

TREATMENT OF FAECAL INCONTINENCE

John W. Briel

ISBN: 90-9013967-2

© 2000, J.W. Briel

All rights reserved. No part of this thesis may be reproduced, stored in a retrieval system of any nature, or transmitted in any form by any means, electronic, mechanical, photocopying, recording or otherwise, included a complete or partial transcription, without the prior written permission of the author, or, when appropriate, of the publishers of the publications.

Printed by Ridderprint BV, Ridderkerk, The Netherlands

TREATMENT OF FAECAL INCONTINENCE

BEHANDELING VAN FAECALE INCONTINENTIE

PROEFSCHRIFT

Ter verkrijging van de graad van doctor
aan de Erasmus Universiteit Rotterdam
op gezag van de Rector Magnificus
Prof.dr.ir. J.H. van Bommel
en volgens besluit van het College voor Promoties

De openbare verdediging zal plaatsvinden op
woensdag 6 september 2000 om 15.45 uur

door

Johan Willem Briel

geboren te Rotterdam

Promotiecommissie

Promotoren:	Prof.dr. H.W. Tilanus Prof.dr. H.A. Bruining
Overige leden:	Prof.dr. C.G.M.I. Baeten Dr. H. van Dekken Dr. J. Stoker
Copromotor:	Dr. W.R. Schouten

This thesis was financially supported by Norgine, Tramedico, Coloplast, Medtronic, American Medical Systems, ConvaTec, Janssen-Cilag, Searle, AstraZeneca, Baxter, B. Braun Medical, Hollister, Schering-Plough, Tyco Healthcare, and Medeco.

*In memory of my mother
For my father
For Maud*

CONTENTS

Chapter 1	9
General introduction and outline of the thesis.	
Chapter 2	53
Clinical value of colonic irrigation in patients with continence disturbances.	
<i>Dis Colon Rectum 1997; 40: 802-5.</i>	
Chapter 3	65
Long-term results of suture rectopexy in patients with faecal incontinence associated with incomplete rectal prolapse.	
<i>Dis Colon Rectum 1997; 40: 1228-32.</i>	
Chapter 4	79
Disappointing results of postanal repair in the treatment of faecal incontinence.	
<i>Adapted from Ned Tijdschr Geneeskde 1995; 139: 23-6.</i>	
Chapter 5	91
Clinical outcome of anterior overlapping external anal sphincter repair with internal anal sphincter imbrication.	
<i>Dis Colon Rectum 1998; 41: 209-14.</i>	
Chapter 6	107
Relationship between sphincter morphology on endoanal MRI and histopathological aspects of the external anal sphincter.	
<i>Int J Colorectal Dis 2000; 15: 87-90.</i>	

Chapter 7	121
External anal sphincter atrophy on endoanal MRI adversely affects continence after sphincteroplasty. <i>Br J Surg 1999; 86: 1322-7.</i>	
Chapter 8	137
Summary and conclusions. Samenvatting en conclusies.	
Dankwoord	147
Curriculum Vitae	149

CHAPTER 1

GENERAL INTRODUCTION AND OUTLINE OF THE THESIS

Introduction

History

No data are available regarding the first description of faecal incontinence, whereas the first description of urine incontinence dates back to 1500 BC in the Georg Ebers Papyrus¹. As a custom, the Egyptians cleansed the intestinal tract regularly to maintain health, not just when ill. A linkage of internal human functioning to the outside world, may have prompted the Egyptians to view the anatomical and physiological makeup of the body as a system of channels '*metu*', similar to the network of canals which spread throughout their land. The heart was at the centre of the system, the station where the '*metu*' delivered and received. The '*metu*' carried blood, urine, tears, sperm and faeces. Around the anus, the channels coalesced into a sort of collecting system where the contents of the rectum could enter the network. Hence, the intestinal contents were cleaned out regularly by emetics, purges and enemas, and the anus became a prime target of medical treatment. The reputation of medical practitioners was consistently high throughout Egypt and the rest of the Mediterranean world. The names of several hundred physicians can be depicted from writings, references, and inscriptions. Among them was Iry, called 'the keeper of the King's rectum', a court physician who, about 2500 BC attended to diseases of the eye, belly, and the anus². Probably, he was the first proctologist.

Detailed information about the anatomy of the pelvic floor and anal canal has been lacking for a long time. Ideas of anatomy in ancient China were reached by reasoning and by assumption rather than dissection or direct observation. The Chinese did not begin systematic, direct anatomical studies since the doctrines of Confucius forbade violation of the body. The best of medieval dissectors was Mondino de Luzzi² of the University at Bologna whose '*Anathomia*' was completed in 1316 AD. However, his treatise was more an instruction book in dissecting techniques rather than a study of gross anatomy. The renaissance produced incredible

advances in many areas. The study of anatomy in this time swept away the many long maintained misconceptions of human anatomy. Andreas Vesalius³ performed his own dissections on cadavers. He described the colon, rectum and anus in meticulous detail as illustrated in his '*De Humani Corporis Fabrica*', published in 1543 AD. The anal sphincters as well as the levator ani muscle were clearly shown.

The risks and consequences of vaginal delivery were already known in Rome in the second century AD. Soranus⁴ wrote on injuries and diseases, but his principal field of work was obstetrics. His papers served as a textbook in the Middle Ages. He noted the complications during delivery due to pelvic abnormalities and the improper presentation of the baby. His methods of correcting abnormal positions were sound, as were his measures to prevent tearing of the pelvic soft parts during delivery. However, repair of the injured anal canal was not described.

For many centuries colonic irrigation was the only treatment modality for patients with faecal incontinence. Although this method, already used by the Egyptians, is widely accepted and recommended, details regarding indication for and functional outcome of colonic irrigation are lacking. After the introduction of biofeedback by Engel⁵ in 1974, the results of this conservative treatment modality are widely investigated and reported. The success-rates are impressive and promising, varying from 50 to 92 per cent^{6,7}. For a long time, the only surgical option was the creation of a colostomy. In 1776, Pillore⁸ was the first to describe the technique of stoma fashioning. The surgical treatment of faecal incontinence due to complete rectal prolapse consists of some sort of rectopexy. Reports on the outcome of this procedure are abundant and vary between 50 and 75 per cent⁹. However, it is questionable whether faecal incontinence associated with incomplete rectal prolapse, which condition was first described by Allingham¹⁰ in 1873, should also be treated by rectopexy.

Anorectal procedures, such as fistula operations and haemorrhoidectomies were already performed by surgeons in the 12th century. However, the first

Introduction

description of anterior sphincter repair dates back to 1875, when Warren¹¹ described the use of the vaginal mucosal flap to protect the repair of the lacerated rectum and anus. Faecal incontinence was called 'idiopathic' when no apparent cause could be found. In 1975, Parks¹² described a technique to reinforce the pelvic floor and external anal sphincter in an attempt to solve the problem of idiopathic faecal incontinence. This procedure was also aimed at restoring the anorectal angle. Results of this so-called postanal repair are varying between 58 and 86 per cent^{13,14}. Unfortunately, in recent years it became clear that the long-term results are less favourable than suggested by those promising short-term results^{15,16}. In 1991, the introduction of endoanal ultrasound was responsible for a dramatic change of view regarding the aetiology of faecal incontinence¹⁷. Using endoanal ultrasound, it became clear that occult anterior sphincter defects could be found in patients with apparently uneventful deliveries. In these patients, sphincter defects are repaired by anterior anal repair instead of postanal repair. In 1940, Blaisdell¹⁸ reported the results of a direct end-to-end anterior sphincter repair and found a failure rate as high as 40 per cent. End-to-end apposition has been criticised because of disappointing results from cutting out the sutures and retracting the muscle ends¹⁹. Overlapping sphincteroplasty, as described by Parks and McPartlin²⁰ in 1971 and later modified by Slade²¹, showed better but not perfect results. Later on, other modifications have been proposed in order to improve surgical outcome following anterior anal repair. Furthermore, new imaging techniques have been developed. In 1994, the use of an endoanal coil in association with MRI was first described²². Using this technique, atrophy of the external anal sphincter could be demonstrated, which is impossible using endoanal ultrasound. However, the accuracy and impact of this novel finding remain to be clarified.

Research in this field has long been lacking. Faecal incontinence in general tends to receive less attention than urinary incontinence. This is further compounded by an apparent lack of professional interest. However, during

the last decades there has been a vast international increase in interest in anorectal physiology and functional disorders of the anorectum. Faecal incontinence is becoming increasingly more described and discussed. Furthermore, obstetric injuries and their sequelae are responsible for an increasing number of medicolegal cases. The most recent developments of awareness in the field of incontinence are reflected by the existence of homepages and information about faecal incontinence on the World Wide Web.

Definition of the Disorder

Despite a general belief, faecal incontinence is a symptom rather than a diagnosis. Definition of faecal incontinence is difficult, due to its many entities. In publications faecal incontinence is also referred to as leakage, seeping, anal incontinence and incontinentia alvi. The latter is the official Latin denomination, meaning that the abdomen is uncontrollable. The following definition of faecal incontinence is generally accepted: faecal incontinence is the impaired ability to control gas or stool. The assessment and grading of faecal incontinence was first described by Parks²³ (*Table 1*).

Table 1. Grading of faecal incontinence according to Parks²³.

Grade	
I	Fully continent
II	Soiling or incontinence for gas
III	Incontinence for liquid stool
IV	Incontinence for solid stool

Because this grading system is simple, it is the one most frequently used. However, it does not take into account the frequency of symptoms.

Introduction

Therefore, other 'faecal incontinence scoring systems' have been proposed²⁴⁻²⁶. In assessing the outcome of surgery these scoring systems have proven to be effective. The use of one generally accepted questionnaire in the assessment of patients with faecal incontinence is stressed, because it may also be used to achieve an objective comparison of results between different series. Unfortunately, none of these questionnaires is widely accepted, except for the Parks grading. This lack of consensus is not the only confounding factor in comparing the results after various treatments: in most reports no attention is paid to the difference between short- and long-term findings.

Psychosocial Aspects

The patient presenting with faecal incontinence does not complain without reason. Although faecal incontinence is one of the more frequent clinical symptoms of the gastrointestinal tract, its assessment is often neglected in clinical practice. The psychosocial aspects of the symptoms are also often overlooked: the number of undetected cases in comparison to those registered, illustrates that not only patients but also physicians avoid this topic during clinical routine²⁷.

Consequences of faecal incontinence for the quality of life have rarely been investigated, but there are specific impacts on family life, specifically with respect to sexuality and employment of those affected. Incontinence often is not discussed due to embarrassment. Many patients with faecal incontinence become housebound because of the stigma associated with the condition. It is a complaint that is socially disruptive. To the individual there is hardly a more distressing symptom than that of faecal incontinence: it is felt to be a mark of social disgrace. Clinical histories reveal the many years that people suffer it before seeking help.

In the elderly, faecal incontinence is frequently an important factor in

deciding whether to institutionalise the patient: along with immobility and confusion, incontinence is considered the primary reason for institutionalisation of the elderly. Elderly persons who experience loss of bladder or bowel control are frequently depressed, isolated, and fearful of discovery. Left untreated, these individuals are prone to mental and social deterioration that may lead to social isolation or institutionalisation. The most common cause in these individuals is faecal impaction with overflow incontinence. Moreover, continence deteriorates considerably with time. In the elderly, faecal incontinence is a major problem with serious consequences. A study among 4232 nursing home residents free of pressure ulcers on admission to a nursing facility revealed that at three months follow-up the determinant faecal incontinence was a major risk factor associated with the formation of stage II-IV pressure ulcers²⁸. Furthermore, incontinence is related to mortality and severe incontinence represents an increased risk factor for mortality in elderly people living at home²⁹. The relationship of urinary tract infection and faecal incontinence is illustrated by the finding that patients with faecal incontinence develop urinary tract infection almost three times as frequently as their continent counterparts³⁰.

For some of the patients with faecal incontinence creation of a stoma might be finally inevitable. This distorts a patient's body image. Orbach and Tallent³¹ reported that all of their patients with a stoma considered their bodily form had been altered in a way deleterious to their physical appearance. A feeling of having been invaded, violated, or even sexually assaulted, is common. Patients feel they have lost control of bowel function and often become preoccupied with elimination. This results in some patients reporting that they have been made 'like an animal'. Some men perceive the colostomy as being similar to a vagina. Patients also have a distorted perception of their health. Many feel that their body has become fragile. These feelings lead to withdrawal from work, leisure and many social responsibilities³²⁻³⁹.

Introduction

In summary, faecal incontinence is a silent affliction that often leads to self-imposed ostracism. For many years, a lack of understanding regarding its pathophysiology and a lack of empathy among many physicians has bedevilled this problem. However, during the last decades, remarkable strides have been made, both in the evaluation and in the treatment of faecal incontinence.

Epidemiology

Faecal incontinence is much more common in the general population than is often realised. Although the problem increases with advancing age and disability, there are large numbers of otherwise healthy adults with this distressing symptom. The first prevalence study of faecal incontinence in the general population was published by Thomas and colleagues⁴⁰ in 1984. Virtually all estimates of the prevalence and incidence of faecal incontinence in the community rely on self-reported continence status. It is known that only a minority of patients with faecal incontinence report these symptoms to their physicians. Epidemiological estimates based on medical chart data, therefore, contain a detection bias, which systematically underestimates the real presence of faecal incontinence⁴¹. As a consequence, reliable epidemiological data on incidence and prevalence of faecal incontinence are scarce, and prevalence figures vary greatly (*Table 2*).

Table 2. Prevalence of faecal incontinence.

	<i>Prevalence (%)</i>
Working population ^{42,43}	0.5-6.8
Non-institutionalised (> 65 years) ^{44,45}	3.7-16.9
Institutionalised ^{46,47}	33-46

The pelvic floor shows sex-related differences. The female pelvic floor and its integrated sphincters are more susceptible to impairment. Ninety per cent of all patients with 'spontaneous incontinence' are women. Furthermore, the spinal centres that govern continence in the female contain significantly fewer ganglion cells than the corresponding centres in the male⁴⁸.

Johanson⁴⁹ analysed 881 individuals, aged 18 years and older. Stratified by frequency 2.7, 4.5, and 7.1 per cent of participants suffered faecal incontinence daily, weekly, or once per month or less, respectively. Incontinence increased progressively with age. Only one-third of individuals with faecal incontinence had ever discussed the problem with a physician. In another study, Nelson and colleagues⁵⁰ determined prevalence of and characteristics associated with faecal incontinence in the general community. A total of 2570 households comprising 6959 individuals were surveyed. Of them, 153 individuals were reported to have faecal incontinence, representing 2.2 per cent of the population. Thirty per cent of the incontinent subjects were older than 65 years, whereas 63 per cent of them were women. Of those with faecal incontinence, 36, 54, and 60 per cent were incontinent to solid stool, liquids, and gas, respectively. Independent risk factors for faecal incontinence included female sex, advancing age, poor general health, and physical limitations.

Studies regarding patients in whom faecal incontinence is associated with urinary symptoms are scarce. This is surprising, considering how common double incontinence is in the elderly and that approximately 12 per cent of patients with faecal incontinence also have genuine stress urinary incontinence⁵¹. Studies of faecal incontinence and urinary incontinence have shown evidence of a neurogenic aetiology for both disorders. It has been postulated that pudendal nerve injury causes a weakened pelvic floor and anal sphincter musculature, which may result in both faecal and urinary incontinence^{52,53}.

To establish the baseline prevalence rate and cost of faecal incontinence

Introduction

in a long-term care hospital, Borrie and Davidson⁴⁷ sent questionnaires to in-patients. The degree of urinary and faecal incontinence and costs of incontinence as measured by nursing time spent dealing with incontinent patients was measured. The prevalence rate of urinary incontinence was 62 per cent, faecal incontinence 46 per cent, and combined incontinence 44 per cent. The mean time spent each day dealing with incontinence was 53 minutes per patient. The total annual cost of incontinence per patient was 9771 Canadian Dollars.

In the Netherlands, the total costs for both urinary and faecal incontinence material, as paid by the associated insurance companies each year, is more than 100.000.000 Dutch Guilders (*Table 3*)⁴².

Table 3. Annual costs for both urinary and faecal incontinence in Dutch Guilders (millions).

Age (years)	Male	Female
< 15	6.8	8.7
15-65	11.1	35.6
> 65	12.2	36.4

Aetiology

Numerous different causes can be identified in the aetiology of this distressing condition. It is virtually impossible to produce an exhaustive list of influencing factors. Faecal incontinence can result from a disturbance in any one of the multiple factors, which maintain continence. The most important anatomic structures in faecal continence are the internal and external anal sphincters, whereas the most important functional factors are believed to be the anorectal angle, rectal compliance, rectal sensitivity, and the discriminatory property of the anal

canal. Closure of the anal canal itself is accomplished by a combined function of the haemorrhoidal cushions and the internal anal sphincter. Under resting conditions and during slow entry of contents into the rectum, continence is maintained by the tonic contraction of the internal anal sphincter supplemented to a lesser or greater degree by contraction of the external anal sphincter⁵⁴⁻⁵⁷. Increases in intra-abdominal pressure, rectal contraction and the rapid entry of larger volumes into the rectum threaten continence and are countered by a compensatory contraction of the external anal sphincter. The external anal sphincter responses depend on adequate afferent mechanisms, properly functioning integrative circuitry in the spinal cord and sufficiently strong muscular contraction. The external sphincter response to rectal distension is critically linked to conscious rectal sensations and must therefore involve cortical modulation. It follows therefore that faecal incontinence could arise from a variety of possible defects.

In practise, faecal incontinence is most commonly seen in women who have had children. This symptom can also be seen in other patient groups, who may, in addition, also have had children. Groups of individuals at high risk for faecal incontinence further include the elderly, the mentally ill, institutionalised patients, and those with neurologic disorders. The principal cause of faecal incontinence is a defect of one or both anal sphincters due to obstetric injury. Other major contributing factors are anorectal surgery, accidental trauma, pudendal nerve neuropathy, complete rectal prolapse and diarrhoea (e.g. due to severe colitis).

Obstetric Trauma

Most patients with faecal incontinence are female, whereas the most common cause of faecal incontinence is obstetric trauma. Rupture of the

Introduction

anal sphincter muscle is a complication of vaginal deliveries, with a reported frequency of 0 to 5 per cent^{58,59}. Certain factors seem to promote this risk, such as primiparity, the use of forceps and vacuum extractor, and young age of the mother⁶⁰⁻⁶². Episiotomy does not prevent a third-degree tear⁶³. Overt obstetric injuries are frequently associated with disruption of the perineal body, division of both anal sphincters, and loss of the distal rectovaginal septum. Usually, such injuries are recognised at the time of delivery and the majority are sutured in the delivery suite, often by junior medical staff who have a poor understanding of anal anatomy⁶⁴. Often an end-to-end repair is performed and primary repair of third-degree tears is, therefore, frequently inadequate. Despite a primary repair, persistent sphincter defects have been reported using endoanal ultrasound varying from 40 to 85 per cent of patients^{65,66}. Bek and Laurberg⁶⁷ assessed the long-term consequences of complete tears and subsequent vaginal delivery (*Table 4*). The major long-term problem was incontinence of flatus.

Table 4. Incidence of faecal incontinence following complete tear during the first delivery and following subsequent delivery.

	<i>Faecal Incontinence</i>	
	<i>Transient</i>	<i>Permanent</i>
Complete tear	41%	7%
Subsequent delivery	39%	17%

Møllerup Sørensen⁶⁸ studied women with and without complete rupture. Surprisingly, the long-term consequences of vaginal delivery comprised dyspareunia in 17 per cent of patients with perineal rupture and 22 per cent of patients without rupture. Urinary incontinence was found in respectively 25 and 11 per cent. After four years of follow-up, 25 per cent was incontinent to flatus, 13 per cent to liquids, and four per cent to solid

stool. Repeated vaginal deliveries increase the risk of minor faecal and urinary incontinence, especially after the third delivery compared with the first and second⁶⁹. Furthermore, patients who sustained a division of the external anal sphincter at delivery are prone to impaired sensation, which persists in the anal canal at six months⁷⁰.

Vaginal delivery may initiate damage to the continence mechanism by direct injury to the pelvic floor muscles, damage to their motor innervation, or both. Additional denervation may occur with ageing, resulting in a functional disability many years after the initial trauma. These factors should be kept in mind when conducting vaginal birth. Obstetricians may be able to reduce pelvic floor injuries by minimising forceps deliveries and episiotomies, by allowing passive descent in the second stage, and by selectively recommending elective caesarean delivery. Midline episiotomies should be avoided. Furthermore, assessment of anal function after delivery is advocated, allowing elective caesarean delivery and sparing of further sphincter damage⁷¹. Elective caesarean birth should also be recommended at term to patients with a foetus greater than 4,000 gram or a strong family history of pelvic prolapse and/or incontinence⁷². Information and routine follow up of all women with obstetric anal sphincter rupture is mandatory.

Anorectal Surgery

Disturbed continence is a frequently neglected but rather common complication following anorectal surgery. Especially fistula surgery may result in impaired continence. Although incontinence for solid stool rarely occurs, minor defects in continence may follow if even a small amount of sphincter muscle is damaged. The incidence after fistulotomy varies from 18 to 52 per cent⁷³⁻⁷⁶. To define the incidence of faecal incontinence following staged fistulotomy using a seton, a five-year retrospective chart review

Introduction

was carried out by Pearl and colleagues⁷⁷. Faecal incontinence occurred in 5 per cent of cases. Internal sphincterotomy may also lead to continence disturbances caused by inadequate closure of the anal canal by the internal sphincter. This condition is usually temporary but may become permanent. Faecal soiling after lateral internal sphincterotomy is reported to vary from 0 to 47 per cent^{78,79}. Anal dilatation was introduced as a treatment for haemorrhoids by Lord⁸⁰ in 1968. In one review faecal incontinence occurred in the first week after anal dilatation in 10 of 55 patients (18 per cent) and as a late complication in 2 patients (4 per cent)⁸¹. Other reported figures vary between 15 and 25 per cent^{82,83}. In modern surgery for haemorrhoids, incontinence is a rare complication. However, if the sphincter mass is inadvertently injured, incontinence may result⁸⁴. Following conventional anterior resection, continence is generally maintained. However, when the anastomosis is performed in the distal third of the rectum, impairment of continence might occur. In a multicentre study to define the incidence of disordered continence after restorative proctocolectomy and ileoanal reservoir 207 patients were included, of whom 156 patients had their ileostomy closed. Minor leakage was observed in 27 per cent of cases, whereas two per cent complained of troublesome faecal soiling. None of them had gross faecal incontinence⁸⁵.

Pudendal Nerve Neuropathy

The current opinion of pudendal nerve neuropathy is that of one sustained during vaginal delivery or chronic straining at stool. Electromyographic studies, performed by Allen and colleagues⁸⁶ showed that there was evidence of re-innervation in the pelvic floor muscles after vaginal delivery in 80 per cent of the studied patients. Women who had a long active second stage of labour and heavier babies showed the most evidence of nerve damage. However, forceps delivery and perineal tears

did not affect the degree of nerve damage seen. Berkelmans⁸⁷ conducted a study to determine whether patients complaining of straining at stool are prone to develop faecal incontinence. The incidence of faecal incontinence after 5 years was higher among women with chronic straining than in a control group. The patients with straining at stool who became incontinent had, at the initial investigation, a lower electromyographic activity of the external anal sphincter during voluntary contraction, greater perineal descent at rest and less elevation of the pelvic floor during maximal contraction of the external anal sphincter than the other women. In fact, women with chronic straining at stool had perineal descent at rest and during straining similar to that of incontinent women. Therefore, women with chronic straining might be prone to develop faecal incontinence.

Ellis and colleagues⁸⁸ suggested pudendal nerve compression as contributing factor in the aetiology of faecal incontinence. The pudendal nerve arises from sacral nerve roots 2, 3, and 4 and traverses the lesser sciatic foramen. To determine if compression of the pudendal nerve could occur in the lesser sciatic foramen, the course of the pudendal nerve was dissected in 79 cadavers (61 female and 18 male). Bilateral compression of the pudendal nerve between the sacrospinous and sacrotuberous ligaments was identified in 20 of the females (33 per cent) but none of the males. Pregnancy results in marked hypertrophy of the pelvic ligaments and could possibly cause compression of the pudendal nerve contributing to faecal incontinence. If this mechanism is confirmed by further studies, it might be worthwhile to decompress the nerve to restore nerve function.

Miscellaneous

Complete rectal prolapse is associated with faecal incontinence in more than

Introduction

50 per cent of the cases¹². It is evident that treatment of faecal incontinence in these patients begins with rectopexy. If incontinence persists following rectopexy, additional sphincter repair might be considered.

Diarrhoea is another contributing cause of faecal incontinence. Patients with sphincter defects following childbirth might be continent to solid stool but incontinent to liquids. In case of frequent diarrhoea they become symptomatic. Diarrhoea as a result of faecal impaction and subsequent overflow incontinence is a symptom rather than a cause. In case of fulminant toxic colitis continence for rectal fluids is impaired due to high concentrations of internal anal sphincter pressure reducing nitric oxides.

Other rare cases, such as diabetic peripheral neuropathy, carcinoma, multiple sclerosis, endometriosis, irradiation, and AIDS, are beyond the scope of this thesis.

Investigation

The evaluation of faecal incontinence begins with comprehensive history taking, which may include overcoming a significant psychosocial barrier. Physical examination is essential. For centuries the structure and function of the pelvic floor and anal sphincters have remained enigmatic. Great strides in new investigation techniques over the last decades have enabled a better understanding of the pathophysiology of this complex and dynamic structure. The assessment of patients with faecal incontinence generally takes place in the anorectal physiology laboratories. Several laboratories have reported results of anorectal physiology measurements, but there is extensive variation between normal values in different laboratories. Also, the value of anorectal physiology tests is debated because: 1) it is not possible to establish a reproducible normal range; 2) abnormal measurements do not correlate

with disease entities or explain symptoms; 3) the results are often unhelpful in diagnosis and management; and 4) clinical outcome after intervention does not correlate with alteration in the measurements obtained.

