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ANAL-SPHINCTER DISRUPTION DURING VAGINAL DELIVERY

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Abstract *Background.* Lacerations of the anal sphincter or injury to sphincter innervation during childbirth are major causes of fecal incontinence, but the incidence and importance of occult sphincter damage during routine vaginal delivery are unknown. We sought to determine the incidence of damage to the anal sphincter and the relation of injury to symptoms, anorectal physiologic function, and the mode of delivery.

Methods. We studied 202 consecutive women six weeks before delivery, 150 of them six weeks after delivery, and 32 with abnormal findings six months after delivery. Symptoms of anal incontinence and fecal urgency were assessed, and anal endosonography, manometry, perineometry, and measurement of the terminal motor latency of the pudendal nerves were performed.

Results. Ten of the 79 primiparous women (13 percent) and 11 of the 48 multiparous women (23 percent) who delivered vaginally had anal incontinence or fecal urgency when studied six weeks after delivery. Twenty-eight of the 79 primiparous women (35 percent) had a sphincter defect on endosonography at six weeks; the defect persisted in all 22 women studied at six months. Of the 48 multiparous women, 19 (40 percent) had a sphincter defect before delivery and 21 (44 percent) afterward. None of the 23 women who underwent cesarean section had a new sphincter defect after delivery. Eight of the 10 women who underwent forceps delivery had sphincter defects, but none of the 5 women who underwent vacuum extractions had such defects. Internal-sphincter defects were associated with a significantly lower mean (±SD) resting anal pressure (61±11 vs. 48±10 mm Hg, $P<0.001$) six weeks post partum, and external-sphincter defects were associated with a significantly lower squeeze pressure (increase above resting pressure, 70±38 vs. 44±13 mm Hg; $P<0.001$). There was a strong association ($P<0.001$) between sphincter defects and the development of bowel symptoms.

Conclusions. Occult sphincter defects are common after vaginal delivery, especially forceps delivery, and are often associated with disturbance of bowel function. (N Engl J Med 1993;329:1905-11.)

CHILDBIRTH may be accompanied by mechanical or neurologic injury to the anal sphincter. Overt sphincter damage due to a third-degree or fourth-degree tear[1] occurs in approximately 0.7 percent of women undergoing vaginal delivery in centers where posterolateral episiotomy is practiced.[2,3] Inadequate primary repair of these sphincter injuries can lead to early fecal incontinence.[3,4] Pudendal-nerve conduction can also become impaired after vaginal delivery,[5] and the later development of fecal incontinence has been attributed to progressive denervation of the anal-sphincter muscles.[6-8] Some women sustain both mechanical and neurologic trauma during vaginal delivery.[9]

Until recently, defects of the external anal sphincter were detected by electromyography,[10] and defects of the internal sphincter were inferred from measurement of a low resting anal pressure.[11] Anal endosonography, however, has allowed accurate imaging of both sphincter muscles,[12,13] leading to the recognition of unsuspected defects of the external sphincter in women thought to have purely neurogenic fecal incontinence[14] and the detection of internal-sphincter damage when only an external-sphincter defect was suspected.[15] In a study of 62 women with fecal incontinence related to obstetrical procedures, anal endosonography revealed an external-sphincter defect in 90 percent and an internal-sphincter defect in 65 percent.[15]

Because most previous studies of anal-sphincter function were either retrospective[14,15] or attributed the development of fecal incontinence directly to pudendal-nerve damage,[5,6,16] we undertook a prospective study of women before and after delivery, using anal endosonography and anorectal neurophysiologic tests to establish the incidence of mechanical and neurologic trauma during childbirth.

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METHODS

Subjects

We studied 202 unselected, consecutive women (median age, 28 years; range, 18 to 43) who had been pregnant for more than 34

weeks. Of the 202 women, 135 had never had a vaginal delivery (including 2 who had had a cesarean section) and 67 had had one or more vaginal deliveries.

