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Quantitative anal sphincter electromyography in primiparous women with anal incontinence

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Abstract

OBJECTIVE—The purpose of this study was to determine whether evidence of denervation/reinnervation of the external anal sphincter is associated with anal incontinence symptoms immediately after delivery.

STUDY DESIGN—After a first vaginal delivery, 42 women completed an anal incontinence questionnaire. They also underwent concentric needle electromyography of the external anal sphincter. For each subject, motor unit action potential and interference pattern parameters were determined.

RESULTS—For the motor unit action potential, no difference was observed between patients with and without anal incontinence symptoms (*t*-test). For the interference pattern, the amplitude/turn was greater in subjects with fecal urgency (318 ± 48 [SD] μ V) and fecal incontinence (332 ± 48 μ V), compared with those without fecal urgency (282 ± 38 μ V) and fecal incontinence (286 ± 41 μ V; *P* = .02, *t*-test).

CONCLUSION—In this group of postpartum women with mild anal incontinence symptoms, interference pattern analysis shows evidence of denervation and subsequent reinnervation.

Keywords

anal incontinence; anal sphincter; postpartum period; quantitative electromyography

Vaginal delivery (especially operative vaginal delivery/episiotomy) is implicated repeatedly in anal incontinence in young healthy women. In a recent statewide survey in Oregon, > 29% of women had symptoms of anal incontinence 6 months after childbirth.¹

Identified sphincter injury at the time of vaginal delivery occurs 3%–24% of the time, but most reports are < 5%.² Recently, the Pelvic Floor Disorders Network reported that primiparous women who sustained a sphincter laceration at vaginal delivery 6 months previously were nearly twice as likely to have anal incontinence as those who delivered vaginally without a tear. The Pelvic Floor Disorders Network also noted, however, that the prevalence of flatal incontinence was 17% in the vaginal delivery control group and 26% in the cesarean group.³ This discrepancy between anal incontinence symptoms immediately after delivery and the

identified sphincter tears (along with the observation that anal incontinence symptoms develop in women who have cesarean deliveries⁴ and the fact that some symptoms do not manifest for years after seemingly uncomplicated deliveries⁵) support the concept of pelvic floor nerve injury with childbirth as cause for altered continence.

Most reports on pelvic nerve injury have relied only on pudendal nerve conduction studies.^{6,7} Pudendal nerve terminal motor latency is an indirect measure of nerve conduction velocity along the terminal portion of the pudendal nerve. It is abnormal only with severe injury to large and heavily myelinated axons and may not detect subtle injury. Few researchers have used comprehensive investigations that include needle electromyography in asymptomatic or only mildly symptomatic women after delivery to detect more subtle injury.⁸ Because the pelvic muscles contract tonically⁹ as postural muscles, electromyography techniques must be modified, compared with the traditional techniques that are used for limb muscles. When testing the biceps muscle, a subject can relax the muscle completely or lift a known weight at a measurable rate. In contrast, it is very difficult for subjects to completely relax pelvic floor muscles or to develop varied states of measurable contraction. Obtaining and analyzing neurophysiologic data are challenging endeavors and require an expanded electromyographic approach.

A quantitative electromyographic technique (interference pattern analysis) is very promising because of its potential ease of use and lack of operator-induced bias.^{10,11} We aimed to determine whether evidence of denervation/reinnervation of the external anal sphincter detected by quantitative electromyography algorithms is associated with anal incontinence symptoms immediately after delivery.

Materials and Methods

Participants

Approval for this investigation was obtained from the Institutional Review Board at Oregon Health and Science University. Between July 2002 and August 2003, 42 women who were 12–40 weeks after delivery were recruited to study the neurophysiologic effects of vaginal delivery to the pelvic floor nerves and external anal sphincter.

These women were recruited originally to fill 2 separate postpartum cohorts. The first group was recently primiparous, had singleton vaginal deliveries within the previous 6 months, and was recruited by posted flyers in the clinics at Oregon Health and Science University. These women had not complained of anal incontinence to their providers and were designated as asymptomatic postpartum (AP) cases. They have been reported on previously in comparison to a nulliparous group.⁸

The other cohort came as a result of participation in Fecal Incontinence Postpartum Research Initiative.¹ Women who had given birth in Oregon between April and September of 2002 received by mail an anonymous survey to assess the prevalence of anal incontinence symptoms within the first 3 months of their delivery. From the 2569 women who admitted anal incontinence symptoms on the survey, we recruited a subset of vaginally primiparous women. These women had identified on a separate postcard that they were willing to be contacted for further investigations. With the use of a random number generator, 105 of these women who lived within the Portland metro area were chosen and contacted to enroll for our planned sample size. They were designated as having fecal incontinence after delivery.