The following anorectal function tests are the most widely used or need further attention:

Anorectal Manometry

Since it was first introduced into clinical investigations in 1960 by Schuster⁸⁹, anorectal manometry has become one of the most important methods to evaluate anorectal sphincter function. Manometry is not a single test but consists of a series of measurements. Manometry permits examination of the resting pressure and the squeeze pressure. Furthermore, it can also be used to elicit the rectoanal inhibitory reflex and to measure rectal compliance. Although the use of anorectal manometry has been well established⁹⁰⁻⁹², there is some debate as to the best method of performing manometry. A variety of systems, including water-perfused catheters, balloons, and microtransducers, have been used⁹³⁻⁹⁵. Data regarding the value of anorectal manometry in predicting outcome following surgery or biofeedback are rather conflicting.

Electromyography

Conventional electromyography enables the localisation of normal external anal sphincter tissue. It also provides a useful tool for the investigation of the activity of the pelvic floor in rest and during squeezing and straining. Single fibre electromyography measures the fibre density of the pelvic floor muscles, reflecting the amount of damage

Introduction

to these muscles. The most common electromyographic findings in patients with faecal incontinence are decreased recruitment of motor units with squeezing and polyphasic motor units potentials, consistent with an injury pattern⁹⁶. Until the introduction of endoanal ultrasound, electromyography was also used for mapping of occult sphincter defects.

Pudendal nerve terminal motor latency/PNTML

Pudendal nerve stimulation, performed transrectally, has been developed from the technique of electro-ejaculation introduced by Brindley⁹⁷ for use in patients with paraplegia. Tests of motor conduction in the pudendal nerves are well established in the diagnosis of pelvic floor disorders⁹⁸⁻¹⁰³. Transrectal measurement of PNTML has proved particularly useful in assessing patients with faecal incontinence^{104,105}. Evidence of pudendal neuropathy is obtained by recording the PNTML in the left and right pudendal nerves and calculating the mean value from these two recordings. A raised mean PNTML has been shown to indicate pudendal nerve damage. The mean PNTML in normal subjects is 2.0 ± 0.2 ms¹⁰⁶. Pudendal neuropathy is found in 37 per cent of patients with faecal incontinence and dramatically increases after 70 years of age¹⁰⁷. This indicates that pudendal neuropathy might be an age-related phenomenon. Pudendal neuropathy is more common in females than in males. Likewise, it is more frequent in patients with pathologic perineal descent and in patients with risk factors, such as difficult labour or excessive straining at stool¹⁰⁸. Interestingly, damage to the motor and sensory components of the pudendal nerve does not occur synchronously¹⁰⁹. The predictive value of PNTML is questioned because data are conflicting.

Defaecography/Proctography

Defaecography is a tool to evaluate the dynamics of defecation¹¹⁰. This technique was first developed by Mahieu¹¹¹ in 1984 and allows visualisation of rectoanal intussusception, rectocele, and other anatomic abnormalities by means of contrast radiology^{112,113}. The anorectal angle can be measured at rest and during straining. Furthermore, the degree of perineal descent can be measured during straining. Proctography is not necessary for the diagnosis of complete rectal prolapse because this abnormality can easily be seen by inspection of the patient, especially during straining. The high prevalence of intussusception in asymptomatic controls cast doubt whether this alteration of rectal morphology is functionally relevant in patients with faecal incontinence¹¹⁴⁻¹¹⁸. The predictive value of proctography in order to assess outcome after sphincter repair is questionable.

Endoanal Ultrasound/Sonography

Endoanal ultrasound has added a new dimension to the investigation of the anal sphincter complex. In 1991, Burnett¹⁷ was the first to report this technique. The endosonic probe causes no more discomfort than routine digital examination of the anus. The technique enables repeated examination of the anal sphincters along the length of the anal canal and permits an assessment of sphincter morphology. The advent of anal endosonography has enabled the identification of anal sphincter defects following vaginal delivery. Sentovich and colleagues¹¹⁹ conducted a study using endoanal ultrasound in three groups of women: 13 incontinent women with obstetric-related sphincter injuries by history, 20 asymptomatic parous women, and 20 asymptomatic nulliparous women. The injuries identified by endoanal ultrasound are shown in *Table 5*:

Introduction

Table 5. Sphincter defects using endoanal ultrasound.

	<i>EAS-Injury</i>	<i>IAS-Injury</i>
Obstetric incontinence	13 (100%)	10 (77%)
Parous	7 (35%)	4 (20%)
Nulliparous	3 (15%)	1 (5%)

EAS: External Anal Sphincter; IAS: Internal Anal Sphincter

Based on these findings, the authors concluded that endoanal ultrasound accurately predicted sphincter injuries in incontinent patients and identified occult sphincter injury in asymptomatic parous women. Intact anal sphincters were also identified by endoanal ultrasound. However, this imaging technique falsely predicted injury in 5 to 15 per cent of patients with intact anal sphincter muscles. Because of the presence of sphincter defects in continent patients and in asymptomatic volunteers, caution should be used in attributing incontinence to anal sphincter defects alone. Both the accuracy and reliability of transanal ultrasound are significantly improved by limiting transanal ultrasound to the distal 1.5 cm of the anal canal¹²⁰.

Using endoanal ultrasound, Felt-Bersma¹²¹ demonstrated sphincter defects following haemorrhoidectomy (13%), fistulectomy (72%), and lateral internal sphincterotomy (88%). In patients presenting with symptoms of impaired continence, sphincter defects could be demonstrated, whereas the other sphincter defects did not produce symptoms.

Endoanal ultrasound has a higher sensitivity than electromyography in detecting sphincter defects and correlates well with surgical findings. Therefore, most authors advocate the use of endoanal ultrasound for sphincter mapping¹²².

Endoanal MRI

Recently, endoanal MRI has been added to the imaging techniques in the assessment of patients with faecal incontinence. In a comparative study, endoanal MRI was more accurate than endoanal ultrasound in the detection of internal and external anal sphincter defects¹²³.

The accurate delineation of the external anal sphincter with endoanal MRI has led to the possibility of evaluating the muscle mass and has revealed the occurrence of external anal sphincter atrophy. However, the prevalence of sphincter atrophy in parous women is unknown.

Disadvantages of endoanal MRI are high costs and limited availability. Therefore, further study is needed to elucidate the impact of external anal sphincter atrophy and the place of endoanal MRI in the assessment of patients with faecal incontinence.

Treatment

Management options for patients with faecal incontinence are limited. There is a need to develop methods, which enable the individual to cope with this distressing symptom, always bearing in mind that the fundamental aim is to correct the handicap and restore a sufficient continence. Basically, only two treatment modalities are available: conservative and surgical (*Table 6*).

Conservative management has to utilise all its possibilities before surgery may become a therapeutic option.

Except for special situations surgery is indicated only after conservative therapy has failed. Patients with failed surgical treatment might still benefit from postoperative conservative treatment modalities.

Introduction

Table 6. Treatment modalities for faecal incontinence.

Conservative Treatment	
Adjustment of diet	
Drugs	
Anal tampon/plug	
Biofeedback	
Colonic irrigation	
Surgical Treatment	
Postanal repair	
Anterior anal repair	
Total pelvic floor repair	
(stimulated) Neosphincter	
Rectopexy	
Ostomy	

Conservative Treatment

Faecal impaction with both incontinence and constipation can be treated by a combination of increasing diet fibre, laxatives and occasionally enemas to regulate defecation in a socially acceptable manner. The diets of persons with faecal incontinence are similar to those of control subjects with normal bowel function. Patients with faecal incontinence may improve their nutritional patterns by lowering sodium and protein intake and increasing dietary fibre and mono-unsaturated fat intake. Calcium and vitamin D supplementation may improve dietary deficiencies and lower disease risks. Including a nutritional assessment and consultation in the care of persons with faecal incontinence to improve general health and prevent disease is recommended, but consideration must be given to altered diet patterns perceived by the patient to prevent

faecal incontinence¹²⁴.

Drug treatment is restricted by the limited number available: only a very few drugs act directly on the anal sphincter mechanism to increase the tone. Since experience with these drugs in the treatment of faecal incontinence is limited, further research is needed. Currently, drug therapy is limited to constipating agents, which can be very useful for people with internal anal sphincter disruption. A useful constipating agent is loperamide. Loperamide is effective in the treatment of diarrhoea with faecal incontinence. Normalisation of colon transit time and an increase in the tone of the internal anal sphincter seem to be the main determinants of efficacy¹²⁵. Additionally, it has an effect on rectal compliance in patients with diarrhoea and associated faecal incontinence¹²⁶. Successful treatment with stool bulking agents, such as psyllium or bran, may be achieved in patients with the pattern of soiling¹²⁷. Activated charcoal is helpful in case of acute diarrhoea and effectively limits the escape of sulphur-containing gases into the environment, which are the major, but not the only, malodorous components of human flatus¹²⁸.

Similar to the vaginal tampon, disposable anal tampons and plugs have been developed¹²⁹. These are available in different sizes. After the first stool has passed the tampon is introduced. The advantage of these tampons is that there is no contact between faeces and skin, reducing the risk of skin-irritation. Christiansen¹³⁰ performed a clinical assessment of anal continence plugs in 14 patients with incontinence for liquid and solid stool. Nine patients (64 per cent) became continent when they used the plug. In 43 percent the plug occasionally slipped out, and 71 percent experienced discomfort to a varying degree, which caused 11 patients to withdraw from the study before the end of the planned study period. The overall conclusion was that the majority of patients would use the plug under special circumstances because it eliminates the fear of faecal leakage but that local discomfort would prevent its daily use.

Besides drug treatment, biofeedback training is the most important basis

Introduction

of conservative therapy. Therefore, pelvic floor exercises have become treatment of choice, most effective with biofeedback support. This is supported by a variety of studies published in the past years, with success-rates varying from 50 to 92 per cent^{10,11}. These studies have presented convincing evidence that this method which derives from psychological learning theory has found clinical importance and relevance, although the exact mode of action is still unknown¹³¹. Some patients never manage to start or complete this treatment. Many lack the motivation or are unconvinced about the possible value of what they perceive to be 'simple exercises'. The treatment will be suitable only for patients who can follow a course of treatment, as the results are largely patient dependent. Future research will need to demonstrate the long-term effects of such management. It is most successful in treating urge incontinence, but also helps some patients with passive leakage. Even in patients with structural anal sphincter damage, some symptom improvement or cure is achieved¹³². Contrary, patients with neurological disease have little or no benefit from biofeedback¹³³.

Surgical Treatment

Postanal repair, anterior anal repair and rectopexy will be discussed in detail in separate chapters of this thesis. Total pelvic floor repair consists of a combination of postanal and anterior anal repair. The procedure is designed for neuropathic faecal incontinence without sphincter defects. Although preliminary short-term results were promising it is becoming clear that total pelvic floor repair rarely renders patients with neuropathic faecal incontinence completely continent. Furthermore, the operation is less successful in obese patients and in those with a history of straining or perineal descent¹³⁴.

Efforts to create a neoanal sphincter have relied principally on the

transposition of skeletal muscle, usually the gracilis and gluteus maximus muscle, around the anal canal. Pickrell¹³⁵ was the first to use this technique in man and although he reported satisfactory results, other investigators found the technique was unreliable. Stimulated anal sphincter replacement offers a new treatment option for patients with severe refractory faecal incontinence. Dynamic graciloplasty consists of transposition of the gracilis muscle to the anus with the implantation of stimulating electrodes. This procedure has been proven to be a safe and reliable technique in patients with severe incontinence and may result in a better quality of life¹³⁶. However, the procedure has significant morbidity that can lead to functional failure. Outcome after dynamic graciloplasty appears to correlate with surgical experience. In contrast to graciloplasty, the use of dynamic gluteoplasty should be limited to investigational purposes¹³⁷. Artificial urinary sphincters have also been implanted around intestinal segments to achieve continence. Although preliminary studies reported that the model of the artificial urinary sphincters appeared to be unsuitable for the treatment of faecal incontinence¹³⁸, other reports are more favourable¹³⁹⁻¹⁴². For treatment of faecal incontinence in patients with neurologic disease that affects the pelvic floor and the muscles of the lower limb it might be the only surgical option.

Full recovery has been achieved in a canine model using pudendal nerve innervation of the semitendinosus muscle neosphincter. Cross-innervation of transposed skeletal muscle by the pudendal nerve creates a physiologic and functional anal sphincter. This technique may be superior to the electrically stimulated wraps for faecal incontinence¹⁴³.

In very advanced cases and for patients who fail all therapeutic options, intestinal stoma may occasionally provide important hygienic and psychological benefits and a better lifestyle than coping with the consequences of faecal incontinence.

Introduction

Miscellaneous

Shafik¹⁴⁴ described patients with pudendal canal syndrome, who were treated by pudendal canal decompression. All complained of faecal incontinence and of additional stress urinary incontinence. Pudendal canal decompression operation comprised a para-anal incision, entering of the ischiorectal fossa, identifying and tracing of the inferior rectal nerve to the pudendal canal, and incising of the roof of the canal. In patients with only short duration of symptoms improvement in faecal and urinary incontinence was found. Postoperatively, there was a significant decrease in pudendal nerve terminal latency. Electromyography of the external anal sphincter and levator muscles showed increased activity. The failures occurred in cases, which presented in advanced stage of the disease, and seem to be due to irreversible nerve or muscle damage. Pudendal canal decompression effected improvement not only in faecal incontinence but also in stress urinary incontinence. A relation between the latter and pudendal neuropathy was suggested.

Shafik¹⁴⁵ also described autologous fat injection in 14 patients with partial faecal incontinence. He used the fat as substitute for the polytetrafluoro-ethylene as he described earlier¹⁴⁶. He proposed that the fat injection did not have the side effects of polytetrafluoro-ethylene, such as granuloma formation and particle migration. The procedure comprised the harvesting of 50 to 60 ml of fat from the abdominal wall. This then was injected submucosally into the rectal neck at 3 and 9 o'clock positions. After the first injection 3 patients were completely continent, whereas after the second and third injection respectively 10 and all 14 patients rendered complete continence.

Electrical stimulators have been used in the treatment of neurogenic faecal incontinence caused by pudendal nerve neuropathy. It is based on repeated neurostimulation of the pudendo-anal reflex arc running

through S3, by altering sphincter and rectal motor function. The preliminary results are promising¹⁴⁷⁻¹⁴⁹.

Introduction

Outline of the Thesis

This thesis is aimed at evaluating the results of different conservative and surgical modalities in the treatment of faecal incontinence. Furthermore, the value of endoanal MRI in the assessment of faecal incontinence is investigated. Continence disturbances, especially faecal soiling, are difficult to treat. Irrigation of the distal part of the large bowel might be considered as a non-surgical alternative for patients with impaired continence. Although this treatment modality is widely accepted, its clinical use has not yet been evaluated. **Chapter 2** describes the clinical value of colonic irrigation. Suture rectopexy is the recommended therapy for complete rectal prolapse that is associated with faecal incontinence. After the introduction of proctography, intussusception or incomplete rectal prolapse as seen on the proctogram, was thought to be a causative factor of faecal incontinence, analogous to complete rectal prolapse. It has been suggested that correction of an incomplete rectal prolapse is also worthwhile for patients with faecal incontinence. In **Chapter 3** the clinical outcome of rectopexy in patients with faecal incontinence associated with incomplete rectal prolapse is described and compared with that obtained from patients with complete rectal prolapse. In patients without apparent cause for their continence disorder, incontinence was called 'idiopathic'. Formerly, idiopathic faecal incontinence was treated by postanal repair. Long-term results of postanal repair are discussed in **Chapter 4**. The introduction of endoanal ultrasound revealed occult sphincter defects in patients with apparent uneventful deliveries. Faecal incontinence in patients with these occult sphincter defects or overt anterior sphincter defects is usually treated by a delayed overlapping repair of the external anal sphincter. However, an obstetric trauma is frequently associated with disruption of the perineal body and loss of the distal rectovaginal septum. Data regarding a combined repair, consisting of restoration of the rectovaginal septum and perineal body, overlapping external anal sphincter repair, and imbrication of the internal anal

sphincter, are scanty. **Chapter 5** compares the long-term results of the rather simple direct sphincter repair with those of the more complex anterior anal repair. Both repairs do not provide restoration of continence in all treated patients. Therefore, it is worthwhile to identify patients preoperatively who might benefit or not. With the introduction of endoanal MRI, atrophy of the external anal sphincter was first visualised. Until now, no study has compared the morphological endoanal MRI findings with histopathological aspects of the external anal sphincter. **Chapter 6** describes the relationship between sphincter morphology on endoanal MRI and histopathology of the external anal sphincter. The predictive value of this atrophy in patients undergoing sphincter repair is discussed in **Chapter 7**. Finally, an algorithm for the treatment of patients with faecal incontinence, based on the conclusions drawn from this thesis is presented in **Chapter 8**.

Questions to be answered in this thesis:

- What are the indications for and results of colonic irrigation?
- What are the long-term results of suture rectopexy in patients with faecal incontinence due to complete rectal prolapse?
- Is rectopexy indicated for patients with faecal incontinence associated with incomplete rectal prolapse?
- Is there still a place for postanal repair?
- Is the complex modified anterior anal repair better than the rather simple direct sphincter repair?
- Can atrophy of the external anal sphincter correctly be identified using endoanal MRI?
- What is the prevalence of external anal sphincter atrophy in female patients with anterior sphincter defects caused by obstetric trauma?
- What is the impact of this sphincter atrophy on functional outcome following sphincteroplasty?

Introduction

References

1. Ebbell B. The Papyrus Ebers: the greatest Egyptian medical document. Copenhagen; Ejnar Munksgaard, 1939.
2. Lyons AS, Petrucelli RJ. Medicine. An illustrated history. Abradale Press, Harry N. Abrams, Inc., Publishers, 1978, New York.
3. Vesalius A. De Humani Corporis Fabrica. Bailleae [ex officina Ioanis Oporini], 1543.
4. De Filippis Cappai C. Soranus of Ephesos and methodism. A gynecologist of the 2nd century A.D. *Minerva Ginecol* 1991; 43: 203-9.
5. Engel BT, Nikoomanesh P, Schuster MM. Operant conditioning of rectosphincteric responses in the treatment of fecal incontinence. *N Engl J Med* 1974; 290: 646-9.
6. Loening-Baucke V. Efficacy of biofeedback training in improving faecal incontinence and anorectal physiologic function. *Gut* 1990; 31: 1395-402.
7. Buser WD, Miner PB Jr. Delayed rectal sensation with fecal incontinence. Successful treatment using anorectal manometry. *Gastroenterology* 1986; 91: 1186-91.
8. Hardy KJ. Surgical history. Evolution of the stoma. *Aust N Z J Surg* 1989; 59: 71-7.
9. Watts JD, Rothenberger DA, Goldberg SM. Treatment of rectal prolapse. In: Henry MM, Swash M, eds. *Coloproctology and the Pelvic Floor*. London: Butterworths Publishing, 1985: 308-39.
10. Allingham W. *Fistula, haemorrhoids, painful ulcer, stricture, prolapses and other diseases of the rectum, their diagnosis and treatment*, 2nd ed. London: J&A Churchill, 1873.
11. Warren JC. A new method of operation for the relief of rupture of the perineum through the sphincter and rectum. *Trans Am Gynecol Soc* 1882; 7: 324.

12. Parks AG. Anorectal incontinence. *Proc R Soc Med* 1975; 68: 681-90.
13. Failes D, Killingback M, Stuart M. The surgical treatment of faecal incontinence. *Aust N Z J Surg* 1979; 49: 345-9.
14. Henry MM, Simson JN. Results of postanal repair: a retrospective study. *Br J Surg* 1985; 72 Suppl: 17-9.
15. Setti-Carraro P, Kamm MA, Nicholls RJ. Long-term results of post-anal repair for neurogenic faecal incontinence. *Br J Surg* 1994; 81: 140-4.
16. Jameson JS, Speakman CTM, Darzi A, Chia YW, Henry MM. Audit of postanal repair in the treatment of fecal incontinence. *Dis Colon Rectum* 1994; 37: 369-72.
17. Burnett SJD, Speakman CTM, Kamm MA, Bartram CI. Confirmation of endosonographic detection of external anal sphincter defects by simultaneous electromyographic mapping. *Br J Surg* 1991; 78: 448-50.
18. Blaisdell PC. Repair of the incontinent sphincter ani. *Surg Gynecol Obstet* 1940; 70: 692-7.
19. Browning GG, Motson RW. Anal sphincter injury; management and results of Parks sphincter repair. *Ann Surg* 1984; 199: 351-7.
20. Parks AG, McPartlin JF. Late repair of injuries of the anal sphincter. *J R Soc Med* 1971; 64: 1187-9.
21. Slade MS, Goldberg SM, Schottler JL, Balcos EG, Christenson CE. Sphincteroplasty for acquired anal incontinence. *Dis Colon Rectum* 1977; 20: 33-5.
22. Hussain SM, Stoker J, Kuiper JW, Schouten WR, den Hollander JC, Laméris JS. MR imaging of anal sphincter complex with an endoanal coil: normal anatomy and pathology. *Radiology* 1994; 193 (P): 445.
23. Parks AG. Anorectal incontinence. *J R Soc Med* 1975; 68: 21-30.
24. Miller R, Bartolo DCC, Locke-Edmunds JC, Mortensen NJMcC. Prospective study of conservative and operative treatment for

Introduction

- faecal incontinence. *Br J Surg* 1988; 75: 101-5.
25. Pescatori M, Anastasio G, Bottini C, Mentasti A. New grading and scoring for anal incontinence. Evaluation of 335 patients. *Dis Colon Rectum* 1992; 35: 482-7.
 26. Österberg A, Graf W, Karlbom U, Pålman. Evaluation of a questionnaire in the assessment of patients with faecal incontinence and constipation. *Scand J Gastroenterol* 1996; 31: 575-80.
 27. Enck P, Gabor S, Von Ferber L, Rathmann W, Erckenbrecht JF. Prevalence of fecal incontinence and degree of information possessed by family physicians and health insurance. *Z Gastroenterol* 1991; 29: 538-40.
 28. Brandeis GH, Ooi WL, Hossain M, Morris JN, Lipsitz LA. A longitudinal study of risk factors associated with the formation of pressure ulcers in nursing homes. *J Am Geriatr Soc* 1994; 42: 388-93.
 29. Nakanishi N, Tatara K, Shinsho F, Murakami S, Takatorige T, Fukuda H, Nakajima K, Naramura H. Mortality in relation to urinary and faecal incontinence in elderly people living at home. *Age & Ageing* 1999; 28: 301-6.
 30. Lara LL, Troop PR, Beadleson-Baird M. The risk of urinary tract infection in bowel incontinent men. *J Gerontol Nurs* 1990; 16: 24-6.
 31. Orbach CE, Tallent N. Modification of perceived body and of body concepts. *Arch Gen Psychiatry* 1965; 12: 126-35.
 32. Devlin HB, Plant JA, Griffin M. Aftermath of surgery for anorectal cancer. *BMJ* 1971; iii: 413-8.
 33. Thomas C, Madden F, Jehu D. Psychosocial morbidity in the first three months following stoma surgery. *J Psychosom Res* 1984; 28: 251-7.
 34. Thomas C, Madden F, Jehu D. Psychosocial effects of stoma. 1.

- Psychosocial morbidity one year after surgery. *J Psychosom Res* 1987; 31: 311-6.
35. Eardley A, George WD, Davis F, Schofield PF, Wilson MC, Wakefield J, Sellwood RA. Colostomy: the consequences of surgery. *Clin Oncol* 1976; 2: 277-83.
 36. Sutherland AM, Orbach CE, Dyk RB, Bard M. The psychological impact of cancer and cancer surgery. 1. Adaptation to the dry colostomy; preliminary report and summary of findings. *Cancer* 1952; 5: 857-72.
 37. Wirsching M, Druner HG, Hermann G. Results of psychosocial adjustment to long-term colostomy. *Psychother Psychosom* 1975; 26: 245-56.
 38. Druss RG, O'Connor JF, Stern LO. Psychologic response to colectomy. II. Adjustment to a permanent colostomy. *Arch Gen Psychiatry* 1969; 20: 419-27.
 39. Williams NS, Durdey P, Johnston D. The outcome following sphincter-saving resection and abdomino-perineal resection for lower rectal cancer. *Br J Surg* 1985; 72: 595-8.
 40. Thomas TM, Egan M, Walgrove A, Meade TW. The prevalence of faecal and double incontinence. *Community Med* 1984; 6: 216-20.
 41. Enck P, Bielefeldt K, Rathmann W, Purmann J, Tschöpe D, Erckenbrecht JF. Epidemiology of faecal incontinence in selected patient groups. *Int J Colorectal Dis* 1991; 6: 143-6.
 42. CBS. Lichamelijke beperkingen bij de nederlandse bevolking 1986/1988. SDU-uitgeverij 1990, 's-Gravenhage: 33.
 43. Roig JV, Garcia GA, Flors AC, Castells FP, Lledo Matoses S. Habitos defecatorios en poblacion laboral normal. *Revista Espanola de Enfermedades Digestivas* 1993; 84: 224-30.
 44. Talley NJ, O'keefe EA, Zinsmeister AR, Melton LJ 3rd. Prevalence of gastrointestinal symptoms in the elderly; a population-based study. *Gastroenterology* 1992; 102: 895-901.

Introduction

45. Kok AI, Voorhorst FJ, Burger CW, Van Houten P, Kenemans P, Janssens J. Urinary and faecal incontinence in community-residing elderly women. *Age & Ageing* 1992; 21: 211-5.
46. Denis P, Bercoff E, Bizien MF, Brocker P, Chassagne P, Lamouliatte H, Leroi AM, Perrigot M, Weber J. Etude de la prevalence de l'incontinence anale chez l'adulte. *Gastroenterol Clin Biol* 1992; 16: 344-50.
47. Borrie MJ, Davidson HA. Incontinence in institutions; costs and contributing factors. *Canadian Medical Association Journal* 1992; 147: 322-8.
48. Stelzner F, Beyenburg S, Hahn N. Acquired disorders of peritoneal cavity muscles. Abdominal wall denervation in pregnancy, denervation incontinence, and continent and incontinent constipation. *Langenbecks Arch Chir* 1993; 378: 49-59.
49. Johanson JF, Lafferty J. Epidemiology of fecal incontinence: the silent affliction. *Am J Gastroenterol* 1996; 91: 33-6.
50. Nelson R, Norton N, Cautley E, Furner S. Community-based prevalence of anal incontinence. *JAMA* 1995; 274: 559-61.
51. Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. *Gut* 1977; 18: 656-65.
52. Neill ME, Parks AG, Swash M. Physiological studies of the anal sphincter musculature in faecal incontinence and rectal prolapse. *Br J Surg* 1981; 68: 531-6.
53. Anderson RS. A neurogenic element to urinary genuine stress incontinence. *Br J Obstet Gynaecol* 1984; 91: 41-5.
54. Sun WM, Read NW, Prior A, Daly J, Cheah SK, Grundy D. The sensory and motor responses to rectal distension vary according to rate and pattern of balloon distension. *Gastroenterology* 1990; 99: 1008-15.
55. Bennett R, Duthie H. The functional importance of the internal anal sphincter. *Br J Surg* 1964; 51: 355-7.