This study was approved by the Research Ethics Committee of the City and Hackney District Health Authority, and all the women gave informed consent.

Delivery and Evaluation

All women were examined during the last six weeks of pregnancy and were asked to return for reevaluation six to eight weeks after delivery. At each assessment an interviewer completed a detailed questionnaire that reported any symptoms of fecal urgency (the inability to defer defecation for more than five minutes) and anal incontinence. Anal endosonography, manometry, studies of pudendal-nerve terminal motor latency, and perineometry were then performed. Information on labor and delivery was obtained from hospital records.

Delivery was managed as deemed appropriate by the attending physician. All episiotomies were posterolateral. Uncomplicated episiotomies were repaired by senior house officers and qualified midwives, whereas complicated episiotomies were repaired by more senior physicians. Tears related to delivery were classified as follows: first-degree tears involved only vaginal epithelium (women with such tears were included among those with no laceration); second-degree tears involved the perineal body but not the external sphincter; third-degree tears involved the external sphincter; and fourth-degree tears involved both the external sphincter and the anal epithelium.

Women with defects on ultrasonography or prolonged terminal motor latency of the pudendal nerves six weeks after delivery were asked to return for reevaluation six months post partum.

Anal Endosonography

Endosonography was performed with an ultrasound scanner with a rotating rectal probe, a 7-MHz transducer (focal range, 2 to 4.5 cm), and a hard sonolucent plastic cone (Bruel and Kjaer, Naerum, Denmark),[12] while the woman lay on her left side. Serial images of the upper, middle, and lower anal canal were recorded (Umatic video recorder, Sony, Tokyo, Japan). The endosonographic interpretation of the appearance of muscle layers has been previously validated[13]: an external-sphincter defect appears as a break in the normal texture of this muscle ring[17,18] (Fig. 1), and an internal-sphincter defect as a gap in the hypoechoic ring[18,19] (Fig. 2). All investigations were performed and the results interpreted by one operator; the results were reviewed independently by a second observer unaware of the first interpretation.

Anal Manometry

Anal manometry was performed with an intracompartmental-pressure monitor (Stryker, Kalamazoo, Mich.) attached to an air-filled microballoon.[20] The maximal resting pressure and the maximal squeeze pressure (i.e., the maximal increase above the resting pressure) were measured according to a stationary pull-through technique.[21]

Measurement of Terminal Motor Latency of the Pudendal Nerves

The terminal motor latency of each pudendal nerve was measured after an electronic stimulus (Medelec stimulator, Old Woking, United Kingdom) from an electrode (Dantec, Skovlunde, Denmark) mounted on a gloved index finger.[22,23] Latency is the time between stimulation of the pudendal nerve at the level of the ischial spine and contraction of the anal sphincter. Prolongation of latency is indicative of damage to the fastest conducting nerve fibers.[24]

Perineometry

The perineal plane (the level of the perineal soft tissues at the point of the anal verge, relative to the bony ischial tuberosities) was measured with a perineometer at rest and during maximal straining effort in the left lateral position.[25] The difference between the two measurements indicates the degree of perineal descent. Descent of the perineum below the level of the ischial tuberosities at rest or on straining was considered abnormal.[25]

Statistical Analysis

Antepartum measurements were compared with postpartum measurements by paired t-tests. Associations between categorical variables were assessed with the chi-square test or Fisher's exact test. Continuous variables in independent groups were compared by two-tailed t-tests. A P value of less than 0.05 was considered to indicate statistical significance. All results are reported as means ±SD.

All variables shown to be significantly associated with sphincter defects on univariate analysis were then entered in a multiple logistic-regression analysis in a stepwise fashion to determine the combination of variables that best predicted sphincter damage. An odds ratio was also calculated.

The statistical analyses were performed with software from Minitab Data Analysis (Minitab, State College, Pa.) and SAS (SAS Institute, Cary, N.C.).

