Pelvic floor injury during vaginal birth is life-altering and preventable: what can we do about it?

John O. L. DeLancey, MD; Mariana Masteling, PhD; Fernanda Pipitone, MD; Jennifer LaCross, DPT, PhD; Sara Mastrovito, MD; James A. Ashton-Miller, PhD

Pelvic floor disorders after childbirth have distressing lifelong consequences for women, requiring more than 300,000 women to have surgery annually. This represents approximately 10% of the 3 million women who give birth vaginally each year. Vaginal birth is the largest modifiable risk factor for prolapse, the pelvic floor disorder most strongly associated with birth, and is an important contributor to stress incontinence. These disorders require 10 times as many operations as anal sphincter injuries. Imaging shows that injuries of the levator ani muscle, perineal body, and membrane occur in up to 19% of primiparous women. During birth, the levator muscle and birth canal tissues must stretch to more than 3 times their original length; it is this overstretching that is responsible for the muscle tear visible on imaging rather than compression or neuropathy. The injury is present in 55% of women with prolapse later in life, with an odds ratio of 7.3, compared with women with normal support. In addition, levator damage can affect other aspects of hiatal closure, such as the perineal body and membrane. These injuries are associated with an enlarged urogenital hiatus, now known as antedate prolapse, and with prolapse surgery failure. Risk factors for levator injury are multifactorial and include forceps delivery, occiput posterior birth, older maternal age, long second stage of labor, and birthweight of >4000 g. Delivery with a vacuum device is associated with reduced levator damage. Other steps that might logically reduce injuries include manual rotation from occiput posterior to occiput anterior, slow gradual delivery, perineal massage or compresses, and early induction of labor, but these require study to document protection. In addition, teaching women to avoid pushing against a contracted levator muscle would likely decrease injury risk by decreasing tension on the vulnerable muscle origin. Providing care for women who have experienced difficult deliveries can be enhanced with early recognition, physical therapy, and attention to recovery. It is only right that women be made aware of these risks during pregnancy. Educating women on the long-term pelvic floor sequelae of childbirth should be performed antenatally so that they can be empowered to make informed decisions about management decisions during labor.

Key words: enlarged hiatus, forceps delivery, levator ani avulsion, occiput posterior, pelvic floor disorders, pelvic floor injury, pelvic organ prolapse, postpartum care, prenatal education, prevention, rehabilitation, stress urinary incontinence, vaginal birth

Introduction

Pelvic floor disorders (PFDs) after childbirth have devastating consequences on quality of life. This is not surprising given the remarkable pelvic floor changes during delivery (Figure 1). Of note, 1 in 4 American women (25%) suffer from these conditions.[1] Moreover, 20% of women will require surgery during their lifetime,[2] and many will suffer from nonsurgical conditions, such as urgency urinary incontinence. Reported symptoms include the protrusion of organs from the vaginal opening (prolapse); leaking urine when laughing, coughing, or exercising; leaking feces; and impaired sexual function. More than 60% of adult women experience some degree of urinary incontinence[3] and 3.3 million women in the United States live with pelvic organ prolapse, with 200,000 pelvic prolapse surgeries performed annually.[4] Vaginal birth is, by far, the single most important modifiable risk factor for the development of PFDs—especially pelvic organ prolapse, with 1.6 times more surgeries than stress urinary incontinence and 9.5 times more surgeries than fecal incontinence performed annually (Figures 2[4–7] and 3[8]).

Anal sphincter injuries that are visible at the time of delivery occur in approximately 3% of vaginal deliveries.[9] Over the last 2 decades, ultrasound and magnetic resonance imaging (MRI) studies of the deeper pelvic floor structures,

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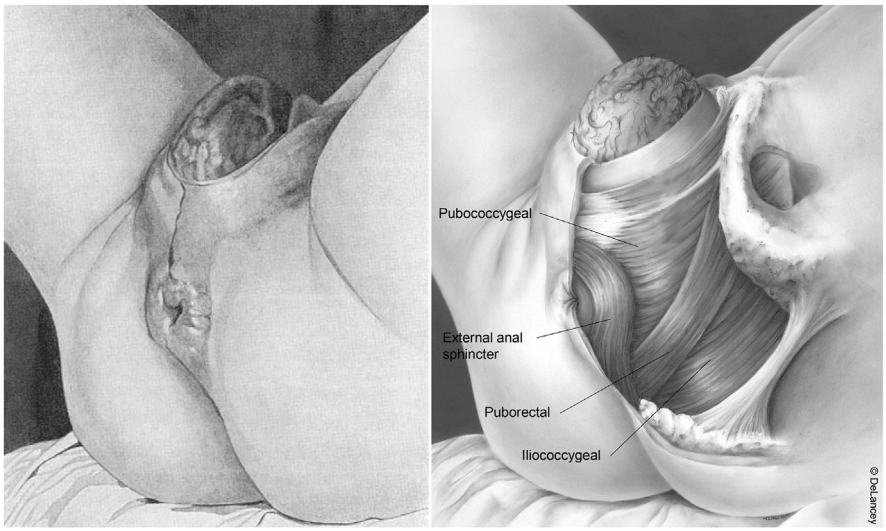
FIGURE 1
Hidden pelvic floor muscles late in the second stage of labor



The perineal membrane is not shown.

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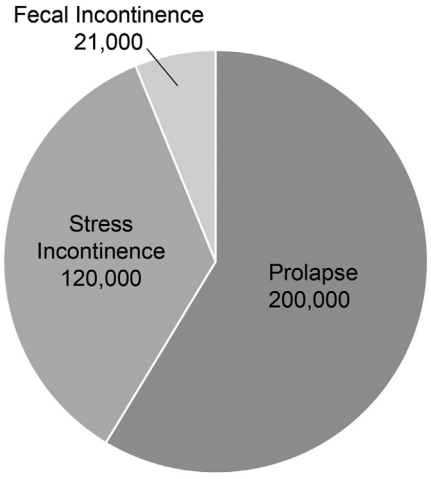
such as the levator ani muscle (Figure 1), have shown that this muscle can be severely injured during vaginal delivery in up to 19% of women, which is 6 times more likely to occur than in anal sphincter injury. This injury does not recover and is a leading cause of PFDs later in life.[10]

FIGURE 2
Annual number of women undergoing pelvic floor surgeries[4–7]



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This expert review will focus on newer information about these hidden injuries. We describe the injuries that occur and the mechanisms of injury. Subsequently, we touch on how these injuries might be reduced in the future. Because these injuries occur in hospitals while women are cared for by obstetricians and midwives, there is an ideal opportunity to use this new knowledge to reduce the number of women injured.

Steps needed to prevent birth injuries

Let us consider the widely adopted eponymous van Mechelen model for preventing injuries (Figure 4).[11,12] It was originally developed as a framework for better understanding the factors leading to sports injuries and testing interventions aimed at preventing those injuries. Here, we apply it to what will be needed to prevent birth injuries in the future. We see that step 1 is to establish the incidence and severity of the pelvic floor injury that will be discussed in subsequent sections. This has been well established for levator ani injury, but we are still establishing the incidence and severity of injury to the perineal body and membrane. Step 2 involves understanding the etiology and pathomechanics of the injuries—an active topic of research to be discussed later in this article. Step 3 is to introduce one or more preventive measures, which, except for perineal compresses and massage, are presently limited.[13] This is because there is currently no simple and reliable assessment strategy to identify, before labor, the up to 20% of women who will sustain a levator ani injury during vaginal delivery. As it makes little sense to apply an intervention to the 80% of women who will not be injured, such a test is sorely needed. Step 4 is to assess the effectiveness of the intervention via randomized clinical trials in different care settings. In summary, the current major knowledge gap is the lack of reliable means to rapidly predict who will be injured in a vaginal delivery so that we can find ways to better prevent these injuries.

