

Committee 15

Anal Incontinence

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A. INTRODUCTION

Faecal incontinence has been a largely neglected topic in the world health care literature. Many patients with faecal incontinence become housebound because of the stigma associated with the condition. It is an embarrassing complaint that is socially disruptive.

I. DEFINITIONS

Faecal incontinence has been variously defined and there are no internationally accepted or accredited definitions available. The Royal College of Physicians has proposed “the involuntary or inappropriate passage of faeces” [1]. An international panel of experts has defined “functional faecal incontinence” as “recurrent uncontrolled passage of faecal material for at least one month, in an individual with a developmental age of at least four years...” [2]. Some authors also include inability to control passage of flatus, or an arbitrary frequency with which symptoms must occur to be included. The “objectively demonstrable” criteria specified by the International Continence Society for urinary incontinence is not practical in clinical practice for faecal incontinence. Therefore, clinicians must rely on patients’ self-report of symptoms.

This chapter will adopt the widely accepted distinction between “anal incontinence” denoting any loss of stool or flatus per anus and “faecal incontinence” as denoting any loss of solid or liquid stool.

The literature is confusing on the subject of definitions. The term “idiopathic incontinence” is often used to denote incontinence not due to trauma, congenital defects or neurological disease. “Neurogenic incontinence” in the colorectal literature often denotes faecal incontinence presumed to be secondary to damage to the pudendal nerve during childbirth, rather than that associated with major neurological disease. It is recom-

mended that this term should not be used since in principal it is identical with idiopathic incontinence and may be confused with incontinence due to neurological disease which is quite distinct. “Sensory incontinence” is not an entity but should be a description added to incontinence of any aetiology when the patient has no defecation urge at all and does not feel the passage of stool. “Motor incontinence” is an ill defined term which mainly covers incontinence in connection with diarrhoea and irritable bowel syndrome. It is recommended that this term should not be used.

The committee proposed the definition “**Anal incontinence is the involuntary loss of flatus, liquid or solid stool that is a social or hygienic problem**” as a working definition for this review. This definition takes cognisance of the fact that people react very differently to the same objective situation. For example, loss of flatus which is hardly noticed by one person is experienced as socially incapacitating by another. We recognise the need for, and would welcome, further debate and refinement of this definition.

II. SERVICES FOR PEOPLE WITH FAECAL INCONTINENCE

In many settings there is a lack of designated services for people with faecal incontinence. Locally, a service may be provided by a colorectal surgeon, gastroenterologist, specialist nurse (e.g. in stoma care, colorectal practice or biofeedback practitioner), but more often there is no interested professional and absent investigation facilities.

People with faecal incontinence are reluctant to seek health care for their problem [3, 4]. It is not clear the relative importance played in this by embarrassment and social taboos, lack of awareness that treatment is possible, lack of services, or a genuine unconcern at the symptom.

III. PROFESSIONAL EDUCATION AND PUBLIC AWARENESS

The majority of health professionals receive little or no training on faecal incontinence and even those who might be supposed to have a special interest, such as continence nurse specialists or colorectal surgeons, often focus on other areas. Public awareness of the symptom is very poor and there have been very limited attempts to alter this as yet.

B. EPIDEMIOLOGY AND THE INCIDENCE OF ANAL INCON- TINENCE; THE MAGNITUDE OF THE PROBLEM

Most discussions of the aetiology of anal incontinence (AI) have been based upon the assumption that women, particularly under the age of 65 years, are more at risk for AI than men. Obstetric injury to the pudendal nerve or sphincter muscle are described as the primary risk factors [5-7], irritable bowel syndrome as second [8] and other aetiologies such as diabetes as a distant third [9]. Yet each population based survey of AI prevalence has shown a surprisingly high prevalence in males [10-15]. Clearly other aetiologies than childbirth must be sought. In the most broadly based survey, it was apparent that factors that impact on an individual's general health or physical capabilities independently of age and gender place that individual at greater risk for anal incontinence than either age or gender [16].

Evidence in epidemiologic investigations comes principally from cohort and cross sectional surveys (level 2 – in which risk can be calculated and expressed as an odds ratio and 95% confidence intervals) in which the statistical significance of the associations can be assessed. Secondly risk factors arise from case series (level 4) and insightful observation (level 5). Systematic reviews of epidemiologic observations have for the most part been difficult to perform, since different studies usually adjust for differing variables and individual patient data are needed to overcome this difficulty. Therefore level 1 evidence does not exist for any risk factor related to faecal incontinence. In the tables below, when odds ratios and confidence intervals are presented it can therefore be assumed that that evidence is level 2 and when only lists of associations are presented, the level is most usually 4, occasionally 5.

I. PREVALENCE

Most reports of AI prevalence have been from single institutions and the patients described therein subject to

referral bias when demographics and aetiology are discussed. The accuracy of prevalence estimates of AI may also be diminished by difficulty in ascertainment. Underestimates of prevalence are common due to patients' reluctance to report symptoms of incontinence in strange settings or to seek support services [9, 17,4] (Table 1). It has been shown that women are more willing to report AI than men [16]. In addition, the character (incontinence to solid faeces, diarrhoea or flatus) and frequency (daily versus episodic) of AI varies greatly in each population. Prevalence depends on the definition of AI. The entry question quoted in one survey was written by a patient with faecal incontinence [16]. That individual regarded the inclusion of gas as critical. The same individual, and many others have said that one does not have to be incontinent all the time or only to solid stool to think that one is incontinent all the time and to be disabled by it.

Only seven population based estimates of prevalence in non-institutionalized individuals have been reported (Table 2). Prevalence has varied in these reports from 0.5% to 11%. The first report was restricted only to individuals over 65 years of age, both living at home and in institutions in New Zealand. The overall prevalence was 3.1%. FI was more prevalent in men, though further details of the population are not given [11]. The second, from Holland, was restricted to women over 60 years of age, and a non-representative sample of this group surveyed for prevalence by mail [12]. The prevalence varied with advancing age from 2.3% to 17.8%. These reports focused, as have most reports, on urinary incontinence. The third report was from Britain and compared prevalence obtained from a survey of community support services concerning individuals known to be incontinent in the region and a follow-up postal survey of all individuals living in the region. There was a marked disparity between those having sought help from community support services for faecal and double (urinary and faecal) incontinence and those reporting incontinence in the postal survey (Table 1). The difference was 8 fold for men below 65, four fold for women below 65 and roughly two fold for both genders over 65 years of age [10]. The total population prevalence of

Table 1 : Prevalence of anal or double incontinence in Middlessex, England [10]

	MEN 15-64 years	MEN > 64	WOMEN 15-64	WOMEN > 64
Community Services Reported	0.5%	4.9%	0.4%	8.8%
Postal Survey Reported	4.2%	10.9%	1.7%	13.3%

Table 2 : Anal incontinence; population based surveys

Country (ref)	Population	Prevalence
New Zealand [11]	> 65 years old	3.1%
Holland [12]	Women > 60 years	4.2% to 16.9 % with rising age
U.K. [10]	Community Service	1.9% (Table 1)
France [13]	All > 45 years	11%, 6% to faeces 60% women
U.S.A. [14]	Market mailing	7% soiling, 0.7% to faeces
U.S.A. [16]	Wisconsin Households	2.2%, 63% women
Australia [15]	Household survey	6.8% Men, 10.9% Women > 15 years old
U.S.A. [19]	Wisconsin Nursing Homes	47%

faecal or double incontinence of that British health region was 1.9% by survey of health service personnel and 4.3% by postal survey.

From France, a prevalence study has been reported limited to individuals over the age of 45 years [13]. FI prevalence was determined in a "Gallup poll" style telephone interview, as well as in the practices of several medical specialists. In the population based survey, an overall prevalence of 11% was reported, roughly half to gas only and half to solid or liquid stool. 60% were women and prevalence was increased independently of gender if subjects had urinary incontinence or neurological disorders. FI prevalence increased with age, with disability resulting in bed confinement, with dementia and with nursing home residence. From the United States, FI prevalence was determined through the addition of questions concerning functional bowel disorders to a commercial marketing survey mailing [14]. Because the means by which the addressees were chosen was proprietary information and was not made available to the investigators, there was great potential for selection bias in this population that cannot be accurately assessed. (Marketers may be more interested in "buyers" than obtaining a group representative of the general population). Response to the survey was also subject to many factors that affect the generalisability of the results. The survey encompassed 5430 respondents, which was 66% of the mailing. Faecal soiling was present in 7.4% of the males in the respondents and 6.9% of the females, with incontinence to solid stool in 0.5% of the males and 0.9% of the females.

The population of the State of Wisconsin was sampled in the 1993 Wisconsin Family Health Survey [16]. Subjects were identified by random digit dialing of Wisconsin residences with telephone interview. The individual within each household identified as most knowledgeable about the health status of all other members of the household was asked about the health status of each member of the household. Approximately 200 households were surveyed each month. The presence of anal incontinence to solid, liquid or gas within the past year, who suffered from it, the frequency of incontinence, and how that individual coped with it were assessed.

2570 Households comprising 6959 individuals were surveyed. 153 individuals were reported to have AI, representing 2.2% of the population (95% confidence interval (CI) = + 0.3%). 30% of the incontinent subjects were greater than 65 years of age; 63% were women. Of those with AI, 36% were incontinent to solid faeces, 54% to liquid and 60% to gas. In a multivariate analysis, independent associations of the following risk factors with AI were found in increasing order of importance: female gender, age, physical limitations and poor general health (Table 3).

From Australia, a population based household survey was reported encompassing 3010 interviews with individuals ranging in age from 15 to 97 years [15]. In face to face interviews, the prevalence of incontinence to either flatus or solid stool was found to be 6.8% and 2.3% in men, and 10.9% and 3.5% in women respectively. The male to female ratio is also very similar to the Wisconsin report, being 64% women for flatus incontinence and 60% for incontinence to solid stool.

II. ASSOCIATIONS

The most prominent association with faecal incontinence by far is nursing home residence. Whereas the prevalence of faecal (not just anal) incontinence is probably around 2% to 3%, and may rise in age with community dwelling individuals to greater than 10%, among nursing home residents the prevalence approaches 50% [19, 20]. Indeed it is one of the most common reasons for nursing home admission. In a survey of residents of Wisconsin nursing homes, risk factors for faecal incontinence, as directly observed by nursing home personnel, were sought [19]. Surprisingly, in this very old population (mean age 84 years), neither age, gender nor diabetes were found to be associated with FI. Positive associations included most prominently the loss of ability to perform the activities of daily living, followed by tube feedings, restraints, diarrhea, dementia, impaired vision, constipation and faecal impaction. Inverse associations were noted with body

Table 3 : Adjusted odds ratios for anal incontinence risk factors [16] (Level 2 evidence)

Risk Factor	Adjusted odds ratio*	95% Confidence interval
AGE @	1.01	1.01 - 1.02
FEMALE GENDER	1.51	1.10 - 2.11
PHYSICAL LIMITATIONS	1.82	1.20 - 2.74
POOR GENERAL HEALTH @	1.64	1.42 - 1.91

* Each factor adjusted for all other factors in the Table @ Age and health (using all five strata from excellent to poor) are continuous variables. The others are dichotomous.

weight, heart disease, arthritis and surprisingly, depression. This was a large survey encompassing over 18,000 residents (Table 4).

Pregnancy, though not the exclusive cause of anal incontinence, is certainly a prominent association [5-7]. Factors leading to incontinence both during [21] pregnancy, immediately after [15, 22, 23] and long after pregnancy [24] have been investigated. Irritable bowel syndrome has been an important correlate with post partum anal incontinence [23] (Table 5). Quantitative assessment of risk related to pregnancy, and various methods of delivery, has only recently been feasible in data presented from an Australian survey [15]. It is most interesting to note how similar risk is comparing caesarean section and vaginal delivery. However, these odds ratios are not adjusted for age or parity and it is not known which caesarean sections were done as an emergency versus electively (Table 6).

Several specific diseases have been associated with AI in case series and mechanisms investigated to explain the associations [25]. These include diabetes, multiple sclerosis, Parkinson's disease, spinal cord injury, systemic sclerosis, myotonic dystrophy and amyloidosis (Table 7). Many of these conditions directly affect a patient's mobility, their ability to perform activities of daily living, or cause diarrhoea or faecal impaction. In addition children with congenital anal anomalies, such as imperforate anus, despite anatomic correction of their deformity, often have life long problems with incomplete evacuation and soiling. A similar group are children born without anomalies, but who for a variety of reasons withhold stool at a point beyond which toilet training should be complete and develop faecal soiling and a megarectum (see section 8, below). Failure to retrain the child at an early age often leads to chronic impaction and AI.

The importance of diarrhoea or liquid stool in AI can-

Table 4 : Associations with faecal incontinence in wisconsin nursing homes. minimum data set reports from 1992 and 1993. Odds ratios and 95% confidence intervals; each variable adjusted for all other variables in the table

ASSOCIATION	1992	1993
Urinary Incontinence	12.6; 11.5-13.7	11.3; 10.3-12.4
Tube Feeding	7.6; 5.6-10.4	8.8; 6.3-12.3
Loss of ADLs	6.0; 4.7-7.7	7.3; 5.5-9.7
Diarrhea	3.3; 2.7-4.2	2.4; 1.9-3.1
Trunk Restraints	3.2; 4.7-7.7	3.0; 2.7-9.8
Pressure Ulcer	2.6; 2.2-3.0	2.3; 2.0-2.6
Dementia	1.5; 1.4-1.7	1.4; 1.3-1.5
Impaired Vision	1.5; 1.4-1.7	1.4; 1.3-1.5
Faecal Impaction	1.5; 1.1-2.1	2.1; 1.3-3.3
Constipation	1.4; 1.3-1.6	1.3; 1.2-1.4
Stroke	1.3; 1.2-1.5	1.2; 1.1-1.3
Male Gender	1.2; 1.1-1.3	1.3; 1.1-1.4
Non-white Race	NS	1.3; 1.0-1.7
Age	1.0; 1.0-1.01	1.0; 1.0-1.01
Body Mass Index	1.0; 1.0-1.3	1.0; 1.0-1.04
Diabetes	NS	NS
Heart Disease	0.9; 0.8-1.0	NS
Arthritis	0.9; 0.8-1.0	0.8; 0.7-0.9
Depression	NS	0.9; 0.8-1.0

2b evidence

ADLs; Activities of Daily Living

NS; Not statistically significant

Age and Body Mass Index are adjusted as continuous variables.

not be overemphasized. One case series noted that 51% of individuals with chronic diarrhoea were incontinent [4]. In the Wisconsin Family Health Survey of AI, 41% of the 25 subjects with AI disclosed in April and May of 1993 lived in Milwaukee [16]. At this specific time there was a water-borne outbreak of cryptosporidium diarrhea in southern Milwaukee [26], reportedly the largest outbreak of water born disease in U.S. history. This is an important reminder that infectious sources of incontinence should be part of the diagnostic evaluation of AI when diarrhoea is present. Non-infectious causes of diarrhoea must also be considered, including those initiated by leisure activities such as running [27]. Additional aetiologies for anal incontinence that have been described include stroke [28] and hospitalisation for acute illness [29].

The surgeon is often concerned that he might be the originator of a patient's AI. On a population wide basis,

Table 5 : Events related to anal incontinence

Running
Pregnancy (That is, during pregnancy)
Vaginal Childbirth; Obstructed Labor;
Rectovaginal Fistula
Hospitalisation For An Acute Illness
Advancing Age
Tube Feedings
Patient Restraints
Faecal Impaction

Running and Hospitalisation are type 4 evidence risk factors. The others are all type 2.

Table 6 : The association of pregnancy and the method of delivery with incontinence to faeces and flatus. Unadjusted Odds Ratios and 95% Confidence Intervals; (level 2 evidence) [15]

Comparison	Faecal Incontinence	Flatus Incontinence
Vaginal Delivery vs. Nulliparous	2.93; 1.23 – 7.33	2.59; 1.58 – 4.28
Instrumented Delivery vs. Nullip.	2.46; 0.87 – 7.12	3.37; 1.93 – 5.91
Cesarean Section vs. Nulliparous	2.54; 0.61 – 9.88	1.76; 0.73 – 4.16
Vaginal Delivery vs. C Section	1.16; 0.38 – 3.94	1.47; 0.69 – 3.23

Table 7 : Diseases associated with anal incontinence (Level 4 Evidence)

Diabetes
Stroke
Multiple sclerosis
Parkinson's disease
Systemic sclerosis
Myotonic dystrophy
Amyloidosis
Spinal cord injury
Imperforate anus
Hirschsprung's disease
Retarded or interrupted toilet training
Procidentia
Any illness causing diarrhea (HIV, Inflammatory Bowel Disease, radiation, infection)
Irritable bowel syndrome

this would seem a fairly insignificant factor, since prior anal surgery has not been an apparent risk factor in the larger surveys. Yet much of a colorectal surgeon's training is directed towards avoidance of this disabling complication. Nevertheless, several operations performed frequently can result in AI (Table 8). The first of these is lateral internal sphincterotomy for fissure in ano. The risk of this procedure causing AI was thought to be insignificant when compared to midline sphincterotomy (which is the operation done most frequently for fistula in ano). A recent reappraisal of the outcome of this operation has shown an AI risk as high as 8% [30]. Similarly fistulotomy was thought to have a negligible risk of AI when compared to fistulectomy. However the risk of AI after fistulotomy has been reported to be as high as 18-52% [31]. New approaches to fissure and fistula have been recently developed specifically to lower this risk [31, 32]. Ileo-anal pouch reconstruction has enabled individuals afflicted with inflammatory bowel disease to live without a stoma, but at high risk of AI. A more proximal anal anastomosis is now commonly done in hopes of diminishing this risk. There is controversy as to whether a pouch should also be made for patients having an ileorectal anastomosis. Low anterior rectal resection has also made it possible for patients with mid-rectal cancer to avoid a permanent stoma, but the functional results, even in the absence of prior radiation, may be poor and new procedures have also been described to deal with this [33]. Lastly, mixing urine and stool has been found to have an adverse effect on anal sphincter control in patients having ureterosigmoidostomy after urinary bladder resection [34].

The development of incontinence in previously continent nursing home residents has also been studied. Significant associations have been found, as would be expected, with dementia, stroke and blindness. However the most prominent association is with the use of patient restraints, even when adjustment has been made for factors that might be associated with restraint, such as immobility and dementia (Table 9) (Nelson; submitted).

III. SUMMARY

Anal and urinary incontinence commonly co-exist, particularly in the elderly [2].

The prevalence of anal incontinence increases with age, but is present in all age groups and both genders varying from 1.5% in children to over 50% in nursing home residents [2].

Though pregnancy is a common association with anal incontinence, in younger and older populations, AI is more common in men [2].

As populations age, comorbid disease becomes a significant component of anal incontinence risk [2].

Table 8 : Operations associated with risk of anal incontinence . Level 4 Evidence

Midline internal sphincterotomy
Lateral internal sphincterotomy
Fistulectomy
Fistulotomy
Ileo-anal reservoir reconstruction
Low anterior rectal resection
Total abdominal colectomy
Ureterosigmoidostomy

IV. RECOMMENDATIONS

Epidemiologic investigations of AI and urinary incontinence should be performed jointly. (D)

The use of truncal restraints in nursing homes should be re-assessed. (D)

Table 9 : Adjusted Odds Ratios* and 95% Confidence Limits Associated With Risk Factors for the Development of Incontinence of Urine, Faeces, and Both Urine and Faeces from 1992 to 1993 in Wisconsin Nursing Home Residents (Level 2 Evidence)

	URINE Incontinent of bladder=940, Not incontinent of bladder=3054,	FAECES Incontinent of bowel=609, Not incontinent of bowel=3386	BOTH Incontinent of both=470, Not incontinent of both =3535
Trunk restraints	2.4 (1.7-3.3)	2.8 (2.0-4.0)	2.4 (1.7-3.6)
Dementia	1.5 (1.3-1.8)	1.6 (1.3-1.9)	1.7 (1.4-2.1)
Impaired vision	NS	NS	NS
Stroke	1.2 (1.0-1.5)	NS	NS
Constipation	NS	NS	NS
Heart disease	NS	NS	NS
Arthritis	0.8 (0.7-1.0)	0.8 (0.6-1.0)	0.7 (0.6-0.9)
Diabetes	NS	NS	NS
Faecal impaction	NS	NS	NS
Race (NW = 1, W = 0)	NS	2.1 (1.3-3.5)	1.8 (1.0-3.0)
Age	1.03 (1.02-1.04)	1.02 (1.01-1.03)	1.02 (1.01-1.04)
Body mass index	NS	0.98 (0.97-1.0)	0.98 (0.96-1.00)
Depression	NS	NS	NS
Diarrhea	NS	NS	NS
Male gender	0.8 (0.6-0.9)	NS	NS
Pressure ulcer	NS	NS	NS
Tube feeding	NS	NS	NS

*Adjusted odds ratios were derived from multivariable logistic regression models with all risk factors included.

C. BOWEL SYMPTOM QUESTIONNAIRES, INCONTINENCE GRADING AND SCORING AND QUALITY OF LIFE STUDIES

In order to better understand anal incontinence as a medical entity, reliable and validated questionnaires and instruments are essential. Although it is considered fundamental by experts in the field of outcomes research that a self-administered questionnaire or other instrument must be psychometrically tested for validity and reliability to have scientific merit or value, this is often overlooked in the published literature. Not uncommonly, bowel symptom questionnaires, anal incontinence grading systems and quality of life scoring have relied on individual clinician's opinions and their subjective collection of only certain data elements that they felt salient. Uncontrolled studies using non-validated instruments to assess populations, measure outcomes and draw conclusions often prevent objective, scientific comparisons within and between many of the studies in the literature.

I. BOWEL SYMPTOM QUESTIONNAIRES

Due to inherent problems in investigating certain aspects of anal incontinence due to the attached social

stigma [4], detailed, objective and pointed questioning is often necessary to delineate symptoms in patients with this condition.

There is a large amount of information that may be obtained and recorded on a suitably constructed bowel symptom questionnaire depending on the particular goals of the researcher in anal incontinence (Table 10). Accordingly, the material covered in the questionnaire may vary with the goal of developing the instrument. The goal of a self-report bowel symptom questionnaire may be multiple including:

1. measuring outcomes including quality of life from medical and surgical interventions
2. assessing individual symptoms and grading the severity of the problem
3. screening for the condition to determine a prevalence or incidence of the condition
4. providing a forum or template from which to begin physician-patient interactions
5. providing an organised basis for recording symptoms in order to better understand the underlying pathophysiology, facilitate diagnosis or improve the management of the condition
6. helping to identify patterns of disease or identifying risk factors.

Bowel symptom questionnaires, although not widely utilised clinically, have been developed and validated for a number of conditions including irritable bowel syndrome [36-39], and Crohn's disease [40-42]. These

Table 10 : Potential data recorded on a bowel symptom questionnaire*

Chief complaint
Bowel pattern Sensation of the urge to defaecate, number of movements per day, consistency: loose, soft, hard, hard pellets, faecal urgency or ability to defer defaecation , evacuation pattern: straining, anal or vaginal digitation
Continence of flatus
Presence of passive soiling
Pain, tenesmus, etc.
Presence of blood or mucus
Sensations of incomplete emptying, or prolapse
Quantification of pad or incontinence pant use
Fluid intake
Toileting access
Past medical/surgical/obstetric history, co-morbid conditions
Medications
Associated risk factors such as diet, smoking, and body weight
Associated symptoms of bladder control
Skin problems due to local irritation
Quality of life assessment

*Adapted from: Norton & Chelvanayagam [35].

questionnaires were developed as general instruments for gastrointestinal disorders, and not specifically for investigating anal incontinence.

Very few anal incontinence specific self-administration questionnaires have been developed and validated. A 38-item questionnaire was tested in a small series of normal controls, patients with anal incontinence and patients with constipation [43]. This instrument performed well in the original patient population but has not been widely adapted or validated by others in the field.

Based on prior work assessing general gastrointestinal symptoms, another anal incontinence specific self-administered questionnaire, the Faecal Incontinence Questionnaire (FIQ), has been developed and validated. This instrument was designed to measure the prevalence of anal incontinence in the community and assess risk factors associated with the condition. When compared to the AUA Symptom Score, it performed favourably [44]. This instrument has subsequently been used to measure the prevalence of anal incontinence in selected populations including those with combined urinary and anal incontinence [45, 46]. Whether this instrument will be widely adopted for clinical and research purposes is unclear.

1. SUMMARY

Objective and complete symptom assessment of anal incontinence is essential.

The information contained on a bowel symptom questionnaire may vary with the goal of the questionnaire both clinically and as a research tool.

At present, there are no widely utilised, validated anal incontinence specific bowel symptom questionnaires.

The FIQ, although lengthy may be a useful instrument in the initial assessment of the patient with AI.

2. RECOMMENDATIONS

- Future work should determine whether the Faecal Incontinence Questionnaire or other subsequently developed bowel symptom questionnaires are applicable, reliable and appropriately validated across all age groups, all languages, as well as all conditions including benign, malignant, acquired and congenital conditions resulting in anal incontinence.

II. ANAL INCONTINENCE GRADING AND SCORING

An anal incontinence scoring system is useful, not only for epidemiological purposes, but also in order to objectively grade the severity of anal incontinence as well as provide a basis for the comparison of outcomes

of both surgical and non-surgical therapies for the condition. A universally agreed upon standard scoring/grading system does not exist.

The ideal scoring system should be reproducible, simple to use and include parameters such as frequency, quantity, and type of incontinence (solid, liquid or gas), descriptions of the circumstances under which anal incontinence occurs (e.g.: passive/active, awareness of urgency, etc.), quantify the use of adjunctive measures such as pads or plugs in an effort to control or manage the condition, as well as assess the effects of anal incontinence on quality of life (occupational, social, etc.). To some degree, classification of incontinence is linked to grading and scoring systems as authors have often devised their own systems in an effort to describe baseline patient symptomatology before and after interventions. These classification/grading/scoring systems are numerous and diverse. Many are not validated and have been used only by the author who devised the system. Therefore reproducibility across and between surgeons, patients, procedures and treatments remains unknown.

Many of the grading/scoring systems recorded in the current literature suffer from a variety of shortcomings including: a lack of objectivity, being primarily descriptive in nature [47, 48] mixing subjective and objective parameters [49], using objective parameters such as anal manometry [50, 51] or difficult to classify subjective parameters [52] which often do not correlate with clinical conditions, or do not account for frequency of the incontinent episodes in individual patients [53-56].

Several anal incontinence grading or scoring systems have been prospectively developed and tested. The Faecal Incontinence Severity Index (FISI) was developed and evaluated as a questionnaire for assessing the severity of anal incontinence [57]. Using a 20 cell matrix table, the researchers constructed the FISI by looking at both type (gas, mucus, liquid and solid) and frequency (5 categories) of incontinence episodes. This generated a graded numerical result. The FISI was administered to both physicians and patients for weighting and scoring, with surgeons and patients responses of severity correlating very well.

Perhaps one of the most widely used scales or grading systems is the Wexner score [59]. This was the first system to account for the use of pads, changes or alterations to lifestyle, consistency and frequency of incontinence. The Wexner Score is derived from numerical values assigned to the frequency of occurrence (scored 0-4) in each of several categories including type of incontinence (solid, liquid, gas), pad use, and lifestyle alteration. A minimum score of 0 indicates perfect continence, and a maximum score of 20 indicates complete incontinence (Table 11).

Table 11 : The Wexner score [59]

Type of incontinence	Frequency				
	Never	Rarely	Some-times	Usually	Always
Solid	0	1	2	3	4
Liquid	0	1	2	3	4
Gas	0	1	2	3	4
Wears Pad	0	1	2	3	4
Lifestyle Alteration	0	1	2	3	4

Never: 0 Rarely: <1month

Sometimes: <1/week, ≥1/month Usually: <1/day, ≥1/week

Always: ≥1/day 0= perfect continence

20=complete incontinence

A modified version of the Wexner score was compared with the original version, a 28 day diary and several other scoring systems in 23 patients and then the results were correlated with an independent clinical assessment by two clinicians [60]. Ten patients were assessed before and after surgery.

The newly devised scale showed the best correlation although all the scales performed well. The modified scale included details regarding urgency, and the use of anti-diarrhoeal medications. Categories scored 0-4 (0=never, to 4=daily) included type of incontinence (solid, liquid, gas), and alteration in lifestyle. Scored separately were the need to wear a pad or plug (0=no, 2=yes), taking anti-diarrhoeal medications (0=no, 2=yes) and lack of ability to defer defecation (0=no, 4=yes). The maximum score in the modified Wexner scale was 24 implying complete incontinence.

1. SUMMARY

Many of the existing grading/scoring scales are empirically derived, and are not prospectively validated.

There are no widely utilised, validated grading/scoring systems for AI.

2. RECOMMENDATIONS

- Faecal incontinence grading and scoring are an important part of the initial assessment of patients with AI. (D)
- Future work should be directed towards development, validation and widespread dissemination of a standardised faecal incontinence grading/scoring system.
- Interventional studies for anal incontinence should utilise a validated grading/scoring system at baseline and following intervention. (D)

III. QUALITY OF LIFE

Quality of Life (QOL) instruments are designed to measure the subjective perception of a given patient's health state including the emotional and social impact of a medical condition. When applied appropriately, this is a reliable outcome measure that can be objectively and scientifically quantified. The optimal methodology by which QOL is measured has been constantly evolving and is not yet ideal [61, 62]. Although the severity of a given condition may correlate with quality of life in some patients, QOL instruments measure different aspects of the health/disease continuum and should not be considered an indicator of disease severity. Both generic and disease-specific QOL instruments have been developed.

Several authors have utilised well-validated generic QOL instruments such as the SF-36 or MOS-36 [63, 64] to study anal incontinence in certain populations [65-67]. Numerous studies in the colorectal and anal incontinence literature have utilised some form of a QOL scale as an outcome measure, but these are for the most part unvalidated instruments. Disease specific QOL instruments have been developed for several gastrointestinal conditions including inflammatory bowel disease [68, 69] and are generally felt to have good reliability and validity [62]. However, with one exception, good quality disease specific QOL instruments for anal incontinence do not yet exist. The Faecal Incontinence Quality of Life Scale (FIQLS) is, however, a QOL instrument developed, tested and psychometrically evaluated for the assessment of patients with anal incontinence [70]. This instrument is a self-administered questionnaire containing 29 items covering 4 scales: Lifestyle, Coping/Behaviour, Depression/Self-perception and Embarrassment. Each of the 29 items are scored 1-5 and a mean score is obtained within each of the 4 scales. Due to unresolved issues regarding potential differential weighting of the 4 scales, it is unclear whether an overall score can be obtained by simply summing the 4 mean scores. In a study of 190 patients, the FIQLS discriminated well between those with anal incontinence and those with other gastrointestinal conditions and also correlated well with the MOS-36 [70]. As the process of validation continues, refinement of the weighting between the four scales are necessary to arrive at an overall score, as is further testing of this instrument in an interventional setting and in different countries, languages and cultures [71].

1. SUMMARY

Patients with AI have a diminished QOL as measured by general QOL instruments [4].

The FIQLS may be a promising disease specific QOL instrument to study those with anal incontinence [4].

2. RECOMMENDATIONS

- A validated, disease specific QOL instrument is essential in the study of anal incontinence. (D)
- General and disease specific QOL instruments should be an integral part of the initial assessment of patients with AI and should be a standardised outcome measure when evaluating interventions. (D)
- Future work should be directed towards development, validation and widespread dissemination of a standardised disease specific QOL instrument. (D)

D. PATHOPHYSIOLOGY AND INVESTIGATION OF FAECAL INCONTINENCE IN ADULTS

I. INTRODUCTION

Normal defecation and faecal continence are the result of a complex interplay among a number of factors. Colonic transit, consistency and volume of stool, rectal compliance and capacity, anal sphincter function and anorectal sensation, and reflexes all play an important role. Faecal incontinence may be the result of abnormalities in any one or more of these functions. Progressive diminution loss of anal sphincter function occurs [72]. Eventually, there may be incontinence to solid stool. The purpose of this section is to describe the investigation of faecal incontinence with particular reference to the value of physiologic tests.

