Pudendal nerve stretch during vaginal birth: A 3D computer simulation

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Objective: The purpose of this study was to determine the increase in pudendal nerve branch lengths using a 3D computer model of vaginal delivery.

Study design: The main inferior rectal and perineal branches of the pudendal nerve were dissected in 12 hemi-pelves from 6 adult female cadavers. Their 3D courses were digitized in the 4 specimens with the most characteristic nerve branching pattern, and the data were imported into a published 3D computer model of the pelvic floor. Each nerve branch was then represented by a stretchable cord with a fixation point at the ischial spine. The length change in each branch was then quantified as the fetal head descended through the pelvic floor. The maximum nerve strains ([final length minus original length/original length] \times 100) were calculated for 5 degrees of perineal descent: reference descent from the literature, 1.25 cm and 2.5 cm caudal and cephalad. The effect of alternative fixation points on resultant nerve strain was also studied.

Results: The inferior rectal branch exhibited the maximum strain, 35%, and this strain varied by 15% from the scenario with the least perineal descent to that with the most perineal descent. The strain in the perineal nerve branch innervating the anal sphincter reached 33%, while the branches innervating the posterior labia and urethral sphincter reached values of 15% and 13%, respectively. The more proximal the nerve fixation point, the greater the nerve strain.

Conclusion: During the second stage: (1) nerves innervating the anal sphincter are stretched beyond the 15% strain threshold known to cause permanent damage in appendicular peripheral nerve, and (2) the degree of perineal descent is shown to influence pudendal nerve strain.

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Vaginal delivery is a major risk factor for developing pelvic floor dysfunction.\(^1\)\(^,\)\(^2\) Pudendal nerve damage is found in women with pelvic floor dysfunction,\(^3\) and has been documented as occurring with vaginal birth.\(^4\)\(^,\)\(^5\) For example, Allen's EMG measurements in nulliparous women before and after their first birth clearly implicated vaginal delivery in pudendal nerve injury. However, the precise mechanisms by which vaginal delivery results in pelvic floor nerve injury remain poorly understood.

Two mechanisms known to cause mechanical damage in appendicular peripheral nerve might be relevant to vaginal birth: excessive compression and stretch. There is indirect evidence from animal models suggesting that transverse compression of a nerve can cause injury.\(^6\)\(^,\)\(^7\) For example, compressive pressures exceeding 80 mm Hg for 2 hours result in permanent nerve conduction failure.\(^8\) Orthopedic experiments have also shown that when a peripheral nerve undergoes stretch exceeding 15% of its original length, permanent nerve conduction failure will result.\(^9\) This effect is exacerbated by the presence of nerve compression,\(^8\) perhaps explaining why branches of the pudendal nerve might be uniquely vulnerable as the fetal head dilates, both compressing the pudendal nerve as well as stretching it during vaginal birth.

In an earlier study we estimated the topographic distribution of the stretch induced in the pelvic floor muscles during the second stage of labor.\(^10\) These studies showed remarkable increases in pelvic floor muscle lengths during the late second stage. The question then arises as to the magnitude of length changes in the pudendal nerve. Although its anatomy is well established,\(^11\)-\(^13\) the geometric changes in the pudendal nerve during vaginal birth have yet to be quantified. Because it is not possible to visualize the changes in the nerve during the second stage of labor, we elected to use modern imaging and computer simulation methods to gain insights into the geometric and mechanical factors that might affect nerve length.

The goal of this paper, therefore, was to extend and refine our earlier model of pelvic floor muscle anatomy by adding representations of each of the pudendal nerve branches based on 3-dimensional imaging, the published anatomic literature, and our own dissections.

**Material and methods**

A total of 12 hemi-pelvises from 6 female formalin-fixed cadavers (mean age 72 years, range: 48-90 years) were dissected to measure the 3-dimensional geometry of pudendal nerve and its main branches. Using a posterior approach, the pudendal nerves were exposed after excising the gluteal muscles and sacrotuberous ligaments. The nerve and its branches were visualized from the sacral foramen to their distal terminations.

Four specimens (ages 48, 48, 80, and 90 years) with the most representative anatomy were selected for the 3-dimensional (3D) geometric study, and firmly attached to a rigid mounting frame. The 3D geometric course of each pudendal nerve branch was digitized from the S2-4 roots to their distal terminations using an Optotrack 3020 optoelectronic digitizing system (Northern Digital, Inc, Waterloo, Canada).

