Quantitative anal sphincter electromyography in primiparous women with anal incontinence

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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] μV) and fecal incontinence (332 ± 48 μV), compared with those without fecal urgency (282 ± 38 μV) and fecal incontinence (286 ± 41 μV; $P = .02$, *t*-test).

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

Key words: anal incontinence, anal sphincter, postpartum period, quantitative electromyography

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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 deliveries[4] and the fact that some symptoms do not manifest for years after seemingly uncomplicated deliveries[5]) 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 tonically[9] 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

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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 $\geq$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 μV/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 Keypoint 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