Childbirth and the pathophysiology of pelvic floor disorders

During a woman's life, many factors affect pelvic floor function. Vaginal birth and genetic, nutritional, and hormonal factors all affect an individual's overall growth to adulthood (Figure 5).

A graphical representation of these factors can illustrate how different life events and age interact to cause symptoms (Figure 5, A and B).[14] The graph has a theoretical y-axis variable representing any theoretical "pelvic floor function." This variable could indicate any single factor, such as the strength of the levator ani muscles or urethra. It might also represent the coordinated actions of several structures, such as the urethral support apparatus that involves muscles, their neural control system, and attaching fascial structures. The x-axis is age.

After reaching full maturity, childbirth, age, and other factors can lead to damage or deterioration of pelvic floor function. The demands that a woman's lifestyle places on the pelvic floor range from mild stress in a healthy but completely sedentary individual to severe stress in someone with a chronic cough or who competes in powerlifting competitions (Figure 5, D and F).

Symptoms result from the interaction between the strength and structural

integrity of the pelvic floor components and the severity of demands placed on them. A woman with an average pelvic floor may not have symptoms in a sedentary life, whereas an active person may have definite symptoms.

Vaginal delivery followed by "normal" repair and recovery does not affect pelvic floor function sufficiently to cause problems (Figure 5, C). Admittedly, however, there are visible changes in the perineal structures of parous women indicating previous vaginal birth. In addition, pelvic floor function can be severely affected if there is more definite injury or defective repair (Figure 5, D). In some cases, with partial recovery, PFDs will appear after 20 to 30 years (Figure 5, D, line 2). Nevertheless, in individuals with extensive damage that the body cannot repair, PFDs occur immediately after birth (Figure 5, D, line 3). Some women have a sufficiently severe injury such that prolapse is immediately visible and does not recover, even temporarily (Figure 6).

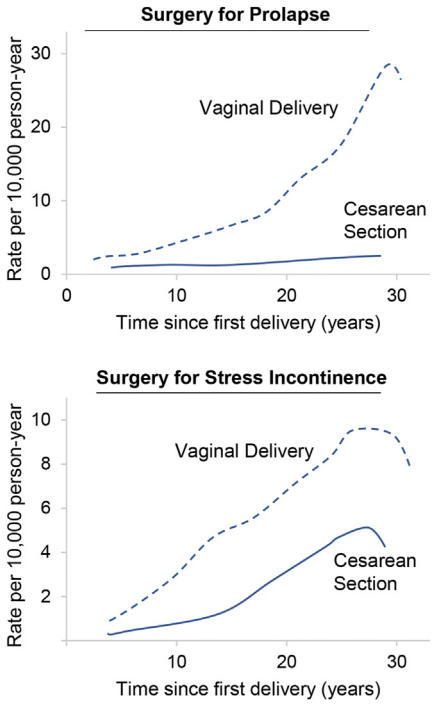
Hiatal enlargement and prolapse

The 2 largest hiatuses in the body, the levator and the urogenital, are the pelvic floor openings through which the fetus is pushed during birth (Figure 7). As explained below, normal hiatal closure is essential to maintaining normal support. In most women, the birth canal recovers after delivery; however, certain women sustain unrecoverable injuries (Figures 6 and 7) that may impair their ability to maintain the closure of these hiatuses effectively.

Successful closure depends on creating a vaginal "high-pressure zone,"[15,16] much like those created by the anal and urethral sphincter muscles to maintain continence. The perineal complex, consisting of the levator ani muscle, perineal body, and perineal membrane (formerly known as the urogenital diaphragm), creates this area of increased vaginal pressure. It is dependent on intricate interactions between the levator ani muscles, their neural control mechanism, the perineal membrane, and the perineal body (Figure 7).

It is now clear that hiatal enlargement is the single most important birth-related

FIGURE 3
Operations for prolapse and stress incontinence by delivery mode



Adapted from Leijonhufvud et al.[8]

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FIGURE 4
Van Mechelen model for preventing injury[11,12]

