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Intrapartum determinants of early postpartum pelvic floor electrophysiological dysfunction and clinical symptoms: a retrospective cohort study linking delivery records and pelvic floor assessment data

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Abstract

Background

Vaginal delivery is the principal modifiable determinant of postpartum pelvic floor dysfunction, yet how specific intrapartum exposures relate to objective pelvic floor electrophysiology and to early clinical symptoms remains incompletely quantified. We aimed to quantify the association between perineal injury severity and early postpartum pelvic floor surface electromyography (sEMG), and to compare the determinants of electrophysiological impairment with those of clinical symptom diagnoses.

Methods

Retrospective cohort study at a single tertiary maternity center in China. Integrated delivery–pelvic floor assessment records (2023–2026) were analyzed for women with vaginal delivery and a valid Glazer-protocol sEMG assessment, grouped by perineal status: intact/first-degree tear (n = 134), second-degree tear (n = 567), or episiotomy (n = 428). Outcomes were five sEMG phases, manual muscle testing, and clinician-recorded diagnoses (pelvic organ prolapse, stress urinary incontinence [SUI], dyspareunia) available for 1,095 women. Multivariable linear, logistic, and ordinal models, restricted cubic splines, and propensity-score matching were used, adjusting for the delivery-to-assessment interval (median 49 days).

Results

Among 1,129 women, contractile sEMG values declined across perineal categories in crude analyses (flicks maximum 30.1 vs 30.5 vs 27.4 μV, P = 0.003), but the gradient was fully attenuated after adjustment (episiotomy vs intact: β=+0.03, 95% CI − 3.11 to 3.17, P = 0.99). Forceps delivery (β=−5.36, 95% CI − 10.01 to − 0.71, P = 0.024) and second-stage duration (β=−1.08 μV per 30 minutes, P = 0.001) were the independent correlates of impaired contractility—confirmed in propensity-score-matched (β=−5.86, P = 0.030) and ordinal models. Second-stage durations ≥ 90 minutes were associated with reduced tonic contraction (β=−2.07, P = 0.010). In contrast, symptom diagnoses showed a reversed crude gradient (SUI 65.4% vs 56.6% vs 47.2%, P < 0.001) that likewise attenuated after adjustment (episiotomy OR 0.64, P = 0.056); multiparity, not intrapartum exposures, independently predicted SUI (OR 1.50, P = 0.018), prolapse (OR 1.57, P = 0.023), and any symptom (OR 1.72, P = 0.005).

Conclusions

Perineal injury severity is a marker of difficult labor rather than an independent driver of early postpartum pelvic floor impairment. Objective electrophysiological dysfunction tracks the intrapartum process (forceps, prolonged second stage), whereas early symptom burden tracks cumulative obstetric history

(parity)—two complementary windows on childbirth-related pelvic floor injury that may warrant distinct referral strategies.

Background

Pelvic floor dysfunction (PFD)—encompassing urinary incontinence, pelvic organ prolapse, and related disorders—affects approximately one in four women and imposes a substantial lifetime burden (1, 2). Vaginal childbirth is the single most important modifiable risk factor: landmark cohort and register-based studies have linked vaginal, and particularly operative vaginal, delivery to levator ani injury, muscle weakness, and PFD symptoms or surgery years to decades after childbirth (3–7), and a recent meta-analysis confirmed higher pelvic floor morbidity after vaginal than cesarean delivery (8).

Despite this progress, three gaps persist. First, most large studies assess pelvic floor outcomes by symptom questionnaires or imaging; objective electrophysiological assessment with vaginal surface electromyography (sEMG) using the Glazer protocol—an accessible, quantitative, and rehabilitation-relevant technique with established reliability (9–11)—has rarely been linked to detailed intrapartum data at scale. Second, imaging studies have established levator ani trauma as a central mechanism of childbirth-related pelvic floor injury (12–17), yet within vaginal deliveries “perineal injury” is usually treated as a single category; whether injury severity tracks electrophysiological impairment in a dose–response manner is unknown (18, 19). Third, perineal outcomes are inextricably confounded by labor difficulty: episiotomy and severe tears cluster with prolonged second stage and operative delivery (20, 21), so any observed association may be a marker rather than a cause—and, critically, almost no study has examined objective electrophysiology and subjective symptoms simultaneously in the same women, leaving open whether the two outcome families share the same determinants.

We therefore analyzed integrated delivery-registry and pelvic floor assessment data from a tertiary maternity center in China to address three questions: (1) does perineal injury severity independently predict early postpartum pelvic floor sEMG dysfunction; (2) which intrapartum factors—forceps, second-stage duration, or perineal injury itself—are the independent correlates of impaired contractility; and (3) do clinician-recorded symptom diagnoses (prolapse, stress urinary incontinence, dyspareunia) share the same determinants as electrophysiological impairment?

Methods

Study design and data sources

This retrospective cohort study was conducted at a tertiary maternity center in Zhejiang Province, China, and is reported following the STROBE statement (22). An integrated dataset (January 2023–2026) linking the hospital delivery registry to pelvic floor clinic assessment records was analyzed. The assessment records contain Glazer-protocol sEMG measurements, manual muscle testing, clinician-recorded pelvic floor diagnoses, and the interval from delivery to assessment; the delivery registry

contains obstetric, intrapartum, and neonatal variables. One assessment record per woman was retained, prioritizing complete sEMG and diagnosis data and then the shortest delivery-to-assessment interval. The ethics committee of Jinhua People's Hospital approved the study with a waiver of individual consent.