A representative course and branching pattern was established for each nerve branch named in standardized anatomic terminology (Terminologia Anatomica\(^14\)): inferior rectal nerve branch (IR), perineal nerve branch, and the dorsal nerve of clitoris branch. The branches relevant to urinary and fecal incontinence were specifically chosen for study, including the inferior rectal nerve branch and the perineal nerve branch, which was further divided into the muscular branch innervating the striated urethra sphincter (Per-US), and muscular branch innervating the external anal sphincter (Per-AS). The remaining part of the perineal nerve, the posterior labial branch (Per-L), was also included for analysis but the dorsal nerve to the clitoris was not modeled during this phase of the research because it is not involved in continence.

After the 3D course and the branching pattern of each pudendal nerve branch was identified, its relationship to the pelvic landmarks was determined by digitizing the locations of 5 pelvic reference landmarks: the midpoint of the pubic symphysis, the ischial spines, and each end of Alcock’s canal. The 3D geometric data from each specimen were then mathematically scaled and transformed so as to correspond to the size and shape of a normal reference pelvis described in Lien et al.\(^10\) Proper anatomic location of these representative nerves was verified by the one of us (J.O.L.D.) based on previous experience in approximately 200 pelvic dissections.

The geometric data describing the course and branching pattern of each branch were imported into a published 3D computer model (Figure 1) of the pelvic floor and vaginal birth in I-DEAS software (UGS Inc, Plano, Tex).\(^10\) The obturator internus muscle, piriformis muscle, coccygeal muscle, and sacrospinous ligament were added to that model, based on data digitized from magnetic resonance images of the healthy 34-year-old nulliparous women from whom the model was derived. Each nerve branch was represented as a stretchable cord routed from the sacral foramen via anatomic landmarks including the ischial spine, the proximal and distal end of Alcock’s canal, and the perineal membrane to their terminations. The model nerves were considered stretchable distal to possible nerve fixation points. In the absence of published data to the contrary, the pudendal nerve was initially assumed to be fixed at the ischial spine based on anatomic inspection. However, given inherent anatomic variability among individuals, we
analyzed the effect of violations of this assumption by considering 2 alternative fixation points that were also observed in our dissections: the sacral foramina and the nerve branching points.

Using methods described in our earlier work, the model was used to estimate the percentage strain ([final length minus original length/original length] × 100) in each pudendal nerve branch during the second stage of labor. As a first-order approximation, the fetal head was simulated by a 9-cm diameter sphere representing the size of the molded fetal head. The pudendal nerve branches and pelvic floor soft tissues were stretched around the “birth tube” formed by the path of the head’s equator as it descends along the curve of Carus (Figure 2). The initial and final length of each pudendal nerve cord was estimated during the second stage of labor. The overall percentage strain in each nerve branch was then also calculated and rounded to the nearest percent strain. Similarly, percentage strains were also calculated for the alternative nerve fixation assumptions described above.

An average value for perineal descent was determined from the only published measurements during birth we were able to locate in the literature. To estimate the sensitivity of pudendal nerve branch strain to different values of perineal descent, simulations of the second stage

Figure 1  Inferior view of the pudendal nerve branches and muscular structure of the female pelvic floor. The urethra (umber), vagina (pink), rectum (light brown), and external anal sphincter (maroon) are shown. Branches of the pudendal nerve (Pud) are shown: the inferior rectal nerve (IR), the muscular branch of perineal nerve innervating external anal sphincter (Per-AS), the posterior labial branch (Per-L), and the muscular branch of perineal nerve innervating the urethral sphincter (Per-US). The red bandlike structures represent the levator ani muscle; the purple band, the puborectal muscle; and the semitransparent blue structure represents the perineal membrane.
of birth were conducted for greater degrees of perineal descent: 1.25 cm and 2.5 cm more caudal to the reference descent; and also for lesser degrees of descent: 1.25 cm and 2.5 cm more cephalad to the reference value, respectively.

**Results**

The maximum strain in any pudendal nerve branch occurred in IR, reaching the value of 35% (Figure 3).