II. FAECAL CONTINENCE

Faecal continence is achieved by a combination of a competent, closed anal sphincter; normal anorectal sensation and sampling reflex; adequate rectal capacity and compliance; and conscious control. Continence is maintained as long as anal canal pressure is greater than rectal pressure [73]. The rectum is supported by the pelvic floor muscles, and the anal sphincter is augmented by the acute rectoanal angle maintained by the sling effect of the puborectal muscle. Reflexes quickly increase muscular activity in response to changes in intra-abdominal pressure to augment mechanical barriers to the aboral movement of rectal content.

Maintenance of faecal continence at times when the anal sphincter is under maximal strain, such as when the rectum is filled with liquid, is associated with marked electrical activity in the anal sphincter complex and puborectal muscle. Pressures measured within the rectal lumen are consistently lower than those recorded in the anal canal as long as continence is preserved. Support for this hypothesis comes from patients in whom a successful postanal repair has been carried out. This operation was originally described for faecal incontinence in the belief that correction of an obtuse anorectal angle would restore faecal continence. A successful operation is associated not with correction of the angle, which often remains unchanged, but rather with a rise in anal sphincter resting and squeeze pressures [74].

The significance of the external anal sphincter in maintaining continence is illustrated by the finding of faecal seepage or soiling in 50% of patients who have a simple defect in the muscle after obstetric tears [75].

The flap valve theory of faecal continence suggests that when intra-abdominal pressure is applied to the rectum, the anterior rectal wall is pressed down onto the upper anal canal and thereby prevents rectal content from gaining contact with the upper anal canal [76]. The anterior rectal wall acts as a plug. In support of this theory is the regular association of an obtuse anorectal angle, and presumably the inability of the anterior rectal wall to close off the upper anal canal, with faecal incontinence. In studies in which the anal sphincter was maximally stressed and the rectum visualised radiographically, however, no contact between rectal wall and anal canal was seen [77]. It is likely that the flap valve effect is effective only when the rectum is less distended. The relevance of the puborectal muscle to continence is illustrated by the high degree of continence in children with congenital absence of internal and external anal sphincters [78] and by the high incidence of incontinence in patients with constipation in whom division of the puborectal muscle has been carried out to relieve outlet obstruction [79].

The flutter valve theory suggests that intra-abdominal pressure applied to a high pressure zone within the lower rectum leads to the occlusion of this area and thereby prevents any rectal content from reaching the anal canal [80]. According to this theory, the anal sphincter serves to provide fine control. The idea is analogous to that observed about the lower oesophageal sphincter, in which a flutter valve mechanism has been proposed to explain control of gastroesophageal reflux. Manometric studies clearly demonstrate, however, that the high pressure zone lies in the mid-anal canal and that no such higher pressure zone is demonstrable in the lower rectum either in static studies or in ambulatory subjects [81].

1. ROLE OF RECTAL COMPLIANCE IN MAINTAINING FAECAL CONTINENCE

The anorectum acts as a functional unit. The first sensation volume of rectal filling varies from 11 to 68 mL in published studies, whereas the mean maximum tolerated volume ranges from 220 to 510 mL [82]. Mean rectal compliance also shows a wide range (4 to 14 mL/cm H₂O). The compliance, and hence the capacitance, of the rectum is integral to this function. Reflex adaptation of the rectum in response to filling occurs in a manner analogous to that of the bladder. The rectum tolerates volumes up to about 300 mL without showing any marked change in intraluminal pressure. Beyond this, pressure waves become more pronounced until tolerance is approached and the feeling of urgency to defaecate develops [83]. Rectal compliance is reduced in patients in the presence of diseases such as Crohn's disease or radiation proctitis. Low compliance leads to increased frequency of bowel actions, urgency of defecation, and often faecal incontinence despite a normal pressure profile of the anal sphincter. In such cases, removal of the diseased rectum and replacement with a compliant neorectum, either an ileal pouch or healthy colon may restore continence [84, 85].

Contractility of the rectal wall increases in response to a meal [86]. The rectum is usually empty [87]. The main reservoir for faeces is located more proximally, and the transverse colon appears to be the principal region for storage and mixing of enteric contents [88, 89]. Solids and liquids are stored equally in the transverse colon [88]. Transit of ¹¹¹indium pellets through unprepared colon shows that although the ascending and transverse colon are sites of storage of solid residue, the descending and rectosigmoid act mainly as conduits [90]. If stool is emptied into the rectum from the sigmoid colon when the tone of the rectum is increased, a more marked increase in intraluminal rectal pressure occurs. This leads to a prompt urge to defecate. In constipated patients, rectal contractility is blunted such that the sensation of rectal filling appears to be less pronounced, and the usual prompt desire to defecate is not appreciated [91].

III. MECHANISM OF DEFAECATION

Events leading to defecation start when the volume of content in the sigmoid colon becomes large enough to initiate contractions, which empty the stool into the rectum. Conscious awareness of a full rectum is brought about by receptors in the rectal wall, pelvic floor, and upper anal canal by way of the rectoanal inhibitory reflex. Rectal filling is recognised at a volume of about 50mL. If the rectum continues to fill, a temporary sen-

sation of an urge to defecate is elicited, followed by a constant urge and eventually pelvic discomfort—the maximal tolerable volume. The puborectal muscle is stretched by rectal pressure waves, and the amplitude of these waves increases [92] as the rectum distends. The urge to defecate is probably mediated by stretch receptors in the pelvic floor because tugging a small balloon against the puborectal muscle produces this feeling [93]. The precise influences of the rate of rectal filling and the nature of its content on conscious awareness are unknown. Distension of the rectum leads to relaxation of the internal anal sphincter and sampling of the contents. The external sphincter contracts. If it is socially convenient, the pressure within the rectum must be made to exceed that of the anal canal. This is largely achieved by increasing the intra-abdominal pressure by contraction of the diaphragm, abdominal muscles, and performance of the Valsalva manoeuvre.

Relaxation of the puborectal muscle allows the anorectal angle to become less acute, increasing from a mean of 92 degrees to a mean of 111 to 137 degrees [94], and this is helped if the individual adopts the squatting position, which increases the anorectal angle more than that achieved by flexion of the hips to 90 degrees [95]. Inhibition of pelvic floor activity allows descent of the pelvic floor by about 2 cm, which further straightens out this angle [96]. As the rectum evacuates, there is prolonged inhibition of the internal anal sphincter and external anal sphincter, and anal pressure falls. Whether physiologically important contractions of the rectal wall occur during defecation is unknown. Conversely, if it is not socially convenient, brief conscious contraction of the external sphincter allows the internal sphincter to recover and the rectal contents are propelled in an oral direction by forceful contraction of the puborectal muscle and the rest of the pelvic floor musculature. Internal anal sphincter tone then returns to normal and the urge to defaecate becomes less imperative.

Defaecation is influenced by the size and consistency of the stool. The time to expel a single solid sphere is inversely proportional to its diameter [97]. More effort is required to evacuate small, hard stools, and the ideal stool diameter is about 2 cm.

Straining at defaecation is associated with the co-contraction of laryngeal, respiratory, and abdominal muscles and the inhibition of anal sphincter muscles, and this patterned recruitment is closely linked to the extrapyramidal system. Paradoxical contraction of the puborectal muscle and the external anal sphincter during defecation is seen in patients with Parkinson's disease [98]. Higher control is needed for appropriate defaecatory responses as illustrated by patients with damage to the frontal lobes [99]. Defaecation occurs without any warning in these patients.

IV. CAUSES OF FAECAL INCONTINENCE

The cause of major incontinence, the total loss of control of solid stool, is poorly understood. Progressive loss of anal sphincter function occurs with no definite time of onset of symptoms. Initially, loss of control of flatus occurs followed by seepage of faecal material onto undergarments and then incontinence to solid stool. Finally, the call to stool is also lost.

Two types of history are typical in colorectal practice. Patients may have a history of prolonged or difficult childbirth with vaginal delivery, with occult sphincter damage [100] or there may be a history of several years' duration of forceful straining at defecation. Incontinence may be passive, in which there is leakage of faeces without the patient being aware, or motor, in which the patient is aware of an urge to defaecate but is unable to prevent the passage of faeces.

1. DYSFUNCTION OF THE INTERNAL ANAL SPHINCTER

Resting anal pressure is usually reduced in patients with faecal incontinence, often to a level similar to that recorded in the rectal lumen. Electrical activity in the internal sphincter as measured by electromyography (EMG) is reduced. Marked descent of the pelvic floor may be apparent, and this laxity may cause disruption of the sympathetic innervation. This denervation is evidenced by atrophy, fibrosis, and increased fibre density. Ultrastructural changes include disruption and loss of smooth muscle cells, increased deposition of collagen, and stretching of elastic tissue [101]. The internal sphincter may relax inappropriately in response to relatively low amounts of rectal distension. Continence then depends on external sphincter activity [102].

Internal sphincter relaxation in response to rectal distension occurs more frequently, is more pronounced, and lasts longer in incontinent patients compared with controls [103]. A subgroup of patients may show not only anal relaxation, but also failure of external sphincter contraction at the time of rectal distention [104]. Endoanal ultrasound has demonstrated that thinning of the internal anal sphincter occurs in patients with idiopathic faecal incontinence, and this correlates with reduced resting anal sphincter pressure [105]. Furthermore, the adrenergic response of the internal sphincter may be impaired [106]. The clinical significance of isolated internal sphincter dysfunction is unknown.

2. DYSFUNCTION OF THE EXTERNAL ANAL SPHINCTER AND PUBORECTAL MUSCLE

Persistent repeated traction on the pudendal nerve leads

to denervation and weakness of the external anal sphincter. There is loss of myelinated and demyelinated nerves and fibrous replacement [107]. Chronic partial denervation of the striated nerves of the pelvic floor is also associated with prolonged vaginal delivery often associated with delivery of a large infant or use of forceps. This situation may result in prolonged stretch being applied to the pudendal nerve or traumatic injury to the sacral nerves by the foetal head. EMG studies before and after breech or forceps deliveries have shown increased fibre density in the external anal sphincter and prolonged pudendal nerve latency times [108]. Interestingly, while vaginal deliveries may result in cumulative injury, women who have undergone caesarean section also show evidence of denervation of the pelvic floor [109]. Partial denervation injury may occur with repeated straining, with denervation secondary to abnormal perineal descent. The pudendal nerve is stretched from its point of fixity in Alcock's canal. Anal sensation is also impaired in this group of patients [110].

3. IMPAIRED RECTAL SENSATION AND COMPLIANCE

Repeated deferral or ignoring the call to stool may lead to faecal impaction. This is a common cause of overflow incontinence in elderly people. Impaired anorectal sensation may be associated with an abnormal response of the external anal sphincter to rectal distension and hence incontinence. Recruitment of external anal muscle activity is reduced [111].

The rectum, if damaged by radiation or Crohn's disease, becomes less compliant and may develop increased rectal pressures. Accommodation of stool is therefore reduced, and patients may complain of symptoms of urgency of defaecation and faecal incontinence.

V. INVESTIGATIONS

1. HISTORY AND CLINICAL EXAMINATION

A careful, thorough history and full physical examination are essential and will identify the majority of causes of faecal incontinence. There may be a history of prolonged or difficult childbirth. A long history of forceful straining at defecation suggests denervation of the pelvic floor. Incontinence may be sensory in which there is leakage of faeces without the patient being aware or motor with patients being aware but unable to prevent the passage of faeces, or a mixture of both. True faecal incontinence must be differentiated from conditions that cause seepage such as grade 3 or 4 haemorrhoids, fistulas, low rectal or anal tumours, and poor perineal hygiene. Diagnostic administration of an enema may be useful in this respect; retention of the enema suggests that the patient does not have clinically

significant faecal incontinence [112].

Severity of faecal incontinence can be classified as a minor if faecal seepage occurs less than once a month, moderate if there is incontinence to solids more than once a month or liquids more than once a week, and severe if there is loss of control of solids several times a week or liquids on a daily basis. An alternative classification grades continence as follows [113]:

Grade 1: Complete

Grade 2: Incontinence of flatus

Grade 3: Incontinence of flatus and liquid stools

Grade 4: Incontinence of flatus, liquid stools, and solid stools.

Physical examination should include inspection of underclothing for soiling and staining by stool, pus, or mucus. Perianal skin should be examined for irritation, fistulous tracks, and hygiene. Scars from previous episiotomies or obstetric tears should be noted. Abnormalities at the anal verge from previous surgery or a gaping anus suggestive of marked loss of function may be present. Digital rectal examination includes assessment of the tone of the anal sphincter (at rest and squeezing) and identification of faecal impaction or a rectal mass. Function of the puborectalis muscle (palpable at the anorectal junction) is assessed by asking the patient to squeeze the sphincter at which time the puborectalis should push the examiner's finger anteriorly. Anoscopy and rigid proctoscopy should be performed to exclude anorectal pathology. Procto-sigmoidoscopy and/or colonoscopy should be performed to exclude anorectal pathology such inflammatory bowel disease, neoplasm or radiation changes. Patients with complete rectal prolapse (Procidentia) often complain of faecal incontinence – examination of the patient while straining on the toilet facilitates determining the severity of the prolapse.

Physiologic and complimentary radiologic tests are used to confirm clinical suspicions and provide objective data on the function of the anorectum. Pelvic floor dysfunction is a complex problem and multiple tests may be needed [114]. An algorithm for the investigation of faecal incontinence is shown in Figure 1.

2. ANORECTAL MANOMETRY

Anal manometry provides a sphincter pressure profile and measures resting and maximum squeeze pressures, length of the anal canal and demonstrates the presence or absence of the anorectal inhibitory reflex [115]. The resting anal pressure reflects the functional status of the internal anal sphincter, the squeeze pressure reflects the functional status of the external sphincter and the anorectal inhibitory reflex demonstrates coordination between rectum and anal canal. The length of the anal

canal can be the only detectable change in anorectal function tests after successful postanal repair or overlapping sphincteroplasty.

The data generated can be used to create a three-dimensional pressure map of the anal canal both at rest and during contraction. Several different catheters have been used, such as air- or water-filled microballoons, water-perfused catheters, sleeve catheters, or solid-state microtransducers. With water-perfused catheters, ports are arranged circumferentially around the catheter tip and generate a 360° pressure image along the length of the anal canal. Recorded pressures vary with type and size of probe used. A station pull-through technique is most commonly used, with resting and squeeze pressures being recorded at 0.5-cm intervals along the lower 6 cm of the anorectum. Directional manometry describes a pressure vector that can identify the site of a sphincter defect. Simple digital examination by an experienced clinician gives a very good estimation of anal sphincter pressures [116].

Unfortunately, there can be considerable overlap between the pressure profiles of normal and incontinent patients; patients with normal resting pressures but reduced squeeze pressures may be incontinent because of failure of the external anal sphincter to contribute to continence at critical times. A significant number of continent patients have impaired pressure profiles and some incontinent patients generate normal pressure profiles. Manometric studies of the anal canal do not always predict the patient's clinical status.

3. ANORECTAL SENSATION

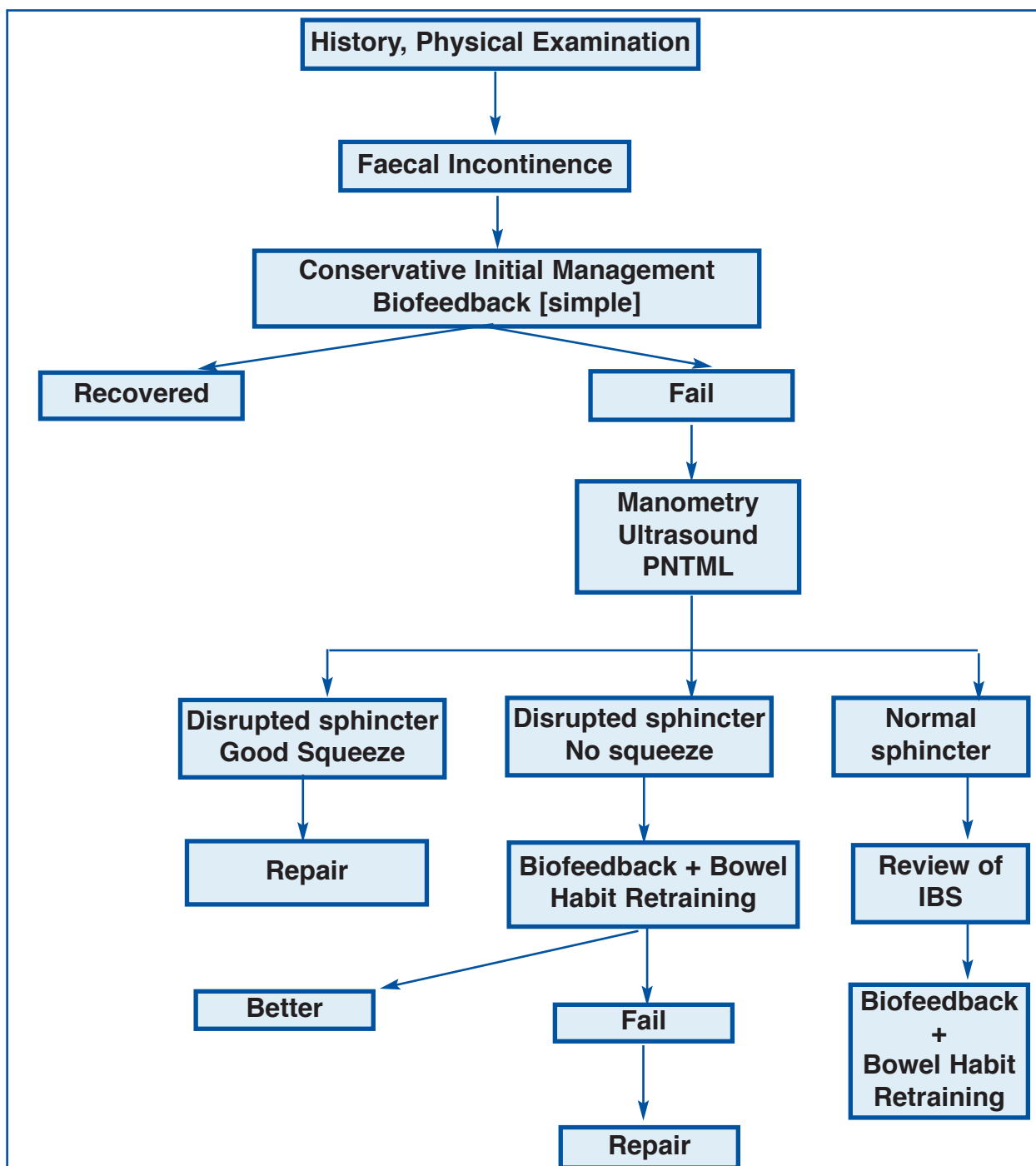
To measure rectal sensation, a balloon is attached to the manometry catheter. The balloon is placed in the rectum and slowly inflated. The patient announces when the sensation of a faecal mass within the rectum is first noticed and filling of the balloon continues to the point at which the patient would wish to defecate. Both blunted and hyperacute rectal sensation can be detected from this test.

Sensation of the anal canal can be measured by means of small electrical probe inserted into the anal canal and connected to a constant current stimulator [110]. The amplitude of current is gradually increased until the patient experiences a tapping or pricking sensation. This threshold to electrostimulation is increased in patients with incontinence but the test is quite specialised and may be of limited value in clinical practice.

4. ELECTROMYOGRAPHY OF THE PELVIC FLOOR AND PUDENDAL NERVE LATENCY

Electromyography (EMG) of the anal sphincter demonstrates electromuscular activity of the puborecta-

Figure 1 : Algorithm for assessment of adults



The newer, investigational surgical approaches are:

Sacral nerve stimulation is appropriate:	Failed standard measures No IBS No anal sphincter defects
--	---

Stimulated graciloplasty is appropriate:	Failed standard measures No IBS Anal defect
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Colon conduit is appropriate:	May be option in the elderly or those with congenital cloacal defects.
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lis and will identify re-innervation of the sphincter typical of pelvic neuropathy. The test can be carried out with single-fibre or concentric needle techniques. Single-fibre electrodes detect potentials that originate from a single motor unit. Fibre density may be determined from the mean number of spikes recorded from the uptake area of a single-fibre EMG electrode sited in 20 different positions [117]. Increased fibre density, suggested by increased number of spike potentials detected by the single-fibre EMG electrode, implies denervation followed by re-innervation as axons sprout from intact neurons to re-innervate muscle fibres. Incontinent patients usually show smaller and prolonged motor unit potentials.

A needle electrode is inserted into either the puborectalis or, more commonly, the external anal sphincter to carry out concentric needle EMG. Electromyography is an uncomfortable investigation that is unpopular with patients and objective interpretation of concentric EMG can be very difficult. An alternative, less painful technique is to use a sponge EMG electrode, which is inserted into the anal canal. Two electrodes, placed 180 degrees apart, record electrical activity of the puborectalis at rest, squeezing, and pushing (which simulates defecation). Normally, the puborectalis generates the greatest signal during contraction and normal defaecation requires relaxation of the puborectalis to permit straightening of the anorectal angle. Incontinent patients may show diminished activity in all phases of activity. It should be noted that EMG of the EAS and pelvic floor is not performed for diagnostic purposes at this time.

Pudendal nerve terminal motor latencies are measured by placing a glove-mounted, stimulating electrode that is inserted through the anal canal onto the nerve as it crosses the ischial spine. A recording electrode, adjacent to the external sphincter, detects the evoked potential associated with muscle contraction [118]. The process is carried out for each pudendal nerve. The normal delay between stimulating and recording is 2.0 +/-0.5 (SD) ms). Increased delay suggests damage to the pudendal nerves.

Although no medical or surgical treatment has been shown to be effective for pudendal nerve damage at present, it is an important diagnosis to make. First, surgical intervention (such as overlapping sphincteroplasty) is usually less successful [119]. Second, if the surgeon is persuaded to attempt a procedure (such as postanal repair), then the surgeon can prepare the patient for the likelihood of failure. Third, some surgeons would argue that some form of neosphincter might be appropriate for this subgroup of incontinent patients.

5. DEFAECATING PROCTOGRAM

Defaecography is a dynamic study of rectal emptying

and the complex process of defaecation. The patient is positioned sitting upright upon a radiolucent commode and barium paste is instilled into the rectum and distal sigmoid. Lateral fluoroscopic images are taken as the patient squeezes the sphincter, while deferring defaecation, and while straining to defaecate. A number of interactive processes can be seen and assessed. A defaecating proctogram assesses the function of the puborectalis, efficiency of emptying length of the anal sphincter, movement of anorectal angle, and perineal descent [120]. Defaecating proctograms are, however, difficult to interpret as 50% of normal subjects will show some abnormality [121]. Occult or overt rectal intussusception may be seen, as well as the presence or absence of a rectocele, sigmoidocele, or enterocele. It should be noted that although a careful history and physical examination may lead to a diagnosis of a rectocele, its role in incontinence may be unclear until clarified by defaecography.

6. ULTRASOUND

Endoanal ultrasound has only recently been used for the investigation of patients with faecal incontinence. The morphology of the internal anal sphincter, part of the external anal sphincter, the puborectalis, and the rectovaginal septum can be seen remarkably well [122] (Figure 2). It is particularly useful if there is a history suggestive of a sphincter defect. Defects, evident as a break in the ring of the sphincter deep to the submucosa, may be identified and mapped. Endoanal ultrasound has shown that up to 85% of patients with incontinence of traumatic origin have external sphincter defects and

Table 12 : Tests of anorectal function for the patient with faecal incontinence

A. Initial Evaluation	1. History of symptoms to include psychosocial history
	2. Physical exam to include visual and digital exam (including examination while straining on the toilet)
B. Prior to Surgical Intervention	1. Anorectal manometry to include compliance (P/V relationship)
	2. Ultrasound of the anal canal
	3. Visualise the colon (proctosigmoidoscopy, colonoscopy)
	4. Abdominal X-ray
C. Optional	1. Pudendal nerve terminal motor latency
	2. Electromyography
D. Research	1. Barostat
	2. MRI
	3. Vector manometry
E. Levels of Evidence for:	
Anorectal Manometry	2—for accuracy 3—for outcomes
Ultrasound	2—for accuracy 3—for outcomes
PNTML	3

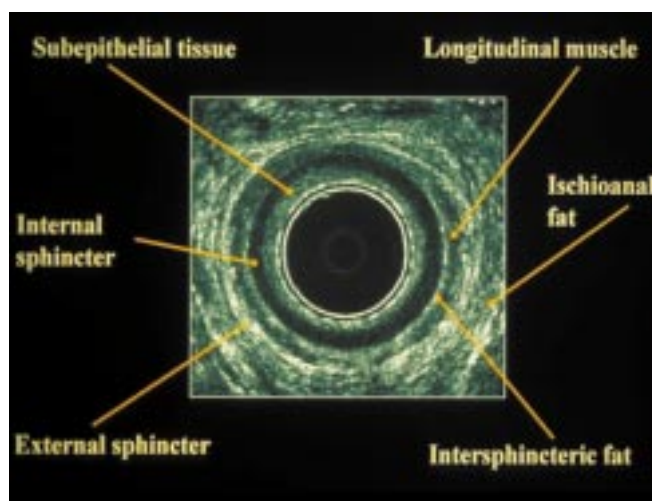


Figure 2 : Normal anal ultrasound

40% of these patients also have disruption of the internal sphincter [105]. Such defects are often associated with a weak puborectalis sling that can be relatively inert on dynamic testing (when the patient is asked to squeeze the sphincter). It may reveal cryptoglandular infection as well as the relative bulk of the upper, middle, and lower parts of the anal sphincter. Incontinent patients with low resting anal pressures may have a thinner internal anal sphincter compared with controls. Endoanal ultrasound is superseding electromyography in the investigation of patients with faecal incontinence.

7. MAGNETIC RESONANCE IMAGING (MRI)

MRI alone and in conjunction with intra-anal and intra-rectal ultrasound may enhance our understanding of pelvic floor function as a whole and delineate abnormalities of function perhaps not seen with more standard tests [123]. Agreement between radiographers regarding the anatomy specifically delineated by MRI remains a problem, however [124].

VI. SUMMARY AND RECOMMENDATIONS

Anorectal incontinence is multifactorial in origin: it rarely presents as a single, easily identifiable abnormality in individual patients. An algorithmic approach (Figure 1) is needed for the evaluation, quantification of anorectal function, and finally, categorization of patients with faecal incontinence. In this way, appropriate conservative medical, aggressive non-surgical (biofeedback), and surgical modalities can be undertaken with an enhanced degree of long-term success.

E. CHILDBIRTH AND ANAL INCONTINENCE

I. INTRODUCTION

Pregnancy and childbirth have a significant impact on the emotional and physical wellbeing of a woman. It is reported that as many as 91% of women report at least one new symptom eight weeks post-partum [125]. A fall in maternal mortality accompanied by an increase in female life expectancy (80 years in the UK) has now shifted the focus of attention towards identification of factors that may minimise morbidity. Although pre-existing bowel symptoms may be aggravated during pregnancy and childbirth, the development of symptoms *de novo* is a more frequent occurrence. Obstetric trauma is the commonest precursor to faecal incontinence. However the onset of symptoms may occur many years after delivery with a peak incidence in the perimenopausal years. This may reflect the effect of contributory factors such as the process of ageing, the effect of the menopause or progression of neuropathy. This section focuses on the association between obstetric trauma and faecal incontinence. However to avoid confusion, the term anal incontinence is used to include incontinence to flatus, liquid and solids.

II. MECHANISM

The mechanism that maintains continence is complex and affected by various factors such as mental function, lack of a compliant rectal reservoir, enhanced colonic transit and changes in stool consistency and volume. However the most important factors are appear to be an anatomical intact anal sphincter complex and neurological function. In about 80% of women with presumed “idiopathic” anorectal incontinence there is histological evidence of denervation of the striated pelvic floor muscles, particularly the puborectalis and external sphincter [126]. This feature has also been demonstrated electro-physiologically by means of an increased fibre density in patients with idiopathic faecal incontinence indicating re-innervation following denervation [127]. Another finding in these patients is a conduction delay in pudendal nerves as measured by pudendal nerve terminal motor latency (PNTML) [128].

Although Hertz in 1909 suggested that pelvic floor damage may result from a normal vaginal delivery, objective scientific evidence for this was only produced in 1984 [129] and a follow-up of 14 patients 5 years later [130]. These authors studied 122 women, 71 after delivery with manometry, perineometry, PNTML and EMG, and 51 before and after delivery with EMG. This

study demonstrated an increase in anal sphincter striated muscle fibre density in the vaginal delivery group at 2 months post-partum indicating evidence of re-innervation following denervation. The fibre density was not altered following elective caesarean section. Thirty three percent of primiparae and 50% of multiparae had prolonged PNTML within 48 hours of delivery. However by 2 months, the PNTML had returned to normal in 60% of these women, indicating that damage to pudendal nerve conduction is reversible. Multiparity, forceps delivery, increased duration of the second stage of labour, third degree perineal tears and high birth weight were important factors leading to pudendal nerve damage. In the 5 year follow-up study of 14 women, only multiparae who did not have a forceps delivery were selected; the denervating process was found to be progressive in the majority of women and 5 women suffered from stress incontinence of urine, 3 of whom were also incontinent to flatus.

In another prospective neurophysiological study, Allen et al [131] studied 96 nulliparous women with EMG, PNTML and vaginal pressure measurements during pelvic floor contraction. They found evidence of re-innervation in the pelvic floor muscles of 80% of primiparae 2 months after vaginal delivery. The only obstetric factors associated with re-innervation were a high birth weight and a longer active stage of labour. Forty five of the original 96 women were studied again 6 years later and they concluded that changes in pelvic floor neurophysiology occur with time and do not appear to be related to further childbearing [132].

A third prospective study [133] measured anal pressures, anal sensation and the perineal plane in 72 antenatal women and repeated 72 hours post-partum and in 41 women 2 months postpartum. Anal sensation was unchanged. Cornes et al [134] measured anal sensation in 96 primiparae within 10 days after delivery and measurements were repeated in 74 women 6 months after delivery. They found that at 6 months anal sensation had returned to normal. Anal sensation remained unchanged after caesarean section. In women who had a torn external sphincter, only impairment of sensation in the upper anal canal persisted at 6 months. More than half the women who admitted to persistent anal incontinence had normal anal sensation. Chaliha et al [135] measured anal electro-sensitivity before and after childbirth and found it unchanged. Anal sensation in isolation therefore probably plays a minor role in the development of obstetric related faecal incontinence.

1. MECHANICAL TRAUMA

Until the recent advent of anal ultrasound, mechanical trauma to the anal sphincters was only suspected when there was a history of a difficult vaginal delivery, parti-

cularly a third or fourth degree tear. Consequently, when anal endosonography was first performed in patients believed to be suffering from "neurogenic" faecal incontinence, unsuspected internal and external sphincter defects were identified [105]. The sonographic appearance of external anal sphincter defects has been verified histologically to represent fibrosis [136] while the appearance of internal sphincter defects have been validated prospectively in patients undergoing lateral internal anal sphincterotomy [137]. Trauma identified by ultrasound may be occult or recognised (third/fourth degree tear).

a) Occult anal sphincter trauma

To date Sultan et al [100] have performed the only prospective study (before and after childbirth) to demonstrate both occult anal sphincter trauma (Figure 3) and pudendal nerve damage during childbirth in both primiparous and multiparous women (n=150). Thirty five percent of primiparous women and 44% of multiparous women developed anal sphincter defects during vaginal delivery. Thirteen percent and 23% respectively developed defaecatory symptoms (faecal urgency and/or anal incontinence) after delivery. Only 2 of the 150 women (both primiparous) had recognised tears of the anal sphincter at the time of delivery. A strong association was demonstrated between the presence of any defect and the development of symptoms. Only 4% of multiparous women sustained new sphincter damage following a subsequent delivery. The single independent factor associated with anal sphincter damage was forceps delivery. The 23 women delivered by caesarean section remained asymptomatic and none developed sphincter defects. No relationship was demonstrated between pudendal latency measurements and defaecatory or urinary symptoms.