Study population

Women were eligible if they (i) had a vaginal delivery (spontaneous or forceps-assisted) during the study period, (ii) underwent at least one pelvic floor sEMG assessment, and (iii) had a documented perineal status at delivery. Women delivered by cesarean section and records with implausible values (sEMG > 200 μV; BMI < 14 or > 50 kg/m²; birthweight < 500 g or > 6000 g) were excluded. The primary analysis set comprised women with complete data on all adjustment covariates. To evaluate selection at this step, included women were compared with otherwise-eligible women excluded for incomplete covariates (Table S1, Additional file 1).

Exposure: perineal injury severity

Perineal status at delivery, as recorded by the attending midwife or obstetrician, was categorized into three groups for the primary analysis: (1) intact perineum or first-degree tear (including minor abrasions); (2) second-degree tear; and (3) episiotomy (medio-lateral or median). No third- or fourth-degree tears occurred in the cohort. Perineal data in the assessment records were used for cross-validation of the registry classification.

Outcomes: sEMG, manual muscle testing, and clinical diagnoses

Vaginal sEMG was performed by trained rehabilitation staff following the Glazer protocol with a vaginal surface electrode (9). Five phases were recorded: pre-contraction rest (pre-rest, μV), rapid phasic contractions (flicks maximum, μV; type II fast-twitch fibers), 10-second tonic contractions (tonic mean, μV; type I slow-twitch), sustained 60-second contraction (endurance mean, μV), and post-contraction rest (post-rest, μV). The protocol shows acceptable test–retest reliability and clinical predictive validity (9–11). Pelvic floor muscle strength was additionally graded by digital palpation on the modified Oxford scale (0–5) separately for type I and type II fibers. Clinician-recorded diagnoses at the assessment visit were coded into binary indicators: pelvic organ prolapse (POP), stress urinary incontinence (SUI), urgency urinary incontinence, dyspareunia, and constipation; a composite “any PFD symptom” indicator was derived.

Covariates and causal framework

Covariates were extracted from the delivery registry and assessment records: maternal age, BMI, gravidity and parity, gestational age at delivery, durations of the first and second stages of labor, forceps delivery, labor analgesia (neuraxial vs other/none), neonatal birthweight, and the delivery-to-assessment interval (days). A directed acyclic graph (DAG; Figure S1, Additional file 2) was drawn a priori: parity, analgesia, and fetal size are upstream determinants of labor difficulty; second-stage duration is a proximate exposure; forceps is modeled as a consequence of prolonged or difficult second stage rather than its cause; perineal injury is a downstream marker of labor difficulty; and sEMG measures and symptom diagnoses are outcome families potentially differing in their determinants and time courses. Adjustment sets were chosen accordingly.

Statistical analysis

Baseline characteristics were compared across perineal groups using one-way ANOVA, the Kruskal–Wallis test, or the chi-square test, as appropriate. Multivariable linear regression was fitted sequentially for sEMG outcomes: model 1 (unadjusted); model 2 (age, BMI, gestational age, parity); and model 3 (model 2 plus second-stage duration, forceps, labor analgesia, birthweight, and delivery-to-assessment interval). Modified Oxford grades were analyzed by proportional-odds ordinal logistic regression. Symptom diagnoses were analyzed by binary logistic regression with the same covariate set as model 3. The dose–response relationship between second-stage duration and flicks maximum was modeled with restricted cubic splines (three knots at the 25th, 50th, and 75th percentiles), with a test for non-linearity and threshold analyses at 60, 90, and 120 minutes. For the forceps association, a propensity-score-matched sensitivity analysis was performed: propensity scores were estimated by logistic regression (age, BMI, second-stage duration, birthweight, assessment interval, primiparity, episiotomy, second-degree tear), and forceps deliveries were matched 1:3 to non-forceps deliveries by nearest-neighbor matching without replacement within a caliper of 0.2 SD of the logit propensity score; balance was assessed by standardized mean differences (SMD). Two-sided $P < 0.05$ was considered significant. Analyses were performed in Python 3.12 (statsmodels 0.14, scikit-learn).

Results

Study population and selection

Of 1,810 integrated records (1,776 women), 527 cesarean deliveries, 19 records with missing perineal status, and 18 duplicate records were excluded, leaving 1,240 women with vaginal delivery and valid perineal grouping; 1,129 with complete covariates and sEMG data formed the primary analysis set (Fig. 1): intact/first-degree tear (n = 134), second-degree tear (n = 567), and episiotomy (n = 428). Forceps-assisted delivery occurred in 41 women (3.6%). The median delivery-to-assessment interval was 49 days (IQR 43–61). Clinician-recorded diagnoses were available for 1,095 women (97.0%). The 1,129 included women did not differ significantly from the 94 excluded women with sEMG data but incomplete covariates in any demographic, intrapartum, or sEMG characteristic (all $P > 0.18$; Table S1, Additional file 1), suggesting minimal selection bias from complete-case analysis.

Baseline characteristics

Women with more severe perineal exposure had substantially more difficult labors (Table 1). The episiotomy group had a nearly fourfold longer median second stage than the intact group (82 vs 23 minutes, P < 0.001), and higher rates of primiparity (83.9% vs 26.9%), neuraxial analgesia (88.8% vs 58.2%), and forceps use (7.9% vs 0%) (all P < 0.001). The delivery-to-assessment interval did not differ across groups (P = 0.85).

