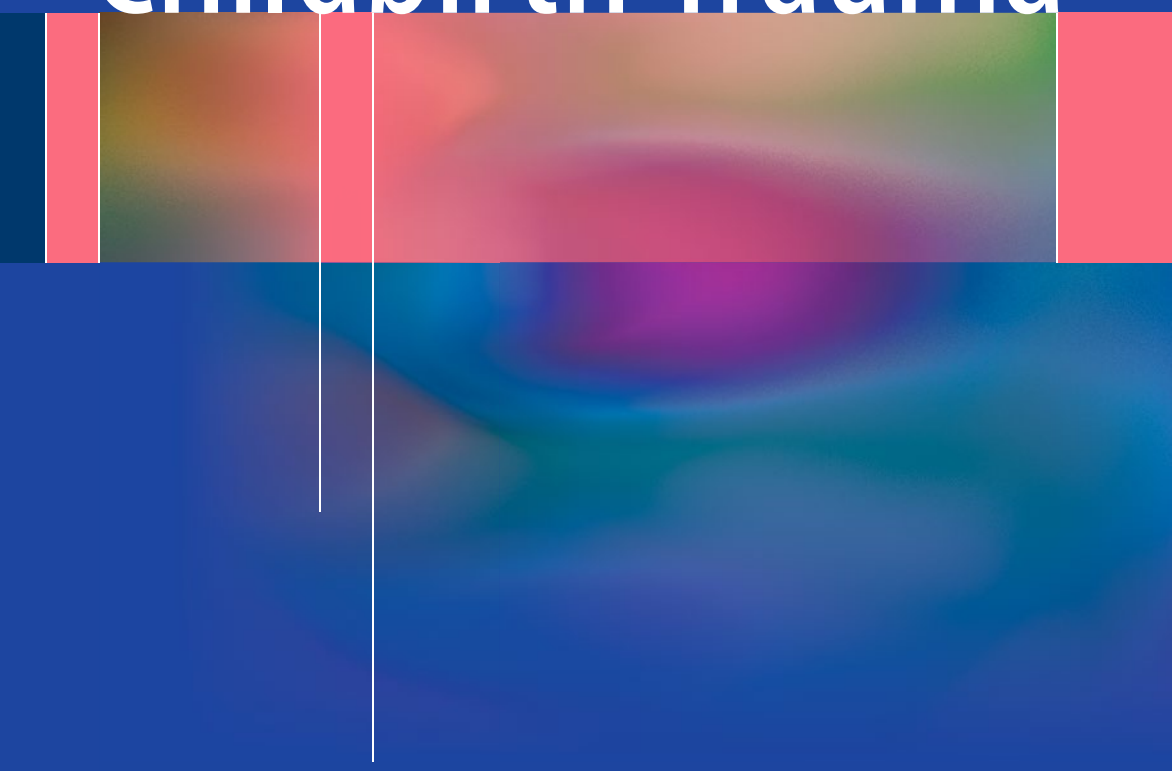
Stergios K. Doumouchtsis
Editor

Childbirth Trauma



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This book is dedicated to my wonderful family, as it would have not been completed without their selfless support, patience and encouragement.

Foreword

Childbirth trauma is an everyday event and is considered as a minor issue and brushed aside. But in reality, the woman suffers in pain, mental anguish and concern for her future sexual and reproductive life. The extent of the injury may be much more serious than what meets the eye on examination, and if not diagnosed properly and managed well, including the post-repair period, there would be short- and long-term consequences.

The editor, Dr. Stergios K. Doumouchtsis, and the authors who contributed to this unique book *Childbirth Trauma* need to be congratulated for their excellent contribution to this important and yet somewhat neglected area. The book consists of 18 well-constructed chapters starting from anatomy of the pelvis and the anorectal anatomy and physiology. This is followed by the effect of pregnancy on the pelvic floor and injuries related to mode of delivery. Episiotomy is one of the most common minor, sometimes unwanted, operations; the details of episiotomy are discussed. The issues related to types of injury, clinical assessment, diagnosis and management are covered next. Short- and long-term follow-up are essential and yet are ignored or not enough attention is paid. These issues are discussed in the next few chapters with an account of the healing process. The final chapters deal with the important aspects of pregnancy, puerperium and pelvic organ prolapse, obstetric fistula, prediction, risk assessment and prevention of childbirth trauma, the role of physiotherapy and the prognosis of childbirth trauma.

The chapters are written in easily 'digestible' language with useful illustrations. The women who are pregnant, those with injuries, midwives, nurses, physiotherapists, psychologists and medical staff would benefit by reading this book. It would be an essential companion for those who deal with childbirth trauma almost on a daily basis, and hence it is highly recommended.

London, UK

Sir Sabaratnam Arulkumaran,
PhD, DSc, FRCS, FRCOG

Preface

Four out of five women sustain some degree of perineal trauma during childbirth. Although in most cases perineal trauma is of minor degree, more significant injuries can be associated with serious physical and psychological morbidities. Childbirth injury to the pelvic floor is one of the most important risk factors for the development of pelvic organ prolapse and is associated with urinary and faecal incontinence and sexual dysfunction with potentially severe impact in women's quality of life.

Historically, childbirth trauma and associated morbidities have been considered part of "being a mother" and have not received sufficient attention. In recent years a systematic approach in clinical diagnosis and management of perineal trauma has been promoted via guidelines, training programmes and raised awareness among healthcare professionals. In addition, advances in imaging anal endosonography, magnetic resonance imaging and 3D/4D ultrasound have improved our understanding of these significant childbirth-related complications, their associated morbidity and our ability to diagnose and manage trauma and its sequelae. Although clinically diagnosed overt anal sphincter injury is relatively rare, with an incidence up to 6.4 %, occult anal sphincter laceration can be identified by ultrasonography, in up to 44 % of parous women.

There has been much focus on anal sphincter injuries and perineal morbidity; however, lower urinary tract as well as lower GI tract dysfunction secondary to childbirth has not always been part of an integrated clinical management or research agenda. In addition, controversies still exist. The mechanisms by which pregnancy and childbirth lead to failure of pelvic organ support are not completely understood. Research with the use of biomechanical modelling techniques has increased our understanding of such injuries to some degree, but still several questions remain unanswered. For example, although episiotomy is globally the second most common surgical procedure after umbilical cord ligation, there is a lack of professional consensus regarding specific episiotomy indications. There is also a variation in the management of anal sphincter injuries. Lack of standardisation in definitions used for levator injuries and defects, resulting in a variation in the reported incidence, is another example.

This book aspires to be an integrated bibliographic reference and to provide evidence-based and up-to-date information on anatomy, physiology of the female pelvis and pelvic organs as well as a comprehensive approach to topics that span the

entire spectrum of childbirth trauma, including different types of trauma, diagnosis, management, prognosis, prediction and prevention.

An international panel of leading experts has contributed chapters and topics from different specialities and disciplines including gynaecological, urological, coloproctology and physiotherapy perspectives. The content of each chapter represents the views of each author. We aimed, however, to contain references from the Royal College of Obstetricians and Gynaecologists (RCOG), American College of Obstetricians and Gynecologists (ACOG), National Institute for Health and Care Excellence (NICE) and Cochrane Database of Systematic Reviews.

This book will be a useful resource to all professionals providing care for women in pregnancy, labour and puerperium and managing women with childbirth trauma.

I would like to express my gratitude and acknowledgements to my colleagues who have supported this book with their expertise, time and overall input. I am grateful to Diane Lamsback, Developmental Editor, Melissa Morton and the rest of the editorial team at Springer for their help, hard work and patience to help complete this book.

I am also very grateful to each of my mentors throughout my career and particularly to Professor Sir Sabaratnam Arulkumaran for his support and inspiration and for being a role model for me over the years.

London, UK Stergios K. Doumouchtsis

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Female Pelvic Anatomy

1

George Iancu

Abstract

Knowledge of female pelvic anatomy helps the clinician recognize correctly childbirth trauma and manage it accordingly. This chapter presents aspects of the anatomy of abdominal wall, external and internal genital organs, anal sphincter and pelvic muscles, including levator ani. The vulva consists of mons pubis, clitoris, hymen, labia minora and majora, vestibule and urethral meatus. The vagina is a virtual cavity between the bladder anteriorly and rectum posteriorly, a musculo-membranous organ that connects the vulva and the uterus. The muscles of the external female genitalia are formed by pelvic muscles and cavernous bodies (ischiocavernosus and bulbocavernosus). The pelvic diaphragm that supports the pelvic load is formed by the levator ani muscle (pubovaginalis, pubourethralis, puborectalis and iliococcygeus muscles) and the coccygeus muscle. The anal sphincter is formed by the external and internal anal sphincter, with different structures and function.

Keywords

Anatomy • Female genital organs • Pelvic diaphragm • Levator ani muscle • Anal sphincter • Pudendal nerve

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Abdominal Wall

The anatomy of the anterior abdominal wall is usually described together with the anatomy of female reproductive system because of the changes in volume and shape in pregnancy and the implications of surgery of the lower abdomen. The anterior abdominal wall is divided into sections, namely from lower to upper parts – hypogastric region in the lower centre, right and left ilio-inguinal regions, umbilical region around the umbilicus, bordered laterally by right and left lumbar regions and epigastric region in the upper centre with right and left hypocondriac regions on the sides. The structure of the anterior abdominal wall is layered, consisting of skin, subcutaneous adipose tissue, muscle fascia, muscle and parietal peritoneum.

The skin is soft and elastic, loosely attached to the underlying tissue, excepting the umbilical region. The orientation of collagen fibers in the dermis forms Langer lines, arranged transversely, as are the tension forces in the abdominal skin. The importance of force distribution in the anterior abdominal wall is illustrated by the fact that vertical skin incisions usually heal with wider scars compared with transverse incisions because of the lateral tension.

Underneath the skin, the subcutaneous adipose tissue is organized into two layers: Camper's fascia, more superficial, consisting essentially of fat tissue, and Scarpa's fascia, placed deeper, a fibroelastic membrane attached to the fascia lata and aponeurosis in the midline.

The rectus sheath is made of strong fibrous tissue that supports the rectus and pyramidalis muscles; it also contains vessels (inferior and superior epigastric vessels) and nerves (terminal branches of lower six thoracic nerves). It is wider superiorly and attaches to the sternum, xiphoid process and lower border of the costal cartilages (seventh to ninth), while inferiorly it is narrow and attaches to the symphysis pubis. The rectus sheath is formed by the aponeuroses of transversus abdominis, internal and external oblique muscles. The internal oblique muscle aponeurosis splits in two lamellae at the lateral border of the rectus muscle cranially and remains unsplit in the lower third of the rectus aponeurosis. The cranial two-thirds of the rectus sheath are formed by anterior wall (external oblique sheath and anterior lamella of internal oblique sheath) and posterior wall (transversus abdominis sheath and posterior lamella of internal oblique sheath); for the caudal one-third, all three aponeuroses fuse anteriorly, and the posterior wall of rectus sheath is formed only by fascia transversalis. The border between the cranial two-thirds and the caudal one-third is called the arcuate line.

The blood supply consists of branches of the femoral artery and external iliac artery. The branches of the femoral artery are (from medial to lateral): superficial external pudendal artery, superficial epigastric and superficial circumflex iliac artery. They originate from the femoral artery immediately below the inguinal ligament, at the level of the femoral triangle. These branches supply the overlying skin and subcutaneous tissue; the superficial epigastric artery has a course towards the umbilicus; it is usually identified during low transverse abdominal incision procedures. The branches from external iliac artery are inferior (deep) epigastric artery and deep circumflex iliac artery; they supply the deeper layers, namely, muscles and fascia of the anterior abdominal wall. The inferior epigastric artery has a course

initially lateral to the rectus muscle, then posterior, between the posterior aspect of the rectus muscle and the sheath. At the level of the umbilicus, it anastomoses with the superior epigastric artery, a branch of the internal thoracic artery. The veins follow the course of the arteries.

Innervation is provided by the iliohypogastric and ilioinguinal nerves originating inferiorly from L1 dermatome, while the superior area is supplied by the abdominal extension of the intercostal (T7-T11) and subcostal (T12) nerves.

External Genital Organs

Vulva

The vulva consists of all the visible structures from pubis to the perineal body: mons pubis, clitoris, hymen, labia minora and majora, vestibule and urethral meatus.

Labia Majora

The labia majora are anatomic structures originating in the mons pubis, consisting essentially of fat tissue, rounded in shape; they terminate posteriorly in the perineum. They correspond embryologically to the male scrotum. The round ligaments terminate in their upper extremities. The overlying skin is covered with hair laterally; it lacks hair on the inner surface. There are also numerous sweat and sebaceous glands. Their size varies with age, height, weight or parity, being approximately 7–9 cm long, 2–4 cm wide and 1–1.5 cm thick. Under the skin there is a rudimentary, poorly developed muscle layer that forms the tunica dartos labialis. The fatty labial structure is abundantly supplied with a rich venous plexus that can develop varicosities in pregnancy due to increased venous pressure. Arterial supply comes from internal and external pudendal arteries.

Labia Minora

Between the labia majora and the vaginal opening there is a pair of thin skin folds named labia minora. They are about 5 cm long, 0.5–1 cm thick and 2–3 cm wide. The labia minora extend from the base of clitoris, where they bifurcate to form the prepuce and the frenulum of the clitoris. Posteriorly, the labia minora fuse at the posterior commissure or fourchette. They consist of connective tissue, mainly elastin fibers, vessels and smooth muscle fibers and nerve endings; they do not contain fat tissue and are covered with stratified squamous epithelium on the lateral aspect and non-keratinized epithelium medially. They lack hair follicles, are smooth and pigmented and contain many sebaceous glands.

The arterial blood supply is from branches of the superficial perineal artery, branch of the dorsal artery of clitoris and the medial aspect of rete of labia majora; they drain in the venous plexus of labia majora and then in the inferior haemorrhoidal veins posteriorly and clitoral veins anteriorly. The lymphatic drainage involves the superficial and deep subinguinal nodes. Innervation is provided by the pudendal nerve through perineal nerves.

Clitoris

The clitoris is the principal erogenous female organ located between the clitoridal hood (prepuce) and external urethral meatus. It is about 2 cm in maximum length and consists of body, glans and two crura. The latter extend laterally at the anterior vulvar part. Usually the glans does not overpass 0.5 cm and is composed of erectile tissue being covered by the prepuce and containing ventrally the frenulum of the clitoris. The erectile body or corpus clitoridis consists of two corpora cavernosa; they extend laterally and form the crura, which lie beneath the ischiopubic ramus bilaterally and deep beneath the ischiocavernosus muscle.

The arterial blood supply is provided by the dorsal artery of the clitoris, terminal ramus of the internal pudendal artery. The venous drainage follows the pudendal vein pathway through pudendal plexus. The superficial inguinal ganglia receive the lymph from the clitoris. Innervation is abundant in the prepuce while it is absent within the glans.

Vestibule and Vestibular Glands

The vestibule is located between the labia minora, clitoris, external surface of the hymen and posterior commissure or fourchette. This is the level of the urethra and vaginal opening, Bartholin and Skene gland ducts. The fossa navicularis is the area between the fourchette and vaginal opening, located posteriorly. The vestibular glands are two Bartholin glands (greater vestibular glands), the paraurethral glands, the largest being Skene glands and the minor vestibular glands.

The bulbs of the vestibule are elongated masses of erectile tissue around the vaginal opening; they join each other anteriorly and end up in the clitoris. At their posterior ends lie the greater vestibular glands (Bartholin). The bulbs are covered posteriorly by the bulbocavernosus muscle.

Between the vestibule and vaginal opening lies the hymen, an elastic membrane with various shapes and openings. In postcoital state, it presents as hymenal remnants or caruncles around the vaginal opening. The external urethral meatus opens at about 2–3 cm posterior to clitoris or 1–1.5 cm below the pubic arch.

Vagina

The vagina is a virtual cavity that lies between the bladder anteriorly and rectum posteriorly. It is a musculo-membranous organ between the vulva and the uterus. The upper third of the vagina originates embryologically from the Müllerian ducts, while the lower two-thirds originate from the urogenital sinus. The vagina is separated from the bladder by the vesicovaginal septum and from the rectum by the rectovaginal septum inferiorly, while the upper vagina is separated by the Douglas cul-de-sac or the rectouterine pouch. The vaginal length varies between individuals, the posterior wall being longer than the anterior wall; consequently, the posterior vaginal cul-de-sac is deeper than the anterior one. Usually, the posterior vaginal wall is about 7–10 cm in length, while the anterior wall is 6–8 cm. The vaginal length and capacity vary with hormonal status and parity. The vaginal lining consists of non-keratinized stratified squamous epithelium; the wall

structure is constituted of smooth muscle and connective fibers (collagen, elastin). It does not contain any glands. Vaginal secretion is produced by transudation from the rich vascular plexus in its structure. Vascular supply is provided by the descending vaginal branches from the cervical branch of the uterine artery; it irrigates the upper vagina. The distal portion of the vagina is irrigated by branches of the internal pudendal artery. The posterior vaginal wall receives branches of the middle rectal artery. The vascular branches on one side anastomose with the contralateral vessels to form rich vascular plexuses. Lymph drains through iliac nodes (external, internal and common) for the upper third of the vagina, while the middle third drains into the internal iliac nodes and the lower third drains into the inguinal nodes.

Perineal Muscles

The muscles of the external female genitalia are the pelvic muscles and cavernous bodies (Fig. 1.1).

Ischiocavernosus Muscle

The origin of ischiocavernosus muscle is, as the name suggests, at the ischial tuberosity and inferior ramus of ischium bone. Its course runs along the inferior surface of the symphysis pubis medially and terminates at the clitoridal base on the anterior surface of the symphysis. The ischiocavernosus muscle sends fibers medially around the proximal urethra to form part of the voluntary urethral sphincter. Its function is to slow venous return and maintain the clitoris erection. Vascular supply is provided by the perforating branches of the perineal artery on its course towards the clitoris. Innervation originates from the pudendal nerve.

Bulbospongiosus Muscle

The bulbospongiosus muscle originates from the central tendon of the perineum; its course runs anteriorly around the vaginal opening, covering the bulb of vestibule. It inserts into the fibrous tissue covering the corpus cavernosus of the clitoris, the fibrous tissue dorsal of the clitoris and sends fibers to the striated urethral sphincter. It contributes to clitoral erection and orgasm, and closes the vagina. Blood supply is ensured by the perineal branches of the internal pudendal artery. Lymphatic drainage is via the superficial inguinal nodes and posteriorly towards the rectal nodes.

Pelvic Floor

The pelvic diaphragm is a fibromuscular structure. It is formed by the levator ani muscle and the coccygeus muscle.

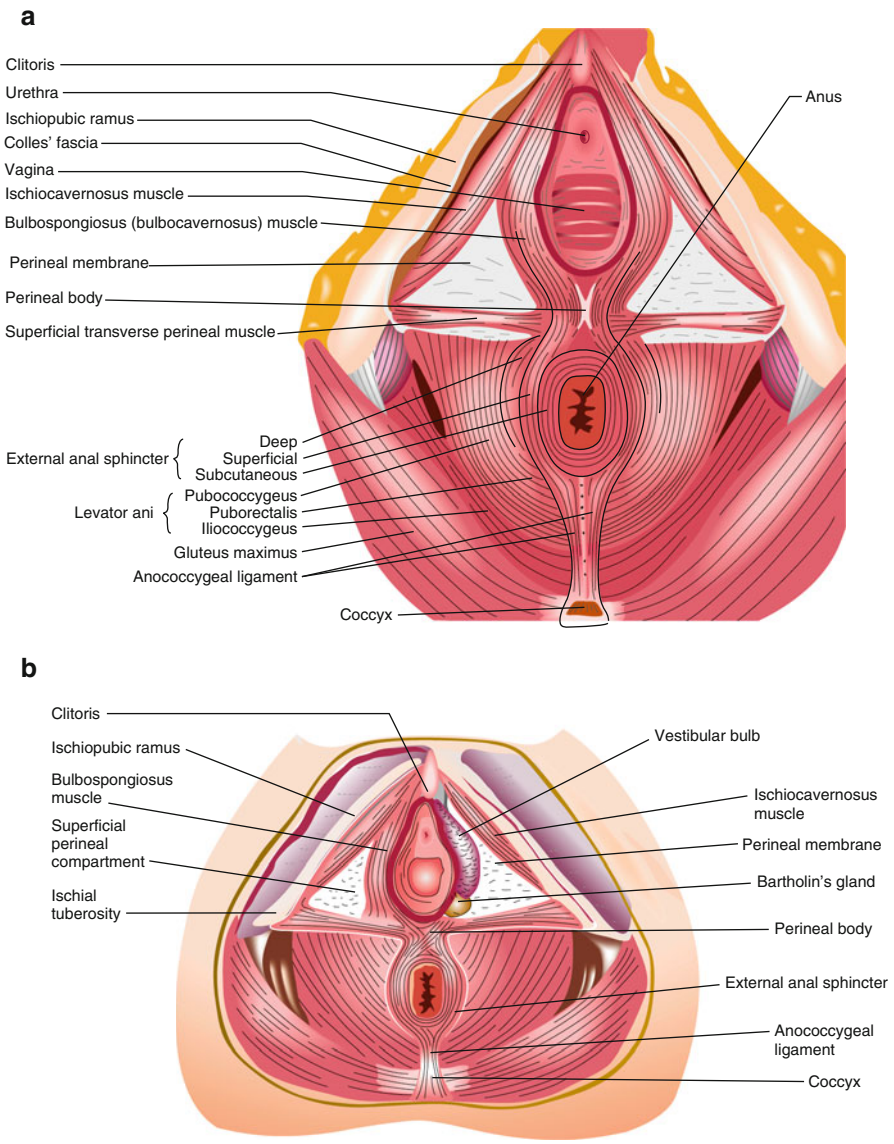

Fig. 1.1 Superficial perineal compartment (superficial transverse perineal muscles (**a**), vestibular and Bartholin's gland after left bulbospongiosus muscle removal (**b**) (With kind permission from Springer Science + Business Media: Thakar and Fenner [5], Figure 1.2, p. 1–12)

Levator Ani

The levator ani is a group of striated muscles with a very important role in pelvic organ support (Fig. 1.2). It is practically the most important supportive structure of the pelvis and forms together with its fascial structures the pelvic diaphragm. It is funnel-shaped and is covered superiorly and inferiorly by connective tissue forming

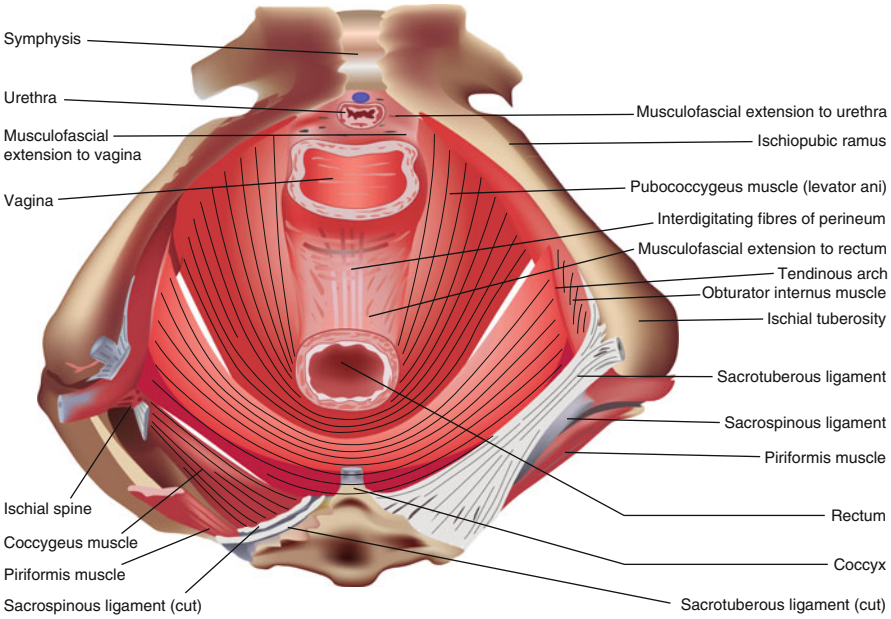

Fig. 1.2 Levator ani muscle (With kind permission from Springer Science+Business Media: Thakar and Fenner [5], Figure 1.7, p. 1–12)

the superior and inferior fasciae of the levator; it is perforated by the urethra, vagina and anal canal as they exteriorize on the perineum. The levator ani is formed by three muscles: pubococcygeus, iliococcygeus and puborectalis muscles. It is frequently damaged during childbirth, mainly with instrumental deliveries [1]. The pubococcygeus is made of puboperinealis, puboanalis and pubovaginalis muscles, according to muscle fibers' insertion. It is named sometimes also pubovisceral muscle because of its insertion on pelvic viscera. The boundaries between levator muscle components are vague and difficult to identify anatomically. The complexity of the levator structure and function is the cause of the confusion in its description and terminology in the literature. Kearney et al. reviewed the literature regarding the origin and insertion points as well as the terminology used to describe the levator and its components (Table 1.1) [2].

The pubococcygeus muscle originates from the inner surface of the pubic bone; its course runs inferiorly and medially to insert into the lateral vaginal walls (pubovaginalis), perineal body (puboperinealis) and anal wall, at the line corresponding to the intersphincteric groove between the two components of anal sphincter (internal and external). The puborectalis muscle is U-shaped, surrounding the anorectal junction.

The iliococcygeus muscle originates laterally from the arcus tendineus levator ani and inner surface of the ischial spines; it forms most of the levator plate. A few fibers attach the inferior sacrum and coccyx, but most of them join the opposite fibers to form the anococcygeal raphe; the raphe continues with the anococcygeal ligament.

Table 1.1 Levator ani structure and function

Levator ani	Origin	Insertion	Function
Pubococcygeus			
Puboperinealis	Pubic bone	Perineal body	Constant tone pulls perineal body ventrally toward pubis
Pubovaginalis	Pubic bone	Lateral vaginal wall (mid-urethral level)	Elevates vagina in region of mid-urethra
Puboanalis	Pubic bone	Intersphincteric groove between internal and external anal sphincter; ends in the anal skin	Elevates the anus and the anal skin
Puborectalis	Pubic bone	Joins contralateral fibers and forms a sling behind the rectum	Closes pelvic floor and forms anorectal angle
Iliococcygeus	Tendinous arch of the levator ani	The two sides fuse in the iliococcygeal raphe	Supportive diaphragm that spans the pelvic canal

Adapted from Kearney et al. [2]

The role of levator ani is mainly supportive. The pubovaginalis supports the lateral vaginal walls and indirectly the urethra, participating in the continence mechanism. The puboperinealis narrows the genital hiatus drawing the perineal body towards the symphysis pubis during contraction. The puboanalis contributes to the narrowing of genital hiatus and elevates the anus. The puborectalis muscle elevates the anorectal junction and is considered part of the anal sphincteric mechanism. The iliococcygeus muscle has an important role in pelvic support, as already mentioned. The levator ani muscle has some particularities that make it different from other muscles: (1) the permanent muscle tone contributes to the normal pelvic support, except during voiding or defecation; (2) it contracts rapidly with coughing or sneezing, maintaining continence; (3) it is distended during labour and delivery, maintaining integrity in the majority of cases, and then contracts and regains normal function [3].

Coccygeus Muscle

The coccygeus muscle originates from the ischial spine and sacrospinous ligament and inserts into the lateral margin of S5 vertebra and coccyx; it supports the bone and pulls it anteriorly when contracting.

Urogenital Diaphragm

While the term urogenital diaphragm has no official entry in Terminologia Anatomica, it is still used occasionally to describe the muscular components of the deep perineal pouch. According to older texts, the urogenital diaphragm comprises of the deep

transverse perineal muscle and sphincter urethrae. Inferiorly and superiorly, it is covered by connective tissue that forms the fascia of the urogenital diaphragm. It strengthens the pelvic diaphragm anteriorly. The transverse perineal muscle originates from the ischial tuberosity and inferior ischial ramus; it runs transversely to insert medially into the central perineal tendon. Often, the fibers interdigitate around the central tendon with bulbospongiosus and puborectalis muscle fibers. The blood supply and innervation is provided by the pudendal bundle. The sphincter urethrae muscle originates from the medial aspect of the ischiopubic rami and inserts into the urethra and vagina. Its main role is to compress the urethra.

Internal Genital Organs

Uterus

Located in the pelvic cavity, between the bladder anteriorly and rectum posteriorly, the uterus has a fibromuscular structure; it is made of body superiorly and cervix inferiorly.

The cervix comprises of a vaginal part with a round convex surface or ectocervix covered with stratified squamous non-keratinized epithelium, while the endocervical canal is lined by columnar epithelium containing mucinous glands. The endocervical canal is about 2–3 cm long; it communicates cranially with the endometrial cavity and caudally with the vagina. Of particular interest is the transformation zone or the dynamic area of squamocolumnar junction that can be the origin of cervical preinvasive and invasive neoplastic pathology. The position of the transformation zone varies depending on woman's age and hormonal status; usually, in young women or during pregnancy, the columnar epithelium extends onto the ectocervix and forms what is known as ectopy, a condition that sometimes causes bleeding with intercourse. During menopause, the squamocolumnar junction is found usually within the endocervical canal.

The uterine body can have different shapes and anatomical position, varying with childbearing status or hormonal profile of the woman. The uterine body lies between the bladder anteriorly and the rectum posteriorly. The peritoneum forms pouches between the three organs, namely the vesicouterine and rectouterine pouches (Douglas). The shape of the uterus corresponds to a flattened pear or pyriform, the upper part being the uterine body and the lower, narrow part, the cervix. The cervix unites with the body through the isthmus that forms the lower uterine segment in pregnancy. The cranial part is the uterine fundus that ends on the sides with uterine cornua; at this level the fallopian tubes originate and run laterally towards the ovaries.

The most common uterine position is described as anteverted and flexed. Occasionally, the uterus can be retroverted or angling posteriorly. The flexion is the angle between uterine body and cervix and the version describes the angle between uterus and the upper vagina.

The uterine cavity is lined with columnar epithelium with mucous secreting glands, forming the endometrium. The cavity is triangular shaped; at this level, the fallopian tubes open through tubal orifices. The endometrium is hormonally

controlled and undergoes shedding monthly during reproductive years. The endometrium covers the muscular layer of the uterine wall, the myometrium. The myometrium forms most of the uterine wall thickness, varying between 1.5 and 2.5 cm; it consists of smooth muscle fibers stratified on layers of various orientation. The interlacing muscular layers play an important role in the haemostatic mechanism during the third stage of labour. The uterus is covered by the visceral peritoneum that forms the uterine serosa, which covers the uterine body and cervix posteriorly, while the cervix anteriorly is covered by the bladder. The neuro-vascular pedicles approach the uterus laterally where the double-layered peritoneum forms the broad ligament. At the level of the uterine cornua, the fallopian tubes and the round ligaments originate, running laterally, the tubes towards the ovaries and the round ligaments towards the internal inguinal orifices; their course continues through the inguinal canal inserting into the labia majora.

Ovaries

The ovaries are the female gonadal structures, paired, lying in the lateral pelvic wall in the ovarian fossa; at this level, they are in close proximity with the ureters, internal iliac and obturator vessels and nerve and with the uterine artery at its origin. The lateral surface faces the pelvic wall, while the medial surface lies in proximity to the uterus and the broad ligament; the fallopian tube approaches the ovary with the fimbria. The ovary has an anterior border that encloses the vascular pedicle (mesovarium) towards the posterior leaf of the broad ligament and a posterior border that faces the peritoneum. The ovary attaches to the uterine cornu through the ovarian ligament, located medially and inferiorly. The ovary is supported and vascularized by the infundibulopelvic ligament, the ovarian ligament and mesovarium.

Anal Sphincter Complex

The anal sphincter is formed by two groups of muscles, the external anal sphincter and the internal anal sphincter, that differ in structure and function (Fig. 1.3).

The external anal sphincter (EAS) consists of three parts – subcutaneous, superficial and deep. It is a striated muscle surrounding the most inferior part of the anal canal [4]. The subdivisions are difficult to identify through anatomical dissection, although information about them could be obtained using imaging studies [5]. The deepest fibers of the EAS mix to some extent with fibers of the puborectalis and transverse perineal muscles anteriorly, with no attachments posteriorly. The middle part of the EAS attaches anteriorly to the perineal body and posteriorly to the coccyx through the anococcygeal ligament. Posteriorly, some fibers of the superficial EAS attach to the anococcygeal raphe. The lowest part of the EAS surrounds the lowest part of the anal canal, with no more internal anal sphincter in between [4].

The internal anal sphincter (IAS) is a smooth muscle that is a continuation of the circular smooth muscle of the bowel. The IAS ends at 6–8 mm above the anal margin; this corresponds to the junction of the subcutaneous with superficial parts of the EAS.

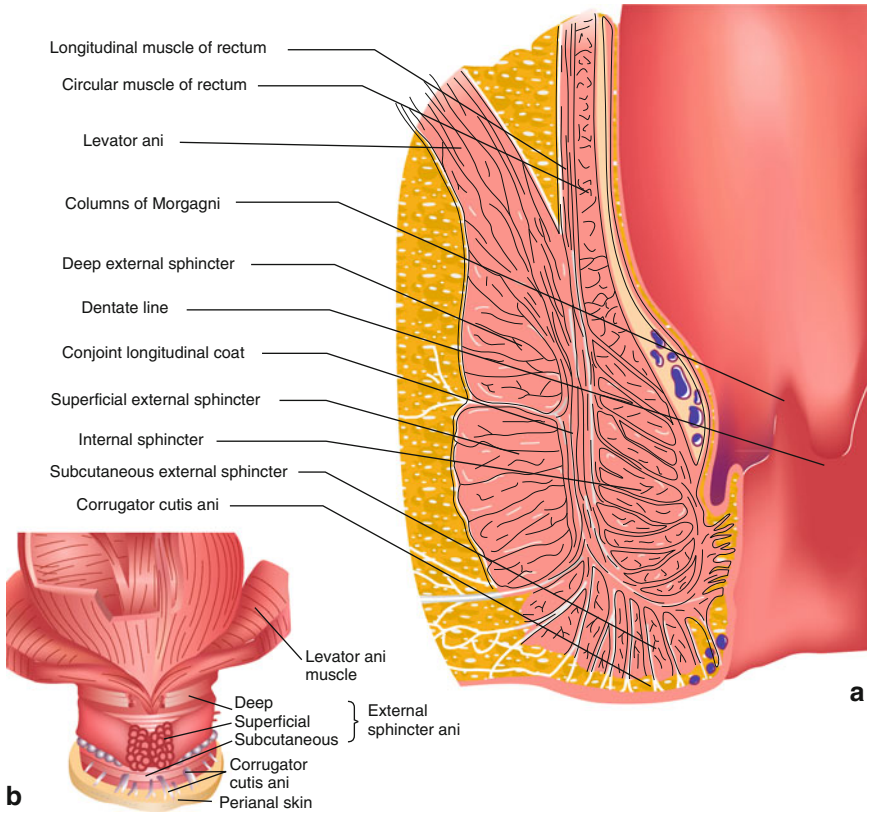

Fig. 1.3 (**a**, **b**) Anal sphincter complex (With kind permission from Springer Science + Business Media: Thakar and Fenner [5], Figure 1.4, p. 1–12)

Between EAS and IAS, there is a vertical muscular layer, the longitudinal anal muscle. It lies between the layers of EAS and IAS and extends along the anal canal from the anorectal junction to the perianal dermis; at this level, it terminates through seven to nine fibroelastic septa, which cross the superficial part of EAS. It consists of outer striated fibers, originating probably from the levator ani and inner smooth muscle fibers, from the rectal longitudinal muscle layer [6].

The Pudendal Nerve

The pudendal nerve is one of the major branches of the sacral plexus, together with the sciatic, superior and inferior gluteal and posterior femoral cutaneous nerves. It is the main perineal nervous structure, having sensory and motor function. It innervates the external anal sphincter (EAS), urethral sphincter, perineal musculature and perineal skin. It originates from the ventral roots of the sacral nerves between S2 and S4 and receives contributions from S1 and S5 [7]. The main branches of the pudendal nerve are inferior rectal (or anal) nerve and two

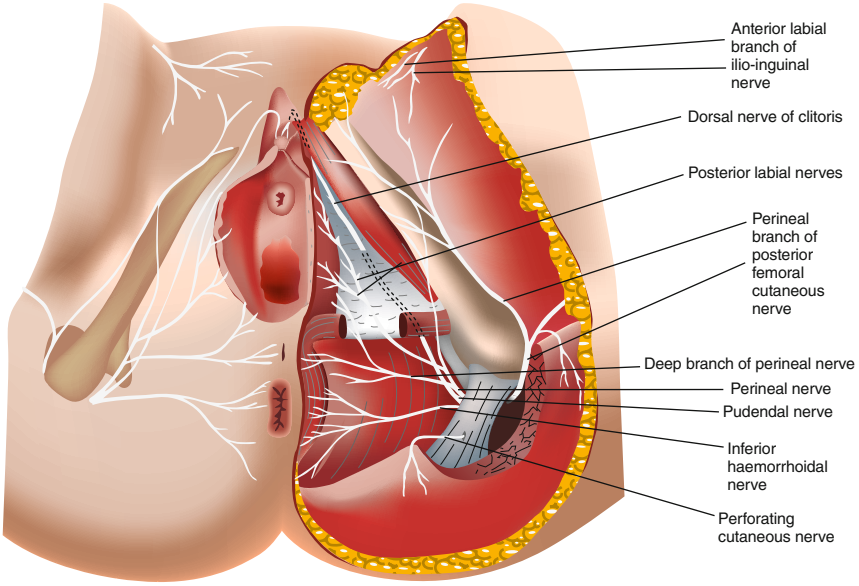

Fig. 1.4 Pudendal nerve – terminal branches (With kind permission from Springer Science+Business Media: Thakar and Fenner [5], Figure 1.8, p. 1–12)

terminal branches, the perineal nerve and dorsal nerve of clitoris. The inferior rectal branch originates from the pudendal nerve before it enters the pudendal canal (Alcock's canal) and supplies the skin and muscle of the anal triangle (EAS). The perineal branch divides into smaller branches for labia majora, transverse perineal muscles, urethral sphincter and perineal skin. The dorsal nerve of the clitoris contains part of the terminal fibers of the nerve, innervating the corpus cavernosum and ending in glans clitoris.

The pudendal nerve passes between the piriformis muscle and coccygeus (ischiococcygeus) muscles and leaves the pelvis through the inferior part of the greater sciatic foramen. After crossing the ischial spine, the pudendal nerve re-enters the pelvis inferiorly through the lesser sciatic foramen and joins the internal pudendal vessels on the lateral pelvic wall at the level of ischiorectal fossa. The obturator fascia splits and generates a sheath that contains the pudendal nerve and vessels – the Alcock's canal. The inferior rectal branch is derived from the pudendal nerve before the latter enters the canal. Rarely, the inferior rectal nerve can originate directly from the sacral plexus [8]. The terminal branches of the pudendal nerve (perineal and dorsal nerve of the clitoris) arise near the midpoint of the pudendal canal and travel together to the end of it. The perineal nerve ends as sensory and motor branches to the perineum and EAS, while the dorsal nerve of the clitoris ends as a true terminal branch at the level of clitoris and infrapubic region (Fig. 1.4). Studies on cadavers identified an additional branch of the pudendal nerve at the level of the sacrospinous ligament that innervates the perineum and levator ani muscle [8].

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Anorectal Anatomy and Physiology

2

Filippo La Torre, Diego Coletta, Federica Costanzo, Fabiano Iaquinandi, and Francesco Velluti

Abstract

The anorectal canal has the important function of regulating defecation and the role of controlling fecal continence. To fulfill its function, it is necessary to integrate muscular and sensory components. Continence in normal conditions is maintained by the acute angle that the contraction of the pubo-rectal muscle creates at the recto-anal junction, the rectal compliance, and the area of high pressure 2 cm from the anal verge. The resting pressure of the channel is attributed by 80 % to the tonic contraction of the internal anal sphincter, and the remaining 20 % is due to the action of the external anal sphincter and a small part of the hemorrhoidal cushions. During the filling of bulb, the distension of the rectum evokes the reflex inhibition of anal canal, with relaxation of the internal sphincter and contraction of the external sphincter. During defecation the external anal sphincter is voluntarily inhibited, the pubo-rectal muscle is relaxed with the aid of the abdominal muscles, and the fecal material is pushed out of the intestine.

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Anatomy of the Anorectal Canal

The rectum and anal canal are the terminal parts of the large bowel. The rectum has a length of 15 cm and follows the pelvic colon, from the rectal-sigmoid junction, at the third sacral level, until the recto-anal verge.

Anatomically the rectum is composed of two portions separated by the insertion of the levator ani muscle into the rectal wall: the rectal ampulla placed in the small pelvis and the anal canal located in the rear perineum [1, 2].

On its way, the rectum shows two curvatures: the first is the sacral curvature situated in front of the anterior sacrum and coccyx, in anterior concavity. The second curvature is the perineal curvature in anterior convexity, alongside prostate apex in men and middle part of vagina in women.

In addition to these curves, there are also two flexuosities on the coronal plane, one convex on the right side and one on the left side, varying depending on the rectal distention.

When the organ is distended, it shows four walls, front side, rear, and two lateral sides, which tend to droop in conditions of vacuity. The walls show on the surface a series of transverse grooves corresponding to internal folds.

This part of intestine is not completely covered by the visceral peritoneum; in fact, the front wall below the pouch of Douglas and a large part of the side walls and the posterior wall [3] are lacking serosal lining. Considering the peritoneum, the rectum can be divided into a peritoneal part and a subperitoneal part, with different relationships depending on the sex. In males, the portion above the peritoneal part corresponds to the rectovesical pouch and is related to intestinal loops, and the subperitoneal part is related to the bladder trigone, the prostate rear wall, the seminal vessel, and the seminal vesicles. In females, the supraperitoneal part walls toward the rectum-uterine pouch, and the peritoneal part is related to the posterior wall of the vagina by which is separated through the rectovaginal septum.

The posterior wall, in both sexes, is separated from the spine by the retrorectal space filled by fibro-adipose material, through which the middle sacral artery runs ending lower with the coccygeal glomus. Finally, the two side walls lean against the walls of the small pelvis [4]. The perineal portion of the rectum is related to nearby organs, according to sex: in men the rectum-urethral trigonum separates it from the prostate apex, urethra, and urethral bulb glands; in women the rectovaginal trigonum is interposed between rectum and rear wall of the vagina.

The inner surface of the organ presents transverse folds, corresponding to the grooves on the outer surface and longitudinal pleats disappearing in case of vacuity. Two inches above the anal orifice, the inner rectum surface lifts in columns that are 5–10 longitudinal pleats presenting an inferior flared base and ending thinner after

about 1 cm distance. Among the bases of the anal columns, we find cross pleats called semilunar valves creating the pectineal line, irregular and with circular course. Each valve together with the wall of the rectum delimits the rectal sinus, a pocket irrigated by the anal glands that secrete mucous. Below the dentate line, we find an area rippled by radiated folds known as the hemorrhoidal ring [5].

The anal orifice is located approximately 3 cm in front of the coccyx, in the posterior perineum, and is the external outlet of the anal canal. At rest shows up a right and left lip that hold on anteriorly and posteriorly with two commissures; when it is expanded instead, it assumes a circular outline. The anal skin is pigmented and haired in the male. At rest the skin rises in radiated folds that disappear when the anus dilates. The perianal skin is rather rich in apocrine sweat glands called circumanal glands.

The rectal tunica mucosa in its upper side is cylindrical and tends to become cuboidal at the bottom; this epithelium continues until the upper third of the anal canal and at the level of the dentate line changes irregularly, becoming stratified squamous epithelium and covering all structures of the anal canal. It is wrong to define the dentate line as "mucocutaneous junction" because this epithelial passage isn't definite; indeed sometimes the squamous epithelium can thrust above the dentate line [6]. The squamous stratified epithelium, beneath the hemorrhoidal ring, shows signs of keratinized layer together with hairs, sebaceous glands, and circumanal glands. The submucosa tunica placed in the ring hemorrhoidal area hosts the hemorrhoidal plexus (Fig. 2.1).

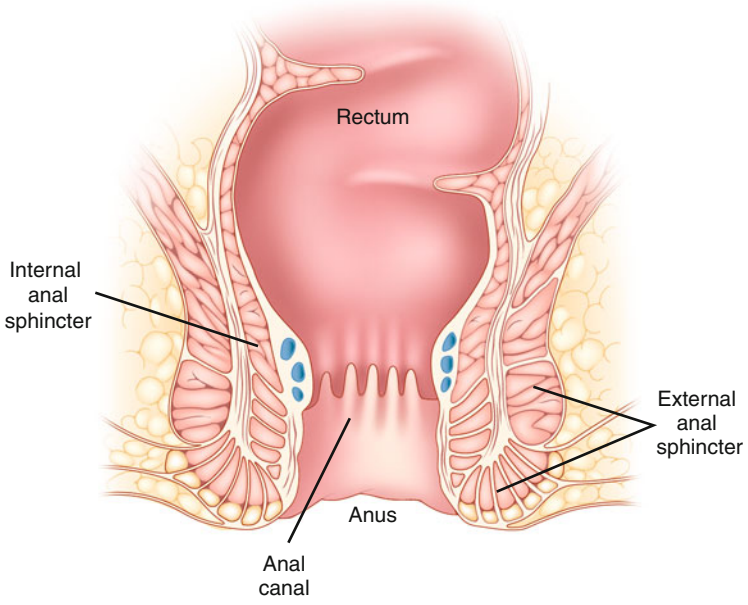

Fig. 2.1 Rectum

Anorectal Muscles

The rectal tunica muscularis, such as in the colon, is composed by one inner circular layer and one external longitudinal layer. The latter is an expansion of colon taenia, which, at the junction of sigmoid-rectal, creates a continuous muscular layer. Concerning rectal reservoir, there are links between the two kinds of musculature, since the longitudinal one opens like a fan within the circular muscle; this particular structure is easy to find at the level of the rectal valves. At the lower part of the rectum, the longitudinal muscular layer merges with the striated muscle of the levator ani muscle and with the fibroelastic tissue from the pelvic fascia overlying the pelvic diaphragm, in order to form the joint longitudinal muscle of the anal canal. In turn, at the level of the anal sinuses, the circular muscle thickening forms the internal sphincter muscle of the anus. This muscle is made by smooth muscle cells, innervated by the autonomic nerves of the intrinsic nervus plexuses (myenteric and submucosal), and terminates with an inferior boundary rounded at the intersphincteric line.

The longitudinal muscle joint (LMJ) broadens inferiorly surrounding the internal sphincter muscle of the anus and, in its turn, is surrounded by the external sphincter muscle of the anus. During its downward course, the LMJ emits a series of fibroelastic and muscular fascicles, which penetrate the internal sphincter muscle of the anus; some of these are joined to the muscularis mucosae of the anal canal composing the muscle of the anus submucosa (or "sustentator mucosae of Kohlrausch"), whose fibers anchor the anoderm of the dentate line to the underlying tissues and to the lower third of the internal sphincter muscle of the anus. Fixed in this way, the dentate line prevents the eversion of the anal canal and supports the overlying internal hemorrhoidal venous plexus during defecation [7]. The LMJ in its lower part before joining with the muscularis mucosa of the anal canal issues a series of fan-shaped fibro-muscular septa, which pass through the submucosal portion of the external sphincter muscle of the anus and are combined with perianal skin forming the corrugator muscle of the skin of the anus. A series of bundles in the opposite side is directed outward, passing through the superficial subcutaneous portion of the external sphincter muscle of the anus, lasting as transverse septum of the ischiorectal fossa. The importance of the LMJ consists in the fact that, along with the levator ani muscle, it exerts an action of lift on the sphincter and anal canal, preventing the spread of any anorectal infections.

The external musculature of the anal canal is formed by the external sphincter muscle of the anus (EAS), arranged in the shape of sleeve so as to surround 2–3 cm the terminal portion of the rectum and the anal canal. The EAS is composed by three parts: a subcutaneous, a superficial, and a deep part. The subcutaneous part about 3–5 mm in diameter surrounds the anal orifice directly above the edge of the anus, below, and slightly to the side to the internal sphincter muscle of the anus; in women, this part of the external sphincter muscle of the anus is more developed, especially anteriorly, where it forms a prominent annular bandage, which is sometimes engraved in the course of an episiotomy.

The subcutaneous part is functionally integrated with the levator ani, through expansions of the longitudinal muscle; they go through it like a fan and they terminate as fibers of corrugator muscle of skin and anus. The superficial part of the external sphincter muscle of the anus has an elliptical shape and is placed deeply and laterally to the subcutaneous part of the EAS. It is the most robust of the three parts of the EAS and originates independently from the rear face and from the tip of the coccyx, so it is sometimes called "coccygeal portion"; in the male it is connected at the front on the tendinous center of the perineum and on the median fascial raphe of bulbocavernosus muscles. In the female, muscle fibers are connected in lower part to the tendinous center; for the most part, however, they connect with the bulbocavernosus muscles. Posteriorly, the fibers form the anococcygeal raphe.

The deep part of the external anal sphincter muscle is for the most part an annular muscle bundle, not joined to the coccyx; posteriorly it is intimately fused with the pubo-rectal muscle since the fibers of this muscle, with a course in sling, pass around the terminal part of the rectum. Prior to the rectum, muscular fibers intersect with those of the opposite side mingling in part with the fibers of the superficial transverse perineal muscles; also in back fibers are mixed to fit in the anococcygeal raphe.

The essential supports that hold in position the recto-anal canal are derived from the muscles that form the pelvic floor: the levator ani muscle, consisting of three parts—the pubococcygeus muscle, the pubo-rectal muscle, and iliococcygeus. These three muscles together are located at the diaphragmatic and subdiaphragmatic floor; their organization is such that, when viewed from above, they appear as a funnel whose point is more declivous and corresponds to the gap of the rectal canal. The levator ani gives stability to the pelvic floor and acts as a fulcrum against the increased abdominal pressure, which is provoked by cough and defecation.

The pubococcygeal and the pubo-rectal part, forming a sling, becomes part of the anorectal muscular ring that surrounds part of the rectal reservoir and in part the upper portion of the anal canal. This ring is composed of the following: on its posterior half it is composed by fibers of the pubo-rectal ring; on the anterior part, by the internal sphincter muscle of the anus and fibers coming from the pubococcygeus muscle (pre-rectal or fibers of Luschka); and by the longitudinal muscle, surrounded by the deep part of the external sphincter muscle of the anus. The posterior half of that ring, along with the pubo-rectal sling, brings the rectum near to the pubic bone and increases anorectal torsion, shortens and narrows the pelvic opening, raises the anus, and collaborates to the closure of the anal canal (Fig. 2.2).

Innervation

The sympathetic and parasympathetic fibers innervate rectum, anal canal, and genitourinary system. The sympathetic innervation is derived from the first three lumbar segments that form the preaortic plexus from which fibers arise that extend below the aortic bifurcation forming the superior hypogastric plexus or presacral nerve. The fibers of the superior hypogastric plexus will lead to the sides of the pelvis

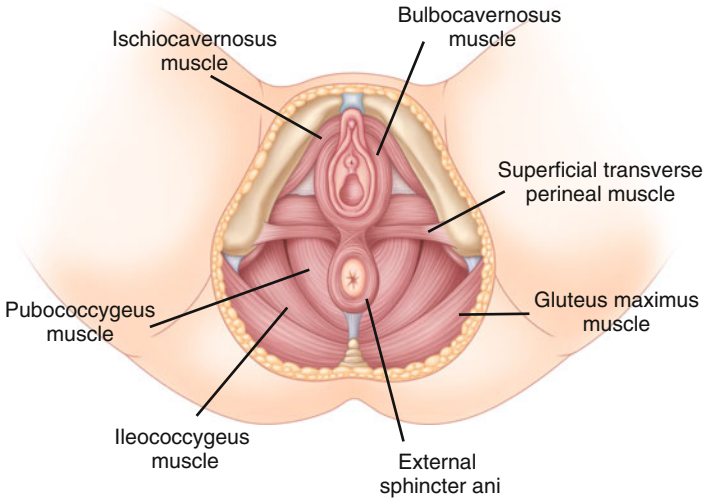

Fig. 2.2 Pelvic floor anatomy, inferior view

where they join with the branches of the parasympathetic nerves to form the inferior hypogastric plexus, or pelvic plexus, in close contact with the rectum [8].

The parasympathetic innervation is derived from the erigentes nerves, or pelvic splachnic, which originate from the II, III, and VI sacral root. The pudendal nerve arises from the sacral plexus at levels S2–S4, which provides motor innervation of the external sphincter and sensory innervation to the perianal skin. The pudendal nerve at the end of its course, at the side wall of the ischiorectal fossa, is divided into three branches: inferior hemorrhoidal, perineal, and dorsal of the penis/clitoris.

The external anal sphincter has a threefold innervation: the perineal muscular branch of the pudendal nerve for the inner or ventral side of the muscle, the anal nerve on the lateral side of the sphincter, and the perineal branch of the fourth sacral nerve for the posterior or caudal area [9]. The arrangement of these fibers is radial, numerous connections are intersegmental, and this explains the functional recovery after nerve section.

The internal sphincter essentially works as a reflex and is constituted by autonomous smooth muscle fibers. Both sympathetic and parasympathetic systems contribute to its innervation [10].

The levator ani muscle, intimately linked to the external sphincter by its puborectal bundle, receives on its upper branches fibers of the levator ani. These nerves can come directly from the third and fourth sacral root or from internal pudendal nerve.

Afferent Nerves

In the wall of the anal canal, there are numerous receptors, distributed all along the channel and also in the thickness of the wall layers.

Muscle receptors and polymodal nociceptive receptors exist in addition to specific mucosal receptors [11]. These muscular receptors are mechanoreceptors of two types: one for slow adaptation, in the internal sphincter, and the others for fast adaptation, in the external sphincter.

The sensory innervation of the anus is richer than the one of the rectum and especially at the mucosa level [12]. The sensitivity of the skin surface area of the anal canal, from the anorectal line, depends on isolated intraepithelial nerve fibers (region discriminative pain). Above this area, receptors with greatest diversity and density allow to analyze lots of information: genital corpuscles (friction), Golgi's corpuscles (pressure), Meissner's corpuscles (touch), Krause's corpuscles (cold), and Pacini's corpuscles (stretching). The sensory nerve fibers run in the hemorrhoidal side branches of the internal pudendal nerve but also with a parasympathetic group toward the hypogastric plexus and the sacral sympathetic trunk through the second and third sacral ganglion. The sensory role is particularly to discriminate accurately the quality of the content of the anal canal, of fundamental importance for continence and evacuation [13].

Nerve Centers of Control

The processing of information takes place at three levels: the enteric nervous system, the paravertebral ganglia of the autonomic nervous system, and the cerebrospinal axis.

The enteric nervous system is the support of local muscle tone. This nervous system is localized in the submucosal plexus of Meissner and between the sphincter muscular layers of myenteric plexus of Auerbach, more voluminous than the last. Regardless of the extrinsic nervous system, a local reflex system, with the presence of interneurons, allows an adjustment control within the same wall. This local nerve plexus is characterized by a wide variety of chemical mediators; cholinergic neurons are present in spontaneous activity permanently, noradrenergic neurons are very common in sphincteric zone, and non-adrenergic and non-cholinergic (purinergic and serotonergic) neurons, as well as numerous neuropeptides, are present in large quantities in the anal canal [14].

Some neuropeptides have a relaxing effect on the internal sphincter, unlike opioid neuropeptides (enkephalinergic), which represent 25 % of the neuronal population and increase sphincter tone.

The autonomic nervous system (autonomic sympathetic and parasympathetic) guarantees a faster connection for the transmission of sensory information through the paravertebral ganglia and the hypogastric plexus.

The cerebrospinal axis receives afferent information from neurons of the ganglionar root placed in the back or in the plexiform ganglion of the vegetative system. At this point, the information can pass through the synapses of preganglionic sympathetic or parasympathetic origin of segmenting reflexes or continue its afference in tractus nervosi of posterior spinal cord, without interruptions up to the brain.

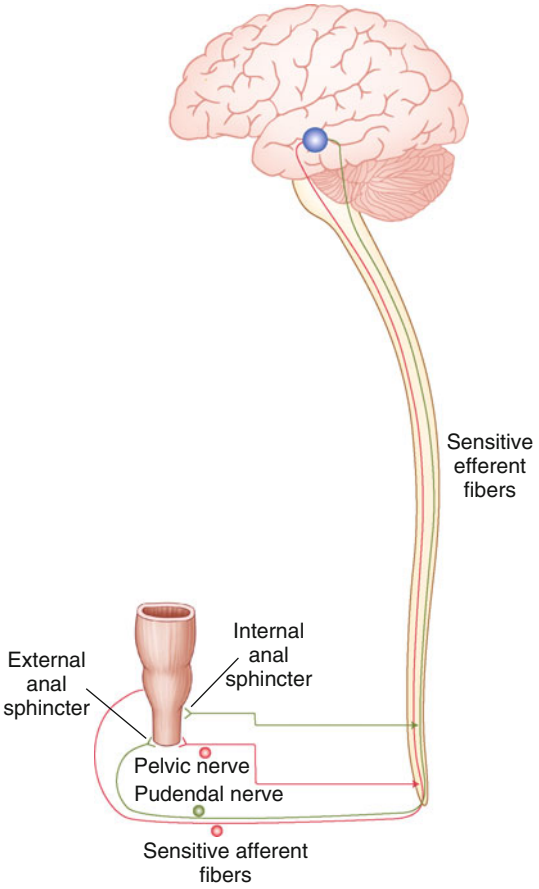

Fig. 2.3 Central nervous control

Brain areas of central regulation are still poorly defined and are located in the brain stem, hypothalamus, limbic system, and neocortex (Fig. 2.3).

Vascularization

The recto-anal canal is perfused from different arteries: the superior rectal artery, branch of the inferior mesenteric artery, middle rectal artery, branch of the internal iliac artery, inferior rectal artery branch of the internal pudendal artery, and branches of the lower sacral artery [15].

The veins that drain the recto-anal channel are the upper, middle, and lower. They come from the submucosal venous plexus or hemorrhoidal plexus. This plexus becomes richer of blood in two regions because they have ampullary expansions: above the dentate line, they form the internal hemorrhoidal plexus, and below the lower edge of the internal sphincter, the exterior hemorrhoidal plexus [16].

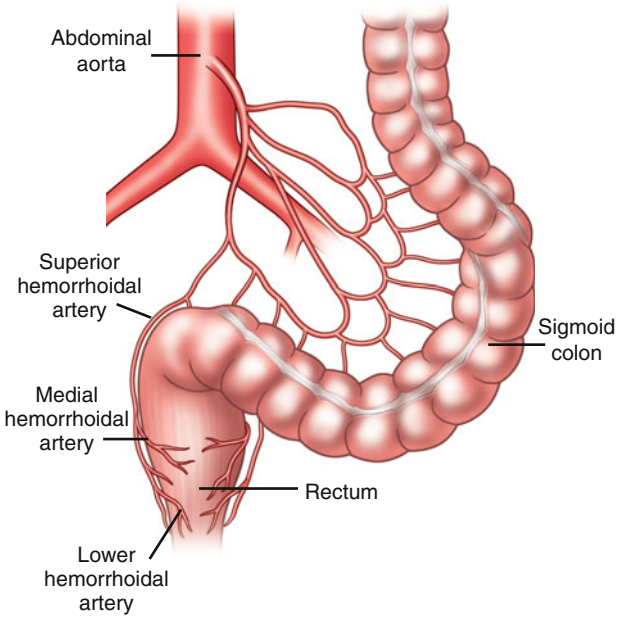

Fig. 2.4 Vascular supply

At the level of the dentate line, there is a communication between the portal and systemic circulation: the upper part of the anal canal is a tributary of the portal system through the superior hemorrhoidal vein and the inferior mesenteric vein; on the contrary, the effluent blood from the lower portion reaches the vena cava through the middle and inferior hemorrhoidal veins, tributary of the hypogastric veins (internal iliac).

As well as the venous system, the lymphatic goes in two directions: by simplifying it can be said that above the dentate line the drainage is toward the hypogastric lymph nodes (Fig. 2.4).

Neurophysiology of Defecation

The recto-anal canal serves two important functions, the fecal continence and defecation, through a complex process that integrates muscle function and somatic and visceral sensory information and checks for local and central control.

Continence

A normal anal continence allows emission voluntarily controlled, periodic, and selective of the various components of the contents of the alimentary canal: gas or

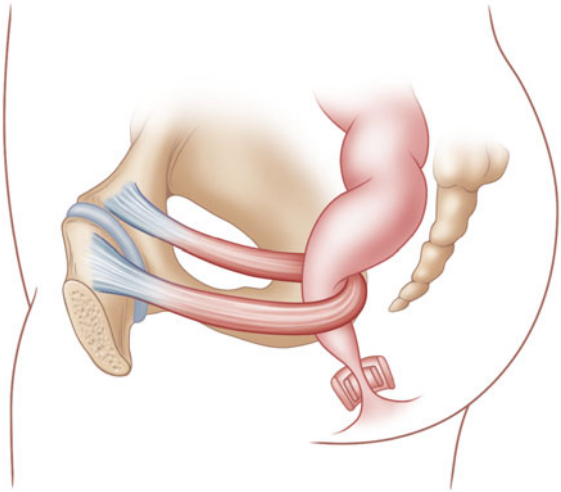

Fig. 2.5 Pelvic floor with pubo-rectal sling

liquid and solid stool. In normal conditions and during the filling of the rectal ampulla, continence is achieved by the contraction of the internal anal sphincter and the hemorrhoidal pillows. During the filling of bulb, the distension of the rectum activates the recto-anal inhibitory reflex with consequent relaxation of the internal sphincter, and this causes a small amount of fecal material to come in contact with the mucous membrane. This is rich in nerve endings and can differentiate the feces from the gas and decide whether or not to defecate [17]. If defecation should be delayed, the voluntary contraction of the external anal sphincter sends back fecal material, postponing the stimulus.

Continence is based on two elements: the ability of the rectum to host feces and anal lock mechanism, which, together with the ability of sensory discrimination of the anal canal, prevents the involuntary leakage of stool (Fig. 2.5).

"Rectal Reservoir" Function

The rectum acts as a physiological and mechanical reservoir, which extends from the angle between the rectum and sigmoid colon to the valves of Houston. It has the ability to relax and adapt, ensuring that the fecal content does not generate an increase in pressure that starts the urge to defecate [18-20].

A high-pressure zone 2 cm from the anal margin and caudal to pubo-rectal sling is important for continence because it acts as a barrier to the feces progression. This pressure difference is due to the fact that this point in the motor activity of the bowel is more pronounced than sigmoid colon, with muscular contractions more frequent and powerful; it is difficult to discriminate which muscle is responsible for this high pressure value because at this level the EAS surrounds the IAS. Nevertheless, several studies in the international literature have demonstrated that the IAS is mainly responsible [22]. The anal canal contributes to form this barrier; it has a differential pressure between its highest and lowest portion. Under normal conditions, the pressure in the rectal reservoir is between 5 and 25 mmHg.

Mechanisms of Anal Lock

The closing mechanism of the anal canal is based on the action of the sphincters and anatomical characteristics of the canal:

1. The pubo-rectal muscle is contracted tonically and causes the acute angle of the recto-anal junction, which is opposed to rectal emptying.
2. The internal anal sphincter is a smooth muscle of visceral origin. It looks like a white ring of 3–4 cm, in continuity with the rectal musculature. It is in permanent nonvoluntary tonic contraction and ensures automatic closure of the anal canal at rest. It produces the majority of the resting pressure, 80 % compared to 20 % produced by the external sphincter, which is recorded with the anorectal manometry (AMR). Its opening is induced by the rectal distension due to reflex of anorectal inhibition. The recording of the electrical activity of smooth muscle cells of the IAS has allowed the identification of sinusoidal waves, called "slow waves," with a frequency of about 16 cycles/min; this frequency does not decrease after the induction of general anesthesia or after paralysis of the external anal sphincter muscle. Other known waves are the "ultraslow electrical waves," with a frequency of about 1.6 cycles/min, identified in patients having a pressure of the anal canal higher than average. It is not clear if there is a correlation between the electrical activity of the cells and pressure measurements of the anal sphincter [21], but electromyographic recordings (EMG) of the IAS are able to demonstrate the correlation between the increase of the baseline blood pressure (with the use of a balloon inflated with air) and the frequency of the slow waves.
3. The external anal sphincter is responsible for a little part of the relaxing pressure (about 20 %); it increases anal pressure when there is a change in the intra-abdominal or intra-rectal (Valsalva maneuver) pressures. The external anal sphincter has a double contractile activity, tonic and phasic type; this may be a reflex action (stimulated by laugh, sneezing, crying with increase of abdominal pressure) or voluntary occurring during the defecatory urgency. Capacity of voluntary contraction is typically two to three times higher than the basal anal tone, and the time varies from 30 to 60 s. The contraction of the external sphincter is undoubtedly the most important mechanism for voluntary continence, but it can be maintained for short periods only, during which the rectum has time to adapt to the new volume reached and the pubo-rectal sling translates forward and at the top the anorectal junction, thus making the angle more acute. All these mechanisms allow to postpone defecation.
4. Reflexes have fundamental importance in the mechanism of fecal continence. We recognize:
   - Reflex of IAS
     If the rectum is filled with air, the IAS is released. Such reflex is also observed in patients with complete section of the spinal cord, suggesting a total independence from the control of the central nervous system.
   - Reflex of EAS
     The muscle activity of the EAS increases not only during voluntary contraction but also during the straining maneuvers (cough or Valsalva maneuver).

The perianal skin stimulation excites the muscles of the EAS, giving rise to what is called "anal reflex." This reflex is also present in paraplegic patients.

- Effect of Rectal Distension
  The rectal distension induced with 50 mL of air increases the activity of the EAS. This response precedes the inhibitory reflex of the IAS and is abolished if the pudendal nerve were blocked. This reflex persists during rectal distension, and the evidence studied proves its independence from the cortex. In normal subjects, stretching the rectum with more than 150–200 mL of air generates a conscious feeling of defecatory urgency, with the automatic inhibition of the EAS and the pubo-rectal muscle. Porter called this response "constant relaxation" [23].
- Effects of Distension of the Anal Canal
  Porter also showed that, in normal subjects, pulling down the anal canal with a finger generates an increase in EMG activity of the EAS and the pubo-rectal muscle. When the anal canal is relaxed, you have a sudden cessation of the contraction. In paraplegics initial peak recorded by EMG follows a complete inhibition for the duration of the anal distension. So in normal subjects such inhibition is masked by cortex of the EAS.
- Recto-anal Inhibitory Reflex RAIR
  At rectal filling, a relaxation of the IAS occurs, which is provoked by the intrinsic inhibitory nerve plexus. This reflex causes the descent of the rectal contents in the anal canal. At this level, the presence of receptors allows discriminating of rectal contents, triggering the act of defecation or the emission of gases.
- Anorectal Sensation
  The ability to keep feces and pass the air requires an "awareness" of the rectal contents. The rectal filling with air or water corresponds to a feeling of fullness in the pelvic floor. Nowadays it is believed that consciousness on rectal filling is due to the presence of pelvic receptors, sensitive to stretching. Despite the absence of specialized receptors in the rectal mucosa, as Pacini and Golgi-Mazzoni corpuscles, there is evidence to suggest that the rectal sensitivity is due to the stimulation of terminationes nervorum and mechanoreceptors in the rectal wall and in adjacent pelvic structures [22]. Furthermore, recent studies in guinea pig models have confirmed the existence of terminationes nervorum intra-ganglionic in the myenteric plexus of rectal wall sensitive to mechanical distension. It ensures gas-stool discrimination, very important for continence. The loss of this sensitive zone is accompanied by sensory incontinence, which appears even if the muscular apparatus is anatomically and functionally normal.

Defecation

The defecation reflex is an act that takes place under the control of will. When the feces reach the rectum, this is stretched, and there is relaxation of the internal anal sphincter (RAIR) and decrease of the pressure of the anal canal; these mechanisms allow the feces to enter in contact with the mucosa of the rectum to enable

discrimination between feces and gas. If social conditions do not allow defecation, the subject can voluntarily postpone it through the contraction of the external anal sphincter and pubo-rectal muscle.

When the distension of the rectum continues and conditions allow it, the person decides to defecate; this decision implies a sitting position with hip flexion that results in the disappearance of the angle between the anal canal and rectum. A complex defecatory mechanism begins that combines the voluntary control of defecation with the reflex of external anal sphincter, the relaxation of the pubo-rectal muscle, and the abdominal pushing.

The effects of defecation reflex are intrinsic and parasympathetic. The first occurs when the rectum is distended by feces; by myenteric plexus start afferent signals that generate a peristaltic wave that push the stool from the descending colon through the sigmoid colon and rectum to the anus. This reflex is, however, weak and to cause defecation must be reinforced by the reflection of the parasympathetic that involves numerous sacral spinal segments; stimulation of rectal afferent fibers gives rise to signals that reach the bone and then, via reflected through the erigendi nerves, the colon to the anus. These parasympathetic signals amplify peristaltic waves and transform the reflex of defecation in a powerful process to allow emptying of the descending colon, sigmoid colon, rectum, and anus. The afferent impulses arriving at the spinal cord give rise to other effects: deep breathing, closure of the glottis, and contraction of the abdominal muscles, to increase the abdominal pressure, which in turn increases the rectal pressure to exceed that produced by the EAS, pushes down the pelvic floor, raises the anus, and helps to push out the feces [22] (see Fig. 2.2).

For defecation to occur, the voluntary mechanism is indispensable as it inhibits the external anal sphincter because this normally contracts with the arrival of stool.

