the perineum several centimeters below the bony outlet of the pelvis during straining. Obstructed defecation, often present for many years, evolves into impairment of fecal continence, considered as a late sign of the syndrome[173]. It is rational to think that perineal descent may simultaneously involve the anterior, middle, and posterior pelviperineal areas in women. Urogynecological structures and proctologic segments are thus all implicated and rectal diseases may be associated with urogynecological diseases[15]. Proctological diseases such as displacement rectocele, anorectal intussusception and rectal prolapse are often combined with POP. Excessive defecatory straining induces muscular stretching which impairs reflex muscular perineal contractions, and repositioning occurs when muscles become lengthened and loose. In this way, the perineum descends more as testified by levator ani muscle and ligament dysfunction. The decline in normal levator ani tone, induced by overstretching, results in an open urogenital hiatus, weakening of the horizontal orientation of the levator plate, and a bowl-like configuration. This new anatomical position involves the entire pelvic floor with effect on all viscera supported by the pelviperineal muscle complex. The combination of the various visceral alterations will vary also according to the stage of the pelviperineal descent: if in the initial phase there is still, even if altered, the defecatory excursion of the pelviperineal plane, in the advanced phase, this excursion will progressively reduce until its complete disappearance because by now the plane is immovably fixed many centimeters below. The descending perineum is considered fixed if defecography at rest shows a pelvic floor descent > 3 cm which increases several centimeters during straining and evacuative maneuvers and returns slowly or not returning to the starting line. The functional anatomy in various stages of development influences the clinical aspect of the disease. The dyssynergic component plays a role in the initial stages of the disease, inducing obstructed defecation. The organic descent of the hypotonic pelvic floor, combined with disease duration, advancing age, pudendal neuropathy, and sometimes uterine surgery, such as abdominal hysterectomy, explains the appearance of late fecal incontinence[174,175]. The complex anatomical disorder explains therapeutic difficulties: surgery, limited to fixed DPS and rehabilitation failures, should be aimed at correcting pelvic floor impairment. The correction is made directly by

means of levatorplasty techniques or indirectly by surgical procedures involving mesh or resection that are aimed at rectal diseases. Results are partial but they can be evaluated with the citation of the glass if half full or half empty. An isolated experience using retroanal levator plate myorrhaphy, in selected patients, reduced perineal descent about 1.08 cm and improved scores of obstructed defecation and fecal incontinence in 87.5 % of subjects but rectocele repair failed in 25% of cases[176].

2. ***Rectal intussusception***. Intussusception of the bowel is defined as the telescoping of a proximal segment of the gastrointestinal tract within the lumen of the adjacent segment. Rectal intussusception is defined also as internal rectal prolapse. Depending on the level reached by the intussusception, it will be identified either as rectorectal intussusception if the intussusception is limited to the rectal ampulla, or as rectoanal intussusception if instead the intussusception reaches the anal canal. In few patients, rectal prolapse may happen as the end point of intussusception evolution. The presence of full-thickness intussusception at defecography may be suspected when rectal fold is greater than 3 mm[177]. The circumferential full-thickness intussusception begins as rectorectal intussusception at ≃ 8 cm from the anal verge during straining at defecation. Precise proctographic measures indicates that the "take-off" point of the intussusception appears correspondingly about 5.5 cm from the anorectal junction, anteriorly[178]. This site corresponds to the deepest peritoneal reflection of the Douglas pouch. The strength vectors, created by intra-abdominal pressure, are channeled in this place, so that, because of forced straining with Valsalva maneuvers, pressure increases are transmitted to the anterior part of the rectum, starting its rectorectal intussusception. The distal transmission of the intussusception will lead to the progressive inosculation of the viscus up to the anal canal with rectoanal intussusception. It should be specified that patients with rectal intussusception have normal rectal wall biomechanics in contrast to those with rectal prolapse, suggesting that they are physiologically distinct[179] even if, within 2 years, 19.2% of patients with rectorectal intussusception on the initial proctogram demonstrates progression to rectoanal intussusception and external prolapse[180]. The different evolutive stage between rectorectal and rectoanal intussusception may explain presence of obstructed defecation and/or appearance of fecal incontinence. Moreover, differentiated therapies may be suggested: rectorectal intussusception may be treated by rehabilitative treatment[181], rectoanal intussusception, after failed conservative therapy, may be cured by surgery[182].

3. ***Solitary rectal ulcer syndrome***. Solitary rectal ulcer syndrome (SRUS) can present with a multifaceted pathological anatomy, either as a mucosal ulceration with hyperemic borders and surrounding induration, or as a polypoid exophytic lesion. The common particularity is that both lesions are found about 6-8 cm from the anal margin, usually on the anterior wall of the rectum[183]. SRUS can occur in patients suffering from obstructed defecation and in those with overt rectal prolapse. SRUS patients without rectal prolapse have high anal canal pressures which increase with straining and a paradoxical puborectalis contraction. In these patients there is an enlargement of muscularis propria that could be the consequence of a rectal wall chronically overloaded by motor activity working against a non-relaxing puborectalis muscle[184]. The mucosal changes in patients with rectal prolapse might be related, instead, to the mechanical trauma of mucosa during the eversion through the anus. Manometric reports show low anal pressures and absence of paradoxical puborectalis contraction.

