ANAL SPHINCTER INJURY AT VAGINAL DELIVERY:

RISK FACTORS AND LONG-TERM CLINICAL CONSEQUENCES

BESCHADIGING VAN DE ANALE SFINCTERS TIJDENS DE VAGINALE BARING:

RISICOFACTOREN EN KLINISCHE GEVOLGEN OP LANGE TERMIJN

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"There are in fact two things, science and opinion;
the former begets knowledge, the latter ignorance."

Hippocrates

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Chapter 1

GENERAL INTRODUCTION

Anal sphincter injury is a known but relatively uncommon complication of vaginal delivery, with a frequency of occurrence of 0.5 – 3% of deliveries in most reports from European countries but a much higher rate of occurrence, of up to 25% of vaginal deliveries, in reports from the United States.[1,2]

Knowledge of the development, anatomy and physiology of the anal canal is a prerequisite to understand the consequences of anal sphincter damage at delivery. In gynecological and obstetrical textbooks these subjects and the technique of primary repair of anal sphincter injury or the management of its complications are often omitted.[3-5]

During the last decades anatomical concepts have changed, and with it, the understanding of the applied physiology.[6] Study of the recent literature on the current knowledge of the anatomy of the anal canal, and its role in the process of normal continence and defecation is therefore necessary.

Reports on the prevalence of fecal incontinence have shown that the problem is frequently underreported because men and women are reluctant to admit that they suffer from this condition.[7,8] In the surgical literature, reviews on the subject of fecal incontinence indicate that traumatic vaginal delivery, especially when complicated by anal sphincter injury, is an important cause of fecal incontinence in women.[9,10]

Until the late 1980's the diagnostic evaluation of patients with fecal incontinence included digital rectal examination, anal manometry, measurements of pudendal terminal motor latencies and, in some cases, EMG-studies of the anal sphincter complex. Many patients were diagnosed as suffering from idiopathic or neurogenic fecal incontinence when structural

lesions could not be demonstrated. The introduction of anal endosonography, some ten years ago, made it possible to reliably demonstrate anal sphincter defects.[11-13] Using this technique it became clear that many patients supposed to suffer from idiopathic or neurogenic incontinence were in fact incontinent for feces because of structural damage of the anal sphincter complex.[14,15] Persisting sphincter defects were now perceived as the primary cause of fecal incontinence following vaginal delivery complicated by recognized and surgically repaired as well as by clinically unrecognized anal sphincter injury.[16-18] The increasing awareness of the relationship between anal sphincter injury at delivery and subsequent anorectal complaints such as fecal incontinence, urgency or soiling, led to multiple case-control studies on this subject. However, the majority of these studies are flawed by small numbers of patients,[17,19-22] lack of control groups,[18,22-24] or an insufficient period of follow-up.[17,19,20,24] Only two studies were published, with contradictory results, in which the relationship between anal sphincter injury and anorectal complaints more than ten years after delivery was addressed,[21,25] and only one of these reports contains a sufficient number of patients to allow reliable conclusions.[25]

In two studies, with only one performed more than one year after delivery, the relationship was addressed between anorectal complaints after anal sphincter injury at delivery and persisting defects or decreased functioning of the anal sphincter complex.[17,18]

Further research, with an adequate period of follow-up and a sufficient number of patients, is therefore mandatory to obtain reliable information on the relationship of anal sphincter injury at delivery and subsequent anorectal complaints and to assess the relationship of anorectal complaints with persisting sphincter defects and decreased functioning of the anal sphincter complex, long after anal sphincter injury has occurred.

In the past most of the knowledge on the relationship between obstetric interventions and anal sphincter injury was derived from case-control studies with their inherent problem of possible selection bias.[26-32] Few randomized controlled trials have been performed in which the relationship between obstetric interventions and anal sphincter injury was studied.[33-35] The results of some these trials were limited by a lack of statistical power,[33] or by the design of the trial protocol that made it difficult to apply the results in daily practice.[34]

In the Netherlands, the existence of the Dutch Perinatal Database (LVR) allows population-based studies on a variety of clinical variables associated with pregnancy, labor and delivery, which limits the possibility of selection bias.[36,37] Considering the drawbacks of the published studies on risk factors for anal sphincter injury at delivery, research on this subject using population-based data may clarify the causal role of various obstetric characteristics and interventions in the occurrence of anal sphincter injury at vaginal delivery.

Based on the considerations presented above, the objectives of this thesis are:

- to analyze the literature concerning the embryonic development and anatomy of the anal canal and anal sphincter complex, and the role of the anal sphincter muscles in the physiology of defecation and fecal continence.
- to assess risk factors for the occurrence of anal sphincter injury at vaginal delivery, using data obtained from the Dutch Perinatal Database (LVR).
- to investigate the causative role of anal sphincter injury at vaginal delivery in the development of anorectal complaints and urinary incontinence, and to identify obstetric risk factors associated with subsequent fecal incontinence.

- to investigate the relationship of anal endosonography and manometry with anorectal complaints in the evaluation of women, long after vaginal delivery complicated by anal sphincter injury.

The studies related to these objectives are described in chapters 2 to 5 of this thesis and followed by a general discussion and conclusions.

Chapter 2

THE FEMALE ANAL CANAL: EMBRYOLOGY, ANATOMY, AND ROLE IN FECAL CONTINENCE AND DEFECATION

2.1 Introduction

Traumatic vaginal delivery complicated by anal sphincter injury constitutes a major risk factor for fecal incontinence in women.[16,38,39] For an exact understanding of the relationship between anal sphincter injury and the development of fecal incontinence, knowledge of the development, morphology and function of the anal canal is essential.[40] In this review the embryonic development, anatomy and physiology of the anal canal are described. Emphasis is placed on the development of modern views on the anatomy of the sphincter complex. In the past 40 years concepts changed and the understanding of the applied physiology changed with them.[6]

2.2 Embryology of the anal canal

The anal canal is derived from two embryonic structures: the hindgut (one of the three parts of the primitive gut) and the proctodeum (anal pit).[41] The terminal portion of the hindgut, the cloaca, is an endoderm-lined cavity in contact with the surface ectoderm. The area of contact, the cloacal membrane, is composed of the endoderm of the cloaca and the ectoderm of the proctodeum.

The cloaca receives the allantois ventrally and the mesonephric ducts laterally, and is divided by a wedge of mesenchym, the urorectal septum, which develops in the angle between the

allantois and the hindgut. As the septum grows caudally towards the cloacal membrane, it develops extensions that produce inward folds into the lateral walls of the cloaca. When these folds fuse, the cloaca is divided into two parts: the rectum and the upper part of the anal canal dorsally, and the urogenital sinus ventrally. The urorectal septum fuses with the cloacal membrane which is then divided into a dorsal anal membrane and a central urogenital membrane. The area of fusion of the anorectal septum and cloacal membrane becomes the central perineal tendon or perineal body.

The urorectal septum divides the cloacal sphincter into two parts. The posterior part develops into the external anal sphincter , whereas the anterior part becomes the superficial transversal perineal muscle, the bulbocavernosus and ischiocavernosus muscles, and the urogenital diaphragm. Around the anal membrane mesenchymal proliferations elevate the surface ectoderm, forming a shallow pit: the proctodeum. The anal membrane is now located at the bottom of the pit and usually ruptures at the end of the eighth embryonic week, establishing the anal canal. This brings the caudal part of the digestive tract into communication with the amniotic cavity

As the superior part of the anal canal is derived from the hindgut, the epithelium at this level is derived from the endoderm of the hindgut. The epithelium of the inferior part of the anal canal is derived from the ectoderm of the proctodeum. The junction of these two types of epithelium is indicated by the pectinate line, the approximate former site of the anal membrane.

2.3 Anatomy of the anal canal

2.3.1 General description

There is an ongoing discussion between anatomists and clinicians about the upper margins of the anal canal.[42] According to the surgical or clinical definition, the anal canal begins where the lower end of the ampulla of the rectum suddenly narrows, passing downwards and backwards to end at the anus. In contrast to this surgical definition, many anatomists and embryologists state that the pectinate line should be used to distinguish the junction of rectum and anal canal, on the basis of the embryonic development.[40,43] Clinically, the pectinate line can be recognized by the anal valves that are situated at this level. Malignant lesions of the epithelium differ in character depending on the site of origin, that is above or below the pectinate line. Because the nervous, venous and lymphatic supply differ for the parts above and below the pectinate line, the surgical management of a disease process is influenced by these differences. This means that the term anorectal junction, which is in widespread use, has no meaning unless it is defined whether the anatomical or the clinical definition of the upper margin of the anal canal is used.

The anterior wall of the anal canal is slightly shorter than the posterior wall. Posteriorly lies a mass of fibrous and muscular tissue, the anococcygeal ligament. In the female, the anal canal is separated on the anterior side from the membranous part of the urethra by the perineal body and the distal part of the vagina. Laterally, it is related to the ischiorectal fossa. Over its whole length it is surrounded by sphincter muscles which normally keep the canal closed.

Based on the surgical definition, the anal canal in the adult is about 4 cm long, measured from the anorectal ring. If, however, the pectinate line is used as the upper landmark, the anal canal

may be little more than 1.3 cm to 2 cm long.[45] The lower margin of the anal canal is also difficult to define. The sloping transition between the moist, hairless and almost glandless lining of the anal canal and the dry peri-anal skin with its appendices is called the anal verge.

2.3.2 Epithelium

The type of epithelium differs depending on the level in the anal canal. The upper half of the canal is lined with mucous epithelium that is plum-colored because of the blood in the underlying internal venous plexus. The epithelium in this region shows interindividual variation; in some individuals it is of the stratified columnar type, in others it is stratified squamous with patches of columnar epithelium, together with stratified polyhedral cells and a single layer of simple columnar epithelium as in the rectum. In this part of the anal canal permanent longitudinal folds in the epithelium, the anal columns, can be recognized.[42,44,46] Each column contains a terminal radicle of the superior rectal artery and vein that form the anal cushions. Enlargement of the venous radicles may cause primary internal hemorrhoids. The lower ends of the columns are joined by small valve-like folds of mucous membrane, the anal valves, situated along the pectinate line . Above each of the anal valves lies a small recess or anal sinus.

The transitional zone, or pecten, extends for about 15 to 20 mm below the anal valves.[44] Its epithelium is stratified and of intermediate thickness as a transition between the epithelium of the upper part of the anal canal and the skin below. This transitional zone lies over the internal rectal venous plexus and is shiny and bluish in appearance. The transitional zone ends in a narrow wavy zone, commonly called the white line (of Hilton). It is situated at the level of the

interval between the subcutaneous part of the external sphincter and the lower border of the internal anal sphincter. A slight groove can sometimes be recognized at this level, the anal intersphincteric groove. Over a distance of approximately 8 mm below the white line the anal canal is lined by true skin containing sweat glands and sebaceous glands.

2.3.3. Arterial, venous and lymphatic supply, and innervation

The arterial blood supply of the anal canal depends on two different systems. The superior part of the anal canal and the mucosa are supplied by the superior rectal artery, a continuation of the inferior mesenteric artery.[40] The inferior part of the anal canal is supplied by the inferior rectal arteries. These arteries originate from the internal pudendal artery in the pudendal canal and traverse the obturator fascia and ischiorectal fossa. Some branches penetrate the external and internal sphincters, others reach the submucosa and subcutaneous tissues of the anal canal.[43,46] The relevance of the middle rectal artery differs individually, mainly depending on the size of the superior rectal artery.[42] It may be absent in 40% of individuals, but it may also have a double or triple presence on one or both sides.[46] When present it gives off branches to the posterior surface of the anal canal.[40]

The venous drainage of the anal canal depends largely on the inferior and middle rectal veins that terminate in the internal iliac vein.[40,42] The inferior rectal veins drain the external rectal venous plexus, situated subcutaneously around the anal canal below the pectinate line. This plexus drains the inferior part of the anal canal and forms external hemorrhoids when dilated. The internal rectal plexus is situated submucosally around the upper part of the anal canal and

the rectal ampulla and empties into the middle rectal vein. Internal hemorrhoids originate from this plexus. The perimuscular plexus, situated more laterally around the upper part of the anal canal and the rectum, receives venous blood from the sphincteric system and part of it flows into the middle rectal vein. The other part of the perimuscular plexus and upper parts of the internal rectal plexus drain into the superior rectal vein that is connected to the inferior mesenteric vein.[40,44] The three venous plexus have extensive communications and may form a portacaval anastomosis.[40,44,46]

The lymphatic drainage of the anal canal depends on the level of the anal canal, above or below the pectinate line.[42,43] Above this level the lymph flows into the middle rectal lymph nodes, connected to the inferior mesenteric and internal iliac nodes. Below the level of the pectinate line the outflow of lymph takes place through the peri-anal and superficial inguinal nodes. In women, lymphatic drainage of the anorectum to the pouch of Douglas, the posterior vaginal wall and the internal genitalia has been described.[42]

According to the embryonic origin of the anal canal, the innervation of the lower part was thought to depend on the somatic inferior rectal nerves and the upper part on innervation by autonomic nerves.[40,47] However, recent experimental work has shown that the motoric innervation of the internal sphincter depends on sympathetic (L5) and parasympathetic nerves (S2, S3,and S4).[42,48] These nerves follow the inferior mesenteric and superior rectal arteries to reach the anal canal. On the basis of cadaver studies, the innervation of the puborectalis muscle was previously thought to be derived from the inferior rectal and perineal branches of the pudendal nerve, or from direct branches of S4 on the perineal side of the levator ani muscle.[48,49] It is now concluded from more recent experimental work that the innervation of the puborectalis muscle depends mainly on nerve branches running on the inner surface of the

levator ani muscles, split off from their mother trunk proximal from the sacral plexus.[48,49] The innervation of the external anal sphincter depends largely on the inferior rectal branch of the pudendal nerve.[42] The motor fibers of this nerve derive mainly from the second sacral nerve, with large interindividual variation.[49] Innervation is also supplied by the perineal branch of the pudendal nerve with its main contribution from S4.

2.3.4 Anal musculature

There is a distinct difference between the muscles surrounding the rectum and those surrounding the anal canal. The smooth musculature of the wall of the rectum consists of two layers: an inner circular and outer longitudinal layer. As the bowel penetrates the pelvic floor striated muscle is added to the smooth muscle and from this point downwards it is also surrounded by sphincteric striated muscle.
In the musculature surrounding the anal canal three layers can be recognized: 1: The internal anal sphincter. 2: The conjoined longitudinal muscle. 3: The external anal sphincter

Internal anal sphincter

Below the level of the pelvic floor the circular musculature of the rectum gradually thickens and ends just above the level of the anal verge. This thickening of about 2.5 mm is known as the internal anal sphincter. Thus, this sphincter represents an increased development of the circular smooth muscle of the gut. Its upper boundary is difficult to distinguish, but it is usually defined at the level of the pelvic floor. For that reason the internal anal sphincter

surrounds the anal canal for about 25 to 30 mm. Its lower border lies 8 to 12 mm below the level of the pectinate line, where it can be palpated as the intersphincteric groove.[42,46]

Conjoined longitudinal muscle

At the level of the pelvic floor the teniae of the large bowel have disappeared and the longitudinal muscle is arranged in an even layer around the circular muscle. At the level of the anorectal junction the longitudinal muscle blends with downwards-oriented fibers of the pubococcygeal muscle (part of the levator ani muscles) to form a conjoined longitudinal muscle. The striated fibers of the pubococcygeal muscle fade out and only few travel distally to the level of the pectinate line.

Classically, the longitudinal muscle is described as a thin, relatively unimportant structure without a well-established function.[42,44,46] However, a recent cadaver study indicates that the longitudinal muscle is much more well-developed than previously thought, and may be as thick as the external sphincter.[50]

At the level of the white line the longitudinal muscle splits into fibro-elastic bundles that spread out. According to classic descriptions these bundles pass mainly through the subcutaneous part of the external sphincter to become attached to the corium of the skin around the anus, the corrugator cutis ani muscles.[44-46] However, recent studies using an endo-anal MRI-coil, and cadaver studies, show that the spreading bundles of longitudinal muscle end in the most distal part of the external sphincter.[50,51] The longitudinal muscle sends some fibers towards the lining of the anal canal, called the musculus mucosae ani.[45,46] These fibers descend through the internal sphincter, eventually join fibers directed upward from below the

subcutaneous part of the external sphincter, and blend with the muscularis mucosae of the anal canal.

