Anal incontinence can have a devastating effect on a woman’s quality of life, and vaginal delivery has been implicated as a significant causal factor. Neurogenic trauma of the pelvic floor and mechanical trauma to the anal sphincters have been shown previously. Denservation injuries of the pubococcygeus and external sphincter muscles occur after 42% to 80% of vaginal deliveries and are associated with multiparity, a long second stage of labor, third-degree tears, and high birth weight. This nerve damage was progressive and noted in women with stress incontinence of urine and incontinence of flatus. In the majority of women, however, this nerve injury returns to normal within 2 months after delivery and does not seem to correlate with symptoms. Postpartum anal endosonographic examination has shown occult sphincter defects that are directly implicated in the pathogenesis of fecal urgency and anal incontinence. The effect of pregnancy and childbirth on anal sphincter pressures and anal sensation, as well as the relationship of these to symptoms of incontinence, is unclear because most studies are small or are not prospective. Decreased anal squeeze pressures and vaginal pressures have been shown after vaginal delivery, as well as transient impairment of anal sensation. However, the relationships between anal sensation and anal sphincter defects or sphincter pressure have not been investigated previously.

The aims of this study were to prospectively establish the incidence of mechanical and neurophysiologic damage to the anal canal in a cohort of nulliparous women before and after delivery. In addition, we aimed to establish the association between specific obstetric variables to identify risk factors for both objective and subjective bowel dysfunction.

Material and methods

The study was approved by the Research and Ethics Committee of St George’s Hospital, and all women gave informed consent. Consecutive nulliparous women who attended the antenatal clinics of St George’s Hospital between April 1996 and August 1997 were invited to participate in this study. All women were evaluated after 34 weeks’ gestation and asked to attend for repeated investigations 12 weeks after delivery. Exclusion criteria included preexisting diabetes mellitus, neurologic disorders, a hi-
tory of anorectal surgery or abnormalities, and inflammatory or irritable bowel syndromes. All women completed a structured symptom questionnaire that included questions about fecal urgency, incontinence of flatus, liquid stool, and solid stool. Fecal urgency was defined as the inability to defer defecation for more than 5 minutes. Fecal incontinence included either incontinence of flatus, liquid stool, or solid stool. To avoid selection bias for recruitment, both hospital and community antenatal clinics were included; therefore the sample is very representative of our local nulliparous population. Of the 549 women who agreed to complete the symptom questionnaire, 286 agreed to attend for investigations (study group).

Anal manometric and sensation evaluations were performed. At 12 weeks postpartum the same investigations were repeated together with anal endosonographic examination. All investigations were performed while subjects were in the left lateral position. Anal manometry was performed with the modified Stryker intracompartmental pressure monitor (Stryker, Kalamazoo, Mich) attached to an air-filled microballoon.1 Anal resting and squeeze pressures were measured at 1-cm intervals along the anal canal from the anal verge with use of a station pull-through technique.1

Anorectal sensation was measured with a bipolar platinum ring electrode (model 21L, 10, Danpec, Bristol, United Kingdom) mounted on a 14-gauge Foley catheter. The electrode was connected to a constant current stimulator (Neuromatic 2000C, Danpec) and recordings were taken at 1-cm intervals from the anorectal margin to the anal verge. The current across the electrodes was increased in increments of 0.1 mA until a threshold sensation of burning or tingling was reported. Three measurements of the threshold were taken at each level, and the mean value was calculated.

Anal endosonographic examinations were performed with use of the Kretz Combison-530 machine (Kretz Technik, Zepf, Austria), with a rotating 7-MHz probe providing a 360-degree image. The probe is initially inserted while the woman is in the left lateral position until the U-shaped puborectalis muscle is identified. As the probe is withdrawn down the anal canal, images of the puborectalis, external anal sphincter, longitudinal muscle, and internal anal sphincter are taken.12

The endosonographic interpretation of the sphincter muscles has been validated previously.15 The internal sphincter appears as a uniformly hypoechoic ring, and the external sphincter lies outside this, appearing as a heterogeneous hyperechoic ring of muscle. An external sphincter defect appears as a break in the hyperechoic texture of the external muscle ring (Fig 1), and an internal sphincter defect appears as a gap in the hypoechoic ring (Fig 2). For those women who did not return for postnatal assessment, the symptom questionnaires were completed by telephone.