Bowel and Anorectal Changes in Pregnancy

Functional changes of the gastrointestinal tract are common in uncomplicated pregnancies. Heartburn, nausea and vomiting, abdominal bloating, constipation, hemorrhoids, anal fissures are most frequent clinical manifestations but also diarrhea, fecal incontinence, and irritable bowel syndrome can occur. Hormonal factors have the major influences on the bowel motility and on the pelvic floor musculature in the first and second trimester, while mechanical changes are associated with advancing gestation [23, 24].

Constipation

It has been documented that constipation is second only to nausea during pregnancy, with up to 40 % of women likely to suffer symptoms at some stage of their pregnancy. The cause of constipation seems to be multifactorial and dependent on hormonal effects, fetus and placenta growth, dietary changes, and decreased physical activity. Small and large bowel hypomotility occurs in pregnancy due to intestinal smooth muscle relaxation secondary to increased progesterone [25, 26].

Even somatostatin may inhibit the release of motilin, a peptide hormone that normally inhibits smooth muscle. Also a polypeptide called relaxin, which inhibits myometrial contraction during pregnancy, could inhibit smooth muscle of the gastrointestinal tract [27–29]. Furthermore, estrogen and progesterone activate the renin-angiotensin-aldosterone system that results in increased colonic water absorption. During the last trimester of pregnancy, the growing uterus and fetal movements can impede onward progression of solid feces, obstruct defecation, and initiate constipation [30].

Hemorrhoids

Hemorrhoids are another typical anorectal disorder during pregnancy and childbirth due to the same factors of constipation. Raising pressure on the superior rectal veins by gravid uterus increased circulating volume, and the effects of progesterone on the vascular system are the protagonists in etiology of hemorrhoids [31].

Anal Fissures

Anal fissures are caused mainly by constipation. Pain related to fissures exacerbates constipation and further promotes fissure formation [32].

Other Disorders

Pregnancy may exacerbate preexisting intestinal disorders like IBS, Hirschsprung's disease, idiopathic megacolon and megarectum, and diseases such as fecal incontinence that is typical of postpartum period (Fig. 2.6) [23].

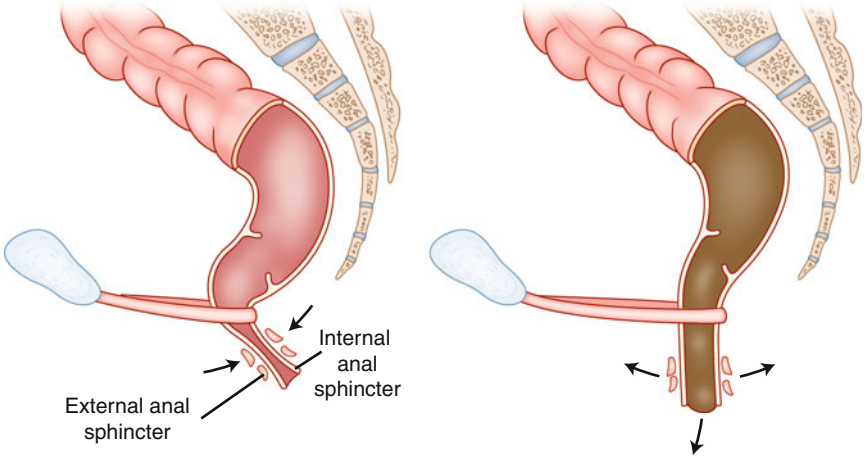

Fig. 2.6 Anorectum at resting and defecation

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Epidemiology of Childbirth Trauma and Associated Pelvic Floor Disorders

3

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Abstract

Pelvic floor disorders such as urinary incontinence, pelvic organ prolapse and anal incontinence affect women of all ages and are strongly associated with a significant economic burden and detriment to a woman's quality of life. Clinical and epidemiologic findings indicate that women who undergo vaginal childbirth are at an increased risk of developing pelvic floor disorders. Trauma to the pelvic floor in the process of vaginal childbirth is common, yet symptomatic development of pelvic floor disorders is difficult to predict. Imaging modalities have provided further information as to the mechanism of pelvic floor trauma, yet the effects of mode of delivery and other modifiable risk factors to implement secondary prevention methods need further investigation.

Keywords

Pelvic organ prolapse • Levator ani trauma • Urinary incontinence • Vaginal delivery • Anal incontinence • Childbirth trauma • Pelvic floor disorders

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Introduction

Pelvic floor disorders include urinary incontinence, pelvic organ prolapse and fecal incontinence. Pelvic floor disorders affect 24 % of US females [1]. In addition to having a strong association with aging, pelvic floor disorders are more prevalent in women who have delivered at least one child, and it is known that pelvic floor trauma commonly occurs at the time of the first vaginal delivery [2–5]. The continuing rising trend towards elective cesarean section [6] is due in part to a growing awareness of the potential deleterious effects of vaginal childbirth and future repercussions on the pelvic floor. Both patients and their doctors increasingly opt for cesarean delivery without maternal or neonatal indications, in part to avoid future morbidity such as urinary incontinence, pelvic organ prolapse or fecal incontinence, all of which have been associated with vaginal childbirth in epidemiologic studies [7, 8]. In addition, women may be at increased disposition to pelvic floor trauma due to inherent weakness in the collagen within the pelvic floor structures [9, 10].

Traditionally, this trauma was thought to involve the anal sphincter complex and the perineal body. Recently, there have been advances in imaging in the form of magnetic resonance imaging (MRI) and three-dimensional (3D) ultrasound, and the role of the levator ani muscle (LAM) as an important component of pelvic floor trauma has become evident. We currently have a better understanding that about 50 % of all women after vaginal delivery have a significant alteration of the pelvic floor anatomy affecting the levator ani muscle [2].

The Pelvic Floor in Childbirth: Risk Factors and Mechanisms

The levator ani muscle plays a major role in childbirth as it is the most substantial soft tissue structure defining the dimensions and biomechanical properties of the birth canal [11]. At the time of vaginal delivery, the birth canal undergoes substantial distension, varying between individuals by at least a factor of 5 [12, 13]. According to research on muscle physiology, skeletal muscle will not stretch to more than twice its length without some structural or macroscopic trauma [14]. Skeletal muscle studies have shown that in passive muscles, a stretch of 50 % is necessary to cause significant injury, whereas in maximally activated muscles a stretch of 30 % results in injury [14]. This finding may explain the suggested protective effects of epidural anesthesia from developing a LAM injury [2].

The association between vaginal parity and POP has been known to have a nonlinear effect, with the first vaginal delivery having its greatest impact as a risk factor for POP [15–19]. Several obstetrical factors have been associated with levator muscle injury after vaginal birth [2, 3, 20, 21]. Results from a small study by Miller et al. reported MRI findings of LA injury in 19 high risk postpartum women, showing 47 % to have LAM injury. However, current experience comes from small studies, or case control data with limitations in clinical application. Based on recent literature, women at highest risk for LAM injury have exposure to risk factors such as perineal injuries, prolonged second stage of labor, instrumented delivery, and

fetal head circumference >35.5 cm. Although these individual risk factors may be associated with LAM injury, little is known about the combination of factors, which increase the risk of LAM injury. While 10–30 % of women will undergo macroscopic LAM trauma, there is an even greater number that will undergo microtrauma, or irreversible distension of the levator hiatus [2–5]. Obstetric predictors of microtrauma may differ from those of levator "avulsion," which is the traumatic dislodgement of the LAM from its bony insertion.

The pelvic floor is a complex three-dimensional structure, with a variety of functional and anatomical areas. It consists of a musculotendinous sheet that spans the pelvic outlet and consists of paired levator ani muscle (LAM). It is broadly accepted that the LAM consists of subdivisions that have been characterized according the origin and insertion points, consisting of the pubococcygeal, puborectal and iliococcygeus portions [22]. The levator ani is further divided into the puboperinealis, pubovaginalis and puboanalis, according to its relationship to the surrounding viscera [22]. Lateral to the LAM is the puborectal division, which forms a sling around and behind the rectum, just cephalad to the external anal sphincter. Lastly, the iliococcygeus division forms a flat, horizontal shelf, spanning both pelvic side walls [23]. Recently, the use of 3D EVUS has been validated to visualize LAM subdivisions previously characterized by MRI studies [24]. These subdivisions were localized in cadaveric dissections, then correlated with images seen in nulliparous women, based on origin and insertion points and were shown to have excellent inter-observer reliability.

The pelvic floor muscles have the unique role of supporting the urogenital organs and the anorectum. Unlike most other skeletal muscles, the LAM maintains constant tone, except during voiding, defecation and a valsalva maneuver [25]. At rest, the LAM keeps the urogenital hiatus closed, by compressing the vagina, urethra and rectum against the pubic bone, and maintains the pelvic floor and pelvic organs in a cephalad direction [23]. Pelvic floor muscles are integral to pelvic organ support, and while functioning properly, provide support to the pelvic organs, keeping the ligament and fascial attachments tension-free.

Consequences of Levator Ani Trauma: The Implications of Childbirth

During parturition, the LAM stretches beyond its limits [12, 26] in order to allow passage of a term infant. Studies have shown that LAM injuries occur in 13–36 % of women who deliver vaginally [3, 27, 28]. There are various definitions of levator ani injury, according to mode of assessment and imaging modality. Assessment of the levator muscles is essential for a complete understanding of pelvic floor anatomy abnormalities, as well as of pelvic floor dysfunction.

Given that skeletal muscle will not stretch to more than twice its length without tearing [14], it is surprising that more women do not sustain LAM injuries. The degree of distension as well as the point of maximum strain of the tissue varies based on MRI-based models [12, 29].

There are numerous definitions of LAM injury depending on the mode of assessment, namely clinical palpation, ultrasonography, or MRI. It is widely believed that nulliparous women do not suffer from LAM injuries [30].

Childbirth and Prolapse

Pelvic organ prolapse is defined as descent of the uterus and vaginal walls into the vaginal canal. Most women have at least some degree of prolapse. Objective prolapse severity is weakly correlated with symptom burden [31–33]. The general trend in clinical studies is that prolapse becomes symptomatic when it descends beyond the hymen, and therefore the hymen represents a clinically significant threshold [33–35]. Women with symptomatic prolapse may experience a high degree of bother and substantial negative impact on physical function and quality of life [36]. Additionally, the public health impact of prolapse is substantial with respect to the incidence of surgery: the lifetime incidence of surgically managed pelvic organ prolapse is as high as 19 %, which is higher than previously estimated [37].

The research on the epidemiology of prolapse has been limited. The gold standard for "evidence-based medicine" is the randomized trial, but the ability to perform a randomized trial in this area is hampered by the long follow-up needed, since there is an inherent latency between obstetric exposures and clinically significant symptom development. As such, most studies have used surrogate markers for prolapse symptoms [38, 39] or surgical management [40, 41]. These surrogate measures may not be reliable and can lead to bias in the estimation of prevalence [19].

More recently, studies using a quantitative or graded approach to measuring prolapse suggest that prolapse is more common among parous compared to nulliparous women [42]. In addition, vaginal childbirth, particularly operative vaginal delivery, has been shown to increase the risk of pelvic organ descent to or beyond the hymen [19, 43]. As early as 6 months postpartum, stage 2 pelvic organ prolapse was noted in 18 % of primiparous spanish women delivered vaginally compared to 7 % of women who delivered by cesarean [44]. Similar findings were shown in a multicenter study from the United States [45]. Current evidence supports that the mode of delivery is a more critical component, rather than the process of labor itself. As such, no difference was seen in prevalence of prolapse in women who delivered by unlabored cesarean, compared to women who delivered by cesarean after active labor and complete cervical dilation [19].

Recently, the role of episiotomy and the development of pelvic organ prolapse have come into question. Episiotomy was first recommended in the 1930s as a means of preventing obstetric lacerations, and many argued that by protecting the mother's perineum this would result in better, pelvic organ support [46]. In 2005, a systematic review observed that the evidence does not support routine episiotomy as means of providing maternal benefit, and the role of episiotomy as it impacts the development of pelvic organ prolapse remains unknown [47].

The potential for an association between spontaneous lacerations and prolapse is suggested by recent literature involving vaginally parous women 5–10 years after

delivery. This study found that women who had more than one spontaneous laceration were more likely to have prolapse to or beyond the hymen [43]. In addition, no increase in pelvic organ prolapse was observed in association with episiotomy. Interestingly, there are recent data suggesting an association between mediolateral episiotomy as a protective factor against developing central support defects of the anterior vaginal wall, which is the most common site of prolapse [48]. The question of episiotomy vs spontaneous lacerations remains, with respect to the risk of prolapse; there is a clear need for future research in this area.

Childbirth and Urinary Incontinence

It is general assumption that urinary incontinence is a sign of a weak pelvic floor, but this assumption is not evidence-based.

Viktrup et al. interviewed 305 primiparous women and found that 39 % had stress incontinence before, during or after pregnancy, and 7 % developed de novo stress incontinence after delivery [10]. In a follow up of this cohort, up to 30 % reported stress incontinence 5 years later. Those without symptoms of urinary incontinence after their first delivery had an incidence of 19 % as compared with 92 % in those who had symptoms at 3 months postpartum [49]. In another prospective study of 949 women, urinary incontinence was experienced by 22 % of women before pregnancy, 65 % during the third trimester and 31 % after delivery [50]. New onset of urinary incontinence was found to be more common in parous as compared with nulliparous women. Even among those having cesarean section, postpartum urinary incontinence was independently associated with incontinence prior to and during pregnancy. Current evidence highlights the high proportion of women who suffer from urinary incontinence and confirm previous observations that prepregnancy and antenatal urinary incontinence increases the risk of future urinary incontinence [51].

There is conflict in the evidence regarding the relative contribution of different obstetric factors in the development of urinary incontinence. It is not clear whether it is pregnancy or the delivery itself, the major contributor to new onset urinary incontinence. Obstetric factors that have been investigated include the duration of the second stage of labor and birthweight [52]. Other investigators have not found a significant correlation between stress incontinence and fetal head circumference [53, 54], second stage of labor [50, 54], or birth weight [53, 55, 56]. In the largest community-based epidemiologic study of incontinence, involving 15,307 participants, (EPICONT study) women who were younger than 65 years, had not delivered or had cesareans or vaginal deliveries only reported that the prevalence of any urinary incontinence was 10 % in the nulliparous group [57]. There was a prevalence of 16 % in the cesarean group, and a prevalence of 21 % in the vaginal delivery group. This implies that pregnancy itself, rather than the process of delivery may also be an important causal factor in the development of urinary incontinence.

Urinary urgency or overactive bladder is reported by 27–45 % of women above the age of 40 [58]. In comparison to other pelvic floor disorders, the association

between overactive bladder and vaginal childbirth has not been well established. For instance, it is not completely clear whether overactive bladder is associated with greater parity since urgency incontinence is reported by similar proportion of both, women who delivered vaginally and nulliparous women [57]. It is also unclear whether overactive bladder is associated more with vaginal versus cesarean birth. For example, the odds of overactive bladder in women 5–10 years after childbirth do not appear to significantly differ in women after a vaginal versus cesarean birth [19]. Interestingly, operative vaginal birth, particularly forceps, may be associated with overactive bladder [19, 43].

Childbirth and Fecal Incontinence

Trauma and laceration of the anal sphincter complicates 2–16 % of vaginal deliveries [59, 60]. Injury to the anal sphincter complex, even those without recognition or repair, contributes to the development of anal incontinence. Several studies have demonstrated significant short-term risk of anal incontinence after exposure to anal sphincter laceration after vaginal childbirth [61, 62]. The prevalence of postnatal fecal incontinence symptoms was reported by a postal questionnaire by 906 women 10 months after delivery to be 4 %. Flatal incontinence was more commonly reported in about 29 % of women at 9 months after delivery in a study of 349 primiparous women [63]. Fecal incontinence is especially common after anal sphincter disruption, with a reported prevalence of 16–47 % [64–67].

Injury to the anal sphincter complex during childbirth is likely due to both, mechanical trauma and denervation injury. The latter injury may occur from traction and straining during the expulsive efforts associated with vaginal childbirth, similar to the mechanisms of nerve damage reported in patients with chronic constipation, which may result in anal incontinence [68]. The presence of neuropathy has been observed to be associated to the length of the second stage of labor, size of baby and instrumental delivery [69]. In studies involving 5 years of follow-up Snooks et al. observed that pudendal nerve terminal motor latencies (PNTML) as measures at the external sphincter were increased after childbirth [69, 70], indicating pudendal nerve damage. As compared to controls, PNTML was increased from 1.9 ms in control subjects to 2.2 ms in normal vaginal delivery and 2.4 ms in forceps delivery. Cesarean delivery appeared protective from such changes.

Several authors have utilized PNTML to investigate pelvic floor innervation, particularly as it applies to anal incontinence. In clinical application, PNTML has questionable clinical usefulness [71], but there is corroborating evidence that PNTML readings are prolonged after vaginal childbirth implying a degree of neuropathy in women [72]. There appears to be some recovery over the first 3 months postpartum, and virtually no change after that [73]. There are still a number of important questions to be answered, but there seems to be little doubt that vaginal childbirth can have significant negative effects on the pudendal nerve and its branches in some women.

The use of ultrasound has enabled the accurate visualization of the anal sphincter complex, revealing a high incidence of previously unrecognized occult anal

sphincter trauma after delivery. On ultrasound, morphologic alterations of the external anal sphincter can be observed in up to 38 % of deliveries [74, 75]. Subsequent studies have demonstrated that most "occult" lacerations can be identified clinically with appropriate training and diligent examination [76]. Therefore, true "occult" sphincter lacerations are probably rare. And, while the consequences of anal sphincter trauma are sometimes severe and clearly apparent, in most instances a primary surgical repair is effective and women remain asymptomatic. Fecal incontinence is probably of a multifactorial etiology. Recent research shows that vaginal delivery, in the absence of sphincter injury, does not appear to increase a woman's odds of anal incontinence [62, 77].

Conclusion

The development of pelvic floor disorders such as urinary and fecal incontinence and pelvic organ prolapse have been associated with vaginal childbirth. Over the last twenty years, history, imaging and physiology studies have revealed mechanisms of injury to the pelvic floor that include direct trauma, disruption to connective tissue and denervation trauma, all of which require a latency period before becoming clinically relevant. Recently, there has been an ongoing debate on whether cesarean delivery will provide a protective effect to reduce future pelvic floor disorders. Epidemiologic studies implicate the parity with urinary incontinence; however, the effect of mode of delivery is less clear. For example, while there is belief that cesarean section may be protective, other evidence suggests that pregnancy itself is the major risk factor. Elective cesarean delivery appears to be protective against the effects of mechanical sphincter disruption during vaginal childbirth, but not the urethral sphincter. When considering cesarean delivery, the benefits must be weighed against the potential morbidity to mother and child [78, 79]. We cannot currently be sure whether avoiding of potential intrapartum pelvic floor trauma of vaginal childbirth is worth the risk and costs of a cesarean section. In order to design and implement potential preventive strategies, future research must first make an effort at identifying the women at highest risk of potential pelvic floor damage.

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The Effect of Pregnancy on the Pelvic Floor

4

Cornelia Betschart and Josef Wisser

Abstract

Knowledge of physiological and anatomical changes during pregnancy is important for optimal prevention and care. Epidemiological and clinical observational studies have focused on bothersome patient symptoms and changes related to the pelvic floor during pregnancy.

Pelvic floor symptoms are common in pregnant women. These are mostly bladder related symptoms like higher micturition frequency, nocturia and stress urinary incontinence.

Quantitative assessments of the pelvic floor anatomy in pregnancy can be performed by clinical tools like the POP-Q or imaging like ultrasound and magnetic resonance technique.

Objectively, changes in the hiatal area, vaginal location, bladder neck position, anal sphincter muscle thickness and levator ani volume can be identified with ultrasound and MRI in pregnant women.

Keywords

Pregnancy • Pelvic floor muscle • Ligament laxity • Anal sphincter • Hiatus • Pelvic Organ Prolapse Quantification (POP-Q) • Pelvic floor symptoms • Ultrasound • MRI

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Physiological Changes of the Pelvic Floor During Pregnancy

Pregnancy exerts physiological alterations in virtually all organ systems. Significant alterations are described in the cardiovascular, gastrointestinal, haematological, endocrine and musculoskeletal systems [1]. The connective tissue of the ligaments and cartilage such as in symphysis pubis or ischiosacral junctions loosens. Hormonal changes are significant. The blood oestrogen levels rise to a hyperoestrogenic state, mostly by additive production by the placenta. Also other steroidal hormones including progesterone, dehydroepiandrosterone and cortisone undergo significant changes and hormones like the human chorionic gonadotropin, chorionic adrenocorticotropin, thyreotropin, relaxin growth hormone-variants and placental peptide hormones like neuropeptide-Y, inhibin, activin and atrial natriuretic peptide (ANP) arise.

During pregnancy the urogenital system undergoes anatomical and physiological changes [2]. Blood flow to the pelvic organs increases significantly, the bladder detrusor smooth muscle loses tone and hormones affect the biochemical composition of pelvic floor tissue. Pregnancy is known to significantly affect the instantaneous stiffness and relaxation behaviour of vaginal tissues in rat [3]. For pregnant human pelvic floor tissues accurate histomorphological findings are lacking.

Pelvic floor symptoms in pregnancy include increased urinary frequency, urgency and incontinence. These symptoms are aggravated in the third trimester as the fetal head engages in the pelvis [4]. Urinary retention may occur in the late first trimester with an estimated prevalence of 1 in 3000 to 1 in 8000 of pregnancies. It is mostly found in women with a retroverted uterus or large fibroids where the uterus exerts mechanical compression to the urethra. Often intermittent or continuous catheterisation is indicated until the uterus is large enough and cannot become incarcerated in the sacral hollow. Several reports of acute retention in pregnancy make special note of the fact that passage of a catheter in patients with retention is not difficult, suggesting that compression of the urethra per se may not be the cause of retention [5]. As urodynamically the bladder capacity increases during pregnancy it is assumed that in some women the high progesterone levels not only result to a weakness of the detrusor contractility but also to incapacity to relax the urethral sphincter [5].

During delivery, it is well established that pelvic floor muscle trauma and denervation occur [6–8]. These injuries are associated with stress urinary incontinence [9], defecation disorders [10] and prolapse [11] postpartum or become apparent decades later in life. Abnormalities are more often found in multiparae [6, 7], correlate with a prolonged second stage of labor [7, 12], forceps delivery [7] and high birthweight [6, 9]. These risk factors have cumulative effects. As pelvic organ prolapse is associated with parity in many clinical and epidemiological studies, it is unknown whether pregnancy, parturition, or a combination of these factors contributes to that. The influence of the hormonal changes during pregnancy on the pelvic floor is not easy to assess and up till now there are only few studies on the effects of pregnancy on the pelvic floor.

To elucidate the effect of pregnancy on the different parts of the pelvic floor, assessment of women before, during and after pregnancy would be required.

Unfortunately, there are significant practical problems in implementing such a study design.

Clinical Assessment of Pelvic Floor Changes

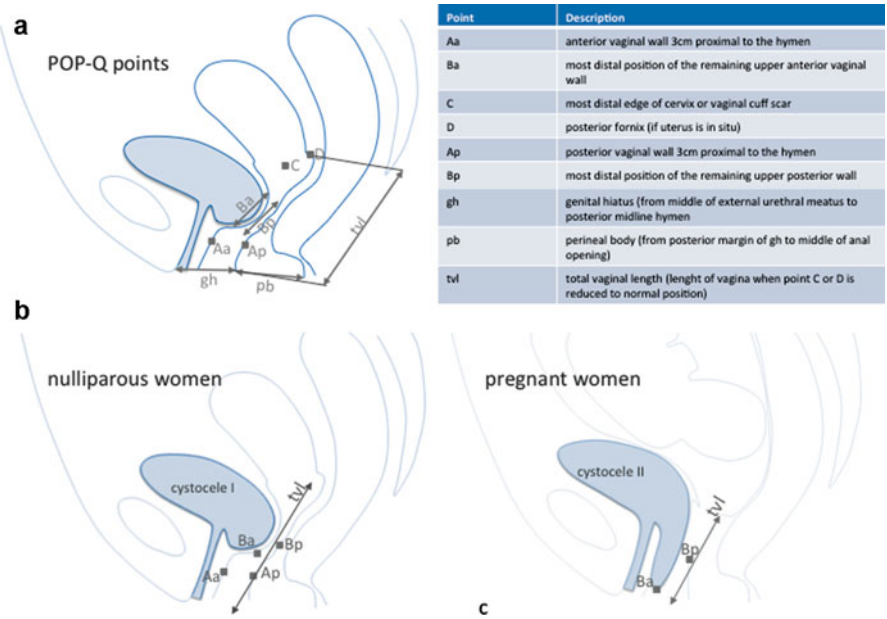
Pelvic floor changes during pregnancy can be assessed by measuring the compressing forces through palpation or vaginally placed balloon-type sensors. Invasive electromyographic studies performed in women with prolapse or after deliveries are not suitable for pregnant women, though surface electromyographic assessments are a feasible evaluation tool also in pregnancy. Clinical visual quantification of the location of pelvic organs can be undertaken using the Pelvic Organ Prolapse Quantification system (POP-Q). Imaging techniques like ultrasound or MRI provide high-resolution images of single structures for analysis or as basis for building 3D models. Those finite-element models containing multiple small pieces that maintain the same properties of the original model allow simulation of different stages of vaginal birth, like maximum levator stretch or also the simulation of prolapse. Until now, no pregnancy related finite models mirror the passive forces of uterine growth on the pelvic floor, vagina or bladder.

Pelvic Organ Prolapse Quantification System (POP-Q)

The change of location of the pelvic organs can be assessed with the Pelvic Organ Prolapse Quantifiction system (POP-Q) [13]. It allows assessment of the extent of movement of the anterior and posterior vaginal wall, the cervix or the vaginal vault between rest and strain, as well as interindividual comparisons (Fig. 4.1a). This semi-objective assessment in pregnant women revealed an increased rate of prolapse stages in the pregnant group compared with age-matched non-pregnant controls [14]. This was shown in a retrospective study of 21 pregnant women in the second or third trimester (average 27.8 weeks of pregnancy). POP-Q stages were significantly higher among pregnant subjects compared with control subjects, indicating a higher incidence of pelvic organ prolapse ($p < 0.001$) (Fig. 4.1b, c). POP-Q points that differed significantly between pregnant and nonpregnant subjects were points Aa, Ba representing the anterior vaginal wall, Ap, Bp for the posterior vaginal wall and the total vaginal length. Measurements at the cervix (point C), the genital hiatus length and the posterior fornix (point D) were not significantly different between the two groups. In the non-pregnant group maximal descent was POP-Q stage 1 in 57 %, whereas in the pregnant group 48 % of the women presented with a POP-Q stage 2. Point Aa, which corresponds to the urethrovesical angle, was significantly more relaxed in pregnant women (Fig. 4.1c).

Furthermore, as pregnancy progresses the prolapse stages become more pronounced [15]. O'Boyle et al. demonstrated that there is a significant difference between the first and third trimester for all anterior and posterior vaginal points in the POP-Q as well as for the total vaginal length and the genital hiatus, not so for the apical suspension (point C/D), though.

Apart from using the Pelvic Organ Prolapse Quantification (POP-Q) system, pelvic organ support can be assessed using relevant quality of life questionnaires. One prospective study looked at pelvic floor symptoms in the course of pregnancy and POP-Q



Point	Description
Aa	anterior vaginal wall 3cm proximal to the hymen
Ba	most distal position of the remaining upper anterior vaginal wall
C	most distal edge of cervix or vaginal cuff scar
D	posterior fornix (if uterus is in situ)
Ap	posterior vaginal wall 3cm proximal to the hymen
Bp	most distal position of the remaining upper posterior wall
gh	genital hiatus (from middle of external urethral meatus to posterior midline hymen
pb	perineal body (from posterior margin of gh to middle of anal opening)
tvl	total vaginal length (lenght of vagina when point C or D is reduced to normal position)

Fig. 4.1 (**a**) POP-Q points after Bump RC et al. [13]. (**b**) All patients in the nonpregnant group had a POP-Q stage of 0 or 1 (**c**) 47.6 % of the pregnant subjects had POP-Q stage 2 ($p < .001$). Point Ba (most distal position of the remaining upper anterior vaginal wall) and point Bp (most distal position of the remaining upper posterior vaginal wall) are significantly different

stages [16]. Quality of life was assessed with the electronic Personal Assessment Questionnaire-Pelvic Floor (ePAQ-PF). Bother with voiding difficulties and stress urinary incontinence increased during pregnancy. Constipation ($p=0.02$) and evacuation subdomains improved significantly ($p=0.009$) between the week 20 to week 36 of pregnancy. Fecal incontinence was not present in either trimester of pregnancy. In the sexual domain, the only subdomain that worsened significantly ($p=0.03$) was "sex and vaginal symptoms" in the course of pregnancy. None of the pelvic floor symptoms impacted the overall quality of life. Objective parameters like genital hiatus and perineal body length showed significantly higher values in the third trimester compared to the second trimester. In this study, other POP-Q points for the anterior or posterior vaginal wall as well as the cervix location did not differ between the second and third trimester.

Other Findings

More data on developmental histomorphological changes in pelvic floor muscles, ligaments, connective tissue and nerve supply in pregnant women would be beneficial. Biomechanical studies on biological tissues like the pelvic ligaments and vaginal tissue have been performed [17] in postmenopausal women with prolapse.

In 29 female cadaveric specimens a correlation between subjective evaluation and objective strength of ligaments was found [18]: the ileopectineal ligament was significantly stronger than the sacrospinous and the arcus tendineus fascia pelvis. But as the influence of hormones during pregnancy is remarkable, these results can not be translated to pregnant women.

At present there is a lack of data about *pregnancy associated* pelvic floor damage by contrast with an abundance of studies on intrapartum injuries and age-related changes contributing to the development of prolapse.

Outcome Assessment of Pelvic Floor Muscle Training (PFMT)

Pelvic floor muscle changes during pregnancy can be assessed through palpation of the pelvic floor muscles or vaginally or rectally placed pressure sensors. These measurements also allow to assess therapy outcome of pelvic floor muscle training, the only recommended therapy during pregnancy to prevent further pelvic floor disorders. The concept of pelvic floor muscle training (PFMT) in pregnancy to prevent urinary incontinence has been supported by recent studies, which showed that PFMT can prevent from urinary incontinence (UI) both during and in the immediate postpartum period [19–21]. In a recent Cochrane review, there was evidence of a statistically significant effect of PFMT during pregnancy on prevention of incontinence at 3 and up to 6 months after delivery [22]. Long-term follow up of participants up to 8 years after their initial randomization showed that 35.4 % of women in the PFMT group versus 38.8 % of women in the control group reported urinary incontinence [21]. Mørkved et al. reported in a conference abstract non-significant differences in the long-term outcome, whereby after 6 years the incontinence rate was higher in the group that had PFMT (urinary incontinence in 23 % of PFMT and 17 % of control women), however the sexual satisfaction was higher in women that had PFMT during pregnancy [19, 22].

According to the Cochrane Database of Systematic Reviews [22], PFMT is especially recommended for women with incontinence prior to pregnancy, women with a bladder neck hypermobility in early pregnancy, or postpartum for women that delivered a large baby and/or had a forceps delivery.

A recent study that assessed the outcome of meticulous PFMT with surface electromyography and quality of life questionnaire in pregnant and postpartum women, demonstrated a successful outcome in all three groups. The pelvic floor muscle contractility increased after the training program ($p = 0.0001$) in the early pregnancy group, in the postpartum group that delivered vaginally with an episiotomy and the postpartum group after an elective caesarean section. Decreases in the scores of both ICIQ-UI SF ($P = 0.009$) and ICIQ-OAB ($P = 0.0003$) were also observed after training in all three groups. One point that has to be investigated further is to evaluate if such effects are maintained after stopping training [23].

Imaging Techniques During Pregnancy

Imaging techniques are beneficial to measure changes of the pelvic floor in a non-invasive, reproducible way. Ultrasound and magnetic resonance imaging, as safe and volume based techniques, allow visualisation of structures and function in real time.

Ultrasound

2D Transperineal Ultrasound and Endovaginal 3D Ultrasound

2D ultrasound has a resolution of up to 0.1 mm. The probe held on the perineum in a sagittal position, allows to depict similarly the symphysis, urethra, bladder neck, bladder, vagina and rectum (Fig. 4.2).

Since the introduction of ultrasound in the early 1960s, safety issues were discussed. Ultrasound, physically longitudinal waves, have an influence on biological systems. Physical effects are thermal effects, as energy gets transformed to warmth/heat and second there is a pressure application that can lead to tissue deformation (cavitation). In the B-mode the power is very low (<10 mW/cm^2) and the duration of pulses are short (<1 μs). Temperature rises are not measurable for the B-mode ultrasound (US).

3D/4D Transperineal Ultrasound

3D-pelvic floor US generates a volume data set, which can be cut in sequential, orthogonal planes to study structures that are not accessible in the 2D-sonography. The advantage of applying 3D US is that reference planes orthogonal to non-linear structures like the vagina or anal canal can be set time-independently after the examination. Those examinations are also safe, as the applied power is far below the level of biological significance (<100 mW/cm^2). Worldwide there are no reports of harmful effects to the fetus through B-mode US which is also the basis for 3D/4D US.

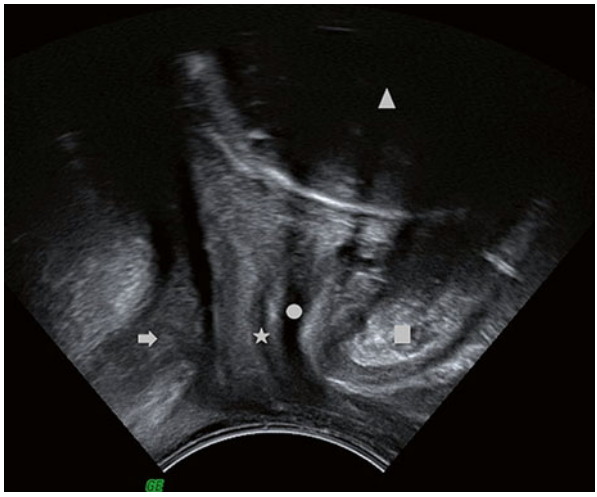


Fig. 4.2 Midsagittal B-mode view of the pelvic floor of a pregnant women in the third trimester. *Arrow* = rectum, *triangle* = fetal head, *asterisk* = vagina, *dot* = urethra (empty bladder), *rectangle* = symphysis

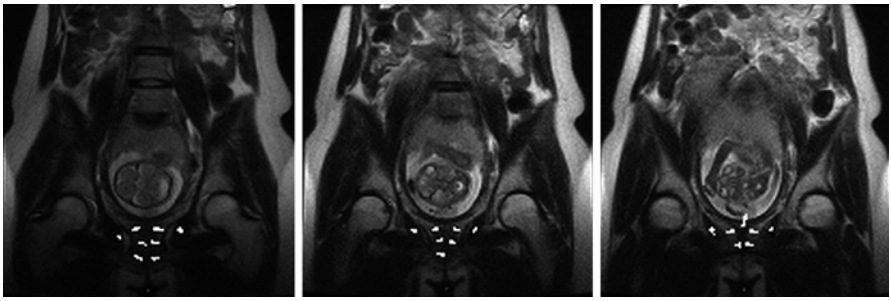


Fig. 4.3 Coronal MRI slides of a women in the second trimester; thickness of slices is 5 mm. The vertical course of the levator ani as thin muscle layer (*white arrow head*), the bulk of the obturator internus muscle (*asterisk*) and the internal cervical os in the third picture (*arrow*) are visible

Magnetic Resonance Imaging

Pelvic floor MRI usually performed in a 1.5 or 3 T superconducting magnet, uses proton density T2-weighted scans, 2-D fast-spin proton density with an echo time of 15 ms and a repetition time of 4000 ms, performed at 5-mm intervals in the axial, sagittal and coronal planes in the supine position. Common settings use a slice thickness of 4 mm with a gap of 1 mm. Like ultrasound, magnetic resonance has no harmful effects to the mother and child (Fig. 4.3). Disadvantages are the higher costs, the narrow space of the MR tube and the noise of the machine.

Pelvic Floor Muscle Anatomy and Function

The female pelvic floor is a complex fibro-muscular-ligamentous unit involved in multiple functions that go beyond the sole support of pelvic organs. Pelvic floor dysfunction globally affects micturition, defecation and sexual activity. Evolutionary modifications like upright walking and the need to deliver fetuses with larger head diameters made the fascial and muscle support of the pelvic floor vulnerable, therefore predisposing women to pelvic organ prolapse and incontinence. The female pelvic floor further undergoes a number of adaptive changes related to pregnancy and endocrine changes.

Animal Studies

Women are not the only upright species that develop pelvic floor disorders during life. Animal studies in primates simulate pregnancy and parturition-related changes. Primates like squirrel monkeys are also known to develop prolapse during their lifetime. To study the effects of pregnancy and parturition on the pelvic floor, MRIs of seven female squirrel monkeys were studied prior to pregnancy, 3 days, and 4 months postpartum [24]. No testing was performed during pregnancy to avoid harming the fetus. The bladder neck and cervix position were measured

dynamically with abdominal squeezing. The pelvic floor muscles are not completely alike in humans, one difference is a prominent coccygeus muscle. The volume of the coccygeus muscle was greater shortly after parturition than before pregnancy or after recovery. The bladder neck position in the relaxed state and with abdominal pressure descended ($p < 0.04$) after delivery and descended further ($p < 0.001$) after recovery. The same happened to the position of the cervix. It seems that parturition-related bladder neck descent in squirrel monkeys is permanent.

Levator Ani Changes in Humans

The appearance of the levator ani (LA) muscle in pregnancy was assessed in 3D-MRI in 84 post-term nulliparas (at 41 weeks of gestation) [25]. This study found a lower levator ani volume in post-term pregnant women of a mean volume of 13.5 ± 3.7 cm^3 than in nulligravida, where LA volumes vary between 32.3 and 46.6 cm^3 [26–29]. In this investigation on post-term pregnancies the LA muscles appear to be thinned. However, it has to be pointed out that this difference might be partly due to different acquisition techniques.

A 3D ultrasound study [30], aimed to predict delivery outcomes in relation to the levator hiatus area, assessed the LA of 61 nulliparous women between 36 and 40 weeks of pregnancy. The mean hiatal area at rest was 11.81 cm^2, at contraction 9.59 cm^2, and at Valsalva 16.03 cm^2. A correlation between levator dimensions and delivery mode could not be demonstrated. However, an inverse correlation was demonstrated between the area of the hiatus, particularly the one on pelvic floor contraction, and length of total second stage. Women with a smaller hiatal area on pelvic floor contraction, indicating a stronger pelvic floor muscle, had a longer second stage of labor [29].

Interestingly, fetal weight was not associated with LA volume. However, the fetal station was associated with a decreasing levator volume. The lower the fetal station/head, in centimeters to the levator ani, the thinner the LA muscle appeared. After adjusting for maternal BMI, this relationship disappeared.

A longitudinal study could answer the question whether the thinning is an effect of pregnancy or whether imaging has more artefacts in pregnancy due to baby movements or measuring technique.

It is not elucidated yet whether it is beneficial for the pelvic floor health in later life to have a strong and thick pelvic floor musculature before pregnancy, that might lead to a longer second stage of labor, which however, may be associated with a higher rate of pelvic floor trauma, or whether a primarily weaker pelvic floor with a larger hiatus and shorter parturition time has lower negative sequelae later in life.

A randomized controlled trial involving 200 women evaluated the possible prevention of pelvic floor disorders using stretching of pelvic floor muscles with the balloon device Epi-No® in late pregnancy. The rationale is based on sports physiology where an increased muscle extensibility might be obtained by intermittent stretching before the exertion. In this trial a non-significant reduction in levator ani muscle avulsion after training with Epi-No®, beginning at 37 weeks' gestation (6 % vs. 13 %) was found [31]. It is to mention that this study was insufficiently powered

with 200 patients included instead of the 660 women that would have been needed according to a power calculation that was aimed to show a 50 % reduction in the incidence of levator avulsion.

Biomechanical Models

Computational models have been demonstrated to be an effective tool in investigating the processes during the first and second stages of parturition. The pelvic floor and the associated structures are one of the most complex regions of the human body and undergo immense stretching in the course of pregnancy and even more during parturition.

Animal models have the inherent problem of the lack of upright position, which is an important factor in studying the pathophysiology and natural history of prolapse. Interdisciplinary collaborative research, involving bioengineers and clinicians, is essential to investigate and simulate the mechanical effects on the pelvic floor along gravity [32]. Geometrical information is gained from high-resolution images, mostly generated by MRI, and processed by segmentation into a finite element by a mathematical tool. The finite element method discretizes a continuous model into small pieces to investigate their mechanical behaviour under load or stress. This has been done by several authors simulating delivery [33–35] or prolapse [36, 37], though no biomechanical models simulating the changes during pregnancy are available yet.

Anatomical Changes of Ligaments

Landon et al. showed that fascia in pregnant women stretches to a much greater length but had less tensile strength compared with fascia from nonpregnant women [38]. The authors demonstrated that the collagen structure changes and the connective tensile strength decreases during pregnancy. The loosening of connective tissue in ligaments and fascia yields to common symptoms like symphysis pubic dysfunction and pelvic girdle pain. The levels of the hormone relaxin were found to be significantly higher in pregnant women that have bothersome joint pain and laxity [39]. Whether there is a relationship between pelvic girdle pain and specific pelvic floor disorders like prolapse is not elucidated yet.

Bladder and Functional Bladder Neck Anatomy during Pregnancy

Epidemiological studies show an increase in lower urinary tract and pelvic floor symptoms during pregnancy [40]. Pregnancy affects bladder function adversely. Urinary incontinence is seen more often in pregnant women than in matched controls and the mean prevalence of stress urinary incontinence (SUI) during pregnancy can be as high as 41 % (18.6–60 %) and increases with gestational age [41].

It is known from another study that urinary incontinence worsens steadily throughout pregnancy [42], which is dependent on changes of the urethrovesical angle [43].

One study examined the displacement of the vesical neck position during pregnancy [44]. The angle or the urethrovesical junction was measured at rest, during Valsalva and during coughing three times, namely at 12–16 weeks, at 28–32 weeks, and 36–38 weeks of pregnancy. The muscle compliance, as calculated during coughing and during Valsalva remained practically unchanged during the whole pregnancy in this study. The significant increase was noted on examination at 6 weeks postnatally, however also returned to normal values 6 months after childbirth. There was no hysteresis in the muscle activity found in this study during pregnancy that would have indicated a failure of tissue to follow the same course during relaxation as it did during distension. The hysteresis is thought to be the result of shifts in the geometrical structure of the fibers with respect to each other, and can be interpreted as a form of internal friction within the tissue.

Conversely, another match-controlled ultrasound study demonstrated higher anterior wall mobility for the bladder compartment in early pregnancy between 10 and 17 weeks' gestation than in non-pregnant controls. This mobility increased and showed a higher organ descent in late pregnancy (32–39 weeks) [45]. The apical (uterus) and posterior compartments (bowel) did not descend significantly in this study. This is in concordance with another functional study that assessed the rate of incontinence in early pregnancy. The numbers of incontinent women in the first trimester were significantly higher (20 %) than in the matched non-pregnant group (4 %) [46]. This finding agrees with the statistically significant change of the urethrovesical junction angle during coughing or Valsalva in the pregnant group.

A urodynamic study by Iosif et al. found that in continent and asymptomatic pregnant women, functional urethral length, maximal urethral pressure, and closure pressure increased by an average of 12 cm H_2O during pregnancy and returned to normal shortly after delivery [47]. In stress urinary incontinent women, the same authors reported lower function urethral lengths and closure pressures than in the continent pregnant controls [48]. The SUI group did not show the same increase in functional urethral length as the continent pregnant women [48].

Genital Hiatus

The urogenital hiatus varies remarkably among non-pregnant individuals [29]. The ratio between the smallest and highest numbers for the urogenital hiatus has been calculated to be 1.6 for width, 2.3 for length and 2.4 for the urogenital hiatus area. In 10 % there was an absence of visible insertion of the levator ani (LA) into the pubic bone. Women where the LA arises only from the arcus tendineus of the levator ani and not from the pubic bone, might have a larger hiatus. As this finding appeared in nulliparous women, it seems to be a variation in normal insertion and not a pathological finding.

It is yet unknown whether this primarily larger hiatus and the missing pubic insertion have consequences for birth-related injuries.

Recently, an observational study was designed to evaluate the potential effect of pregnancy on pelvic floor function [49]. 3D ultrasound volume data of 688 nulliparae assessed in the late third trimester and again 4 months postpartum were compared with data of 74 nulliparous nonpregnant volunteers. Outcome parameters were the hiatal dimension and the urethral mobility adjusted for BMI and age. The comparison of those two groups revealed a 27 % increase in hiatal area at rest and a 41 % increase on Valsalva comparing the non-pregnant group and the nulliparae of the late third trimester. About 70 % of this difference in hiatal dimension was observed when comparing nonpregnant controls with women 4 months after prelabor or 1st stage caesarean section. For the urethral mobility, similar results were found with the remark that this effect was irreversible postpartum, as the higher urethral mobility persisted and did not recover at 4 months postpartum. Another recent 3D/4D ultrasound study confirmed increased absolute values of hiatal dimensions and increased contractility and distensibility of the levator hiatus at 36 weeks' gestation compared to 12 weeks' gestation [50].

In twin pregnancies the hiatal measurements become higher than the ones measured in singleton pregnancies, with changes in coronal diameters reaching significance at rest and during contraction. The sagittal diameters in the course of pregnancy do not differ between twin and singleton mothers. The higher changes in the width of the hiatus suggest that pelvic support undergoes greater changes during twin pregnancy [51].

Anal Sphincter

The sonographic appearance of the anal sphincter in the first trimester of pregnancy [52] has been evaluated using perineal ultrasound with a 7.5-MHz transvaginal probe placed at the posterior fourchette without distending the anal sphincter. The ratio of the anterior and posterior internal anal sphincter (a/p-ratio) was compared in 14 nulliparous women in the first trimester with 26 women after elective caesarean section (Fig. 4.4). There are high interindividual differences in internal anal

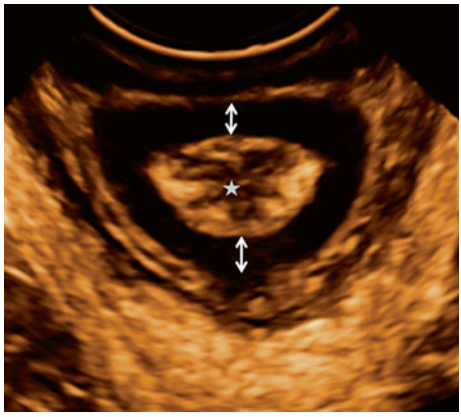


Fig. 4.4 3D ultrasound measurement of the thickness of the anterior and the posterior anal sphincter muscle in the reference plane. The a/p-ratio in this patient is 1.0 (*double-head arrow*). *Asterisk* = rectal mucosa

sphincter thickness. The anterior internal anal sphincter ranges from 1.0 to 3.0 mm in nulliparous pregnant women and 0.5 to 4.2 mm in post-caesarean women, and the posterior internal anal sphincter from 1.3 to 3.0 mm and 0.8 to 4.5 mm, respectively. The a/p-ratio was significantly higher in the nulliparous women (mean 1.03) than in the group after elective cesarean section (mean 0.88), $p < 0.01$). The two groups differ primarily in the exposure to the hormonal changes of the second half of pregnancy.

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Mode of Delivery and Perineal Trauma

5

Nivedita Gauthaman and Stergios K. Doumouchtsis

Abstract

Perineal trauma during childbirth is common. Although in the majority of cases perineal trauma does not have a major impact in the woman's future pelvic floor function, severe degrees of childbirth trauma such as levator ani trauma and obstetric anal sphincter injuries (OASIS) may result in significant pelvic floor dysfunction including urinary and faecal incontinence. Levator ani muscle trauma is diagnosed in one third of women who have vaginal birth. Levator trauma can lead to weakening of the pelvic floor muscles, widening of the urogenital hiatus and pelvic organ prolapse in the future. In this chapter, the biomechanics of the second stage of labour and its effects on the pelvic floor and levator ani complex are discussed along with the effects of prolonged second stage on the pelvic floor. Different modes of delivery including instrumental deliveries have a different impact on the risk of perineal trauma and levator ani complex. The effect of fetal malposition and malpresentation and multiple pregnancies on perineal trauma as well as the role of episiotomy are also discussed in this chapter.

Keywords

Obstetric anal sphincter injuries (OASIS) • Biomechanics • Second stage of labour • Levator ani trauma • Perineal injury • Forceps delivery • Ventouse delivery • Episiotomy • Malpresentation • Malposition

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Introduction

The second stage of labour and various types of vaginal birth have been associated with a variable impact on risks of pelvic floor trauma. Severe degrees of perineal trauma, particularly OASIS and levator ani trauma, can have long-term consequences such as faecal and urinary incontinence, pelvic floor dysfunction and pelvic organ prolapse in the future. Our understanding of the biomechanics of the second stage of labour has improved in recent years with the advent of dynamic imaging modalities and research based on modelling techniques in an effort to simulate vaginal childbirth and its effects on the pelvic floor muscles and the perineum. Fetal malpositions, malpresentations, multiple pregnancies, instrumental deliveries and the use of episiotomy have been identified in several studies as factors affecting the risks of pelvic floor trauma.

The Second Stage of Labour and Its Effects on the Pelvic Floor

Evidence on the effects of the second stage of labour per se on the pelvic floor prior to the birth of the fetus is limited. Studies to evaluate the impact of the second stage on the pelvic floor are challenging due to technical reasons and possible ethical concerns. With biomechanical modeling, changes on tissues of the pelvic floor have been studied to some extent. These are part of various changes to the pelvic floor during the different stages of labour. During the first stage there is increasing strength and frequency of uterine smooth muscle contractions, progressive dilatation of the cervix and descent of the fetal presenting part into the pelvis. The second stage of labour commences after the cervix is fully dilated and there is further descent of the fetal presenting part. This stage is complete when expulsion of the fetus from the introitus takes place with maternal voluntary efforts. There is progressive increase in the intrauterine pressures during uterine contractions and voluntary pushing efforts and an additional increase in intrauterine pressures has been estimated with ventouse traction of 113 N [1] and forceps traction of 200 N [2].

During the second stage of labour the descent of the fetal head causes progressive distension of the perineum and anal dilatation. The stage of fetal crowning occurs when the vulvovaginal opening is persistently dilated by the fetal head, the biparietal diameter has passed through the level of the ischial spines and there is no retraction of the fetal head between contractions. The perineum is thinned out and may undergo spontaneous tearing in primigravidae. This is less common in multiparous women. Episiotomy is usually performed at the stage of crowning to facilitate delivery with less risk of anterior perineal trauma.

Levator Ani Trauma and the Second Stage of Labour

The levator ani muscle complex consists of five parts and is responsible for closing the urogenital hiatus against the opening forces exerted by intraabdominal pressure. The muscle is distributed in a U-shape around the urethra, vagina and rectum, and the force

caused during the muscle contraction compresses the rectum, vagina and urethra, from back to front. Using 3D/4D ultrasound imaging and dynamic MRI, detachment of the puborectalis muscle from its insertion to the inferior ramus of the pubic bone can be visualised and is noted in one third of all women having a normal vaginal birth [3]. However, 85–90 % of primiparous women can have a vaginal birth without disruption of the pubovisceral muscles, a result of compensatory stretching of the perineal body. This has been called the "fusible link hypothesis" [4]. Geometric models have suggested that muscle damage in second stage may be due to overstretching. The pubococcygeal/pubovisceral muscle, which is the most medial in the levator ani complex sustains the largest tissue strain with a stretch ratio of 3.26 [3]. MRI studies of simulated vaginal childbirth have also demonstrated the maximum stretch ratio to be 3.5 corroborating similar findings with the geometric models [4]. The maximum stretch ratio is usually achieved at the time of crowning of the fetal head, therefore factors which increase the stretch ratio such as instrumental deliveries and increased head circumference of the fetus or malposition would further increase the risk of levator ani trauma.

The pudendal nerve is also at risk of injury during vaginal delivery. It is located relatively superficially in the pelvis and is therefore prone to stretch damage during childbirth. Stretch injury of the pudendal nerve has been reported in 38–42 % of vaginal deliveries [5]. Although the nerve injury is usually reversible due to re-innervation, severe injuries or total transection may result in protracted restoration of nerve function as shown in simulated childbirth using rat models [6]. Pudendal nerve trauma during childbirth has been implicated in postpartum faecal and urinary incontinence and is usually attributed to increased stretch and pressure injury during the second stage of labour.

Prolonged Second Stage of Labour and the Pelvic Floor

The American College of Obstetricians and Gynecologists defines prolonged second stage of labour as longer than 3 h in a nulliparous woman with regional anaesthesia and 2 h without regional anaesthesia [7]. Prolonged second stage of labour can be associated with malpositions, fetal macrosomia and relative cephalopelvic disproportion. A combination of raised intrauterine pressure in the second stage of labour due to intense contractions and maternal voluntary pushing efforts can lead to ischaemic injury of the pelvic nerves and muscles. This can lead to denervation injuries, which in some cases can be permanent, particularly if the active phase of the second stage of labour is prolonged. A neurophysiological study using concentric needle electromyography (EMG), pudendal nerve conduction tests and perineometer in 96 nulliparous women showed EMG evidence of re-innervation after vaginal delivery in 80 % of cases. Prolonged active second stage of labour (>1 h in primiparous women) and heavier babies showed the most significant EMG evidence of nerve damage, whereas passive second stage of labour did not increase the risk of denervation injury to the pelvic floor [8]. An MRI study in women 9–12 months after the first delivery showed that the use of forceps, anal sphincter rupture and episiotomy were associated with increased risk of levator defects. Women with levator injury were also found to have a 78 min longer second stage of labour in this study [9].

Regional Anaesthesia and Pelvic Floor Trauma

The relationship between epidural analgesia and perineal trauma during childbirth is not clear. It can be inferred that epidural analgesia may be associated with an increase in the rate of severe perineal trauma indirectly due to the increased likelihood of operative vaginal delivery and episiotomy associated with instrumental delivery. However, it can also be postulated that epidural analgesia in labour can exert a protective effect against severe perineal trauma by reducing the uncontrollable urge to push during delivery. To evaluate the independent risk of epidural analgesia in labour, logistic regression analysis was used in a study and epidural was not found to be an independent predictor of perineal injury [10]. A large population based study has shown that epidural analgesia in labour does not increase the risk of perineal trauma [11].

The effect of epidural analgesia on levator trauma is not clear. As epidural analgesia increases the duration of second stage with higher risk of instrumental delivery particularly forceps, it can potentially lead to levator ani injury and microtrauma. Prolonged pushing in second stage can lead to neuromuscular or vascular injury due to distension and stretching and longer second stage of labour has been associated with levator trauma [9]. However, intrapartum epidural may be beneficial by preventing premature pushing and may exert a protective effect on levator trauma by muscle relaxation of the pelvic floor with an effective epidural analgesia [12].

Spontaneous Vaginal Delivery and Perineal Trauma

It is estimated that 85 % of women who deliver vaginally will suffer some degree of perineal trauma in the UK. The incidence of OASIS varies between 1.7 and 18 % [13–15]. Variations are related to the standards of reporting and training to recognise such tears and the methods of identifying OASIS. Various other factors, such as instrumental deliveries, use of episiotomy and parity, also affect the incidence of OASIS, hence the variation in the rates. Gurol-Urganci et al. reported that the rate of anal sphincter injuries tripled in England between 2000 and 2012 (1.8–5.9 %). Improved recognition of these tears, standardisation of the classification of perineal trauma and the decline in the use of routine episiotomy probably contributed to the increasing incidence of OASIS [16].

Episiotomy is one of the commonest procedures performed in labour. Albeit a fairly common practice, there is no robust scientific evidence in support of performing episiotomies and it seems that it has just crept into clinical practice. Episiotomy is usually performed to increase the vaginal orifice shortly prior to the delivery of the presenting part. It is also performed by obstetricians prior to instrumental delivery particularly forceps, breech vaginal deliveries and in deliveries where shoulder dystocia is anticipated such as fetal macrosomia. Episiotomy is also given for indications such as rigid and inflexible perineum particularly in primigravidae, to expedite vaginal delivery in cases of fetal distress during the second stage of labour, and to avoid multiple vaginal tears by performing a controlled surgical incision.

A Cochrane review has recommended the practice of restrictive episiotomy as it has the benefit of reducing severe perineal trauma and posterior perineal trauma although there was an increased risk of anterior perineal trauma [17]. This review included 8 studies with a total of 5541 women. In the routine episiotomy group, 75.15 % of women actually had episiotomies. In the group with restrictive episiotomy, 28.40 % had episiotomy. The restrictive episiotomy group had shown less risk of severe perineal trauma (RR 0.67, 95 % CI 0.49–0.91), less need for suturing (RR 0.71, 95 % CI 0.61–0.81) and lesser complications with wound healing (RR 0.69, 95 % CI 0.56–0.85). The use of restrictive episiotomy was associated with a higher incidence of anterior perineal trauma (RR 1.84, 95 % CI 1.61–2.10). This review did not recommend a specific type of episiotomy (midline versus mediolateral) and left the choice of type of episiotomy with the accoucheur due to lack of evidence.

Intrauterine Fetal Demise and Perineal Trauma

Women delivering vaginally following intrauterine fetal demise appear to have a lower overall risk of perineal trauma compared to women with a live birth in a retrospective case matched study. This study included 323 women who delivered vaginally following intrauterine fetal death and was controlled for age, parity, gestational age and birth weight and excluded other significant factors, which contribute to perineal trauma such as instrumental delivery and episiotomy. The study concluded that women with intrauterine fetal death had a lower risk of perineal trauma (RR 0.16, 95 % CI 0.12–0.22) as well as lower risk of OASIS (RR 0.12, 95 % CI 0.03–0.50), which may be due to differences in biomechanics of childbirth in cases of intrauterine fetal demise [18].

Multiple Births and Perineal Trauma

Twin vaginal births generally tend to happen in earlier gestations than singleton vaginal births. It can be surmised that the lower birth weight and head circumference of twins may cause less perineal trauma than their singleton counterparts. Data looking at perineal trauma in twin pregnancies are limited. A recent retrospective cohort study [19] comparing twin vaginal deliveries (1538) and singleton vaginal deliveries (91,312) in a single tertiary unit identified nulliparity (twins adjusted OR 5.9, 95 % CI 1.7–20.9; singletons adjusted OR 3.9, 95 % CI 3.5–4.4), occipitoposterior position (twins adjusted OR 3.00, 95 % CI 1.1–8.0; singletons adjusted OR 1.6, 95 % CI 1.3–2.00), instrumental delivery (twins adjusted OR 4.3, 95 % CI 1.2–15.4, singletons adjusted OR 2.4, 95 % CI 2.2–2.6) and birth weight (twin adjusted OR 1.1, 95 % CI 1.0–1.2; singletons adjusted OR 1.07, 95 % CI 1.06–1.08) to be independent risk factors for OASIS both in vaginal twin and singleton deliveries. The authors also concluded that no single risk factor posed a higher risk in twins than in singleton pregnancy. The OASIS rate in twin vaginal deliveries (1.27 %) was approximately half than in singleton deliveries (2.55 %) in this study. This is not

different to the risk factors which have already been identified to increase the risk of perineal trauma in singleton vaginal births however it is still clinically relevant in counseling women with twin pregnancies prior to making decisions about mode of delivery.

Instrumental Delivery and Perineal Trauma

Ventouse and Forceps Delivery

Instrumental delivery either forceps or ventouse aims to expedite delivery of the fetus in the second stage of labour for various indications and accounts for 11 % of vaginal births in the UK [20]. Maternal indications include maternal exhaustion, prolonged second stage of labour and medical conditions such as pre-eclampsia, placental abruption, acquired or congenital heart disease. The most common fetal indication is fetal distress in the second stage of labour.

The choice of the instrument depends on operator's preference and expertise, local variations in practice, clinical indications and type of maternal analgesia. Other factors which influence choice of instrument include fetal position, fetal station and availability of instruments. There are advantages and disadvantages in the use of ventouse versus forceps.

There are various types of ventouse available, namely soft cup (silastic) – usually recommended for occipitoanterior positions and anticipated easy delivery, semi-rigid cups – made from flexible plastic and more effective with higher success rates than soft cups, handheld disposable ventouse cups (Kiwi Omnicup®) – used for rotational and non-rotational ventouse delivery, rigid metal cups – anterior and posterior cups – useful for both rotational and non-rotational ventouse delivery. Common cups used are the Bird's and the 'malmstrom' cups. Ventouse delivery has a number of advantages over forceps delivery as it is known to have less potential for trauma to maternal tissues compared to forceps delivery, can be performed without need for regional anaesthesia and episiotomy is not always required. The disadvantages of ventouse are that it cannot be used in preterm fetuses, it is contraindicated in face presentation, has higher failure rates leading to the use of sequential instruments and higher risk of injuries to fetal scalp and cephalhaematoma [21].

A Cochrane review [21] supports the use of ventouse as first line when the procedure is expected to be easy (occipitoanterior position, no cephalopelvic disproportion). Soft ventouse cup is recommended rather than the metallic cup due to less scalp injury (9 studies with 1517 women; RR 0.67, 95 % CI 0.53–0.86) and cephalhaematoma (6 studies with 669 women; RR 0.61, 95 % CI 0.39–0.95). However ventouse is more likely to fail to achieve a vaginal birth compared to forceps which increases the chances of sequential use of instruments, usually forceps, to complete the delivery and attendant risks to the mother and the fetus. The review also supported that forceps were more likely to be successful in achieving vaginal birth in studies including 2419 women (RR 0.65, 95 % CI 0.45–0.94).

There are various types of forceps available. The commonest ones used in the UK are Anderson's and Neville Barnes forceps. These are typically used for mid-cavity and low forceps deliveries. Wrigley's forceps are less commonly used and predominantly for outlet deliveries. There are no randomised controlled trials available to assess the efficacy of one type of forceps over the other and the choice depends on the operators familiarity and preference. Forceps are preferred over ventouse in prematurity (<36 weeks) and malpositions such as face presentation where ventouse is contraindicated, as well as for delivery of the aftercoming head in breech presentations where ventouse delivery is not applicable. Forceps are generally more successful in achieving vaginal birth over ventouse due to higher force applied but require training and expertise in assessing the correct fetal position and station. Forceps are generally associated with increased trauma to the maternal tissues and need for episiotomy.

Forceps are associated with significant maternal trauma such as episiotomy, third or fourth degree tears with or without episiotomy (RR 1.89, 95 % CI 1.51–2.37), vulval and vaginal trauma (RR 2.48, 95 % CI 1.59–3.87) and higher incidence of facial injury of the fetus (RR 5.10, 95 % CI 1.12–23.25) compared to ventouse delivery [21].

Rotational (Kielland's) forceps are used to achieve rotation of the fetal head from the occipito transverse or occipitoposterior position at the midcavity level of the pelvis. Ventouse can be used in these situations as well, however is fraught with increased risks of failure. Over the past 20 years the rising trend in the use of ventouse, fear of complications and subsequent litigation [22, 23], lack of experience with modern obstetric training and increasing obstetrician's preference to resort to caesarean sections in second stage [24–27] has led to a decline in the use of rotational forceps. As rotation of the fetal head occurs at the midcavity level, the risk of perineal trauma should not be significantly increased over other types of forceps deliveries. Recent publications [28–30] have shown comparable risks of OASIS with the use of Kielland's forceps compared to other non –rotational forceps/rotational ventouse deliveries. Rotational instrumental deliveries have a higher risk of injuries to the levator muscle complex [31]. Levator ani avulsion has been linked to higher risk of female pelvic organ prolapse [32]. Currently there are no techniques adapted to repair levator avulsion at the time of delivery, nonetheless this type of injury is often not recognised at the time of delivery.

Sequential Instruments

The use of sequential instruments usually involves completion of a vaginal delivery by forceps when a primary application of ventouse has failed. The main concern with the use of sequential instruments is the risk of neonatal morbidity such as retinal haemorrhage, intracranial haemorrhage and feeding difficulty compared to primary forceps deliveries as shown in a large retrospective study [33]. In another retrospective study comparing successful ventouse deliveries with failed ventouse deliveries either completed by forceps or second stage caesarean sections, the use of

sequential forceps was found to increase the risk of OASIS significantly, although neonatal outcomes were comparable [34]. However in choosing sequential instruments considerable thought should be given to achieving a safe vaginal birth with minimum risks to the mother and fetus. The Royal College of Obstetricians and Gynaecologists recommends that this should be carefully considered versus the potential risks of a second-stage caesarean section [35].

In summary, although forceps appear to be more effective in achieving a vaginal birth thereby avoiding a second stage caesarean section there is risk of significant perineal trauma to the mother. Ventouse, particularly use of metal cups increases the risk of cephalhaematoma albeit with significantly less maternal perineal trauma. Soft ventouse cups have lower risks of cephalhaematoma and less perineal trauma but increased risks of failure to achieve a vaginal birth and subsequent use of a forceps with added maternal perineal trauma.

Episiotomy and Its Role in Instrumental Deliveries

Although the intention of performing an episiotomy is to facilitate delivery of the presenting part and to avoid extensive tears in the perineum by performing a controlled surgical cut, there is still controversy as to the benefit of episiotomy in preventing severe perineal tears. Midline episiotomies are generally thought to increase the risk of OASIS when compared to mediolateral episiotomies [36, 37].

The role of episiotomy in the prevention of OASIS in instrumental deliveries is debatable. A large retrospective population based study [38] including 284,783 vaginal deliveries obtained from the Dutch National Obstetric Database, showed an overall risk of third degree tears of 1.94 %. The study concluded that mediolateral episiotomy strongly protected against damage to the anal sphincter complex during delivery (OR 0.21, 95 % CI 0.20–0.23). Another population based retrospective observational study [39] of 21,254 women delivered by ventouse and 7478 women delivered by forceps showed that mediolateral episiotomy significantly protected against OASIS in both the ventouse group (OR 0.11, 95 % CI 0.09–0.13) and forceps delivery (OR 0.08, 95 % CI 0.07–0.11). However other studies did not support these findings. A retrospective cohort study including 33,842 vaginal births [40] showed that operative vaginal delivery particularly in combination with midline episiotomy was associated with an increased risk of OASIS in primi and multigravidae (nullipara OR 4.5, 95 % CI 3.7–5.4; multipara OR 14.6, 95 % CI 10.4–20.5). A prospective non randomised study in the UK [41] comparing the use and non-use of episiotomy for all operative vaginal deliveries showed that episiotomy did not reduce or greatly increase OASIS (9.9 % versus 7.1 %, adjusted OR 1.11, 95 % CI 0.66–1.87). A pilot randomised controlled trial conducted in two maternity units in the UK [42] involving nulliparous women did not show any conclusive evidence of the protective effect of routine episiotomy over restrictive episiotomy against anal sphincter injuries (OR 0.72, 95 % CI 0.28–1.87) in women who underwent operative vaginal deliveries. To date there is no conclusive evidence basis for routine use of episiotomy to avoid third/fourth degree tears with instrumental deliveries and operator judgement in the use of episiotomy is recommended by the RCOG [35].

Malpositions, Malpresentations and Perineal Trauma

Occipitoposterior position is the commonest malposition in labour and is diagnosed in 5 % of deliveries. It is due to deflexion of the fetal head either due to mechanical factors such as flat pelvis or due to weak uterine contractions leading to inadequate flexion of the fetal head. Occipitoposterior position is associated with primigravida, prolonged labour and epidural analgesia and often increases the risks of instrumental delivery which is also known risk factor for OASIS. Persistent occipitoposterior position has been associated with a sevenfold increase in the risk of OASIS [43]. Increased levator ani stretch can be attributed to occipitoposterior positions due to the higher diameter of the presenting part, however this association needs to be evaluated further.

Breech presentation is the commonest malpresentation and occurs in 3–4 % of term pregnancies. In modern obstetric practice caesarean section is increasingly performed for breech presentations, although there is a role for breech vaginal delivery particularly in multiparous women and favourable breech presentations such as flexed breech and frank breech with an average sized fetus. There are no studies which have specifically looked at perineal trauma in breech vaginal deliveries. Episiotomy would be preferred in breech vaginal deliveries in order to increase the outlet diameter and also to enable use of various manoeuvres for assisted vaginal breech delivery. Forceps has been used to facilitate the delivery of the aftercoming head in vaginal breech deliveries. The effect of episiotomy and the use of forceps for the aftercoming head may increase the risk of perineal trauma and OASIS but there are no published studies to confirm this. A case of the fetal foot causing isolated rectal tears in frank breech delivery with intact anal sphincters has been reported [44].

Shoulder Dystocia and Perineal Trauma

Shoulder dystocia (SD) is an obstetric emergency which is defined as failure to deliver the anterior, posterior or both shoulders of the fetus requiring additional manoeuvres to achieve delivery. It occurs when the shoulder of the fetus cannot pass below the pubic symphysis causing delay in internal rotation, fetal descent and delivery. It can be associated with serious neonatal morbidity and mortality such as brachial plexus injury, birth asphyxia and upper limb, clavicular and rib fractures. Maternal complications usually relate to perineal trauma particularly obstetric anal sphincter injuries (OASIS). There is limited evidence of associations between specific manoeuvres for shoulder dystocia and risk of OASIS.

