A rectocele consists of rectal mucosa, and represents a pulsion diverticulum of the lower anterior rectum. The mucosa herniates through a defect in the midline in the lower anterior rectal muscle wall. This defect then extends into the internal and external anal sphincters. It is a consistent finding in women with faecal incontinence due to anal sphincter disruption. If the anterior rectal muscle wall is repaired first, anal sphincter repair is facilitated.

The nature and pathology of rectocele and anal sphincter disruption have received more attention since the advent of improved ultrasound imaging in this anatomical area.1-4 Most clinicians attribute faecal incontinence to anal sphincter deficits, while obstructed defaecation is related to rectocele.4-5 In general, these are regarded as two separate problems. At present, there is no clear definition of rectocele, while the anal sphincter deficit is self-explanatory.

The vast majority of patients who present with symptoms of faecal incontinence and difficulty in evacuation are female. The pelvic floor disruption is generally attributed to childbirth. If anal sphincter damage is recognised immediately at the time of delivery, it is usually repaired by the attendant obstetrician.6 However, most women only present with symptoms later – even many years later. Delayed repair is usually performed by colorectal surgeons.6,7

The approach in delayed repair of anal sphincter disruption is usually through the perineum. A number of different approaches have been described for rectocele repair, with some preferring a transabdominal approach.6 More commonly a translabial approach is used.6,9 Recently the use of mesh has become popular, although there are sporadic reports of infection and dyspareunia.8,10 There are no specific reports of repair of both rectocele and anal sphincter at the same time through a transperineal approach.

This paper presents individual experience of a significant number of selected female patients with faecal incontinence and/or obstructed defaecation. All had a history of vaginal delivery, with or without evidence of associated perineal injury at the time. In treating these patients, the underlying muscular defect was investigated and an appropriate method of repair instituted. This new understanding has resulted in surgical correction of the underlying rectal muscle and anal sphincter anatomical defect, rendering the use of mesh unnecessary.

Methods
Patient selection
All female patients presenting from 1995 to 2007 were entered into the study. All patients were subjected to a standard history and clinical examination in the consulting room. This included a digital rectal examination and proctoscopy. Patients were then referred for a carefully directed translabial ultrasound scan of the rectum and anal sphincters. The ultrasound examination was performed by a radiologist with specific understanding of the pathology. Conventional two-dimensional ultrasound imaging was performed using a 4 - 8 MHz curved array probe. The patient was in the left lateral position, and a translabial scan was performed in the axial and sagittal planes. Three- or four-dimensional endo-ultrasound was not available (Fig. 1). Anal manometry was available but was not performed routinely. The overall findings were discussed with the individual patient and informed consent for surgery was obtained.

Summary
This study reports on the surgical anatomy and technique of perineal repair in a selected group of parous women with faecal incontinence and/or difficulty in evacuation. Anal sphincter muscle damage is usually attributed to childbirth, although most of these women present for the first time years later.

Consecutive patients with the above symptoms were examined clinically and then investigated with a perineal ultrasound scan. During the perineal operation for repair, further investigation by transillumination and measurements with calipers were done in 50 patients. All patients received routine postoperative care, and were followed up for at least 6 months.

From 1995 to 2009 a total of 117 patients, all female, underwent perineal repair by a single surgeon. The age range was 24 - 82 years. In the last 50 consecutive patients, transillumination was positive prior to repair in all, and negative after. The average thickness of the rectocele wall was 2.4 mm prior to repair and 4.8 mm after. In all patients, a rectocele was found in conjunction with the anal sphincter defect. The results of combined repair were satisfactory in 109 of 117 patients (93%).

Fig. 1. Ultrasound scan showing defect in sphincters.
Transillumination
Once the patient was anaesthetised and placed in the lithotomy position, the rectocele was investigated with transillumination. Flexible sigmoidoscopy was performed to reach the mid-sigmoid colon. On withdrawal, the rectocele was identified and the tip of the sigmoidoscope passed into it. Transillumination was positive if the light was clearly visible (Fig. 2).

Caliper measurement
The combined thickness of the rectocele wall and posterior vaginal wall was then measured at the apex of the rectocele with a Harpenden Skinfold Caliper (Baty, Sussex, UK). This is a registered class I medical device with the following specifications: dial graduation 0.2 mm; measuring range 0 - 80 mm; measuring pressure 10 g/mm², constant over range; accuracy 99.0%; repeatability 0.2 mm.