Results

One hundred fifty women (of whom 100 were primiparous and 50 multiparous) returned for postpartum examination a median of 49 days (range, 35 to 105) after delivery. Twenty-three women (21 primiparous and 2 multiparous) had delivered by cesarean section, and 127 vaginally (73 women were white, 71 black, and 6 of other races).

Vaginal Delivery

Of the 127 women who had a vaginal delivery, 79 were considered primiparous (including 2 women who had had previous children, but by cesarean section) and 48 multiparous, of whom 38 (79 percent) had only one previous vaginal delivery. Two primiparous women (3 percent) and 10 multiparous women (21 percent) had symptomatic urinary stress incontinence on examination at six weeks.

Bowel Symptoms

None of the 79 primiparous women had diabetes mellitus or neurologic or anorectal disease. Post partum, 10 women (13 percent) reported having one or more new bowel symptoms; 8 (10 percent) had fecal urgency, and 4 (5 percent) had anal incontinence (3 had flatus, and 1 had flatus and liquid stool). Two women had temporary incontinence of flatus for less than three weeks post partum. Among the 48 multiparous women, 9 (19 percent) had one or more bowel symptoms before delivery. These symptoms began after a previous vaginal delivery (seven women had fecal urgency, three were incontinent of flatus, and five were incontinent of liquid stool). After delivery, three women (6 percent) had new symptoms (two had urgency and incontinence of flatus, and one had urgency alone). One woman with incontinence of liquid stool before delivery had no symptoms afterward. Therefore, after delivery, 11 (23 percent) of the multiparous women had bowel symptoms.

Altogether, of the 127 women who delivered vagi-

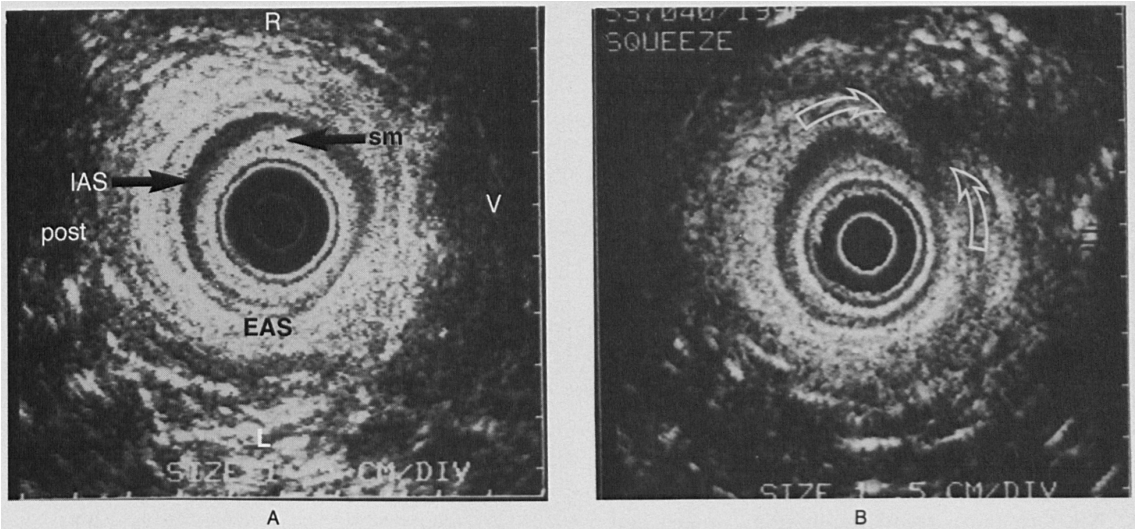

Figure 1. Results of Anal Endosonography before and after Delivery in a Primiparous Woman with a Postpartum Defect of the External Anal Sphincter.