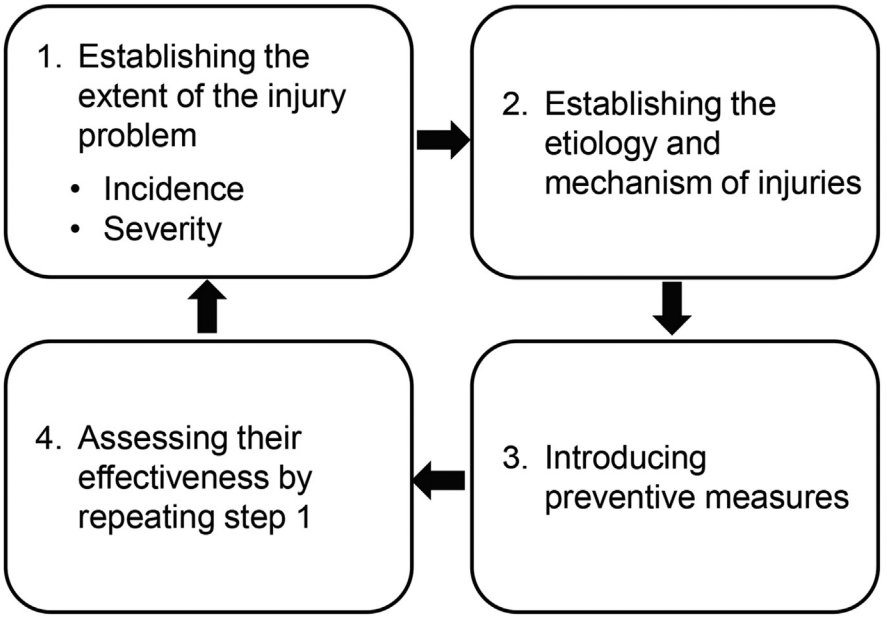

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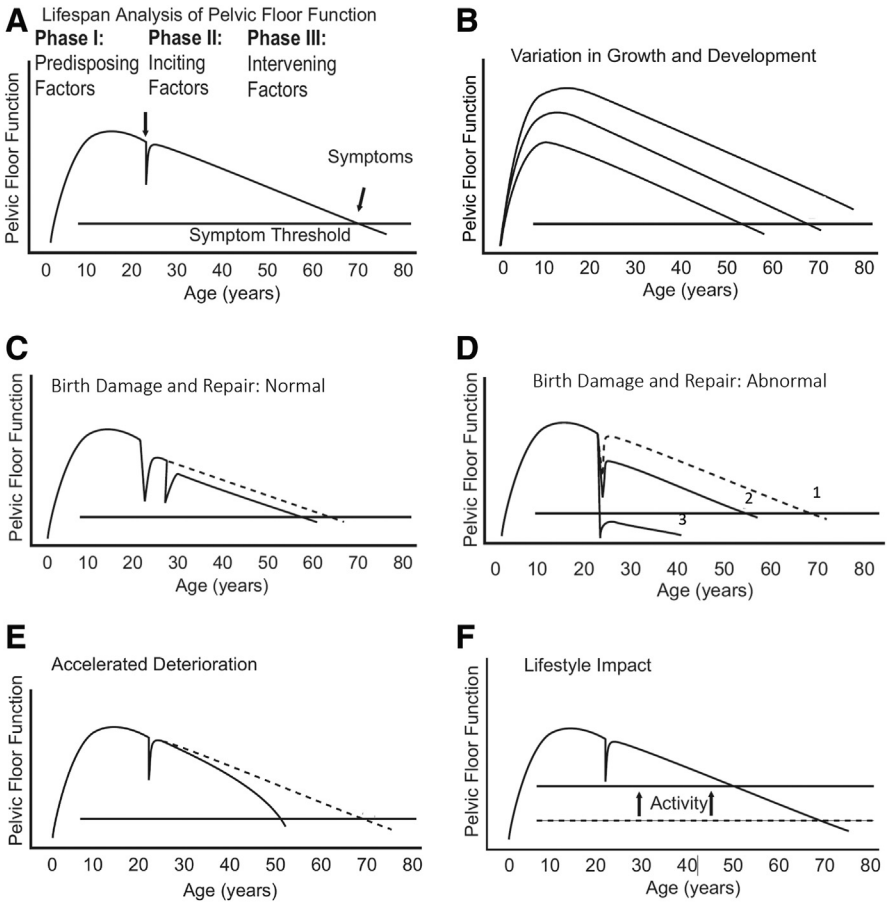
FIGURE 5
Graphical display of the concept of pelvic floor function



A, Phases of a woman's life span. **B,** Different degrees of functional reserve. **C and D,** Variations in birth damage and repair. **E,** Accelerated deterioration. **F,** Lifestyle impact. Adapted from DeLancey et al.[14]

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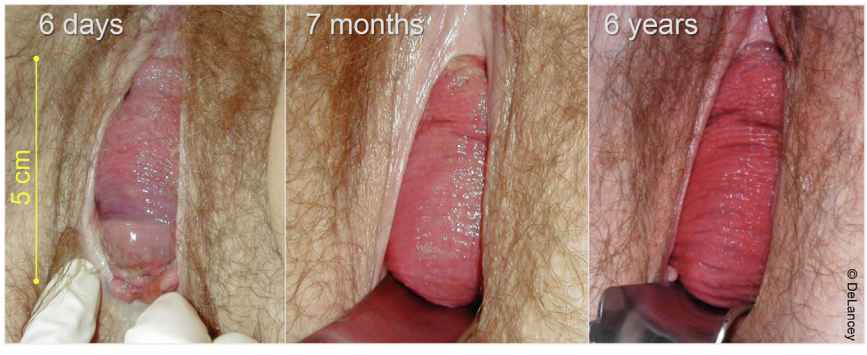
FIGURE 6
Enlarged hiatus 6 days after birth persisting for 6 years



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factor associated with pelvic organ prolapse (Figure 7).[17–20] Increasing hiatus size is clearly associated with an increasing likelihood of developing prolapse (Figure 8[21]).[22] In addition, hiatal enlargement precedes the occurrence of prolapse, indicating a potentially causal relationship (Figure 6).[23]

During the first 15 to 20 years after birth, approximately 25% of women with an enlarged straining hiatus (≥3 cm) followed prospectively developed prolapse at least 1 cm below the hymenal ring; this number increased to more than 60% of women if the hiatus was ≥4 cm.[21] The estimated median time to develop prolapse was 33 years for a woman with a 3.0-cm hiatus, whereas it was only 6 years for a woman with a 4.5-cm hiatus.[21] Of note, 2 recent studies have shown that <25% of the variation in hiatus size is attributable to the degree of muscle injury present on MRI.[20,24] This fact indicates that levator injury is not the only factor involved in an enlarged hiatus. The other changes involved likely relate to the connective tissue components of the perineal complex (perineal membrane and body), whose contribution to an enlarged hiatus has not been studied to the same extent as levator injury. When several aspects of pelvic floor hiatus closure are examined (muscle strength, perineal elevation with muscle contraction, descent during the Valsalva maneuver, and visible muscle on MRI), they are each found to be independent contributing factors.[20] Correlations between these factors revealed that no 1 factor explains more than 20% of the variation in others.

What causes prolapse?

Understanding why pelvic floor damage results in prolapse requires an understanding of the interactions between muscle impairment and connective tissues that attach the pelvic organs to the pelvic walls. To provide pelvic organ support, the muscles and ligaments must resist the downward force applied on the pelvic floor by the weight of the abdominal organs and the dynamic forces that arise from increases in abdominal pressure during coughing or

sneezing or from inertial loads placed on it, such as when landing from a jump (Figure 9).

This normal load sharing between the adaptive action of the muscles and the energy-efficient support from the connective tissues is part of the elegant load-bearing design of the pelvic floor. When injury to 1 of these 2 components occurs, the other must carry the increased demands placed on it. For example, it is a fundamental biomechanical principle that, in a situation where muscle and connective tissue both resist a load in parallel, while that muscle is injured, the connective tissue will have to carry more of the load. If this load exceeds the strength of the pelvic tissues, they may be stretched or broken, and prolapse may result.[25] This forms a causal chain of events by which pelvic muscle injury may influence pelvic organ prolapse. In addition, there is accumulating evidence that women operated on for pelvic organ prolapse have higher postoperative failure rates than women who have undamaged muscles if they have levator ani muscle impairment assessed by biopsy,[26] muscle function testing,[27] and ultrasound.[28] Moreover, there are early differences in pelvic organ support seen after surgery depending on whether a levator defect is present.[29] Similarly, muscle avulsion is seen more commonly in women with anatomic recurrence at 2 years after reconstructive surgery than in women with no recurrence.[30] Therefore, it is the disruption of the normal load sharing between the active levator muscles and related connective tissue structures (perineal body and membrane) that normally maintain hiatal closure that leads to the development of pelvic organ prolapse.

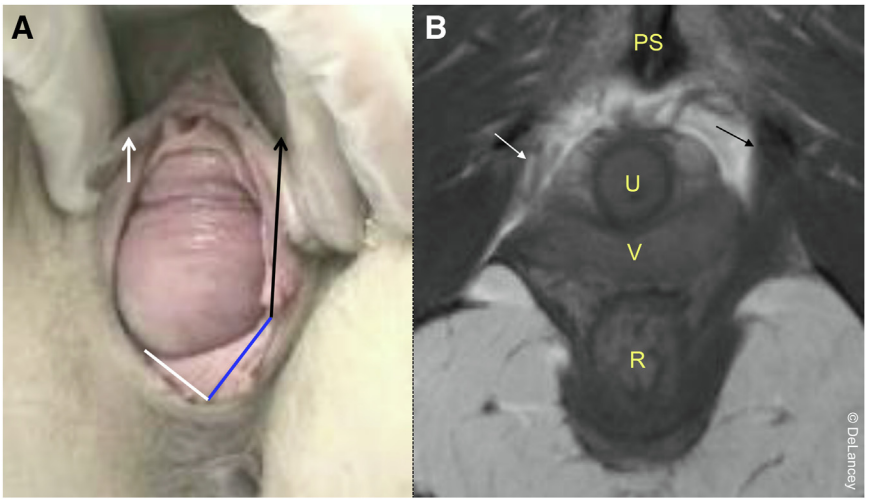
Levator ani and perineal complex

As described briefly above, there are 3 structures that participate in the closure of the lower vaginal canal that we refer to simply as the "perineal complex": (1) the levator ani muscles, (2) the perineal membrane, and (3) the perineal body, along with the associated fascial connective tissues that bind these structures together (Figure 10). They surround the urogenital hiatus and affect pelvic floor closure by creating a high-pressure zone in the lower third of the vagina.[15,16] Damage to any 1 of the 3 components of the perineal complex (levator, perineal membrane, or perineal body) can affect the other 2 elements in the complex.