In a recent retrospective study [45] which included cases of shoulder dystocia over a period of 5 years in a tertiary teaching unit, SD was associated with a threefold increase in the risk of OASIS. The use of internal manoeuvres (OR 2.182: 95% CI 1.173-4.059), increased number of manoeuvres ≥4 (OR 4.667: 95% CI 1.846-11.795), Wood's screw manoeuvre (OR 3.096: 95% CI 1.554-6.169), reverse

Woods' screw manoeuvre (OR 4.848: 95% CI 1.647-14.277) and removal of the posterior arm (OR 2.222: 95% CI 1.117-4.421) were all associated with a significant increase in the likelihood of OASIS. The authors concluded that to effectively manage shoulder dystocia with consideration of perineal trauma, these factors need to be considered in designing further prospective studies and developing management protocols for shoulder dystocia in the future.

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Episiotomy

6

Vladimir Kalis, Zdenek Rusavy, and Matija Prka

Abstract

Episiotomy is a surgical incision to the perineum made during the last part of labour to facilitate delivery. It should always be defined by the location of the beginning, direction, length, and timing. Seven episiotomy types have been identified. However, only three (midline, mediolateral, and lateral) are routinely used. Exact placement of episiotomy incision is important regarding perineal trauma. Lateralisation of episiotomies significantly decreased OASIS incidence. While midline episiotomy increases the risk of OASIS, the protective role of mediolateral episiotomy depends on the correct identification of the risk group and correct incision. A protective effect of lateral episiotomy on primiparous women has been consistently demonstrated. Mediolateral episiotomy at an angle of at least 60° from the midline or lateral episiotomy are recommended. A restrictive policy regarding episiotomy is recommended: <30 % in total, <50 % for primiparas, <10 % in multiparas. Episiotomy is clearly indicated for fetal compromise, and, consensually, instrumental deliveries. Perineal mapping is helpful in deciding whether episiotomy might be useful. A qualified approach to the protection of the perineum should be applied to all deliveries including those with episiotomy. A continuous non-locking suturing technique for all layers using fast-absorbing synthetic material is currently the recommended standard for episiotomy repair.

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Keywords
Episiotomy • Definition • Classification • Midline episiotomy • Mediolateral episiotomy • Lateral episiotomy • Perineal trauma • Healing • Pain • Sexuality • Dyspareunia • Incontinence • Anal incontinence

Historical Landmarks

Episiotomy is globally the second most common surgical procedure after umbilical cord ligation (also obstetrical) [1]. It was first described more than 270 years ago [2]. During the eighteenth and nineteenth centuries this procedure was rarely applied [3] until 1921 when De Lee advised using mediolateral episiotomy during forceps deliveries [4]. The labour was seen to be a disease producing, and therefore, "decidedly pathologic process." Historically, physicians were trained to intervene in to a disease process, including protecting the mother from the morbidity of childbirth [5]. Coincidently, at that time, a movement from home to hospital delivery was taking place, with a significant increase in all obstetrical operations. As a result of these factors, an increase in the use of episiotomy was registered over the following years. Another turning point occurred in 1982 when Banta and Thacker contested the well-established opinion that routinely performed episiotomy reduces maternal and neonatal morbidity [6, 7]. Their findings had a great impact on the global scientific community. This can clearly be seen in the continuous growth in the number of articles published annually from 1983 until the present day, by searching for the keyword "episiotomy" in the PubMed database [8] (Fig. 6.1). The result was the overthrow of routine episiotomy and the introduction of a more restrictive use of episiotomy in everyday obstetric practice.

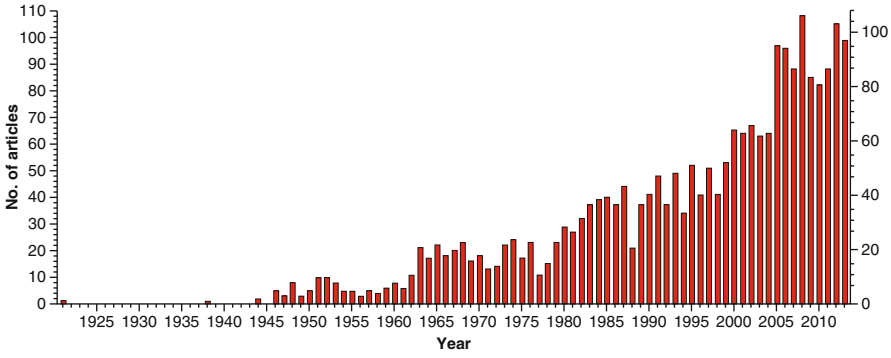

Fig. 6.1 Number of published articles per year (1921–2013) searching for the keyword *episiotomy* in the PubMed database [8]

Definition, Classification, Types of Episiotomy

Episiotomy is a surgical enlargement of the vaginal orifice by an incision to the perineum during the last part of the second stage of labor or delivery [7, 9]. It should always be defined by the following combination of parameters; the location of the beginning of the incision, the direction, the length, and the exact timing of the procedure. A recent analysis [10] revealed that the standard research texts usually describe only two main types of episiotomy (midline and mediolateral) [9, 11–13]. The term lateral episiotomy has only lately started to be re-used [14–19]. Therefore, a classification system of episiotomies [10] is important in order to improve the quality of methodology in future research and to facilitate the comparison of different studies.

A thorough analysis of the literature [10] revealed seven main types of episiotomy: midline, modified median, J-shaped, mediolateral, lateral, radical lateral, and anterior (Fig. 6.2).

Midline (median, medial) **episiotomy** begins at the fourchette and extends to a half of the length of the perineal body [10, 11, 20].

Modified median episiotomy differs from the previous type by two transverse cuts in opposite directions slightly anteriorly of the expected margins of the external anal sphincter (EAS) [21]. Transversely, only subcutaneous tissues, not the skin, may be incised.

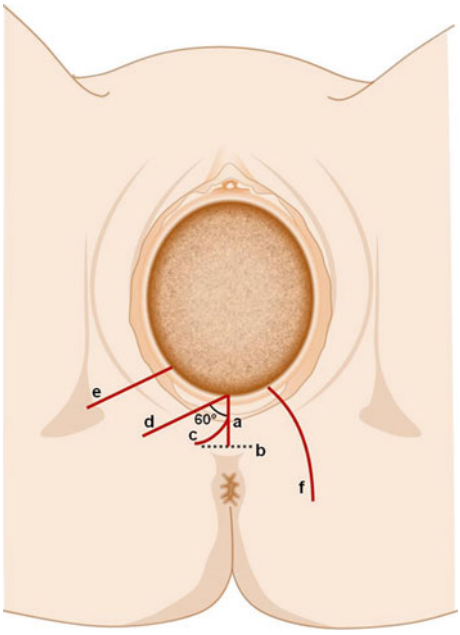

Fig. 6.2 Types of episiotomy. Key: *a* midline episiotomy; *b* modified median episiotomy; *c* J-shaped episiotomy; *d* mediolateral episiotomy; *e* lateral episiotomy; f radical lateral (Schuchardt incision) (Illustration adapted from Hakan Soken, MD, Eskisehir Military Hospital, Turkey, hsoken@hotmail.com)

"J-shaped" episiotomy runs initially as a midline incision and then at approximately 2.5 cm from the anus is curved to avoid the anal sphincter [10, 22, 23]. The latter part of episiotomy is directed towards the ischial tuberosity [24].

Mediolateral episiotomy is a compromise between midline and lateral episiotomy. The results of recent research clearly demonstrate that the definition of mediolateral episiotomy has thus far been unsatisfactory [25–31]. A wide variety in the clinical performance of mediolateral episiotomy has been observed between countries and institutions [29] as well as between individual doctors and midwives [27, 28]. Based on studies by Tincello et al. [27], Eogan et al. [32], and Kalis et al. [30, 33] evaluating the placement of episiotomy, an angle of episiotomy of 60° has been proposed as part of the definition [10]. Therefore, mediolateral episiotomy is defined as an incision starting at the posterior fourchette in the midline and directed at an angle of at least 60° towards the ischial tuberosity [10, 33].

Lateral episiotomy begins in the vaginal introitus 1–2 cm laterally from the midline and is directed towards the ischial tuberosity [24, 34–37]. Lateral episiotomy is often non-mentioned in obstetric literature [8, 11, 12, 38]. This type has been reported in only one RCT [19]. The Cochrane review [9] suggests that: "*There is a pressing need to evaluate which episiotomy technique (mediolateral or midline) provides the best outcome*" thus not taking lateral episiotomy into account [9]. Also, a review analyzing seven commonly sold general textbooks [39] evaluates whether *"both methods of performing episiotomy (median/mediolateral)"* are discussed in the texts, so again no other type of episiotomy is mentioned. However, it has been found that lateral episiotomy has in fact been used, albeit unintentionally by wider medical community, in Europe [28, 29]. In both Finland and Greece this type of episiotomy is used routinely [14, 40, 41].

Radical lateral (Schuchardt incision) is an original non-obstetrical **e**pisiotomy performed at the beginning of radical vaginal hysterectomy or trachelectomy [42–44], starting as lateral episiotomy but passing around the rectum in a downward, lateral curve [45]. Only rarely it is recommended as an aid to childbirth during complicated deliveries [35, 37, 45].

Anterior episiotomy (deinfibulation – opening the scar associated with female genital mutilation). A potential choice for labour and also antenatally [20, 46], the anterior scar tissue is incised in the midline up to the urethra [47]. Due to the possibility of tissue stretching at the end of delivery, it may be deemed necessary to employ an alternative type of episiotomy.

To improve the methodological quality of studies evaluating episiotomy, the authors present the following proposal for a detailed classification of episiotomies, Table 6.1.

Significance of the Placement of Episiotomy

An evaluation of studies and reviews, where the majority focussed on mediolateral episiotomy, has found that the methodology is very often poorly organised [48]. Four main problems were defined: diagnostics and classification of the perineal trauma, and the definition and practical implementation of mediolateral or lateral

Table 6.1 Types and characteristics of episiotomies [10]

Type of episiotomy	Location of the initial incision	Direction of the cut
Midline	within 3 mm of the posterior fourchette (midline)	between 0 and 25° of the midline
Modified median	within 3 mm of the posterior fourchette (midline)	between 0 and 25° of the midline
"J shaped"	within 3 mm of the posterior fourchette (midline)	At first midline, then "J" is directed towards the ischial tuberosity
Mediolateral	within 3 mm of the posterior fourchette (midline)	Directed laterally at an angle of at least 60° towards the ischial tuberosity
Lateral	1–2 cm from the midline	Towards the ischial tuberosity
Radical lateral (Schuchardt incision)	1–2 cm from the midline	Towards the ischial tuberosity and around the rectum
Anterior	Midline	Midline, directed towards the pubis

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episiotomy [48]. Evaluating the methodology of studies included in the Cochrane review [9], only two studies looked at median episiotomy [49, 50], and none of the others described the positioning of mediolateral episiotomy sufficiently [25, 51–55], suggesting that the methodology did not fulfill current requirements and no clear conclusions may be drawn, especially with regards to severe perineal trauma.

There is growing evidence that the exact placement of episiotomy plays an important role in the degree of perineal trauma [14, 15, 32, 57–59]. At crowning, the perineal body is particularly exposed to a high degree of deformation. After delivery, the strain on the perineal tissues and edema recedes, and subsequently the deformation disappears. The significant alterations in the geometry of the perineal region result in a difference between the observed episiotomy locations: at the time of incision, after repair, and later postpartum [59]. Based on perineo-anthropometric studies [30, 32, 33] three new terms have been introduced: incision angle, suture angle and scar angle of episiotomy [33]. It has been shown that a mean incision angle of 40° falls to 20° after suture [30] while that of 60° falls to 45° [33]. Analysis showed a 50 % relative reduction in the risk of obstetric anal sphincter injuries (OASIS) for every 6.3° that the episiotomy scar lies away from the perineal midline [32]. This finding was later supported by Stedenfeldt who also found that episiotomy cut too short and at too wide an angle also carried a higher risk of OASIS [56].

In many retrospective studies [60–63] as well as in a recent RCT [50] midline episiotomy was associated with an increased risk of OASIS and consequent functional damage. A significantly higher incidence of OASIS was also found for midline episiotomy in comparison to the mediolateral type [26, 64–67]. Mediolateral episiotomy has consistently been found to be protective in instrumental vaginal deliveries [68–70] whereas midline episiotomy has been associated with a significant increase in OASIS [71]. Lateralisation of episiotomies as a part of a set of obstetric interventions contributed to an immediate significant decrease in OASIS

in Norway [57, 58, 72]. Lateral episiotomies have consistently been found to be protective in primiparous women [16, 17], the effect of mediolateral episiotomies has differed between studies [73–77], and midline episiotomy has never been found protective [7, 9, 78, 79]. A recent RCT evaluating properly performed mediolateral and lateral episiotomies has shown a low incidence of OASIS amongst primiparous women [80]. Two procedures that appear to be among those that best divert principal perineal tissue strain away from the midline appear to be appropriately executed mediolateral episiotomy as well as properly executed lateral episiotomy [80].

Conclusion

The exact placement of incision of episiotomy has a significant role in the subsequent severity of perineal trauma. Lateralisation of episiotomies has significantly decreased the incidence of OASIS. Either mediolateral episiotomy at an angle of at least 60° from the midline or lateral episiotomy are recommended if indicated. If in a study design the type of episiotomy differs from internationally recognized mediolateral or lateral episiotomy, then specific details of the episiotomy characteristics are required.

Episiotomy Goals and Indications

The performance of episiotomy to expedite delivery in cases of non-reassuring fetal status by shortening the second stage of labour is currently a generally accepted approach. There does however remain a lack of professional consensus regarding other specific episiotomy indications. The commonly argued goals and indications include:

1. Prevention of OASIS and of pelvic floor dysfunction either in general, or in the following cases: short perineum, instrumental delivery *(*see the section "Episiotomy and instrumental deliveries"), fetal macrosomia, prolonged second stage of labor, imminent perineal tear, history of episiotomy or OASIS in previous delivery.
2. Providing space for the facilitation of difficult deliveries (i.e., shoulder dystocia, persistent occiput posterior presentation, breech delivery),
3. Lack of self-control or cooperation of the mother.

The Role of Episiotomy in Prevention of OASIS

The protective effect of episiotomy against OASIS is a matter of controversy and largely depends on the type of episiotomy. The estimated risk of OASIS in women undergoing midline episiotomy is six times higher than in the case of mediolateral episiotomy [64], while women giving birth without a mediolateral episiotomy were

1.4 times more likely to experience OASIS [75]. In spite of the fact that mediolateral episiotomy was occasionally identified as an independent risk factor for OASIS [76], a large Dutch retrospective study demonstrated its protective effect [73]. Interestingly, no difference was observed in the prevalence of OASIS when comparing maternity units with either a restrictive or routine approach to episiotomy [73]. While midline episiotomy increases the risk of OASIS, the role of mediolateral episiotomy in OASIS prevention depends upon the correct identification of the risk group of patients and upon its correct execution. A protective effect of lateral episiotomy was consistently demonstrated [81].

The Role of Episiotomy in Prevention of Pelvic Floor Dysfunction

Episiotomy has not been found to confer benefits with respect to preserving continence or pelvic floor muscle function within a period of months or years after birth [82]. Mediolateral episiotomy was, in some studies, associated with a lowered strength of the pelvic floor muscles in comparison with spontaneous perineal lacerations [83]. On the other hand, a recent prospective cohort study suggested that while women with perineal lacerations in two or more deliveries were at a significantly higher risk of prolapse 5–10 years after the first delivery, women with a history of even multiple episiotomies showed no increase in the risk of suffering a prolapse [84]. Nevertheless, although well established in clinical practice, the prevention of pelvic floor dysfunction alone, as an indication for episiotomy, is hardly justifiable at present.

Short Perineum

The association between short perineal body length and the risk of OASIS is controversial. A perineum shorter than 4 cm in the first stage of labor was associated with traumatic vaginal delivery [85]. However, the mean perineal length ranges from 3.6 to 4 cm [86]. The phenomenon of perineal second-stage stretching is to be considered. Second-stage perineal stretching >150 % was found to be predictive of perineal damage and assessment of perineal stretching was suggested to avoid unnecessary episiotomies [87]. However, the stretching did not correlate with the degree of trauma among multiparous women [86]. No data exist suggesting any benefit of performing episiotomy in cases of a short perineum or low perineal stretching. Apart from a short perineum, the range of anal dilation in the final phase of labour may contribute to the degree of perineal trauma [88].

Fetal Macrosomia

It is generally acknowledged that fetal macrosomia is an important risk factor of OASIS. Prevalence estimates of OASIS based on published odds ratios have demonstrated no preventive effect of episiotomy in the delivery of a macrosomic fetus.

However, the type of episiotomy used was not provided in these studies and could possibly have been midline [89–91]. The type of episiotomy certainly plays an important role; however, there is no data evaluating the benefits of different types of episiotomy in deliveries of macrosomic infants.

Imminent Perineal Tear

A German RCT demonstrated benefit of avoiding episiotomy in cases of impending perineal tear. This practice was associated with an increased frequency of intact perineum or minor trauma, reduction of postpartum perineal pain, and with no increase in maternal or neonatal morbidity [51]. A follow-up of this study proved that episiotomy at the time of impending perineal tear is not beneficial for the preservation of pelvic floor function [92].

History of Episiotomy or Severe Perineal Trauma in a Previous Delivery

Episiotomy performed at a first vaginal delivery is a significant independent risk factor of repeated episiotomy and spontaneous perineal tears in a subsequent delivery [93]. Episiotomy at first delivery was associated with more than a four-fold risk of perineal laceration in subsequent childbirth [94]. The data encouraged further restrictions in episiotomy use. There are no data supporting routine episiotomy in a childbirth with previous OASIS.

Space for Necessary Interventions or Maneuvers in Difficult Deliveries

Preventive episiotomy is commonly performed to facilitate maneuvers in difficult deliveries such as malpresentations or anticipated shoulder dystocia. In spite of historical recommendations that episiotomy should be performed for brachial plexus injury prevention when shoulder dystocia is encountered, recent evidence has demonstrated no neonatal benefit of this practice [95]. Performing fetal manipulations without midline episiotomy in severe shoulder dystocia leads to a reduction in the risk of OASIS without incurring a greater risk of brachial plexus injury [96]. Moreover, use of mediolateral episiotomy in instrumental delivery did not reduce the risk of shoulder dystocia [97]. Therefore, episiotomy in cases of shoulder dystocia should be reserved for cases where maneuvers to effect delivery cannot be reasonably achieved without episiotomy [95].

There is not enough evidence regarding the relationship between persistent occiput posterior position, episiotomy and perineal trauma. A French cohort retrospective study found that mediolateral episiotomy is not protective against OASIS in cases of persistent occiput posterior positions [98].

Likewise, there is not enough data regarding the relationship between episiotomy and breech delivery. Although episiotomy is quite common for breech delivery in clinical practice, a restrictive approach can also be employed. Based on a Dutch perinatal register, mean episiotomy rate in term breech delivery was 72 % in 1990, with a wide variation among hospitals (19–100 %) [99].

Self-Control of the Woman

The risk of laceration is increased in a patient, who is not capable of good self-control (i.e. unable to respond to directions) and some accoucheurs prefer to cut an episiotomy. Nevertheless, no data exist regarding the benefits of this practice.

Conclusion

Analysis of episiotomy indications is an important step in the identification of patients, who could really benefit from this obstetric intervention. This approach leads to a reduction in the frequency of episiotomy while preserving, or even improving the standard of care. Apart from a clear indication for episiotomy, i.e., shortening of the second stage of labour in case of suspected fetal compromise, there are many other indications of episiotomy. While the existing evidence suggests that most of these indications are not justified per se, there are circumstances in which a prudent clinical judgment necessitates an episiotomy. In these cases, mediolateral or lateral episiotomy should be preferred.

Episiotomy and Instrumental Deliveries

Traditionally, episiotomy has been a routine component of instrumental delivery, the primary aim being to avoid OASIS. However, the use of instrumental delivery in combination with midline episiotomy was associated with a significant increase in the risk of OASIS in both primiparous and multiparous women [71]. Time trends support a reduction in OASIS by restricting the liberal use of the two modifiable variables: midline episiotomy and forceps delivery [100].

Routine use of mediolateral episiotomy in instrumental delivery is recommended by the National Institute for Health and Care Excellence (NICE) [101]. National surveys in the UK and Ireland revealed that two-thirds of obstetricians held the view that routine use of episiotomy decreases the likelihood of OASIS for a forceps delivery while having a divided view as to vacuum extraction [102]. In the only RCT comparing routine versus restrictive use of episiotomy for instrumental delivery, routine use of episiotomy was not associated with a statistically significant difference in the incidence of OASIS (8.1 % vs. 10.9 %) [103]. However, subsequently and with regards to the same population, Macleod et al. [104] found that restrictive use of episiotomy for instrumental delivery may increase immediate

postpartum morbidity, in particular the incidence of perineal pain and stress urinary incontinence. The type of episiotomy or its precise placement were not recorded and neither were the complete spectrum of other obstetric interventions [103, 104].

Two large retrospective population-based register studies from the Netherlands suggested that mediolateral episiotomy reduces the risk of OASIS in instrumental delivery [68, 69]. De Leeuw et al. demonstrated that mediolateral episiotomy significantly protected against OASIS in both vacuum extraction and forceps [68]. Twelve mediolateral episiotomies were needed to prevent one case of OASIS concerning vacuum extraction, whereas five mediolateral episiotomies could prevent one case of OASIS with regards to forceps. Another Dutch group found a sixfold decrease in the risk of OASIS when mediolateral episiotomy was performed in women undergoing instrumental deliveries [69]. According to this study, the known adverse effects of mediolateral episiotomy (e.g., short-term perineal pain, dyspareunia) cause less morbidity compared with the known adverse effects of OASIS (e.g., fecal incontinence).

In a similar Finnish study evaluating vacuum extraction, lateral episiotomy decreased the incidence of OASIS by 46 % in primiparous but not in multiparous women [17].

Conclusion

The significant risk-reducing effect of mediolateral or lateral episiotomy warrants their use in all instrumental deliveries at least with regards to primiparous women, as opposed to the use of midline episiotomy which carries a considerable risk of the occurrence of OASIS in instrumental deliveries.

Episiotomy Rate

Although there is a growing general consensus about restricting the use of episiotomy, no such agreement has emerged as to what constitutes an appropriate episiotomy rate [105]. Carroli and Belizán have established that a restrictive episiotomy rate above 30 % is not clinically justified [25]. Episiotomy rates around the world range from as low as 9.7 % in Sweden to 100 % in Taiwan, while half of all countries exceeded the recommended rate of 30 % [40, 105]. Moreover, episiotomy rates vary with regards to parity. Results from large epidemiologic studies from restrictive episiotomy settings where total episotomy rate remained under 30 % showed an episiotomy rate of 55–65 % in primiparous women [106, 107].

When defining the lower limit for “safe” episiotomy rate, it is important to take into account the type of episiotomy being used and the quality indicator for determining the success of the restrictive approach. The quality indicator commonly used is the OASIS rate.

In the USA, a restrictive approach to midline episiotomy in spontaneous deliveries resulted in a reduction in the OASIS rate from 5 to 3.5 % [108]. In Australia a significant correlation was registered between increasing mediolateral episiotomy use, from 12.6 to 20.1 %, and a reduction in the OASIS rate, from 4.4 to 2.1 % [109]. Both lateral

(in Finland and Norway) and mediolateral episiotomy (in Sweden, Denmark and Norway) are used in Nordic countries. Within the last 10 years a falling trend in the use of episiotomy was registered in Denmark (10 % vs. 5 %) and Sweden (9 % vs. 6 %) while the rate remained unchanged in Norway (20 % vs. 19 %) and stayed higher in Finland (42 % vs. 24 %). However, OASIS incidence in Finland has been notably lower (0.7–1 %) than in the other Nordic countries (2.3–4.2 %). A significant and constant reduction in OASIS incidence has only been observed in Norway (from 4.1 to 2.3 %, $p<0.001$) [110, 111]. This reduction occurred simultaneously with the introduction of a national intervention program of improved delivery techniques aiming at reducing the incidence of OASIS [57, 111].

Conclusion

Nowadays, taking into account different episiotomy types (midline, mediolateral, lateral), it is necessary to find a balance between the lowest reported (total: 5 %, primiparas: 10 %, multiparas: <5 %) and the optimal (total: <30 %, primiparas: 50 %, multiparas: <10 %) episiotomy rates for both spontaneous and instrumental deliveries with regards to the OASIS rate of between 1 % and 5 % depending on the strengths of the restrictive approach and the type of episiotomy applied.

Timing of Episiotomy

The optimal time for performing an episiotomy is unclear and depends largely on the indication. In cases where prophylactic episiotomy is performed, i.e. to facilitate a forceps delivery or to expedite delivery, it is recommended to perform episiotomy when the head is visible during a contraction to a diameter of 3–4 cm [20]. However, with restrictive approach to episiotomy, the indication often arises during the crowning. It is important to bear in mind the significant difference in the change of the angle of mediolateral episiotomy between time of cut and after repair depending on the timing of the episiotomy [30]. Performing episiotomy early before crowning of the fetal head is associated with increased blood loss [112]. Some authors have argued that performing episiotomy too late compromises the protection of the maternal perineum. According to their opinion, at the time of crowning, the fetal head has already torn the perineal muscles and the damage of the supporting structures has already occurred [24, 113]. However, no valid studies have been performed to support this expert opinion.

Episiotomy Repair

Reduction of maternal discomfort during episiotomy repair and short- and long-term maternal morbidity following this procedure can be achieved with the use of an appropriate type of analgesia, the choice of quality suture materials and the application of modern suturing techniques.

The level of analgesia/anaesthesia should be adequate for the episiotomy repair. If the patient received an adequate epidural anaesthesia during labour, it can be used to provide analgesia for the repair. Pudendal nerve block or local field block is generally adequate if there is no pre-existing analgesia.

Several studies have shown the advantages of fast-absorbing polyglactin 910 for episiotomy repair [114–117]. Meta-analysis revealed that, comparing standard synthetic with fast-absorbing sutures for repair of episiotomy and second-degree tears, short- and long-term pain scoring was similar [118]; in one trial fewer women with fast-absorbing sutures reported using analgesics at 10 days (RR 0.57) [117]. More women in the standard synthetic group required suture removal compared to those in the fast-absorbing group (RR 0.24) [117, 118].

For more than 80 years, researchers have been suggesting that continuous non-locking suture techniques for repair of the vagina, perineal muscles and skin are far better than "traditional" interrupted methods in terms of reduced postpartum pain [117, 119–121]. Recent meta-analysis showed that continuous suture technique, when compared with interrupted sutures for episiotomy or second-degree tear repair (in all layers or perineal skin only), are associated with less perineal pain for up to 10 days postpartum (RR 0.76) [122]. There was an overall reduction in analgesia use associated with the continuous subcutaneous technique versus interrupted stitches for repair of perineal skin (RR 0.70). There was also a reduction in suture removal in the continuous suturing groups versus interrupted (RR 0.56), whereas no significant differences were seen in the need for re-suturing of wounds or in long-term pain.

Several case studies and one small randomized trial have suggested that tissue adhesives could be used instead of stitches for episiotomy repair [123–126]. However, these agents are expensive and not all are widely available so further research is needed to determine the safety and efficacy of this approach.

Conclusion

Continuous non-locking suturing technique for all layers using fast-absorbing synthetic material is currently the recommended standard for the episiotomy repair. See Fig. 6.3a–d.

Episiotomy and Healing Complications. Resuturing of Episiotomy

Complications can occur in any healing process. In episiotomy and/or any degree of perineal trauma, the following variables are usually evaluated: episiotomy dehiscence and need for surgical re-intervention, infection of episiotomy and need for antibiotic treatment, haematoma in episiotomy, and the need for removal of suture material [9]. These variables have not been evaluated in any significant detail and extensive data are not available due to the relatively low prevalence of these complications which vary between 0.1 and 2.1 % [127–130].

For an overall evaluation of healing complications in episiotomy suture, the REEDA scale is generally used [131] in spite of some limitations to the

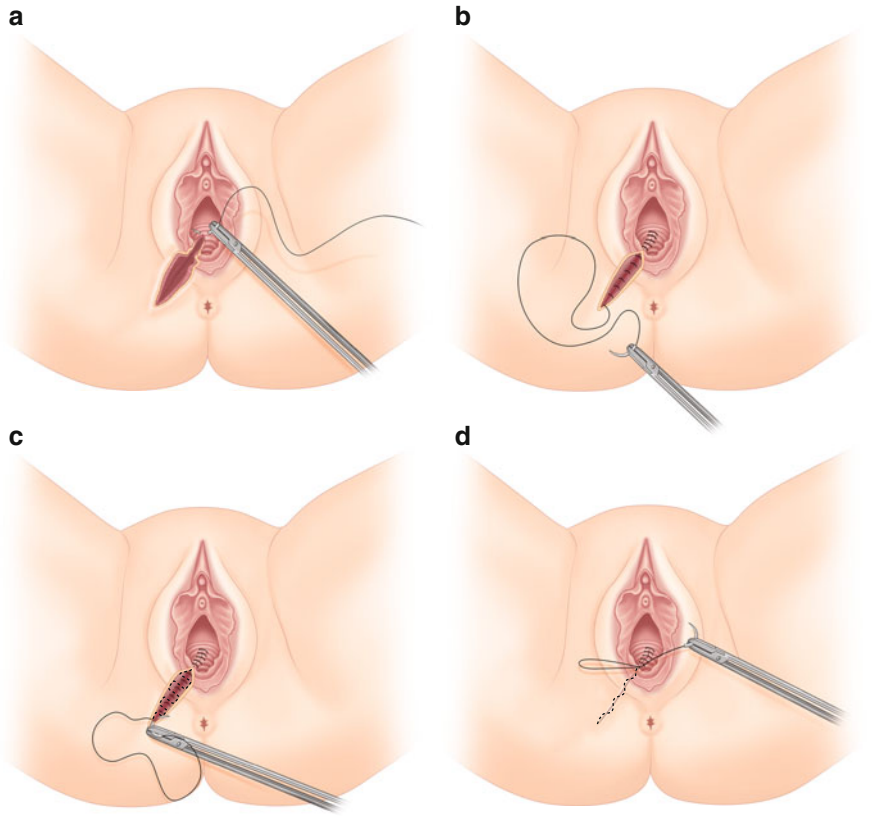

Fig. 6.3 Episiotomy repair using continuous non-locking suture (all layers). (**a**) Episiotomy repair, suturing of vaginal wall. After perineal infiltration with local anaesthetic, carefully insert first stitch to the vagina above the apex of episiotomy cut and tie a knot there. (**b**) Episiotomy repair, suturing of perineal muscles. (**a**) Episiotomy repair, suturing of vaginal wall. Appose divided perineal muscles and deep subcutaneous tissue. Approximate skin edges as much as possible. (**c**) Episiotomy repair, suturing of perineal skin. Starting from the bottom edge of the episiotomy cut, close perineal skin in the opposite direction towards the vaginal orifice using subcuticular continuous suture. (**d**) Episiotomy repair, knotting of the stitch. Place the subcuticular stitch in the vagina just above the remnants of the hymen and tie a knot there (Illustrations adapted from Hakan Soken, MD, Eskisehir Military Hospital, Turkey, hsoken@hotmail.com)

interrater reliability evaluation [132]. This scoring system contains five domains: redness, edema, ecchymosis, discharge and approximation of the edges of the suture [131].

Dehiscence

In a recent study, dehiscence of episiotomy suture requiring further treatment was found in 1 % [127]. The technique of episiotomy repair, choice of material, instrumental delivery, OASIS, perineal body length and neonatal weight along with a

surgeon‘s inexperience were all found to be contributing factors for dehiscence of episiotomy suture [128, 133–141].

There is no current consensus on the definition of episiotomy dehiscence. Dehiscence may vary in severity from a mild superficial detachment of the skin to extensive separations involving a complex of anal sphincters and rectal mucosa [133]. For a more specific clinical definition a wound gaping of more than 0.5 cm [142] or complete separation of epithelium of at least 50 % of the episiotomy length [133] have been used.

Infection

Infection is defined clinically by the presence of sero-purulent or purulent discharge or fever [127]. Infection is a major cause of episiotomy dehiscence occurring in 0.05–0.5 % [143, 144]. In a study by Uygur et al. [127] 67 % of dehiscences were infected. Microbiological or imaging examinations are not required to confirm the diagnosis except in severe cases requiring re-hospitalization [141]. Poor postpartum perineal hygiene or hematoma in episiotomy suture line might be contributing factors [133].

Prevention of Dehiscence and/or Infection in Episiotomy

Prevention involves reducing exposure to the risk factors (see above) combined with adequate postpartum care. A satisfactory standard of episiotomy repair technique minimizes the risk of haematoma, tissue ischaemia, and inadequate approximation. Postpartum daily routine inspections of the perineal area are essential [145, 146].

Resuturing of Episiotomy

There is a paucity of evidence on the management of episiotomy dehiscence. A recent Cochrane review [147] includes only two small studies [143, 148] with a total of 52 participants. Conservative therapy consists of cleaning of the dehiscence with local antiseptics and local or systematic application of antibiotics. The process of granulation results in spontaneous healing [127, 148].

Nowadays, some guidelines [38] and the majority of studies suggest good results with active local therapy including irrigation, devitalized tissue debridement and a sitz bath several times a day with an eventual systematic application of antibiotics followed by early resuturing, usually within 4–10 days [127, 148–150]. An earlier resumption of sexual intercourse has been observed after resuturing compared to conservative management [148]. Resuturing should be performed after careful debridement when the surface of the dehiscence is clean and its margins covered by pink granulation tissue [149]. During surgery, debridement of this granulation tissue is also performed [149]. It is not important whether a continuous running suture or interrupted sutures are used. However, mid-term absorbable suturing material is recommended [127, 133]. After resuturing, sitz baths should be continued. Administration of antibiotics is considered on an individual basis.

Conclusion

The recommended technique of episiotomy repair, adequate hygiene and regular postpartum inspection of the perineum reduce complications of the healing process.

Episiotomy and Perineal Pain

Episiotomy is a common cause of postpartum perineal pain [9, 26, 55, 151, 152]. The presence and intensity of the pain is associated with the degree of perineal injury [151, 153–155], instrumental delivery, parity, duration of delivery [152, 156], type of suturing material [114–116, 118] repair technique [117, 122] or analgesia used [157–164]. Currently, the most commonly used scoring systems are two components of SF-MPQ [165]: The Visual Analogue Scale (VAS) and Present Pain Intensity (PPI), and the four-point Verbal Rating Score (VRS) [166].

Post-episiotomy pain affects up to 97 % of women on the first day [26, 152] and up to 71 % of women 7–10 days postpartum [152]. Comparing routine and restrictive approaches, the current version of the Cochrane review [9] has found a higher incidence of pain in the routine approach at discharge [25] but no difference at 3 and 10 days and 3 months postpartum [54, 55]. Women with a routine approach to episiotomy registered more maximum intense pain within the first 5 days postpartum [51]. However, in the long term, there was no difference observed in the prevalence of perineal pain between routine and restrictive approaches [167, 168].

There are very few studies comparing different types of episiotomy and perineal pain. In a quasi-randomized trial comparing midline and mediolateral episiotomies no difference in pain was observed 3 months after delivery [26]. The only study, with a retrospective design, evaluating perineal pain after mediolateral and lateral episiotomies and only one day postpartum found no difference in perception of pain [18].

When deliveries with episiotomy were compared to deliveries without episiotomy, the incidence of short-term episiotomy pain was similar on the 1st, 7th or 10th postpartum day regarding spontaneous first- and second-degree perineal tears, but higher than figures for an intact perineum and lower than those for OASIS [151, 152]. No difference was observed at 6 weeks [152]. At 3 months the incidence of post-episiotomy perineal pain was similar compared to spontaneous first- and second-degree tears but the frequency and intensity were higher in the episiotomy group [151].

Prevention

Antenatal perineal massage [169, 170], application of warm perineal packs/compresses during the second stage [171, 172] and manual perineal protection (MPP) [173] may decrease the rate of postpartum perineal pain. If episiotomy is indicated, midline episiotomy should not be selected. Midline episiotomy significantly increases the risk of OASIS, the main cause of intense and long-term perineal pain. Another type of episiotomy should be used.

Current standards of episiotomy repair reduce post-episiotomy pain [117]. An epidural provided during labor can be used to relieve any immediate pain. If an epidural has not been provided, immediate pharmacological analgesia (rectal, oral, occasionally subcutaneous or intramuscular) can lower the maximum intensity of postpartum pain usually occurring during the first 24 h [155, 157–164]. Application of local cooling tools can reduce the subsequent development of oedema and haematoma, which contribute to perineal pain [164, 174, 175].

Treatment

Post-episiotomy pain can be significantly reduced using analgesics. There are a number of products available and several methods of administration (oral, local, rectal, etc.) can be used. A combination can enhance the effect.

A variety of oral analgesics can be used. The effects of non-steroidal anti-inflammatory drugs (NSAID): acetaminophen, celecoxib, diclofenac, indomethacin, ketoprofen or paracetamol alone or in combination were reported [155, 157, 159–161]. Diclofenac administered either orally or rectally has been found to be more effective or faster acting than others [157, 159, 160]. However, oral celecoxib has shown a larger reduction of pain score on VAS compared to oral diclofenac [161]. Rectal suppositories showed the best effect compared to oral analgesics or ice packs [155]. No trials included in the Cochrane review showed any difference in pain relief when a local anaesthetic was compared with placebo [163]. Several non-pharmacological methods have also been tested. Application of ice packs and cold gel decreased the pain in comparison with cases when no treatment was applied while gel pads were preferred over ice packs or no treatment [155, 164].

Conclusion

Current data suggest that there is no difference in perception, frequency and intensity of pain between different types of episiotomy. However, there is a paucity of literature addressing this problem. Post-episiotomy pain seems to be slightly (not significantly) increased compared to spontaneous first- and second-degree tears in the short to mid-term. However, the short-term pain is reducible with the use of analgesic agents.

Episiotomy and Sexual Function

Any childbirth, and particularly vaginal delivery, may change the qualitative level of sexual function. There are many sexual function related outcome measures to be followed. The main sexual components – desire, arousal, lubrication, orgasm, satisfaction and pain – are included in the most common tool used to evaluate postpartum sexuality, the Female Sexual Function Index (FSFI) [176]. Another scoring system frequently used is the McCoy Female Sexuality Questionnaire [177].

Apart from episiotomy and perineal trauma, sexual function after delivery can be subject to other variables: maternal age [178–180], partnership status [180, 181], breastfeeding [151, 179, 181–186], overall health and mental [181, 187] and physical status (including the partner's) [180], pre-pregnancy dyspareunia [182, 183, 188], instrumental delivery [189, 190] or parity [151, 181, 191, 192].

Resumption of Sexual Intercourse

After vaginal delivery with episiotomy, one-third of women has resumed vaginal sex by 6 weeks, two-thirds by 3 months and 90 % by 6 months [179–182, 184, 193]. At 12 months 95–100 % of women in all groups have resumed vaginal sexual intercourse [180, 181].

Primiparous women with episiotomy re-initiated their vaginal sexual intercourse later than those after vaginal delivery with an intact or unsutured perineum [180]. Comparisons with women after caesarean section have been conflicting [180, 189, 193–199], a large RCT reported no effect on resumption of sexual activity or sexual dysfunction [199]. There has been no significant difference found when episiotomy was compared to spontaneous sutured tears [180, 193]. However, women with first- and second-degree tears had less pain at first postpartum sexual intercourse than women with episiotomy [151].

Sexual Function in the Short Term

In comparing restrictive and routine approaches [9] the Cochrane review has included the data of only one trial [167] evaluating a resumption of intercourse and dyspareunia at only 3 months after the index delivery. No significant difference was noted in either of these [9, 167]. In other Cochrane reviews short-term absorbable synthetic sutures when compared to catgut [118], and continuous technique of repair for all layers when compared to interrupted stitches [122] resulted in significantly lower rate of dyspareunia at 3 months.

The rates of dyspareunia after mediolateral or midline episiotomy vary between 8 and 73 % at 3 months [179, 182, 189] and 11 and 36 % at 6 months [33, 179, 182, 189]. In a study by Barret et al. [182] the rate of dyspareunia after episiotomy compared to that after spontaneous perineal tears was non-significant and was higher than in women with an intact perineum at 6 months. Vaginal tearing has been found to be a higher risk factor than episiotomy [181, 182]. There has not yet been any data gathered on the consequences after lateral episiotomy.

Sexuality in the Long Term

In a study by Ejegård et al. and Bühling et al. [183, 189] there was no difference in sexual satisfaction or sexual function between women with or without episiotomy at 12–18 months postpartum. However, dyspareunia [183, 189, 200] and

vaginal dryness [183] were more frequent in women after episiotomy. Long-term comparisons to second-degree tears are conflicting [183, 189]. Anyway, the most significant risk factor for long-term postpartum dyspareunia was previous dyspareunia [182, 183, 188]. Also, long-term postpartum dyspareunia seemed to be related more closely to the mother's experience of delivery than to perineal trauma [188].

Conclusion

Human sexuality is a complex interaction involving biological, sociocultural, and psychological factors in which episiotomy plays a limited role. The current data regarding postpartum sexual function are unclear because of the high variety of measured outcomes [201]. Breastfeeding [151, 179, 181–186], previous dyspareunia [182, 183, 188], instrumental delivery [189, 190] and OASIS [151, 153, 179, 181, 193] are consistent risk factors for postpartum dyspareunia or impairment of sexual activity.

Reducing perineal trauma (i.e., episiotomy or spontaneous tears) during delivery to the greatest extent possible is important for the resumption of sexual intercourse after childbirth [151, 179]. Episiotomy is occasionally considered to be more significant for short-term postpartum dyspareunia compared to spontaneous tears without OASIS. However, overall sexual satisfaction seems to be equal. Adequate episiotomy repair significantly decreases the rate of postpartum dyspareunia.

Episiotomy and Incontinence

Urinary Incontinence

Urinary incontinence (UI), the involuntary loss of urine, is a frequent consequence of pregnancy and childbirth. The cumulative incidence of de novo UI during pregnancy is 39 % [202]. Furthermore, 33 % of women reported symptoms of UI 3 months postpartum [203] and 31 % of women 6 months after delivery [204]. No difference was reported in the frequency of postpartum stress urinary incontinence (SUI) in patients with and without episiotomy at 3 months postpartum (13 % vs. 12 % [83] and 29 % vs. 35 % [205]). Regarding urge urinary incontinence (UUI), two North American studies found episiotomy to be statistically significant in univariate, but not multivariate analysis 4 and 7 months after delivery [206, 207]. In a retrospective Italian study, women after laterally positioned episiotomies registered a non-significantly lower rate of UUI and significantly lower King's Health Questionnaire (KHQ) scores compared to a group with no episiotomy 12 months after delivery [208].

Anal Incontinence

Anal incontinence (AI), the involuntary loss of flatus, liquid or solid stool, is a serious and distressing condition with a devastating effect on quality of life including

occupational, social and sexual aspects. Fecal urgency (FU), the inability to suppress the sensation of necessity to defecate for more than 15 min, was proven to be closely associated with EAS dysfunction irrespective of rectal sensitivity and internal anal sphincter dysfunction [209]. Many tools have been developed for scoring of anal incontinence severity. The Wexner (Cleveland) score [210] is currently the most frequently used scoring system globally. However, St. Mark's score [211] or a more complex scoring tool that takes FU into consideration, i.e., Fecal Incontinence Quality of Life (Rockwood) score [212] were recommended for the follow-up of patients with childbirth trauma [213] since FU is commonly associated with EAS injury [209].

The incidence of accidental bowel leakage was reported in 6.4 % of patients 6 weeks after delivery and 5.3 % of patients 1 year post partum [214]. A Spanish study reported a 10.3 % cumulative incidence rate of de novo AI in nulliparous women during pregnancy and after delivery [202]. A large retrospective cohort study demonstrated that midline episiotomy is not effective in protecting the perineum and sphincters during childbirth and may impair anal continence. Women who had midline episiotomies had a significantly higher risk of fecal incontinence at 3 and 6 months postpartum compared to women who delivered with an intact perineum (OR 5.5 and 3.7, respectively). Even when compared with spontaneous laceration, midline episiotomy tripled the risk of fecal incontinence and doubled the risk of flatus incontinence at 3 and 6 months postpartum [60].

Contrary results were observed in cases of mediolateral episiotomy [215]. The incidence of AI in nulliparous women 10 months after delivery was comparable between those having mediolateral episiotomy, intact perineum and spontaneous laceration. Amongst multiparas the risk was higher in the episiotomy group [216]. A Dutch retrospective cohort study demonstrated a lower risk of subsequent fecal incontinence development (OR 0.17) in primiparas with OASIS after mediolateral episiotomy compared to OASIS without episiotomy [217].

Conclusion

No significant effect of episiotomy on postpartum SUI and UUI was demonstrated. No protective effect of mediolateral episiotomy on the development of AI in an uncomplicated delivery was proven. However, a significant increase in the risk of postpartum AI development in association with midline episiotomy was observed. Mediolateral episiotomy was found to be protective of postpartum fecal incontinence in cases where OASIS occurred.

Episiotomy as a Part of Complex Perineal Protection

Episiotomy is only one of many possible interventions that can be made during the final stage of vaginal delivery. Many others have been suggested either to facilitate or accelerate delivery of the fetus or to protect the perineum: obstetric gel [172, 218, 219], warm compresses [171, 172], second stage perineal massage [172, 219], water birth [220], variety of maternal positions [221], forceps, vacuum-extraction

[68, 69, 71], Thierry's spatula [222], fundal expression (Kristeller maneuver) [223, 224], slowing of the expulsion of the fetal head [40, 57, 110, 225], control of maternal co-operation, and manual perineal protection (MPP) during the expulsion of the fetal head [57, 110, 174, 225–227] or during the delivery of fetal shoulders. Moreover, as these interventions are often interrelated it is clinically difficult to analyze them separately. The complexity of the relationship between episiotomy and all other interventions is outlined in a recent population-based study [81]. During the study period the protective effect of lateral episiotomy in primiparous women has moved towards a positive association with OASIS only because of a more restrictive approach to episiotomy, while the incidence of OASIS amongst women without episiotomy has decreased [81]. The episiotomy rate in this study has just served as a surrogate for other unmeasured confounding factors [81].

A modern clinical approach is to evaluate the whole set of clinical interventions [40, 57, 58, 110, 225, 228] involving some steps of MPP, the slowing of the expulsion of the fetal head and episiotomy technique. In Norway, currently the only country with a reversed trend in the incidence of OASIS, a further lateralisation of episiotomy has been recommended [40, 57, 58, 110, 225]. The style of execution of episiotomies and MPP was derived from Finland [229] which traditionally has the lowest incidence of OASIS from all Nordic countries [40].

Norwegian studies have shown that not only known and recognized risk factors such as forceps, midline episiotomy, primiparity, fetal macrosomia or occiput posterior presentation have an impact on OASIS [231]. Some preventive steps are also possible. Lateralisation of episiotomies has not been the only one [10, 14, 15, 19, 32, 33, 40, 56–58, 66, 72, 110, 225]. At least some of the other implemented interventions – the slowing of the passage of the fetal head expulsion or a Finnish modification of MPP – have reduced the rate of OASIS [40, 57, 58, 72, 110, 225]. Recently, a potentially beneficial effect of MPP has been shown in a computerized study based on biomechanical principles where a simulation of the Viennese modification of MPP significantly reduced the perineal tension, thus supporting the concept of MPP [226]. A complex approach to protecting the perineum should be applied to all deliveries including deliveries with episiotomy. Recent studies have shown that there has been a similar reduction of OASIS amongst low- and high-risk women [57, 58, 225], and the most recent study found the most significant decrease of OASIS in deliveries of low-risk women [72].

The Role of Episiotomy in Modern Obstetrics

Throughout its history, episiotomy has been subject to various views from academic circles, media and the general population. Sometimes praised for its effect, at other times deemed too minor to be worthy of analysis, only to be condemned as useless, painful and harmful. It depends on the philosophical approach as to what rates of OASIS, pain, dyspareunia, AI and other perineal adverse outcomes are acceptable. Four percent of OASIS in Norway has recently been found to be unacceptable and led to a national audit and intervention. However, 4 % might be considered acceptable elsewhere.

Evidence suggests that correct execution of the episiotomy incision can have significant implications for the degree of perineal trauma. If episiotomy is to be performed, all its characteristics have to be clearly stated: the point of the beginning, direction and length. Midline episiotomy carries an unacceptable risk of OASIS and its consequences and should be avoided. Properly executed mediolateral or lateral episiotomy should be undertaken when indicated.

Episiotomy facilitates the delivery of the neonate and should be performed in cases of fetal compromise. It seems that properly performed mediolateral or lateral episiotomy is also protective in instrumental deliveries. The role of episiotomy either in protecting or in impending the anatomic and/or functional integrity of the perineum in general has not yet been fully explained. There is a current international consensus that a restrictive approach to episiotomy should be exercised. However, if more than a hundred mediolateral or lateral episiotomies are reported to be necessary to protect against one case of OASIS [14, 15, 231] in spontaneous deliveries, the acceptable rate of episiotomies is still undecided for the restrictive approach.

Current data suggest that if a recommended type of episiotomy repair is performed, there are no significant differences in perception, frequency and intensity of pain, dyspareunia and overall sexual function between different types of episiotomy. No significant effect of episiotomy on postpartum SUI or UUI has been found. A protective effect of mediolateral episiotomy on AI in deliveries without OASIS has not been demonstrated.

Episiotomy is merely one of many obstetric interventions considered during the second stage of labor. A complex approach to perineal protection in all vaginal deliveries is essential in order to reduce the rates of perineal trauma and, subsequently, the risk of adverse outcome.

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Types of Pelvic Floor Injury During Childbirth

7

Jorge Milhem Haddad, Lilian R. Fiorelli, and Thais V. Peterson

Abstract

Pelvic floor dysfunction has a high incidence mainly in aging women. Parity and vaginal childbirth are strongly associated with pelvic organ prolapse and stress urinary incontinence. Muscle injury, neurovascular injury and connective tissue remodeling may explain this association. In this chapter, the main mechanisms of injury are discussed, as well as the role of related risk factors, such as episiotomy, operative delivery and prolonged second stage of labor.

Keywords

Pelvic floor • Childbirth • Delivery • Urinary incontinence • Pelvic organ prolapse • Fecal incontinence • Risk factors • Forceps • Menopause • Episiotomy

Introduction

The incidence of pelvic floor dysfunction, such as pelvic organ prolapse and urinary and fecal incontinence increased by 45 % over the last 40 years. Increased life expectancy has significantly contributed since this occurrence increases over the woman's lifetime [1, 2].

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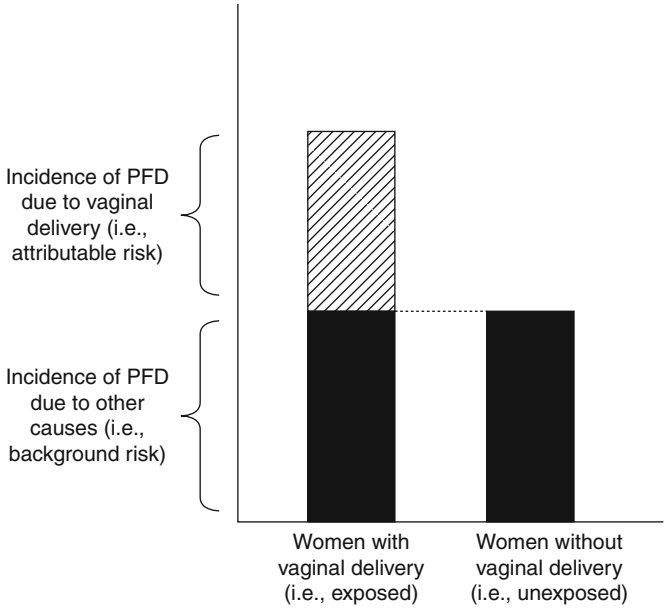

Fig. 7.1 Pelvic floor dysfunction in women with or without vaginal delivery (Reprinted from Patel et al. [4], Copyright 2006, with permission from Elsevier)

Main risk factors for pelvic floor dysfunction (PFD) include factors associated with increased intra-abdominal pressure such as obesity, multiparity, chronic cough, factors associated with the breakdown of collagen, such as smoking, deficiencies of collagen, menopause, and factors associated with local trauma, such as birth trauma, including vaginal delivery with or without the use of forceps [3]. Figure 7.1 shows the incidence of pelvic floor disorders associated with vaginal delivery [4].

Vaginal delivery increases the incidence of genital prolapse 4–11 times [1] and the incidence of urinary incontinence 2.7 times [5]. Important genes in inflammation, collagen breakdown, and smooth muscle inhibition are upregulated in patients who had vaginal delivery and develop incontinence and genital prolapse [6].

Types of Injury in Vaginal Delivery

Intact neuromuscular function and pelvic support are crucial to pelvic stability. Pregnancy and delivery contribute to pelvic floor disorders due to compression, stretching, or tear of nerve, muscle and connective tissue. We describe below the main mechanisms of obstetrical injury.

Mechanical Injury

Levator ani muscle complex integrity is very important to pelvic floor support. Comprising puborectalis, pubococcygeus and ileococcygeus, this muscle provides support to urethra, distal vagina and rectum. During labor, stretching and damage of

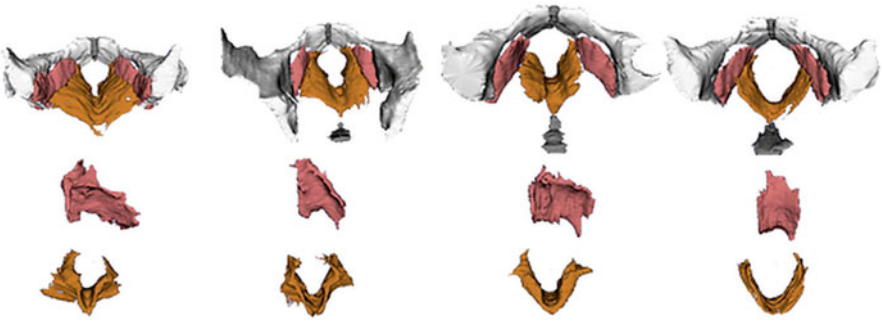

Fig. 7.2 Three-dimensional reconstruction of MRI of levator ani in nuliparous, asymptomatic multiparous, symptomatic multiparous and elderly (Reprinted from Singh et al. [10], Copyright 2003, with permission from Elsevier)

these muscles can occur, particularly to the pubococcygeus, since it is the shortest and most medial muscle of the complex [7–9].

Levator trauma can lead to widening of the genital hiatus and thus be a risk factor for pelvic organ prolapse. It can also be associated with urinary incontinence.

Imaging studies may help in elucidating the types of injuries of levator ani muscle, especially transvaginal or transperinaeal ultrasonography and magnetic resonance imaging of the pelvis with or without three-dimensional reconstruction [7–9].

In three-dimensional reconstruction, it is possible to observe the distinct anatomical difference of the levator ani when comparing nulliparous, asymptomatic multiparous, symptomatic multiparous and elderly, as shown in Fig. 7.2 [10].

Nerve Injury

Injury to pudendal nerve can also be associated with pelvic floor disorders, especially urinary and fecal incontinence. The pudendal nerve innervates the external urethral and anal sphincters. During labor, nerve compression and stretching can occur, leading to incontinence. This process is reversible in most cases, with complete return to continence in the postpartum period. Severe cases of injury can lead to persistent incontinence [11, 12].

Connective Tissue Remodeling

During pregnancy, collagen and elastin, components of connective tissue, experience some modifications in order to increase vaginal distensiblity [13].

During labor, extensive stretching promotes collagen degradation. The endopelvic fascia and other connective tissue elements are at risk of stretch and detachment from their bony attachments during childbirth [13]. Pubic bone edema and subcortical fracture are common, and magnetic resonance shows that they persist until 7

weeks after delivery without clinical findings [14]. In rare cases, pubic symphysis rupture can occur [15].

After delivery, there is a substantial remodeling of the connective tissue components. However, this new tissue is not as strong as the original [13].

Injuries at level I of DeLancey are responsible for the appearance of prolapse of the uterus. The increase in intra-abdominal pressure generated by the pregnancy itself can cause rupture or stretching of these structures, especially if it is a twin pregnancy, macrosomic fetus or increased amniotic fluid as in gestational diabetes.

At level I, proximal transverse defect may occur with a detachment of the rectovaginal fascia from the pericervical ring, leading to the descent of the small bowel, omentum or sigmoid through the vaginal canal, called enterocele or protrusion of the rectum, called high rectocele [3, 16].

Structures of level II of DeLancey in labor are molded to hold the fetus. However, especially in cases of large fetuses or prolonged labor or even accelerated labor, rectovaginal fascia can break or stretch or it can detach from arcus tendineus. These injuries may result in the appearance of rectoceles in varied degrees and types, associated with central or transverse rupture of fascia or side rupture of fascia [17].

At level II, if there is a lesion of pubocervical fascia, the anterior vaginal wall may protrude into the vagina. When this prolapse involves the protrusion of the bladder it is called cystocele. Rarely, enterocele may occur via anterior vaginal wall, but is anatomically classified as apical prolapse; it originates from the detachment of the pubocervical fascia from the pericervical ring [7, 17]. Moreover, vaginal childbirth is associated with loss of tenting of the vaginal fornices, independent of levator trauma, and also with impaired anterior vaginal wall support. The existence of paravaginal defects may imply a role for such defects in the causation of anterior vaginal wall prolapse [18].

The expulsion phase of labor can cause injuries to level III of DeLancey as pubocervical fascia and the urethra can prolapse into the vaginal lumen, called urethrocele. In some cases it can cause hypermobility of the bladder neck and stress urinary incontinence (usually during medium or large efforts). Still, if there is injury to the urethral sphincter during the expulsion phase, it will decrease the intra-urethral pressure and, therefore, also cause stress urinary incontinence (usually during minimal efforts) [7, 17].

The second stage of labor can also be associated with level III lesions in the posterior compartment. Lesions in the rectovaginal fascia at this level cause rectoceles. Lesions in the perineal body and perineal muscles can cause perineal rupture. In these cases the patient complains of "a large or gaping vagina." If the anal sphincter is affected, the patient may develop fecal incontinence [19].

Table 7.1 summarizes the time of pregnancy or childbirth, the types of injuries that can occur according to DeLancey levels and their clinical consequences.

It is important to note that in some cases urinary urgency and urgency incontinence/overactive bladder can be caused by anterior vaginal prolapse. Vesical receptors present in the base of the bladder in contact with the vaginal epithelium are activated during bladder filling. However, in most cases, the etiology of overactive bladder is unknown [20].

Table 7.1 Types of injuries during labor

Pregnancy or childbirth	Levels of De Lancey	Place of injury	Diagnosis by ICS/IUGA(2011) and by region [16]
Pregnancy and primary stage of labor	Level I	Uterosacral ligament Cardinal ligament Pericervical ring	Apical prolapse: Uterine prolapse After total hysterectomy: vaginal vault prolapse After subtotal hysterectomy: cervix prolapse
		Pubocervical fascia	Anterior prolapse: Cystocele Enterocele (rare)
		Rectovaginal fascia	Enterocele High rectocele
Second stage of labor: active phase	Level II	Pubocervical fascia	Urethrocele Cystocele
		Rectovaginal fascia	Rectocele anal
Second stage of labor: expulsive phase	Level III	Pubocervical fascia	Urethrocele Stress urinary incontinence by hypermobility of bladder neck
		Urethral sphincter	Stress urinary incontinence by intrinsic sphincter deficiency
		Rectovaginal fascia	Rectocele anal
		Perineal body	Perineal rupture (if there is lesion of sphincter can cause fecal incontinence)

Obstetric and Maternal Factors in Pelvic Floor Disorders

The use of forceps appears to increase the risk of pelvic organ prolapse and anal sphincter laceration, which increases the risk of fecal incontinence. Forceps delivery can affect the muscles of the levator ani (OR = 14.7 and 95 % CI: 4.9–44.3). When there is injury of these muscles, a concomitant injury of the external anal sphincter may occur (OR = 8.1 and 95 % CI: 3.3–19.5). Women with levator injury were 3.5 years older in a study by Kearney et al. and had a 78-min longer second stage of labor [7].

Episiotomy is discussed extensively elsewhere in this book. The role of episiotomy on pelvic floor disorders is unclear. The routine use of episiotomy is decreasing, and there is no evidence that this procedure prevents pelvic floor dysfunction.

Levator tear during labor is associated with levator weakness and posterior-vaginal wall descent [14].

Third-degree perineal tear is when the external anal sphincter is affected; fourth-degree tear is when external and internal anal sphincter and rectal mucosa are

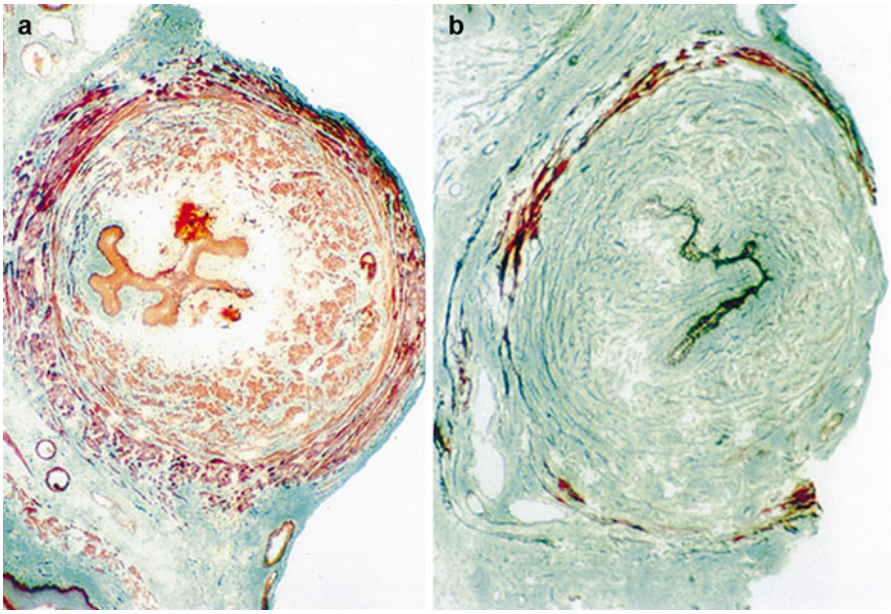

Fig. 7.3 Rat urethra after simulated birth trauma. Cross-section of the midurethra from (**a**) a continent rat and (**b**) an incontinent rat. The first one shows abundant smooth and striated muscle (**a**); the last one, a marked decrease (**b**). Trichrome stain, original magnification ×40 (Reprinted from Lin et al. [24], Copyright 1998, with permission from Elsevier)

affected. Risk factors for these lesions are primiparity (OR = 1.8 and 95 % CI: 1.65–1.95), Asian ethnicity (OR = 1.1 and 95 % CI: 1,09–1,23), use of forceps delivery (OR = 1.8 and 95 % CI: 1.65–1.95) and male fetus (OR = 1.3 and 95 % CI: 1.27–1.34) [19].

Both third- and fourth-degree perineal tears and vaginal sidewall tears are independently associated with levator avulsion (p = 0.004 and 0.012, respectively) and consequently future pelvic floor disorder [21]. Meta-analysis of non-randomised studies showed a significant reduction in the risk of obstetric anal sphincter injuries with manual perineal support [22].

Increasing maternal age and birth weight are associated with PFD [23].

A prolonged second stage may increase soft tissue injury and neuromuscular damage to the pelvic floor. Childbirth injuries to the pelvic floor are also found in experimental rat models. After the simulation of labor with vaginal balloon, rats can develop urinary incontinence. The damage to the urethral sphincter is shown in Fig. 7.3, and the levator ani muscle is shown in Fig. 7.4 [24].

It is noteworthy that the lesions mentioned do not occur in all deliveries. Moreover, even after a cesarean section, the woman may develop genital prolapse or incontinence because these conditions occur after a combination of risk factors. Cesarean section has its own indications and cannot be indicated just for the prevention of pelvic floor injuries. Gestational urinary incontinence can be one of the

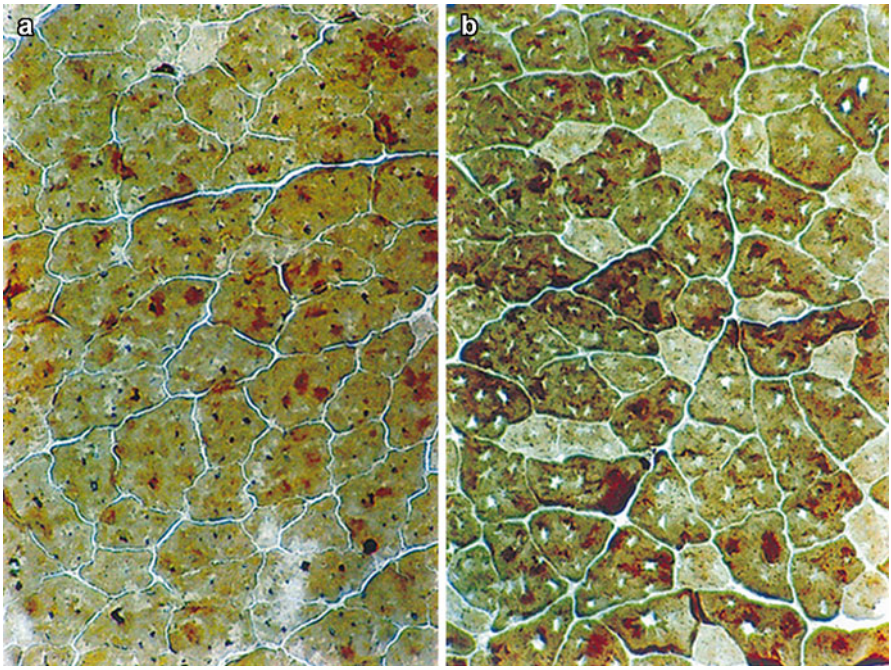

Fig. 7.4 Rat urethra after simulated birth trauma. ATPase stain of the levator muscle from (**a**) a continent rat and (**b**) an incontinent rat. In the latter the amount of slow-twitch fiber (lighter stain) is increased (Reprinted from Lin et al. [24], Copyright 1998, with permission from Elsevier)

predictors of urinary incontinence immediately after delivery and up to 2 years after birth. Weight gain during pregnancy is a risk factor for pelvic floor muscle dysfunction [25, 26].

In most cases it is observed that the genital prolapse or incontinence does not immediately appear post-partum. Other risk factors like obesity, chronic cough, constipation, smoking, collagen diseases, and especially menopause need to be present too. At menopause there is a decrease of estrogen resulting in breakdown of collagen fibers. The structures already weakened, such as fascia, ligaments and muscles, no longer support the pelvic organs, which then herniate through the vaginal canal. In addition, genital atrophy increases the occurrence of urinary incontinence [27].

Prophylactic pelvic floor muscle exercises performed during pregnancy help to decrease the short-term risk of urinary incontinence, but there is limited evidence on its long-term benefits [26]. It is important to note that there are no significant changes in sexual function after childbirth trauma with levator avulsion [28].

Knowledge of the anatomical parameters of the pelvic floor is important for the understanding of urogenital disorders such as pelvic organ prolapse and urinary incontinence that can be secondary to childbirth trauma in order to try to prevent these pathologies or propose the most appropriate treatment.

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Principles of Assessment of Childbirth Injury

8

Maya Basu

Abstract

Perineal trauma is the most common type of maternal morbidity encountered by healthcare professionals, and it may result in significant effects on quality of life. An understanding of relevant anatomy is necessary for a full evaluation of the underlying trauma, and the extent of a tear will be classified according to the structures involved. Assessment requires good lighting, good analgesia and good positioning of the patient to ensure adequate visualization. The available evidence suggests that structured and multi-professional training enhances detection of severe perineal trauma. Limited evidence suggests that ultrasound imaging of the anal sphincter may also improve detection, but this is an area that will require further study to more precisely delineate its role in management.

Keywords

Perineum • Trauma • Postpartum • Anal sphincter • Assessment • Diagnosis • Endoanal ultrasound • Training

Introduction

Childbirth injury affects millions of women worldwide, and is the most common form of maternal morbidity encountered by obstetric and maternity healthcare professionals. Although childbirth injury has conventionally been taken to refer to perineal and vaginal trauma following delivery, this term can also be taken to include

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trauma to the levator ani muscles, which is a more recent concept. Recent population studies have reported that the incidence of perineal trauma is over 91 % in nulliparous women and over 70 % in multiparous women [1]. A clinical diagnosis of obstetric anal sphincter injury (OASIS) is made in between 1 and 11 % of women following vaginal delivery [2, 3]. There is evidence that increased awareness and training with regards to OASIS is associated with an increase in the reported incidence [2].

A good working knowledge of the assessment of childbirth trauma, both in the acute delivery room setting and in the later postnatal setting, is essential for any clinicians involved in obstetric care. Inadequate assessment may lead to incorrect diagnosis, with consequent inappropriate management. Both short- and long-term symptoms following repair of childbirth trauma can have a significant effect on daily functioning, psychological well-being and sexual function.

Assessment of Childbirth Trauma in the Delivery Room Setting

In the acute setting, injury to any segment of the female genital tract may be encountered. A systematic approach to assessment is necessary prior to any repair, in order to establish the type of repair needed, who should carry out the repair and where it should take place. Injury to the uterus and its lateral anatomical relations is outside the scope of this chapter and will not be discussed.

Certain women will be at higher risk of severe lower genital tract trauma, e.g., obstetric anal sphincter injury, and this should be considered in the assessment of women in the delivery room. The following are risk factors for spontaneous lower genital tract trauma [1]:

- Nulliparity
- Forceps delivery
- Duration of second stage
- Episiotomy
- Labour dystocia
- Macrosomia

Although these risk factors have been consistently identified in retrospective studies, most cannot be feasibly used to prevent or predict the occurrence of an obstetric anal sphincter injury [4].

