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ABSTRACT

Disruption of the anal sphincters or injury to the sphincter innervation during childbirth is an important cause of anorectal incontinence among female soldiers. This study sought to determine the incidence of damage to the anal sphincter and the relation of injury and method of delivery to anorectal symptoms and function.

Ninety active duty soldiers were studied at 24 weeks gestation and 12 weeks after delivery by comparing transanal ultrasonography, anal manometry, and pudendal nerve terminal motor latencies. Although only three percent of the subjects sustained clinically apparent injury to the anal sphincters during vaginal delivery 41% of the subjects demonstrated anal sphincter damage on their post-partum transanal ultrasonography. Twenty-one percent of the soldiers had anorectal symptoms after delivery and fifteen percent had at least occasional incontinence of flatus or liquid stool at 12 weeks post-partum. The incidence and severity of anorectal symptoms appear to diminish within a six-month time frame, but the structural damage to the anal sphincters appears to remain. The long-term effects of anal sphincter damage during childbirth remain to be determined.

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INTRODUCTION

Pelvic floor trauma associated with vaginal delivery is an important cause of both immediate and long-term pelvic floor dysfunction in a significant number of women.[1] Permanent anal incontinence is reported to complicate 4-9% of vaginal deliveries and has been ascribed to both mechanical and neurologic trauma during the process.[2]

Overt anal sphincter damage due to third or fourth-degree tears occurs in approximately 0.7% of women undergoing vaginal deliveries in centers where mediolateral episiotomy is practiced[3] and 6-8% of deliveries in regions where midline episiotomies are the preferred method.[4] Inadequate primary repair of these sphincter injuries can lead to a variety of pelvic floor dysfunctions to include fecal incontinence.[5] Investigations utilizing anal endosonography have demonstrated that the internal anal sphincter can sustain damage as well, leading to post-partum fecal urgency and incontinence.[6] Burnett studied 62 women with fecal incontinence related to obstetrical procedures by anal endosonography and found external sphincter defects in 90% as well as internal sphincter defects in 65%.[7]

Impairment of pudendal nerve conduction at delivery has been attributed to progressive denervation of the anal sphincter muscles and the later development of fecal incontinence.[8] Kafka found pudendal neuropathy to be the only parameter differentiating female subjects with solid stool incontinence from those with leakage.[9]

Accurate imaging of both sphincter muscles by anal endosonography demonstrated unsuspected defects of the external anal sphincter in 36% of women thought to have purely

neurologic fecal incontinence.[10] Sultan[6] in a prospective study of women before and after delivery, using anal endosonography and anorectal neurophysiologic tests found significant changes in pudendal nerve terminal motor latency and anal sphincter defects in 35% of primiparous and 40% of multiparous women who delivered vaginally.

The exact mechanism of post-partum fecal incontinence is doubtless complex and likely depends upon multiple factors to include a woman's anatomical, muscular, cellular, and neurologic composition as well as the stresses to which the pelvic floor is exposed.

Sampsille[11] studied the effect of pelvic floor exercises on urinary incontinence and pelvic muscle strength in primigravidas during pregnancy and post-partum, and concluded that those with stronger pelvic floor muscles tended to rehabilitate from the stresses of vaginal birth sooner. Taskin[12] found that primiparous subjects who practiced ante-partum Kegel exercises and were given prophylactic episiotomies were the only group delivering vaginally who demonstrated no major post-partum pelvic floor defects. These studies suggest that the strength of a woman's pelvic floor musculature may be a factor in determining pelvic floor damage and anal incontinence during childbirth.

In a previous study, we had determined that nine percent of female soldiers complained of fecal incontinence to the extent that it was socially embarrassing, or interfered with the performance of their duty. Since no published studies have addressed the incidence of post-partum fecal incontinence among young women in the peak of physical condition, we undertook a prospective study of female soldiers before and after delivery, using anal endosonography and anorectal neurophysiologic tests to establish the incidence of mechanical and neurologic trauma to the posterior pelvic floor during childbirth in this population.

BODY

Methods

We studied 101 female soldiers from Fitzsimons Army Medical Center, Aurora, Colorado and Madigan Army Medical Center, Fort Lewis, Washington. Subjects were recruited by means of a poster and lectures given at prenatal classes. The study was approved by the Institutional Review Board and the Human Use Committee at Fitzsimons and Madigan Army Medical Centers. All subjects gave informed consent. Subjects who related a history of incompetent cervix, previous premature deliveries, premature labor, multiple gestation during the present pregnancy, or major medical problems such as insulin dependent diabetes were excluded from participation.