Levator ani muscle anatomy

The levator ani muscle consists of 3 portions: the pubovisceral (also known as the pubococcygeal), the iliococcygeal, and the puborectal (Figures 1 and 10).[31,32] Our published studies of their lines of action show that the pubovisceral muscles lift the perineal structures and close the hiatuses on the pelvic floor.[33] The puborectal muscle arises lateral to the pubovisceral muscle and passes dorsal to the anorectal junction. Although both the pubovisceral and puborectal muscles can act to close the pelvic floor, only the pubovisceral muscle can lift the perineal structures cranially because of its more vertical orientation.[33] The iliococcygeal muscle is a thin sheet of muscle that spans the pelvic canal from the tendinous arch of the levator ani to the midline iliococcygeal raphe. Pelvic floor closure in level III is provided by the pubic portions of the levator ani muscles and their connections to the perineal membrane and perineal body in the perineal complex.

FIGURE 7
Anterior wall prolapse in a woman with a unilateral levator muscle tear



A, Intact muscle seen using magnetic resonance imaging scan (*black arrow*). **B,** Missing muscle (expected location indicated with *white arrow*) that results in the asymmetry of the perineal body (**A**) that is attached on one side (solid black arrow) and not on the other (separated by a *white arrow*).

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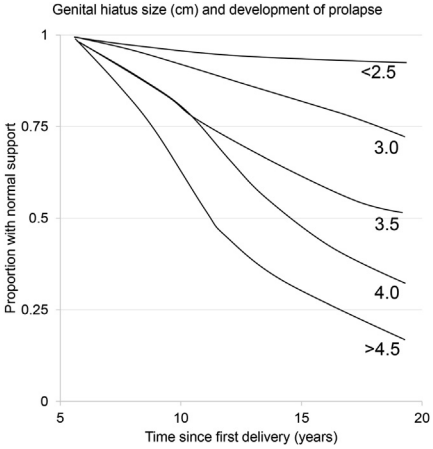
FIGURE 8
The proportion of individuals maintaining normal support when followed longitudinally



Each plot relates to the size of the urogenital hiatus during straining on examination from 2.5 to 4.5 cm. A decline in normal support represents an increase in prolapse at or below the hymen. Adapted from Handa et al.[21]

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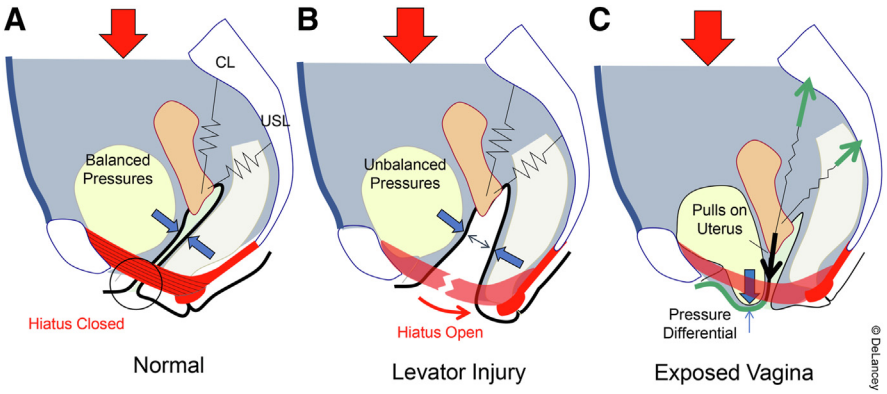
FIGURE 9
Interactions among levator, anterior wall, and apical ligaments



The *red arrows* represent the force created by gravity and abdominal pressure. With normal levator function (*red band*) (**A**), the hiatus is closed, and the vaginal walls are in apposition; the anterior and posterior pressures are equal and cancel (*blue arrows*). Levator damage (**B**) results in hiatal opening, and the vagina becomes exposed to a pressure differential between abdominal and atmospheric pressures. This pressure differential (**C**) makes the vaginal wall protrude and creates a traction force on the cardinal ligament and uterosacral ligament.

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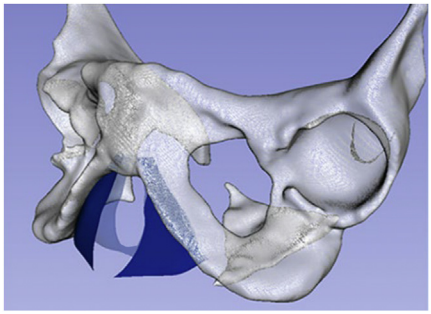
FIGURE 11
Birth-associated changes in the perineal membrane



Normal view is presented in *light blue*. Postpartum view is presented in *dark blue* showing separation of the 2 sides and the resulting "swinging door" rotation and descent. Adapted from Pipitone et al.[34]

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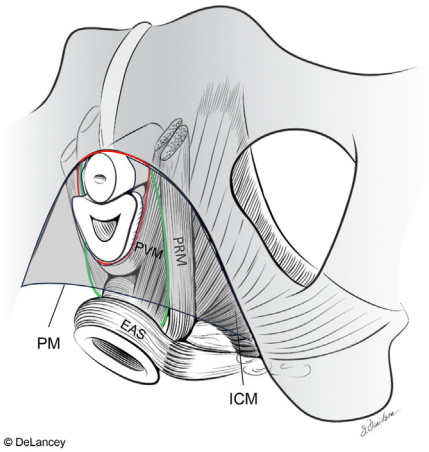
FIGURE 10
Pelvic floor seen from below after removal of the vulvar structures



The urogenital hiatus is outlined in *red*, and the levator hiatus is outlined in *green*.

EAS, external anal sphincter; *ICM*, iliococcygeal muscle; *PM*, perineal membrane; *PRM*, puborectal muscle; *PVM*, pubovisceral muscle.

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Perineal body and membrane

The perineal membrane and body are altered by pregnancy and childbirth and are abnormal in women with prolapse. These structural alterations have been demonstrated using a recently developed MRI-based reconstruction technique (Figure 11)[34] showing a caudal rotation of the membrane as a pregnancy effect and separation of the 2 sides of the membrane from the midline as the most prominent childbirth-related structural change. Because of their intimate connection with the levator ani, this lateral rotation is likely associated with diastasis of the levator ani and thereby enlargement of the urogenital hiatus.

How are structures injured?

There have been many competing theories proposed to explain the cause of birth-related pelvic floor injury: altered neural function (see for example[35]), evidence from blood samples suggesting ischemia and reperfusion[36] from compression, and levator tearing.[37,38] As efforts at prevention must be based on a proper understanding of why the injury occurs, it is necessary to decide between these hypotheses (Table 1[39–44]). Current evidence shows that tearing is the plausible hypothesis for levator injury.

Edema is the first response to overstretching and initiates the healing process. The large amount of edema seen in the pubovisceral muscle (Table 1[39–44]) supports the theoretical studies showing that this region of the levator ani is stretched the most, up to 300%, during vaginal birth.[45] The magnitude of levator ani muscle tears did not substantially change by 8 months after delivery, but levator ani muscle edema and bone injuries showed total or near-total resolution.[39–41] The magnitude of unresolved musculoskeletal injuries correlated with the magnitude of reduced levator ani muscle force and posterior vaginal wall descent showing failed hiatal closure.