56. Frenckner B, Ihre T. Influence of pudendal block on the function of the anal sphincter. *Gut* 1975; 16: 482-9.
57. Lestar R, Penninckx, Kerremans R. The composition of anal basal pressure-an in vivo and in vitro study in man. *Int J Colorectal Dis* 1989; 4: 118-22.
58. Thacker SB, Banta HD. Benefits and risks of episiotomy: an interpretative review of the English language literature 1860-1980. *Surg Gynecol Obstet* 1983; 38: 322-38.
59. Venkatesh KS, Ramanujam PS, Larson DM, Haywood MA. Anorectal complications of vaginal delivery. *Dis Colon Rectum* 1989; 32: 1039-41.
60. Walker JL. Complete perineal incision for delivery. *Southern Medical Journal* 1975; 67: 265-8.
61. Detlefsen GU, Vinther S, Larsen P, Schroeder E. Median and mediolateral episiotomy. *Ugeskr Læger* 1980; 142: 3114-6.
62. Fisher SR. Factors associated with the occurrence of perineal lacerations. *J Nurse Midwifery* 1979; 24: 18-26.
63. 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.
64. Sultan AH, Hudson CN. Are junior doctors and midwives adequately trained to repair the perineum? *J Obstet Gynaecol* 1993; 13: 484-5.
65. 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: 1317-22; discussion 1322-3.
66. 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.
67. Bek KM, Laurberg S. Risks of anal incontinence from subsequent

Introduction

- vaginal delivery after a complete obstetric anal sphincter tear. *Br J Obstet Gynaecol* 1992; 99: 724-6.
68. Møllerup Sørensen S, Bondesen H, Istre O, Vilmann P. Perineal rupture following delivery: long-term consequences. *Acta Obstet Gynecol Scand* 1988; 67: 315-8.
 69. Ryhammer AM, Bek KM, Laurberg S. Multiple vaginal deliveries increase the risk of permanent incontinence of flatus and urine in normal premenopausal women. *Dis Colon Rectum* 1995; 38: 1206-9.
 70. Cornes H, Bartolo DC, Stirrat GM. Changes in anal canal sensation after childbirth. *Br J Surg* 1991; 78: 74-7.
 71. Dorrance HR, Duthie GS, Rainey Livingston JB. The detection of occult sphincter injuries during pregnancy: should this affect mode of delivery? *Dis Colon Rectum* 1994; 37: P15.
 72. Bost BW. Should elective cesarean birth be offered at term as an alternative to labor and delivery for prevention of complications, including symptomatic pelvic prolapse, as well as stress urinary and fecal incontinence? *Obstet Gynecol* 2000; 95: S46.
 73. Seow-Choen F, Nicholls RJ. Anal fistula. *Br J surg* 1992; 79: 197-205.
 74. Marks CG, Ritchie JK. Anal fistulas at St. Marks Hospital. *Br J Surg* 1977; 64: 84-9.
 75. Lilius HG. Fistula in ano: investigation of human foetal anal ducts and intramuscular glands, and a clinical study of 150 patients. *Acta Chir Scand (Suppl)* 1968; 383: 7-88.
 76. Parks AG, Gordon PH, Hardcastle JD. A classification of fistula-in-ano. *Br J Surg* 1976; 63: 1-9.
 77. Pearl RK, Andrews JR, Orsay CP, Weisman RI, Prasad ML, Nelson RL, Clintron JR, Abcarian H. Role of the seton in the management of anorectal fistulas. *Dis Colon Rectum* 1994; 36: 573-7; discussion 577-9.

78. Abcarian H. Surgical correction of chronic anal fissure: results of lateral internal sphincterotomy vs. fissurectomy-midline sphincterotomy. *Dis Colon Rectum* 1980; 23: 31-6.
79. Hardy KJ, Cuthbertson AM. Lateral sphincterotomy: an appraisal with special reference to sequelae. *Aust N Z J Surg* 1969; 39: 91-3.
80. Lord PH. A new regime for the treatment of haemorrhoids. *Proc R Soc Med* 1968; 61: 935-6.
81. MacIntyre IMC, Balfour TW. Results of the Lord non-operative treatment for haemorrhoids. *Lancet* 1972; I: 1094-5.
82. Fusell K. Follow-up of Lord's procedure for haemorrhoids. *Lancet* 1972; 20: 1094-6.
83. Snooks SJ, Henry MM, Swash M. Faecal incontinence after anal dilatation. *Br J Surg* 1984; 71: 617-8.
84. Goligher JC. *Surgery of the anus, rectum and colon*. 3rd ed. London: Bailliere Tindall, 1975: 116-69.
85. Pescatori M, Mattana C. Factors affecting anal incontinence after restorative proctocolectomy. *Int J Colorectal Dis* 1990; 5: 213-8.
86. Allen RE, Hosker GL, Smith AR, Warrell DW. Pelvic floor damage and childbirth: a neurophysiologic study. *Br J Obstet Gynaecol* 1990; 97: 770-9.
87. Berkelmans I, Heresbach D, Leroi AM, Touchais JY, Martin PA, Weber J, Denis P. Perineal descent at defecography in women with straining at stool: a lack of specificity or predictive value for future anal incontinence? *Eur J Gastroenterol Hepatol* 1995; 7: 75-9.
88. Ellis CN, Coyle DJ, Blakemore WS. Pudendal nerve compression: a possible etiology of fecal incontinence. *Dis Colon Rectum* 1993; 36: P30.
89. Schuster MM, Tow DE, Sherbourne DH. Anal sphincter abnormalities characteristic of myotonic dystrophy. *Gastroenterology* 1965; 49: 641-8.

Introduction

90. Coller JA. Clinical application of anorectal manometry. *Gastroenterol Clin North Am* 1987; 16: 17-33.
91. Collins CD, Brown BH, Whittaker GE, Duthie HL. New method of measuring forces in the anal canal. *Gut* 1969; 10: 160-3.
92. Williamson JL, Nelson RL, Orsay C, Pearl RK, Abcarian H. A comparison of simultaneous longitudinal and radial recordings of anal canal pressures. *Dis Colon Rectum* 1990; 33: 201-6.
93. Bartolo DC. Pelvic floor disorders: incontinence, constipation and obstructed defecation. In: Schrock T, ed. *Perspect Color Surg* 1988; 1: 1-29.
94. Miller R, Bartolo DCC, Roe AM, Mortensen NJ. Assessment of microtransducers in anorectal manometry. *Br J Surg* 1988; 75: 40-3.
95. Lane RH. Clinical application of anorectal physiology. *Proc R Soc Med* 1975; 68: 28-30.
96. Wexner SD, Marchetti F, Salanga VD, Corredor C, Jagelman DG. Neurophysiologic assesment of the anal sphincters. *Dis Colon Rectum* 1991; 34: 606-12.
97. Brindley GA. Electroejaculation: its technique, neurological implications and uses. *Journal of Neurology, Neurosurgery and Psychiatry* 1981; 44: 9-18.
98. Kiff ES, Swash M. Slowed conduction in the pudendal nerves in idiopathic (neurogenic) faecal incontinence. *Br J Surg* 1984; 71: 614-6.
99. Kiff ES, Swash M. Normal proximal and delayed distal conduction in the pudendal nerves of patients with idiopathic (neurogenic) faecal incontinence. *J Neurol Neurosurg Psychiatry* 1984; 47: 820-3.
100. Swash M. New concepts in incontinence. *Br Med J* 1985; 290: 4-5.
101. Snooks SJ, Swash M, Henry MM, Setchell MM. Risk factors in childbirth causing damage to the pelvic floor innervation. *Int J*

- Colorect Dis 1986; 1: 20-4.
102. Kiff ES, Barnes PRH, Swash M. Evidence of pudendal neuropathy in patients with perineal descent and chronic straining at stool. *Gut* 1984; 25: 1279-82.
 103. Snooks SJ, Badenoch D, Tiptaft R, Swash M. Perineal nerve damage in genuine stress urinary incontinence: an electrophysiological study. *Br J Urol* 1985; 57: 422-6.
 104. Snooks SJ, Swash M, Henry MM. Abnormalities in central and peripheral nerve conduction in patients with anorectal incontinence. *J R Soc Med* 1985; 78: 294-300.
 105. Snooks SJ, Henry MM, Swash M. Anorectal incontinence and rectal prolapse: differential assessment of the innervation to puborectalis and external anal sphincter muscles. *Gut* 1985; 26: 470-6.
 106. Snooks SJ, Nicholls RJ, Henry MM, Swash M. Electrophysiological and manometric assessment of the pelvic floor in solitary rectal ulcer syndrome. *Br J Surg* 1985; 72: 131-3.
 107. Vaccaro CA, Cheong DM, Wexner SD, Nogueras JJ, Salanga VD, Hanson MR, Phillips RC. Pudendal neuropathy in evacuatory disorders. *Dis Colon Rectum* 1995; 38: 166-71.
 108. Roig JV, Villoslada C, Lledo S, Solana A, Buch E, Alos R, Hinojosa J. Prevalence of pudendal neuropathy in fecal incontinence. Results of a prospective study. *Dis Colon Rectum* 1995; 38: 952-8.
 109. Gee AS, Mills A, Durdey P. What is the relationship between perineal descent and anal mucosal electrosensitivity? *Dis Colon Rectum* 1995; 38: 419-23.
 110. Broden B, Snellman B. Procidentia of the rectum studied with cineradiography: a contribution to the discussion of causative mechanism. *Dis Colon Rectum* 1968; 11: 330-47.
 111. Mahieu P, Pringot J, Bodart P. Defecography: I. description of a new procedure and results in normal patients. *Gastrointest Radiol* 1984; 9: 247-51.

Introduction

112. Wexner SD, Jagelman DG. Chronic constipation. *Postgrad Adv Color Surg* 1989; 1: 1-22.
113. Bartolo DC, Bartram CI, Ekberg O, Fork FT, Kodner I, Kuijpers JH, Mahieu PH, Shorvon PJ, Stevenson GW, Womack N. Proctography symposium. *Int J Colorectal Dis* 1988; 3: 67-89.
114. Bielefeldt K, Enck P, Zamboglou N, Erckenbrecht JF, Modder U. Radiologische Diagnostik bei analer Inkontinenz. *Rontgenblätter* 1990; 43: 256-60.
115. Goei R, van Engelshoven J, Schouten H, Baeten C, Stassen C. Anorectal function: defecographic measurement in asymptomatic subjects. *Radiology* 1989; 173: 137-41.
116. Shorvon PJ, McHugh S, Diamant NE, Somers S, Stevenson GW. Defecography in normal volunteers: results and implications. *Gut* 1989; 30: 1737-49.
117. Bartram CI, Turnbull GK, Lennard-Jones JE. Evacuation proctography: an investigation of rectal expulsion in 20 subjects without defecatory disturbances. *Gastrointest Radiol* 1988; 13: 72-80.
118. Freimanis MG, Wald A, Caruana B, Bauman DH. Evacuation proctography in normal volunteers. *Invest Radiol* 1991; 26: 581-5.
119. 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: 1430-4.
120. Sentovich SM, Wong WD, Blatchford GJ. Accuracy and reliability of transanal ultrasound for anterior anal sphincter injury. *Dis Colon Rectum* 1998; 41: 1000-4.
121. Felt-Bersma RJF, van Baren R, Koorevaar M, Strijers RL, Cuesta MA. Unsuspected sphincter defects shown by anal endosonography after anorectal surgery: a prospective study. *Dis Colon Rectum* 1995; 38: 249-53.
122. Oliveira L, Nogueras JJ, Weiss EG, Pfeifer J, Gonzalez A, Wexner

- SD. The role of endoanal ultrasonography for fecal incontinence. *Dis Colon Rectum* 1995; 38: P30.
123. Rociu E, Stoker J, Eijkemans MJC, Schouten WR, Laméris JS. Fecal incontinence: endoanal US versus endoanal MR imaging. *Radiology* 1999; 212: 453-8.
124. Bliss DZ, McLaughlin J, Jung HJ, Lowry A, Savik K, Jensen L. Comparison of the nutritional composition of diets of persons with fecal incontinence and that of age- and gender-matched controls. *J Wound Ostomy Continence Nurs* 2000; 27: 90-97.
125. Sun WM, Read NW, Verlinden M. Effects of loperamide oxide on gastrointestinal transit time and anorectal function in patients with chronic diarrhoea and faecal incontinence. *Scand J Gastroenterol* 1997; 32: 34-8.
126. Musial F, Enck P, Kalveram KT, Erckenbrecht JF. The effect of loperamide on anorectal function in normal healthy men. *J Clin Gastroenterol* 1992; 15: 321-4.
127. Hoffmann BA, Timmcke AE, Gathright JB Jr, Hicks TC, Opelka FG, Beck DE. Fecal seepage and soiling: a problem of rectal sensation. *Dis Colon Rectum* 1995; 38: 746-8.
128. Suarez FL, Springfield J, Levitt MD. Identification of gases responsible for the odour of human flatus and evaluation of a device purported to reduce this odour. *Gut* 1998; 43: 100-4.
129. Mortensen N, Humphreys MS. The anal continence plug: for patients with anorectal incontinence. *Lancet* 1991; 338: 295-7.
130. Christiansen J, Roed-Petersen K. Clinical assessment of the anal continence plug. *Dis Colon Rectum* 1993; 36: 740-2.
131. Enck, P. Biofeedback training in disordered defecation. A critical review. *Dig Dis Sci* 1993; 38, 1953-60.
132. Norton C, Kamm MA. Outcome of biofeedback for faecal incontinence. *Br J Surg* 1999; 86: 1159-63.
133. Macleod SH. Biofeedback in the management of partial anal

Introduction

- incontinence. *Dis Colon Rectum* 1983; 26: 242-6.
134. Korsgen S, Deen KI, Keighley MR. Long-term results of total pelvic floor repair for postobstetric fecal incontinence. *Dis Colon Rectum* 1997; 40: 835-9.
 135. Pickrell KL, Broadbent TR, Master FW, Metger JT. Construction of a rectal sphincter and restoration of anal incontinence by transplanting the gracilis muscle; a report of four cases in children. *Ann Surg* 1952; 135: 853-63.
 136. Baeten CG, Geerdes BP, Adang EM, Heineman E, Konsten J, Engel GL, Kester AD, Spaans F, Soeters PB Anal dynamic graciloplasty in the treatment of intractable fecal incontinence. *N Engl J Med* 1995; 332: 1600-5.
 137. Madoff RD, Rosen HR, Baeten CG, LaFontaine LJ, Cavina E, Devesa M, Rouanet P, Christiansen J, Faucheron JL, Isbister W, Kohler L, Guelinckx PJ, Pählman L. Safety and efficacy of dynamic muscle plasty for anal incontinence: lessons from a prospective, multicenter trial. *Gastroenterology* 1999; 116: 549-56.
 138. Weston PM, Morgan JD, Hussain J, Stephenson TP. Artificial urinary sphincters around intestinal segments-are they safe? *Br J Urol* 1991; 67: 150-4.
 139. Wong, WD, Jensen LL, Bartolo DCC, Rothenberger DA. Artificial anal sphincter. *Dis Colon Rectum* 1996; 39: 1345-51.
 140. Vaizey CJ, Kamm MA, Gold DM, Bartram CI, Halligan S, Nicholls RJ. Clinical, physiological, and radiological study of a new purpose-designed artificial bowel sphincter. *Lancet* 1998; 352: 105-9.
 141. Lehur PA, Glemain P, Bruley des Varannes S, Buzelin JM, Leborgne J. Outcome of patients with an implanted artificial anal sphincter for severe faecal incontinence. *Int J Colorectal Dis* 1998; 13: 88-92.
 142. Christiansen J, Rasmussen OØ, Lindorff-Larsen K. Long-term

- results of artificial anal sphincter implantation for severe anal incontinence. *Ann Surg* 1999; 230: 45-8.
143. Congilosi SM, Johnson DR, Medot M, Tretinyak A, McCormick SR, Wong WD, Rothenberger DA, Madoff RD. Experimental model of pudendal nerve innervation of a skeletal muscle neosphincter for faecal incontinence. *Br J Surg* 1997; 84: 1269-73.
 144. Shafik A, el-Sherif M, Youssef A, Olfat ES. Surgical anatomy of the pudendal nerve and its clinical implications. *Clin Anat* 1995; 8: 110-5.
 145. Shafik A. Perianal injection of autologous fat injection for treatment of sphincteric incontinence. *Dis Colon Rectum* 1995; 38: 583-7.
 146. Shafik A. Polytetrafluoro-ethylene injection for the treatment of partial fecal incontinence. *Int Surg* 1993; 78: 159-61.
 147. Binnie NR, Kawimbe BM, Papachrysostomou M, Smith AN. Use of the pudendo-anal reflex in the treatment of neurogenic faecal incontinence. *Gut* 1990; 31: 1051-5.
 148. Matzel KE, Stadelmaier U, Hohenfellner M, Gall FP. Permanent electrostimulation of sacral spinal nerves with an implantable neurostimulator in treatment of fecal incontinence. *Chirurg* 1995; 66: 813-7.
 149. Vaizey CJ, Kamm MA, Roy AJ, Nicholls RJ. Double-blind crossover study of sacral nerve stimulation for fecal incontinence. *Dis Colon Rectum* 2000; 43: 298-302.

CHAPTER 2

CLINICAL VALUE OF COLONIC IRRIGATION IN PATIENTS WITH CONTINENCE DISTURBANCES

JW Briel¹, WR Schouten¹, EA Vlot¹, S Smits², and I van Kessel².

From the Departments of ¹General Surgery and ²Stomacare,
University Hospital Dijkzigt, Rotterdam, The Netherlands

Dis Colon Rectum 1997; 40: 802-5.

Abstract

Continence disturbances, especially faecal soiling, are difficult to treat. Irrigation of the distal part of the large bowel might be considered as a nonsurgical alternative for patients with impaired continence. This study is aimed at evaluating the clinical value of colonic irrigation. Thirty-two patients (16 females; median age 47 (range 23-72) years) were offered colonic irrigation on an ambulatory basis. Sixteen patients suffered from faecal soiling (Group I), whereas the other 16 patients were treated for faecal incontinence (Group II). Patients were instructed by enterostomal therapists how to use a conventional colostomy irrigation set to obtain sufficient irrigation of the distal part of their large bowel. Patients with continence disturbances during daytime were instructed to introduce 500 to 1000 ml of warm (38° C) water within 5 to 10 minutes after they passed their first stool. In addition, they were advised to wait until the urge to defecate was felt. Patients with soiling during overnight sleep were advised to irrigate during the evening. To determine clinical outcome, a detailed questionnaire was used. Median duration of follow-up was 18 months. Ten patients discontinued irrigation within the first month of treatment. Symptoms resolved completely in two patients. They believed that there was no need to continue treatment any longer. Irrigation had no effect in two patients. Despite the fact that symptoms resolved, six patients discontinued treatment because they experienced pain (n=2) or they considered the irrigation to be too time-consuming (n=4). Twenty-two patients are still performing irrigations. Most patients irrigated the colon in the morning after the first stool was passed. Time needed for the washout varied between 10 and 90 minutes. Frequency of irrigations varied from two times per day to two times per week. In Group I, irrigation was found to be beneficial in 92 per cent of patients, whereas 60 per cent of patients in Group II considered the treatment as a major improvement to the quality of their lives. If patients who discontinued treatment because of washout-

related problems are included in the assessment of final outcome, the success-rate is 79 per cent and 38 per cent respectively. Patients with faecal soiling benefit more from colonic irrigation than patients with incontinence for liquid or solid stools. If creation of a stoma is considered, especially in patients with intractable and disabling soiling, it might be worthwhile to treat these patients first by colonic irrigation.

Introduction

Disturbed continence is a frequently neglected but rather common complication of anorectal surgery. Faecal soiling, for example, occurs frequently after fistulotomy, lateral internal sphincterotomy, and haemorrhoidectomy. Impaired continence may persist after an attempt to restore continence for liquid or solid stool by external anal sphincter plication. Both faecal soiling and faecal incontinence are distressing conditions for the patient and can cause social isolation. For some of these patients creation of a stoma might finally be inevitable.

Irrigation of a colostomy has been used for a long time. This treatment modality has been proven to be safe and provides the opportunity to avoid use of a colostomy bag¹. In 1989, Iwama *et al.*² described rectal application of this method to a group of ten patients who complained of difficulty in defecation following anterior resectosigmoid resection. Based on our personal experience with colostomy irrigation and results obtained from the study by Iwama *et al.*², we decided to offer colonic irrigation to a selected group of patients with impaired continence. Because there are no data available regarding the potential role of colonic irrigation in the treatment of continence disturbances, it seems worthwhile to report our experience with this treatment modality.

Patients & Methods

Thirty-two patients were offered colonic irrigation on an ambulatory basis during the time period between 1989 and 1995. Group I comprised 16 patients with faecal soiling (11 males; median age 45 (range 23-59) years), Group II consisted of 16 patients with faecal incontinence (5 males; median age 52 (range 25-72) years). History of the patients is listed in *Table 1*. Hospital records and outpatient clinic charts were analyzed, and follow-up information was obtained both from review of charts and personal telephone communication by one author (EAV) who had not participated in any of the instructions. A questionnaire was used to determine clinical outcome. This form included the prevalence of accidental bowel movements, use of pads, patient satisfaction, and degree of social isolation.

Colonic Irrigation

The patient was instructed by enterostomal therapists. All patients received the same instructions, both verbal and written. A conventional colostomy irrigation set was used. The set consisted of an irrigation bag, a tube, and a cone tip (Biotrol Iryflex, B. Braun Medical BV, *Fig. 1*).

Patients were instructed to hang the irrigation bag at shoulder height or 1 meter above the toilet seat. The volume of warm water to be used for irrigation was 500 to 1000 ml. It was essential to run water through the feeding tube prior to insertion to avoid inflating the colon with air. When the patient sat on the toilet seat, the lubricated cone tip was introduced into the anal canal. Irrigation water was instilled within five to ten minutes. The patient was instructed to wait several minutes, until the urge to defecate was felt before taking out the cone tip. After removal of the cone tip, expulsion of irrigation solution could take place. Patients with continence disturbances during the daytime were advised to irrigate after they passed

Table 1. History of the patients.

<i>History</i>	<i>Group I</i>	<i>Group II</i>
Fistulotomy	7	3
Following sphincter repair	2	4
Idiopathic (neurogenic)	-	4
Haemorrhoidectomy	1	2
Sphincterotomy	2	-
Lord procedure	2	-
Irritable bowel syndrome	1	1
Anorectal malformation	-	2
Coloanal anastomosis	1	-

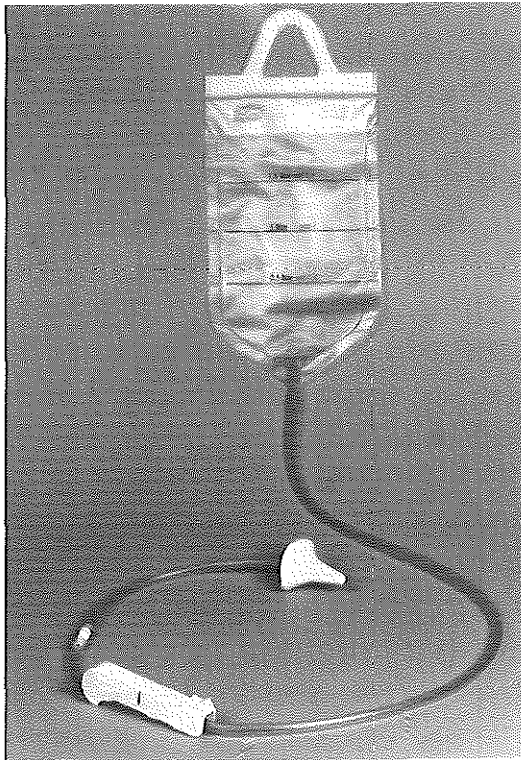


Figure 1. Irrigation bag with the cone tip at the end of the tube.

Colonic Irrigation

their first stools, whereas patients with soiling during the night sleep were advised to irrigate during the evening. Some cautionary remarks were made when the patient was instructed. Water to be used for the washout had to be approximately 38° C. Cold water may lead to collapse. To prevent nausea, it is recommended that washout be performed at least two hours after having a meal.

Results

Median duration of follow-up was 18 months. Ten patients decided to discontinue irrigations within the first month of treatment. In two incontinent patients, this decision was based on the fact that they observed no effect of the treatment. Subsequently, in these two patients, a stoma was created. In two patients in Group I, original complaints disappeared, and thereby the need for irrigation. Painful sensation during irrigation and the time consuming aspect of treatment were arguments to discontinue the treatment in, respectively, two and four patients. Twenty-two patients still perform colonic irrigation.

In almost three-fourths of the patients, the time period shortly after the first stool was found to be the most appropriate time for irrigation, because these patients experienced incontinence or soiling during daytime. Twenty-four per cent of patients decided to perform colonic irrigation before sleep to avoid soiling during overnight sleep. Median duration of the procedure was 30 (range 10-90) minutes. In 87 per cent of patients one or more colonic irrigations per day were required.

Seventeen patients (77 per cent) of the 22 who are still continuing the treatment reported a successful outcome of the colonic irrigation. Results and indications for irrigations are listed in *Table 2*. If patients who discontinued treatment because of irrigation-related problems are included in the assessment of final outcome, the success-rate drops (*Table 3*).

Table 2. Clinical outcome in patients who continued treatment.