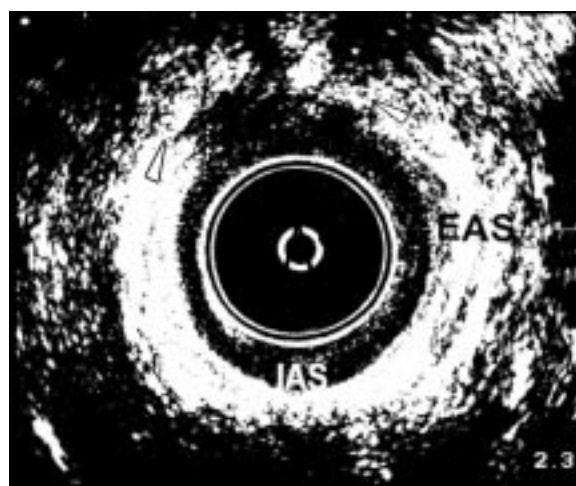


Figure 3 : Anal ultrasound image of the mid anal canal. EAS= external anal sphincter. IAS= internal anal sphincter. The area between the arrows at 10 and 12 o'clock represents an external anal sphincter defect.

Donnelly et al [22] interviewed 219 nulliparae regarding bowel habit in the third trimester and performed anal vector manometry. At 6 weeks postpartum 184 women returned and the same bowel symptom questionnaire was completed and anal vector manometry plus PNTML measurements were performed. Anal endosonography was performed in 81 women with altered faecal continence or abnormal physiology. Instrumental vaginal delivery and a passive second stage of labour prolonged by epidural analgesia were significantly associated with the greatest risk of anal sphincter trauma and impaired faecal continence. As instrumental delivery is a known risk factor (8 fold increased risk of sphincter trauma), early use of oxytocin was recommended to shorten the second stage. A continuation of the same study [138] reported that pudendal nerve latencies were prolonged and the squeeze pressure increment was reduced in those women who had a caesarean section in the late first stage (>8cm cervical dilatation) or second stage.

Chaliha et al [135] measured anal sensation and mano-

metry in 286 nulliparae during the third trimester and repeated in 161 women postpartum when anal endosonography was also performed. Anal endosonography revealed sphincter defects in 38% of women and this was associated with a lowering of anal squeeze and resting pressures. Threshold anal electrosensitivity remained unchanged and bore no relationship to symptoms. Postpartum sphincter defects were associated with perineal laceration and vaginal delivery.

Abramowitz et al [139] performed a prospective study of 233 women who had anal endosonography performed before and 6 to 8 weeks after childbirth. Of the 233 women (118 primiparae), 202 had a vaginal delivery. Postpartum anal incontinence in the 233 women was reported by 13% of primiparae and 8.5% of multiparae and anal sphincter defects in 21% and 12% respectively. However, the prevalence of anal sphincter defects amongst those that had a vaginal delivery (n=202) was 26% and 13% respectively. Previous studies [140, 141] including others mentioned in Table 13 and 14 have shown that the first delivery is at greatest risk for anal

Table 13 : Prospective studies before and after vaginal delivery of “occult” anal sphincter injury and anal incontinence excluding faecal urgency

Study	Parity Numbers	Vaginal delivery postpartum	FU in weeks	Sphincter Defects	Anal incontinence
Sultan et al 93 [100]	Primi	79	6	35%	5%
	Multi	48	6	44%	19%
Donnelly et al 98 [22]*	Primi	168	6	35%	25%
Rieger et al 98 [144]	Primi	37	6	41%	8%
Zetterstrom et al 99 [145]	Primi	38	9	20%	18%
Fynes et al 99 [142]*	Multi	59	6-12	37%	17%
Abramowitz et al 00 [139]	Primi	202 including	8	26%	15%
	Multi	multi	13%	10%	
MEAN	Primi			31%	14%
	Multi			31%	15%

*modified continence score questionnaire used and may include urgency

Table 14 : Studies of “occult” anal sphincter injury sustained during vaginal delivery and anal incontinence excluding faecal urgency only done postnatally

Study	Vaginal delivery	Parity	FU postpartum	Defects	Anal incontinence
Varma et al 99 (143)*	78	Primi	4 weeks	11.5%	0%
	31	Multi	4weeks	19%	0%
Damon 00 [146]	197	Primi	3 months	34%	6%
Faltin 00 [147]**	150	Primi	3 months	28%	15%

*Ultrasound performed < 1 week after delivery

** anal ultrasound performed immediately after delivery before perineal repair

sphincter trauma but this study is at variance as it claimed that secundiparous females have the same risk as primiparous women. However this finding remains unsubstantiated and is further disputed by a more recent prospective study [142].

Fynes et al [142] undertook a prospective study of 59 previously nulliparous women through 2 successive pregnancies and found that 34% had anal sphincter injury after their first delivery but only 2 new injuries occurred after the second delivery confirming the findings in Sultan's study [100]. An important finding in this study was that 42% of women (5 of 12) who had occult sphincter injury during their first delivery (squeeze pressure increment < 20mmHg or anal sphincter defect > one quadrant) developed anal incontinence after the second delivery.

In three further studies [143-145] anal ultrasound was performed only after delivery and defects were identified in 11.5 to 34% (Table 14). Varma et al [143] studied 159 postnatal women (105 primiparous and 54 secundiparous) and found occult anal sphincter defects in 11.5% of primiparous and 19% of secundiparous vaginal deliveries but 80% of forceps deliveries. None of their patients suffered faecal incontinence but only 72% of questionnaires were returned. However their cohort had a high caesarean section rate (25%) and a low forceps rate (4%).

b) Third/fourth degree obstetric tears

It is unclear as to whether the sonographic anal sphincter defects described above represent tears that have been missed at delivery or true "occult" defects that may not be visible to a trained doctor or midwife. Groom and Patterson [148] conducted a study in which they demonstrated that the rate of third degree tears rose to 15% when all "second degree tears" were examined by a second person confirming that at least some tears are being missed. This reflects inadequate training and was highlighted by Sultan et al [149] who reported that 91% of doctors who had done at least 6 months of training in obstetrics and 60% of midwives indicated inadequate training in perineal anatomy and 84% and 61% respectively reported inadequate training in identifying third degree tears. Another possible reason for under-diagnosis is that tears of the anal sphincter have been wrongly classified and therefore anal sphincter tears have been under-reported. Any involvement of the anal sphincter should be classified as third degree. However 41% of doctors and 16% of midwives classified a torn anal sphincter as a second degree tear [149]. Sultan and Thakar reviewed every relevant text book (n=65) in the library of the Royal College of Obstetricians and Gynaecologists (RCOG) and found that there was a lack of consistency in classification and in about 40% the classification was omitted or wrong [150].

Furthermore, previous classifications are incomplete because they do not incorporate depth of external sphincter rupture or involvement of the internal sphincter. This therefore has epidemiological, clinical and medicolegal implications. If a third degree tear is incorrectly classified as second degree, then inappropriate repair could result in sub-optimal outcome (see below). Sultan (151) has therefore proposed the following classification to be incorporated in the 29th RCOG green top guidelines:

First degree: laceration of the vaginal epithelium or perineal skin only.

Second degree: involvement of the perineal muscles but not the anal sphincter.

Third degree: disruption of the anal sphincter muscles and this should be further subdivided into:

3a: <50% thickness of external sphincter torn.

3b: >50% thickness of external sphincter torn.

3c: internal sphincter torn also.

Fourth degree: a third degree tear with disruption of the anal epithelium.

An isolated rectal tear without involvement of the anal sphincter is rare and should not be included in the above classification.

Primary sphincter repair of a third or fourth degree obstetric tear is usually performed by obstetricians using the end-to-end repair technique [152]. However as shown in Table 15, anal incontinence occurs in 15 to 59% and in addition, urgency can affect a further 6 [152, 153] to 28% [154]. Frank faecal incontinence affected 9% (range 2 [155] to 23% [156]). In five studies [152, 154, 157-159] anal endosonography was performed to demonstrate persistent anal sphincter defects following repair in 40 to 91% of women. Forceps delivery, first vaginal delivery, large baby, shoulder dystocia and a persistent occipito-posterior position have been identified as the main risk factors for the development of a third/fourth degree tear [139, 140, 152, 157].

The most popular method of repair of the external sphincter is the end-to-end technique but colorectal surgeons prefer the overlap technique for secondary repair because of better outcome [160]. It is now known that like other incontinence procedures outcome can deteriorate with time and one study has reported 50% continence at 5-year follow-up [161]. However some women in this study had more than one attempt at sphincter repair [161]. Sultan et al [160] were the first to describe the overlap technique for acute anal sphincter rupture and in addition advocated the separate identification and repair of the internal sphincter. Compared to matched historical controls [152] who had an end-to-

Table 15 : Prevalence of anal incontinence following primary repair of obstetric anal sphincter rupture

Authors	Year	Country	N	Follow-up Months	Anal incontinence
Sangalli et al [153]	2000	Switzerland	177	13 years	15%
Wood J et al [162]	1998	Australia	84	31	17%*
Walsh et al [163]	1996	UK	81	3	20%
Sander et al [164]	1999	Denmark	48	1	21%
Crawford et al [165]	1993	USA	35	12	23%
Sorensen et al [166]	1993	Denmark	38	3	24%
Nielsen et al [167]	1992	Denmark	24	12	29%
Go & Dunselman [168]	1988	Netherlands	20	6	30%
Uustal Fornell et al [169]	1996	Sweden	51	6	40%
Poen et al [157]	1998	Netherlands	117	56	40%
Sultan et al [152]	1994	UK	34	2	41%
Zetterstrom et al [155]	1999	Sweden	46	9	41%
Sorensen et al [170]	1988	Denmark	25	78	42%
Tetzschner et al [171]	1996	Denmark	72	24-48	42%
Kammerer-Doak et al [159]	1999	New Mexico	15	4	43%
Haadem et al [172]	1988	Sweden	62	3	44%
Bek & Laurberg [173]	1992	Denmark	121	?	50%
Fitzpatrick et al [154]	2000	Ireland	154	3	53%
Gjessing H et al [156]	1998	Norway	38	12-60	57%
Goffeng et al [158]	1998	Sweden	27	12	59%
MEAN					37%

* Includes 2 with secondary sphincter repair

end repair, anal incontinence could be reduced from 41% to 8% using the overlap technique and separate repair of the internal sphincter [156]. However as the two operators performing the repair had a specialised knowledge of the anal sphincter anatomy it could have biased their results and they therefore recommended a randomised trial. The only published randomised trial published to date is by Fitzpatrick et al in Dublin [154] who found no significant difference between the 2 methods of repair although there appeared to be trend towards more symptoms in the end-to-end group. There were methodological differences in that the torn internal sphincter was not identified and repaired separately and they used a constipating agent for 3 days after the repair. Nevertheless as the authors concur, a better outcome would be expected with both techniques as a

consequence of focused education and training in anal sphincter repair. A longer-term follow-up is awaited and further randomised trials using the described technique [160] are currently in progress.

• MANAGEMENT OF SUBSEQUENT PREGNANCY

All women who have sustained a third/fourth degree tear should be assessed in hospital by a senior obstetrician 6 to 8 weeks after delivery. Some centres have established dedicated multidisciplinary perineal clinics. It is important that a careful history is taken regarding bowel, bladder and sexual function. As these symptoms are embarrassing, a structured questionnaire may be useful. A careful vaginal and rectal examination should be performed to check for complete healing, scar tenderness and sphincter tone [174, 175]. We

recommend that all women should have ano-rectal investigations (endosonography and manometry) but if such facilities are unavailable locally then at least symptomatic women should be referred for investigations (Figure 4) [150]. Mild incontinence (faecal urgency or flatus incontinence) may be controlled with dietary advice, constipating agents (loperamide or codeine phosphate), physiotherapy or biofeedback. However women who have severe incontinence should, in addition, be offered secondary sphincter repair by a colorectal surgeon. Asymptomatic women must be advised to return if symptoms develop.

There are no randomised studies to determine the most appropriate mode of a subsequent delivery. Women who have had a successful secondary sphincter repair for faecal incontinence should be delivered by caesarean section [176]. Some women with faecal incontinence may chose to complete their family prior to embarking on anal sphincter surgery. It remains to be established whether these women should be allowed a vaginal delivery as it could be argued that damage has already occurred and risk of further damage is minimal and possibly insignificant in terms of the outcome of surgery.

Very few text books discuss management of a subsequent pregnancy but some indicate that caesarean section should be considered particularly after a difficult primary repair [150]. It has been suggested that a cae-

sarean section should be performed even after transient anal incontinence [171] but this has been questioned [177].

In order to counsel women with previous third/fourth degree tears appropriately, we find it useful to have a symptom questionnaire, anal ultrasound (Figure 2) and manometry results. If vaginal delivery is contemplated then these tests should be performed during the current pregnancy unless performed previously and found to be abnormal (Figure 4). Current evidence suggests that if a large sonographic defect (more than one quadrant) is present or if the squeeze pressure increment is less than 20 mmHg then the risk of impaired continence is increased to 42% after a subsequent delivery [142]. One policy is to counsel these women and offer a caesarean section especially especially to those who are symptomatic. Asymptomatic women who do not have compromised anal sphincter function can be allowed a normal delivery by an experienced accoucher. Although 11% of textbooks recommend a prophylactic episiotomy [150] there is no evidence that an elective episiotomy prevents subsequent anal sphincter disruption. Some studies have indicated that episiotomy may increase the prevalence of anal sphincter disruption. Preliminary results of a multicentre study (Sultan, unpublished data) do not support the practice of prophylactic episiotomy and therefore it should only be performed if clinically indicated.

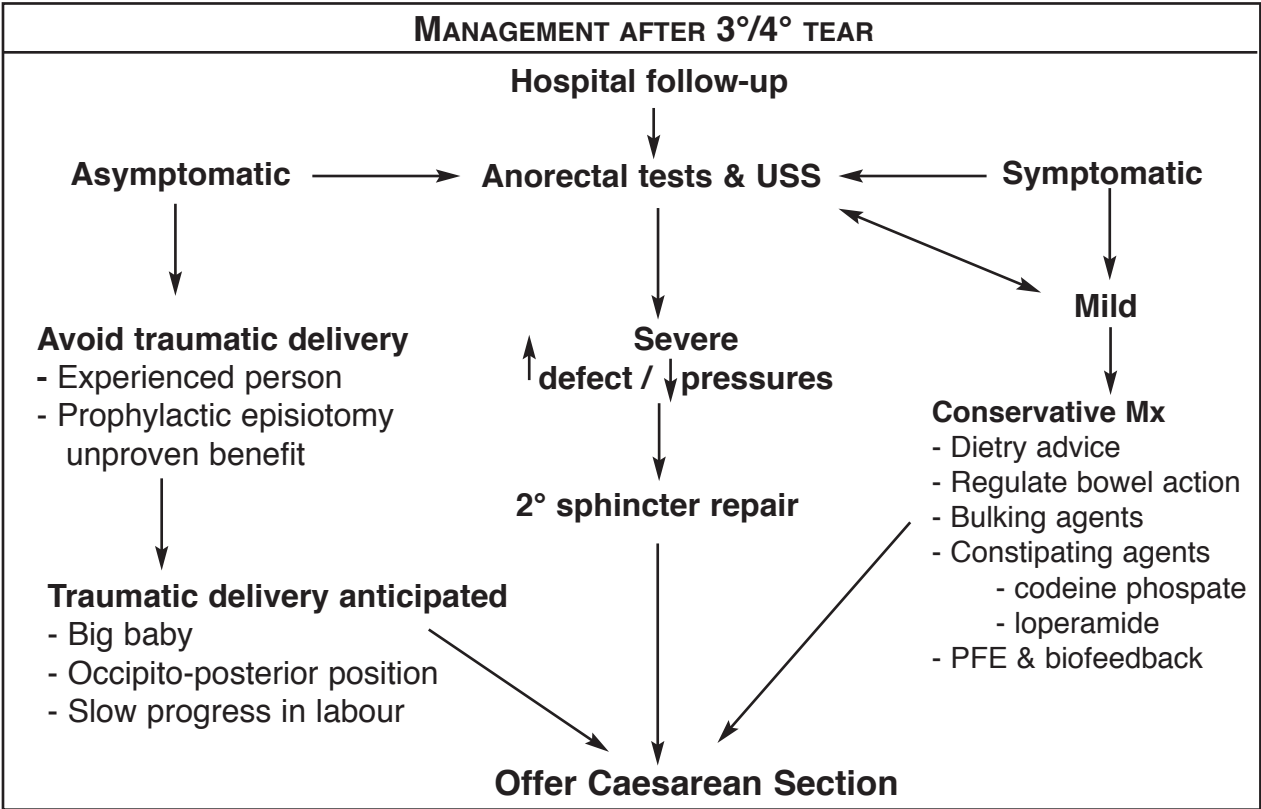


Figure 4 : Management of obstetric injury

III. EFFECTS OF INSTRUMENTAL VAGINAL DELIVERY

Although only 4% of women delivered by forceps sustain a third/fourth degree tear, up to 50% of those that do tear have an instrumental delivery [152]. Vacuum extraction is associated with fewer third/fourth tears than forceps and this view is supported by 2 large randomised studies [178, 179]. A UK study [178] where mediolateral episiotomy is practised reported severe vaginal lacerations in 17% of forceps compared to 11% of vacuum deliveries and a Canadian study [179] where midline episiotomy is practised reported third/fourth tears in 29% of forceps compared to 12% of vacuum deliveries. In a Cochrane review (ten trials) [181] use of the vacuum extractor instead of forceps was associated with significantly less maternal trauma (odds ratio 0.41, 95% confidence interval 0.33 to 0.50) and with less need for general and regional anaesthesia. There were more deliveries with vacuum extraction (odds ratio 1.69, 95% confidence interval 1.31 to 2.19) and fewer caesarean sections were carried out in the vacuum extractor group. However the vacuum extractor was associated with an increase in neonatal cephalhaematoma and retinal haemorrhages. Serious neonatal injury was uncommon with either instrument.

The reduction in cephalhaematoma and retinal haemorrhages seen with forceps may be a compensatory benefit. A 5 year follow-up of infants who participated in a randomised study of forceps and vacuum delivery has confirmed that there is no difference in terms of neurological development and visual acuity with use of either instrument [59]. Occult trauma to the anal sphincter has also been identified more frequently in forceps delivery occurring in up to 80 percent [100, 143, 182]. A small randomised study (n=44) confirmed this by identifying occult anal sphincter defects in 79% of forceps compared to 40% of vacuum deliveries [182]. Trauma occurs more frequently when a second instrument is used to attempt vaginal delivery [182] and therefore if delivery fails with the appropriate technique and vacuum cup, one should resort to a caesarean section. Metal cups appear to be more suitable for 'occipito-posterior', transverse and difficult 'occipito-anterior' position deliveries [183]. The soft cups seem to be appropriate for straightforward deliveries as they are significantly more likely to fail to achieve vaginal delivery (odds ratio 1.65, 95% confidence interval 1.19 to 2.29). Although they were associated with less scalp injury (odds ratio 0.45, 95% confidence interval 0.15 to 0.60), there was no difference between the two groups in terms of maternal injury. Farrell et al [184] performed a prospective study of 690 primigravid women and found that forceps delivery was associated with a higher incidence of flatal incontinence (RR 2.6) com-

pared to vaginal delivery and both flatal (RR 2.6) and faecal (RR 3.6) incontinence compared to caesarean delivery. Vacuum delivery did not increase the risk of flatal incontinence. In another recently study MacArthur et al [185] performed the largest questionnaire based multicentre study to establish the prevalence of faecal incontinence at 3 months post-partum. They reported a prevalence of 9.2%, with 4.2% reporting it more often than rarely. Forceps delivery was associated with almost twice the risk of developing faecal incontinence whereas vacuum extraction was not associated with this risk. These studies support the recommendation by the U.K. Royal College of Obstetricians and Gynaecologists (RCOG) that the vacuum extractor should be the instrument of choice [186].

IV. EPISIOTOMY

There is now considerable observational data to indicate that a reduction in episiotomy rate is not associated with an increase in anal sphincter rupture [187]. The Cochrane database [188] shows that restricting the use of episiotomy is associated with less posterior vaginal trauma. Although anterior perineal trauma was increased it had no effect on the development of urinary incontinence. Henrikssen et al [189, 190] performed an observational study in which they noted that when midwives who previously had a high episiotomy rate reduced their rate, the prevalence of anal sphincter rupture also reduced. However this beneficial effect was abolished when midwives with a low rate of episiotomy attempted to reduce it even further. Based on this evidence, it was suggested that the ideal episiotomy rate should lie between 20 to 30% and no more. Midline episiotomies are more popular in North America as it is believed that they are more comfortable and recovery is less complicated. However Coats et al [191] performed a randomised study of 407 primiparae and found 12% of midline episiotomies extended into the anal sphincter compared to 2% of mediolateral episiotomies. Although the perineum was significantly less bruised in the midline group and sexual intercourse commenced earlier, pain and wound breakdown was similar in both groups.

V. DELIVERY TECHNIQUES

Pirhonen et al [192] compared the frequency of anal sphincter rupture in low risk deliveries between two Scandinavian countries (26,541 vaginal deliveries) and found the risk to be 13 times higher in Sweden (Malmö) vs Finland (Turku). They speculated that the only explanation for this was a difference in manual support given to the baby's head during crowning and pushing the perineum under the chin.

VI. TRAINING

There is evidence from one study [149] that perineal anatomy is poorly understood by midwives and trainee doctors, who perform the bulk of deliveries in the UK. In this study 41% of trainees and 16% of midwives incorrectly classified a partial or complete tear of the EAS as 'second degree'. Inconsistency in classification of tears would allow many injuries to pass unrecognised. Intensive and focused training in perineal anatomy and repair should therefore become an essential module in the programme for trainees.

VII. IRRITABLE BOWEL SYNDROME (IBS)

IBS affects 3-17% in selected populations and the cause remains unknown. Donnelly et al [23] recruited 312 primiparous women and reported that 11% of young primiparous women (n= 34 of 208) suffered from pre-existing IBS prior to their first pregnancy. Twenty four percent reported symptoms of impaired faecal continence in the puerperium but symptoms were found significantly more frequently in those with IBS compared to those with normal bowel habit (71% vs 18%). However women suffering from IBS are no more likely to incur mechanical or neurologic injury to the anal sphincter. Women with IBS delivered by caesarean section did not have altered continence postpartum. However 6 months postpartum there were no symptomatic differences between those with IBS and those without, but only 90 of the 107 women who had either impaired faecal continence or abnormal anal manometry were studied. Treatment is directed towards the predominant symptom and although antispasmodics such as hyoscine, mebeverine and dicyclomine are used widely to relax intestinal smooth muscle, they should be avoided during pregnancy.

VIII. RECOMMENDATIONS

- a) Compared to forceps the vacuum extractor is associated with less perineal and anal sphincter trauma and should therefore be the instrument of choice. (Level 1)
- b) Compared to midline episiotomy, mediolateral episiotomy is associated with a lower risk of anal sphincter rupture (12% vs 2%). (Level 1)
- c) Liberal use of episiotomy is not beneficial (Level 1) and restricting the rate of episiotomy to about 30% may reduce the risk of trauma to the anal sphincter. (Level 4)

- d) A prolonged active second stage of labour is associated with denervation of the pelvic floor and one study has suggested that this also occurs with a prolonged passive second stage of labour with epidural analgesia. In these circumstances, early use of oxytocics in the second stage of labour may be useful. (Level 4)
- e) Selective use of caesarean section particularly in those who have evidence of compromised anal sphincter function and those who have had previous successful continence or prolapse surgery. (Level 5)
- f) The value of antenatal pelvic floor exercises in the prevention of incontinence and prolapse could be of benefit but is currently being evaluated in randomised studies.
- g) Modification in techniques of delivery of the baby may reduce anal sphincter injury and further research is needed (Level 5)
- g) A more focused training program for doctors and midwives needs to be implemented. There is a poor understanding of perineal and anal sphincter anatomy and hence identification of anal sphincter trauma, incorrect classification and poor outcome of repair (Level 5)

F. CONSERVATIVE MANAGEMENT OF FAECAL INCONTINENCE IN ADULTS

I. DRUG THERAPY

Patients who experience urge incontinence of faeces associated with loose stool, or who have a passive anal seepage of soft stool may benefit from low dose constipating medication [193]. Loperamide has been found to reduce faecal incontinence, improve stool consistency, and reduce stool weight compared to placebo. Continence to rectally infused saline improved on 4mg three times per day in 26 patients with chronic diarrhoea, and there was additionally a small increase in resting anal pressure [194]. Loperamide oxide has a similar effect [195] There is also some evidence that loperamide increases water absorption by slowing colonic transit and, in an animal model, that it decreases internal anal sphincter relaxation in response to rectal distension [196] and increases mucosal fluid uptake [197].

Codeine phosphate is a naturally occurring opiate which also acts to increase transit time. It is prescribed in doses of 30 to 120mg daily in divided doses. The use of codeine is limited by the relatively high incidence of side-effects, principally drowsiness. Additionally, long term usage of codeine may be associated with tolerance and dependence. Despite these problems, codeine is a useful agent in the treatment of faecal incontinence [198] although formal evidence from trials is lacking. It may be given in addition to loperamide when maximal doses have failed to reduce symptoms.

Some patients may choose to effectively stop spontaneous evacuation by using these agents and then plan evacuation at a convenient time by using suppositories or a micro-enema. Although not ideal, this regime can at least give the patient an element of control and predictability to enable a reasonable quality of life. This has been found to be effective in a nursing home environment, even when staff compliance with prescribed regimes is not good [199].

One study has suggested some benefit from hormone replacement therapy (HRT) in post-menopausal women with faecal incontinence [200]. Resting and squeeze anal pressures were increased, as was the maximum volume tolerated in the rectum. The presence of an anal sphincter defect did not preclude symptomatic benefit. 90% of women reported some improvement and 25% became symptom-free. These results must be viewed with caution as it was a small (20 patient) uncontrolled study, and it has been suggested with urinary incontinence that subjective well-being rather than objective improvement may be the mode of action [201]. It is known that hormone replacement can increase collagen and elastic tissue in the pelvic floor, that there are oestrogen receptors in the external anal sphincter [202], and that anal pressures have a tendency to decrease with advancing age [203]. It is theoretically possible that HRT could decrease or even reverse this trend.

A more recent pharmacological approach has been to modulate anal sphincter activity. Alpha-stimulant medication has been found to increase anal resting tone in healthy volunteers [204] and to improve incontinence in patients after formation of an ileo-anal pouch [205]. However, initial results in patients with “idiopathic” incontinence have been disappointing [206].

II. BIOFEEDBACK

Biofeedback is a technique which gives the patient immediate feedback about normally subconscious body processes. Equipment is used to detect and amplify a physiologic response [207]. Biofeedback has been extensively used in clinical practice and has been advocated to be “the treatment of choice” for faecal inconti-

nence [208]. Many different treatment modalities have been used in the name of “biofeedback”. As described by the original authors, it was thought to be an operant conditioning therapy [209]. The aim was for the patient to learn to enhance the presumed reflex contraction of the external anal sphincter (EAS) in response to a reflex relaxation of the internal anal sphincter (IAS) induced by stimulating the recto-anal inhibitory reflex by distension of a rectal balloon [209]. It has subsequently become evident that the EAS response is mostly a voluntary (if usually subconscious) response [210]. Later authors have recognised this and have focused on training the patient to improve this voluntary response.

1. MODALITIES OF BIOFEEDBACK

Three main modalities, with many variations and many adjunctive measures, have been described, although there is a wide variety of methods used within each modality, and some studies give very little detail on equipment or training programme used. No two studies have described exactly the same treatment as “biofeedback”.

The first method is the use of an intra-anal electro-myographic (EMG) sensor, anal manometric probe (measuring intra-anal pressure), or perianal surface EMG electrodes to teach the patient how to exercise the anal sphincter, usually as a variation of pelvic floor muscle or “Kegel” exercises more commonly used for treatment of urinary incontinence [211]. Some have used this simply to demonstrate correct isolation and use of an anal squeeze in response to rectal filling or an urge to defaecate. Others have devised a programme of home exercises and use the clinic biofeedback sessions to demonstrate correct technique and monitor progress in achievement. Early studies tended to focus on the peak muscle strength (squeeze increment) [212], while later workers have suggested that it is the overall muscle capacity (strength and endurance of the squeeze) that is important [213, 214].

The second modality is the use of a 3-balloon system to “train” the patient to correctly identify the stimulus of rectal distension and to respond without delay by immediate and forceful EAS contraction to counteract reflex inhibition of the internal anal sphincter. Some have felt that sensory delay is an important factor in faecal incontinence and that abolishing any delay in response to the sensation of distension is the crucial element in successful therapy [215, 216].

The third method is the use of a rectal balloon to “retrain” the rectal sensory threshold, usually with the aim of enabling the patient to discriminate (and thus respond to) smaller rectal volumes. However, tolerance of larger volumes by the use of progressive distension and urge resistance is also reported [217, 218].

One study has used anal ultrasound to show patients contraction of the anal sphincter on a screen [219].

Two studies have attempted to evaluate the different components of biofeedback in randomised controlled trials [216, 220]. Unfortunately, both had very complex designs which makes analysis of the different components impossible from the data presented, except that improving (lowering) the threshold for sensing rectal distension did seem to be beneficial in reducing symptoms [216].

2. RESULTS OF BIOFEEDBACK

A systematic review found 46 studies in adults published in English, including a total of 1364 patients treated [221]. In all but four studies the majority of patients were female. Ages in the included studies ranged from 6-97 years (studies where the majority of participants were children were excluded). Most were mixed or unstated aetiology groups, but a few studies related to specific patients groups (e.g. obstetric [222, 223], or elderly [224]) or to those with a common surgical history (e.g. sphincter repair [225], or anterior resection of the rectum [226]). Two studies involved patients undergoing formation of an ileo-anal pouch and looked at the prophylactic rather than treatment effect of biofeedback; these patients initially had a covering stoma, so it was not known how many had the symptom of faecal incontinence [217, 227]. One study followed a cohort of women who had a repaired third degree obstetric tear, treating symptomatic women more intensively than those without symptoms [223].

Studies have used between one and twenty-eight sessions over 2 days to one year duration of therapy. A wide variety of outcome measures has been employed in studies of biofeedback. Most take reduction of episodes of faecal incontinence as their primary endpoint, although few explicitly stated that diaries or questionnaires were used to gather this information. Criteria for success varied from 90% [228, 229] to 50% [230] reduction in episodes of incontinence. Success rates ranged from 0% [231] to 100% [232]. Thirty four studies quoted an overall response rate (usually combined cure and improvement). Of these, only 3 had a response rate under 50%; 13 had a response rate of 50-75% and 19 studies had a response rate of 75% or greater (6 over 90%).

Of those studies which gave data on patients who were free of the symptom of faecal incontinence, 275 of 566 patients (48.6%) were said to have no incontinence at the end of the study or follow up period. Criteria for "responders" varied greatly, but overall, 617 of 861 patients were reported as responders (71.7%). Many did not specify a follow-up period. Some had no follow up and reported results at the end of the treatment period

only [218, 222]. Of those that had longer follow up, most included a wide variation within a mean. Only 4 studies reported results at 2 or more years for all patients [216, 233-5]. One study reported immediate results and then at 3 years follow-up, with success deteriorating from 22 to 15 of 37 patients over time [235].

Adjunctive therapies have included the use of electrical stimulation [222, 223]. Others mention the use of medication (bulking agents or anti-diarrhoeals) and dietary modification. The majority of studies mention that patients were instructed to practice at home, but few specified in detail what instructions were given to the patients. Some instructed patients to squeeze whenever rectal distension was felt; other gave a structured exercise programme.