External anal sphincter

The anatomy of the external sphincter and puborectalis muscle is still debated. In the conventional description of the external anal sphincter, three parts are distinguished, a subcutaneous, a superficial and a deep portion, the latter being intimately related to the puborectalis muscle.[6,44]

The subcutaneous part is classically regarded as a multifascicular ring of muscle without distinct ventral or dorsal attachments, lying inferior and lateral to the internal anal sphincter, split by bundles of the conjoined longitudinal muscle. The superficial part is described as an elliptical muscle slightly above and medially of the subcutaneous part of the external sphincter. It consists mainly of anteroposteriorly directed fibers that pass from the central tendineous point of the perineum to the anococcygeal raphe attached to the coccyx. The deep portion of the external sphincter is closely related to the puborectalis muscle. There is general agreement that its fibers usually fail to make contact with the coccyx, but they intersect on the anterior side and blend with the deep part of the transverse perineal muscle.[45]

Since the 1950's concepts with a subdivision in two parts, or no subdivision at all, came in favor. Oh and Kark describe an external anal sphincter consisting of a superficial and a deep part. In their view, the superficial part consists of the subcutaneous and the superficial part in the old concept with three parts, the deep part combines the deep portion of the sphincter of the three part concept with the puborectalis muscle.[52] Other investigators describe the

external sphincter as a single continuous mass, explaining the subdivisions as a result of thorough dissection.[6,46,53] These studies consider the puborectalis muscle to be part of the levator ani, and they were recently supported by studies using magnetic resonance imaging with an endo-anal coil.[51] (Figure 2.1)

2.4 Physiology of fecal continence and defecation

Fecal continence is the ability to be aware of rectal contents, to retain them and to excrete them at a convenient moment. This ability is a complex of inborn and acquired reflexes by which the anal canal can be kept closed. However, the ability to maintain continence does not

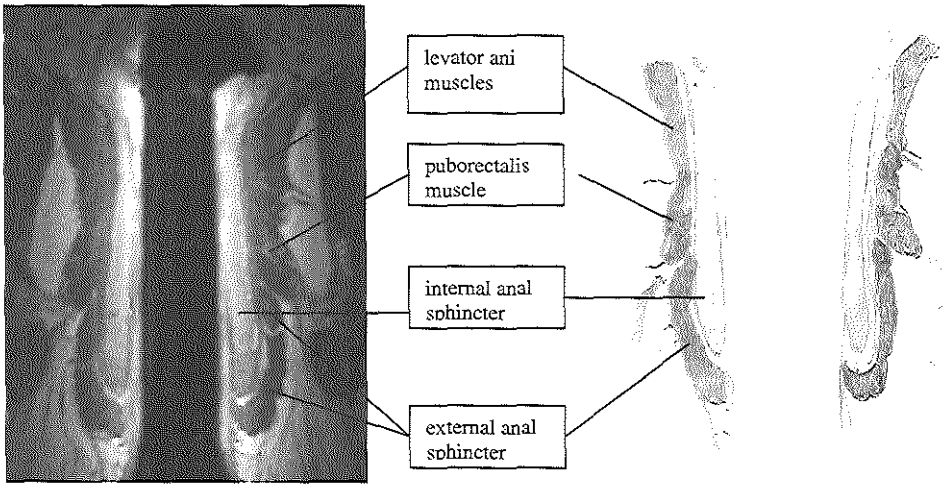


Figure 2.1: *Coronal view of the anal sphincter musculature with endo-anal MRI and schematic drawing (MRI-picture by courtesy of J. Stoker MD, PhD, Dept. of Radiology, Academic Medical Centre, Amsterdam)*

entirely depend on these reflexes. The entire colon, its contents and the special anatomical relationship between rectum and anal canal also contribute to fecal continence.

The colon works in an intermittent manner by low pressure activity. This type of activity, called segmentation, occurs over short lengths of the colon, and is responsible for kneading and turning over of the fecal contents. If the colon passes loose stools to the rectum in an uncontrolled way, normal anorectal physiological mechanisms may not be able to guarantee continence.[54]

In the resting state the anal canal is kept closed mainly by the internal anal sphincter, which accounts for about 55% of the resting pressure.[42,55,56] Other contributors to the resting pressure are the anal cushions and the external sphincter. The anal cushions add to the sealing of the anal canal by vascular distension, and may contribute 15-20% of the anal resting pressure. [54] In the resting state, the external anal sphincter maintains a continuous unconscious resting muscle tone, and its contribution to the resting pressure is estimated to account for 25 to almost 50%.[42,54]

Another factor in maintaining continence in the resting state is the reservoir capacity of the rectum. The rectum can often tolerate more than 300 ml before a feeling of fullness develops that may cause an urgent desire to defecate. Rectal distension causes regular rectal contractions, with rising rectal pressure with each contraction. In the case of low compliance, increased frequency and increased urgency of defecation may develop.[42,54]

The special anatomy of the anorectum may also contribute to maintaining continence. Parks has suggested the presence of a flap-valve mechanism of the rectum and anal canal.[57] In normal conditions, an almost right angle exists between the lower rectum and the anal canal. The angle depends on the puborectalis muscle. When the intra-abdominal pressure rises, e.g. because of coughing, the forces are transferred to the anterior rectal wall which is pushed in

caudal direction onto the top of the anal canal. According to this theory, continence is maintained by occlusion of the top of the anal canal by the anterior rectal mucosa.[55,57] Other investigators have failed to demonstrate this phenomenon.[42] Increased activity of the puborectalis muscle may also contribute to fecal continence by further sphincteric occlusion of the anal canal.[42]

With increasing filling and distension of the rectum, the upper part of the anal canal opens because of relaxation of the internal anal sphincter. This relaxation or inhibition reflex of the internal sphincter is a locally mediated intramural reflex that is not affected by denervation. The reflex can be tested by rapid inflation and deflation of a rectal balloon.[42,54,55] When the upper part of the anal canal opens, rectal contents get into contact with the sensitive epithelium of the upper part of the anal canal that is capable of discriminating flatus from feces. This "sampling mechanism" occurs in continent subjects up to seven times per hour.[54] If desired, the external anal sphincter responds with recruitment of muscle activity in the distal anal canal, thus maintaining the high pressure zone in this region and continence if desired. When rectal filling increases another mechanism is recruited to maintain continence. Mediated by stretch receptors in the levator ani muscle, the muscle tone in the external sphincter increases. At first this inflation reflex occurs involuntarily and without the individual noticing, but when rectal filling increases further the individual will become aware of the higher pressure in the rectum. Active contraction of the striated muscle complex will prevent loss of feces.[55] However, the voluntary contraction can only be maintained for 40 to 60 seconds. As rectal peristaltic waves last less than a minute, with the peak of each wave lasting only a few seconds, this period of increased intraluminal pressure is sustained long enough to maintain continence.[42] At a convenient moment and place relaxation of the anal

anal is allowed, intrarectal pressure will exceed the anal canal pressure, and defecation will occur.

In the squatting position, the axis of the rectum and the anal canal is straightened, allowing alignment of the forces permitting rectal evacuation. At the same time , the tonic activity of the pelvic floor and puborectalis muscle is inhibited, leading to a descent of the pelvic floor and further straightening of the anorectal angle.[42,55] Passage of rectal contents will result in prolonged internal sphincter inhibition and a fall in upper anal canal pressure. The tonic external anal sphincter activity is also inhibited, which leads to a further decrease in anal canal pressure. A rise in intra-abdominal pressure will then result in increased intrarectal pressures that lead to expulsion of the fecal bolus. Whether rectal motor activity is of any significance in defecation is still unclear. Most experts feel that defecation depends mainly on abdominal straining and ascribe minimal importance to rectal contraction.[55]

Many aspects of the mechanism of defecation are still poorly understood. This appears to be due to a large extent to the unphysiological circumstances in which individuals were investigated in many studies.[55] Further research under physiological conditions is needed for a better understanding of the mechanism of defecation.

Chapter 3

RISK FACTORS FOR ANAL SPHINCTER INJURY AT VAGINALDELIVERY*

3.1 Introduction

Traumatic vaginal delivery is considered the most important risk factor for fecal incontinence in women.[38] Fecal incontinence may happen after recognized anal sphincter injury, but can also occur after apparently non-traumatic vaginal delivery.[14,16-18,25,58,59] Studies using endo-anal ultrasonography have shown that fecal incontinence is mainly caused by persisting sphincter defects and not, as was previously believed, by neurological damage.[14,17,18,59] After third and fourth degree perineal ruptures, up to 85% of women have persistent sphincter defects and up to 50% have anorectal complaints, despite apparently adequate repair.[17,18,25,58,59] Therefore, assessment of risk factors for the occurrence of third and fourth degree perineal ruptures is essential, in order to allow primary prevention.
Randomized trials showed no prophylactic effect of the routine use of episiotomy.[33,34] Previous case-controlled studies on risk factors and putative preventive interventions concerned only small groups of women or groups with a small number of anal sphincter injury, which may limit the significance of the results.[29,30] Other studies dealt with risk factors for anal sphincter injury in particular clinical conditions, such as instrumental compared with spontaneous vaginal delivery.[28-32,36]

* *The main substance of this chapter was published in: de Leeuw JW, Struijk PC, Vierhout ME, Wallenburg HCS. Risk factors for third degree perineal ruptures during delivery. Br J Obstet Gynaecol 2001;108:383-87.*

The existence of the Dutch National Obstetric Database (LVR) allows population-based studies on a variety of clinical variables associated with pregnancy, labor and delivery.[36,37] The present study was designed to analyze risk factors for the occurrence of anal sphincter injury using the LVR database.

3.2 Methods

Study population

In the Netherlands, the independent midwife and general practitioner are responsible for providing primary obstetric care of healthy pregnant women and for identifying pathology during pregnancy or delivery. If risks or pathology are identified, the obstetrician/gynecologist is consulted and the patient may be referred to secondary care, if considered necessary.

Deliveries performed in primary and secondary care are registered separately in the LVR. All deliveries beyond 16 weeks gestation, including stillbirths or terminations of pregnancy remote from term, are entered into the database on a voluntary basis. The validity of the data is assessed by the Stichting Informatiecentrum voor de Gezondheidszorg (SIG; Dutch Centre for Health Care Information) using a plausibility program based on obstetric knowledge. For our study we combined both parts of the database to make the population comparable to populations in other countries. In 1994 and 1995, the years included in this study, 82.5% of all deliveries in the Netherlands were recorded in the LVR. The study was approved by the Privacy Committee of the SIG, according to the LVR privacy regulations.

Data collection

The total LVR database of 1994 and 1995 contained 321,726 deliveries, 125,851 (39.1%) in primary care and 195,875 (60.9%) in secondary care. All 32,148 (10.0%) deliveries by cesarean section were excluded, after which 289,578 vaginal deliveries remained. Of those, 829 (0.26%) were excluded because of incomplete data, and 3,966 (1.23%) were excluded because of obvious erroneous data, e.g. birthweight of less than 100 grams, term vaginal delivery with transverse lie, negative duration of second stage, second stage duration of more than 3 hours. The remaining database with complete data contained 284 783 deliveries, with 238 503 spontaneous and 46280 assisted vaginal deliveries. Of all deliveries characteristics of pregnancy and labor such as parity, induction of labor, duration of second stage, interventions during delivery and fetal characteristics were analyzed for risk factors. In case of multifetal pregnancies, the characteristics of the first infant were used for analysis, because the passage of the first baby was thought to carry the largest risk for damage to the birth canal. Anal sphincter injury was defined as any perineal rupture involving the anal sphincter muscles, with or without rupture of the anal mucosa, i.e. third or fourth degree perineal tears.

Statistical analysis

We calculated incidences of third and fourth degree perineal ruptures for each potential risk factor, known from previous studies on this subject and available in the LVR-database. Where possible, factors were grouped: parity, fetal presentation, episiotomy, induction of labor and assisted vaginal delivery. The incidence of third or fourth degree ruptures for each risk factor was compared with the incidence in the most frequently occurring physiological condition in

each group, e.g. occipitoposterior versus occipito-anterior presentation or no episiotomy versus mediolateral episiotomy. We have expressed this as the relative risk of the occurrence of third or fourth degree ruptures for these specific risk factors. Adjusted odds ratios (OR) with 95%-confidence intervals (CI) were calculated for all factors, by modelling the data to control for possible confounding variables, using multiple logistic regression analysis. SPSS for Windows version 7.0 (SPSS Inc., Chicago, Il) was used for the statistical calculations.

3.3 Results

The overall risk of third and fourth degree perineal ruptures in the study group was 1.94% (5528/284,783). The various risk factors analyzed and their association with third and fourth degree perineal tears are summarized in Table 3.1.

Primiparity was found to be significantly associated with an increased risk of third and fourth degree perineal ruptures. Higher parity appeared to be a protecting factor for anal sphincter injury; the odds halved for each following delivery, up to a maximum of six (OR: 0.52, 95%-CI: 0.50-0.55). Fetal occipitoposterior position increased the risk of third degree ruptures significantly. Breech presentation was associated with fewer sphincter injuries than occipito-anterior position, but after regression analysis the association disappeared. Separate analysis of complete and incomplete breech showed no relationship with anal sphincter injury. Other presentations, e.g. brow or face presentations, increased the risk significantly.

The total episiotomy rate in the study group was 35.4%. In 34.1% of all deliveries a mediolateral episiotomy was performed, whereas in only 1.3% of cases a median episiotomy was performed. The use of median episiotomies was significantly associated with multiparity ($p < 0.01$) and spontaneous deliveries ($p < 0.01$). Mediolateral episiotomy appeared to be

strongly protective for anal sphincter injury, whereas median episiotomy showed a weak protective effect. After separate logistic regression analysis of all spontaneous deliveries mediolateral episiotomy was still strongly associated with a reduced risk of third and fourth degree perineal ruptures (OR: 0.34, 95%-CI: 0.31-0.37).

Induction of labour was found to be weakly associated with the occurrence of anal sphincter injurys.

All types of assisted vaginal delivery were associated with an increase in the risk of third and fourth degree perineal tears. Uterine fundal expression, to expedite delivery, was applied in 4.6 % of all vaginal deliveries, either alone or in combination with other types of intervention, and appeared to be significantly associated with an increased risk of anal sphincter damage.

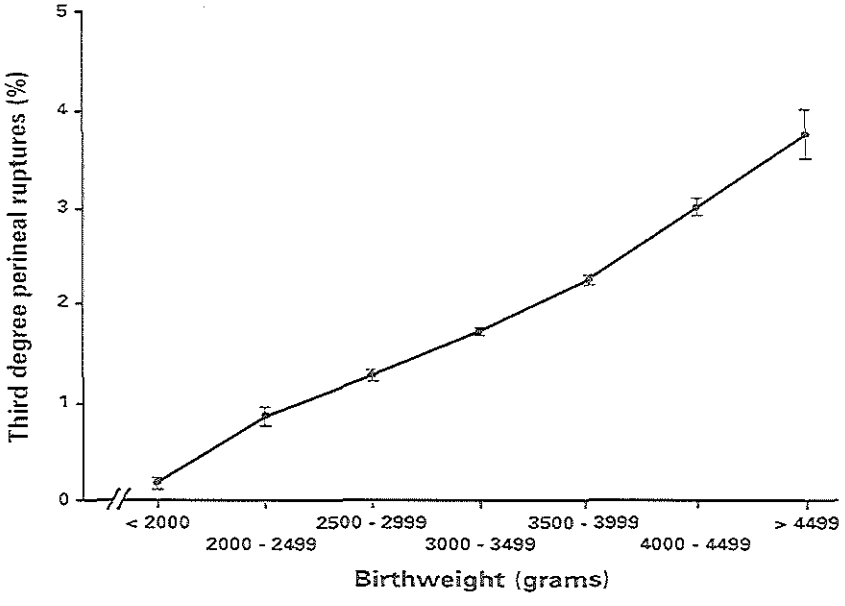


Figure 3.1: Risk of anal sphincter injury per 500 grams birthweight

Vacuum extraction was also significantly associated with anal sphincter tears, but carried a lower risk. Forceps delivery, of all forms of assisted vaginal delivery, appeared to carry the strongest risk for the occurrence of third degree perineal tears. Combined use of different types of assisted vaginal delivery appeared to increase the risk for third and fourth degree perineal ruptures in comparison with the exclusive use of one of both types.

Birthweight was found to be significantly associated with anal sphincter injury, with an odds ratio per increase of birthweight with 500 grams of 1.47 (95%-CI: 1.43-1.51) (Figure 3.1).

Also duration of the second stage of labour appeared to be significantly associated with anal sphincter tears, with an odds ratio of 1.12 (95%-CI: 1.10-1.14) per 15 minutes (Figure 3.2).

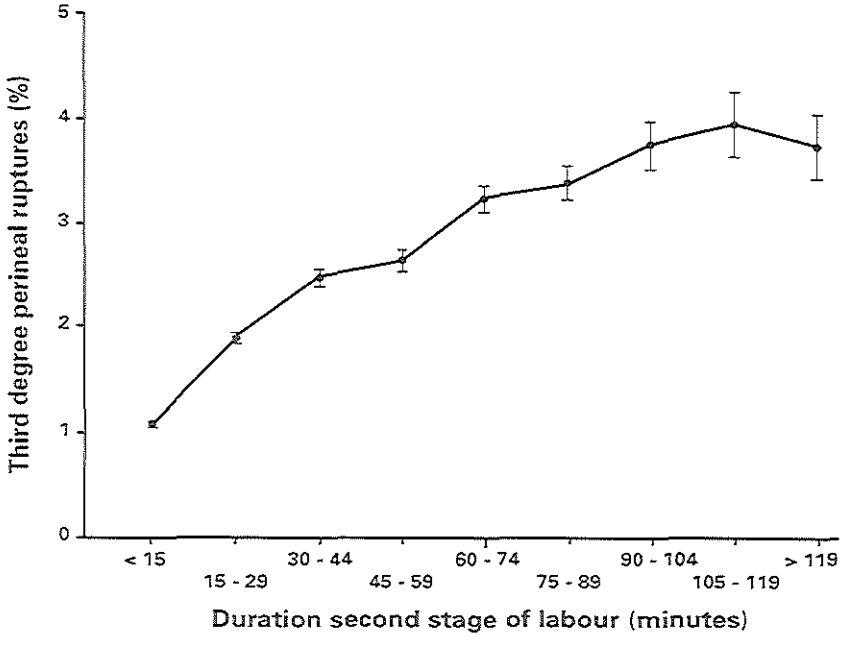


Figure 3.2: Risk of anal sphincter injury per 15 minutes duration of second stage of labor

3.4 Discussion

To our knowledge this is the largest study concerning risk factors for the occurrence of third and fourth degree perineal ruptures published to date. The study comprises the majority of all deliveries in the Netherlands over a period of two years and contains a sufficient number of deliveries and third and fourth degree perineal tears to draw firm conclusions. By using a national obstetric database, potential selection bias in data from single institutions is avoided. In the database only obstetric data are registered, which does not allow analysis of associated clinical problems such as fecal incontinence.