The levator ani muscle tears can involve one or both sides of the muscle and can be graded as high or low grade depending on the amount of muscle involved. In the case of a full tear, the muscle detaches from its origin at the pubic bone. This happens when the muscle is overstretched during the second stage of labor, when there is a larger-than-normal tensile force on the muscle (every muscle physiologist knows that an active muscle can only be torn when it is forcibly lengthened in a so-called

TABLE 1
Review of 3 competing hypotheses for why the levator ani are injured during the second stage of labor[a]

	Hypothesis		
	Overstretch and tearing	Compression	Neuropathy
Scientific evidence	Proven.	Not causal for visible injury.	Not causal for visible injury.
Evidence	Seen immediately and will not resolve with time.	If compression is the mechanism, edema of both the internal obturator and the levator ani muscle should be present, as they are adjacent and both would be equally compressed.	If neuropathy, normal muscle bulk would atrophy over time.
Supporting evidence[39,40]	29% of patients had levator ani avulsion; 66% of patients had pubic bone marrow edema; and 29% of patients had a subcortical fracture. From Pipitone et al[41] on same population: 51% pubovisceral muscle edema; 5% puborectal muscle edema; and 5% iliococcygeal muscle edema.	Only levator edema was present, with no evidence of internal obturator edema. Edema always resolved. The levator edema is likely caused by stretching.	None of the women showed a pattern supportive of atrophy because of neural injury, where an initially normal-appearing muscle became atrophic.
Clinical issues	Given the viscoelastic nature of muscle and connective tissue, allowing a slow and gradual delivery would be the right solution (ie, slow delivery of fetal head). Relaxing the muscle decreases the risk of stretch-related injury.[42–44]	If causal, decrease the duration of time during which the tissues are compressed would be logical (ie, reducing the length of second stage of labor).	Nerve compression or stretching would be presumed mechanism and is associated with birth but not visible levator tear.

[a] Injuries are visible postpartum as levator abnormalities on ultrasound or MRI.

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eccentric or lengthening contraction, which can double the force acting in the muscle),[42,46] and when the muscle exceeds its ultimate tensile strength (Figure 12).

In addition, there are neurologic changes to the pelvic floor with childbirth that might play a role in PFDs, but they are not the cause of levator injury.[47] The pudendal nerve innervates the voluntary urethral and anal sphincters; the levator receives its nerve supply from the sacral plexus.[48] The stretching of the pelvic floor tissues during delivery (Figure 13[49]) might cause nerve stretching and neuropathy, as seen in the abnormal electromyography findings in the pelvic floors of 29% of women at 6 months after delivery[35] and in women with prolapse and stress incontinence.[50]

Birth injury biomechanics

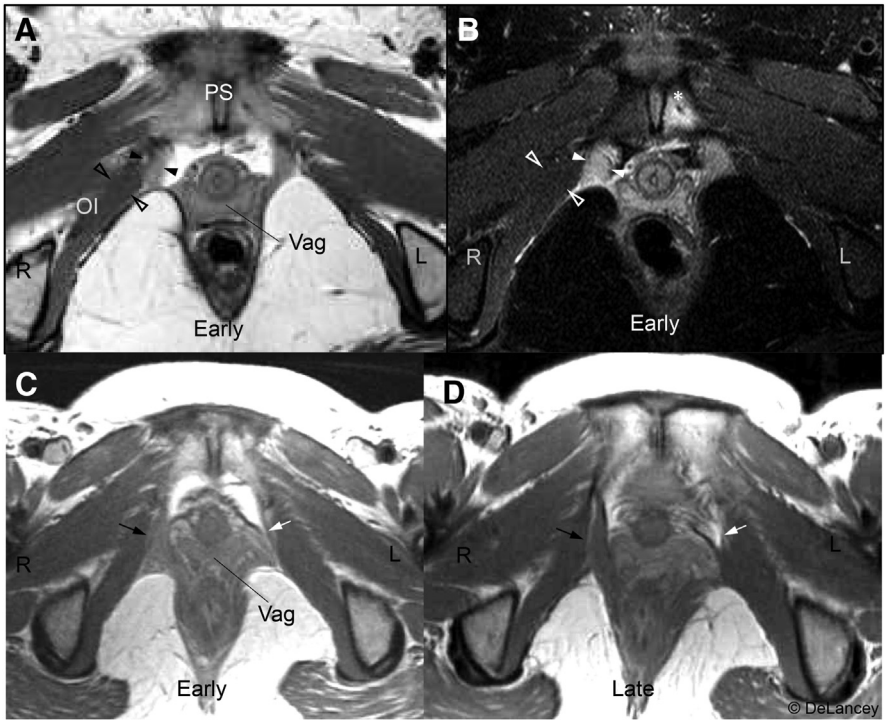
The pelvic floor tissues start changing in preparation for delivery during pregnancy.[51] During the third trimester of pregnancy, the area of the levator hiatus at rest increases up to 29%.[52] These changes start at the molecular level. Pregnant murine models show 20% to 30% sarcomere elongation and a 50% to 140% increase in the extracellular matrix of pelvic floor tissues in late pregnancy.[53] The most remarkable changes occur to the viscoelastic properties of the pelvic floor tissues (increased "stretchiness"), which allow a 300% stretch to occur with relative ease in most people (Figure 14).[45,54–56] We are most interested in understanding what happens in people where this stretch does not occur successfully and injury occurs. The cellular and molecular factors responsible for this phenomenon need to be established.

Tracy et al[57] examined the factors affecting the size (geometric capacity) of the lower birth canal to accommodate delivery of fetal heads of different diameters (demand)—for simplicity, referred to as capacity-demand. When this analysis was updated to include the measured viscoelastic properties of the lower birth canal, some 15% of women were predicted to be at risk of stretch-related injury to this region.[56,57] This analysis holds the promise to be able to identify which specific women are at greatest risk of levator ani muscle injury. For example, a woman with a relatively small hiatus and a large fetal head might be able to deliver successfully if her tissues are "stretchy" but might suffer a birth injury if they are too stiff. Injury prediction using these 3 factors (maternal hiatal dimensions, fetal head size, and birth canal viscoelastic properties) might allow the likelihood of injury to be considered in delivery planning before a long labor.

Diagnosing levator injury

Imaging women in the postpartum period can improve early diagnosis. For symptomatic women, it provides objective evidence to explain and validate the symptoms and problems that they are having. It can also suggest the need for referral to physical therapy to help

FIGURE 12
Midurethral axial MRI where the pubococcygeal muscle is normally seen



A, Proton density scan where the *solid arrow heads* mark the pubococcygeal muscle and the *open arrow heads* show the obturator internus. Signal intensity is lower (*lighter*) in the pubococcygeal muscle (*arrowhead*) than the adjacent internal obturator muscle (*open arrowhead*). **B,** Fluid-sensitive scan. This difference is more apparent, and an *asterisk* marks pubic bone edema and fracture. **C,** Normal pubococcygeal muscle (*black arrow*) is seen between the vagina and internal obturator (*black arrow*), although it is absent on the left. This pattern persists in the late scan (**C**).