Panel A shows a normal image of the middle portion of the anal canal at 34 weeks of pregnancy. R denotes right, L left, IAS internal anal sphincter (hypoechoic), EAS external anal sphincter (hyperechoic), post posterior, sm submucosa (hyperechoic), and V vagina. The probe lies medial to the submucosa. Panel B shows the canal six weeks after delivery. The woman had incontinence of flatus after a forceps delivery with an episiotomy. A hypoechoic defect of the external anal sphincter is present between the open arrows. The damage to the anal sphincter was not recognized during repair of the episiotomy.

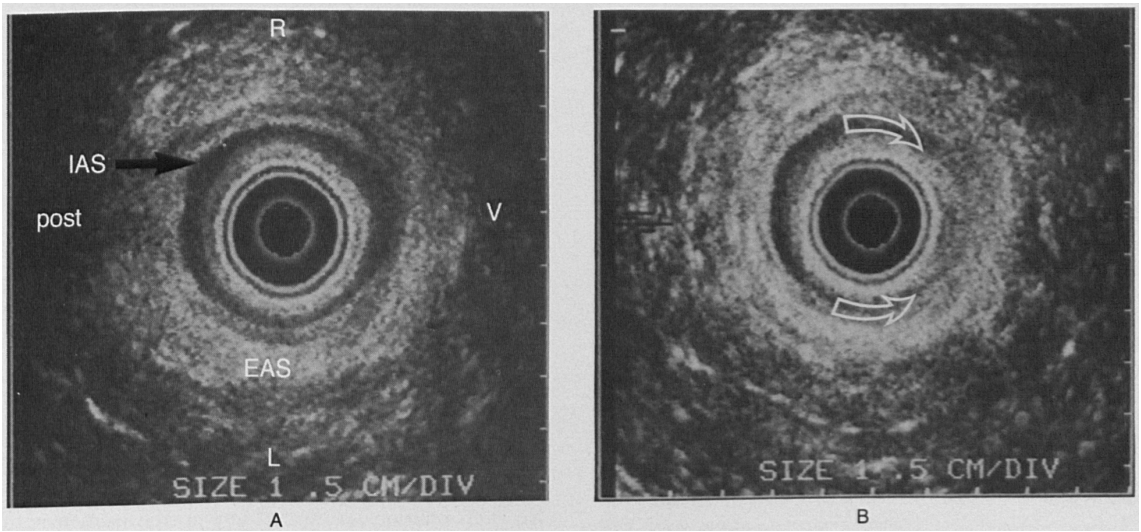

Figure 2. Results of Anal Endosonography before and after Delivery in a Primiparous Woman with a Postpartum Defect of the Internal Anal Sphincter.

Panel A shows the middle portion of the anal canal at 34 weeks of pregnancy. R denotes right, L left, IAS internal anal sphincter, EAS external anal sphincter, post posterior, and V vagina. Panel B shows the canal six weeks after delivery. The woman had no symptoms after a spontaneous vaginal delivery but had a second-degree tear (not involving the anal sphincter). The hyperechoic defect in the internal anal sphincter is present between the open arrows.

nally, 13 (10 percent) had one or both bowel symptoms (urgency and incontinence) after delivery.

Anal Endosonography

No sphincter defect was detected before delivery in any primiparous woman. Six weeks after delivery, 28 women (35 percent) had defects of either the internal sphincter (Fig. 2B), the external sphincter (Fig. 1B), or both (Table 1). Twenty-three women (29 percent) had a defect of the internal sphincter; the defect involved the entire length of the internal sphincter in 14 women and the distal portion in 9. Among the 15 women (19 percent) with a defect of the external sphincter, the defect involved the full length of the sphincter in 11 women, the proximal portion in 2, and the distal portion in 2. Nine women had only partial thickness defects, and six had complete defects. Ten of the 15 women with an external-sphincter defect also had an internal-sphincter defect.

Nineteen (40 percent) multiparous women had a sphincter defect before delivery, and 21 (44 percent) after delivery (Table 1). Two women had a new defect, and two with an internal-sphincter defect before delivery had a new, external-sphincter defect after delivery. All three multiparous women who reported new bowel symptoms after delivery had combined sphincter defects, which were new in two of the women. All these defects occurred in the anterior portion of the sphincter.