From these studies, there are few clear indicators of which patients are likely to benefit from biofeedback, and some of the evidence is contradictory. Few studies had the benefit of anal ultrasound to define sphincter structural defects. Two studies did not find that success correlated with defects [236, 237], while another obtained better results if the anal sphincters were structurally intact [218]. Pudendal nerve damage was the common factor in the only study to find no benefit at all from biofeedback [231], but others have found that success is independent of pudendal nerve function [236]. Although patients with pudendal neuropathy failed to increase their anal squeeze pressure, this did not preclude symptomatic improvement [236]. Pre-treatment manometry does not seem to predict success or failure [237-9], but sensitivity to balloon distension with air may be important, with some authors concluding that those with an initially insensitive rectum are less likely to respond [229], and others finding that decreasing sensory threshold is the single most important element of biofeedback [216]. Many studies have shown improvement in both resting and squeeze anal pressures, but improvement does not necessarily correlate with symptomatic relief. For example, one study found that pressures increased in both successes and failures [228]; another found that even those who failed to improve their pressures could improve their symptoms [236]. Two studies concluded that duration rather than strength of squeeze was the important element [213, 214].

A Cochrane review of controlled studies of biofeedback and exercises for faecal incontinence has concluded that "there is not enough evidence from trials to judge whether these treatments are helpful, nor which aspects of the treatment are the most helpful and which patients are the most likely to be helped" [240]. Four trials reported in published papers and one reported in abstract form met the inclusion criteria of a randomised or quasi-randomised controlled trial [216, 220, 222, 224, 241]. The total number of participants was 109. All

included trials but one [222] presented with potential methodological bias. The quality of allocation concealment was judged to be adequate in one trial [222], unclear in three trials [220, 216, 241] and inadequate in one trial [224]. Only two of the included trials provided data in a form suitable for statistical analysis.

No two studies employed the same outcome measures and consequently a quantitative synthesis was not possible. Results from a single trial in this review suggest that anal biofeedback is superior to vaginal, and that electrical stimulation might enhance the results of exercises [222]. However, the trial focused on the use of electrical stimulation as an adjunct to biofeedback and compared two very different types of interventions (vaginal pelvic floor manometric pressure biofeedback and home exercise with anal EMG biofeedback and home exercises in combination with anal electrical stimulation) and did not just single out the effects of electrical stimulation or biofeedback. Moreover, it is difficult to know how much of this improvement is a consequence of the natural history of faecal incontinence following childbirth as a no-treatment group was not included.

It appears also that training to enhance rectal discrimination of sensation may be helpful in reducing faecal incontinence, at least in the short term [216]. It may be that adding this technique to the more commonly-available pelvic floor muscle training would enhance results, but this cannot be a strong recommendation in view of the small numbers and lack of follow-up data.

The evidence suggests that biofeedback and exercises have an effect in clinical practice and a role in the treatment of patients with faecal incontinence, with only one treatment study reporting no benefit at all and the majority improving at least 50% of patients at least to some degree. However, it cannot be claimed that “biofeedback” is the effective element in what is inevitably a complex package of care, including assessment and the opportunity to discuss these taboo symptoms, time and attention with an enthusiastic therapist, patient information and teaching, and often supplementary advice on diet, medication and lifestyle. It is not known whether these elements, exercises alone, or the biofeedback itself, are the determining factor in symptom improvement. Well-designed controlled trials are needed to clarify these issues.

a) Summary

It is not possible to draw definitive conclusions for practice from the currently available evidence. Research does not provide sufficient evidence on which to judge the effectiveness of sphincter exercises and or biofeedback therapy in the management of people with faecal incontinence. In particular there is not enough

evidence on which to select patients suitable for anal sphincter exercises and/or biofeedback, nor to know which modality of biofeedback or exercises is optimal.

b) Recommendations

No study has reported adverse events from biofeedback and it seems unlikely that the treatment itself could cause worsening of symptoms. Given this, and the potential for adverse events from surgery [161], biofeedback may be considered as a first-line option in therapy for many patients with faecal incontinence which has not responded to simple dietary advice or medication, with surgery reserved for those with major structural abnormalities of the anal sphincter, severe symptoms and those who fail to respond to biofeedback (D). Although some results from the studies reviewed here are equivocal, the majority report at least some positive benefit.

III. ELECTRICAL STIMULATION

Electrical stimulation was first described for treatment of faecal incontinence nearly 40 years ago [242] and was described as efficacious in several uncontrolled studies over the subsequent decades [243-5]. Only one study has reported minimal clinical benefit, and this in patients with major pudendal neuropathy, who probably could not be expected to respond [246].

Stimulation has never been properly evaluated in a controlled manner. A recent review of controlled trials of electrical stimulation concluded “Only one eligible trial with 40 participants was identified. At present, there are insufficient data to allow reliable conclusions to be drawn on the effects of electrical stimulation in the management of faecal incontinence. There is a suggestion that electrical stimulation may have a therapeutic effect, but this is not certain” [247]. There are no controlled studies comparing different stimulation parameters or active with sham stimulation. There have been very few studies and most have been very small, with some using inappropriate stimulation parameters, particularly too low a frequency, for optimum muscle hypertrophy. Some have not even stated their stimulation parameters. Interferential therapy has been judged ineffective, in this and many other areas of physiotherapy [248]. Many have used very few treatments, or over a period of time too short to reasonably expect change. Some have used home exercise [244] and/or biofeedback [222] as well (often instructions not stated), so it is not possible to tell which has an effect. One controlled study has compared biofeedback and exercise with adjunctive electrical stimulation, but in this study different therapists were used to compare vaginal biofeedback with anal biofeedback plus electrical sti-

mulation, and so electrical stimulation was not the only variable and the difference in outcome found could have been the effect of a different therapist or biofeedback method [222]. One attempted controlled study after repair of third degree tears during childbirth abandoned stimulation as it caused discomfort [223].

Most have used unselected patient groups and some have not had the benefit of any physiological investigations to ascertain the cause of incontinence. Few have had the benefit of endoanal ultrasound and so the structural status of the internal and external anal sphincters is unknown in most studies.

a) Recommendations

Given the reasonable success rates of treatment for urinary incontinence [211], and clinical observations of some therapeutic effect in clinical practice, this is a treatment modality which warrants proper investigation (D).

IV. BOWEL MANAGEMENT IN THE NEUROLOGICAL PATIENT

While there is a considerable literature on the prevalence and pathophysiology of bowel dysfunction in neurological disease or injury, there has been remarkably little work done on practical management. In particular, there are very few controlled trials of interventions, and many articles finish their review of causes with rather vague advice on the importance of a good fluid intake and balanced diet. Many patients with neurological problems will be treading a fine dividing line between constipation and faecal incontinence. Great care is needed that in treating one, the other is not precipitated [249, 250].

Many patients will prefer constipation to incontinence as being more socially acceptable, but care is needed that side-effects of constipation such as exacerbation of bladder symptoms [251] or limb spasticity [249] or faecal impaction with overflow diarrhoea do not in turn cause problems. In patients with high spinal lesions impaction can precipitate autonomic dysreflexia and so is potentially life-threatening [252]. Many patients with neurological problems will need to actively plan bowel evacuation rather than wait until problems develop.

The importance of a multidisciplinary assessment in order to identify and minimise impairments disabilities and handicaps related to bowel dysfunction, and in setting person-centred goals in line with desired post-rehabilitation lifestyle are often emphasised [253]. Any bowel schedule has to fit with other activities, of the patient and any carer involved.

Bowel control is often easier to manipulate than blad-

der control as, if complete evacuation can be achieved, the rectum is normally empty, mass movements are an infrequent event and stool consistency can be regulated by diet or medication. Most patients, with careful planning of a bowel regime, should be able to achieve bowel continence and adequate evacuation. Often this will involve a “package” of care rather than a single intervention - attention to diet, bowel habit, evacuation techniques, and in selected cases, use of medication or surgery.

1. MEDICATION IN NEUROLOGICAL PATIENTS

There are no data on the use of constipating agents in patients with neurological problems and faecal incontinence.

Dunn et al have compared “therevac” micro enemas (5ml stimulant enemas) to bisacodyl suppositories in spinal cord injury (SCI) patients and found that patients need over 45 minutes to respond to the suppositories. The micro enemas reduced both digital stimulation time and evacuation time, and could potentially reduce carer time by up to one hour per day, with obvious cost savings [254]. Another two randomised studies have found that the carrier is important for suppositories, with hydrogenated vegetable oil bisacodyl taking longer to work than polyethylene glycol based bisacodyl, assumed to be the result of sooner bioavailability as the former has to dissolve in body heat [255, 256].

2. BOWEL TRAINING IN NEUROLOGICAL PATIENTS

Many different interventions have been described as “bowel training” [257]. Most extol the virtues of a high fibre diet, adequate fluid intake, as much exercise as feasible, and a regular toileting regime to capitalise on the gastro-colic reflex. Where this fails to establish a habit, use of a suppository or digital stimulation is often advocated. In practice many patients resort to evacuation by Valsalva or straining. However, this can eventually lead to problems with haemorrhoids or even rectal prolapse.

Children with spina bifida have been found to respond to behaviour modification, a regular toilet habit and dietary manipulation, with laxative medication or evacuants as indicated [258]. Most programmes stress the importance of teaching the child to self-initiate toileting after each meal, rewarding successful defaecation and use of suppositories or an enema if there is no self-initiated bowel motion in 2 days.

A protocol of a step-wise programme for bowel management in spina bifida, has been developed [259]. In a prospective evaluation of this protocol 40 patients with spina bifida were entered, “social continence” (defined

as one or less episodes of faecal incontinence per month) was increased from 13 to 60% of subjects. Success was associated with younger age at onset of the programme, compliance with the prescribed regime and presence of the anocutaneous reflex. Of those who had been previously incontinent and who complied, 79% achieved continence.

Patients following a CVA have been found to develop a consistent elimination pattern by a programme of daily digital rectal stimulation [260]. Daily stimulation was more effective than alternate day stimulation, with 24/25 patients achieving success.

The most developed protocols for bowel management are with SCI patients. Patients with an upper motor neurone lesion are usually taught to stimulate reflex bowel emptying every 1-3 days by gentle rotation of a gloved finger in the anus until the rectal wall is felt to relax, or flatus is passed and the stool comes down. In a survey of 424 members of the Danish Paraplegic Association it was found that 65% use digital stimulation [261]. Lower motor neurone lesion patients often need to manually evacuate the rectum once or more per day to stay continent.

In a nursing home population, frequency defaecation and number of continent stools was increased significantly in a trial of prompted voiding for urinary incontinence [262]. The authors suggest that this incidental finding may relate to increase opportunity to sit on the toilet, together with improved mobility and fluid intake. This suggests that dependent individuals may increase bowel frequency simply by being able to access a toilet more frequently. This has particular relevance for people with a urinary catheter who may seldom be offered use of the toilet.

3. BOWEL WASHOUT REGIMES IN NEUROLOGICAL PATIENTS

There is limited research on optimal regimes and techniques, or on which fluids are the most effective. Shandling has developed a catheter incorporating an inflatable balloon and reported 100% success with 40% of 112 children with spina bifida, but did not make selection criteria clear or state length of follow-up [263]. Others are more cautious in their appraisal. Thirty one children with spina bifida who were dissatisfied with current bowel management were started using the catheter, using saline at 20ml/kg body weight every 24 or 48 hours [264]. There were 6 immediate dropouts and a further 9 had stopped using the catheter, at 30 months. Of those who continued to use the catheter the percentage of continent stools rose from 28 to 94% and the percentage of constipated stools dropped from 55 to 15%.

One group report 83 of their 190 patients with spina bifida using irrigation daily or on alternate days via a cone in the anus, with good compliance [265]. Details of the technique are given by Scholler-Gyure and colleagues [266]. Of 41 patients with spina bifida who had failed other bowel management, 66% were continent at a mean follow up of 33 months, seven had monthly incontinence and 7 weekly incontinence; none had daily incontinence. Minor side effects included abdominal pain, headaches and poor appetite, but these were rare. Parental satisfaction was high in 63% and good in 37%. 66% of the children rated continence as the most important advantage, but half felt that it took up too much time and energy. Six found irrigation painful and 3 unpleasant; five were dependent on others to help.

Where a rectal washout is incomplete a catheterisable continent conduit may be surgically constructed to achieve antegrade colonic irrigation (see ACE procedure, section G, below).

4. MANUAL EVACUATION IN NEUROLOGICAL PATIENTS

There is a group of patients who have little or no reflex activity in the lower colon (and so cannot stimulate peristalsis), who have little alternative to planned manual evacuation either done by themselves or a carer or nurse. The majority of SCI patients need to use this regularly [267]. The UK Royal College of Nursing has reviewed manual evacuation and suggested a procedure and safety points [268].

V. GENERAL MEASURES AND ADVICE

Patients with bowel problems often need considerable psychological support, together with teaching and information on the normal working of the bowel. Knowledge about peristalsis, the gastro-colic reflex and the normal co-ordination of internal and external anal sphincters can enable planning and development of coping strategies. A few patients present following sexual abuse and may need intensive psychological support.

Fibre supplements have been found to contribute to faecal incontinence in frail immobile people [198, 269]. Softer stool is more difficult to hold during an urge to defaecate, and is also more likely to passively leak. There are no trials on the effect of fibre reduction on faecal continence, but clinically a lot of patients derive benefit from moderating their fibre intake [270]. People with faecal incontinence which is not secondary to severe constipation may find it helpful to keep the stool firm and formed by moderating the fibre content of their diet. Immobile people are often found to have a

colon loaded with soft stool or coinstipation, which is more likely to leak than firm stool [198, 271]. Conversely, those with incontinence secondary to loose stool or constipation may benefit from additional fibre [272]. Fibre must be used with caution in immobile people as it has been found to increase the risk of faecal incontinence compared to placebo [269] and a soft impaction can be caused [271].

Caffeine seems to act as a gut stimulant for some people and clinically some patients with urgent defaecation benefit from caffeine reduction [270].

VI. MANAGING FAECAL INCONTINENCE

There are no perfect answers to the problem of coping with faecal incontinence. It is very difficult to find anything that reliably disguises bowel leakage and smell and very few products have been designed specifically for faecal leakage.

Most of the disposable pads used for urinary incontinence can be used for containment, but some people find them unnecessarily thick, bulky, and not exactly the right shape to contain anal leakage.

An anal plug has been developed to help people with faecal incontinence. It is designed to be worn inside the rectum to plug the entrance to the anus from the inside and is available in two sizes. Some people find an anal plug uncomfortable, or that it gives a constant feeling of needing to open the bowels. It has to be taken out before a bowel action, and so is not suitable for someone who needs to open the bowels very frequently. Christiansen [273] found that 11 of 14 patients (71%), withdrew because of discomfort. In another evaluation [274], the majority of patients (14/20) could not tolerate the plug due to discomfort. Four patients (20%) wished to continue to use a plug on a regular basis after the study, and two others on an occasional basis. However, for this minority who could tolerate a plug, it was highly successful in controlling faecal incontinence. There was no association between comfort in using the plug and anorectal sensitivity as measured by electrophysiological tests. It was not possible to predict which patients would benefit from plug use.

G. SURGERY FOR FAECAL INCONTINENCE

Standard surgical treatment for faecal incontinence has been postanal repair for idiopathic incontinence and external sphincter repair for traumatic faecal incontinence.

Although idiopathic faecal incontinence should be defined as incontinence in subjects where neither trauma nor underlying disease are recognized as cause of the condition, the patients with idiopathic faecal incontinence treated by postanal repair are usually confined to middle-aged and older women. The introduction of intra-anal ultrasound has revealed that up to 60% of these patients have an overlooked obstetric lesion of the external sphincter [275] which means that the standard treatment should be sphincter reconstruction and not postanal repair. The recognition that a large fraction of the patients previously treated as idiopathic incontinence in fact has an external sphincter lesion may explain some of the conflicting results published.

I. POST-ANAL REPAIR

The post-anal repair was originally designed to improve continence by restoring an obtuse anorectal angle and to elongate the functional length of the anal canal [76]. The operation is carried out with the patient in the lithotomy or prone jack-knife position according to the surgeons preference. Bowel preparation and perioperative antibiotic prophylaxis is used. The technique consists of a transverse post-anal incision and dissection of the intersphincteric space, opening of Waldeyers fascia and mobilisation of the rectum off the sacrum by finger dissection. The central pelvic floor muscles, i.e. the puborectalis, the ischiococcygeus and the pubococcygeus are sutured behind the rectum either by mass suture or by a layered suture. After the operation the patients should be instructed not to strain during defecation and a bulk laxative may be advisable. Pelvic floor exercises should be encouraged 3-4 weeks after the operation.

The initial results reported were encouraging, obtaining continence of faeces in 60 to 75% of the patients [276, 277] but later, critical assessment by independent observers could not confirm these results. In a study from St. Marks hospital where this technique was first described, only 26% of the patients were continent to faeces at a median follow-up of 6 years [278]. Similar

results were reported by Jameson et al [279] where only 28% of the patients experienced a major improvement after a median follow-up of 2 years compared to 83% after 6 months. A recent study from United States reported satisfactory continence in 35% of the patients after an average follow-up of 3 years [280].

Hypothetically, the same effect on anal continence should be obtained whether the puborectalis sling is shortened by suturing the muscle posterior or anterior to the anal canal. This was eventually demonstrated [281-3] and it was furthermore shown that neither post-anal repair nor anterior perineoplasty decreased the anorectal angle [282, 283]. A randomised study comparing anterior and posterior pelvic floor repair with total pelvic floor repair (combined anterior and posterior repair) showed a significantly better results for the latter operation which also was the only of the three which increased the length of the anal canal [284]. This difference may partly be explained by the knowledge provided by intranal ultrasound examination that 60% or more of these patients may have had an anterior external sphincter defect which would be repaired by the anterior sphincteroplasty.

Persisting incontinence after rectopexy for complete rectal prolapse, a condition which manometrically resembles idiopathic faecal incontinence, has also been treated by post-anal repair with results which do not differ from those described above [285, 286].

In conclusion, the long term effect of postanal repair for idiopathic anal incontinence is poor and there are no predictors for a successful outcome. It has been argued [280] that despite the low success rate, the absence of mortality and the low morbidity may justify its use in selected patients in whom conservative treatment has failed and who are not candidates for other surgical procedures.

II. ANAL SPHINCTER REPAIR

Traumatic lesion of the external anal sphincter, which in the large majority of patients is due to an obstetric tear, is treated by reconstruction of the external sphincter usually by an overlapping suture. The defect, which is diagnosed by intra-anal ultrasound, will in obstetric lesions always be located anteriorly whereas defects following anal fistula surgery are usually located in the posterior half of the external sphincter. Patients should go through conservative treatment including biofeedback training before sphincter reconstruction is performed. It is our experience, however, that patients with external sphincter lesion and severe incontinence (comparing to a Wexner score of 10 or more) usually will require surgery.

The procedure is carried out with the patient either in

the lithotomy or prone jack-knife position and with bowel preparation and antibiotic prophylaxis. In patients with obstetric lesions an anterior curvilinear incision parallel to the anal verge is made. The length of the incision should be limited to approximately 180 degrees antero-laterally. The scar tissue is dissected from the posterior vaginal wall to the level of the anorectal ring and lateral dissection is continued until solid external sphincter muscle is reached. The ends of the external sphincter are mobilised and the repair is performed by placing 3 to 4 2-0 polyglycolic acid mattress sutures which overlap the cut ends of the muscle and finally another 3-4 sutures to complete the overlap repair. In patients without a total disruption of the muscle an imbrication without division of the muscle may be performed. In patients with a lax internal sphincter imbrication of this muscle should be done [287] and in patients with signs of perineal descent 2 or 3 anterior levator sutures may be added. Faecal diversion is unnecessary since it offers no benefit in terms of wound healing or functional outcome, and is a source of morbidity [288].

Early results of external sphincter repair for obstetric lesions have generally been favourable with satisfactory continence in approximately 75% of the patients [287-293] but after 3 to 5 years this figure has decreased to roughly 50% [293, 294, 161]. Furthermore, higher age seems to reduce the success rate with inferior results reported in women over 40 and 50 years [292, 295]. Whether the repair is performed as an overlapping suture or as a simple end to end suture seems of no importance [296].

Some of the poor results are due to an insufficient repair, whether insufficiently performed or due to post-operative dehiscence of the repair, which can be demonstrated by intra-anal ultrasound at follow-up. Dehiscence may be due to infection which occurs in up to 24% resulting in disruption in about 10% [297]. In a study of 10 consecutive patients who had undergone external anal sphincter reconstruction by overlapping suture for obstetric lesions and who had no infection or wound disruption, ultrasound revealed sphincter defects in 4 of 5 patients who were still incontinent [298]. In such patients, repeated external sphincter reconstruction may be performed successfully [299] indicating that intra-anal ultrasound should be part of the follow-up procedure in patients with persisting incontinence.

Damage to the pudendal nerve expressed by a prolonged pudendal nerve terminal motor latency has been claimed to be a predictor of poor outcome of sphincter repair for anal incontinence due to obstetric lesion [119]. Recent studies have, however, do not support this [292, 294].

In conclusion, external sphincter repair for traumatic,

mainly obstetric, lesions results in satisfactory long-term continence in approximately 50%. The principal predictors for a successful outcome are age and post-operative infection.

III. NOVEL SURGICAL APPROACHES

In patients with refractory end stage faecal incontinence who have failed or who are not candidates for sphincter repair or other standard therapy more advanced surgical procedures may be tried. These are dynamic graciloplasty, implantation of an artificial anal sphincter and sacral nerve stimulation.

1. DYNAMIC GRACILOPLASTY

Graciloplasty for faecal incontinence was originally developed for use in paediatric surgery [300]. It gained a certain use also for incontinence in adults [301, 302], but the functional results were too unreliable. Since striated muscles are composed of a mixture of fatigue-resistant type 1 fibres and fatigue-prone type 2 fibres they are usually not able to sustain contraction for more than a few minutes which means that it can only to a limited degree act as a sphincter. By electrical stimulation which replaces voluntary contraction, fatigue-prone fibres are converted to fatigue-resistant fibres which is the physiological basis for the use of a stimulated gracilis transplant. The anatomical basis for use of the gracilis muscle as an anal sphincter is the fact, that the muscle receives its vascular and nerve supply through one bundle at its proximal end which allows mobilisation of the full length of the muscle from its insertion at the tibial tuberosity.

The operation is carried out with the patient in the lithotomy position. The gracilis muscle is mobilized to its insertion in the tibial tuberosity through one long or two short incisions. Care must be taken not to damage the neurovascular bundle. The muscle is transposed around the anal canal in subcutaneous tunnels through two perianal incisions at 3 and 9 o'clock. The tendon of the muscle is usually fixed to the ischial spine, but may also be fixed to the skin. Intramuscular electrodes are implanted at the site of nerve entry and connected through a subcutaneous tunnel to a neurostimulator placed in a subcutaneous pocket in the abdominal wall. Prophylactic antibiotics are given from 1 to 3 days. Before continuous stimulation is applied the muscle is trained for 4 to 8 weeks according to a protocol. The amplitude, pulse width, rate and duty cycle can be programmed telemetrically.

Satisfactory continence has been reported in 50 to 73% of the patients [302-4]. Complication rate is rather high and especially infectious complications which occur in

up to 30% of the patients are followed by a high rate of therapy failure [304, 305]. The most serious of these complications are necrosis of the transplant and erosion through the anal canal. Impaired rectal emptying without sign of anatomical obstruction but with a severe impact on quality of life has been described in all series. A multicentre study showed a convincing relationship between volume of patients and success rate as well as rate of complications indicating that this therapy should only be carried out in specialist centres with a reasonably large number of patients [306].

Since impaired rectal emptying with severe impact on quality of life has occurred in a few patients in all series, a history of obstructed defecation should probably be a contraindication for this procedure.

2. ARTIFICIAL ANAL SPHINCTER

Implantation of an artificial anal sphincter was first reported in 1987 [308]. The sphincter used was originally designed for treatment of urinary incontinence but subsequently the device has been modified to meet the demands of a bowel sphincter, i.e. higher closing pressure and increased strength of the cuff. The system consists of an inflatable cuff placed around the upper anal canal, a pressure-regulating balloon to maintain closure of the cuff placed in the subperitoneal space lateral to the bladder and a control pump accessible to the patient to empty the cuff for defaecation placed in the scrotum or labium. There is a button on the pump which allows for deactivation and activation of the system. The cuff is available in lengths from 10 to 12 cm and in widths from 2 to 3.4 cm. In the reported series the size mostly used has been a length of 11 cm and a width of 2.9 cm. The pressure-regulating balloon contains 40 ml of fluid and is available in pressure ranges from 60 to 110 cm H₂O.

The patients should have a full bowel preparation and peri-operative antibiotic prophylaxis from 1 to 3 days. The procedure begins with implantation of the cuff around the anal canal. This is done either through two perianal incisions at 3 and 9 o'clock or through one transverse perineal incision. A tunnel is created around the anal canal preferably at the anorectal junction and posteriorly above the anococcygeal ligament. Through a suprapubic incision the pressure regulating balloon is placed in the laterovesical subperitoneal space. From this incision the pump can be placed in the scrotum or labium by blunt dissection and the components are connected by tubings through subcutaneous tunnels. The system is left deactivated for 4 to 6 weeks. No diverting stoma is used.

In the first published series of 12 patients the indication had mainly been faecal incontinence due to neuro-muscular disease, a condition where no other treatment was

available [308]. Subsequent series have mainly included patients where sphincter repair for traumatic lesions has failed and patients with idiopathic incontinence and anal atresia [309-313].

All series are relatively small with 6 to 14 patients from each participating centre. Acceptable continence has been obtained in approximately 70% of the patients. One series with long-term follow up (more than 5 years) showed that 7 of 17 patients had the system removed due to infection, malfunction or obstructed defaecation. It should be emphasised, however, that the majority of these patients had an implant either with an unmodified urinary sphincter or with one of the earlier modifications of the sphincter; the patients available for follow-up all had good to acceptable continence [314].

Complication rate in most series has been relatively high with infection around the device being the most serious and responsible for removal in up to 23% of the patients [315]. Technical complications like rupture of the cuff which occurred frequently with the earlier modifications of the device are now rare. Obstructed defecation without anatomical stenosis as described for dynamic graciloplasty has also occurred in most series and has in some patients required explantation. Other complications leading to explantation have been erosion of the cuff through the skin or into the anal canal which emphasises the importance of placing the cuff as close to the anorectal junction as possible and not to overpressure the system. At present a total removal rate of 20-25% probably must be expected, an important fact to discuss with patients prior to the operation.

Implantation of the artificial anal sphincter may be done on the same indications as for dynamic graciloplasty except in patients with previous perianal infections or with a thin and scarred perineum where a muscle transplant is preferable. It should be emphasised that due to the relatively high risk of treatment failure and of complications requiring re-operation patient selection for both procedures should be very strict.

3. SACRAL NERVE STIMULATION

One of the newer modalities for faecal incontinence is stimulation of the sacral nerves through the foramina of the sacrum. The technique is at present under evaluation but in contrast to dynamic graciloplasty and the artificial sphincter it is not a sphincter replacement therapy and consequently requires an anal sphincter with some degree of function.

This means that its main indications probably will be incontinent patients with an intact anal sphincter, i.e. idiopathic incontinence, patients who are still incontinent after external sphincter repair for traumatic lesions, incontinence after spinal lesions and patients with persisting incontinence after surgery for rectal pro-

lapse and possibly also patients with continence disturbances after low anterior resection for rectal cancer.

Unlike the two other operations this is a minimally invasive procedure which is usually performed in two steps. The operation is carried out with the patient in the prone position. The outline of the sacral bone is marked on the skin and the sacral foramina are located. Sheathed needles are inserted in the sacral foramen of S2, S3 and S4. After placement of the needles current is applied in graduated amplitude until a muscle response is obtained, monitored visually and by anal electromyography and pressure recording. Typically, S2 stimulation results in contraction of the perineum and usually also in an inward movement of the heel and contraction of the toes and foot, S3 stimulation results in a clamp-like contraction of the levator ani and plantar flexion of the great toe, and S4 stimulation in contraction of the levator ani but no leg or foot activity. The nerve that gives the most efficient contraction is chosen for temporary stimulation.

An incision is made over the sacrum with the needle left in place and when the foramen is identified the needle is replaced by a stimulating electrode which is fixed to the periosteum of the sacrum with two non-absorbable sutures. After having checked the efficiency of stimulation, the electrode is through a subcutaneous tunnel to the flank connected to a temporary pulse generator. The system is now tested for 2 to 3 weeks, and if the patient gains satisfactory continence a permanent pulse generator is implanted by the same technique as described for dynamic graciloplasty. The operation requires no bowel preparation but antibiotic prophylaxis from 1 to 3 days is recommended.

It is possible in some patients to replace the first step of the operation with an even less invasive test procedure simply by inserting temporary stimulating electrodes into the sacral foramina through the needles (percutaneous needle electrodes).

The first results with this technique was published by Matzel et al in 1995 [316] who described a successful outcome in three patients with traumatic incontinence in two and incontinence after rectopexy in one. Similar encouraging results both for short term and permanent stimulation have been reported from other centres [317, 318]. Satisfactory long-term continence has been described in a series of 6 patients with a follow up from 5 to 66 months [319].

4. INJECTABLE BIOMATERIAL

Perianal injection of fat [320], glutaraldehyde cross-linked collagen [321] and silicone biomaterial [322] in patients with passive anal incontinence due to internal sphincter dysfunction or disruption have been successfully reported in small series.

IV. ANTEGRADE LAVAGE (ACE PROCEDURE)

An option which should be considered in patients unsuitable for the procedures described above is the antegrade colon lavage through an appendicostomy originally described for faecal incontinence in children [323]. A recent study where the technique was used for faecal incontinence in 10 adults showed that 6 gained satisfactory continence [324].

V. COLOSTOMY

A number of patients in whom the surgical procedures described above are inapplicable or do not lead to satisfactory bowel control should be offered a permanent colostomy. With the stoma appliances now available a colostomy offers the patient a far more social acceptable life than severe uncontrolled faecal incontinence. There are no published studies comparing quality of life in patients with anal incontinence with and without a colostomy.

VI. SUMMARY

There is a number of surgical options for the treatment of faecal incontinence.

Post anal repair is associated with poor long term results (4).

Overlapping anal sphincter repair achieves good long term results in about 50% of patients (4).

In patients where simple sphincter reconstruction fails or is unsuitable there is a number of newer options available. The role of these is as yet not well-established, nor are the long-term success rates. Some of these methods carry a considerable risk of side effects and consequently a careful patient selection is mandatory.

Sacral nerve stimulation for faecal incontinence is a promising therapy but indications and contraindications are still not clarified.

VII. RECOMMENDATIONS

Surgery should normally be reserved for patients who have failed conservative therapy, except in cases of severe sphincter disruption and symptoms (D).

Surgery is seldom, if ever, indicated for minor symptoms (D).

H. FAECAL INCONTINENCE IN CHILDREN

I. INTRODUCTION

Faecal incontinence during childhood may be a symptom of delayed acquisition of toileting skills or may reflect serious underlying organic or functional pathology. Whatever the cause, the social and psychological consequences for the child and their family are often profound. For management to be successful the physical and psychological components will need to be addressed.

There are considerable cultural differences in toilet training and across the world children acquire bowel control at anytime between 1 year and 4 years of age [325-7]. Children tend to be trained earlier when the carer is able to immediately respond to signals that the child is about to defaecate. The trend in Western cultures has changed in the past 30 years to allow a more child-oriented approach in which the behaviour of the child dictates when toilet training is introduced [328]. In the USA the average age at which bowel control is gained is 28 months and the vast majority of children will have acquired bowel control by 4 yrs of age [329].

The pattern of bowel actions changes during early life from an average of 3 stools per day in the neonate to 1.7 stools per day at 1 year of age. 97% of preschool children in the UK will pass stool within the range 3 times per day to alternate days [330].

Epidemiological data on faecal incontinence in the normal childhood population is variable. Bellman observed a prevalence of 1.5 % among 7 yr old Swedish children [331] and Rutter in the UK reported a similar prevalence in 10-11 year olds [332]. It accounts for 3% of referrals to a medical clinic in Boston [333] and 25% of referrals to specialist paediatric gastroenterology clinics.