The strain in Per-AS also reached a large value, 33%. The strains in Per-L and Per-US reached more moderate values of 15% and 13%, respectively. Despite Per-AS having a similar initial length to Per-US and Per-L, it underwent higher strain than the other perineal nerve branches.

The predicted strain in each nerve branch showed a characteristic sigmoid relationship with fetal head descent (Figure 3). The IR was first engaged, followed by the Per-AL and Per-US, and then by Per-L. The maximal increase in nerve strain that occurred with each increment of fetal head descent (shown by the steepest slope of each curve in Figure 3) occurred in IRN between 4.4 and 6.4 cm fetal head descent. Maximal Per-AL strain occurred between 6.4 and 7.9 cm of fetal head descent. Finally, both maximal Per-L and Per-US strain occurred between 7.9 and 9.1 cm of fetal head descent.

Perineal descent altered predicted pudendal nerve strain during birth (Figure 4). The maximum strain in IR increased from an average of 34.5% to 40.6% when perineal descent was 2.5 cm greater than average. Strains in the other branches were also increased: Per-AS from 32.9% to 36.3%; Per-L from 14.7% to 23.2%; and Per-US from 12.5% to 15.3%, respectively. For a perineal descent 2.5 cm less than average, strains were reduced to 18.9% in IR, 28.9% in Per-AS; 6.8% in Per-L, and 9.0% in Per-US. Hence, greater-than-average descent most affected Per-L strain, while less-than-average descent most affected IR strain.

Alternative nerve fixation points also affected the resulting nerve strain (Figure 5). If the fixation point was assumed to be at the sacral foramina rather than the ischial spine, nerve stretch was reduced by 19.1% for IR, 10.5% for Per-AS, 9.1% for Per-L, and 7.9% for Per-US (because of its greater original length). On the other hand, when the fixation points were assumed to be located at the pelvic side wall, where the nerve branched from the main body of the nerve, pudendal nerve strain increased markedly by the following amounts compared with fixation at the ischial spine: IR, 52.4%; Per-AS, 77.4%; Per-L, 35.2%; and Per-US, 27.2%.

**Comment**

The sentinel observation that spawned interest in nerve injury and pelvic floor dysfunction was Parks’ finding that anal sphincter denervation in women with fecal incontinence was associated with vaginal birth. Subsequent studies have added further detail to this original observation. Electrodiagnostic studies, including electromyography (EMG) and pudendal nerve terminal latencies (PNTML) have been used to demonstrate pudendal nerve injury. Allen’s EMG test before and after childbirth showed that changes in pudendal nerve
function were seen in 80% of women vaginally delivered and, in the 4 women with the greatest change, immediate symptoms of urinary and or fecal incontinence occurred. Snooks found increased fiber density (via EMG) and prolonged PNTML after vaginal delivery indicating evidence of nerve injury and repair. By contrast, no change in EMG fiber density was found after cesarean section. This suggested that the pudendal nerve is injured during vaginal delivery but not during pregnancy. Animal models simulating the childbirth trauma with vaginal distension in rat support this hypothesis.

Our analysis provides the first estimates of pudendal nerve strain during the second stage of labor. In addition, it suggests which portions of the nerve are at greatest risk for stretch-induced injury. The 34.5% maximum strain in the inferior rectal nerve, and 32.9% in the perineal nerve to the anal sphincter found in this study exceed the 15% strain threshold known to result in rabbit tibial nerve injury and the 15% to 20% injury threshold in rat. These values may also be compared with the 17% to 25% distraction limit orthopedists employ to avoid nerve palsies resulting from operative lengthening of the human leg. The branches to the labia and urethral sphincter, because of their lateral course along the pelvic wall, have lesser degrees of strain (14.7% and 12.5%, respectively) of the pudendal nerve. If injury can be caused by nerve stretch exceeding a maximal permissible value, then the inferior rectal nerve and perineal nerve to the anal sphincter appear to be at greater risk for injury than the other pudendal nerve branches during the second stage of labor. These findings are consistent with the fact that damage to the anal sphincter was the first, and best, documented birth-induced pudendal nerve injury.