The overall risk of anal sphincter injury in our study, defined as any rupture of the perineum involving anal sphincter muscle, is 1.94%. This incidence is higher than that in some European reports,[17,25-28] comparable to that in other studies from the continent,[27,58] but much lower than the incidence reported from the United States.[29-32]

Our observation of an elevated risk in primiparae, which may be due to relative inelasticity of the perineum, and a reduction in risk with increasing parity is in line with earlier reports.[17,26,27,29,30,32]

Fetal presentation appears to be an important discriminating factor for the occurrence of anal sphincter injury. As previously reported, a persisting occipitoposterior position of the fetal head increases the risk of anal sphincter damage.[26,27] After logistic regression the risk of anal sphincter damage in breech deliveries appeared to be comparable with that in cephalic occipito-anterior deliveries. This may explained by selection before and during breech delivery, in which expected obstetric problems are avoided by performing a cesarean section resulting in a elevated cesarean section rate in breech deliveries of 41.6% versus 10.0%

Table 3.1 *Analysis of potential risk factors for the occurrence of anal sphincter injury (n= 284,783).*

Risk Factor	Present*	%	Relative Risk	Logistic Regression	
				Adj. OR†	95-% CI
Parity					
Multiparity	2173/159903	1.35	1		
Primiparity	3355/124880	2.69	1.99	2.39	2.24 – 2.56
Fetal presentation					
Occipito-anterior	5082/264426	1.92	1		
Occipitoposterior	250/ 7624	3.28	1.71	1.73	1.52 – 1.98
Breech presentation	103/ 9842	1.05	0.54	1.00	0.78 – 1.26
Other presentation	93/ 2891	3.21	1.67	1.59	1.28 – 1.98
Episiotomy					
No episiotomy	4185/183919	2.28	1		
Mediolateral episiotomy	1234/ 97250	1.27	0.56	0.21	0.19 – 0.23
Median episiotomy	109/ 3614	3.02	1.33	0.81	0.66 – 0.98
Induction of labor					
No induction	4556/238383	1.91	1		
Induced labor	972/ 46400	2.09	1.10	1.19	1.11 – 1.28

Table 3.1 continued

Risk Factor	Present*	%	Relative Risk	Logistic Regression Adj. OR†	95-% CI
Assisted vaginal delivery					
No intervention	4052/238503	1.70	1		
Fundal expression‡	191/ 9176	2.08	1.23	1.83	1.57 - 2.14
Fundal expr. + Vacuumextr.	74/ 2661	2.78	1.64	1.78	1.40 – 2.28
Fundal expr. + Forceps	27/ 522	5.17	3.04	4.62	3.09 – 6.89
Vacuum extraction‡	646/ 21254	3.03	1.79	1.68	1.52 – 1.86
Vacuumextr. + Forceps	51/ 656	7.77	4.58	4.74	3.49 – 6.45
Forceps delivery‡	348/ 7478	4.65	2.73	3.53	3.11 – 4.02
Interv. for shoulder dystocia‡	46/ 1180	3.89	2.29	2.03	1.49 – 2.74
Breech extraction‡	27/ 1284	2.10	1.24	2.91	1.88 - 4.51

*: Present is defined as the number of women with third or fourth degree perineal rupture/ total number of women.
†: Adj. OR = adjusted odds ratio
‡: Applied with exclusion of any other type of assisted vaginal delivery

overall. In the group of other presentations, such as brow or face presentations, the risk of sphincter damage was also significantly elevated, but the number of deliveries and third and fourth degree perineal tears was too small to draw firm conclusions.

Our study shows a strong protective effect of mediolateral episiotomies against the occurrence of anal sphincter injury in spontaneous and assisted vaginal deliveries, which was not influenced by parity. In contrast to results of earlier studies, median episiotomy was not found to increase the risk of anal sphincter tears. This may be explained by the fact that median

episiotomies were almost exclusively used in spontaneous deliveries, and were strongly associated with multiparity. Our results confirm the results of Anthony et al,[36] who found a similar protective effect in uncomplicated vertex deliveries. Other studies have questioned the beneficial effect of mediolateral episiotomies to prevent anal sphincter injury. Møller Bek and Laurberg reported that the liberal use of mediolateral episiotomies increased the risk of anal sphincter damage.[26] Two randomized trials showed no protective effect of routine mediolateral episiotomy,[33,34] but because of very small numbers the statistical power of one of these was too low to allow firm conclusions.[33] The episiotomy rate in our study group was much lower than that in the group with anal sphincter injury in the study of Møller Bek and Laurberg (34.1% *vs.* 84.9%) and comparable to the rate in the group with selective use in the Argentinean trial (34.1 % *vs.* 30.1%).[26,34] A protective effect of selective use of mediolateral episiotomy, cannot be ruled out on the basis of these previous studies, and is strongly supported by the results of our study, and mediolateral episiotomy may thus protect against resulting fecal incontinence.

Induction of labor was found to be associated with a slightly increased risk of anal sphincter damage, which confirms the results of Poen et al.[27] Indications for induction of labor are not included in the LVR and can therefore not be analyzed. The mechanism by which induction of labor results in a higher risk of anal sphincter damage remains unclear and needs further study.

All types of assisted vaginal delivery were found to be associated with an increased risk of anal sphincter lacerations. Our study showed a marked increase in the risk when vacuum extraction was performed. The fact that earlier studies showed no independent effect of vacuum extraction can be explained by the small number of third and fourth degree perineal

ruptures and small study groups.[17,26,27] Forceps delivery appeared to be the strongest risk factor, which is in line with results of earlier studies.[17,26,27] With respect to the prevention of anal sphincter damage, vacuum extraction is to be preferred over forceps delivery, if the obstetric situation permits use of either instrument. The combined use of forceps with fundal expression or vacuum extraction appeared to increase the risk for the occurrence of anal sphincter injury even further, and should therefore be avoided, whenever possible. Interventions used to resolve shoulder dystocia were also associated with an increased risk of anal sphincter damage, which confirms the results of Møller Bek and Laurberg.[26]

Our results show a significant positive correlation between birthweight and the occurrence of anal sphincter injury. Shiono et al reported a significant odds ratio of 1.10 per 100 grams increase in birthweight,[31] and other studies have shown an elevated risk with fetal birthweight exceeding 4000 grams.[17,27]

Although earlier studies failed to show a relationship between the duration of the second stage of labor and anal sphincter damage,[26,27,32] our study shows a significant increase in the risk of third and fourth degree perineal tears with increasing duration of the second stage. Stretching of the perineum for a longer period of time may lead to ischemia, which may increase the risk of ruptures of the perineum. Whether the use of upper time limits for the duration of second stage will lower the risk of anal sphincter damage remains doubtful, as this will lead to an increase in operative vaginal deliveries with an even greater risk of sphincter injuries.

Chapter 4

ANAL SPHINCTER INJURY AT VAGINAL DELIVERY: FUNCTIONAL OUTCOME AND RISK FACTORS FOR FECAL INCONTINENCE*

4.1 Introduction

Anal sphincter injury due to third and fourth degree perineal tears is a known but relatively rare complication of vaginal delivery. Sequelae such as perineal pain, sexual dysfunction, and fecal incontinence, urgency or soiling may develop.

The incidence of third and fourth degree perineal ruptures at delivery appears to vary in reports from different countries. European studies report incidences between 0.5 and 3 %, whereas studies from the United States show rates up to 25 %.[1,2] Studies on the functional outcome of primary repair of anal sphincter injury have shown that fecal incontinence may develop in up to 57 % of women. Most of these studies, however, contain small numbers of patients,[17,19-22] lack control groups,[18,22-24] or a sufficient follow-up period, which hampers a reliable interpretation of these results.[17,19,20,24]

We present the results of a large retrospective case-control study, with a median follow-up of 14 years. The aim of our study was to assess the functional outcome after primary repair of anal sphincter injury in comparison with the outcome in controls with a vaginal delivery without anal sphincter damage, and to analyze obstetric risk factors for the development of anorectal complaints after anal sphincter damage complicating vaginal delivery.

* *The main substance of this chapter was published in: de Leeuw JW, Vierhout ME, Struijk PC, Hop WCJ, Wallenburg HCS. Anal sphincter damage after vaginal delivery: functional outcome and risk factors for fecal incontinence. Acta Obstet Gynecol Scand 2001;80:830-34*

4.2 Methods

The study was designed as a retrospective case-control study with matched controls and was approved by the Medical Ethics Committee of the Ikazia Hospital, Rotterdam. All 171 women who underwent primary repair of anal sphincter injury between January 1st 1971 and December 31st 1990 in the Ikazia Hospital were included in the study. This group comprised women who were delivered in the hospital attended by the obstetrician-gynecologist, as well as women referred (73 %) after home delivery under supervision of an independent midwife or general practitioner. The first woman after the index case, matched for parity, who had a vaginal delivery without anal sphincter damage in our hospital was selected as a control. All relevant data were obtained from the hospital records. Perineal tears with anal sphincter damage were classified in three groups: Partial rupture of the anal sphincters (third degree-a), complete rupture of the sphincters with intact anal mucosa (third degree-b), and complete rupture of the anal sphincters and mucosa (fourth degree).

In the 20-year period covered by the study, the surgical technique of primary repair remained unchanged. Sphincter muscle ends were approximated end-to-end using interrupted chromic catgut sutures. The anal mucosa was closed separately with interrupted chromic catgut sutures if necessary. A nylon suture through the perineal skin and both sphincter ends was used and left in place for one week, to secure approximation of both sphincter ends. Vaginal mucosa, perineal muscles and skin were repaired as in routine second-degree rupture or episiotomy. All women received prophylactic antibiotic treatment.

A questionnaire was sent to all patients and matched controls with questions about the obstetric and medical history, general health, daily defecatory pattern, and complaints of fecal

soiling, fecal and urinary incontinence or urgency (Appendix A). If the questionnaire was not returned after three weeks a reminder was sent. Complaints of incontinence were scored positive if they were reported to occur more than once a week during a period of at least one year. The severity of complaints of fecal incontinence was classified according to Parks' classification.[57] The frequency of complaints was classified as less than once a week, one to six times per week, one to five times a day or more than five times a day.

Statistical testing of comparisons between index cases and controls regarding general and obstetric characteristics was performed using McNemar's test or Wilcoxon's signed-rank test for qualitative or continuous data. Comparisons of the functional outcomes between both groups were evaluated with the Mantel-Haenzsel common odds ratio estimate for matched case-control studies. Univariate analysis of risk factors for the development of anorectal complaints after anal sphincter damage was performed with calculations of odds-ratios with 95%-confidence intervals. Multiple logistic regression analyses were performed to asses independent risk factors. A two-sided p-value of 0.05 was considered to be the limit of statistical significance. Analyses were done with the Statistical Package for Social Sciences, version 7.0 for Windows (SPSS Inc., Chicago, IL).

4.3 Results

Of 171 women with anal sphincter damage, 147 (86%) returned a completed questionnaire; 10 women refused participation in the study, and 14 were lost to follow-up. Of 171 controls, 131 (73%) returned a completed questionnaire; 27 refused participation and 13 women were

lost to follow-up. Of 147 index cases and 131 controls, 125 matched pairs remained and formed the subject of this study.

In the case group, 67 women (54%) had a third degree-a rupture, 36 women (29%) a third degree-b rupture, and 22 women (18%) a fourth degree rupture.

Table 4.1. General characteristics. Values are presented as median (range) or total number [%].

	Cases (n=125)	Controls (n=125)
Age at delivery (yrs)	26 (18-41)	28 (19-38)
Age at questionnaire (yrs)	40 (27-59)	41 (24-58)
Duration of follow-up (yrs)	14 (5-24)	14 (5-24)
Gestational age (wks)	39 (36-42)	38 (35-41)
Parity	1 (1-3)	1 (1-4)
Number of subsequent deliveries	1 (0-4)	1 (0-6)
Birthweight (gm)	3620 (2060-5700)	3430 (1870-4380)*
Vacuum extraction	7 [5.6]	13 [10.4]
Forceps delivery	2 [1.6]	0
Occipitoposterior presentation	5 [4.0]	1 [0.8]
Breech delivery	4 [3.2]	6 [4.8]
Mediolateral episiotomy	47 [37.6]	70 [56.0]*

* $p < 0.05$

Table 4.1 lists the general characteristics of both groups. All episiotomies were of the mediolateral type. There were no significant differences between both groups except a higher birthweight in the case group and more mediolateral episiotomies in the control group. The

median follow-up in both groups was 14 years. Separate analysis comparing responders and nonresponders within both study groups showed no differences.

All forms of fecal incontinence were significantly more common in the group with sphincter damage (Table 4.2).

Table 4.2 *Prevalence of complaints. Values are presented as n (%)*

	Cases (n=125)	Controls (n=125)	Mantel-Haenszel Common Odds-ratio [95%-CI]
Anorectal complaints	50 (40)	19 (15)	3.64 [1.87 - 7.09]
Fecal incontinence	39 (31)	16 (13)	3.09 [1.57 - 6.10]
Grade-II	28 (22)	14 (11)	
Grade-III	10 (8)	2 (2)	
Grade-IV	1 (1)	0	
Fecal urgency	32 (26)	7 (6)	7.25 [2.55 - 20.62]
Fecal soiling	12 (10)	1 (1)	12.00 [1.56 - 92.29]
Urinary incontinence	65 (52)	52 (42)	1.46 [0.91 - 2.37]
Stress-incontinence	63 (50)	50 (40)	1.46 [0.91 - 2.37]
Urge-incontinence	32 (26)	28 (22)	1.16 [0.68 - 1.98]

A total of 40% of women in the case group reported some kind of anorectal problem, compared to 15% of women in the control group.

Separate analysis of women with anorectal complaints showed that in the group of women with sphincter damage complaints started significantly earlier compared to controls. In 69% of cases complaints started in the first three months after delivery, compared to 31% in controls (p=0.003). Classified according to Parks' classification, complaints of fecal

incontinence were more severe in cases compared to controls ($p<0.001$). Also the rate of occurrence was significantly higher in the case group ($p=0.004$).

In more than 90% of women with anorectal complaints these were still present at the time of the questionnaire. Only a minority underwent earlier treatment for their complaints; 14 were treated conservatively with dietary measures or physiotherapy, whereas two women underwent anterior sphincter repair.

In contrast to anorectal complaints, neither stress- nor urge-incontinence for urine were found to be associated with previous anal sphincter damage during delivery.

Characteristics such as maternal age at delivery and current age, number of subsequent vaginal deliveries, and obstetric factors such as parity, gestational age, mode of delivery, fetal birthweight and presentation, extent of sphincter damage and presence of an episiotomy were tested as potential risk factors for the development of fecal incontinence after anal sphincter damage. Using univariate analysis only the extent of anal sphincter damage and the presence of a mediolateral episiotomy appeared to be associated with the development of fecal incontinence (Table 4.3). Women with a third degree-a rupture reported complaints in 21%, women with a third degree-b rupture in 31%, and women with fourth degree ruptures in 64% of cases. Stepwise logistic regression analysis confirmed the extent of sphincter damage to be the primary independent risk factor for the development of fecal incontinence. Using the subdivision of perineal tears in degrees three through four, the odds for the development of fecal incontinence increased more than twofold with each step (Table 4.3).

While univariate analysis suggested that mediolateral episiotomy had a weak protective effect for the development of fecal incontinence, multivariate analysis showed that this effect was only present in primiparae. Of the primiparae without a mediolateral episiotomy and anal

sphincter damage, 46 % developed fecal incontinence, compared to 12% of the primiparae with anal sphincter damage combined with a mediolateral episiotomy (p=0.003). In multiparae these figures were resp. 32% and 44% (p=0.47). The odds for primiparae with episiotomy in the case group to develop fecal incontinence, adjusted for the extent of sphincter damage, was reduced by 83 % (p=0.005), compared to other women.

Table 4.3. *Univariate and multivariate analysis of various risk factors for fecal incontinence after anal sphincter damage during delivery*[*].*

	Univariate analysis		Multivariate analysis	
Extent of perineal damage[†]	2.44 [1.46 – 4.06]	< 0.001	2.54 [1.45 – 4.45]	0.001
Subsequent vaginal delivery	1.09 [0.50 – 2.34]	0.83	2.32 [0.85 – 6.33]	0.10
Primiparity[‡]	0.79 [0.37 – 1.69]	0.55	1.16[a] [0.41 – 3.29]	0.78
			0.15[b] [0.02 – 0.98]	0.05
Mediolateral episiotomy[‡]	0.38 [0.15 – 0.91]	0.03	0.17[c] [0.05 – 0.61]	0.007
			1.25[d] [0.27 – 5.83]	0.78

[*] values are presented as odds ratios with [95%-confidence interval] and P-values
[†] per grade: grade-IV vs. grade-IIIb vs. grade-IIIa
[‡] significant difference: [a]without episiotomy versus. [b]with episiotomy, and [c]primiparity versus. [d]multiparity

Women who had one or more vaginal deliveries following the delivery with anal sphincter damage reported complaints in 41%, compared to 39% of those who did not deliver vaginally after the delivery in which the sphincter damage occurred. Multivariate analysis showed that fecal incontinence was not significantly positively associated with subsequent vaginal deliveries. In none of the analyses an association was found between fecal incontinence and age at the moment of delivery or the duration of follow-up, the latter being minimally 5 years.