II. DEFINITIONS

Differences in definition can lead to confusion when comparing literature. In the USA encopresis is defined as "involuntary faecal soiling in the presence of functional constipation, in a child over the age of 4 years". Within the UK and Australian literature this is usually called "soiling" with the term encopresis being reserved for those where there appears to be voluntary passage of stool into a socially unacceptable place, implying a large behavioural element. This is a far less common cause of incontinence.

A Multinational Consensus Document On Functional Gastrointestinal Disorders: Rome II [334] identifies functional faecal retention and functional non-retentive soiling as separate entities with the following descriptions:

“Functional faecal retention is the most common cause of constipation and faecal soiling in children. It consists of repetitive attempts to avoid defecation because of fears associated with defaecation. Consequently, a faecal mass accumulates in the rectum.”

“Functional non-retentive soiling may be a manifestation of an emotional disturbance in a school-aged child. Soiling episodes may have a relationship to the presence of a specific person (e.g., a parent) or time of day, and may represent impulsive action triggered by unconscious anger.”

III. CAUSES OF CONSTIPATION AND FAECAL INCONTINENCE IN CHILDHOOD

1. ORGANIC

Table 16 gives the most common organic causes of childhood bowel symptoms.

The first role of the paediatrician is to identify those few children with underlying organic disease such as Hirschsprung’s Disease, anorectal anomaly and those with a neuropathic bowel. One in 5000 children is born with Hirschsprung’s disease. Reports of faecal incontinence after surgery vary widely. Several studies show a prevalence of 10 – 20% [335, 336] but Catto-Smith and

Table 16 : Organic causes of constipation and faecal incontinence in childhood

Neuropathic – meningocele, spinal dysraphism, spinal tumour, sacral agenesis, Cerebral palsy, dystrophia myotonica

Anorectal anomaly – anal atresia, stenosis, ectopic anus

Acquired anal conditions - anal fissure, Group A streptococcal infection, lichen sclerosis et atrophicus, anal sexual abuse

Congenital bowel disorders – Hirschsprung’s, neuronal intestinal dysplasia, chronic intestinal pseudo-obstruction

Miscellaneous medical conditions

Hypothyroidism
Hypercalcaemia
Cow’s milk protein intolerance

Drugs

Opiate analgesia
Loperamide
Anticonvulsants
Oxybutynin

colleagues in Melbourne have described soiling in 80% of 60 patients followed up by questionnaire and interview [337].

Ninety percent of those with meningocele will experience faecal incontinence of some degree [338]. It is important to identify children with less overt spinal abnormalities, such as spinal dysraphism and sacral agenesis as the majority in this group will also have a neuropathic bladder. Preservation of the upper renal tract from reflux and infection is a priority in order to protect future renal function.

2. FUNCTIONAL RETENTIVE SOILING

95% of children who soil have no inherent bowel or neurological abnormality but are incontinent as a result of functional constipation. There has been considerable debate as to the relative influences of psychological, behavioural and physical factors in the development of constipation but the cause of severe constipation is probably multifactorial.

In young children, stool holding is a common antecedent of constipation seen in 13% of 480 children followed through the toilet training process by Taubman [329]. Parents may describe manoeuvres and postures adopted by the child that almost certainly represent avoidance of defaecation. Such behaviour is reinforced by episodes of painful defaecation, as with anal fissure. In older children, reluctance to use toilet facilities at nursery and school may also precipitate stool holding and subsequent constipation (Ross and Price – personal communication).

The stool in overflow soiling may be loose and therefore interpreted as diarrhoea. 10-20% of children are thought by their parents not to be constipated [339, 333]. However in these children, the fact that they have little sensation of the passage of this stool should alert the paediatrician to the likelihood of constipation as the underlying cause of the soiling.

3. FUNCTIONAL NON-RETENTIVE SOILING

Children over four years who do not have underlying constipation and have no organic or anatomical abnormality are defined as having “non-retentive soiling”. They often pass normal stools into clothing at normal stool frequencies. Day and night time enuresis is commonly associated.

Primary and involuntary soiling may be due to delayed toilet training. This may be associated with general developmental delay or with the group of interrelated conditions that includes developmental coordination disorder, autism, other language impairments and attentional difficulties such as Attention Deficit Hyperactivity Disorder

[340]. Functional constipation is also common in this group.

Secondary or voluntary soiling is more likely to have an underlying psychological cause especially when it is related to specific and identifiable triggers. Children with a history of abuse or neglect often have continence problems with soiling in 26% [341]. However chronic faecal retention needs to be excluded.

IV. SECONDARY EFFECTS

1. PSYCHOLOGICAL AND BEHAVIOURAL

Whatever the cause of faecal incontinence, loss of bowel control is one of the most devastating symptoms a child can suffer. Soiling results in increased anxiety and loss of self-esteem. There are significant negative effects on relationships with family members and at school where bullying can become a serious problem. Children are often blamed for being “lazy” as it is not understood that they have little or no bowel control. Many children respond by denial and will hide soiled pants rather than admit to an accident. The whole family, not just the affected child, can experience guilt and failure with associated shame and secrecy leading to isolation [342]. Parents and other carers often give confusing messages by being cross and punitive at times but encouraging and forgiving at others. Continuing problems can lead to “learned helplessness” as all attempts to control the soiling fail [343-5]. Behaviour problems defined by parents are found in up to 40% of children with soiling (344, Butler, personal communication) but these are not generally as severe as in children referred to child mental health services [346]. Most of these behaviour difficulties resolve with successful treatment of soiling indicating they are likely to be secondary. However some children with severe behavioural difficulties associated with poor intra family relationships do not have good outcomes [347]. Breaking these negative cycles by engaging the child and family with appropriate explanations in a non-blaming fashion is vital. It should generate the motivation to cooperate with the management of the condition.

2. EDUCATIONAL

Although up to 40% of children will not have gained full bowel control by entry into nursery at 3 years of age, lack of adequate provision for these children can cause difficulties. In the UK, Department of Health guidance suggests that schools should “recognise the need for unrestricted access to non-threatening toilet facilities”. However, school toilets are often unsatisfactory or viewed as such by children [348].

V. ASSESSMENT

The aim of assessment is to identify the cause of faecal soiling and especially identify those children where there may be an underlying organic condition. Other related problems need exploring and an appropriate management plan developed that takes into account the whole child and family.

1. HISTORY

A careful history is vital. The areas that should be covered are given in Table 17.

2. EXAMINATION

The soiling child should be examined as follows:

Growth and general overview to exclude failure to thrive and neglect.

Abdomen – distension, palpable faecal mass.

Anus and genitalia – careful inspection for abnormality.

Perianal sensation, inspect lower back, spine and buttocks, check lower limb reflexes to exclude any suggestion of neuropathy.

Rectal examination is not usually necessary and may cause distress to younger children.

3. INVESTIGATIONS

In functional constipation few investigations are indicated. Cow’s milk protein intolerance may present as intractable constipation and should be considered in an atopic child [349].

Anal manometry and rectal biopsy are indicated if Hirschsprung’s disease is a possibility. This should be suspected with a history of delayed passage of meconium or severe constipation starting shortly after birth. Endoanal sonography may provide information about external anal sphincter configuration.

Where there is doubt and perhaps when parents need convincing evidence a plain abdominal X ray can show significant faecal loading and gross rectal enlargement. A rectopelvic ratio greater than 0.61 has been suggested as demonstrating enlargement [350]. Scoring systems have been devised in an attempt to quantify faecal loading [351, 352]. Measurement of bowel transit time using radiological markers may contribute to the clinical picture [353].

• Anal Manometry

Anal manometry is an invasive investigation and is not regarded as routine. It may contribute useful information in children suspected of having a neuronal abnormality or where response to conventional treatment is poor.

Table 17 : History in childhood faecal incontinence

	Reason
Age of onset, any initiating factors. Primary or secondary	Constipating event?
Present stool habits – interval, size (any huge) and consistency	
Soiling – interval, amount, consistency, when and where.	
Coping strategies. Hiding soiled pants? School involved?	
Attitude of child, parent, school friends etc.	
Co existing conditions. Congenital or acquired disorders	Neuropathy?
Atopy	Cow's milk intolerance?
Medication (e.g. anticholinergics, anticonvulsants, opiates)	Constipating effect
Learning or attentional difficulties, language impairment.	Delayed toileting
Behavioural problems	1° or 2° to soiling?
Previous surgery – especially related to GI tract	Association
Previous GI symptoms	Constipation associated
Birth history – delay in passage of meconium, early constipation	Hirschsprung's
Family history of bowel related difficulties	Genetic or dietary
Social history, any past history of any type of abuse	Abuse associated
Toilet training history – delay, holding, toilet refusal.	Constipation
Age when could identify need to pass stool without prompting	1° or 2° soiling
Nocturnal enuresis, day wetting, frequency, urgency	Association
Diet (fibre content and balance)	Very low fibre, restricted
Fluid intake and type.	Poor total intake or milk in excess

Children with chronic constipation have significantly increased rectal volume [353] and rectal myohypertrophy [354]. 50% of 34 constipated children studied by Loening-Baucke had an abnormal increase in external sphincter activity during attempts to pass a balloon [354]. There is no evidence for any underlying abnormality in rectal sensation or rectal wall compliance in children with faecal impaction [350]. Anal manometry abnormalities have been shown to persist even after effective treatment of constipation and encopresis [354].

VI. MANAGEMENT

Most authors advocate a multidisciplinary approach in which psychosocial and biological issues are both addressed [356, 344]. Nolan and colleagues in a large randomised found a multimodal approach (disimpaction, maintenance laxatives and behaviour modification) and to be superior to behaviour therapy alone [357].

There are various components to a successful treatment plan (Figure 5):

1. EXPLANATION / “DEMYSTIFICATION”

It is crucial that parents, carers and the child understand the reason why soiling is occurring. The child is likely to have little or no sensation of soiling episodes. Acknowledging this is a relief for children who previously have not been believed, although it may cause guilt for those parents.

2. TOILETING PROGRAM

Establishing a normal and regular pattern of bowel evacuation is central to eventual success for children with soiling from any cause. Star charts and reward systems can be used to reinforce this behaviour. Externalisation of the bowel problem by using ideas such as goal scoring charts or beating that “sneaky poo” can be helpful. Behavioural programmes like these on their own have been shown to be of benefit [358] but are even more successful when used in conjunction with appropriate laxative medication [357]. Continuing follow up and support to maintain motivation is important.

3. DIET AND FLUID INTAKE

A well balanced diet with a reasonable fibre intake is likely to be helpful. Experimental studies have shown that increasing fibre results in shorter bowel transit times and stool with greater volume and water content [359]. Mean daily fibre intake in constipated children was statistically lower than that of controls in a series from Greece [360] but low fibre intake is not thought to be the only causative factor. Excessive consumption of milk or poor fluid intake probably contributes.

Figure 5 : Flow chart for management of faecal incontinence in children

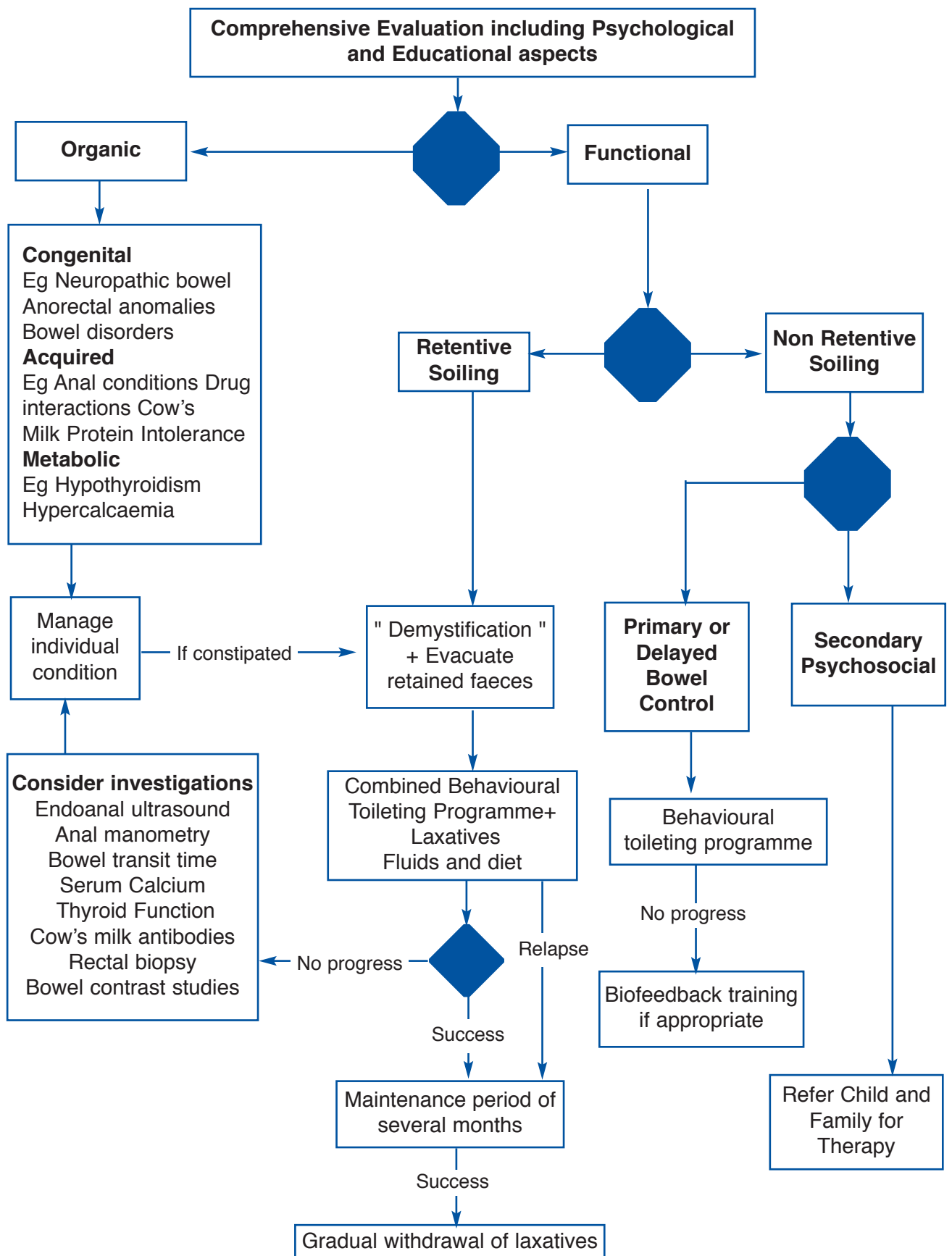


Figure 5 : Algorithmic approach to management of childhood soiling

4. LAXATIVES

There is general consensus that the child with constipation and overflow soiling requires laxative treatment with the aim of evacuating retained stool and maintaining regular bowel actions thereafter [343, 361, 362]. The evidence base to support the choice of laxatives is however small. Within the UK and Australia the common practise is to combine osmotic laxatives such as lactulose with stimulants such as senna, sodium picosulphate or sodium dioctyl. There are however very few relevant clinical trials and none which contribute significantly to the debate. Lubricants such as mineral oil provide the mainstay of treatment in USA often in combination with laxatives such as senna. Lipoid pneumonia has been described with mineral oil treatment and this should be used with caution in a child at risk of aspiration [363]. Faecal impaction can often be cleared with oral laxatives and lubricants in adequate doses, but in more resistant cases, enemas may be required. Many children find these distressing and effective evacuation of stool is often possible without resorting to rectally administered treatment [364]. Isotonic intestinal lavage with polyethylene glycol is effectively and clears retained faeces in severe refractory constipation [365, 366].

Once retained stool is cleared soiling will dramatically reduce. Various approaches have been used to maintain regular bowel actions – the mainstay being laxative treatment with behavioural approaches, designed to establish a regular toileting routine, enhance compliance and maintain motivation.

5. BIOFEEDBACK

Biofeedback training appears to have short term benefits [367, 368] but more recent controlled studies have not demonstrated that these are greater than the success following standard combined behavioural and laxative therapy with supportive follow up [369, 370]. There is some evidence it may be helpful in children who have nonretentive soiling [371].

6. COMPLEMENTARY THERAPIES

Abdominal massage with or without aromatology, reflexology, homeopathy and acupuncture can all be helpful, sometimes in conjunction with standard management, where they assist in establishing a regular toileting routine. Evidence base for these therapies is poorly established.

7. GENERAL SUPPORT

Faecal incontinence has socially isolating effects for the whole family. The network of support is generally less than for other chronic conditions but appropriate literature and advice can be very helpful. Within the UK, the

charitable organisation ERIC, Enuresis Resource and Information Centre [372], has done much to raise the profile of this disabling childhood problem.

8. SPECIALIST PSYCHOLOGY AND PSYCHIATRY SERVICES

Children whose soiling is associated with complex family functioning difficulties may need the expertise of a child and family mental health team.

VII. ASSOCIATED DISABILITIES

In those with structural or neuropathic abnormalities the aim is to achieve social continence. The treatment approach, once corrective surgery is complete, is remarkably similar – namely to remove faecal impaction and maintain regular bowel actions. Laxatives and regular toileting plans (with physical aids for those with additional disabilities) may be sufficient but in those with inadequate bowel emptying additional techniques such as use of enemas or rectal washouts may be required to prevent overflow soiling. Malone in 1990 introduced the surgical technique of the antegrade colonic enema (ACE) whereby the large bowel is irrigated via a caecostomy tube or appendix stoma [373, 374]. By keeping the large bowel empty in this way overflow soiling can be largely abolished. This technique has also been used in severe intractable functional constipation with megacolon [375].

VIII. OUTCOME OF CHILDHOOD FAECAL INCONTINENCE

Several studies have demonstrated the chronicity of this condition. The prognosis seems better in those diagnosed before the age of 4 years with recovery in 63% of children followed up by Loening Baucke [376]. In older children approximately 50% will have discontinued laxatives at 12 month follow up, with a further 20% coming off laxatives in the next 2 years [377-9]. In Clayden's series of over 300 children with severe constipation, laxative treatment was required by 56% for over 12 months [343]. At a mean of 6.8 years after treatment nearly 70% of 43 constipated children reviewed by Sutphen were entirely asymptomatic. Mild constipation persisted in 13. Faecal incontinence persisted in 3 of the 17 children who first reported it [380].

IX. SUMMARY

Most children have gained bowel control by 4 years of age [2].

The prevalence of faecal incontinence is around 1.5% at 10 – 11 years of age (2).

Functional results of reconstruction of congenital ano-rectal anomalies (eg imperforate anus and Hirschsprung's disease) may be poor. These children require long term follow up (3).

The vast majority of soiling children have functional retentive soiling secondary to constipation with no underlying organic abnormality. Stool holding is a common antecedent of constipation (3).

Psychological and behavioural problems are common and are usually secondary to the soiling. These improve when the child becomes continent (3).

Functional non-retentive soiling is less common and may be due to delay in establishing bowel control or to significant psychological and behavioural problems associated with other family and relationship difficulties.

Biofeedback training has been found useful for some children with functional non-retentive soiling (3).

A multidisciplinary approach to the management of functional retentive soiling is more likely to be successful. Parents and children need a clear understanding of the reasons why soiling is occurring in order to prevent intolerance and encourage compliance with the programme. Behavioural issues need to be addressed in conjunction with a combined laxative and toileting programme (2).

Laxative therapy may be needed for many months to maintain regular bowel actions (2).

Outcome is generally better when the condition is diagnosed early (2).

Figure 5 gives an algorithm for management of soiling in children.

X. RECOMMENDATIONS

- A comprehensive and holistic assessment is necessary with consideration of family, psychological and educational issues (D).
- The few children with organic causes of faecal incontinence must be identified, investigated and managed appropriately. Children with a neuropathic bowel or congenital bowel anomalies should be managed within specialist paediatric units (D).
- There is often a considerable delay before children with FI present to knowledgeable health professionals indicating a need for general health promotion and professional training in this area.

- Levels of evidence and research into the most common cause of FI in children – functional retentive soiling – are generally poor although combined laxative and behavioural toileting programmes have been shown to be more effective than either alone (B).

I. ANAL INCONTINENCE IN THE OLDER ADULT

I. INTRODUCTION

Few medical symptoms are as distressing and social isolating for older people as anal incontinence (AI), a condition which places them at greater risk of morbidity, dependency, hospital admissions, and institutionalisation. Many older individuals with AI will not volunteer the problem to their general practitioner, and regrettably, doctors and nurses do not routinely enquire about the symptom. This 'hidden problem' therefore leads to social isolation and a downward spiral of psychological distress, dependency, and poor health. The condition takes its toll on carers also; anal incontinence surpasses even dementia as a leading reason for requesting nursing home placement.

• Data sources

A computer-assisted search of the English language literature using Medline (to present), Cochrane Library databases, reference lists from recent systematic reviews and book chapters.

II. EPIDEMIOLOGY OF ANAL INCONTINENCE IN OLDER PEOPLE – PREVALENCE AND RISK FACTORS

Details are presented in Section B (above); the following is a summary of current evidence. The studies reviewed include one prospective short-term cohort study [381]; all the others are cross-sectional surveys [45, 16, 382-6]. Risk factor analyses were unadjusted in several studies [383, 385], and retrospectively conducted in all but one [381].

1. SUMMARY

The prevalence of AI increases with age, particularly in the 8th decade and beyond (2). The prevalence of AI also varies with the general health of the patient, and is

therefore higher in the acute hospital and nursing home setting than in the community (2). The prevalence of AI in elderly people is equal or greater in men than women. Nursing home studies show great variability in prevalence between institutions (2). AI is often combined with urinary incontinence in older individuals (2). Impaired mobility, dementia and loose stool are primary risk factors for AI in older people (2).

2. RECOMMENDATIONS

Bowel continence status should be identified by direct questioning and/or direct observation in nursing home residents, hospital inpatients aged 65 and over, people aged 80 and beyond living in the community, and older adults with impaired mobility and/or cognition (B).

The high prevalence of AI in older men (B) should prompt exploration of aetiologies other than child-birth in ageing adults.

Variability of AI prevalence between nursing homes (B) implies that different standards of care may impact the occurrence of the condition.

Standardised definition of AI in older people would be useful in both clinical and research arenas.

Further epidemiological studies are required to further characterise AI in older people, particularly in the acute hospital and primary care settings. Such studies should include evaluation of unmet need for patients and carers.

III. ANAL INCONTINENCE IN OLDER ADULTS - THE 'HIDDEN' PROBLEM

In a British primary care study of patients aged 75 years and over, only half of those reporting AI (or their carers) had discussed the problem with a health care professional, and only one out of eight patients with daily AI had done so. The GP's involved in this study reported full knowledge of incontinence status in only 33% of patients with a continence problem [382].

In Australian survey of acutely hospitalised patients of all ages, only 1 in 6 of those reporting AI had the symptom documented by ward nursing staff [384].

A US nursing home study showed that nursing staff were aware of AI in only 53% of residents self-reporting the condition [387]. The kappa value for concordance between resident and nurse reporting of AI was only 0.34 (0.24-0.43 95% CI).

In an English nursing home study, only 4% of patients

with long-standing AI had been referred to their general practitioner for further assessment of this problem [199].

1. SUMMARY

Physicians and nurses have a low awareness of AI (2). Within the nursing home, there is a low rate of referral by nursing staff of residents to their primary care physicians for further assessment of AI (2), which may reflect a tendency toward conservative nursing management (e.g. use of pads only without further evaluation).

2. RECOMMENDATIONS

General practitioners, primary care nurses, hospital ward staff, and nursing home nursing staff should routinely enquire about bowel incontinence in older patients (B).

Older patients with AI should be further assessed for reversible causes, regardless of their institutionalisation status (B).

IV. THE AGEING LOWER BOWEL AND PATHOPHYSIOLOGY IN OLDER ADULTS WITH ANAL INCONTINENCE

1. QUALITY OF DATA

The findings from physiological studies of the lower bowel in older adults tend to be variable due to a) a variety of different techniques used in measuring anorectal function, b) unclear definition of the normative range of manometric measures for older people, c) poor matching between cases and controls of clinical factors which may affect gut function (e.g. level of mobility), or inadequate clinical information [388, 389] and d) small subject numbers in some cases [389-91]. Studies reviewed are cohort case-control to evaluate age-effect [392-4], young-old healthy subject comparisons [390, 391], and age- and sex-matched case-control studies of continent versus incontinent patients [388, 389, 395].

• Anorectal function in healthy older adults

Studies of age effect in healthy volunteers have shown a linear reduction with ageing in squeeze pressures in women after the age of 70, and in men from the 9th decade onwards [392, 393]. Age beyond 70 years was associated with reduction in basal pressures in both genders, but to a greater degree in women [392, 393]. A study of healthy adults in early old age showed no differences in sphincter pressures when compared with healthy young people [391].

Rectal motility appears to be unaffected by healthy ageing [390], but an age-related increase in anorectal sensitivity thresholds has been observed, starting at an earlier age in women than men [394].

2. ANORECTAL FUNCTION IN OLDER ADULTS WITH FAECAL INCONTINENCE

Rasmussen et al found prolonged pudendal nerve terminal motor latency ($>2.2\text{ms}$) in 34% of women aged over 50 with AI, though no relationship was observed between pudendal neuropathy and basal or squeeze pressures [396]. Advancing age was however related to declining basal pressures. Vaccaro et al similarly found an age-related increase in pudendal neuropathy in incontinent women, again unrelated to squeeze pressures [397]. Single fibre EMG in incontinent patients aged over 70 years showed increased fibre density in the external anal sphincter muscles compared with continent subjects, indicating some local reinnervation of these muscles following neurogenic damage [389].

Barrett et al. examined anorectal function in elderly medically frail incontinent patients and continent age- and sex-matched controls [388]. Individuals with AI had reduced internal anal sphincter pressures, and a lower threshold for expulsion of a rectal balloon. Patients with AI and dementia were more likely to exhibit multiple rectal contractions in response to rectal distension, though the role of these ‘uninhibited’ contractions in causing incontinence was unclear [388, 398].

A similar matched case-control study showed that elderly patients with rectal impaction and soiling had impaired rectal sensation (needing a larger volume before feeling the presence of a rectal balloon and the urge to void), lower rectal pressures during rectal distension, and impaired anal and perianal sensation (‘rectal dyschezia’) [395]. Basal and squeeze pressures were however unimpaired in these patients, and the rectoanal inhibitory response was well-preserved. The authors concluded that overflow AI is primarily due to locally secreted mucus from around an irritative rectal faecal mass leaking out, despite well-preserved anal sphincter integrity.

a) Summary

Anorectal function in healthy older persons is characterised by a tendency towards an age-related reduction in internal anal tone (2), and a more definite decline in external anal sphincter tone, especially in older women (2). An age-related decline in anorectal sensitivity in women has been observed (3), but rectal motility is well-preserved (3). Ageing alone however, appears to have little impact on anorectal function until later old age – from the 7th decade upwards in women and even later in men (2).

Age-related internal anal sphincter dysfunction is an important factor in faecal incontinence in elderly people (2).

Although pudendal neuropathy is an age-related phenomenon in women with AI (2), its significance as a predisposing factor for incontinence is unclear.

Stool impaction predisposing to overflow is related to rectal dyschezia in frail older adults, a condition characterised by reduced tone, increased compliance and impaired sensation (3b). Overflow AI is due to mucus secretion from around a rectal faecal bolus, rather than to impaired sphincter function (3).

b) Recommendations

- Overall, the physiological data suggests that AI should not be considered an inevitable consequence of ageing (B).
- Older adults with AI should be evaluated for age-related reduction in internal and external sphincter function (B).
- Older patients with AI require a digital examination to identify rectal stool impaction causing overflow (B). Patients who are unaware of the presence of a large faecal bolus in the rectum may have rectal dyschezia, and should be considered at risk of recurrent impaction with overflow (B)

V. CAUSES OF FAECAL INCONTINENCE IN OLDER PEOPLE

1. QUALITY OF DATA

Risk factor studies have been reviewed above, and there is little additional data on causes of AI in this population. The causes of AI in older people, unlike in younger adults, are often multifactorial. The aim of this section is to categorise AI in the older adult by primary cause in a clinically meaningful way, emphasising the identification of potentially reversible factors.

a) Overflow incontinence secondary to constipation and stool impaction

The prevalence of faecal impaction and overflow AI is high in nursing home patients, implying that institutionalisation may place older individuals at greater risk of this condition. Tobin et al showed overflow to be the underlying problem in 52% of nursing home residents with long-standing AI [199], while Read reported that faecal impaction was the main reason for acute hospitalisation in 27% of nursing home residents admitted over the course of a year [395].

While there is virtually no published data on causes of impaction and overflow, there are several studies evaluating potentially modifiable risk factors for constipation in older adults. Only one of these was prospective; Robson et al looked at baseline characteristics predictive of new-onset constipation in elderly nursing home patients, using the US Minimum Data Set instrument. Seven percent (n=1,291) developed constipation over a 3-month period. Independent predictors were white race, decreased fluid intake, Parkinson's disease, decreased mobility, arthritis, greater than 5 medications and dementia [399]. In mainly retrospective studies, other researchers have identified the following causes of constipation in older people:

Immobility - Symptoms of constipation, and level of laxative use have been shown to be less in older people who are more physically active [400-402].

Medication side-effects - Drugs most implicated are iron supplements, calcium supplements, calcium channel blockers, opiates, and non-steroidal anti-inflammatory drugs [403, 404]. Another important group of drugs affecting the bowel are those with anticholinergic properties (e.g. antipsychotics, tricyclic antidepressants, oxybutynin) which may induce irreversible colonic laxity with long-term use [402].

Parkinson's disease patients may suffer greatly from constipation because of dual pathologies of primary degeneration of dopaminergic neurons in the enteric nervous system resulting in prolonged transit throughout the entire gut [405], and pelvic dyssynergia resulting in rectal outlet delay and prolonged straining (406)

Low dietary fibre intake is common in the elderly, and has been associated (through meta-analysis) with increased stool weight and decreased transit time in healthy and constipated adults [407].

Low fluid intake in older adults has been related to slow colonic transit [408], and in young male volunteers over a 1-week period has been shown to reduce stool output [409]. Elderly people are at greater risk of dehydration due to impaired thirst sensation, and less effective hormonal responses to hypertonicity, while those in nursing homes and hospitals may in addition have functional reasons for being unable to drink.

Dementia predisposes individuals to rectal dyschezia [395, 398], possibly partly through ignoring the urge to defecate; a study in which young men deliberately suppressed defecation resulted in a prolongation of transit through the rectosigmoid with a marked reduction in frequency of bowel movements [410].

2. FUNCTIONAL INCONTINENCE

Functional incontinence occurs in individuals who are

unable to access the toilet in time due to impairments in mobility, dexterity, or vision. These patients may even have normal lower gut function. Epidemiological studies of nursing home residents (see above) have repeatedly shown that poor mobility is a strong risk factor for AI after adjustment for other variables [381, 16, 386].

3. DEMENTIA-RELATED INCONTINENCE

Patients with advanced dementia may have a neurologically disinhibited rectum, with a tendency to void formed stool once or twice daily following mass peristaltic movements. Tobin et al documented dementia as the primary cause of AI in 46% of nursing home residents [199]. These individuals are very commonly incontinent of urine also.

4. COMORBIDITY-RELATED INCONTINENCE

The following diseases may cause AI, and are more common in older people:

Stroke - Immediately following a stroke 40% of individuals are incontinent, and 10% remain so 6 months after the acute event [29]. There is very little data examining the pathophysiological basis for AI following stroke. AI in 3-month stroke survivors was shown to be more strongly associated with potentially modifiable factors of anticholinergic medication use, and functional difficulties in getting to the toilet, than with stroke severity or location in a multivariate adjusted analysis [411].

Diabetes mellitus - AI may occur in people with diabetic neuropathy affecting the gut through the dual mechanisms of a) bacterial overgrowth resulting from severe prolongation of gut transit causing the characteristic nocturnal diarrhoea and b) multifactorial anorectal dysfunction [412]. Case-control studies show that diabetic patients with AI have reduced basal and squeeze pressures, spontaneous relaxation of the internal anal sphincter, reduced rectal compliance, and abnormal rectal sensation [412, 413].