Manometry

The maximal resting anal pressure fell significantly after delivery in both the primiparous and the multiparous women (Table 2). It was also significantly lower in women with an internal-sphincter defect than in those without such a defect (Table 3). The decrement in resting pressure (the difference between the value after delivery and the value before delivery) was significantly greater in women with an internal-sphincter defect than in those without (Table 3). The relation between resting pressure and external-sphincter defects was not significant (Table 3).

The squeeze pressure fell significantly after delivery in both the primiparous and the multiparous women (Table 2). It was also lower in women with an external-sphincter defect than in those without such a defect (Table 3). The decrement in squeeze pressure after delivery was greater in the women with an external-sphincter defect than in those without (Table 3). There was no relation between the squeeze pressure and internal-sphincter defects (Table 3).

The 23 women in whom an internal-sphincter defect developed had a significantly shorter anal canal before delivery than the 56 women in whom such a defect did not develop (36±5 vs. 39±5 mm, P = 0.01). No such relation was found among the women with external-sphincter defects.

Table 1. Incidence of Anal-Sphincter Defects in Women Evaluated by Anal Endosonography after Vaginal Delivery, According to Parity.

Parity Group	Anal-Sphincter Defects			
	Internal Sphincter	External Sphincter	Internal and External	Total
	no. with defect (%)			
Primiparous women (n = 79)				
Before delivery	0	0	0	0
After delivery	13 (16)	5 (6)	10 (13)	28 (35)
Multiparous women (n = 48)				
Before delivery	8 (17)	2 (4)	9 (19)	19 (40)
After delivery	7 (15)	2 (4)	12 (25)	21 (44)

Table 2. Anal Pressure, Perineal Descent, and Pudendal-Nerve Terminal Motor Latency before and Six Weeks after Childbirth, According to Type of Delivery.*

Type of Delivery†	Antepartum Value	Postpartum Value	P Value
Vaginal delivery			
Primiparous women (n = 79)			
Anal pressure (mm Hg)			
Resting	61±10	57±12	<0.001
Squeeze	88±41	64±36	<0.001
Perineal descent (mm)	11±6	14±7	<0.001
Pudendal-nerve latency (msec)			
Right nerve (n = 63)	1.9±0.2	2.0±0.2	<0.001
Left nerve (n = 64)	2.0±0.2	2.1±0.2	<0.001
Multiparous women (n = 48)			
Anal pressure (mm Hg)			
Resting	57±12	53±14	0.004
Squeeze	60±31	52±22	0.006
Perineal descent (mm)	14±6	17±7	<0.001
Pudendal-nerve latency (msec)			
Right nerve (n = 33)	1.9±0.2	2.0±0.2	0.002
Left nerve (n = 33)	2.0±0.2	2.1±0.2	0.009
Cesarean section (n = 23)			
All women			
Anal pressure (mm Hg)			
Resting	62±12	61±13	0.29
Squeeze	72±32	71±32	0.69
Women with elective procedure (n = 7)			
Perineal descent (mm)	9±2	10±2	0.19
Pudendal-nerve latency (msec)			
Right nerve	2.0±0.2	2.0±0.2	1.00
Left nerve	1.9±0.2	2.0±0.2	0.08
Women with indicated procedure (n = 9)‡			
Perineal descent (mm)	11±6	12±5	0.39
Pudendal-nerve latency (msec)			
Right nerve	1.9±0.1	2.0±0.2	0.12
Left nerve	1.9±0.1	2.1±0.3	0.01

*Plus–minus values are means ±SD.

†Resting denotes maximal resting anal pressure; squeeze, maximal increase above resting pressure; and perineal descent, the difference between the plane of the perineum at rest and its plane during a straining effort.