Table 1
Baseline characteristics by perineal injury group (n = 1,129 unless otherwise stated)

Characteristic	Intact/1st° (n = 134)	2nd° tear (n = 567)	Episiotomy (n = 428)	P value
Age, years	30.9 ± 4.1	30.7 ± 3.8	30.0 ± 3.4	0.005
BMI, kg/m²	22.6 ± 2.8	22.8 ± 3.1	22.8 ± 2.9	0.768
Gestational age, weeks	39.1 ± 1.6	39.4 ± 1.0	39.5 ± 1.1	0.001
Neonatal birthweight, g	3229 ± 493	3266 ± 376	3337 ± 408	0.005
Delivery-to-assessment interval, days, median (IQR)	49 (44–59)	48 (43–62)	49 (43–61)	0.851
Second stage, min, median (IQR)	23 (13–47)	42 (22–70)	82 (50–126)	< 0.001
First stage, min, median (IQR)	258 (160–390)	301 (185–420)	390 (275–520)	< 0.001
Forceps delivery, n (%)	0 (0)	7 (1.2)	34 (7.9)	< 0.001
Neuraxial analgesia, n (%)	78 (58.2)	423 (74.6)	380 (88.8)	< 0.001
Primiparous, n (%)	36 (26.9)	339 (59.8)	359 (83.9)	< 0.001

Data are mean ± SD unless otherwise stated.

Perineal severity and sEMG: crude associations

In crude analyses, all three contractile sEMG phases differed across perineal categories (Table 2): flicks maximum 30.1 ± 14.6 (intact/first-degree) vs 30.5 ± 14.8 (second-degree) vs 27.4 ± 14.4 μV (episiotomy), P = 0.003; tonic mean 20.2 ± 11.2 vs 20.0 ± 10.4 vs 18.0 ± 10.4 μV, P = 0.007; endurance mean 17.4 ± 9.8 vs 16.7 ± 8.9 vs 15.2 ± 9.4 μV, P = 0.014 (Fig. 2). Manual muscle strength likewise declined (type I P < 0.001; type II P = 0.005). Pre-rest and post-rest activity did not differ (P = 0.45 and P = 0.12).

Table 2
Pelvic floor sEMG, manual muscle strength, and clinical diagnoses by perineal injury group (crude)

Outcome	Intact/1st° (n = 134)	2nd° tear (n = 567)	Episiotomy (n = 428)	P value
Pre-rest, μV	4.3 ± 3.2	4.5 ± 5.7	4.1 ± 4.3	0.447
Flicks maximum, μV	30.1 ± 14.6	30.5 ± 14.8	27.4 ± 14.4	0.003
Tonic contraction mean, μV	20.2 ± 11.2	20.0 ± 10.4	18.0 ± 10.4	0.007
Endurance mean, μV	17.4 ± 9.8	16.7 ± 8.9	15.2 ± 9.4	0.014
Post-rest, μV	3.6 ± 2.9	3.6 ± 2.6	3.3 ± 2.4	0.122
Manual type I grade, median (IQR)	2 (1–2)	2 (1–2)	2 (1–2)	< 0.001
Manual type II grade, median (IQR)	2 (2–3)	2 (2–3)	2 (1–3)	0.005
Stress urinary incontinence, % (n/N)	65.4 (87/133)	56.6 (312/551)	47.2 (194/411)	< 0.001
Pelvic organ prolapse, % (n/N)	19.5 (26/133)	20.0 (110/551)	23.1 (95/411)	0.445
Dyspareunia, % (n/N)	4.5 (6/133)	6.0 (33/551)	5.6 (23/411)	0.801
Any PFD symptom, % (n/N)	78.2 (104/133)	72.8 (401/551)	66.4 (273/411)	0.015

Adjusted analyses: complete attenuation of the perineal effect

The perineal-severity gradient was fully attenuated after adjustment (Table 3). For flicks maximum, the crude episiotomy difference (β=−2.72, 95% CI − 5.56 to 0.12, P = 0.061) shrank to β=+0.03 (95% CI − 3.11 to 3.17, P = 0.99) in the fully adjusted model; corresponding estimates for tonic and endurance means were β=−0.07 (P = 0.95) and β=−0.05 (P = 0.96). Second-degree tears were null in all models.

Table 3
Association between perineal injury and sEMG outcomes: sequential multivariable models (β, 95% CI)

Outcome / Model	2nd° tear vs intact/1st°	Episiotomy vs intact/1st°
Flicks maximum, μV		
Crude	+ 0.41 (− 2.35 to 3.16)	−2.72 (− 5.56 to 0.12)
Demographic-adjusted	+ 1.15 (− 1.68 to 3.98)	−1.65 (− 4.71 to 1.41)
Fully adjusted	+ 1.25 (− 1.57 to 4.06)	+ 0.03 (− 3.11 to 3.17)
Tonic mean, μV		
Crude	−0.12 (− 2.10 to 1.85)	−2.13 (− 4.17 to − 0.10)*
Demographic-adjusted	+ 0.51 (− 1.51 to 2.54)	−1.24 (− 3.43 to 0.95)
Fully adjusted	+ 0.54 (− 1.47 to 2.55)	−0.07 (− 2.31 to 2.17)
Endurance mean, μV		
Crude	−0.69 (− 2.42 to 1.04)	−2.15 (− 3.93 to − 0.36)*
Demographic-adjusted	−0.05 (− 1.82 to 1.72)	−1.22 (− 3.14 to 0.70)
Fully adjusted	+ 0.01 (− 1.75 to 1.77)	−0.05 (− 2.02 to 1.91)

Fully adjusted model: age, BMI, gestational age, parity, second-stage duration, forceps, labor analgesia, neonatal birthweight, delivery-to-assessment interval. *P < 0.05.

