

Cesarean Delivery Is Independently Associated with Postpartum Pelvic Floor Hypertonicity: Mirror-Image Risk Profiles of Hypertonic and Hypotonic Phenotypes in 1,796 Women

JiaPing Lu

Jinhua People's Hospital

Heng Xu

xheng0248@sina.com

Jinhua People's Hospital

Qiulian Xu

Jinhua People's Hospital

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Abstract

Background

Postpartum pelvic floor research has focused almost exclusively on weakness (hypotonic phenotype). Elevated resting activity (hypertonicity) is linked to pelvic pain and dyspareunia, yet its obstetric correlates remain unstudied.

Objective

To identify independent factors associated with early postpartum pelvic floor hypertonicity measured by surface electromyography (sEMG), and to compare its risk profile with that of the hypotonic phenotype.

Methods

In this retrospective cross-sectional study of 1,796 women (median 49 days postpartum), hypertonicity was defined as resting baseline amplitude $> 10 \mu\text{V}$. We built sequential logistic regression models (demographic $\rightarrow$ obstetric $\rightarrow$ symptomatic) and performed subgroup, interaction, and sensitivity analyses. A parallel model used hypotonicity (fast-twitch $< 20 \mu\text{V}$) for contrast.

Results

Hypertonicity was present in 197 women (11.0%). Cesarean delivery was the dominant independent correlate (adjusted OR 6.86, 95% CI 3.40–13.86), followed by younger age (OR 0.91 per year) and dyspareunia (OR 2.55). The association with cesarean was robust across all sensitivity analyses (OR range 6.7–8.7) with no interaction by age or parity. The hypertonic and hypotonic phenotypes displayed mirror-image risk profiles: cesarean protected against hypotonicity (OR 0.20), and age effects ran in opposite directions; only 0.7% exhibited both phenotypes.

Conclusions

Postpartum pelvic floor hypertonicity is a distinct phenotype strongly associated with cesarean delivery and dyspareunia, with a risk profile mirroring that of weakness. Postpartum screening should assess both strength and resting tone to guide phenotype-specific rehabilitation.

Introduction

Pelvic floor disorders affect up to half of parous women and represent a major determinant of long-term quality of life (1, 2). Vaginal childbirth is the strongest established risk factor for pelvic floor weakness,

acting through stretch injury and avulsion of the levator ani, pudendal nerve damage, and perineal trauma (3–5); conversely, cesarean delivery exerts a partially protective effect against stress urinary incontinence and pelvic organ prolapse (2, 6, 7). This literature, however, is almost entirely built on a single conceptual axis, that of the hypotonic pelvic floor: weak muscles, prolapse, and incontinence.

A second, largely neglected phenotype exists at the opposite end of the tonus spectrum. Pelvic floor hypertonicity, defined as an inability to relax the pelvic floor muscles and reflected electrophysiologically by elevated resting sEMG amplitude, is a recognized substrate of chronic pelvic pain, dyspareunia, and dysfunctional voiding in non-pregnant populations (8–10). In postpartum women, hypertonicity has been linked to dyspareunia and perineal pain in small clinical series (11, 12), yet no large study has systematically examined which obstetric and demographic factors predispose women to an elevated resting tone after delivery, or how this risk profile relates to the classical weakness phenotype.

The Glazer protocol provides a standardized, objective, five-phase surface electromyographic assessment of pelvic floor muscle function (pre-rest baseline, rapid [fast-twitch] contractions, sustained [slow-twitch] contractions, endurance, and post-rest baseline) and is widely used in Chinese postpartum rehabilitation programs (13, 14). Its resting phases offer a quantitative window into hypertonicity that does not depend on subjective palpation.

We therefore analyzed 1,796 women undergoing routine early postpartum sEMG evaluation to (1) identify independent obstetric and demographic correlates of pelvic floor hypertonicity, (2) test the robustness of these associations across prespecified sensitivity and subgroup analyses, and (3) directly contrast the risk factor architecture of hypertonic versus hypotonic phenotypes. We hypothesized that hypertonicity would constitute a distinct phenotype with a risk profile that differs qualitatively from that of pelvic floor weakness.

Materials and Methods

Study design and population

This was a single-center retrospective cross-sectional study of women who delivered at Jinhua People's Hospital, between January 2023 and June 2026 and attended the postpartum pelvic floor rehabilitation clinic for a standardized sEMG assessment before any pelvic floor intervention. Of 1,810 women with a baseline assessment, 14 were excluded because of missing or invalid delivery-mode coding, leaving 1,796 women in the analytic sample (Fig. 1). The study protocol was approved by the institutional ethics committee; the requirement for individual informed consent was waived owing to the retrospective design.

Pelvic floor sEMG assessment

All women underwent pelvic floor sEMG using the Glazer protocol (13) with a vaginal surface electrode, using the Medlander MLD A2 (Deluxe) sEMG biofeedback system (Nanjing Medlander Medical Technology Co., Ltd., Jiangsu, China). Five phases were recorded: pre-rest baseline (60 s), five rapid contractions (fast-twitch maximum amplitude), five sustained 10-s contractions (mean amplitude), a 60-s endurance contraction (mean amplitude), and post-rest baseline (60 s). A composite Glazer score was computed by the device software; physiologically implausible scores (> 200) were treated as data-entry errors and set to missing. Assessments were performed a median of 49 days (interquartile range [IQR] 43–62) after delivery, before any pelvic floor muscle training, electrical stimulation, or magnetic stimulation.

Outcome definition

The primary outcome was pelvic floor hypertonicity, defined as a pre-rest or post-rest baseline amplitude $> 10 \mu\text{V}$, consistent with thresholds used in prior sEMG studies (8, 13) and with the measurement framework for pelvic floor muscle overactivity summarized in a recent systematic review (19); the threshold was further corroborated by the present data, in which the 90th percentile of the maximal resting amplitude was $10.32 \mu\text{V}$ (Fig. 2). Prespecified alternative definitions ($> 8 \mu\text{V}$, $> 12 \mu\text{V}$, and the empirical 90th percentile) were examined in sensitivity analyses. For the comparative phenotype analysis, hypotonicity was defined as a fast-twitch maximum amplitude $< 20 \mu\text{V}$, approximating the lower reference range of the Glazer protocol (13, 14).

Covariates

Obstetric variables were extracted from the structured delivery record: delivery mode (vaginal vs cesarean), perineal status (intact/abrasion, first-degree tear, episiotomy, second-degree tear, or third-degree tear; ordinal grade 0–3), forceps assistance, neonatal birth weight (grams; macrosomia $\geq 4,000$ g), gestational age at delivery, second-stage duration (parsed from HHMM fields; applicable to vaginal births only), and estimated blood loss (postpartum hemorrhage > 500 mL). Demographic variables included maternal age, body mass index (BMI), parity (nulliparous vs multiparous), and the interval from delivery to testing. Symptoms and comorbidities were abstracted from the clinic's diagnostic coding field (prolapse, stress urinary incontinence [SUI], urge incontinence, dyspareunia, constipation) and the admission record (pain, gestational diabetes, hypertensive disorders of pregnancy, premature rupture of membranes). Birth weight was recorded in grams despite a kilogram column label; values were re-parsed accordingly, and for twin deliveries the larger twin was used.

Statistical analysis

Continuous variables were summarized as medians (IQR) and compared with the Mann–Whitney U test; categorical variables were compared with the chi-square or Fisher exact test. Univariable logistic regression screened candidate predictors; multivariable models were built sequentially: Model 1

(demographic: age, BMI, parity, interval from delivery to testing), Model 2 (Model 1 + obstetric factors: delivery mode, perineal injury grade, macrosomia), and Model 3 (Model 2 + symptoms: pain, dyspareunia). Second-stage duration was analyzed only within the vaginal-delivery subgroup, because cesarean-delivered women have no second-stage exposure and including it would have systematically excluded the entire cesarean group from the model. Multicollinearity was assessed with variance inflation factors (VIF), discrimination with the area under the receiver operating characteristic curve (AUC), and calibration with the Hosmer–Lemeshow test and calibration plots. Prespecified subgroups included delivery mode, parity, maternal age (< 35 vs ≥ 35 years), and pain status; interactions were tested with product terms. Sensitivity analyses varied the hypertonicity threshold, restricted the testing window (≤ 90 and ≤ 60 days postpartum), excluded women reporting pain, restricted the sample to nulliparous women, and refitted the model among women with complete diagnostic coding to assess detection bias in symptom ascertainment. Because no hypertonic events occurred among the 41 forceps deliveries (complete separation), this association was quantified with the Fisher exact test and a Haldane–Anscombe-corrected odds ratio rather than maximum-likelihood regression. For the comparative phenotype analysis, the full multivariable model was refitted with the hypotonic outcome. Two-sided $p < 0.05$ was considered statistically significant. Analyses were performed in Python 3.12 (statsmodels 0.14, scikit-learn).

Results

Cohort characteristics and prevalence

The analytic sample comprised 1,796 women (median age 30 years, IQR 28–33; median BMI 22.5 kg/m², IQR 20.8–24.6; 527 [29.3%] cesarean deliveries; median testing interval 49 days). Hypertonicity was identified in 197 women (11.0%). Compared with non-hypertonic women, hypertonic women were younger (median 30 vs 31 years, $p = 0.006$), more often nulliparous, more often delivered by cesarean (68.0% vs 24.6%, $p < 0.001$), and had less perineal trauma of grade $\geq$ II (15.9% vs 37.4%, $p < 0.001$) (Table 1). None of the 41 forceps-assisted births occurred in hypertonic women ($p = 0.011$). Hypertonic women more frequently reported pain (10.7% vs 6.1%) and dyspareunia (12.7% vs 5.3%), but less frequently SUI (41.1% vs 52.1%) and prolapse (8.1% vs 17.2%) (all $p < 0.01$).