Sacral cord dysfunction - The neuropathophysiology of rectal dyschezia [18] is compatible with diminished parasympathetic outflow from the sacral cord. Conditions in older persons that could impair sacral cord function are ischaemia and spinal stenosis.

5. ANORECTAL INCONTINENCE

Age-related anal sphincter dysfunction is likely to contribute to the increased prevalence of AI in men and women in their 80's and beyond (see above). Rectal prolapse is also a cause of AI which occurs more commonly in older adults [393].

6. LOOSE STOOLS

Loose stool increases the risk of AI in normally continent older adults by overwhelming a functional but age-compromised sphincter mechanism. Forty-four percent of cases of AI in a prospective nursing home study had diarrhoea as a primary cause [386].

Potentially reversible causes of loose stools in older adults are:

Excessive use of laxatives - One-third of community-dwelling people aged 65 and over regularly take laxatives, far exceeding the prevalence of constipation in this population [414]. Laxative use has been linked to AI in nursing home residents, in particular ‘Codanthramer’ [385].

Lactose intolerance is an age-related phenomenon - Goulding et al. found a lactose malabsorption rate of 15% in healthy women aged 40-59 years as compared with 50% in those aged 60-79 [415].

Antibiotic-related diarrhoea - Among hospitalised patients, age, female gender and nursing home residency significantly increases the risk for Clostridium difficile-associated diarrhoea associated with antibiotic use [416].

7. SUMMARY

Overflow incontinence secondary to stool impaction is a primary cause of AI in nursing home residents (3).

There are multiple potentially modifiable causes of constipation in older people (2)

Older people may be incontinent because they are unable to use the toilet for functional reasons (2)

Dementia-related AI is an important cause of AI in nursing home residents (2b)

AI is a common complication following stroke (2), but causes other than stroke status itself may also contribute to incontinence in stroke survivors (2).

Loose stools predispose elderly people to soiling (2), and have potentially reversible causes (2).

8. RECOMMENDATIONS

- Nursing home residents with AI should be evaluated for the presence of overflow and impaction (B)
- Older patients with constipation should be carefully assessed for potentially modifiable causes of immobility, medication side-effects, low fibre intake, and low fluid intake (B).
- Functional AI should be identified early on in the assessment process (B)
- Cognitive status should be assessed when evaluating causes of AI in older individuals (B)
- The symptom of loose stools should be elicited in older people, and reversible causes sought (B).

VI. ASSESSMENT AND DIAGNOSIS OF FAECAL INCONTINENCE IN OLDER ADULTS

Table 18 summarises the clinical evaluation of faecal incontinence in older people. The emphasis is on a structured comprehensive clinical approach. In most cases this approach will provide sufficient diagnostic information on which to base a feasible management plan without resorting to more specialised tests and assessments.

Table 18 : Assessment and diagnosis of faecal incontinence in older people

Emphasis in older people is on a *structured clinical approach* to identify all contributing factors for faecal incontinence

• History

Duration of faecal incontinence

Frequency of episodes

Type (soiling, small amounts, complete bowel movement)

Stool consistency Stool-stained mucus

Passive or urge AI

Constipation symptoms / current laxative use

Systemic illness (confusion, depression, weight loss, anemia)

Antibiotic use

• General examination

Abbreviated Mental Status Examination (score of 6 or less out of 10 suggests significant cognitive impairment)

Mood assessment (for depression)

Neurological profile (stroke, autonomic neuropathy, Parkinson’s disease)

• Toilet access

Evaluate ability to use toilet based on muscle strength, coordination, vision, limb function, and cognition.

Place in context of current living environment.

• Specific examination

Abdominal inspection for distension and tenderness

Perineal inspection for skin breakdown, dermatitis, surgical scars

Observe for excessive downward motion of the pelvic floor when asking patient to bear down in the lateral lying position

Digital examination for stool impaction

Digital examination for evaluation of impaired sphincter tone

- Anal gaping, and/or easy insertion of finger (internal sphincter)

- Reduced squeeze pressure (external sphincter)

Ask patient to strain while sitting on commode and observe for rectal prolapse

• Tests

An abdominal Xray should be performed to look for stool impaction in the colon

In the case of diarrhoea – send stool sample for culture, including C.Difficile

Blood tests for full blood count, glucose, urea and electrolytes, calcium, thyroxine

There is however much room for improvement in this clinical area; current surveys indicate a lack of thoroughness by doctors and nurses in assessing faecal incontinence in older people in all settings (community, acute hospital, and nursing home), with failure to obtain an accurate symptom history or to perform rectal examinations. A recent UK survey of physician practice in performing rectal examinations in acutely hospitalised elderly patients found that of the 5% (n=10) of patients with AI, only two had undergone a rectal exam [417].

• **Data quality**

There are few quality data evaluating methods of assessing and diagnosing AI in older adults. The information presented below is largely derived from review articles, and non-age specific studies.

• **Results**

Documentation of the *type of incontinence* is diagnostically helpful. Self-report of bowel symptoms relating to AI have been shown to be reliable and reproducible in older cohorts [43, 39]. Constant leakage of loose stool or stool-stained mucus is characteristic of overflow around an impaction, while patients with anal sphincter dysfunction tend to leak small amounts of stool. Engel et al. showed in younger adults that where external anal sphincter weakness predominates the patient often reports urgency prior to leaking (urge AI), while those with internal sphincter dysfunction tend to have unconscious leakage of stool (passive AI) [418]. Patients with dementia-related incontinence often pass complete bowel movements, especially after meals in response to the gastrocolic reflex.

Evaluation of *toilet access* should be multidisciplinary, and include a broad functional assessment (e.g. Barthel Index), mobility test (e.g. 'up and go' test), visual acuity test (count fingers), upper limb dexterity assessment (undoing buttons), and cognitive measure (e.g. Abbreviated Mental Test Score). For community patients, the health care provider should be aware of the physical layout of the patients home, and in particular bathroom details (location, distance from main living area, width of doorway for accommodating walking aids, presence of grab rails or raised toilet seat). Low lighting levels, high degree of clutter and hard to manage clothing may also be relevant.

Digital examination can reasonably assess anal sphincter tone in the clinical setting. Easy finger insertion with gaping of the anus on finger removal indicates poor internal sphincter tone, while reduced squeeze pressure around the finger when asking the patient to 'squeeze and pull up' suggests external sphincter weakness. Hallan et al. found that digital assessment of squeeze and basal tone was as sensitive and specific as manometry in discriminating sphincter function between continent and

incontinent patients aged over 50 [116].

A digital rectal examination is essential for identifying stool impaction, although an empty rectum does not exclude the diagnosis of constipation [400]. All incontinent patients without evidence of rectal stool impaction should ideally undergo a *plain abdominal radiograph* in order to a) establish or rule out the diagnosis of overflow, b) measure the extent and severity of faecal loading, c) evaluate the degree of bowel obstruction secondary to impaction, d) rule out acute complications of impaction such as sigmoid volvulus and stercoral perforation and e) identify colonic motility/dysmotility [419-21].

1. SUMMARY

Current evidence suggests that assessment of AI in older adults is suboptimal (2).

Documentation of the type of incontinence by self-report, proxy report or observation is diagnostically helpful (2).

Evaluation of ability to access and use the toilet should be multidisciplinary.

Digital assessment of sphincter tone can accurately discriminate between continent and incontinent adults (3).

2. RECOMMENDATIONS

- Standardisation of assessment methods of older adults with AI in all health care settings is required
- The emphasis in older people is on a structured clinical approach to identify multiple causes of AI (D), including cognitive and functional assessments (B).
- A careful bowel symptom history and assessment of voiding pattern should form part of the assessment (B).
- Digital assessment of sphincter tone in older people should be a first-line approach (B), with anorectal manometry testing reserved for a minority in whom surgery or biofeedback seems feasible (D).
- Digital rectal examination is essential in assessing older patients with AI, but those without evidence of rectal stool impaction should undergo a plain abdominal radiograph to rule out higher impaction and other problems (C).

VII. TREATMENT OF FAECAL INCONTINENCE IN OLDER ADULTS (Figure 6)

Quality of data

There are very few published trials of treatment of AI in

older people, and no trials on prevention of AI. The studies reviewed suffer from small numbers [199, 224], poor methodology (e.g. not applying intent-to-treat rule, unclear reporting of drop-outs) [199, 422], and no blinding (all). Randomised controlled trials examining effective laxative treatment for constipation in older adults also largely lack power, and are therefore unlikely to detect effects of treatment [423].

1. TREATMENT OF FAECAL IMPACTION AND OVERFLOW ANAL INCONTINENCE IN OLDER PEOPLE

Tobin et al. evaluated a therapeutic intervention in 52 nursing home residents with AI, based on treatment recommendations to general practitioners [199]. Patients with rectal impaction and continuous faecal soiling were classed as having overflow and recommended treatment with enemas until no further response followed by lactulose - complete resolution of incontinence was achieved in 94% of those in whom full treatment compliance could be obtained.

A French nursing home study of 206 frail elderly nursing home residents found that treatment of constipation was only effective in improving overflow AI (incontinence at least once weekly associated with impaired rectal emptying) when long-lasting and complete rectal emptying (monitored by weekly rectal examinations) could be achieved using daily lactulose, daily suppositories and weekly tap-water enemas [422]. Patients randomised to the group receiving lactulose only showed no reduction in frequency of AI. The analysis was not performed on an intent-to-treat basis and there was no description of the high number of subjects lost to follow-up.

Over half of nursing home residents take laxatives at least once daily, prompting speculation that nonpharmacological approaches to optimise management of constipation may be under-utilised in this setting [385]. A recent systematic review of effective laxative treatment in elderly persons found that RCT's in this area are potentially flawed due to small numbers, but nevertheless commented that significant improvements in bowel movement frequency have been observed with cascara and lactulose, while bulk laxative psyllium and lactulose have been reported to improve stool consistency and related symptoms in placebo-controlled trials [423]. Observational studies of nursing home residents (i.e. those at risk of impaction with overflow) suggest that stimulant and osmotic laxatives (lactulose and polyethylene glycol) may be effective [423, 424].

Although enemas are considered useful in the clinical setting for acute disimpaction and treatment of overflow, there are no data examining effectiveness other

than case-reports. Case-series suggest that phosphate enemas should be used with caution in people with renal failure (no more than once weekly) [425].

2. TREATMENT OF DEMENTIA-RELATED FAECAL INCONTINENCE

Ouslander et al showed that prompted voiding programmes significantly increased the number of continent bowel movements in an uncontrolled study of elderly nursing home residents with dementia-related incontinence over a period of a few weeks, but no impact was seen on frequency of AI [262].

Tobin et al evaluated a bowel programme in 25 nursing home residents with dementia-related AI, consisting of daily codeine phosphate and twice weekly enemas; continence was achieved in 75% of those fully treated [199].

3. TREATMENT OF AI IN OLDER ADULTS DUE TO ANAL SPHINCTER WEAKNESS

Biofeedback treatment for AI in older people resulted in a 75% reduction in incontinent episodes short-term in one small study of a highly selected group of patients with no cognitive impairment, good motivation and intact anorectal sensation [224].

Loperamide is recommended for treatment of anorectal incontinence [193], though there are no data on its efficacy in older people. In one small placebo-drug crossover trial in older adults, loperamide oxide significantly reduced visual analogue scores for incontinence and urgency, and both prolonged colonic transit and increased basal tone [195].

a) Summary

There are too few data to determine what constitutes effective laxative treatment of faecal impaction with overflow AI (1) Although good quality data are lacking, stimulant and osmolar laxatives may be effective in treating constipation in older people at risk of overflow (2).

Treatment approaches to dementia-related AI (prompted toileting, controlled evacuation) may be helpful in the nursing home setting (3).

Biofeedback is unlikely to help more than a minority of older adults with AI (3).

Loperamide is a useful drug treatment for anorectal AI in the absence of constipation (4).

b) Recommendations

All underlying causes of AI in each older individual should be identified and treated (D).

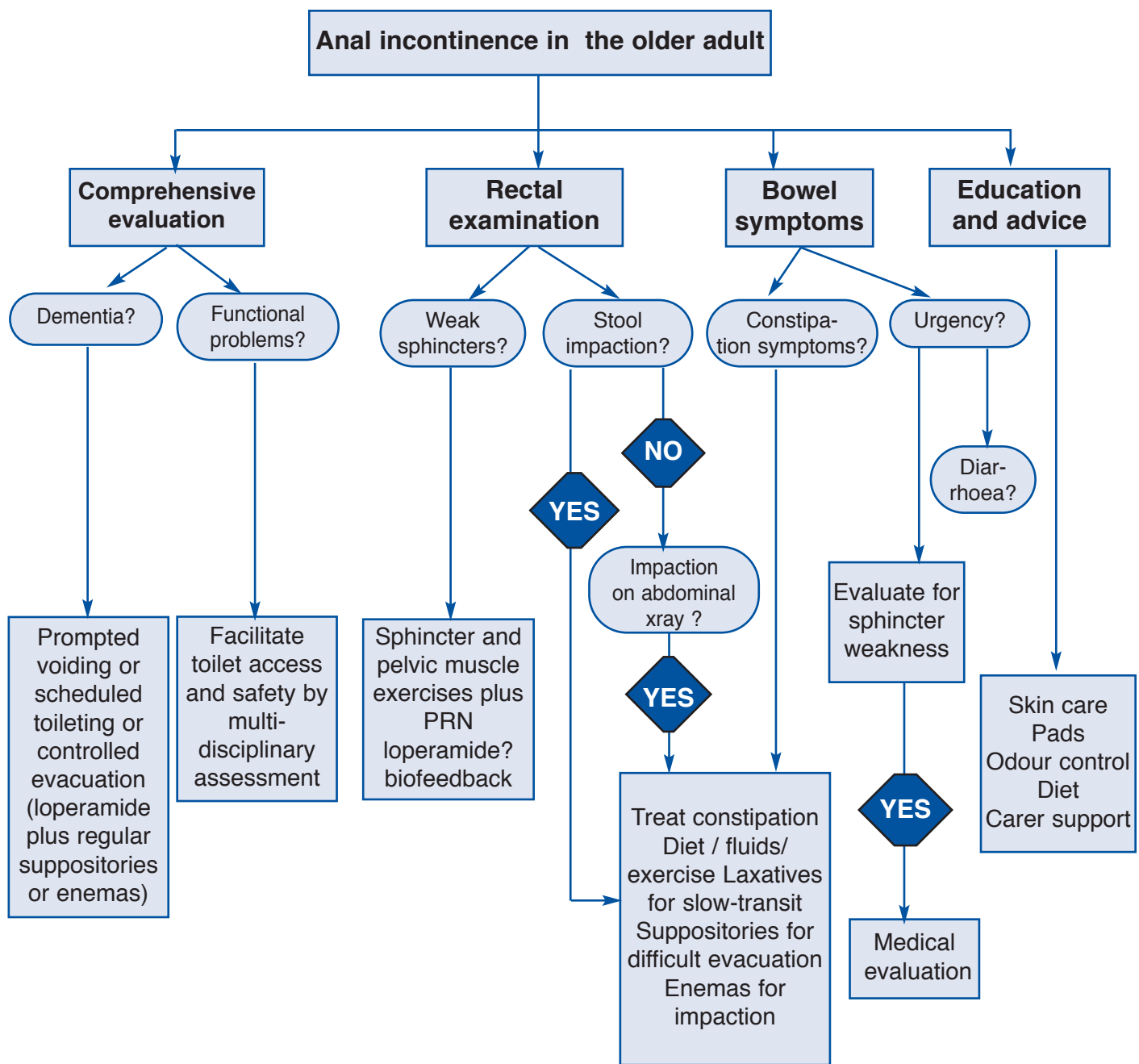


Figure 6 : Algorithmic approach to management of AI in older adults

As older adults with AI are not a homogenous group, a multicomponent research approach in the form of a randomised controlled trial evaluating a structured management protocols versus usual care would be helpful. Such a study should be sufficiently large to permit subgroup analysis of targeted treatment protocols are required.

RCT's of laxative and nonpharmacological treatment and prevention of faecal impaction in and overflow are needed to optimise standards of prescribing and care.

A multidisciplinary RCT assessing a step-wise approach to management of dementia-related AI in nursing home residents (prompted voiding in those with mild to moderate dementia, scheduled toileting plus suppositories next step, bowel programme of controlled evacuation in those with persistent incontinence) would be of great value.

Greater emphasis needs to be placed on systematic and effective management of faecal incontinence in older people backed up by sound communications between doctors and nurses, especially in the nursing home and acute hospital setting (D)

Patient and carer education should be undertaken to promote self-efficacy and other coping mechanisms, and where appropriate self-management (e.g. reducing risk of constipation and impaction through dietary and lifestyle measures, advice on how to take loperamide) (D). Advice on skin care, odour control, and continence aids is also important (D).

Figure 6 shows an algorithmic approach to management of AI in older adults.

REFERENCES

- Royal College of Physicians. Incontinence. Causes, management and provision of services. A Working Party of the Royal College of Physicians. *Journal of the Royal College of Physicians of London* 29, 272-274. 1995.
- Whitehead, W.E., Wald, A., Diamant, N.E., Enck, P., Pemberton, J.H. and Rao, S.S.C. Functional disorders of the anus and rectum. *Gut* 45, 1155-1159. 1999.
- Johanson, J.F. and Lafferty, J. Epidemiology of fecal incontinence: the silent affliction. *Am.J.Gastroenterol.* 91, 33-36. 1996
- Leigh, R.J. and Turnberg, L.A. Faecal incontinence: the unvoiced symptom. *Lancet* 1, 1349-1351. 1982.
- Small KA and Wynne JM. Evaluating the pelvic floor in obstetric patients. *Aust. New Zeal. Obstet. Gynecol.*, 30:41-45, 1990.
- Madoff RD, Williams JG and Caushaj PF. Current Concepts; Fecal Incontinence. *NEJM.*, 326:1002-7, 1992.
- Abou-Zahr C. Obstructed labour. In Murray CJL, Lopez AD. Health dimensions of sex and reproduction: the global burden of sexually transmitted diseases, HIV, maternal conditions, perinatal disorders and congenital anomalies. Cambridge. Harvard University Press, 1996.
- Drossman DA. What can be done to control incontinence associated with the irritable bowel syndrome? *Am. J. Gastroent.*, 84:355-57, 1989.
- Schiller LR, Sanat Ana CA, Schmulen AC, Hendler RS, Harford WV and Fordtran JS. Pathogenesis of fecal incontinence in diabetes mellitus. *NEJM.*, 307:1666-71, 1982.
- Thomas TM, Egan M, Walgrove A and Meade TW. The prevalence of faecal and double incontinence. *Comm. Med.*, 6:216-20, 1984.
- Campbell AJ, Reinken J and McCosh L. Incontinence in the elderly: prevalence and prognosis. *Age and Aging*, 14:65-70, 1985.
- Kok ALM, Voorhorst FJ, Burger CW, van Houten P, Kenemans P and Jansens J. Urinary and faecal incontinence in community-residing elderly women. *Age & Aging*, 21:211-15, 1992.
- Denis P, Bercoff E, Bizien MF, Brocker P, Chassagne P, Lamouliatte H, Leroi AM, Perrigot M and Weber J. Etude de la prevalence de l'incontinence anale chez l'adulte. *Gastroent. Clin Biol.*, 16:344-50, 1992.
- DA, Li Z, Andruzzi E, Temple RD, Talley NJ, Thompson WG, Whitehead WE, Janssens J, Funch-Jensen P, Corazziari E, Richter JE and Koch GG. U.S. Householder survey of functional GI disorders: prevalences, sociodemography and health impact. *Dig. Dis. & Sciences*, 38:1569-80, 1993.
- MacLennan AH, Taylor AW, Wilson DH, Wilson D. The prevalence of pelvic floor disorders and their relationship to gender, age, parity and mode of delivery. *BJOG*, 107:1460-1470, 2000
- Nelson RL, Norton N, Cautley E and Furner S. Community based prevalence of anal incontinence. *JAMA*, 274:559-562, 1995.
- Enck P, Bielefeld K, Rathmann W, Purmann J, Tschöpe D, Erkenbrecht JF. Epidemiology of faecal incontinence in selected patient groups. *Int J Colorectal Dis.*, 6:143-146, 1991
- Wisconsin Family Health Survey, Center for Health Statistics, Division of Health, Department of Health and Social Services, P.O. Box 309, Room 172, Madison Wisconsin 53701-0309, 1994.
- Nelson RL, Furner S, Jesudason V. Fecal incontinence in Wisconsin nursing homes. *Dis Colon & Rectum*, 41:1226-1229, 1998
- Borrie MJ, Davidson HA. Incontinence in institutions: costs and contributing factors. *CMAJ*, 147:322-328, 1992.
- Højberg KE, Salvig JD, Winslow NA, Bek KM, Laurberg S, Secher NJ. Flatus and faecal incontinence: prevalence and risk factors at 16 weeks of gestation. *BJOG*, 107:1097-1103, 2000.
- Donnelly V, Fynes M, Campbell D, Johnson H, O'Connell PR, O'Herlihy C. Obstetric events leading to anal sphincter damage. *Obstet Gynecol.*, 92:955-961, 1998.
- Donnelly VS, O'Herlihy C, Campbell DM, O'Connell PR. Postpartum fecal incontinence is more common in women with irritable bowel syndrome. *Dis. Colon & Rectum*, 41:586-589, 1998.
- Nygaard IE, Rao SSC, Dawson JD. Anal incontinence after anal sphincter disruption: a 30 year perspective. *Obstet. Gynec.*, 89:896-901, 1997.
- Wald A. Systemic diseases causing disorders of defecation and continence. *Sem Gastroint Dis.*, 6:194-202, 1995.
- MacKenzie WR, Hoxie NJ, Proctor ME, Gradus MS, Blair KA, Petersen DE, Kazmierczak JJ, Addiss DG, Fox KR, Rose JB and Davis JP. A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. *NEJM*, 331:161-7, 1994.
- Sullivan SN, Wong C. Runner's diarrhea. Different patterns and associated factors. *J. Clin Gastroent.*, 14:101-104, 1992.

28. Bliss DZ, Johnson S, Savik K, Clabots CR, Gerding DN. Fecal incontinence in hospitalized patients who are acutely ill. *Nursing Res.*, 49:101-108, 2000.
29. Nakayama H, Joergensen HS, Pedersen PM, Raaschou HO, Olsen TS. Prevalence and risk factors of incontinence after stroke: The Copenhagen stroke study. *Stroke*, 28:58-62, 1997
30. Pernikoff BJ, Eisenstat TE, Rubin RJ, et al. Reappraisal of partial lateral internal sphincterotomy. *Dis. Colon & Rectum*, 37:1291-5, 1994.
31. del Pino A, Nelson RL, Pearl RK, Abcarian H. Island flap anoplasty for treatment of transsphincteric fistula-in-ano. *Dis. Colon & Rectum*, 39:224-6, 1996.
32. Gorfine SR. Treatment of benign anal disease with topical nitroglycerin. *Dis. Colon & Rectum*, 38:453-7, 1995.
33. von Flue M, Harder F. A new technique for pouchanal reconstruction after total mesorectal excision. *Dis. Colon & Rectum*, 37:1160-2, 1994.
34. Ishigooka M, Hashimoto T, Izumiya K, Sasagawa I, Nakada T. Incidence of anal incontinence after long-term follow-up of patients treated by ureterosigmoidostomy. *Int Urol Nephrol.*, 25:455-460, 1993.
35. Norton, C. and Chelvanayagam, S. A nursing assessment tool for adults with fecal incontinence. *Journal of Wound, Ostomy, & Continence Nursing* 27, 279-291, 2000.
36. Talley, N.J., Phillips, S.F., Melton, L.J., Wiltgen, C., Zinsmeister, A.R.: A patient questionnaire to identify bowel disease. *Ann Intern Med.* 111:671-4, 1989.
37. Talley, N.J., Phillips, S.F., Wiltgen, C.M., Zinsmeister, A.R., Melton, L.J.: Assessment of functional bowel disease: The bowel disease questionnaire. *Mayo Clin. Proc.* 65:1456-79, 1990.
38. Manning, A.P., Thompson, W.G., Heaton, K.W., Morris, A.F.: Towards positive diagnosis of the irritable bowel. *Br. Med. J.* 2:653-4, 1978.
39. O'Keefe, E.A., Talley, N.J., Tanagalos, E.G. and Zinsmeister, A.R.: A bowel symptom questionnaire for the elderly. *Journal of Gerontology*. 47(4):M116-M1, 1992.
40. Best, W.R., Beckett, J.M., Singleton, J.W., Kern, F.: Development of a Crohn's disease activity index: National Cooperative Crohn's Disease Study. *Gastroenterology* 70:439-44, 1976.
41. Harvey, R.F., Bradshaw, J.M.: A simple index of Crohn's disease activity. *Lancet* 1:514, 1980.
42. Van Hees, P.A.M., Van Elteren, P.H., Van Lier, H.J.J., Van Tongeren, J.H.M.: An index of inflammatory activity in patients with Crohn's disease. *Gut* 21:279-86, 1980.
43. Osterberg, A., Graf, W., Karlsson, P., Pahlman, L.: Evaluation of a questionnaire in the assessment of patients with faecal incontinence and constipation. *Scand J. Gastroenterol.* 31:575-580, 1996.
44. Reilly, W.T., Talley, N.J., Pemberton, J.H. and Zinsmeister, A.R.: Validation of a questionnaire to assess fecal incontinence and associated risk factors. *Dis. Colon Rectum* 43:146-154, 2000.
45. Roberts, R.O., Jacobsen, S.J., Reilly, W.T., Pemberton, J.H., Lieber, M.M. and Talley, N.J.: Prevalence of combined fecal and urinary incontinence: A community based study. *J. Am. Geriatric Soc.* 47:837-841, 1999.
46. Bishoff, J.T., Motley, G., Optenberg, S.A., Stein, C.R., Moon, K.A., Browning, S.M., Sabanegh, E., Foley, J.P. Thompson, I.M.: Incidence of fecal and urinary incontinence following radical perineal and retropubic prostatectomy in a national population. *J Urol.* 160:454-458, 1998.
47. Hiltunen, K.M., Matikainen, M., Auvinen, O., Hietanen, P.: Clinical and manometric evaluation of anal sphincter function in incontinent patients. *Am J Surg.* 151:489-92, 1986.
48. Broden, G., Dolk, A., Holmstrom, B.: Recovery of the internal anal sphincter following rectopexy: a possible explanation for continence improvement. *Int. J. Colorectal Dis.* 3:23-8, 1988.
49. Kelly, J.H.: Cineradiography in anorectal malformation. *J. Pediatric Surg.* 4:538, 1968 and Parks, A.G.: Anorectal incontinence. *J. R. Soc. Med.* 68:21-30, 1975.
50. Shelton, A., Madoff, R.: Defining anal incontinence: establishing a uniform continence scale. *Semin. Colon Rectal Surg.* 8:54-60, 1997.
51. Holschneider, A.M.: Treatment and functional results of anorectal continence in children with imperforated anus. *Acta Chir Belg* 3:191-204, 1983.
52. Keighley, M.R. and Fielding W.L.: Management of faecal incontinence and results of surgical treatment. *Br. J. Surg* 70:463-468, 1983.
53. Rudd, W.W.: The transanal anastomosis: a sphincter saving operation with improved continence. *Dis. Colon Rectum* 22:102-5, 1979.
54. Womack, N.R., Morrison, J.F., Williams, N.S.: Prospective study of the effects of post-anal repair in neurogenic fecal incontinence. *Br. J. Surg.* 75:48-52, 1988.
55. Corman, M.: Gracilis muscle transposition for anal incontinence. Late results. *Br. J. Surg* 72:S21-22, 1985.
56. Rainey, J.B., Donaldson, D.N., Thomson, J.P.: Post-anal repair: which patients derive most benefit? *J.R. Coll Surg. Edinb* 35:101-105, 1990.
57. Rockwood, T.H., Church, J.M., Fleshman, J.W., Kane, R.L., Mavrantonis, C., Thorson, A.G., Wexner, S.D., Bliss, D., and Lowry, A.C.: Patient and surgeon ranking of the severity of symptoms associated with fecal incontinence. The Fecal Incontinence Severity Index. *Dis. Colon Rectum*. 42:1525-1532, 1999.
58. Pescatori, M., Anastasio, G., Bottini, C., Mentasti, A.: New grading and scoring for anal incontinence. *Dis. Colon Rectum* 35:482-487, 1992.
59. Jorge, J.M.N., Wexner, S.D.: Etiology and management of fecal incontinence. *Dis. Colon Rectum* 36:77-97, 1993.
60. Vaizey, D.J., Carapenti, E., Cahill, J.A. and Kamm, M.A.: Prospective comparison of faecal incontinence grading systems. *Gut*. 44:77-80, 1999.
61. Gill, T.M., Feinstein, A.R.: A critical appraisal of the quality of Quality of Life measurements. *JAMA* 272(8):619-626, 1994.
62. Maunder, R.G., Cohen, Z., McLeod, R.S., Greenberg, G.R.: Effect of intervention in inflammatory bowel disease on Health-Related Quality of Life: A critical review. *Dis. Colon Rectum* 38:1147-1161, 1995.
63. Stewart, A.L., Greenfield, S., Hays, R.D. et al: Functional status and well-being of patients with chronic conditions. Results from the Medical Outcomes Study. *JAMA* 262:907-912, 1989
64. Stewart, A.L., Hay R.D., Ware, J.E.: The MOS short form general health survey: reliability and validity in a patient population. *Medical Care* 26:724-35, 1988.
65. Hassink, A.A.M., Rieu, P.N.M.A., Brugman, A.T.M., Festen, C.: Quality of Life after operatively corrected high anorectal malformation: A long-term follow-up study of patients aged 18 years and older. *Journal of Pediatric Surgery*. 29:773-776, 1994.
66. Tiainen, J., Matikainen, M.: Health related quality of life after ileal J-pouch anal anastomosis for ulcerative colitis: Long term results. *Scand J. Gastroenterol.* 34:601-605, 1999.
67. Fazio, V.W., O'Riordan, Lavery, I.C., Church, J.M., Lau, P., Strong, S.A., Hull, T.: Long term functional outcome and quality of life after stapled restorative proctocolectomy. *Annals of Surgery* 230:575-586, 1999.