All subjects completed a questionnaire at the initial visit and at the 12 week post-partum visit which reviewed pertinent anorectal history. This questionnaire included an incontinence scoring system based on a simple tabular form completed by the patient. It listed and rated the frequency of any bowel symptoms which the subjects were experiencing. These symptoms included fecal urgency and/or incontinence of gas, liquid or solid stool. They were asked to indicate if the frequency of the symptom was less than once a month, more than once a month but less than once a week, or more than once a week. In addition the subjects were asked to indicate if the symptoms significantly altered their lifestyle or interfered with job performance.

Transanal Ultrasonography (TAUS)

Endosonography was performed at approximately 24 weeks gestation and 12 weeks post-partum using a B&K Medical Systems Inc. (Marlborough, MA) 3535 scanner with a 10 mHz

transducer and a sonolucent plastic cap over the transducer. Data was recorded from the proximal, middle and lower anal canal, and stored on a 3.5 inch diskette. Both real time and static images were obtained. Static images were obtained at .5cm intervals from the anal verge. All subjects were scanned in the dorsal lithotomy position to reduce pelvic organ displacement caused by gravity in the left lateral decubitus position.

Muscle injury was determined using the criteria outlined by Stentovich,[13] in that the continuity of either or both anal sphincters had to be interrupted by a hyperechoic, hypoechoic or mixed echogenic defect in the muscle ring. The defect had to be present on more than one .5cm level of the canal so that an artifact seen on a single level would not be misinterpreted as an injury. Sphincter thickness was measured anteriorly in the proximal, middle, and distal anal canal. Investigations were performed and the results interpreted by multiple operators and the results were reviewed independently by a second observer unaware of the first interpretation.

Anal Manometry

Anal manometry was performed at approximately 24 weeks gestation and 12 weeks post-partum using BioLAB equipment with Analgraph software from Sandhill Scientific, Inc. (Littleton, CO). Standardized anorectal physiologic data were recorded for each patient during resting and squeezing to include pressure vector volume, vector symmetry index, maximal pressure, maximal average pressure, standardized pressure, and sphincter length.

Measurements were obtained with an eight-channel flexible catheter, 4.8mm in diameter with a spiral and radial configuration of the eight ports beginning 3cm from the distal tip. The resting and squeeze pressure profiles were obtained at 1/2cm levels beginning 7cm above the anal verge.

Pudendal Nerve Terminal Motor Latency

Pudendal nerve terminal motor latency was obtained with a Dantec Medical Inc. (Cambell, CA) Cantata™ EMG/Nerve Conduction System. Bilateral pudendal nerve latency determinations were obtained with Dantec St. Mark's Pudendal Nerve Electrodes. Comparisons were made to normal pudendal nerve latency (2.0 +/- 0.2 milliseconds) and the subjects ante-partum latency. The pudendal nerve terminal motor latency was defined as the time between stimulation of the pudendal nerve at the level of the ischial spine and contraction of the anal sphincter.

Delivery and Post-Partum Evaluation

All women were examined at approximately 24 weeks gestation and 12 weeks post-partum. At each assessment, the subject completed an identical questionnaire which reviewed her anorectal history and included an incontinence scoring system. Anal endosonography, anal manometry, and pudendal nerve terminal motor latency were performed. Information on the course of labor and delivery was obtained from the patient and the hospital record.

Delivery was managed as deemed appropriate by the attending physician. All episiotomies were midline. Uncomplicated episiotomies were repaired by the house officers, and the more complicated episiotomies were repaired under the supervision of senior staff. Tears or incisions related to delivery were classified as follows: first degree tears involved only the vaginal epithelium; second degree tears involved the perineal body but not the external anal sphincter; third degree tears involved the external anal sphincter; and fourth degree tears involved both the external sphincter and the anal epithelium. All subjects were asked to return for re-evaluation six months post-partum.