Table 1
Baseline characteristics by hypertonicity status

Variable	Full cohort (n = 1,796)	Hypertonic (n = 197)	Non-hypertonic (n = 1,599)	p value
Age, years	30.0 (28.0–33.0)	30.0 (28.0–32.0)	31.0 (28.0–33.0)	0.006
BMI, kg/m ²	22.5 (20.8–24.6)	22.9 (21.0–25.0)	22.4 (20.8–24.5)	0.019
Parity	1.0 (1.0–2.0)	1.0 (1.0–1.0)	1.0 (1.0–2.0)	0.004
Neonatal birth weight, g	3300.0 (3050.0–3600.0)	3350.0 (3100.0–3600.0)	3300.0 (3050.0–3600.0)	0.227
Gestational age at delivery, weeks	39.4 (38.9–40.1)	39.3 (38.9–40.0)	39.4 (38.9–40.1)	0.161
Interval from delivery to testing, days	49.0 (43.0–62.0)	51.0 (44.0–65.0)	49.0 (43.0–61.5)	0.138
Second stage of labor, min	51.0 (26.8–88.2)	65.0 (24.2–89.8)	51.0 (27.0–88.0)	0.733
Cesarean delivery	527 (29.3%)	134 (68.0%)	393 (24.6%)	< 0.001
Forceps-assisted delivery	41 (2.4%)	0 (0.0%)	41 (2.7%)	0.011
Perineal injury ≥ grade II	624 (35.0%)	31 (15.9%)	593 (37.4%)	< 0.001
Macrosomia (≥ 4,000 g)	110 (6.2%)	12 (6.2%)	98 (6.2%)	1.000
Postpartum hemorrhage (> 500 mL)	37 (3.2%)	2 (3.4%)	35 (3.2%)	0.709
GDM	211 (11.7%)	25 (12.7%)	186 (11.6%)	0.640
Hypertensive disorders of pregnancy	46 (2.6%)	7 (3.6%)	39 (2.4%)	0.338
Premature rupture of membranes	291 (16.2%)	32 (16.2%)	259 (16.2%)	1.000
Pain	118 (6.6%)	21 (10.7%)	97 (6.1%)	0.001
Dyspareunia	109 (6.1%)	25 (12.7%)	84 (5.3%)	< 0.001
Stress urinary incontinence	914 (50.9%)	81 (41.1%)	833 (52.1%)	0.004
Pelvic organ prolapse	291 (16.2%)	16 (8.1%)	275 (17.2%)	< 0.001

sEMG characteristics

The hypertonic phenotype was not merely an isolated elevation of resting tone: all sEMG phases were shifted upward (Table 2). Median pre-rest (11.4 vs 3.7 μ V) and post-rest (10.4 vs 3.1 μ V) amplitudes were roughly threefold higher, while fast-twitch (42.6 vs 28.8 μ V), sustained-contraction (28.9 vs 18.2 μ V), and endurance (24.7 vs 15.1 μ V) amplitudes were also substantially greater (all $p < 0.001$). Hypertonic women thus exhibited higher, not lower, contractile-phase amplitudes than non-hypertonic women, although sEMG amplitude does not equate to muscle strength and may partly reflect differences in motor unit recruitment strategy.

Table 2
Five-phase sEMG measures by hypertonicity status

sEMG measure	Hypertonic (n = 197)	Non-hypertonic (n = 1,599)	p value
Pre-rest baseline, μ V	11.4 (10.5–13.6)	3.7 (2.1–5.7)	< 0.001
Fast-twitch maximum, μ V	42.6 (31.9–52.2)	28.8 (19.5–39.3)	< 0.001
Sustained contraction mean, μ V	28.9 (22.3–35.0)	18.2 (11.8–26.1)	< 0.001
Endurance mean, μ V	24.7 (19.5–31.2)	15.1 (10.0–21.6)	< 0.001
Post-rest baseline, μ V	10.4 (8.0–12.2)	3.1 (1.6–5.1)	< 0.001
Glazer total score	67.4 (60.6–71.9)	61.1 (48.9–71.9)	< 0.001

Independent correlates of hypertonicity

In univariable analyses (Supplementary Table S1), cesarean delivery (OR 6.53, 95% CI 4.74–8.99), younger age, higher BMI, nulliparity, lower perineal injury grade, pain, and dyspareunia were associated with hypertonicity. In the fully adjusted obstetric model (Model 2; Table 3, Fig. 3), cesarean delivery remained the dominant correlate (adjusted OR 6.86, 95% CI 3.40–13.86, $p < 0.001$), together with younger age (OR 0.91 per year, 95% CI 0.87–0.95, $p < 0.001$); perineal injury grade and macrosomia were no longer significant after adjustment for delivery mode. Adding symptoms (Model 3) revealed dyspareunia as an independent symptomatic correlate (OR 2.55, 95% CI 1.36–4.77, $p = 0.003$), whereas pain was attenuated (OR 1.09, 95% CI 0.64–1.88). Model discrimination was good (Model 2 AUC 0.768; Model 3 AUC 0.776) with excellent calibration (Hosmer–Lemeshow $\chi^2=3.80$, $p = 0.875$; Fig. 7) and no relevant multicollinearity (all VIF < 3).

Table 3
Multivariable logistic regression models for hypertonicity

Model	Variable	Adjusted OR	95% CI	p value
Model 1 (Demographic)	Age	0.95	0.91–1.00	0.030
Model 1 (Demographic)	BMI	1.05	1.01–1.09	0.016
Model 1 (Demographic)	Multiparity	0.68	0.47–0.98	0.040
Model 1 (Demographic)	Testing interval, days	1.0	1.00–1.01	0.155
Model 2 (+ Obstetric factors)	Age	0.91	0.87–0.95	< 0.001
Model 2 (+ Obstetric factors)	BMI	1.04	0.99–1.09	0.095
Model 2 (+ Obstetric factors)	Multiparity	0.83	0.56–1.22	0.339
Model 2 (+ Obstetric factors)	Interval from delivery to testing, days	1.0	1.00–1.01	0.222
Model 2 (+ Obstetric factors)	Cesarean delivery	6.86	3.40–13.86	< 0.001
Model 2 (+ Obstetric factors)	Perineal injury grade	0.92	0.60–1.43	0.719
Model 2 (+ Obstetric factors)	Macrosomia	0.61	0.31–1.20	0.155
Model 3 (+ Symptoms)	Age	0.91	0.87–0.95	< 0.001
Model 3 (+ Symptoms)	BMI	1.04	1.00–1.09	0.076
Model 3 (+ Symptoms)	Multiparity	0.81	0.55–1.19	0.283
Model 3 (+ Symptoms)	Interval from delivery to testing, days	1.0	1.00–1.01	0.258
Model 3 (+ Symptoms)	Cesarean delivery	6.66	3.29–13.48	< 0.001
Model 3 (+ Symptoms)	Perineal injury grade	0.90	0.58–1.40	0.654
Model 3 (+ Symptoms)	Macrosomia	0.62	0.32–1.23	0.171
Model 3 (+ Symptoms)	Pain	1.09	0.64–1.88	0.742
Model 3 (+ Symptoms)	Dyspareunia	2.55	1.36–4.77	0.003

Subgroup, interaction, and sensitivity analyses

The cesarean–hypertonicity association was consistent across all prespecified subgroups (Supplementary Table S2; Fig. 4): nulliparous (OR 7.56, 95% CI 5.15–11.08) and multiparous women (OR 4.66, 95% CI 2.54–8.53), women aged < 35 (OR 6.92) and ≥ 35 years (OR 8.38), and women with (OR 5.19) and without pain (OR 6.58). No significant interaction was detected for cesarean delivery with age ($p = 0.656$) or parity ($p = 0.228$). Within vaginal deliveries, second-stage duration > 60 min (OR 1.45, 95% CI 0.87–2.43) and perineal injury grade (OR 0.89, 95% CI 0.59–1.35) were not independently associated with hypertonicity. Sensitivity analyses confirmed robustness: adjusted ORs for cesarean delivery ranged from 6.71 to 8.65 across alternative thresholds (> 8, > 12 μ V, empirical P90), restricted testing windows, exclusion of women with pain, and restriction to nulliparous women (Table S3). To address potential detection bias in symptom coding, the model was refitted among the 1,692 women with complete diagnostic records; the associations were unchanged or stronger (cesarean OR 6.28, 95% CI 3.08–12.83; dyspareunia OR 2.67, 95% CI 1.42–5.03).

Mirror-image risk profiles of hypertonic and hypotonic phenotypes

Four phenotypes were almost mutually exclusive: 1,189 women (66.2%) were normal, 410 (22.8%) purely hypotonic, 185 (10.3%) purely hypertonic, and only 12 (0.7%) displayed both. Refitting the full model with hypotonicity as the outcome produced a risk profile that was the mirror image of the hypertonic profile (Table 4, Fig. 5): cesarean delivery was strongly protective against weakness (OR 0.20, 95% CI 0.13–0.31) while strongly predisposing to hypertonicity (OR 6.86); older age increased the risk of weakness (OR 1.06 per year) but protected against hypertonicity (OR 0.91); higher BMI showed opposite-direction trends. Cesarean-delivery rates across the four groups followed the same geometry (normal 29.3%, hypotonic 11.0%, hypertonic 69.2%). Symptom spectra were likewise segregated (Table 5, Fig. 6): pure hypertonicity carried the highest rates of pain (13.5%) and dyspareunia (13.0%) but the lowest rates of prolapse (7.0%) and SUI (41.6%), whereas the hypotonic group showed the reverse pattern.

Table 4
Mirror-image risk profiles: parallel multivariable models

Outcome phenotype	Exposure	OR	95% CI	p value
Hypertonic	Age	0.91	0.87–0.95	< 0.001
Hypertonic	BMI	1.04	0.99–1.09	0.095
Hypertonic	Multiparity	0.83	0.56–1.22	0.339
Hypertonic	Interval from delivery to testing, days	1.0	1.00–1.01	0.222
Hypertonic	Cesarean delivery	6.86	3.40–13.86	< 0.001
Hypertonic	Perineal injury grade	0.92	0.60–1.43	0.719
Hypertonic	Macrosomia	0.61	0.31–1.20	0.155
Hypotonic	Age	1.06	1.03–1.09	< 0.001
Hypotonic	BMI	0.94	0.91–0.98	0.003
Hypotonic	Multiparity	0.78	0.60–1.02	0.070
Hypotonic	Testing interval, days	0.99	0.99–1.00	0.050
Hypotonic	Cesarean delivery	0.20	0.13–0.31	< 0.001
Hypotonic	Perineal injury grade	0.86	0.70–1.06	0.158
Hypotonic	Macrosomia	1.3	0.79–2.14	0.295

Table 5
Symptom spectrum across the four phenotypes

Symptom	Normal	Purely hypotonic	Purely hypertonic	Combined	p value
Pain	6.6%	5.9%	13.5%	0.0%	0.021
Dyspareunia	5.6%	4.4%	13.0%	8.3%	< 0.001
Stress urinary incontinence	52.4%	51.2%	41.6%	33.3%	0.030
Pelvic organ prolapse	16.1%	20.5%	7.0%	25.0%	< 0.001

Discussion

Principal findings

In this large single-center cohort with objective five-phase sEMG phenotyping, early postpartum pelvic floor hypertonicity affected one in nine women and was independently associated with cesarean delivery (adjusted OR ≈ 7), a finding that was robust across threshold definitions, testing windows, and subgroups. The hypertonic phenotype was electrophysiologically distinct, with globally elevated sEMG amplitudes including the contractile phases, and carried a symptomatic signature of pain and dyspareunia rather than incontinence and prolapse. The risk factor architecture of hypertonicity was a near-mirror image of that of hypotonicity, and the two phenotypes almost never coexisted in the same woman.