<i>Indication</i>	<i>No. of Patients</i>	<i>Success-rate (%)</i>
Faecal soiling	12	92
Faecal incontinence	10	60
Total	22	77

Table 3. Overall clinical outcome.

<i>Indication</i>	<i>No. of Patients</i>	<i>Success-rate (%)</i>
Faecal soiling	14	79
Faecal incontinence	16	38
Total	30	57

Discussion

Disturbed continence is a frequently neglected but rather common complication following anorectal surgery. Fistula surgery may result in impaired continence. Although incontinence for solid stool rarely occurs, minor defects in continence may follow if even a small amount of sphincter muscle is damaged. The incidence after fistulotomy varies from 18 to 52 per cent³⁻⁶. Internal sphincterotomy may also lead to continence disturbances caused by inadequate closure of the anal canal by the internal anal sphincter. This condition is usually temporary but may become permanent. Faecal soiling after lateral internal sphincterotomy is reported to vary from 0 to 47 per cent^{7,8}. Continence disorders also occur after nonmuscle-splitting

Colonic Irrigation

procedures, such as anal stretch procedures (15-25 per cent)⁹⁻¹¹. In modern surgery for haemorrhoids, incontinence is a rare complication. However, if the sphincter mass is inadvertently injured, incontinence may result (20 per cent)¹². Following conventional anterior resection, continence is generally maintained. However, when anastomosis is performed in the distal third of the rectum, impairment of continence might occur. Lewis *et al.*¹³ pointed out that continence after low anterior resection is related to the level of the anastomosis and to an appropriate sampling response in the anal sphincter to activity within the neorectum. This in turn, is directly related to length of the residual rectum, which is, therefore, of crucial importance to function¹³. Childbirth is the most common cause of incontinence¹⁴. Primary repair of obstetric anal sphincter injury is advisable whenever feasible. Despite this primary gynaecologic repair, persistent defects can be demonstrated using endoanal ultrasound in 85 per cent of patients¹⁵. A substantial part of patients with such an occult defect will become incontinent with increasing age. In this group, a secondary repair might be necessary. However, despite a secondary repair, faecal incontinence may still persist. Following postanal repair, continence disturbances persist in up to 54 per cent of patients¹⁶, whereas the failure-rate at long-term follow-up of anterior anal repair is as high as 53 per cent¹⁷. Recently, it has been shown that these results cannot be improved by combining anterior and postanal repair¹⁸. For patients with persistent incontinence after conventional treatment, such as sphincter repair and biofeedback, only a few options are left. Until recently, in most patients creation of a stoma was considered. Nowadays the dynamic graciloplasty seems to be an excellent, rather expensive alternative. Faecal soiling, caused by internal anal sphincter damage after anal surgery, is a troublesome condition, which is rather difficult to treat. Until now, there have been no data available regarding the potential role of irrigations in the treatment of postoperative soiling. In this study colonic irrigation was found to be beneficial in 92 per cent of patients with faecal soiling, whereas 60 per cent of patients with faecal incontinence considered

the treatment as a major improvement to quality of their lives. If patients who discontinued treatment because of washout-related problems are included in the assessment of final outcome, the success-rate is 79 and 38 per cent, respectively. As a last resort for patients with disabling continence disturbances, construction of a colostomy may become necessary. If creation of a stoma is considered, especially in patients with intractable soiling, it might be worthwhile to treat these patients first by colonic irrigation.

References

1. Binkley GE. Construction and care of abdominal colostomy. *Am J Surg* 1952; 83: 807-91.
2. Iwama T, Imajo M, Yaegashi K, Mishima Y. Self washout method for defecational complaints following low anterior rectal resection. *Jpn J Surg* 1989; 19: 251-3.
3. Seow-Choen F, Nicholls RJ. Anal fistula. *Br J Surg* 1992; 79: 197-205.
4. Marks CG, Ritchie JK. Anal fistulas at St. Marks Hospital. *Br J Surg* 1977; 64: 84-9.
5. Lilius HG. Fistula in ano: investigation of human foetal anal ducts and intramuscular glands, and a clinical study of 150 patients. *Acta Chir Scand (Suppl)* 1968; 383: 1-88.
6. Parks AG, Gordon PH, Hardcastle JD. A classification of fistula-in-ano. *Br J Surg* 1976; 63: 1-9.
7. Abcarian H. Surgical correction of chronic anal fissure: results of lateral internal sphincterotomy versus fissurectomy-midline sphincterotomy. *Dis Colon Rectum* 1980; 23: 31-6.
8. Hardy KJ, Cuthbertson AM. Lateral sphincterotomy: an appraisal with special reference to sequelae. *Aust N Z J Surg* 1969; 39: 91-3.
9. Fussell K. Follow-up of Lord's procedure for haemorrhoids. *Proc R Soc Med* 1973; 66: 246-7.
10. Macintyre IM, Balfour TW. Results of the Lord non-operative treatment for haemorrhoids. *Lancet* 1972; 1 (760): 1094-5.
11. Snooks S, Henry MM, Swash M. Faecal incontinence after anal dilatation. *Br J Surg* 1984; 71: 617-8.
12. Goligher JC. *Surgery of the anus, rectum and colon*. 3rd ed. London: Bailliere Tindall, 1975: 116-69.
13. Lewis WG, Martin IG, Williamson ME, Stephenson BM, Huldsworth PJ, Finan PJ, Johnston D. Why do some patients experience poor

functional results after anterior resection of the rectum for carcinoma? *Dis Colon Rectum* 1995; 38: 259-63.

14. Ctercteko GC, Fazio VW, Jagelman DG, Lavery IC, Weakly FL, Melia M. Anal sphincter repair: a report of 60 cases and review of the literature. *Aust N Z J Surg* 1988; 58: 703-10.
15. Sultan AH, Kamm MA, Hudson CN, Bartram CI. Third degree obstetric anal sphincter tears: risk factor and outcome of primary repair. *BMJ* 1994; 308: 887-91.
16. Briel JW, Schouten WR. Disappointing results of postanal repair in the treatment of fecal incontinence. *Ned Tijdschr Geneeskd* 1995; 139: 23-6.
17. Laurberg S, Swash M, Henry MM. Delayed external sphincter repair for obstetric tear. *Br J Surg* 1988; 75: 786-8.
18. Keighley MR, Korsgen S. Long-term results and predictive parameters of outcome following total pelvic floor repair. *Dis Colon Rectum* 1996; 39: A15.

CHAPTER 3

LONG-TERM RESULTS OF SUTURE RECTOPEXY IN PATIENTS WITH FAECAL INCONTINENCE ASSOCIATED WITH INCOMPLETE RECTAL PROLAPSE

JW Briel, WR Schouten, and MO Boerma.

From the Department of General Surgery,
University Hospital Dijkzigt, Rotterdam, The Netherlands

Dis Colon Rectum 1997; 40: 1228-32.

Abstract

Suture rectopexy is the recommended therapy for complete rectal prolapse that is associated with faecal incontinence. It has been suggested that correction of an incomplete rectal prolapse is also worthwhile for patients with faecal incontinence. Aims of this study were 1) to evaluate the clinical outcome of suture rectopexy in a consecutive series of patients with incomplete rectal prolapse associated with faecal incontinence, and 2) to compare these results with those obtained from patients with complete rectal prolapse. Between 1979 and 1994, suture rectopexy was performed in 13 incontinent patients (3 males; median age 65 (range 45-77) years) with incomplete rectal prolapse (Group I) and in 24 incontinent patients (3 males; median age 71 (range 24-86) years) with complete rectal prolapse (Group II). After a median follow-up of 67 months, continence was restored in 5 out of 13 (38 per cent) patients with incomplete rectal prolapse and in 16 out of 24 (67 per cent) patients with complete rectal prolapse. In both groups, all male patients became continent. For the majority of incontinent patients with incomplete rectal prolapse, a suture rectopexy is not beneficial. The clinical outcome of this procedure is only good in incontinent patients with complete rectal prolapse. Based on these data, it is questionable whether incomplete rectal prolapse plays a causative role in faecal incontinence.

Introduction

Complete rectal prolapse is associated with faecal incontinence in more than 50 per cent of cases¹. It is well documented that suture rectopexy is beneficial for those individuals, restoring continence in 50 to 75 per cent of patients². Incomplete rectal prolapse, first described by Allingham³ in 1873, is generally considered as the precursor of complete rectal prolapse^{4,7}.

Therefore, it has been suggested that this condition also plays a causative role in the origin of faecal incontinence. Surgical treatment of incomplete prolapse has followed the same lines as that of complete rectal prolapse, i.e., some type of rectopexy. Until now, only a few authors have reported the results of rectopexy in incontinent patients with rectal intussusception⁸⁻¹⁰. Aims of the present study were to 1) to evaluate the clinical outcome of suture rectopexy in a consecutive series of patients with incomplete rectal prolapse, which was associated with faecal incontinence, and 2) to compare these results with those obtained from patients with complete rectal prolapse.

Patients & Methods

During a 15-year period between 1979 and 1994, 13 incontinent patients with incomplete rectal prolapse (Group I: 3 males, 10 females; median age 65 (range 45-77) years) and 24 incontinent patients with complete rectal prolapse (Group II: 3 males, 21 females, median age 71 (range 24-86) years) were studied. Complete prolapse was defined as full-thickness protrusion of the rectal wall through the anal orifice. Additional investigation was not performed in patients with such a complete prolapse. During the period studied all other patients with faecal incontinence underwent evacuation proctography. Incomplete prolapse was defined as an intussusception of the rectum that did not transgress the anal canal. Patients underwent rectopexy if physical examination did not reveal a sphincter defect and if an intussusception of the rectum was found on evacuation proctography. In these patients, the incomplete prolapse was considered the cause of their problems with continence. Only during the last years of the period studied additional sphincter-mapping and anorectal manometry was available. Hospital records from the time of the operation and outpatient clinic charts were analyzed, and follow-up information was

Rectopexy

obtained both from review of charts and from personal telephone communication by one author (MOB) who had not participated in any of the operations. The degree of incontinence was assessed and graded as described by Parks¹¹: Grade I, fully continent; Grade II, soiling and incontinence for gas; Grade III, incontinence for liquids; Grade IV, incontinence to solid stool. Restoration of continence from Grade IV to Grade II or I, or from Grade III to Grade I, was defined as a successful result. During the telephone communications, postoperative grading was assessed without the knowledge of the preoperative continence status.

Surgical Treatment

The suture rectopexy was performed after conventional preoperative bowel preparation (polyethylene glycol). With the patient placed in the supine position an abdominal incision was made to mobilize the rectum to the level of the pelvic floor. The lateral ligaments were preserved. Fixation of the posterior rectal wall to the presacral fascia was performed using absorbable sutures. No slings or other foreign material were utilized.

Results

No operative deaths occurred in this series. Early postoperative complications included urinary tract infection and a wound abscess in, respectively, three and four patients. Median duration of follow-up was 67 (range 10-181) months. Preoperatively, 30 patients were fully incontinent (Grade IV), whereas in 7 patients, incontinence was scored as Grade III. Treatment was successful in 5 of 13 (38 per cent) patients in Group I and in 16 of 24 (67 per cent) patients in Group II. In both groups, all male patients became continent. The overall success-rate was 57 per cent. None of the

patients with restored continence presented with recurrent incontinence during the follow-up period. Recurrence of complete rectal prolapse was not observed. Eight patients who remained incontinent after suture rectopexy were treated by postanal repair. This procedure was successful in only one patient. One incontinent patient required a subsequent ileostomy.

Discussion

Complete rectal prolapse is a disabling condition which is more common in females and is encountered more frequently with increasing age. Its earliest description dates back to 1500 B.C. in the Ebers Papyrus¹². Recurrent prolapse following the Ripstein procedure has been reported in 0 to 13 per cent of patients^{13,14}. Following surgery, continence is restored in 40 to 64 per cent of patients². Faecal incontinence in patients with complete rectal prolapse may result from injury to the nerve supply and subsequent denervation of the pelvic floor and anal sphincters caused by chronic stretching and from direct repetitive muscle damage caused by the prolapse itself^{6,15}. Rectal prolapse often precedes incontinence, and in these patients, it seems probable that the prolapse itself is caused by a weakness of the pelvic floor muscles, as has been described in patients with cauda equina lesions¹⁶. In those patients in whom rectal prolapse occurs without incontinence on the other hand, the pelvic floor musculature (as assessed by clinical examination, anorectal manometry, and electromyography) is frequently normal¹⁷. Other contributing factors include chronic inhibition of the internal anal sphincter *via* the rectoanal inhibitory reflex¹⁸ and loss of anorectal sensation⁸. Electromyographic studies showed increased fiber density in the puborectalis muscle and external anal sphincter of incontinent patients with a complete rectal prolapse compared with normal controls and continent patients with rectal prolapse^{17,19}. The mechanisms responsible for restoration of continence after rectopexy are not fully understood, although

Rectopexy

improved internal anal sphincter function might be of importance¹⁸. Recovery of continence is associated with abolition of high-pressure rectal waves, which produce maximal inhibition of sphincter activity before operation²⁰. Furthermore, recovery of electromyographic activity of the internal anal sphincter²¹, improvement in anorectal sensation^{22,23}, and an increase in anal resting pressures²¹⁻²⁷ have been reported following rectopexy for complete rectal prolapse. Regardless of the mechanism involved, most authors agree that complete rectal prolapse must be operated on before irreversible damage to the anal sphincters has occurred. In our series, continence was restored in 16 of 24 (67 per cent) patients with complete rectal prolapse. These results are concomitant with those obtained in other studies on the effect of rectopexy on complete rectal prolapse².

It has been reported that incomplete rectal prolapse is also associated with faecal incontinence⁸⁻¹⁰. Rectal intussusception is considered to be a stage towards the development of rectal prolapse, because evacuation proctography reveals that rectal intussusception starts at the same level as in complete rectal prolapse^{5,7}. Surgical treatment of incomplete rectal prolapse has followed the same lines as that of complete rectal prolapse. There have been only a few reports that specifically focus on restoration of continence after surgical repair of this condition⁸⁻¹⁰. In our series continence was restored in 5 of 13 (38 per cent) patients with incomplete prolapse.

Ihre²⁸ reported that internal intussusception is three times more common than complete rectal prolapse. Women are affected six times more commonly than men, with a peak incidence for women in the fifth decade²⁹. Although rectal intussusception has been considered as a precursor of complete rectal prolapse, it has been questioned whether the incomplete prolapse, as demonstrated by evacuation proctography, is abnormal or not. Goei *et al.*³⁰ demonstrated rectal intussusception in 23 per cent of asymptomatic women after menopause. Shorvon and colleagues³¹ reported even higher figures; they found intussusception in 50 per cent of young

female volunteers. Although Wiersma³² was not able to confirm this finding, neither in women, nor in men, it is generally agreed that rectal intussusception is a common finding in asymptomatic controls^{30,31,33-35}. Therefore, most authors have questioned the clinical relevance of intussusception. This uncertainty is underscored by the findings of our study because the majority of our patients with rectal intussusception did not experience restoration of continence after rectopexy. In contrast, Ihre and Seligson⁸ and Delemarre *et al.*⁹ noted restoration of continence after surgical correction of incomplete rectal prolapse in, respectively, 77 and 62 per cent of their patients. The differences in clinical outcome are difficult to explain. Duration of follow-up in both studies was within the same range as that of our present study. Both authors performed an original Ripstein procedure, characterized by a posterior rectopexy using Marlex mesh. It might be possible that a suture rectopexy is less successful than the rectopexy using Marlex mesh. However, Duthie and Bartolo²³ found no significant differences regarding clinical outcome between different methods of rectopexy. So, it seems unlikely that the conflicting results are caused by differences in surgical technique. It might also be possible that persistent incontinence in our patients is due to recurrent intussusception. However, the recurrence rate on evacuation proctography is reported to be very low^{6,9,36-38}. Moreover, in our series, there were no recurrences after rectopexy for complete rectal prolapse. Therefore our fixation technique seems to be adequate. Ihre and Seligson⁸ defined improvement of continence as a successful outcome. However, they did not use the classification according to Parks¹¹. In the present study, we defined restoration of continence from Grade IV to Grade II or I, or from Grade III to Grade I, as a successful result only. In this respect, it might be possible that Ihre included patients with minor improvement in continence in his success-rate⁸. On the other hand, Delemarre and colleagues⁹ used the same classification in their study as we did and had better results. Because intussusception of the rectum is considered to be a precursor of complete

Rectopexy

rectal prolapse, it seems likely that the extent of the intussusception is related to the final outcome after prolapse repair. If this hypothesis is true, it might be possible that in the series of Ihre and Seligson⁸ and Delemarre *et al.*⁹ patients presented with an intussusception of a greater extent than in our series and had subsequently a better result. Recently, Schultz *et al.*¹⁰ studied 12 patients in whom the rectal intussusception was associated with unacceptable faecal incontinence. The authors reported improvement of continence after rectopexy. However, their results can not be compared with those of the present study because Schultz¹⁰ and coworkers graded faecal incontinence in a different way.

Although rectal intussusception is considered to be a stage toward development of rectal prolapse, progression of incomplete prolapse to complete prolapse is not inevitable. Ihre²⁸ noted that only 1 of 20 patients with incomplete prolapse who were not treated surgically went on to develop complete prolapse. Similarly, Broden and Snellman⁷ demonstrated that during two years, only 2 of their 21 patients developed complete rectal prolapse. Therefore, they concluded that rectopexy cannot be recommended prophylactically in these patients.

Conclusions

For the majority of patients with incomplete prolapse associated with faecal incontinence, a rectopexy is not beneficial. Because incomplete prolapse is frequently seen on defaecography in asymptomatic controls, it is questionable whether this abnormality plays a causative role in faecal incontinence. Regardless of the causative factor, these patients suffer from faecal incontinence. Although studies suggested a high frequency of mechanical disruption of anal sphincters in women who presented with incontinence, the incidence of such damage after childbirth was unknown until Sultan *et al.*³⁹ studied 202 consecutive pregnant women. Thirty-five

per cent of primiparae who delivered vaginally developed a sphincter defect involving one or both muscles, and this damage persisted at six months. Furthermore, when endoanal ultrasound was first applied to a consecutive series of women presenting with faecal incontinence, in whom the only apparent risk factor was obstetric damage, 90 per cent were found to have a structural abnormality in one or both sphincters⁴⁰. These findings also cast doubt on the importance of incomplete rectal prolapse as a factor in the pathogenesis of faecal incontinence. It seems likely that in patients with incomplete rectal prolapse and associated faecal incontinence, this condition is the result of occult anterior sphincter defects rather than the incomplete prolapse itself.

In our series, all male patients became continent. In male patients with complete rectal prolapse, this is most likely the result of removal of the dilating activity of the prolapse itself. In the male patients with rectal intussusception, the improvement may be the result of impaired rectal evacuation induced by the procedure itself. In female patients in Groups I and II, rectopexy was successful in, respectively, 20 per cent (2/10) and 62 per cent (13/21) of patients. The low success-rate in female patients might be explained by the presence of an occult sphincter defect. This assumption is underlined by the fact that the history of all female patients with persistent faecal incontinence after rectopexy revealed obstetric tear or episiotomy.

Because the clinical outcome of rectopexy is only good in incontinent patients with complete rectal prolapse, the authors advocate a conservative attitude toward surgery in patients with faecal incontinence and associated incomplete prolapse. These patients should be considered as candidates for endoanal ultrasound and subsequent sphincter repair.

References

1. Parks AG. Anorectal incontinence. *Proc R Soc Med* 1975; 68: 681-90.
2. Watts JD, Rothenberger DA, Goldberg SM. Treatment of rectal prolapse. In: Henry MM, Swash M, eds. *Coloproctology and the Pelvic Floor*. London: Butterworths Publishing, 1985: 308-39.
3. Allingham W. *Fistula, haemorrhoids, painful ulcer, stricture, prolapses and other diseases of the rectum, their diagnosis and treatment*, 2nd ed. London: J&A Churchill, 1873.
4. Ripstein CB. Symposium: Procidentia of the rectum: internal intussusception of the rectum (stage I rectal prolapse). *Dis Colon Rectum* 1975; 18: 458-60.
5. Hoffman MJ, Kodner IJ, Fry RD. Internal intussusception of the rectum: diagnosis and surgical management. *Dis Colon Rectum* 1984; 27: 435-41.
6. Kuijpers HC, Schreve RH, ten Cate Hoedemakers H. Diagnosis of functional disorders of defecation causing the solitary ulcer syndrome. *Dis Colon Rectum* 1986; 29: 126-9.
7. Broden B, Snellman B. Procidentia of the rectum studied with cineradiography: a contribution to the discussion of causative mechanism. *Dis Colon Rectum* 1968; 11: 330-47.
8. Ihre T, Seligson U. Intussusception of the rectum-internal procidentia: treatment and results in 90 patients. *Dis Colon Rectum* 1975; 18: 391-6.
9. Delemarre JB, Kruijt RH, Posthuma BJ, Gooszen HG. Effects of posterior rectopexy on false urgency, perineal pain and faecal continence in patients with rectal prolapse or rectal intussusception. In: *Aspects of anorectal physiology*. Karstens Drukkers bv, Leiden, The Netherlands (ISBN: 90-9005906-7), Thesis, Leiden 1993: 63-73.
10. Schultz I, Mellgren A, Dolk A, Johansson C, Holmström B.

Continence is improved after the Ripstein rectopexy: different mechanisms in rectal prolapse and rectal intussusception? *Dis Colon Rectum* 1996; 39: 300-6.

11. Parks AG. Anorectal incontinence. *Proc R Soc Med* 1975; 68: 681-90.
12. Mann CV. Rectal prolapse. In Morson BC: *Diseases of the Colon, Rectum and Anus*. Apleton-Century-Crofts 1969: 238-50.
13. Ripstein CB. Symposium. Procidentia: Definitive corrective surgery. *Dis Colon Rectum* 1972; 15: 334-46.
14. Launer DP, Fazio VW, Weakley FL, Turnbull RB Jr, Jagelman DG, Lavery IC. The Ripstein procedure: a 16 year experience. *Dis Colon Rectum* 1982; 25: 41-5.
15. Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. *Gut* 1977; 18: 656-65.
16. Butler EC. Complete rectal prolapse following removal of tumours of the cauda equine. Two cases. *Proc R Soc Med* 1954; 47: 521-2.
17. Neill ME, Parks AG, Swash M. Physiological studies of the anal sphincter musculature in faecal incontinence and rectal prolapse. *Br J Surg* 1981; 68: 531-6.
18. Holmström B, Brodén G, Dolk A, Frenckner B. Increased anal resting pressure following the Ripstein operation: a contribution to continence? *Dis Colon Rectum* 1986; 29: 485-7.
19. Snooks SJ, Henry MM, Swash M. Anorectal incontinence and rectal prolapse: differential assessment of the innervation to puborectalis and external anal sphincter muscles. *Gut* 1985; 26: 470-6.
20. Farouk R, Duthie GS, MacGregor AB, Bartolo DC. Rectoanal inhibition and incontinence in patients with rectal prolapse. *Br J Surg* 1994; 81: 743-6.
21. Farouk R, Duthie GS, Bartolo DC, MacGregor AB. Restoration of continence following rectopexy for rectal prolapse is associated with recovery of the internal anal sphincter electromyogram. *Br J Surg*

Rectopexy

- 1992; 79: 439-40.
22. Duthie GS, Bartolo DC. Abdominal rectopexy for rectal prolapse: a comparison of techniques. *Br J Surg* 1992; 79: 107-13.
 23. Duthie GS, Bartolo DC. A comparison between Marlex and resection rectopexy. *Neth J Surg* 1989; 41: 136-9.
 24. Yoshioka K, Hyland G, Keighley MR. Anorectal function after abdominal rectopexy: parameters of predictive value in identifying return of continence. *Br J Surg* 1989; 76: 64-8.
 25. Spencer RJ. Manometric studies in rectal prolapse. *Dis Colon Rectum* 1984; 27: 523-5.
 26. Sainio AP, Voutilainen PE, Husa AI. Recovery of anal sphincter function following transabdominal repair of rectal prolapse: cause of improved continence? *Dis Colon Rectum* 1991; 34: 816-21.
 27. Brodén G, Dolk A, Holmström B. Recovery of the internal anal sphincter following rectopexy: a possible explanation for continence improvement. *Int J Colorectal Dis* 1988; 3: 23-8.
 28. Ihre T. Internal procidentia of the rectum: treatment and results. *Scand J Gastroenterol* 1972; 7: 643-6.
 29. Goldberg SM, Gordon PH, Nivatvongs S. *Essentials of anorectal surgery*. 1st ed. Philadelphia: JB Lippincott, 1980.
 30. Goei R, van Engelshoven J, Schouten H, Baeten C, Stassen C. Anorectal function: defecographic measurement in asymptomatic subjects. *Radiology* 1989; 173: 137-41.
 31. Shorvon PJ, McHugh S, Diamant NE, Somers S, Stevenson GW. Defecography in normal volunteers: results and implications. *Gut* 1989; 30: 1737-49.
 32. Wiersma TG. Intussusception. In: *Dynamic rectal examination: clinico-radiological correlation*. Drukkerij Arnhem, Arnhem, The Netherlands (ISBN: 90-801844-2-X), Thesis, Utrecht 1994: 104-9.
 33. Bartram CI, Turnbull GK, Lennard-Jones JE. Evacuation proctography: an investigation of rectal expulsion in 20 subjects

- without defecatory disturbances. *Gastrointest Radiol* 1988; 13: 72-80.
34. Mahieu P, Pringot J, Bodart P. Defecography: II. Contribution to the diagnosis of defecation disorders. *Gastrointest Radiol* 1984; 9: 253-61.
 35. Freimanis MG, Wald A, Caruana B, Bauman DH. Evacuation proctography in normal volunteers. *Invest Radiol* 1991; 26: 581-5.
 36. Goei R, Baeten C. Rectal intussusception and rectal prolapse: detection and postoperative evaluation with defecography. *Radiology* 1990; 174: 124-6.
 37. Selvaggi F, Canonico S, Riegler G, et al. Surgical treatment of internal rectal prolapse. *Coloproctology* 1994; 16: 341-5.
 38. Christiansen J, Hesselheldt P, Sorensen M. Treatment of internal rectal intussusception in patients with chronic constipation. *Scand J Gastroenterol* 1995; 30: 470-2.
 39. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *New Engl J Med* 1993; 329: 1905-11.
 40. Burnett SJ, Speakman CT, Kamm MA, Bartram CI. Confirmation of endosonographic detection of external anal sphincter defects by simultaneous electromyographic mapping. *Br J Surg* 1991; 78: 448-50.