One measurement was taken before repair and one on completion (Fig. 3).

Surgical technique (Figs 4 - 6)
The operation is performed through a transverse perineal incision. The sphincters are dissected in the midline only, and then the rectocele is dissected from the posterior vaginal wall and reduced. The upper limit of the rectocele is identified as divarication of the longitudinal muscle of the lower rectum. The repair starts at this point, and is completed in layers. As the first layer is plicated from proximal to distal in the midline, the rectocele is obliterated, the longitudinal muscle of the rectum approximated, and distally the internal anal sphincter reconstructed. The second layer approximates the external anal sphincter. This is reinforced by plication of the levator ani muscle at the level at which it blends into the exter-
nal sphincter. Finally the perineal body is reconstituted. A urinary catheter is inserted on completion, and the patient is kept in hospital until after the first bowel movement.

Postoperative care
All patients were seen routinely in the consulting room and reviewed finally after 6 months. Patients were subsequently followed up on an ad hoc basis or during consultations for other complaints.

Results
Over the 13-year period 1995 - 2009, a total of 117 patients were operated on, all female. The age range was 24 - 85 years (Fig. 7).

Clinical findings
No specific scoring system was applied to the faecal incontinence. In every case it was socially unacceptable and had restricted the patient's lifestyle. The patients with difficulty in evacuation used various forms of digital assistance. In the initial clinical evaluation, digital rectal examination was invariably diagnostic.

Radiological findings
In the last 50 consecutive patients a degree of disruption of both the internal and external sphincter was visible on careful review of the ultrasound scan. The rectocele was not demonstrated (Fig. 1).

Intra-operative measurement
In the last 50 consecutive patients, caliper measurements and transillumination were performed. The pre-operative caliper measurement was taken at the apex of the rectocele and the postoperative measurement just above the reconstituted sphincters (Figs 2 and 3). The results are set out in Table I. Transillumination was positive in all 50 cases before repair, and negative after repair. Interestingly, a rectocele is not easily identified on routine endoscopic inspection, even under anaesthesia. A conscious effort and manual guidance are necessary for the sigmoidoscope to enter the diverticulum.

Results of surgery
In all patients with anal sphincter disruption, there was a concomitant rectocele. Of the 117 patients, 109 had satisfactory improvement with regard to faecal incontinence after 6 months. All 43 patients with incontinence and obstructed defaecation reported normal evacuation. Complications occurred in 2 patients, who developed gross perineal sepsis in the immediate postoperative period and required a defunctioning colostomy. After this, the perineum healed well, and the colostomy was closed approximately 4 months later.

Discussion
Rectocele is variously described as a tear in the rectovaginal septum or a prolapse of the posterior vaginal wall. Some describe it as a prolapse of the anterior rectal wall into the posterior vagina. These varied descriptions make it clear that the exact nature of rectocele is not well understood. From this personal series it is apparent that a rectocele comprises rectal mucosa only, and is a pulsion diverticulum of the anterior lower rectum. Without this understanding, long-lasting functional sphincter repair can be quite difficult to achieve. It is well recorded that repair of the sphincter alone results in good initial improvement in continence, but that there is deterioration after 1 or 2 years. The presence of a rectocele was a constant finding in all the cases reported above, in association with the sphincter deficit. If this is not repaired at the same time, it may be a factor in the later disruption of the sphincter repair.

Transillumination is a simple but valid clinical observation. It certainly contributes to an understanding of the nature of the rectocele. As the rectocele consists only of the rectal mucosa, it allows transmission of light. Once the muscle layer is reconstituted, the light is no longer visible.

A literature search of anatomy and pathology textbooks yielded no data on the normal thickness of the muscle layer of the rectal wall. During surgical dissection, it is difficult to identify the muscle layer of the rectum unless there is a clear understanding of the nature of the defect and the thickness of the muscle layer. It is also important to recognise that the defect invariably extends into the internal anal sphincter, and often into the external anal sphincter as well. The variation in symptomatology represents a spectrum that is probably related to the variation in size and extent of the defect. In other words, it is not necessary to distinguish types or extent of incontinence as all will be repaired in the same way. Similar to a hernia, the pulsion diverticulum (and muscle divarication) enlarges over time, possibly accounting for the delay in presentation.

There is no doubt that the technology and technique of ultrasound scanning in this anatomical region has improved. The extent of the sphincter disruption is usually clearly visible. In the patients reported here, only conventional 2D ultrasound imaging was available. However, this was more than adequate to demonstrate the need for surgical repair.