Comparison with previous work

The pelvic floor consequences of childbirth have traditionally been quantified on the weakness axis, namely levator avulsion, reduced contractility, incontinence, and prolapse, for which vaginal (particularly instrumental) delivery is the dominant risk factor and cesarean delivery partially protective (2–7, 15). Our hypotonic-side results replicate this canonical pattern almost exactly (cesarean OR 0.20), which strengthens confidence in the measurement platform. The hypertonic side of the tonus spectrum has been characterized mainly in chronic pelvic pain populations (8–10, 16, 19) and in small postpartum dyspareunia series (11, 12). The most direct predecessor of the present work is the cross-sectional study by Gao et al. (18), who analyzed pelvic floor sEMG in 845 postpartum women and reported that cesarean delivery was associated with higher rates of abnormal resting muscle tone, including the post-rest stage, alongside a reduced risk of urinary incontinence. Our findings corroborate this association in a cohort more than twice as large and extend it in three ways: we validate the resting-tone threshold against the empirical distribution of the data ($P_{90} = 10.32 \mu V$ vs the prespecified $10 \mu V$), we quantify the association with multivariable adjustment and extensive sensitivity analyses (adjusted OR 6.86; robust range 6.3–8.7), and we add two elements absent from prior work: formal phenotype modeling demonstrating near-mutual exclusivity of the hypertonic and hypotonic states, and a direct mirror-image comparison of their risk factor architectures.

Possible mechanisms

Several non-exclusive mechanisms may explain the cesarean–hypertonicity association. First, absence of the vaginal passage may deprive the pelvic floor of the physiological stretch–relaxation cycle of the second stage, leaving postpartum tone unmodulated. Second, surgical stress, peritoneal irritation, and postoperative pain may drive reflex guarding and sustained elevator activation, an established pathway in abdominal and pelvic surgery-related myofascial pain (9, 16). Third, confounding by indication deserves detailed consideration. Indications cluster into two groups with opposing implications: obstetric indications (placenta previa, scarred uterus, breech presentation, macrosomia) typically lead to prelabor cesarean, in which case the observed association would reflect the absence of vaginal passage rather than the indication itself; psychosocial pathways (maternal request or “social factor” cesarean, fear of childbirth, antenatal anxiety) could plausibly elevate pelvic floor tone directly and inflate the association. The replication of a directionally consistent association in an independent Chinese cohort with a different case mix (18) argues against an explanation purely by indication, but cannot exclude it.

The inverse observation, zero hypertonic events among 41 forceps deliveries, is consistent with the traumatic-stretch model: severe birth-canal trauma directs the pelvic floor toward denervation weakness and low tone, as documented for levator avulsion after instrumental delivery (4, 15).

Clinical implications

If hypertonicity is as strongly patterned by delivery mode as weakness is, but in the opposite direction, then postpartum pelvic floor assessment should be explicitly bidirectional. A woman screened only for “low strength” after cesarean delivery will be declared normal while carrying a substantially elevated risk of a hypertonic, pain-prone phenotype. The phenotype segregation also has direct rehabilitative implications: strengthening-oriented training (the default postpartum program) is mechanistically appropriate for the hypotonic majority but potentially counterproductive for the hypertonic minority, for whom relaxation, downtraining, and myofascial approaches are indicated (8, 11, 16). The near-mutual-exclusivity of the two phenotypes means that simple phase-based rules derived from the routine Glazer assessment could support phenotype-stratified rehabilitation pathways at the point of care.

Strengths and limitations

Strengths include the large sample, a fully standardized objective sEMG protocol, data-driven validation of the hypertonicity threshold, prespecified sensitivity and subgroup analyses, and the internal replication provided by the hypotonic contrast. Several limitations deserve emphasis. First, the cross-sectional design precludes causal inference, and the interval from delivery to testing (15–196 days) introduces recovery-stage heterogeneity, although results were unchanged when restricted to ≤ 60 days. Second, we could not distinguish elective from intrapartum cesarean deliveries: the cesarean-indication field was populated for only 65 of 527 cesarean-delivered women (12.3%) and the emergency-cesarean field for a single woman, so indication-stratified analysis was not feasible. As detailed above, obstetric and psychosocial indication clusters would bias the association in opposite directions, and residual confounding by indication cannot be excluded. Third, symptoms were abstracted from routine clinic diagnostic coding rather than standardized inventories: PFDI-20 and PISQ-12 were missing in $> 98\%$ of records, and the completeness of symptom coding differed slightly by delivery mode (94.3% after cesarean vs 96.8% after vaginal delivery, $p = 0.021$), raising the possibility of detection bias. Two features argue against a material impact: the direction of differential completeness (lower in the cesarean group) would if anything attenuate the observed associations, and the complete-record sensitivity analysis yielded unchanged or stronger estimates (dyspareunia OR 2.67). Fourth, elevated resting sEMG is an electrophysiological finding, not a clinical diagnosis of pelvic floor myofascial pain, and sEMG amplitudes do not directly equate to muscle strength (19). Fifth, forceps deliveries were few ($n = 41$), limiting precision for that exposure. Finally, this was a single-center Chinese cohort; generalizability requires external validation.

Conclusions

Postpartum pelvic floor hypertonicity is a common, objectively measurable phenotype that is independently associated with cesarean delivery and linked to dyspareunia. Its risk factor profile mirrors that of pelvic floor weakness, and the two phenotypes are nearly mutually exclusive. Bidirectional postpartum screening, assessing both contractile function and resting tone, together with phenotype-specific rehabilitation strategies, may improve postpartum pelvic floor care. Prospective studies with preconception or antenatal baseline measurements are needed to establish causality and to test whether phenotype-stratified rehabilitation improves outcomes.

Declarations

Ethics approval and consent to participate

This study was approved by the institutional ethics committee of Jinhua People's Hospital. Given the retrospective nature of the study and the use of fully de-identified data, the requirement for informed consent was waived. All procedures were conducted in accordance with the Declaration of Helsinki.

Consent for publication

Not applicable. This manuscript does not contain any individual person's data in any form.

Competing interests

The authors declare that they have no competing interests.

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Author Contribution

Lu Jiaping was responsible for data collection, database sorting, preliminary statistical analysis and manuscript drafting. Xu Qiulian participated in clinical data screening, case inclusion and exclusion judgment, and revision of the results section. Xu Heng conceived and designed the research framework, guided the whole statistical analysis process, revised the full manuscript repeatedly, and acted as the corresponding author of this paper. All authors read and approved the final submitted manuscript.

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Availability of data and materials

The datasets analyzed during the current study are not publicly available due to institutional privacy policies but are available from the corresponding author on reasonable request and with appropriate institutional approval.

References

1. Kepenekci I, Keskinilic B, Akinsu F, et al. Prevalence of pelvic floor disorders in the female population and the impact of age, mode of delivery, and parity. *Dis Colon Rectum*. 2011;54(1):85–94.
2. Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S, Norwegian EPINCONT. Study. Urinary incontinence after vaginal delivery or cesarean section. *N Engl J Med*. 2003;348(10):900–7.
3. Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. *Obstet Gynecol*. 2005;106(4):707–12.
4. Memon HU, Blomquist JL, Dietz HP, Pierce CB, Weinstein MM, Handa VL. Comparison of levator ani muscle avulsion injury after forceps and vacuum-assisted vaginal childbirth. *Obstet Gynecol*. 2015;125(5):1080–7.
5. Rogers RG, Leeman LM, Borders N, et al. Contribution of the second stage of labour to pelvic floor dysfunction: a prospective cohort comparison of nulliparous women. *BJOG*. 2014;121(9):1145–54.
6. Handa VL, Harris TA, Ostergard DR. Protecting the pelvic floor: obstetric management to prevent incontinence and pelvic organ prolapse. *Obstet Gynecol*. 1996;88(3):470–8.
7. Memon H, Handa VL. Pelvic floor disorders following vaginal or cesarean delivery. *Curr Opin Obstet Gynecol*. 2012;24(5):349–54.
8. Butrick CW. Pelvic floor hypertonic disorders: identification and management. *Obstet Gynecol Clin North Am*. 2009;36(3):707–22.
9. Butrick CW. Pathophysiology of pelvic floor hypertonic disorders. *Obstet Gynecol Clin North Am*. 2009;36(3):699–705.
10. Ross V, Detterman C, Hallisey A. Myofascial pelvic pain: an overlooked and treatable cause of chronic pelvic pain. *J Midwifery Womens Health*. 2021;66(2):148–60.
11. Dionisi B, Senatori R. Effect of transcutaneous electrical nerve stimulation on the postpartum dyspareunia treatment. *J Obstet Gynaecol Res*. 2011;37(7):750–3.
12. Ram-Weiner M, Hayman-Mendelson A, et al. Postpartum dyspareunia: clinical evaluation, causes, and treatment outcomes. *J Sex Med*. 2023;20(3):324–30.
13. Glazer HI, Hacac CR. The Glazer Protocol: evidence-based medicine pelvic floor muscle (PFM) surface electromyography (SEMG). *Biofeedback*. 2012;40(2):75–9.

14. Glazer HI, Rodke G, Swencionis C, Hertz R, Young AW. Treatment of vulvar vestibulitis syndrome with electromyographic biofeedback of pelvic floor musculature. *J Reprod Med.* 1999;44(4):283–90.
15. Volløyhaug I, Taithongchai A, Van Gruting IMA, Sultan AH, Thakar R. Levator ani muscle morphology and function in women with obstetric anal sphincter injury. *Ultrasound Obstet Gynecol.* 2019;53(3):410–6.
16. Cosgriff L, Ramanathan A, et al. Pelvic floor disorders and sexual function: a review. *Obstet Gynecol Clin North Am.* 2024;51(2):263–77.
17. Barber MD, Walters MD, Bump RC. Short forms of two condition-specific quality-of-life questionnaires for women with pelvic floor disorders (PFDI-20 and PFIQ-7). *Am J Obstet Gynecol.* 2005;193(1):103–13.
18. Gao Q, Wang M, Zhang J, Qing Y, Yang Z, Wang X, et al. Pelvic floor dysfunction in postpartum women: A cross-sectional study. *PLoS ONE.* 2024;19(8):e0308563.
19. Worman RS, Stafford RE, Cowley D, et al. Evidence for increased tone or overactivity of pelvic floor muscles in pelvic health conditions: a systematic review. *Am J Obstet Gynecol.* 2023;228(6):657–74.