Statistical Analysis

Ante-partum measurements were compared with post-partum measurements by ANOVA. Associations between categorical variables were assessed with Fisher's exact T-test. Continuous variables in independent groups were compared by two tailed T-tests. P-values of less than 0.05 were considered to indicate statistical significance. All results were compared by plus or minus the standard deviation. The statistical analysis was performed using software from the Department of Clinical Investigation, Madigan Army Medical Center, and confirmed by independent statisticians.

Results

One-hundred and three gravid soldiers entered the study and ninety returned for post-partum examination at a median of 86 days (range of 72-98) after delivery. Thirteen women did not complete the study mainly because of transfer or base re-alignment and closure during the course of the study. Of the ninety subjects who completed the study, on entry, forty-two were multiparous and forty-eight were primiparous.

The median age of the multiparous subjects was twenty-eight years with a range of 22-39. The median age of the primiparous subjects was twenty-four years with a range of 19-40. Of the forty-two multiparous subjects three (7%) had previously delivered by cesarean section. Thirty-nine of the subjects had delivered vaginally, with sixteen having more than one vaginal delivery.

Twenty-eight (72%) of those previously delivering vaginally had an episiotomy performed, eleven (28%) had forceps and three (7%) had a vacuum extraction at delivery. Seven

subjects (18%) reported having a vaginal laceration at delivery. The median weight of the last infant at delivery was 3,302 grams with a range of 2,508 - 3,803 grams.

Seventy-eight of the ninety women (86%) delivered vaginally during the course of the study. Twelve subjects (14%) required cesarean sections for delivery. Four multiparous subjects required cesarean section: one unsuccessful vaginal birth after cesarean section, one for fetal indications, and two for arrested labor. Eight of the primiparous subjects were delivered by cesarean section: two for fetal indications and six for arrested labor. No cesarean sections were electively scheduled.

Forty (83%) of the forty-eight primiparous subjects delivered vaginally. Thirty-one (78%) had an episiotomy performed and seven (18%) were reported to have sustained a vaginal laceration at delivery. Three lacerations were second degree and the remainder first degree. Two episiotomies (6%) extended into the anal mucosa (fourth degree) and the remainder were first or second degree episiotomies. Six primiparous subjects were delivered with forceps (five outlet and one with a rotational delivery). The mean weight of the infants at birth was 3,270 grams with a range of 2,721 - 4,082 grams.

Thirty-eight (90%) of the forty-two multiparous subjects delivered vaginally. Fifteen (39%) had an episiotomy performed during the delivery. One subject had a fourth degree episiotomy performed for shoulder dystocia and the remainder of the episiotomies were first or second degree. Vaginal lacerations were reported in four (12%) subjects and all were superficial lacerations. The mean weight of the infants delivered by the multiparous group was 3,281 with a range of 2,820-4,236 grams.

Bowel Symptoms

When surveyed at the initial examination at 24 weeks gestation no primiparous subject responded that they currently had problems with anal incontinence, although 16% indicated they were incontinent of flatus less than once a month. Two subjects indicated they occasionally (less than one episode per month) had incontinence of liquid stool, and no primiparous subject was incontinent of solid stool. No primiparous subjects indicated that the anal symptoms significantly altered her lifestyle.

Of the forty-two multiparous women, nine (21%) indicated that they had one or more bowel symptoms before the present pregnancy. They all dated the onset of their symptoms to a previous delivery. Five of the subjects stated that their symptoms significantly altered their lifestyle or ability to perform their duties. Six women (14%) had fecal urgency, five (12%) were incontinent of liquid stool, and one was incontinent of solid stool but less than once a month. Five subjects had multiple symptoms.

Seven (15%) of the forty-eight primiparous subjects reported experiencing one or more new bowel symptoms after delivery. Five (10%) had fecal urgency, four (8%) had anal incontinence, three had incontinence of flatus, and one had incontinence of flatus and liquid stool. Three women had temporary incontinence of flatus for up to five weeks post-partum.

After delivery four multiparous subjects (10%) had new anorectal symptoms. Two had incontinence of liquid stool and flatus as well as urgency. Two had incontinence of flatus only. After delivery thirteen multiparous subjects (31%) had bowel symptoms. When primiparous and multiparous were considered together 21% had anorectal symptoms at the end of the study.