Relevant Anatomy

The muscles of the pelvic floor, perineum or anal sphincter may be disrupted by childbirth trauma. The perineal body is a fibromuscular structure situated at the centre of the perineum, which acts as an insertion point for several different muscles. Fibres from muscles including the bulbospongiosus, external anal sphincter and superficial transverse perineal are incorporated into the perineal body. The most

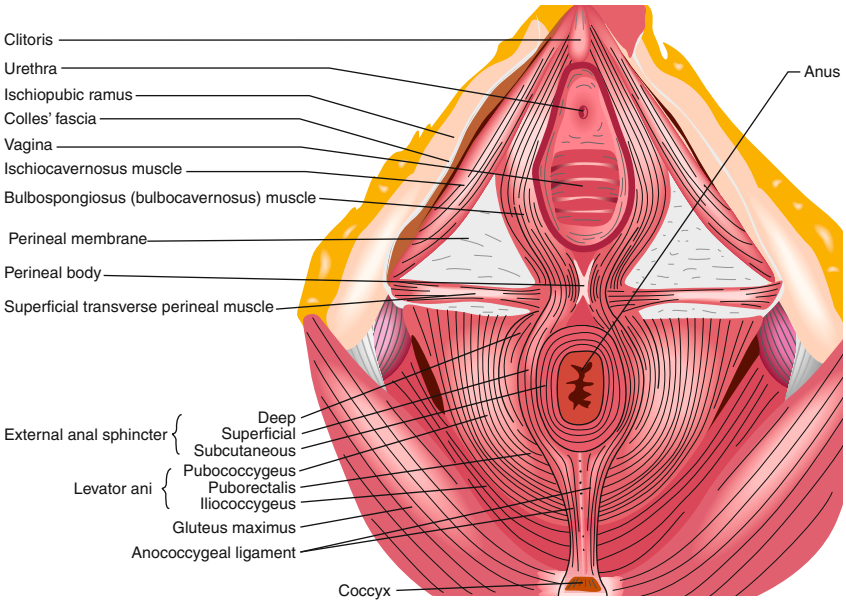

Fig. 8.1 Schematic of the perineal muscles (With kind permission from Springer Science + Business Media: Thakar and Fenner [12], p. 1–12)

superficial of the perineal muscles are the superficial transverse perineal muscle, bulbospongiosus and ischiocavernosus. The superficial transverse perineal muscle arises from the ischial tuberosity and inserts into the perineal body. The bulbospongiosus muscle runs either side of the introitus. The ischiocavernosus is situated on either side on the medial aspect of the ischiopubic ramus (Fig. 8.1).

Obstetric anal sphincter injury involves the muscles of the anal triangle. The anal canal is 3–4 cm long and is lined by an epithelial cell layer with the anal sphincter complex being situated externally. The anal sphincter complex is separated into external (EAS) and internal (IAS) components by a layer of fibromuscular and connective tissue. The EAS consists of striated muscle fibres and permits voluntary squeeze (via the pudendal nerve) as well as reflex contractions. The IAS consists of circular smooth muscle under autonomic control, and is responsible for the majority of the resting tone of the sphincter complex. The anatomical configuration of the anal sphincter complex is illustrated in Fig. 8.2.

The term "pelvic floor" is used to refer to a muscular layer that spans the pelvic outlet and is comprised mainly of the paired levator ani muscles, which are found deep to the muscles of the perineum and anal sphincter complex. These muscles arise from the arcus tendineous fascia pelvis on each side and are subdivided according to their bony attachments into three main portions- iliococcygeus, pubococcygeus and ischiococcygeus. Medial fibres from the pubococcygeus are arranged to form a hammock-like configuration around the rectum, and are designated the puborectalis.

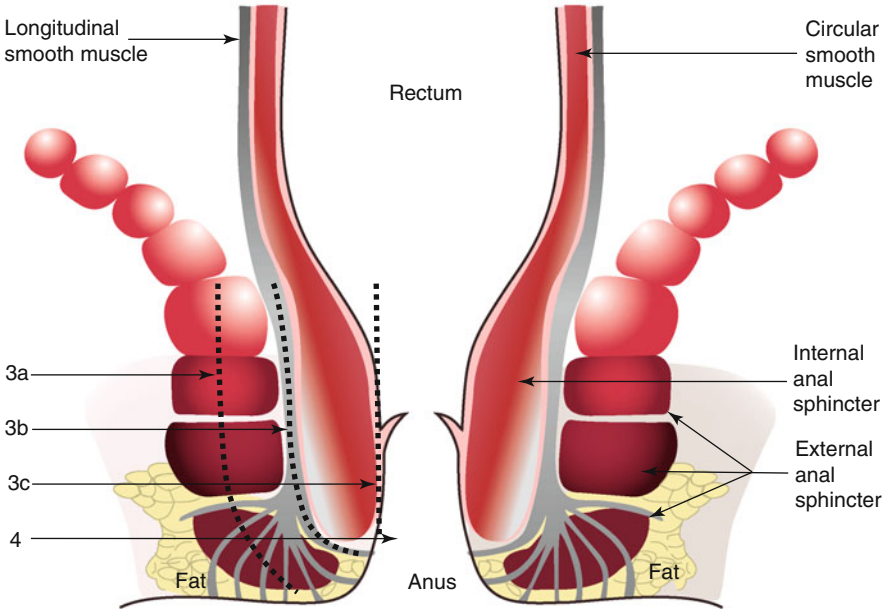

Fig. 8.2 The anal sphincter complex (With kind permission from Springer Science + Business Media: Sultan and Kettle [13], p. 13–19)

Perineal trauma may occur spontaneously with vaginal delivery, or it may be iatrogenic, i.e., an episiotomy. An episiotomy may also extend to involve other structures. Although perineal trauma is conventionally thought of as incorporating trauma to the posterior vaginal wall and the muscles of the perineum and anal sphincter complex, tears may also be seen which involve the anterior vaginal wall, urethra, clitoris or labia. Tears can sometimes be highly complex, involving multiple compartments of the vagina in a "spiral" fashion, or with complete detachment of the vaginal epithelium from underlying structures.

The following classification has been recommended for use in the assessment of perineal trauma [2]:

- *First degree*: Injury to the vaginal epithelium or perineal skin only
- *Second degree*: Involvement of the superficial perineal muscles (bulbospongiosus, transverse perineal) and sometimes the pubococcygeus muscle, but with no involvement of the anal sphincter.
- *Third degree*: Involvement of the anal sphincter complex (Fig. 8.3). Can be further subdivided into:

3A	Less than 50 % thickness of external anal sphincter torn
3B	More than 50 % thickness of external anal sphincter torn
3C	External and internal anal sphincters torn

Fig. 8.3 External view of a third-degree tear

- *Fourth Degree*: A third-degree tear with additional involvement of the anorectal mucosa

A *buttonhole* tear refers to an isolated tear of the rectal mucosa into the vagina, without involvement of the anal sphincters. These sit outside of the classification above as they do not involve the muscles of the perineum. They may be difficult to detect without a thorough assessment including a digital rectal examination. Detection is essential in order to avoid debilitating consequences such as rectovaginal fistula, although there is no association between buttonhole tears and continence outcomes.

Prior to assessment for genital tract trauma, it is recommended that the examining clinician ensures the following criteria are met [5]:

1. The woman understands what will be done and why (including specific verbal consent for a vaginal and rectal examination).
2. Effective analgesia has been provided (epidural or inhalational).
3. Lighting is adequate.
4. The woman is positioned comfortably such that the genital structures can be clearly visualized; the lithotomy position may be necessary to facilitate good visualization if there are deep or complex tears.

Once these criteria are met, a vaginal examination should be performed to evaluate the extent of the vaginal tear, plus visualization of the perineum.

- Although parting the labia is usually adequate to visualize the tear, complex or deep tears may necessitate the use of a Sims speculum to identify the apex of the tear(s).
- A Sims speculum and two sponge-holding (Rampley's) forceps will be necessary for evaluation if a cervical tear is suspected; the sponge-holding forceps should be used to gently grasp the cervix in quadrants in order to systematically inspect for any disruption.
- Following a full vaginal examination, a digital rectal examination should be performed to evaluate the integrity of the anal sphincter complex, and to exclude buttonhole tears of the anal epithelium.

- Clear visualization is necessary to diagnose a third- or fourth-degree tear.
- The EAS is striated muscle that is red in appearance; the IAS is smooth muscle and is paler in appearance.
- The examiner's index finger should be inserted into the rectum and the labia parted with the other hand to inspect for any tears to the EAS and IAS (remembering that anal sphincter damage can still be present with an intact perineum).
- The woman can be asked to contract her anal sphincter around the examiners finger; a defect will be felt anteriorly if there is anal sphincter damage.
- If there is any doubt about whether a tear involves the anal sphincter complex, an assessment should be undertaken by a more experienced professional.
- If adequate assessment cannot be undertaken to exclude anal sphincter trauma due to inadequate analgesia, it may sometimes be necessary to consider additional measures such as pudendal or regional blockade.

Once a full assessment has been undertaken by a suitably experienced individual as outlined above, the tear can be classified and arrangements for repair made. Repair should be undertaken by a trained healthcare professional in a suitable environment. The exact requirements will depend on the classification of the tear and the comfort and analgesia requirements of the patient.

Ultrasound as an Assessment Method in the Acute Setting

Although clinical assessment remains the most commonly used modality to detect and correcty classify perineal trauma in the immediate postpartum period, other methods, such as ultrasound, have been explored. Figure 8.4 shows the normal ultrasonographic appearance of the anal sphincter complex.

An evaluation of endoanal ultrasound performed postpartum in 150 primiparous women revealed clinically undiagnosed anal sphincter defects to be present in 28 %

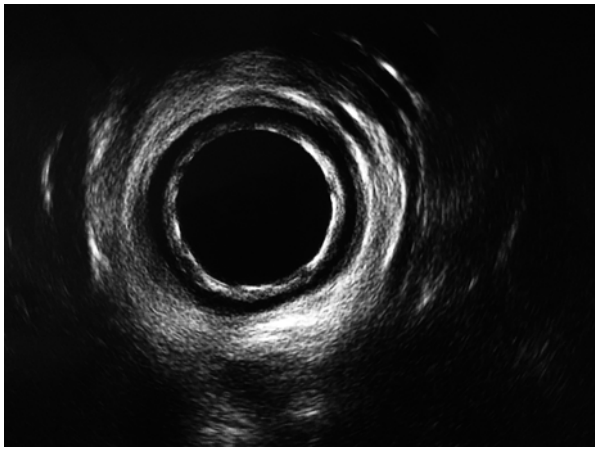

Fig. 8.4 Endoanal ultrasound images showing normal appearance of the internal (IAS) and external (EAS) anal sphincters

[6]. The sensitivity of anal endosonography was 68 % and the positive predictive value 37 %. A subsequent randomized trial evaluating endoanal ultrasonography against standard clinical assessment showed postpartum ultrasound examination to be associated with a significant improvement in diagnosis of anal sphincter tears, together with a lower incidence of faecal incontinence at 3 months [7]. A further study of 154 primiparous women who underwent a transperineal ultrasound scan 6–24 h after their first delivery found ultrasound evidence of anal sphincter damage to be associated with a higher risk of anal incontinence symptoms up to 6 months after delivery [8].

The limited available evidence does seem to suggest that ultrasound evaluation of the anal sphincter complex improves detection of anal sphincter injuries in the immediate postnatal period, however widespread use of this tool is influenced by resource and training limitations. Therefore drives to improve care of women with perineal trauma tend to focus on optimizing clinical assessment.

Improving Clinical Assessment of Perineal Trauma

Over recent years, the focus has been on improving detection of severe perineal trauma (i.e., injury to the anal sphincter complex) by training in standardized assessment methods. The rationale behind this is that improved detection will ensure adequate repair. An evaluation of clinicians who attended a structured training course in the detection and management of obstetric anal sphincter injury reported improved identification and classification of tears after the course, together with a shift towards more evidence-based practice [9]. An analysis of third-degree tears repaired by doctors assessing and repairing tears after a structured training programme showed the incidence of persistent sphincter defects to be lower than that reported in the literature, with no significant deterioration in anal incontinence symptoms [10].

A randomized controlled trial of an enhanced cascaded multiprofessional training programme focusing on evidence based perineal management was conducted amongst 3681 women who sustained a second-degree tear. The primary outcome measure was perineal pain on sitting and walking at 10–12 days post partum. Although there was no difference between the intervention and no intervention for the primary outcome measure, implementation of the training programme was associated with significant improvements in evidence based practice; this again supports the use of structured training for perineal assessment [11].

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Management of Childbirth Injury

9

Maya Basu

Abstract

Perineal trauma is the most common form of morbidity experienced during childbirth. Accurate assessment by an appropriately qualified professional and adequate analgesia are essential before undertaking repair. Repair should be undertaken as soon as possible after delivery, with good lighting, and an aseptic technique. The principles of repair of second, third and fourth degree tears are outlined in this chapter. There appears to be insufficient evidence to recommend either overlapping or end to end repair of the external anal sphincter in terms of continence outcomes. Symptoms of anal incontinence are seen in up to 43 % of women following obstetric anal sphincter injury, but the aetiology of this is likely to be multifactorial. Careful debriefing and assessment post-natally is of importance, and this is ideally done within a specialized clinic such that persisting symptoms can be managed effectively in a standardized manner. Advice for management in subsequent pregnancies will depend on factors such as symptoms of anal incontinence, endoanal scan and anorectal manometry findings, as well as patient choice.

Keywords

Perineal trauma • Obstetric anal sphincter injury • Endoanal ultrasound scan • Perineal pain

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Introduction

Lower genital tract trauma is the most common form of morbidity seen in postnatal women. All practitioners caring for postnatal women therefore require a good knowledge of the management of women with all types of trauma, from first degree to fourth degree tears. In addition, women sustaining obstetric anal sphincter injuries will require assessment for, and management of sequelae including faecal incontinence. Planning for delivery in future pregnancies is also an essential component in such women. Complications such as perineal wound breakdown can be hugely distressing for women and so require careful management. In this chapter, an overview of pertinent management issues in women sustaining childbirth trauma will be discussed.

Management of Perineal Trauma in the Acute Setting

The first stage of managing any childbirth injury in the acute setting will be a comprehensive assessment of the structures involved in the injury by a suitably qualified practitioner. There is evidence that hands-on training workshops improve knowledge of perineal anatomy and recognition of anal sphincter injury [1], and training in recognition and repair of obstetric anal sphincter injury is now a mandatory component of obstetrics training in the UK. A prospective study reported sonographic evidence of persistent sphincter defects in 10 % of women following repair by doctors who had undergone structured training in repair of anal sphincter injuries, with no deterioration in symptoms at 1 year postnatal; this compares very favourably with previous reports of sphincter defects of up to 92 % [2].

Certain basic surgical principles should be adhered to when approaching repair of lower genital tract trauma [3]:

- Repair should be undertaken by an appropriately qualified practitioner.
- Repair of perineal tears should be undertaken as soon as possible after delivery to reduce the risk of bleeding and tissue oedema, which may make repair technically more difficult.
- Adequate analgesia making the woman comfortable enough to allow for visualization and good approximation of the tear is an essential step. Local anaesthesia with 1 % lidocaine, or an epidural top up are suitable options.
- Basic surgical principles of asepsis, good lighting and ensuring swab counts are correct should be followed.

To Suture or Not to Suture?

Whilst the general approach of most practitioners is to suture vaginal and perineal tears, the question of whether leaving the skin unsutured has been asked by some researchers. A trial of 80 women with first- or second-degree tears were

randomized to suturing or non-suturing (40 in each arm) and followed up at up to 6 months for pain and healing. There were no differences between the groups in terms of healing defects or pain scores, although more women in the sutured group visited the midwife in the early postnatal period because of discomfort [4]. This study should be viewed with caution however as non-standardised instruments were used for data collection. A later randomized trial of suturing versus non-suturing of first and second degree tears reported a significant difference in healing between the two groups, with women in the non-sutured group having a significantly higher incidence of poor wound approximation [5]. There was no difference in pain scores and depressive symptoms between the two groups; however the findings of this trial should also be interpreted with caution since the eventual sample size led to it being underpowered. A non-randomised observational study of pain and pelvic floor function in 172 women with sutured second degree tears, non-sutured perineal tears and intact perineums reported an increase in analgesic use in women with sutured second degree tears in the early postnatal period, but no differences in pain, resumption of sexual activity and bladder and bowel function at 12 weeks' postpartum [6].

Other authors have focused on whether the presence of sutures in the skin causes excess pain. A randomized trial of 1780 women with a first- or second-degree tear or an episiotomy compared leaving the skin unsutured with skin closure using interrupted or subcuticular sutures. At up to 10 days postpartum there was no difference in the incidence of perineal pain between the two groups, although at 3 months there was a higher incidence of pain in the sutured group [7]. There was also a higher incidence of dyspareunia in the sutured group, with fewer patients having resumed sexual activity in this group. Conversely a trial of 400 women randomized to skin suturing or not suturing reported no differences in pain scores, wound gaping, dyspareunia and patient satisfaction between the two groups [8].

Overall, there does not seem to be enough consistent evidence to support a change in practice of leaving perineal trauma unsutured. More randomized studies allowing for meta-analysis are necessary to answer this question.

What Type of Suture Should Be Used?

There have been a relatively large number of studies on this issue, meaning that the level of evidence for this question is high. The question of whether chromic catgut or the now more commonly used multifilament polyglactin 910 should be used has been addressed in several randomized trials, which have reported less short term pain in the polyglactin 910 group with a trend towards a lower requirement for resuturing [9]. A head to head trial of chromic catgut versus rapidly absorbed polyglactin found significantly less short term pain, wound dehiscence, wound infection and discomfort from the sutures in the rapidly absorbed polyglactin group [10]. A systematic review of the available trials has confirmed a lower incidence of short term pain with absorbable synthetic sutures [11]. Other trials have evaluated standard polyglactin 910 versus rapidly absorbed polyglactin 910. In one large trial there was

no difference in pain at 10 days or dypareunia at 3 months between the two groups, but there was a higher requirement for suture removal in the standard polyglactin 910 group [12].

Technique for Repair of First- or Second-Degree Tears

- After obtaining informed consent and ensuring adequate pain relief, the woman should be positioned such that the vaginal and perineal components of the tear can be clearly and easily visualised; this may require the lithotomy position.
- A digital rectal examination should be carried out prior to repair to check for anal sphincter trauma or buttonhole tears between the vagina and rectum.
- The first suture should be inserted and tied above the apex of the vaginal component of the tear to ensure haemostasis.
- The vaginal part of the wound should then be sutured with a continuous, non-locking technique; this has been found to be associated with less pain and dyspareunia than interrupted sutures [13].
- The perineal muscles should be apposed and sutured with the same continuous suture, aiming to approximate the muscle such that the skin edges can be closed without tension; if the defect in the muscle layer is deep, this may require two layers of continuous sutures.
- The perineal skin should be closed with a continuous subcuticular suture.
- Following repair, a vaginal and rectal examination should be carried out to ensure the repair is complete, and that there is no other trauma.

Technique for the Repair of Third- and Fourth-Degree Tears [14]

- In the case of third- and fourth-degree tears, repair should be undertaken by a clinician who has undergone formal training and attained competence in repair of obstetric anal sphincter injury.
- A trial of immediate versus delayed repair (8–12 h) concluded that there is no difference in functional outcome if the repair is delayed [15], e.g., because of lack of trained staff; however, it is good practice to repair perineal trauma as soon as possible after the delivery.
- Repair should be carried out in an operating theatre environment under regional or general anaesthesia; this permits muscle relaxation, which allows for easier identification of the torn ends of the anal sphincter.
- A pre-repair rectal examination may identify a buttonhole tear between the rectum and vagina; this should be repaired using two layers of interrupted polyglactin sutures to minimise the risk of a fistula. In the case of gross faecal contamination of the wound, an opinion should be sought from a colorectal surgeon.
- In the case of a fourth-degree tear, trauma to the anal epithelium should be repaired with interrupted 3/0 polyglactin sutures with the knots tied in the anal lumen [14].

- Any trauma to the internal anal sphincter should be repaired separately with interrupted sutures using a fine suture such as 3/0 polydioxanone (PDS) or polyglactin. Separate identification and repair of the internal anal sphincter is associated with better continence outcomes [16].
- The torn ends of the external anal sphincter are held with Allis tissue forceps and sutured using either an overlap (if the muscle is completely torn, i.e., 3B/3C) or end-to end approximation (see below); a systematic review found no difference in perineal pain, dyspareunia, faecal incontinence or flatal incontinence between the two techniques, although there is some evidence of a lower incidence of faecal urgency and lower anal incontinence symptom scores in the overlap group [17].
- Following repair of the sphincter, it is important to perform a robust reconstruction of the perineal body to provide support to the repaired sphincter muscles and to minimise the risk of the perineum being deficient and more vulnerable to subsequent trauma.
- Repair of the vagina and perineum should proceed as for a second degree tear.
- A rectal examination should be carried out to ensure that the repair is complete and that no sutures have been placed inadvertently through the rectal mucosa.
- An indwelling catheter should be left in the bladder for 12–24 h.
- Patients should be given an intravenous dose of broad spectrum antibiotics (including cover for anaerobic organisms) at the time of the repair, plus oral antibiotics for 5–7 days after, since the development of infection and breakdown around the sphincter repair will result in a high risk of fistula formation or anal incontinence [18].
- Comprehensive documentation of the extent of the tear and the type of repair undertaken, including diagrams, is useful for debriefing the woman at a later date, and also in the case of potential litigation.
- Laxatives are recommended in the post-natal period to avoid passage of a hard stool, which could disrupt the repair. The use of a laxative will lead to earlier and less painful bowel opening when compared with a constipating agent. Clinical guidelines recommend the use of a stool softener plus a bulking agent for 10 days [14]; however, there is some evidence that there is a higher incidence of anal incontinence in the early postnatal period with this regime compared to stool softeners alone [19]. There are no long-term differences in long-term symptoms or pain between the two regimes.
- Good pain relief should be prescribed for the post-operative period, avoiding the use of constipating agents if possible. Evidence supports the use of diclofenac suppositories following perineal repair [20], but there are no trials evaluating its use in third- and fourth-degree tears specifically.

Techniques for Repairing the External Anal Sphincter: Overlap or End to End?

If the external sphincter is completely torn, reconstruction may proceed by either overlapping one end over the other or by approximating the two torn edges together. As mentioned above, there have been a number of studies evaluating whether either one of these techniques is associated with superior outcomes.

A randomised trial of primiparous women who had sustained an obstetric anal sphincter injury with complete disruption to the external sphincter used validated questionnaires to assess rates of faecal and flatal incontinence in women undergoing an end to end repair versus an overlapping repair at 1, 2 and 3 years following delivery. At one year, there was a significantly lower risk of flatal incontinence in women who had undergone an end to end repair (31 % vs. 56 %, p=0.01), with a trend towards a lower risk of faceal incontinence that did not reach significance (7 % versus 16 %, p=0.1) [21]. However there were no long term differences in symptoms between the two groups. An earlier study with a similar design evaluated symptom outcomes at 3 months postnatally, and identified no significant differences in faecal or flatal incontinence or urgency between the two methods of repair [22]. This study also evaluated the endoanal ultrasonographic appearance of the anal sphincter. Although there was no significant difference in the proportion of women with a significant (more than one quadrant) defect, it is interesting to note that more that two thirds of the cohort had a residual full thickness defect on imaging. Another smaller study of 64 women randomised to overlap or end to end repair again reported differing results. For the primary outcome measure of faecal incontinence, there was a significant difference favouring overlap repair (0 % vs. 24 %) [23]. Faecal urgency was also significantly more likely in the end to end group.

Overall, the available evidence is somewhat conflicting. A recent meta-analysis of the data available at the time reported that overlap repair was associated with a lower risk of anal incontinence [17]. However, the trials included did not take operator experience into account, and the authors therefore concluded that there is insufficient evidence to recommend one method over the other.

Continence Outcomes

There have been a number of cohort studies reporting outcomes after repair of obstetric anal sphincter injury. The reported incidence of anal incontinence following OASIS seems to be lower in more recent studies than in past studies, and this is likely to be due to improvements in training for recognition and repair.

In the first 12 months after delivery, symptoms of anal incontinence have been reported in approximately 20–43 % of women with a previous OASIS [24, 25]. Sonographic evidence of internal anal sphincter injury in the early postnatal period has been identified as a significant risk factor for the development of anal incontinence [24], underlining the importance of carefully identifying and repairing trauma to the internal anal sphincter. Other independent factors associated with a higher risk of anal incontinence include fourth-degree tears [25] and evidence of persistent sphincter defects. Interestingly, a prospective study evaluating risk factors for postnatal anal incontinence in a cohort of unselected women (i.e., including all modes of delivery, with and without perineal trauma) found that anal sphincter defects account for only 45 % of cases of anal incontinence overall [26]. This implies that the aetiology

of anal incontinence in postnatal women is multifactorial. Other factors such as pre-existing anal incontinence and intrapartum injury to the pudendal nerve may be relevant factors.

Follow-Up Care and Future Pregnancies

It is recommended that all women who sustain a third- or fourth-degree tear be seen by a senior obstetrician at between 6 and 12 weeks following delivery [14]. A more recent concept is that of a dedicated Perineal Clinic, where women can be seen by a professional with training in perineal trauma, with access to endoanal imaging and anal manometry; this allows for follow-up assessment and management of postnatal women as well as counselling of women antenatally with regards to mode of delivery [27]. Although there is no direct evidence to support mandatory assessment with anal manometry and ultrasonography, this has been suggested based on expert opinion, since endoanal sonography has been found to be more accurate than purely clinical assessment at diagnosis of sphincter defects [28]. There is a significant association between sonographic sphincter defects, anal incontinence symptom scores and low rest and squeeze pressure differentials [29, 30]. A multivariate analysis of risk factors for faecal incontinence following sphincter injury in 500 women showed significant internal anal sphincter defects specifically to be associated with the development of symptoms at 3 months [31]. Sonographic sphincter defects are also predictive of the development of faecal incontinence in later life [32].

Women with anal incontinence symptoms are managed according to the severity of their symptoms. Those with mild symptoms such as faecal urgency may be successfully managed by dietary modification, constipating agents and physiotherapy with bowel retraining and biofeedback. Women with more severe incontinence symptoms should be referred to a colorectal surgeon for further management. Secondary sphincter repair has been the surgical option for refractory symptoms for many years. Although this will usually lead to an improvement in symptoms, the rate of complete resolution of symptoms is poor [33]. This is likely to be because the aetiology of anal incontinence in post birth injury patients is likely to be multifactorial. A newer treatment for faecal incontinence is sacral nerve stimulation. An evaluation of medium term results from sacral nerve stimulation in faecally incontinent patients reported a decrease in median number of incontinence episodes from 11 per week to 0, with significant improvements seen in urgency [34]. A small study specifically evaluating the use of sacral nerve stimulation in 8 patients with faecal incontinence following obstetric anal sphincter injury reported similarly good outcomes at a median follow up of 26 months [35]. Sacral nerve stimulation has been found to be an effective treatment in the presence of pudendal neuropathy or after a previous sphincter repair [36].

The benefit of incorporating endoanal ultrasonography into the follow up assessment lies in the ability to use the information gained to advise on mode of delivery in subsequent pregnancies. A suggested pathway for the management of future pregnancies is given in Fig. 9.1 [38].

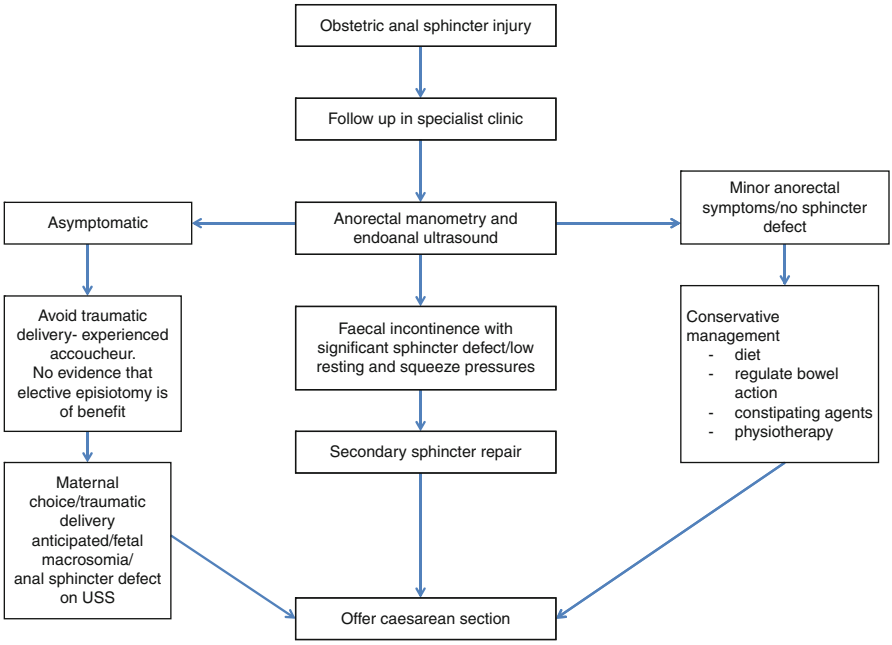

Fig. 9.1 Suggested algorithm for management of subsequent pregnancies after obstetric anal sphincter injury

Generally speaking, women with symptoms of anal incontinence may be offered delivery by caesarean section, since a second vaginal delivery may be associated with a deterioration in symptoms [37]. A study using validated questionnaires and endoanal ultrasonography to evaluate the effect of a second vaginal delivery found that significant sphincter defects were present in 53 % of women after their first delivery and were associated with higher anal incontinence scores. The risk of incontinence was highest in women with a significant sphincter defect who had undergone a second vaginal delivery (39 %) [39]. A further study of women undergoing a second vaginal delivery following a previous forceps delivery found a 26 % risk of developing new or worsening anal incontinence symptoms in women with a significant anal sphincter defect identified prior to the second delivery, although none developed severe symptoms [40]. This implies that women with an asymptomatic defect following obstetric anal sphincter injury may be offered delivery by caesarean section in subsequent pregnancy; however, there is currently no evidence that this will alter outcomes.

Management of Complications of Perineal Trauma

Although the healing process and complications are presented in detail in Chapter 13, we will outline here the salient points on the management of the common complications for ease of reference.

Perineal Pain and Dyspareunia

Perineal pain is common after any degree of perineal trauma and has been reported to affect 92 % of women, resolving by 2 months' postnatally in the majority of cases [41]. Whilst most perineal pain is self-limiting and manageable with simple analgesia, a small proportion of women will develop longer term symptoms. In the short term, women should be reassured and treated with analgesia as necessary. Perineal trauma is known to be associated with a decrease in sexual function at 6 months postpartum, with second degree tears leading to an 80 % increased risk of dyspareunia and third/fourth degree tears leading to a 270 % increased risk of dyspareunia [42]. Pain, refractory to conservative measures may be addressed with a variety of interventions, but there is no extensive evidence base to support these measures. Local perineal injections with hydrocortisone, marcaine and hyaluronidase are well tolerated and have been reported to lead to a significant fall in pain visual analogue scale scores (from 6.1 to 4.1) and resolution of dyspareunia in 89 % of sexually active women [43], but there is currently no randomised control trial evidence to support these data. There are very little other data in the literature on strategies for long term perineal pain in the obstetric population. Dyspareunia secondary to scarring or tightness at the fourchette following suturing is generally initially treated with dilators and topical oestrogens, but there are no data on outcomes of this intervention. In the presence of obvious scarring and anatomical constriction of the fourchette, women with refractory symptoms may be considered for a surgical revision of the perineum. This will generally involve a longitudinal incision of the scarred area with mobilisation of the underlying tissues and suturing transversely to ensure that a persistent skin bridge does not form. As with other interventions in this patient group, there is little in the literature on outcomes. A prospective study of 9 women who underwent a perineal revision for dyspareunia following perineal trauma reported a significant decrease in pain scores from 6.1 to 0.5 and an increase in coital frequency and satisfaction in 89 % of the cohort [44]. In the presence of associated spasm of the levator muscles, botulinum toxin injections to the levator muscles may also be an effective treatment [45].

Perineal Wound Infection and Breakdown

It is estimated that 11 % of women having had a perineal tear will have a wound infection [46], with prolonged rupture of membranes and instrumental delivery being significant risk factors. Administration of prophylactic antibiotics at the time of third/fourth-degree tear repair has been found to lead to a significantly lower risk of wound infection [47], which is important after a sphincter repair; however, women with second-degree tears are not routinely given antibiotics since there is no proven benefit. Wound infections should be treated with broad spectrum antibiotics including anaerobic cover, unless sensitivities based on wound swabs suggest otherwise. The majority of perineal infections will resolve with a course of antibiotics and good perineal hygiene. Perineal wound breakdown is less common, but can lead to considerable distress and impact on quality of life for affected women. As with other postnatal

perineal complications, there is a poor evidence base to guide management. By convention, most practitioners manage the wound expectantly, treating any infection, and awaiting healing by secondary intention. Women should be seen at intervals by an experienced professional to keep the wound under review and provide reassurance. A recent systematic review of suturing versus expectant management for perineal wound breakdown identified only two small randomised trials of poor quality [48]. Only one of these trials evaluated wound healing as a primary outcome measure, and although there was a trend towards better healing in the resuturing group, this did not reach statistical significance and the authors also did not specify how this outcome was quantified. The authors concluded that there is currently insufficient evidence to favour either treatment. An adequately powered randomised trial of these two interventions is underway to evaluate for the primary outcome measure of wound healing, in order to effectively evaluate these management strategies [49].

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Further Investigations and Follow-Up: Pelvic Floor Ultrasound

10

Ghazaleh Rostami Nia and S. Abbas Shobeiri

Abstract

The purpose of this chapter is to describe the 3D ultrasonography's utility in the assessment of pelvic floor trauma associated with vaginal delivery. These pathologies are associated with urinary incontinence and cystocele. 3D ultrasonography has been overlooked in favor of MR imaging. However, this technique can be a powerful tool in the hands of the obstetricians who suspect levator ani trauma.

Keywords

Ultrasound • Trauma • Pelvic floor

Introduction

Pregnancy and childbirth change the anatomy and function of the pelvic floor. Physicians who provide care to women can easily distinguish a multiparous from a nulliparous on pelvic examination. MRI-based computer modelling of the levator ani muscles have shown increase in length of the pubococcygeus fibers by a factor of 3 or more during crowning of the fetal head [1, 2]. The area of the minimal levator hiatus in young nulliparous women varies from 6 to 36 cm^2 on Valsalva maneuver

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[3]. The area of the average fetal head in the plane of minimal diameters measures 70–90 cm^2 (equivalent to a head circumference of 300–350 mm), requiring marked distension and deformation of the levator complex. Widened genital hiatus, less vaginal epithelium rugeation in addition to decreased muscle tone are most significant signs of multiparity during pelvic examination. There is ample scientific evidence that the vaginal childbirth process is associated with neuromuscular and soft tissue injuries to the pelvic floor. The pelvic organ support system is multifaceted and includes the endopelvic fascia, the perineal membrane, and the levator ani muscle that are controlled by nervous system. All these structures are at risk of injury in pregnancy and during vaginal delivery. These injuries can lead to short- and long-term pelvic floor structural changes. Pelvic organ prolapse, urinary and fecal incontinence, and chronic pelvic pain are considered inevitable sequelae for some women who experience injuries during birth. There is little ongoing debate about these findings and the focus has been on risk factor assessment and injury reducing interventions, however; this goal cannot be achieved without detailed understanding of structural and functional changes after delivery. Ultrasonography is taking an increasingly central role in defining birth related changes in pelvic floor support system. Levator ani muscle injury, levator hiatus enlargement, levator plate descent, widened anorectal angle and anal sphincter complex defects are examples of birth related traumas that can be diagnosed mostly by ultrasound techniques. Physical examinations even in very experienced hands are not precise enough for detection of these defects and are quite simply inadequate. The aim of this chapter is to review the role of ultrasound in evaluating the pelvic floor after pregnancy and labor.

Ultrasound

In recent years, with advances in magnetic resonance imaging (MRI) and three-dimensional (3D) ultrasound, we know that pelvic floor trauma goes beyond perineal, vaginal and anal sphincter lacerations which we can identify in the labor room. It has become evident that levator ani injury forms an underappreciated component of pelvic floor trauma. The levator ani muscle consists of three functional parts: the puboperineal and puboanal portions, the puborectalis, and iliococcygeus and pubococcygeus muscles which form the pubovisceralis complex [4]. Mechanisms of levator muscle injury became more clear with continuous works of Ashton-Miller and DeLancey on the biomechanics of vaginal birth [5]. Based on a geometric model they suggested that some muscle damage during the second stage of labor may come from overstretching [1]. The pubococcygeus portion of pubovisceralis muscle was the portion that underwent the greatest degree of stretch, and the second area of observed injury was iliococcygeus portion of the pubovisceralis.

Endoanal ultrasound was utilized by Sultan in 1993 using a series of elegant correlative histologic studies to document anal sphincter injury during labor [6, 7]. The same authors performed endovaginal ultrasound in 1994 to visualize the anal sphincter complex and incidentally visualized the levator ani muscles but the significance of these muscles at that point was unknown and they went largely unnoticed [8]. Shortly thereafter, the same group acknowledged the pelvic floor trauma during labor and suggested

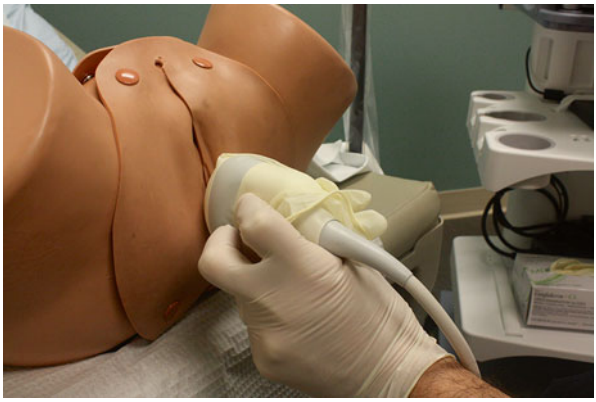


Fig. 10.1 A GE probe applied transperineally to obtain 3D/4D data volumes

strategies to protect the pelvic floor, perineum, and the anal sphincter during labor [9, 10]. Strohben reported on visualization of levator ani muscles using MRI with anatomic correlation in 1996 [11] and in 1999 Tunn reported on levator ani recovery after vaginal birth in 14 women [12]. In this group of patients, for the first time they reported injury to the levator ani muscle of one patient. Transperineal imaging was reported for visualization of the levator ani muscle activity in 2001 and concluded that this modality could be used to teach pelvic floor biofeedback (Fig. 10.1). The use of transperineal ultrasound for anal sphincter imaging was reported in 2002 [13] and concluded that digital examination could discriminate between an adequate and inadequate anal sphincter laceration repair. In 2005, Dietz et al. reported on the use of 3D transperineal ultrasound (TPUS) to document a 36 % incidence of levator ani avulsion which is the total separation of the levator ani from the pubic bone [14]. Unfortunately, TPUS had not been authenticated by cadaveric and histologic correlation and the authors' terminology named "puborectalis" as the injured muscle. Later on, levator ani muscle avulsion by TPUS was defined as an obvious detachment of the puborectalis muscle from the pelvic sidewall and if an abnormality was defined as defect seen in three or more slices [15] (Figs. 10.2 and 10.3). Different scoring systems with TPUS has been used to show the severity of muscle injury [16–18]. Zhuang et al. used the same terminology and defined full avulsion diagnosed if the "puborectalis"-to-ipsilateral sidewall attachment was not seen on any of the three central slices. Partial avulsion was diagnosed when the "puborectalis" attachment to the ipsilateral sidewall is not seen on at least one slide [19]. More recent studies have shown that the minimal levator hiatus is mostly lined with pubococcygeus [20] which is consistent with 3D modelling studies [1].

The description of the levator ani muscle subdivisions by 3D endovaginal ultrasonography (EVUS) was reported in 2008 and published in 2009 [21]. EVUS methodology and description of muscles was authenticated in a systematic manner. First the anatomic correlation [21] and subsequently histologic correlations were made in nulliparous women [22]. Interrater and interdisciplinary reliability of EVUS were subsequently described in nullipara [23]. Interrater reliability assessments of these measurements during pregnancy and postpartum have been performed.

Compared with other imaging modalities, ultrasound imaging is widely available, easy to perform, and familiar to many medical specialties. These advantages

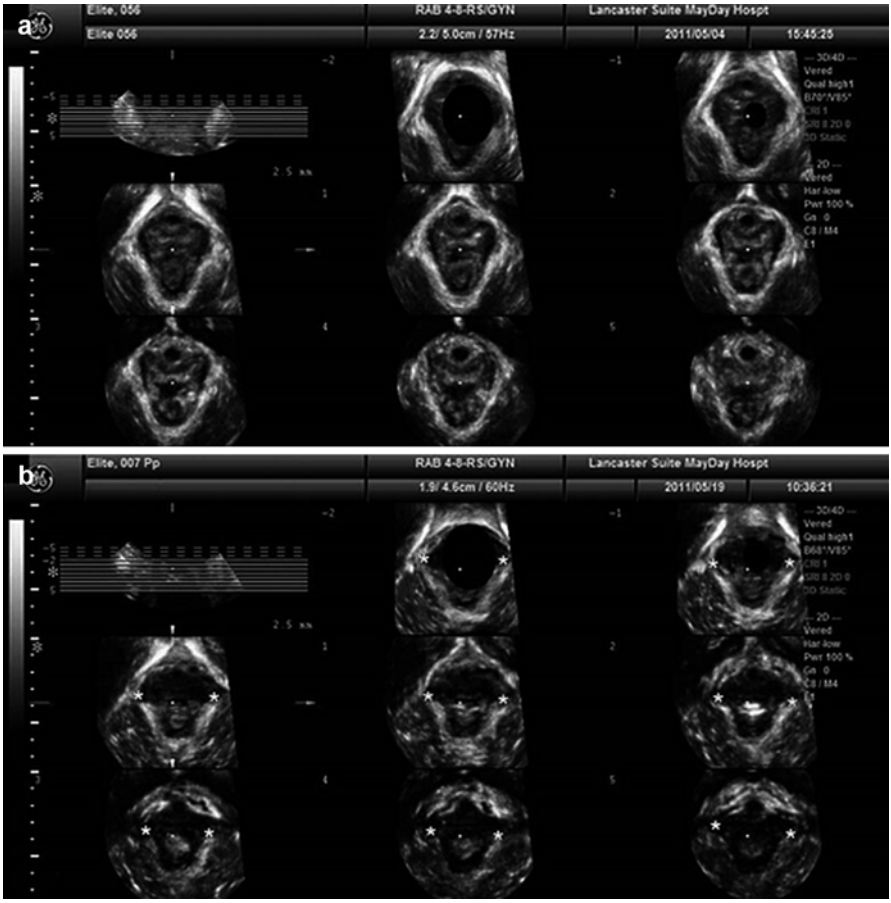


Fig. 10.2 Tomographic ultrasound images obtained using transperineal ultrasound of (**a**) a typical intact levator ani muscle (LAM) in a nulliparous woman and (**b**) a bilateral LAM avulsion in a multiparous woman. Slices were obtained at 2.5-mm intervals below and above the level of minimal hiatal dimension (*). LAM defects are indicated in (**b**) by * (Reprinted from Schwertner-Tiepelmann et al. [63]; with permission from John Wiley and Sons

have made it feasible for many researchers to study early postpartum pelvic floor injuries and structural changes and further correlate them with obstetric factors.

Levator Ani Muscle Trauma

During vaginal delivery, overstretching of levator ani muscle can predispose muscle to disconnect from its insertion on the inferior pubic ramus and pelvic side wall. Based on studies using 3D/4D pelvic floor ultrasound, the prevalence of levator ani muscle trauma after vaginal delivery is 13–40 % [14, 24, 25]. In a study of 114 postpartum women with EVUS, one third of primiparous women delivering vaginally developed levator ani muscle hematomas within hours of delivery diagnosed using high

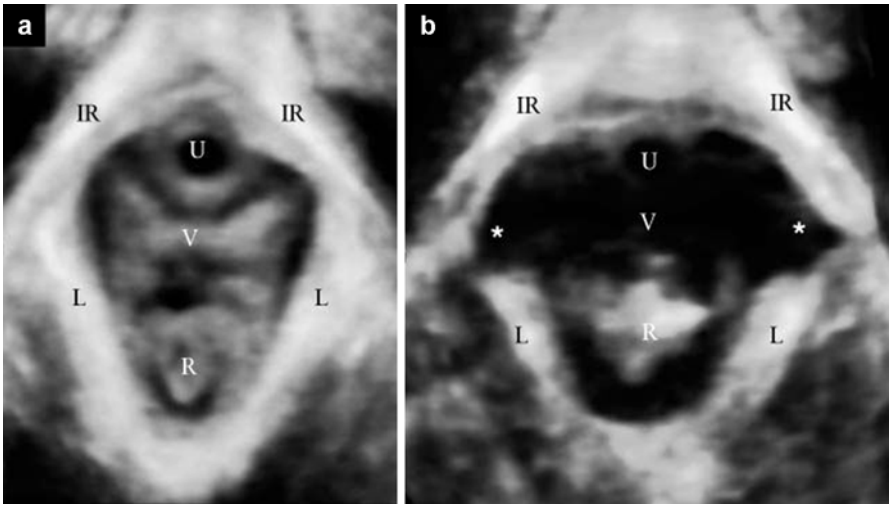


Fig. 10.3 Three-dimensional rendered volumes obtained on transperineal ultrasound showing: (**a**) an intact levator ani muscle (LAM) displayed in an oblique axial plane in a nulliparous woman and (**b**) a bilateral avulsion injury. LAM defects are indicated in (**b**) by (*). *IR* inferior ramus os pubis, *L* levator ani muscle, *R* rectum, *U* urethra, *V* vagina (Reprinted from Schwertner-Tiepelmann et al. [63]; with permission from John Wiley and Sons)

frequency EVUS. When hematoma was located in the attachment zone of the levator ani muscle to the pubic bone, levator ani muscle detachment from the pubic symphysis was almost always identified three months postpartum. When hematoma was located away from the attachment zone, a defect was most often not seen three months postpartum [26]. The mode of delivery has an impact on levator ani muscle injury. In a study on 157 postpartum women, the risk of levator defect after vaginal delivery was more than seven times higher than after cesarean section [27]. Instrumental delivery by forceps was one the most important risk factors as levator injury was detected in 60–64 % of women who had been delivered by forceps [28, 29]. Another study reported a prevalence of 18 % of levator lesions among women who had a non-instrumental vaginal delivery, 14 % among women who delivered by vacuum and 40 % among women who delivered by forceps, but reported no levator lesions in the cesarean delivery group [30]. Chan et al. showed similar findings in Chinese women after first vaginal delivery. The rate of muscle injury for spontaneous vaginal delivery, ventouse extraction and forceps delivery were 15.4 %, 33.3 %, and 71.4 %, respectively. There was no levator muscle injury in cesarean section groups [31].

EVUS Technique for Visualization of Levator Ani Muscle

3D endovaginal ultrasound can give us high-quality images of levator ani muscle subdivisions. It is not known if the EVUS can detect LA subdivisions better than MRI. Imaging is obtained using the BK Medical Flexfocus (Peabody, MA, USA) and a 2052/8838 transducer (Fig. 10.4). All ultrasound scans are

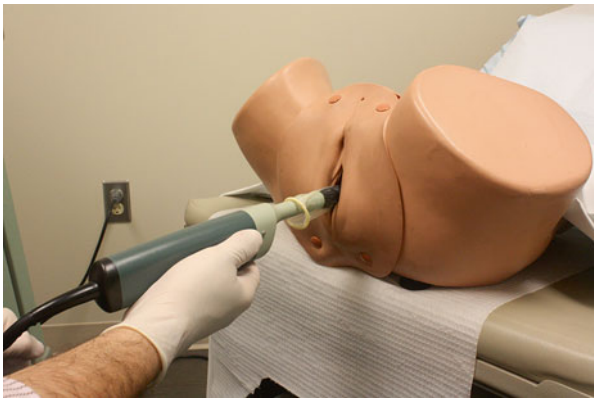


Fig. 10.4 A BK probe applied endovaginally to obtain 3D volume

performed in the office setting, with the patient in dorsal lithotomy position, with hips flexed and abducted. No preparation is required and the patient is recommended to have a comfortable volume of urine in the bladder. No rectal or vaginal contrast is used. To avoid excessive pressure on surrounding structures that might distort the anatomy, the probe is inserted into the vagina in a neutral position. It has been shown that endovaginal probe does not have any adverse effect on anatomy comparing to transperineal ultrasound [32]. Three hundred axial images over a distance of 6 cm are taken in 60 s; 360° EVUS volumes are digitally stored for further analysis.

The approach to 3-dimensional endovaginal ultrasound takes into account certain, easily recognizable anatomic landmarks [21]. We delineated three ascending levels with level 1 being the most caudal and level 3 the most cephalad (Fig. 10.5). This categorization was utilized for interrater reliability validation. Level 1 contained muscles that insert into the perineal body, namely the superficial transverse perinei, puboperinealis and puboanalis. The superficial transverse perinei served as a reference point (Fig. 10.6a, b). Level 2 contained the attachment of the pubovaginalis, puboperinealis, puboanalis and puborectalis, and iliococcygeus to the pubic bone (see Fig. 10.6c–f). Level 3 contained subdivisions visible cephalad to the inferior pubic ramus, namely the pubococcygeus and iliococcygeus, which winged out toward the ischial spine (see Fig. 10.6g–j). This standardized approach of endovaginal ultrasound assessment of levator ani muscle subdivisions has been verified with good to excellent interobserver and interdisciplinary reliability, with kappa values of 0.6–1 [21].

Levator Ani Deficiency

The majority of patients with pelvic floor disorders remote from delivery did not have evidence of birth-related injury at the level of the pubic symphysis, rather they had global atrophy and deficiency of the muscle. Based on functional

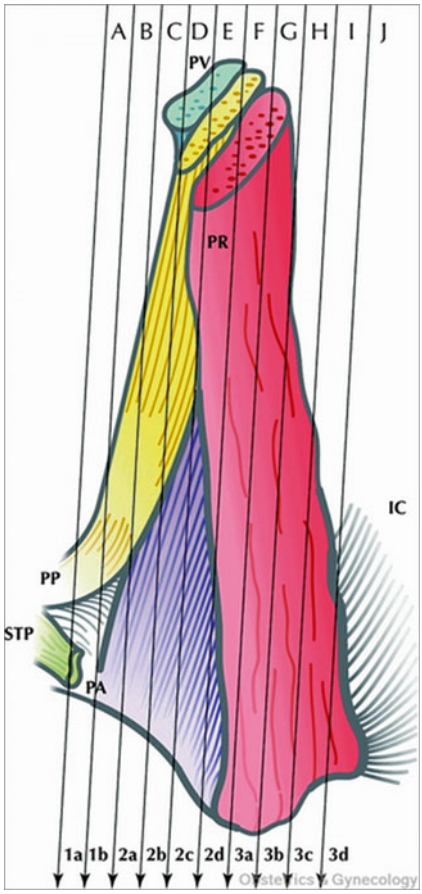
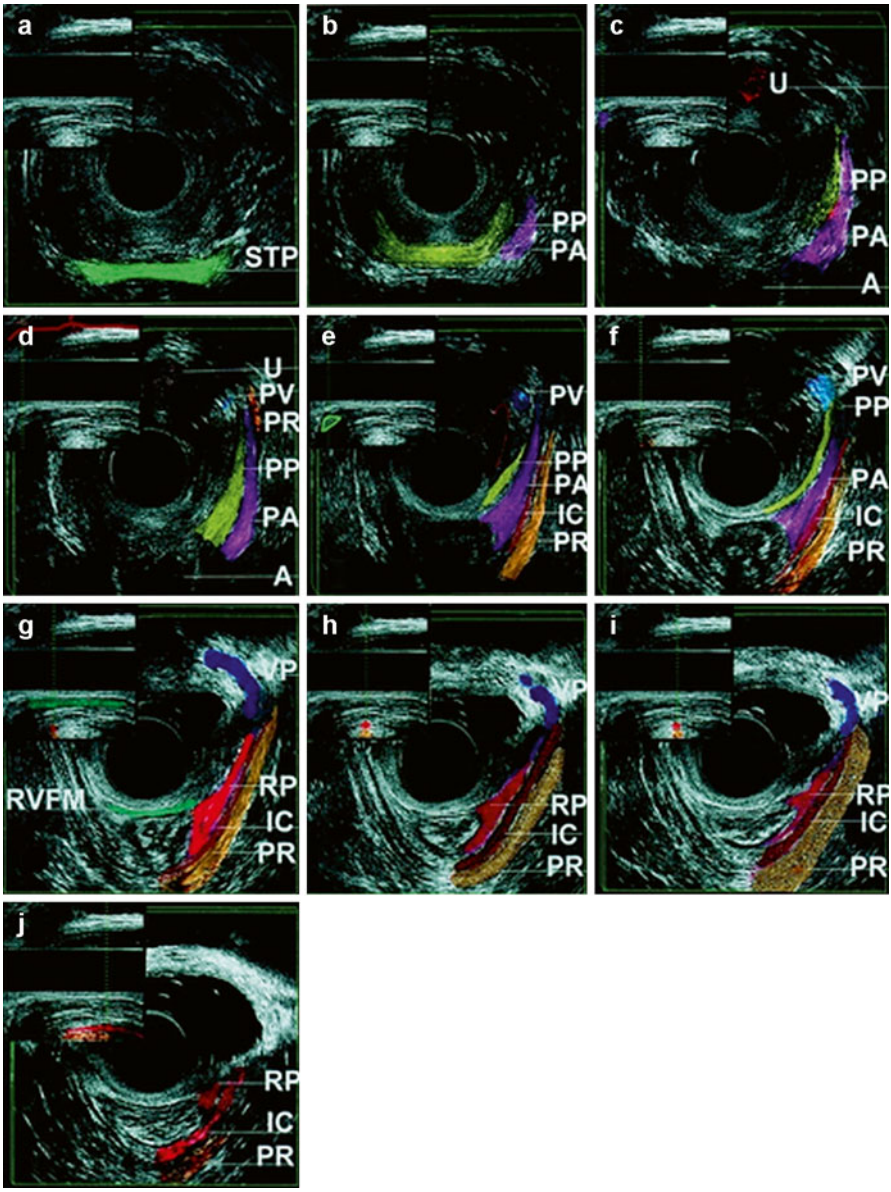


Fig. 10.5 The relative position of levator ani subdivisions during ultrasound imaging. Levels 1–3 are identified below the figure. The A-J markings on top of the figure correspond to the ultrasound images shown in Fig. 10.4. *IC* iliococcygeus, *PP* puboperinealis, *STP* superficial transverse perinea, *PA* puboanalis (Reprinted with permission from Shobeiri et al. [21])

anatomy of subdivisions of the levator ani muscle a scoring system for evaluation the severity of levator ani deficiency (LAD) was described for EVUS [33]. By EVUS, levator ani muscle subdivisions were evaluated in their specific axial plane where the full length of muscle could have been visualized and were scored (0 = no defect, 1 = minimal defect with <50 % muscle loss, 2 = major defect with >50 % muscle loss, 3 = total absence of the muscle) on each side based on thickness and detachment from the pubic bone as previously used in the MRI studies [34]. Each muscle pair score ranged from 0, indicating no defects, to maximum score of 6, indicating total muscle absence. For the entire levator ani muscle group, a cumulative levator ani deficiency (LAD) score that ranged between 0 and 18 was possible [33] (Fig. 10.7a–c). Although patients with normal support can have severe LAD, no patient with advanced prolapse demonstrates normal musculature.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Pelvic Floor Biometry

One of more recent applications of cross-sectional imaging of the female pelvic floor is pelvic floor biometry. Minimal levator hiatus dimensions, levator plate mobility, bladder neck mobility, urethral sphincter volume, anorectal angle, and pelvic organ mobility are the most common measurements that have been used in this area [20, 30, 35–38]. Researchers work on structural changes of the pelvic floor by using these measurements and try to find associations between these changes and pelvic floor dysfunction symptoms as they believe distorted anatomy can lead to malfunction.

3D ultrasound has made it feasible to visualize morphological changes of the pelvic floor after delivery. Levator hiatus distensibility, urethral sphincter volume and bladder neck mobility were assessed using ultrasound imaging in pregnancy, 6 weeks and 6 months after delivery in 156 women. It was shown that vaginal delivery is strongly associated with a larger, more distensible levator hiatus and a greater degree of bladder neck mobility both antenatally and postnatally [39]. Using 3D perineal ultrasound in 130 primiparous on second day postpartum, women with vaginal or operative vaginal delivery had a significantly larger hiatal area and transverse diameter than women who delivered by caesarean section [40]. Shek et al.

Fig. 10.6 Levator ani subdivisions seen at different levels. Midline structures are identified in lateral views with corresponding colors in the picture inserts at the upper left corner of the ultrasound images at each level. The green vertical line in the insert corresponds to the relative position in the vagina where the image was obtained. (**a**) Level 1A. At 0 cm, the first muscle seen is the superficial transverse perinei (*green*) with mixed echogenicity. (**b**) Level 1B. Immediately cephalad to the superficial transverse perinei is the puboperinealis (*yellow*), which can be traced to PB with manipulation of the three-dimensional cube. It comes in at a 45° angle as a mixed echoic band to join the perineal body. Lateral to it, the puboanalis is seen as a hypoechoic triangle (*pink*). (**c**) Level 2A marks the attachment of the muscles to the pubic arch. The external urethral meatus is visible (*dark red*). Puboperinealis and puboanalis insertions are highlighted. (**d**) Level 2B. Pubovaginalis (*blue*) and puborectalis (*mustard*) insertions come into view. The urethra and the bladder are outlined (*red*) in the lateral view. (**e**) Level 2C. The heart-shaped vaginal sulcus (*outlined in red*) marks the pubovaginalis insertion. Iliococcygeus fibers (*red*) come into view. The perineal body is outlined in the lateral view. (**f**) Level 2D. The puboanalis is starting to thin out. The puborectalis is seen in the lateral view. (**g**) Level 3A. The puboperinealis and puboanalis become obscure. Anatomically, the puboanalis becomes a thick, fibromuscular layer forming a tendineus sheet-the rectal pillar (RP). The perivesical venous plexus is prominent (*purple*). The rectovaginal fibromuscularis is shown (*green*) in the sagittal view as a continuous, mixed, echogenic structure approaching the perineal body and laterally attaching to the RP. (**h**) Level 3B. The RP (*orange*) is seen easily. The iliococcygeus becomes prominent and widens. (**i**) Level 3C. The iliococcygeus widens further and inserts into the arcus tendineus fascia pelvis. (**j**) Level 3D. The puborectalis and iliococcygeus fade out of view. The puborectalis (*mustard*) and iliococcygeus (*red*) are outlined in the lateral view, showing their entire course (Reprinted with permission from Shobeiri et al. [21])

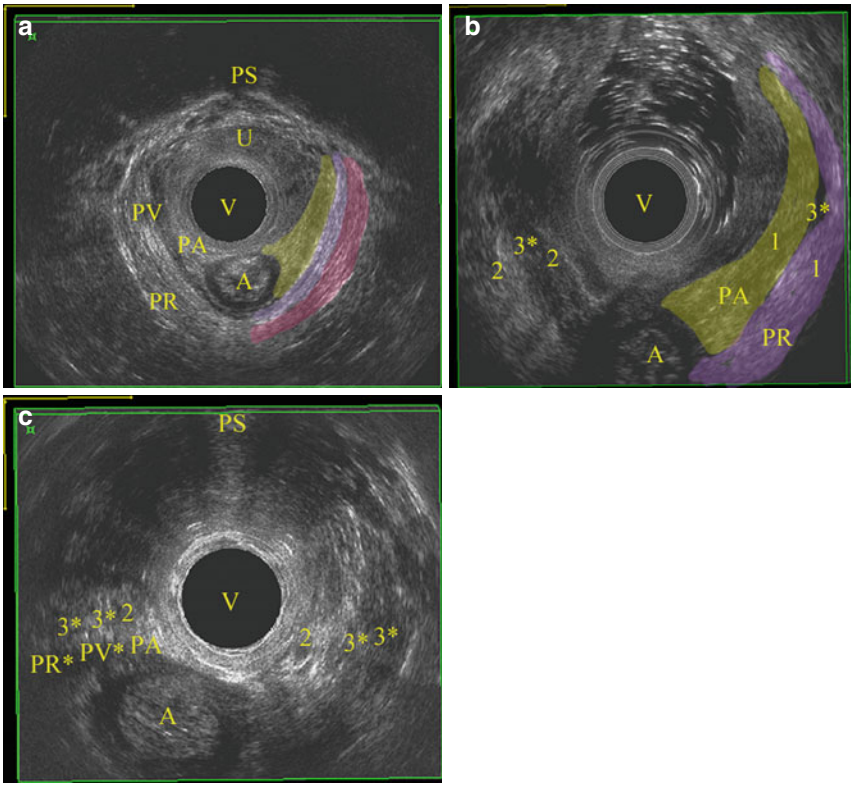

Fig. 10.7 (**a**) The axial view of pelvic floor muscles with no LA muscle deficiency. *A* anus, *LA* levator ani, *PA* puboperinealis/puboanalis, *PR* puborectalis, *PS* pubic symphysis, *PV* pubovisceralis, *V* vagina. * denotes a missing muscle. Numbers are muscle scores. (**b**) The axial view of pelvic floor muscles with moderate LA muscle deficiency. * denotes a missing muscle and numbers are muscle scores. *A* anus, *LA* levator ani, *PA* puboperinealis/puboanalis, *PR* puborectalis, *PS* pubic symphysis, *PV* pubovisceralis, *V* vagina.* denotes a missing muscle. Numbers are muscle scores. (**c**) The axial view of pelvic floor muscles with severe LA muscle deficiency. * denotes a missing muscle and numbers are muscle scores. *A* anus, *LA* levator ani, *PA* puboperinealis/puboanalis, *PR* puborectalis, *PS* pubic symphysis, *PV* pubovisceralis, *V* vagina. * denotes a missing muscle. Numbers are muscle scores (Reprinted with permission from Rostaminia et al. [33])

have shown that both hiatal dimensions and urethral mobility were markedly higher in late pregnancy and at 4 months after labour compared to nulliparous controls [41]. It was also shown that vaginal childbirth results in enlargement of the levator hiatus, especially after an avulsion. However, even without major levator trauma, there may be increased distensibility of the hiatus, which may be another mechanism leading to enlargement of the hiatus and pelvic organ prolapse [30]. Using 3-4D TPUS, levator hiatal area was significantly higher after forceps delivery [29]. Perineal body and anorectal junction mobility can be assessed by 3D pelvic floor ultrasound and it was shown than vaginal delivery increased the mobility of the perineal body and the anorectal junction [42].

3D EVUS Technique for Pelvic Floor Biometry

EVUS has been reliably used for pelvic floor biometry values such as minimal levator hiatus (MLH) and anorectal angle (ARA) with good to excellent interobserver and intradisciplinary reliability, with kappa values of 0.6–0.9 [23, 43].

Minimal Levator Hiatus Dimensions and Area

3D EVUS volumes obtained by 360° endovaginal probe has been used for this measurement. The mid-sagittal plane is used to identify the minimal distance between hyperechoic posterior aspect of the symphysis pubis and the hyperechogenic anterior border of the levator plate (Fig. 10.8). The shortest line between the levator plate and pubic symphysis corresponding to the anterior-posterior line or height of the minimal levator hiatus is drawn (Fig. 10.9). Ultrasound volume can be tilted and axial plane at the level of this line is used for minimal levator hiatus dimensions measurements (Fig. 10.10).

Puborectalis Hiatus

Puborectalis hiatus is not a term frequently used. However, it is important to emphasize that the points of attachment and direction of some of the puborectalis fibers are

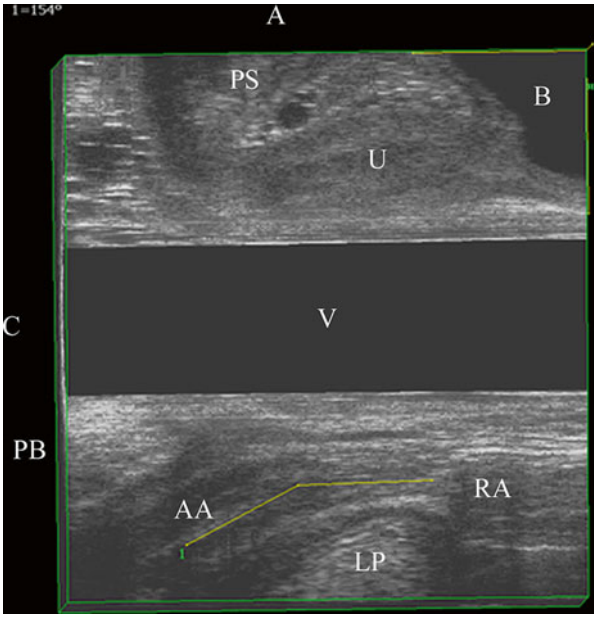

Fig. 10.8 Anorectal angle in mid-sagittal view by transvaginal 360° ultrasound. Anal axis (AA) and the rectal axis (RA) lines form the anorectal angle. *A* anterior, *B* bladder, *C* caudad, *LP* levator plate, *PS* pubic symphysis, *U* urethra, *V* vagina (Reprinted with permission from Shobeiri et al. [20])

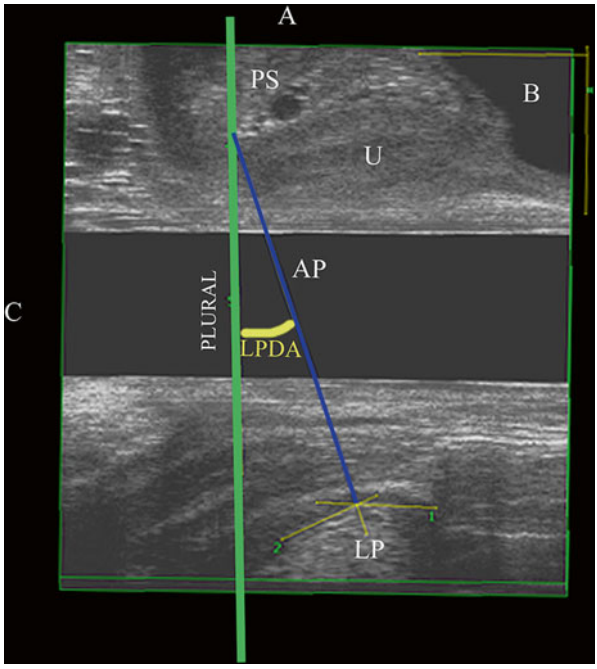

Fig. 10.9 Levator plate descent angle in mid-sagittal view by transvaginal 360° ultrasound. *A* anterior, *AP* antero-posterior line of minimal levator hiatus (*blue line*), *B* bladder, *C* caudad, *LP* levator plate, *LPDA* levator plate descent angle, *PLURAL* Pubic Levator Ultrasound Reference Assessment Line (*green line*), *PS* pubic symphysis, *U* urethra, *V* vagina (Reprinted with permission from Shobeiri et al. [20])

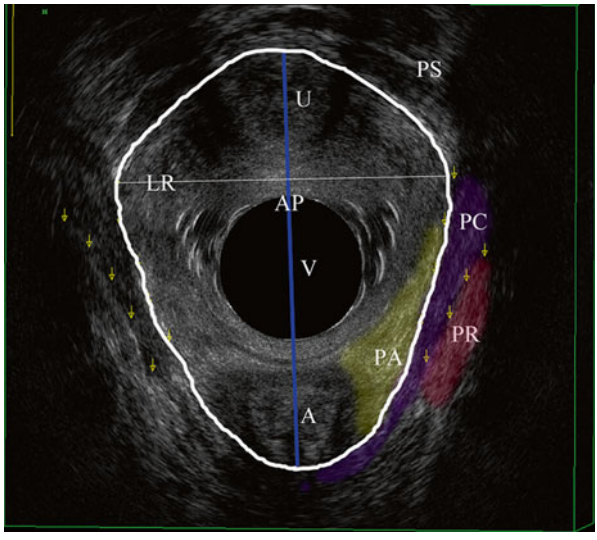

Fig. 10.10 Minimal levator hiatus area in axial plane by transvaginal 360° ultrasound. AP line is in blue. Puborectalis pubococcygeus border is delineated with small arrows. *A* anterior, *AP* antero-posterior line of minimal levator hiatus (*blue line*), *LR* left-right axis of minimal levator hiatus, *PC* pubococcygeus, *PR* puborectalis, *PS* pubic symphysis, *U* urethra, *V* vagina (Reprinted with permission from Shobeiri et al. [20])

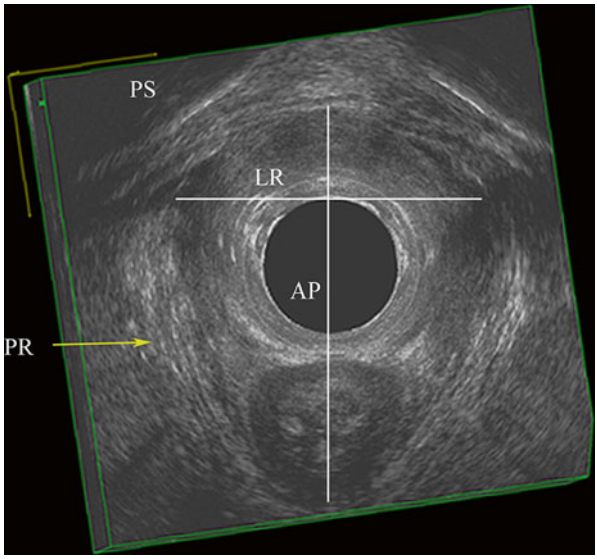

Fig. 10.11 Puborectalis hiatus in the same patient in axial view by transvaginal 360° ultrasound. *AP* antero-posterior line of minimal levator hiatus, *LR* left-right axis of minimal levator hiatus, *PR* puborectalis, *PS* pubic symphysis (Reprinted with permission from Shobeiri et al. [20])

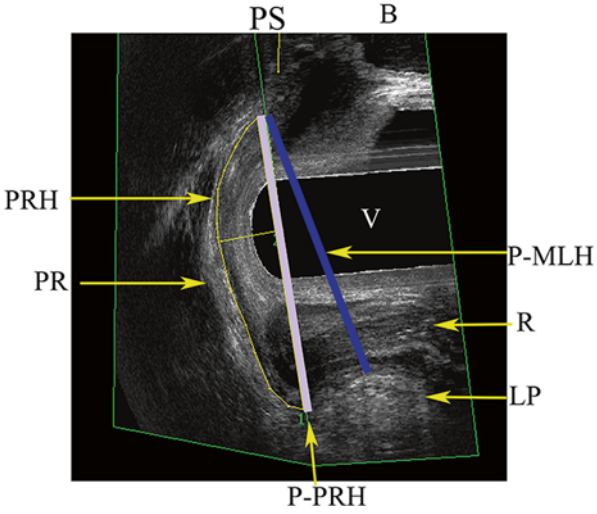

Fig. 10.12 Ultrasound showing plane of MLH and PRH in right sagittal view. *AP* antero-posterior line of minimal levator hiatus (*blue line*), *B* bladder, *LP* levator plate, *LR* left-right axis of minimal levator hiatus, *P-MLH* plane of minimal levator hiatus (*blue line*), *MLH* minimal levator hiatus, *P-PRH* plane of puborectalis hiatus (*purple line*), *PR* puborectalis, *PS* pubic symphysis, *R* rectum, *U* urethra, *V* vagina (Reprinted with permission from Shobeiri et al. [20])

different from that of the minimal levator hiatus (Fig. 10.11). Anteriorly, minimal levator hiatus is comprised of pubococcygeus fibers, although laterally some puborectalis fibers exist as well (Fig. 10.12). Puborectalis contributes significantly to the mid and distal edge of the levator plate.