In the fully adjusted model, independent correlates of flicks maximum were: forceps β=−5.36 (95% CI − 10.01 to − 0.71, P = 0.024); second stage β=−1.08 μV per 30 min (95% CI − 1.72 to − 0.44, P = 0.001); age β=−0.39 per year (P = 0.002); BMI β=+0.34 per kg/m² (P = 0.020). In the propensity-score-matched sample (39 forceps vs 114 non-forceps), forceps remained associated with flicks maximum (β=−5.86, 95% CI − 11.13 to − 0.58, P = 0.030) and endurance mean (β=−3.68, 95% CI − 6.80 to − 0.56, P = 0.021).

Independent correlates of electrophysiological impairment: forceps and second-stage duration

In the fully adjusted model (Table 3, Fig. 3), forceps delivery was the strongest intrapartum correlate of impaired contractility: flicks maximum β=−5.36 μV (95% CI − 10.01 to − 0.71, P = 0.024), tonic mean β=−3.54 (P = 0.037), endurance mean β=−3.42 (P = 0.021). Second-stage duration showed a dose-dependent association with all contractile phases (flicks β=−1.08 μV per 30 minutes, 95% CI − 1.72 to − 0.44, P = 0.001; tonic β=−0.87, P < 0.001; endurance β=−0.80, P < 0.001). Older age was independently associated with lower contractile amplitudes (β≈−0.3 to − 0.4 μV per year, all P ≤ 0.002), and higher BMI with better flicks maximum (β=+0.34 per kg/m², P = 0.020). The delivery-to-assessment interval was not associated with any contractile outcome (all P > 0.17), and neuraxial analgesia and birthweight were null throughout. Ordinal models confirmed the pattern: forceps (type I OR 0.52, 95% CI 0.28–0.97, P = 0.040;

type II OR 0.60, 95% CI 0.33–1.10, P = 0.10) and second-stage duration per 30 minutes (type I OR 0.85, P < 0.001; type II OR 0.90, P = 0.015).

Spline analysis confirmed the second-stage dose–response with significant non-linearity (P = 0.005): adjusted flicks maximum was stable through approximately 60–70 minutes and declined progressively thereafter (Fig. 4). Threshold analyses concurred: ≥90 minutes was associated with reduced flicks maximum (β=−2.77, 95% CI − 4.97 to − 0.58, P = 0.013) and tonic mean (β=−2.07, 95% CI − 3.64 to − 0.50, P = 0.010), whereas the 60-minute threshold was weaker (flicks P = 0.10).

Propensity-score-matched sensitivity analysis

Thirty-nine of the 41 forceps deliveries were matched 1:3 to 114 non-forceps deliveries (seven of eight post-match SMDs < 0.14). The forceps association persisted in the matched sample: flicks maximum β=−5.86 (95% CI − 11.13 to − 0.58, P = 0.030) and endurance mean β=−3.68 (95% CI − 6.80 to − 0.56, P = 0.021), supporting robustness to measured selection into operative delivery.

Clinical symptom diagnoses: a different determinant structure

Symptom prevalences were high in this clinic-attending cohort: SUI 54.2% (593/1,095), POP 21.1% (231), dyspareunia 5.7% (62), urgency incontinence 1.1% (12), constipation 0.5% (5); 71.1% had at least one diagnosis. In stark contrast to sEMG, the crude symptom gradient ran in the *opposite* direction across perineal groups: SUI 65.4% (intact/first-degree) vs 56.6% (second-degree) vs 47.2% (episiotomy), P < 0.001; any symptom 78.2% vs 72.8% vs 66.4%, P = 0.015 (Table 2). After adjustment, these associations attenuated (SUI: episiotomy OR 0.64, 95% CI 0.40–1.01, P = 0.056; any symptom: OR 0.78, P = 0.33), and neither forceps (SUI OR 0.84, P = 0.60) nor second-stage duration (OR 0.95 per 30 minutes, P = 0.30) predicted any symptom outcome (Fig. 5). Instead, multiparity was the consistent independent predictor: SUI OR 1.50 (95% CI 1.07–2.09, P = 0.018), POP OR 1.57 (95% CI 1.06–2.33, P = 0.023), any symptom OR 1.72 (95% CI 1.18–2.50, P = 0.005). For POP, episiotomy showed a borderline positive adjusted association (OR 1.71, 95% CI 0.99–2.95, P = 0.056) in the opposite direction to its crude “protective” gradient.

Discussion

Principal findings

In this cohort of 1,129 women assessed with Glazer-protocol sEMG a median of 49 days after vaginal delivery, perineal injury severity showed a monotonic gradient with contractile dysfunction in crude analyses that was fully—indeed, numerically completely—attenuated once labor difficulty was taken into account. The independent intrapartum correlates of electrophysiological impairment were forceps delivery and second-stage duration, acting specifically on contractile phases. The novel second finding is a determinant dissociation: clinician-recorded symptom diagnoses in the same women showed a

reversed crude gradient (driven by the parity structure of the perineal groups) and, after adjustment, were predicted by multiparity rather than by any intrapartum exposure. Objective muscle electrophysiology and subjective symptom burden thus appear to be two complementary, non-interchangeable windows on childbirth-related pelvic floor injury.