Transanal Ultrasonography (TAUS)

Transanal ultrasonography did not initially reveal any sphincter defects in the forty-eight primiparous subjects. Twelve weeks after delivery transanal ultrasonography demonstrated either internal or external anal sphincter defects, or both, in 16 (33%) of the primiparous subjects. Nine (19%) of these subjects had a defect in the internal sphincter limited to the distal 1.5cm, but in seven subjects (14%) the defect appeared to involve the entire length of the internal sphincter.

Nine primiparous subjects (19%) had a defect in the external anal sphincter demonstrable by TAUS.

In four of these primiparous subjects, the defect appeared to involve the full length of the external anal sphincter. The distal portion only was involved in three subjects and the proximal portion of the external anal sphincter in two subjects. Five of the subjects had only partial thickness defects and four had defects completely through the external anal sphincter muscle. All nine subjects with external anal sphincter defects also had internal anal sphincter defects.

Eighteen (43%) of the forty-two multiparous subjects had a sphincter defect (internal, external, or both) before the delivery, and three more developed a sphincter defect after delivery (7%) for a total rate of 50% among the multiparous patients. All of these defects occurred in the anterior portion of the sphincter. All three who developed combined internal and external anal sphincter defects after delivery reported new onset of bowel symptoms (urgency or incontinence of flatus or liquid stool).

Anal Manometry

Resting Pressures

The maximal resting anal pressure fell significantly after delivery in both the primiparous and the multiparous groups (Table 1 and Table 2). The greatest significance was in those women who demonstrated internal anal sphincter defects during TAUS (Table 3). The resting anal pressures were also significantly lower in those with anal symptoms, particularly those with flatal incontinence.

Squeeze Pressures

The squeeze pressures in both the primiparous and multiparous subjects were also significantly lower after delivery. The difference was greater in the primiparous group (Table 1).

The squeeze pressure was lower in women with external sphincter defects than those who demonstrated no external sphincter defect on TAUS (Table 3). The decrement in squeeze pressure after delivery was greater in those women who demonstrated an external sphincter defect on TAUS. Resting pressure was not related to external sphincter defects and there was no significant relationship between changes in squeeze pressures and internal sphincter defects.

Both the resting and squeeze vector symmetry index was reduced significantly (-0.18) ($P = < .001$) in the primiparous subjects who delivered vaginally, but not in the multiparous subjects who delivered vaginally or those subjects who delivered by cesarean section. All nine of the primiparous subjects as well as the three multiparous subjects who developed both internal and external sphincter defects had a post-partum vector symmetry index of less than 0.75 (both resting and squeeze pressures). Seven of the nine primiparous subjects with external anal sphincter defects had a squeeze vector symmetry index of less than 0.75. Thirteen of the sixteen primiparous subjects with internal anal sphincter defects had a post-partum resting vector symmetry index of less than 0.75.

Pudendal Nerve Terminal Motor Latency

Bilateral pudendal nerve terminal motor latency was measured in each of the forty-two multiparous and forty-eight primiparous subjects before and after delivery (Table 4). Latency was significantly increased in both nerves in the primiparous and multiparous groups who underwent vaginal delivery, and in nine of the twelve (75%) who delivered by cesarean section (Table 3). Three multiparous subjects (7%) had abnormal pudendal nerve terminal motor latency before delivery.

There was no significant relationship between the change in pudendal nerve terminal motor latency and the development of symptoms or changes in manometric readings. There was however, a significantly longer post-partum pudendal nerve latency in those primiparous subjects who developed sphincter defects ($P = 0.03$). The one multiparous subject who complained of solid stool incontinence did demonstrate prolonged pudendal nerve terminal motor latency before and after delivery.

Sphincter Defects in Relationship to Obstetric Variables

The mother's age, race, length of each stage of labor, infant's weight, induction of labor or spontaneous superficial perineal tears were not significantly related to the development of either internal or external sphincter defects. No subject in this study had a second stage of labor in excess of two hours.

External sphincter defects were associated with augmentation of labor ($P = 0.02$), midline episiotomy ($P = 0.03$), spontaneous tears into the sphincter capsule ($P = 0.04$), and forceps delivery ($P = 0.001$). Forceps delivery appeared to be the factor most correlated with the development of both internal and external sphincter defects.

Internal anal sphincter defects were significantly associated with forceps delivery, midline episiotomy, and spontaneous tears extending into the external anal sphincter. Internal sphincter defects were found on TAUS in four women who delivered over an intact perineum. Two patients had an occiput posterior delivery - both demonstrated internal sphincter defects and one had external sphincter defects on post-partum TAUS examination. There were no breech deliveries or twin gestations in the groups studied.