Delivery was managed according to the standard protocol for active management of labor in our unit. All episiotomies were right mediolateral, and any trauma to the anal sphincter was classified as third degree. Pregnancy and delivery details were obtained from the patient case records after the postnatal visit. Obstetric variables analyzed included mode of delivery, epidural usage, augmentation of labor, duration of labor, perineal trauma, fetal weight, and fetal head circumference.

Information on labor and delivery were transferred directly to the Access database (Microsoft, Redmond, Wash) from the case notes after the investigations were completed. Stata statistical software (release 5.0, Stata Corp, College Station, Texas) and BIOCNF (www.sghms.ac.uk/depts/staff/jmb/jmbsoft.htm) were used for analysis.

Symptoms before, during, and after pregnancy were compared with use of the McNemar test for paired dichotomous data. The paired t test was used to compare antenatal and postnatal anorectal physiologic measurements. Associations between categorical variables were assessed with use of the χ² test. Differences in the mean values of continuous variables between groups were assessed with use of the 2-sample t test. Log-transformations were used when appropriate. A P value <.05 was considered to be significant, and the test statistic has been presented when appropriate. All reports are presented as mean values (SD). Confidence intervals for proportions were calculated by the exact binomial method because some of the numerators were very small.

Results

Two hundred eighty-six women attended for antenatal assessment, and 161 of these returned 12 weeks postpartum. The obstetric and neonatal data of the study population was compared with the overall nulliparous population registered during the same period. The demographic and delivery data were compared and appeared to be representative, although there was a higher rate of deliveries with forceps and epidural usage in our study population compared with the overall nulliparous population. The mean gestation at attendance was 37 weeks (range, 34 to 41 weeks), and the age at attendance was 29 years (range, 17 to 43 years). Of the 161 women who returned for postnatal investigations, 89 had a spontaneous vaginal delivery, 31 had a caesarean section (9 of which were performed before labor), and 41 had an instrumental delivery. The mean duration of labor was 582 (SD, 349) minutes for the first stage, 53 (SD, 33) minutes (defined as the time from full cervical dilatation to the onset of maternal pushing) for the passive second stage, and 36 (SD, 31) minutes (defined as the time from the onset of maternal pushing to delivery) for the active second stage. Three (1.9%) women sustained a third-degree tear.

In the 286 women who attended for antenatal investigations, the prevalence of fecal urgency before, during,
and after pregnancy was 3 (1%), 27 (9.4%), and 30 (10.5%), respectively; the prevalence of fecal incontinence was 4 (1.4%), 20 (7.0%), and 25 (8.7%), respectively. In those who attended both the antenatal and postnatal investigations (n = 161), the prevalence of fecal urgency before, during, and after pregnancy was 3 (1.8%), 18 (11.2%), and 29 (18.1%), respectively (Table I). Of these women, 25 had urgency or fecal incontinence in pregnancy; in 3 of these 25 women, incontinence had begun before pregnancy. Twenty-nine (18%) women had new symptoms postpartum, 10 (6.2%) of whom had symptoms of fecal incontinence. In this group of women, new symptoms were reported by 14 (15.7%) women in the spontaneous vaginal delivery group (n = 89), 8 (19.5%) women in the instrumental delivery group (n = 41), and 7 (22.5%) women in the cesarean section group (n = 31). Two women reported new symptoms after an elective caesarean section. In the caesarean section group, 3 women reported urgency, 3 women reported incontinence of flatus, and 1 woman reported incontinence of both flatus and urgency. The prevalence of fecal symptoms was higher in those women who returned for investigations compared with those who did not return, and these differences were statistically significant.

The mean maximum anal squeeze pressures decreased significantly after delivery, from 108 ± 28 mm Hg to 99 ± 29 mm Hg. There were no significant changes in maximum resting pressures and anal sensation. Vaginal delivery, particularly instrumental delivery, resulted in a significant decrease in maximum squeeze pressure (P = .015), and this was not dependent on type of instrument used (Table II). The maximum resting pressure was also decreased in the instrumental delivery group compared with the cesarean section group (P = .002). There was no relationship between antenatal or postnatal anal sensation and anal manometry measurements.