‡An indicated procedure was performed after the onset of labor.

Pudendal-Nerve Terminal Motor Latency

The terminal motor latency of each pudendal nerve was measured in 63 primiparous and 33 multiparous women. Latency was significantly increased in both nerves in women from both groups who underwent vaginal delivery (Table 2). The values were higher

Table 3. Anal Pressure in Relation to the Presence or Absence of Sphincter Defects in 79 Women Six Weeks after Vaginal Delivery.*

TYPE OF PRESSURE†	DEFECT	NO DEFECT	P VALUE
	anal pressure (mm Hg)		
Internal sphincter	(n = 23)	(n = 56)	
Resting (post partum)	48±10	61±11	<0.001
Change	−9±9	−2±11	0.01
Squeeze (post partum)	61±32	66±37	0.49
Change	−29±36	−22±25	0.37
External sphincter	(n = 15)	(n = 64)	
Resting (post partum)	52±13	59±12	0.07
Change	−8±8	−3±11	0.15
Squeeze (post partum)	44±13	70±38	<0.001
Change	−47±27	−18±26	<0.001

*Plus–minus values are means ±SD.

†Resting denotes maximal resting pressure; squeeze, maximal squeeze pressure; and change, the difference between postpartum and antepartum values.

than the upper limit of the antepartum normal range in 10 primiparous women (16 percent) and 5 multiparous women (15 percent) six weeks post partum; only 1 multiparous woman (3 percent) had abnormal values before delivery.

There was no relation between the change in the pudendal-nerve terminal motor latency and the development of symptoms or the results of anal manometry. However, there was a significant association (P = 0.02) between abnormal latency and the development of a sphincter defect in primiparous women.

Perineometry

Perineal descent increased significantly after vaginal delivery in both primiparous and multiparous women (Table 2). The 36 women with abnormal perineal descent on straining after delivery had a significantly longer mean pudendal-nerve motor latency post partum than the 35 with normal descent (2.1±0.2 vs. 2.0±0.2 msec, P = 0.01).

The duration of active pushing during the second stage of labor correlated significantly with the plane of the perineum at rest (P = 0.04, r = 0.23) and during straining (P = 0.008, r = 0.30): the women with a longer active second stage of labor had a greater descent.

Sphincter Defects in Relation to Obstetric Variables

A defect involving at least one of the sphincter muscles occurred in 8 of the 10 women (80 percent) who had forceps deliveries (9 with outlet [low] deliveries and 1 with a rotational delivery), but in none of the 5 who had vacuum-extractor deliveries (Table 4). All instrumental deliveries were carried out when the infant presented below the ischial spines.

The deliveries of two women were complicated by shoulder dystocia; one of these women had a forceps delivery and was later found to have an external-sphincter defect. In addition, one woman underwent a twin delivery (no defects), another a breech delivery (internal-sphincter and external-sphincter defects), and a third an occipitoposterior delivery (external-sphincter defect).

Internal-sphincter defects developed in three women although their perineum was intact after delivery. External-sphincter defects were detected only in women who underwent episiotomy or sustained a spontaneous perineal tear.

Univariate analysis showed that internal-sphincter defects were significantly associated with forceps delivery (P = 0.004), epidural analgesia (P = 0.005), and the presence of an episiotomy (P = 0.04). Stepwise logistic-regression analysis revealed that forceps delivery was associated with a significant risk of an internal-sphincter defect (odds ratio, 7.0). When this factor was controlled for, epidural analgesia was not found to contribute to the development of an internal-sphincter defect.

External-sphincter defects were associated with augmentation of labor (P = 0.03), epidural analgesia (P = 0.03), posterolateral episiotomy (P = 0.02), and forceps delivery (P = 0.001) on univariate analysis. On stepwise logistic-regression analysis, the single independent factor associated with the development of an external-sphincter defect was forceps delivery (odds ratio, 11.1).