Of the eight women delivered by forceps, a defect involving at least one sphincter was detected in six (75%) in the post-partum TAUS examinations. Forceps delivery was the one factor most correlated with the development of both external and internal sphincter defects.

Cesarean Section

Out of the twelve subjects who required a cesarean section, none demonstrated any significant change in sphincter integrity on TAUS, but maximal squeeze manometry pressures were significantly reduced on post-partum manometry ($P = .0015$).

None of the twelve cesarean sections were elective, and one was a failed vaginal birth after a cesarean section. The pudendal nerve terminal motor latency was significantly increased in nine of the twelve at the 12 week post-partum testing. The vector symmetry index did not change significantly with either resting or squeeze pressure after cesarean section (Table 5).

Six Month Follow-up Examination

Fifty-six of the ninety women returned six months after delivery for a third evaluation. Twenty-four were practicing Kegel exercises regularly (more than five times a week) and eighteen had received biofeedback training. All subjects had returned to full military duty. This

group included six with anal incontinence and eleven with fecal urgency at the 12 week post-partum examination.

Five of the patients with fecal urgency stated that it was either no longer a problem or occurred less than once a month. Two of the women with anal incontinence stated that it was no longer a problem (both had received biofeedback). No subject regardless of changes in symptomatology demonstrated any changes in the sphincter defects outlined at the 12 weeks post-partum examination. Anal manometry did not demonstrate any significant changes from the values recorded 12 weeks post-partum, even in the two subjects who had improvement with biofeedback. Pudendal nerve terminal motor latency showed a significant improvement (decrease) bilaterally in the patients ($P = 0.003$). Pudendal nerve latency had returned to normal in 56% of those who had abnormal values at the 12 weeks examination.

Discussion

This study demonstrates that vaginal delivery is frequently associated with mechanical disruption of the anal sphincters even in a young population of women in excellent physical condition.

Klein[14] studied risk factors for severe vaginal perineal trauma and determined that while the incidence of episiotomy or spontaneous tears was not influenced by exercise, a strong exercise profile was associated with fewer third and fourth degree tears in the presence of episiotomy. Exercise did not influence the rate of sulcus tears. Sampsille[11] found that the practice of pelvic

muscle exercise by primiparous resulted in fewer urinary incontinence symptoms in late pregnancy and post-partum.

Two (5%) of the primiparous women studied and one multiparous woman sustained an injury to the anal sphincters during vaginal delivery which was clinically apparent, i.e., a third or fourth degree laceration. Post-partum transanal sonography, however, revealed sphincter damage in 33% of the primiparas and new sphincter damage in 7% of the multiparas. The incidence of sphincter defects demonstrated by TAUS at 12 weeks post-partum among the primiparous women was comparable to the multiparous before delivery. The 7% increase in the occurrence of new sphincter defects noted at the multiparous subject post-partum TAUS suggests that the risk of sphincter damage is greatest during the first vaginal delivery.

The majority of external sphincter damage occurred in those subjects who had a tear or episiotomy at delivery. External sphincter damage occurred in two multiparous subjects who reportedly had no tears and no episiotomy. The internal sphincter was disrupted more frequently than the external sphincter and was frequently damaged when the perineum remained intact. Although the examination performed at six months post-partum suggests a gradual return of most neurological function in over half of the subjects, TAUS studies suggest that the structural damage is permanent.

Data obtained from anal manometric readings are often difficult to interpret clinically. A wide range of anal manometric pressure has been reported due to a variety of equipment, techniques, and the population studied. The resting anal sphincter pressures decrease in females throughout their lifetime, but do not decrease in males significantly until they reach 60 years of age. After age 60 there is a very steep decrease in resting pressures showing no significant difference in gender after 80 years of age.[15] Several studies have documented that males have stronger external anal sphincter than females.[8, 15] Females also have a lifelong steady decline in

anal squeeze pressures, starting at about 22 years of age, followed by a marked drop again at 66 years of age.[15]

The subjects with anal sphincter defects, particularly those with both internal and external sphincter defects, had reduced anal pressures, but there was considerable overlay in values between the women who developed anal symptoms and those who did not. Anorectal manometry is time consuming and occasionally uncomfortable to the patient.