Anal endosonographic examinations were performed on 156 women postpartum (5 women refused this investigation), of whom 59 (38%) revealed evidence of sphincter damage. Ten (6%) had an internal sphincter defect, 35 (23%) an external sphincter defect, and 14 (9%) had combined internal and external sphincter defects. Only 1 woman sustained trauma to the external sphincter after a caesarean section.

The presence of a sphincter defect was associated with a significant decrease in anal resting pressures (P = .02), and squeeze pressures (P = .001) but no effect on anal sensation (Table III). In women with isolated external sphincter defects, the maximum squeeze pressures were significantly lower than those in women with intact sphincters (P = .006). There was no effect on anal pressures or sensation in those who had internal sphincter defects only. In women with combined sphincter defects, the resting pressures (P = .002) and squeeze pressures (P = .0005) were significantly lower than those in women with intact sphincters. The presence of symptoms was not related to anal pressures, sensation, or sphincter integrity.

Perineal trauma (episiotomy and second- and third-degree tears) was associated with a significant decrease in maximum squeeze pressures compared with women with an intact perineum (intact versus tears, P = .022; intact versus episiotomy, P = .032). Women with intact perineum, spontaneous perineal tears, and episiotomies had mean squeeze pressures of 105.8 ± 26.4 mm Hg, 92.3 ± 30.3 mm Hg, and 92.2 ± 29.7 mm Hg, respectively.

Vaginal delivery, perineal trauma, a longer active second stage, and a longer total length of the second stage of labor.
were associated with postpartum sphincter trauma (Tables IV and V). Because vaginal delivery were the most significant factor in this analysis ($P < .001$), the data were reanalyzed for those women who had vaginal delivery only, and it was found that the presence of sphincter trauma was still associated with the occurrence of significant perineal trauma at delivery ($P = .02$; defined as greater than first-degree tears and episiotomies; Tables IV and V).

**Comment**

This large prospective study shows that a woman’s first pregnancy and delivery is associated with a high prevalence of objective and subjective bowel dysfunction. The prevalence of fecal symptoms was higher in those women who returned for investigations compared with those who did not return, and that might have influenced their decision to return for follow-up. It may also explain why only 56% of women returned because these tests may have been uncomfortable and women with more symptoms may have been more motivated to return. Vaginal delivery and not cesarean section resulted in a significant decrease in anal squeeze pressure and postpartum sphincter trauma, indicating that delivery, rather than pregnancy alone, predisposes women to pelvic floor trauma. The significant association between reduced anal squeeze pressures and external and combined internal and external sphincter defects confirms that mechanical disruption is the major contributory factor. This is consistent with studies that have shown that the external sphincter contributes mainly to anal squeeze pressure and the internal sphincter mainly to anal resting pressure. Accordingly, combined internal and external sphincter defects were associated with a decrease in both resting and squeeze pressures, showing the contribution of both sphincters to overall anal canal pressures.

Reduced anal squeeze pressures may also result from pudendal nerve damage, connective tissue damage, and pelvic floor support disruption inasmuch as it was also observed in cases in which the ultrasound findings were normal. The reduction in anal squeeze pressures may show recovery with time. Cornes et al\(^\text{11}\) found an improvement in anal squeeze pressures at 6 months postpartum, although the pressures were still lower than control subjects and women who had cesarean sections. As in this study, Cornes et al\(^\text{11}\) did not find a relation of symptoms to a lower anal squeeze pressure. This is either because anal squeeze pressure is not responsible for continence or because it can be compensated for by adequate basal anal resting pressures. This overall decrease in anal pressures may be permanent because it has been shown to be lower in women who have undergone previous vaginal and forceps deliveries compared with primigravidae.\(^\text{10}\)

The high prevalence of sphincter trauma observed in 38% of nulliparous women is in agreement with previous studies that used anal ultrasound alone\(^\text{1}\) and anal ultrasound combined with anal vector manometry.\(^\text{2}\) Despite the high prevalence of sphincter defects, fewer than half of the women who had sphincter defects had symptoms, which contrasts with Sultan et al\(^\text{1}\) who found a significant association between the presence of a defect and symptoms. This may be the result of differences in our study population because our women had a high prevalence of antenatal symptoms. Furthermore, we found that the predominant sphincter defect in this study involved the external rather than the internal sphincter. However, it does confirm that sphincter trauma is a common occurrence in nulliparous women after a vaginal delivery. This study evaluated alterations in anal sensation in a pregnant population by use of a conventional validated form of electrostimulation. In the only other prospective study, a transcutaneous electrical nerve stimulation skin stimulator—a technique that has not been validated—was used, and no change was found in anal electrosensitivity after vaginal delivery.\(^\text{9}\) Cornes et al\(^\text{11}\) measured anal sensation 10 days postpartum in 96 primiparae and again in 76 of these women at 6 months postpartum. They noted that at 10 days postpartum, anal sensation was significantly impaired after vaginal and forceps delivery at all levels in the anal canal, as well as in the midanal canal after a ventouse delivery. In contrast, anal sensation was unaffected by cesarean section. However, these early changes may have been a consequence of decreased anal pressures, which may affect the validity of anal sensation measurements inasmuch as laxity of the anal mucosa.