The primary outcome variables were the quantitative electromyography motor unit action potential (MUAP) parameter duration and amplitude that was obtained as described later.

Using data regarding these parameters in the anal sphincter from previous reports,^{10,12} we required approximately 25 subjects in each group.

Questionnaires

Each woman completed a questionnaire that had been designed to detect episodes of difficult bowel movements (using Rome II criteria)¹³ or anal incontinence to either gas, mucus, liquid, or solid stool similar to the Fecal Incontinence Severity Index.¹⁴ As in the Childbirth and Pelvic Symptoms study, *incontinence* was defined as involuntary leakage at a frequency of ≥ 1 time per month.³ The presence of fecal urgency (defined as a “yes” response to the question) was also recorded separately.

Evaluations

As described previously,⁸ we performed standardized examinations that included a pelvic organ prolapse quantitative method (POP-Q),¹⁵ 3-dimensional endoanal ultrasound imaging,¹⁶ and a comprehensive pelvic floor neurophysiologic examination that included pudendal and perineal nerve terminal motor latency determinations, sacral reflexes (clitoral-anal, urethral-anal, and bladder-anal), and quantitative concentric needle electromyography of the external anal sphincter. Before needle electromyography, a small amount of topical anesthesia (lidocaine/prilocaine cream) was placed at the anal verge. We performed quantitative concentric needle electromyography using a Medtronic Keypoint electromyography machine (Medtronic Corporation, Minneapolis, MN) that was equipped with multiple MUAP (multi-MUAP) and interference pattern expert analysis software. Filter settings were 5 Hz to 10 kHz; the gain was 100 $\mu\text{V}/\text{div}$ to 2 mV/div and the sweep speed was 10 msec/div. Examinations were performed by 1 examiner (W.T.G.) who was masked to the responses that were provided on the questionnaire. Labor and delivery characteristics were culled from the medical record of the hospital at which the woman delivered. Each woman was compensated modestly for her participation.

Anal sphincter electromyography technique

Using the same technique as previously published reports,^{8,17} we used a 37-mm concentric needle to sample 4–5 unique motor unit territories on each side of the external anal sphincter. A finger was placed in the anus to gauge the direction of the anal canal, and the needle was advanced in a direction that was parallel to the anal canal (perpendicular to the longitudinal direction of the external anal sphincter muscle fibers) approximately 0.5 cm for each sample acquisition. The electromyography signal was recorded in a digital format on a Sony TCD-D7 digital audio tape recorder (Sony Corporation, Tokyo, Japan; frequency response: 2 Hz to 22 kHz) with the optical digital output interface of the Medtronic Key-point machine. The original uncompressed digital data were then played through the optical digital input (bypassing any digital-analog conversions) and analyzed with the software on the Keypoint system after the completion of the experimental protocol. The time stamp on the digital audio tape recorder was noted with each new site to ensure that the analysis, which was performed from the digital audio tape recording, acquired motor units from different areas in the muscle and prevented the sampling of previously analyzed motor units.

Multi-MUAP analysis

We encouraged the subjects to relax the pelvic floor as much as they were able. At baseline muscle contraction, the multi-MUAP algorithm on the electromyography machine automatically obtained the following parameters: amplitude (positive peak to negative peak), duration (initial, main spike, and terminal portions), area (under the entire delineated waveform), area/amplitude ratio (also known as “thickness”), phases (baseline crossings plus 1), and turns (shifts in waveform directions of a minimum voltage).^{18,19} Any MUAP with an

unstable baseline was discarded, because this precluded accurate analysis. No further user interaction was required for determination of these quantitative parameters.

Interference pattern analysis

We additionally asked subjects to increase voluntary contraction incrementally at each site to maximum to recruit more motor units, thereby creating an interference pattern. The interference pattern was sampled at baseline muscle activity and at the consciously increased muscle activity. We did not measure simultaneous force or pressure readings.