The group of 16 women with fecal incontinence in the control group was too small to analyze for risk factors.

4.4 Discussion

During the last decade the relationship between vaginal delivery and subsequent urinary and fecal incontinence has received increasing interest, in particular with regard to the contribution of anal sphincter damage (Table 4.4).[16-25,38,58,60-63] These studies indicate a significant but variable association between anal sphincter damage following vaginal delivery and subsequent anorectal complaints. The variability in results may be attributed, at least in part, to small study size and short follow-up, or both. Our questionnaire-based study contained large numbers in case and control groups, with high response rates, which makes significant selection bias unlikely. The extensive period of follow-up allows assessment of long-term consequences of anal sphincter damage during delivery.

The study and control groups were similar regarding their general characteristics, except for a lower median fetal birthweight and a higher incidence of episiotomy in controls (Table 1). These differences may be explained by the recruitment of the control group entirely from women who delivered in the hospital under specialist care, with more pregnancies and deliveries at risk than in the case group, 73% of which were delivered at home.

Anal sphincter damage was found to be significantly associated with fecal incontinence, which is in accordance with the results of earlier studies.[17-25,58] The occurrence of fecal urgency in women with anal sphincter damage in our study is similar to that reported by Sultan et al.[17] Our findings with regard to fecal soiling confirm the results of earlier studies in

which fecal soiling is reported in 7 to 10 percent of women with anal sphincter damage after delivery.[18,25]

Table 4.4: *Follow-up studies after anal sphincter injury at delivery, with or without controls*

				Fecal Incontinence (%)				
				Anal sphincter injury				*Controls*
Authors, year	*Ref.nr.*	*Nr. cases*	*Follow-up*	*Grade-II*	*Grade-III*	*Grade-IV*	*Total*	
Haadem et al, 1988	64	59	41 months	25	7		32	0
Sørensen et al, 1988	65	24	78 months	25	13	4	42	0
Go et al, 1988	66	9	29 months	0	33	0	33	---
Haadem et al, 1990	67	21	3 months	43	5		48	0
Møller Bek et al, 1992	23	121	2-13 years	---	---	---	48	---
Nielsen et al, 1992	59	24	12 months	17	9	4	30	---
Sørensen et al, 1993	68	38	3-12 months	13	24		37	0
Crawfordet al, 1993	19	35	9-12 months	17	3	3	23	6
Sultan et al, 1994	17	34	6 wks-2 years	32	9	0	41	6
Tetzschner et al, 1996	58	94	2-4 years	25	17		42	---
Walsh et al, 1996	24	81	3 months	12	7		20	---
Uustal et al, 1996	20	51	6 months	24	16	0	40	35
Nygaard et al, 1997	21	29	32 years	31	28		59	30
Haadem et al, 1997	25	41	16-21 years	23	10	7	39	5
Franz et al, 1998	69	82	21 weeks	30	---	---	30	10
Poen et al, 1998	18	117	10-135 months	23	6	0	29	---
Goffeng et al, 1998	70	34	12 months	59	11		70	13
Wood et al, 1998	71	84	2-7 years	7	10		17	---
Gjessing et al, 1998	22	35	41 months	34	11	11	57	---
Zetterström et al, 1999	72	38	9 months	42	---	---	42	---
Kammerer et al, 1999	73	15	4 months	---	---	---	43	20
Sangalli et al, 2000	74	179	13 years	6	7	2	15	---

The results of the three previously published studies with a follow-up of more than ten years, with regard to the occurrence of anorectal complaints, are contradictory.[21,25] Nygaard et al. reported no significant difference in the rate of frequent flatus incontinence in women with anal sphincter damage compared to women with episiotomy only, and frequent fecal incontinence was even significantly more common in the latter group.[21] This may be explained by the high rates for frequent flatus and fecal incontinence in the control group of 30.3% and 18.0%, respectively. A recent study from the United States showed similar high incontinence rates in women after midline episiotomy without visible extension.[75] These rates are much higher than those reported in our control group and in control groups of other European studies.[17,20,25,58] The differences may be explained by a high incidence of unrecognized sphincter damage after midline episiotomy. This procedure is known to increase the risk of anal sphincter damage which may be difficult to recognize.[2,16] Our study confirms the results of the study of Haadem et al. and Sangalli et al in which anorectal complaints were significantly more often present in women with anal sphincter damage almost two decades after delivery compared to women without anal sphincter damage.[25,74]

Our study shows that in women with anal sphincter injury at delivery complaints start significantly earlier after delivery and are more severe than in controls, an issue not addressed in any of the earlier studies. The fact that only a minority of women underwent treatment for their complaints is in line with previous reports and indicates that many women may be reluctant to discuss the problem with their physician, or that their complaints are not taken seriously.[21,25]

Findings with regard to urinary incontinence in our study confirm the results of Nygaard et al. and Haadem et al. and support evidence that the development of urinary incontinence after

delivery may mainly be due to general damage or denervation of the pelvic floor, which is not significantly affected by rupture of the anal sphincter complex.[21,25,63]

Knowledge of risk factors for the development of fecal incontinence is needed for adequate counseling of women with previous sphincter damage. Using stepwise logistic regression analysis we identified the extent of anal sphincter damage as an independent risk factor for the development of fecal incontinence. Tetzschner et al.,[58] using a different classification of anal sphincter damage, found no association between the extent of
damage and subsequent fecal incontinence. Their classification with a very discrete classification of sphincter damage may be difficult to use in daily practice and is liable to misclassification. Our findings confirm the results of Poen et al. and Haadem et al., who also found an increased risk for development of fecal incontinence after involvement of the anal mucosa.[27,64]

In contrast to the findings of Møller Bek and Laurberg,[23] who reported an increased risk of fecal incontinence after subsequent vaginal delivery in women with mild or transient symptoms, and Sangalli et al.,[74] who reported an increased risk of fecal incontinence following subsequent vaginal delivery in women with anal sphincter injury with involvement of the anal mucosa, our findings showed that subsequent vaginal deliveries were not associated with an increased the risk of fecal incontinence after anal sphincter damage during a previous delivery. The observed protective effect of mediolateral episiotomy for the development of fecal incontinence in primiparous women is of note. Only mediolateral episiotomies were performed, as is common practice in the Netherlands. The protective effect may be explained by reduced stretching of the perineum, as prolonged stretching of the pelvic floor and the pudendal nerve may aggravate complaints of fecal incontinence.[58,76]

Further study is necessary to elucidate the role of mediolateral episiotomy in the development of fecal incontinence after anal sphincter damage during delivery.

Chapter 5

ANAL SPHINCTER INJURY AFTER VAGINAL DELIVERY: RELATIONSHIP OF ANAL ENDOSONOGRAPHY AND MANOMETRY WITH ANORECTAL COMPLAINTS

5.1 Introduction

Fecal incontinence is an embarrassing health problem that may lead to social isolation.[8] It is reported to occur in approximately 2.2% of the general population.[77] In a recent American study women of 50 years of age reported fecal incontinence in 13.1%, whereas women of 80 years and older reported fecal incontinence in 20.7% of cases.[78] During the last decade increasing awareness has developed that injury to the anal sphincters associated with childbirth is a major cause of the development of fecal incontinence in women.[38,61,63] Anal manometry and anal endosonography are considered to be the methods of choice to evaluate the condition of the anal sphincter complex after vaginal delivery.[15,79,80] Anal manometry may indicate the presence of anal sphincter malfunction when anal resting and squeeze pressures are reduced,[79] and anal endosonography allows reliable visualization of damage of the anal sphincters.[15,81] However, results of follow-up studies of women who suffered anal sphincter injury during delivery using anal manometry and endosonography are conflicting.[17,18,22,59,67,68,70,82] In some studies anal manometry showed lower resting and squeeze pressures in women with sphincter damage compared to controls,[17,67,82] whereas other studies showed differences in only one of these parameters, or no differences at all.[18,68,70] Anal manometry showed no differences between women with and without complaints after anal sphincter damage.[22,59,67,68,82]

Studies using anal endosonography showed significantly more persisting sphincter defects in women with anal sphincter damage during delivery than in controls.[17,18,70] On the other hand, some studies using anal endosonography in women with and without anorectal complaints after anal sphincter damage showed significant differences between these groups,[17,22] whereas others found only differences in the number of defects in one of the sphincters, or no differences.[18,59,70] The majority of these studies were done shortly after anal sphincter damage had occurred,[17,18,59,67,68,70] or lacked control groups.[22,59]

The aim of our study was to assess the relationship of anal manometry and endosonography with anorectal complaints in women who suffered demonstrated anal sphincter injury during vaginal delivery, after primary repair and longtime follow-up.

5.2 Methods

Thirty-four women who underwent primary repair of a third or fourth degree perineal tear in our department in the period 1971-1990 were investigated using a questionnaire, anal manometry and anal endosonography. A third degree rupture was defined as a perineal tear with partial or complete rupture of the anal sphincters with intact anal mucosa, a fourth degree tear with in addition laceration of the anal mucosa. The first woman with an uncomplicated vaginal delivery after the index case and no anorectal complaints was selected from the delivery records and invited to take part in the study as a control. Of the 34 women who were approached only 12 agreed and formed the control group. All women gave their informed consent. The study was approved by the Medical Ethics Committee of the Ikazia Hospital, Rotterdam, the Netherlands.

Primary repair

The method of primary surgical repair of third and fourth degree perineal tears remained unchanged throughout the period of time covered by the study. If necessary, the anal mucosa was closed with interrupted chromic catgut sutures. Sphincter muscle ends were approximated end-to-end using interrupted chromic catgut sutures. A nylon suture through the perineal skin and both sphincter ends was used and left in place for one week, to secure approximation of both sphincter ends. Vaginal mucosa, perineal body and skin were repaired as usual in second-degree perineal rupture or episiotomy. All women received prophylactic antibiotic treatment.

Questionnaire

A questionnaire was sent to all patients and controls with questions about the obstetric and medical history, general health, daily defecatory pattern, and complaints of fecal soiling, fecal and urinary incontinence or urgency (Appendix A). If the questionnaire was not returned after three weeks a reminder was sent. Complaints of incontinence were scored positive if they were reported to occur more than once a week during a period of at least one year. The severity of complaints of fecal incontinence was classified as incontinence for flatus only, for flatus or loose stools or for all stools. The frequency of complaints was classified as less than once a week, one to six times per week, one to five times a day, or more than five times a day.

Anal manometry

Anal manometry was performed with the patient in left lateral position with flexed knees and hips, without bowel preparation. A catheter of 3-mm diameter with a microtransducer (Gaeltec, Synetics Medical AB, Sweden), was placed in the rectum and left to accommodate for several minutes. The catheter was then withdrawn in 1-cm steps. The maximum anal resting pressure (MARP), expressed in mmHg, was determined by pulling the catheter through the anal canal three times, and calculating the mean value of the three measurements. After positioning the transducer at the location of the MARP, the patient was asked to squeeze maximally three times to obtain the maximum anal squeeze pressure (MASP), expressed in mmHg. The mean value of three recordings was taken as the MASP (Figure 5.1 and 5.2). Rectal sensitivity was tested by inflating a silicone balloon catheter positioned in the rectum with increments of 10cc of air, until the patient sensed the balloon (volume of first sensation, FSV), felt the urge to defecate (urge volume, UV), and experienced pain (maximum tolerable volume, MTV).

Anal endosonography

Anal endosonography was performed with a Bruel and Kjaer ultrasound system (type 2203) with a 7-10 MHz, 360°-rotating endoprobe (type 1850) covered by a water-filled hard sonolucent plastic cone with an external diameter of 1.7 cm. Serial radial images were

obtained at the level of the puborectal muscle, the central level and the subcutaneous level of the anal canal (Figures 1 and 2). Defects were recorded directly from the screen. A defect in the external sphincter was defined as a break in the continuity of the normal sonographic texture of the muscle, usually with a hypo-echoic appearance or an appearance of mixed echogenicity. A defect in the internal sphincter, represented as a homogeneous hypo-echoic

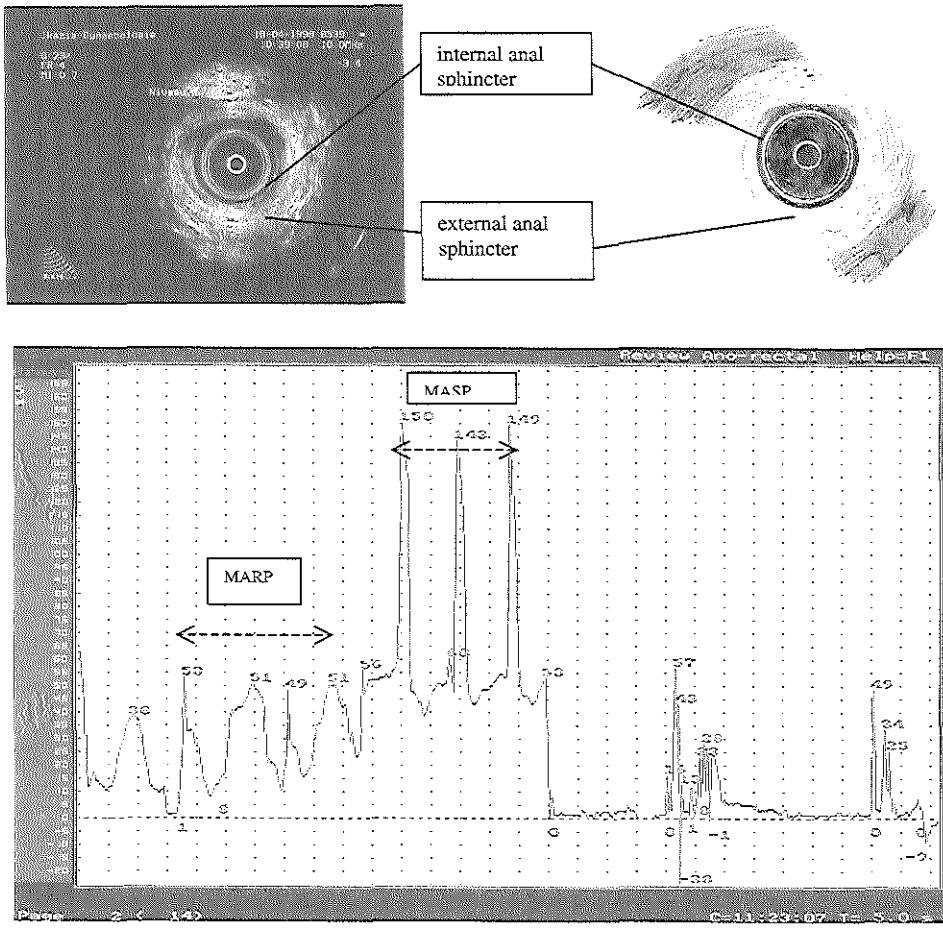
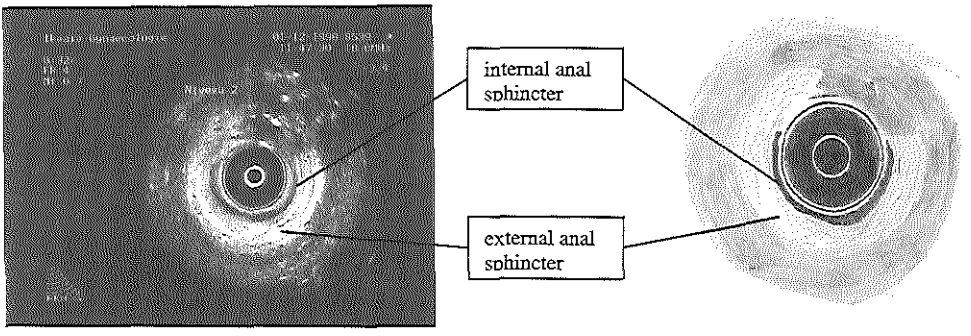


Figure5.1: *Anal endosonographic picture and results of anal manometry of a 41-year old woman without anorectal complaints. 12 years after she delivered a healthy girl of 4230 grams without complications. Anal endosonography and manometry showed no abnormalities.*

ring, was defined as a break in the continuity of the ring. The presence of defects at different levels of the anal canal was recorded to establish the cranio-caudal length (mm) of the defects.