Comparison with existing literature

Our crude sEMG findings parallel the dominant narrative—that more severe perineal trauma tracks worse pelvic floor outcomes (18, 19)—but the adjusted results reframe it, consistent with Handa and colleagues' longitudinal observation that spontaneous laceration, not episiotomy, was associated with later pelvic floor disorders (18). Imaging studies have established forceps as the leading obstetric risk factor for levator ani avulsion (13, 15, 17); our electrophysiological estimates (β≈−5 to − 6 μV; type I manual-strength OR ≈ 0.5) are directionally concordant and extend this evidence to functional electrophysiology. Absolute sEMG values are comparable to a large 42-day postpartum screening cohort from Chengdu (23), and our adjusted null for episiotomy helps reconcile a Chinese prospective report of lower early-postpartum EMG indices after episiotomy in unadjusted comparisons (24). The second-stage dose–response—with functional decline accelerating beyond approximately 90 minutes—adds electrophysiological support to prospective evidence implicating the second stage in postpartum PFD (20) and to large secondary analyses of prolonged second stage (21). The high early symptom prevalence we observed exceeds general-population estimates, as expected in a clinic-attending sample, but the parity gradient mirrors population epidemiology in which cumulative births, rather than index-delivery mechanics, drive long-term symptom risk (3, 6, 7).

Mechanisms and clinical implications

Two non-exclusive mechanisms plausibly underlie the electrophysiological findings: direct mechanical trauma to the levator complex and pudendal nerve stretch injury, both scaling with fetal descent time and instrumented extraction (12, 15, 25). The dissociation between electrophysiology and symptoms has a plausible interpretation: at six to eight weeks postpartum, sEMG captures the acute muscular and neural insult of the index delivery, whereas reported symptoms reflect cumulative pelvic floor wear across the woman's obstetric career—primiparous women dominate the episiotomy group precisely because episiotomy is a first-delivery intervention, and their lower symptom burden reflects a single delivery rather than a protective incision. Consistent with this, the crude "protective" SUI gradient of episiotomy vanished (and, for POP, reversed to borderline harmful) once parity was adjusted. Neuraxial analgesia showed no independent association with any outcome, in line with prospective data (27). Clinically, our results support two distinct, testable referral strategies: intrapartum markers (forceps, second stage beyond 90 minutes) to target women for early electrophysiological evaluation and rehabilitation referral—an application supported by the effectiveness of pelvic floor muscle training (29) but requiring prospective confirmation—and routine parity-based counseling for symptom surveillance (26, 28).

Strengths and limitations

Strengths include the large sample for sEMG-linked intrapartum research; the dual outcome structure (objective electrophysiology plus clinician-recorded diagnoses in the same women); a recorded delivery-to-assessment interval, incorporated into all adjusted models; the DAG-guided decomposition strategy; robustness confirmed by propensity-score matching; and internal consistency across sEMG and digital palpation.

Several limitations temper interpretation. First, this is a single-center retrospective study of women attending a pelvic floor clinic; attendance is symptom- and referral-driven, which inflates absolute symptom prevalences and may distort symptom-level associations—although the included-versus-excluded comparison showed no meaningful differences, and the internal exposure contrasts for sEMG are less vulnerable to such selection. Second, symptom outcomes were clinician-recorded diagnoses rather than validated questionnaires (PFDI-20, PISQ-12, IIQ-7 were available for only ~ 1% of women); misclassification is likely non-differential but limits comparability with questionnaire-based epidemiology. Third, urgency incontinence (1.1%) and constipation (0.5%) were too rare for adjusted analysis. Fourth, forceps deliveries were few (n = 41), limiting subgroup and interaction power despite matched-analysis confirmation. Fifth, residual confounding—by maternal positioning, pushing technique, fetal head circumference, and levator status—is possible, and the DAG encodes unverifiable assumptions; between-day sEMG variability is documented (30), although the null association of the assessment interval with outcomes is reassuring. Finally, symptom and electrophysiological time courses may differ; longitudinal follow-up is needed to test whether early electrophysiological impairment predicts later symptoms.

Conclusions

Perineal injury severity is best understood as a marker of difficult labor rather than an independent cause of early postpartum pelvic floor impairment. Forceps delivery and prolonged second-stage duration—particularly beyond 90 minutes—are the dominant intrapartum correlates of electrophysiological contractile dysfunction, whereas early symptom burden tracks cumulative obstetric history. Objective electrophysiology and subjective symptoms capture distinct dimensions of childbirth-related pelvic floor injury and should not be used as proxies for one another in etiologic research or referral pathways.

Abbreviations

BMI
body mass index
CI
confidence interval
DAG
directed acyclic graph
IQR
interquartile range

OR
odds ratio
PFD
pelvic floor dysfunction
POP
pelvic organ prolapse
sEMG
surface electromyography
SMD
standardized mean difference
SUI
stress urinary incontinence
SVD
spontaneous vaginal delivery.

Declarations

Ethics approval and consent to participate

This study was approved by the Ethics Committee of Jinhua People's Hospital (approval No. 2026-Research-110). The requirement for informed consent to participate was waived by the Ethics Committee of Jinhua People's Hospital because of the retrospective nature of the study and the use of fully de-identified data. 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

JiaPing Lu: Conceptualization, Data curation, Investigation, Writing-original draft, Visualization.Heng Xu*: Conceptualization, Methodology, Formal analysis, Supervision, Project administration, Writing-review & editing, Validation.Qiulian Xu: Data curation, Resources, Investigation, Writing-review & editing.