Transanal sonography is rapidly and easily performed with minimal discomfort to the patient and appeared to be best correlated with the development of clinical anorectal symptoms. Still, less than half of the women with sphincter defects demonstrable on TAUS complained of any bowel symptoms during the six month follow-up period.

The long term clinical significance of asymptomatic defects in the internal and/or external anal sphincters observed at TAUS has yet to be determined. Snooks[2] reported a five year follow-up of patients who delivered vaginally and recorded manometric and neurophysiological evidence of pelvic floor weakness from partial denervation of the pelvic floor musculature at delivery. This weakness was more marked in those women with incontinence. He concluded that pudendal neuropathy due to vaginal delivery persists and may worsen with time.

Younger women with well developed levator ani and anal sphincters may have sufficient residual function to maintain anal continence, but those with sphincter defects may be at a greater risk to develop symptoms as they age. Lash[16] found that women lose 2% of functional pelvic floor musculature yearly, compared to 1% of other skeletal muscles. The effects of aging, menopause, and subsequent deliveries may not become apparent until a woman's fifth or sixth decade when anorectal incontinence reaches its peak. It is not known whether women with occult sphincter damage are at increased risk for subsequent rectal incontinence.

We found no association between bowel symptoms and prolonged pudendal nerve terminal motor latency. Kafka[9] found pudendal neuropathy to be the only parameter differentiating liquid stool leakage from incontinence of solid stool. He concluded that the presence of bilaterally normal pudendal nerves could partially compensate for muscular dysfunction.

Sultan[6] found that although vaginal delivery, particularly the first vaginal delivery, resulted in significant increases in pudendal nerve terminal motor latency post-partum, he did not find these increases in motor latency to be correlated with post-partum bowel symptoms. He concluded that 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 relationship.

None of the subjects who underwent cesarean section developed post-partum bowel complaints. However, nine of the twelve had prolonged pudendal nerve terminal motor latency post-partum. All of these subjects were in advanced labor (greater than 8cm dilated).

Fynes[17] studied anal sphincter injury after vaginal delivery and found, as did we, that although pudendal nerve terminal motor latency was prolonged and anal squeeze pressure increment reduced, the vector symmetry index was unchanged in women delivered by cesarean section late in labor. Fynes concluded that these findings indicated neurological injury to the anal sphincter mechanism, rather than mechanical trauma as occurs in vaginal delivery.

None of the women with bowel function disturbances had spontaneously reported their symptoms or sought medical attention. Bowel urgency or incontinence to any degree is particularly stressful to the female soldier. Incontinence poses challenges to the female soldier unparalleled by her civilian counterparts. Duty requirements often subject the soldier to marked

and sudden increases in intra-abdominal pressure making it more difficult to control incontinence. Field conditions often provide limited access to hygienic measures commonly utilized by the incontinent patient.

The psychosocial impact of fecal incontinence can be more devastating than the health consequences and has multiple and broad reaching effects that influence daily activities, social interactions and self-perceptions of health status.

In a recent study at Madigan Army Medical Center, nine percent of female soldiers reported that they commonly experienced bowel symptoms such as urgency or incontinence to the extent that it interfered with lifestyle or performance of duty. This study suggests that the incidence of these disorders is likely under reported. The question of how to prevent injury to the pelvic floor during first and subsequent vaginal deliveries is a difficult one - but deserves much more study.

CONCLUSIONS

Although only 3% of the subjects sustained clinically apparent injury to the anal sphincters during vaginal delivery 41% of the subjects demonstrated anal sphincter damage on post-partum transanal ultrasonography. Twenty-one percent of the soldiers complained of anorectal symptoms and fifteen percent had some degree of anorectal incontinence 12 weeks post-partum.

The incidence and severity of anorectal symptoms appear to diminish over the short term, but the structural damage to the anal sphincters appears to remain. The long term effects of anal sphincter damage during childbirth remain to be determined.

This study confirms that instrumental deliveries, particularly forceps deliveries, are an independent risk factor in the development of posterior pelvic floor defects and both structural damage as well as the resultant bowel symptomatology. However, this study also suggests that cesarean delivery in labor does not entirely prevent the pudendal nerve injury associated with labor.