68. Guyant, G., Mitchell, A., Irvine, E.J. et al: A new measure of health status for clinical trials in inflammatory bowel disease. *Gastroenterology* 96:804-810, 1989.
69. Drossman, D.A., Lesserman, J., Li, Z., Mitchell, C.M., Zagami, E.A., Patrick, D.L.: The rating form of IBD patient concerns: A new measure of health status. *Psychosom Med* 53:701-12, 1991.
70. Rockwood, T.H., Church, J.M., Fleshman, J.W., Kane, R.L., Mavrantonis, C., Thorson, A.G., Wexner, S.D., Bliss, D., Lowry, A.C.: Fecal incontinence quality of life scale: Quality of life instrument for patients with fecal incontinence. *Dis Colon Rectum* 43:9-17, 2000.
71. McLeod, R.S: Re: Fecal incontinence quality of life scale: Quality of life instrument for patients with fecal incontinence (editorial) *Dis Colon Rectum* 43:16-17, 2000.
72. McHugh SM, Diamant E: Effect of age, gender, and parity on anal canal pressures. Contribution of impaired anal sphincter function to fecal incontinence. *Digestive Diseases & Sciences* 32 (7): 726-36, 1987.
73. Ferrara A, Pemberton JH, Hanson RB: Preservation of continence after ileoanal anastomosis by the coordination of ileal pouch and anal canal motor activity. *AM J Surg* 163:83-89, 1992.
74. Womack NNR, Morrison JFB, Williams NS: Prospective study of the effects of post anal repair in neurogenic faecal incontinence. *Br J Surg* 75:48-52, 1988.
75. Parks AG, Porter NH, Melzak J: Experimental study of the reflex mechanism controlling the muscles of the pelvic floor. *Dis Colon Rectum* 5:407-414, 1962.
76. Parks AG: Anorectal incontinence. *Proc Roy Soc Med* 68:681-690, 1975.
77. Bartolo DCC, Roe AM, Locke-Edmonds JC: Flap valve theory of anorectal incontinence. *Br J Surg* 73:1012-1014, 1986.
78. Varma KK, Stephens D: Neuromuscular reflexes of rectal continence. *Aust N Z J Med* 41:263-272, 1972.
79. Barnes PR, Hawley PR, Preston DM, et al: Experience of posterior division of the puborectalis muscle in the management of chronic constipation. *Br J Surg* 72:475-477, 1985.
80. Phillips SF, Edwards DAW: Some aspects of anal continence and defecation. *Gut* 6:396-405, 1965.
81. Taylor BM, Beart RW, Jr., Phillips SF: Longitudinal and radial variations of pressure in the human anal sphincter. *Gastroenterology*, 86(4):693-7, 1984.
82. Rasmussen OO: Anorectal function. *Dis Colon Rectum* 37:386-403, 1994.
83. O'Connell PR, Pemberton JH, Kelly KA: Motor function of the ileal J pouch and its relation to clinical outcome after ileal pouch-anal anastomosis. *World J Surg* 11:735-741, 1987.
84. Pemberton JH, Kelly KA, Beart RW, Jr., Dozois RR, Wolff BG, Ilstrup DM: Ileal pouch-anal anastomosis for chronic ulcerative colitis. Long-term Results. *Annals of Surgery*, 206(4):504-13, 1987.
85. Beart RW, Jr., Dozois RR, Wolff BG, Pemberton JH: Mechanisms of rectal continence. Lessons from the ileoanal procedure. *American Journal of Surgery*, 149(1): 31-4, 1995.
86. Grotz RL, Pemberton JH, Levin KE, et al: Rectal wall contractility in healthy subjects and in patients with chronic severe constipation. *Ann Surg* 218:761-768, 1993.
87. McNeil NI, Rampton DS: Is the rectum usually empty? A quantitative study in subjects with and without diarrhea. *Dis Colon Rectum* 23:596-599, 1981.
88. Hammer J, Phillips SF: Fluid loading of the human colon: Effects on segmental transit and stool composition. *Gastroenterology* 105:988-998, 1993.
89. B, Malmud L, D'Ercole F, et al: Colonic transit scintigraphy: A physiologic approach to the quantitative measurement of colonic transit in human. *Gastroenterology* 91:1102-1112, 1986.
90. Proano M, Camilleri M, Phillips SF, et al: Transit of solids through the human colon: Regional quantification in the unprepared bowel. *Am J Physiol* 248:G856-862, 1990.
91. Ferrara A, Pemberton JH, Grotz RL, Hanson RB: Prolonged ambulatory recording of anorectal motility in patients with slow-transit constipation. *American Journal of Surgery*, 167(1):73-9, 1994 January.
92. Haynes WG, Read NW: Anorectal activity in man during rectal infusion of saline: A dynamic assessment of the anal continence mechanism. *J Physiol* 330:45-56, 1982.
93. Scarli AF, Kiesewetter WB: Defecation and continence. *Dis Colon Rectum* 13:81-107, 1970.
94. Womack NR, Williams NS, Holmfield JH, et al: New method for the dynamic assessment of anorectal function in constipation. *Br J Surg* 72:994-998, 1985.
95. Barkel DC, Pemberton JH, Pezim ME, Phillips SF, Kelly KA, Brown ML: Scintigraphic assessment of the anorectal angle in health and after ileal pouch-anal anastomosis. *Annals of Surgery* 208(1):42-9, 1988 July.
96. Womack NR, Williams NS, Holmfield JHM, et al: Anorectal function in the solitary rectal ulcer syndrome. *Dis Colon Rectum* 30:319-323, 1987.
97. Ambroze WL, Pemberton JH, Bell AM, et al: The effect of stool consistency on rectal and neorectal emptying. *Dis Colon Rectum* 34:1-7, 1991.
98. Christmas TJ, Kempster PA, Chapple CR: Role of subcutaneous apomorphine in Parkinsonian voiding dysfunction. *Lancet* 2:1451-1453, 1988.
99. Andrew J, Nathan P: Lesions of the anterior frontal lobes and disturbances of micturition and defaecation. *Brain* 87:233-262, 1964.
100. Sultan AH, Kamm MA, Hudson CN, et al: Anal sphincter disruption during vaginal delivery. *N Engl J Med* 329:1905-1911, 1993.
101. Swash M, Gray A, Lubowski DZ, et al: Ultrastructural changes in internal anal sphincter in neurogenic faecal incontinence. *Gut* 29:1692-1968, 1988.
102. Read NW, Bartolo DCC, Read MG: Differences in anal function in patients with incontinence to solids and in patients with incontinence to liquids. *Br J Surg* 71:29-42, 1984.
103. Sun WM, Read NW, Miner PB, et al: The role of transient sphincter relaxation in faecal incontinence. *Int J Colorect Dis* 5:31-36, 1990.
104. Bannister JJ, Read NW, Donnelly TC, et al: External and internal anal sphincter responses to rectal distension in normal subjects and in patients with idiopathic faecal incontinence. *Br J Surg* 76:617-621, 1989.
105. Law PJ, Kamm MA, Bartram CI: Anal endosonography in the investigation of faecal incontinence. *Br J Surg* 78:312-314, 1991.
106. Speakman CTM, Hoyle CHV, Kamm MA: Adreneregic control of the internal anal sphincter is abnormal in patients with idiopathic faecal incontinence. *Br J Surg* 77:1342-1344, 1990.
107. Parks AG, Swash M, Urlich H: Sphincter denervation in anorectal incontinence and rectal prolapse. *Gut* 18:656-666, 1977.
108. Snooks SJ, Setchell M, Swash M: Injury to the innervation of the pelvic floor causing damage to the pelvic floor musculature in childbirth. *Lancet* 2:546-550, 1984.
109. Fynes M, Donnelly VS, O'Connell PR, O'Herlihy C: Cesarean delivery and anal sphincter injury. *Obstetrics & Gynecology* 92(4 Pt 1):496-500, 1998.

110. Miller R, Bartolo DCC, Cervero F, et al: Differences in anal sensation in continent and incontinent patient with perineal descent. *Int J Colorectal Dis* 4:45-49, 1989.
111. Read NW, Abouzekry L: Why do patients with faecal impaction have faecal incontinence? *Gut* 27:283-287, 1986.
112. Madoff RD, Williams JG, Caushaj PF: Fecal incontinence. *N Engl J Med* 326:1002-1007, 1992.
113. Fleshman JW, Dreznick Z, Fry RD, et al: Anal sphincter repair for obstetric injury: Manometric evaluation of functional results. *Dis Colon Rectum* 34:1061-1067, 1991.
114. Pemberton JH: Anorectal and pelvic disorders: Putting physiology into practice. *J Gastroenterol Hepatol* 5:127-143, 1990.
115. Barkel DC, Pemberton JH, Pezim ME, et al: Scintigraphic assessment of the anorectal angle in health and following ileal pouch-anal anastomosis. *Ann Surg* 208:42-49, 1988.
116. Hallan RI, Marzouk DEMM, Waldron DJ, et al: Comparison of digital and manometric assessment of anal sphincter function. *Br J Surg* 76:973-975, 1988.
117. Neill ME, Swash M: Increased motor unit fibre density in the external anal sphincter muscle in anorectal incontinence: a single fibre EMG study. *J Neurol Neurosurg Psychiatry* 43:343-347, 1980.
118. Snooks SJ, Barnes PRH, Swash M: Damage to the innervation of the pelvic floor musculature in chronic constipation. *Gastroenterology* 89:977-981, 1985.
119. Laurberg S, Swash M, Henry MM: Delayed external sphincter repair for obstetric tear. *British Journal of Surgery* 75(8):786-8, 1988.
120. Bartram GI, Turnbull GK, Lennard-Jones JE: Evacuation proctography: An investigation of rectal expulsion in 20 subjects without defecatory disturbance. *Gastrointest Radiol* 13:72-80, 1988.
121. Goie R: Anorectal function in patients with defaecation disorders and asymptomatic subjects. *Radiology* 174:121-123, 1990.
122. Law PJ, Kamm MA, Bartram CI: Anal endosonography: Technique and normal anatomy. *Gastrointest Radiol* 14:349-353, 1989.
123. Stoker J, Halligan S, Bartram CI: Pelvic floor imaging. *Radiology* 218(3):621-41, 2001 March
124. Malouf AJ, Halligan S, Williams AB, Bartram CI, Dhillon S, Kamm MA: Prospective assessment of interobserver agreement for endoanal MRI in fecal incontinence. *Abdominal Imaging* 26(1):76-8, 2001.
125. Glazener CMA, Abdalla M, Stroud P, Naji S, Templeton A, Russell IT: Postnatal maternal morbidity: extent, causes, prevention and treatment. *Br J Obstet Gynaecol* 102:282-87, 1995.
126. Beersiek F, Parks AG, Swash M: Pathogenesis of anorectal incontinence: a histometric study of the anorectal musculature. *J Neurol Sci* 42:111-127, 1979.
127. Neill ME, Swash M: Increased motor unit fibre density in the external anal sphincter in ano-rectal incontinence: a single fibre EMG study. *J Neurol Neurosurg Psychiatry* 43:343-347, 1980.
128. Kiff ES and Swash M: Slowed conduction in the pudendal nerves in idiopathic (neurogenic) faecal incontinence. *B J Surg* 71:614-616, 1984.
129. Snooks SJ, Swash M, Setchell M, Henry MM: Injury to innervation of pelvic floor sphincter musculature in childbirth. *Lancet* 2:546-50, 1984.
130. Snooks SJ, Swash M, Mathers SE and Henry MM: Effect of vaginal delivery on the pelvic floor: a 5-year follow-up. *Br J Surg* 77:1358-60, 1990.
131. Allen RE, Hosker GL, Smith ARB, Warrell DW: Pelvic floor damage and childbirth: a neurophysiological study. *Br J Obstet Gynaecol* 97:770-779, 1990.
132. Mallett V, Hosker G, Smith ARB, Warrell D: Pelvic floor damage and childbirth: a neurophysiologic follow up study. *Neurorol Urodyn* 13:357-8, 1994.
133. Small KA, Wynne JM: Evaluating the pelvic floor in obstetric patients. *Aust NZ J Obstet Gynaecol* 30:41-5, 1990.
134. Cornes H, Bartolo DCC, Stirrat GM: Changes in anal canal sensation after childbirth. *Br J Surg* 78:74-7, 1991.
135. Chaliha C, Sultan AH, Kalia V, Monga AK, Stanton SL: Anal incontinence during pregnancy and following childbirth *Am J Obstet Gynecol* 2001, in press.
136. Sultan AH, Kamm MA, Talbot IC, Nicholls RJ, Bartram CI: Anal endosonography: Precision of identifying sphincter defects confirmed histologically. *Br J Surg* 81:466-9, 1994.
137. Sultan AH, Kamm MA, Nicholls RJ, Bartram CI: Internal anal sphincter division during lateral sphincterotomy. Prospective ultrasound study. *Dis Colon Rectum* 37:1031-33, 1994.
138. Fynes M, Donnelly VS, O'Connell PR, O'Herlihy C: Cesarean delivery and anal sphincter injury. *Obstet Gynecol* 92:496-500, 1988.
139. Abramowitz L, Sobhani I, Ganasia R, Vuagnat A, Benifla JL, Darai E, Madelenat P, Mignon M: Are sphincter defects the cause of anal incontinence after vaginal delivery? Results of a prospective study. *Dis Colon Rectum* 43:590-8, 2000.
140. DeLeeuw JW, Struijk PC, Vierhout ME, Wallenburg HCS: Risk factors for third degree perineal ruptures during delivery. *Br J Obstet Gynaecol* 108:383-7, 2001.
141. Buchhave P, Flatow L, Rydhystroem H, Thorbert G: Risk factors for rupture of the anal sphincter. *Eur J Obstet Gynecol Reprod Biol* 87:129-32, 1999.
142. Fynes M, Donnelly V, Behan M, O'Connell PR, O'Herlihy C: Effect of second vaginal delivery on anorectal physiology and faecal incontinence: a prospective study. *Lancet* 354:983-6, 1999.
143. Varma A, Gunn J, Gardiner A, Lindow SW, Duthie GS: Obstetric anal sphincter injury. Prospective evaluation of incidence. *Dis Colon Rectum* 42:1537-43, 1999.
144. Rieger N, Schlothe A, Saccone G, Wattchow D: A prospective study of anal sphincter injury due to childbirth. *Scand J Gastroenterol* 33:950-5, 1998.
145. Zetterstrom J, Mellgren A, Jensen LJ, Wong WD, Kim DG, Lowry AC, Madoff RD, Congilosi SM: Effect of delivery on anal sphincter morphology and function. *Dis Colon Rectum* 42:1253-60, 1999.
146. Damon H, Henry L, Bretones S, Mellier G, Minaire Y, Mion F: Postdelivery anal function in primiparous females. Ultrasound and manometric study. *Dis Colon Rectum* 43:472-7, 2000.
147. Faltin DL, Boulvain M, Irion O, Bretones S, Stan C, Weil A: Diagnosis of anal sphincter tears by postpartum endosonography to predict fecal incontinence. *Obstet Gynecol* 95:643-7, 2000.
148. Groom KM, Paterson-Brown S: Third degree tears: are they clinically underdiagnosed? *Gastroenterology International* 13(2):76-7, 2000.
149. Sultan AH, Kamm MA, Hudson CN: Obstetric perineal tears: an audit of training. *Journal of Obstetrics and Gynaecology* 15:19-23, 1995.
150. Sultan AH, Thakar R: Lower genital tract and anal sphincter trauma. In: Baskett and Arulkumaran: Operative and Intrapartum Surgery. Bailliere's Best Practice & Research – Clinical Obstetrics and Gynaecology, 2002 in press.
151. Sultan AH: Editorial: Obstetric perineal injury and anal incontinence. *Clinical Risk* 5 (5):193-6, 1999.
152. Sultan AH, Kamm MA, Hudson CN, Bartram CI: Third degree obstetric anal sphincter tears: risk factors and outcome of primary repair. *BMJ* 308:887-91, 1994.

153. Sangalli MR, Floris L, Weil A. Anal incontinence in women with third or fourth degree perineal tears and subsequent vaginal deliveries. *Aust N Z J Obstet Gynaecol* 40:244-8, 2000.
154. Fitzpatrick M, Behan M, O'Connell R, O'Herlihy C. A randomized clinical trial comparing primary overlap with approximation repair of third degree obstetric tears. *Am J Obstet Gynecol* 183:1220-4, 2000.
155. Zetterstrom J, Lopez A, Anzen B, Norman M, Holmstrom B, Mellgren A. Anal sphincter tears at vaginal delivery: Risk factors and clinical outcome of primary repair. *Obstet Gynecol* 24:21-8, 1999.
156. Gjessing H, Backe B, Sahlin Y. Third degree obstetric tears; outcome after primary repair. *Acta Obstet Gynecol Scand* 77:736-40, 1998.
157. Poen AC, Felt-Bersma RJF, Strijers RLM, Dekkers GA, Cuesta MA, Meuwissen SGM. Third-degree obstetric perineal tear: long-term clinical and functional results after primary repair. *Br J Surg* 85:1433-8, 1998.
158. Goffeng AR, Andersch B, Berndtsson I, Hulten L, Oresland T. Objective methods cannot predict anal incontinence after primary repair of extensive anal tears. *Acta Obstet Gynecol Scand* 77: 439- 43, 1988.
159. Kammerer-Doak DN, Wesol AB, RogersRG, Dominguez, Dorin MH. A prospective cohort study of women after primary repair of obstetric anal sphincter laceration. *Am J Obstet Gynecol*;181(6):1317-23, 1999
160. Sultan AH, Monga AK, Kumar D, Stanton SL. Primary repair of obstetric anal sphincter rupture using the overlap technique. *Br J Obstet Gynaecol* ;106:318-323, 1999.
161. Malouf AJ, Norton CS, Engel AF, Nicholls RJ, Kamm MA. Long term results of overlapping anterior anal-sphincter repair for obstetric trauma. *Lancet* ;355: 260-5, 2000.
162. Wood J, Amos L, Rieger N. Third degree anal sphincter tears: risk factors and outcome. *Aust N Z J Obstet Gynaecol* ;38:414-7, 1998
163. Walsh CJ, Mooney EF, Upton GJ, Motson RW. Incidence of third-degree perineal tears in labour and outcome after primary repair. *Br J Surg* ; 83:218-21, 1996.
164. Sander P, Bjarnesen L, Mouritsen A, Fuglsang-Frederiksen A. Anal incontinence after third- /fourth degree laceration. One-year follow-up after pelvic floor exercises. *Int J Urogynecol*;10:177-81, 1999.
165. Crawford LA, Quint EH, Pearl ML, DeLancey JOL. Incontinence following rupture of the anal sphincter during delivery. *Obstet Gynecol* ;82:527-31, 1993.
166. Sorensen M, Tetzschner T, Rasmussen OO, Bjarnesen J, Christiansen J. Sphincter rupture in childbirth. *Br J Surg* ; 80:392-94, 1993.
167. Nielsen MB, Hauge C, Rasmussen OO, Pedersen JF, Christiansen J. Anal endosonographic findings in the follow-up of primarily sutured sphincteric ruptures. *Br J Surg* ; 79: 104-6, 1992.
168. Go PMNYH, Dunselman GAJ. Anatomic and functional results of surgical repair after total perineal rupture at delivery. *Surg Gynecol Obstet* ;166:121-4, 1988.
169. Uustal Fornell EK, Berg G, Hallbook O, Matthiesen LS, Sjodahl R. Clinical consequences of anal sphincter rupture during vaginal delivery. *J AM Coll Surg* ;183:553-8, 1996.
170. Sorensen SM, Bondesen H, Istre O, Vilmann P. Perineal rupture following vaginal delivery. *Acta Obstet Gynecol Scand* :67:315-8, 1988.
171. Tetzschner T, Sorensen M, Lose G, Christiansen J. Anal and urinary incontinence in women with obstetric anal sphincter rupture. *Br J Obstet Gynaecol* ;103:1034-40, 1996.
172. Haadem K, Ohrlander S, Lingman G. Long-term ailments due to anal sphincter rupture caused by delivery - a hidden problem. *Eur J Obstet Gynecol Reprod Biol* ; 27: 27-32, 1988.
173. Bek KM, Laurberg S. Risks of anal incontinence from subsequent vaginal delivery after a complete obstetric anal sphincter tear. *Br J Obstet Gynaecol* ;99:724-7, 1992.
174. Sultan AH, Abulafi MA. Anal incontinence – the role of the obstetrician and gynaecologist. In: Sturdee D, Olah K, Keane D (eds) *The yearbook of obstetrics and gynaecology*. Vol 9. London: RCOG press. :170-187, 2001.
175. Nichols DH. *Gynecologic and Obstetric Surgery*. Boston: Mosby. :1053-4, 1993.
176. Sultan AH, Stanton SL. Preserving the pelvic floor and perineum during childbirth - elective caesarean section? *Br J Obstet Gynaecol* ;103:731-4, 1996.
177. Sultan AH, Monga AK. Anal and urinary incontinence in women with obstetric anal sphincter rupture. *British Journal of Obstetrics and Gynaecology* ; 104: 753-4, 1997.
178. Johanson RB, Rice C, Doyle M et al. A randomised prospective study comparing the new vacuum extractor policy with forceps delivery. *Br J Obstet Gynaecol* ;100: 524- 30, 1993.
179. Bofill JA, Rust OA, Schorr SJ et al. A randomized prospective trial of the obstetric forceps versus the M-cup vacuum extractor. *Am J Obstet Gynecol* ;175:1325-30, 1996.
180. Johanson RB, Menon BKV. Vacuum extraction versus forceps for assisted vaginal delivery (Cochrane Review). In: *The Cochrane Library*, Issue 2. Oxford: Update Software 2001
181. Johanson RB, Heycock E, Carter J, Sultan AH, Walklate K, Jones PW. Maternal and child health after assisted vaginal delivery: five-year follow up of a randomised controlled study comparing forceps and ventouse. *Br J Obstet Gynaecol* ;106:544-9, 1999.
182. Sultan AH, Johanson RB, Carter JE. Occult anal sphincter trauma following randomized forceps and vacuum delivery. *Int J Gynecol Obstet* ; 61:113-9, 1998.
183. Johanson RB, Menon BKV. Soft versus rigid vacuum extractor cups for assisted vaginal delivery; Vacuum extraction versus forceps for assisted vaginal delivery. In: *The Cochrane Library*, Issue 1. Oxford: Update Software 2001.
184. Farrell SA, Allen VM, Baskett TF. Anal incontinence in primiparas. *J Soc Obstet Gynaecol Can* ; 23(4):321-6, 2001.
185. MacArthur C, Glazener CMA, Wilson PD, Herbison GP, Gee H, Lang GD, Lancashire R. Obstetric practice and faecal incontinence three months after delivery. *Br J Obstet Gynaecol*;108:678-83, 2001.
186. RCOG Audit Committee. *Effective procedures in obstetrics suitable for audit*. Manchester: RCOG Medical Audit Unit, 1993.
187. Woolley RJ. Benefits and risks of episiotomy: A review of the English language literature since 1980. *Obstet Gynecol Surv*;50:806-35, 1995.
188. Carroli G, Belizan J. Episiotomy for vaginal birth (Cochrane Review). In: *The Cochrane Library*, Issue 2. Oxford: Update Software, 2001.
189. Henriksen TB, Bek KM, Hedegaard M, Secher NJ. Methods and consequences of changes in use of episiotomy. *BMJ* ;309:1255-8, 1994.
190. Henriksen TB, Bek KM, Hedegaard M, Secher NJ. Episiotomy and perineal lesions in spontaneous vaginal deliveries. *Br J Obstet Gynaecol* ; 99:950-4, 1992.
191. Coats PM, Chan KK, Wilkins M, Beard RJ. A comparison between midline and mediolateral episiotomies. *Br J Obstet Gynaecol* ; 87: 408-12, 1980.
192. Pirhonen JP, Grenman SE, Haadem K, Gudmundsson S, Lindqvist P, Sihola S, Erkkola RU, Marsal K. Frequency of anal sphincter rupture at delivery in Sweden and Finland – result of

- difference in manual help to the baby's head. *Acta Obstet Gynecol Scand* ;77:974-7, 1998.
193. Kamm MA. Faecal incontinence: clinical review. *British Medical Journal* ; 316: 528-532, 1998.
 194. Read M, Read NW, Barber DC, Duthie HL. Effects of loperamide on anal sphincter function in patients complaining of chronic diarrhoea with faecal incontinence and urgency. *Digestive Diseases and Sciences* ; 27: 807-814, 1982.
 195. Sun WM, Read NW, Verlinden M. Effects of loperamide oxide in gastrointestinal transit time and anorectal function in patients with chronic diarrhoea and faecal incontinence. *Scandinavian Journal of Gastroenterology* ; 32: 34-38, 1997.
 196. Rattan S, Culver PJ. Influence of loperamide on the internal anal sphincter in the opossum. *Gastroenterology* ; 93: 121-128, 1987.
 197. Kamm MA. Functional disorders of the colon and anorectum. *Current Opinion in Gastroenterology* ; 11: 9-15, 1987.
 198. Barrett JA. Faecal incontinence and related problems in the older adult. London: Edward Arnold, 1993.
 199. Tobin GW, Brocklehurst JC. Faecal incontinence in residential Homes for the elderly: prevalence, aetiology and management. *Age and Ageing* ; 15: 41-46, 1986.
 200. Donnelly V, O'Connell PR, O'Herlihy C. The influence of oestrogen replacement on faecal incontinence in postmenopausal women. *British Journal of Obstetrics & Gynaecology* ; 104: 311-315, 1997.
 201. Fantl JA, Cardozo L, McClish D. Estrogen therapy in the management of urinary incontinence in postmenopausal women: a meta-analysis. First report of the hormones and urogenital therapy committee. *Obstetrics & Gynecology* ; 83: 12-18, 1994.
 202. Haadem K, Ling L, Ferno M, Graffner H. Oestrogen receptors in the external anal sphincter. *American Journal of Obstetrics & Gynecology* , 164: 609-610, 1991.
 203. Laurberg S, Swash M. Effects of aging on the anorectal sphincters and their innervation. *Dis.Colon Rectum* ; 32: 737-742, 1989.
 204. Carapeti EA, Kamm MA, Evans BK, Phillips RK. Topical phenylephrine increases anal sphincter resting pressure. *British Journal of Surgery* ; 86: 267-270, 1999.
 205. Carapeti EA, Kamm MA, Nicholls RJ, Phillips RKS. Randomized, controlled trial of topical phenylephrine for fecal incontinence in patients after ileoanal pouch construction. *Dis.Colon Rectum* ; 43: 1059-1063, 2000.
 206. Carapeti EA, Kamm MA, Phillips RKS. Randomized controlled trial of topical phenylephrine in the treatment of faecal incontinence. *British Journal of Surgery* ; 87: 38-42, 2000.
 207. Burgio KL, Engel BT. Biofeedback-assisted behavioral training for elderly men and women. *Journal of the American Geriatrics Society* ; 38: 338-340, 1990.
 208. Whitehead WE, Drossman DA. Biofeedback for disorders of elimination: fecal incontinence and pelvic floor dyssynergia. *Professional Psychology: Research and Practice* ; 27: 234-240, 1996.
 209. Engel BT, Nikoomanesh P, Schuster MM. Operant conditioning of rectosphincteric responses in the treatment of faecal incontinence. *New England Journal of Medicine* ; 290: 646-649, 1974.
 210. Whitehead WE, Orr WC, Engel BT, Schuster MM. External anal sphincter response to rectal distension: learned response or reflex. *Psychophysiology* ; 19: 57-62, 1981
 211. Schussler B, Laycock J, Norton P, Stanton S. Pelvic floor re-education: principles and practice. London: Springer-Verlag, 1994.
 212. MacLeod JH. Management of anal incontinence by biofeedback. *Gastroenterology* ; 93: 291-294, 1987.
 213. Chiarioni G, Scattolini C, Bonfante F, Vantini I. Liquid stool incontinence with severe urgency: anorectal function and effective biofeedback treatment. *Gut* ; 34: 1576-1580, 1993.
 214. Patankar SK, Ferrara A, Larach SW, et al. Electromyographic assessment of biofeedback training for faecal incontinence and chronic constipation. *Dis.Colon Rectum* ; 40: 907-911, 1997.
 215. Buser WD, Miner PB. Delayed rectal sensation with faecal incontinence. Successful treatment using anorectal manometry. *Gastroenterology* ; 91: 1186-1191, 1986.
 216. Miner PB, Donnelly TC, Read NW. Investigation of the mode of action of biofeedback in the treatment of faecal incontinence. *Digestive Diseases & Sciences* ; 35: 1291-1298, 1990.
 217. Oresland T, Fasth S, Nordgren S, Swenson L, Akervall S. Does balloon dilatation and anal sphincter training improve ileo- anal pouch function? *International Journal of Colorectal Disease* ; 3: 153-157, 1988.
 218. Norton C, Kamm MA. Outcome of biofeedback for faecal incontinence. *British Journal of Surgery*; 86: 1159-1163, 1999.
 219. Solomon MJ, Rex J, Eysers AA, Stewart P, Roberts R. Biofeedback for fecal incontinence using transanal ultrasonography: novel approach. *Dis.Colon Rectum* ; 43: 788-792, 2000.
 220. Latimer PR, Campbell D, Kasperski J. A components analysis of biofeedback in the treatment of faecal incontinence. *Biofeedback and Self-Regulation* ; 9: 311-324, 1984.
 221. Norton C, Kamm MA. Anal sphincter biofeedback and pelvic floor exercises for faecal incontinence in adults - a systematic review. *Alimentary Pharmacology & Therapeutics* ; 15: 1147-1154, 2001.
 222. Fynes MM, Marshall K, Cassidy M, et al. A prospective, randomized study comparing the effect of augmented biofeedback with sensory biofeedback alone on fecal incontinence after obstetric trauma. *Dis.Colon Rectum* ; 42: 753-758, 1999.
 223. Sander P, Bjarnesen J, Mouritsen L, Fuglsang-Frederiksen A. Anal incontinence after obstetric third- /fourth-degree laceration. One-year follow-up after pelvic floor exercises. *International Urogynecology Journal & Pelvic Floor Dysfunction* ; 10: 177-181, 1999.
 224. Whitehead WE, Burgio KL, Engel BT. Biofeedback treatment of faecal incontinence in geriatric patients. *Journal of the American Geriatrics Society* ; 33: 320-324, 1985.
 225. Jensen LL, Lowry AC. Biofeedback improves functional outcome after sphincteroplasty. *Dis.Colon Rectum* ; 40: 197-200, 1997.
 226. Ho YH, Chiang JM, Tan M, Low JY. Biofeedback therapy for excessive stool frequency and incontinence following anterior resection or total colectomy. *Dis.Colon Rectum* ; 39: 1289-1292, 1996.
 227. Jorge JM, Wexner SD, Morgado PJ, James K, Noguera JJ, Jagelman DG. Optimization of sphincter function after the ileoanal reservoir procedure. *Dis.Colon Rectum* ; 37: 419-423, 1994.
 228. Berti Riboli E, Frascio M, Pitto G, Reboa G, Zanolla R. Biofeedback conditioning for faecal incontinence. *Archives of Physical & Medical Rehabilitation* ; 69: 29-31, 1988.
 229. Cerulli MA, Nikoomanesh P, Schuster MM. Progress in biofeedback conditioning for faecal incontinence. *Gastroenterology* ; 76: 742-746, 1979.
 230. McHugh S, Kersey K, Diamant NE. Biofeedback training for faecal incontinence: outcome according to physiological parameters. *Gastroenterology* 94: A295, 1988.
 231. van Tets WF, Kuipers JH, Bleijenberg G. Biofeedback treatment is ineffective in neurogenic fecal incontinence. *Dis.Colon Rectum* ; 39: 992-994, 1996.
 232. Magrini P, Pallotta L, Koch M, Capurso L. Manometric biofeedback in the management of faecal incontinence and pelvic floor dyssynergia. *International Journal of the Proctological and Perineal Diseases* ; 1: 269-270, 1997.