The infant's weight, the infant's head circumference, induction of labor, the length of each stage of labor, spontaneous perineal tears, the mother's age, and race were not significantly related to the development of sphincter defects.

Sphincter Defects in Relation to Bowel Symptoms

All women except one (a primiparous woman) who had either fecal urgency or anal incontinence after delivery had sphincter defects. There was a strong

Table 4. Obstetrical Variables in Relation to the Development of Anal-Sphincter Defects in 79 Women with Vaginal Deliveries.

VARIABLE — NO. OF WOMEN (%)*	NO. OF WOMEN WITH SPHINCTER DEFECTS			
	INTERNAL SPHINCTER	EXTERNAL SPHINCTER	INTERNAL AND EXTERNAL	TOTAL
Noninstrumental delivery — 64				
Induction — 6 (9)	2	0	0	2
Augmentation — 20 (31)	4	2	2	8
Epidural analgesia — 16 (25)	5	1	2	8
Episiotomy — 22 (34)†	4	2	3	9
Second-degree tears — 24 (38)	4	2	0	6
Third-degree tears — 2 (3)	0	0	2	2
No laceration — 17 (27)	3	0	0	3
Instrumental delivery — 15				
Forceps — 10 (67)	2	1	5	8
Vacuum extractor — 5 (33)	0	0	0	0
Induction — 8 (53)	0	1	4	5
Augmentation — 8 (53)	1	1	4	6
Epidural analgesia — 10 (67)	1	1	5	7
Episiotomy — 14 (93)†	2	1	5	8

*Some women in each group (noninstrumental delivery and instrumental delivery) underwent more than one procedure.

†All episiotomies were posterolateral.

association ($P<0.001$) between the development of either symptom and sphincter defects (Table 5).

Cesarean Section

No woman who underwent cesarean section had any bowel symptom after delivery or any significant change in anal pressure (Table 2). Pudendal-nerve terminal motor latency was measured in 16 of these women. None of the seven in whom this procedure was elective had a significant change in latency; however, the nine women in whom the procedure was indicated after labor had begun had a significant increase in the latency of the left pudendal nerve (Table 2).

There was no significant change in perineal descent in 16 of these women (Table 2). However, the perineal plane on straining was significantly lower after delivery in the nine women who underwent a cesarean section after the onset of labor ($P = 0.05$).

Follow-up Evaluation at Six Months

Thirty-two women returned a mean of six months after delivery for a third evaluation. This group included 10 women with fecal urgency and 7 with anal incontinence six weeks post partum. Fecal urgency was no longer a problem in 4 of the 10 women with this symptom, but it developed in 2 others. Two of the seven women with anal incontinence had improvement, and another woman (with no sphincter defect) had had no further episodes of incontinence.

Repeat anal endosonography performed at six months in women with sphincter defects six weeks after delivery showed no change in the defects. Anal manometry did not reveal any significant change from the values recorded six weeks post partum. Measurement of pudendal-nerve terminal motor latency was repeated in 22 women and showed a significant decrease in latency in both the right ($P = 0.002$) and left ($P = 0.04$) pudendal nerves. Latency had returned to normal in 8 of the 12 women who had abnormal values six weeks after delivery.

Discussion

We found that vaginal delivery is frequently associated with mechanical disruption of the anal sphincters. Three percent of the primiparous women studied, but none of the multiparous women, sustained an injury to the anal sphincters during delivery that was apparent on clinical examination — i.e., a third-degree or fourth-degree tear. Endosonography, however, revealed sphincter damage in 35 percent and 44 percent, respectively. The incidence of sphincter defects among the primiparous women six weeks after delivery was comparable to that among the multiparous women before delivery, most of whom had had only one previous vaginal delivery. There was only a slight increase in the incidence among the multiparous group post partum (4 percent), suggesting that the risk of sphincter damage is greatest during the first vaginal delivery.