During each site sampling, 1–2 crisp-sounding 500-msec epochs (coinciding with each incremental change in muscle contraction efforts) were obtained and analyzed for the following electromyography parameters: number of turns/second (any peak or trough of the signal where amplitude changes by 100 microvolts), amplitude/turn (change in volts between 2 turns), number of short segments (parts of electromyography signal that have sharp activity characterized by very short rise times), percent activity (percent of time during the epoch that sharp activity occurs), and envelope (peak to trough amplitude exceeded only by 1% of amplitudes during epoch). Those rare data points that were recorded as a value of “zero” (ie, an epoch of little or no crisp electromyography activity) were discarded.

Data analysis and statistics

After unmasking the questionnaire responses, we noted that many of the women in the AP group also had anal incontinence symptoms. We elected to pool the postpartum women for the analysis and then compared the symptoms of anal incontinence to the electromyography parameters. After pooling was complete for each subject, MUAP and interference pattern parameter means were determined from the data points that were sampled from that subject. These MUAP and interference pattern parameters were checked for normal distribution; the Student *t* tests were used to compare these neurophysiologic parameters between those with and without anal incontinence symptoms. In addition, chi-square and Fisher’s exact tests were used to evaluate the association of delivery characteristics between those with and without anal incontinence symptoms. We used Pearson’s correlations to assess the potential bivariate associations between the neurophysiology parameters and POP-Q measurements.

Results

The mean age of the group was 26.7 ± 5.7 (SD) years; mean body mass index was 26.3 ± 6.3 kg/m²; and most of the women were white. Twenty-three women were from the AP cohort, and 19 women were from the fecal incontinence after delivery cohort. The mean gestational age at vaginal delivery was 39.5 ± 1.1 weeks; the length of stage 2 was 83.8 ± 59 minutes, and the mean birth weight was 3565 ± 438 g. At the time of the investigations, the women were 24.7 ± 2.0 weeks after delivery. Four women (9%) had operative vaginal deliveries; 9 women (21%) underwent a mid-line episiotomy, and 6 women (14%) had recognized sphincter tears at delivery that were repaired. Four of the 9 women who had an episiotomy sustained a recognized sphincter laceration. Overall, 3 women (7%) had anal sphincter lacerations that were detected by postpartum ultrasound examination, and all were symptomatic subjects from the fecal incontinence postpartum cohort. In each of the sphincter disruptions that were detected by ultrasound imaging, both the external and internal sphincter was involved. Two of the sphincter tears that were seen by ultrasound imaging were occult (ie, only 1 represented a persistent sphincter disruption). There was no statistically significant association of these delivery characteristics with fecal urgency or incontinence. POP-Q and pelvic floor muscle strength assessments given are in Table 1. There were no differences in POP-Q measurements or pelvic floor muscle strength between those women with or without fecal urgency or incontinence.

Questionnaires

Twenty-eight of the 42 women (66%) had flatal incontinence; 8 women (19%) had fecal incontinence, and 15 women (36%) had fecal urgency. Of the 23 women who were recruited for the AP group, 10 women (43%) had flatal incontinence, and 2 women (9%) had fecal incontinence.

Sacral reflexes

Sacral reflex results are also given in Table 1, and there were no differences between those women with or without fecal urgency or incontinence.

MUAP parameters (Table 2)

With MUAP parameters that were obtained by multi-MUAP techniques, no parameter showed any difference between those postpartum women with and without anal incontinence symptoms.

Interference pattern parameters (Table 2)

The parameter amplitude/turn was higher in subjects with fecal urgency, compared with those subjects without fecal urgency (318 ± 48 vs 282 ± 38 μ V, $P = .02$, t -test). For those women with incontinence to liquid stool, amplitude/turn was higher than in those women without incontinence to liquid stool (320 ± 47 vs 278 ± 35 μ V; $P = .02$, t -test). Amplitude/turn was also higher for those women with incontinence to solid stool than in those women without incontinence to solid stool (332 ± 27 vs 286 ± 41 μ V; $P = .003$, t -test). There was no difference in amplitude/turn in those women with flatal incontinence, compared with those women without. For all subgroups of anal incontinence, the other interference pattern parameters were not different.