233. Enck P, Daublin G, Lubke HJ, Strohmeier G. Long-term efficacy of biofeedback training for faecal incontinence. *Dis.Colon Rectum* ; 37: 997-1001, 1994.
234. Guillemot F, Bouche B, Gower-Rousseau C, et al. Biofeedback for the treatment of fecal incontinence. Long-term clinical results. *Dis.Colon Rectum* , 38: 393-397, 1995.
235. Ryn A-K, Morren GL, Hallbook O, Sjudahl R. Long-term results of electromyographic biofeedback training for faecal incontinence. *Dis.Colon Rectum* ; 43: 1262-1266, 2000.
236. Leroi AM, Dorival MP, Lecouturier MF, et al. Pudendal neuropathy and severity of incontinence but not presence of an anal sphincter defect may determine the response to biofeedback therapy in fecal incontinence. *Dis.Colon Rectum* ; 42: 762-769, 1999.
237. Rieger NA, Wattochow DA, Sarre RG, et al. Prospective trial of pelvic floor retraining in patients with faecal incontinence. *Dis.Colon Rectum* ; 40: 821-826, 1997.
238. Ko CY, Tong J, Lehman RE, Shelton AA, Schrock TR, Welton ML. Biofeedback is effective therapy for fecal incontinence and constipation. *Arch.Surg.* 132: 829-833, 1997.
239. Wald A. Biofeedback therapy for faecal incontinence. *Annals of Internal Medicine* ; 95: 146-149, 1981.
240. Norton C, Hosker G, Brazzelli M. Effectiveness of biofeedback and/or sphincter exercises for the treatment of faecal incontinence in adults. *Cochrane electronic library* 2000.
241. McHugh S, Walma K, Diamant NE. Faecal incontinence: a controlled trial of biofeedback. *Gastroenterology* ; 90: 1545-1545, 1986.
242. Caldwell KPS. The electrical control of sphincter incompetence. *Lancet* ; ii: 174-175, 1963.
243. Mills PM, Deakin M, Kiff ES. Percutaneous electrical stimulation for ano-rectal incontinence. *Physiotherapy* ; 76: 433-438, 1990.
244. Pescatori M, Pavesio R, Anastasio G, Daini S. Transanal electrostimulation for faecal incontinence: clinical, psychologic and manometric prospective study. *Dis.Colon Rectum* ; 34: 540-545, 1991.
245. Jost WH. Electrostimulation in fecal incontinence: relevance of the sphincter compound muscle action potential. *Diseases of the Colon & Rectum* ; 41: 590-592, 1998.
246. Scheuer M, Kuijpers HC, Bleijenberg G. Effect of electrostimulation on sphincter function in neurogenic fecal continence. *Dis.Colon Rectum* ; 37: 590-593, 1994.
247. Hosker G, Norton C, Brazzelli M. Effectiveness of electrical stimulation for faecal incontinence in adults. *Cochrane electronic library* 2000.
248. Sylvester KL, Keilty SEJ. A pilot study to investigate the use of interferential in the treatment of ano-rectal incontinence. *Physiotherapy* ; 73: 207-208, 1987.
249. Hinds JP, Wald A. Colonic and anorectal dysfunction associated with multiple sclerosis. *Am.J.Gastroenterol.* ; 84: 587-595, 1989.
250. Wald A. Systemic diseases causing disorders of defaecation and continence. *Seminars in Gastrointestinal Disease* ; 6: 194-202, 1995.
251. O'Regan S, Yazbeck S, Schick E. Constipation, bladder instability, urinary tract infection syndrome. *Clin Nephrol* ; 23: 153-154, 1985.
252. Banwell JG, Creasey GH, Aggarwal AM, Mortimer JT. Management of the neurogenic bowel in patients with spinal cord injury. *Urologic Clinics of North America* ; 20: 517-526, 1993.
253. Stiens SA, Bergman SB, Goetz LL. Neurogenic bowel dysfunction after spinal cord injury: clinical evaluation and rehabilitation management. *Archives of Physical Medicine & Rehabilitation* ; 78: S-86-S-104, 1997.
254. Dunn KL, Galka ML. A comparison of the effectiveness of Thevac SB and bisacodyl suppositories in SCI patients' bowel programs. *Rehabilitation Nursing* ; 19 : 334-338, 1994.
255. Glen House J, Stiens SA. Pharmacologically initiated defaecation for persons with spinal cord injury: effectiveness of three agents. *Archives of Physical Medicine & Rehabilitation* ; 78: 1062-1065, 1997.
256. Stiens SA. Reduction in bowel programme duration with polyethylene glycol based bisacodyl suppositories. *Archives of Physical Medicine & Rehabilitation* ; 76: 674-677, 1995.
257. Doughty D. A physiologic approach to bowel training. *Journal of Wound, Ostomy, & Continence Nursing* ; 23: 46-56, 1996.
258. Younoszai MK. Stooling problems in patients with myelomeningocele. *Southern Medical Journal* ; 85: 718-724, 1992.
259. King JC, Currie DM, Wright E. Bowel training in spina bifida: importance of education, patient compliance, age, and anal reflexes. *Archives of Physical Medicine & Rehabilitation* ; 75: 243-247, 1994.
260. Munchiando JF, Kendall K. Comparison of the effectiveness of two bowel programs for CVA patients. *Rehabilitation Nursing* ; 18: 168-172, 1993.
261. Krogh K, Nielsen J, Djurhuus JC, Mosdal C, Sabroe S, Laurberg S. Colorectal function in patients with spinal cord lesions. *Dis.Colon Rectum* ; 40: 1233-1239, 1997.
262. Ouslander JG, Simmons S, Schnelle J, Uman G, Fingold S. Effects of prompted voiding on fecal continence among nursing home residents. *Journal of the American Geriatrics Society* ; 44: 424-428, 1996.
263. Shandling B, Gilmour RF. The enema continence catheter in spina bifida: successful bowel management. *J.Pediatr.Surg.* ; 22: 271-273, 1987.
264. Lipak GS, Revell GM. Management of bowel dysfunction in children with spinal cord disease or injury by means of the enema continence catheter. *Journal of Pediatrics* ; 120: 190-194, 1992.
265. De Kort LMO, Nesselaar CH, Van Gool JD, De Jong TPVM. The influence of colonic enema irrigation on urodynamic findings in patients with neurogenic bladder dysfunction. *Br.J.Urol.* ; 80: 731-733, 1997.
266. Scholler-Gyure M, Nesselaar CH, van Wieringen H, Van Gool JD. Treatment of defaecation disorders by colonic enemas in children with spina bifida. *European Journal of Pediatric Surgery* ; 6: 32-34, 1996.
267. Glickman S, Kamm MA. Bowel dysfunction in spinal-cord-injury patients. *Lancet* ; 347: 1651-1653, 1996.
268. Addison, R. Digital rectal examination and manual removal of faeces. London, Royal College of Nursing, 1995.
269. Ardron ME, Main ANH. Management of constipation. *British Medical Journal* ; 300: 1400, 1990.
270. Norton C, Kamm MA. Bowel control - information and practical advice. Beaconsfield: Beaconsfield Publishers, 1999.
271. Barrett JA. Effects of wheat bran on stool size. *British Medical Journal* ; 296: 1127-1128, 1988.
272. Bliss,D.Z., Jung,H., Lowry,A.C., Savik,K., Jensen,L., LeMoine,M. and Werner,C. Effects of dietary fiber therapy for fecal incontinence. *Gerontologist* ; 325-325, 1997.
273. Christiansen J, Roed-Petersen K. Clinical assessment of the anal continence plug. *Dis.Colon Rectum* ; 36: 740-742, 1993.
274. Norton C, Kamm MA. Anal plug for faecal incontinence. *Colorectal Disease* ; 3: 323-327, 2001.
275. Rasmussen OØ, Christiansen J, Tetzschner T, Sørensen M. Pudendal nerve function in idiopathic fecal incontinence. *Dis Colon Rectum* ;43:633-635, 2000.
276. Browning GGP, Parks AG. Postanal repair for neuropathic fae-

- cal incontinence: correlation of clinical results and anal canal pressures. *Brit J Surg* ;70:101-104, 1983.
277. Keighley MRB. Postanal repair. *Int J Colorectal Dis* ;2:236-239, 1987.
278. Carraro PS, Kamm MA, Nicholls RJ. *Br J Surg* ;81:1401-44, 1994.
279. Jameson JS, Speakman CTM, Darzi A, Chia YW, Henry MM. Audit of postanal repair in the treatment of fecal incontinence. *Dis Colon Rectum* ;37:369-372, 1994.
280. Matsuoka H, Mavrantonis C, Wexner SD, Oliveira L, Gilliland R, Pikarsky A. Postanal repair for fecal incontinence-is it worthwhile?. *Dis Colon Rectum* ;43:1561-1567, 2000.
281. Christiansen J, Skomorowska E. Persisting incontinence after postanal repair treated by anterior perineoplasty. *Int J Colorectal Dis* ;7:2:9-11, 2000.
282. Miller R, Orrom WJ, Cornes H, Duthie G, Bartolo DCC. Anterior sphincter plication and levatorplasty in the treatment of faecal incontinence. *Br J Surg* ;76:1058-1060, 1989.
283. Orrom WJ, Miller R, Cornes H, Duthie G, Mortensen NJM, Bartolo DCC. Comparison of anterior sphincteroplasty and postanal repair in the treatment of idiopathic fecal incontinence. *Dis Colon Rectum* ;34:305-310, 1991.
284. Deen KI, Oya M, Ortiz J, Keighley MRB. Randomized trial comparing three forms of pelvic floor repair for neuropathic faecal incontinence. *Br J Surg* ;80:794-798, 1991.
285. Keighley MRB, Williams NS. *Surgery of the Anus Rectum and Colon*. Saunders. London 1993 p.548.
286. Carraro PS, Nicholls RJ. Postanal repair for faecal incontinence persisting after rectopexy. *Br J Surg* ;81:305-307, 1994.
287. Wexner SD, Marchetti F, Jagelman DG. The role of sphincteroplasty for fecal incontinence reevaluated: a prospective physiological and functional review. *Dis Colon Rectum* ;34:22-30, 1991.
288. Hasagawa H, Yoshioka K, Keighley MR. Randomized trial of fecal diversion for sphincter repair. *Dis Colon Rectum* ;43:961-964, 2000.
289. Sitzler PJ, Thompson JP. Overlap repair of damaged anal sphincter. A single surgeons series. *Dis Colon Rectum* ;39:1356-1360, 1996.
290. Engel AF, Kamm MA, Sultan AH, Bartram CI, Nicholls RJ. Anterior sphincter repair in patients with obstetric trauma. *Br J Surg* ;81:1231-1234, 1994.
291. 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* ;3:494-497, 1990.
292. Rasmussen OØ, Puggaard L, Christiansen J. Anal sphincter repair in patients with obstetric trauma. *Dis Colon Rectum* ;42:193-195, 1999.
293. Karoui S, Leroi AM, Koning E, Menard JF, Michot F, Denis P. Results of sphincteroplasty in 86 patients with anal incontinence. *Dis Colon Rectum* ;43:813-820, 2000.
294. Morren GL, Hallböök O, Nyström PO, Baeten CGM, Sjö Dahl R. Audit of anal-sphincter repair. *Colorectal Dis* ;3:17-22, 2001.
295. Nikiteas N, Korsgen S, Kumar D, Keighley MRB. Audit of sphincter repair. Factor associated with poor outcome. *Dis Colon Rectum* ;39:1164-1170, 1996.
296. Arnaud A, Sarles JC, Sielezneff I, Orsoni P, Joly A. Sphincter repair without overlapping for fecal incontinence. *Dis Colon Rectum* ;34: 744-747, 1991.
297. Cook TA, Mortensen NJM. Management of faecal incontinence following obstetric injury. *Br J Surg* ;85:293-299, 1998.
298. Nielsen MB, Dammegaard L, Pedersen JF. Endosonographic assessment of the anal sphincter after surgical reconstruction. *Dis Colon Rectum* ;37:434-438, 1994.
299. Engel AF, Brummelkamp WH. Secondary surgery after failed postanal or anterior sphincter repair. *Int J Colorectal Dis* ;9:187-190, 1994.
300. Pickrell K, Georgiade N, Maguire C, Crawford H. Gracilis muscle transplant for rectal incontinence. *Surgery* ;40:349-363, 1956.
301. Corman ML. Follow-up evaluation of gracilis muscle transposition for fecal incontinence. *Dis Colon Rectum* ;23:552-555, 1980.
302. Christiansen J, Rasmussen OØ, Lindorff-Larsen K. Dynamic graciloplasty for severe anal incontinence. *Br J Surg* ;85:88-91, 1998.
303. Baeten CGMI, Geerdes BP, Adang EMM, Heineman E, Konsten J, Engel GL, Kester ADM, Spaans F, Soeters PB. Anal dynamic graciloplasty in the treatment of intractable fecal incontinence. *N Engl J Med* ;332:1600-1605, 1995.
304. Madoff RD, Rosen HR, Baeten CG, LaFontaine LJ, Cavina E, Devesa M, Rouanet PR, Christiansen J, Faucheron J-L, Isbister W, Köhler L, Guelinckx J, Pählman L. Safety and efficacy of dynamic muscle transplant for anal incontinence: lessons from a prospective multicenter trial. *Gastroenterology* ;116:649-556, 1999.
305. Geerdes BP, Heineman E, Konsten J, Soeters PB, Baeten CGMI. Dynamic graciloplasty. Complications and management. *Dis Colon Rectum* ;39:912-917, 1996.
306. Baeten CGM and the Dynamic Graciloplasty Study Group. *Dis Colon Rectum* ;43:743-751, 2000.
307. Christiansen J, Lorenzen M. Implantation of artificial sphincter for anal incontinence. *Lancet* ;1:244-245, 1987.
308. Christiansen J, Sparsø B. Treatment of anal incontinence by an implantable prosthetic anal sphincter. *Ann Surg* ;215:383-386, 1992.
309. Wong DW, Jensen LR, Bartolo DCC, Rothenberger DA. Artificial anal sphincter. *Dis Colon Rectum* ;39:1345-1351, 1996.
310. Lehur P-A, Michot F, Denis P, Grise P, Leborgne J, Teniere P, Buzelin J-M. Results of artificial sphincter in severe anal incontinence. *Dis Colon Rectum* ;39:1352-1355, 1996.
311. Lehur P-A, Glemain P, Bruley des Varannes S, Buzelin JM, Leborgne J. Outcome of patients with an implantable artificial anal sphincter for severe anal incontinence. *Int J Colorectal Dis* ;13:88-92, 1998.
312. 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* ;352:105-109, 1998.
313. Dodi G, Melega E, Masin A, Infantino A, Cavallari F, Lise M. Artificial bowel sphincter (ABS) for severe faecal incontinence: a clinical and manometric study. *Colorectal Dis* ;2:207-211, 2000.
314. Christiansen J, Rasmussen OØ, Lindorff-Larsen K. Long-term results of artificial anal sphincter implantation for severe anal incontinence. *Ann Surg* ;230:45-48, 1999.
315. Spencer M, Wong W, Congilosi S. Artificial anal sphincter: preliminary results of a multicenter prospective trial. *Dis Colon Rectum* ;41: A15, 1998.
316. Matzel KE, Stadelmaier U, Hohenfellner M, Gall FP. Electrical stimulation of sacral spinal nerves for treatment of faecal incontinence. *Lancet* ;346:1124-1126, 1995.
317. Vaizey CJ, Kamm MA, Turner IC, Nicholls RJ, Wolosko J. Effects of short term sacral nerve stimulation on anal and rectal function in patients with anal incontinence. *GUT* ;44:407-412, 1999.
318. Malouf AJ, Vaizey CJ, Nicholls RJ, Kamm MA. Permanent sacral nerve stimulation for fecal incontinence. *Ann Surg* ;232:143-148, 2000.
319. Matzel KE, Stadelmaier U, Hohenfellner M, Hohenberger W. Chronic sacral spinal nerve stimulation for fecal

- incontinence: long-term results with foramen and cuff electrodes. *Dis Colon Rectum* ;44:59-66, 2001.
320. Shafik A. Perianal injection of autologous fat for treatment of sphincteris incontinence. *Dis Colon Rectum* ;38:583-87, 1995.
 321. Kumar D, Benson M. Glutaraldehyde cross-linked collagen in the treatment of faecal incontinence. *Br J Surg* ; 85:978-79, 1998.
 322. Malouf AJ, Vaizey CJ, Norton CS, Kamm MA. Internal anal sphincter augmentation for fecal incontinence using injectable silicone biomaterial. *Dis Colon Rectum* ;44: in press 2001.
 323. Griffiths DM, Malone PS. The Malone antegrade continence enema. *J Pediatric Surg* ;30:68-71, 1995.
 324. Krogh K, Laurberg S. Malone antegrade continence enema for faecal incontinence and constipation in adults. *Brit J Surg*; 85:974-977, 1998.
 325. Vries MW, de Vries MR. Cultural relativity of toilet training readiness: a perspective from East Africa. *Pediatrics*, 60(2):170-7, 1977.
 326. Largo RH, Stutzle W. Longitudinal study of bowel and bladder control by day and at night in the first six years of life. I: Epidemiology and interrelations between bowel and bladder control. *Developmental Medicine and Child Neurology*, 19(5):598-606, 1977.
 327. Largo RH, Stutzle W. Longitudinal study of bowel and bladder control by day and at night in the first six years of life. II: The role of potty training and the child's initiative. *Developmental Medicine and Child Neurology*, 19(5): 607-613, 1977.
 328. Brazelton TB: A child oriented approach to toilet training. *Pediatrics*, 29: 121, 1962.
 329. Taubman B. Toilet training and toileting refusal for stool only: a prospective study. *Pediatrics*, 99(1): 54-8, 1997.
 330. Weaver LT, Steiner H. The bowel habit of young children. *Arch. Dis. Child*, 59: 694-652, 1984.
 331. Bellman M. Studies on encopresis. *Acta Paediatr Scand Suppl*:170: 1-151, 1966.
 332. Rutter M. *Helping Troubled Children*. Harmons-Worth, England, Penguin Education 1995.
 333. Levine MD. Children with encopresis: a descriptive analysis. *Pediatrics*, 56(3):412-416, 1975.
 334. Rasquin-Weber A, Hyman PE et al. Childhood functional gastrointestinal disorders. *Gut*, 45 Supp II 60-68, 1999.
 335. Marty T et al. Gastrointestinal function after surgical correction of Hirschprung's disease: Long term follow up in 135 patients. *J Pediatr Surg*, 30:655 – 658, 1995.
 336. Moore et al. Clinical outcome and Long term quality of life after surgical correction of Hirschprung's disease. *J Pediatr Surg*, 31(11): 1496 – 1502, 1996.
 337. Catto-Smith Ag, Coffey CM, Nolan TM, Hutson JM. Fecal incontinence after surgical treatment of Hirschprung disease. *J Pediatr*, 127:954-957, 1995.
 338. Loening-Baucke VA, Desch L, Wolraich M. Biofeedback training for patients with meningomyelocoele and fecal incontinence. *Dev Med Child Neurol*, 30; 78-90, 1998.
 339. Davidson M, Kugler MM, Bauer CH. Diagnosis and management in children with severe and protracted constipation and obstipation. *J Pediatr*, 62:261, 1963.
 340. Johnston BD, Wright JA. Attentional dysfunction in children with encopresis. *J. Dev & Beh. Ped*, 14; 381-385, 1993.
 341. Gohlke BC, Khadilkar VV, Skuse D, Stanhope R. Recognition of children with psychosocial short stature: a spectrum of presentation. *J Ped Endocrinology & Metabolism*, 11: 509 – 517, 1998.
 342. Levine MD. Encopresis: its potentiation, evaluation and alleviation. *Pediatr. Clin. North Am*, 29(2): 315-30, 1982.
 343. Clayden GS. Management of chronic constipation. *Arch Dis Child*, 67:340, 1992.
 344. Buchanon A. *Children who Soil*. Wiley. Chichester ISBN 0-471-93479-8, 1992.
 345. Slukin A. Behavioural social work with encopretic children, their families and the school. *Child Care, Health & Dev*, 7: 67-80, 1981.
 346. Gabel S, et al. prevalence of behavior problems and mental health utilisation among encopretic children: Implications for behavioral pediatrics. *J. Dev & Beh Ped*, 7(5) 293-297, 1986.
 347. Young M, Brennen L, Baker R, Baker S. Functional encopresis: Symptom reduction and behavioral improvement. *J Dev & Beh Ped*, 16(4) 226-232, 1995.
 348. Jewkes RK, O' Connor . Crisis in our schools: survey of sanitation facilities in schools in Bloomsbury health district. *BMJ*, 301(6760):1085-7, 1990.
 349. Iacono G, Cavataio F, Montalto G, et al. Intolerance of cow's milk and chronic constipation in children. *N Engl J Med*, 339:1100-1104, 1998.
 350. van der Plas RN, Benninga MA et al. Megarectum in constipation. *Arch Dis Child*, 83(1): 52-58, 2000.
 351. Blethyn AJ, Verrier Jones K, Newcombe R, Roberts GM, Jenkins HR. Radiological assessment of constipation. *Arch Dis Child*, 73:532-533, 1995.
 352. Leech SC, McHugh K, Sullivan PB. Evaluation of a method of assessing faecal loading on plain abdominal radiographs of children. *Pediatr Radiol*, 29(4): 255-8, 1999.
 353. Papadopoulou A, Clayden GS, Booth IW. The clinical value of solid marker transit studies in childhood constipation and soiling. *Eur J Pediatr*, 153:560-564, 1994.
 354. Loening-Baucke V. Abnormal rectoanal function in children recovered from chronic constipation and encopresis. *Gastroenterology*, 87:1299, 1984.
 355. Clayden GS, Lawson JON. Investigation and management of long-standing chronic constipation in childhood. *Arch Dis Child*, 51: 918-923, 1976.
 356. Poenaru D, Roblin N, Bird M, Duce S et al. The pediatric bowel management clinic: initial results of a multidisciplinary approach to functional constipation in children. *J Pediatr Surg*, 32(6) 843-848, 1997.
 357. Nolan T, DeBelle G, Oberklaid F, Coffey C. Randomised trial of laxatives in treatment of childhood encopresis. *Lancet*, 338;523-527, 1991.
 358. Taitz LS, Wales JKW, Urwin OM et al . Factors associated with outcome in management of defaecation disorders. *Arch Dis Child*, 61:472, 1986.
 359. Graham YD, Moser ES, Estes KM. The effect of bran on bowel function in constipation. *Am J Gastroenterol*, 77: 599 – 603, 1982.
 360. Roma E, Adamidis D et al. Diet and chronic constipation in children: The role of fibre. *J Ped Gastro & Nutr*, 28(2); 168-174, 1999.
 361. Loening-Baucke V. Encopresis and Soiling. *Ped Clin of N Am*, 43 (1) 279-298, 1996.
 362. *Drug and Therapeutics Bulletin*. Managing Constipation in Children, 38(8): 57-60, 2000.
 363. Bandla HP, Davis SH, Hopkins NE . Lipoid pneumonia: a silent complication of mineral oil aspiration. *Pediatrics*, 103(2): E19, 1999.
 364. Gleghorn EE, Heyman MB, Rudolph CD: No enema treatment for idiopathic constipation and encopresis. *Clin Pediatr*, 30:669-672, 1991.
 365. Ingebo KB , Heyman MB. Polyethylene glycol-electrolyte solu-

- tion for intestinal clearance in children with refractory encopresis. *AJDC*, 142:340-342, 1988.
366. Sondheimer JM, Sokol RJ, Taylor SF, Silverman A, Zelasney B. Safety, efficacy and tolerance of intestinal lavage in pediatric patients undergoing diagnostic colonoscopy. *J Pediatr*, 119:148-52, 1991.
367. Loening-Baucke V. Modulation of abnormal defaecation dynamics by biofeedback treatment in chronically constipated children with encopresis. *J Pediatr*, 116:214, 1990.
368. Benninga MA, Büller HA, Taminiau JAJM. Biofeedback training in chronic constipation. *Arch. Dis Child*, 68: 126-129, 1993.
369. van der Plas RN, Benninga MA, Buller HA et al. Biofeedback training in treatment of childhood constipation: a randomised controlled study. *Lancet*, 348 (9030):776-80, 1996.
370. Loening-Bauke V. Biofeedback treatment for chronic constipation and encopresis in childhood: long-term outcome. *Pediatrics*, 96: 105-110, 1995.
371. van Ginkel R, Benninga MA et al. Lack of benefit of laxatives as adjunctive therapy for functional nonretentive fecal soiling in children. *J Pediatr*, 137:808-813, 2000.
372. ERIC (Enuresis resource and Information Centre, Bristol, UK) Helpline +44 117 960 3060. www.enuresis.org.uk
373. Malone PS, Ransley PG, Keily EM. Preliminary report; the antegrade continence enema. *Lancet*, 336:1217-1218, 1990.
374. Griffiths DM, Malone PS. The Malone antegrade continence enema. *J Pediatr Surg*, 30 68-71, 1995.
375. Vos A, Cuesta M, Meuwissen S. Antegrade colonic enema (ACE): a new therapeutic approach to chronic constipation. *Acta Gastroenterol Latinoam* 26(4):225-6, 1996.
376. Loening-Baucke V. Constipation in early childhood: patient characteristics, treatment and long term followup. *Am Fam Physician*, 49:397, 1994.
377. Loening-Baucke V. Factors determining outcome in children with chronic constipation and fecal soiling. *Gut*, 30:999,1989.
378. Loening-Baucke V. Factors responsible for persistence of childhood constipation. *J Pediatr Gastroenterol Nutr*, 6:915, 1987
379. Rockney R, McQuade, W et al. Encopresis treatment outcome: Long-term follow-up of 45 cases. *J Dev & Beh Ped*. 17(6): 380-385, 1996.
380. Sutphen JL, Borowitz SM, Hutchinson RL, Cox DJ. Long-term follow-up of medically treated childhood constipation. *Clin Pediatr (phila)* 34(11):576-80, 1995.
381. Chassagne P, Landrin I, Neveu C, Czernichow P, Bouaniche M, Doucet J, Denis P, Bercoff E. Fecal incontinence in the institutionalized elderly: incidence, risk factors, and prognosis. *Am J Med* 106(2):185-190 1999;1999.
382. Prosser S, Dobbs F. Case-finding incontinence in the over-75's. *Br J Gen Pract* ; 47:498-500, 1997.
383. Peet SM, Castleden CM, McGrother CW. Prevalence of urinary and fecal incontinence in hospitals and residential and nursing homes for older people. *Br Med J* ;311:1063-4, 1995.
384. Schultz A, Dickey G, Skoner M. *Urologic Nursing* ;17:23-8, 1997.
385. Brocklehurst J, Dickinson E, Windsor J. Laxatives and faecal incontinence in long-term care. *Nursing Standard* ;52:32-36, 1999.
386. Johanson JF, Irizarry F, Doughty A. Risk factors for fecal incontinence in a nursing home population. *J Clin Gastroenterol* ; 24:156-60, 1997.
387. Harari, D, Gurwitz JH, Choodnovskiy I, Avorn J, Minaker KL. Constipation: Assessment and management in an institutionalised population. *J Am Geriatr Soc* ;4 2:1-6, 1994.
388. Barrett JA, Brocklehurst JC, Kiff ES, Ferguson G, Faragher EB. Rectal motility studies in faecally incontinent geriatric patients. *Age and Ageing* ;19:311-317, 1990.
389. Percy JP, Neill ME, Kandiah TK et al. A neurogenic factor in faecal incontinence in the elderly. *Age Ageing* ;11:175-179, 1982.
390. Loening-Baucke V, Anuras S. Sigmoidal and rectal motility in healthy elderly. *J Am Geriatr Soc* ;32:887-891, 1984.
391. Loening-Baucke V, Anuras S: Effects of age and sex on anorectal manometry. *Am J Gastroenterol* ;80(1):50-53, 1985.
392. McHugh SM, Diamant NE. Effect of age, gender, and parity on anal canal pressures. *Dig Dis Sci* ;32(7):726-736, 1987.
393. Matheson DM, Keighley MRB: Manometric evaluation of rectal prolapse and faecal incontinence. *Gut* ;22:126-129, 1981
394. Ryhammer AM, Laurberg S, Bek KM. Age and anorectal sensibility in normal women. *Scand J Gastroenterol* ;32:278-84, 1997
395. Read NW, Abouzekry L, Read MG, Howell P, Ottewill D, Donnelly TC. Anorectal function in elderly patients with fecal impaction. *Gastroenterology* ;89:959-966, 1985
396. Rasmussen OO, Christiansen J, Tetzschner T, Sorensen M. Pudendal nerve function in idiopathic fecal incontinence. *Dis Colon Rectum* 43:633-6, 2000;
397. Vaccaro CA, Cheong DM, Wexner SD, Nogueras JJ, Salanga VD, Hanson MR, Phillips RC. Pudendal neuropathy in evacuatory disorders. *Dis Colon Rectum* ;38:166-71, 1995
398. Barrett JA, Brocklehurst JC, Kiff ES et al. Anal function in geriatric patients with fecal incontinence. *Gut* ;30:1244-1251, 1989
399. Robson KM, Kiely DK, Lembo T. Development of constipation in nursing home residents. *Dis Colon Rectum* ;43:940-3, 2000
400. Donald IP, Smith RG, Cruikshank JG, Elton RA, Stoddart ME. A study of constipation in the elderly living at home. *Gerontology* ;31:112-8, 1985.
401. Kinnunen O. Study of constipation in a geriatric hospital, day hospital, old people's home and at home. *Aging* ;3:161-170, 1991.
402. Harari, D, Gurwitz JH, Choodnovskiy I, Avorn J, Minaker KL. Correlates of regular laxative use in frail elderly persons. *Am J Med* ;99(4):513-8, 1995.
403. Harari D, Gurwitz JH, Minaker KL. Constipation in the elderly. *J Am Geriatr Soc* 41(10):1130-1140, 1993.
404. Jones RH, Tait CL. Gastrointestinal side-effects of NSAIDs in the community. *Br J Clin Pract* ;49:67-70, 1995.
405. Singaram C, Ashraf W, Gaumnitz EA et al. Dopaminergic defect of enteric nervous system in Parkinson's disease patients with chronic constipation. *Lancet* ;346:861-64, 1995.
406. Bassotti G, Maggio D, Battaglia E et al. Manometric investigation of anorectal function in early and late stage Parkinson's disease. *J Neurol Neurosurg Psychiatry* ;68:768-770, 2000.
407. Muller-Lissner SA: Effect of wheat bran on the weight of stool and gastrointestinal transit time: a meta analysis. *Br Med J* 296:615-617, 1988.
408. Towers AL, Burgio KL, Locher JL, Merkel IS, Safaiean M, Wald A. Constipation in the elderly: Influence of dietary, psychological, and physiological factors. *J Am Geriatr Soc* ;42:701-6, 1994.
409. Klauser AG, Schindlbeck NE, Muller-Lissner SA: Low fluid intake lowers stool output in healthy male volunteers. *Z Gastroenterol* 28(11):606-609, 1990.
410. Klauser AG, Voderholzer WA, Heinrich CA, Schindlbeck NE, Muller-Lissner SA. Behavioural modification of colonic function: can constipation be learned? *Dig Dis Sci* ;35:1271-5, 1990.
411. Harari D, Patel M, Rudd SG, Wolfe CDA. Prevalence, corre-

- lates, and impact of new-onset faecal incontinence following stroke. *J Cerebrovascular Dis* (in press).
412. Wald A, Tunuguntla AK. Anorectal sensorimotor dysfunction in fecal incontinence and diabetes mellitus. *NEJM* ;310:1282-7, 1984.
 413. Sun WM, Katsinelos P, Horowitz M, Read NW. Disturbances in anorectal function in patients with diabetes mellitus and faecal incontinence. *Euro J Gastroenterol Hepatology* ; 8:1007-12, 1996.
 414. Harari D, Gurwitz JH, Avorn J, Bohn R, Minaker KL. Bowel habits in relation to age and gender: Findings from the National Health Interview Survey and clinical implications. *Arch Int Med*;156:315-320, 1996.
 415. Goulding A, Taylor RW, Keil D, Gold E, Lewis-Barned NJ, Williams SM. Lactose malabsorption and rate of bone loss in older women. *Age and Ageing* ; 28:175-180, 1999.
 416. Al-Eidan FA, McElnay JC, Scott MG, Kearney MP. Clostridium difficile-associated diarrhoea in hospitalised patients. *J Clin Pharm Therapeutics* ;25:101-9, 2000.
 417. Morgan R, Spencer B, King D. Rectal examinations in elderly subjects: attitudes of patients and doctors. *Age and Aging*; 27:353-6, 1998.
 418. Engel AF, Kamm MA, Bartram CI, Nicholls RJ. Relationship of symptoms in faecal incontinence to specific sphincter abnormalities. *Int J Colorect Dis* ;10:152-5, 1995.
 419. Harari D, Minaker KL. Megacolon in patients with chronic spinal cord injury. *Spinal Cord* ;38:331-339, 2000.
 420. McKay LF, Smith RG, Eastwood MA et al: An investigation of colonic function in the elderly. *Age Ageing* 12:105-110, 1983.
 421. Starrveld JS, Pols JS, Van Wijk MA et al: The plain abdominal radiograph the assessment of constipation. *Z Gastroenterol* 28:335-338, 1990.
 422. Chassagne P, Jeco A, Gloc P et al. Does treatment of constipation improve faecal incontinence in institutionalized patients. *Age Ageing* :29:159-164, 2000.
 423. Petticrew M, Watt I, Sheldon T. Systematic review of the effectiveness of laxatives in the elderly. *Health Technology Assessment* ;1(13):i-iv,1-52, 1997.
 424. Puxty JA, Fox RA. Golytely: a new approach to faecal impaction in old age. *Age Ageing* ;15(3):182-184, 1986.
 425. Korzets A, Dicker D, Chaimoff C, Zevin D. Life-threatening hyperphosphatemia and hypocalcemic tetany following the use of Fleet enemas. *J Am Geriatr Soc* 40:620-1, 1992.