4. ***Megarectum***. Idiopathic megarectum (IM) is characterized by abnormal, pronounced rectal dilatation in the absence of identifiable organic pathology[185]. Qualitative and

quantitative histological examination[186] found that patients with idiopathic megarectum, compared with controls, had significant thickening of their muscularis mucosae, circular muscle and longitudinal muscle ($P < 0.005$). Fibrosis of the longitudinal muscle was seen in 58%, of circular muscle in 38%, and of muscularis mucosae in 29% of patients. The density of neural tissue in the longitudinal muscle seemed to be reduced in patients with idiopathic megarectum. Functional abnormalities of the latter remain a possible cause of smooth muscle hypertrophy. Almost all patients with idiopathic megarectum develop symptoms in childhood or adolescence and present with fecal impaction and soiling, both two due to altered rectal sensitivity as consequence of the abnormal volumetric rectal enlargement[187]. The consequence is the frequent manual disimpaction. High values of Conscious Rectal Sensitivity Threshold, Constant Sensation and Maximal Tolerated Volume are all together the main impaired manometric values while RAIR is normally present, even if induced by high threshold values, unlike Hirschsprung megacolon where RAIR is absent.

4. PELVIC PAIN

Chronic pelvic pain is defined as pain perceived to originate from the pelvis, typically lasting more than 6 months, and is often associated with negative cognitive, behavioral, sexual, and emotional consequences and symptoms suggestive of lower urinary tract, sexual, bowel, myofascial, or gynecologic dysfunction[188]. Chronic pelvic pain is associated with many visceral, neurological, musculoskeletal, and psychological symptoms and multiple pelvic pain syndromes often coexist in the same patient. Visceral structures (uterus, bowel, and bladder) and somatic structures (skin, muscles, fascia, and bones) in the pelvis share neural pathways resulting in similar symptoms and making it difficult to differentiate somatic from visceral causes of pain.

Pudendal neuralgia, included among chronic pelvic pain diseases, is the neuropathic pain component of the syndrome caused by pudendal nerve entrapment and neuropathy. It is commonly a bilateral process with a characteristic perineal pain, aggravated by sitting and relieved by standing or lying, which is present in over 50% of affected patients.

1. Pudendal nerve

The pudendal nerve emerges from the S2, S3, and S4 roots' ventral rami of the sacral plexus. It carries sensory, motor, and autonomic fibers; however, an injury to the pudendal nerve causes more sensory effects than motor. It initially courses between two muscles, the piriformis and coccygeus muscles, then departs the pelvic cavity through the greater sciatic foramen ventral to the sacrotuberous ligament. It passes medial to and under the sacrospinous ligament at the level of the ischial spine to re-enter the pelvic cavity through the greater sciatic foramen. The pudendal nerve then courses in the pudendal canal, also called the Alcock canal. The terminal branches of pudendal nerve, branched out after its passage through the Alcock canal, are the inferior rectal branch, perineal branch and dorsal sensory nerve of the penis or clitoris.

2. Pudendal nerve entrapment syndromes

The pudendal nerve entrapment syndromes are subdivided into four types based on the location of the compression.

Type I - Entrapment below the piriformis muscle as the pudendal nerve exits the greater sciatic notch.

Type II - Entrapment between sacrospinous and sacrotuberous ligaments is the most common site of pudendal nerve entrapment.

Type III - Entrapment in the Alcock canal.

Type IV - Entrapment of terminal branches.

Labat *et al* published the "Nantes" criteria to diagnose pudendal nerve entrapment[189]. Pelvis MRI is recommended as it can help rule out other causes of chronic pain. The advancement of MRI techniques in evaluating peripheral nerves provides a detailed description of the anatomy, fascicular details, the blood supply of the nerve, and detailed 3-D anatomy[190]. It also helps in localizing the exact site of entrapment. MRI testing is strongly recommended prior to any surgery for pudendal entrapment. High-frequency ultrasound is helpful in the detection of the site of compression. Compressed nerves and associated veins appear flat, whereas inflamed nerves appear edematous[191]. Functional tests can be used. The electromyographic bulbocavernosus reflex is a well-known somatic reflex that is useful for gaining information about the state of the sacral spinal cord segments. When present, it is indicative of intact spinal reflex arcs (S2–S4 spinal segments) with afferent and efferent nerves through the pudendal nerve. Its absence diagnoses a lower motor neuron lesion while a conduction delay suggests pudendal neuropathy. Finally pudendal nerve block injections with a local anesthetic have been recommended to help confirm the diagnosis of pudendal nerve entrapment, especially if the injection is done directly into Alcock's canal using image guidance[192]. This is often used as an indicator of which patients are most likely to benefit from decompressive surgery. Some therapeutic options may be explored. The overall response to pudendal nerve blocks in properly selected patients is roughly 80%, but the relief typically lasts only about 30 days in most patients. A repeat injection or an alternative treatment would then need to be utilized. The best long-term cures are from decompressive surgery, where the response rates are 60% to 80%[193]. Patients who fail decompressive surgery, possibly up to 80%, can still obtain relief from pudendal neuromodulation[194].