CHAPTER 4

DISAPPOINTING RESULTS OF POSTANAL REPAIR IN THE TREATMENT OF FAECAL INCONTINENCE

JW Briel and WR Schouten.

From the Department of General Surgery,
University Hospital Dijkzigt, Rotterdam, The Netherlands

Adapted from Ned Tijdschr Geneeskd 1995; 139: 23-6.

Abstract

To evaluate the long-term results after postanal repair in patients with faecal incontinence. Thirty-seven patients, who underwent postanal repair during the period 1984 to 1992, were contacted by telephone communication to determine the current functional status. The median duration of follow-up was 38 (range 4-94) months. One year after the operation 24 patients (65 per cent) were continent. In 22 patients this successful outcome was observed within the first half year. In seven patients (29 per cent) incontinence recurred. In 13 patients (35 per cent) the functional results were disappointing from the very beginning. The final outcome, which was good in only 46 per cent of patients, was not influenced by the underlying aetiology. Postanal repair is not beneficial for the majority of patients with faecal incontinence. Therefore, it should be considered if other treatment modalities might be preferable.

Introduction

Faecal incontinence is the disability to control timing and place of defecation. This troublesome condition is frequently encountered in the elderly. The exact incidence of faecal incontinence is unknown. Estimations based upon the UK population, predict the number of Dutch patients with faecal incontinence varying between 80.000 and 240.000¹. Because most incontinent patients are ashamed of their condition, social isolation is common. Professional help is asked for relatively late. Damage to the anal sphincters is the most common cause of faecal incontinence. Such a sphincter defect can be identified by careful history taking and digital examination. However, in some patients the sphincters are intact and no apparent cause for the continence disturbance can be found. In these patients faecal incontinence is called "idiopathic". In recent years the

aetiologic factors contributing to faecal incontinence have been pointed out by electromyography. This so-called idiopathic incontinence is thought to be caused by denervation of the pelvic floor and external anal sphincter. Tonic activity of the pelvic floor, and the puborectal muscle in particular, is decreased by the innervation damage. It is thought that in normal patients the anorectal angle is about 90° ². During coughing and sneezing, this angle is maintained by an increased intra-abdominal pressure. The anal canal is closed by the flap-valve mechanism. It is postulated that the anorectal angle becomes more obtuse by denervation of the puborectal muscle, causing the flap-valve mechanism to become less efficient.

In 1975, Parks³ described a technique to reinforce the pelvic floor and external anal sphincter in an attempt to solve the problem of idiopathic (*i.e.* neurogenic) incontinence. This procedure was also aimed at restoring the anorectal angle. Results of this so-called postanal repair are varying between 58 and 86 per cent^{4,5}. Unfortunately, in recent years it became clear that the long-term results are less favourable than suggested by those promising short-term results⁶⁻⁹. In this study the results of postanal repair in 37 patients with faecal incontinence are described.

Patients & Methods

During the period between 1984 and 1992, 37 patients underwent postanal repair. This group of patients consisted of 30 female and 7 male patients. Duration of symptoms ranged from 6 months to 40 years, with a median duration of 24 months. Classification of faecal incontinence was assessed using the scoring system as described by Parks¹⁰. Only patients with faecal incontinence Grade III and IV were operated on. Twenty-nine patients were operated on by one surgeon. In 24 patients, the cause of faecal incontinence could not be found. Thirteen patients underwent postanal repair for a posterior sphincter defect, caused by previous anorectal surgery

Postanal Repair

or trauma. Prior to physical examination, a detailed history was taken. Patients were asked to describe the severity of their faecal incontinence and the degree of social isolation. Contributing factors for the continence disturbance were assessed (sphincter disruption during childbirth, episiotomy, perianal trauma, previous anorectal surgery). Then, physical examination was performed. The perineum was inspected carefully for the presence of scars. Furthermore, the patient was asked to strain, to exclude a rectal prolapse. Following, digital examination was performed to identify defects of the anal sphincters.

Technique of Surgery

In early years, conventional preoperative preparation by mechanical cleansing and antibiotic sterilization of the large bowel during three days was used. Later, polyethylene glycol was used for bowel preparation. Cefuroxime (1500 mg) and metronidazole (500 mg) were administered intravenously after the induction of general endotracheal anaesthesia. Postoperatively, this administration was repeated twice.

All patients were operated on in the lithotomy position. A curved skin incision was made 1 to 2 cm behind the anus. The distal portions of both internal and external anal sphincter muscles were identified and the intersphincteric plane was developed laterally and posteriorly. Dissection in the midplane was carried out upwards until Waldeyer's fascia. This fascia was incised which offered the possibility to mobilize the rectum anteriorly. Next, a row of absorbable sutures (2x0) was laterally placed in the muscles and left untied until all sutures were correctly placed. By tying the sutures, starting cranially, the pelvic floor muscles, and especially the puborectalis and the external anal sphincter muscle were approximated behind the rectum. The subcutis was closed, whereas the skin was left open. An indwelling bladder catheter was placed postoperatively.

Functional outcome following postanal repair was assessed by personal telephone communication. Pre- and postoperative continence status was compared. Improvement from Grade IV to Grade II or I, or from Grade III to Grade I was classified as successful outcome.

Results

In the early postoperative period (<30 days) four patients suffered bladder infection. Two patients developed superficial wound infection. Median duration of follow-up was 38 (range 4-94) months. One year following postanal repair, 24 (65 per cent) patients were continent, which was observed in 22 patients within the first half year. Unfortunately, incontinence recurred in seven patients (29 per cent) after a median period of 16 months following postanal repair. In 13 patients outcome was poor from the beginning. Long-term outcome was only good in 46 per cent of patients. There was no difference between patients with idiopathic incontinence and patients with a posterior sphincter defect (11/24 *versus* 6/13). Eventually, in four patients a stoma was fashioned to treat severe faecal incontinence.

Discussion

Until Parks³ introduced his postanal repair in 1975, the outcome of surgical treatment for idiopathic faecal incontinence was poor. He aimed to increase the length of the anal canal, and to restore the anorectal angle, in order to correct the flap-valve mechanism. This hypothesis is based on proctographic findings, suggesting that the anorectal angle is obtuse in patients with idiopathic faecal incontinence^{3,11-15}. In contrast, other investigators demonstrated anorectal angles within the normal ranges in these patients¹⁶⁻

Postanal Repair

¹⁹. Therefore, the role of the flap-valve mechanism has been questioned. Defaecography has revealed that an increase of intra-abdominal pressure does not close the anal canal²⁰. Furthermore, the flap-valve mechanism will only work if pressures in the proximal compartment are higher than in the distal compartment. Because during elevated intra-abdominal pressures, rectal pressure does not exceed anal pressures, it is unlikely that the flap-valve mechanism plays an important role in the conservation of continence²¹. Although postanal repair is aimed at reducing the anorectal angle, this could not be demonstrated in most studies^{11,16-19}. Only one published report confirms correction of the anorectal angle following postanal repair¹⁴. However, this study was based on proctographic investigation, using a contrast filled balloon. It is doubted that the anorectal angle is measured correctly in this way.

Anorectal manometry has revealed that anal canal pressures in patients with idiopathic faecal incontinence are lower than in control patients, both at rest and during squeeze^{18,19,22-26}. Some investigators reported increased resting and squeezing pressures following postanal repair^{17,19,27,28}. Until now, only one study pointed out that outcome is related to elevated anal canal pressures²⁹. Others concluded that postanal repair could be beneficial even without increased pressures^{11,18}. Contrary, despite elevated anal pressures, patients can still suffer from incontinence¹⁸. In other studies no increase in pressures could be demonstrated at all^{16,18,23,24,30,31}. Therefore, the effect of postanal repair on anal canal pressures remains to be clarified.

Also, the length of the anal canal has been studied. It has been reported that the anal canal length is increased following postanal repair^{7,29}. Others were not able to confirm these findings^{18,30,32}. Consequently, it is still unclear why patients, following repair, may benefit or not.

Denervation can be demonstrated by electromyography of the pelvic floor and external anal sphincter. Also, defects of the anal sphincters can be revealed in this way. Denervation signs have been noted in patients with idiopathic faecal incontinence. Prolonged straining at defecation might

stretch the pudendal nerve³³. Moreover, damage to the pudendal nerve, sustained during childbirth results in prolonged latencies of the pudendal nerve²¹. In most patients with idiopathic faecal incontinence pudendal nerve terminal motor latencies are delayed^{23,34,35}. Ongoing of denervation following postanal repair has been reported²³. Moreover, neurogenic injury might be worsened by the procedure itself³⁶. Poor outcome has been attributed to injury to and prolonged stretching of the pudendal nerve²³. It is clear that this neuropathy cannot be treated by postanal repair. Probably denervation of the pelvic floor is not the sole contributory factor to the poor outcome of postanal repair. Endoanal ultrasound has revealed occult sphincter defects in 63 per cent of patients with idiopathic faecal incontinence⁷. Such occult sphincter defects, that cannot be detected by digital examination, are usually caused by obstetric trauma. Sphincter defects can be identified in 37 per cent of women with apparently uneventful deliveries³⁷. These findings illustrate the impact of delivery in the pathogenesis of faecal incontinence and the subsequent overestimation of idiopathic faecal incontinence.

In the present study, postanal repair was not only performed for idiopathic faecal incontinence. Posterior sphincter defects were also treated with this repair. In 13 of the studied patients former anorectal surgery was identified as cause for the incontinence. In 24 patients no cause could be found using the investigations available at that time. Therefore, incontinence in these patients was classified as idiopathic. Endoanal ultrasound was not performed in any of the patients. Therefore, it seems likely that these patients might have had anterior sphincter defects as result of obstetric trauma.

Despite encouraging short-term results of postanal repair (65 per cent), long-term results are disappointing (46 per cent). This outcome is comparable with that reported in literature⁶⁻⁹. Obviously, endoanal ultrasound is mandatory in the work-up of patients with faecal incontinence to reveal occult anterior sphincter defects. It is hypothesized that in the

Postanal Repair

absence of sphincter defects other treatment modalities are more beneficial for this group of patients. Biofeedback, aimed at reactivating the remaining function of pelvic floor and anal sphincter muscles has been propagated. In literature success ranges from 50 to 92 per cent^{38,39}. These results are so promising, that is has been advocated to treat all patients with faecal incontinence with biofeedback in the first place.

References

1. Mandelstam DA. Faecal incontinence. In: Henry MM, Swash M (eds). *Coloproctology and the pelvic floor: pathophysiology and management* (1st edition). London: Butterworths, 1985; 217-21.
2. Mahieu P, Pringot J, Bodart P. Defecography: Description of a new procedure and results in normal patients. *Gastrointest-Radiol* 1984; 9: 247-51.
3. Parks AG. Anorectal incontinence. *Proc R Soc Med* 1975; 68: 681-90.
4. Failes D, Killingback M, Stuart M. The surgical treatment of faecal incontinence. *Aust N Z J Surg* 1979; 49: 345-9.
5. Henry MM, Simson JN. Results of postanal repair: a retrospective study. *Br J Surg* 1985; 72 Suppl: 17-9.
6. Rainey JB, Donaldson DR, Thompson JPS. Postanal repair: which patients derive most benefit? *J R Coll Surg Edinb* 1990; 35: 101-5.
7. Setti-Carraro P, Kamm MA, Nicholls RJ. Long-term results of post-anal repair for neurogenic faecal incontinence. *Br J Surg* 1994; 81: 140-4.
8. Engel AF. Surgical and physiological aspects of faecal incontinence [proefschrift]. Amsterdam, 1994.
9. Jameson JS, Speakman CTM, Darzi A, Chia YW, Henry MM. Audit of postanal repair in the treatment of fecal incontinence. *Dis Colon Rectum* 1994; 37: 369-72.
10. Parks AG. Anorectal incontinence. *J R Soc Med* 1975; 68: 21-30.
11. Womack NR, Morrison JF, Williams NS. Prospective study of the effects of postanal repair in neurogenic faecal incontinence. *Br J Surg* 1988; 75: 48-52.
12. Kerremans R. Morphological and physiological aspects of anal continence and defecation. Brussels: Editions Arscia, 1969.
13. Hardcastle JD, Parks AG. A study of anal incontinence and some

Postanal Repair

- principles of surgical treatment. *Poc R soc Med* 1970; 63 Suppl: 116-8.
14. Preston DM, Lennard-Jones JE, Thomas BM. The balloon proctogram. *Br J Surg* 1984; 71: 29-32.
 15. Bartolo DCC, Jarratt JA, Read MG, Donnelly TC, Read NW. The role of partial denervation of the puborectalis in idiopathic faecal incontinence. *Br J Surg* 1983; 70: 664-7.
 16. Keighley MRB, Fielding JWL. Management of faecal incontinence and results of surgical treatment. *Br J Surg* 1983; 70: 463-8.
 17. Miller R, Bartolo DCC, Locke-Edmunds JC, Mortensen NJMc. Prospective study of conservative and operative treatment for faecal incontinence. *Br J Surg* 1988; 75: 101-5.
 18. Yoshioka K, Hyland G, Keighley MRB. Physiological changes after postanal repair and parameters predicting outcome. *Br J Surg* 1988; 75: 1220-4.
 19. Orrom WJ, Miller R, Cornes H, Duthie G, Mortensen NJ, Bartolo DCC. Comparison of anterior sphincteroplasty and postanal repair in the treatment of idiopathic fecal incontinence. *Dis Colon Rectum* 1991; 34: 305-10.
 20. Bartolo DCC, Roe AM, Locke-Edmunds JC, Virjee J, McMortensen NJ. Flapvalve theory of anorectal continence. *Br J Surg* 1986; 73: 1012-4.
 21. Bannister JJ, Gibbons C, Read NW. Preservation of fecal incontinence during rises in intra abdominal pressure: is there a role for the flap valve? *Gut* 1987; 28: 1242-5.
 22. Hiltunen KM. Anal manometric findings in patients with anal incontinence. *Dis Colon rectum* 1985; 28: 925-8.
 23. Snooks SJ, Swash M, Henry M. Electrophysiologic and manometric assessment of failed postanal repair for anorectal incontinence. *Dis Colon Rectum* 1984; 27: 733-6.
 24. Hunter RA, Saccone GT, Sarre R, Cain T, Toouli J. Faecal inconti-

- nence: manometric and radiological changes following postanal repair. *Aust N Z J Surg* 1989; 59: 697-705.
25. Farouk R, Duthie GS, Pryde A, McGregor AB, Bartolo DCC. Internal anal sphincter dysfunction in neurogenic faecal incontinence. *Br J Surg* 1993; 80: 259-61.
 26. Eckhardt VF, Jung B, Fischer B, Lierse W. Anal endosonography in healthy subjects and patients with idiopathic fecal incontinence. *Dis Colon Rectum* 1994; 37: 235-42.
 27. Scheuer M, Kuijpers HC, Jacobs PP. Postanal repair restores anatomy rather than function. *Dis Colon Rectum* 1989; 32: 960-3.
 28. Braun J, Tons C, Schippers E, Fass J, Schumpelick V. Ergebnisse der posterioren Raffung nach Parks bei der idiopathischen analen Inkontinenz. *Chirurg* 1991; 62: 206-10.
 29. Browning GGP, Parks AG. Postanal repair for neuropathic faecal incontinence: correlation of clinical result and anal canal pressures. *Br J Surg* 1983; 70: 101-4.
 30. Van Vroonhoven ThJMV, Schouten WR. Postanal repair in the treatment of faecal incontinence. *Neth J Surg* 1984; 36: 160-2.
 31. Yoshioka K, Keighley MRB. Critical assessment of the quality of continence after postanal repair for faecal incontinence. *Br J Surg* 1989; 76: 1054-7 [erratum, *Br J Surg* 1990; 77: 356].
 32. Deen KI, Oya M, Ortiz J, Keighley MR. Randomized trial comparing three forms of pelvic floor repair for neuropathic faecal incontinence. *Br J Surg* 1993; 80: 794-8.
 33. Ortiz J, Oya M, Bacelar T, Chattapady G, Panagamura B, Keighley MRB. Cause of deteriorating continence after postanal repair. *Gut* 1991; 32: A1236.
 34. Kiff ES, Swash M. Slowed conduction in the pudendal nerves in idiopathic (neurogenic) faecal incontinence. *Br J Surg* 1984; 71: 614-6.
 35. Kiff ES, Swash M. Normal proximal and delayed distal conduction in

Postanal Repair

- the pudendal nerves of patients with idiopathic (neurogenic) faecal incontinence. *J Neurol Neurosurg Psychiatry* 1984; 47: 820-3.
36. Laurberg S, Swash M, Henry MM. Effect of postanal repair on progress of neurogenic damage to the pelvic floor. *Br J Surg* 1990; 77: 519-22.
 37. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *N Engl J Med* 1993; 329: 1905-11.
 38. Buser WD, Miner PB. Delayed rectal sensation with fecal incontinence. *Gastroenterology* 1986; 91: 1186-91.
 39. Loening-Baucke V. Efficacy of biofeedback training in improving faecal incontinence and anorectal physiologic function. *Gut* 1990; 31: 1395-402.

CHAPTER 5

CLINICAL OUTCOME OF ANTERIOR OVERLAPPING EXTERNAL ANAL SPHINCTER REPAIR WITH INTERNAL ANAL SPHINCTER IMBRICATION

JW Briel¹, LM de Boer¹, WCJ Hop², and WR Schouten¹.

From the Departments of ¹Surgery, and ²Epidemiology and Biostatistics,
University Hospital Dijkzigt, Rotterdam, The Netherlands

Dis Colon Rectum 1998; 41: 209-14.

Abstract

Faecal incontinence caused by overt anterior sphincter defects sustained during childbirth is usually treated by a delayed overlapping repair of the external anal sphincter. However, an obstetric trauma is frequently associated with disruption of the perineal body and loss of the distal rectovaginal septum. Data regarding a combined repair, consisting of restoration of the rectovaginal septum and perineal body, overlapping external anal sphincter repair, and imbrication of the internal anal sphincter, are scanty. This prospective study was aimed at the following: 1) evaluating the clinical outcome of such an anterior anal repair in patients with faecal incontinence caused by obstetric trauma; 2) comparing the functional results with those obtained in a historical group of patients who underwent a conventional direct sphincter repair. During the period between 1973 and 1989, 24 female patients (median age 44 (range 28-67) years) with faecal incontinence underwent direct sphincter repair (Group I). During the period between 1989 and 1994, a consecutive series of 31 female patients (median age 46 (range 23-78) years) with faecal incontinence underwent anterior anal repair (Group II). At two years of follow-up, continence had been restored in 15 patients (63 per cent) in Group I, whereas restoration of continence was successful in 21 patients (68 per cent) in Group II. The more complex anterior anal repair fails to confer clinical benefit compared with the rather simple direct sphincter repair.

Introduction

Faecal incontinence requiring surgical correction is generally the result of an acquired sphincter muscle injury sustained during childbirth or anorectal surgery^{1,2}. Such an injury might be overt or occult. The incidence of overt obstetric perineal injury following normal vaginal delivery is about 5 per

cent³. Overt obstetric injuries are frequently associated with disruption of the perineal body, division of both anal sphincters, and loss of the distal rectovaginal septum³. Usually, such an injury is corrected by the gynaecologist shortly after delivery. Despite such a primary repair, persistent sphincter defects have been reported using endoanal ultrasound in 85 per cent of patients⁴. The incidence of faecal incontinence among those women is approximately 4 per cent³. The incidence of occult sphincter damage after childbirth was unknown until Sultan *et al.*⁵ studied a consecutive group of 202 pregnant women. On endoanal ultrasound, 35 per cent of primiparae who delivered vaginally developed a sphincter defect involving one or both muscles, and this damage persisted at six months. Furthermore, Burnett *et al.*⁶ showed that in a consecutive series of women presenting with faecal incontinence, in whom the only apparent risk factor was obstetric damage, 90 per cent were found to have an anterior defect in one or both sphincters. Because sphincter muscle injury, sustained during childbirth, is frequently associated with disruption of the perineal body and loss of the distal rectovaginal septum, reinforcement of the rectovaginal septum and overlapping pubo-sphincteroplasty has been advocated for the treatment of obstetric faecal incontinence. In 1985, Corman⁷ reported excellent results after anterior reefing with anoplasty for incontinence after obstetric injury. Later, these promising results were confirmed by Stricker *et al.*⁸ and Abcarian *et al.*⁹

The aims of this study were to prospectively assess the functional results of anterior anal repair, consisting of combined restoration of the rectovaginal septum and perineal body, overlapping external anal sphincter repair, and imbrication of the internal anal sphincter, by comparing these results with those obtained in a historical group of patients who underwent a conventional direct sphincter repair.

Patients & Methods

During the period between 1973 and 1989, 24 female patients (median age 44 (range 28-67) years) with faecal incontinence as a result of obstetric trauma underwent direct sphincter repair (Group I) and were retrospectively evaluated. During the period between 1989 and 1994, a consecutive series of 31 female patients (median age 46 (range 23-78) years) with faecal incontinence underwent anterior anal repair (Group II) and was prospectively evaluated. Duration of symptoms ranged from 3 months to 23 years in Group I (median 84 months) and from 7 months to 21 years in Group II (median 45 months).

In both groups, the origin of sphincter defects was obstetric. None of the patients had associated rectal prolapse. Three patients in Group I and four patients in Group II had undergone at least one previous attempt at surgical correction. In Group II, associated lesions included seven patients with rectoceles and two patients with rectovaginal fistulas. These anomalies were treated simultaneously with anterior anal repair.

Hospital records from the time of the operation and outpatient clinic charts were analyzed, and follow-up information was obtained both from review of charts and personal telephone communication by one author (LMdB) who had not participated in any of the operations. The degree of incontinence was assessed and graded as described by Parks¹⁰: Grade I, fully continent; Grade II, soiling and incontinence for gas; Grade III, incontinence for liquids; Grade IV, incontinence for solid stool.

Preoperatively, degree of incontinence was equally distributed. In Group I, 14 patients presented with faecal incontinence Grade IV and 10 patients with Grade III. In Group II, 17 patients suffered from Grade IV and 14 patients from Grade III faecal incontinence.

Restoration of continence from Grade IV to Grade II or I or from Grade III to Grade I, was defined as a successful outcome. During telephone com-

munication, postoperative grading was assessed without knowledge of the preoperative continence status.

Preoperative Care

Preoperatively, patients in Group I were given a clear liquid diet and received an enema for cleansing the bowel. Patients in Group II underwent complete mechanical bowel preparation (polyethylene glycol). Metronidazole (500 mg) together with cefamandole (1 g) in Group I or cefuroxime (1500 mg) in Group II was administered intravenously after the induction of general endotracheal anaesthesia. All patients were placed in the lithotomy position with an indwelling bladder catheter in place.

Direct Sphincter Repair

A curvilinear incision paralleling the outer edge of the superficial part of the external anal sphincter was used to separate the anoderm from the underlying external anal sphincter. The arc of the incision was limited to 180 to 240° anterolaterally to avoid pudendal nerve damage. Then both ends of the external anal sphincter were identified. Next, an overlapping repair of the external anal sphincter with interrupted absorbable sutures (2x0) was performed (*Fig. 1*).

Anterior Anal Repair

A similar incision was made. Then, cephalad mobilization was extended. The posterior vaginal wall was separated from the anterior rectal wall. Next, the intersphincteric plane was entered bilaterally to separate the external

Anterior Anal Repair

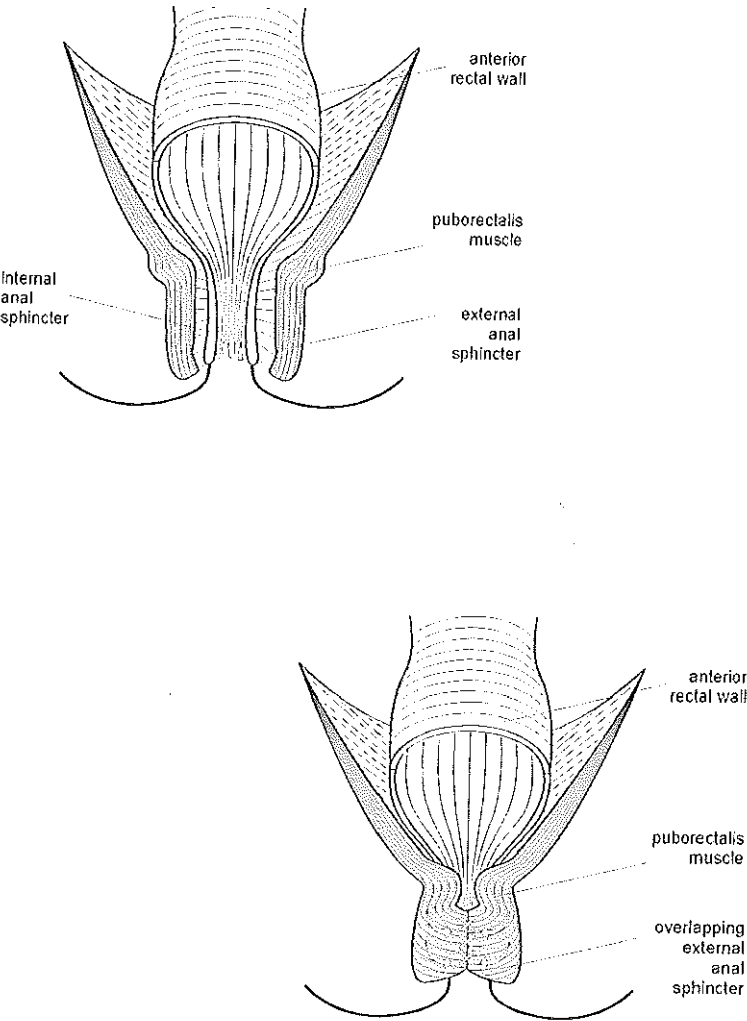


Figure 1. Diagrammatic sketch of direct sphincter repair.