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strengthen the uninjured parts of the muscle to compensate for the loss of 1 portion of the muscle. This is especially useful for women after a first vaginal birth with a high risk of pelvic floor injury (>35 years old, operative delivery, shoulder dystocia, vaginal birth after cesarean delivery, occipital-posterior presentation, rotational delivery, large vaginal tears, or primary obstetrical anal sphincter injury repair).[58]

Recognizing women who are at risk can be as simple as measuring the size of the urogenital hiatus with a ruler during a pelvic examination.[59] Although palpation during pelvic examination is the easiest diagnostic method to implement in routine clinical care, the value of this method is limited, as there is a considerable learning curve and only moderate interrater reliability compared with other imaging diagnostic methods. Palpation often relies on the comparison of findings with a supposedly intact contralateral side, which makes bilateral defects much more difficult to detect with finger palpation than on imaging.[32,60–62]

Both 3-dimensional transperineal ultrasound and MRI are noninvasive imaging techniques used clinically to evaluate the pelvic floor. Although MRI is considered the gold standard (Figure 12),[37] perineal ultrasound is more accessible, is easier to implement in routine clinical care, and has reasonable agreement with MRI in detecting levator defects.[63] To assess other injury mechanisms, MRI is still superior for identifying muscle atrophy and edema as a sign of trauma seen as an increased signal intensity on fluid-sensitive scans.[39–41]

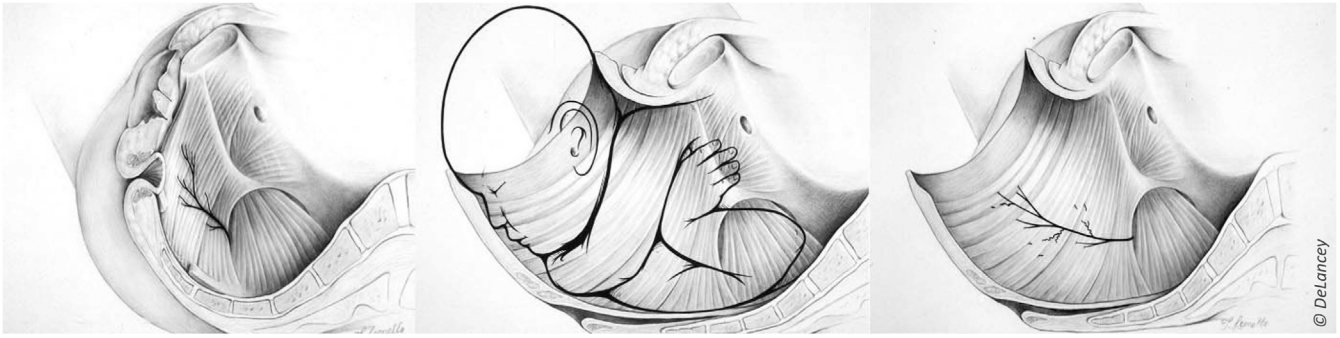
Risk factors and prevention strategies

Levator injury is a multifactorial event; however, there are interventions with the potential to reduce the risk of injury that are clinically plausible and those that are under development (Table 2[44,55-57,64-78]). Ineffective risk-reducing actions, including the use of the EPI-NO; the position, pattern, intensity, and types of pushing during the second stage of labor; manual perineal support; and water birth are described in the Appendix. From a prevention standpoint, pelvic floor muscle training is ineffective in preventing levator injury and urinary incontinence. Its therapeutic role will be discussed in the Interventions and rehabilitation section.

There is moderately robust long-term population-based epidemiologic data on major risk factors for PFDs. Among these are (1) urinary incontinence before pregnancy[79]; (2) ethnicity, with higher rates in Asian American and White women than in African American women[80]; (3) older age at birth of the first child[80–82]; (4) greater BMI[80,81,83,84]; (5) family history of PFDs[80]; 6) baby's weight and maternal height (if the baby's weight is >4 kg and the mother's height is <160 cm); and (7) operative delivery.[8,85,86]

Of note, 1 obvious way to reduce levator ani muscle injury is to avoid obstetrical practices that cause injury whenever possible. For example, when it would be equally feasible to use a vacuum delivery vs forceps delivery, the increased risk of pelvic floor injury with forceps delivery would make vacuum delivery the better choice.[87] Similarly, manually rotating the fetal head from occiput posterior to occiput anterior for delivery allows a smaller diameter fetal head to pass through the pelvic floor hiatuses. This should reduce the stresses on the birth canal[88] and reduce the risk of injury. These simple changes in practice require no special training or increased cost, so they can be considered (Table 2[44,55-57,64-78]). In addition, practices shown in randomized trials to

FIGURE 13
Circumferential and downward levator ani and nerve stretch during birth



Similar changes could occur to the innervation of the external anal sphincter.[49]

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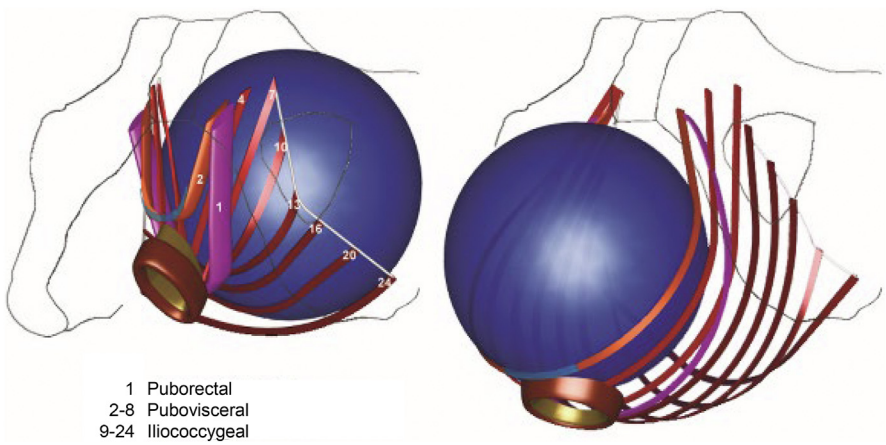
reduce the risk of perineal tear (warm compresses and perineal massage) may make sense, as they might help reduce levator ani muscle damage; however, randomized trials will be needed to test their efficacy.[75] There are also ongoing trials to dilate the lower birth canal in the first stage of labor to reduce levator injury risk (see for example,[77] and the EASE clinical trial #NCT03973281), but formal results are not available at the time of writing. Furthermore, the current practice of inducing labor at 39 weeks of gestation has the potential to reduce injury.[89] It is well established in injury science that a contracted muscle is much more vulnerable to injury than a relaxed muscle, and so it makes theoretical sense to coach women to relax their muscles during the late second stage—a practice often already done to facilitate head descent.[42–44]

Another approach would be to identify women before labor who are almost certainly going to have an injury during birth and offer potential cesarean delivery. Naturally, this would depend on accurate predictions so as not to unnecessarily increase cesarean delivery; however, given the fact that these births would also likely be associated with prolonged labor, shoulder dystocia, severe lacerations, and hemorrhage, there would be additional benefits beyond injury reduction. Obstetricians and gynecologists have extensive experience in estimating risk and practicing targeted prevention, so the research needed to prove or disprove such an approach could easily be designed and conducted to determine optimal cutoff values. The framework using the capacity-demand model is available,[57] which is similar to analyzing whether a truck could fit under a bridge. The height of the bridge is the capacity, divided by the height of the truck, the demand. A ratio of <1 indicates that the truck will not pass. This strategy would involve an ultrasound assessment later in the third trimester of pregnancy to measure the size of the urogenital and levator hiatuses, arch of the pubic bones, and size of the fetal head. A table of risk can be generated with a woman's risk of sustaining a permanent injury. Women with a value significantly <1 could be evaluated for potential cesarean delivery—especially if they only plan 1 birth or are at increased risk because of older age. Proof of concept using postnatal fetal head size