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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.

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Figures

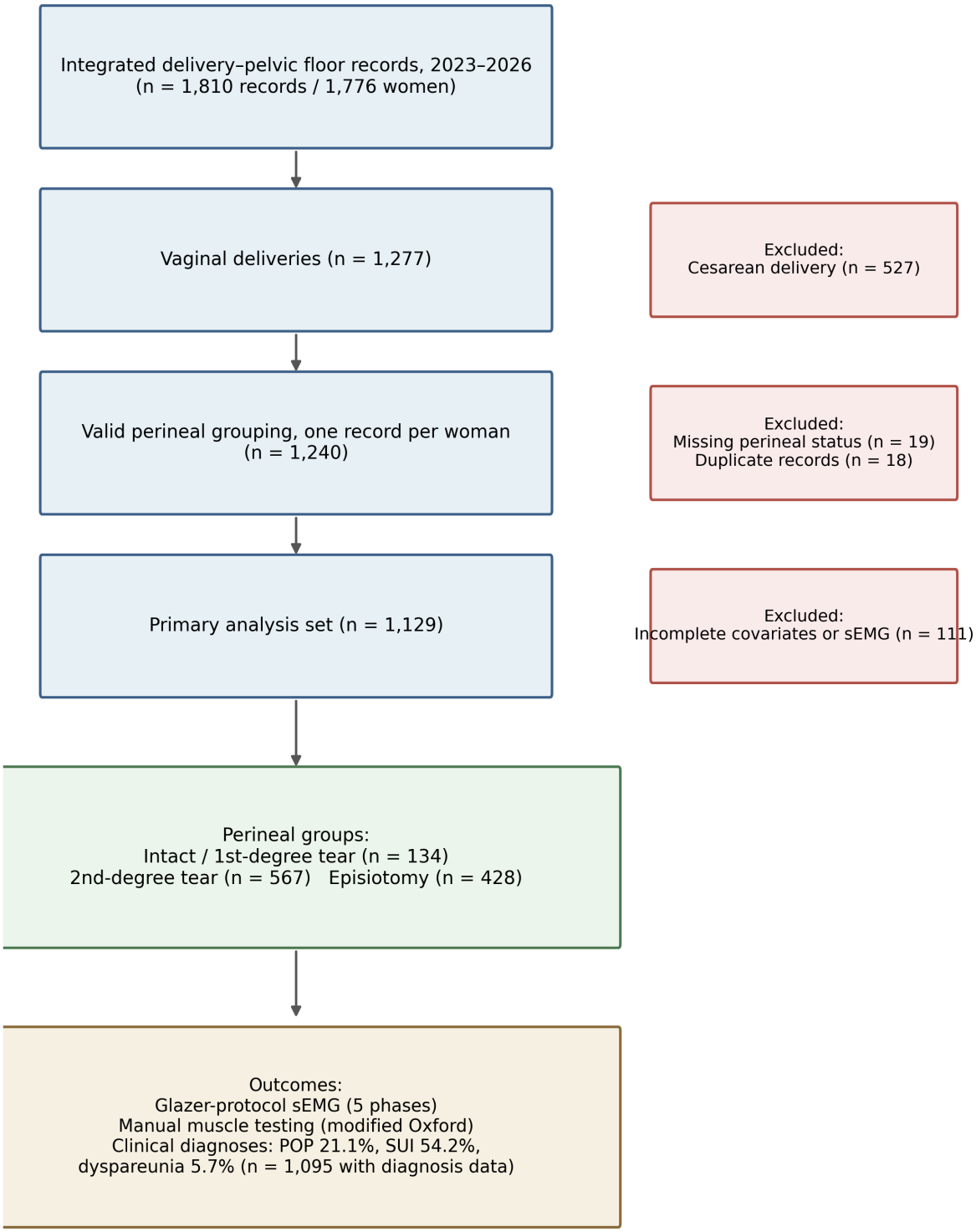


Figure 1

Study flow diagram.