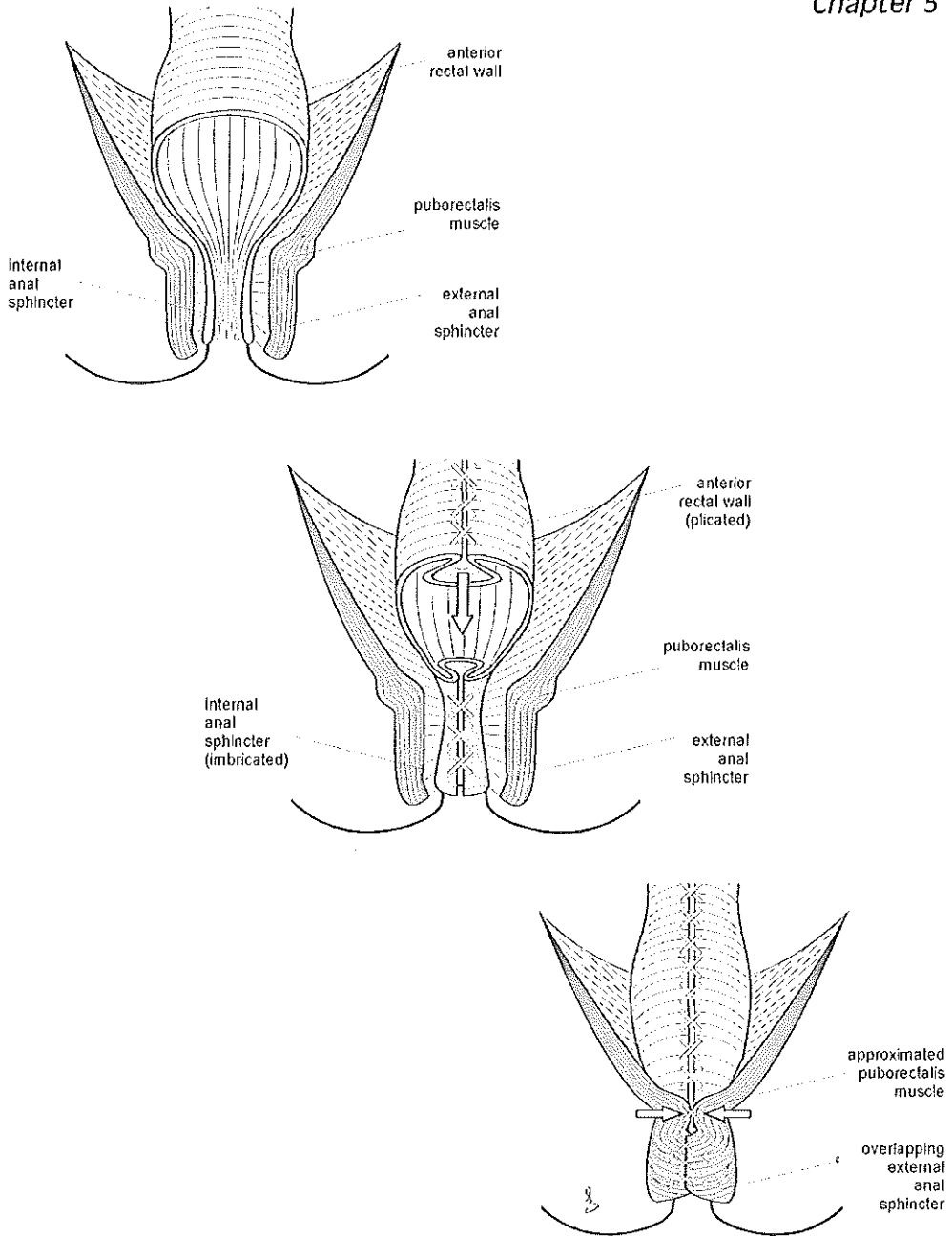


Figure 2. Diagrammatic sketch of anterior anal repair.

Anterior Anal Repair

anal sphincter and the internal anal sphincter. The entire scar tissue in the anterior midline was divided completely. Then, the anterior rectal wall was plicated (3x0), the internal anal sphincter was imbricated (2x0), both parts of the puborectal sling were approximated (2x0), and an overlapping repair of the external anal sphincter was performed (2x0; *Fig. 2*).

After sphincter repair in both groups, the subcutaneous layer was closed and the skin was left open. Temporary covering colostomies were not fashioned in either group.

Postoperative Care

Postoperatively, three doses of gentamicin (80 mg) and metronidazole (500 mg) were administered on a daily basis for a period of two days in Group I, whereas three doses of cefuroxime (1500 mg) and metronidazole (500 mg) were administered on a daily basis for a period of five days in Group II. Patients were instructed to retain strict bed rest for three days and for five days, respectively. All patients were kept on a clear liquid diet for five days. Three times daily, sitz baths began on the first postoperative day, and the bladder catheter was usually removed on the fourth day. Pain medication was required for approximately seven to ten days. After starting with a regular diet, stool softeners were prescribed. Patients usually left the hospital in seven to ten days.

Results

Within the first 30 days postoperatively, anal sphincter reconstruction was complicated by a wound abscess in three patients in Group I and in three patients in Group II. Two patients suffered a urinary tract infection in Group

I, whereas none of the patients in Group II presented with a urinary tract infection. Long-term complications comprised one perineovaginal fistula and one rectovaginal fistula in Group II. One patient complained about disabling dyspareunia after anterior anal repair. In this patient the anterior sphincteroplasty was broken down, and within the same procedure a postanal repair was performed.

At 24 months of follow-up, continence had been restored in 15 patients (63 per cent) in Group I, whereas in Group II the procedure was successful in 21 patients (68 per cent). The functional outcome in both groups deteriorated with the length of follow-up (*Fig. 3*).

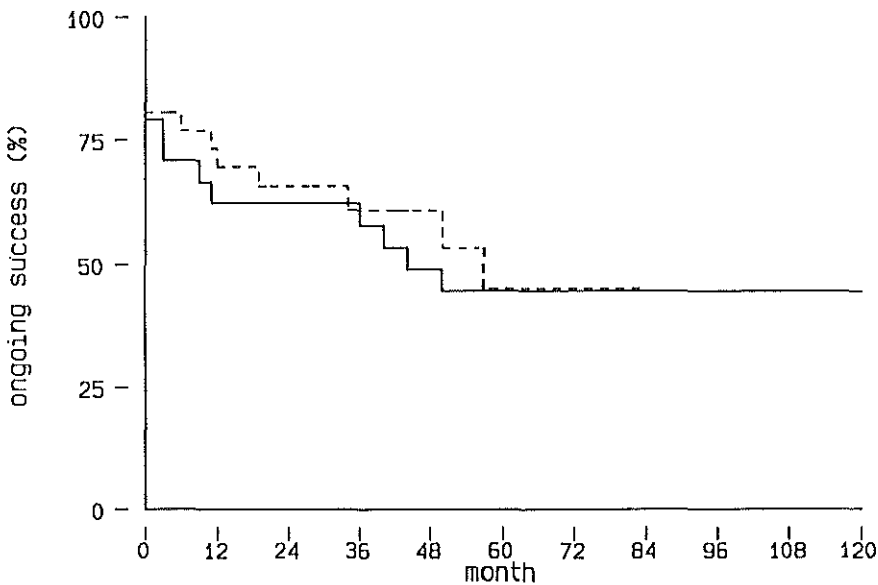


Figure 3. Ongoing success-rate after direct sphincter repair (solid curve: --) and anterior anal repair (dashed curve: - -). Log-rank test, $P=0.86$

In Group I, the outcome in patients without previous sphincter repair was better than in patients who underwent a previous attempt to restore their continence (62 *versus* 33 per cent; not significant). Similarly, in Group II,

Anterior Anal Repair

the success-rate was greatly reduced when patients had already undergone sphincter repair compared with those who underwent a first attempt (50 *versus* 81 per cent; not significant).

The associated lesions in Group II did not affect the final outcome. The two coexisting rectovaginal fistulas were healed after anterior anal repair, whereas continence had also been restored in these two patients. Four of the seven patients (57 per cent) with an accompanying rectocele regained their continence after anterior anal repair.

Discussion

Laceration of the perineum during delivery might sever the anal sphincters. Usually such an injury is repaired by the gynaecologist shortly after childbirth. Despite such a repair, quite a number of women complain about faecal incontinence. The incidence of this complication varies between 4 and 7 per cent^{3,11}. By using endoanal ultrasound, it has been shown that after primary repair, persistent defects are present in 85 per cent of patients⁴. It is likely that patients with such an occult defect might become incontinent with increasing age. In these patients, a secondary repair is necessary. In 1882, Warren¹² was the first to describe a perineal repair for anorectal laceration. In 1940, Blaisdell¹³ reported the collective results of the American Proctological Society using a direct end-to-end sphincter repair. His survey revealed that this technique resulted in a failure rate as high as 40 per cent. End-to-end apposition has been criticized because of disappointing results from cutting out the sutures and retracting the muscle ends¹⁴. Overlapping sphincteroplasty, as described by Parks and McPartlin in 1971¹⁵ and later modified by Slade *et al.*¹⁶, showed better results. By overlapping the sphincter muscle, the surface area in contact is greatly increased. By using this technique, tearing out of the sutures was thought to be less likely. Functional results have been reported, varying from 47 to

96 per cent of patients^{17,18}. However, sphincter muscle injury sustained during childbirth is not confined to the external anal sphincter only. Frequently, both anal sphincters are divided. Furthermore, both the perineal body and the distal rectovaginal septum may be disrupted³. In 1991, Wexner and colleagues¹⁹ performed overlapping sphincteroplasty with internal anal sphincter imbrication in 16 female patients with an isolated anterior external anal sphincter defect. After ten months of follow-up, 76 per cent of patients had an excellent or good result¹⁹. It is questionable whether addition of internal anal sphincter imbrication is worthwhile or not, because similar results have been reported after conventional overlapping sphincter repair^{17,18}. Corman⁷ and Stricker *et al.*⁸ advocated anterior sphincter repair combined with anoplasty. Twenty-eight patients in the series by Corman⁷ underwent anterior reefing with anoplasty for incontinence after obstetric injury, and all had improvement at three months to three years. Similarly, Stricker *et al.*⁸ pointed out that the addition of anoplasty showed much better results than anterior reefing without anoplasty, both for the short-term and the long-term. Abcarian *et al.*⁹ reported excellent functional results in patients with traumatic cloaca after childbirth. He pointed out the advantages of extended repair: 1) it constructs a new perineal body and gives support to the sphincteroplasty; 2) it creates a distance between the anus and vagina; 3) it places normal, healthy layer of muscle between the posterior vaginal and anterior anorectal walls⁹. Based on these reports, we decided to conduct a prospective study in a consecutive series of patients. The repair consisted of external and internal anal sphincter plication, reinforcement of the rectovaginal septum, and approximation of the puborectal sling. The results in this series were compared with a historical group of patients who underwent a conventional direct sphincter repair. As shown by the present study, clinical outcome in both groups was similar (*Fig. 3*).

It has been suggested that separation of the internal and external anal sphincter affects outcome¹⁸. The effect of additional anoplasty might,

Anterior Anal Repair

therefore, be undone by imbrication of the internal anal sphincter. The study conducted by Wexner and coworkers¹⁹ does not provide evidence that additional imbrication of the internal anal sphincter is beneficial, because the outcome in this series is similar with that obtained after conventional sphincter repair^{17,18}. The lack of benefit of additional anoplasty is also supported by a recent study conducted by Österberg *et al.*²⁰. They reported excellent and good results in 40 of 54 (74 per cent) patients who had an obstetric injury after anterior levatorplasty alone. This result was sustained in the long-term²⁰.

The process of regaining continence after sphincter repair is far more complex than supposed. Electromyographic studies demonstrated severe denervation and pudendal nerve damage in patients who remained incontinent^{17,19,21}. Wexner *et al.*¹⁹ suggested that pudendal nerve terminal motor latency may be a much more significant predictor of outcome than either age or preoperative manometric pressures. Laurberg *et al.*¹⁷ and Fleshman and colleagues²² demonstrated that complete control of continence could be achieved if anal sphincter length, resting pressure, squeeze pressure, and anal sensation were restored to normal. These findings could not be confirmed by Luukkonen and Jarvinen²³. Cterkteko *et al.*²⁴ studied a number of factors in relation to the results of sphincteroplasty. He found that the older the patient, the more likely were problems with continence after the operation, the necessity of wearing pads, and dissatisfaction with the result of the operation. The longer the patient had been incontinent before operation, the more likely the results would be poor. With respect to cause, patients with obstetric sphincter injuries were less likely to remain incontinent than patients with iatrogenic defects. Patients who had a previous attempt at repairs tended to have worse results²⁴. This was also demonstrated in the present study, both in patients who underwent direct sphincter repair and those who had anterior anal repair.

The rather complex and extended anterior anal repair carries more risk than a conventional sphincter repair. In our opinion, it is only worthwhile to

perform an anterior anal repair provided the results of this procedure are significantly better than the conventional direct sphincter repair. Because anterior anal repair seems to fail to confer clinical benefit, a prospective, randomized study using both techniques would be needed to further support this conclusion.

References

1. Corman ML. Colon and rectal surgery. Philadelphia: JB Lippincott, 1989.
2. Goldberg SM, Gordon PH, Nivatvongs S. Essentials of anorectal surgery. Philadelphia: JB Lippincott, 1980: 282-90.
3. Venkatesh KS, Ramanujam PS, Larson DM, Haywood MA. Anorectal complications of vaginal delivery. Dis Colon Rectum 1989; 32: 1039-41.
4. Sultan AH, Kamm MA, Hudson CN, Bartram CI. Third degree obstetric anal sphincter tears: risk factor and outcome of primary repair. BMJ 1994; 308: 887-91.
5. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. New Engl J Med 1993; 329: 1905-11.
6. Burnett SJD, Speakman CTM, Kamm MA, Bartram CI. Confirmation of endosonographic detection of external anal sphincter defects by simultaneous electromyographic mapping. Br J Surg 1991; 78: 448-50.
7. Corman ML. Anal incontinence following obstetrical injury. Dis Colon Rectum 1985; 28: 86-9.
8. Stricker JW, Schoetz DJ Jr, Collier JA, Veidenheimer MC. Surgical correction of anal incontinence. Dis Colon Rectum 1988; 31: 533-40.
9. Abcarian H, Orsay CP, Pearl RK, Nelson RL, Briley SC. Traumatic cloaca. Dis Colon Rectum 1989; 32: 783-7.
10. Parks AG. Anorectal incontinence. J R Soc Med 1975; 68: 21-30.
11. Bek KM, Laurberg S. Risks of anal incontinence from subsequent vaginal delivery after a complete obstetric anal sphincter tear. Br J Obstet Gynaecol 1992; 99: 724-6.

12. Warren JC. A new method of operation for the relief of rupture of the perineum through the sphincter and rectum. *Trans Am Gynecol Soc* 1882; 7: 324.
13. Blaisdell PC. Repair of the incontinent sphincter ani. *Surg Gynecol Obstet* 1940; 70: 692-7.
14. Browning GG, Motson RW. Anal sphincter injury; management and results of Parks sphincter repair. *Ann Surg* 1984; 199: 351-7.
15. Parks AG, McPartlin JF. Late repair of injuries of the anal sphincter. *J R Soc Med* 1971; 64: 1187-9.
16. Slade MS, Goldberg SM, Schottler JL, Balcos EG, Christenson CE. Sphincteroplasty for acquired anal incontinence. *Dis Colon Rectum* 1977; 20: 33-5.
17. Laurberg S, Swash M, Henry MM. Delayed external sphincter repair for obstetric tear. *Br J Surg* 1988; 75: 786-8.
18. Fang DT, Nivatvongs S, Vermeulen FD, Herman FN, Goldberg SM, Rothenberger DA. Overlapping sphincteroplasty for acquired anal incontinence. *Dis Colon Rectum* 1984; 27: 720-2.
19. Wexner SD, Marchetti F, Jagelman DG. The role of sphincteroplasty for fecal incontinence reevaluated: a prospective physiologic and functional review. *Dis Colon Rectum* 1991; 34: 22-30.
20. Österberg A, Graf W, Holmberg A, Pålman L, Ljung A, Hakelius L. Long-term results of anterior levatorplasty for fecal incontinence: a retrospective study. *Dis Colon Rectum* 1996; 39: 671-5.
21. Jacobs PP, Scheuer M, Kuijpers JH, Vingerhoets MH. Obstetric fecal incontinence: role of pelvic floor denervation and results of delayed sphincter repair. *Dis Colon Rectum* 1990; 33: 494-7.
22. Fleshman JW, Dreznik Z, Fry RD, Kodner IJ. Anal sphincter repair for obstetric injury: manometric evaluation of functional results. *Dis Colon Rectum* 1991; 34: 1061-7.
23. Luukkonen P, Jarvinen HJ. Anal sphincter reconstruction. Surgical results and functional outcome. *Acta Chir Scand* 1990; 156: 723-7.

Anterior Anal Repair

24. Ctercteko GC, Fazio VW, Jagelman DG, Lavery IC, Weakley FL, Melia M. Anal sphincter repair: a report of 60 cases and review of the literature. *Aust N Z J Surg* 1988; 58: 703-10.

CHAPTER 6

RELATIONSHIP BETWEEN SPHINCTER MORPHOLOGY ON ENDOANAL MRI AND HISTOPATHOLOGICAL ASPECTS OF THE EXTERNAL ANAL SPHINCTER

JW Briel¹, DDE Zimmerman¹, J Stoker², E Rociu², JS Laméris², WJ Mooi³,
and WR Schouten¹.

From the Departments of ¹General Surgery, ²Radiology, and ³Pathology,
University Hospital Dijkzigt, Rotterdam, The Netherlands

Int J Colorectal Dis 2000, 15: 87-90.

Abstract

Atrophy of the external anal sphincter can be shown only on endoanal magnetic resonance imaging (MRI). Until now, no study has compared the morphological endoanal MRI findings with histopathological aspects of the external anal sphincter. The aim of this study was to validate the MRI interpretation of the external anal sphincter using histology as a 'gold standard'. In this prospective study 25 consecutive unselected women (median age 48 (range 27-72) years) with faecal incontinence due to obstetric trauma were assessed preoperatively with endoanal MRI. All patients underwent anterior sphincteroplasty within 6 months of the preoperative assessment. During sphincter repair, a biopsy specimen was taken both from the left and right lateral parts of the external anal sphincter. Interpretation of MRI was performed by one of the radiologists (JS), and biopsy specimens were evaluated by the pathologist (WJM). Both were blinded to the interpretation of the other. MRI revealed external anal sphincter atrophy in 9 of the 25 patients (36 per cent). Histopathological investigation confirmed these findings in all but one. In one additional patient atrophy was detected on histological investigation, while the morphology of the external anal sphincter was classified as normal on MRI. In detecting sphincter atrophy endoanal MRI showed: 89% sensitivity, 94% specificity, 89% positive predictive value, and 94% negative predictive value. MRI correctly identified sphincter morphology in 23 of 25 cases (92 per cent). This study demonstrates that endoanal MRI accurately identifies normal and abnormal external anal sphincter morphology. Endoanal MRI is therefore a valuable preoperative diagnostic tool.

Introduction

Endoanal magnetic resonance imaging (MRI) has recently been introduced

in the diagnostic workup of patients with faecal incontinence¹⁻⁵. The high intrinsic contrast resolution of MRI and the high spatial resolution of an endoanal coil facilitate detection of defects of the internal and external anal sphincter. It has been shown that endoanal MRI also enables the detection of external anal sphincter atrophy^{1,4,5}. Atrophy of the external anal sphincter cannot be demonstrated, using conventional endoanal ultrasound. The poor inherent contrast of endoanal ultrasound causes cumbersome identification of the external anal sphincter, and therefore measurements of the muscle diameter of the anal sphincters by endoanal ultrasound are found to be unreliable⁶. The main disadvantages of endoanal MRI are the time and the cost of an examination, which are both greater than for endoanal ultrasound. Therefore preoperative endoanal MRI is justified only if it accurately identifies sphincter morphology. This prospective study compared the MRI interpretation of the external anal sphincter with the histological findings.

Patients & Methods

Written informed consent was obtained from all patients. In this prospective study 25 consecutive unselected women (median age 48 (range 27-72) years) with faecal incontinence due to obstetric trauma were assessed preoperatively by means of history, digital examination, anal manometry, endoanal ultrasound, and endoanal MRI. All patients underwent anterior sphincteroplasty within 6 months after preoperative assessment. Patients underwent anterior anal repair as described earlier⁷. This procedure was performed by one surgeon (WRS). During sphincter repair, a biopsy specimen was taken both from the left and right lateral parts of the external anal sphincter. MRI results were interpreted by one of the radiologists (JS), and biopsy specimens were evaluated by the pathologist (WJM). Both were blinded to the interpretation of the other.

Endoanal MRI

MRI was performed at 0.5 T (Gyrosan T5-II, Philips Medical Systems, Best, The Netherlands). An endoanal coil with a diameter of 19 mm (Philips) was used³⁻⁵. Axial T2-weighted three-dimensional gradient echo and coronal and sagittal T2-weighted turbo spin-echo sequences were performed. An anterior sphincter defect was present in all patients. The remaining sphincter muscle was assessed. External anal sphincter morphology was classified as normal or atrophied. Normal endoanal MR findings were used as a reference to determine sphincter atrophy with endoanal MRI as no criteria exist³. Atrophy of the external anal sphincter was defined as extreme thinning of its sphincter fibers or generalized fatty infiltration.

Histopathological Examination

During surgery full-thickness biopsy specimens, transversing the entire band of tissue, were taken from the left and right lateral parts of the external anal sphincter. Biopsy specimens were fixed in 4 per cent neutral buffered formaldehyde, and processed using standard procedures. Paraffin sections were stained with haematoxylin and eosin. Histologically, the external anal sphincter was considered to be atrophied when the striated muscle tissue exhibited diminished diameter of some muscle fibers, in association with replacement by fatty tissue.

Results

Both ultrasound and MRI demonstrated anterior sphincter defects in all patients. Histologically this was found to correspond to replacement with

fibrous tissue. MRI showed the morphology of the remaining external anal sphincter to be normal in 16 patients (*Fig. 1*).

In 15 of these cases biopsy sampling showed normal external anal sphincter morphology, apart from scattered fibrosis (*Fig. 2*), and in the remaining case histopathological examination revealed external anal sphincter atrophy. Endoanal MRI diagnosed external anal sphincter atrophy in 9 patients (*Fig. 3*).

Atrophy of the external anal sphincter was demonstrated only on MRI. Histopathologically these findings were confirmed in 8 patients (*Fig. 4*). The biopsy samples of the other patient were found to be normal.

The correlation between radiological and histological findings in the detection of sphincter atrophy was high, with endoanal MRI showing 89% sensitivity, 94% specificity, 89% positive predictive value, and 94% negative predictive value. In two patients MRI showed an asymmetrical aspect of the external anal sphincter. One lateral side was found to be normal, while the other lateral side showed marked atrophy of the external anal sphincter; in both cases these asymmetrical findings were confirmed histologically.

The prevalence of sphincter atrophy was 36 per cent, detected by either endoanal MRI or histopathology. MRI correctly identified sphincter morphology in 23 of 25 cases (92 per cent). There was no relationship between the patient's age and the presence of external anal sphincter atrophy. Furthermore, the prevalence of sphincter atrophy did not differ in patients with immediate-onset or late-onset faecal incontinence.

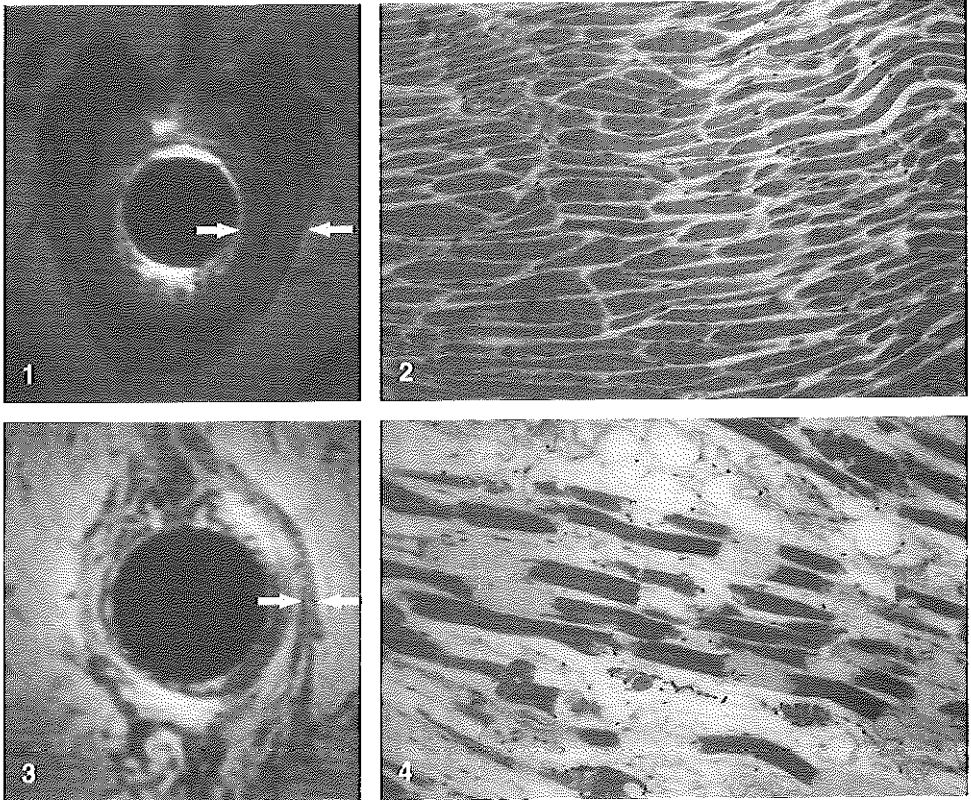


Figure 1. Normal external anal sphincter on endoanal MRI with anterior defect. *Arrows* relatively dark gray external anal sphincter

Figure 2. Biopsy specimen of normal external anal sphincter.

Figure 3. Atrophy of the external anal sphincter on endoanal MRI with anterior defect. *Arrows* relatively dark gray external anal sphincter

Figure 4. Biopsy specimen of atrophic external anal sphincter.