FIGURE 14
The effect of birth on the length of the levator ani muscle fibers



Representative muscle bands for different components are shown before and after dilation during the second stage of labor. Note that the pubovisceral muscle fibers (slings 2—8) are the shortest before birth and undergo the most elongation (and, therefore, are at highest risk of stretch injury). Modified from Lien et al.[45]

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TABLE 2
Factors associated with levator ani injury that have been considered for risk-reducing interventions, both clinically plausible and in development[a]

Potential risk-reducing actions	Hypothesized mechanism	Evidence for altered risk
Clinically plausible interventions		
Vacuum rather than forceps	Reduced traction force and smaller perimeter.	Forceps has an OR of up to 5.9 for levator injury.[64–68] Traction force and peak pressure are up to 2-fold higher when using forceps.[69–71]
Manual rotation from occiput posterior to occiput anterior	Smaller presenting fetal head diameter.	Occiput posterior delivery has an OR of 3.9 for the development of levator injury.[68,72]
Perineal massage or compresses	Change in tissue properties because of stretch and/or heat application.	Perineal massage has an RR of 0.49 for incidence of severe perineal trauma, RR of 1.40 for an intact perineum, and RR of 0.56 for incidence of episiotomy.[73,74] The use of warm compresses has an RR of 0.46 for developing third- and fourth-degree perineal tears, but no effect on the incidence of first-degree tears.[75]
Early induction of labor	Smaller infant requires less stretch of the pelvic floor tissues.	Head circumference above 35.5 cm (>50th percentile) has an OR of 3.3 for the development of levator injury.[72,76]
Slow gradual delivery	Allows increased stretch of tissues before injury (viscoelastic tissue relaxation).	Incomplete or absent levator ani muscle relaxation seems to be associated with a longer second stage of labor (clinical opinion).[44]
Interventions in development		
Predilating the tissues during labor	Predilating the birth canal tissues during the first phase of labor might reduce the risk of overstretch during the second phase of labor.	Preclinical trials have shown no adverse effect, currently undergoing large multisite clinical trial (NCT03973281).[77]
Capacity-demand risk assessment and selective cesarean delivery	Identifies only those at the highest risk for prevention, such as cesarean delivery.	Engineering analysis and preliminary clinical studies show that the risk of levator injury is lower when the maternal levator hiatus (capacity) is large and the fetal head diameter (demand) is small, identifying women who might benefit from cesarean delivery.[55–57,78]

OR, odds ratio.

[a] Ineffective interventions are listed in the Appendix.

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coupled with antenatal hiatus and pelvic bone measurements demonstrated an 80% ability to predict injury,[90] even in the absence of other factors used in the Tracy model (described above in the Birth injury biomechanics section).

Birth and stress urinary incontinence

Stress incontinence occurs 2 to 3 times more often in women who deliver vaginally than in those who deliver via cesarean delivery (Figure 3[8]). There are 2 possible explanations for this: (1) damage to the muscular and fascial tissues that support the urethra and (2) damage to urethral closure normally generated by the smooth and striated muscles in the urethral wall.

Primiparous women who developed de novo stress incontinence that persisted until at least 9 months after vaginal birth had 25% lower maximal urethral closure pressure at rest and 31% greater vesical neck movement during cough, showing that both factors were involved. Compared with nulliparous women of similar age and race, primiparous continent women had only 7% lower maximum urethral closure pressure. Table 3[91,92] shows the effect sizes for these parameters. This is similar to the value found in a longitudinal study that assessed women from 8 weeks of gestation to 8 weeks after delivery. They found a 6% drop in urethral function between 36 weeks of gestation and 8 weeks after delivery.[93] Therefore, there is evidence both for a change in sphincter function and for urethral support.

The explanation for why normal birth only represents a 6% to 7% change while women with de novo stress incontinence have a 25% lower maximum urethral closure pressure is likely explained by wide variation in urethral function among different women, even in the absence of childbirth (Figure 15[92]). Plausibly, women with weak urethras

TABLE 3
Relative contributions of maximal urethral closure pressure and urethral support to the cause of SUI as expressed as effect sizes comparing women with stress incontinence to asymptomatic women of similar age, race, and parity in the postpartum period vs middle age[91,92]

	Effect size		
Variable	Maximum urethral closure pressure	Urethral support	Levator injury
Primiparous younger women (9 mo after delivery)	0.9 (25% lower in the SUI group)	0.8 (vesical neck movement 31% greater in the SUI group)	29% in the SUI group vs 12% in the control group
Older women (mean age of 47 y after delivery)	1.5 (42% lower in the SUI group)	0.6	38% in the SUI group vs 32% in the control group

SUI, stress urinary incontinence.

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(lower closure pressure) before pregnancy may be more likely to develop incontinence if their support is damaged, so incontinence might continue in the postpartum period. Therefore, the major initial factor associated with vaginal birth is a change in urethral support. It should be noted that, later in life, after urethral function has declined by 15% per decade (Figure 15[92]), urethral failure becomes the dominant causal factor in women seeking care for incontinence.[91]

In addition, it is likely that when 1 pelvic floor structure is injured, other adjacent structures may be injured or affected as well.[79,94–100] Overall, women with damaged levator muscles have a 24% lower urethral closure pressure during a maximal pelvic muscle contraction than women without damage (65.9 vs 86.8 cm H_2O; P=.004).[101] However, levator injury does not necessarily affect urethral sphincter contraction in all women. Reduced closure function is present in some women after levator ani muscle injury, but not in all—suggesting a field effect where injury in 1 area (eg, the levator ani) makes it more likely that there will be an injury to adjacent structures (eg, the urethra).[102] The exact combination of these factors that eventually explains incontinence remains unknown. Therefore, changes in urethral support because of pelvic floor injury in women with weak urethras are the primary factors explaining de novo stress incontinence after vaginal birth.

Interventions and rehabilitation

Although it will be possible to reduce the occurrence of pelvic floor injury, it will not be possible to eliminate it. To date, limited experience with surgical repair of the muscle is not sufficient to establish its effectiveness.[103] Help for women recovering from birth is essential to fulfill our responsibility to aid the injured. Postpartum perineal clinics ("Healthy Healing After Delivery") have emerged to fill the gap between hospital discharge and initial obstetrical follow-up.[104,105] These collaborative multidisciplinary care centers, which include urogynecologists, nurses, physical therapists, and other advanced practitioners, provide early individualized assessment, education, and intervention for pelvic floor symptoms in pregnant and postpartum women.[106–108] Women are seen from as early as 1 week and up to 1 year after delivery,[106,108] with a median initial visit occurring 24 days after delivery.[107] This timeframe is consistent with the American College of Obstetricians and Gynecologists recommendation that all women should have contact with maternal care providers within the first 3 weeks after delivery.[109] Postpartum clinics not only provide an opportunity for early pelvic floor and mental health screening,[107] intervention, and prevention of long-term health issues[106] but also help to offload obstetrical practices[106] and provide referrals for needed rehabilitation services, such as physical therapy (15.8% referral rates).[94,106,108] It should be emphasized that the principles that underlie this care can be performed by any practice providing obstetrical care by (1) recognizing women with difficult deliveries, (2) assessing them early on, (3) working with pelvic floor physical therapists to help with muscle training, (4) decreasing levator spasm when present, and (5) providing educational materials.