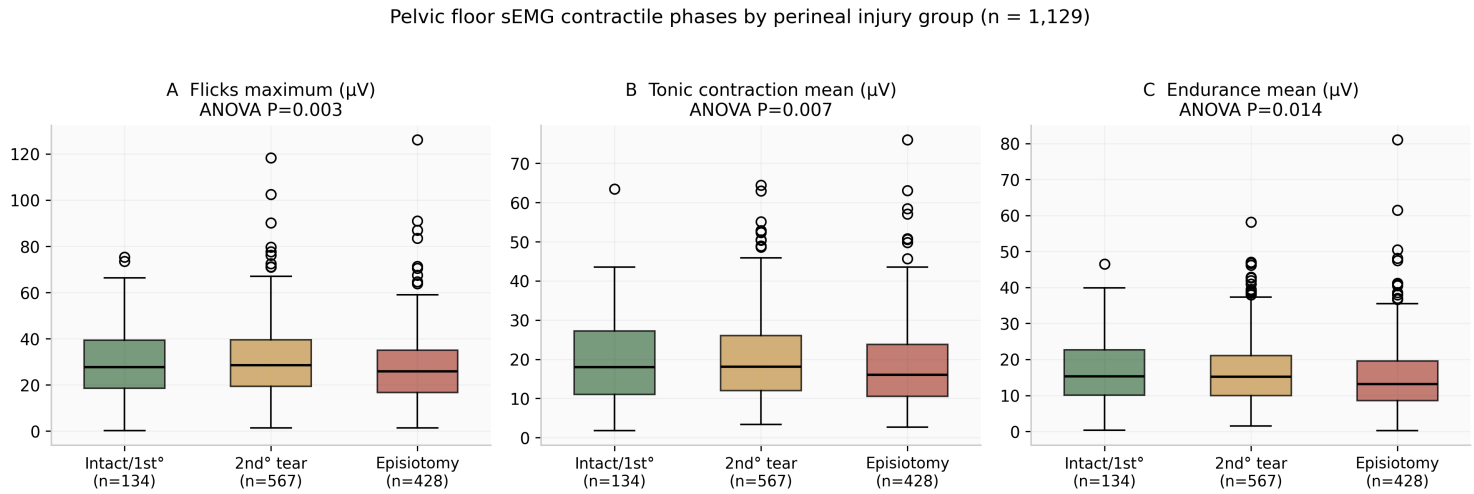


Figure 2

Pelvic floor sEMG contractile phases by perineal injury group: (A) flicks maximum, (B) tonic contraction mean, (C) endurance mean. Boxes show median and interquartile range; whiskers show 1.5×IQR.

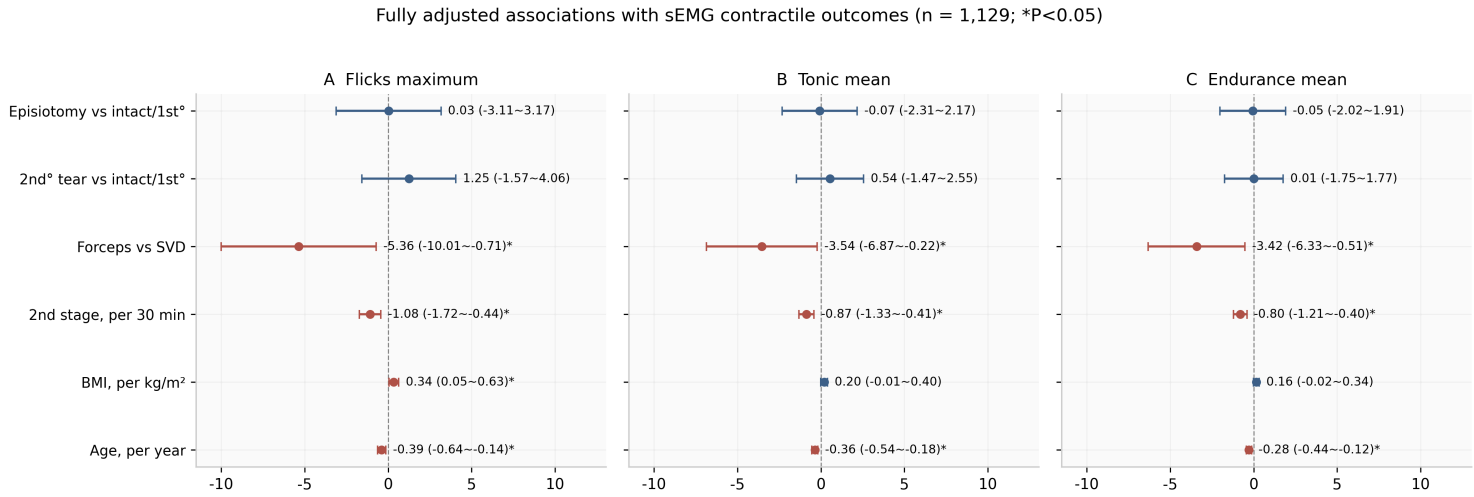


Figure 3

Forest plot of fully adjusted associations between key intrapartum exposures and sEMG contractile outcomes (linear models). β coefficients with 95% CI; *P<0.05.