Figures

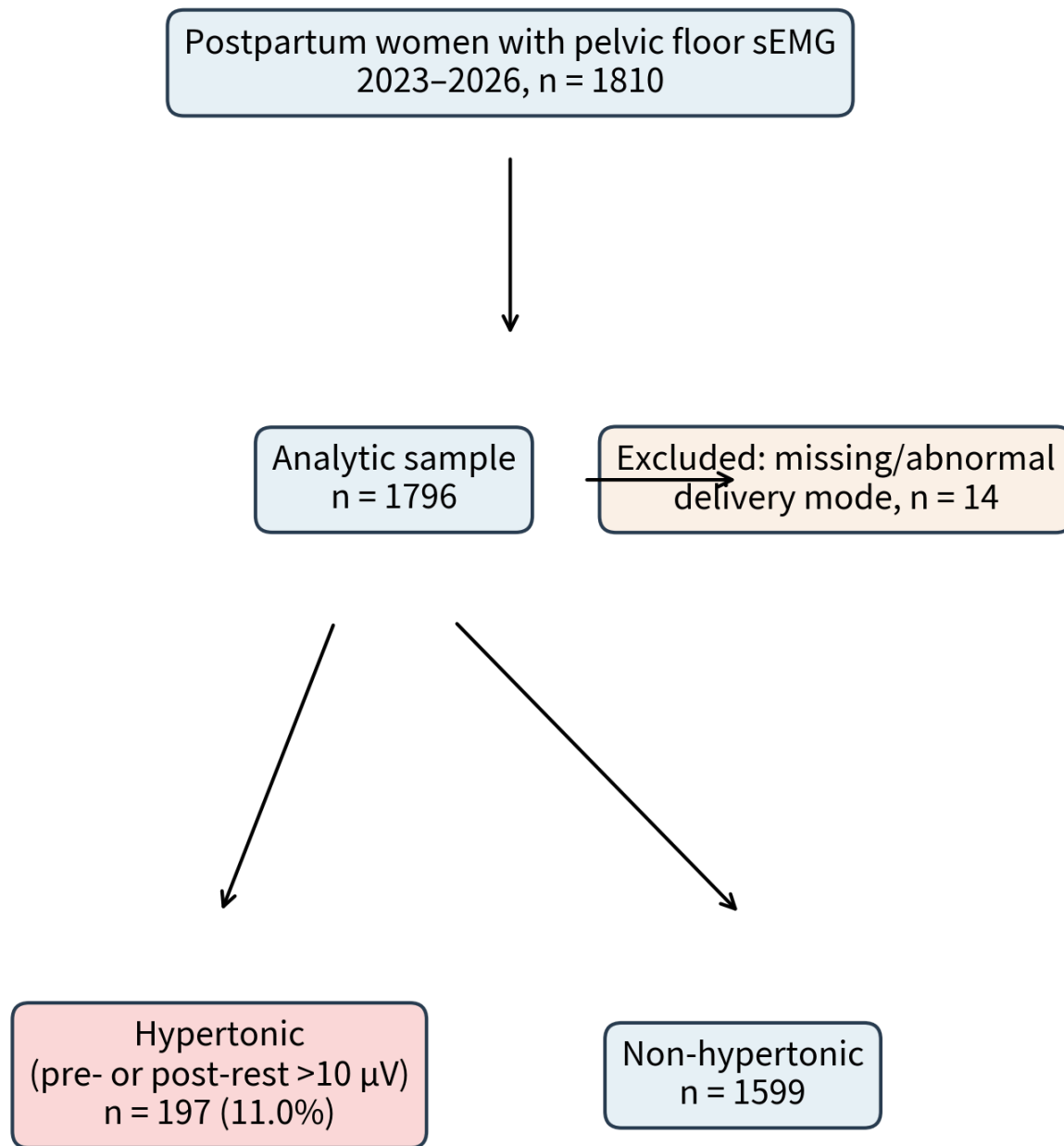


Figure 1

Study flow diagram. Of 1,810 postpartum women assessed with pelvic floor sEMG between 2023 and 2026, 1,796 were analyzed; 197 (11.0%) met the definition of pelvic floor hypertonicity.

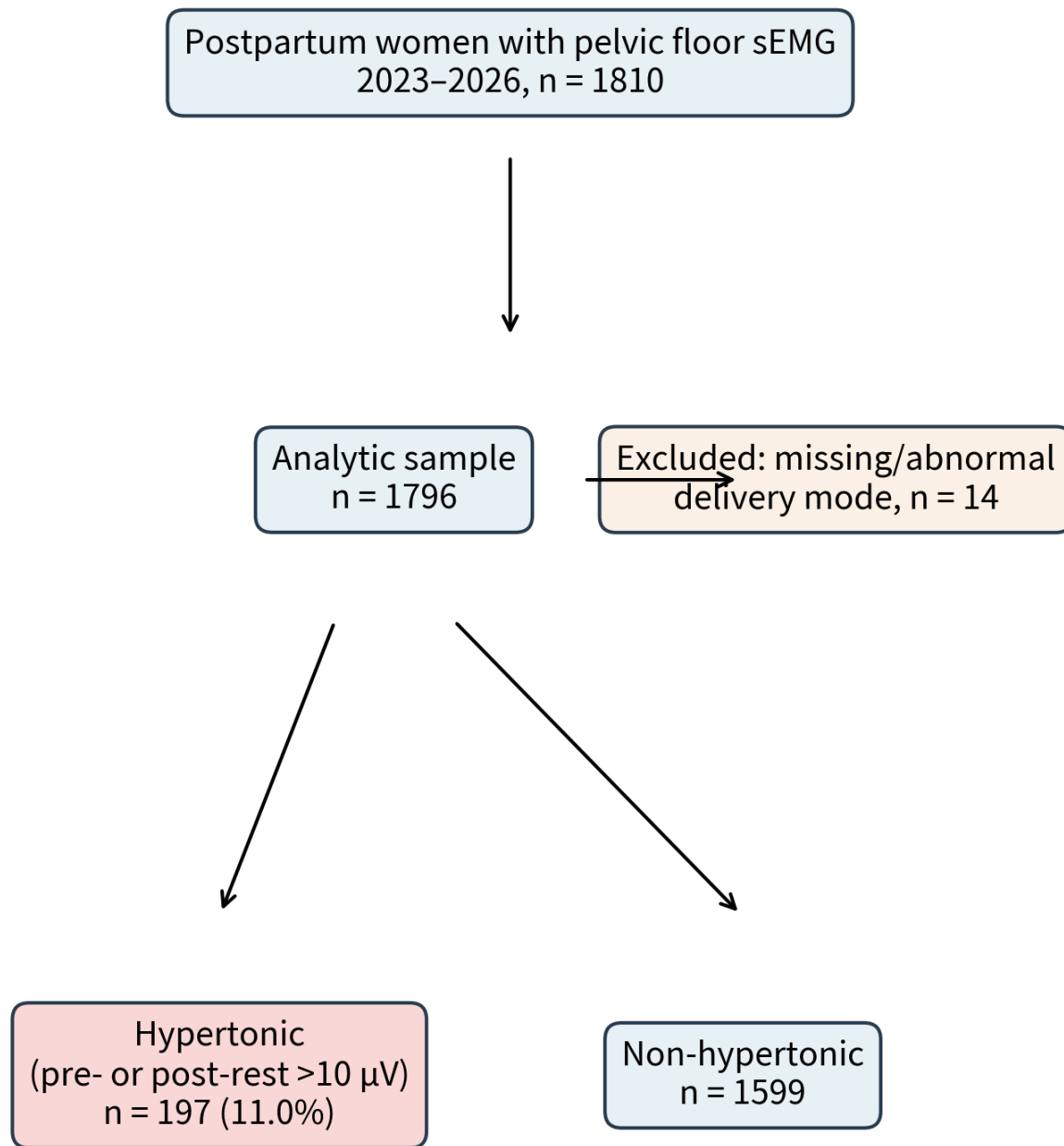


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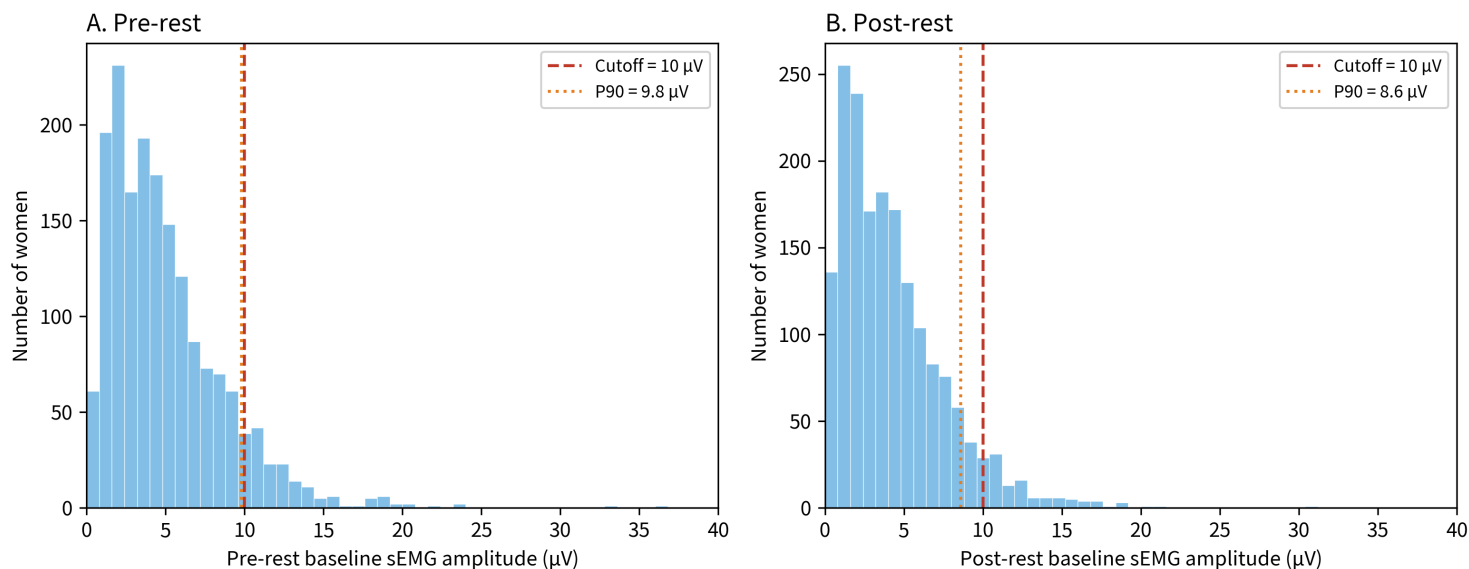


Figure 2

Distribution of (A) pre-rest and (B) post-rest baseline sEMG amplitudes. Dashed red line: prespecified hypertonicity cutoff (10 μV); dotted orange line: empirical 90th percentile (9.8 and 8.6 μV , respectively; P90 of the maximal resting amplitude was 10.32 μV).