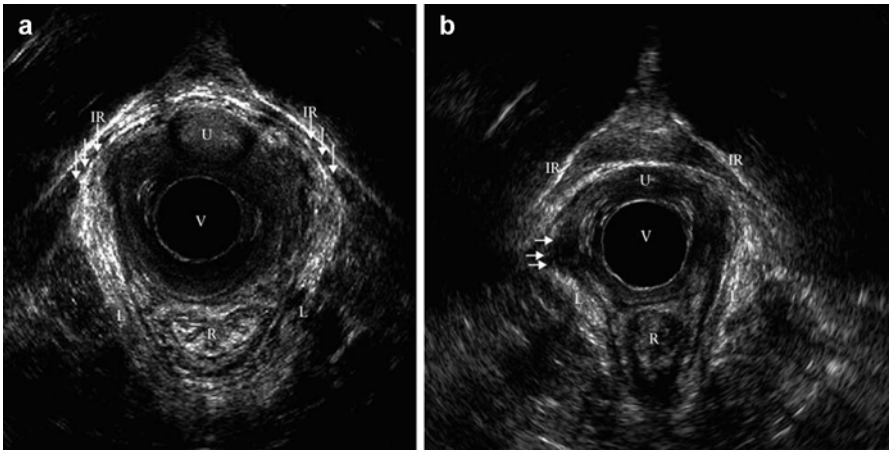

Fig. 10.13 Images obtained by EVUS in (**a**) a nulliparous woman with intact levator ani muscle (LAM) at level 3 and (**b**) a primiparous woman after forceps delivery involving a right mediolateral episiotomy and a third-degree tear with unilateral avulsion injury (axial plane at level 3). In (**a**) the LAM attachment is indicated by *arrows*. In (**b**) *arrows* indicate missing LAM muscle on patient's right side indicating the levator ani-pubic gap. *IR* inferior rami os pubis, *L* levator ani muscle, *R* rectum, *U* urethra, *V* vagina (with endovaginal probe) (Reprinted from Schwertner-Tiepelmann et al. [63]; with permission from John Wiley and Sons

Levator Pubic Gap

The plane of minimal levator hiatus can be used to assess levator ani muscle attachment to the pubic bone. In case of a levator ani detachment at the level of the pubic bone, the levator pubic gap will be measured as the distance between the remnants if the levator muscle to the original insertion on the pubic bone (Fig. 10.13).

Levator Muscle Thickness

The plane of minimal levator hiatus can be used to assess muscle thickness at 3 and 9 o'clock in coronal plane. However, interrater and intrarater reliability of this measure has not been good in unpublished studies.

Levator Plate Descent Angle

3D-ultrasound cubes obtained by 360° endovaginal probe are used for this measurement. Midsagittal plane is the measurement plane. The shortest line between the levator plate and pubic symphysis corresponding to the anterior-posterior line or height of the minimal levator hiatus is drawn. By drawing a reference vertical mid-symphysial line in the midsagittal plane, we can calculate the angle between the two lines as a measure of the relative location of the levator plate in relation to

the pubic symphysis in the resting position, the reference vertical line is referred to as the *P*ubic *L*evator plate *U*ltrasound *R*eference *A*ssessment *L*ine, or PLURAL (see Fig. 10.9).

Anorectal Angle

3D-ultrasound volumes obtained by 360° endovaginal probe can be used for this measurement. In the mid-sagittal plane one can measure the anorectal angle (ARA) as an angle between the axis of the anal canal and axis of the rectum (see Fig. 10.8). Caution should be used reporting these values as the ARA values obtained by EVUS are different from those obtained by MRI. Generally, EVUS ARA values >170° are associated with severe LAD.

Urethral Thickness

3D-ultrasound volumes obtained by 360° endovaginal probe can be used for this measurement. Midsagittal plane should be used to identify urethral lumen length and longitudinal axis. The midpoint of urethra will be marked. Using axial plane at the level of mid urethra, image should be tilted to become perpendicular to the longitudinal axis of the urethral lumen, visualized in the midsagittal plane. The urethral thickness can be measured in this plane. As with levator ani thickness, urethral thickness measurements are up for debate. That is because just as with muscles the thickness of a person's muscle or the urethra are determined by genetics and do not necessarily indicate pathology nor does it have correlation with continence status [36].

Obstetric Factors

Antepartum prediction of levator muscle trauma is difficult or maybe impossible [25] but there are reports of obstetric risk factors associated with pelvic floor trauma which can suggest modification of obstetric practice. Levator trauma at the time of first delivery has been associated with vaginal delivery, forceps and a longer second stage. Epidural pain relief may exert a protective effect [17]. It is likely that birth weight, length of second stage, size of fetal head and forceps delivery increase the probability of muscle injury [24, 27–29]. Valsky et al. showed an OR of 2.27 for muscle injury detected by ultrasound when the second stage was more than 110 min and OR of 3.34 for muscle injury when fetal head circumference was more than 35.5 cm [24]. Falkert et al. also found a positive correlation with weight and head circumference of baby and area of levator hiatus [40]. There are controversies regarding maternal age at first delivery. In some studies increased maternal age contributes to muscle injury [44]; however, this has not been reproduced by other studies [24, 25]. Shek et al. found that women with lower BMI were at higher risk of sustaining muscle injury, but this finding's clinical significance is questionable as BMI thresholds identified were 27.85 kg/m^2

vs 30.01 kg/m^2 [25] and this association was not confirmed by another study [40]. TPUS technique identified the interpubic gap and the infrapubic arc with a high inter-rater agreement, however; it was shown that infrapubic angle is not associated with length of second stage of labour and the occurrence of levator defects [45].

Birth related detachment of the levator ani muscle from the pubic bone seems to persist, whereas. levator ani stretch injury in the body of the muscle may resolve over time [26]. Although some earlier studies placed the rate at about 36 % [14], more recent MRI, and EVUS studies place the true overall rate closer to 13 % [26, 46]. Breaking down this rate by risk factors, in a recent MRI study, major defect rates were: 42 % for forceps and short second stage; 63 % for forceps and second stage arrest; and 6 % for spontaneous delivery. The odds ratios for major injury were: 11.0 for forceps and short second stage compared with spontaneous delivery; 25.9 for forceps and second stage arrest compared with spontaneous delivery; and 2.3 for forceps and second stage arrest compared with short second stage ($P=0.07$) [47].

It has been established that levator ani muscle injury increases the risk of pelvic organ prolapse (POP), and specifically cystocele formation initially [33, 48]. Observational studies have shown that there is a direct correlation between the size of the muscle defect and the symptoms or/and signs of prolapse [49] and women with bilateral avulsions are more likely to suffer from uterine prolapse [50]. The relationship between levator muscle injury and urinary incontinence is controversial [14, 51–54]. Although periurethral smooth and striated muscles have been implicated as potential mechanisms by which childbirth might affect urinary continence [36], using EVUS their presence or absence is not associated with continence status [36]. Associations between anal incontinence and birth related changes in pelvic floor support system have been investigated [55–57].

Conclusion

Imaging studies have improved our understanding of the pathophysiology of pelvic floor trauma in labor [58]. From this information, we can devise population based prevention strategies, and pelvic floor trauma repair strategies (Fig. 10.14). The information about the relationship between forceps delivery

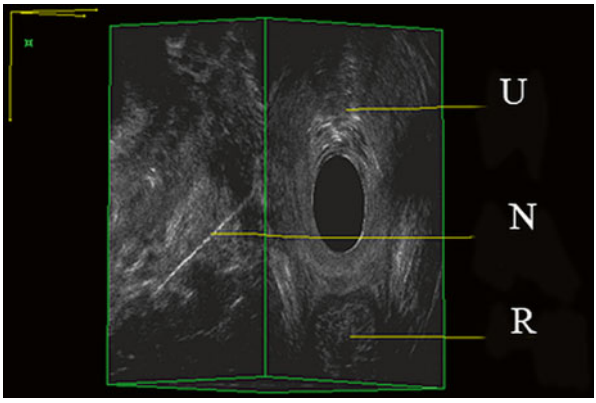

Fig. 10.14 J-hook needle in puborectalis muscle by endovaginal 360° ultrasound. *N* needle, *R* rectum, *U* urethra (With kind permission from Springer Science + Business Media: Rostaminia et al. [61])

and pelvic floor trauma has trickled to obstetricians in the United States, and forceps deliveries have declined significantly. Other efforts have been aimed at obstetrician education to repair pelvic floor trauma. Anal sphincter repair workshops (OASIS) help obstetricians to repair anal sphincter injury in an evidence based and reproducible manner [59]. Despite preliminary attempts at repair of levator ani trauma [60–62], the results are not applicable to the general population.

EVUS provides a reliable vehicle for investigating pathophysiology of pelvic floor trauma associated with labor as it provides detailed anatomy of levator ani subdivisions and can be deployed in large scale for population-based studies. Additionally, now the endovaginal 3D ultrasonography has surpassed investigational use and is used routinely by more clinicians for evaluation of the levator ani muscles, evaluation of defecatory dysfunction, vaginal mesh, vaginal cysts and masses just to name a few of the indications.

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Childbirth and Lower Urinary Tract Complications

11

Ali Abdel Raheem, Ahmed Zoaier, and Ahmed El-Sherbiny

Abstract

Effects of childbirth on women's health are often multiple, including changes in the mechanisms of urinary continence and pelvic floor support. Women after delivery may suffer from a wide range of lower urinary tract dysfunction such as voiding difficulty, urinary retention, urinary incontinence and acute or chronic injury to the urinary tract. Hormonal changes during pregnancy, regional anaesthesia and prolonged labour or instrumental delivery may predispose to postpartum urinary retention. Proper bladder care during and after labour is essential to prevent undiagnosed bladder overdistension and subsequent long-term bladder dysfunction. Stress urinary incontinence is the most common type associated with pregnancy and puerperium which results from levator ani damage, urethral and bladder neck hypermobility and/or ischaemic urethral injury. There is evidence that pelvic floor muscle training during antenatal and postnatal period can prevent urinary incontinence, while, the role of caesarean section in the prevention of incontinence is still debatable. Although intraoperative bladder and ureteric injury is uncommon, it is a potentially serious complication of caesarean delivery if unrecognised. Special attention in high risk patients is essential to avoid such injuries. Obstetric fistula is a devastating public health problem in the developing countries.

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Introduction

Childbirth effects on women's health are often multiple, including changes in the mechanisms of urinary continence and pelvic floor support. Some of these changes take much longer to resolve, and some may never fully revert to the prepregnant state. Up to one-third of premenopausal women and about one-half of postmenopausal women experience some type of pelvic floor disorder during their life, including urinary or anal incontinence or pelvic organ prolapse (POP) [1].

During pregnancy, the urinary system undergoes different anatomical and functional changes due to the hormonal effects of pregnancy, the increased mechanical pressure on the bladder from the gravid uterus, and the increased urine production from increased glomerular filtration rates. At the end of pregnancy, the effect of the delivery process on the pelvic floor function has been well documented. The urinary symptoms that women experience may be related to either pregnancy or delivery or both [2, 3]. Other symptoms may be iatrogenic such as urinary retention due to epidural anaesthesia and postpartum urinary tract infection (UTI) after urethral catheterization.

There are anatomical alterations in the pelvic floor muscle (PFM) following pregnancy and vaginal delivery; however, the long-term effects of pregnancy and their relationship with the mode of delivery and development of lower urinary tract (LUT) symptoms remain controversial. Despite this, the association between childbirth and postpartum urinary symptoms is well documented [4–7]. It is still unclear whether primary elective caesarean section (CS) prevents these symptoms in the long-term or not. Primiparous women delivered vaginally have a twofold incidence of incontinence than those delivered by CS. However, the protective effect of caesarean deliveries on urinary incontinence (UI) decreases with age [8].

In this chapter, we aim to describe the different postpartum LUT complications associated with vaginal and operative childbirth, present the incidence of each complication and the risk factors and mechanism for its occurrence. Moreover, it is important to appraise the early detection and appropriate management in the postpartum period to avoid long-term sequelae.

Classification of Childbirth-Related LUT Complications

Women after delivery may suffer from a wide range of LUT dysfunction and complications, which could be classified as follows:

1. Postpartum voiding difficulty and urinary retention (PPUR).
Overt urinary retention
Covert urinary retention
2. Postpartum urinary incontinence (PPUI).
Stress urinary incontinence (SUI)
Urgency urinary incontinence (UUI)
Mixed urinary incontinence (MUI)
3. Urinary tract injury:
Acute complications:
Iatrogenic bladder injury/laceration
Iatrogenic ureteric injury/laceration
Bladder rupture
Chronic complications: genitourinary fistulas

Postpartum Voiding Difficulty and Urinary Retention (PPUR)

Voiding difficulty and urinary retention are relatively common puerperal conditions among women in the immediate postpartum period. To ensure a rapid recovery and return to normal bladder function after delivery, it is important to accurately diagnose and properly manage this potentially serious condition [9].

Definition

PPUR is defined as inability to void, with a painful (usually), palpable or percussable bladder and need for catheterization to provide relief [6]. In the postpartum period, there are two main types of urinary retention:

- Overt (acute, painful or symptomatic) urinary retention: it refers to sudden onset of inability to pass urine spontaneously within 6 h of vaginal birth. Typically it is characterized by suprapubic pain, palpable or percussable bladder and patients usually need catheterization.
- Covert (chronic, painless or asymptomatic) urinary retention: it refers to increased post void residual volumes (PVR) of more than 150 ml measured by ultrasound or catheterization and no symptoms of urinary retention [10–12]. However, ultrasound measurement of postpartum bladder volumes can be inaccurate. Sometimes patients with covert urinary retention may present with frequency and feeling of incomplete emptying. However, it is usually a self-limiting condition, which often resolves within a few days [13].

Persistent PPUR may be defined as the inability to void spontaneously despite the use of an indwelling catheter for 3 days [6].

Incidence

The exact incidence of PPUR is uncertain and its range varies widely. Estimated PPUR incidences range from 0.05 to 37 % (Table 11.1) [6, 14]. This could be explained by the inconsistency in PPUR definitions and method of diagnosis. Moreover, most studies' figures are different depending on whether these studies comment either on overt or covert urinary retention or both types.

Although in most patients urinary function improves with time, a small number (0.05 % [6] up to 1 % [15]) continue to have persistent PPUR, beyond the early puerperium. These women, if not recognized and adequately treated in the early postnatal period, may develop acute prolonged bladder overdistension (ApBO) and risk of significant long-term bladder dysfunction.

Mechanism and Pathophysiology

Several mechanisms have been suggested to explain the pathophysiology of PPUR.

Hormonal Effect on the Bladder

In the postpartum period the bladder is hypotonic, and this is a result of the physiological hormonal changes such as elevated progesterone levels during normal pregnancy and puerperium, affecting the normal bladder function, as it is a hormone-responsive organ [21]. This reduction in smooth muscle tone usually starts after the third month of pregnancy with the bladder gradually increasing in capacity as pregnancy progresses and remaining so for a number of days following birth [22]. In a small number of patients these changes may persist for days or longer especially in those patients with risk of developing PPUR (see risk factors below).

Table 11.1 The incidence of PPUR among women

Authors	Number of patients in study	Number of patients with PPUR	Incidence (%)
Groutz et al. (2001) [6]	8402	4	0.05
Kekre et al. (2011) [12]	771	82	10.6
Yip et al. (1997) [13]	164	19	11
Ismail and Emery (2008) [14]	100	37	37
Andolf et al. (1994) [15]	539	8	1.5
Liang et al. (2002) [16]	605	101	16.7
Hee et al. (1992) [17]	51	23	45
Demaria et al. (2004) [18]	154	55	36
Yip et al. (2005) [19]	691	101	14.6
Saultz et al. (1991) [20]	–	–	1.7–17.9

Regional Anaesthesia

Spinal, epidural and pudendal nerve block are common causes of PPUR. This may be due to suppression of the sensory stimuli from the bladder to the pontine micturition centre after afferent neural blockade, followed by inhibition of the reflex micturition mechanism, which may result in reduction of bladder contractility and urinary retention [9]. In a retrospective case controlled study of 11,322 women, 51 women (0.46 %) had overt signs of PPUR following vaginal delivery and 98 % of these were associated with epidural anaesthesia [11].

Acute Prolonged Bladder Overdistension (ApBO)

Sometimes in a woman with an epidural or following spinal anaesthesia the discomfort associated with bladder overdistension is masked and if not recognized and treated properly, ApBO occurs. One episode of overdistension is considered enough to cause bladder damage [23]. ApBO pathogenesis likely consists of 2 consecutive stages. A primary temporary neurogenic dysfunction leads to acute urinary retention; if neglected and not treated properly postnatally, it will be followed by secondary myogenic detrusor damage. Recovery depends on whether reversible or irreversible damage has occurred [24]. With irreversible damage, the detrusor becomes atonic and unable to respond; this leads to long term voiding difficulties [25].

Prolonged Labour and Instrumental Delivery

Prolonged labout and delivery may lead to PPUR by two mechanisms:

- Denervation pudendal nerve injury: The pudendal nerve (S2–4) travels along the posterior wall of the pelvis and exits the pelvis to innervate the external genitalia. Due to its length and position, it becomes more vulnerable to compression, pelvic floor tissue overstretching and damage when the fetal head is compressed against the pelvic floor for a long time during vaginal delivery. Pudendal nerve damage results in diminished reflex and voluntary mechanism required for normal voiding [11, 13]. Some studies described the status of pudendal nerve injury after vaginal delivery using electrophysiological tests and showed a significant increase in pudendal nerve terminal motor latencies, which may take a few months to recover post-delivery [26–28].
- Mechanical obstruction: Another possible mechanism that may lead to transient PPUR is extensive tissue oedema around the urogenital area which results from prolonged compression of the fetal presenting part onto the birth canal or instrumental/assisted delivery or extensive vaginal and perineal laceration. As tissue oedema resolves within few days of delivery, PPUR gradually improves.

In a prospective observational study of 2866 women, instrumental delivery accounted for 16.7 % of the retention group compared with 4.7 % of the control group [4].

Risk Factors for PPUR

It is difficult to accurately predict the group of women who are more at risk of PPUR. However, in a large systematic review and meta-analysis including 23 observational studies with original data Mulder et al. concluded that "*instrumental delivery, epidural analgesia, primiparity and episiotomy"* can be identified as the main four clinical risk factors for development of overt or covert PPUR [29].

Therefore, special attention and careful follow-up should be offered to this group of high-risk women. They must be educated to seek a review and advice if they develop any symptoms suggestive of voiding difficulties in the early postpartum period.

Clinical Presentation of PPUR

Symptoms of PPUR differ according to its type. In patients with overt PPUR symptoms are much more obvious than in those with covert type, as they are not able to void and they usually experience associated suprapubic bladder pain.

Symptoms of overt "acute" PPUR include:

- Pain (should not be misdiagnosed as caesarean wound pain)
- Hesitancy
- Slow or intermittent stream
- Straining to void
- Sensation of incomplete bladder emptying

Symptoms of covert urinary retention include:

- Lack of pain or other symptoms of overt urinary retention
- No urgency to void – reduced bladder sensation
- Overflow incontinence [19]

Symptoms may be masked and patients may be asymptomatic especially if they had epidural anaesthesia. On the other hand, some patients may present with overflow UI owing to overdistension of the bladder and be misdiagnosed with SUI.

Diagnosis of PPUR

The diagnosis of PPUR can be difficult especially if a woman is asymptomatic. It is important to have a high level of suspicion if a patient develops any symptoms of overt or covert retention. Sometimes absence or presence of symptoms may be misleading and make the diagnosis challenging. This necessitates targeted clinical investigations and further evaluation. In a study by Groutz et al. up to 45 % of women complain of significant voiding symptoms in early

puerperium, however, of these patients, only 22 % will have low flow rates. Pain was the most common symptom (63 %) followed by weak stream, intermittent stream and hesitancy (44, 38, and 33 %, respectively). In this study, the symptomatic group had significantly longer 2nd stage of labour and more instrumental deliveries (ventouse) [30]. Ramsay et al. showed that 43 % of patients in the immediate post-partum period had abnormal flow rates, although, the majority of them were asymptomatic [31].

On examination some patients may have suprapubic tenderness and bulge and a palpable and percussable bladder. Clinically, the diagnosis of overt PPUR could be made if the woman is unable to void in the first 6 h after delivery and requires urethral catheterization to drain >600 ml [32]. On the other hand, covert PPUR diagnosis could be made if the patient have PVR >150 ml and is unable to void adequate amount of urine (>50 % of normal bladder capacity) [13].

Postvoid residual (PVR) volume measurements could be carried out by two methods, either by catheterization or bladder ultrasound. Catheterization is more accurate than bladder scan; however, it potentially carries increased risks of UTI. Bladder scanning is a popular non-invasive method of measuring PVR and although its accuracy has been debated, some authors believe that ultrasound assessment is highly accurate even in the postpartum period as the bladder maintains an ellipsoid shape [33].

Management and Prevention of PPUR

The bladder management during the early postpartum period is aimed to:

- Maintain the normal bladder function
- Minimize the risk of damage to the urethra and bladder
- Provide adequate management strategies for women who have problems with bladder emptying
- Prevent long-term complications associated with bladder emptying

Postpartum Bladder Care

In the first days after birth the production of urine is increased, as extracellular fluid is excreted. The following instructions are recommended in the early postpartum period to prevent the development of PPUR [20, 23, 25, 34–36]:

- The timing and volume of the first voided urine should be monitored.
- Timed voiding should be encouraged every 3–4 h.
- PVR should be measured.
- Frequency volume charts should be filled with strict follow-up for urinary input and output to ensure adequate fluid balance in the first 24 h post-delivery.

- Women should be counseled to report any of the following problems if occur: insentience episode, lack of awareness or desire to void urine, hesitancy, inability to empty her bladder fully; urinary frequency; pain on voiding; any other related concerns.
- If urine has not been passed within 6 h after birth, efforts to assist urination should be advised, such as taking a warm bath or shower.
- If bladder emptying has still not occurred, then either the bladder must be emptied by catheterization and the volume of urine recorded, or bladder volume estimated by ultrasound, followed by catheterization as appropriate.

What to Do if PPUR Is Suspected or Confirmed?

If the voided volume is <150 ml or PVR is >150 ml by ultrasound, the patient should be managed with in/out catheterization and PVR should be accurately measured. PVR of at least 150 ml may imply a degree of bladder dysfunction. At this stage a fluid balance chart is mandatory [32].

If Still Unable to Void after a Further 6 h, Insert an Indwelling Catheter

If urinary drainage is >500 ml in 1 h, leave the catheter for 24 h. If urinary drainage is >1000 ml in 1 h, leave the catheter for 48 h. Some authors advocate for the catheter to remain in situ for 1 week [32]. We should rule out UTI by sending a midstream or catheter urine sample for culture and if UTI is documented prescribe appropriate antibiotics [37]. Intermittent self-catheterization or indwelling catheter is recommended if a trial without catheter failed or there is persistent high PVR in the absence of infection. The urine volume drained initially can be used as a predictor for repeat catheterization. In a study by Burkhart et al. it was found that if the initial volume of PVR was <700 ml, patients did not require repeat catheterization, however, if PVR was >1000 ml, 20 % of patients required repeat catheterization [38].

Discharge Planning

After delivery all women should void of ≥400 ml before discharge. Women with risk factors and who develop PPUR must have three documented voids with normal voiding parameters post removal of catheter before discharge. Women who require bladder retraining and management should be counselled and referred to a health professional with appropriate training and expertise in the treatment options for urinary retention.

Postpartum Urinary Incontinence (PPUI)

Urinary incontinence (UI) is a common problem among adult women. It is more frequent during pregnancy and the postnatal period, which may be the first time that many women experience UI. The presence of incontinence during pregnancy may be predictive of PPUI [39]. Moreover, PPUI in the short term may be predictive of

longer-term problems. Women with persistent postpartum SUI at 3 months have a 92 % risk of having stress urinary incontinence at 5 years [40].

Definitions

There is no exact definition for the term PPUI; however, various definitions of UI types are standardized according to the International Continence Society (ICS) and International Urogynecological Association (IUGA). In general UI is defined as "any complaint of involuntary leakage of urine" [41].

There are three main types of UI:

- Stress urinary incontinence (SUI) is defined as "the complaint of involuntary leakage on effort or exertion or on sneezing or coughing."
- Urgency urinary incontinence (UUI) is defined as "the complaint of involuntary leakage preceded by urgency."
- Mixed urinary incontinence (MUI), combination of SUI and UUI [41]

Of these types of UI, SUI is the most common type associated with pregnancy and puerperium [42–44].

Epidemiology

The prevalence of UI in adult women over 20 years is 25 % and increases with age [45]. Pregnancy and parity are considered the main risk factors for developing UI in women. Both could have a significant impact on the pelvic floor, leading to damage of the pelvic floor support (muscles, ligaments, fascias and peripheral nerves) and result in pelvic floor dysfunction with undesired sequelae regarding urogenital tract.

The prevalence of UI in women seems to increase during pregnancy and decrease following delivery; however, the prevalence of UI during the postpartum period is still higher than before pregnancy [46, 47]. The prevalence of UI during pregnancy and postpartum varies, and reports show a wide range of 7–64 % during pregnancy [48–50], while PPUI ranges from 3 to 40 % [46, 51, 52]. It is important to estimate the prevalence of PPUI to accurately assess the public health burden of this problem and also to calculate sample sizes when designing research studies.

A Norwegian large population-based study of 12,679 primiparous women found the prevalence of PPUI at 6 months to be as high as 31 %. Incontinence was most common among women with an instrumental delivery (36 %) or spontaneous vaginal delivery (34 %) and lowest among women with emergency CS (17 %) or elective CS (13 %). The prevalence of SUI was twice that of UUI in this study (Tables 11.2 and 11.3) [51]. In 2009, another large population-based cross-sectional study from the United States (Boyles et al.) estimated the incidence of PPUI among 5599 primiparous women. The incidence of PPUI at 6 months after delivery was 10 % (see Tables 11.2 and 11.3) [53].

Table 11.2 The incidence of PPUI among primiparous and multiparous women

Author	Number of patients	Postpartum assessment time (month)	Parity
Wilson et al. (1996) [44]	1505	3	12 % Primiparous 21 % Multiparous
Wesnes et al. (2009) [51]	12,679	6	21 % Primiparous
Boyles et al. (2009) [53]	5599	6	10 % Primiparous
Eliasson et al. (2005) [54]	665	12	21 % Primiparous
Farrell et al. (2001) [55]	595	6	26 % Primiparous
Glazener et al. (2006) [56]	3405	3	15 % Primiparous
Solans-Domenech et al. (2010) [57]	1128	2	5 % Primiparous
Burgio et al. (2003) [58]	523	3	10 % Multiparous
Foldspang et al. (2004) [39]	1232	>12	14 % Multiparous
Iosif (1981) [59]	1411	6–12	19 % SUI Multiparous
Dimpfl et al. (1992) [60]	350	3	4 % SUI Primiparous 4 % Multiparous

Table 11.3 The prevalence of PPUR among primiparous and multiparous women

Author	Number of patients	Postpartum assessment time (month)	Parity
Wesnes et al. (2009) [51]	12,679	6	31 % Primiparous
Boyles et al. (2009) [53]	5599	6	17 % Primiparous
Ekstrom et al. (2008) [61]	389	3	13 % SUI, 4 % UUI Primiparous
Eliasson et al. (2005) [54]	665	12	49 % Primiparous
Foldspang et al. (2004) [39]	1232	>12	26 % Primiparous
Sampselle et al. (1996) [62]	59	6	67 % SUI Primiparous
Thomason et al. (2007) [63]	121	6	45 % Primiparous
Yang et al. (2010) [64]	1889	6	10 % Multiparous
Ege et al. (2008) [65]	1749	12	20 % Multiparous
Ewings et al. (2005) [66]	723	6	45 % Multiparous
Hvidman et al. (2003) [78]	642	3	3 % Multiparous
Mason et al. (1999) [68]	717	3	10 % Primiparous 31 % SUI Multiparous
Raza-Khan et al. (2006) [69]	113	–	46 % Primiparous 43 % Multiparous
Schytt et al. (2004) [70]	2390	12	18 % SUI Primiparous 24 % SUI Multiparous

In a systematic review and meta-analysis of 33 population-based studies, Thom and Rortveit reported that the pooled prevalence of PPUI is 33 % in all women during the first 3 months postpartum. The mean prevalence of weekly and daily

incontinence was 12 and 3 %, respectively. The mean prevalence was double in the vaginal delivery group (31 %) compared to the CS group (15 %) [7].

Mechanism and Pathogenesis

Stress Urinary Incontinence (SUI)

Most studies reported that the main underlying mechanism for development of SUI in women is the presence of intrinsic sphincter deficiency and/or bladder neck and urethral hypermobility with evidence that both pathologies are present together in most of patients with SUI. Pregnancy and childbirth are the main risk factors that predispose to both pathologies based on the hypothesis that pelvic organ support can be impaired by vaginal childbirth [71]. Peschers et al. demonstrated an impaired strength of PFM immediately after vaginal birth, which improves gradually with time [72]. Persistent PPUI could be the result of partial irreversible functional and anatomical changes of the PFM, which have a more serious prognosis than SUI developing during pregnancy [43].

The main underlying changes that occur after vaginal delivery and have a role in the development of PPUI are levator ani trauma, bladder neck and urethral hypermobility, and urethral sphincter injury.

Levator Ani Trauma

The PFM (the pubococcygeus–puborectalis complex) insert on the pelvic sidewall from the pubic rami to the ischial spine and form a V-shaped or U-shaped sling around the anorectal junction. The levator hiatus is the space bordered by this sling which contains the urethra anteriorly, the vagina centrally, and the anorectum posteriorly [73]. In nulliparous women, the levator hiatus varies from 6 to 36 cm^2 on valsalva manoeuvre [74]. During labour, the average fetal head measures 60–90 cm^2 in the plane of minimal diameters, requiring marked distension and deformation of the levator complex. Lien et al. showed that the most inferior and medial parts of the levator complex may have to increase in length by a factor of 3 or more during crowning of the fetal head [75], which explain that 36 % of women after vaginal delivery have a levator ani avulsion [73, 74]. Hoyte et al. found a significant decrease in levator muscle volume and levator hiatus widening in women with SUI [76]. An MRI study showed that nulliparous women do not have defects of the levator ani musculature, while up to 20 % of primiparous women have levator ani defects after vaginal birth [77].

Bladder Neck and Urethral Hypermobility

Both have been assessed as predictors of postpartum SUI. King and Freeman found that increased bladder neck mobility antenatally was predictive of postpartum SUI and they suggest that collagen susceptibility to changes during pregnancy, measured by changes in bladder neck mobility, might predict PPUI [78]. DeLancey et al. reported that low maximum urethral closure pressure was the most common factor associated with SUI in primiparous women followed by bladder neck

hypermobility [79]. Toozs-Hobson and colleagues stated that bladder neck mobility was significantly greater in women who delivered vaginally compared to women with CS [80].

Urethral Sphincter Injury

The normal urethral sphincter function may be adversely affected after prolonged labour or instrumental vaginal delivery because of pressure-induced ischaemic injury [81]. In addition, in multiparous women there may be repeated urethral injury and greater urethral dysfunction following each vaginal birth [82]. Cannon et al. observed histologically in rats that prolonged vaginal distension results in extensive disruption and marked thinning of the urethral skeletal muscle fibres and regarding urethral sphincter function, there was associated lower leak point pressure with increased severity of SUI [83]. These findings suggest that ischaemic injury to the urethral sphincter is important in the development of SUI after prolonged vaginal distension.

Risk Factors

Several risk factors are found to be associated with the development of postpartum SUI and the persistence of symptoms such as:

- Preconception UI, chronic cough and smoking [84]
- Prolonged labour, second stage >1 h and large birth weight [61, 85]
- Weak pelvic floor collagen, which might be relevant during pregnancy where connective tissue is weaker than in the non-pregnant [86]
- Joint hypermobility has been proposed as a marker for connective tissue weakness and subsequent development of SUI and prolapse [87]. However, in other studies no differences were found except for elbow hyperextension [88].
- Obesity is known to be a possible risk factor for postpartum SUI [89].

Urgency Urinary Incontinence

Effects of childbirth on UUI are less well described. Postpartum UUI may occur as commonly as SUI affecting 30 % of women [90]. Some risk factors may be associated with increased symptoms of UUI such as forceps delivery, episiotomy [90], and fetal macrosomia [42].

Diagnosis of Urinary Incontinence (UI)

History: For diagnosis a full history should be taken to evaluate patients' pattern of voiding and leakage, which may determine the type of incontinence. Obstetric history including difficult deliveries, grand multiparity, forceps use, obstetric lacerations, and large babies, history of pelvic surgery especially prior incontinence procedures, hysterectomy, or pelvic floor reconstructive procedures should be asked about. In addition, the patient should be asked if she takes medications that affect LUT function.

Physical examination: Adequate physical examination is essential for the diagnosis of women with UI. If possible, it is optimal to examine the patient with a comfortably full bladder. This may aid confirmation of leakage of urine with a cough test during examination *(cough-stress test)*. Pelvic examination by vaginal speculum and bimanual examination may reveal vaginal atrophy, vulvo-vaginitis, POP or pelvic masses. In addition, weakness of the PFM could be assessed. Spinal reflex activity of L5–S5 is assessed by bulbocavernosus reflex testing (squeezing of the clitoris induces anal sphincter contraction) and spinal reflex activity of S4–S5 nerve roots by anal reflex testing.

Urinalysis and culture: A urine dipstick assessment should be performed to exclude a urinary tract infection (UTI) and a specimen sent for microscopy and culture if abnormal. UTI should be excluded and treated first before shifting to other test.

Non-invasive urodynamic testing:

- Bladder diary*:* the patient should keep a bladder diary form (3–7 days), record the times of urination, voided volumes, daily fluid intake, episodes of urine leakage, and estimated amounts of leakage.
- Uroflowmetry and PVR measurement: provides information about the voiding pattern "continuous, fluctuating or interrupted" and numerical values (maximum flow rate "Qmax," average flow rate, voided volume and PVR).

Invasive urodynamic studies: These studies include many tests such as cystometry, pressure flow study, urethral pressure profilometry, videourodynamics. These tests evaluate the bladder dynamics during the filling and voiding phases. They may be indicated for diagnosis if MUI is suspected, before surgical intervention, refractory cases, failed previous surgery or in the setting of research work.

Treatment and Prevention of Urinary Incontinence (UI)

Successful treatment of UI must be tailored to the specific type of incontinence and its cause. Patient symptoms, age and any underlying medical problems may also affect the treatment outcomes. A wide range of treatment options has been successfully used in the treatment of UI (SUI, UUI and MUI) and many guidelines and recommendations in the literature have described extensively the appropriate line of therapy for each type according to patient clinical presentation and diagnosis [91].

The following treatments could be considered for the treatment of UI:

1. Conservative interventions: such as physical therapies (e.g. pelvic floor muscle training [PFMT] and biofeedback), lifestyle modification (e.g. weight loss, caffeine and alcohol restriction, weight reduction and smoking cessation), behavioural training (e.g. timed voiding and bladder training). According to several guidelines conservative treatment should be tried as the first line of therapy for all patients with UI.

2. Pharmaceutical therapies: such as anticholinergics, alpha-adrenergic agonists, topical oestrogens, duloxetine.
3. Surgical therapies: for SUI: midurethral tapes, bulking agents, Burch colposuspension and pubovaginal sling and for UUI: botulinum toxin injections, sacral neuromodulation and bladder augmentation.

Role of PFMT During Pregnancy and Puerperium

Physiotherapy such as pelvic floor muscle training (PFMT) is more appropriate during pregnancy and puerperium than drugs or surgery for the treatment of UI. Some drugs are contraindicated or best avoided during pregnancy and breastfeeding, while surgery is not recommended until a woman has completed her family [92].

PFMT was first popularized by Arnold Kegel for the treatment of UI [93], and is recommended as the first treatment of choice for UI [91]. The rationale for PFMT is based on two functions of the PFM; support of the pelvic organs and a contribution to the sphincteric closure mechanism of the urethra [94, 95].

In a Cochrane Database Systematic Review Hay-Smith et al. determined the effect of PFMT for the prevention and treatment of UI in antenatal and postnatal women. They found that pregnant women without prior UI who were randomized to intensive antenatal PFMT were less likely to report UI than women randomized to no PFMT or usual antenatal care in late pregnancy (about 56 % less) and up to 6 months postpartum (about 30 % less). Postnatal women with persistent UI 3 months after delivery and who received PFMT were less likely to report UI than women who did not receive treatment or received usual postnatal care (about 20 % less) 12 months after delivery. The authors concluded that there is some evidence that PFMT in women having their first baby can prevent UI in late pregnancy and postpartum and that PFMT is an appropriate treatment for women with persistent PPUI [92].

Does CS Reduce PPUI?

The impact of delivery mode and controversy over CS versus vaginal delivery with regard to pelvic floor trauma and subsequent development of UI has been discussed and debated. Damage to important pelvic floor muscles and nerves is primarily attributable to vaginal delivery [96]. Many reviews conclude that CS may be more protective against PPUI. However, these commonly held perceptions have been challenged in recent years [42]. The protective role of CS against injury to the pelvic floor cannot be justified because pregnancy itself may increase the prevalence of UI as a result of mechanical compression on the pelvic floor [97–99]. On the other hand, some studies found that there was no significant difference between CS and vaginal delivery in the protection against SUI suggesting that the labour process, rather than vaginal birth itself, may be implicated in pelvic floor damage [100, 101].

A systematic review by Press et al. compared the prevalence of postpartum UI after CS and vaginal birth after exclusion of instrumental delivery. They reported that CS reduced the risk of postpartum SUI from 16 to 9.8 % in 6 cross sectional studies, and from 22 to 10 % in 12 cohort studies. They concluded that the short-term occurrence of any degree of postpartum SUI is reduced with CS; however the risk of severe SUI and UUI did not differ by mode of birth [96].

With the trend towards elective CS, women who request an elective CS appear to be motivated by the desire to prevent pelvic floor damage, including UI. These women should be offered detailed information and proper counseling which should include the overall risks associated with a caesarean section in order to make an informed decision.

Urinary Tract Injury During Childbirth

Childbirth either vaginal or caesarean delivery might carry a risk for urinary tract injuries, which are divided into two categories:

1. Acute complications
   - Iatrogenic bladder injury/laceration
   - Iatrogenic ureteric injury/laceration
   - Bladder rupture
2. Chronic complications: Genitourinary fistulas

Acute Complications

Intraoperative urological injury is an infrequent but potentially serious complication of CS. It is important to identify risk factors that place patients at higher risk for urological injury. Identification of high-risk patients preoperatively allows for careful planning by the surgeon and greater caution during surgical dissection. Immediate recognition of bladder or ureteric injury ensures that further sequelae can be minimized [102].

Iatrogenic Bladder Injury

Although uncommon, iatrogenic bladder injury is the most common urological injury occurring at the time of CS [103, 104]. Delayed recognition of the injury can be associated with serious short-term and long-term complications. Potential complications include prolonged operative time, UTI, prolonged indwelling catheterization, urinary ascites and fistula formation [105–108].

Incidence

The incidence of bladder injuries has been reported with a range from 0.08 % up to 0.81 % [109–115]. For primary caesarean deliveries it is estimated to be 0.2 % [115] and its incidence increases with repeat cesarean deliveries to reach 0.43–0.81 % [113, 114].

Mechanism of Bladder Injury at CS

1. During pregnancy: The urinary system undergoes some changes that increase the risk of injury. The bladder is elevated and placed high in the abdomen that follows the growth of the gravid uterus [116].
2. During CS:
   - During repeat CS: A previous CS favors the adhesion of the bladder to the lower uterine segment or in a position much higher on the uterus, thereby blocking access to the lower uterine segment and increases the likelihood of bladder injury during the hysterotomy [117].
   - During emergency CS: In urgent cases due to fetal distress bladder injury may occur when visualization and dissection of the correct surgical planes is suboptimal [102].
   - During CS after a prolonged second stage: The vagina may be incised because of difficulty identifying the interface between uterus and vagina, resulting in an increased risk of bladder injury [102]. This risk is also high due to the deeply impacted presenting part of the fetus.

Risk Factors for Caesarean-Related Bladder Injury

Many risk factors increase the likelihood of bladder injury during CS have been described in several studies:

1. Previous CS and adhesions: A prior CS is the most common risk factor. Many reports show that this risk factor represents from 63 % up to 72.4 % of cases of bladder injury [110–112].
2. Emergency CS: up to 69 % of injuries may occur during this type of surgery [103].
3. Other risk factors are previous pelvic surgery, obstetric haemorrhage, and post-caesarean hysterectomy, prolonged labour before caesarean delivery and concurrent uterine rupture.

Treatment and Prevention

Most bladder injuries could be detected easily intra-operatively. A high index of suspicion for bladder injury should be noted in high-risk patients. Signs of bladder injury include:

- Leakage of urine into the operative field
- Visualization of the Foley catheter balloon
- The presence of blood-stained urine in the catheter bag

During caesarean delivery, the commonest site of bladder injury occurs in the dome, away from the trigone and ureters [102]. The prognosis of a bladder injury that is diagnosed immediately and repaired adequately is very good and without serious sequelae, especially if the injury does not involve the trigone [118].

Treatment is carried out by identification of the extent of injury and surgical repair through bladder wall closure in two layers (water tight closure) using absorbable suture (3/0). The urethral catheter should be left in situ for 7–10 days postoperatively for adequate bladder drainage. Some prefer to perform retrograde cystogram before urethral catheter removal to ensure complete healing of the bladder.

To avoid injury to the urinary tract during childbirth, the obstetrician must have an accurate understanding of the pelvic anatomy, use a meticulous surgical technique and maintain a constant high degree of vigilance during delivery, especially in difficult cases, where the risk of injury is increased. The risk of bladder injury during CS can be reduced if the following tips are considered:

- Enter the peritoneal cavity at the most superior segment of the abdominal incision.
- Reflect the bladder using sharp dissection to separate a densely adherent bladder from the uterus.
- In cases of a large fetus and a prolonged second stage of labour that requires CS, a low vertical uterine incision may be preferable to reduce the risk of extensions into the broad ligament [102].

Since prior CS is the most common risk factor for bladder injury, it is very important to counsel women requesting primary elective caesarean deliveries about the potential for significant surgical complications in repeat CS when discussing the indications for a primary elective caesarean delivery [110–112].

Iatrogenic Ureteric Injury

At the time of CS ureteric injury is less common than bladder injury, with a reported incidence of 0.10–0.27 % [115, 119].

Causes of Ureteric Injury

Most ureteric injuries at the time of CS occur in patients who have no identifiable risk factors and injury can occur as a result of the following causes: [120]

- During repair of an extension of the hysterotomy.
- After sutures have been placed to control bleeding in the broad ligament.
- During internal iliac artery ligation performed to control postpartum haemorrhage.

The most common sites of ureteric injury during hysterectomy are along the pelvic wall lateral to the uterine artery, the area of ureterovesical junction, and the base of infundibulopelvic ligament [121, 122].

Treatment and Prevention

During the course of a difficult CS, it is important to avoid compromising adequate blood supply to the ureter and prevent its devascularization by carefully dissecting

the ureter from the pelvic sidewall while preserving its attachment medially to the peritoneum [120].

If a ureteric injury is suspected at the time of a difficult CS, cystoscopy should be performed intraoperatively and intravenous administration of indigo carmine or methylene blue dye to visualize efflux from the ureteric orifices. Patients with an occult ureteric injury during CS may present later with elevated creatinine levels, fever, flank pain, or symptoms of ileus. If these symptoms are noted, the patient should undergo renal ultrasonography or intravenous urography to assess for ureteric injury [123].

After ureteric injury diagnosis is confirmed, immediate surgical repair by releasing the suture ligation or ureteric stenting is recommended.

Bladder Rupture

Bladder rupture during childbirth is an extremely rare complication and only present in the literature as a few case reports [124–127]. Usually bladder rupture occurs during attempt of vaginal delivery in a woman who has undergone a prior CS as it occurs simultaneously and as a result of uterine rupture. If the injury is severe, it may extend into the urethra [126]. It may be complicated with fistula formation between the bladder and uterus (VUF) [125].

Both urologists and obstetricians should keep bladder injuries in mind, as potentially serious complications during labour, which can become life threatening. Surgical treatment is the preferred management approach in most cases.

Chronic Complications (Obstetric Fistulas)

Obstetric fistula (OF) is a devastating complication. Even with the recent developments in medical technologies and therapeutic advances, it is still one of the major public health problem with negative impact on women's quality of life especially in developing countries of some parts of Africa and Asia, where unattended obstructed labour is common and maternal mortality rate is high. Obstetric fistula is discussed more extensively in another chapter of this book. We will present some salient points on the management of OF in this chapter.

Definition

OF is an abnormal opening between the genital and the urinary system or the gastrointestinal system, resulting in uncontrollable leakage of urine and/or faeces. OF may be one of the following:

- A vesico-vaginal fistula (VVF) occurs between the bladder and the vagina.
- A vesico-uterine fistula (VUF) occurs between the bladder and the uterus.
- A urethro-vaginal fistula (UVF) occurs between the urethra and the vagina.
- A recto-vaginal fistula (RVF) occurs between the rectum and the vagina.
- A uretero-vaginal fistula (UTVF) occurs between the ureter and the vagina.

- A double fistula occurs between the vagina, the bladder and the rectum.
- An iatrogenic fistula occurs inadvertently, for example, during CS.

Epidemiology

Obstetric fistulas are most common in the developing countries of sub-Saharan Africa and South Asia. More than two millions young women live with untreated OF in these regions. Each year between 50,000 and 100,000 new women worldwide develop OF [128]. However, the global prevalence and incidence of OF are largely unknown because many patients with OF are unable to access medical care. Indeed, one study found that one million women are affected by OF in Nigeria alone, and another one suggests that 70,000 new cases occur annually in Bangladesh [129]. On the other hand, OF is extremely rare in the industrialized countries as a result of skilled attendance in labour and comprehensive emergency obstetric and neonatal care.

The most common type of OF is VVF and accounts for 79–100 % of cases, while RVF accounts for 1–8 %, and combined VVF and RVF have been reported in 1–23 % of cases [130]. Other types of OF are very rare.

Mechanism and Pathophysiology

The main underling aetiology is prolonged obstructed labour, which accounts for 76–97 % of cases. Usually this condition starts when the descending fetus is unable to pass through the mother's pelvis due to cephalopelvic disproportion. This will lead to ischaemic damage of the surrounding soft tissues. Usually the mother loses her child and the necrotic tissue sloughs off leaving a hole between the vagina and bladder (Fig. 11.1) or vagina and rectum (RVF), through which urine and/or stool leak. Patients can be affected by multiple devastating medical and psychosocial sequelae [131]. The second most common cause of OF is the direct tearing of the soft tissue during precipitous delivery or obstetric maneuvers. A least common cause is elective abortion [132, 133]. Some reports showed that sexual violence and abuse may be a cause.

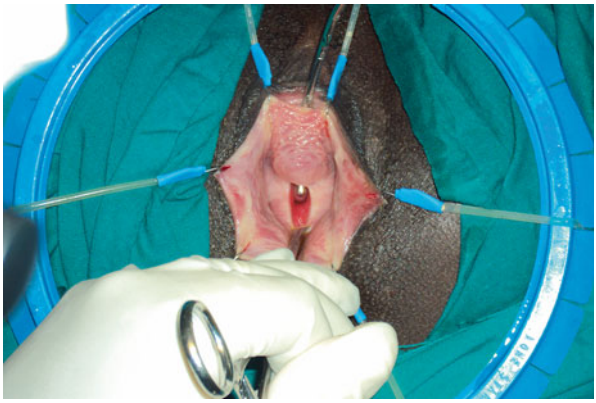

Fig. 11.1 Obstetric VVF during surgical repair

Risk Factors

Risk factors implicated in the pathogenesis of OF in developing countries are the following:

- Young age at marriage [134, 135]
- Short stature with small-sized foot [136, 137]
- Illiteracy and lower socioeconomic status [136]
- Primiparity [136]
- Lack of prenatal care (lack of skilled attendance at birth, lack of emergency obstetric care) [133, 138]
- Rural location and lack of transportation [133]

Classification

Several methods have been used to classify OF. The WHO classification divides OF into two main groups according to the anticipated difficulty of repair (simple or complex fistula) and most surgeons nowadays use this classification.

1. Simple fistula (fistula with good prognosis):
   Characterized by:
   - Non-recurrent single fistula (VVF)
   - Less than 4 cm in size
   - No urethral involvement
   - No scarring of the vaginal tissue
   - Minimal degree of tissue loss
   - Intact ureters
2. Complex fistula (fistula with uncertain prognosis):
   Characterized by:
   - Multiple fistulas
   - RVF or mixed VVF/RVF that involve the cervix
   - Fistula greater than 4 cm in size
   - Urethral involvement
   - Vaginal scarring
   - Complete separation of the urethra from the bladder
   - Extensive tissue loss
   - Ureteric involvement
   - Stones in the bladder
   - And/or failed surgical attempts [129]

Presentation

Women with OF typically complain of persistent leakage of urine through the vagina (VVF), or leakage of flatus and stool through the vagina (RVF) or both. Both VVF and RVF are associated with persistent offensive odour leading to isolation from family and many women are divorced [138, 139].

Patients with a VUF most often present with complaints of urinary leakage or haematuria, and may also experience cyclic haematuria (menouria), amenorrhea, and UTI [140, 141].

Obstructed labour causes injury that may involve different pelvic organs, and patients may have other chronic debilitating comorbidities in addition to urinary or fecal incontinence such as:

- Footdrop from lumbo-sacral nerve damage
- Vaginal stenosis and infertility
- Chronic pelvic pain
- Chronic vulvar dermatitis and skin excoriation from chronic irritation of the skin by urine and feces
- Psychological disorders: depression, social stigma, and posttraumatic stress disorder. Some women may attempt suicide [142, 143].

Diagnosis

Diagnosis is established by careful history taking and meticulous clinical examination. The physician could identify the fistula easily, however, it is important not to miss a small fistula or another associated pathology, such as ureteric involvement because this will increase the probability of failure of the surgical repair.

Women typically give a history of obstructed labour for days with fetal death followed later by leakage of urine and/or stool. A history of emergency CS or sexual violence may also be presented. Pelvic and vaginal examination using bivalved vaginal speculum aims to assess the fistula and its surrounding tissues and also to evaluate for prolapse or UI, which may alter the surgical plan.

Dye tests (methylene blue test) may be used to detect the site of leakage and if it is a single or multiple fistulas. If the methylene blue dye test is negative, the patient can be given pyridium orally and have a tampon placed in the vagina. A subsequent yellow stained tampon following a negative bladder fill is indicative of UTVF.

Retrograde cystogram may show a communication between the bladder and the vagina or pooling of the dye in the vagina after bladder emptying. Intravenous urography may show hydroureteronephrosis if UTVF present.

Other diagnostic procedures such as cystoscopy, contrast- enhanced computed tomography and magnetic resonance imaging may confirm the presence and location of fistula, identify any associated lesions and add more information to the diagnosis.

Treatment of OF

Conservative treatment of a fresh fistula by bladder drainage through 18 F urethral catheter may be an option in some patients with small fistulas (<1 cm) [144–147], but if healing has not taken place within a few weeks, surgical repair is required.

The treatment of choice for OF is surgery with the majority of reports showing success rates over 80 % for simple fistulas at the time of first operation [148–154].

The best chance for successful fistula closure is at the first operation and closure rates tend to diminish with each subsequent attempt of operative repair [155].

Surgical procedures for OF may be undertaken through vaginal, abdominal, combined vaginal and abdominal or laparoscopic approach.

Surgical principles for the treatment of simple OF include:

- Wide exposure and dissection of the fistula with ureters' protection by stents.
- Adequate tissue mobilization to ensure tension-free closure.
- Closure of the bladder or rectum in one or two layers and vagina separately.
- Adequate hemostasis after closure.

Other special considerations for the surgical repair of complex OF:

- The patient should be referred to a fistula center.
- Advanced training and surgical skills are prerequisites for treating this type of fistula.
- Proper selection of the most suitable approach as the patient may require urethral reconstruction or bladder augmentation during reconstruction and rarely some intractable cases may require urinary diversion.
- Relaxing posterolateral episiotomies may be needed.
- Well-vascularized inter-positioning tissue flap may be beneficial under certain circumstances (complex fistula, recurrent fistula and fistula associated with a difficult closure).

Postoperative Care

Adequate continuous bladder drainage for 2–3 weeks through urethral or suprapubic catheter. Antibiotics and anticholinergics are commonly used. Sexual abstinence for at least 3 months is recommended by many surgeons.

Prevention of OF

To reduce OF rates, effective preventive programmes that include family planning, prenatal care, safe labour, delivery and postpartum care are needed. Moreover, adequate support is required for interventions that focus on improving access to maternal health care, emergency obstetric care, and facilities for cesarean delivery when this is indicated [156].

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Childbirth Trauma and Lower Gastrointestinal Tract Complications

12

Filippo La Torre, Giuseppe Giuliani, and Francesco Guerra

Abstract

Pelvic floor and lower gastrointestinal tract complications as a consequence of vaginal childbirth are issues that have a great impact on women's well-being. They are also of great social and economic importance, even if the role of childbirth on pelvic and anorectal disorders is still an area of debate. Levator ani and anal sphincter injuries are associated with several complications, and women seem to be only partially protected by caesarean section. Pelvic organ prolapse (POP) and fecal incontinence (FI) are clinical consequences of childbirth trauma. In postpartum women, the first-line approach with medical and behavioral treatments often proves ineffective in treating FI. Rehabilitative therapy and less invasive procedures are preferred before performing standard intervention, while invasive procedures are to be discouraged.

Keywords

Childbirth trauma • Fecal incontinence • Levator ani muscle injury • Pelvic organ prolapse • Rehabilitative therapy

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Introduction

Despite an increased use of caesarean section, often chosen as elective option with the intent of decreasing morbidity associated with vaginal delivery [1, 2], the incidence of pelvic floor and lower gastrointestinal tract lesions as a result of vaginal childbirth remains an issue of great impact on women's well-being as well as an issue of social and economic importance. The higher prevalence of morbid obesity in the female population and the higher age of primiparous females, especially in western countries, have led to an increase in complications associated with vaginal delivery.

In fact, the role of the main protagonist in the pathogenesis of anorectal and perineal lesions is attributed by some authors to pregnancy per se, rather than to the trauma sustained at the time of delivery. To date there is no consensus about the real risk of serious injury from trauma associated with vaginal delivery [3–5].

What are the traumatic effects to the pelvic floor, and particularly anorectum, during vaginal childbirth? Anorectal tract disorders will be discussed, focusing on anatomical defects, specifically muscular structures, describing pathophysiology and symptoms associated with them, addressing clinical and diagnostic evaluation and general principles of medical and surgical treatment.

Injuries affecting lower gastrointestinal function will be treated separately, primarily muscle injuries associated with pelvic floor and those associated with anal sphincteric apparatus.

Pelvic Floor Injuries

Pelvic floor muscles, which can be identified as the complex of the *levator ani* muscle (LAM), is shaped like a slingshot that starts from the lateral pelvic wall, envelops the distal gastrointestinal tract – the anorectal junction – and runs on the opposite side to fit on the contralateral pelvic wall. The *levator hiatus*, which includes the urethra anteriorly, the vagina in its central part, and the anorectum posteriorly, has a surface of about 5–40 cm when stimulated with straining. The risk of injury that can result from the passage of the fetus is particularly related to the passage of the head, which has an approximate area of 70–100 cm^2 [6].

Numerous imaging studies have been conducted in this regard, often using MRI methods. The advent and spread of radiologic methods based on the use of 3D ultrasound allowed a much more detailed and dynamic study of the changes present in multiparous compared to nulliparous women [6]. In this regard, the size of the levator hiatus varies consistently if measured in nulliparous women, but prospective studies have demonstrated that the lesion of levator ani muscles is present in 13–36 % of women following vaginal delivery. It seems that the degree of muscle stretch varies widely in general population. Brooks and colleagues demonstrated that in passive muscles, individual strains of 50 % are necessary to produce significant lesions, while muscles in maximum activity exposed to a stretching of 30 % can be damaged, even if the degree of distension which can be considered maximal varies widely among individuals, according to MRI-based models. Lien and

collaborators have shown that the largest tissue strain occurs in the medial component of the pubococcygeus muscle due to its small original length and for its location at the midline. Overall, there are differences in ability to be strained due mainly to changes in muscle length at maximum Valsalva maneuver and the intrinsic properties of muscle. Indeed, it has been assumed that the hormonal effects of pregnancy may affect the properties of levator ani muscles. In this regard, Balmforth and coworkers have explored the importance of connective tissue's biochemical configuration, as an important factor in the development of pelvic organ prolapse (POP), stress incontinence, and normal progress of labor [7–12].

Even today, there are different definitions of LAM injury, based on clinical or imaging evaluations, by means of ultrasound or MRI assessment.

The definition of LAM injury according to Novellas and colleagues includes one of the following abnormalities: hypersignal muscle thickening or thinning, ruptured muscle's insertion, and abnormality in the iliococcygeus muscle, which can unilaterally or bilaterally appear flat or concave. Regardless of the definition used, the prevalence of trauma affecting the LAM in parous women varies depending on the technique used from 13 to 36 % [8, 13, 14]. Comparisons between different studies are often difficult, due to heterogenous patient selection or difference in obstetric practice. Certainly the lack of a universal definition of LAM injury remains the key issue on which a further clarification is strongly needed.

One of the main risk factors is forceps delivery: damage was demonstrated by means of ultrasound evaluation in 35–64 % of women after childbirth, with an odds ratio of 3.4–14.7. Anyway, it is not clear if the real injury is caused by the rapid passage of the head or by the type of forceps used [8, 15–17].

There is also evidence that a prolonged second stage of labor is associated with LAM injury, while ventouse delivery does not seem to increase the risk of LAM injury. Indeed, as stated by Kearney and coworkers, a second stage of labor longer than 78 min is associated with LAM injury. Furthermore Valsky revealed an OR of 2.3 when a second stage of labor was greater than 110 min [7, 18, 19].

The age at first birth also appears to be a risk factor (RF). The greater the age at first delivery, the greater the probability of LAM injury, although this correlation was not found significant by all study groups. The role of body mass index (BMI) does not seem to be clear: Shek and Dietz found that women with low BMI had a higher risk of LAM injury, although the clinical significance is quite questionable. On the other hand, epidural anesthesia proved to have a protective effect against LAM injuries [8].

Concerning lower gastrointestinal tract, the major problems associated with damage to the pelvic floor can be traced to fecal incontinence and pelvic organ prolapse.

Bowel Disorders

As described by Sultan et al. [20], up to 13 % of primiparous will develop fecal incontinence mainly to flatus.

There seems to be a clear relationship between LAM injury and fecal incontinence (FI) in older women, highlighting the importance of the LAM in preserving anal continence. This was demonstrated in a study conducted by Weinstein, in which an US-detected abnormality of LAM was correlated with an increased risk of developing FI compared to controls. On the other hand, major LAM injuries are significantly more common in women with sphincter tears than in those who delivered vaginally without sphincter tears or by caesarean. One in five women with a sphincter tear had a major LAM injury on MRI in a study by Heilbrun and colleagues, who found no major LAM injuries in women who delivered by caesarean without labor [21, 22].

Although the probability to develop postpartum fecal incontinence is highest in women after a vaginal delivery, an elective caesarean is not completely protective against the development of fecal incontinence [23]. Postpartum fecal incontinence is more likely if birth follows a late stage of labor with an emergency caesarean section.

Other risk factors to the development of fecal incontinence seem to be epidural anesthesia prolonging the second stage of labor resulting in pudendal nerve injury by stretching and compression lesions; the use of episiotomy and of forceps in women with epidural anesthesia are other risk factors to the development of fecal incontinence [24].

If a clear relationship between childbirth and fecal incontinence is really consistent, the link of constipation to childbirth is not so clear. Parity in general has been considered a predisposing factor for the outlet-type constipation due to pelvic floor prolapse and particularly rectocele. Although these conditions are present among patients who suffer from constipation, a correlation between the severity of prolapse and the prevalence of constipation and other bowel dysfunctions has not been confirmed [25].

Pelvic Organ Prolapse (POP)

Parity increases the risk for both POP and surgery for POP. In a study of Samuelsson et al., 31 % of 487 Swedish women had some degree of POP on examination: parity and age increased the risk of POP, after adjusting for other variables.

In a study designed to determine risk factors for surgical interventions for POP in women less than 45 years of age, Rinne and coworkers demonstrated that women with POP had more deliveries and babies with higher birthweight than age-matched controls operated for benign ovarian tumors.

LAM injury increases the risk of POP. Avulsion of levator ani appears to double the risk of anterior and central compartment prolapse, with minor effect on prolapse of the posterior compartment, although a link between rectal intussusception and avulsion has been suggested. A direct correlation between the size of the defect and symptoms and/or signs of prolapse has been suggested, and women with a bilateral lesion are more likely to suffer from uterine prolapse.

However, it is unclear if all women with LAM injuries develop a prolapse: is it only a question of time? In a case control study including a group of 151 women

with POP and 135 controls, DeLancey and colleagues found a significant OR of 7.3 for major LAM injuries but an equal number of minor defects. It was also demonstrated that, in women with major LAM defects, outcomes following surgical correction of POP appear to deteriorate in the short term and the risk of recurrence is increased [4, 15, 26, 27].

Acute LAM injury can be clinically diagnosed at inspection and digital examination, when the levator ani avulsion is associated with a large vaginal laceration; also a chronic detachment from the lower aspect of the pubis can be clinically detected, with the finger positioned laterally and parallel to the urethra and pushed up to the bladder neck: in this way the insertion of the puborectalis muscle on the pubic bone can be palpated immediately lateral to the finger.

According to Laycock [28], a strong pubococcygeus muscle of a young woman can be palpated like an elastic band of about 1–2 cm. An avulsion defect is diagnosed if the lower aspect of the pubis feels free from the muscle when moving the finger sideways. Palpation should be performed at rest and during contraction, to identify the presence of small portions of muscle. Overall, clinical evaluation by different clinicians was proved to moderately correlate with LAM defects. On the contrary, it has been shown that US evaluation leads to highly reproducible findings among different operators. Avulsion is diagnosed if a discontinuation between hyperechogenic fibers of puborectalis muscle and pelvic wall is present, with the insertion being replaced by a hypoechoic area representing the vaginal wall [21, 29–35].

Injuries to Anal Sphincteric Apparatus

The damage caused to the anal sphincters is common but underdiagnosed at the time of delivery. Between one-third and two-thirds [36] of women who have a significant laceration recognized during childbirth will suffer from fecal incontinence. In those women who present with symptoms of anal incontinence which continue to exist postpartum or those in whom FI develops subsequently, the incidence of anal sphincter injury (both external and internal anal sphincter, or EAS and IAS) is high, although severity of symptoms does not strongly correlate with damage entity. Bidimensional endoanal ultrasound (EAUS)-based studies suggest that injury involving one or both anal sphincter muscles occurs in up to one-third of primiparous women [20], although the true incidence of injuries is probably closer to 11 % [37] (Fig. 12.1).

On the other hand, many patients with significant defect at ultrasound may be clinically asymptomatic.

Afro-Caribbean women have a lower incidence of severe trauma at delivery than white European or Hispanic. Conversely, Asian women have an increased risk, which might be related to their relatively shorter stature [38]. Again, both obesity and high birthweight seem to increase the risk of perineal trauma [39, 40]. Furthermore, in women with a history of intermittent episodes of fecal incontinence after first delivery, there is an increased risk of overt fecal incontinence after

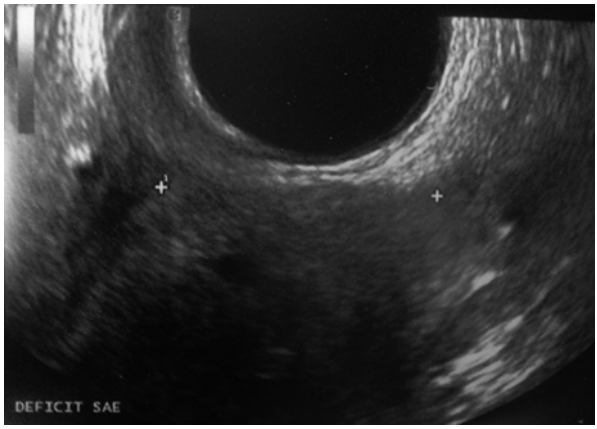

Fig. 12.1 EAUS of postpartum EAS lesion

subsequent delivery. In fact, in women with sphincter injury, the risk of further severe lesions is seven times greater compared to women with healthy muscles [36].

A persistent occipito-posterior position of fetal head is associated with higher risk of third- to fourth-degree anal sphincter injury. Instrument-assisted delivery, episiotomy, and conversion to caesarean section are often required in such cases. With occipito-posterior position, sphincter laceration occurs in 42 % of patients undergoing assisted deliveries with suction cup and in 52 % of those with forceps [36].

Likewise, the prolongation of the second stage of labor is associated with an increased risk operative vaginal delivery and anal sphincter injuries, with one-third of women who have a second stage of labor more than 4 h sustaining a third- or fourth-degree injury. Women encouraged to push immediately after full cervical dilatation have an increased risk of perineal trauma compared to those where pushing is delayed. Traditionally, it is taught that applying pressure against the perineum and the descent of the baby's head during delivery reduce the incidence of perineal injury. According to other studies, using the "hand-poised" method of childbirth whereby the accoucheur avoids touching the perineum and verbally guides the parturient, with occasional gentle support of the head, severe anal sphincter injuries and the need of episiotomy seem to be reduced, although these findings failed to reach significance [41, 42].

The risk of fecal incontinence following a non-extended midline episiotomy is three times higher when compared with spontaneous laceration. Injuries are more significant in cases of midline than those of mediolateral episiotomies.

Forceps-assisted delivery is associated with symptoms of fecal incontinence in more than 59 % of women with an incidence varying from 13 to 83 % in different studies. The risk seems to increase with occipito-posterior presentation.

Vacuum extraction compared with forceps delivery is associated with a lower incidence of clinically significant anal and perineal trauma. Delivery by caesarean section appears to play a protective role against anal sphincter injury when carried out as elective procedure or in the early stage of labor. Conversely, pelvic floor does

not seem to be fully protected by caesarean section, and abdominal delivery should be considered in women at risk of further trauma after precedent vaginal delivery resulting in anatomical defects, as well as in women with symptoms of fecal incontinence after previous vaginal birth [36, 43–47].

Diagnostic Aspects

Clinical Examination

The presence of pelvic floor or even perineal skin trauma should raise suspicion for injury to sphincters, immediately after childbirth.

For example, the presence of a large vaginal laceration after childbirth may be associated with acute LAM injuries [48]. A chronic detachment of LAM from the inferior ramus of the pubic bone can be diagnosed by vaginal examination. With the patient in lithotomy position, a finger is inserted 4 cm laterally and parallel to the urethra, with the fingertip at the level of the bladder neck. The puborectal muscle insertion to the pubic bone can be palpated lateral to the index finger about 2 cm proximal to the introitus and the pubococcygeus of a young women, and according to Laycock [17], it is felt like a 1- to 2-cm firm band. Hence a chronic detachment of LAM from the inferior ramus of the pubic bone is diagnosed when moving the finger laterally, whereby the inferior ramus appears free of muscle [4].

A rectal examination is necessary before any type of instrument assessment: low anal sphincter resting tone associated with a low pressure during the contraction raises the suspicion of anal sphincter injuries. It must be considered that significant injury of one or both sphincters may be evident in the absence of LAM injuries.