**Table I.** Fecal symptoms before, during, and after pregnancy in women who attended for antenatal and postnatal investigations.

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Before pregnancy</th>
<th>During pregnancy</th>
<th>Postnatal</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No. (%)</td>
<td>95% CI</td>
<td>No. (%)</td>
</tr>
<tr>
<td>Fecal urgency</td>
<td>3 (1.8%)</td>
<td>0.4% to 9.3%</td>
<td>18 (11.2%)</td>
</tr>
<tr>
<td>Flatus incontinence</td>
<td>1 (0.6%)</td>
<td>0.0% to 3.4%</td>
<td>12 (7.5%)</td>
</tr>
<tr>
<td>Soiling of underwear</td>
<td>0</td>
<td>0.0% to 2.3%</td>
<td>4 (2.5%)</td>
</tr>
<tr>
<td>Liquid stool incontinence</td>
<td>0</td>
<td>0.0% to 2.3%</td>
<td>1 (0.6%)</td>
</tr>
<tr>
<td>Solid stool incontinence</td>
<td>0</td>
<td>0.0% to 2.3%</td>
<td>0</td>
</tr>
<tr>
<td>Any incontinence</td>
<td>1 (0.6%)</td>
<td>0.0% to 3.4%</td>
<td>15 (9.3%)</td>
</tr>
<tr>
<td>Urgency or incontinence</td>
<td>3 (1.9%)</td>
<td>0.4% to 5.3%</td>
<td>25 (15.5%)</td>
</tr>
</tbody>
</table>

CI, Confidence interval.
would decrease contact with the stimulating probe. The recorded changes in anal sensation were not related to anal incontinence symptoms, and by 6 months postpartum they had returned to normal. Changes in anal sensation may therefore be transient and recover with time.

The association of perineal trauma and vaginal delivery with sphincter defects has important implications for obstetric practice. Vaginal muscle disruption may predispose the anal sphincters to direct trauma from passage of the fetal head during vaginal delivery because the incidence of sphincter damage increases significantly when an episiotomy occurs together with a perineal tear. Method to minimize perineal trauma and the potential risk of sphincter trauma include use of antenatal perineal massage, preference of vacuum extraction over forceps, and correct management of the active second stage of labor.

Recognition of perineal trauma is poor and may account for the discrepancy between the low incidence of third-degree tears compared with the higher incidence of occult sphincter trauma. Poor recognition of perineal trauma and consequent inadequate repair will predispose women to the development of incontinence. Both doctors and midwives need improved and focused training in the recognition and repair of sphincter trauma.

The association between vaginal delivery and sphincter trauma suggests that caesarean section may reduce the incidence of sphincter trauma.
cidence of occult sphincter trauma. It may be difficult, however, to justify the overall increase in death and disease associated with cesarean section over vaginal delivery, especially if such defects may not always lead to the development of incontinence, as shown in this study. Cesarean section may be advisable in cases in which transient anal incontinence is observed after vaginal delivery because relapse of symptoms is more likely after subsequent deliveries. In women who have had previous anal sphincter tears or who already have symptoms, additional deliveries may further compromise pelvic floor function. Elective cesarean section may be beneficial in these women, although it is preferable to perform anorectal physiologic studies to exclude pelvic floor compromise before vaginal delivery is advised.

In conclusion, this study has shown that a high prevalence of sphincter defects are associated with vaginal delivery and perineal trauma. However, the clinical relevance of these findings is uncertain because the prevalence of anal incontinence was much lower in this population. Physiologic studies to exclude pelvic floor compromise before vaginal delivery is advised.

### REFERENCES