It is our opinion that the use of transanal ultrasonography (TAUS) provides the clinician with more useful information concerning anal sphincter defects than either anal manometry or pudendal nerve studies. It also appears to be more cost effective and can be performed in a more timely manner with less discomfort to the patient.

Long term studies need to be instituted to determine the exact role of TAUS and other studies of pelvic floor functions with reference to pelvic floor damage during labor and delivery. Only through a better understanding of the factors leading to pelvic floor damage can we reduce the incidence of these injuries to the female soldier and all women who give birth.

TABLE 1

Anal Pressures Before and 12 Weeks after Vaginal Delivery

Primiparous Women N = 40

	Ante-Partum	**Post-Partum**	**P Value**
Resting Pressure	**cm H_2O**	**cm H_2O**	
Mean Max P	131	116	< .0001
Mean Max Av P	104	91	< .0001
Mean Std P	75	65	< .0001
Squeeze Pressure	**cm H_2O**	**cm H_2O**	
Mean Max P	305	191	< .0001
Mean Max Av P	233	154	< .0001
Mean Std P	180	115	< .0001
Manometric Sphincter Length	**Cm**	**cm**	
Mean Resting	2.9	2.5	< .0010
Mean Squeeze	3.6	3.5	.3932

TABLE 2

Anal Pressures Before and 12 Weeks After Vaginal Delivery

Multiparous Women N= 38

	Ante-Partum	**Post-Partum**	**P Value**
Resting Pressure	**Cm H_2O**	**cm H_2O**	
Mean Max P	113	106	.0367
Mean Max Av P	88	82	.0324
Mean Std P	60	55	.0685
Squeeze Pressure	**Cm H_2O**	**cm H_2O**	
Mean Max P	184	162	.0059
Mean Max Av P	146	127	.0144
Mean Std P	110	96	.0010
Manometric Sphincter Length	**cm**	**cm**	
Mean Resting	2.7	2.6	.3946
Mean Squeeze	3.39	3.40	.3234

TABLE 3

Ante-partum and Post-partum anal Pressure Changes and the Relationship to Anal Sphincter Defects in Primiparous Woman

External Anal Sphincter	**Defect on TAUS N = 9**	**No Defect on TAUS N = 39**	**P Value**
Mean Change in resting pressure	-10 ± 11 cm H_2O	-6 ± 8 cm H_2O	0.15
Mean Change in squeeze pressure	-56 ± 30 cm H_2O	-22 ± 28 cm H_2O	< 0.001
Internal Anal Sphincter	**Defect on TAUS N = 16**	**No Defect on TAUS N = 24**	**P Value**
Mean Change in resting pressure	-12 ± 14 cm H_2O	-4 ± 11 cm H_2O	< 0.001
Mean Change in squeeze pressure	-31 ± 41 cm H_2O	-24 ± 28 cm H_2O	0.39

TABLE 4

Pudendal Nerve Terminal Motor Latency Before and 12 Weeks Post-Partum Vaginal Delivery

	Ante-partum	**Post-partum**	**P Value**
Primiparous Women N = 34			
Right nerve	1.9 ± 0.2M/Sec	2.1 ± 0.2 M/Sec	< 0.001
Left nerve	2.0 ± 0.2 M/Sec	2.1 ± 2.35 M/Sec	< 0.001
Multiparous Women N = 38			
Right nerve	2.0 ± 0.2 M/Sec	2.1 ± 02 M/Sec	0.004
Left nerve	2.0 ± 0.2 M/Sec	2.2 ± 0.2 M/Sec	0.008
Cesarean Section Women N = 12			
Right nerve	1.9 ± 0.2 M/Sec	2.0 ± 0.2 M/Sec	0.14
Left nerve	1.9 ± 0.2 M/Sec	2.1 ± 0.3 M/Sec	0.015

TABLE 5

Cesarean Sections
N = 12

	Ante-partum Max P	**Post-partum Max P**	**P Value**
Mean Resting Pressure	126 cm H_2O	114 cm H_2O	.0281
Mean Squeeze Pressure	267 cm H_2O	185 cm H_2O	.0015
	Ante-partum Vector Symmetry Index	**Post-partum Vector Symmetry Index**	**P Value**
Mean Resting Pressure	.82	.82	.2906
Mean Squeeze Pressure	.79	.78	.2016

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