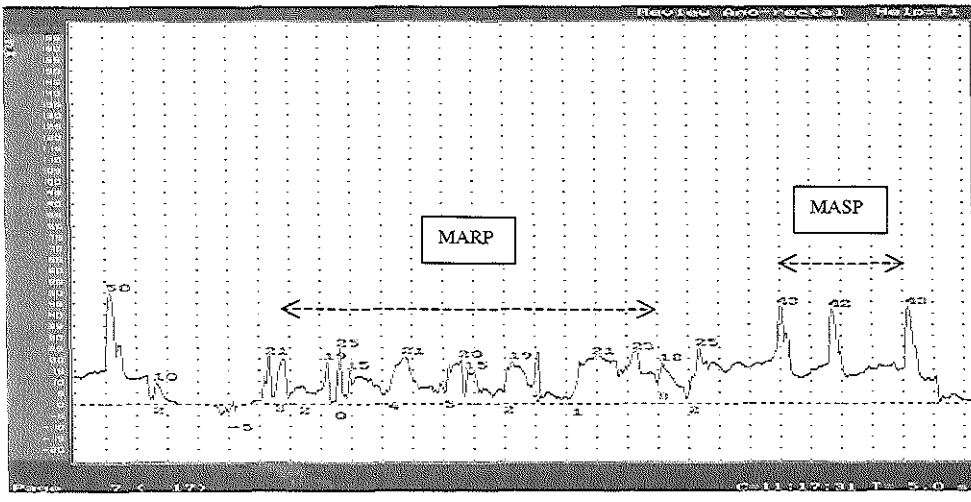


Figure 5.2: *Anal endosonographic picture and results of anal manometry of a 52-year old woman suffering from fecal urgency and grade-III incontinence, one to five times daily, 21 years after she delivered a boy of 3430 gram at home, complicated by a grade III(b) perineal rupture. Anal endosonography showed a defect in the internal sphincter from 10 to 2 o'clock and a defect in the external sphincter from 12 to 2 o'clock. Anal manometry showed abnormally low maximum anal resting and squeeze pressures.*

The number and radial extent of sphincter defects, expressed in degrees, were determined in each subject.

Statistical analysis

Comparisons of general characteristics and results of anal manometry and endosonography were tested with the Kruskal-Wallis and Mann-Whitney-U test for continuous variables, and Chi-square and Fisher's exact-test for categorical variables. A two-sided p-value of 0.05 was considered the limit of statistical significance. Analyses were done with the Statistical Package for Social Sciences, version 9.0 for Windows (SPSS Inc., Chicago, IL).

5.3 Results

General characteristics of the study group at the time of the vaginal delivery associated with anal sphincter damage, and of the controls are presented in Table 5.1. Based on the results of the questionnaire, the study group was divided into a subgroup of women without anorectal complaints and one of women with complaints. No significant differences between groups are apparent. Of the 34 women with a history of anal sphincter damage, 12 (35%) reported incontinence for flatus, and 7 (21%) incontinence for loose stools. Fecal urgency was reported by 12 (35%) women, whereas fecal soiling was reported by 8 (24%) women. A total of 22 (65%) women reported anorectal complaints. Of these, 14 reported complaints to occur one to six times per week, five women reported complaints one to five times a day and three women reported complaints to occur more than five times a day.

Results of anal manometry and rectal sensitivity tests are presented in Table 5.2.

Both MARP and MASP were significantly lower in women with anal sphincter damage compared to controls. In the group of women with anal sphincter damage and complaints, nine women (41%) had an MARP that is usually considered abnormally low (< 30 mm Hg) and 16 (73%) had an abnormal MASP (< 70 mm Hg), whereas none of the controls had an abnormal MARP and only one had an MASP below 70 mm Hg ($p < 0.05$ and $p < 0.001$, respectively).

Table 5.1: General characteristics. Values are presented as median (range).

	Anal sphincter damage		
	Complaints (n=22)	No complaints (n=12)	Controls (n=12)
Age at delivery (years)	33 (25 – 42)	31 (26 - 40)	31 (27 –36)
Age at questionnaire (years)	46 (32 – 64)	44 (36- 62)	47 (36 –49)
Duration of follow-up (years)	18 (6 – 23)	14 (5 – 24)	13 (7 –19)
Parity	1 (1 – 3)	1 (1 – 3)	1 (1 – 3)
Subsequent deliveries (n)	1 (0 – 2)	2 (0 – 3)	1 (0 – 6)
Fetal birthweight (grams)	3535 (2430 – 4130)	3675 (3170 – 4380)	3602 (2350 – 4380)
Mediolateral episiotomy (n)	9	7	6

When comparing women with anal sphincter damage without complaints with controls, only the number of women with an abnormal MASP differed significantly between both groups (50% vs. 8%, $p < 0.05$). The proportion of women with an abnormal MARP or MASP, or both, was not different in women with a history of anal sphincter injury with and without anorectal complaints.

The mean MARP was significantly lower in women with anorectal complaints, whereas the mean MASP showed no significant difference between women with and those without

anorectal complaints. Both median MARP and MASP showed considerable overlap between continent and incontinent women, as shown in Figure 3. No differences were observed between the three groups with regard to rectal sensitivity.

Table 5.2: Results of anal manometry and rectal sensitivity tests. Values are presented as median (range).

	Anal sphincter damage		
	Complaints (n=22)	No complaints (n=12)	Controls (n=12)
MARP* (mm Hg)	31*‡ (21-54)	42† (25-66)	52‡ (33-108)
MASP* (mm Hg)	55‡ (31-97)	69§ (45-96)	112‡§ (61-170)
FSV (ml)	60 (30-120)	90 (20-180)	60 (50-120)
UV (ml)	110 (50-180)	120 (50-210)	120 (90-190)
MTV (ml)	170 (90-240)	215 (90-340)	230 (110-300)

* $p < 0.001$ for three groups (Kruskal-Wallis test)
† $p = 0.02$ for cases with vs. cases without complaints
‡ $p < 0.001$ for cases with complaints vs. controls
§ $p < 0.001$ for cases without complaints vs. controls

Table 5.3 presents the results of anal endosonography. All sphincter defects were located in one of the anterior quadrants. Isolated defects of the internal anal sphincter were not observed. Isolated defects of the external anal sphincter and combined defects of the internal and external anal sphincters were significantly associated with previous anal sphincter damage. Within the group of women with a history of anal sphincter injury, persisting anal sphincter defects were not associated with presence of anorectal complaints. No association was found between the cranio-caudal length or the radial extent of the sphincter defects, proven by anal endosonography, with previous anal sphincter damage or the presence of anorectal

complaints. The results of anal endosonography were in agreement with the results of anal manometry, when compared in the entire study group. In women with anal sphincter defects shown by anal endosonography, the mean MARP and MASP were significantly lower than in women without anal sphincter defects ($p < 0.05$ and $p < 0.001$, respectively). However, after subdivision of the study group, no difference could be demonstrated. In the group of 22 women with previous anal sphincter injury with complaints, 11(58%) of 19 women with

Table 5.3: *Frequency and extent of anal sphincter damage by anal endosonography. Values are presented as n (%) or median [range]*

	Anal sphincter damage		
	Complaints (n=22)	No complaints (n=12)	Controls(n=12)
No. of defects			
Internal sphincter	0	0	0
External sphincter	6 (27)	2 (17)	0
Both sphincters	13* (59)	6 (50)	1* (8)
Total	19† (86)	8‡ (67)	1†‡ (8)
Radial extent of damage (°)			
Internal sphincter	73 [0-144]	9 [0-140]	36§
External sphincter	26 [0-174]	21 [0-59]	46§

* $p = 0.009$ for cases with complaints vs. controls
† $p < 0.001$ for cases with complaints vs. controls
‡ $p = 0.009$ for cases without complaints vs. controls
§ concerns one patient

sphincter defects found by anal endosonography had an MARP of more than 30 mm Hg and four (21%) out of these 19 had an MASP of more than 70 mm Hg, a non-significant difference. In the group of women with previous anal sphincter damage without complaints,

six out of eight women with sphincter defects shown by anal endosonography had a normal MARP, whereas three had a normal MASP.

5.4 Discussion

The study describes the relationship of anal endosonography and manometry with anorectal complaints, at least 10 years after anal sphincter injury occurred during delivery.
For comparison of the results of anal manometry and endosonography in women who suffered anal sphincter damage during delivery, we sought to establish a control group of women who had an uncomplicated vaginal delivery at approximately the same time and no anorectal complaints. Enrollment of those healthy women into the study proved to be difficult, and we had to be satisfied with only 12 women in the control group. General and obstetric characteristics were similar between the study group and the controls.
The results of anal manometry were significantly related to previous anal sphincter injury. Both MARP and MASP were significantly lower in women with previous anal sphincter damage with complaints compared to controls, although in women with previous anal sphincter damage without complaints only the MASP differed significantly from that in controls. Haadem et al. and Sultan et al. showed that MARP and MASP were significantly reduced in women with anal sphincter damage shortly after delivery, regardless of the presence of complaints.[17,67] Sørensen et al. found significantly lower MARP and MASP in women with anal sphincter damage compared to controls three months after delivery, but these differences had disappeared twelve months after delivery.[68] Our results indicate that anal sphincter injury during delivery is associated with decreased anal squeeze pressures even more than ten years after delivery, regardless of the presence of anorectal complaints, whereas

decreased anal resting pressures are only associated with previous anal sphincter injury in women with complaints.

In women with anal sphincter damage the MARP was significantly lower in women with anorectal complaints compared to those without complaints, but the MASP was not different between both groups. The large overlap between MARP and MASP in both groups, as apparent from Figure 5.3, limits the predictive value of anal manometry, in accordance with results of earlier studies.[22,59,67,68,82]

Our results showed no differences in any of the parameters of rectal sensitivity between the three groups. Reports on rectal sensitivity tests after anal sphincter damage in the literature are scarce. Poen et al. reported only an increased volume of first sensation in women with anal sphincter damage, but no differences in other parameters.[18] On the basis of these results,

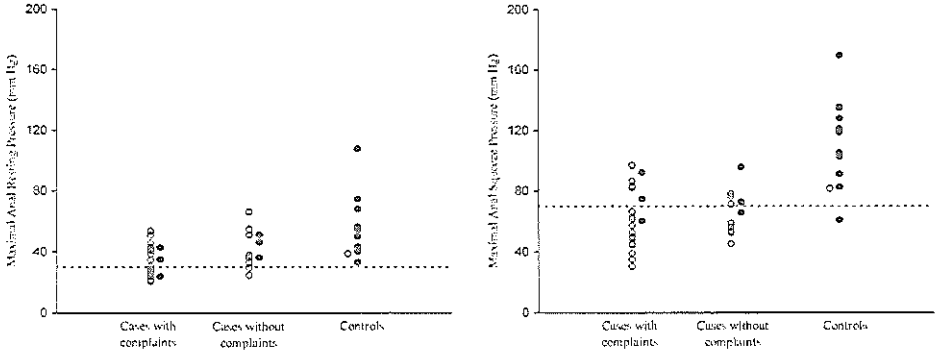


Figure 5.3: *Relationship of maximum anal squeeze pressures and endosonographic sphincter defects in patients with a history of anal sphincter damage during delivery with (n=22) and without (n=12) complaints of fecal incontinence, and in controls with a history of uncomplicated delivery and no complaints (n=12). The dotted line divides normal and abnormal levels. ○: Endosonographic sphincter defects ● : No endosonographic sphincter defects*

testing of rectal sensitivity in the evaluation of women with anal sphincter damage appears to be of limited clinical importance.

In our study ultrasonographic defects in the anal sphincter complex were strongly associated with anal sphincter damage during delivery, in accordance with results of previous studies.[17,18,22,70] Results of previous studies on the relationship between anorectal complaints and anal endosonography are contradictory. Some studies showed a strong association between findings of anal endosonography and the occurrence of anorectal complaints,[17,22] whereas others found no relationship between fecal incontinence and sphincter defects.[18,59,70] In our study, sphincter defects tended to be more common in women complaining of fecal incontinence, but this did not reach statistical significance (Table 5.3).

In accordance with findings reported by Poen et al., we could not demonstrate a difference in the radial extent of sphincter damage between women with anal sphincter damage and controls.[18] Contrary to observations reported by Sultan et al. we found no significant difference in the cranio-caudal length of the defects in women with anal sphincter damage compared to controls.[17]

In the entire group of 46 women, the results of anal endosonography were in agreement with those of anal manometry. Sultan et al. reported only lowered resting pressures in women with internal sphincter defects, whereas in women with external sphincter defects no difference was found with regard to maximum squeeze pressures.[17] After subdivision of our study group according to the clinical history, the association of the results of anal endosonography with the results of anal manometry could no longer be demonstrated. Our results of anal endosonography were more in line with the clinical history than with the results of anal manometry, as shown in Table 5.3 and Figure 5.3.

Although the results of both anal manometry and anal endosonography were found to be associated with anal sphincter damage during delivery, our results suggest that in the evaluation of women with anorectal complaints after anal sphincter damage, anal endosonography is more useful than anal manometry. With anal endosonography a sphincter defect can be demonstrated in the vast majority of these women, also in the presence of normal anal resting and squeeze pressures. The possibility to locate a sphincter defect is of clinical importance, because secondary repair is one of the therapeutic options in these patients.[83]

Chapter 6

GENERAL DISCUSSION AND CONCLUSIONS

In the past, the relationship between anal sphincter injury at delivery and subsequent anorectal complaints received little attention in obstetric textbooks. During the last decade awareness has grown that anal sphincter injury at delivery does not always heal properly and women may suffer from long-lasting anorectal complaints afterwards. That awareness formed the basis of the studies presented in this thesis. The results have led to the following considerations and conclusions:

6.1 Anatomy and physiology of the female anal canal

Views on the anatomy of the external sphincter changed when the application of modern techniques of visualization, such as anal endosonography and endo-anal MRI, produced new morphologic data. The analysis of the pertinent literature presented in Chapter 2 shows that:

1. The external anal sphincter muscle is built and functions as a single unit.

Classically, the external anal sphincter was described as consisting of three distinctive parts, each with a distinct function in the physiology of maintaining fecal continence. Later, a two-part anatomical concept came into favor, but recent studies provide evidence that the external sphincter is built as a single unit. Earlier concepts may be explained by thorough anatomical

dissection and variation in anatomy between individuals. The understanding that the external sphincter is built and functions as a single unit has important consequences in case it becomes damaged during vaginal delivery. Based on the three-part concept, it was conceivable that the deeper parts of the external sphincter would function normally when the subcutaneous part of the sphincter was damaged. Now that it has become clear that the external sphincter is built and functions as a single unit, it can be understood that injury at delivery that has not healed properly after primary repair may have a significant influence on the function of the sphincter as a whole. This may explain from the anatomical point of view why the consequences of anal sphincter injury at delivery for fecal continence are far more serious than previously recognized.

2. The puborectalis muscle is part of the levator muscles and its innervation differs from that of the external sphincter.

It has been extensively debated in the anatomical literature whether the puborectalis muscle is to be considered part of the sphincter complex or of the levator muscles. During voluntary contraction it is impossible to contract the external anal sphincter without simultaneous contraction of the puborectalis (and levator ani) muscle. The most recent anatomical and MRI-studies showed a clear distinction between the upper part of the external anal sphincter and the puborectalis muscle. Because the puborectalis muscle forms part of the levator muscles instead of the external anal sphincter, persisting damage of the anal sphincter complex due to delivery may be expected to have no, or only minor, effect on the function of the puborectalis muscle. This may explain why in our study the presence of echo-proven anal sphincter defects in women with a history of anal sphincter injury at delivery was not found

associated with the presence of anorectal complaints. An intact function of the puborectalis muscle may serve as a compensating mechanism for the loss of function of the anal sphincter complex in maintaining fecal continence. Furthermore, besides innervation by the inferior rectal branch of the pudendal nerve, the puborectalis muscle receives innervation from direct sacral branches running on the abdominal surface of the levator muscles. This may have clinical consequences in the treatment of patients with fecal incontinence, with or without a history of anal sphincter injury at delivery, because sacral neurostimulation of these branches at the level of S3 or S4 may serve as a possibility for treatment when anterior sphincter repair has failed or is thought to be useless.

3. The (conjoined) longitudinal muscle may have the same caliber as the external anal sphincter and its fibro-elastic septa end in the subcutaneous part of the external anal sphincter.

Studies using anal endosonography suggested that the (conjoined) longitudinal muscle is a thin muscular structure with no clear function in maintaining fecal continence. However, MRI-studies in vivo as well as recent cadaver studies indicate that the conjoined longitudinal muscle may be as thick as the external anal sphincter. The fact that the fibro-elastic septa of the longitudinal muscle end in the subcutaneous part of the external anal sphincter supports the concept that the longitudinal muscle has a role in normal defecation by everting the subcutaneous part of the sphincter and shortening the anal canal.

6.2 Risk factors for the occurrence of anal sphincter injury at delivery

The population-based study reported in Chapter 3 allows assessment of risk factors of anal sphincter injury at vaginal delivery without apparent selection bias and the limitations of a trial protocol. Our study shows that:

4. Increasing birthweight and longer duration of the second stage of labor are associated with increased risk of anal sphincter injury.

The fact that higher birthweight is associated with an increased risk of third and fourth degree perineal tears seems logical and has been previously reported. It implies that in case of delivery of an expected large infant a balance must be found between the risk of anal sphincter injury, especially with instrumental delivery, and the risks and benefits of a cesarean section.

Our study is the first to show an association of the duration of the second stage of labor with an increasing risk of anal sphincter injury. However, it remains doubtful if the use of strict upper limits for the duration of the second stage will reduce the risk of anal sphincter injury because such an approach may be expected to lead to an increase in assisted vaginal deliveries with an even greater risk of sphincter injury. With regard to anal sphincter injury, the benefits of awaiting spontaneous delivery in the absence of signs of fetal distress may outweigh the risk of instrumental delivery.

5. Mediolateral episiotomy has a protective effect on the occurrence of anal sphincter injury during delivery, when used selectively.

Our study shows a strong protective effect of mediolateral episiotomy against the occurrence of third and fourth degree perineal tears at spontaneous and assisted vaginal deliveries. Two randomized clinical trials, comparing the liberal versus the selective use of mediolateral episiotomies, showed no beneficial effect of the liberal use of mediolateral episiotomies.[33,34] Because of small numbers the statistical power of one of these trials was too low to draw reliable conclusions. In the other trial the episiotomy rate in the group with selective use was comparable to the episiotomy rate in our study group (30.1% versus 34.1%) Other studies have reported an ideal episiotomy rate of approximately 30% in vaginal deliveries, balancing the unnecessary use of mediolateral episiotomies with the risk of anal sphincter injury. On the basis of previous studies a protective effect of selective use of mediolateral episiotomy cannot be ruled out, and is strongly supported by the results of our study.