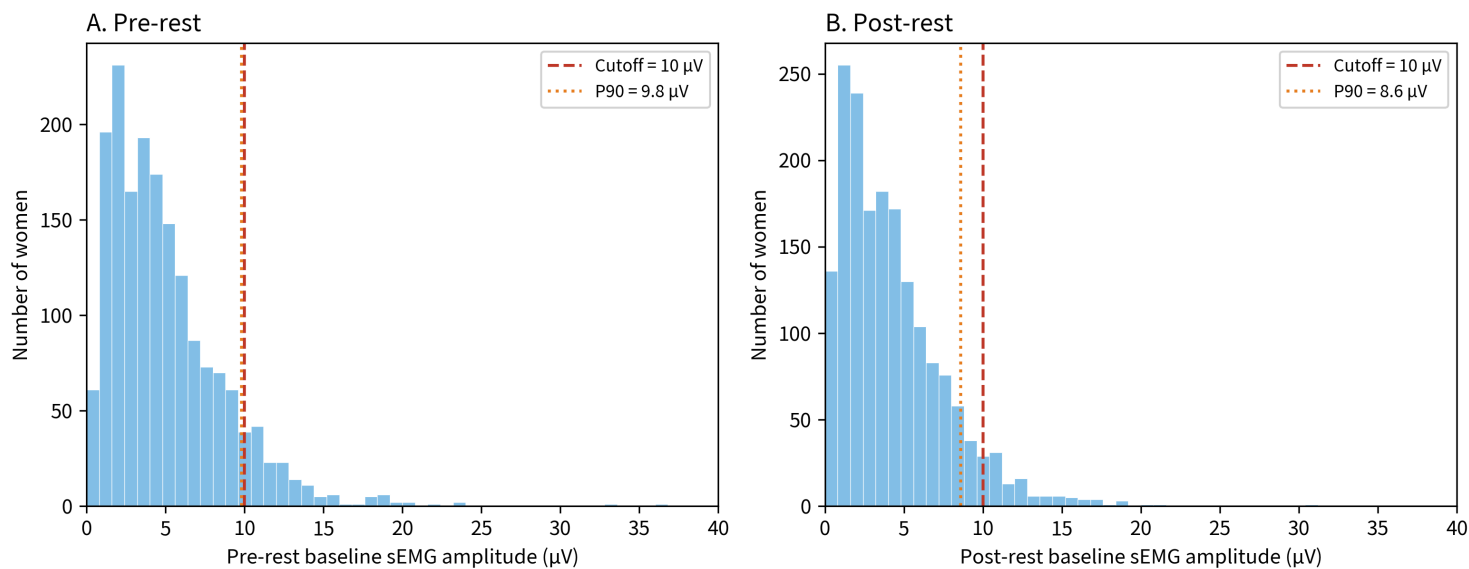


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Figure 3. Independent factors associated with pelvic floor hypertonicity

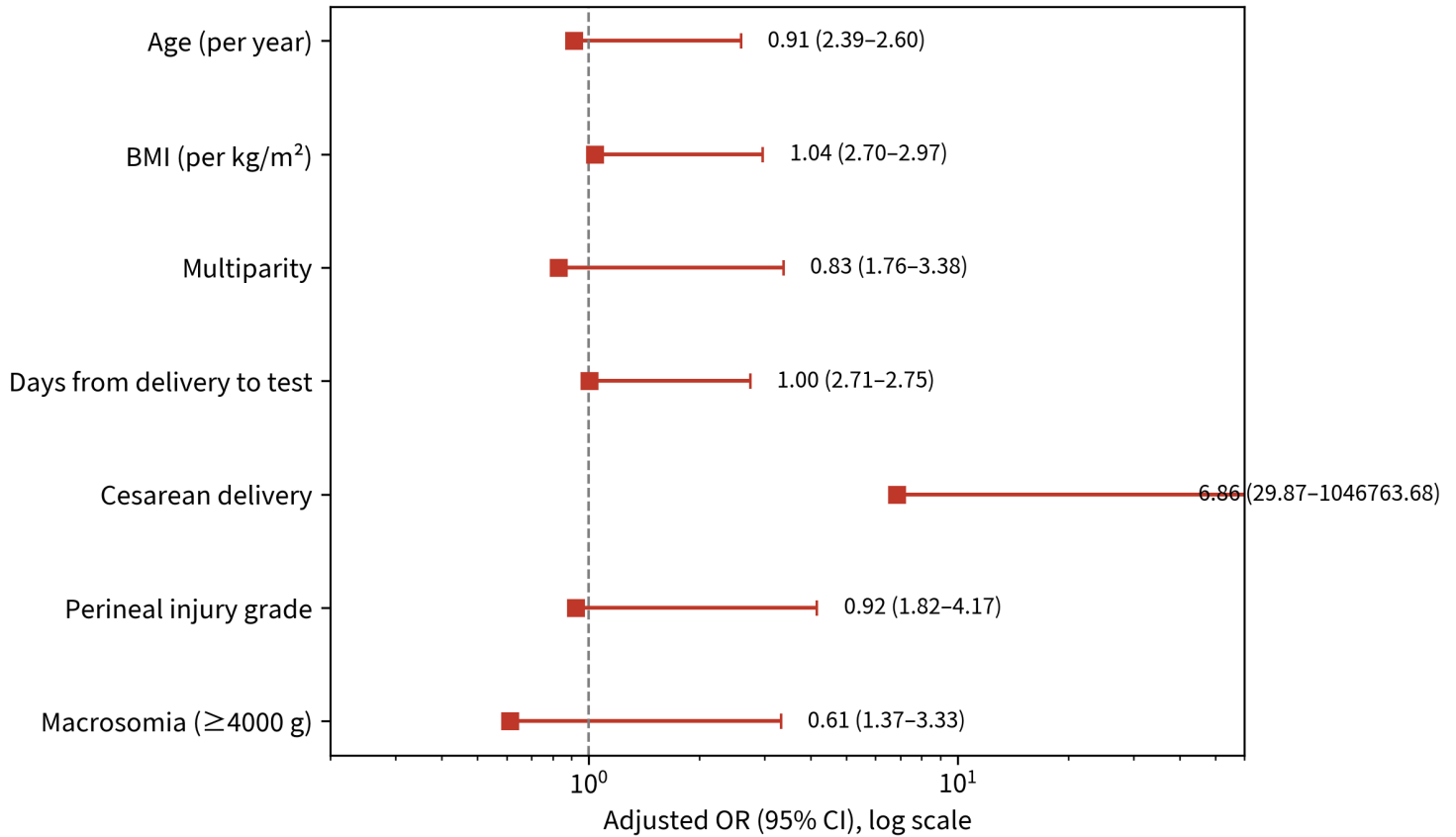


Figure 3

Forest plot of adjusted odds ratios from multivariable Model 2 (demographic + obstetric factors; n=1,761). Cesarean delivery is the dominant independent correlate of postpartum pelvic floor hypertonicity.

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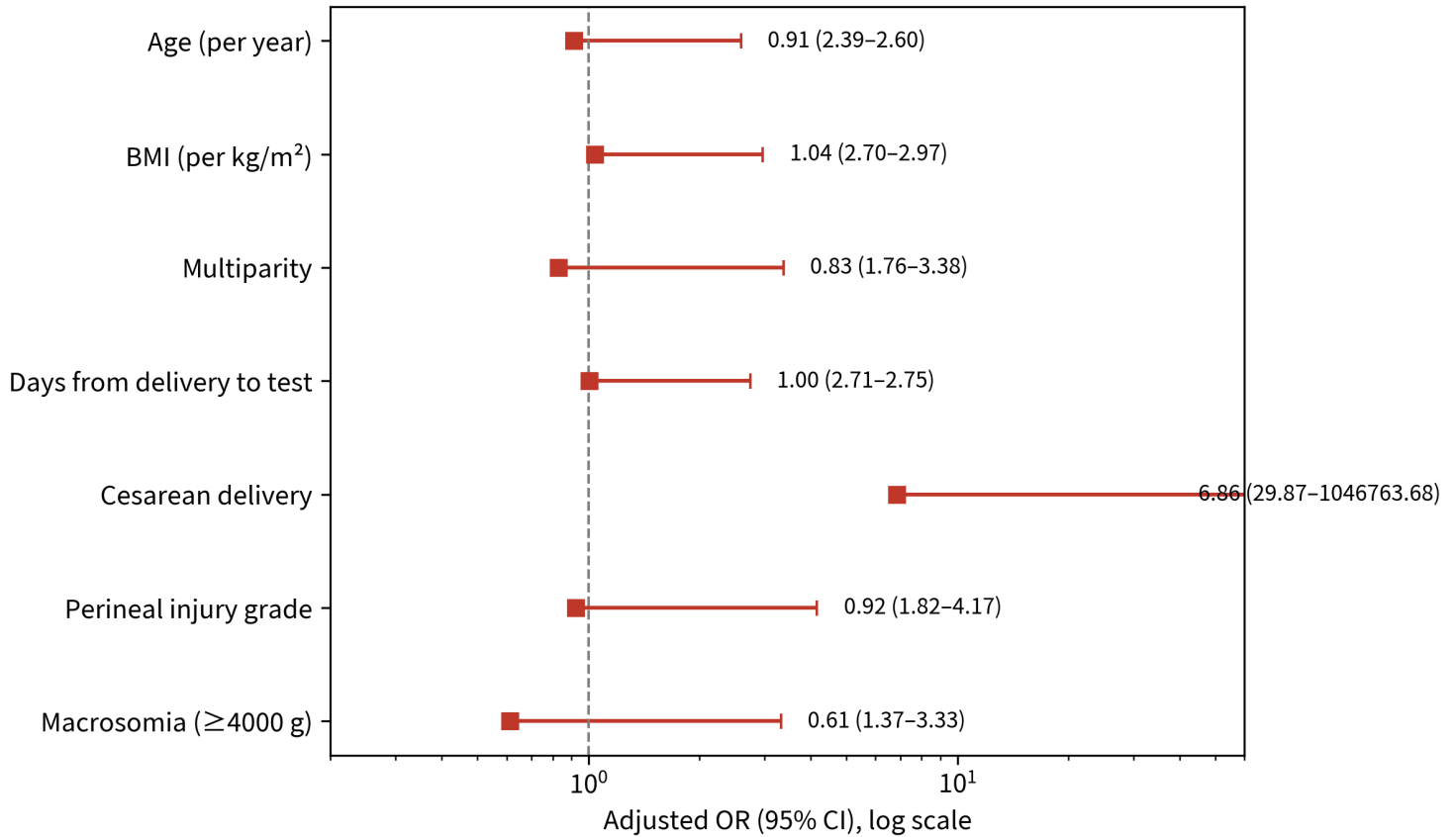