Stretch of a nerve is only one of several possible mechanisms that may be involved in birth-induced nerve injury. Rydevik, in his seminal research on nerve injury mechanisms, demonstrated the inter-related nature of nerve compression and nerve stretch. As a nerve lengthens, its diameter must decrease due to the Poisson effect, thereby constricting intraneural blood flow. When external compression is applied in a radial or transverse direction, further constriction of endoneural blood flow occurs, exacerbating the effect of the stretch in constricting blood flow. Indeed, experimental evidence in animal models shows that placing an inflated balloon in the vagina is able to cause denervation. However, the specific relationship between nerve lengthening and nerve compression deserves further investigation. The

Figure 3  The relationship between fetal head descent (abscissa, in cm) and the resulting nerve strain (ordinate, in percent) for the 4 pudendal nerve branches. IR, inferior rectal nerve branch. Perineal nerve branches: Per-AS, anal sphincter; Per-L, labial; and Per-US, urethral sphincter.
low injury rate of other nerves within the pelvis, such as the obturator nerve, branches of the sacral plexus, and lumbosacral trunks, which undergo compression but not stretch indicates that an interaction between stretch and compression may be necessary for damage.

There are several methodologic limitations involved in this type of research that must be considered in properly interpreting our findings. First, the assumption of uniform stretch along the nerve fiber leads to a conservative estimate of nerve strain: it may be greater in certain regions of the nerve, especially where the nerve bends around structures, such as the sacrospinous ligament. Second, the process of dissection, necessary to define the course of the nerve, frees the nerve from its
surrounding attachments; therefore, we did not study the points of nerve fixation in this study. We therefore made reasonable assumptions about possible fixation points and evaluated several candidate scenarios. We cannot exclude the possibility that higher local strain might occur at fixation points. This could possibly explain the injury of the perineal nerve branch to the urethral rat nerve observed by Damaser. As the perineal nerve penetrates the perineal membrane, its local adhesion might lead to damage despite limited overall length changes. Further study of these issues seems warranted.

Third, the pudendal nerve is a complex structure with multiple terminal branches. We therefore had to simplify this arrangement to a representative central nerve for each region in order to render the analysis feasible. Analyses of the strain in additional nerve branches may uncover local phenomena of potential importance. However, the overall trends predicted by the model should still be valid. Fourth, we did not consider time-dependent changes (i.e., how fast the nerve is stretched or time- or force-dependent material property effects) on the calculated nerve stretch. Although these effects may affect the strain history of the nerve, they will not affect our estimates of the maximum average pudendal nerve stretch, since the nerve branches still have to wrap around the fetal head to reach downward displaced structures, regardless of the force needed. We have also not considered the magnitude of any forces acting on the nerve branches, but this would be a logical extension of our research. Again, these analyses would not affect our estimates of maximal nerve strains. Fifth, this study focused on the longitudinal stretch in the nerve fibers. Other injury factors such as the compression and nerve bending need further examination. Sixth, we did not consider pelvic floor changes that occur during the last several weeks in preparation for delivery that might lessen the maximal nerve strain.

Seventh, the calculated pudendal nerve strains were generated from computer simulation. Clinical verification is needed but this must await the development of suitable methods for measuring pudendal nerve strain in vivo. However, the regions of nerve at high risk suggested by this model are consistent with clinical observations of nerve impairment, providing indirect validation to our model. Finally, the threshold value of stretch required for permanent loss of pudendal nerve conduction is unknown, as is the time that this stretch must be maintained, as are also the effects of hormones on this injury threshold during labor. We cannot exclude the possibility, for example, that pudendal nerve injury thresholds may be elevated during labor.

In this study, a generalized pudendal nerve course and a mean-size pelvis were used. Effects of pelvic size and shape variations on pudendal nerve strain need further study. A smaller or narrower pelvis has shorter pudendal nerves that have the potential to undergo larger stretch during birth of a fetus of given size. Racial differences in pelvic size and shape (for example) might also affect risk for pudendal nerve injuries, but this possibility requires further study.

There are several potential clinical implications of these findings that deserve further consideration. Of interest, we found that different degrees of pelvic floor descent affect nerve stretch. This raises the issue of whether specific obstetric factors, such as occipitoposterior position during delivery, which may lead to greater degrees of perineal descent, may increase the potential for nerve injury. Also, early episiotomy, by widening the vaginal opening, might lead to lesser degrees of descent. This contentious question deserves serious evaluation in order to resolve important yet unresolved issues. Further research studying the relative contribution of compression and lengthening of the pelvic nerves seems justified.

References


Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.ajog.2005.01.032