Imaging

Transanal bidimensional ultrasound is still the fastest method to study anal canal anatomy, although evaluating childbirth-related trauma provides discordant results according to the timing of investigation. An EAS or IAS is simple to recognize with endoanal ultrasound, a hyperechoic (EAS) or hypoechoic (IAS) ring.

As demonstrated by Santoro et al. with endovaginal ultrasound, it is possible to achieve good visualization of the LAM described as a hyperechoic sling lying posterior to the anorectum and attaching to the pubic bone, which resembles a “gothic arch” [4]; moreover with endovaginal ultrasound, LAM injuries can be diagnosed, with good to very good interobserver and interdisciplinary reliability [49, 50].

MRI is a second-line investigation that can be performed after an ultrasound assessment, if an ultrasound examination is inconclusive.

Functional Investigations

Functional tests remain useful after imaging evaluation to obtain clinically relevant information. The first, simple, and fast examination that must be performed is anorectal manometry that can measure resting and squeeze pressures and also anal

canal length. Resting pressure is impaired if an IAS defect is present; a reduction of squeeze pressure is typical finding of EAS injury.

The use of an intrarectal balloon can also evaluate the rectal compliance, sensitivity, and the rectoanal inhibitory reflex.

Altered rectal sensitivity or anal incontinence is usually transient after childbirth, with anorectal manometry showing a reduction in both resting and squeezing pressure immediately after delivery; but clinical evidence is generally poor and when significant tends to spontaneously heal. It seems that this condition is due to traction pudendal neuropathy postpartum; but clinical symptoms usually recover in about 2 months in 60 % of women. Anal incontinence may persist when weakness of pelvic floor coexists, but not all studies confirm this [51–55].

Another functional test is the pudendal nerve terminal motor latency testing (PNTML) that assesses the pudendal nerve function: although normal latencies do not exclude nerve damage, this examination is important before sphincter repair surgery because a prolonged value is a prognostic indicator of poor long-term functional success after surgery [56].

Finally there is no correlation between altered PNTML examination and presence of a sphincter defect [57].

Treatment

Pharmacological treatments are employed substantially in order to solidify the stool and prolong the intestinal transit. Constipating agents are indicated in most cases of postpartum women suffering from passive incontinence (leakage) or urgency with the intent of reducing the fecal mass and the frequency of bowel emptying. Reductions in episodes of fecal incontinence, fecal urgency, and loose stools have actually been demonstrated. Associated to clinical improvement, in patients treated with constipating agents, a reduction in weight and fecal content has been achieved. Loperamide is the most commonly used agent due to its minimal side effects and because of its greater efficacy compared to codeine and difenoxin associated with atropine. Agents promoting evacuation such as osmotic laxatives or glycerine suppositories can be adopted in cases of post-defecatory leakage or when overflow incontinence is present, often due to the presence of fecal impaction in chronically constipated individuals.

Rehabilitative therapy in the form of pelvic floor muscle training (PFMT) involves training on the right use of the pelvic floor muscles during contracting and straining, breathing, and changes in abdominal pressure, based on the use of electrostimulation and/or biofeedback. Biofeedback consists of exercises to strengthen anal sphincter and pelvic floor muscles, by improving rectal sensation and voluntary contraction of the EAS. Pelvic floor physiotherapy associated with changes in lifestyle is effective in treating patients suffering from fecal incontinence of different causes. In a study conducted by Norton and Kamm on 100 patients, it

was demonstrated that a lesion of EAS alone does not seem to affect the efficacy of treatment with biofeedback, while a lesion of the IAS appears to decrease the effectiveness of the treatment. In a subsequent randomized controlled trial on 171 women conducted by Norton, in both patients with intact sphincter muscles and those with sphincter disruption, including women with childbirth trauma, effectiveness rate was comparable. However, there seems to be a direct relationship between the extent of the defect in the EAS and the effectiveness of the treatment [68, 69].

Normally only patients suffering from severe anal incontinence, in whom conservative treatment failed, require surgery. Anal sphincter injury repair can be performed at the time of childbirth or later. Delayed surgery is performed by colorectal surgeons. EAS repair may have a short-term effectiveness in up to 54 % of patients, but symptoms can worsen over time. Indeed at 3-month follow-up, a residual muscle defect at ultrasound is still detectable in more of 90 % of patients and between 30 and 61 % of patients present with fecal incontinence. Although at the short term sphincter repair seems to improve symptoms, with increased resting and squeezing manometric pressures, symptoms seem to deteriorate progressively. In fact, 10 years after surgery, only 20 % of patients remain continent to liquids and solids [36].

Other surgical procedures, such as graciloplasty, the implantation of an artificial anal sphincter (AAS), or prosthetic trans-obturator sling (TOT) (Fig. 12.2), are far more invasive and generally not recommended due to frequency and number of complications and low success rates [58, 59].

Sacral nerve stimulation (SNS) and tibial nerve stimulation (TNS) improved stool continence in several studies. SNS consists of a direct electrical stimulation to sacral plexus by means of an inplanted electrode. TNS (Fig. 12.3) provides electrical stimulation with a needle electrode (percutaneous tibial nerve stimulation) or from two pad electrodes (transcutaneous tibial nerve stimulation) both inserted into

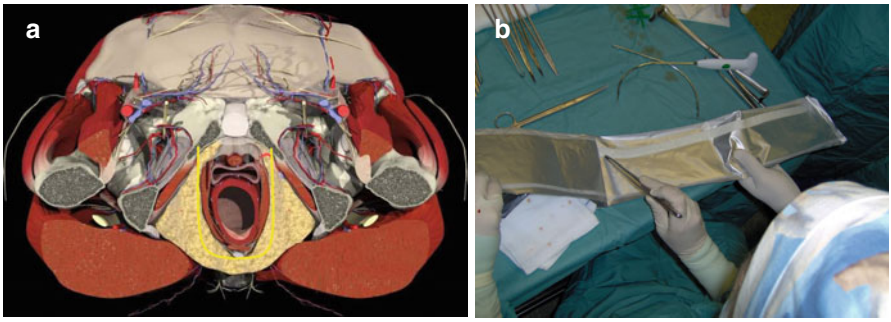

Fig. 12.2 (**a**) Anal sling: elastic structure that surrounds anorectal canal bilaterally fastened to obturator foramen. (**b**) Anal sling: the device patented in Italy, not on the market, includes the device and the *hammock* instruments to implant the prosthesis formed by a central body in biological material and four ends in nonabsorbable material to "suspend" the rectum and reposition it in the anatomical position

Fig. 12.3 PTNS device (Uroplasty®)

the lower, inner aspect of the leg, slightly cephalad to the medial malleolus aiming to transmit stimulation through the tibial nerve to the sacral plexus. Both SNS and PTNS showed low rate of complications and morbidity. PTNS treatment has shown an efficacy in the short term of up to 83 % [60] and in the long term, 53 %, reported by La Portilla after 2 years without treatment [61]. While for TTNS there are no studies with long-term outcomes, in the short term the efficacy is up to 60 %; in patients treated with bilateral TTNS, the efficacy is 85 % [62, 63].

In a meta-analysis that examined 34 studies published between 2000 and 2008 and included 790 patients, of whom 665 received a SNS permanent implant, SNS is an effective treatment for patients with FI compared to conservative treatment. In a multicenter, prospective nonrandomized trial that evaluated SNS in patients with FI, 83 % of 106 patients had a 50 % improvement and 40 % became fully continent, maintaining the improvement for 3 years [64, 65].

According to Wexner et al, the presence of an IAS defect, compared with its absence, is statistically associated with a lower likelihood of SNS treatment success [65].

According to Tan et al, the most common complications related with permanent SNS implantation are pain o local disconfort (6%), infection (3%) or seroma (3%),lead displacement or breakag (4%) [64].

Use of perianally injected bulking agents (BA) is a minimally invasive method for treating fecal incontinence, especially useful in those patients with higher risk of comorbidity in whom an open surgical procedure should be avoided, such as postpartum females. The procedure involves injecting prosthetic or autologous fillers into the submucosal tissues of the anorectum increase their volume and coaptation, thus preventing from incontinence episodes. Numerous studies have reported favorable short-term results with injectable perianal bulking agents, but according to Guerra et al. in the long-term follow-up, bulking agents seem to lose effectiveness and the ultrasound assessment of bulking agents suggests they are absorbed almost completely with time and the implants are no longer effective in treating incontinence [66, 67].

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Healing Process and Complications

13

Kostis I. Nikolopoulos and Stergios K. Doumouchtsis

Abstract

The general healing cascade involves four interrelated and overlapping phases, which are haemostasis, inflammation, cellular and matrix proliferation and wound remodeling. Multiple factors, such as the extent of the trauma, surgical skills, suture technique, the type of suture material and poor maternal hygiene, can affect the magnitude and degree of the associated short- and long-term morbidity.

Complications of wound healing, including wound haematoma, infection, dehiscence, breakdown, labial fusion, introital narrowing, pain and dyspareunia, can be extremely distressing for the new mother.

Understanding the various factors associated with wound complications is critical in antepartum counseling of patients, as well as intrapartum and postpartum practices. Modification of these factors may be essential in avoiding short-term complications and preventing long-term consequences.

Keywords

Childbirth trauma • Perineal tear • Tissue healing • Healing process • Healing complications • Paravaginal haematomas • Pubic bone injuries • Perineal infection • Wound dehiscence • Introital narrowing • Labial fusion

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Introduction

Pregnancy and childbirth can result in significant changes of pelvic floor anatomy and function. Changes range from observable trauma to the genital tract at the time of delivery to occult injury to nerves and muscles of the pelvic floor. Disorders caused by pelvic floor injury are multifactorial and include urinary and anal incontinence, prolapse, sexual dysfunction, and perineal pain [1].

Complications of wound healing, such as wound haematoma, infection, dehiscence, breakdown, labial fusion and introital narrowing can be extremely distressing for the new mother. Considering these complications and understanding the various associated factors, is critical in the antepartum counseling of patients, as well as intrapartum and postpartum practices.

Healing

Natural Wound Healing Process in Soft Tissue Injuries

Regardless of the type of injury, the wound healing process has common features among all soft tissues, with differences in duration of phases, and interactions between key mediators and time of healing [2–4]. The healing process begins at the time of wounding and involves soluble mediators, many cell types, and extracellular matrices. Wound healing cannot be considered a generic term but as a cascade of carefully regulated, interrelated processes. These processes, are initiated at the time of injury and proceed through repair [2].

The general healing cascade involves four interrelated and overlapping phases:

1. Haemostasis
2. Inflammation
3. Cellular and matrix proliferation, which is the most significant phase of healing and begins within days of trauma
4. Wound remodeling, the longest phase, which may lead to formation of scar tissue [5, 6]

Haemostasis and coagulation must occur quickly to prevent exsanguination. Immediately following trauma, capillary leak allows the recruitment of haemostatic factors and inflammatory mediators. The coagulation cascade is activated, leading to platelet aggregation, clot formation, and development of an extracellular matrix construct [7]. Mediators that are activated during this first phase, such as platelet-derived growth factor (PDGF) and thrombin peptides have overlapping regulatory effects on many of the cellular elements of early tissue repair such as fibroblasts and endothelial cells [2]. Platelets adhere to exposed collagen and circulating extracellular matrix proteins, which triggers the release of bioactive factors from alpha granules (growth factors, chemokines, and cytokines, in addition to pro-inflammatory mediators such as, bradykinin, serotonin, prostaglandins, thromboxane, histamine and prostacyclins) [8, 9].

Normal pregnancy is associated with major changes in many aspects of haemostasis, all contributing to maintain placental function during pregnancy and to prevent excessive bleeding in delivery. Most changes in blood coagulation and fibrinolysis create a state of hypercoagulability. This phenomenon protects the woman from haemorrhage during delivery, but predisposes her to thromboembolism both during pregnancy and in puerperium. These changes include a decrease in platelet count, increases in a number of clotting factors, a decrease in protein S levels, a significant fall in the activity of activated protein C and inhibition of fibrinolysis. The peak in clotting and platelet activity occurs immediately after placental delivery, whereas the peak of fibrinolytic activity during the first 3 h post-partum, as reflected by an increase in d-dimer levels [10, 11].

The second, **cellular and humoral inflammatory phase** must proceed in order to establish an immune barrier against external microorganism assault [2].

Neutrophils are the first subpopulation of leucocytes to appear at the injury site. They constitute the first line of defense against infectious agents that penetrate the body's physical barriers [7]. Chemoattractant agents begin to summon neutrophils to the traumatized site within 1–2 h of the injury. Later (around 48–72 h post-injury), **macrophages** appear in the wound and play the leading role in wound debridement and regulation of inflammation. They are also involved in recruiting fibroblasts and endothelial cells. Finally, the last cells to enter the wound during the late inflammatory phase are **lymphocytes**.

During pregnancy, there is physiologic regulation of the innate immune responses to prevent the rejection of the fetal allograft. Central to this adaptation are changes in cytokine production. Functionally, the cytokines can be divided into two groups: those involved in Th1 reactions (cell-mediated immunity) and those involved in Th2 reactions (humoral immunity). During pregnancy, there is an increase in regulatory T cells, as well as a shift in the responses from a Th1 to a non- inflammatory Th2 type. The factors responsible for Th2 related immune responses include hormones and cytokines secreted by the placenta including progesterone. The increase in the number of regulatory cells and the alteration in the Th1/Th2 balance are likely important reasons why several autoimmune diseases are mitigated during pregnancy [12].

The third, **cellular and matrix proliferation phase**, lasts from 48 h up to 6 weeks in part, because the cells involved serve as a metabolic engine, driving tissue repair. These cells originate from pluripotent progenitor cells in adjacent tissues. Two to three days following injury, macrophages and chemotactic, mitogenic, and angiogenic growth factors recruit **fibroblasts** and **epithelial cells** to infiltrate the site of injury [13]. Once in the wound, **fibroblasts synthesize collagen** and change to their myofibroblast phenotype, thus facilitating wound contraction. These cells produce Type III collagen, which appears in about four days, and is random and immature in its fiber organisation. Capillary germination occurs, supplying with nutrition the injured area, and collagen cross-linking begins. As the process proceeds, the number of fibroblasts decreases while more collagen is laid down. This phase ends with the beginning of wound contracture and shortening of the margins of the injured area. Angiogenesis and formation of granulation tissue are also important aspects during the proliferative phase of healing [14].

The final phase of the healing process involves **wound maturation and remodeling.** During this phase, growth factors such as PDGF, TGF-β (transforming growth factor–beta 1) and fibronectin, stimulate fibroblast proliferation, migration, and synthesis of the components of extracellular matrix (ECM) [15, 16]. The remodeling phase is tightly regulated to maintain the balance between degradation and synthesis. Gradually, cross-linking and shortening of the collagen fibers promote formation of a tight, strong tissue (scar tissue). **Final aggregation, orientation, and arrangement of collagen fibers occur during this phase.** Complete recovery of original tissue strength is rarely achieved, because repaired tissue remains less organized than non-injured tissue [17]. Type I collagen replaces Type III collagen, proteoglycan, and fibronectin through a process referred to as "creeping substitution" to form a more robust matrix with increased tensile strength [18]. The maturation phase varies in duration depending on the extent of the traumatized area, the individual characteristics, as well as specific tissue healing capabilities of the tissue involved [19, 20]. Pathophysiological, iatrogenic and metabolic factors can also affect wound healing. These factors include local causes, such as tissue hypoxia, ischaemia, necrosis and infection, as well as systemic causes, such as metabolic disease and nutritional status [21].

Surgical Techniques and Suture Material Affect Healing in Childbirth Trauma

Suturing Techniques

In addition to the extent of the trauma, surgical skill, suture technique and the type of material used for perineal trauma repair after childbirth can affect the magnitude and degree of the associated short- and long-term morbidity.

The preferable technique for perineal trauma repair should not be time consuming, (may prevent excessive blood loss), requires less use of materials which causes less short and long term complications, and requires less of a need to remove the sutures and lower resuturing rates [22].

There is limited evidence available to guide the choice between surgical repair and non-surgical management of first degree perineal tears sustained during childbirth. At present there is insufficient evidence to suggest that one method is superior to the other with regard to healing and recovery in the early or late postnatal periods. The discomfort tends to be of a different nature between women with surgical repair and non-surgical management. Typical symptoms in women with non-sutured trauma involve burning sensation, whereas in those with surgical repair mainly "stinging" and "pulling" discomfort. Clinicians' decisions whether to suture or not should be based on clinical judgment and the women's preference after informing them about the lack of long-term outcomes and the possible chance of a slower wound healing process, if left unsutured [23, 24].

Skin adhesive, a method for repair of cutaneous lesions, has been shown to be a safe and rapid alternative to traditional sutures in many skin locations. This material does not require subsequent removal, gives some elasticity to the wound site and seems to cause little foreign body reaction. In addition, octyl-2-cyanoacrilate has

been shown in vitro to be an effective barrier against microbial penetration [25–28]. A randomized clinical trial from 2009, showed that perineal skin closure using adhesive glue is faster than subcuticular suture, and associated with a similar incidence of complications and pain in the first 30 days [29].

For more than 70 years, researchers have been suggesting that continuous non-locking suture techniques for repair of the vagina, perineal muscles and skin are associated with less perineal pain than traditional interrupted methods. In 2012, a Cochrane review on suturing techniques for the repair of episiotomy or second-degree tears, concluded that continuous suturing techniques for perineal closure, compared with interrupted methods, are associated with less short-term pain (for up to 10 days' postpartum), need for analgesia and suture removal. Furthermore, there is also evidence that the continuous techniques used less suture material as compared with the interrupted methods (one packet compared to two or three packets, respectively) [30, 31].

Some obstetricians repair obstetric anal sphincter injuries by reapproximating the torn ends of the external anal sphincter (the end-to-end technique). Reports, however, have noted that 15–59% (mean 37%) of women have persistent anal incontinence symptoms despite primary repair [32]. This worrisome figure may be attributed to physicians' poor understanding of perineal anatomy, inadequate proficiency in surgical techniques, or possibly low efficacy of the end-to-end technique. However, in cases of delayed or secondary anal sphincter repair when women present with fecal incontinence, colorectal surgeons prefer to reapproximate the disrupted ends of the external anal sphincter by using the overlap technique. A modification of this technique for primary anal sphincter repair was first described in 1999 [33, 34]. According to the Royal College of Obstetricians and Gynaecologists guidelines, for the repair of the external anal sphincter, either an overlapping or end-to-end (approximation) method can be used, with equivalent outcome. Where the IAS (internal anal sphincter) can be identified, it is advisable to repair separately with interrupted sutures [35].

Abramov et al suggested that repair of obstetric anal sphincter tears by the overlapping sphincteroplasty technique with reconstruction of the internal anal sphincter and perineum, seems to carry favorable clinical outcome and to reduce the risk for anal incontinence, perineal pain, and dyspareunia [34].

Impact of Suture Material on Healing Process

A Cochrane review from 2010, provides evidence that perineal repair with catgut, which is still used in some settings, may increase short-term pain and wound breakdown, compared to synthetic sutures. Furthermore, the review reports few differences between standard polyglactin 910 (Vicryl) and rapidly absorbed synthetic sutures (Vicryl Rapide), however fewer women allocated to the rapidly absorbed suture material needed suture removal up to 3 months postpartum, compared to those who had standard material. This is important as women requiring removal of perineal sutures may find this particularly uncomfortable. In addition, if sutures remain in the tissues for a longer period than is required, they may cause a significant inflammatory response and increase the risk of infection [36–42].

According to the RCOG, when repair of the EAS (external anal sphincter) muscle is being performed, either monofilament sutures such as polydioxanone (PDS) or modern braided sutures such as polyglactin (Vicryl®) can be used with equivalent outcome. Also, when repair of the IAS muscle is being performed, fine suture size such as 3-0 PDS and 2-0 Vicryl may cause less irritation and discomfort and burying of surgical knots beneath the superficial perineal muscles is recommended to prevent knot migration to the skin [35].

Healing Complications

Postpartum Haemorrhage

Operative vaginal delivery, especially when using forceps, is a well-known cause of cervical and vaginal laceration, which may be responsible for postpartum haemorrhage. Postpartum haemorrhage due to vaginal or cervical laceration is generally controlled with appropriate emergency surgical repair and vaginal packing. However, in cases of persisting bleeding after suturing, pelvic transcatheter arterial embolization (TAE) may be indicated. TAE has been shown to be safe and effective in the treatment of postpartum haemorrhage due to cervical or vaginal lacerations following operative delivery with forceps. In addition, TAE helps perform subsequent and successful cervical and vaginal suturing when coagulation disorders are present and allows evacuating an associated paravaginal hematoma [43].

Haematomas

Paravaginal (Infralevator and Supralevator) Haematomas

The levator ani muscles divide the paravaginal space into an upper or supralevator fossa and a lower or infralevator fossa. A paravaginal hematoma is typically confined to the upper or lower compartment, although massive haemorrhage can break through the levator barrier.

Haemorrhage into an infralevator space causes massive swelling and ecchymosis of the labia, perineum and lower vagina on the affected side with severe vulval and perineal pain (Fig. 13.1). Anorectal tenesmus may result from extension into the ischiorectal fossa, while urinary retention may succeed spread ventrally into the paravesical space.

A supralevator haematoma, on the contrary, is not visible externally (Fig. 13.2). It can be felt as an insensitive rubbery mass protruding into the vaginal wall and potentially occluding the canal and causing vaginal or rectal pain and pressure symptoms.

In small not expanding infralevator haematomas, ice packs, analgesia and bladder catheterization may be effective, whereas surgical management is indicated for large or expanding haematomas, in order to prevent pressure necrosis, septicaemia and further haemorrhage.

Treatment options for supralevator haematomas are conservative with vaginal packing for 12–24 h and haemoglobin check, but if bleeding is intractable, internal

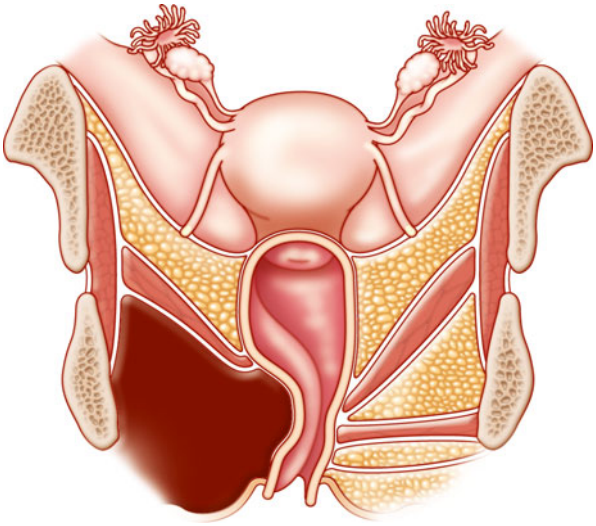

Fig. 13.1 Infralevator haematoma

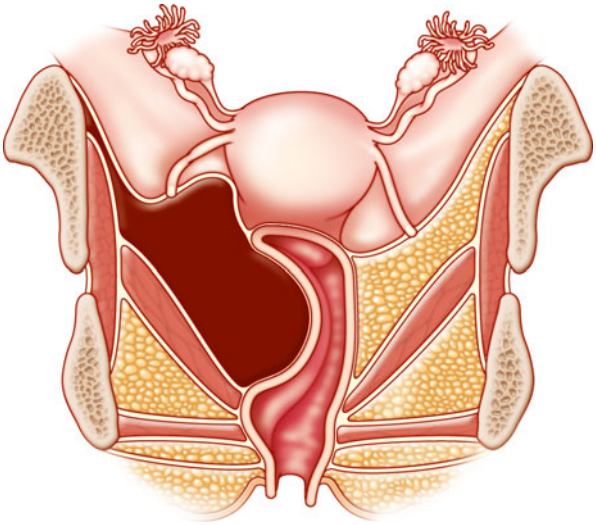

Fig. 13.2 Supralevator haematoma

iliac artery embolization or ligation may be indicated. The use of a Bakri tamponade balloon for haemostasis has been reported [44–46].

Vulval Haematoma

Vulval haematomas usually result from injuries to the branches of the pudendal artery during spontaneous vaginal or operative delivery or in conjunction with episiotomy. These vessels are typically located in the superficial fascia of the anterior (urogenital) or posterior pelvic triangle.

The superficial compartment of the anterior triangle communicates with the subfascial space of the lower abdomen below the inguinal ligament. Colles' fascia (superficial peritoneal fascia) and the urogenital diaphragm limit extension of bleeding into the anterior triangle, while the anal fascia limits extension of bleeding into the posterior triangle. As a result, bleeding is directed towards the skin where the loose subcutaneous tissues have little resistance to haematoma formation.

Superficial haematomas can extend from the posterior margin of the anterior triangle (at the level of the transverse perineal muscle) anteriorly over the mons to the fusion of fascia at the inguinal ligament. Necrosis caused by pressure and rupture of the tissue surrounding the haematoma may lead to external haemorrhage.

Large haematomas usually require exploration in the operating theatre. Initial resuscitation with IV fluids may be required, and blood should be sent for full blood count, coagulation screen and cross match.

An adequate linear skin incision should be made, the haematoma evacuated and bleeding points identified and ligated. The dead space is obliterated with interrupted sutures and the skin incision closed appropriately. Prophylactic antibiotics, urinary catheter, rectal examination and adequate postoperative analgesia are advisable [46].

Pain

Pain after delivery is common. Bone and soft tissue trauma might explain why some women have difficulty recovering postpartum owing to refractory pain or activity intolerance and the diverse clinical presentations of symphysis pubis dysfunction [47]. For most women, the associated pain and discomfort is temporary but in a minority it persists as chronic pain and discomfort [48].

Perineal Pain

Perineal discomfort and pain in the days after a vaginal delivery is common. Abraham et al. showed that perineal pain may persist up to 6 months after vaginal delivery, 20 % experiencing discomfort for more than 2 months [49].

Pain following obstetric anal sphincter injury (OASI) can be severe. Severe perineal pain has been reported by 100 % (N/N) of women on day 1 and by 91 % (N/N) of women on day 7 following third-degree and fourth-degree tears [50, 51].

Treatment options for perineal pain include oral and in severe cases rectal analgesia [51]. Following primary repair after OASI, laxative use is recommended. Stool softeners prevent from faecal impaction and possible damage to the recently repaired sphincter. Also, laxatives lead to a significantly earlier and less painful first bowel motion and earlier discharge [51].

The beneficial impact of massage on postnatal pain is noteworthy. Women who undertook perineal massage had lower perineal pain scores than those who did not. The practice of antenatal massage enables women understand the anatomy of their perineum and manage effectively their postnatal perineal pain.

Chronic perineal pain that does not respond to analgesics and massage may require perineal injections with local anaesthetic and steroids, which appear effective [52].

Levator Ani Muscle Injuries

The reported incidence of levator ani muscle (LAM) trauma is as high as 15 % at first vaginal births [53–55]. To detect these injuries, imaging techniques like MRI, transperineal ultrasound and endovaginal sonography can be used [56]. Acute LAM injuries can be diagnosed clinically by visualization and digital examination. Levator avulsion can be associated with a large vaginal tear. Levator avulsion appears to double the risk of significant anterior and apical compartment prolapse, with less effect on posterior compartment prolapse. There is a direct correlation between the size of the defect and the symptoms and/or signs of prolapse [57].

Pubic Bone Injuries

Injuries to the pubic bones and pubic symphysis, are known to occur, and can be evaluated by magnetic resonance imaging (MRI).

An observational study of women who underwent MRI after delivery, showed pubic bone fractures in 38 % of women at high risk for pelvic floor injury (risk factors: second-stage labour >150 min or <30 min, anal sphincter tear, forceps, maternal age >35 years and birth weight >4000 g) and in 13 % of women at low risk for pelvic floor injury. In the same study, levator ani muscle tears were present in 44 % of high-risk women and in 9 % of the low-risk women and bone marrow oedema in the pubic bones was present in 61 % of women studied across delivery categories [47].

Separation of the pubic symphysis is a recognized complication of childbirth with an incidence of 1 in 600 to 1 in 30,000 deliveries [58]. However, a more recent review showed an incidence of peripartum pubic symphysis diastasis to be 1 in 500 [59].

These injuries are often associated with significant pain and disability for prolonged periods of time after delivery. Although conservative treatment often leads to resolution, invasive orthopaedic treatments are sometimes required if pain is significant or the diastasis fails to resolve [58, 60].

Perineal Wound Infection

Approximately one in ten women who sustained a perineal tear at vaginal delivery that required suturing, develop perineal wound infection. Instrumental deliveries and prolonged rupture of membranes predispose women to this complication [61–63]. By 2 weeks postpartum, patients who received prophylactic antibiotics at the

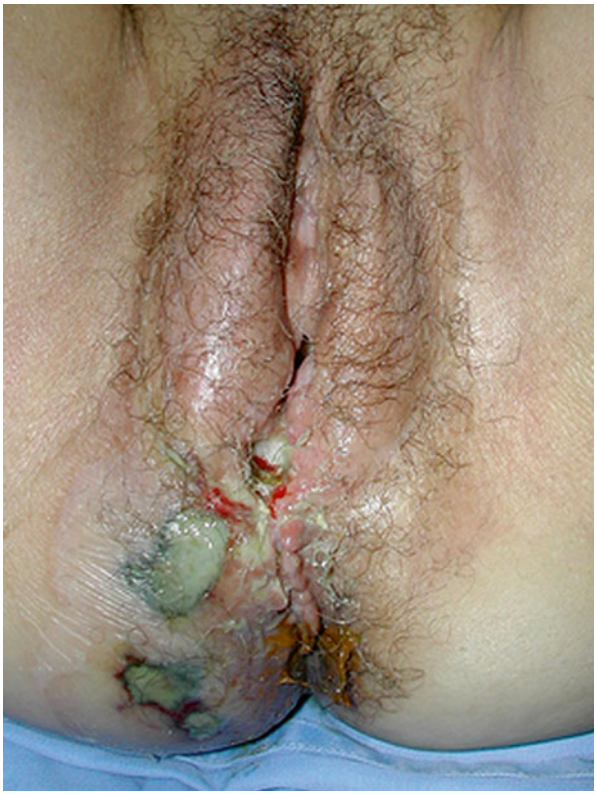

Fig. 13.3 Necrotizing perineal infection. (With kind permission from Springer Science+Business Media: Silva-Filho [81])

time of third- or fourth-degree tear repair had a lower rate of perineal wound infectious complications than patients who did not [63].

The development of infection poses a greater risk for wound breakdown, fistula formation and anal incontinence. Given the severity of these complications, most authorities consider it prudent to prescribe antibiotic cover for both aerobic and anaerobic bacteria following primary repair [35, 51] (Figs. 13.3 and 13.4).

Wound Dehiscence/Breakdown and Management

Perineal wound breakdown is one of the most devastating complications, with an incidence of 0.1–4.6% [64–66]. Although uncommon, perineal wound breakdown can lead to significant morbidity.

Up to 80 % of wound dehiscence cases are secondary to wound infection. The possible contributing factors of genital infection, which may lead to dehiscence, can be divided into antepartum, intrapartum and postpartum.

Antepartum risk factors include extremes of maternal age, smoking, poor maternal hygiene, poor nutrition and preexisting medical conditions such as diabetes,

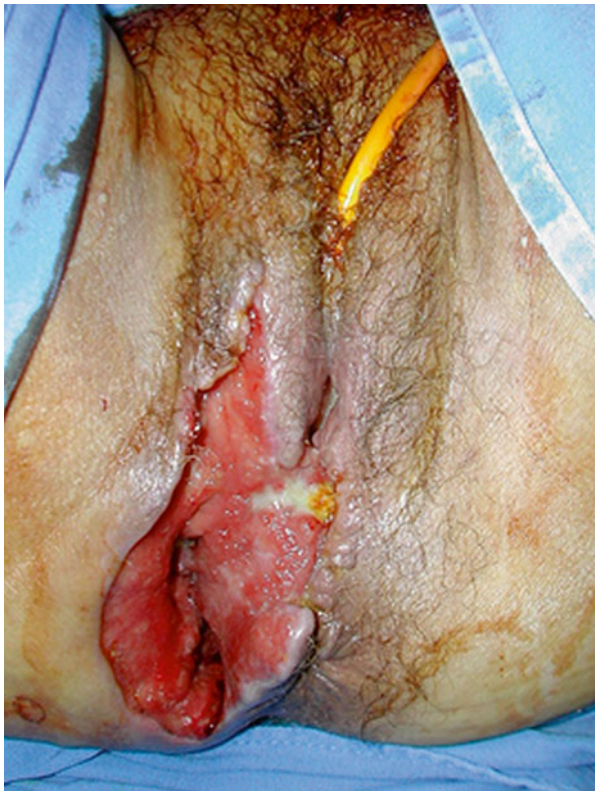

Fig. 13.4 Debridement of necrotizing perineal infection (With kind permission from Springer Science + Business Media: Silva-Filho [81])

immunocompromise, severe anaemia and bacterial vaginosis, chlamydia, gonorrhoea or trichomoniasis.

Intrapartum factors include prolonged rupture of membranes, thick meconium staining, prolonged labour, intrapartum pyrexia, multiple internal examinations, operative vaginal delivery, poor aseptic technique, manual removal of placenta and retained products of conception.

Postpartum factors include delayed or omitted prophylactic antibiotics, suboptimal haemostasis, haematoma, contamination of wound or surgical site and residual dead space following wound closure [67].

Obesity remains an independent risk factor for wound infection and this may also apply to the perineum. A large retrospective study compared maternal outcomes in nearly 800 women based on pre-pregnancy weight. Women who were moderately obese (pre-pregnant weight, 90–120 kg) were 1.6 times (95% CI: 1.31–1.95) more likely to have caesarean wound and episiotomy infections when compared with non-obese women, and women who were severely obese (pre-pregnant weight, >120 kg) were 4.45 times (95 % CI: 3.00–6.61) more likely to have a wound infection when compared with non obese women [68].

Interestingly, a retrospective case – control study on 47 women whose episiotomies dehisced in the immediate postpartum period, found that human papilloma

virus (HPV) infection was associated with poor healing of episiotomy repairs, as HPV was detected in up to 30 % of patients with episiotomy breakdown [69].

The management of wound breakdown varies depending on individual clinician's preferences, as there is limited evidence and lack of guidelines on best practice. Most practitioners manage these cases conservatively, whereas, others offer secondary suturing.

The traditional approach is to allow healing by secondary intention, whereby the dehisced area fills with granulation tissue that gradually contracts to bring the wound edges together; however, this is a slow process and can take several weeks for the wound to heal completely. This approach may result in a protracted period of significant morbidity for women whereas re-suturing of perineal wound dehiscence within the first 2 weeks following childbirth may result in a reduction of perineal pain during the healing process for up to 6 months post-delivery, an improvement of dyspareunia symptoms, continuation of exclusive breastfeeding for up to 6 months and increased satisfaction with the aesthetic result of the perineal wound [70]. There is currently insufficient evidence available to support or refute secondary suturing for the management of broken down perineal wounds following childbirth.

Sexual Morbidity Secondary to Healing Complications

Childbirth trauma causes considerable maternal morbidity such as perineal pain, vulval and vaginal stenosis, scar formation and gaping wound that leads frequently to decreased libido, dyspareunia and decreased sexual satisfaction in the months following vaginal delivery. General practitioners should consider referral to hospital for consideration of secondary repair if indicated [71].

Dyspareunia is defined as any pain that occurs during sexual intercourse, and affects a significant number of women following childbirth, approximately 20 % at 3 months postpartum [51, 72]. Twenty percent of women take longer than 6 months before sexual intercourse becomes comfortable. Inadequate repair of an episiotomy or vaginal tear can also lead to longstanding perineal discomfort and dyspareunia, so attention to anatomy and good surgical technique is important [73]. Women with obstetric anal sphincter injuries are at increased risk for dyspareunia after their delivery [74].

Traditional treatment includes perineal massage, topical oestrogens, perineal injections and, more recently, the use of intravaginal electrical stimulation.

De Oliveira Bernardes and Bahamondes [75] showed that electrical stimulation applied vaginally, was effective treatment for chronic pain with significantly fewer complaints of dyspareunia following treatment – an effect that lasted 7 months after treatment [51].

Surgical treatment is used for introital enlargement following failed manual dilatation. The appropriate surgical procedure depends on the site and extent of the vaginal constriction, the state of the surrounding tissue, and the overall length and caliber of the vagina. Fenton's procedure, Z-plasty, vaginal incision of constriction ring, vaginal advancement, or placement of free skin graft are the most commonly used techniques [76] (Fig. 13.5).

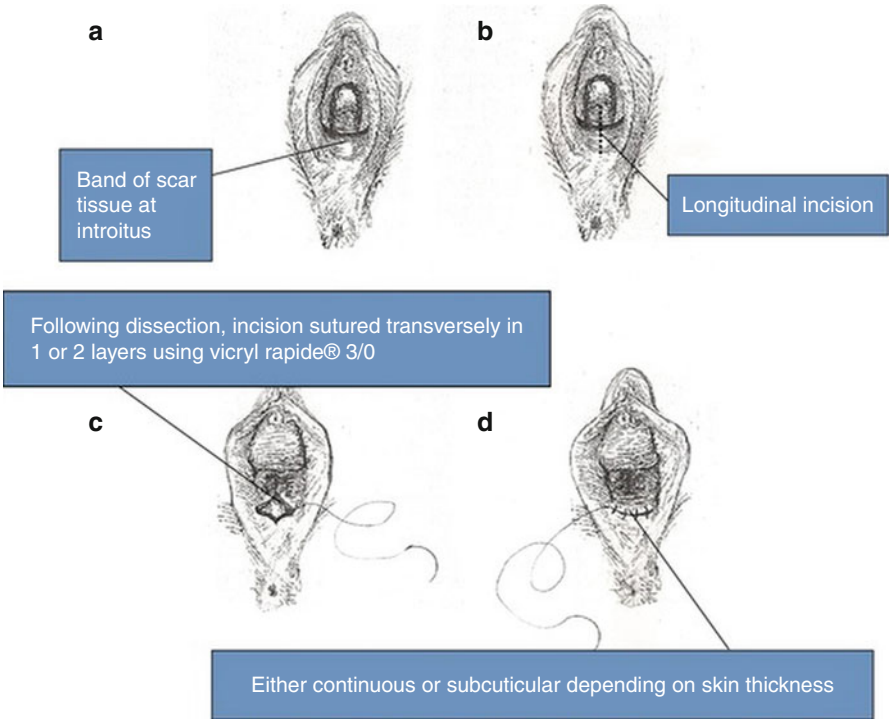

Fig. 13.5 Fenton's procedure. (**a**) Band of tissue. (**b**) Longitudinal incision. (**c**) Transverse suturing. (**d**) Final result (With kind permission from Springer Science + Business Media: Chandru S [82])

Labial Fusion: Introital Asymmetry

Spontaneous approximation of lacerations of the labia may lead to distorted anatomical healing, with resultant dyspareunia, among other distressing symptoms. Prevention of labial or clitoral adhesions may be possible through personal hygiene techniques of instructing women to manually gently separate the labia several times a day while urinating. Oestrogen cream has been used for the management of adhesions of epithelium of the external genitalia, Surgical correction may be necessary when medical treatment fails [77].

Obstetric Fistula

Obstetric fistula, an opening that forms between the vagina and the bladder and/or the rectum, is most frequently caused by unattended prolonged labour, when the pressure of the baby's head against the mother's pelvic tissues cuts off blood supply to delicate tissues until it causes necrosis. Obstetric fistula is one of the most severe childbirth injuries that occur when labour is allowed to progress for a long period

without timely intervention. Estimates indicate that more than two million women worldwide live with vesicovaginal fistula (VVF) or rectovaginal fistula (RVF) and the majority of these women reside in Africa and Asia [78–80]. Obstetric fistula is discussed in more detail elsewhere in this book.

Conclusion

Perineal wound complications can be associated with significant morbidity. Understanding the various factors associated with wound healing and complications is critical in antepartum counseling of patients, as well as intrapartum and postpartum practices. Modification of these factors may be critical in preventing long-term consequences such as wound infection, wound breakdown, fistula formation and introital narrowing. Furthermore, identification of these factors will determine the need for follow up of these patients in the postpartum period.

Health professionals must recognize that women may not volunteer information about dyspareunia and faecal incontinence in particular and, therefore, they must obtain a full history of symptoms by direct albeit tactful questioning.

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Pregnancy, Puerperium and Pelvic Organ Prolapse

14

Jittima Manonai

Abstract

Pelvic organ prolapse (POP) is a common condition that develops secondary to multiple factors. POP is associated with affected quality of life, loss of productivity, and increased financial burden on the healthcare system. Many studies in literature show that pregnancy and childbirth negatively affect pelvic floor structure and function. Vaginal delivery clearly has an influence on subsequent POP, especially the first childbirth that is critical for the major change of the pelvic floor. Obstetric pelvic floor trauma (levator ani muscle injury) has been linked to the pathogenesis of POP and this condition can be demonstrated using magnetic resonance imaging, 3/4D transperineal ultrasound and 3D endovaginal ultrasound. Pelvic organ prolapse in pregnancy may cause maternal and fetal complications. The reported prevalence of POP after childbirth is 15–48 %. Previous studies define vaginal delivery, usage of forceps and length of the second stage of labour as risk factors for postpartum POP.

Keywords

Pelvic organ prolapse • Pregnancy • Childbirth • Puerperium • Levator ani muscle injury

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Introduction

Pelvic organ prolapse (POP) is defined as the descent of the vaginal walls, the uterus, or the apex of the vagina [1]. It is a major health issue for women in both developed and developing countries [2–4]. Although the etiology is multi-factorial, injury to the pelvic floor from childbirth is one of the most important risk factors for the development of POP [5, 6]. Several studies reported that POP is more common among parous women than nulliparous women [7–9]. Estimates based on population studies in North America have shown that 50 % of multiparous women develop a mild form of pelvic organ prolapse, while nulliparous women account for only 2 % of prolapse cases [10]. In the literature, pregnancy is one of the most frequently cited lifestyle-related risk factors for POP, accounting for 20–40 % of the risk, with severity increased by gravidity, parity, vaginal delivery and weight of an infant delivered vaginally [11]. It seems that pelvic floor injuries that take place during pregnancy, labour and delivery may predispose to POP later in life.

Vaginal childbirth is found to be associated with prolapse symptoms [12]. The major change in pelvic floor status seems to occur after the first pregnancy and delivery. Furthermore, the first vaginal delivery is the time when women sustain the most significant pelvic floor damage [13]. The relationship between the number of vaginal births and prolapse to or beyond the hymen was examined in a cross-sectional study of women seeking gynecology care. The authors found a 10-fold increase in the odds of prolapse with a single vaginal birth but no significant increase in the odds of this condition for additional vaginal births [14]. This chapter will focus on the effect of pregnancy and childbirth on POP as well as POP during pregnancy and the long-term sequelae of pelvic floor injuries after childbirth.

Pregnancy, Childbirth and Pelvic Organ Prolapse

Pelvic Organ Support and Pelvic Organ Prolapse

Pelvic organ prolapse results from attenuation of the supportive structures, whether by actual tears or breaks or by neuromuscular dysfunction or both. Pelvic support structures include the muscles and connective tissue of the pelvic floor, the fibromuscular tissue of the vaginal wall and the endopelvic connective tissue. Endopelvic connective tissue includes (1) the cardinal/uterosacral complex (2) lateral connective tissue attachment of the anterior vaginal wall to the arcus tendineus fascia pelvis and of the posterior vaginal wall to the fascia of the levator ani and to the posterior arcus tendineus near the ischial spine and (3) less dense areolar connective tissue surrounding retroperitoneal portion of the pelvic organs [15].

The muscles of the pelvis include those of the lateral wall and those of the pelvic floor. The pelvic diaphragm forms the primary supporting structure for the pelvic contents. It is composed of the levator ani and the coccygeus muscles. The levator ani muscles are composed of the pubococcygeus (including the pubovaginalis and pubourethralis), puborectalis and the iliococcygeus [16]. The levator ani assists the

anterior abdominal wall muscles in containing the abdominal and pelvic contents. Levator ani muscles are innervated solely by a nerve traveling on the superior (intrapelvic) surface of the muscles. The levator ani nerve originates from S3, S4 and/or S5 and innervates both the coccygeus and the levator ani muscle complex [17].

Effects of Pregnancy and Childbirth on Pelvic Organ Support

The mechanisms by which pregnancy and childbirth lead to failure of pelvic organ support are not completely understood. During pregnancy the urogenital system and pelvic floor itself undergo anatomical and physiological changes. An increase of all hiatal dimensions as well as bladder neck mobility was found from 21 weeks to 37 weeks of gestation in nulliparous pregnant women using three-dimensional and four-dimensional transperineal ultrasonography at rest, during contraction, and during Valsalva maneuver [18]. Pregnancy itself, by means of mechanical changes of pelvic statics and changes in hormones, can be a significant risk factor for pelvic floor damage. During childbirth, the levator ani supports the fetal head while the cervix dilates. Thereafter, the passage of the baby through the birth canal is thought to result in a mechanical distortion that damages the pelvic floor connective tissue and muscular supportive structures, as well as the nerves and vessels that supply these structures [19–22]. The endopelvic fascia and other connective tissue elements of the pelvic floor are at risk of stretching and detachment from their bony attachments during childbirth. Ultimately, these changes may lead to persistent or permanent modifications in the proper function of pelvic floor muscles.

Additionally, difficult or prolonged labour may exceed the stretch limits of the soft tissue, causing imbalance in the reparative and degenerative processes leading to progression of pelvic organ prolapse [23]. After delivery, there is substantial remodeling of the connective tissue components. This is accomplished by an increase in collagen and elastin synthesis in response to mechanical stretch [24]. However, the new tissue that results from healing after childbirth is not as strong as the original tissue that it replaces. Collagen levels, history of collagen disease and childbirth-related pelvic floor trauma are associated with pelvic organ prolapse [25].

Regarding route of delivery, vaginal delivery and emergency cesarean section have been associated with denervation injuries of the levator ani muscle with an incidence of 30 % [26]. Prolapse is noted to be more common in women after a vaginal delivery than after a cesarean section [27]. One study found that nulliparous pregnant women had an increased Pelvic Organ Prolapse Quantification (POP-Q) stage as compared with nonpregnant controls [28]. Thus, both pregnancy and delivery are important causal factors for the development of pelvic organ prolapse. Increasing parity also leads to a linear increase in the probability of developing prolapse [9]. In a British study, women with one child were four-times more likely, and those with two children were eight-times more likely to develop pelvic organ prolapse when compared with nulliparous women [8].

Levator Ani Muscle Injuries and POP

The anterior portion of the levator ani complex, serves to close the urogenital hiatus and pull the urethra, vagina, perineum, and anorectum toward the pubic bone. The horizontally oriented posterior portion serves as a supportive diaphragm behind the pelvic organs. Injury to the levator ani muscle is attributed to an important explanation of the effect of vaginal delivery on the development of POP. Loss of normal levator ani tone, through denervation or direct muscle trauma during vaginal delivery, results in laxity of the urogenital hiatus [29]. Increasing urogenital hiatus size is associated with pelvic organ prolapse and uterine cervix descent at straining was correlated with the hiatus size and levator plate angle at straining [30, 31].

Recently, several studies have been conducted to evaluate the association of levator ani muscle injury and the symptoms and stage of POP [32, 33]. There is evidence that 36 % of women with prolapse have an underlying levator ani muscle avulsion using four-dimensional translabial ultrasound. Women with levator avulsion defects are about twice as likely to have POP of stage II or higher than those without [34]. Several studies confirm that irreversible overdistension of the levator hiatus due to vaginal delivery is related to levator avulsion [35–37] and is a risk factor for POP [38, 39]. Levator avulsion appears to double the risk of significant anterior and apical compartment prolapse, with less effect on posterior compartment prolapse [34]. In addition, a moderate positive correlation was demonstrated between levator ani deficiency score and stage of prolapse [40].

Howard Gainey first described defects in pelvic floor muscles following childbirth in 1943 [41], with a second report from the same author in 1955 [42]. There are various definitions of levator ani muscle injury, according to method and technique of assessment, i.e., clinical palpation, ultrasonography or magnetic resonance imaging [43]. The effect of vaginal childbirth on pelvic floor structures, such as the levator ani and puborectalis muscles, and the pelvic fascial structures, has recently been studied using advanced techniques such as 3- or 4-dimensional ultrasound and magnetic resonance imaging [30, 35, 38, 39, 44, 45].

MRI has previously shown abnormalities in the levator ani in women with stress incontinence and prolapse (Fig. 14.1) [30, 46]. Regarding the MRI studies in nulliparous women and women after their first vaginal birth, 20 % of primiparous women had a visible defect in the pubovisceral or iliococcygeal portion of the levator ani muscle. Major and minor defects in the pubovisceral muscle were seen in 13.7 % and 4.4 %, respectively. These defects were not seen in nulliparous women [44, 47].

Later on, levator ani muscle defects have been found using translabial ultrasound. Vaginal delivery resulted in an increased prevalence and size of a defect in the rectovaginal septum. These defects are associated with symptoms of pelvic organ prolapse and obstructed defecation [48, 49]. In another study by this group [45], it was shown that the avulsion of the levator ani from the pelvic sidewall was demonstrated in one-third of women who had vaginal delivery. This avulsion occurs in 14–22 % of women during the first vaginal delivery using three or four-dimensional translabial ultrasonography (Fig. 14.2) [50–53] by stretching and tearing of the muscle from the insertion on the inferior pubic ramus [54].

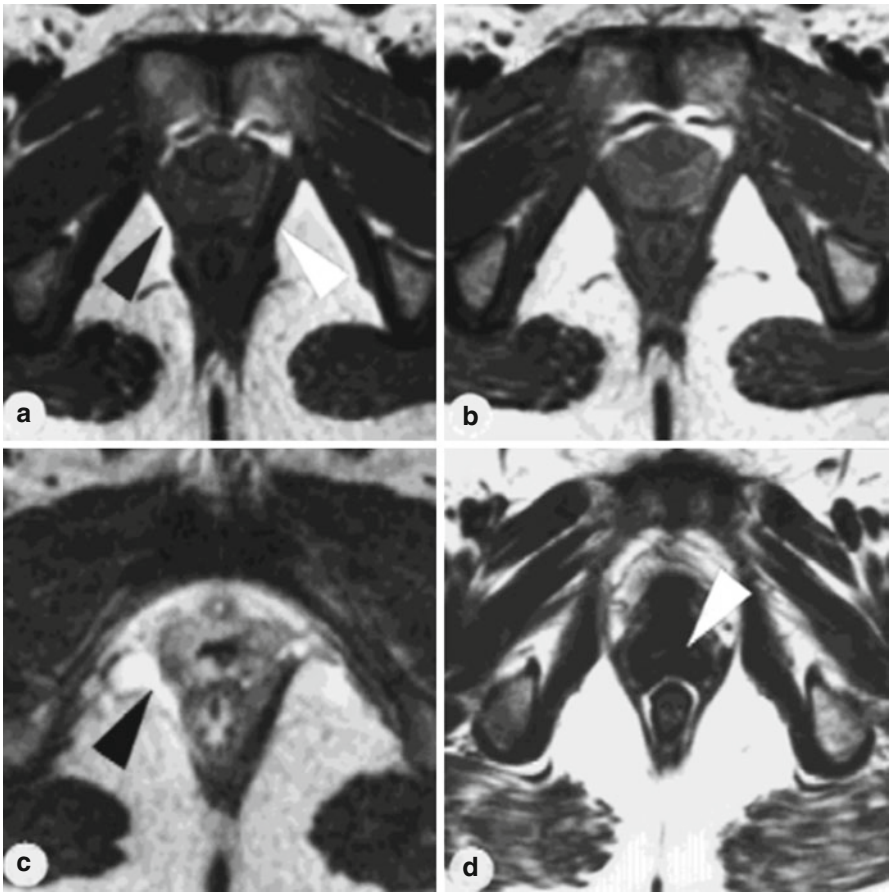

Fig. 14.1 Magnetic resonance imaging findings of levator musculator (LM) at the level of the proximal urethra. (**a**) Control subject, intact LM on both sides. LM subjectively thinner on the right (*black arrow*) and thicker on the left (*white arrow*) due to pronounced chemical shift artifact. (**b**) Similar appearance of the LM on both sides. Minimal chemical shift. (**c**) No demonstration of striated muscle tissue in the right LM (*black arrow*); status post-forceps delivery. (**d**) Loss of the hammock-like configuration of the vagina (*white arrow*), thin LM on both sides (Reprinted from Tunn et al. [46] with permission from John Wiley and Sons)

Recently, an observational longitudinal cohort study aimed to establish the incidence of levator ani muscle avulsion in primiparous women and to develop a clinically applicable risk prediction model. Nulliparous women at 36 weeks of gestation and 3 months postpartum were recruited. Four-dimensional transperineal ultrasound was performed during both visits. Tomographic ultrasound imaging at maximum contraction was used to diagnose no, minor or major LAM avulsion.

Following vaginal delivery, the overall incidence of LAM avulsion was 21.0 %. Minor and major LAM avulsions were diagnosed in 4.9 % and 16.1 %, respectively.

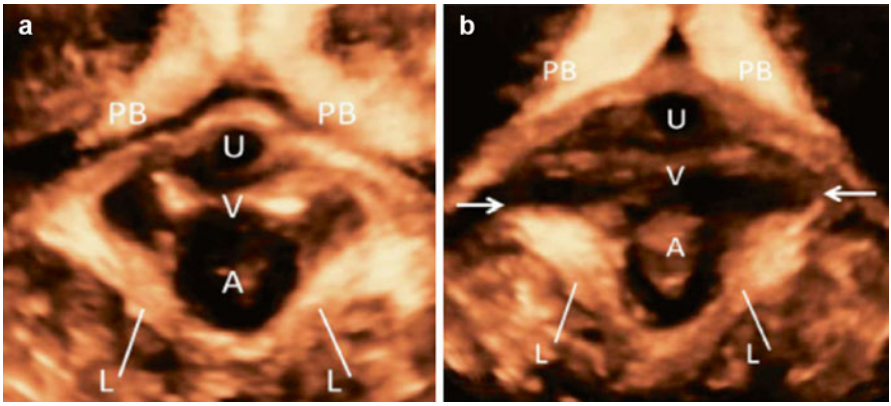

Fig. 14.2 Transperineal ultrasound findings of lavator hiatus at rest in rendered volume. *PB* pubic bone, *U* urethra, *V* vagina, *A* anus, *L* levator ani muscle. (**a**) Normal antenatal levator hiatus. (**b**) Abnormal postnatal levator hiatus. *Arrows* indicate bilateral LAM avulsion (Reprinted from van Delft et al. [53] with permission from John Wiley and Sons)

Risk factors were obstetric anal sphincter injuries (odds ratio 4.4, 95 % CI 1.6–12.1), prolonged active second stage of labour per hour (odds ratio 2.2, 95 % CI 1.4–3.3) and forceps delivery (odds ratio 6.6, 95 % CI 2.5–17.2) [55].

3D endovaginal ultrasonography (EVUS) has been used for visualization of the levator ani muscle injury. The terminology for levator ani defect for EVUS is different and levator ani muscle is divided into three subdivisions: (1) pubovaginalis (puboperinealis + puboanalis), (2) puborectalis, and (3) pubovisceralis (pubococcygeus + iliococcygeus) [56]. Concerning the role of endovaginal ultrasonography on levator ani muscle injury detection, transperineal and endovaginal ultrasound can both be used to analyze hiatus area and anteroposterior diameter with the patient at rest and to diagnose levator avulsion (Fig. 14.3) [43, 57].

A prospective, observational study showed that defects of the pubovisceral muscle were identified with 3D endovaginal ultrasonography in 27 % of women with faecal incontinence that had undergone vaginal delivery. Furthermore, vaginal delivery results in enlargement of the levator hiatus and a lower position of the anorectal junction and bladder neck compared with nulliparous women [58].

Primigravid women were examined using EVUS prior to delivery, early postpartum and 3 months postpartum. The results showed that puborectalis avulsion was found in 15.7 % and 13.3 % of women at 20 h and 13 weeks postpartum, respectively [59].

Pelvic Organ Prolapse in Pregnancy

Prevalence and Natural History

Uterine or cervical prolapse complicating pregnancy is a rare event, with an estimated incidence of 1 in 10,000–15,000 deliveries [60, 61]. An earlier study had

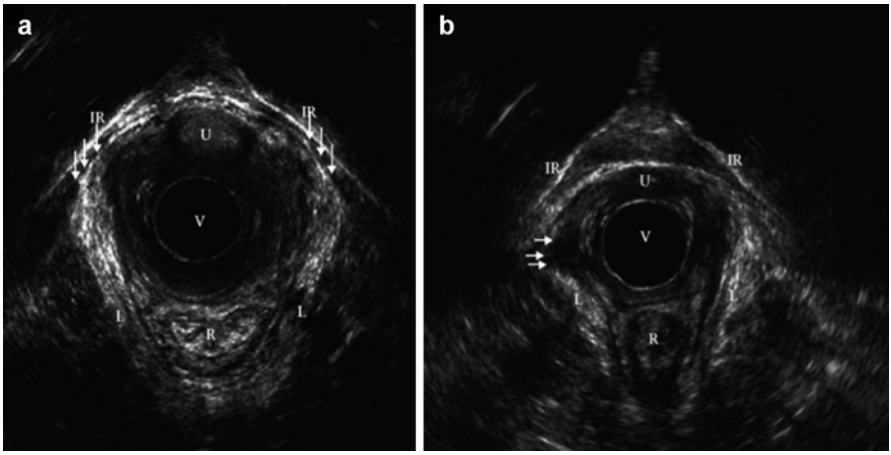

Fig. 14.3 Endovaginal ultrasound findings of levator ani muscle (LAM). *IR* inferior rami os pubis, *L* levator ani muscle, *R* rectum, *U* urethra, *V* vagina (with endovaginal probe). (**a**) A nulliparous woman with intact LAM. (**b**) A primiparous woman after forceps delivery involving a right mediolateral episiotomy and a third-degree tear with unilateral avulsion injury. *Arrows* indicate missing LAM on patient's right side (Reprinted from Schwertner-Tiepelmann et al. [43] with permission from John Wiley and Sons)

identified only one case among more than 13,000 obstetric admissions during a 14-year period [61]. This condition is due to poor cardinal ligament and uterosacral ligament support; therefore, it is categorized as an apical compartment prolapse. However, descent of the uterine cervix may also be aggravated by pregnancy as a result of physiological increases in cortisol and progesterone, which leads to a concomitant softening and stretching of the pelvic tissues, thus causing prolapse during pregnancy [60]. The physiologic changes of pregnancy- in terms of cervical elongation and hypertrophy- with pregnancy-related hormonal changes such as increased progesterone and decreased relaxin, may lead to reduced strength and supportive function of the pelvic floor muscle, can also contribute to prolapse. Even though uterine prolapse frequently complicates pregnancy in multiparous women, POP of nulliparous women has also been reported. Of note, a case report described uterine prolapse in a primiparous woman who had multiple asymptomatic fibroids [60, 62, 63].

Uterine prolapse is equally likely to develop at any time during pregnancy even during labour [64, 65]. This condition can be classified as prolapse that is present before pregnancy and prolapse that occurs during pregnancy. Even though some degree of prolapse is present before pregnancy, most cases with prolapse resolve with progression of the pregnancy, and spontaneous correction can be expected by the end of the second trimester when the uterus becomes an abdominal organ, pulling the cervix up into the vagina [60, 64, 66]. Even if prolapse resolves transiently during pregnancy, the prolapse that preceded pregnancy may persist or even recur after childbirth because the prolapse is secondary to the pelvic floor dysfunction caused by mechanical damage to the pelvic support system [67]. Prolapse that

develops during pregnancy is usually first noted in the second and third trimester owing to the considerable changes of pelvic organ support [68].

Maternal and Fetal Complications

Complications of POP in pregnancy are common. This condition could predispose pregnant women to discomfort; cervical ulceration, acute urinary retention, preterm labour and fetal and maternal morbidity [68]. Significant complications may develop during pregnancy and childbirth. Urinary retention and urinary tract infection [69], cervical dystocia and obstructive labour, as well as cervical laceration and infection are documented [70]. Major complications, such as spontaneous abortion, fetal demise, preterm labour, fetal death, maternal sepsis or even death have been reported [66, 71, 72].

Antepartum Complications

The oedematous protruding cervix due to venous obstruction and impaired arterial blood flow in pregnancy is susceptible to mechanical trauma, which could lead to its ulceration and infection. In addition, the ulceration and infection of the oedematous cervix secondary to mechanical trauma may be the cause of the high incidence of abortion [73] and preterm labour [60]. Preterm labour is one of the serious complications of uterine cervical prolapse complicating pregnancy due to impaired blood flow induced by cervical trauma and vascular congestion. Urinary tract infection and acute urinary retention have also been reported as complications of uterine prolapse during pregnancy caused by mechanical obstruction and subsequent infection [72].

Intrapartum Complications

Intrapartum complications of POP in pregnancy include inability for cervical dilatation, cervical dystocia and prolonged or obstructed labour as cervical dilatation may begin outside the introitus, and difficulty is added by oedema or fibrosis of cervix [62]. During pregnancy, cervical lacerations followed by infection are quite common, which may lead to cervical fibrosis. This condition also leads to prolonged labour due to cervical dystocia. When the prolapsed uterus causes obstructed labour, rupture of lower uterine segment and intrapartum fetal death or even maternal death may occur.

A case report described an extensive, irreducible uterine prolapse during labour of a patient without any antenatal care, which resulted in the arrest of labour and stillbirth [74].

Postpartum Complications

Prematurity was the main cause of fetal death after preterm delivery, while infection was the most frequent reason for maternal death in association with POP in the early twentieth century [72]. A review of a total of 170 pregnancies with uterine prolapse (from 1925 to 1940) revealed fetal and maternal mortality during this period to have been 22.1 % and 6.3 %, respectively. And this review reported one case of maternal death due to sepsis [61]. Another complication reported is postpartum bleeding due to uterine atony [75].

Recommendations for Management

Management of POP in pregnancy depends on the severity of prolapse, gestational age and the woman's preference. Management options range from conservative management with bed rest to aggressive and ambiguous operative procedures, i.e., cervical incision or cesarean hysterectomy. If managed appropriately, the patients without obstetric complication are considered to have favourable outcomes [64], achieving a spontaneous vaginal delivery rate of 84.8 % [70].

- Good genital hygiene is essential to prevent cervical and urinary tract infection [76]. Local antiseptics should be applied in the event of ulcerations or infected cervix [72]. Topical magnesium solution has been used to prevent cervical dystocia and lacerations for a prolapsed cervix that is oedematous [77]. The mechanism proposed was due to the osmotic diuretic properties of magnesium.
- Bed rest in a moderately Trendelenburg position can be advised in order to reduce oedema and displacement of the uterus. In addition, this position in combination with good genital hygiene can protect the cervix from local desiccation, trauma, oedema and infection or even preterm labour [62].
- Several authors have also recommended placement of pessaries particularly support pessary, i.e., ring with support or dish pessary after reducing the prolapse. Reduction of the prolapsed uterus during pregnancy will protect the cervix from local trauma and prevent the possibility of incarceration [66, 78].
- Alternatively, in cases where conservative methods have failed or when prolonged bed rest is impossible, minimally invasive surgery in a pregnant woman may be considered. Few cases of laparoscopic uterine suspension during pregnancy were reported with successful outcome [62, 79]. There is a new laparoscopic option for the treatment of uterine prolapse in early pregnancy, namely modified Gilliam suspension.
- Regarding route of delivery in cases of prolapse during pregnancy, normal vaginal delivery can be achieved [80]. Although vaginal delivery with forceps may be an option if required and if the cervix is fully dilated, continued stretching of the lower segment to the point of uterine rupture due to cervical dystocia has been reported [72]. In this situation, cesarean delivery becomes the inevitable choice for women with a thick, oedematous, trapped, and irreducible cervix. Duhrssen's cervical incision and forceps application for vaginal delivery in a situation that emergency cesarean section is not available has been reported [60, 75].
- Cesarean hysterectomy with subsequent suspension of the vaginal cuff might be a therapeutic option for women who have completed their families [81] particularly in developing countries where access to healthcare is limited [68].
- Prophylactic bilateral uterine artery ligation can be considered to prevent lower uterine segment atony and postpartum hemorrhage [82].

Puerperium and Pelvic Organ Prolapse

During childbirth, the pelvic floor is extended due to direct pressure of the fetal presenting part and maternal pressure efforts. The decline of the levator ani muscle tone is caused either by denervation or by direct muscle trauma. This results in an open urogenital hiatus, which combined with functional and anatomic alterations in the muscles and nerves of the pelvic floor, contributes to the development of POP in the puerperium.

Incidence and Prevalence

There are numerous reports on the incidence, prevalence and degree of pelvic organ prolapse after childbirth. The reported wide range (15 %–48 %) in the prevalence of POP after childbirth is mainly a result of differences in study populations and varying classification of POP [83–86]. Moreover, there are little data that describe the quantification of prolapse in primiparous women at and beyond 6 weeks from childbirth regarding POP-Q system. Incomplete recovery of pelvic organ support in nulliparous women defined using objective measures ranged from 33 to 79 % for women evaluated at various time-points between 6 weeks and 1 year postpartum [87–91].

- The puerperium is the period of time encompassing the first few weeks following childbirth. The duration of this period is considered between 4 and 6 weeks. A study from China reported 100 and 87.5 % rates of POP after vaginal delivery and elective cesarean delivery 6 weeks postpartum. These women had at least stage I prolapse. However, prolapse symptoms were not evaluated [90]. The incidence of POP from this study was higher than a previous study, which demonstrated that 32 % of women who had spontaneous vaginal delivery and 35 % of cesarean delivery group during active labour developed at least stage II prolapse when compared to their 36-week antepartum [88].
- At 3 months after vaginal delivery, predominantly Hispanic primiparous women were evaluated with POP-Q examination and multichannel urodynamic testing was conducted in Dallas, Texas, USA. The results showed that with respect to the cumulative stage of prolapse, 56 % had stage II, and none had stage III prolapse or greater [92].
- At 6 months after vaginal delivery, magnetic resonance imaging was used to quantify the changes that occur in the levator ani muscles. Levator ani signal intensities and thickness, in areas of the urogenital and the levator hiatus were assessed prospectively. The authors reported that recovery of connective tissue and complete pelvic floor muscles contractility takes up to 6 months after vaginal delivery [93]. According to a prospective study conducted in Albuquerque, NM, USA, nulliparous women were recruited and evaluated at 6 months postpartum. At the 6-months postpartum visit, the vaginal birth group was more likely to have a higher stage of prolapse than the cesarean delivery group; the POPQ

differences were limited to the anterior vaginal wall [94]. The prevalence is consistent with data from an observational study in the primigravid women evaluated 6 months postpartum in Barcelona, Spain. In terms of POP-Q system stage, the authors found that 19.4 % of women were assessed as POPQ stage II [95]. These findings are slightly lower than the 31.2 % of stage II prolapse reported by the Pelvic Floor Disorders Network at 6 months postpartum [89].

- At 1 year after delivery. A prospective, observational study was conducted in Wenzhou, Zhejiang, China. Pelvic organ support was assessed at 36–38 weeks of gestation, before the onset of labour, as well as at 6 weeks, 6 months and 1 year postpartum using the POP-Q system. Stage II prolapse was present in 35 and 37 % of women in unlaboured cesarean delivery (UCD) and trial of labour (TOL) at 36–38 weeks of gestation. After delivery, the likelihood of stage II prolapse declined during the first year postpartum in the whole cohort. The TOL group was much less likely to recover from stage II prolapse compared with the UCD. The continued changes in the pelvic floor were shown from 36 to 38 weeks of gestation to 1 year postpartum, therefore, the process by which the reproductive tract returns anatomically to a normal non-pregnant state after delivery might be more than 6 weeks [96].
- 5 years after childbirth. A longitudinal observational cohort study was conducted in the UK to assess the pelvic organ support stage and pelvic floor symptoms in the second trimester, at 14 weeks after delivery, 1 year and 5 years. The results showed that in women who had a vaginal delivery, the change in average POP-Q stage score was significantly increased from baseline score at 14 weeks, 1 year and at 5 years. In the caesarean delivery group the change in average POP-Q stage score from baseline was only significantly increased at 14 weeks postpartum. Prolapse symptoms were not significantly altered from baseline at 14 weeks, 1 and 5 years in both groups. The authors suggest that although pelvic organ support stage and some symptoms worsen after one vaginal delivery, they do not affect condition-specific QOL [91].
- 12 years after childbirth. All of the women who delivered in three maternity units: in Aberdeen (UK), Birmingham (UK) and Dunedin (New Zealand) were surveyed. The main research question was whether delivery mode history was associated with either prolapse symptoms or prolapse signs at 12 years after the index birth. A questionnaire survey of the 7725 women was conducted around 12 years after their index delivery. Women were also invited to a clinical examination to assess any degree of pelvic organ prolapse using the POP-Q system. Compared with women whose births were all spontaneous vaginal deliveries, women who had all births by caesarean section were the least likely to have prolapse (OR 0.11, 95 % CI 0.03–0.38), and there was a reduced risk after forceps or a mixture of spontaneous vaginal delivery and caesarean section. The authors concluded that prolapse symptoms and objective prolapse may not be in concordance [97].
- 20 years after childbirth. A national survey of pelvic floor dysfunction, the SWEPOP (SWEdish Pregnancy, Obesity and Pelvic floor) study was conducted in 2008 to assess pelvic floor function in women 20 years after one single pregnancy terminating either in a vaginal or a surgical delivery. Symptomatic pelvic

organ prolapse (sPOP) was diagnosed according to a validated five-item questionnaire. The overall prevalence of sPOP was 12.8 %. The prevalence of sPOP was doubled after vaginal delivery compared with caesarean section, two decades after a single birth. The odds of sPOP 20 years after birth increased by 255 % after vaginal delivery compared with caesarean section [83].