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Figure 4. Subgroup analyses: robustness of key associations

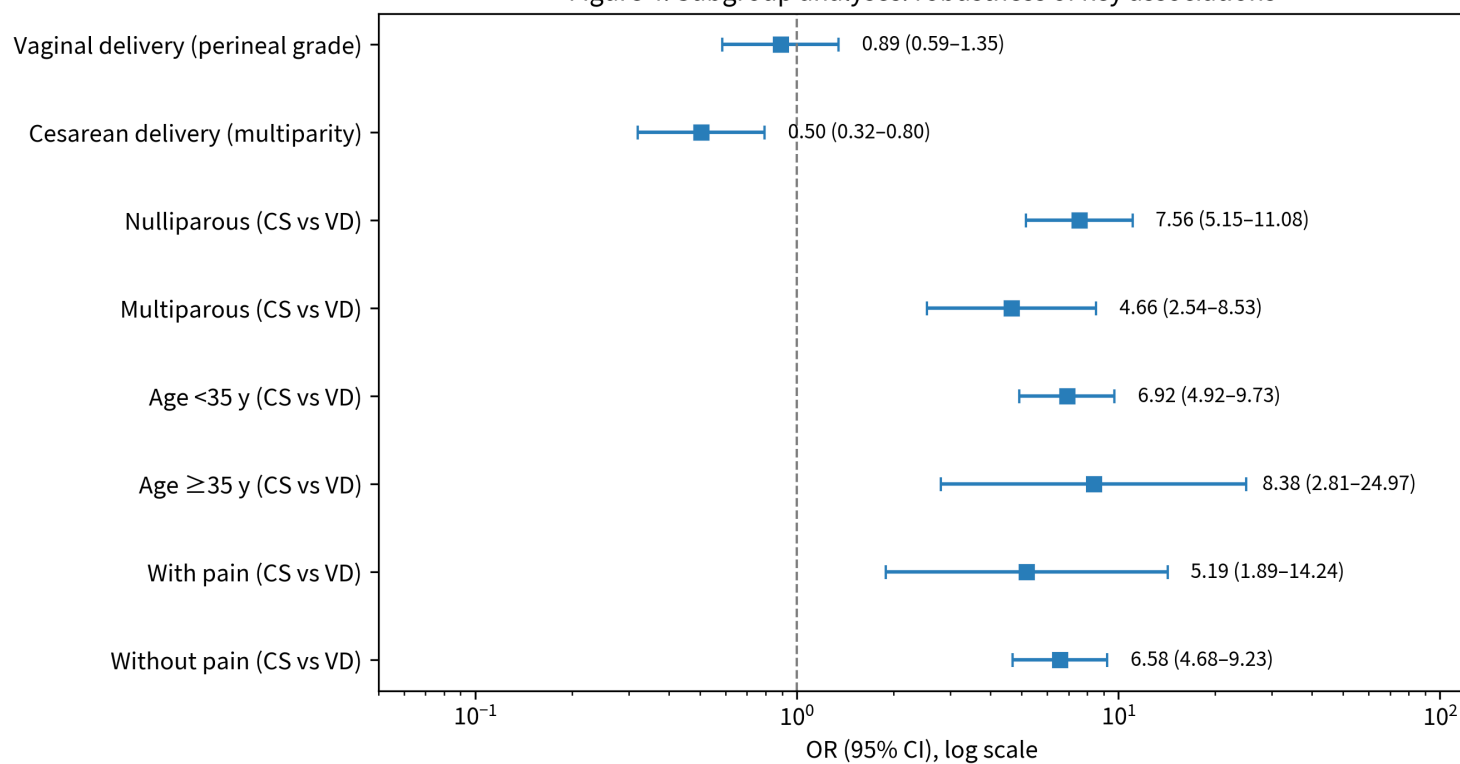


Figure 4

Subgroup analyses. The association between cesarean delivery and hypertonicity is consistent across parity, age, and pain strata; within delivery-mode strata, perineal injury grade (vaginal) and multiparity (cesarean) are shown.

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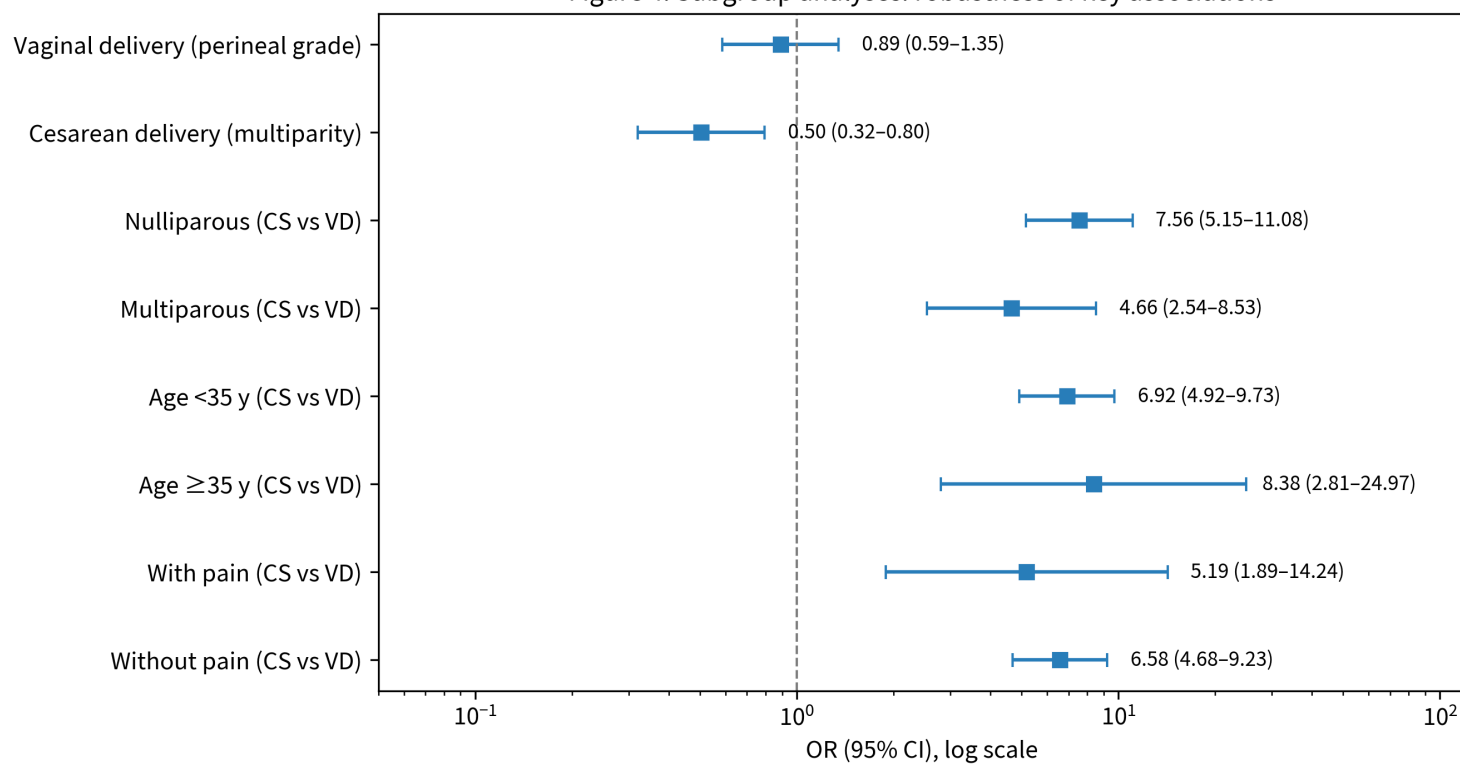


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Figure 5. Mirror-image risk profiles of hypertonic vs hypotonic phenotypes