6. The use of forceps is associated with the largest risk of anal sphincter injury associated with vaginal delivery.

When used exclusively, forceps delivery was found to be associated with a threefold increase in the occurrence of third and fourth degree perineal ruptures. Although the use of vacuum extraction was also found to be associated with a significantly elevated occurrence of anal sphincter injury, the risk was much smaller than that associated with forceps delivery. Some published reports suggest that with the use of vacuum extraction the risk of anal sphincter injury is not different from that in spontaneous deliveries. The fact that we found a small but statistically significant increase in the risk of anal sphincter injury when vacuum extraction was used may be explained by the small sample size of previous studies, with limited statistical power. Therefore, our results support the view that when intervention by

instrumental delivery is indicated and the obstetric situation permits use of forceps or vacuum extractor, vacuum extraction is to be preferred over forceps delivery with respect to the prevention of anal sphincter injury.

7. Nulliparity is an independent risk factor for the occurrence of anal sphincter injury at delivery.

This observation is in line with previous reports, and the relative inelasticity of the perineum in nulliparous women seems to be the logical explanation. It implies that in nulliparous women extra attention should be given to prevention of anal sphincter injury, e.g. by choosing the optimum type of instrumental delivery with regard to the risk of third and fourth degree or by applying mediolateral episiotomy.

6.3 Anal sphincter injury at delivery: functional outcome and risk factors for fecal incontinence

From the retrospective case-control study with matched controls reported in Chapter 4 it can be concluded that:

8. Anal sphincter injury at vaginal delivery, despite primary repair, is strongly associated with subsequent fecal incontinence, urgency and soiling.

The results of our study confirm the results of most earlier studies with regard to the relationship between third and fourth degree perineal ruptures and subsequent anorectal complaints, and with regard to the relative number of women who suffer from anorectal complaints after anal sphincter injury.

Until present three studies, with a median follow-up of more than ten years, addressing the relation of anal sphincter injury at delivery and anorectal complaints have been published.[21,25,74] Two studies, both from the European continent, showed a clear relationship between anal sphincter injury and subsequent anorectal complaints, whereas the other study, from the United States, failed to show such a relationship. The fact that in the latter study no difference in risk was found may be explained by the high rates of anorectal complaints in the control group. It may be hypothesized that this could be due to the widespread use in the Unites States of median episiotomies known to be related with a high risk of unrecognized anal sphincter defects.

9. When anorectal complaints developed they started within one year after delivery in 75% of women with anal sphincter injury, and were still present after a median follow-up of 14 years in more than 90%.

Our finding that complaints usually start shortly after anal sphincter injury has occurred is in line with earlier follow-up studies. These findings contradict earlier reports that suggested that complaints of fecal incontinence often start many years after delivery. This may be explained by the fact that these studies selected women who presented for treatment of fecal incontinence. Most women with these complaints are only prepared to undergo surgical

treatment when their complaints become severe, which may occur after menopause with deterioration of compensatory mechanisms of the pelvic floor.

Our observation that only a minority of women with complaints consulted their physician, in some cases because they were unaware of the possible relationship of their complaints with anal sphincter injury and in others because they were reluctant to discuss the problem, implies that an active approach by obstetricians and midwives is necessary to counsel women with anal sphincter injury, especially in the first year after delivery.

10. The extent of anal sphincter injury is an independent risk factor for the development of fecal incontinence in women with third and fourth degree perineal tears.

Comparison of women with perineal ruptures that involved the anal mucosa with women with only a partial rupture of the anal sphincter muscles showed that the risk of developing fecal incontinence was three times higher in women in the former group (64% versus 21%). Earlier studies on this subject using different subdivisions of sphincter damage with discrete increments of damage showed no relationship between the extent of sphincter damage and subsequent complaints. However, the method of subdivision used in these studies is difficult to use in daily practice and liable to misclassification, and may therefore have led to false conclusions.

The high percentage of women with complaints after anal sphincter injury with involvement of the anal mucosa emphasizes the need for active counseling and follow-up of these women.

11. Subsequent vaginal delivery in women who suffered anal sphincter injury at a previous delivery was not found to be significantly associated with the development of fecal incontinence.

Our study shows that the risk of fecal incontinence following subsequent vaginal delivery after anal sphincter injury at previous vaginal delivery is minor. It is obvious that avoiding vaginal delivery by primary cesarean section will completely prevent anal sphincter injury. However, the findings of our study imply that many primary cesarean sections have to be performed in subsequent pregnancies of women who suffered anal sphincter injury in a preceding vaginal delivery to prevent the development of fecal incontinence in one woman. Whether this is a desirable option is doubtful.

12. Mediolateral episiotomy protects for development of fecal incontinence in primiparous women with anal sphincter injury at delivery.

Our study is the first to report this association. It may be explained by the relationship between damage of the pudendal nerve caused by stretching of this nerve during delivery and the development of fecal incontinence. Performing a mediolateral episiotomy may prevent maximal stretching of the perineum and pelvic floor at the end of the second stage of labor, especially in nulliparous women. The protective effect of episiotomies will be much less in multiparous women, which may be the explanation that the protective effect of mediolateral episiotomy for the development of fecal incontinence could not be demonstrated in these women.

6.4 Anal sphincter injury after vaginal delivery: relationship of anal endosonography and manometry with anorectal complaints

The study on the relationship of anal endosonography and manometry with anorectal complaints after anal sphincter injury at delivery reported in Chapter 5 is the first to address this issue in patients more than ten years after delivery. The study led to the following conclusions:

13. Anal sphincter injury at delivery is significantly associated with the presence of echo-proven anal sphincter defects.

Echo-proven sphincter defects were present in a high proportion of women with and without anorectal complaints after third and fourth degree perineal tears at delivery. The fact that anal sphincter defects were demonstrated in almost 80% of women with a history of anal sphincter injury at delivery confirms the results of previous studies and demonstrates the need for a better method of primary repair in these patients. Evaluation of techniques of primary repair different from the classical end-to-end repair, e.g. the recently proposed technique with overlapping repair of the torn sphincter muscle, is needed. The fact that sphincter defects could be demonstrated in 67% of women without complaints after anal sphincter injury proves that in many women with anal sphincter defects compensatory mechanisms are able to maintain fecal continence for many years. Whether these women are at a higher risk to become incontinent after the menopause compared to women without echo-proven sphincter defects could be the subject of a new study.

14. Median maximum anal resting and squeeze pressures were significantly lower in women with anal sphincter injury at delivery, compared to controls.

In women with a history of a third or fourth degree perineal tear at delivery, the median maximum anal resting pressure was significantly lower in women with anorectal complaints compared to those without complaints. Our findings in the comparison of median maximum resting and squeeze pressures between women with a history of anal sphincter injury with uncomplicated controls are in agreement with the results of earlier studies. In contrast to earlier studies in which no differences in maximum anal resting and squeeze pressures were demonstrated between women with and without anorectal complaints after anal sphincter injury, our study showed a significant difference in median maximum anal resting pressure between these two groups. However, only 40% of women with anorectal complaints after anal sphincter injury had a maximum anal resting pressure below 30 mmHg, the cut-off level of abnormality applied in our study. For that reason, the clinical importance of anal manometry in the evaluation of women with anal sphincter injury at delivery is limited. In contrast, persisting sphincter defects can be detected by anal endosonography in almost 90% of these women, which may have important consequences because anal sphincter repair is one of the therapeutical options in these patients.

SUMMARY

CHAPTER ONE presents a general introduction to the problem of anal sphincter injury at vaginal delivery and its long-term consequences. Knowledge of the development, anatomy and physiology of the anal canal is a prerequisite to understand the consequences of anal sphincter injury.

Previous studies have shown contradictory results with regard to the risk factors for the occurrence of anal sphincter injury at delivery. In the Netherlands the Dutch Perinatal Database (LVR) allows population-based assessment of clinical variables associated with anal sphincter injury at delivery.

There is also disagreement in the existing literature with respect to the relationship between anal sphincter injury, persisting sphincter defects, and anorectal complaints. The introduction of anal endosonography made it possible to reliably demonstrate anal sphincter defects, but its clinical advantage over anal manometry in the assessment of long-term anorectal complaints following vaginal delivery remains disputed.

Based on these considerations, the objectives of this thesis are summarized as follows:

- to analyze the literature on the embryonic development and anatomy of the anal canal and anal sphincter complex, and the role of these structures in the physiology of defecation and fecal continence.
- to assess risk factors for the occurrence of anal sphincter injury at vaginal delivery, using data derived from the Dutch Perinatal Database (LVR).
- to investigate the causative role of anal sphincter injury at delivery in the development of anorectal complaints and urinary incontinence, and to identify obstetric risk factors associated with subsequent fecal incontinence.

- to investigate the relationship of anal endosonography and manometry with anorectal complaints, long after vaginal delivery complicated by anal sphincter injury.

CHAPTER TWO describes the embryonic development, anatomy and physiology of the anal canal. The relationship of the embryonic origin of the different parts of the anal canal with consequences for (patho)physiology in later life is discussed. The anatomy of the anal sphincter complex and its role in defecation and fecal continence are described, with special emphasis on concepts of the anatomy of the external anal sphincter. Because the external sphincter is built and functions as a single unit, persisting structural damage following injury at vaginal delivery enhances the risk of anorectal complaints.

CHAPTER THREE describes a population-based observational study to determine risk factors for the occurrence of anal sphincter injury at delivery. All 284 783 vaginal deliveries of 1994 and 1995 recorded in the Dutch Perinatal Database (LVR) were included in the study. Primiparity, increasing birthweight, and increasing duration of the second stage of labor were found to be associated with an elevated risk of anal sphincter damage. Mediolateral episiotomy appeared to protect against damage to the anal sphincter complex during delivery (OR: 0.21; 95%-CI: 0.20-0.23). All types of assisted vaginal delivery were associated with anal sphincter injury at delivery, with forceps delivery (OR: 3.33; 95%-CI: 2.97-3.74) carrying the largest risk of all assisted vaginal deliveries. Combined use of forceps with other types of assisted vaginal delivery appeared to increase the risk even further. It is concluded that mediolateral episiotomy protects against the occurrence of anal sphincter injury and may thus serve as a method of primary prevention of fecal incontinence. Forceps delivery is a

stronger risk factor for anal sphincter injury than vacuum extraction. If the obstetric situation permits use of either instrument, the vacuum extractor should be the instrument of choice with respect to the prevention of fecal incontinence.

CHAPTER FOUR reports a retrospective case-control study with matched controls to assess the role of anal sphincter injury at delivery in the development of anorectal complaints and urinary incontinence, and to identify obstetric factors associated with subsequent fecal incontinence. A postal questionnaire was used and delivery and operation records were analyzed of all women who underwent primary repair of a third or fourth degree perineal rupture in our hospital between 1971 and 1991 and their controls, matched for date of delivery and parity. In the period studied, 171 women underwent a primary repair, 147 of which returned the questionnaire (86%), compared with 131 of the controls (73%). Analysis was performed on 125 matched pairs with a median follow-up of 14 years. Fecal incontinence was reported by 39 patients and 16 controls (OR: 3.09; 95%-CI: 1.57-6.10). Urinary incontinence was reported by 65 patients and 52 controls (OR:1.46; 95%-CI: 0.91-2.37). Among women with anal sphincter damage, the extent of anal sphincter damage was an independent risk factor for fecal incontinence (OR: 2.54; 95% CI: 1.45-4.45). Subsequent vaginal delivery was not associated with the development of fecal incontinence (OR: 2.32; 95% CI: 0.85-6.33). In primiparous women mediolateral episiotomy protected for fecal incontinence after anal sphincter damage (OR: 0.17; 95% CI: 0.05-0.60).

It is concluded that anal sphincter injury at delivery is significantly associated with subsequent anorectal complaints, but not with urinary incontinence.

CHAPTER FIVE presents a study to assess the relationship of anal endosonography and manometry with anorectal complaints in the evaluation of women a long time after vaginal delivery complicated by anal sphincter injury. Thirty-four women with anal sphincter injury at delivery, 22 with and 12 without anorectal complaints, and 12 controls without anorectal complaints underwent anal endosonography, manometry and rectal sensitivity testing. Complaints were assessed by questionnaire, with a median follow-up of 14 years. Maximum anal resting and squeeze pressures were significantly lower in women with anal sphincter injury ($P < 0.001$ for both). Maximum anal resting pressures were significantly lower in women with anorectal complaints after anal sphincter injury, compared with women without complaints ($P = 0.02$). Results of anal manometry showed a large overlap between all groups. Rectal sensitivity showed no significant differences between the three groups. Persisting defects shown by anal endosonography were significantly more often present in women with anal sphincter injury at delivery, with and without complaints, than in controls ($P < 0.001$ and $P = 0.009$, respectively). No differences in the number of echo-proven sphincter defects were found between women with or without anorectal complaints after anal sphincter injury. Although anal manometry showed significant differences between women with anal sphincter injury and controls, the method is of limited therapeutic importance in the evaluation of these women because of the large overlap in results between groups.

CHAPTER SIX presents a general discussion and conclusions of the findings reported in the previous chapters. Views on the anatomy of the external anal sphincter changed during the last decades and were confirmed when modern techniques of visualization in vivo were applied. The external anal sphincter is built and functions as a single unit. This may explain

why the consequences for fecal incontinence of anal sphincter injury at delivery are far more serious than previously recognized.

Our studies show that nulliparity, increasing birthweight, long duration of the second stage of labor, and all forms of assisted vaginal delivery are associated with an elevated risk of anal sphincter injury. The use of forceps carries the largest risk. Mediolateral episiotomy was shown to protect against anal sphincter injury at delivery. Careful clinical consideration of assisted vaginal delivery as a factor of elevated risk and mediolateral episiotomy as a factor of reduced risk may contribute to prevention of anal sphincter injury at delivery. On the basis of the results of our studies the issue whether or not a cesarean section should be offered to women with anal sphincter injury in their next pregnancy to prevent the development of anorectal complaints remains debatable.

The observation that persisting sphincter defects are often demonstrated by anal endosonography in women a long time after anal sphincter injury at delivery, independent of the presence or absence of anorectal complaints, demonstrates the need for a improved technique of primary repair. The large overlap of the results of anal manometry between women with and without anorectal complaints after sphincter injury and controls limits the clinical applicability of this technique in the follow-up of women with anal sphincter injury at delivery. Anal endosonography provides reliable information on the location of a sphincter defect and is therefore to be considered the most important technique in the evaluation of women with a history of anal sphincter injury at delivery.

SAMENVATTING

HOOFDSTUK EEN beschrijft een introductie tot het probleem van beschadiging van de anale sfincters tijdens de vaginale bevalling en de klinische gevolgen op lange termijn. Kennis van de embryonale ontwikkeling, anatomie en fysiologie van het anale kanaal is een voorwaarde voor een goed begrip van de gevolgen van beschadiging van de anale sfincters. Eerdere onderzoeken hebben tegenstrijdige resultaten opgeleverd ten aanzien van risicofactoren voor beschadiging van de anale sfincters tijdens de vaginale baring. De Landelijke Verloskundige Registratie (LVR) maakt het mogelijk om in Nederland onderzoek uit te voeren naar klinische variabelen gerelateerd aan beschadiging van de anale sfincters tijdens de vaginale baring.

Het verband tussen beschadiging van de anale sfincters tijdens de vaginale baring en daaropvolgende anorectale klachten staat in de huidige literatuur ter discussie. Ook over de relatie van persisterende defecten van de anale sfincters na beschadiging tijdens de baring met anorectale klachten bestaat verschil van inzicht. Met de introductie van endo-anale echografie is het mogelijk geworden om defecten van de anale sfincters betrouwbaar aan te tonen. Over het voordeel van endo-anale echografie boven anale manometrie voor het beoordelen van anorectale klachten op lange termijn na beschadiging van de anale sfincters tijdens de baring zijn de meningen verdeeld.

Op basis van deze overwegingen zijn de volgende doelstellingen van dit proefschrift geformuleerd:

- een overzicht geven van de literatuur over de embryonale ontwikkeling en anatomie van het anale kanaal en het complex van anale kringspieren, en de rol van deze structuren in de fysiologie van defaecatie en handhaving van fecale continentie

- het vaststellen van risicofactoren voor beschadiging van de anale kringspieren tijdens de vaginale baring met behulp van gegevens verkregen uit de Landelijke Verloskundige Registratie (LVR).
- het onderzoeken van de bijdrage van beschadiging van de anale kringspieren tijdens de vaginale baring aan het ontstaan van fecale en urine-incontinentie en het identificeren van obstetrische risicofactoren voor het ontwikkelen van fecale incontinentie.
- het onderzoeken van het verband tussen endo-anale echografie, anale manometrie en anorectale klachten, lang nadat beschadiging van de anale kringspieren tijdens de baring is opgetreden.

HOOFDSTUK TWEE behandelt de embryonale ontwikkeling, anatomie en fysiologie van het anale kanaal. De relatie tussen de embryonale oorsprong van de diverse delen van het anale kanaal en (patho)fysiologie op latere leeftijd wordt besproken. De anatomie van het anale sfinctercomplex en de rol hiervan bij defaecatie en handhaving van de fecale continentie wordt beschreven, met nadruk op de opvattingen betreffende de anatomie van de externe anale sfincter. Omdat de externe anale sfincter is gebouwd en functioneert als één geheel verhoogt blijvende beschadiging na trauma tijdens de vaginale baring de kans op anorectale klachten.