5. PELVIC INFLAMMATION

Common causes for pelvic abscesses are numerous and include postoperative septic complications, inflammatory or infectious gastrointestinal tract disease (Crohn's disease, appendicitis, diverticulitis, post-radiotherapy enteritis, fistula-in-ano-related perianal abscesses), tube-ovarian abscesses and pelvic inflammatory disease. The formation of an abscess is usually the consequence of a visceral perforation but sometimes it can result from the diffusion by contiguity of an inflammatory phenomenon located in another neighboring organ.

Fistula in ano.

Anal fistula is a small and infected channel that connects the anal canal with the surrounding skin. In agreement with the cryptoglandular theory, the fistula is the outcome of an infection of anal glands which, as it progresses, degenerates into an abscess that will open onto the outer skin. In 1976 Park, Gordon, and Hardcastle[195] proposed a detailed classification of fistulas based primarily on the internal and external sphincter involvement in the fistula (Table 4).

Table 4. Parks' Classification of anorectal fistulas.

Fistula type	Pathological highlights
Inter-sphincteric fistula	Intramural fistula, between internal and external anal sphincter
Trans-sphincteric fistula (high or low)	Penetrating portion of the external sphincter
Supra-sphincteric fistula	Fistulous tract passing above the levator muscles
Extra-sphincteric fistula	Often iatrogenic or related to Crohn's disease

A fistula can also be categorized as simple or complex. The former includes those with an inter-sphincteric or low trans-sphincteric track that involves less than 30% of the sphincter complex. A fistula in the presence of inflammatory bowel disease, malignancy, incontinence, chronic diarrhea or previous irradiation should be considered complex as should recurrent fistulas and those with an anterior tract in a female patient[196]. Preservation of continence is the target of anal fistula surgery and explains the development of various surgical techniques. Surgical techniques include fistulotomy and fistulectomy, with or without primary sphincteroplasty, cutting seton, and so-called sphincter-sparing techniques such as advancement flaps, ligation of the inter-sphincteric fistulous tract (LIFT), laser closure (FILaC) and video assisted anal fistula treatment (VAAFT). Fistula type is a risk factor for anal incontinence due to loss of muscle mass after surgical intervention. Since high trans-sphincteric and supra-sphincteric fistulas tend to persist and recur more often than simpler, more distally located fistulas, they are more likely to be associated with repetitive surgical interventions, which further increases the risk. When there is no preoperative incontinence, 9.4% of patients can develop it postoperatively[197]. However, most patients have low grade incontinence following surgery. The greatest significance is in incontinence to flatus ($p < 0.013$) and only a few patients have significant soiling from solid feces[198]. This type of passive incontinence is usually due to internal anal sphincter lesions but also excessive ablation of anal mucosa can be responsible. In any case, regardless of the anal incontinence model, morphological evaluation of the anal canal with endoanal ultrasound is mandatory: it is the optimal method for studying the anal sphincter complex, showing sphincteric lesions and measuring their size in degrees and length. Anorectal manometry is useful for the study of anorectal function: low anal resting pressure is related to internal anal sphincter malfunctioning, low maximal anal contraction indicates the external anal sphincter dysfunction, and impaired rectal sensitivity and/or rectal compliance suggests rectal involvement[199]. Anorectal manometry is also useful for suggesting which rehabilitation program and techniques should be adopted[200]. Rehabilitation is considered first-line treatment and may be useful for correcting obstructed defecation when combined with anal fistula. It should be

emphasized that rehabilitative treatment is not indicated if the postoperative anal sphincter defect exceeds 95°. If rehabilitation fails, other types of treatment can be hypothesized including sacral neuromodulation, the SphinkeeperTM procedure and sphincteroplasty.

6. *IBD*

Inflammatory bowel diseases (IBD) represent a group of chronic systemic inflammatory conditions with predilection to gastrointestinal tract and include Crohn's disease and ulcerative colitis. If the IBD cannot be further specified, the term unclassified IBD is used. Localization in the rectum can occur in all forms and does not offer particularities of rectal functional anatomy, except for the involvement of the full thickness of the wall in Crohn's disease with possible formations of fistulas. The impairment of rectal functionality results in alterations of the bowel habit, with frequent association of proctorrhagia. In the previous chapters dedicated to the colon and rectum, data relating to the functional anatomy of the rectum and its post-surgical modifications are amply illustrated. Refer to those descriptions for further discussion of the topic of functional anatomy in IBD.

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