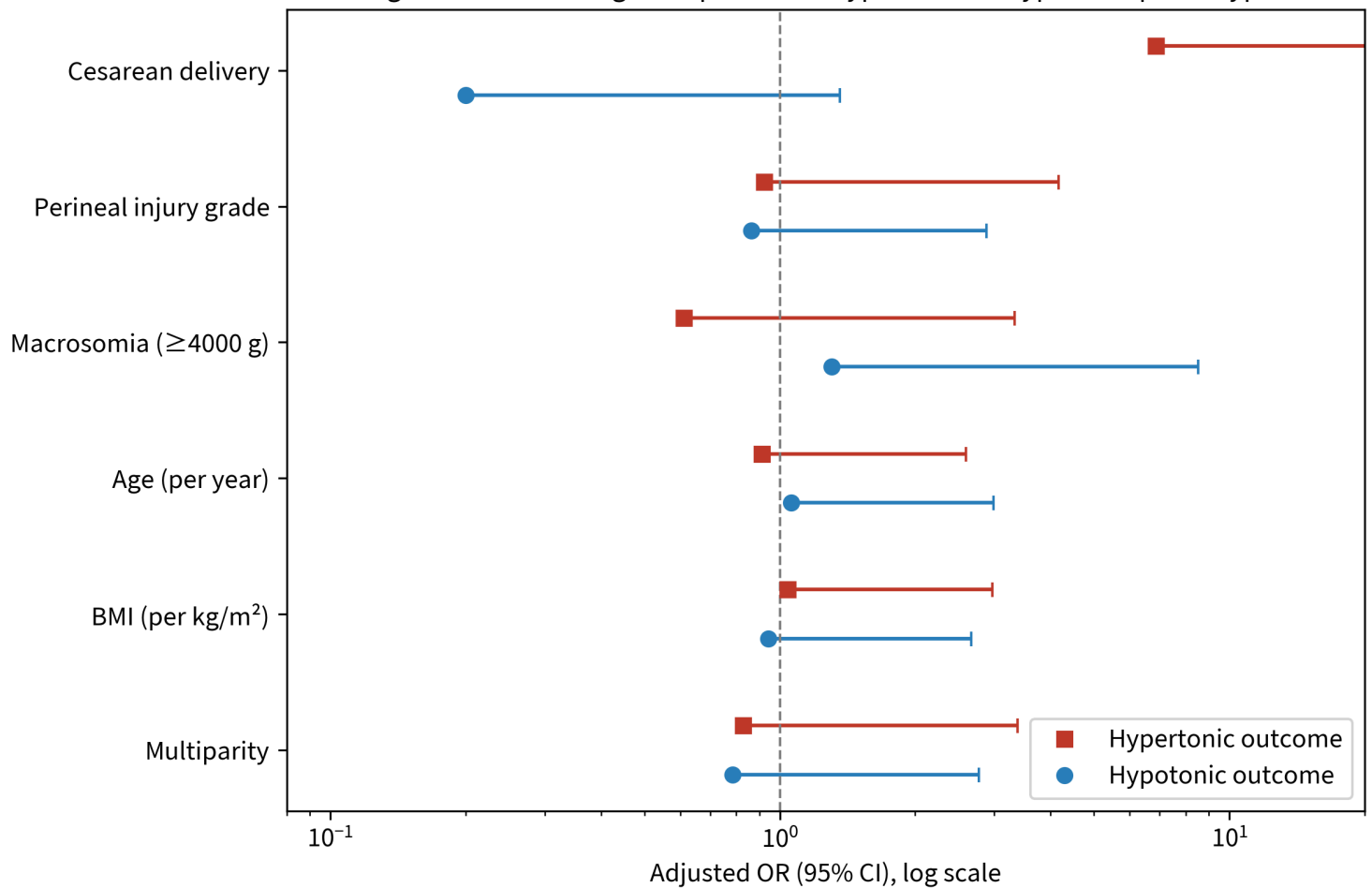


Figure 5

Mirror-image risk profiles. Adjusted odds ratios for the hypertonic (red squares) and hypotonic (blue circles) outcomes from parallel multivariable models. Cesarean delivery and age act in opposite directions on the two phenotypes.

Figure 5. Mirror-image risk profiles of hypertonic vs hypotonic phenotypes

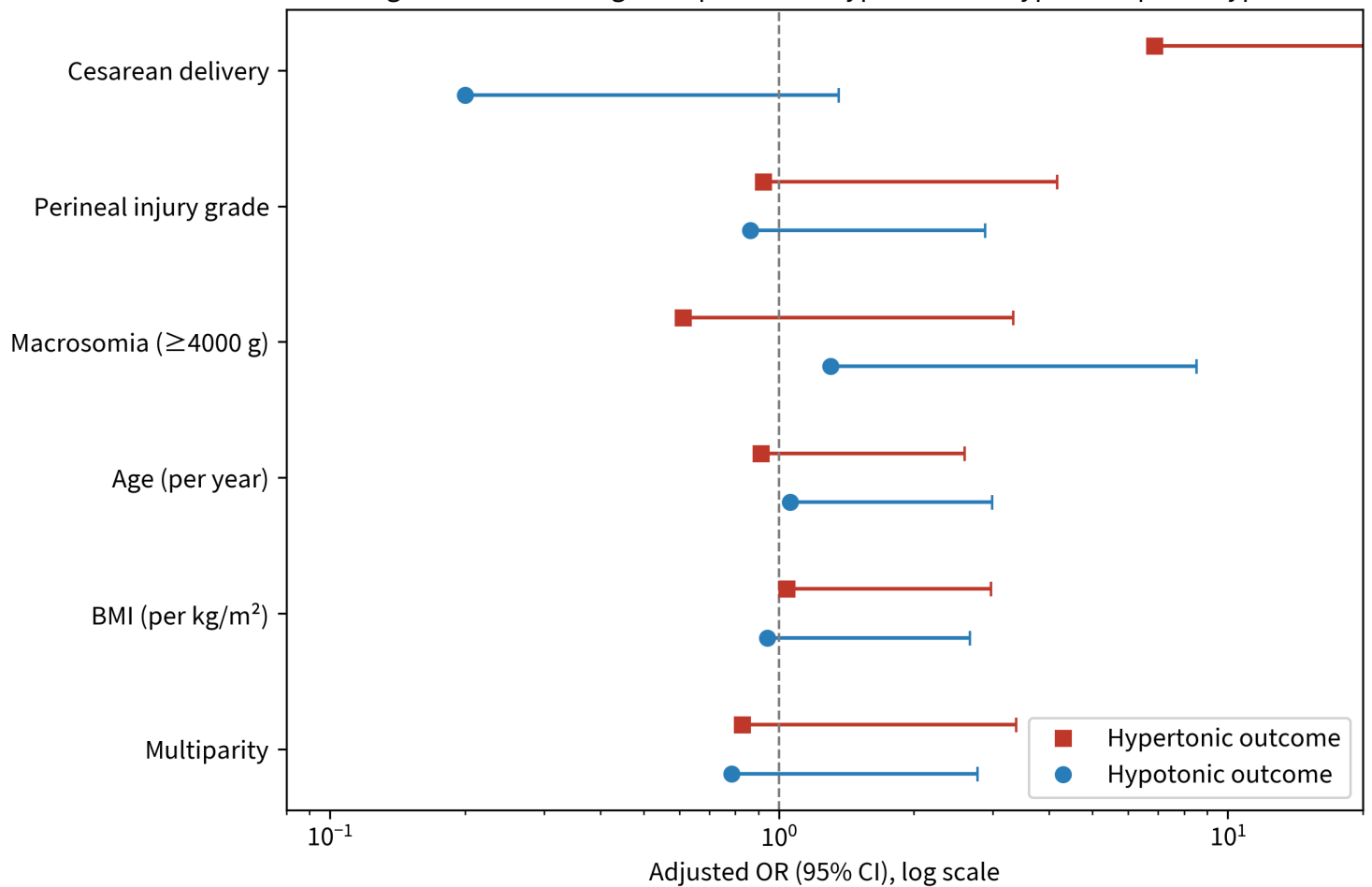


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Figure 6. Symptom spectrum across four pelvic floor phenotypes

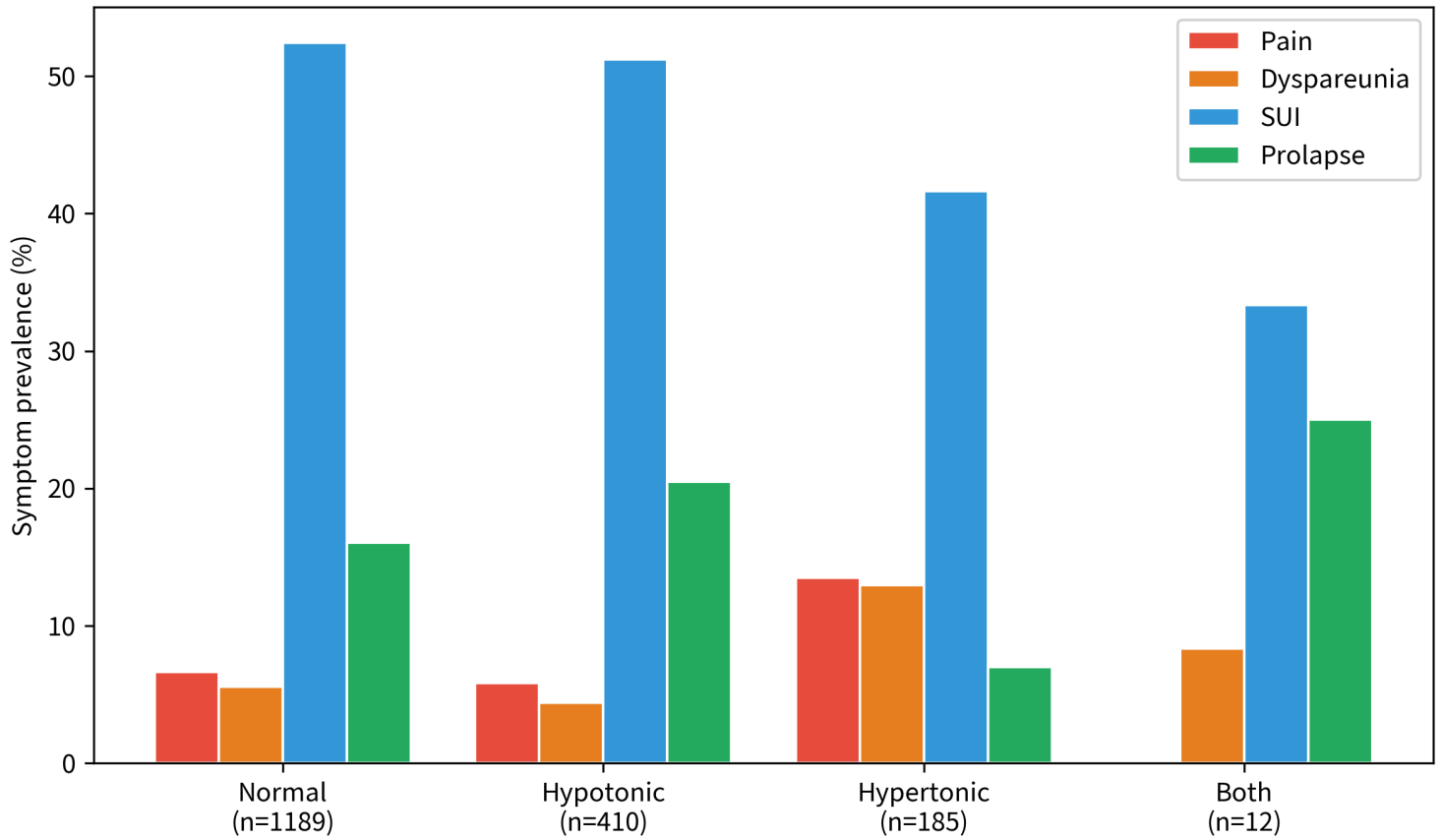


Figure 6

Symptom spectrum across the four pelvic floor phenotypes (normal, purely hypotonic, purely hypertonic, both). The hypertonic phenotype carries the highest pain and dyspareunia rates and the lowest prolapse rate.