Associated Factors

Mode of Delivery

Several studies have linked vaginal childbirth to pelvic organ prolapse [97, 98]. An observational study was undertaken to evaluate the influence of mode of delivery on pelvic organ support of primigravid women after childbirth. Pelvic organ support was evaluated at 6 months postpartum using the POP-Q system. Specifically, spontaneous vaginal delivery was found to more than treble the risk (OR 3.19; 95 % CI 1.07–9.49), while with instrumental vaginal delivery it increased more than fivefold (OR 5.52; 95 % CI 1.79–17.30). Stage II prolapse was found in only 7.7 % women who had undergone cesarean sections [95]. This finding is similar to other authors [94, 95, 97, 99], who observed a low prevalence of POP after cesarean section.

A cross-sectional study conducted in Turkey confirms such association as well. Vaginal delivery was associated with an odds ratio of 2.92 (95 % confidence interval 1.19–7.17) for prolapse when compared with nulliparity [100]. Moreover, each vaginal delivery increased the risk of POP (odds ratio 1.23; 95 % confidence interval 1.12–1.35) after controlling for all confounding factors [100]. The odds for symptomatic pelvic organ prolapse increased with number of childbirths and were 3.3-fold higher among mothers of 4 than among mothers of 1 [85].

Operative Vaginal Delivery

Operative vaginal delivery or the instrumental vaginal delivery refers to the use of traction devices to assist uterine contractions and maternal expulsive efforts during the second stage of labour to achieve delivery of the fetus. Forceps and vacuums are the most commonly used instruments for this purpose. Forceps delivery was found to increase risk of levator ani muscle avulsion during the first vaginal delivery (OR 6.6, 95 % CI 2.5–17.2) [55]. Forceps delivery increased the odds of POP (OR 1.95, 95 % CI 1.03–3.70) in a cohort study [101]. The result suggests that one additional woman would have development of POP for every eight women who experienced at least one forceps birth (compared with delivering all her children by spontaneous vaginal birth).

Prolonged Second Stage of Labour

The second stage of labour is characterized by progressive descent of the fetal head through a completely dilated cervix. This is achieved by the expulsive forces generated by uterine contractions and maternal effort. During these contractions, intrauterine pressure is high. Maternal pushing can additionally increase intrauterine pressure [102]. Ischemic necrosis of the pelvic tissues (including nerves and

muscles) and stretch injuries, leading to permanent denervation of the tissues, can occur if this pressure continues for an extended duration [103]. As a result, a prolonged second stage may increase soft tissue injury and neuromuscular damage to the pelvic floor.

This finding is further supported by the suggestion that prolonged pushing for more than 1 h during the second stage of labour is associated with denervation injuries to the pelvic floor in primiparous women [20]. A study using four-dimensional transperineal ultrasound found that prolonged active second stage of labour increased the risk of levator ani muscle avulsion (OR 2.2, 95 % CI 1.4–3.3) [54]. A small Japanese study identified duration of the second stage of labour of more than 30 min as a risk factor for POP in primiparous women [104].

Conclusion

The development of pelvic organ prolapse has been associated with pregnancy and childbirth. Hormonal changes during pregnancy and mechanical injury to the pelvic floor support, which are direct muscle trauma, disruption of connective tissue support and denervation, are some of the underlying mechanisms for development of pelvic organ prolapse. Over the past two decades, imaging techniques that include ultrasonography and magnetic resonance imaging have revealed mechanisms of injury to the pelvic floor with the time of greatest risk of damage during the first vaginal delivery.

Pelvic organ prolapse in pregnancy is a rare condition. Early recognition is essential in order to avoid possible maternal and fetal risks. In puerperium, continuous changes occur in the pelvic floor from childbirth to 1 year postpartum. Associated factors with pelvic organ prolapse after childbirth are vaginal delivery, forceps delivery and prolonged second stage of labour.

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Obstetric Fistula

15

George Iancu

Abstract

Obstetric fistula is common in developing countries as a result of unattended prolonged obstructed labour. Evaluation of obstetric fistula is based on history and clinical examination. Continuous urinary or stool leakage started soon after a long labour that ended usually with stillbirth in a low resource area is highly suggestive of obstetric fistula. To date, there is no consensus regarding the classification of obstetric fistula. Multiple classification systems have been proposed, but all the present classifications of obstetric fistula are of limited clinical use because of the lack of impact on treatment outcome. Surgical treatment with closure of fistulous tract and reconstruction of local anatomy is the mainstay of therapy. Persistent urinary incontinence after successful surgical repair is the most concerning problem; additional continence surgery is usually needed. In experienced hands, the success rate of surgery varies around 80–90 % after the first surgical repair. Postoperative care should be focused on appropriate bladder drainage and early identification of complications.

Keywords

Obstetric fistula • Obstructed labour • Urinary incontinence • Low-resource area • Incontinence surgery • Fistula recurrence • Continuous bladder drainage

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Background and Definition

The genital fistula is an abnormal communication between the genital tract and urinary or digestive system. Fistula is classified as genitourinary or genito-anorectal fistula depending on the type of communication. In developed countries, most fistulae are the consequence of surgery, while in developing countries, it is the obstetric cause that leads. The maternal mortality and morbidity due to inappropriate access to medical resources is still unacceptably high in developing countries [1, 2]. Obstetric fistula is one of the most difficult to manage complications of a dystocic delivery with a great impact on women's quality of life.

Epidemiology and Risk Factors

The incidence of obstetric fistula cited by World Health Organization is about 0.3 % of deliveries. The true prevalence of the disease is unknown. There are epidemiological studies that estimate the incidence of obstetric fistula to be around 0.35 % of deliveries [3]. The prevalence is reported to be high especially in Africa, in countries such as Sudan, Ethiopia, Ghana, Nigeria and in South Asia, mainly Bangladesh [4]. The prevalence of the condition is likely to be even higher in the rural areas of the developing world where access to healthcare facilities is usually difficult and available data are limited.

Risk factors associated with obstetric fistula are primiparity, young age, delivery of a large fetus, malpresentation, short statured patient, lack education [5, 6].

Pathogenesis

The aetiology of obstetric fistula is mainly ischaemic due to prolonged obstructed labour. The main cause is probably cephalopelvic disproportion, fistulae being often located at the bladder neck or vault. The mother's soft tissues are compressed against the bony pelvis during labour that lasts usually for several days. The continuous pressure diminishes the blood supply to the soft tissues and generates extensive damage with tissue necrosis and fistula, scarring and alteration of normal pelvic anatomy. Often, the injury involves not only the bladder and the vagina, but the urethra, uterus or rectum as well; urogenital, gastrointestinal, neurologic and musculoskeletal systems are usually involved. Other causes of obstetric fistula encountered less frequently are iatrogenic trauma during caesarean section, difficult instrumental deliveries or other obstetric maneuvers [7].

Clinical Diagnosis

Symptoms and Signs

The obstetric fistula is usually a consequence of prolonged abnormal labour; one study estimated the average length of labour of almost 4 days (mean 3.9 days, range 1–6), with 92.7 % ending as stillbirths [5]. Usually, it takes about 3–10 days for the

communication to develop after the necrotic ischaemic slough is drained out through the vagina (Fig. 15.1). The patient becomes continuously incontinent after fistula formation. Some patients are rendered incontinent for stool, if a rectovaginal fistula has concomitantly developed. Obstructed labour trauma that results in fistula formation is different from the surgical injury; it involves often other organ systems as well and the fistula is usually wider due to extensive ischaemic injury. The average size of obstetric fistula treated in a fistula hospital in Addis Ababa was 2.3 cm long (range 0.1–8 cm) and 2.5 cm wide (range 0.1–10 cm) [8].

Vesicovaginal fistulae are the most common type of fistulae. The site of vesicovaginal fistula varies depending on the level of impaction of labour; if the mechanical conflict is at the level of the pelvic inlet, usually the fistula develops intra- or juxtacervical (Fig. 15.2). If the impaction occurs lower in the pelvis, usually the urethra is involved (Fig. 15.3), with severe compromise of the continence mechanisms in the long term [9]. Urethral involvement is usually a predictor for poor prognosis regarding the continence outcome [10]. It can occur in up to one third of obstetric fistula patients and about 5 % of the cases can present total urethral loss [8]. Ureteral lesions

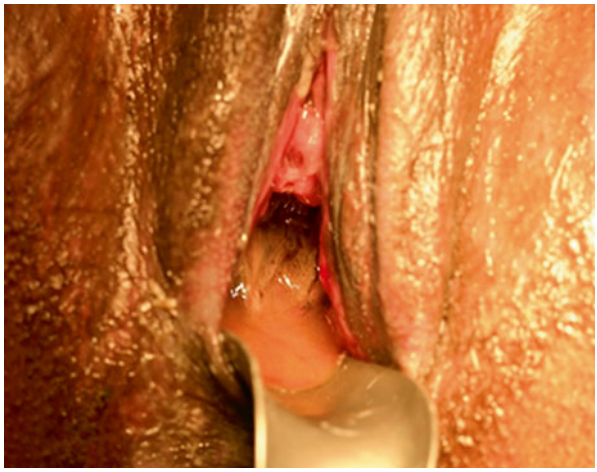

Fig. 15.1 Early vesicovaginal fistula with sloughing tissue draining out through the vagina (Reproduced with permission of Dr. Andrew Browning)

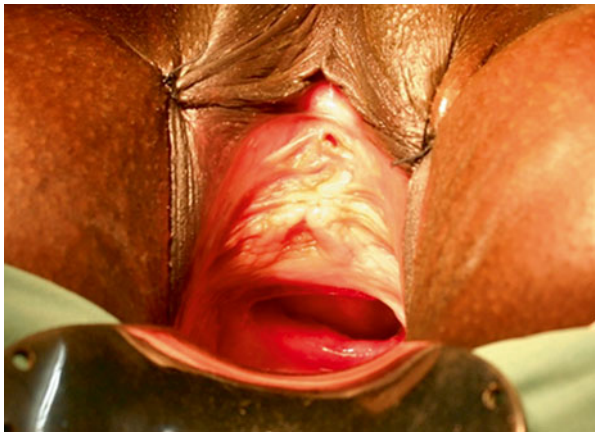

Fig. 15.2 Large vesicovaginal fistula at the level of midvagina, juxtacervical (Reproduced with permission of Dr. Andrew Browning)

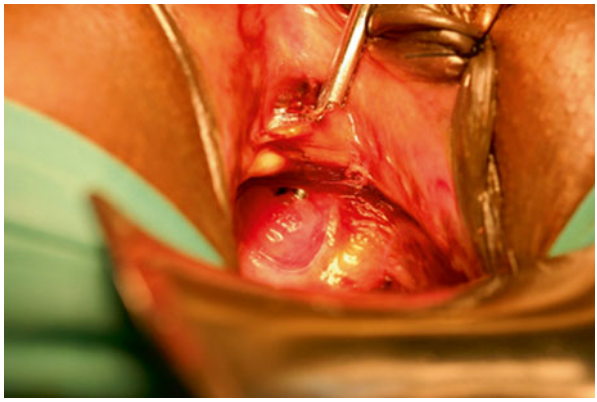

Fig. 15.3 Small circumferential urethrovaginal fistula (Reproduced with permission of Dr. Andrew Browning)

can lead to genito-urinary fistula as well; in a small number of cases, involvement of the distal ureter is followed by uretero-vaginal fistula, with continuous free drainage of urine into the vagina [11]. The fistulous tract can involve the uterus as well, though more rare and usually due to operative injury after caesarean section [12]. Usually, they manifest as vaginal urinary leakage or sometimes as cyclical haematuria.

Obstetric trauma can involve the digestive tract as well. Most commonly, rectovaginal fistulae are the consequence of fetal impaction against the rectum, followed by ischaemic necrosis of the rectovaginal septum. A study revealed a prevalence of recto-vaginal fistula of 1–8 % of obstetric fistula and 1–23 % for combined vesico-vaginal and recto-vaginal fistula; vesico-vaginal fistula accounted for the vast majority of obstetric fistula (over 80 %) [13]. The level of the fistula is important because involvement of anal sphincter can compromise the fecal or flatal continence mechanism.

In developing countries, genitourinary fistulae can be associated with upper renal tract damage, from mild hydronephrosis to non-functioning kidney requiring nephrectomy [11, 14]. The upper urinary tract damage is usually secondary to obstructive uropathy caused by the scarred ureter. Bladder stones form due to recurrent infections, reduced water intake followed by concentrated urine, insertion of foreign bodies in the vagina that act as promoters for calculogenesis [7]. The continuous leakage of urine irritates the perineal skin, causing dermatitis, excoriations, superficial infections or hyperkeratosis.

Regarding the extent of the changes of the reproductive tract, these vary from minimal or mild changes in anatomy (though this is usually the case with surgical fistula) to extensive damage, with vaginal injuries up to necrosis of the whole vagina, torn cervix and involvement of the uterus. Vaginoplasty is required in about one third of the cases [8].

The reproductive outcome is frequently severely affected. Amenorrhoea occurs in half of the patients with obstetric fistula [8]. The aetiology has been debated; amenorrhoea is probably due to the stress of delivery and presence of fistula leading to social isolation, low BMI, pituitary failure after obstetric haemorrhage or shock in long labour, Asherman's syndrome or obstructed outflow and subsequent haematometra [7]. The pregnancy rate in a patient previously treated for fistula is as low as

19 % [15]; small series of pregnant patients delivering post fistula repair showed high recurrence rates of fistulae after vaginal delivery (27 %) and good outcomes with no recurrence after caesarean delivery [16, 17].

Apart from the local anatomical changes related to the pathophysiology of the disease, there are also associated conditions that manifest commonly in patients with obstetric fistula. Obstetric fistula is usually associated with social consequences and an important impact on mental health due to its circumstances of occurrence: young women from low resource country that laboured for days and usually lost the baby, divorced and living in isolation because of the debilitating condition [5, 18]. The vast majority of fistula patients present with mental health problems; one study revealed 97 % of the patients with obstetric fistulae screened positive for mental disorders [19]. Other associated conditions cited in the literature are malnutrition or limb contractures [7].

Assessment

History and clinical examination are the first tools for the assessment of a patient with suspected obstetric fistula. Continuous incontinence that started soon after a long labour, ending usually with stillbirth, in a low resource area is characteristic for obstetric fistula. The clinical examination will enable the diagnosis of a large fistula, its location and the extent of scarring. If the size of the fistula is small and it cannot be identified during naked eye examination, a dye test can be undertaken. Swabs are placed in the vagina, the bladder is catheterized and methylene blue is retrograde injected in the bladder. After a few minutes, the swabs are checked for leakage. After identifying and localizing the genitourinary fistula, the posterior vaginal wall is carefully checked for rectovaginal fistula. The integrity of anal sphincter should be ascertained as well. When a rectovaginal fistula is identified close to the external anal sphincter, reconstruction of the sphincter might be required. Sometimes, when the patient is symptomatic (flatus or stool incontinence) and there is no obvious fistula on examination, a small fistula can be diagnosed using instillation of dye per rectum with the aid of a Foley catheter.

Other more complex diagnostic tests (intravenous urography etc) may be required to establish a diagnosis of fistula. The fistula location and its relationship with the ureteral orifices and urethra are very important to plan management. Large fistulae may allow the identification and catheterization of ureteral orifices vaginally through the fistula tract. Cystoscopy is helpful for small fistulae or for fistulae located high, and are difficult to be accessed vaginally.

Classification of Fistulae

To date, there is no consensus regarding the classification of obstetric fistulae. A valid classification should follow criteria according to the impact on treatment outcome; there are no prospective studies to evaluate the prognosis of different

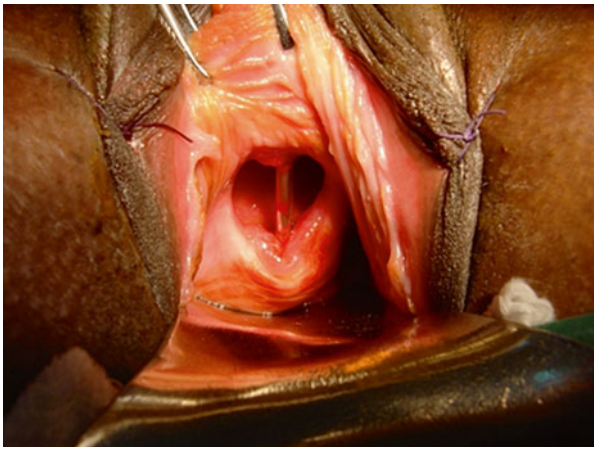

Fig. 15.4 Medium vesicovaginal fistula at the level of midvagina. No urethral involvement is seen (Reproduced with permission of Dr. Andrew Browning)

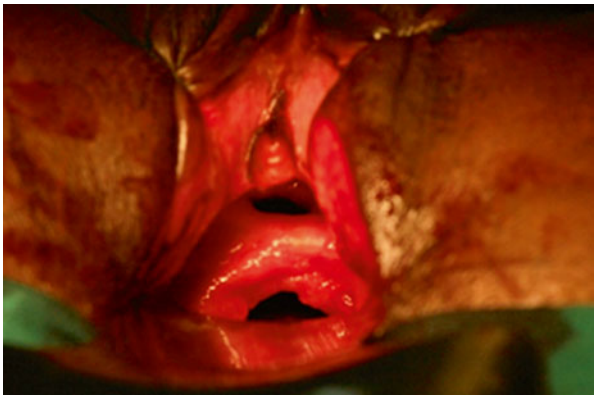

Fig. 15.5 Complicated fistula: double urethrovaginal and vesicovaginal, circumferential, with almost complete urethral loss (Reproduced with permission of Dr. Andrew Browning)

categories of fistulae. Accordingly, all the present classifications of obstetric fistulae are of limited clinical use concerning the impact on treatment outcome [20].

The World Health Organization (WHO) proposed a classification of obstetric fistulae. This classification takes into account the difficulty of the surgical repair. Depending on the complexity, there are two types of fistulae [21]:

- Simple fistulae, with good prognosis, that can be repaired by surgeons trained to treat uncomplicated fistula; they are usually single vesico-vaginal fistulae, under 4 cm diameter, without urethral or ureteral involvement, minimal vaginal scarring and tissue loss, no previous fistula surgery (Fig. 15.4).
- Complicated fistula, requiring usually referral to be repaired only by specialist fistulae surgeons; they are multiple, recto-vaginal or combined vesico- and recto-vaginal, involvement of cervix, urethra, ureters draining into the vagina, vaginal tissue scarring, circumferential loss, recurrent fistulae after failed repairs (Fig. 15.5).

Table 15.1 Classification systems of vesicovaginal fistulae of obstetric origin

Author (year)	Criteria considered	Classification/type
Waaldijk (1995) [22]	Urethral closing mechanism involvement	**I.** Urethral closing mechanism intact **IIAa**. Urethal closing mechanism affected, without (sub)total urethral involvement or circumferential defect **IIAb**. Without (sub)total urethral closing mechanism involvement, with circumferential defect **IIBa**. (Sub)total urethral involvement, without circumferential defect **IIBb**. (Sub)total urethral involvement and circumferential defect **III**. Ureteric involvement; other rare fistulae.
Browning (2004) [9]	Vaginal scarring Bladder volume	1. Simple – reduced vaginal scarring and normal bladder volume 2. Complex – severe vaginal scarring and/or reduced bladder volume; needs vaginoplasty or reconstruction
Goh (2004) [23]	Distance to urethral meatus Size of fistula Vaginal fibrosis/length/capacity	1. distal edge > 3.5 cm from external urethral meatus (EUM) 2. distal edge at 2.5–3.5 cm from EUM 3. distal edge 1.5–2.5 cm from EUM 4. distal edge <1.5 cm from EUM (a) size < 1.5 cm in maximal diameter (b) size 1.5–3 cm in maximal diameter (c) size > 3 cm in maximal diameter (i) minimal or absent vaginal/perifistular fibrosis, vaginal length > 6 cm, normal vaginal capacity (ii) moderate to severe fibrosis and/or reduced vaginal length and/or capacity (iii) special considerations (ureteric involvement, previous repair, circumferential fistula)

Other classification systems have also been proposed (Table 15.1).

The prognostic factors associated with the outcome of the repair of obstetric fistula are the size of the fistula, concurrent lesions (rectovaginal fistula), degree of scarring around the fistula and involvement of the continence mechanism, including urethral damage [10, 24]. Apart from the factors already mentioned, the vaginal route of repair instead of abdominal route and duration of catheterization more than 14 days have been associated with increased risk of failure of the repair [25].

Management

When the management of obstetric fistula is considered, general aspects should be discussed (Table 15.2).

The timing of repair is very important especially for obstetric fistula where the main aetiologic factor is extensive ischaemic necrosis; the time of surgical intervention should be carefully selected, due to the need for good quality tissue for the fistula to heal properly and to avoid recurrences. Usually, it is recommended to wait for at least 3–6 months from the causative injury. The waiting time should allow the necrotic tissue to separate from the normal one that will be used in the repair. Some authors advocate for immediate repair to avoid issues related to potential social rejection experienced by the patient. They claim, there is no significant difference between early repair and the classic repair performed after a few months. The cited study consisted of more than 1700 cases operated by an experienced fistula surgeon with roughly 95 % success rate for first attempt repair; however, the results might not be reproducible in other services with less experience in fistula treatment [26].

Early treatment of fistula could be achieved using continuous bladder drainage as sole therapy, especially for a small fistula that can be closed conservatively thus avoiding surgery. The bladder in this circumstance should be kept on free drainage for 3–4 weeks depending on the fistula size and extension of necrosis. The reported cure rates vary between 7 and 15 % [26, 27]. The success rates depend on the degree of atrophy secondary to menopause, size of the fistula or scarring extent. If closure does not occur after 4 weeks of continuous drainage, surgical treatment is usually needed [28].

A fistula can be repaired vaginally or abdominally. The route of repair depends on the accessibility to the surgical site and experience and skills of the surgeon. The abdominal route is used for vault fistula, juxtacervical or vesicouterine location [7]; ureteral injury, need for augmentation cystoplasty or concomitant abdominal pathology mandate abdominal approach as well. The abdominal approach is associated

Table 15.2 General considerations in obstetric fistula management

Aspects of management of obstetric fistula
Timing of surgery
Abdominal/vaginal approach
Excision/conservation of fistulous tract
Tissue interposition:
Omental flap
Labial fat pad (Martius graft)
Peritoneal flap
Muscle flap
Concomitant procedures: stress urinary incontinence treatment, cystoplasty, vaginoplasty
Adjuvant treatment: postoperative drainage, anticholinergic therapy, antibiotics, HRT

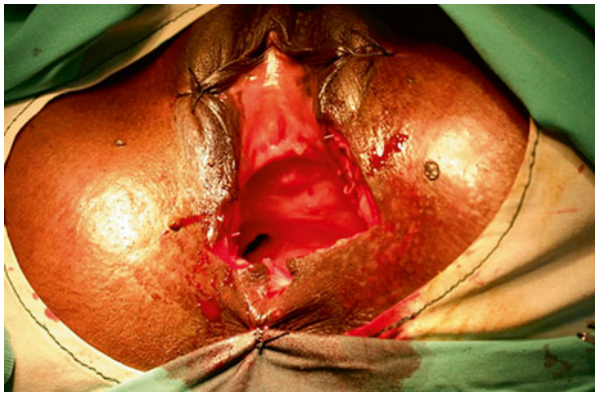

Fig. 15.6 Relaxing perineal incision for juxtacervical ureterovaginal fistula with narrowed introitus (Reproduced with permission of Dr. Andrew Browning)

with morbidity related to laparotomy and requires cystotomy to access fistula site. When the exposure of the superior vagina is difficult, relaxing perineal incisions can be used to facilitate accessibility via the vaginal route (Fig. 15.6) [29]. A study suggested though that for particular indications, the abdominal approach might have better success rate than the vaginal route. Factors like extensive scarring, ureteric, trigonal or supratrigonal involvement, vesicouterine or vesicocervical location were followed by better outcomes of the repair when approached transabdominally; the relative risk of failure for vaginal approach was 1.41 [30].

Regarding the excision of fistula tract, the opinions are divided. Some authors suggest excising it to reduce the recurrence rate, while others advocate a surgical repair without excising the fistula tract which would increase the fistula size and might require electrocautery to control bleeding, thus creating more nonviable tissue. The fistula tissue provides good support for the first layer of sutures. It is important to dissect and mobilize the bladder wall in order to avoid any tension on the repair site with suturing. Care should be taken to avoid ureteral injuries, during surgical repair. The ureters should be catheterized for all fistulae involving the trigone or located supratrigonally [7]. Because of the big size of obstetric fistulae, ureteral catheterization can often be undertaken through the fistula tract.

A flap of tissue or graft is occasionally used to repair a fistula involving urethra or urethrovesical junction to limit the scarring and avoid recurrence. The options are labial or bulbospongiosus fat pad (Martius graft), omental flap, peritoneal flap, gluteal or gracilis flap [29]. The use of tissue interposition techniques optimized the success rate of the procedure in a couple of studies [31, 32]. However, the use of vascular tissue flaps is still controversial. Authors with vast experience in fistula surgery report similar success rates independent of the graft use [33].

Most commonly, the graft used is harvested from the labia majora (Martius graft); a longitudinal incision is made along the labia majora, exposing the underlying fat. The flap of fat is then developed, conserving the posterior vascular pedicle to ensure viability. The flap is then rotated medially, reaching the fistula site through a tunnel created in front of the pubic ramus and behind the bulbospongiosus muscle.

The flap is anchored at the site of fistula to promote healing. Some surgeons drain the labial site to prevent hematoma formation.

Important principles that should be followed for the surgical treatment of fistula include mobilization of the fistulous tract enough to enable closure without tension, water-tight closure of the injury site and careful postoperative management, providing adequate bladder drainage to promote wound healing [34].

When approaching the fistula vaginally, the patient is placed in lithotomy position and the entire fistula tract is exposed. The vaginal opening of the fistula is incised circumferentially and the fistula mobilized so that the fistula margins can be brought together without tension to close the communication between urinary and genital tract. After mobilization of the fistulous tract and the surrounding tissue, the edges of the fistula are closed with absorbable material, either interrupted or continuous depending on the surgeon's preference. The second layer of sutures in the bladder wall could be considered in the absence of extensive scarring. It is very important to ensure that the suture in the bladder wall is water-tight. Therefore, 100–250 ml of diluted methylene blue or indigo carmine dye are instilled in the bladder and the suture line is checked for leakage. If leakage occurs, revision of the suture line is considered until it is water-tight. The instilled fluid should not exceed 250 ml to avoid bladder over-distension and break-down of the fresh repair. The vaginal epithelium is closed with running absorbable sutures.

Urinary Incontinence After Repair

Persistent urine loss after fistula repair can have multiple causes. Recurrence of fistula should be excluded first: the patient should be examined to rule out a recurrence and if transurethral incontinence is diagnosed, other causes of incontinence should be considered. Frequently, urinary incontinence occurs after surgical treatment of fistula, either due to loss of the sphincteric mechanism of vesicourethral junction and urethral injury or reduced bladder capacity and urgency urinary incontinence. Browning assessed the risk of urinary incontinence after fistula closure on 318 patients and divided them into two groups: simple fistulae with minimal scarring and good bladder volume and complex fistulae with severe scarring and/or reduced bladder volume. The risk of incontinence after fistula repair was 50 % in the first group and 100 % in the complex fistulae group [9].

Persistent fistula is ruled out by vaginal examination and dye test. If leakage through the repair site is diagnosed, it is advisable to wait for 6–8 weeks to allow healing and thereafter to consider another surgical intervention if the leakage persists. If the fistula tract is small, continuous bladder drainage can result in spontaneous closure.

When transurethral incontinence is diagnosed and a recurrent fistula is excluded, the type of urinary incontinence should be ascertained. Urodynamic tests are mandatory for the evaluation of these patients; unfortunately, in resource-limited settings, urodynamic facilities are usually not available. A recent study on urodynamic evaluation of residual urinary incontinence after obstetric fistula repair showed a

prevalence of almost 50 % for stress incontinence, over 40 % mixed urinary incontinence and only 3 % detrusor overactivity [35].

The management of patients treated for urogenital fistula and diagnosed with postoperative transurethral urinary incontinence proves to be often difficult especially in cases of preexisting damage of the urethral sphincter, reduced bladder capacity and detrusor overactivity. The use of synthetic mesh sling in patients with a history of obstetric fistula was associated with higher rates of erosion and lower rates of success, advocating the use of autologous sling in this clinical scenario [36]. The use of a urethral plug was proposed as a non-invasive therapy for stress urinary incontinence after fistula repair. The plug should be removed every three hours by the patient to allow bladder emptying [37].

Urinary diversion and bladder augmentation are therapeutic measures of last resort for patients with persistent urinary incontinence [34]. They are sometimes the only options for patients with most of the bladder injured. Patients with bladder augmentation with intact urethra often require intermittent self-catheterization that may not be an option in low resource areas due to lack of catheters and equipment. Urinary diversion can be achieved by ureterosigmoidostomy, Mainz II pouch or ileal conduit [7].

Particular Clinical Forms of Urogenital Fistulae/Complex Fistulae

The criteria to consider a fistula complex are variable. The size of the fistula is important (greater than 4–6 cm), involvement of the continence mechanism (urethra partially or completely absent, bladder capacity reduced), recurrent fistulae, extensive scarring or association of rectovaginal fistula.

Circumferential Fistula

This type of fistula remains one of the most challenging to repair. It is a result of complete transection of the urethra that is separated from the bladder. It is usually due to impaction of the presenting part against the symphysis pubis. In the vast majority of patients, the continence mechanism is impaired and needs to be addressed usually at the time of the repair. The surgical correction requires extensive mobilization of the bladder from the vagina and symphysis pubis and re-anastomosis to the urethra.

Complete/Partial Absent Urethra

Extensive damage and large defects are common features of obstetric fistula. When obstructed labour injury involves the urethra causing it to slough away (Fig. 15.7), surgical reconstruction that restores the anatomy and continence is challenging for

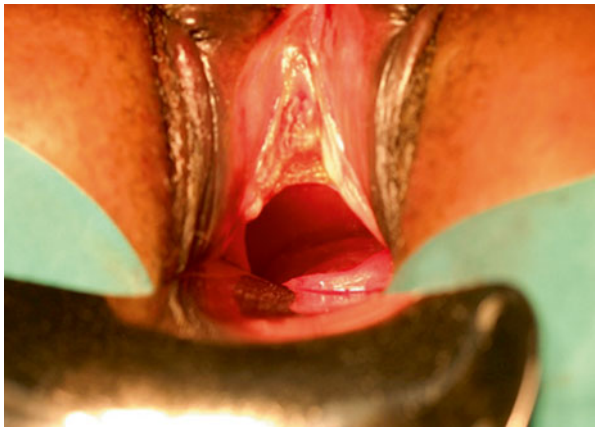

Fig. 15.7 Large circumferential vesicovaginal fistula at the level of midvagina with partial absent urethra (Reproduced with permission of Dr. Andrew Browning)

the surgeon. This clinical form was recognized decades ago and its management was largely debated; a cohort of 50 women with totally destroyed urethra after prolonged obstructed labour was published in 1969. The urethra was reconstructed using skin and connective tissue covering the pubic bone and inferior margin of the symphysis pubis. The reconstructed urethra was reinforced with gracilis muscle and/or labial fat graft [38].

Extensive Vaginal Injury

For large fistulae with extensive scarring and damage of the vaginal wall, vaginoplasty is usually recommended. It can be required in about one third of the obstetric fistula cases [16]. Depending on the extent of vaginal scarring, reconstructive procedures can vary from a Fenton type procedure to complex vaginal reconstruction with rotational flaps use from labia or gluteal skin or other tissue (sigmoid, ileum [39]).

Rectovaginal Fistulae

The coexistence of rectovaginal fistula with urogenital fistula increases the complexity of therapeutic management. The association between the two was estimated in one large cohort to be 17 %; rectovaginal fistulae alone were found in 4 % of patients [40]. The principles of management are essentially the same: mobilization of fistula, excision of excessive scar tissue, closure of the fistula tension free, use of grafts (Martius) rarely necessary, closure of vaginal epithelium. The surgical repair of rectovaginal fistula has a lower success rate than for vesicovaginal fistula, regardless of the association of the two, but the continence

outcome after successful repair of rectovaginal fistula is better than after vesicovaginal fistula [8].

Outcomes and Complications of Obstetric Fistula Surgery

The success rates after fistula surgery vary because of different definitions used by different authors. Success of fistula repair is commonly reported either as closure of abnormal communication or defect or as continence after repair or dryness. Currently, there is a lack of consensus regarding definitions and clinical success related to obstetric fistula [41]. Usually, success is considered the closure of the fistula without considering the continence outcome or patient's quality of life. The reported success rate for fistula of obstetric origin varies among different authors, depending on the surgeon's expertise, severity and complexity of fistula and possibly surgical technique used (Table 15.3). Success rates vary essentially between 80 and 90 % in the majority of studies; success rates over 95 % are usually reported by surgeons with vast experience in fistula treatment, working in centers with great workload. Particularities of fistula such as degree of scarring, urethral involvement, fistula size and location, circumferential fistula or reduced bladder capacity, history of failed repair can be used to stratify the prognosis. Other factors, such as female genital mutilation, parity or antibiotic use are not predictors for fistula outcome [10, 42].

Table 15.3 Success rates after obstetric fistula surgery

Author	No of patients	Success rate
Rafique et al. (2002–2003) [45]	42 patients	85.7 %
Husain et al. (2005) [46]	50 patients	63 % after primary repair 61 % after multiple repairs
Singh et al. (2010) [27]	42 patients	80.1 % after primary repair
Arrowsmith et al. (1994) [47]	98 patients	81 % after primary repair 96 % after multiple repairs
Kliment et al. (1992) [48]	41 patients	85.4 % after primary repair
Kayondo et al. (2011) [49]	77 patients	77.9 % after primary repair
Chigbu et al. (2006) [50]	78 patients	82 % after primary repair
Morhason-Bello et al. (2008) [51]	71 patients	79.2 % after primary repair
Rijken et al. (2007) [44]	407 patients	94.1 % after primary repair
Hilton (2012) [52]	348 pt (2/3 gynecologic origin, 1/3 obstetric origin)	95.7 % overall (98.2 % – primary repair; 88.2 % – previously failed repairs)
Browning et al. (2006) [33]	413 patients	97.6 % after primary repair
Browning et al. (2007) [53]	316 patients	97.5 % after primary repair
Nardos et al. (2012) [54]	189 patients	95.2 % after primary repair
Waaldijk K (2004) [26]	1716 patients	95.2 % after primary repair

Table 15.4 Complications after fistula surgery

Recurrent fistula
Urinary/fecal incontinence
Ureteral injury
Reduced bladder capacity
Urinary tract infection
Voiding dysfunction (urinary retention to anuria)
Bladder stones
Amenorrhea
Vaginal stenosis/atresia
Asherman's syndrome
Leg weakness, contractures
Haemorrhagic complications
Wound infection

Regarding the time frame when success should be assessed, there are different opinions as well. Browning et al. (2008) suggest that persistent incontinence after fistula repair improves usually after 6 months [43]. Rijken et al. (2007) reported that over 50 % of cases of persistent incontinence after repair resolved by 6 months follow-up [44].

Apart from persistent urinary incontinence after surgical repair, which is the most concerning complication and has already been discussed, there are complications such as voiding difficulty and incomplete bladder emptying; they are consequence of neuropathic injury due to obstructed labour trauma and extensive dissection to mobilize a fistula located at the trigone. The rate of erosion associated with synthetic suburethral sling in obstetric fistula patients is higher than in non-fistula patients [36]. Biological slings are often preferred for urinary incontinence treatment in patients with history of fistula [9, 55, 56]. Bladder stones develop when the repair was performed using non-absorbable sutures. Other complications are common to other types of gynecologic surgery as well (Table 15.4).

Postoperative Care

Careful continuous drainage of the bladder for 10–14 days postoperatively with nursing support is the mainstay of the postoperative management for patients with urogenital fistula [54]. A full bladder could result in pressure on the repair site and failure of the surgical treatment. Imaging studies (cystogram, CT scan) may be indicated to be performed prior to the removal of catheter [57]; this can be problematic in low-resource settings. Ureteral catheters should be removed at the end of the procedure if ureteral orifices are far from the repair site or kept in situ for 5–7 days if they are in its proximity. Vaginal packing for 24–48 hours should be placed to ensure compressive haemostasis. Early mobilization and high fluid intake are also advisable.

Obstetric fistula continues to represent an important burden for the women's health internationally. Occurring mainly in developing countries, it urges mobilization of medical resources to improve access to healthcare facilities and provide adequate maternity care.

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Prediction, Risk Assessment, and Prevention of Childbirth Trauma

16

Anushuya Devi Kasi and Stergios K. Doumouchtsis

Abstract

Eighty-five percent of women sustain perineal trauma of varying degrees during childbirth. Overall the risk of anal sphincter injury is 1 % of all vaginal deliveries. There are numerous risk factors associated with the development of perineal trauma. The non-modifiable factors are ethnic origin, nulliparity and maternal age. The potentially modifiable risk factors are mainly obstetric: macrosomia, epidural anaesthesia, prolonged second stage, instrumental delivery and episiotomy.

Even with optimal obstetric management, childbirth injuries are still a common occurrence. Strategies designed to prevent diseases can act at three different levels. Primary prevention strategies aim to prevent by modifying the risk factors prior to the onset of a condition. Secondary prevention strategies aim to identify and treat people with preclinical disease. Tertiary prevention strategies focus on treating and managing people with the disease and attempt to treat or prevent further complications.

Primary prevention strategies include elective caesarean section, antenatal pelvic floor muscle training, warm compresses and antenatal perineal massage. The secondary prevention strategies are related to perineal massage during second stage, maternal position during delivery, whirl pool baths, vacuum and forceps delivery, perineal support, pushing during second stage, episiotomy, perineal

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hyaluronidase injection, EPI-NO®. The tertiary prevention strategies aim to address the mode of delivery in subsequent pregnancies for women with previous childbirth injuries to the pelvic floor.

Preventing perineal trauma would prove to be a significant health benefit factor in childbearing women. It would also reduce the cost and complications that follow it.

Keywords

Childbirth trauma • Perineal trauma • Childbirth injuries • Risk assessment • Prevention strategies • Primary prevention • Secondary prevention • Tertiary prevention • EPI-NO® • Prediction

Introduction

Eighty-five percent of women sustain some degree of perineal trauma during childbirth [1]. The overall risk of obstetric anal sphincter injury is 1 % of all vaginal deliveries [2]. Childbirth trauma is associated with increased maternal morbidity both in the short term and long term. The morbidities could be both physical and psychological. In a study conducted by MacArthur et al. [3], about 47 % of women experienced at least one or more of the following symptoms within 3 months after delivery, which included backache, headache, haemorrhoids, depression, bowel and bladder symptoms and persisted for at least 6 weeks; 23–42 % have postpartum pain and discomfort for 10–12 days postpartum, 7–10 % have long-term pain (3–18 months following delivery) [4–6].

Twenty-three percent of women experience superficial dyspareunia at 3 months, 3–10 % report faecal incontinence [7, 8], and 24 % have urinary problems [4, 5] after perineal trauma. Levator ani avulsions occur in 13–36 % of women mainly during the first vaginal delivery [9]. This is mainly by stretching and shearing of the muscle from its attachment during vaginal delivery.

Urinary, faecal incontinence and uterovaginal prolapse are considered to be sequelae of childbirth. The first vaginal delivery is the time when women sustain most of the damage [10]. Clinical and epidemiological studies strongly indicate that women who have a vaginal delivery are at higher risk of subsequent incontinence than nulliparous women and those who are delivered by caesarean section. Urinary or anal incontinence are related to direct damage of the pelvic floor and disruption of the nerve supply. Some women are more prone to get prolapse than others due to an inherent weakness of collagen within the pelvic floor fascia [11].

A systematic review has estimated the prevalence of any post-partum urinary incontinence after vaginal delivery to be 31 % (95 % CI: 30–33 %), and weekly or daily incontinence 12 % (95 % CI: 11–13 %) and 3 % (95 % CI: 3–4 %), respectively [12].

Sexual dysfunction and postpartum perineal pain may also occur. A large prospective survey of Swedish postpartum women reported that 8 % (167/2154) of

women had not had sexual intercourse within 6 months after childbirth; of those with an anal sphincter injury the proportion was higher at 13.6 % [13].

The above epidemiological data highlight the scale of the burden of vaginal childbirth on the pelvic floor and the associated sequelae, as well as the importance of developing strategies for the prediction and primary and secondary prevention. Risk assessment is the cornerstone of these strategies.

Risk Assessment

Risk Factors for Perineal Injury

For the past 20 years attempts have been made to identify, modify and prevent the risk factors for perineal trauma. Numerous factors are associated with the development of perineal trauma.

The non-modifiable factors are ethnic origin, nulliparity, maternal age. The potentially modifiable risk factors are mainly obstetric: macrosomia, epidural anaesthesia, prolonged second stage, instrumental delivery, episiotomy.

Asian Ethnicity

A meta-analysis by Pergialiotis et al. [14] showed that Asian ethnicity was associated with an increased risk of severe perineal laceration (OR 2.74 [95 % CI, 1.31–5.72]; $P<0.01$). This review included 111,697 women from four studies [15–18]. They were admittedly heterogenous. Groutz et al. [15] included a total of 566 Asian women in their study. This study showed an incidence of severe perineal trauma as high as 2.5 % in Asian women, tenfold higher than the incidence in the general obstetric population. High prevalence of severe perineal tears among Asian women delivering in Western countries was previously reported by others, and is thought to be associated with a relatively short perineum that is less likely to stretch well, or relatively higher birthweights secondary to dietary changes [16, 19–22]. Another possible explanation was the lack of effective communication between these women and the local midwifes during the course of labour and delivery [16]. Data regarding obstetric parameters and prevalence of perineal tears among Asian women in their own countries are limited. Nakai et al. [23] studied the incidence and risk factors for severe perineal tears among Japanese women. Of 7946 singleton vaginal vertex deliveries that occurred between 1997 and 2005, 135 women (1.7 %) had severe perineal tears. The severe perineal lacerations in this study included third- or fourth-degree tears.

Maternal Age

Pelvic floor disorders are common among older women. This is mainly because of the adverse impact of aging on tissue integrity and elasticity. Hornemann et al. [24], in a retrospective study including 2967 women, found maternal age to be the second most important risk factor for severe perineal lacerations. However, owing to the methodology of the study, clear cut-off threshold values for maternal age could not

be defined. Rortveit and Hunskaar [25] however, showed that maternal age older than 25 years at the first delivery increased the risk for urinary incontinence, and specifically stress urinary incontinence, compared to younger nulliparas. Groutz et al. [26] showed that maternal age of more than 37 years at the time of delivery was a risk factor for postpartum urinary incontinence.

Obesity

Obesity is recognized as a risk factor for pelvic floor disorders following vaginal delivery [27, 28]. The threshold values and morbidity associated with different modes of delivery are not, however, defined. Obesity was found to be a risk factor for pelvic organ prolapse in women with one child. Dolan et al. [27] found that there is a fourfold increased likelihood of severe SUI in obese women.

Parity

Nulliparous women are more likely to sustain pelvic floor trauma during labour. Studies have shown that vaginal delivery increases the risk of both anal sphincter complex injury and urinary incontinence in nulliparous women by two- to five-fold [29]. A study by Sultan et al. confirmed that anal sphincter defects were common in nulliparous women who had vaginal deliveries [31]. A possible explanation is reduced tissue elasticity of the pelvic floor in nulliparous women. Petersen and Uldbjerg demonstrated that the content of hydroxyproline and the strength of the collagen in the uterine cervix of multiparas is reduced [30]. The incidence of clinically detected obstetric sphincter tears is usually less than 3 % in nulliparous women. According to Handa et al., nulliparous women had more than six times the risk of anal sphincter laceration compared to multiparous women [31]. A meta-analysis by Oberwalder et al. [32] included 5 studies with 717 deliveries and revealed a 26.9 % incidence of anal sphincter defects in nulliparous women and an 8.5 % incidence of new sphincter defects in multiparous women. A prospective observational study by Smith et al. [33] including 2574 women with a planned singleton vaginal delivery in one obstetric unit, three freestanding midwifery-led units and home settings in South East England concluded that the proportion of multiparous women who delivered with an intact perineum were three times higher than that of the nulliparous women. The multivariate analysis of this study showed that multiparity was associated with reduced odds of obstetric anal sphincter injuries. A retrospective cohort study by Lowder et al. [34], which assessed 20,674 deliveries also concluded that anal sphincter laceration occurred in 16 % of women with first vaginal deliveries and 18 % with vaginal birth after caesarean section (VBAC). In this study multivariable logistic regression modeling for primary anal sphincter laceration showed that first vaginal delivery had OR of 5.1 and 95 % CI 4.4, 5.9, and VBAC had OR of 5.1, 95 % CI 4.2, 6.2 when compared with the reference group with second vaginal delivery. A study from Norway also showed that a nulliparous woman is a dominant risk factor for obstetric anal sphincter injury [35].

Mant et al. [36] showed strong association between parity and vaginal delivery with pelvic organ prolapse (POP). Compared with nulliparous women, women with

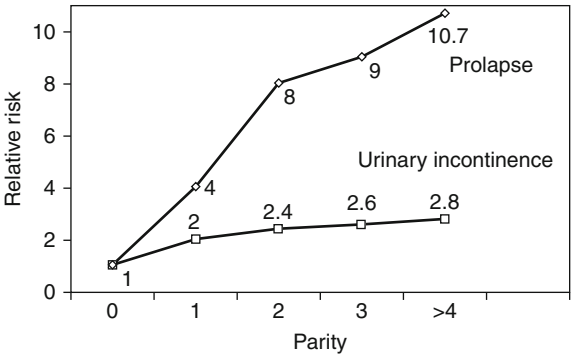

Fig. 16.1 Graph of the effect of vaginal parity on the development of urinary incontinence and pelvic organ prolapse (Reprinted from Lavy et al. [129]; with kind permission from Springer Science and Business Media)

one child were four times more likely and women with two children were 8.4 times more likely to experience POP.

Leijonhufvud et al. [37] also showed an increased risk of both stress incontinence and genital prolapse with increasing parity in 63,229 women delivered by vaginal delivery between 1973 and 1983.

Rortveit et al. [25] found a strong association between parity and stress UI, with relative risks of 1.9 (95 % CI, 1.6–2.2) for primiparous women and 2.3 (95 % CI – 2–2.6) for women with two deliveries.

See Fig. 16.1 for a graph of the effect of vaginal parity on the development of urinary incontinence and pelvic organ prolapse.

Instrumental Delivery

All types of assisted vaginal delivery are considered as an independent risk factor for severe perineal trauma [38]. Among the potentially modifiable variables, forceps leads the list.

Use of forceps and vacuum has shown to increase the risk of fecal incontinence by two- to seven-fold [7, 39]. Sultan et al. [7], MacArthur et al. [40], and Combs et al. [41] found an increased risk of perineal trauma with forceps but not vacuum. The force of forceps against the pelvic floor muscles and surrounding tissues has been estimated at 75 psi [42]. Forceps delivery markedly increases the risk of third and fourth degree tears and also causes pelvic neuropathy.

In the study by Sultan et al. [7], 81 % of forceps deliveries caused endosonographic anal sphincter defects, compared with 24 % of vacuum extractions. Defaecatory symptoms were also much more frequent in the forceps delivery group. Vacuum extraction is generally thought to be less traumatic than forceps delivery. Handa et al. [31] found an increased risk of lacerations with both forceps and vacuum. Others [41, 43], including a meta-analysis by Eason et al. [44], also have implicated both forms of operative delivery, with forceps carrying a greater risk than vacuum. A prospective, randomized trial by Johanson et al. [45] showed significantly less perineal trauma in the vacuum-extraction group.

A Cochrane review [46], which included 10 trials, concluded that the use of the vacuum extractor for assisted vaginal delivery, compared with forceps, was associated with significantly less maternal trauma.

Forceps has a higher rate of maternal morbidity than vacuum but this also depends on the experience of the operator. This is probably because the shanks of the forceps stretch the perineum and can cause injury to the perineum especially to the anal sphincter if pulled excessively in the posterior direction to encourage flexion of the fetal head.

Prolonged Second Stage

Prolonged second stage of labour carries a twofold increased risk of incontinence. Prolonged second stage of labour is associated with neuromuscular injury [47, 48]. A retrospective cohort study of 15,759 nulliparous women by Cheng et al. [49] concluded that maternal complications like postpartum haemorrhage, third- or fourth-degree lacerations were increased when second stage of labour was increased more than an hour. Rates of caesarean delivery increased when the second stage of labour was prolonged >3 h. When the second stage lasted >4 h, the caesarean delivery rate increased to 32.8 %. Similarly, rates of vaginal delivery declined rapidly from >80.0 % in the 1- to 2-h interval to 56.6 % when the second stage ended during the 2- to 3-h interval. It further declined to 18.8 % when the second stage was prolonged >4 h. In contrast, the rate of operative vaginal delivery increased with time, approaching 50 % after a second stage of >4 h. The frequency of postpartum hemorrhage increased from 7.1 % in cases of 0- to 1-h interval of second stage to 30.9 % when second stage was prolonged >4 h. There was an increase in the rates of third and fourth degree tears from 11.6 % in the 0- to 1-h interval to 34.2 % when the second stage lasted more than 4 h.

Pelvic floor damage may occur even in the first stage of labour. Therefore a caesarean section performed after the start of labour is not necessarily protective.

In summary, the longer the duration of the second stage of labour, the higher the risks of adverse maternal and perinatal outcomes in both nulliparous and multiparous women. The increased risks appear independent of the mode of delivery.

Epidural Analgesia

Women who had epidural anaesthesia for pain relief in labour were almost twice as likely to have third- or fourth-degree perineal lacerations than those who did not have epidurals [50]. The reason for that association was increased operative deliveries and episiotomies with epidurals. The effect of epidural analgesia during labour on the occurrence of perineal tear is complex, as there are many confounding factors. There is evidence that nulliparous women use epidural analgesia at a statistically significant higher rate than parous women; women who use epidural analgesia demonstrate a prolonged second stage of labour; an increased use of augmentation of labour; and more use of epidural analgesia with occipito-posterior position may increase the risk of anal sphincter damage [51, 52]. On the other hand, women undergoing epidural analgesia have a higher rate of episiotomy and epidural analgesia results in women being more relaxed and under control with a slower and more controlled second stage that might reduce the risk [51]. In another study by Eskandar et al. [53], epidural analgesia reduced the rate of severe perineal tear by 12 %. Combs et al. [41] in a study of 2832 consecutive operative vaginal deliveries, also

reported no effect of epidural on third- or fourth-degree lacerations. The combination of operative vaginal delivery, epidural and episiotomy increases the risk of childbirth injuries.

Malpresentation and Malposition

Persistent occipitoposterior position leads to a larger presenting diameter and often to a difficult delivery with increased risks of postpartum incontinence [46]. Face and brow presentations also increase the risk of incontinence because of the larger presenting diameter. Breech deliveries do not appear to increase the risk.

Macrosomia

Birthweight more than 4 kg is associated with potential fetal problems like birth trauma, shoulder dystocia and lower apgar scores. On the maternal side, potential complications include higher rates of perineal trauma especially third- and fourth-degree tears [47], pudendal nerve injury, and significantly weaker anal squeeze pressures [54]. Vaginal delivery of at least 4000 g raises the risk of long term stress incontinence [55]. A study by De Leeuw et al. [43] showed a significant correlation between birthweight and the occurrence of third-degree tears. Shiono et al. [56] reported a significant odds ratio of 1.10 per 100-g increase in birthweight. Other studies have also confirmed an increased risk with fetal birthweight exceeding 4 kg [57, 58].

Macrosomic babies disrupt the fascial supports of the pelvic floor. Injury to the pelvic and pudendal nerves may also occur. Shoulder dystocia is also associated with perineal and anal sphincter trauma. It is not clear whether shoulder dystocia per se causes perineal trauma. Manoeuvres used in shoulder dystocia are associated with an increased risk of anal sphincter damage. This was confirmed by Moller Bek and Laurbeg [59]. However, a decision analysis of elective caesarean section for macrosomia determined that 539 elective caesarean sections need to be performed to prevent one case of anal incontinence.

De Leeuw et al. [43] found that birthweight is significantly associated with third degree perineal tears, with an odds ratio per increase of birthweight by 500 g of 1.47 (95 % CI: 1.43–1.51) (Fig. 16.2).

Episiotomy

Although episiotomy is the commonest operation performed in obstetrics, there is little evidence to demonstrate any benefit with its routine use.

In a number of trials [5, 60–66], restrictive use of episiotomy appeared to reduce perineal trauma that required suturing [60]. The weighted risk difference in sutured perineal trauma between the restrictive and liberal episiotomy was –0.23 (95 % CI –0.35,–0.11), i.e., a 23 % absolute decrease in the risk of sutured perineal trauma with restrictive episiotomy. Avoiding routine episiotomy in 4.4 women would prevent one case of perineal trauma that requires suturing.

In the episiotomy trials, the weighted risk difference in anal sphincter tears between restrictive and liberal episiotomy group was –0.004 (95 % CI –0.02, 0.01). Restricting the analysis to mediolateral episiotomy trials [5, 60, 63, 64] did not

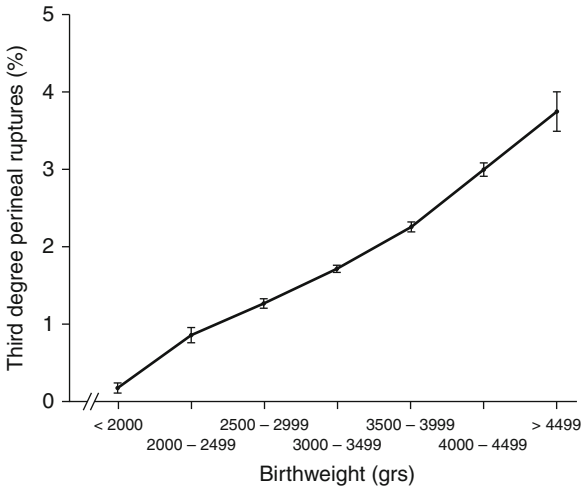

Fig. 16.2 De Leeuw et al. found that birthweight was significantly associated with third degree perineal tears, with an odds ratio per increase of birthweight by 500 g of 1.47 (95 % CI: 1.43–1.51) (Reprinted from De Leeuw et al. [43]; with permission from John Wiley and Sons)

change the risk difference. The trial by Klein et al. [65] on median episiotomy reported no difference in anal sphincter tears between groups randomized to liberal or restrictive episiotomies. Coats et al. trial compared median to mediolateral episiotomies [67]. Anal sphincter trauma occurred in 24 % of cases with median and 9 % with mediolateral episiotomies. This study has been criticized because the group allocation was not masked.

In Klein's RCT of median episiotomy [65], 7.6 % (53 of 698) of women had anal sphincter tears, compared with 1.1 % (44 of 3952) in the mediolateral episiotomy trials [5, 60, 63, 64].

Legino et al. [68] showed that there was a sharp rise in third-degree tears when episiotomy technique switched from mediolateral to median technique. Although mediolateral episiotomy does not protect the anal sphincter, median episiotomy clearly increases the risk of anal sphincter injuries.

Liberal episiotomy did not reduce pain or dyspaerunia. A large Argentine [60] trial found more pain with the use of liberal episiotomies. Routine episiotomy did not prevent urinary incontinence at 3 months postpartum according to Klein et al. [65] and at 3 months and 3 years according to Sleep et al. [5].

Prevention of Childbirth Injuries

Even with optimal obstetric management, childbirth injuries are still a common occurrence. Nevertheless, several strategies have been put forward to minimise the risks. Strategies designed to prevent diseases can act at three different levels. Primary prevention strategies aim to prevent by modifying the risk factors prior to the onset of a condition. Secondary prevention strategies aim to identify and treat people with preclinical disease. Tertiary prevention strategies aim to treat or prevent further complications by managing patients with the disease.

Interventions to Prevent Perineal Trauma

Primary Prevention Strategies

Elective Caesarean Section

Elective caesarean delivery is the only true primary prevention strategy. Caesarean delivery after the onset of labour is not protective of injuries to the pelvic floor. Some functional loss to the pelvic floor might be avoided by elective caesarean section but not by emergency caesarean section [57, 69].

Elective cesarean section can certainly prevent mechanical trauma to the anal sphincter but not neurological trauma [70], but this has not been demonstrated for the urethral sphincter. The surgical and anaesthetic risks of a caesarean section and the risks associated with repeat caesarean sections for future pregnancies need to be considered in making an informed decision.

Studies have shown an increased risk of long-term urinary incontinence [71], and surgery for POP and/or SUI [37], following vaginal delivery; most epidemiological studies suggest that caesarean section provides only partial protection. This is for both emergency and elective caesarean section. Eight or nine caesarean sections need to be performed to avoid one case of urinary incontinence [71]. A 12 year large cohort study by Mcarthur et al has concluded that caesarean section is not protective of urinary incontinence unless all the women had only caesarean deliveries. Even after exclusive caesarean deliveries the prevalence of urinary incontinence was as high as 40 % [109].

With regards to POP, patients delivered exclusively by caesarean section have a significantly reduced risk of objectively measured POP 12 years after delivery [73] and a reduced risk of symptoms by 20 years [71]. Yet the lifetime risk of undergoing a single operation for POP and UI is estimated to be 11.1 % [74], which suggests that the development of PFD may be attributable to factors beyond vaginal versus caesarean deliveries. An intervention such as primary elective caesarean delivery for all births could potentially cause harm to a proportion of women who would otherwise have been delivered vaginally and not have experienced pelvic floor disorders.

Nevertheless, there may be a role for elective caesarean delivery in women with non-modifiable risk factors as part of an alternative primary prevention strategy.

Antenatal Pelvic Floor Muscle Training

Pregnancy and birth trauma are risk factors for urinary incontinence. The incidence of stress urinary incontinence during pregnancy has been reported in the range of 19.9–70 % in nulliparous women [75–77] while in the postpartum period after one vaginal delivery it ranges from 0.7 to 35 % [78, 79].

Antenatal pelvic floor muscle training has been shown to reduce the incidence of postnatal SUI in the short term [80–82]. However, a 6-year [83] and 8-year [84] follow-up showed no significant improvement. By 8 years there was no difference in the quality of life between the study and control group. This was mainly because of the poor compliance with PFMT. Only 38 % of the women performed pelvic floor muscle training (PFMT) twice per week or more. There was no difference in

outcome between those who performed regularly compared with those who performed less frequently. However the National Institute of Clinical Excellence (NICE) [85] recommends PFMT for all women in a first pregnancy for prevention of SUI based on data from two randomized controlled trials (RCTs) [81, 82]. An RCT on antenatal pelvic floor exercises to prevent and treat urinary incontinence by Ko et al. [86] on 300 women, concluded that there was a significantly lower incidence of self reported urinary incontinence in the PFMT group than the control group. It also showed that women who delivered vaginally experienced more postpartum leakage than those who delivered by caesarean section.

Warm Compresses and Antenatal Perineal Massage

Antenatal perineal massage has been proposed as a method of decreasing the incidence of perineal trauma. Daily antenatal perineal stretching massage by the woman or her partner has been found to prevent perineal trauma and perineal pain [87]. Antenatal perineal stretching massage has a protective effect in nulliparous women and is associated with an overall reduction in the incidence of trauma requiring suturing as well as lower incidence of episiotomy. These findings were significant for women without previous vaginal birth only. No differences were seen in the incidence of first- or second-degree perineal tears. No significant differences were observed in the incidence of instrumental deliveries, sexual satisfaction, or incontinence of urine, faeces or flatus for any women who practiced perineal massage compared with those who did not.

Five studies [88–92] evaluated the effectiveness of perineal massage. Two of those studies failed to analyse by intention to treat [88] and two of the studies failed to randomize [89]. Two large and similar trials and a pilot study [90] evaluated massage with sweet almond oil for 5–10 min daily from 34 weeks until delivery. The Labrecque and colleagues' trial [91] reported that in women with first vaginal births, 24.3 % of those that had been randomized to massage had intact perineums compared with 15.1 % of controls who did not massage. A simultaneous trial with subsequent or second births showed no significant benefit of massage [91]. A similar trial by Shipman et al. [92] in nulliparous women found a 6.2 % increase of intact perineums in the massage group compared to the controls. The weighted risk difference from all those trials was – 0.08, i.e., one case of perineal trauma that required suturing would be avoided for every 13 women who did antenatal perineal massage [44].

Secondary Prevention Strategies

Secondary prevention strategies should aim to address obstetric practices that can be modified during labour and delivery.

Perineal Massage During Second Stage

Some accoucheurs ease the perineum back over the crowning head, whereas others believe that manual stretching increase the local tension and causes laceration. There are no randomised controlled trials to provide evidence for or against perineal stretching massage in the second stage. One study from Turkey on 396 nulliparous women who delivered vaginally, concluded that perineal massage during labour

decreases the rate of episiotomy and lacerations [93]. Another randomized controlled trial by Stamp et al. [94] on 1340 women concluded that the practice of perineal massage in labour does not increase the likelihood of an intact perineum or reduce the risk of pain, dyspaerunia, or urinary and fecal problems. This trial was underpowered and did not assess the outcome of third degree tears. A Cochrane review showed there was a significant effect of warm compresses and perineal massage during the second stage of labour on reduction of perineal trauma and suturing [95] but suggested that further research is required to see if it prevents OASIS. The procedure has shown to be acceptable to women and midwives.

Maternal Position During Delivery

Randomised controlled trials have evaluated birthing position for the second stage of labour. Two studies [60, 61] assessed squatting or other unsupported upright position compared with recumbent positions for the second stage of labour. Seven randomised controlled trials [96–102] comparing upright and recumbent position, showed that women delivering in upright position were less likely to have an episiotomy, had more lacerations and required repair. A Cochrane review on position in the second stage of labour for women without epidural anaesthesia [103] stated that there was no difference in second degree perineal tears and fewer episiotomies were performed in the birthing stool or squatting position. The rates of episiotomy were also lower for women using birth chair, but second degree perineal tears were increased. However, when considering all women, there was a higher risk of second degree tears in the upright positions except when the birthing cushion was used. The current evidence on the effectiveness of various delivery positions in the prevention of perineal trauma still remains inconclusive. So, it is suggested that women should be encouraged to deliver in whichever position is most comfortable for them.

Whirlpool Baths

Whirlpool baths have a place mainly in low risk midwifery units, but there was only one trial of Jacuzzi whirlpool baths in labour albeit not for birth. This showed less perineal trauma in the group assigned to the Jacuzzi [104].

Vacuum Versus Forceps Delivery

Perineal trauma after forceps and vacuum delivery has been compared in many RCTs [44, 105, 106]. Eason et al. [44] compared seven randomized controlled trials and showed that women delivered by forceps had more anal sphincter trauma than women delivered by vacuum extraction. Compared to vacuum extraction, forceps delivery was associated with almost twice the risk of developing faecal incontinence [105]. Vacuum delivery therefore causes significantly less maternal trauma compared to forceps. The weighted risk difference for anal sphincter trauma was −0.06 (95 % CI −0.10,−0.02). Obstetricians would need to deliver 18 women by vacuum than forceps to prevent one case of anal sphincter tear.

Perineal Support

A recent trend has been towards hands off (not to support the perineum) approach for normal vaginal delivery during crowning. There is lack of evidence for this

change of practice. In a recent survey of midwifery practice in England, 50 % of the midwives preferred a hands off/poised approach [107]. A recent Cochrane review has suggested no difference in OASIS rates between 'hands on' or 'hands poised/off' [95]. One of the studies (Decosta et al. [108]) was small and did not give any estimable effect. Mayerhofer et al. [109] reported only on third-degree tears. McCandlish et al. [4] reported on both third- and fourth-degree tears. There was considerable heterogeneity in the studies. The perineal techniques of the studies were different. The outcome measures were different. The terms "hands on," "hands off," "standard care" and "perineal support" meant different things across the studies and were not always defined sufficiently. In the McCandlish et al. study [4], "hands off" not only meant no hand on the perineum and infant's head until the head was born, but also no manual assistance for the birth of the shoulders, while Mayerhofer et al. [109] defined "hands off" as no hands on the perineum or fetal head until the head was born, but made no distinction between "hands on" and "hands off" for the assistance of the birth of the shoulders. In the Albers' study [110] "hands off" only meant no hands on the perineum until crowning of the head. Although the standard care or "hands on" manual support techniques are poorly described in most of the studies, it is clear that all studies implied a slow and controlled delivery of the head. Another randomized trial by Albers' et al which compared hands off the perineum technique and warm compress did not show any advantage or disadvantage in reducing the obstetric genital trauma [111]. Evidence from Finland suggests that their lower OASIS rate (0.6 %) is a result of the more frequent use of perineal support and episiotomy, compared with other Nordic countries (OASIS rates 3.6–4.2 %) [112]. In Norway, recent implementation studies of the 'hands on' method have shown a 50 % reduction in OASIS rates [113, 114], supporting a return to the use of the traditional method of perineal support as a method of prevention [115].

Pushing During Second Stage

During the second stage of labour, women may be encouraged to bear down throughout a contraction. A prolonged second stage, in which strong voluntary pushes are encouraged, has been implicated in denervation injury [69]. Parnell et al. [116] and Thompson [117] found no difference in perineal trauma between women who only pushed spontaneously and those who were directed to push throughout the contraction.

Episiotomy

The role of episiotomy has already been discussed elsewhere. Whether episiotomy should be used as a form of prevention is still controversial. However, a quasi randomized study by Coats et al. [67] in nulliparous women showed that midline episiotomies had 12 % anal sphincter tears compared with a 2 % anal sphincter tears in mediolateral episiotomies. A prospective study of almost 300,000 vaginal deliveries reported that the selective use of mediolateral episiotomy did protect against damage to the anal sphincter complex [43].

Perineal Hyaluronidase Injection for Reducing Perineal Trauma

Perineal hyaluronidase injection has been used during the second stage of labour for reducing perineal trauma. Four randomised controlled trials involving 599 woman were included in a Cochrane review [118]. Two trials compared hyaluronidase with placebo injection and three trials compared hyaluronidase with no intervention. In comparison to the group that received no intervention, the group, which received hyaluronidase during the second stage of labour, had a lower incidence of perineal trauma but no difference when compared with placebo injection. The role of perineal hyaluronidase during second stage is yet to be established.

Birth and Vaginal Training Devices - EPI-NO®

EPI-NO® – a new birth and vaginal training device – was developed to reduce the number of episiotomies and increase the incidence of an intact perineum by training with the device. A German doctor, Wilhelm Horkel, designed it. It consists of an inflatable silicone balloon connected to a hand pump. EPI-NO® is designed to dilate the vagina with the aim of adaptation of vagina and perineum to the delivering fetus. Furthermore, women can train their pelvic floor muscles and are able to develop a feeling for pushing process during labour. First results of a German trial demonstrated not only a significant decrease of perineal trauma (42 %) and much lower episiotomy rates (33 %) but also a significant reduction of analgesics, patient anxiety of birth and shortening the duration of second stage of labour after training with EPI-NO® [119].

Similar experience was reported by Kok et al. [120] who found a significant reduction in episiotomies and a tendency towards lower rates of injured perineum (90 % vs. 96.6 %). Similar results with significantly higher rates of intact perineum and a lower rate of perineal tears were observed in an Australian trial [121]. Another prospective randomized multicentre trial concluded that training with EPI-NO® is safe for both mother and child, easy to use, helps to avoid unnecessary episiotomies and increases the likelihood of having an uninjured perineum [122]. Further improvement of these results by means of combining EPI-NO® and perineal massage should be evaluated. A randomized controlled pilot study of EPI-NO® on levator trauma by Shek et al. [123] showed a weak trend towards a lower incidence of levator avulsion and irreversible overdistension in women allocated to EPI-NO® group than in those who actually used the device. It also concluded that a larger sample study size was needed to determine the efficacy on EPI-NO® on levator muscle. Shek et al. [123] also performed a subgroup analysis in women who delivered by prelabour or first-stage caesarean section. No significant differences in peripartum changes of hiatal areas and pelvic organ descent between the control and EPI-NO® groups were found.

Tertiary Prevention Strategies

Tertiary prevention strategies aim to address the mode of delivery in subsequent pregnancies for women with previous childbirth injuries to the pelvic floor.

A study by Fynes et al. [124] evaluated the effect of a second vaginal delivery on anal sphincter structure and function. This study assessed women at risk for

cumulative anal sphincter injury and development of anal incontinence and included 59 women who went through two successive vaginal deliveries. After two vaginal deliveries 25.4 % of women reported symptoms of anal incontinence. Seven out of eight women with anal incontinence during their second pregnancy noticed deterioration of symptoms after their second vaginal delivery. The study concluded that women with persistent or transient anal incontinence, and asymptomatic women with anal sphincter defects after their first vaginal delivery, are at high risk for cumulative injury. The study concluded that elective caesarean delivery and a repeat sphincter repair should be given as an option for women with persistent anal incontinence and a sphincter defect. For asymptomatic women with anal sphincter defects appropriate counselling regarding risk of anal incontinence after a second vaginal delivery should be given, so the women can make an informed decision about their subsequent delivery. The Royal College of Obstetricians and Gynaecologists also recommends that all women who have sustained an obstetric anal sphincter injury in a previous pregnancy and are symptomatic or have abnormal endoanal ultrasonography and or manometry should have the option of elective caesarean birth.

Peleg et al. [125] found that women with a history of a third- or fourth-degree tear were 2.3 times as likely to have a repeat tear in a subsequent delivery than women without a history of third- or fourth-degree tear.