Comment

This study shows that the anal sphincter of postpartum women who have fecal incontinence to liquid and solid stool and fecal urgency show evidence of denervation/reinnervation, when compared with asymptomatic women after a first vaginal delivery. These findings are congruent with our understanding of the pathophysiologic impact of nerve injury and repair. A *motor unit* is defined as a single motor nerve cell and all the muscle fibers that it serves. When partial nerve injury occurs, axons from adjacent motor units “sprout” and innervate those muscle fibers that have lost their nerve connection (denervated). The surviving motor unit becomes “larger” by incorporating more muscle fibers into the same nerve cell. Similar to an electrocardiogram, needle electromyography detects a waveform generated by single motor unit called a MUAP. After denervation and with reinnervation, the waveform representing the single motor unit becomes longer in duration, larger in amplitude, and more complex in appearance. These parameters can be quantified.¹⁹ Isolating single motor units in the external anal sphincter by electromyography is challenging. The muscle is always contracted (to maintain continence), and multiple motor units overlap, often creating an “interference pattern” with summation and cancellation of action potential components.

As the requirement for greater muscle contraction and force increases, more and larger motor units are recruited to contribute to the interference pattern (Henneman’s principle).²⁰ This leads to both an increase in the amplitude of the signal and the number of turns in the polarity per second. When nerve injury occurs, fewer motor units are recruited to contribute to the creation of force during increased muscle contraction. This creates a “reduced” interference pattern that can be seen and heard subjectively (Figure) and measured quantitatively with quantitative interference pattern algorithms such as described earlier.²¹ Because there may be fewer motor

units at any location, the voltage that is generated by each of those surviving motor units is greater. Additionally, the competition for the polarity of the electrical signal at any given time is decreased. This leads to fewer “turns” in polarity than expected for the amplitude of the signal measured.

Podnar et al¹² previously suggested that reinnervation changes may be more prominent in “high threshold motor units” (ie, those activated at the higher levels of muscle force generation requirements). Our findings are consistent with that suggestion. We were able to demonstrate differences between those women with and without anal incontinence symptoms using only the interference pattern analysis technique, which has an opportunity to analyze these higher threshold motor units that are activated to generate strong voluntary contraction beyond the baseline tone. In contrast, simultaneously performed and used multi-MUAP techniques did not show a similar statistical evidence of difference.

There are limitations from this project that should be addressed. The project was sized originally to detect differences in the MUAP parameters that can be detected at lower contraction force ranges. Our data revealed smaller differences in the MUAP parameters than anticipated, and we would have needed to examine 160 women to demonstrate statistically significant differences for both MUAP amplitude and MUAP duration. Although not statistically significant in our small number of women, this finding may also be related to the study group characteristics. Despite the fact that we originally designed this project to recruit both asymptomatic and incontinent women, we instead found that we had recruited women in each cohort with similar, generally mild incontinence. As a result, we did not collect enough data from 2 ends of the continence-incontinence spectrum.

An additional limitation is that we did not use a validated symptom index or quality of life tool. Nonetheless, for consistency and relevance, we did define incontinence similarly to the Pelvic Floor Disorders Network.³ Our tool was modeled from 2 commonly used instruments, the Wexner²² score and the Fecal Incontinence Severity Index.¹⁴ At the time these studies were performed, the Fecal Incontinence Severity Index was used less widely.

Finally, although this study shows aggregate evidence of more profound nerve injury in postpartum women who have anal incontinence symptoms, compared with postpartum women who do not, because of the small sample sizes involved and its cross-sectional design, the study does not define which obstetric practices or maternal or fetal characteristics lead to increased pelvic floor nerve injury. It also fails to answer whether simply carrying the pregnancy produces the same effect, because these patients all delivered vaginally and there were no patients who underwent a cesarean delivery.

Some authors have suggested that occult injury to the sphincter is noted in 35% of primiparous women. The effect that such an injury has in the future remains unknown. Similarly, occult pelvic floor nerve injury may subject a woman to pelvic floor disorders that include anal incontinence in the future. We demonstrated previously that a group of primiparous postpartum women had evidence of denervation/reinnervation, when compared with nulliparous women. Many of those women did have anal incontinence symptoms as we have defined here. An interference pattern analysis (as is discussed here) was not done for that report. This report, on the other hand, specifically separates those women without symptoms from those with symptoms. Although not tested, it is interesting to note that the MUAP parameters in the women without anal incontinence symptoms are of the same magnitude as the values that were shown to be greater statistically in that report. It still remains to be seen whether this phenomenon represents appropriate immediate neurophysiologic rehabilitation and functional recovery, but a risk for future pelvic floor disorders when age-related sarcopenia^{23,24} and motor neuron loss²⁵ occurs years or even decades later.