Discussion

Faecal incontinence is a troublesome condition. It may have any of various causes, but sphincter damage sustained during childbirth is the most common. It is generally thought that vaginal delivery also damages the innervation of the muscles of the pelvic floor. The major risk factors include a prolonged second stage of labour, delivery of a large baby and forceps delivery. The most commonly used tools in the diagnostic work-up of patients presenting with faecal incontinence consist of digital examination, anal manometry, proctography and electromyography. Recently, the assessment of sphincter injuries has been revolutionized by the introduction of endoanal ultrasound⁸. Cadaveric studies show a close correlation between ultrasonographic images and anatomical structures^{9,10}. Internal and/or external anal sphincter defects can be identified accurately. Endoanal ultrasonography is quick to perform and comparatively inexpensive¹¹. However, a major limitation of endoanal endosonography is the poor inherent contrast, which hinders identification of the external anal sphincter. Endoanal ultrasound does not define the echogenic external anal sphincter well against the perianal fat. Measurements of the diameter of the anal sphincter muscles are unreliable since these measures are not reproducible⁶. More recently endoanal MRI was added to the arsenal of sphincter assessment. It has been shown that the use of an endoanal coil also enables the detection of anal sphincter defects. The two techniques are equally accurate in detecting sphincter defects. However, the sphincter defects are better delineated with endoanal MRI¹². Correlation studies between MRI and in vitro anatomy have confirmed that MRI correctly identifies the various layers of the anal sphincter complex, and that endoanal MRI findings show a good correlation with cross-sectional anatomy^{13,14}. The sensitive region of the endoanal coil extends beyond the region normally visualized with endoanal ultrasound. Endoanal MRI, with its high intrinsic contrast resolution and the high spatial resolution of an

Sphincter Morphology

endoanal coil, enables the detection of external anal sphincter atrophy^{1,4,5}. Atrophy of the external anal sphincter is characterized by extreme thinning of its sphincter fibers or generalized fatty infiltration.

Until now, the specific morphological aspects of the external anal sphincter as shown by endoanal MRI, such as atrophy, have not been validated, and it was to validate these MRI findings that the present study was carried out. The authors used the outcome of histological examination as the 'gold standard'. Endoanal MRI accurately identified normal and abnormal external anal sphincter morphology. Sphincter atrophy was found in 36 per cent of the patients. We found no relationship between external anal sphincter atrophy and the patient's age. There was also no difference between the prevalence of sphincter atrophy in patients with immediate-onset and late-onset faecal incontinence. Therefore, it seems likely that sphincter atrophy is caused by the denervating effect of obstetric trauma, rather than ageing of the patient. Factors such as menopause and the resulting lack of estrogen hormones do not seem to be significant in causing sphincter atrophy.

The introduction of endoanal ultrasound emphasized the importance of internal and external anal sphincter defects. The introduction of endoanal MRI has not only confirmed these findings but also revealed the coexistence of sphincter atrophy. Sphincter defects are now considered as the main cause of continence disturbances following obstetric injury. External anal sphincter atrophy may play also an important role. Roberts *et al.*¹⁵ studied women with faecal incontinence after vaginal delivery, and reported that, although specific anterior defects may be expected following obstetric trauma, global defects of the sphincter, as assessed by anorectal manometry catheter, are not to be expected and may support the role of injury to the innervation of the musculature of the pelvic floor as a contributing cause of faecal incontinence after childbirth. Therefore, anal sphincter rupture and denervation may coexist in patients with faecal incontinence due to obstetric injury^{16,17}.

Denervation of the pelvic floor has serious consequences. Atrophy of the external anal sphincter is most likely the result of denervation injury. The external anal sphincter is composed of striated voluntary muscle and is controlled by the somatic nervous system. The efferent innervation of the external anal sphincter for both tonic and phasic contractions originates in spinal cord segment S2 and travels through the pudendal nerve¹⁸. The pudendal nerve supplies the ipsilateral external anal sphincter muscles, but not the puborectalis muscle¹⁹. The puborectalis muscle is innervated by a branch of the sacral nerve (S3 and S4). The puborectalis and the external anal sphincter muscles are thus innervated by different motor nerves, although both originate in the same spinal cord segments. Cross-innervation from side to side has been observed in monkeys, with a substantial overlap in the pudendal innervation on the two sides²⁰. This overlapping innervation may permit partial reinnervation from the opposite side when there is pudendal nerve damage. However, fiber hypertrophy may itself lead to secondary degenerative changes in muscles, and thus to failure of compensation²¹.

Until now, denervation of the external anal sphincter has been determined only by either electromyography or pudendal nerve terminal motor latencies. However, normal latencies do not exclude nerve damage because only the most rapidly conducting fibers are recorded. Also, denervation of the external anal sphincter and its detection fade in time while the sphincter damage remains. This probably explains why DeSouza *et al.*²² found that atrophy of individual components of the external anal sphincter is not related to the degree of delay in pudendal nerve conduction. Although further information on reinnervation may be obtained from single fiber electromyography, this is painful for the patient. Since electromyography is a blind procedure, loss of electrical muscular activity may also occur as a result of needle placement outside the external anal sphincter or within the internal sphincter. In contrast, endoanal MRI enables the direct detection of external sphincter atrophy, rather than

Sphincter Morphology

leaving its existence to be presumed. Endoanal MRI is well tolerated since it causes no more discomfort than digital examination. The present study validates the endoanal MRI findings. In our opinion, endoanal MRI renders electromyography and assessment of the pudendal nerve terminal motor latency largely superfluous.

References

1. deSouza NM, Puni R, Gilderdale DJ, Bydder GM. Magnetic resonance imaging of the anal sphincter using an internal coil. *Magn Reson Q* 1995; 11: 454-6.
2. deSouza NM, Kmiot WA, Puni R, Hall AS, Burl M, Bartram CI, Bydder GM. High resolution magnetic resonance imaging of the anal sphincter using an internal coil. *Gut* 1995; 37: 284-7.
3. Hussain SM, Stoker J, Laméris JS. Anal sphincter complex: endoanal MR imaging of normal anatomy. *Radiology* 1995; 197: 671-7.
4. Stoker J, Hussain SM, van Kempen D, Elevelt AJ, Laméris JS. Endoanal coil in MR imaging of anal fistulas. *AJR* 1996; 166: 360-2.
5. Hussain SM, Stoker J, Schouten WR, Hop WC, Laméris JS. Fistula in ano: endoanal sonography versus endoanal MR imaging in classification. *Radiology* 1996; 200: 475-81.
6. Enck P, Heyer T, Gantke B, Schmidt WU, Schafer R, Frieling T, Haussinger D. How reproducible are measures of the anal sphincter muscle diameter by endoanal ultrasound? *Am J Gastroenterol* 1997; 92: 293-6.
7. Briel JW, de Boer LM, Hop WCJ, Schouten WR. Clinical outcome of anterior overlapping external anal sphincter repair with internal anal sphincter imbrication. *Dis Colon Rectum* 1998; 41: 209-14.
8. Law PJ, Kamm MA, Bartram CI. Anal endosonography in the investigation of faecal incontinence. *Br J Surg* 1991; 78: 312-4.
9. Sultan AH, Nicholls RJ, 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.
10. Sultan AH, Kamm MA, Hudson CN, Nicholls RJ, Bartram CI. Endosonography of the anal sphincters: normal anatomy and comparison with manometry. *Clin Radiol* 1994; 49: 368-74.
11. Bartram CI, Sultan AH. Anal endosonography in faecal incontinence.

Sphincter Morphology

- Gut 1995; 37: 4-6.
12. Rociu E, Stoker J, Eijkemans MJC, Schouten WR, Laméris JS. Fecal incontinence: Endoanal US versus endoanal MR imaging. *Radiology* 1999; 212: 453-8.
 13. Van Beers BE, Kartheuser A, Delos MA, Grandin C, Detry R, Jamart J, Pringot J. MRI of the anal canal; correlation with histologic examination. *Magn Reson Imaging* 1996; 14: 151-6.
 14. Hussain SM, Stoker J, Zwamborn AW, Den Hollander JC, Kuiper JW, Entius CA, Laméris JS. Endoanal MRI of the anal sphincter complex: correlation with cross-sectional anatomy and histology. *J Anat* 1996; 189: 677-82.
 15. Roberts PL, Collier JA, Schoetz DJ Jr, Veidenheimer MC. Manometric assessment of patients with obstetric injuries and fecal incontinence. *Dis Colon Rectum* 1990; 33: 16-20.
 16. Jacobs PPM, Scheur M, Kuijpers JHC, Vingerhoeds MH. Obstetric faecal incontinence. Role of pelvic floor denervation and results of delayed sphincter repair. *Dis Colon Rectum* 1990; 33: 494-7.
 17. Cuesta MA, Meyer S, Derksen EJ, Boutkan H, Meuwissen SG. Anal sphincter imaging in fecal incontinence using endosonography. *Dis Colon Rectum* 1992; 35: 59-63.
 18. Whitehead WE, Schuster MN. Anorectal physiology and pathophysiology. *Am J Gastroenterol* 1978; 82: 478-97.
 19. Percy JP, Neill ME, Swash M, Parks AG. Electrophysiological study of motor nerve supply of pelvic floor. *Lancet* 1981; 1: 16-17.
 20. Wunderlich M, Swash M. The overlapping innervation of the two sides of the external anal sphincter by the pudendal nerve. *J Neurol Sci* 1983; 59: 91-109.
 21. Swash M, Schwartz MS. Implications of longitudinal fibre splitting in neurogenic and myopathic disorders. *J Neurology Neurosurg Psychiatry* 1977; 40: 1152-9.
 22. DeSouza NM, Puni R, Zbar A, Gilderdale DJ, Coutts GA, Krausz T.

MR imaging of the anal sphincter in multiparous women using an endoanal coil: correlation with in vitro anatomy and appearances in fecal incontinence. *AJR* 1996; 167: 1465-71.

CHAPTER 7

EXTERNAL ANAL SPHINCTER ATROPHY ON ENDOANAL MAGNETIC RESONANCE IMAGING ADVERSELY AFFECTS CONTINENCE AFTER SPHINCTEROPLASTY

JW Briel¹, J Stoker², E Rociu², JS Laméris², WCJ Hop³, and WR Schouten¹.

From the Departments of ¹General Surgery, ²Radiology, and ³Epidemiology
and Biostatistics,
University Hospital Dijkzigt, Rotterdam, The Netherlands

Br J Surg 1999; 86: 1322-7.

Abstract

There is still considerable debate about the value of preoperative anorectal physiological parameters in predicting the clinical outcome after sphincteroplasty. Recently it has been reported that atrophy of the external anal sphincter can be clearly shown with endoanal magnetic resonance imaging (MRI). The aims of this study were to investigate the prevalence of external anal sphincter atrophy in women with anterior sphincter defects due to obstetric injury and to determine the impact of external anal sphincter atrophy on the outcome of sphincteroplasty. In this prospective study, 20 consecutive women (median age 50 (range 28-75) years) with faecal incontinence due to obstetric trauma were assessed before operation with endoanal ultrasonography and endoanal MRI. The external anal sphincter was examined and evaluated for the presence of atrophy. The clinical outcome of sphincteroplasty was interpreted without knowledge of the magnetic resonance and ultrasonographic images. In all patients anterior sphincter defects could be demonstrated with ultrasonography and MRI. External anal sphincter atrophy could only be demonstrated on MRI. Eight of 20 patients had external anal sphincter atrophy (40 per cent). Continence was restored in 13 patients (65 per cent). Outcome was significantly better in those without external anal sphincter atrophy (11 of 12 patients *versus* two of eight; $P=0.004$). External anal sphincter atrophy can only be visualized on endoanal MRI and affects continence after sphincteroplasty. Endoanal MRI is valuable in the preoperative assessment of patients with faecal incontinence.

Introduction

Childbirth is the most common cause of faecal incontinence¹. Following primary gynaecological repair of perineal tears, persistent defects can be

demonstrated in 85 per cent of patients². A substantial part of patients with an occult sphincter defect will become incontinent with increasing age. The incidence of occult sphincter damage after childbirth was unknown until *Sultan et al.*³ studied a consecutive group of 202 pregnant women. On endoanal ultrasonography, 35 per cent of primiparae who delivered vaginally developed a sphincter defect involving one or both muscles, which persisted at 6 months. Although sphincter defects due to obstetric trauma can be restored adequately by overlapping sphincteroplasty, incontinence persists in a substantial number of patients. The reported failure rates at long-term follow-up vary between 4 per cent⁴ and 53 per cent⁵.

Preoperative evaluation of patients with faecal incontinence usually includes anorectal manometry, evacuation proctography, assessment of pudendal nerve terminal motor latency, and endoanal ultrasonography. The role of these preoperative anorectal function tests in predicting the outcome of sphincteroplasty is still controversial. Reports about the predictive value of these tests are numerous and conflicting.

Recently, it has been shown that endoanal magnetic resonance imaging (MRI) enables the detection of external anal sphincter atrophy⁶⁻¹⁰ (*Figs. 1 and 2*).

Using conventional endoanal ultrasonography, atrophy cannot be demonstrated. The main disadvantages with MRI are the time required and the cost of an examination, which are both greater than those of endoanal ultrasonography. Therefore preoperative endoanal MRI is only justified if it influences surgical decision-making or predicts final outcome. A prospective study was conducted to investigate the prevalence of external anal sphincter atrophy in women with anterior sphincter defects due to obstetric injury. The impact of external anal sphincter atrophy on the outcome of sphincteroplasty was also studied.

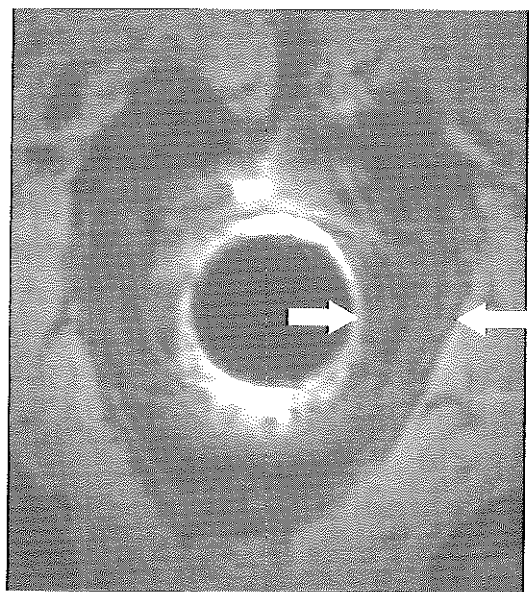


Figure 1. Normal external anal sphincter on endoanal MRI with anterior defect. *Arrows* relatively dark gray external anal sphincter

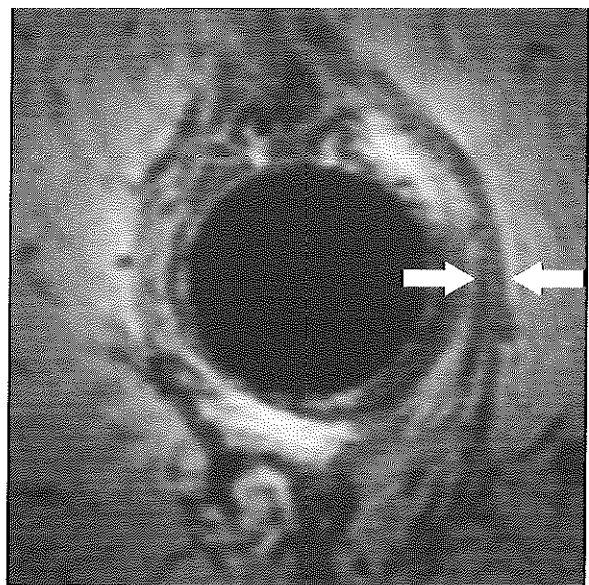


Figure 2. Atrophy of the external anal sphincter on endoanal MRI with anterior defect. *Arrows* relatively dark gray external anal sphincter

Patients & Methods

Twenty consecutive women (median age 50 (range 28-75) years) with faecal incontinence due to obstetric trauma were assessed clinically by means of history and digital examination, and by preoperative anal manometry, endoanal ultrasonography, and endoanal MRI. Both sphincters were examined and external anal sphincter atrophy was scored. Within 6 months of the preoperative assessment, all patients underwent anterior anal repair, as described earlier¹¹. All repairs were performed by one surgeon (WRS). Clinical outcome was evaluated using the grade and frequency of faecal incontinence, the need for pads, the grade of social isolation and patient satisfaction after a median follow-up of 1 year. The degree of incontinence was graded as described by Parks¹²: grade I, fully continent; grade II, soiling and incontinence for gas; grade III, incontinence for liquids; and grade IV, incontinence for solid stool. Restoration of continence from grade IV to grade II or I, or from grade III to grade I, was defined as a successful outcome. Outcome was interpreted without knowledge of the magnetic resonance and ultrasonography images.

The decision to perform surgery was based solely on endosonography. MRI was considered additional and did not influence treatment.

Endoanal Sonography

A Brüel & Kjaer (Naerum, Denmark) ultrasound scanner was used with a rotating probe providing a 360° image. A 7-MHz transducer, with minimum beam width of 1.1 mm and a focal length of about 3 cm, was used. Transverse images were obtained, at least at four different levels, through the anal canal.

Endoanal Magnetic Resonance Imaging

MRI was performed at 0.5 T (Gyrosan T5-II; Philips Medical Systems, Best, The Netherlands). An endoanal coil with a diameter of 19 mm (Philips Medical Systems) was used⁷⁻⁹. Axial T2-weighted three-dimensional gradient echo and coronal and sagittal T2-weighted turbo spin-echo sequences were performed.

Determination of Atrophy

External anal sphincter atrophy was assessed on hard copy by one radiologist (JS). To determine sphincter damage with endoanal MRI, as no criteria exist, normal endoanal MRI findings were used as a reference⁷. Atrophy of the external anal sphincter was defined as extreme thinning of its sphincter fibres or generalized fatty infiltration. The magnetic resonance images were also evaluated quantitatively using a work station with commercially available software (Gyrovu HR; Philips Medical Systems, Best, The Netherlands). In all cases the anterior part of the external anal sphincter could not be identified because of an anterior sphincter defect. External anal sphincter width at the posterior side and at both lateral sides was determined. Furthermore, the arc of the anterior defect was noted. The area of the remaining part of the external anal sphincter was measured (*Fig. 3*).

All measurements were performed on the MRI slice located at the mid-level of the anal canal, in each patient. This level of the anal canal was chosen based on normal anatomy findings using endoanal MRI⁷. At the lowest part of the anal canal, the external anal sphincter consists of two halves embedded within the ischioanal space. Cranial to this level, the two lateral halves of the external anal sphincter are connected to each other anteriorly. Just slightly cranial to this level the external sphincter becomes completely

circular, representing the mid-level, which was measured in the present study. More cranial to this level, the puborectalis muscle is first seen on endoanal MRI.

To investigate the possibility of interobserver bias, the results of quantitative MRI assessment performed by two investigators (JWB and ER) were compared. Each was blinded to the measurements of the other.

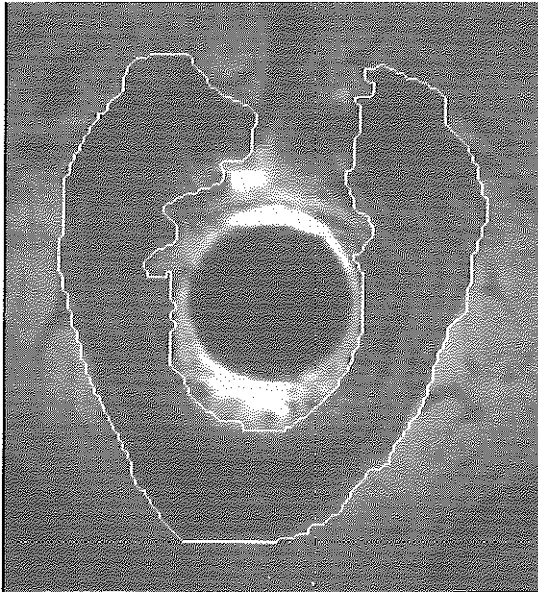


Figure 3. Endoanal magnetic resonance imaging showing delineation of area on the image. Area is calculated by the integral software.

Statistical Analysis

Fisher's exact test was used for the comparison of percentages. Mann-Whitney test was used to compare continuous data between groups. To determine agreement between measurements of different investigators, the intraclass correlation coefficient was used. $P=0.05$ was considered the limit of significance.

Results

In all patients anterior sphincter defects were demonstrated with ultrasonography and MRI. Qualitative or semi-quantitative measurements of the thickness of the external sphincter muscle was not feasible on endoanal ultrasonography. External anal sphincter atrophy could only be demonstrated on endoanal MRI. The prevalence of external anal sphincter atrophy (determined by one of the radiologists) was 40 per cent (eight of 20 patients). Continence was restored in 13 patients (65 per cent). Comparing patients with and without atrophy, clinical outcome was significantly better in those without external anal sphincter atrophy (11 of 12 *versus* two of eight; $P=0.004$). The MRI measurements are listed in *Table 1*.

Table 1. Magnetic resonance imaging measurements in patients with poor and good outcome after sphincter repair.

	<i>Good Outcome</i>	<i>Poor Outcome</i>	<i>P</i>
Width (mm)			
Posterior	9.3 (2.0-23.5)	9.3 (4.7-13.1)	0.12
Left	4.3 (1.1-8.8)	2.5 (1.1-5.6)	0.22
Right	3.9 (1.1-9.0)	3.0 (1.1-6.0)	0.73
Arc (°)	55 (19-146)	85 (51-190)	0.04
Area (mm ²)	393 (121-1350)	218 (87-360)	0.002

Values are median (range). Mann-Whitney test.

Determination of external anal sphincter width at the posterior side and both lateral sides was shown not to be useful in predicting the outcome of sphincter repair. However, there was a significant relationship between the outcome of sphincteroplasty and the arc of the external anal sphincter defect ($P=0.04$). Furthermore, the area of the remaining external anal

sphincter was related to outcome ($P=0.002$). Of these two parameters, the area of the remaining external anal sphincter was the better predictor (*Table 1*).

For area of the remaining external anal sphincter, the cut-off point for successful outcome was determined. The greatest area in patients with a poor outcome after sphincteroplasty was 360 mm² (*Fig. 4*).

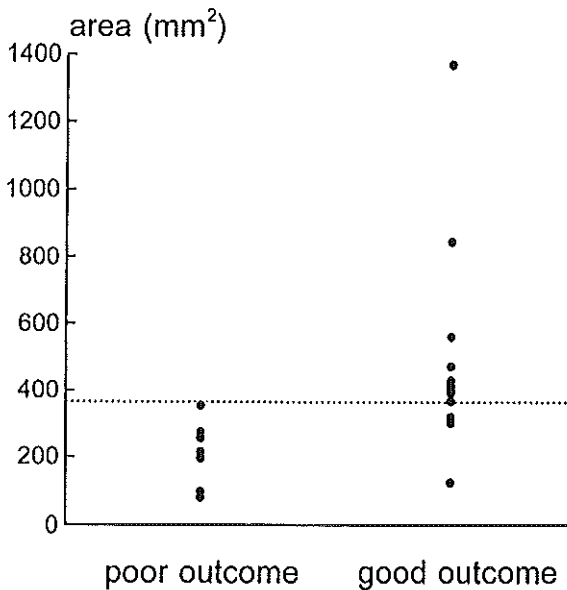


Figure 4. Outcome of sphincter repair related to preoperative external anal sphincter area.

Of the ten patients with an area of 360 mm² or less, only three had a successful outcome after sphincter repair (30 per cent). All ten patients with an external anal sphincter area greater than 360 mm² regained continence (100 per cent). A receiver operating characteristic curve was calculated for the cut-off point of area (*Fig. 5*).

For a cut-off point of 300 mm², the sensitivity and specificity were 92 and 86 per cent respectively. For an area of 360 mm², they were 77 and 100 per cent.

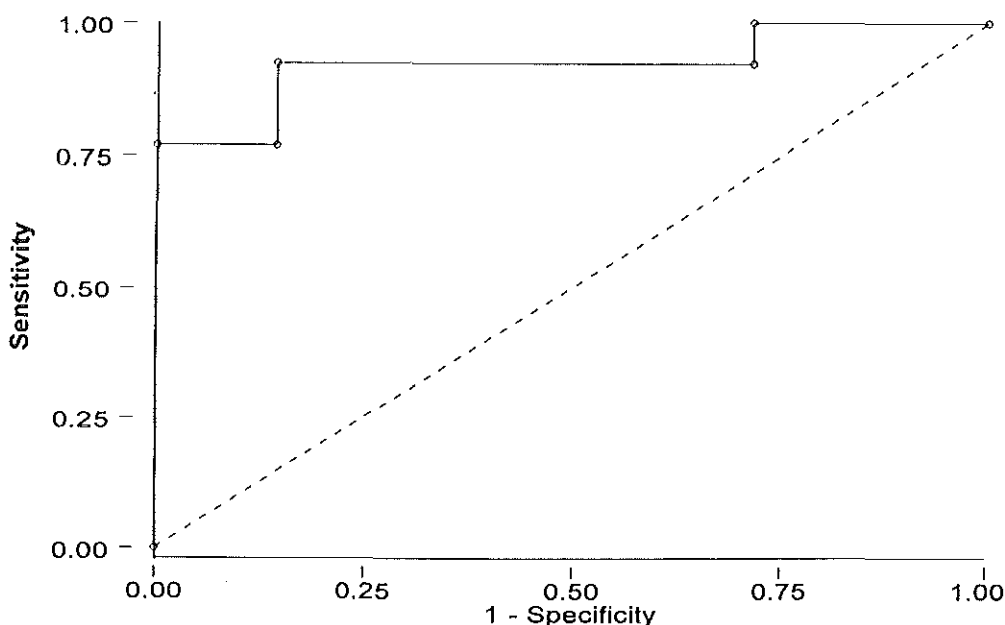


Figure 5. Receiver operating characteristic curve (solid line) to demonstrate cut-off points of area.

Atrophy, as assessed by one radiologist, and the quantitative measurement of area on MRI were well related ($P=0.001$). In patients with atrophy, as described by the radiologist, the measured area was significantly lower than in patients without radiological signs of external anal sphincter atrophy (214 mm^2 versus 399 mm^2 ; $P=0.001$).