The nature and extent of rectocele are difficult to demonstrate with conventional two-dimensional, and even three-dimensional, ultrasound imaging. The rectocele can only be fully assessed at the time of surgical dissection. Starting with rectocele repair makes the sphincter anatomy easier to demonstrate, and extensive lateral dissection is not required. This probably contributes to obtaining

### Table I. Rectocele Wall Measurements (N=50)

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<thead>
<tr>
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<th>Pre-operative</th>
<th>Postoperative</th>
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</thead>
<tbody>
<tr>
<td>Average</td>
<td>2.4 mm</td>
<td>4.8 mm</td>
</tr>
<tr>
<td>Range</td>
<td>2.2 - 2.6 mm</td>
<td>4.5 - 5.3 mm</td>
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Fig. 7. Age distribution of 117 patients undergoing perineal repair.
a longer-lasting repair. Although the longitudinal muscle layer of the lower rectum is relatively thin, it obviously plays a crucial role in continence and evacuation. It must be free to contract physiologically and not be held rigid, as when a non-absorbable mesh is placed.

In older women, there is a tendency to recurrence of pelvic floor weakness. Reinforcement with an absorbable mesh is an attractive option and may bolster the middle pelvic compartment, preventing subsequent rectal prolapse and/or enterocele. However, this aspect obviously requires further careful study.

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Invited comment

The nature and pathophysiology of a rectocele are poorly understood. Our view is that it is a prolapse of the anterior rectal wall into the posterior vagina. It can also be seen as a pulsion diverticulum of the anterior lower rectum. Divarication of the longitudinal muscle of the lower rectum is not always present. In fact, our impression is that it is present in a minority of patients, although the muscle is invariably stretched out.

Rectoceles are almost always associated with a perineal body defect (anal sphincter defect). Excessive stretching of the perineum during childbirth with incomplete recovery afterwards is the main cause of such a defect. A visible obstetric tear is not a prerequisite. Not all perineal body or anal sphincter defects are associated with anal incontinence. A perineal body defect can also contribute towards obstructed defecation. There is no recognised definition of a perineal body defect. In our unit we regard it as a perineal thickness of less than 5 mm just above the superficial external anal sphincter. The defect mainly involves the deep part of the external sphincter.

Transperineal ultrasound has greatly contributed to the diagnosis and evaluation of rectocele. The muscular content of the perineal body can be distinguished from its fibrous tissue and the muscle thickness can be measured (unpublished data). However, ultrasound is insufficient for complete evaluation of a rectocele and perineal body defect.

In our unit, we use the ‘pyramid sign’ for diagnosing a rectocele. With a finger in the lower rectum, the anterior rectal wall is firmly pushed anteriorly. A measurement is made from where the skin and vaginal epithelium meet (fourchette) to the top of the ‘pyramid’: 1 cm is stage 1, 2 cm is stage 2 and 3 cm is stage 3. When the rectocele clearly extends upwards (superiorly) in the presence of a stage 3, we call it stage 4 (unpublished data).

With the index finger still in the rectum, the thumb is pressed on the perineal skin and the perineal thickness is evaluated between the two fingers.

For many years, we have combined repair of rectocele and perineal body defect. Since we have been using transperineal ultrasound routinely, we have recognised sub-clinical rectoceles covered by mesh after a previous repair. Currently we do not regard mesh as a suitable treatment method for a rectocele and a perineal body defect.

Our treatment for rectocele consists of plication of the rectocele. With a finger in the rectum, longitudinal absorbable sutures are placed over the entire rectocele and tied. Four to six sutures are used in one layer. The perineal body is repaired by first opening the pararectal spaces on both sides as distally as possible. This allows proper placement of usually three absorbable sutures in the deep part of the external sphincter. The muscle is pulled over the ano-rectum and sutured in the midline. Levator plication is avoided owing to the risk of dyspareunia.

Our short-term results are similar to the results mentioned in the above article. Obstructive symptoms improved from 62.1% to 3.9% and constipation improved from 58.6% to 3.9% (N=29) (data still unpublished).

In summary, we agree with the author on several issues. Perineal body defects (anal sphincter defect) and rectoceles are separate entities and treated differently. Mesh is not a treatment for either of these conditions. A rectocele is treated by plication (or a STARR procedure in selected cases), and a perineal body defect by repair of the sphincter. There is no consensus on the evaluation and staging of these conditions.

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