Prevention or Prediction?

Preventing perineal trauma would prove to be a significant health benefit factor in childbearing women. It would also reduce the cost and complications that follow it. Hamilton et al. developed a prediction model using CART analysis [126]. Classification and regression trees (CART) analysis is also known as recursive partitioning. It is seldom described in obstetric literature. Hamilton et al. developed their model from a large dataset that included information from several institutions. CART analysis takes individual risk factors, determines the one that best separates patients with or without the problem of interest, and divides the dataset on that basis. The process is carried out repeatedly, identifying the next most discriminating factor in turn. It continues until no variables with discriminating ability are left. At the end of CART analysis, patients are grouped and the risk factors are assessed for a given outcome for each of the groups. William Grobman [127] critically evaluated this model. Firstly, the discriminatory capacity of the original model does not appear to be better. Secondly, this demonstrates that populations other than the one in which the model was developed will still have their outcomes accurately predicted by the model. Several specific aspects of the model presented by Hamilton et al. [126] also are important to consider. Hamilton et al. considered episiotomy of primary importance in the model. This suggests a potential weakness as episiotomy is not a routine in many institutions. Thus, in these circumstances, the ability of the tree to provide further clinically usable information is extremely limited for the vast majority of the population. If a woman does not have an episiotomy, the only other discriminatory factor is the length of the second stage, and this can be used to distinguish women with point-estimate probability of 1.7 % from those with a

point-estimate probability of 8.9 %. The authors also documented the combination of episiotomy, instrumental delivery, and birthweight, noting, for example, that every one of the women who had episiotomy, vacuum, and a newborn with a birthweight 4312 g or more ended up with a third- or fourth-degree laceration. This was based on a sample size of just seven women, so that the confidence interval for this probability was wide. The use of birth weight in this model is an important one. The birth weight is an acceptable factor to use if the CART is used for the purpose of an understanding of the factors. However, if the purpose of the CART is to help in prediction, then a factor such as birthweight is unlikely to be helpful as it is not known accurately before birth. This cannot be simply substituted for the actual birthweight used in the model because the predicted probabilities may no longer be valid. CART analysis should be validated in different populations before it is to be used as a prediction model.

Awareness of the risk factors does not always help to predict which women will sustain a sphincter tear and tears occur in women without risk factors. Approximately one third of births are in nulliparous women. An occipito-posterior position of the fetal head is probably present in about one in five women at the start of labour. In many of these women the fetal head rotates to an occipito-anterior position, although this may not occur until late in labour or at the time of an assisted delivery. A persistent occipito-posterior position cannot be predicted.

Risk prediction models have been tried but whether they effectively assist in prevention is debatable. Introduction of high-quality prediction models into clinical practice may result in reduced incidence of childbirth trauma. However, validation of such prediction models is important.

A contentious debate regarding the role of primary elective caesarean has gained interest. An important aspect of this debate relates to the potential benefits of caesarean in the prevention of pelvic floor dysfunction. However the development of appropriate prevention strategies has been impossible by a lack of adequate data. Patel et al. [128] developed an epidemiological approach for the assessment of prevention opportunities at delivery. The study focused mainly on primary prevention. However, they also accepted that the development of appropriate prevention strategies has been stalled by lack of data. Primary prevention denotes an action taken to prevent the development of a disease in a person who is well and does not have the disease in question. Epidemiological studies usually aim to determine the incidence and risks of a disease in a population. The science of epidemiology is based on the determination of whether an association between an exposure and an outcome reflects a causal association. With respect to prevention, measures based on absolute differences are preferred, because they provide an estimate of the excess risk that is associated with a given exposure. Attributable risk measures indicate the potential for prevention if the exposure could be eliminated, given that the exposure and outcome are linked causally. The basic concept of absolute risk is that it subtracts the incidence of the outcome in the unexposed (e.g., incidence of PFD in women who did not undergo vaginal delivery) from that in the exposed (e.g., incidence of PFD in women who have undergone vaginal delivery). On the basis of these epidemiological data, Patel et al. calculated the attributable risk percentage from a cohort

study using the following formulas: [incidence in exposed – incidence in unexposed]/incidence of exposed × 100 or ([relative risk – 1]/relative risk × 100. However the data required to calculate the excess risk of POP attributable to vaginal delivery are not available. Long-term prospective studies are required to provide data that in turn could be used for the development of targeted prevention strategies. Further epidemiological studies are still awaited for a definitive recommendation.

In conclusion, it seems that elective caesarean section before labour is the only true primary prevention strategy for childbirth injuries to the pelvic floor but the risks of an elective caesarean section should be taken into account while counselling a woman. Alternative primary prevention strategies include antepartum pelvic floor exercises, use of vaginal training devices and perineal massage. Secondary prevention strategies must focus on modifying obstetric practices that predispose women to childbirth injury. These factors may include restrictive use of episiotomy, mediolateral episiotomy when necessary, spontaneous over forceps-assisted vaginal delivery, vacuum extraction over forceps delivery, and perineal massage during second stage. Tertiary prevention strategies should address the mode of delivery recommended for women with childbirth injuries who desire future pregnancies.

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Pelvic Floor Physiotherapy for the Prevention and Management of Childbirth Trauma

17

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Abstract

Pregnancy and childbirth are known risk factors for weakening and injury to the perineum and pelvic floor muscles. Stretch and rupture of peripheral nerves, connective tissue and pelvic floor muscles may cause pelvic floor dysfunctions such as urinary and anal incontinence. Controlled trials have found pelvic floor muscle training to be effective in both prevention and treatment of incontinence. Common factors for all trials reporting a positive effect of pelvic floor muscle exercises (PFMT) in pregnancy or postpartum are found to be thorough clinical assessment of the women's ability to perform a voluntary pelvic floor muscle contraction, close individual or group follow-up, and high adherence to the exercise protocol. Symptoms of incontinence before or during pregnancy have been found to be the main risk factors for incontinence symptoms postpartum. Similarly, women with symptoms of incontinence in the first year after delivery have an increased risk of long

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term incontinence symptoms. The cost of incontinence-related illness is a substantial economic and human burden, highlighting the need for effective forms of prevention and management.

Keywords
Physiotherapy • Pelvic floor muscle training • Urinary incontinence • Anal incontinence • Faecal incontinence

Pregnancy and childbirth are known risk factors for weakening and injury to the perineum and pelvic floor muscles (PFM). Stretch and rupture of peripheral nerves, connective tissue and PFM may cause urinary (UI) and anal incontinence (AI), pelvic organ prolapse, sensory and emptying abnormalities of the lower urinary tract, defecation dysfunction, sexual dysfunction and chronic pain syndromes [1]. UI is defined as "any involuntary loss of urine" [2]. Anal incontinence (AI) is defined as "involuntary loss of faeces or flatus," faecal incontinence as involuntary loss of faeces and flatal incontinence is the involuntary loss of flatus [2]. About 50 % of women lose some of the supporting function of the pelvic floor due to childbirth [3], and recent research using ultrasound and MRI report prevalence of major injuries to the PFM of 20–26 % following vaginal delivery [4–6]. Hence, vaginal delivery may be considered equivalent to a major sports injury, but unfortunately PFM injuries have not been given the same attention concerning prevention or treatment as their sports-related counterparts.

Pelvic Floor Muscle Training and Incontinence

Pelvic floor muscle training (PFMT) has been a part of exercise programs in Chinese Taoism for over 6000 years [7]. PFMT has been found to have a high cure rate as treatment for UI after being popularized by the American gynecologist Arnold Kegel in the late 1940s [8]. The concept of intensive PFMT was introduced by Bø and co-workers in the 1990s [9] and PFMT is now recommended as first line treatment for stress urinary incontinence (SUI), urge urinary incontinence as well as mixed urinary incontinence in the adult female population [7]. Compared to UI, there is scarce documentation on the general effect of PFMT on AI symptoms in pregnancy and postpartum [10–13].

The recommendations for effective strength training to increase general muscle cross-sectional area and strength are 3 sets of 8–12 close to maximum contractions 3–4 times per week [14]. The effect of an exercise regimen is also influenced by factors such as type of exercise, frequency, intensity and duration of the training, as well as adherence [9, 14]. Furthermore, the success of PFMT depends on the ability to effectively contract the PFM and it has been estimated that on the first attempt, 30 % of women are unable to contract the PFM [15, 16]. After a brief standardized verbal instruction, only 49 % were found able to perform an ideal voluntary pelvic floor muscle contraction (VPFMC) [15]. Dinc and co-workers (2009) [17], found

that only 68 % of pregnant women were able to perform a correct VPMFC 1 week after thorough individual instruction. Vaginal palpation is the method most commonly used by physiotherapists to evaluate the function and strength of the PFM. However ultrasound is a more objective measure and is becoming an important clinical tool [18].

A variety of hypotheses have been suggested as to why PFMT might help prevent and treat incontinence in pregnancy and after delivery. Strength training of the PFM builds muscle volume, elevates the location of the PFM and pelvic organs, and closes the levator hiatus thus providing improved structural support for the pelvic floor as well as more optimal automatic function [19]. Neural adaptations and motor learning may explain increases in muscle function before hypertrophy occurs, as muscle function improves with increasing number of recruited motor units [20]. Morphological changes such as increased muscle thickness, narrowed resting area of the levator hiatus, reduced pubovisceral length and elevated resting position of the bladder and the rectal ampulla after PFMT have been documented after an intensive PFMT program in non-pregnant women [21]. The functional changes (elevated resting position of the bladder and rectum and the reduced pubovisceral length and hiatus size at maximum Valsalva) may be explained by increased "stiffness" in the muscle-connective tissue complex [21].

During pregnancy, the growing uterus will compress the bladder. Strengthening the PFM results in better structural support for the bladder neck and a strong contraction of the PFM ensures continence during an abrupt increase in the abdominal pressure [22]. Further, PFMT during pregnancy may also aid in counteracting the increased intra-abdominal pressure caused by the growing fetus, the hormonally mediated reduction in urethral pressure, and the increased laxity of fascia and ligament in the pelvic area [23]. The effect of PFMT as a successful treatment of incontinence may also be explained by behavior modification in addition to increased PFM strength. A total of 80 % of women with de novo SUI in pregnancy week 35 reported being able to reduce leakage during coughing by using the "Knack" maneuver (i.e., tightening of the PFM in preparation for a known leakage-provoking event), with 55 % eliminating leakage completely [24]. The rationale for teaching women how to perform a conscious contraction of the PFM before and during increases in abdominal pressure [24] is that the urethra and bladder base is prevented from descending [22].

Another hypothesis is that a trained muscle may be less prone to injury, and previously trained muscles may be easier to retrain after damage as the appropriate motor patterns are already learned. It may be that previously trained muscles has a greater reserve of strength so that injury to the PFM muscles, or the nerve supply, does not cause sufficient loss of muscle function to reach the threshold where reduced urethral pressure results in leakage [23]. Further, PFMT may in theory improve the mechanism maintaining anal continence and closure of the anal canal by improving the strength and function of the puborectalis, internal and external anal sphincter muscles in largely the same manner as indicated in the treatment of UI [7, 11]. However, it has been questioned whether it is possible to distinguish between a voluntary contraction of the external anal sphincter muscle as compared to a general VPFMC [13].

Due to the physiological and hormonal changes occurring during pregnancy as well as after delivery, it may be that the effect of PFMT differs between pregnant and postpartum women [11]. We have thus reviewed the evidence of PFMT and treatment efficacy in pregnancy and postpartum in current literature separately.

UI and PFMT in Pregnancy

In a systematic literature search on PubMed, two trials [25, 26] were found addressing primary prevention of UI during pregnancy including only continent women (Table 17.1), and five trials included a mixed population with both continent and incontinent women [12, 27–30]. Further, three trials assessed the effect of treatment of UI during pregnancy including only incontinent women (Table 17.2) [17, 31, 32]. Seven of the trials assessing PFMT during pregnancy found UI to be less prevalent in the intervention group [12, 17, 25, 28–30, 32], and three trials found no differences between groups [26, 27, 31]. One long-term follow-up study found that the difference in UI symptoms and quality of life (QoL) was not present 8 years after the index delivery [33]. Two of the trials [12, 27] included PFMT in a general fitness program for pregnant women with diverging results. In the trial by Stafne and co-workers (2012) [12], UI was found to be less prevalent in the intervention group. Bø and Haakstad (2011) [27] randomised sedentary pregnant women to either usual care or PFMT as part of a general fitness class twice weekly and found no difference in UI between groups. However, this trial was underpowered, included no assessment of the participants' ability to perform a correct VPFMC, had high dropout rates and low adherence to the exercise training protocol. A recent Cochrane review of trials including continent as well as incontinent primiparae [23] concluded that PFMT may prevent UI up to 6 months postpartum. Further, PFMT was found to be an effective treatment option for women with persistent postpartum UI [11].

AI and PFMT in Pregnancy

No randomised or quasi-randomised trials reporting on the effect of PFMT on primary prevention of AI/FI in pregnancy were identified. Two trials including both continent and incontinent women reported on AI/FI in pregnancy. Bø and Haakstad (2012) [27] found no differences in AI symptoms between intervention and control groups. Although not reaching statistical significance, Stafne and co-workers (2012) [12] found fewer women with FI in the intervention group. In a subgroup analysis, however, performing PFMT in the second half of pregnancy was shown to have a protective effect on late pregnancy FI in multiparous and not in primiparous women [12]. These findings indicate that even among women with potential weakening or injury to the PFM or obstetric anal sphincter injury from a previous pregnancy or delivery, specific training of the PFM may prevent or reduce the severity of incontinence in subsequent pregnancies. However, as FI was not the primary outcome measure of these studies, both were underpowered to assess FI.

Table 17.1 Controlled trials assessing the effect of pelvic floor muscle training as primary prevention of UI during pregnancy and postpartum including women continent at inclusion only

Author	Subjects	Design/intervention	Outcome	Results (IG vs CG)	Comment
Mason et al. (2010) [26]	N=311 Nullipara No previous SUI Included between 11 and 14 weeks of pregnancy	2 arm RCT single blind 1. Control: usual care and instructions in PFMT. 2. Intervention: attending a physiotherapy class (45 min) with PFMT 1×/month for 4 months and to perform daily PFMT at home (8–12 close to maximum PFMC each held 6–8 s twice daily). Individual assessment of correct VPFMC was performed in most women.	Bristol Female Lower Urinary Tract Symptoms Questionnaire (BFLUTS) Leicester Impact Scale (LIS) Self-reported number of leakage episodes last 3 days	36 weeks of pregnancy: UI: 24/60 (40 %) vs. 51/96 (53 %) (p=0.14) 3 months PP: UI: 23/68 (33.8 %) vs. 33/80 (41.3 %) (p=0.40) No sig difference in symptoms and episodes of UI between groups in late pregnancy and PP	8 % were lost to follow-up. 23 % failed to return any of the questionnaires, and only 31 % completed all three sets of questionnaires Only 65 % of IG women attended ≥1 exercise class The trial was underpowered

(continued)

Table 17.1 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs CG)	Comment
Reilly et al. (2002) [25]	N=268 Nullipara Continent with increased bladder neck mobility Included at 20 weeks of pregnancy	2 arm RCT 1. Control: routine antenatal care likely to include verbal instructions in PFMT. 2. Intervention: Individual PFMT with physiotherapist at monthly intervals from 20 week' of pregnancy until delivery, with additional home exercises. 3 sets of 8 contractions (each held for 6 s), repeated twice daily. At 34 weeks of pregnancy the number of contractions per set was increased from 8 to 12. Women were instructed to contract the PFM when coughing or sneezing. Performing PFMT was recorded in a personal training diary.	Self-reported UI Symptom questionnaire Bladder neck mobility measured by ultrasound	3 months PP: UI: 19 % vs. 33 % (p=0.02) QoL: higher score in the exercise group (p=0.004) Pad test: no difference Bladder neck mobility: no difference PFM strength: no difference	Lost to follow-up: 14 % Adherence: 46 % IG women performed PFMT ≥28 days 51 % of CG did unsupervised PFMT
Agur et al. (2008) [33] 8 year follow-up	N=164		Self-reported UI Performing PFMTSelf-reported UI Performing PFMT	8 years after index delivery: UI: 35 % vs. 39 % (p=0.75) Little or no difference in UI severity or QoL between groups. Performing PFMT >1/weekly: 38 % in IG, CG not reported.	Drop-out: 34 % vs 23 % (p=.004)

Abbreviations: *IG* intervention group, *CG* control group, *PFM* pelvic floor muscles, *PFMC* pelvic floor muscle contraction, *PFMT* pelvic floor muscle training, *QoL* quality of life, *RCT* randomized controlled trial, *SUI* stress urinary incontinence, *UI* urinary incontinence, *VAS* visual analogue scale, *VPFMC* voluntary pelvic floor muscle contraction

UI and PFMT Postpartum

In a systematic literature search on PubMed, ten randomised trials were found where six trials included a mixed population of continent and incontinent women [34–39] and four trials included incontinent women only [40–43] (Table 17.3). All four trials assessing the effect of PFMT in treatment of urinary incontinence postpartum had significant reductions in UI among intervention group women. Kim and co-workers (2012) [40] compared a PFMT programme with abdominal strengthening exercises and trunk stabilisation with or without supervision and found reductions in clinical symptoms of UI, including QoL measures in both groups. The improvements were greater in the supervised group, however, this was a pilot study and included only 20 postpartum women. In addition, two studies reported on 6 and 7 years follow up. Glazener and co-workers (2005) [44] found no differences between groups in UI after 6 years, whereas Dumoulin and co-workers (2013) [45] found that over 50 % of the women in the PFMT groups were still continent after 7 years.

In the trials with a mixed population, an improvement in UI was found in three out of six studies [36–38]. Mørkved and Bø (2000) [46] reported that the effect of PFMT was still present 1 year after the cessation of the training programme, while Chiarelli and Cockburn (2004) [47] found no persistent effect after 1 year. However, they found that continued adherence to PFMT at 12 months was predictive of UI at that time. Three trials found no differences between intervention and control group [34, 35, 39], however, in two of the trials there was little distinction between the intervention offered and the care given to the control group [35, 39]. The trial by Hilde and co-workers (2013) [34] included primiparous women 6 weeks postpartum. All participants were given thorough instructions in correct VPFMC, with both ultrasonography and digital palpation prior to randomisation to either usual care or weekly PFMT class and home PFM exercises. After the 16 weeks of intervention period, there were no differences between groups with regards to prevalence of UI or effect size.

AI and PFMT Postpartum

In the three studies reporting on mixed prevention and treatment of FI/AI postpartum, there was no evidence favouring PFMT over standard or usual care [27, 39] or no PFMT [37]. However, none of these studies were designed with FI or AI as the main outcome measure, the confidence intervals tended to be wide, indicating that the trials may have been underpowered in order to evaluate the effect of the intervention on postpartum FI/AI. In the study by Sleep and Grant (1987) [39], both study groups received standard instructions of home PFMT prior to discharge home from hospital after delivery, and one group also received reinforcement of these instructions during home visits by community midwives during the first 4 weeks postpartum. The effect of these interventions was assessed 2 months later and revealed no differences in UI or FI between groups [39]. Two trials have reported on the prevalence of AI/FI and

Table 17.2 Controlled trials assessing the effect of pelvic floor muscle training during pregnancy to prevent and treat incontinence including women with and without UI or AI at inclusion

Author	Subjects	Design/intervention	Outcome	Results (IG vs. CG)	Comments
Stafne et al. (2012) [12]	N = 855 Nulli-/multiparae Continent/incontinent Included between 18 and 22 weeks of pregnancy	2 arm RCT 1. Control: standard care (including written instructions in PFMT) 2. Intervention: attending weekly standard exercise program (60 min) and performing a home exercise twice weekly (45 min). The group and home exercise program consisted of aerobic activity and strength exercises including PFMT. Encouraged to perform 3×8–12 close to maximum PFMC each held for 6–8 s, 3 fast contractions added at the end of the contraction. Individual instruction on correct PFM, VPFMC checked Intervention period lasted 12 weeks	Self-reported UI and FI	After 12 weeks intervention (in pregnancy week 32–36): UI ≥1 times per week: 11 % vs. 19 % (p = 0.006) SUI ≥ 1 times per week: 7 % vs. 13 % (p = 0.03) FI: 3 % vs. 5 % (p = 0.24)	Loss to follow-up: 11 % Adherence: 55 % adhered to the exercise protocol (exercising ≥3 times per week at the end of the intervention period)

Sangsawang et al. (2011) [32]	N = 70 Nulli-/multiparae SUI (≥1 episode of urinary leakage last month) Included between 20 and 30 weeks of pregnancy.	Quasi-experimental study, pre-post test with control group 1. Control: usual nursing care (including written instructions in PFMT) 2. Intervention: written and individual oral instruction in correct VPFMC (no vaginal palpation, but instructions in "stop-test" i.e. trying to stop or slow urinary flow) and PFMT. PFMT (45 min) in groups every second week for 6 weeks. Home exercises, with 40 repetitions daily, 5×/week (slow contractions with 10 s holding time and 10 fast contractions at the end of holding time). Performing PFMT was recorded in a training diary.	SUI severity (frequency and amount) SUI severity (VAS)	After 6 weeks intervention: No leakage: 39 % vs. 0 % (p = 0.001) Frequency SUI: 2 ± 4 vs. 15 ± 10 (p < 0.001) Severity SUI (VAS): 1.3 ± 1.3 vs. 6.8 ± 2.2 (p < 0.001)	Loss to follow up: 4 (IG) Adherence: 100 % in IG adhered to the exercise protocol (≥28 days of PFMT) Women in the IG were matched to CG women regarding age, parity and severity of SUI Women who failed to perform PFMC were categorized as dropout No assessment of correct VPFMC

(continued)

Table 17.2 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs. CG)	Comments
Bø and Haakstad (2011) [27]	N = 105 Nulliparae Continent/incontinent Sedentary Included within 24 week of pregnancy	2 arm RCT 1. Control 2. Intervention: attending aerobic fitness classes including PFMT, 2–3×/week, and performing 10 daily PFMC at home. The PFMT instructed in classes consisted of three sets of close to maximum contractions of 8–12 repetitions with holding periods of 6–8 s performed in different positions. Women were encouraged to be physically active ≥30 min daily. Intervention period lasted 12–16 weeks.	UI and AI reported in a personal interview	36–38 weeks of pregnancy: UI: 17/42 vs. 16/42 (p = 0.82) Flatus: 11/42 vs. 9/16 (p = 0.61) AI: 1/42 vs. 1/42 6–8 weeks postpartum: UI: 12/43 vs. 13/47 (p = 0.99) Flatus: 10/43 vs. 8/47 (p = 0.46) AI: 1/43 vs. 3/47 (p = 0.62)	Loss to follow-up: 20 % Adherence: 40 % of IG women attended at least 80 % of the exercise sessions No assessment of correct VPFMC Classes led by an aerobic instructor, verbal instructions were given No data on level of exercise and PFMT in the CG

Ko et al. (2011) [28]	N = 300 Nulliparae Continent/incontinent Included between 16 and 24 week of pregnancy Performing PFMT was an exclusion criteria	2 arm RCT 1. Control: received regular prenatal care. 2. Intervention: weekly PFMT in group led by a physiotherapist (45 min). And daily home exercises (3 repetitions of 8 contractions each held for 6 s) performed twice daily. Women were individually instructed in anatomy and correct VPFMC (by observation of inward movement of perineum during contraction).	UI reported in a personal interview Incontinence Impact Questionnaire (IIQ-7) Urogenital Distress Inventory (UDI-6)	36 weeks of pregnancy: UI: 34 % vs. 51 % ($p<0.01$) 3 days postpartum: UI: 30 % vs. 41 % ($p=0.07$) 6 weeks postpartum: UI: 2 % vs. 35 % ($p=0.06$) 6 months postpartum: UI: 16 % vs. 27 % ($p=0.04$) The IG had lower scores in total UDI-6 and IIQ-7 than CG in late pregnancy and postpartum No differences in pregnancy outcome	Loss to follow-up: 0 Adherence (defined as performing ≥75 % of PFMT): 87 %

(continued)

Table 17.2 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs. CG)	Comments
Dinc et al. (2009) [17]	N = 92 Nulli-/multiparae Incontinent (having complaints of stress/ mixed UI in their history) Included between 20 and 34 weeks of pregnancy	2 arm RCT 1. Control 2. Intervention: thorough instructions in correct PFMC and a home exercise programme with gradually increasing repetitions and holding time until 3 sets of 15 repetitions with 10 s holding time and fast contractions added, repeated 3×/day.	Self-reported UI	36–38 weeks of pregnancy: UI: 43 % vs. 71 % 6–8 weeks postpartum: UI: 17 % vs. 38 % Significant difference in episodes of UI, urgency, number of voids and amount of urine in pad test in favor of the IG both at 36–38 weeks pregnancy and at 6–8 weeks postpartum	Loss to follow-up: 12 Correct VPFMC checked at enrolment in both groups
Woldringh et al. (2006) [31]	N = 264 Nulli-/multiparae Incontinent (≥2 episodes of loss of urine last month) Included between 17 and 20 weeks of pregnancy	2 arm RCT 1. Control: routine care. 2. Intervention: four sessions of individual PFMT; three sessions (with 2 week interval) between 23 and 30 weeks of pregnancy and a fourth session 6 weeks PP. Written information including a detailed PFMT programme.	Self-reported severity of UI Incontinence Impact Questionnaire (IIQ)	No difference between IG and CG with respect to the severity of UI and impact of UI on daily life	Lost to follow-up: 50 % Adherence: 37 % reported to exercise almost every day No vaginal palpation of VPFMC, but observation and palpation of the perineal body

Mørkved et al. (2003) [29]	N = 301 Nulliparae Continent/incontinent Included at 20 weeks of pregnancy	2 arm RCT single blind 1. Control: customary information given by their midwife or general practitioner. Correct VPFMC checked at inclusion. 2. Intervention: 12 weeks of weekly PFMT in groups led by physiotherapist, with additional home exercises (10 close to maximum PFMC each held for 6 s, 3–4 fast contractions added on the last 4 exercises. Repeated 2×/day). Correct VPFMC checked at inclusion. Intervention period between 20 and 36 weeks of pregnancy	Self-reported UI (women reporting UI ≥1/week were categorized as incontinent)	36 weeks of pregnancy: UI: 32 % vs. 48 % (p = 0.07) 3 months postpartum: UI: 20 % vs. 32 % (p = 0.02)	Dropout rate, 4 % Adherence: 81 %

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Table 17.2 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs. CG)	Comments
Sampselle et al. (1998) [30]	N = 72 Nulliparae Continent/incontinent Included at 20 weeks of pregnancy	2 arm RCT single blind 1. Control: routine care. 2. Intervention: PFMT tailored to the woman's individual ability, with muscle identification exercises preceding strength-building efforts. 30 PFMC daily at maximum or near-maximum intensity was recommended for strength building. Correct VPFMC were checked.	Self-reported UI (results reported as change in mean UI symptom score)	35 weeks of pregnancy: Less UI symptoms were seen in the IG vs. CG (p = 0.04) 6 weeks postpartum: Less UI symptoms were seen in the IG vs. CG (p = 0.03) 6 months postpartum: Less UI symptoms were seen in the IG vs. CG (p = 0.04)	Adherence 85 % Study participants were financially compensated, $150

Abbreviations: *AI* anal incontinence, *IG* intervention group, *CG* control group, *PFMC* pelvic floor muscle contraction, *PFMT* pelvic floor muscle training, *RCT* randomized controlled trial, *SUI* stress urinary incontinence, *UI* urinary incontinence, *VAS* visual analogue scale, *VPFMC* voluntary pelvic floor muscle contraction

Table 17.3 Controlled trials assessing the effect of pelvic floor muscle training postpartum to prevent and treat incontinence including women with and without UI or AI at inclusion

Author	Subjects	Design/intervention	Outcome	Results (IG vs CG)	Comment
Urinary incontinence as primary outcome measure					
Hilde et al. (2013) [34]	N = 175 Primiparae with no 3rd or 4th grade perineal tear Continent/incontinent Included at 6 weeks after vaginal delivery, stratified by major levator ani muscle defects	2-arm RCT, single blind 1. Control: oral and written instruction on how to perform correct PFMC at inclusion, no further intervention 2. Intervention: weekly PMFT, in group led by physiotherapist. Daily home exercises: 3 sets of 8–12 close to maximum PFMC each held for 6–8 s, 3 fast contractions added on the last 4 exercises. Intervention period was 16 weeks. VPFMC checked at inclusion in both groups. Exercise diary of home exercises, group attendance recorded by physiotherapist.	Self-reported UI (ICI-Q UI SF) Pad test	Baseline: UI: 39 % vs 50 % 6 months postpartum: UI: 35 % vs 39 %, effect size: 0.89 (95 % CI: 0.6–1.3) Similar results in stratum with or without major leavtor ani defects, effect size: 0.89 (95 % CI: 0.5–.16) vs 0.90 (95 % CI: 0.5–1.5), respectively. No sign differenced in pad test between groups.	Loss to follow up: 8.6 %; Higher drop-out rate in IG: 12/87 vs. 3/88 96 % adhered to the exercise protocol (≥80 % of class sessions and daily home training Number of women performing PFMT at baseline: 35 % vs 50 % (p = .10)
Kim et al. (2012) [40]	N = 20 Parous women Incontinent Included <6 weeks after normal vaginal delivery.	2-arm RCT, pilot study 1. Control: Unsupervised PFMT 2. Intervention: Supervised PFMT Intervention period was 8 weeks. VPFMC checked at inclusion in both groups. Both groups were instructed to perform PFMT daily. All women received a booklet and exercise diary.	UI (Bristol female lower urinary tract symptoms) Vaginal squeeze pressure	Significant difference in UI and vaginal squeeze pressure in favour of the supervised PFMT group on after the intervention period.	Loss to follow up: 2/20 Adherence: not stated

(continued)

Table 17.3 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs CG)	Comment
Ewings et al. (2005) [35]	N = 234 Parous women Continent/incontinent	Nested RCT 1. Control: usual postnatal care including verbal promotion of postnatal PFMT and leaflet explaining how to do PFMT. 2. Intervention: taught one to one with physiotherapist in hospital, with intervention to attend PFMT group at 2 and 4 months after delivery. No details of PFMT programme given.	UI	6 months postpartum: UI: 60 % vs 47 % (p = .10)	Loss to follow-up: 19 % (27/90 vs 17/100) Adherence to PFMT in the intervention group: 5/90 (5.6 %)

Dumoulin et al. (2004) [41]	N = 64 Parous women Weekly SUI at ≥3 months after their last delivery Recruited during annual gyenaecological visit	3-arm RCT 1. Control: 8 weekly sessions of massage 2. PFM rehabilitation: Weekly sessions supervised by physiotherapist for 8 weeks: 15-minutes electrical stimulation (biphasic rectangular form; frequency 50 Hz; pulse with 250 ms; duty cycle, 6 s on and 18 s off for the first 4 weeks and 8 s on and 24 s off for the last 4 weeks; maximal tolerated current intensity) + 25 min PFMT with biofeedback + home training 5 days per week. 1. PFM rehabilitation (as group 2) + 30 min deep abdominal muscle training	Pad test Self-reported weekly UI	Less than 2 g urine on pad test after intervention period: CG: 0/19 PFM rehabilitation: 14/20 PFM rehabilitation + deep abdominal muscle training: 17/23 Significant difference in favour of the intervention groups (p = 0.001) Non-significant difference between the two intervention groups Incontinence Impact Questionnaire: Significant difference in favour of the intervention groups PFM strength: Non-significant difference between groups.	Drop-out rate: 6 % High adherence
Dumoulin et al. (2013) [45] 7 year follow-up	N = 35 Combination of the previous two intervention groups		Pad test Self-reported UI	Less than 2 g urine on pad test (performed by 26 out of 35 women): 14/26 (53 %) Incontinence Impact Questionnaire: Significantly better than at baseline	61.4 % of the participants from the original studies agreed to participate in the follow-up study Percentage performing any PFMT: 54 %

(continued)

Table 17.3 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs CG)	Comment
Chiarelli & Cockburn (2002) [36]	N = 720 Primi-/multiparae Continent/incontinent Postnatal women following forceps or ventouse delivery, or birthweight 4000 g or more Included while in hospital postpartum	2-arm parallel group 1. Control: usual care, leaflet on PFMT 2. Intervention: continence promotion. One contact with physiotherapist on postnatal ward and another at 8 weeks postpartum (correct VPFMC checked at second visit). Intervention included individually tailored PFMT, use of transversus abdominus contraction, the 'Knack', techniques to minimise perineal descent, postpartum wound management. Written and verbal information.	Self-reported UI Urinary diary (3 days)	Baseline: UI: 18 % vs 17 % 3 months postpartum: UI: 31 % vs 38 % (p = .04)	Drop-out rate: 6 % in each group Adherence: 84 % vs 58 % (p = .001)
Chiarelli et al. (2004) [47] 12 month follow-up			Self-reported UI (telephone interview)	12 months postpartum: No significant difference in UI status IG significantly more likely to perform PFMT at adequate levels, continued adherence predictive of continence at 12 months postpartum	Drop-out rate: 30 %

Glazener et al. (2001) [42] N = 747 Primi-/multiparae UI at 3 months postpartum	2-arm RCT 1. Control: no visit 2. Intervention: assessment of UI by nurses, with conservative advice on PFM exercises (80–100 fast/slow contractions daily) 5, 7, and 9 months after delivery supplemented by bladder training if appropriate at 7 and 9 months	Self-reported UI and FI PFMT	3 months postpartum (baseline): Severe UI: 57 % vs 54 % FI: 16 % vs 15 % 12 months postpartum: Any UI: 60 % vs 69 % (p = .037) Severe UI: 20 % vs 32 % (p = .002) Any FI: 4 % vs 11 % (p = .012) Severe FI: 2 % vs 5 % (p = .075)	Loss to follow-up at 12 months postpartum: 25 % vs 35 %
Glazener et al. (2005) [44] N = 516 6 year follow-up			6 years postpartum: Severe UI: 58 % vs 51 % Any FI: 14 % vs 14 %	Loss to follow-up at 6 years postpartum: 30 % (263/371 vs 253/376) Percentage performing any PFMT at 6 years postpartum: 50 % vs 50 %

(continued)

Table 17.3 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs CG)	Comment
Meyer et al. (2001) [37]	N = 107 Nulliparae Continent/incontinent Recruited between 12 and 39 weeks postpartum (mean 29 ± 7 weeks)	2-arm controlled study, assigned in alternating manner 1. Control: no PFM education until after intervention period 2. Intervention: 12 sessions of PFMT, followed by 20 min biofeedback and 15 min electrical stimulation. Intervention period was between 2 and 10 months postpartum. VPFMC was checked prior to intervention.	Self-reported UI and FI	Base line: UI: 14/51 vs 18/56 AI: 0/51 vs 0/56 10 months postpartum: UI: 6/51 vs 8/56 AI: 2/5| vs 3/56 Cure rate: 19 % vs. 2 % (p = 0.002)	Loss to follow up, drop-out or adherence to PFMT protocol not reported. PFMT protocol not reported
Wilson and Herbison (1998) [43]	N = 230 Primi-/multiparae Incontinent at 3 months postpartum. Stratified by parity, UI severity and mode of delivery.	2-arm RCT 1. Control: standard post natal PFMT taught by physiotherapist while in hospital 2. Intervention: 4 sessions (3, 4, 6, 9 months postpartum) with PFME instructed by physiotherapist. IG further randomised into 3 groups; (a) PFMT (8–10 sessions of a total of 80–100 daily PFMC + biofeedback with vaginal perionometer); (b) PFMT & cone weights (PFMT + 15 min training with vaginal cone weight daily) (c) Cone group (15 min training with vaginal cones) daily	Self-reported UI and FI Home pad test PFMT in the past months	Baseline: UI: 89 % vs 89 % PFMT in last month: 79 % vs 77 % 12 months postpartum: UI: 50 % vs 76 %* FI: 22 % vs 22 % PFMT in last month: 89 % vs 65 % 24–44 months postpartum: PFMT in last month: 58 % vs 54 %	Loss to follow up: 85/230 (IG: 59/113 CG: 26/117) *Significantly less UI in IG, however due to high drop-out rate in IG, results must be interpreted with caution

Mørkved and Bø (1997) [38]	N = 198 Primi-/multiparae Continent/incontinent	Prospective matched control study 1. Control: customary written postpartum instructions from the hospital. Not discouraged from performing PFM exercises on their own. Correct VPFMC checked at enrolment. 2. Intervention: 8 weeks of intensive pelvic floor muscle training (in a group) led by physiotherapist with additional home exercises. The intervention started 8 weeks postpartum. Correct VPFMC checked.	Self-reported SUI Pad test Urodynamics	16 weeks postpartum: UI: 14 % vs 28 % (p = .015) Pad test: 3 % vs 13 % (p = .009) PFM strength improvement: 5.3 vs 0.8 (p = <.01)	Drop-out IG: 7/99 Percentage performing PFMT between 8 and 16 weeks postpartum: 100 % vs 65 %
Mørkved and Bø (2000) [46] One year follow-up	N = 180		Self-reported UI Pad test PFM strength in women performing PFMT less or more than 3 times per week	12 months postpartum: UI: 17 % vs 38 % (p < .003) Pad test: 3 % vs 13 % (p < .001) Change in PFM strength in women performing PFMT 3 times or more per week: 4.9 (95 % CI: 3.7–6.2) vs. performing PFMT less or more than 3 times per week: 1.8 (95 % CI: 0.8–2.7)	Drop-out: 18 pairs Percentage performing PFMT between 16 weeks and 1 year postpartum: 53 % vs 24 %

(continued)

Table 17.3 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs CG)	Comment
Sleep and Grant (1987) [39]	N = 1800 Continent/incontinent Recruited within 24 h of vaginal delivery	2 arm RCT 1. Control: Current standard antenatal and postnatal care. Recommended to do VPFMC as often as remember and mid stream urine stop. 4 week health diary 2. Intervention: As above plus one individual session daily while in hospital with midwifery co-ordinator. Four weeks health diary including additional section recommending a specific PFMT task each week (all tasks related to integrating VPFMC with usual daily activity).	Self-reported UI	3 months postpartum: UI: 22 % vs 22 %	Drop-out rate: 107/900 vs 84/900 Performing PFMT at 3 months postpartum: 58 % vs 42 %
Anal or faecal incontinence as primary outcome measure					
Peirce et al. (2013) [74]	N = 120 Continent/incontinent Primiparae recruited prior to discharge home from hospital postpartum	2-arm RCT 1. Home PFMT: Standard PFMT 5 min twice daily for 3 months 2. Early EMG biofeedback + PFMT: use of home biofeedback programme (5 s hold, 10 s rest × 10) twice daily and PFMT as for group 1 Participants recorded adherence to PFMT protocol in an training diary	Self-reported FI (Jorge Wexner score) QoL (Fecal Incontinence QoL scale) Anal resting and squeeze pressures (Manometry)	After 3 months of treatment: No differences in incontinence scores, QoL scores or anal resting/squeeze pressures between groups.	Drop-out: 0 Adherence: 7/30 did not use ES as per protocol due to lack of time and absence of FI symptoms. Adherence not reported in BF group.

Mahony et al. (2004) [48]	N = 60 Incontinent Primi-/multiparae recruited 12 weeks following obstetric injury	2-arm RCT, single blind 1. BF: weekly intra-anal biofeedback (alternating slow twitch (hold 5 s, relax 8 s) and fast twitch (3 rapid maximum VPFMC for 5 s, relax 8 s) for 10 min) with physiotherapist for 12 weeks. Daily PFMT home exercises. 2. BF + ES: weekly intra-anal electrical stimulation (35 Hz, 20 % ramp modulation, 5 s on/8 s off for 20 min, intensity eliciting contraction of the external anal sphincter muscle) and intra-anal biofeedback (as for group 1) for 12 weeks	Self-reported AI (Jorge Wexner score) QoL (Fecal Incontinence QoL scale) Mean maximum anal squeeze and resting pressures Endoanal ultrasound	After 12 weeks of treatment: Continent (FI): 8/28 (BF + ES) vs 6/26 (BF) Significant reductions in incontinence scores and QoL scores in both groups after treatment. Significant improvement in anal squeeze pressure and anal squeeze pressure increments in both groups. No change in anal resting pressures. 3 % had a normal endoanal ultrasound scan.	Loss to follow-up: 6/60 (BF + ES: 2; BF: 4) Adherence to PFMT not reported

(continued)

Table 17.3 (continued)

Author	Subjects	Design/intervention	Outcome	Results (IG vs CG)	Comment
Fynes et al. (1999) [49]	N = 40 Incontinent Primi-/multiparae consecutively recruited from perineal clinic following obstetric anal sphincter injury	2-arm RCT 1. Sensory BF: weekly sessions of sensory vaginal biofeedback PFMT (20 short maximum PFMC of 6–8 s hold, 10 s relax and 30 s hold PFMC) + Standard home PFMT 2. Augmented BF: weekly anal electrical stimulation (20 % ramp modulation and 20 Hz 5 s on/8 s off for 10 min followed by 50 Hz, 8 s on/30 rest for 10 min) and EMG biofeedback (PFMC alternating between hold 5 s and rapid maximum squeezes during 5 s and 8 s relax for 15 min) + Standard home PFMT	Self-reported FI PFM strength (manometry)	After 12 weeks of treatment: Median FI: 0 (AugBF) vs 4 (SensBF) ($p < .0001$) Asymptomatic FI: 15/20 (AugBF) vs 7/19 (SensBF) Significant increases in the mean maximum resting and squeeze pressures and squeeze increments in AugBF group. No significant difference in SensBF group Poorer outcome in participants with full thickness defects of the external anal sphincter muscle.	Loss to follow-up: 1/40

Abbreviations: *AI* anal incontinence, *BF* biofeedback, *CI* confidence interval, *EMG* electromyography, *ES* electrical stimulation, *FI* faecal incontinence, *IG* intervention group, *CG* control group, *PFM* pelvic floor muscles, *PFMC* pelvic floor muscle contraction, *PFMT* pelvic floor muscle training, *RCT* randomized controlled trial, *SUI* stress urinary incontinence, *UI* urinary incontinence, *VAS* visual analogue scale, *VPFMC* voluntary pelvic floor muscle contraction

the treatment effect of PFMT in women with UI at inclusion. In the study by Wilson and co-workers (1998) [43], more than half of the women randomized to the intervention groups receiving reinforced PFMT with or without cone weights withdrew before the end of the study, and the study was thus underpowered in order to show any differences in prevalence of AI between groups. The findings in the study by Glazener and co-workers (2001) [42] suggest that PFMT may reduce co-existing AI in patients referred with UI. Further, a significant difference in the number of daily PFM contractions performed was found, favouring the PFMT group [42]. At 6 years postpartum, however, these group differences did not persist, as the number of women who reported performing daily PFM contractions was halved, and the prevalence of FI/AI was similar in the PFMT and the control groups [44].

Two studies have compared the effect of postpartum PFMT in conjunction with other treatment aids such as biofeedback and electrical stimulation in women with AI/FI postpartum [48, 49]. Both studies had a low drop-out rate and the results show an increased ability to perform VPFMC and a reduction of FI/AI symptoms following PFMT [48, 49]. Mahony and co-workers (2004) [48] offered women experiencing AI symptoms postpartum PFMT with biofeedback or PFMT with biofeedback and a standardised electrical stimulation programme for 12 weeks. Due to ethical reasons, the study included no control group. A total of 85 % of participants reported improvements in FI symptoms and 26 % became asymptomatic. Further, participants reported significant improvements in QoL scores. Both groups showed a significant improvement in median PFM squeeze pressure, however, a larger increment was found in the group performing PMFT with biofeedback and no electrical stimulation. Similar results were found by Fynes and co-workers (1999) [49], who compared PFMT and vaginal biofeedback to PFMT and anal biofeedback augmented by a standardised electrical stimulation programme during a 12 week treatment period. Continence scores improved in both treatment groups, however, more participants became asymptomatic in the group receiving PFMT and biofeedback augmented with electrical stimulation and only the women in the augmented biofeedback group had increased resting and squeeze pressures.

Cost of Incontinence

The cost of incontinence-related illness is a substantial economic and human burden, highlighting the need for effective forms of prevention and management [50]. Symptoms of UI or AI before or during pregnancy have been found to be the main risk factors for incontinence symptoms postpartum [51–62]. In a 12 year prospective study, Viktrup and co-workers (2006) [57] reported that women with onset of UI in pregnancy or shortly after their first delivery had increased risk of long-lasting symptoms. Among women who were continent during their first pregnancy and the postpartum period, the prevalence of UI 12 years after the first delivery was 33 % compared to 66 % in women who became incontinent during their first pregnancy and the postpartum period [63]. Similarly, women with AI symptoms in the first year after delivery have an increased risk of long term AI symptoms [64, 65].

Experiencing UI or AI/FI 6 months postpartum has been found to have a negative impact on health related QoL [66], and poorer QoL has been reported among women experiencing severe symptoms or both UI and AI/FI in pregnancy and postpartum [66–68].

Using 1995 estimates, the direct annual cost of UI in women was similar to the annual expenditures of other chronic diseases, and estimated to be $12.4 billion [69]. It has been estimated that the financial burden of incontinence for patients as well as society is relatively large. Furthermore, the financial burden may increase in the future due to an aging population and direct costs related to medical care and treatment, as well as indirect costs related to factors such as the loss of productivity [69–72]. Increasing costs of FI have been shown to be associated with symptom severity and female gender, whereas age was found to be associated with reduced costs among patients experiencing AI for more than 1 year [70]. Among older patients with longstanding FI, loss of productivity in paid and unpaid work accounted for half the estimated total cost of FI [71]. Considering that incontinence symptoms in pregnancy and the first year postpartum have been found to be strongly associated with incontinence symptoms in the long term, identifying women with AI symptoms affecting QoL in the first year postpartum may reduce the long term adverse effect of AI both with regards to personal as well as societal costs.

Criteria for Successful Treatment Outcomes and Recommendations

In the current literature, there are insufficient details on the PFMT protocols in order to assess their potential to improve PFM function and subsequently reduce UI and AI/FI [11]. However, common factors for all trials reporting a positive effect of PFMT in pregnancy or postpartum were found to be thorough clinical assessment of the participants' ability to perform VPFMC, close individual or group follow-up, and high adherence to the exercise protocol [11, 34]. In contrast, trials with little or no effect tended to have an inadequate training dosage, infrequent or no follow up of participants during the intervention period or low adherence to the exercise protocols. Further, considering the spontaneous changes occurring to the PFM during the first 6 months postpartum [73], it may be that the duration of the intervention in some of these studies was insufficient and introduced as well as assessed too early postpartum in order to result in any clinical improvement in PFM strength and function [34, 74].

Furthermore, it is suggested that individual and supervised PFMT interventions may be more successful than group-based interventions in women with severe incontinence symptoms, major levator ani or sphincter ani muscle defects or reduced ability to perform PFM contractions [34]. Nevertheless, the effect of PFMT during pregnancy to prevent UI and AI appears to still be open to question [23, 75, 76]. There is a lack of trials investigating the effect of implementing PFMT in a more general training program for pregnant women. Boyle and co-workers (2012) [11] strongly recommend that all future trials of PFMT during pregnancy or postpartum should collect data on AI as well as UI, and highlight the need for large, pragmatic trials with population-based approaches, using adequate PFMT

intensity. Further, considering the complex and multifactorial pathophysiology of AI/FI in particular, multifactorial interventions have been recommended in future trials on the effect of PFMT on reducing AI/FI symptoms in pregnancy and postpartum aiming at reducing the frequency of AI/FI, improving rectal sensibility and changing stool quality [77].

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Prognosis of Childbirth Trauma

18

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Abstract

An accepted risk factor for pelvic floor disorders is vaginal birth, and its concomitant birth trauma. Much of the effect is mitigated over the life span, with conflicting evidence and unclear causal and mechanistic explanations in the literature of the role of birth trauma. The greatest impact of vaginal birth trauma is on the incidence and prevalence of pelvic organ prolapse [1]. The prognosis of vaginal birth trauma on development or severity of urinary incontinence and anal incontinence is more difficult to determine because of the profound impact that aging has on these conditions. Immediately after birth, little is known about the sequelae of specific trauma other than in the short-term postpartum period. In what follows, we review the literature and aim to elucidate the role of birth trauma, its impact, and prognosis on a parous woman's pelvic floor.

Keywords

Birth injury • Fecal incontinence • Urinary incontinence • Pelvic pain • Perineal injury • Postpartum • Prolapse • Risk factors

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Introduction

The prevalence of pelvic floor disorders (PFD) as well as their surgical management creates a large burden on patients, providers and the health care system in general. Surgical management of PFDs is common, with a lifetime risk of undergoing a surgery for pelvic organ prolapse or incontinence by age 80 being 11.1 % [2]. Projections from United States Census Bureau data indicate that the prevalence of symptomatic PFD will increase by 56 % from 28.1 million to 43.8 million from 2010 to 2050 [3].

An accepted risk factor for PFDs is vaginal birth, and concomitant birth trauma. Pelvic floor disorders at this stressful and exciting time of life offer their own challenges including interruptions of early parenting, frustration with unmet expectations, plus significant time and cost. Within this, it is beneficial to understand the prognosis of common issues and complications that arise within this period in a woman's life. In what follows, we will address the literature regarding prognosis for the issues surrounding birth and birth trauma.

Obstetric Anal Sphincter Injury (Oasis)

Of the spectrum of pelvic floor disorders with the most profound effect on quality of life in the postpartum period, fecal and anal incontinence are arguably most disruptive. Fecal incontinence is the complaint of involuntary loss of solid or liquid feces and anal incontinence includes the complaint of involuntary loss of feces or flatus. What is often overlooked in considering these issues is fecal urgency with or without incontinence, including the sudden compelling desire to defecate that is difficult to defer [4]. The most common cause of these disorders in young women is anal sphincter injury at childbirth. The prevalence of anal incontinence reported in the literature among women with sphincter injuries ranges from 20 to 50 % reporting some sort of anal incontinence symptoms in the near postpartum period [5–7]. Anal incontinence can occur in up to one-third of women with obstetrical sphincter injuries with immediate or delayed onset of symptoms [8]. Because of occult injury, the incidence of anal sphincter damage at the time of vaginal delivery is higher than the number of observed injuries would suggest. Overt anal sphincter injury is relatively rare in women without episiotomy or operative vaginal delivery, with an incidence that ranges from 0 to 6.4 % [8–11]. The incidence of occult anal sphincter laceration identified by ultrasonography, ranges from 6.8 to 44 % in parous women [7, 12]. Additionally, data from a large US population-based study indicated that 29.3 % of postpartum women suffer from fecal incontinence (including flatus) when assessing for immediate postpartum symptoms and one in five of these women had undergone a cesarean delivery [13]. Clearly, this is a multifactorial problem that is prevalent not only with vaginal delivery but also with the cesarean delivery population.

In differentiating fecal or flatal incontinence, a systematic review of comparative studies-with short term follow up, showed that anal incontinence was increased after spontaneous vaginal delivery as compared to cesarean delivery (OR: 1.32;

95 % CI 1.04–1.68). However, the risk of severe anal incontinence was not significantly increased between these two groups, even though there was a trend towards more symptoms in the vaginal delivery group [14]. Likewise, in a longitudinal cohort study of women 5–10 years after their first delivery, there was no significant difference in anal incontinence symptoms in women who had been delivered by cesarean compared to those with spontaneous or instrumented delivery [15].

Episiotomy and operative vaginal delivery increase the incidence of severe pelvic floor trauma, yet were performed in 29 % and 9 % of vaginal births, respectively, in 2001 [16, 17] A meta- analysis of six randomized trials compared restrictive to liberal use of episiotomy in 4,850 women concluded that liberal use of episiotomies conferred no benefit and was associated with other complications [18]. Much of the incidence of OASIS depends upon the type of episiotomy performed. In these cases where mediolateral episiotomies are practiced, the rate of OASIS is 1.7 % in all comers and 2.9 % in primiparous patients [19]. Much higher rates are noted in those instances of midline episiotomy, at rates of 12 % for all comers [20] and 19 % in primiparous patients [21]. Operative vaginal delivery was similarly reviewed in 2,582 women and it was concluded that vacuum delivery was associated with a much lower risk of anal sphincter laceration than delivery with forceps (relative risk: .41; 95 % CI .33 to .50) [22]. Prevention of anal sphincter laceration and subsequent development of anal incontinence partly lies in decreasing the use of these interventions at the time of delivery.

While vaginal birth alone is not clearly a risk for fecal incontinence, OASIS increases the risk of subsequent fecal incontinence. Estimates range from 9 to 28 % [11, 23–25]. Likewise the risk of fecal incontinence is increased when there is a disruption of the internal anal sphincter, as compared to the external anal sphincter alone [26]. Although debilitating in younger life, studies of older women in their 50–60s, seem to eradicate the correlation of birth injury in explaining fecal incontinence. Most convincingly, a study of over 2600 women in their 50s demonstrated no significant difference between the prevalence of fecal incontinence between nulliparous, primiparous and multiparous women. These groups had fecal incontinence rates of 11.3 %, 9.0 %, and 10.4 % respectively. This similarity prevailed among parous women, irrespective of mode of delivery [27]. DeLeeuw et al. reported a retrospective cohort study of 125 matched pairs with median follow-up of 14 years after index delivery. Fecal incontinence was reported in 39 women with sphincter lacerations compared to 16 controls (OR: 3.1; 95 % CI 1.57–6.10) [28]. In an American cohort of sphincter injury patients followed at 6 months, the presence of fecal incontinence was associated with white race, antenatal UI, 4th- versus 3rd-degree sphincter tear, older age at time of delivery and higher BMI. There were no factors associated with fecal incontinence at the 6-month postpartum mark in the vaginal delivery group without OASIS or who had undergone a cesarean delivery [26].

The role of midline versus mediolateral episiotomy has been identified as a possible causal factor in explaining the higher rates of anal and fecal incontinence involved in an American cohort, where episiotomies, when performed are midline versus mediolateral. Careful evaluation of findings and subsequent outcomes need

to assess this mechanism of OASIS versus that which occurs in the setting of mediolateral episiotomy [29].

In counseling patients for outcomes of primary repair, rates of fecal and anal incontinence vary greatly, not surprisingly, based on the variations in repair techniques, as well as the study design and the manner in which data was collected. A recent prospective study of 241 women at their first vaginal delivery, 59 of whom experienced OASIS, with subsequent repair with trained providers showed no fecal incontinence and no difference in flatal incontinence as compared to those women who had not had a sphincter disruption at 4 years postpartum [30]. What is most hopeful about this study, is that when evidence based protocols are established and implemented, not surprisingly, patient outcomes improve, and thus prognostic indicators improve as well.

In counseling women about future route of delivery, it appears that there is only a modest increase in risk for recurrent OASIS. A retrospective review of a large American cohort (n = 658) showed recurrent OASIS in only a small percentage of women at 3.2 %, with operative vaginal delivery and birth weight of ≥4000 g to be associated with recurrent OASIS [31]. This is consistent with a large Swedish cohort which although showed an increase of sixfold in incidence of sphincter rupture, the incidence was only 3 %. It was however somewhat lower than in another large studies (n = 774), which showed a rate that was still quite low at 7.5 % [21].

Incontinence

The role of vaginal birth as it leads to stress urinary incontinence is well established. The case for urgency incontinence is less so. Rates in the initial postpartum period vary, while 21 % of women are known to have urinary incontinence of some type within 10 weeks of delivery (Scheer). Prevalence of stress (SUI) and urgency (UUI) incontinence 5 years after first vaginal delivery has been shown to be 30 % and 15 % respectively with presence of symptoms at 3 months post-partum being predictive of more and longer lasting symptoms [32].

Most studies are short term in their follow-up, but in the observational analysis by Altman et al. women were followed 10 years out from their first delivery. They found that there were significant increases in stress as well as urgency symptoms at 10 years follow up compared with baseline compared with the 10 years preceding delivery. Most of those in the analyzed cohort experienced mild to moderate symptoms, with a five to six time increase in incidence of urinary incontinence episodes from the time of their first vaginal delivery [33]. Surrogates for perineal trauma did not correlate with the presence of incontinence 10 years after the first delivery, nor did repeat vaginal deliveries [33]. In an American cohort 5–10 years after vaginal or cesarean delivery, spontaneous vaginal birth was associated with a significantly greater odds of SUI (OR 2.9; 95 % CI 1.5–5.5) as compared to cesarean without labor [15]. These findings are not dissimilar to the large population study of Rortveit et al. which found 14.7 % of parous women having symptoms of SUI, as compared to 4.7 % of nulliparous women. In this cohort of greater than 15,000 women, a

relative risk of 2.4 for developing SUI was noted, and the number of vaginal deliveries was of limited importance for the outcome as compared to that of the first vaginal delivery [34].

Predictors of postpartum urinary incontinence in several studies include leaking during pregnancy [35, 36] and predictors of incontinence at the 1 year mark, include persistent leakage 4–8 weeks postpartum [37]. Association of other factors with incontinence is worthy of investigation, with findings indicative of OASIS being associated with both pure urge and mixed incontinence. In a large American cohort of 943 women, urge incontinence alone was found in 16.2 % of women and mixed incontinence was found in 14.6 % of women. Stress symptoms were present in 21.3 % of women [38].

Assessments of persistence of urinary incontinence are difficult to obtain. In a longitudinal comparison of women undergoing spontaneous vagina delivery complicated by OASIS, vaginal delivery without OASIS and cesarean delivery without labor of all of the women reporting urinary incontinence at 6 weeks, about 40 % did not report incontinence at 6 months, and about one-third of the urinary incontinence reported at 6 months was not in women who reported incontinence at the 6 week postpartum point [26].

Understanding the significant association is only a first step in the analysis. Further understanding of the structures involved and the mechanism by which the damage occurs can be helpful in assessing pathology and prognosis. In an analysis of primiparous stress incontinent versus continent women at 9–12 months postpartum, maximal urethral closure pressure was 25 % lower in stress incontinent women. In the same analysis comparing primiparous stress continent to nulliparous women, the two groups had similar values [39]. This points to sphincter function as a key component in the continence mechanism and a potential target for therapeutic interventions.

Additionally, primparous women with SUI are twice as likely to have visible LA defects compared to content primiparas [39]. However further analysis of this relationship showed that urethral function measured as a urodynamic variable did not differ in women with and without levator ani muscle injury. This is both frustrating and hopeful. It requires a careful analysis of the continence mechanism of urethral closure pressure and levator ani support, as after birth MUCP change may not necessarily accompany LA change or other anatomical change [40] Second, birth events that injure the LA do not necessarily limit a woman's ability to augment MUCP with a Kegel effort in the postpartum period as well as later in life [40, 41].

Levator Injury

It is well established that vaginal delivery leads to higher rates of levator ani damage particularly involving both the pubovisceral portion of the levator ani muscle [42]. In an assessment of 160 primiparous women, 32 of the 160 were found to have levator ani defects on MRI. These women with muscle defects were more likely to have had an a difficult delivery with an odds ratio of 14.7 for forceps delivery, 8.1 for anal

sphincter rupture, 3.1 for episiotomy [43]. Later in life, these levator defects are more commonly found in women with prolapse (55 %) as compared to normal controls (16 %), leading to an adjusted odds ratio of 7.3 for prolapse in those with a levator ani defect as compared to their counterparts without a muscle defect [1].

The consequences of levator ani injury in the short term are not completely clear other than those with a muscle defect were found to have weaker pelvic floor muscles in the 9–12 month postpartum period as compared to controls [39]. Birth related changes to the levator ani muscles persist in both function and structure with a significant amount of remodeling present in the course of normal postpartum healing. Analysis of the dynamic MRIs of those patients who had experience those factors putting them at risk for levator tear demonstrated that at rest diameters of the urogenital and levator hiatus were smaller on late scans (~7 months postpartum) compared with early scans (~1 month postpartum) by 7.7 and 3.2 mm respectively ($p < .05$) [44]. These findings were independent of the status of the levator muscles in this cohort. It was also demonstrated by Tunn et al., that the at rest locations of the perineal body, levator and urogenital hiatus locations improve greatly from the 1 day and 2 weeks postpartum mark [45]. This points to an aggressive early resolution of postpartum change in position, just in virtue of time, without any intervention. There was, however, no statistically significant difference in the ability to displace structures during Kegel and Valsalva in the comparison of 1 month and 7 month scans, showing that in this group there is little change in function of the muscles from the early to the later postpartum period [44].

In those patient who had undergone a vaginal delivery, pelvic floor muscle strength 6–11 years after vaginal delivery was similarly assessed with a significant reduction in both strength and duration of contraction in those who had undergone either spontaneous or assisted vaginal delivery. Further, among women with at least one vaginal delivery, pelvic muscle strength was lower among the women with a pelvic floor disorder as compared to those without ($p = 0.12$) This finding was additionally associated with the obstetric variables at delivery of macrosomia, perineal laceration, episiotomy, anal sphincter laceration, as well as the number of vaginal deliveries [46]. Further, 5–10 years after vaginal delivery an associate of prolapse to or beyond the hymen was found (OR: 5.6; 95 % CI 2.2–14.7) as compared to cesarean without labor [15].

Low Back and Pelvic Pain

Low back and pelvic pain (LBPP) is often reported during pregnancy and postpartum with rates of up to 70 % reported in the literature [47]. The prognosis as measured by the prevalence of LBPP in the postpartum period varies greatly. In one analysis, even at 3 years after giving birth, up to 20 % of women report that their symptoms have still persisted [48]. Another analysis also found a similar prevalence of LBPP of up to 20 % at 6 years postpartum [49], with serious pain affecting approximately 7 % of women at 18 months postpartum [50]. Clearly this is an often neglected but disabling condition facing women in pregnancy and the postpartum period.

In assessing LBPP postpartum, a large cohort out of Sweden, assessed by telephone survey, demonstrated that the prevalence of persistent LBPP 6 months after delivery was 43 % [51]. In this group, approximately one-third of the subjects reported having a previous experience of low back pain or pelvic pain in life before pregnancy. This indicates a multifactorial etiology of LBPP rather than just pregnancy and delivery alone. This is supported by a follow-up study which indicated that an elective cesarean led to an increased risk of persistent LBPP as compared to the vaginal birth cohort [52]. Additionally, in a large Norwegian study at the 6 month postpartum point, neither planned nor emergent cesarean section was associated with increased prevalence of pelvic girdle syndrome (PGS) (pain in anterior and bilateral posterior pelvis). However, planned cesarean section was associated with severe PGS; with 3.1 % of women undergoing a planned cesarean delivery reporting severe PGS as compared to 1.1 % of women who had an unassisted vaginal delivery [53]. In a large Dutch cohort, prevalence of pelvic girdle pain was 74 % at 30 weeks of pregnancy 48 % between 0 and 6 weeks postpartum and 43 % between 6 and 12 weeks postpartum. Predictors for postpartum pelvic girdle pain included more disability at 6 weeks postpartum, higher mean pain scores at 6 weeks postpartum and even the presence of pelvic girdle pain at 6 weeks postpartum, higher birth weight of the baby and higher somatization during pregnancy and at 6 weeks postpartum, and uncomfortable postures at work. Days of bed rest, even just 1–2 days (more so with 3–4 days) decreased the risk of postpartum pelvic girdle pain as compared to no days of bed rest after delivery [54].

For prognosis and improvement, appropriate recognition and treatment is key. In studies by Norén et al., it was found that although the prevalence of residual back pain at 3 years postpartum was 20 %, on exam the pain itself was found to consist of lumbar back pain, posterior pelvic pain and a combination of both. Not surprisingly, findings of disability, muscle endurance and increasing difficulty with exercise was worse in those with a combination of lumbar back pain and posterior pelvic pain. This analysis, rather than accepting "back pain" as a single type of pain, identifies two separate phenomen: lumbar back pain and posterior pelvic pain [49]. Findings such as these and other studies that show that LBPP persists postpartum points to a mechanism different from that often understood as merely joint-related and one in fact more likely to be muscle-related. This is hopeful in that close follow-up with an intervention of physical therapy after delivery can help in rehabilitating and re-training muscles that may have been compromised during pregnancy, and thereby help to avoid serious long-term impairment [49].

Sexual Function

It is well known that the prevalence of sexual dysfunction is high in the female population. More so, there is a large body of epidemiologic data describing short term post-partum sexual dysfunction. Like any postpartum dysfunction, sexual function is a manifestation of multiple factors, including the new parenting responsibility, sleep deprivation, adjustment of the family members, hormonal changes and not least of all pre-pregnancy sexual function and intimacy.

One component of sexual dysfunction in the postpartum period is pain with intercourse. Several studies have pointed to the consequence of worsening perineal trauma in the form of assisted vaginal delivery as a predictor of increased sexual pain postpartum. A large cross-sectional study out of Australia, using mail surveys at 6–7 months postpartum showed a nearly five-fold increased risk of perineal pain, and a two-fold risk of sexual problems with vacuum or forceps delivery as compared to spontaneous vaginal delivery (OR 4.69; 95 % CI 3.2–6.8 and OR 2.06; 95 % CI 1.4–3.0, respectively). This was the case even after controlling for the duration of labor, infant birth weight and degree of perineal trauma [55]. An American cohort had similar findings with no resumption of sexual intercourse at 7 weeks postpartum in those undergoing an assisted delivery. This same group had also endorsed that the delivery had adversely affected their experience of sexual activity as compared to those who had undergone spontaneous vaginal delivery [56].

In light of the prevalence of sexual dysfunction, it is important to keep in mind that nonetheless resumption of sexual activity after delivery occurs relatively soon after the traditional interval of "vaginal rest." Data indicates that approximately half of women resume sexual activity by 5–6 weeks postpartum [57]. A somewhat lower number of 40 % of women reported being sexually active at the 7 week postpartum point. 241 patients were included in this prospective analysis, 98 of whom underwent episiotomy (mediolateral). In this cohort, being sexually active was not affected by the type or degree of perineal trauma that occurred with delivery [58]. At the 6-month postpartum point, an American cohort of over 500 women reported 94 % having resumed sexual activity. This cohort demonstrated slightly lower rates in those having undergone OASIS (88 %) and cesarean delivery (86 %) [59].

Most frustrating in any analysis of postpartum sexual function is the lack of attention it garners. In a London teaching hospital with a large obstetrics unit, only 15 % of women with a sexual problem post-partum felt comfortable raising these issues with their health care provider and only 18 % of all obstetric patients reported receiving information about changes in sexual function post-partum. Although as reported, a vast majority of women have resumed intercourse at the 3 month postpartum mark, analysis of a different cohort demonstrated that at the 2 month postpartum mark, 55 % of women experienced painful penetration and 45 % experienced painful intercourse [60]. Determining who these patients will be is more complicated than merely screening those who in their delivery experienced perineal trauma or assisted delivery. Clearly, pre-delivery sexual function plays a role as lack of satisfaction with one's relationship at the 1 year postpartum mark was predicted by not being sexually active at 12 weeks of pregnancy [61].

As with many postpartum issues, longer term analysis is somewhat confounding. Studies of identical twins demonstrated that those who were sexually active were more likely to be premenopausal and multiparous as compared to their opposite counterparts. However, beyond that, nulliparous women who were sexually active reported superior sexual satisfaction scores compared with parous women, regardless of age and mode of delivery of their parous counterparts [61]. Another population based study of a cohort 40 years old or older demonstrated no significant associations between parity or mode of delivery and the outcomes of low sexual

desire, less than monthly sexual activity, or low overall sexual satisfaction. This was the case with the exception that those who had undergone operative vaginal delivery were more likely to report low sexual desire (OR-1.38; 95 % CI 1.04–1.83) [62]. These studies point to some resolution or at least an adaptation to the short term postpartum effects on sexual function. In the setting of a paucity of intervention based versus observational research on the consequences of parity, birth and birth trauma on sexual function and the high prevalence of sexual dysfunction in women, we would be well served to screen women during their pregnancy and in the postpartum period.

Conclusion

In general, medicine has a limited ability to determine prognosis in complicated multifactorial cases. Nowhere is this more apparent than in predicting prognosis of birth-related injury and trauma. There is a clearly a large need for well-designed randomized controlled trials for interventions in the postpartum period. Much of this work is likely to come from the well-established postpartum perineal clinics.

In light of the prevalence of disorders, there needs to be an emphasis on screening new mothers and newly delivered multiparous women for the consequences and signs of the various manifestations of post-partum sexual dysfunction and pelvic floor disorders.

In summary, when determining the prognosis of women with pelvic floor trauma, we can make some generalizations. Women that develop urinary incontinence during pregnancy are more likely to suffer urinary incontinence after delivery. If she continues to leak urine after 3 months, she may improve, but is likely to have some persistent symptoms. Anal incontinence immediately after delivery is common with a sphincter laceration, but in the vast majority of cases will resolve. As the woman ages, she may be at increased risk for developing FI, but the data is unclear. Levator ani muscle tears are associated with pelvic organ prolapse, UI and FI. The immediate impact of the muscle tears are not well studied. There are strong associations with LA tears and pelvic organ prolapse as the women age. But again not all women with LA tears have clinically relevant prolapse and we do not know the prognostic factors to determine which women will go on to develop problems later in life. Sexual function within the first 6 months of delivery is often painful for women, especially if there has been a perineal laceration or sphincter tear. Fortunately most discomfort will resolve by a year.

Some women are no doubt innately more prone to the development of pelvic floor disorders based on genetic factors, body weight and muscle mass, levels of physical activity etc. A birth injury in one woman may lead to devastating consequences, while another woman with the same injury may heal and suffer no symptoms. Our challenge now is to determine which women will suffer the injuries, what are the modifiable risk factors, and how can we stop the progression of disease and symptoms. Without a clear understanding, we are limited in describing associations, and our counseling of patients is thereby limited as well.

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