HOOFDSTUK DRIE beschrijft een observationeel onderzoek in de Nederlandse populatie naar risicofactoren voor beschadiging van de anale sfincters tijdens de vaginale baring. Alle 284.783 vaginale bevallingen in 1994 en 1995, geregistreerd in de Landelijke Verloskundige Registratie (LVR), werden geanalyseerd. Primipariteit, toenemend geboortegewicht en

toename van de uitdrijvingsduur vertoonden een significante samenhang met een stijging van het risico van beschadiging van de anale sfincters. Mediolaterale episiotomie bleek te beschermen tegen beschadiging van de anale sfincters (OR: 0,21; 95%-BI: 0,20-0,23). Alle vormen van kunstverlossing waren geassocieerd met een toename van het risico van beschadiging van de anale sfincters tijdens de vaginale baring, waarbij de forceps het risico het sterkst bleek te verhogen (OR: 3,33; 95%-BI: 2,97-3,74). Forcipale extractie in combinatie met andere methoden van kunstverlossing deed het risico nog verder toenemen.
Geconcludeerd wordt dat de mediolaterale episiotomie beschermt tegen het optreden van beschadiging van de anale sfincters en kan dienen als methode van primaire preventie voor het optreden van fecale incontinentie. In het licht van preventie voor het optreden van fecale incontinentie verdient vacuümextractie de voorkeur boven het gebruik van de forceps, als de verloskundige omstandigheden kunstverlossing noodzakelijk maken en het gebruik van beide instrumenten toestaan.

HOOFDSTUK VIER behandelt een retrospectief cohortonderzoek met gepaarde controlepatiënten. Het onderzoek werd uitgevoerd om de rol te bepalen van beschadiging van de anale sfincters tijdens de vaginale baring bij het ontstaan van anorectale klachten en urine-incontinentie én om obstetrische risicofactoren te identificeren voor het ontwikkelen van fecale incontinentie.
Alle 171 vrouwen bij wie in het Ikazia Ziekenhuis Rotterdam tussen 1971 en 1991 anale sfincterschade onmiddellijk na de baring operatief werd hersteld ontvingen een schriftelijke enquête. Vrouwen zonder beschadiging van de anale sfincters, gematched voor datum van bevalling en pariteit, dienden als controles en ontvingen dezelfde enquête. Van beide groepen werden gegevens van de bevalling en de operatieve ingreep geanalyseerd. Analyse werd

uitgevoerd op 125 patiënt-controle paren met een mediane follow-up van 14 jaar. Fecale incontinentie werd gemeld door 39 vrouwen met beschadiging van de anale sfincters en 16 controles (OR:3,09; 95%-BI: 1,57-6,10). Vijfenzestig vrouwen met sfincterbeschadiging en 52 controles gaven aan last te hebben van incontinentie voor urine (OR: 1,46; 95%-BI: 0,91-2,37). De uitgebreidheid van het sfincterletsel bleek een onafhankelijke risicofactor te vormen voor het ontwikkelen van fecale incontinentie (OR: 2,54; 95%-BI: 1,45-4,45). Een vaginale baring volgend op de baring waarin beschadiging van de anale sfincters was opgetreden was niet geassocieerd met het ontwikkelen van fecale incontinentie (OR: 2,32; 95%-BI: 0,85-6,33). Bij primiparae bleek een mediolaterale episiotomie te beschermen tegen het optreden van fecale incontinentie (OR: 0,17; 95%-BI: 0,05-0,60). Uit het onderzoek wordt geconcludeerd dat klachten van fecale incontinentie significant samenhangen met letsel van de anale sfincters tijdens de vaginale baring, in tegenstelling tot incontinentie voor urine.

HOOFDSTUK VIJF beschrijft een onderzoek naar het verband tussen de resultaten van endo-anale echografie en anale manometrie en anorectale klachten bij vrouwen met een voorgeschiedenis van beschadiging van de anale sfincters tijdens de baring, lang na het optreden van de beschadiging. Vierendertig vrouwen met een voorgeschiedenis van anale sfincterschade, 22 met en 12 zonder klachten, en 12 controles met een ongecompliceerde vaginale baring in de voorgeschiedenis en geen anorectale klachten werden onderzocht met behulp van endo-anale echografie en anale manometrie. Het bestaan van anorectale klachten werd vastgesteld door middel van een schriftelijke enquête, met een mediane follow-up van 14 jaar.

Zowel de maximale anale rustdruk als de maximale knijpdruk was significant lager bij vrouwen met een voorgeschiedenis van beschadiging van de anale sfincters ($p < 0,001$ voor

beide). In de patiëntengroep was de maximale anale rustdruk significant lager bij vrouwen met anorectale klachten, vergeleken met vrouwen zonder klachten. ($p = 0,02$).
Vergelijking van de resultaten van anale manometrie toonde een grote overlap tussen de drie groepen. Voor wat betreft de sensitiviteit van het rectum waren geen significant verschillen aantoonbaar tussen de drie groepen. Endo-anale echografie liet bij vrouwen met en zonder anorectale klachten na beschadiging van de anale sfincters significant meer persisterende defecten in de sfincters zien dan bij vrouwen met een ongecompliceerde voorgeschiedenis ($p < 0,001$ resp. $p = 0,009$). Het aantal sfincterdefecten verschilde niet tussen vrouwen met en zonder anorectale klachten na beschadiging van de sfincters tijdens de baring. De grote overlap tussen de resultaten van anale manometrie inde drie groepen beperkt de toepasbaarheid van deze techniek voor evaluatie van vrouwen met beschadiging van de anale sfincters tijdens de vaginale baring.

HOOFDSTUK ZES geeft een algemene discussie en conclusies van de resultaten beschreven in de voorgaande hoofdstukken.
De visie op de anatomie van de externe anale sfincter is veranderd gedurende de laatste decennia en werd bevestigd met behulp van moderne beeldvormende technieken. De externe anale sfincter is gebouwd en functioneert als één geheel. Dit kan verklaren waarom beschadiging van de anale sfincters tijdens de baring meer nadelige gevolgen heeft voor de fecale continentie dan vroeger werd aangenomen.
Onze onderzoeken tonen aan dat primipariteit, alle vormen van kunstverlossing en toename van geboortegewicht en uitdrijvingsduur zijn geassocieerd met een verhoogd risico voor het optreden van beschadiging van de anale sfincters. De kans op letsel van de anale sfincters is het grootst bij een forcipale extractie. Een mediolaterale episiotomie biedt bescherming tegen

beschadiging van de anale sfincters tijdens de baring. Zorgvuldige afweging van de vaginale kunstverlossing als risicoverhogende factor en de mediolaterale episiotomie als beschermende factor voor het optreden van beschadiging van de anale sfincters kan bijdragen aan de preventie van deze letsels. De vraag of aan vrouwen met beschadiging van de anale sfincters in hun volgende zwangerschap een sectio caesarea dient te worden aangeboden om fecale incontinentie te voorkomen kan op basis van onze onderzoeken niet worden beantwoord. Persisterende defecten van de anale sfincters, aangetoond door middel van endo-anale echografie, komen vaak voor bij vrouwen met een voorgeschiedenis van beschadiging van de anale sfincters tijdens de baring, onafhankelijk van de aanwezigheid van klachten. Dit toont de noodzaak aan van het ontwikkelen van een verbeterde chirurgische techniek voor het primair herstellen van deze beschadigingen. De resultaten van anale manometrie bij vrouwen met en zonder klachten na beschadiging van de sfincters en die bij controles zonder sfincterbeschadigigng in de voorgeschiedenis tonen een grote overlap. Dit beperkt de klinische toepasbaarheid van deze methode voor de follow-up van vrouwen met beschadiging van de anale sfincters tijdens de baring. Met behulp van endo-anale echografie kunnen eventuele sfincterdefecten betrouwbaar worden gelokaliseerd. Endo-anale echografie is daarom te beschouwen als de belangrijkste methode van onderzoek voor de evaluatie van vrouwen met een voorgeschiedenis van letsel van de anale sfincters tijdens de baring.

REFERENCES

1. Thacker SB, Banta HB. Benefits and risks of episiotomy: an interpretive review of the English language literature. Obstet Gynecol Surv 1983;38:322-38.
2. Woolley RJ. Benefits and risks of episiotomy: a review of the English-language literature since 1980. Part 1. Obstet Gynecol Surv 1995;50:806-35.
3. Cunningham FG, MacDonald PC, Gant NF, Leveno KJ, Gilstrap LC. Williams Obstetrics. 19th ed. East Norwalk (Conn): Appleton & Lange; 1993.
4. Heineman MJ, Bleker OP, Evers JLH, Heintz APM, editors. Obstetrie en Gynaecologie, de voortplanting van de mens. 3rd rev. ed. Maarssen (the Netherlands): Elsevier/Bunge;1999.
5. Scott JR, Di Saia PJ, Hammond CB, Spellacy WK. Danforth's Obstetrics and Gynecology. 8th ed. Philadelphia (PA): Lippincott Williams & Wilkins; 1999.
6. Dalley AF. The riddle of the sphincters. The morphophysiology of the anorectal mechanism reviewed. Am Surg 1987;53:298-306
7. Leigh RJ, Turnberg LA. Fecal incontinence: The unvoiced symptom. Lancet 1982;i:1349-51.
8. Johanson JJ, Lafferty J. Epidemiology of fecal incontinence: the silent affliction. Am J Gastroenterol 1996;91:33-6.
9. Madoff RD, Williams JG, Caushaj PF. Fecal incontinence. N Engl J Med. 1992;326:1002-7.
10. Jorge JMN, Wexner SD. Etiology and management of fecal incontinence. Dis Colon Rectum 1993;36:77-97.

11. Law PJ, Bartram CI. Anal endosonography: technique and normal anatomy. Gastrointestinal Radiol 1989;14:349-53.

12. Sultan AH, Nicholls, Kamm MA, Hudson CN, Beynon J, Bartram CI. Anal endosonography and correlation with *in vitro* and *in vivo* anatomy. Br J Surg 1993;80:508-11.

13. Sultan AH, Kamm MA, Talbot IC, Nicholls RJ, Bartram CI. Anal endosonography for identifying external sphincter defects confirmed histologically. Br J Surg 1994;81:463-65.

14. Burnett SJD, Spence-Jones C, Speakman CTM, Kamm MA, Hudson CN, Bartram CI. Unsuspected sphincter damage following childbirth revealed by anal endosonography. Br J Radiol 1991;64:225-27.

15. Felt-Bersma RJF, Cuesta MA, Koorevaar M, Strijers RLM, Meuwissen SGM, Dercksen EJ, et al. Anal endosonography: relationship with anal manometry and neurophysiologic tests . Dis Colon Rectum 1992;35:944-49.

16. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal sphincter disruption during vaginal delivery. N Engl J Med 1993;329:1905-11.

17. Sultan AH, Kamm MA, Hudson CN, Bartram CI. Third degree obstetric anal sphincter tears: risk factors and outcome of primary repair. BMJ 1994;308:887-91.

18. Poen AC, Felt-Bersma RJF, Strijers RLM, Dekker GA, Cuesta MA, Meuwissen SGM. Third-degree obstetric perineal tear: long-term clinical and functional results after primary repair. Br J Surg 1998;85:1433-38.

19. Crawford LA, Quint EH, Pearl ML, DeLancey JOL. Incontinence following rupture of the anal sphincter during delivery. Obstet Gynecol 1993;82:527-31.

20. Uustal Fornell EK, Berg G, Halböök O, Matthiesen LS, Sjödahl R. Clinical consequences of anal sphincter rupture during vaginal delivery. J Am Coll Surg 1996;183:553-58.

21. Nygaard IE, Rao SSC, Dawson JD. Anal incontinence after anal sphincter disruption: a 30-year retrospective cohort study. Obstet Gynecol 1997;89: 896-901.

22. Gjessing H, Backe B, Sahlin Y. Third degree obstetric tears: outcome after primary repair. Acta Obstet Gynecol Scand 1998;77:736-40.

23. Møller Bek K, Laurberg S. Risk of anal incontinence from subsequent vaginal delivery after a complete obstetric anal sphincter tear. Br J Obstet Gynaecol 1992;99:724-26.

24. Walsh CJ, Mooney EF, Upton GJ, Motson RW. Incidence of third degree perineal tears in labour and outcome after primary repair. Br J Surg 1996;83:218-21.

25. Haadem K, Gudmundsson S. Can women with intrapartum rupture of anal sphincter still suffer after-effects two decades later? Acta Obstet Gynecol Scand 1997;76:601-03

26. Møller Bek K, Laurberg S. Intervention during labor: risk factors associated with complete tear of the anal sphincter. Acta Obstet Gynecol Scand 1992;71:520-24.

27. Poen AC, Felt-Bersma RJF, Dekker GA, Devillé W, Cuesta MA, Meuwissen SGM. Third degree obstetric perineal tears: risk factors and the preventive role of mediolateral episiotomy. Br J Obstet Gynaecol 1997;104:563-66.

28. Brink Henriksen T, Møller Bek K, Hedergaard M, Secher NJ. Episiotomy and perineal lesions in spontaneous vaginal deliveries. Br J Obstet Gynaecol 1992;99:950-54.

29. Green JR, Soohoo SL. Factors associated with rectal injury in spontaneous deliveries. Obstet Gynecol 1989;73:732-38.

30. Combs CA, Robertson PA, Laros RK. Risk factors for third-degree and fourth-degree perineal lacerations in forceps and vacuum deliveries. Am J Obstet Gynecol 1990;163:100-04.

31. Shiono P, Klebanoff MA, Carey JC. Midline episiotomies: more harm than good? Obstet Gynecol 1990;75:765-70.

32. Wilcox LS, Strobino DM, Baruffi G, Dellinger WS. Episiotomy and its role in the incidence of perineal lacerations in a maternity center and tertiary hospital obstetric service. Am J Obstet Gynecol 1989;160:1047-52.

33. Sleep J, Grant A, Garcia J, Elbourne D, Spencer J, Chalmers I. West Berkshire perineal management trial. BMJ 1984;289:587-90.

34. Argentine Episiotomy Trial Collaborative Group. Routine vs. selective episiotomy: a randomised controlled trial. Lancet 1993;342:1517-18.

35. Johanson RB, Rice C, Doyle M, Arthur J, Anyamwu L, Ibrahim J, et al. A randomised prospective study comparing the new vacuum extractor policy with forceps delivery. Br J Obstet Gynaecol 1993;100:524-30.

36. Anthony S, Buitendijk SE, Zondervan KT, van Rijssel EJC, Verkerk PH. Episiotomies and the occurrence of severe perineal lacerations. Br J Obstet Gynaecol 1994;101:1064-67.

37. Van Hemel OJ, Elferink-Stinkens PM, Brand R. How to compare and report department specific mortality rates for peer review using the Perinatal Database of the Netherlands. Eur J Obstet Gynecol Reprod Biol 1994;56:1-7.

38. Kamm MA. Obstetric damage and fecal incontinence. Lancet 1994;344: 730-33.

39. de Leeuw JW, Vierhout ME, Struijk PC, Hop WCJ, Wallenburg HCS. Anal sphincter damage after vaginal delivery: functional outcome and risk factors for fecal incontinence. Acta Obstet Gynecol Scand (in press).

40. Godlewski G, Prudhomme M. Embryology and anatomy of the anorectum. Basis for surgery. Surg Clin North Am 2000;80:319-43.

41. Moore KL. The Developing Human. Clinically oriented embryology. 3rd ed. Philadelphia: Saunders, 1982.

42. Jorge JMN, Wexner SD. Anatomy and physiology of the rectum and anus. Eur J Surg. 1997;163:723-31.

43. Skandalakis JE, Gray SW, Ricketts R. The colon and rectum. In: Skandalakis JE, Gray SW. Embryology for surgeons. 2nd ed. Baltimore, Md: Williams and Wilkins, 1994

44. Williams PL, Warwick R, editors. Gray's anatomy. 36th ed. Edinburgh: Churchill Livingstone, 1980.

45. Hollinshead H. Anatomy for surgeons, vol. 2: the thorax, abdomen and pelvis. 2nd ed. New York: Harper and Row, 1971

46. Goligher JC, Duthie HL, Nixon HH. Surgical anatomy and physiology of the colon, rectum and anus In: Surgery of the Anus, Rectum and Colon. 5th ed. London: Ballière Tindall, 1984; 1-47.

47. Skandalakis JE, Gray SW, Rowe JS Jr. Anatomical Complications in General Surgery. New York, NY: McGraw-Hill Inc; 1983.

48. Wood BA, Kelly AJ. Anatomy of the anal sphincters and pelvic floor. In: Henry MM, Swash M, eds. Coloproctology and the pelvic floor. 2nd ed. Oxford: Butterworth Heineman, 1992; 3-19.

49. Matzel KE, Schmidt RA, Tanagho EA. Neuroanatomy of the striated muscular anal continence mechanism, implications for the use of neurostimulation. Dis Colon Rectum 1990;33:666-73.

50. Gerdes B, Köhler HH, Stinner B, Barth PJ, Celik I, Rothmund M. The longitudinal muscle of the anal canal. Chirurg 1997;68:1281-85.

