

Second-Stage Labor Duration, Epidural Analgesia, and Early Postpartum Pelvic Floor Electromyography: A Retrospective Cohort Study of 1,127 Vaginal Deliveries

JiaPing Lu

Jinhua People's Hospital

Heng Xu

xheng0248@sina.com

Jinhua People's Hospital

Qiulian Xu

Jinhua People's Hospital

Research Article

Keywords: second stage of labor, epidural analgesia, pelvic floor, surface electromyography, dose-response, mediation analysis, postpartum

Posted Date: September 23rd, 2026

DOI: <https://doi.org/10.21203/rs.3.rs-10791734/v1>

License: © ⓘ This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Additional Declarations: No competing interests reported.

Second-Stage Labor Duration, Epidural Analgesia, and Early Postpartum Pelvic Floor Electromyography: A Retrospective Cohort Study of 1,127 Vaginal Deliveries

Running title: Second-stage duration and pelvic floor sEMG

Authors: JiaPing Lu¹, Heng Xu^{1*}, Qiulian Xu¹

¹ Department of Obstetrics, Jinhua People's Hospital, Jinhua, China

Corresponding Author:

Heng Xu^{1*}

Department of Obstetrics, Jinhua People's Hospital, Jinhua, China

Tel: 8618757829616

Email: xheng0248@sina.com

Abstract

Background: Prolonged second stage of labor is usually analyzed dichotomously, and the association between epidural analgesia and pelvic floor function remains contested, with electromyographic evidence limited and unadjusted for labor duration. We quantified the association of second-stage duration with early postpartum pelvic floor muscle electrophysiology and reassessed the epidural association using standardized surface electromyography (sEMG).

Methods: Single-center retrospective cohort of 1,127 singleton vaginal deliveries (January 2023 to January 2026) with Glazer-protocol pelvic floor sEMG at postpartum screening (median 49 days). Outcomes were fast-twitch maximum, sustained-contraction mean, and endurance mean amplitudes (μ V). Multivariable linear models were mutually adjusted for forceps, perineal injury grade, epidural analgesia, parity, age, BMI, birth weight, and days postpartum, with a 50-

dataset multiple-imputation sensitivity analysis and an exploratory bootstrap mediation analysis of the epidural association.

Results: Each additional 10 minutes of second stage was associated with $-0.39 \mu\text{V}$ of fast-twitch amplitude (95% CI -0.59 to -0.19 , $p < 0.001$). No statistical evidence of departure from linearity was identified (P for nonlinearity = 0.55), although power was limited at the tails. The association was stronger in women tested within 42 days postpartum (-2.20 vs $-0.88 \mu\text{V}$ per 30 min; $n = 247$ vs $n = 880$; interaction $P = 0.042$) and remained stable after multiple imputation. Forceps delivery was independently associated with $-6.08 \mu\text{V}$ ($p = 0.010$); perineal injury grade showed no independent association ($p = 0.438$). Epidural analgesia was not directly associated with fast-twitch amplitude ($+0.93 \mu\text{V}$, 95% CI -1.29 to $+3.15$, $p = 0.413$), sustained contraction ($p = 0.411$), or resting tone ($p = 0.996$), but was associated with a 20.3-minute longer second stage ($p < 0.001$). The only statistically significant indirect path ran through that prolongation in exploratory mediation analysis (indirect effect $-0.79 \mu\text{V}$, 95% CI -1.26 to -0.40), suggesting that the lower sEMG amplitudes in epidural labors were statistically accounted for by longer labor.

Conclusions: Second-stage duration showed a quantifiable, graded association with early postpartum pelvic floor muscle electrophysiology, with no statistical evidence of departure from linearity. No direct association between epidural analgesia and postpartum pelvic floor muscle electrophysiology was detected; absence of evidence is not evidence of safety. These findings are hypothesis-generating, and whether modifying second-stage management improves pelvic floor outcomes requires prospective evaluation.

Keywords: second stage of labor; epidural analgesia; pelvic floor; surface electromyography; dose-response; mediation analysis; postpartum

1. Introduction

Nearly one in four US women report at least one symptomatic pelvic floor disorder (PFD) [1], and delivery mode is strongly associated with the risk of incontinence and prolapse decades later [2 – 4]. The mechanical strain is concentrated in the second stage: the levator ani is

stretched around the descending head, approaching its tissue tolerance limits in simulation models [5], and the pudendal nerve is compressed against the pelvic sidewall [6].

Yet in practice, second-stage duration is almost always dichotomized— ≥ 60 minutes or ≥ 120 minutes, depending on the guideline [7 – 8]. Although useful for protocols, this does not match clinical decision-making. When progress slows, the choice is typically between waiting another 30 minutes or intervening now. The relevant question is not whether a threshold has been crossed, but how much each additional minute is associated with the outcome.

A second, long-standing controversy runs in parallel. Epidural analgesia relieves pain effectively but lengthens the second stage by roughly 15 – 30 minutes in most studies [9 – 10], and because it may reduce pelvic floor tone during the block, concern persists that it might contribute to postpartum pelvic floor impairment. The electromyographic evidence is weaker than it appears. Although some sEMG cohorts are large—one recent Chinese series included over 3,000 primiparas [11]—none has linked second-stage duration to sEMG with continuous duration data. The only sEMG study that specifically examined epidural analgesia ($n = 124$) did not adjust for labor duration [12], which is arguably a key confounder or mediator in this association. Whether epidural analgesia is associated with postpartum muscle impairment directly, or only because the labors it accompanies run longer, is not merely academic: the two answers imply opposite counseling approaches.