Comparing the quantitative measurements of the two investigators (JWB and ER), there was no systemic difference ($P=0.67$). The interobserver agreement was good (intraclass correlation coefficient: 0.91).

Age and surgical outcome were not related. There was also no significant correlation between age and atrophy. Furthermore, age was not related to any of the MRI measurements (width, arc or area).

Discussion

The use of conventional whole-body MRI in the evaluation of patients with faecal incontinence has been proven to be of great value in assessing patients with congenital anorectal anomalies¹³. However, anal endosonography is the imaging technique of choice for the detection of abnormalities in the region of the anal sphincters, when compared with MRI using a whole-body receiver coil¹⁴⁻¹⁶. The spatial resolution of endoanal sonography is superior to that of body coil MRI. However, a major limitation of anal endosonography is the poor inherent contrast, which makes it difficult to identify the external anal sphincter. Recently, it has been shown that the use of an endoanal coil enables the detection of anal sphincter defects as well as external anal sphincter atrophy on MRI^{6,8,9}. Moreover, the sensitive region of the coil extended beyond the region normally visualized with endoanal ultrasonography. In this study the value of preoperative endoanal MRI was assessed.

Anterior sphincteroplasty was found to be successful in 13 of 20 patients (65 per cent) in the present study. This outcome is in accordance with other series^{4,5}. External anal sphincter atrophy was present in eight of 20 patients (40 per cent). In 1977, Parks¹⁷ examined biopsies of the external anal sphincter of patients with idiopathic faecal incontinence. The biopsies of all incontinent patients showed histological evidence of denervation. He therefore suggested that this type of faecal incontinence could result from denervation of muscles of the anal sphincter mechanism. This could be the result of entrapment or stretch injury of the pudendal or perineal nerves occurring as a consequence of rectal descent induced during repeated defecation straining in constipated patients¹⁸, or from injuries to these nerves associated with childbirth¹⁹. Nowadays this type of faecal incontinence is called neurogenic. In patients with faecal incontinence due to obstetric injury both anal sphincter rupture and denervation may coexist²⁰⁻²². Several authors have reported that denervation of the pelvic

Endoanal MRI

floor and subsequent failure of surgical repair can be predicted by preoperative electromyography^{5,20,23-25}. In contrast, other authors were not able to confirm these findings²⁶⁻²⁸. Also, denervation of the external anal sphincter and its detection fade in time while the sphincter damage remains. Furthermore, electromyography of the pelvic floor is generally considered to be distressing. It seems likely that in patients with both denervation and sphincter defects, denervation of the pelvic floor will persist, regardless of the outcome of repair of the sphincter defect. It has been shown that functional results of sphincter repair in patients with neurogenic faecal incontinence are poor²⁹. Therefore, in patients with both denervation and sphincter defects, a poor outcome might be expected. If traumatic denervation leads to atrophy of the external anal sphincter, the finding of atrophy of this sphincter muscle influences outcome after surgery. This hypothesis was confirmed by the present study. Outcome was significantly better in those without external anal sphincter atrophy. The area of the remaining external anal sphincter on endoanal MRI appeared to be of significant value in identifying patients with a favourable outcome. The objective quantitative measurement of this area was of equal value as the rather subjective determination of atrophy on hard copies of magnetic resonance images by the radiologist; the two methods were well correlated ($P=0.001$). The separate measurements of sphincter width at the posterior side and both lateral sides did not relate to outcome. This might be explained by the isolated value of the width measurement. The computed area consists of numerous adjacent sphincter width measurements, thereby overcoming the effect of sphincter irregularity. Determination of the presence of atrophy on endoanal MRI may enable the prediction of outcome after sphincteroplasty. Patients below the cut-off area (360 mm^2) are less likely to benefit from sphincter repair. Confirmation of this cut-off value in another group of patients is necessary.

Endoanal MRI is the first imaging technique to predict functional outcome after sphincter repair. Moreover, it directly enables the detection of external

sphincter atrophy, rather than assuming atrophy by detection of pelvic floor denervation assessed by either electromyography or pudendal nerve terminal motor latencies. The authors advocate the inclusion of endoanal MRI in the preoperative work-up of patients with faecal incontinence to help predict the outcome of surgery in individual patients.

The alternatives for patients with persistent faecal incontinence after sphincteroplasty are either creation of a stoma or dynamic gracilis plasty. Since dynamic gracilis plasty is technically easier in patients in whom previous attempts to restore continence (i.e. sphincteroplasty) have not been made, selection of patients is preferable. Therefore, patients with external anal sphincter atrophy as seen on endoanal MRI might be candidates for dynamic gracilis plasty in the first place.

References

1. Ctercteko GC, Fazio VW, Jagelman DG, Lavery IC, Weakly FL, Melia M. Anal sphincter repair: a report of 60 cases and review of the literature. *Aust N Z J Surg* 1988; 58: 703-10.
2. Sultan AH, Kamm MA, Hudson CN, Bartram CI. Third degree obstetric anal sphincter tears: risk factor and outcome of primary repair. *BMJ* 1994; 308: 887-91.
3. Sultan AH, Kamm MA, Hudson CN, Thomas JM, Bartram CI. Anal-sphincter disruption during vaginal delivery. *New Engl J Med* 1993; 329: 1905-11.
4. Fang DT, Nivatvongs S, Vermeulen FD, Herman FN, Goldberg SM, Rothenberger DA. 'Overlapping sphincteroplasty for acquired anal incontinence. *Dis Colon Rectum* 1984; 27: 720-2.
5. Laurberg S, Swash M, Henry MM. Delayed external sphincter repair for obstetric tear. *Br J Surg* 1988; 75: 786-8.
6. deSouza NM, Puni R, Gilderdale DJ, Bydder GM. Magnetic resonance imaging of the anal sphincter using an internal coil. *Magn Reson Q* 1995; 11: 454-6.
7. Hussain SM, Stoker J, Laméris JS. Anal sphincter complex: endoanal MR imaging of normal anatomy. *Radiology* 1995; 197: 671-7.
8. Stoker J, Hussain SM, van Kempen D, Elevelt AJ, Laméris JS. Endoanal coil in MR imaging of anal fistulas. *AJR Am J Roentgenol* 1996; 166: 360-2.
9. Hussain SM, Stoker J, Schouten WR, Hop WC, Laméris JS. Fistula in ano: endoanal sonography versus endoanal MR imaging in classification. *Radiology* 1996; 200: 475-81.
10. deSouza NM, Kmiot WA, Puni R, Hall AS, Burl M, Bartram CI, Bydder GM. High resolution magnetic resonance imaging of the anal sphincter using an internal coil. *Gut* 1995; 37: 284-7.
11. Briel JW, de Boer LM, Hop WCJ, Schouten WR. Clinical outcome of

- anterior overlapping external anal sphincter repair with internal anal sphincter imbrication. *Dis Colon Rectum* 1998; 41: 209-14.
12. Parks AG. Anorectal incontinence. *J R Soc Med* 1975; 68: 21-30.
 13. Sato Y, Pringle KC, Bergman RA, Yuh WT, Smith WL, Soper RT, Franken EA Jr. Congenital anorectal anomalies: MR imaging. *Radiology* 1988; 168: 157-62.
 14. Sultan AH, Kamm MA, Hudson CN, Nicholls JR, Bartram CI. Endosonography of the anal sphincters: normal anatomy and comparison with manometry. *Clin Radiol* 1994; 49: 368-74.
 15. Sultan AH, Nicholls RJ, 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.
 16. Schafer A, Enck P, Furst G, Kahn T, Frieling T, Lubke HJ. Anatomy of the anal sphincters. *Dis Colon Rectum* 1994; 37: 777-81.
 17. Parks AG, Swash M, Urich H. Sphincter denervation in anorectal incontinence and rectal prolapse. *Gut* 1977; 18: 656-65.
 18. Kiff ES, Barnes PRH, Swash M. Evidence of pudendal neuropathy in patients with perineal descent and chronic straining at stool. *Gut* 1984; 25: 1279-82.
 19. Snooks SJ, Swash M, Setchell M. Injury to the innervation of pelvic floor sphincter musculature in childbirth. *Lancet* 1984; ii: 546-50.
 20. Jacobs PPM, Scheur M, Kuijpers JHC, Vingerhoeds MH. Obstetric faecal incontinence. Role of pelvic floor denervation and results of delayed sphincter repair. *Dis Colon Rectum* 1990; 33: 494-7.
 21. Felt-Bersma RJF, Cuesta MA, Koorevaar M, Strijers RI, Meuwissen SG, Dercksen EJ, Wesdorp RT. Anal endosonography; relationship with anal manometry and neurophysiologic tests. *Dis Colon Rectum* 1992; 35: 944-9.
 22. Cuesta MA, Meyer S, Dercksen EJ, Boutkan H, Meuwissen SG. Anal sphincter imaging in fecal incontinence using endosonography. *Dis Colon Rectum* 1992; 35: 59-63.

23. Yoshioka K, Keighley MRB. Sphincter repair for fecal incontinence. *Dis Colon Rectum* 1989; 32: 39-42.
24. Infantino A, Melega E, Negrin P, Masin A, Carnio S, Lise M. Striated anal sphincter electromyography in idiopathic fecal incontinence. *Dis Colon Rectum* 1995; 38: 27-31.
25. Wexner SD, Marchetti F, Jagelman DG. The role of sphincteroplasty for fecal incontinence reevaluated: a prospective physiologic and functional review. *Dis Colon Rectum* 1991; 34: 22-30.
26. Young CJ, Mathur MN, Eysers AA, Solomon MJ. Successful overlapping anal sphincter repair: relationship to patient age, neuropathy, and colostomy formation. *Dis Colon Rectum* 1998; 41: 344-9.
27. Engel AF, Kamm MA, Sultan AH, Bartram CI, Nicholls RJ. Anterior anal sphincter repair in patients with obstetric trauma. *Br J Surg* 1994; 81: 1231-4.
28. Chen AS, Luchtefeld MA, Senagore AJ, MacKeigan JM, Hoyt C. Pudendal nerve latency. Does it predict outcome of anal sphincter repair? *Dis Colon Rectum* 1998; 41: 1005-9.
29. Briel JW, Schouten WR. Disappointing results of postanal repair in the treatment of fecal incontinence. *Ned Tijdschr Geneeskde* 1995; 139: 23-6.

CHAPTER 8

SUMMARY AND CONCLUSIONS

SAMENVATTING EN CONCLUSIES

Summary

Faecal incontinence is a common but infrequently reported, imperfectly understood, multifactorial condition with far-reaching socio-economic and psychological implications. Limited success with somewhat empirical surgical procedures implies that patients should be investigated fully, indications for surgery should be clear, and disability should be serious enough to demand surgical intervention. Dietary adjustments and medical treatment should be tried first. In **Chapter 2** the results of colonic irrigation in patients with continence disturbances following failed repair for obstetric injury or iatrogenic sphincter trauma are described. Patients with faecal soiling benefit more from colonic irrigation than patients with incontinence for liquid or solid stools. Successful outcome is 79 and 38 per cent, respectively. If creation of a stoma is considered, especially in patients with intractable and disabling soiling, it might be worthwhile to treat these patients first by colonic irrigation. The long-term results of suture rectopexy in patients with faecal incontinence are discussed in **Chapter 3**. Because the clinical outcome of rectopexy is only acceptable in incontinent patients with complete rectal prolapse (67 versus 38 per cent), a conservative attitude toward surgery in patients with faecal incontinence and associated incomplete prolapse is recommended. These patients should be considered as candidates for endoanal ultrasound and subsequent sphincter repair. Furthermore, it makes proctography largely superfluous in the evaluation of patients with faecal incontinence since complete rectal prolapse can be seen on physical examination. Formerly, postanal repair was considered for patients with 'idiopathic' faecal incontinence. The long-term results are presented in **Chapter 4**. It is concluded that postanal repair is not beneficial for the majority of patients with faecal incontinence (success-rate: 46 per cent). Nowadays, endoanal ultrasound will reveal occult anterior sphincter defects, which might be treated by subsequent anterior anal sphincter repair. In patients with true neuropathic faecal incontinence other treatment modalities are preferable. In **Chapter 5** the long-term results of surgical repair for anterior sphincter defects are

discussed. Initial good results deteriorate at follow-up. The more complex anterior anal repair fails to confer clinical benefit compared with the rather simple direct sphincter repair (68 versus 63 per cent). In the search for new imaging techniques to enhance understanding and solving the problem of faecal incontinence, endoanal MRI was introduced. Endoanal MRI is the only imaging technique that reveals atrophy of the external anal sphincter. In **Chapter 6** the close relationship between endoanal MRI findings and actual sphincter morphology is confirmed (89 per cent sensitivity, 94 per cent specificity). The prevalence of external anal sphincter atrophy is 36 per cent. The predictive value of endoanal MRI and the consequences of atrophy of the external anal sphincter are revealed in **Chapter 7**: atrophy of the external anal sphincter adversely affects continence after sphincter repair. In a selected group of patients without external anal sphincter atrophy it is possible to restore continence in 92 per cent of the treated patients. Now that preoperative selection of patients is possible using endoanal MRI, patients with atrophy of the external anal sphincter might be candidates for dynamic graciloplasty in the first place. Unwelcome though it is, colostomy may be the ultimate remedy in some patients.

Based on the findings of this thesis, it is concluded that:

- Colonic irrigation is worthwhile in patients with faecal soiling.
- Rectopexy is only indicated for patients with faecal incontinence due to complete rectal prolapse.
- Proctography is not necessary in the assessment of patients with faecal incontinence.
- Postanal repair for faecal incontinence is not indicated.
- Results of anterior anal repair are comparable with direct sphincter repair.

Summary

- Atrophy of the external anal sphincter is correctly identified by endoanal MRI.
- Preoperative endoanal MRI in the assessment of patients with faecal incontinence is worthwhile because it predicts outcome after sphincteroplasty.

Faecale incontinentie is een frequent voorkomende, onvoldoende begrepen, multifactoriële aandoening met verstrekende socio-economische en psychische gevolgen. Patiënten dienen volledig onderzocht te worden, indicaties voor chirurgie dienen helder te zijn, en de klacht van de patiënt ernstig genoeg om chirurgische interventie te overwegen. Dieet maatregelen en medicamenteuze behandeling moeten eerst worden geprobeerd. In **Hoofdstuk 2** worden de resultaten van rectum spoelingen bij patiënten met continentie problemen na een mislukte operatie voor obstetrisch of iatrogeen kringspier letsels beschreven. Patiënten met soiling hebben meer baat bij rectum spoelingen dan patiënten met incontinentie voor dunne ontlasting. Het succespercentage is respectievelijk 79 en 38 procent. Indien het aanleggen van een stoma wordt overwogen, is het aantrekkelijk eerst nog met rectum spoelingen te behandelen, met name bij patiënten met ernstige soiling. De lange-termijn resultaten van rectopexie bij patiënten met faecale incontinentie worden besproken in **Hoofdstuk 3**. Omdat het klinisch resultaat van rectopexie uitsluitend goed is bij incontinente patiënten met een uitwendige rectum prolaps (67 versus 38 procent), wordt een afwachtende houding geadviseerd ten aanzien van chirurgisch ingrijpen bij patiënten met faecale incontinentie en een inwendige rectum prolaps. Deze patiënten dienen endoanale echografie te ondergaan met aansluitende kringspier plastiek. Defaecografie is overbodig bij de beoordeling van patiënten met faecale incontinentie omdat een uitwendige rectum prolaps gezien kan worden bij het lichamelijk onderzoek. Vroeger werd een achterste reefplastiek uitgevoerd bij patiënten met 'idiopathische' faecale incontinentie. De lange-termijn resultaten worden gepresenteerd in **Hoofdstuk 4**. De conclusie is dat de achterste reefplastiek niet gunstig is voor het merendeel van de patiënten met faecale incontinentie (succespercentage: 46 procent). Tegenwoordig laat endoanale echografie bij deze patiënten een verborgen kringspier defect zien aan de voorzijde van het anale kanaal, hetgeen met een voorste kringspier plastiek dient te worden behandeld. Bij patiënten waarbij sprake is van echte

Summary

neurogene faecale incontinentie, hebben andere therapeutische opties de voorkeur. In **Hoofdstuk 5** worden de lange-termijn resultaten van het herstel van anterieure kringspier letsels besproken. Initieel goede resultaten zijn minder gunstig bij nacontrole. De meer complexe voorste reefplastiek blijkt niet beter te zijn dan de relatief eenvoudige overlappende kringspier plastiek (68 versus 63 procent). Om het probleem van faecale incontinentie beter te begrijpen is gezocht naar nieuwe beeldvormende technieken. Endoanale MRI werd recent geïntroduceerd en toont als enige beeldvormende techniek atrofie van de uitwendige kringspier. In **Hoofdstuk 6** wordt de nauwe relatie tussen de bevindingen van endoanale MRI en histopathologisch onderzoek van de uitwendige kringspier nader belicht (sensitiviteit: 89 procent; specificiteit: 94 procent). De voorspellende waarde van atrofie van de uitwendige kringspier wordt opgehelderd in **Hoofdstuk 7**: Atofie van de uitwendige kringspier beïnvloedt het resultaat van voorste reefplastiek negatief. In een geselecteerde groep patiënten zonder atrofie van de uitwendige kringspier is het mogelijk gebleken de continentie te herstellen in 92 procent van de geopereerde patiënten. Nu preoperatieve selectie mogelijk is door middel van endoanale MRI, is het de vraag of patiënten met atrofie van de uitwendige kringspier niet direct een dynamische gracilis plastiek zouden moeten ondergaan. Uiteindelijk zal een stoma in sommige patiënten de definitieve behandeling bieden.

Op grond van de resultaten van dit onderzoek zijn de volgende conclusies getrokken:

- Rectaal spoelen is zinvol bij patiënten met soiling.
- Rectopexie is uitsluitend geïndiceerd voor patiënten met faecale incontinentie als gevolg van een uitwendige rectum prolaps.
- Defaecografie is van geen waarde bij de beoordeling van patiënten met faecale incontinentie.

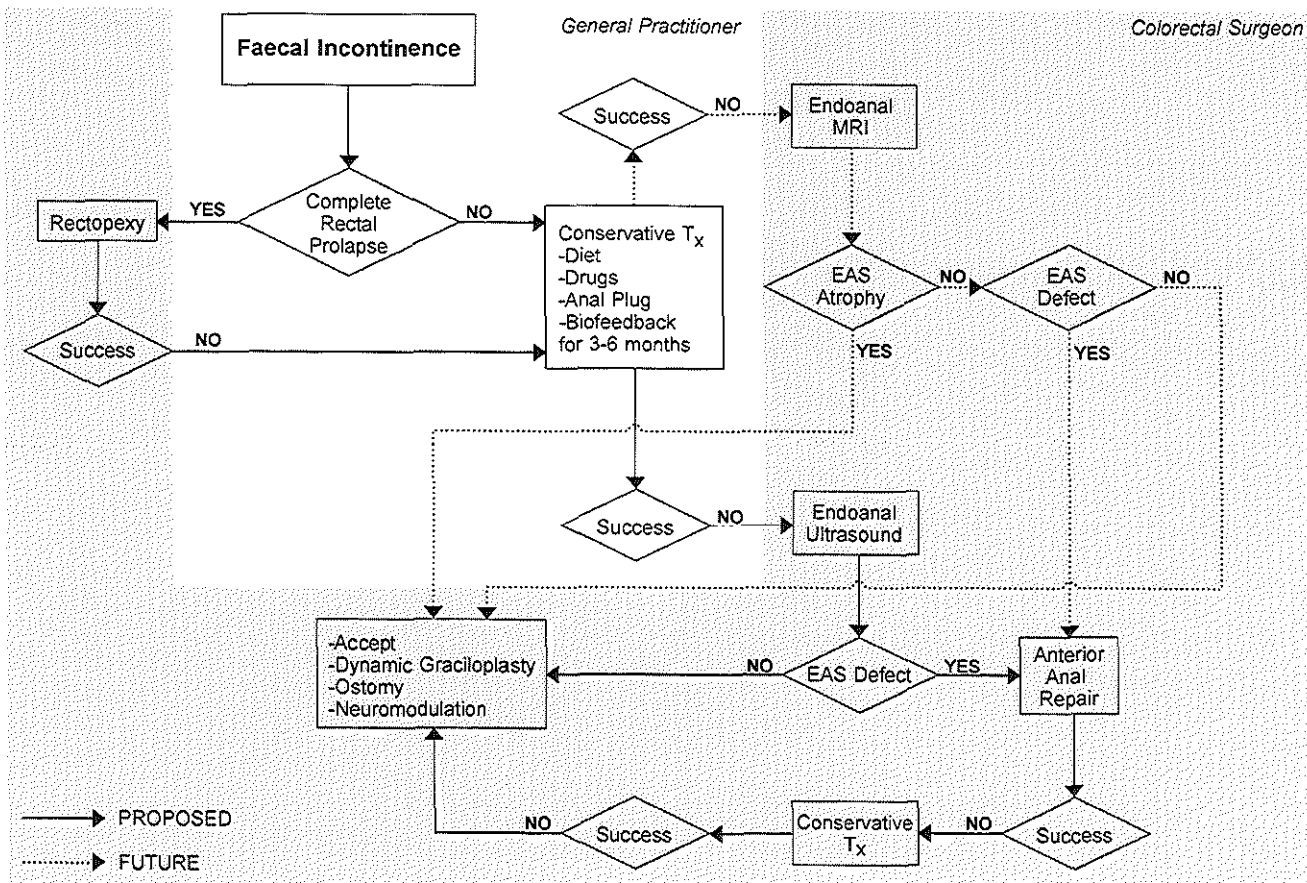
- Er is geen plaats voor de achterste reefplastiek bij de behandeling van patiënten met faecale incontinentie.
- De resultaten van de complexe voorste reefplastiek zijn vergelijkbaar met die van de relatief simpele overlappende kringspierplastiek.
- Atrofie van de uitwendige kringspier is aan te tonen door middel van endoanale MRI.
- Preoperatieve endoanale MRI bij de beoordeling van patiënten met faecale incontinentie is zinvol, omdat het resultaat van voorste reefplastiek hiermee voorspeld kan worden.

Summary

Algorithm

Based on the conclusions drawn from this thesis, the following algorithm for the investigation and treatment of patients with faecal incontinence is presented.

Algorithm for Faecal Incontinence



DANKWOORD

Aan de totstandkoming van dit proefschrift hebben velen direct of indirect, inhoudelijk of ondersteunend, een bijdrage geleverd. Zonder de illusie te hebben volledig te kunnen zijn wil ik een aantal mensen in het bijzonder danken.

In de eerste plaats, mijn copromotor, Dr. W.R. Schouten. Beste Ruud, zonder jouw nimmer aflatend en ook onnavolgbaar enthousiasme zou dit proefschrift nooit tot stand zijn gekomen. Juist door deze aanstekelijkheid werd mijn belangstelling voor dit promotieonderzoek al in een vroeg stadium gewekt. Ik bedank je voor de persoonlijke en wetenschappelijke begeleiding van deze promotie. De vele (internationale) discussies die we voerden hebben altijd weer tot resultaat geleid. De vrijheid bij het verrichten van het onderzoek was zeer stimulerend. Bedankt!

Mijn beide promotoren, Prof.dr. H.W. Tilanus en Prof.dr. H.A. Bruining ben ik veel dank verschuldigd voor het in mij gestelde vertrouwen.

Prof.dr. C.G.M.I. Baeten en Dr. H. van Dekken ben ik erkentelijk voor de vlotte beoordeling van het manuscript.

Dr. J. Stoker; beste Jaap, dank voor jouw bereidheid de eindeloze berg MRI's te (her-) beoordelen en mij te introduceren in de wereld van de endoanale MRI. Jouw kritische kanttekeningen en suggesties waren zeer waardevol.

Prof.dr. Th.J.M. Helmerhorst, Prof.dr. J. Jeekel en Prof.dr. E.J. Kuipers dank ik voor hun bereidheid zitting te nemen in de promotiecommissie.

Elk hoofdstuk van dit proefschrift was een nieuwe uitdaging, welke ik met verschillende mensen aanging. Ik wil Eline Vlot, Sandra Smits, Ingrid van Kessel, Michiel Boerma, Luuk de Boer, Wim Hop, Elena Rociu, Prof.dr. J.S. Laméris en Prof.dr. W.J. Mooi dankzeggen voor hun waardevolle bijdrage aan de verschillende hoofdstukken.

Mijn beide paranimfen, Pieter-Joost van Wattum en David Zimmerman dank ik voor hun vriendschap en steun bij de verdediging van dit proefschrift.

Pieter-Joost, hoewel jij al weer geruime tijd aan de andere kant van de grote plas verblijft houden wij de goede wederzijdse betrekkingen regelmatig in stand. Het is een goed gevoel om tijdens de verdediging van dit proefschrift een psychiater aan mijn zijde te hebben staan.

David, jij was van onschatbare waarde tijdens mijn verblijf in het St. Clara ziekenhuis. Ook bij de voltooiing van dit proefschrift ben jij van grote betekenis geweest. Het speelse gemak waarmee jij de meest exotische creaties uit een computer weet te toveren is opzienbarend. Beste David, dank voor al jouw bemoeienissen.

Ik wil mijn vader en, in dierbare herinnering, mijn moeder bedanken voor alle mogelijkheden die zij mij hebben geboden en voor hun vertrouwen waarop ik altijd kon rekenen.

Tenslotte, lieve Maud, ik dank je voor je geduld en steun, en, omdat het leven met jou zo ongecompliceerd mooi is.

CURRICULUM VITAE

10 februari 1969	Geboren te Rotterdam
1981-1987	VWO, Gymnasium ß Johannes Calvijn, Rotterdam
1987-1994	Doctoraal Examen Geneeskunde Erasmus Universiteit Rotterdam
1994-1996	Artsexamen Erasmus Universiteit Rotterdam
1997-1999	Arts-assistent chirurgie St. Clara Ziekenhuis, Rotterdam Opleider: Dr. T.I Yo
2000-heden	Arts-assistent chirurgie Academisch Ziekenhuis Dijkzigt, Rotterdam Opleider: Dr. H.J. Bonjer