The ability to identify whether an individual woman is at risk for denervation/reinnervation would help with pre-natal counseling and ultimately decrease obstetrically related pelvic floor disorders. One of the mandates of the recent National Institutes of Health State of the Science Conference on Cesarean Delivery on Maternal Request was to identify pregnancy- and birth-induced injuries while controlling for effects of aging on pelvic floor and to identify modifiable factors that would decrease the risk of future pelvic floor disorders without having to perform cesarean delivery.²⁶ To minimize maternal childbirth injury and optimize postpartum recovery with immediate postpartum rehabilitation algorithms, future investigations at our center will use these and other electromyography characteristics to associate denervation/reinnervation that occurs as a result of pregnancy and childbirth with identifiable maternal characteristics and appropriate modifiable obstetric practices.

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FIGURE. Interference pattern examples

A, Full interference pattern in a normal subject. **B,** Reduced interference pattern in a different subject with significant neuropathy, which shows fewer turns, most with large amplitudes.

TABLE 1

Exam characteristics

Postpartum POP-Q (cm)	Mean (SD) ^a
Aa	-1.1 (1)
Ba	-1.1 (1)
C	-6.0 (1.1)
gh	3.2 (1.1)
pb	3.3 (1.1)
tvI	9.2 (0.9)
Ap	-2.1 (0.7)
Bp	-2.1 (0.7)
D	-8.3 (1)
Pelvic muscle strength (Oxford)	2.9 (1.2)
Pudendal nerve latencies (msec)	
Pudendal nerve terminal motor latency	2.3 (0.8)
Perineal nerve terminal motor latency	2.3 (0.9)
Sacral Reflexes (msec)	
Clitoral-anal reflex	47.6 (11.1)
Urethral-anal reflex	71.4 (13.4)

^aMean standard deviations of selected pelvic floor exam and neurophysiologic characteristics.

TABLE 2

Motor multi-MUAP and IP parameters by AI symptom

Anal incontinence										
Fecal incontinence										
Parameter	Gas			Liquid			Solid			P value
	Yes	No	P value	Yes	No	P value	Yes	No	P value	
MUAP										
Duration (msec)	6.3 ± 1.7	6.1 ± 1.1	.6	6.0 ± 1.3	6.4 ± 1.3	.4	6.2 ± 1.1	6.1 ± 1.4	.9	6.2 ± 1.5
Amplitude (μV)	343 ± 72	311 ± 67	.2	333 ± 72	305 ± 62	.2	346 ± 66	308 ± 68	.1	335 ± 76
Area (msec μV)	317.9 ± 121.7	262.4 ± 61.1	.15	283.7 ± 93.5	277.0 ± 84.4	.8	312.9 ± 78.2	264.2 ± 90.7	.1	321.9 ± 114.8
Area/amplitude	0.9 ± 0.2	0.9 ± 0.1	.6	0.86 ± 0.1	0.91 ± 0.1	.2	0.89 ± 0.1	0.88 ± 0.2	.9	0.86 ± 0.12
Phases (n)	3.2 ± 0.5	3.0 ± 0.3	.2	3.0 ± 0.3	3.1 ± 0.4	.5	3.1 ± 0.4	3.0 ± 0.4	.6	3.2 ± 0.5
Turns (n)	3.0 ± 0.6	2.7 ± 0.4	.1	2.8 ± 0.5	2.8 ± 0.5	.8	2.9 ± 0.5	2.8 ± 0.5	.4	3.0 ± 0.7
Interference pattern										
Amplitude/turn (μV)	318 ± 48	282 ± 38	.02	299 ± 38	287 ± 53	.4	320 ± 47	278 ± 35	.003	332 ± 48
Turns/second (n)	224 ± 87	204 ± 64	.4	218 ± 72	199 ± 71	.4	219 ± 70	205 ± 73	.6	216 ± 94
Activity (%)	10.5 ± 4.9	9.2 ± 4.3	.4	9.9 ± 4.2	9.1 ± 4.9	.6	10.5 ± 3.7	9.1 ± 4.8	.3	9.7 ± 5.4
No. of short segments (n)	137.2 ± 57.9	121.5 ± 51.6	.4	132.4 ± 50.2	117.1 ± 57.7	.4	137.8 ± 44.2	119.8 ± 57.3	.3	123.9 ± 63
Envelope (μV)	780.3 ± 286.6	648.8 ± 163.1	.08	689.3 ± 207.1	691.2 ± 241.5	.9	810.3 ± 217.5	627.7 ± 196.1	.01	824.8 ± 338.4

Independent *t*-test for all; data are given as mean ± SD. The data in bold type signifies statistically significant findings.