51. Hussain SM. Imaging of the anal sphincter complex (dissertation). Rotterdam: Erasmus University, 1996.

52. Oh C, Kark AE. Anatomy of the external sphincter. Br J Surg 1972;59(9):717-23.

53. Ayoub SF. Anatomy of the external sphincter in man. Acta Anat (Basel) 1979;105:25-36.

54. Maxwell PR, Heriot AG, Davies DC, Kumar D. Anorectal sensation and Continence. Scand J Gastroenterol 1999;34:113-6.

55. Duthie GS, Bartolo DCC. Faecal continence and defaecation. In: Henry MM, Swash M, eds. Coloproctology and the pelvic floor. 2nd ed. Oxford: Butterworth Heineman, 1992;86-97.

56. Sangwan YP, Solla JA. Internal anal sphincter: advances and insights. Dis Colon Rectum 1998;41:1297-1311.

57. Parks AG. Anorectal incontinence. Proc R Soc Med 1975;68:681-90.

58. Tetzschner T, Sørensen M, Lose G, Christiansen J. Anal and urinary incontinence in women with obstetric anal sphincter rupture. Br J Obstet Gynaecol 1996;103:1034-40.

59. Nielsen MB, Hauge C, Rasmussen OØ, Pedersen JF, Christiansen J. Anal endosonographic findings in the follow-up of primarily sutured sphincteric ruptures. Br J Surg 1992;79:104-06.

60. Ryhammer AM, Møller Bek K, Laurberg S. Multiple vaginal deliveries increase the risk of permanent incontinence of flatus and urine in normal premenopausal women. Dis Colon Rectum 1995;208:1206-09.

61. MacArthur C, Bick DE, Keighley MRB. Faecal incontinence after childbirth. Br J Obstet Gynecol 1997; 104:46-50.

62. Connolly AM, Thorp JM. Childbirth-related perineal trauma: Clinical significance and prevention. Clin Obstet Gynecol 1999;42: 820-35.

63. Chaliha C, Sultan AH, Stanton SL. Changes in the pelvic floor following childbirth. Fetal Matern Med Rev 1999;11:41-54.

64. Haadem K, Ohrlander S, Lingman G. Long-term ailments due to anal sphincter rupture caused by delivery- a hidden problem. Eur J Obstet Gynecol Reprod Biol 1988;27:27-32.

65. Sørensen SM, Bondensen H, Istre O, Vilmann P. Perineal rupture following vaginal delivery, long-term consequences. Acta Obstet Gynecol Scand 1988;67:315-18.

66. Go PM, Dunselman GAJ. Anatomic and functional results of surgical repair after total perineal rupture at delivery. Surg Gynecol Obstet 1988;166:121-24.

67. Haadem K, Dahlström JA, Lingman G. Anal sphincter function after delivery: a prospective study in women with sphincter rupture and controls. Eur J Obstet Gynecol Reprod Biol 1990;35:7-13.

68. Sørensen M, Tetzschner T, Rasmussen OØ, Bjarnesen J, Christiansen J. Sphincter rupture in childbirth. Br J Surg 1993;80:392-94.

69 Franz HB, Benda N, Gonser M, Bäckert IT, Jehle EC. Clinical effects of childbirth with median episiotomy and anal sphincter injury on fecal incontinence of primiparous women. Zentralbl Chir. 1998;123:218-22. (German)

70. Goffeng AR, Andersch B, Andersson M, Berndtsson, Hulten L, Öresland T. Objective methods cannot predict anal incontinence after primary repair of extensive anal tears. Acta Obstet Gynecol Scand 1998;77:439-43.

71. Wood J, Amos L, Rieger N. Third degree anal sphincter tears: risk factors and outcome. Aust NZ J Obstet Gynecol 1998;38:414-17.

72. Zetterström JP, López A, Anzén B, Dolk A, Norman M, Mellgren A. Anal incontinence after vaginal delivery. Br J Obstet Gynaecol 1999;106:324-30.

73. Kammerer-Doak DN, Wesol AB, Rogers RG, Dominguez CE, Dorin MH. A prospective cohort study of women after primary repair of obstetric anal sphincter laceration. Am J Obstet Gynecol 1999;181:1317-23.

74. Sangalli MR, Floris L, Faltin D, Weil A. Anal incontinence in women with third or fourth degree perineal tears and subsequent vaginal deliveries. Aust NZ J Obstet Gynecol 2000;40:244-48.

75. Signorello LB, Harlow BL, Chekos AK, Repke JT. Midline episiotomy and anal incontinence: retrospective cohort study. BMJ 2000;320:86-90.

76. Allen RE, Hosker GL,Smith ARB, Warrel DW. Pelvic floor damage and childbirth: a neurophysiologic study. Br J Obstet Gynaecol 1990; 97:770-79.

77. Nelson R, Norton N, Cautley E, Furner S. Community-based prevalence of anal incontinence. JAMA 1995;274:559-61.

78. Roberts RO, Jacobsen SJ, Reilly WT, Pemberton JH, Lieber MM, Talley NJ. Prevalence of combined fecal and urinary incontinence: a community-based study. J Am Geriatr Soc 1999;47:837-41.

79. Rao SSC, Patel RS. How useful are manometric tests of anorectal function in the management of defecation disorders? Am J Gastroenterol 1997;92:469-75.

80. Sentovich SM, Blatchford GJ, Rivela LJ, Lin K, Thorson AG, Christensen MA. Diagnosing anal sphincter injury with transanal ultrasound and manometry. Dis Colon Rectum 1997;40:1430-34.

81. Sultan AH, Kamm MA, Talbot IC, Nicholls RJ, Bartram CI. Anal endosonography for identifying external sphincter defects confirmed histologically. Br J Surg 1994;81:463-65.

82. Tetzschner T, Sørensen M, Rasmussen OØ, Lose G, Christansen J. Pudendal nerve damage increases the risk of fecal incontinence in women with anal sphincter rupture after childbirth. Acta Obstet Gynecol Scand 1995;74:434-40.

83. Cook TA, Mortensen NJ. Management of fecal incontinence following obstetric injury. Br J Surg 1998;85:293-99.

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CURRICULUM VITAE

1963	Born in Rossum, the Netherlands
1975 – 1981	Atheneum-B, OSG "Buys Ballot", Zaltbommel
1981 – 1988	Medical School, State University Limburg, Maastricht
1988 – 1992	Residency in Obstetrics and Gynecology, St. Elisabethkliniek Heerlen, Ikazia Ziekenhuis Rotterdam, and Reinier de Graaf Gasthuis Delft
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1998 – present	Consultant in Obstetrics and Gynecology, Ikazia Ziekenhuis and Havenziekenhuis, Rotterdam

APPENDIX A

ENQUETEFORMULIER

ONGEWILD VERLIES VAN URINE EN ONTLASTING

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PERSOONLIJKE GEGEVENS

1 NAAM: .....

2 HUIDIG ADRES: .....

.....

3 GEBOORTEDATUM: .....

4 HUISARTS: .....

5 JAAR BEVALLING: .....

6 TELEFOONNUMMER: .....

INDIEN BOVENSTAANDE GEGEVENS NIET JUIST OF ONVOLLEDIG ZIJN, WILT U DAN EVENTUELE CORRECTIES HIERONDER AANGEVEN ?:

1 NAAM: .....

2 HUIDIG ADRES: .....

3 GEBOORTEDATUM: .....

4 HUISARTS: .....

5 JAAR BEVALLING: .....

6 TELEFOONNUMMER: .....

7 Bent U sinds de onder 5 genoemde bevalling nog een of meerdere keren bevallen ?

___ Ja, namelijk nog ... keer
(Ga door met vraag 8)
___ Nee (Ga door naar vraag 12)

Gegevens 1e bevalling nadien

8 Datum: .....

9 Onder leiding van: Huisarts/Verloskundige/Gynaecoloog*

Naam: .....

en/of Naam Ziekenhuis: .....

Gegevens 2e bevalling nadien

10 Datum: .....

11 Onder leiding van: Huisarts/Verloskundige/Gynaecoloog*

Naam: .....

en/of Naam Ziekenhuis: .....

12 Hoe vaak bezoekt U het toilet voor ontlasting ?

___ Meer dan 5 x per dag
___ 1 tot 5 x p. dag
___ 1 tot 6 x p. week
___ Minder dan 1 x p. week

13 Hoe ziet uw normale ontlasting eruit ?

___ Waterdun
___ Brijig
___ Vast
___ Hard

14 Gebruikt U weleens laxeermiddelen om uw ontlasting op gang te houden ?

___ Nooit
___ Minder dan 1 x per week
___ 1 tot 6 x p. week
___ Dagelijks

15 Gebruikt U weleens dieetmiddelen (bijvoorbeeld sennathee. pruimen e.d.) om uw ontlasting op gang te houden ?

___ Nooit
___ Minder dan 1 x per week
___ 1 tot 6 x per week
___ Dagelijks

16 Hebt U wel eens het idee dat U niet alle ontlasting in een keer kwijt raakt bij toiletbezoek ?

___ Nooit
___ Soms
___ Vaak
___ Altijd

17 Moet U weleens uw vingers gebruiken om uw ontlasting kwijt te raken ?

___ Nooit
___ Soms
___ Vaak
___ Altijd

18 Hebt U sinds de onder 5 genoemde bevalling last gehad of nog steeds last van het volgende probleem ?:
Het niet in staat zijn om de ontlasting meer dan vijf minuten op te houden, nadat U aandrang hebt bemerkt ?

___ Ja (Ga door met vraag 19)
___ Nee (Ga door met vraag 22)

19 Wanneer zijn deze klachten begonnen ?

___ Reeds voor de bevalling
___ Binnen 3 maanden na de bevalling
___ 3 tot 12 maanden na de bevalling
___ Meer dan 1 jaar na de bevalling

20 Hoe lang duurden deze klachten ?

___ Minder dan 6 weken
___ 6 weken tot 12 maanden
___ Meer dan 1 jaar
___ Tot op heden

21 Hoe vaak hebt U last (gehad) van deze klachten ?

___ Minder dan 1 x per week
___ 1 tot 6 x per week
___ 1 tot 5 x per dag
___ Meer dan 5 x per dag

22 Hebt U sinds de onder 5 genoemde bevalling last gehad of nog steeds last van het volgende probleem ?:
Het verlies van ontlasting zonder dat U dit zelf merkt op het moment dat dit werkelijk gebeurt (U bemerkt 's avonds een vieze plek in uw onderbroek)

___ Ja (Ga door met vraag 23)
___ Nee (Ga door met vraag 26)

23 Wanneer zijn de klachten begonnen ?

___ Reeds voor de bevalling
___ Binnen 3 maanden na de bevalling
___ 3 tot 12 maanden na de bevalling
___ Meer dan 1 jaar na de bevalling

24 Hoe lang duurden deze klachten ?

___ Minder dan 6 weken
___ 6 weken tot 12 maanden
___ Meer dan 1 jaar
___ Tot op heden

25 Hoe vaak hebt U last (gehad) van deze klachten ?

___ Minder dan 1 x per week
___ 1 tot 6 x per week
___ 1 tot 5 x per dag
___ Meer dan 5 x per dag

26 Hebt U sinds de onder 5 genoemde bevalling last gehad of nog steeds last van het volgende probleem ?:
Het verlies van ontlasting of "windjes" op momenten of plaatsen dat U dat niet wilt

___ Ja (Ga door met vraag 27)
___ Nee (Ga door met vraag 31)

27 Als U hiervan last hebt, verliest U:

___ Alleen "windjes"
___ Alleen "windjes" of dunne ontlasting, maar geen vaste ontlasting
___ Alle soorten ontlasting

28 Wanneer zijn de klachten begonnen ?

___ Reeds voor de bevalling
___ Binnen 3 maanden na de bevalling
___ 3 tot 12 maanden na de bevalling
___ Meer dan 1 jaar na de bevalling

29 Hoe lang duurden deze klachten ?

___ Minder dan 6 weken
___ 6 weken tot 12 maanden
___ Meer dan 1 jaar
___ Tot op heden

30 Hoe vaak hebt U last (gehad) van deze klachten ?

___ Minder dan 1 x per week
___ 1 tot 6 x per week
___ 1 tot 5 x per dag
___ Meer dan 5 x per dag

DE VRAGEN 31 TOT EN MET 42 ALLEEN INVULLEN INDIEN VRAAG 18, 22 OF 26 MET JA HEBT BEANTWOORD. INDIEN U DEZE DRIE VRAGEN MET NEE HEBT BEANTWOORD, KUNT U DOORGAAN MET VRAAG 40

31 Hebt U al eens een behandeling zelf geprobeerd of ondergaan voor uw klachten van incontinentie voor ontlasting ?

___ Ja (Ga door met vraag 32)
___ Nee (Ga door met vraag 35)

32 Welke therapie heeft U zelf geprobeerd of ondergaan ?
(meerdere antwoorden zijn mogelijk)

___ Bekkenbodemoefeningen zelf
___ Bekkenbodemoefeningen o.l.v. (fysio)therapeut
___ Dieetmaatregelen
___ Operatie(s)

33 Indien U een operatie onderging:
In welk jaar en in welk ziekenhuis onderging U deze operatie ?

Jaar (evt. datum): .....

Ziekenhuis: .....

34 Welke specialist was uw behandelaar (bijv. chirurg, gynaecoloog etc.) ?

Specialist: .....

Naam arts: .....

35 Hebt U, sinds U moeite hebt gekregen met het ophouden van ontlasting, gemerkt dat U:
Bepaalde lichamelijke activiteiten (bijv. sport)
vermijdt, om minder problemen te hebben met het ophouden van ontlasting ?

___ Ja
___ Nee

Zo ja, ...
evt. toelichting: .....

36 Hebt U, sinds U moeite hebt gekregen met het ophouden van ontlasting, gemerkt dat U:
Bepaalde sociale activiteiten (bijv. feestjes) vermijdt, omdat U bang bent dat anderen uw problemen opmerken ?

___ Ja
___ Nee

Zo ja, ...
evt. toelichting:
.....

37 Belemmeren uw problemen met het ophouden van ontlasting U in uw dagelijks werk ?

___ Ja
___ Nee

Zo ja, ...
evt. toelichting
.....

38 Gebruikt U hygienische verbandmiddelen voor het opvangen van ontlasting ?

Zo ja, welke ? (meerdere antwoorden mogelijk).

___ Geen
___ Inlegkruisjes
___ Maandverband
___ Incontinentieverband
___ Andere: .....

39 Zo ja, hoeveel van deze verbandmiddelen gebruikt U ?

___ Maximaal 1 per dag
___ 1 tot 5 per dag
___ Meer dan 5 per dag

40 Heeft U na de onder 5 genoemde bevalling een of meerdere sporten beoefend ?

___ Ja (Ga door met vraag 41)
___ Nee (Ga door met vraag 42)

41 Welke sport(en) heeft U in welke periode beoefend ?

Sport: ..... van 19... tot 19...

Sport: ..... van 19... tot 19...

Sport: ..... van 19... tot 19...

42 Oefent U een beroep uit ? Zo ja, welk beroep ?

___ Ja, namelijk: .....
___ Nee

43 Hebt U een of meerdere van de volgende ziekten (of gehad), zo ja: Sinds wanneer ?

___ Suikerziekte, sinds 19....
___ Multiple Sclerose, sinds 19....
___ Hernia (Nucleus Polposi), sinds 19...

44 Gebruikt U medicijnen ?

___ Ja (Ga door met vraag 45)
___ Nee (Ga door met vraag 46)

45 Welke medicijnen gebruikt U ?

.....

.....

.....

46 Verliest U wel eens urine op momenten of plaatsen dat U dat niet wilt ?

___ Ja (Ga door met vraag 47)
___ Nee (Ga door met vraag 54)

47 Wanneer zijn deze klachten begonnen ?

___ Reeds voor de bevalling
___ Binnen 3 maanden na de bevalling
___ 3 tot 12 maanden na de bevalling
___ Meer dan 1 jaar na de bevalling

48 Hoe vaak verliest op deze manier urine ?

___ Minder dan 1 x per week
___ 1 tot 5 x per week
___ 1 tot 5 x per dag
___ Meer dan 5 x per dag

49 Verliest U wel eens urine bij drukverhogende momenten (bijv.hoesten, niezen, persen, tillen etc.)

___ Ja
___ Nee

50 Verliest U wel eens urine op weg naar het toilet nadat U aandrang hebt bemerkt ?

___ Ja
___ Nee

51 Gebruikt U hulpmiddelen voor het opvangen van urine ? Zo ja, welke ?

___ Geen
___ Inlegkruisjes
___ Maandverband
___ Incontinentieverband
___ Andere: .....

52 Zo ja, hoeveel van deze verbandmiddelen gebruikt U ?

___ Maximaal 1 per dag
___ 1 tot 5 per dag
___ Meer dan 5 per dag

53 Indien U problemen of klachten hebt gekregen waar in deze enquete niet naar gevraagd is, maar die U wel wijt aan de opgetreden ruptuur, kunt U deze dan hieronder invullen ?

.....
.....
.....

54 Bent u bereid om in de toekomst eventueel mee te doen aan nader onderzoek (bijvoorbeeld lichamelijk onderzoek), naar mogelijke gevolgen van een totaalruptuur ?

___ Ja
___ Nee

55 Hebt U interesse in de uiteindelijke resultaten van deze enquete ? Zo ja, dan krijgt U na afloop van het onderzoek een samenvatting van de resultaten toegestuurd.

___ Ja
___ Nee

HARTELIJK DANK VOOR HET INVULLEN VAN DEZE ENQUETE !

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