Figure 6. Symptom spectrum across four pelvic floor phenotypes

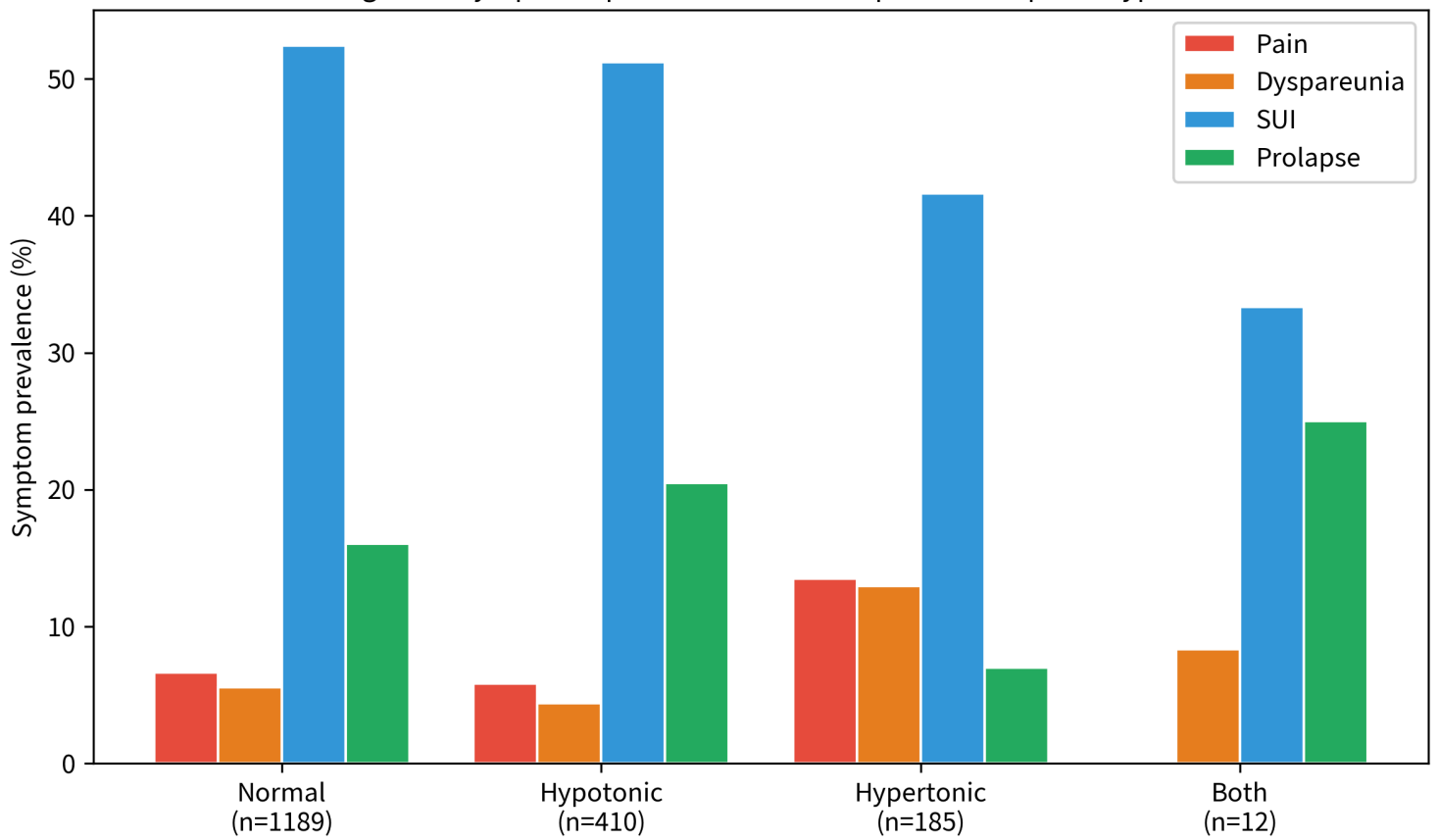


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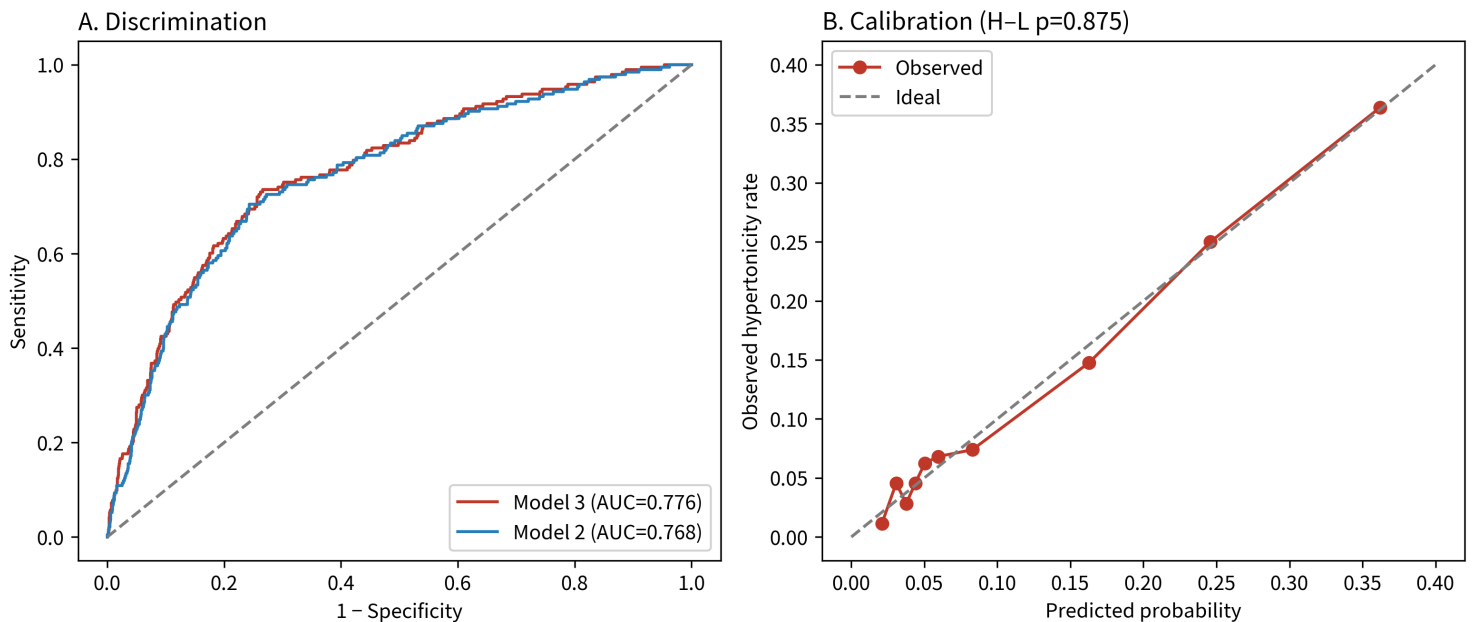


Figure 7

Model performance. (A) Receiver operating characteristic curves for Model 2 (AUC=0.768) and Model 3 (AUC=0.776); (B) calibration plot for Model 3 (Hosmer–Lemeshow $p=0.875$).

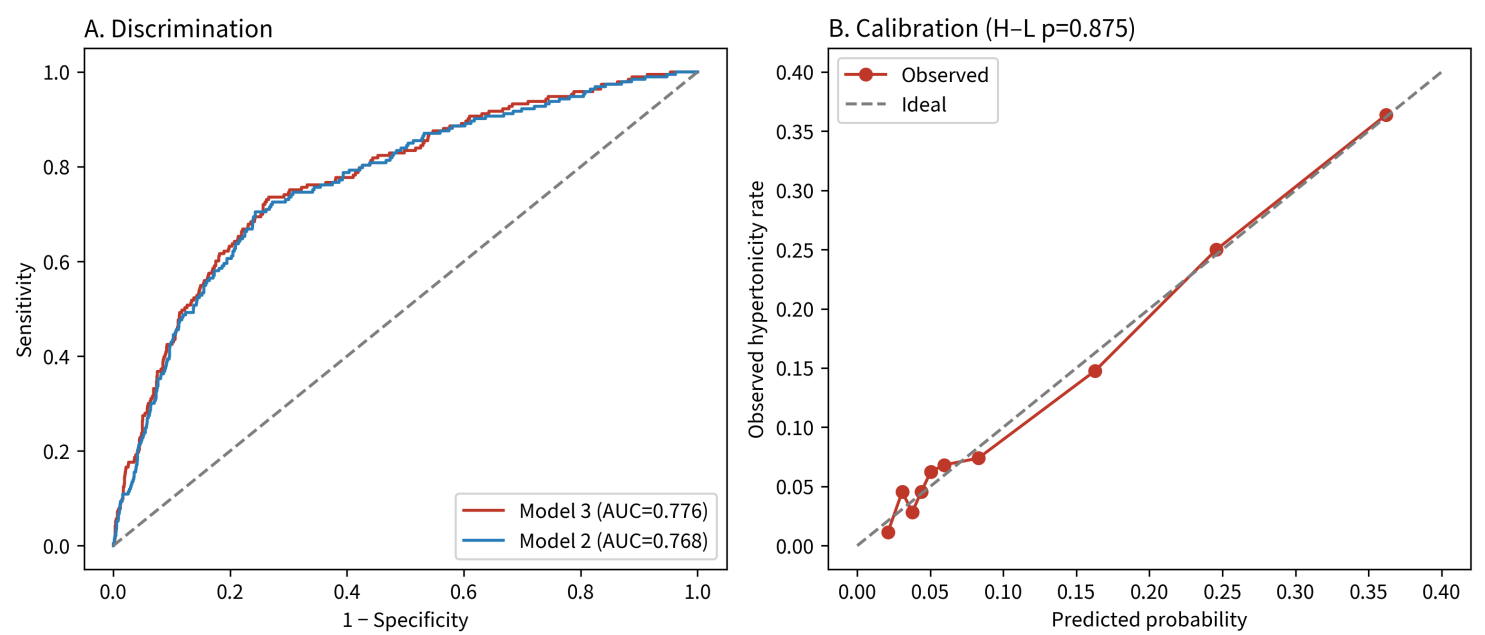


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Supplementary Files

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