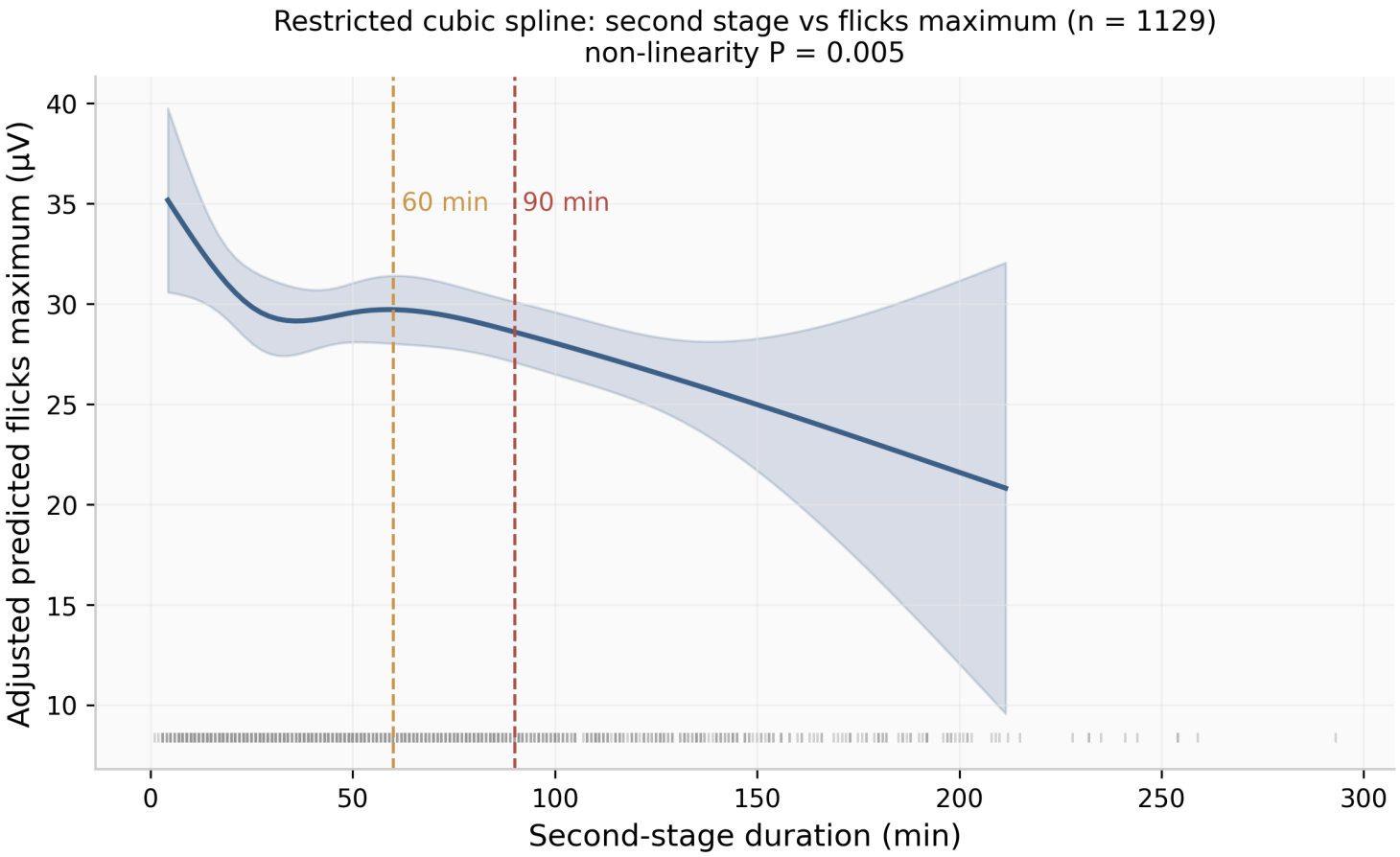


Figure 4

Restricted cubic spline analysis of second-stage duration and predicted flicks maximum (fully adjusted). Shaded band: 95% CI. Rug plot: distribution of second-stage durations. Non-linearity test P=0.005; dashed lines mark the 60- and 90-minute thresholds.

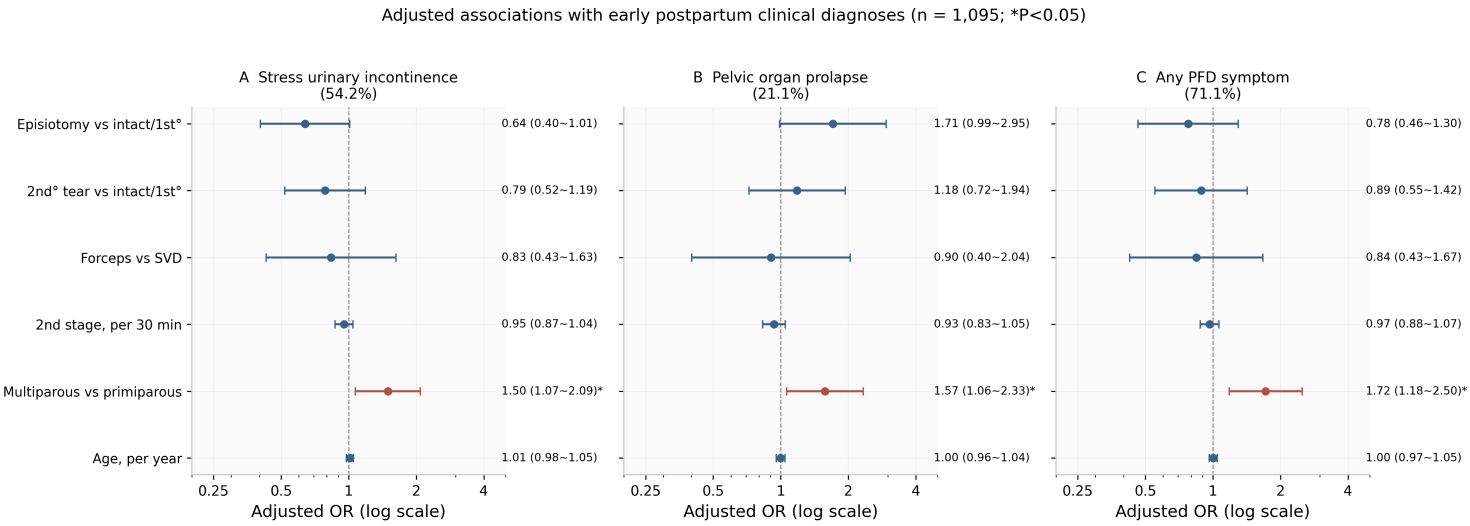


Figure 5

Forest plot of adjusted associations with clinician-recorded early postpartum diagnoses: (A) stress urinary incontinence, (B) pelvic organ prolapse, (C) any PFD symptom (logistic models). Adjusted ORs with 95% CI on a log scale; *P<0.05.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- Additionalfile1TableS1.docx
- Additionalfile3STROBEchecklist.docx
- Additionalfile2FigureS1DAG.png