The suggested timeframe for referral to physical therapy is 2 to 6 weeks after delivery.[109,110] Women who report definite symptoms (ie, pain, incontinence, or pelvic pressure) or who sustain a major pelvic floor injury should be given referral priority, as they would maximally benefit from early guided rehabilitation to address tissue- and activity-

FIGURE 15
Relationship between age and maximal urethral closure pressure in nulliparas[92]

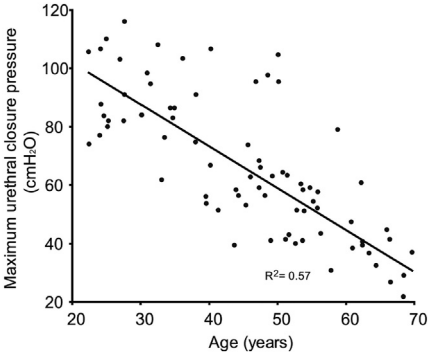

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TABLE 4
Prenatal discussion points for the risk of pelvic floor injury and subsequent development of symptomatic pelvic floor disorders

Discussion points
• Awareness of the fact that some pelvic floor injuries can be prevented but that the risk cannot be entirely eliminated • Potential risks of operative deliveries on pelvic floor injury • Educate on pelvic floor–related changes and symptoms during prenatal visits • Screen for PFD during pregnancy (especially in the third trimester of pregnancy) • Educate on potentially beneficial interventions: ■ Pelvic floor muscle training (grade A evidence)[94–96] ■ Avoidance of smoking (grade A evidence)[79,96] ■ Avoidance of constipation (grade B evidence)[79,96] ■ Maintaining normal BMI (grade A evidence)[79,96]
For women with significantly increased risk of PFD
Elective cesarean delivery[a,79,97]
Support materials
Educational videos and pamphlets for pregnant women (see References for examples)[98–100]

BMI, body mass index; *PFD*, pelvic floor disorder.

[a] Always in consideration of the benefits balanced with the risks of repeated cesarean deliveries, particularly with complications of placenta previa and placenta accreta.

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level impairments. To compare birth injury to a common orthopedic injury, there are approximately 200,000 anterior cruciate ligament (ACL) injuries per year in the United States alone.[111] Birth injuries are similar to ACL injuries in that their presentation is highly variable; thus, management must be personalized to consider patient preferences, injury severity, and long-term prognosis.[112] Considering the annual vaginal birth rate of 3 million and a levator ani injury rate of 19%,[10] 570,000 women sustain a birth injury per year. Therefore, birth injury affects almost 3 times the number of people sustaining ACL injuries. Even though physical therapy is part of the standard of care management for an ACL injury,[113] far fewer women are receiving care for injuries resulting from birth—injuries that, when left untreated, can lead to short- and long-term disabilities. Physical therapists are well positioned to serve as care team members to assist in pain management, functional restoration, and disability prevention in this patient population.[114]

Improving prenatal education about pelvic floor disorder

Until recently, the extent of morbidity after childbirth has gone unrecognized among both parturients and healthcare providers, and there has been a paucity of evidence supporting the effectiveness of our care. Population studies have demonstrated a lack of knowledge among pregnant women and a need for education programs to fill this gap.[115–118] Women's reluctance to seek help for pelvic floor conditions stems from the feeling that those are part of the process of childbearing and from low awareness, poor knowledge of PFDs, embarrassment, or feeling that they should not trouble health professionals.[119,120] Thus, it is important to educate women on the implications of vaginal delivery on the pelvic floor to empower and improve their ability to make informed decisions regarding their perinatal and postpartum care.

PFD prevention should be discussed both as an ethical obligation and from a legal standpoint with every pregnant woman as part of routine antenatal care, even though most women will have a childbirth experience with minimal or reversible pelvic floor damage (Table 4).[97] Identifying those most susceptible to birth injury allows practitioners to reassure parturients who will unlikely experience any harm if opting for vaginal delivery and, simultaneously, warn those who are at higher risk of medium- to long-term pelvic floor damage.[79]

Furthermore, scoring systems are being developed to help provide women with evidence-based prelabor advice in an attempt to avoid unnecessarily high incidences of PFDs requiring future surgeries.[79,121] When validated and used consistently, scoring systems may empower more women to approach the labor process assured of minimizing the chance of long-term consequences on their pelvic floor.

Research needs

The research needs are as follows:

1. Intervention trials to assess the efficacy of prevention strategies are needed to reduce injuries.
2. Mechanistic and epidemiologic studies are needed to assess the risk factors for perineal body and perineal membrane injuries during vaginal birth.
3. The cellular and molecular mechanisms underlying changes in viscoelasticity ("softening" or "stretchiness") of the distal birth canal tissues need to be identified so that interventions can address inadequate ripening of these tissues when needed.

4. A rapid and reliable screening strategy is needed to identify the 10% to 15% of women at highest risk of pelvic floor injury during vaginal birth so that interventions can be targeted only where they are needed.

Conclusions

Birth-related injuries to the levator ani causing an enlarged urogenital hiatus are the best-studied cause of pelvic organ prolapse, but injury to the perineal body, perineal membrane, and associated fascial tissues are also likely to be important. To date, surgical repair of levator injury is not a generally accepted intervention. Overstretching and tearing are the mechanisms of levator ani muscle injury. Forceps and occiput posterior delivery, along with advanced maternal age, are the largest risk factors. Women should be coached to relax their pelvic floor muscles during pushing—not only to speed delivery by the muscles being "more stretchy" but also to minimize the risk of avulsion because of the viscoelastic properties of the muscles. Contracting the muscle as it is stretched by the descending head during a push places increased tension on the muscle origin, thereby increasing the risk of injury. Better information is needed for expectant mothers on their risk of pelvic floor injuries and the implications of these injuries on lifetime pelvic floor function. ■

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Appendix

SUPPLEMENTAL TABLE

Interventions that have shown to be ineffective in the prevention of levator ani injury or urinary incontinence in the postpartum period

Potential risk-reducing actions	Hypothesized mechanism	Evidence for altered risk
Pelvic floor muscle training for preventing injury or incontinence	Exercised muscles would be less prone to injury.	Pelvic floor muscle training did not reduce the risk of levator avulsion or urinary incontinence in the postpartum period.[1,2]
EPI-NO	Predilating the birth canal tissues during pregnancy reduces the risk of pelvic floor injury.	Levator injury, hiatal enlargement, and sphincter injury were not different in EPI-NO users compared with controls.[3]
Position of pushing	Reduced tension on perineal tissues.	Upright position has been associated with less stress incontinence but not pelvic floor trauma.[4,5]
Pushing pattern	Reducing the number of pushing cycles decreases the risk of levator injury.	The risk of postpartum urinary or fecal incontinence is not different between the intervention group (pushing only twice during each contraction and resting regularly for 1 contraction in 5 without pushing) and controls (pushing 3 times during each contraction with no contraction without pushing).[6]
Immediate vs delayed pushing	Mechanism is unclear.	In nulliparous women at term with epidural anesthesia, there is no difference in levator ani injury between delayed and immediate pushing.[7]
Manual perineal support	Slows the delivery and minimizes distension dimensions.	There is insufficient evidence that manual perineal support (hands on or hands off) reduced pelvic floor trauma.[8]
Water birth	Mechanism is unclear.	In low-risk women, water birth has an increased risk of obstetrical anal sphincter injury.[9]

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