Table 5. Symptoms of Fecal Urgency or Anal Incontinence in Relation to the Presence of Any Anal-Sphincter Defect after Vaginal Delivery in 127 Women.

Symptom	Defect (N = 49)	No Defect (N = 78)	P Value*
	no. of women		
Fecal urgency			
Yes (n = 18)	18	0	<0.001
No (n = 109)	31	78	
Anal incontinence			
Yes (n = 11)	10	1	<0.001
No (n = 116)	39	77	

*By Fisher's exact test. Both symptoms were strongly associated with the presence of a defect.

External-sphincter damage occurred only in the presence of a tear or episiotomy, suggesting that it occurs as part of a direct continuation of perineal disruption. As others have reported,[1] a posterolateral episiotomy did not appear to protect the patient against the development of sphincter defects (Table 4). Midline episiotomies are associated with a higher incidence of third-degree or fourth-degree tears than posterolateral episiotomies.[1,26] There is therefore no reason to suspect that our findings would have been any different in women who underwent a midline episiotomy.

The internal sphincter was injured more frequently than the external sphincter and was sometimes damaged when the perineum remained intact. Shearing forces produced by the descent of the infant's head may cause isolated damage to the internal sphincter, a mechanism different from that causing injury to the external sphincter. Women with a shorter anal canal may be more prone to this form of trauma, since internal-sphincter disruption was positively associated with a shorter canal before delivery.

Endosonographic examination suggested that the structural damage to the sphincters was permanent, since the defects were present at six months, and the incidence of defects among the primiparous women after delivery was similar to that among the multiparous women before delivery.

There was a definite relation between the presence of sphincter defects, anal pressure (Table 3), and bowel symptoms (Table 5). These ultrasonographically identified sphincter defects therefore appear to have physiologic and clinical importance. The single best predictive test of clinical dysfunction was anal endosonography. Although anal pressures were reduced in women with sphincter defects, there was considerable overlap of values between the women with symptoms and those without them.

Only about one third of the women with sphincter defects had bowel symptoms. Women with defects who do not have symptoms may have sufficient residual sphincter function to maintain continence. Long-term follow-up of such women will be necessary to determine whether they are at greater risk for in-

continence later in life. Since the peak incidence of fecal incontinence among women occurs in the fifth and sixth decades,[27] the cumulative effect of subsequent deliveries, the effects of aging,[27,28] the menopause,[27,28] and the progression of a neuropathy[6] may all contribute to sphincter weakness in the long term. Women with an occult sphincter defect may be at greater risk.

None of the women with disturbances of bowel function had spontaneously reported their symptoms or sought medical attention. Underreporting of such symptoms is well known[29] and may explain why these problems are not widely recognized in obstetrical practice.

Sphincter defects developed in 8 of the 10 primiparous women who had a forceps delivery but in none of the 5 who had a vacuum-extractor delivery. These findings are consistent with those of other reports indicating that vacuum extraction is associated with less trauma to the perineum than forceps delivery.[30,31]

In two previous studies,[5,16] pudendal-nerve terminal motor latency in women who delivered vaginally was not increased two months post partum, as compared with latency in a control group of women[5]; latency had not been measured before delivery. When we compared postpartum values directly with antepartum values, we found that vaginal delivery, particularly a first vaginal delivery, resulted in a significant increase in pudendal-nerve terminal motor latency and in perineal descent six weeks after delivery. There was no association between bowel symptoms and prolonged pudendal-nerve terminal motor latency or abnormal perineal descent, which is not the case with idiopathic fecal incontinence[32] that develops in middle age and is believed to be related to neuropathy.[8] The association of a prolonged pudendal-nerve terminal motor latency with a sphincter defect probably reflects a traumatic cause common to these two factors, rather than a causal relation between them.

In conclusion, vaginal delivery causes bowel symptoms, mechanical trauma to the anal sphincter, and injury to the pudendal nerve in many women. The use of the obstetrical forceps is particularly associated with a higher risk of sphincter damage.

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