Pelvic floor sEMG following the Glazer protocol provides standardized, quantitative readouts of fast-twitch and sustained contractile function and of resting tone [13 – 14], is consistent with internationally standardized assessment terminology [15], and is sensitive to delivery mode and obstetric factors [16 – 17]. Because it is performed routinely at postpartum screening in China [18], it can generate large consecutive series with linkable delivery records. We used one such series to pursue two connected aims: to quantify the association of second-stage duration with early postpartum pelvic floor sEMG, and to reassess the association between epidural analgesia and pelvic floor sEMG by separating its direct component from the component operating through longer labor.

2. Methods

2.1 Design and population

This is a single-center retrospective cohort study at Jinhua People's Hospital. We screened all women who underwent standardized postpartum pelvic floor sEMG between January 2023 and January 2026 and whose electronic delivery records could be linked ($N = 1,810$). Twin pregnancies ($n = 31$) and cesarean births ($n = 506$) were excluded, leaving 1,273 singleton vaginal deliveries; 1,248 had a parseable second-stage duration. When a woman had more than one postpartum sEMG record during the study period (identified by matching name and telephone number), only the first examination was retained, so that each woman contributed a single observation (7 repeat records excluded; 1,241 eligible women). Of these, 1,127 had complete data on all model covariates and formed the analytic sample (Figure 1). All exclusions and derivations—including the one-record-per-woman rule—were applied by a single scripted analysis pipeline; no manual case-level editing was performed. The study protocol was approved by the Ethics Committee of Jinhua People's Hospital (approval No. 2026-Research-110); given the retrospective design and the use of de-identified routinely collected data, the requirement for individual informed consent was waived by the ethics committee. Reporting follows STROBE.

2.2 sEMG outcomes

Pelvic floor surface electromyography was performed with the MLD-A2 pelvic floor sEMG analysis system (Medlander Medical Technology, Nanjing, China) using the manufacturer's pear-shaped vaginal electrode (model MLD-A2-E01). In our center, women undergo pelvic floor screening at the routine postpartum visit, scheduled at approximately 42 days after delivery (median actual assessment 49 days; range 15 – 195). Examinations were performed by trained technicians with the woman in the lithotomy position after bladder emptying and vulvar cleansing. Signals were sampled at 1000 Hz with a built-in 20 – 500 Hz band-pass filter. The examination followed the manufacturer's Glazer-based protocol: five rapid (phasic) contractions, five 10-s sustained (tonic) contractions, and an endurance series of five repeated 10-s contractions, separated by standardized 10-s rest intervals; pre-resting and post-resting tone were also recorded. The primary outcome was fast-twitch maximum amplitude (μV), defined as the

highest RMS amplitude among the five rapid contractions; secondary outcomes were sustained-contraction mean and endurance mean amplitudes, calculated as the mean RMS amplitude across valid contraction segments, and pre-resting tone. A contraction segment was considered valid if the technician confirmed adequate electrode contact and the waveform showed no artifact. The technician performing the examination was not aware of the woman's intrapartum details at the time of assessment.

Quality control followed a standardized institutional procedure: the technician verified electrode depth and contact before recording, monitored the waveform in real time, and repositioned the electrode and repeated the test whenever artifacts were observed. At the analysis stage, physiologically implausible values were set to missing using pre-specified thresholds (fast-twitch maximum $> 120 \mu V$, sustained or endurance amplitude $> 60 \mu V$, resting tone $> 60 \mu V$, and Glazer total score > 200), based on the manufacturer's reference range and local quality-control data. Among the 1,248 women with parseable second-stage durations, these thresholds affected 1 fast-twitch, 6 sustained, 2 endurance, and 3 resting-tone recordings, and 1 Glazer total score.

2.3 Exposures and covariates

Second-stage duration was extracted from the electronic labor record and defined as the interval from documented full cervical dilatation (10 cm) to delivery of the fetus. For women with epidural analgesia, this interval includes the passive phase of the second stage; delayed pushing was routinely practiced for women receiving neuraxial labor analgesia at our institution throughout the study period (2023–2026). Durations are recorded to the nearest 5 minutes. Implausible values (< 0 or > 300 minutes) were reviewed against the original labor records and set to missing when they could not be confirmed.

We also extracted forceps assistance, perineal status, labor analgesia type, neonatal birth weight, parity, maternal age, and BMI. Operative vaginal delivery in this cohort consisted exclusively of forceps-assisted delivery; vacuum extraction is not practiced at our institution. Perineal status was coded as intact/abrasion = 0, first-degree tear or episiotomy = 1, second-degree tear = 2, and third-degree tear = 3; the distribution of the raw recorded categories is reported in Supplementary Table S6. No third- or fourth-degree (obstetric anal sphincter injury)

tears occurred in this cohort; the grade-3 category was empty. Because grouping episiotomy with first-degree tears is debatable, we additionally modeled episiotomy as a separate indicator in sensitivity analyses. Labor analgesia was categorized as epidural versus none or non-neuraxial methods, parsed from anesthesia text fields. Implausible birth weight (<1,000 or >6,000 g) and BMI (<12 or >60) values were set to missing.

Neuraxial labor analgesia followed a standardized institutional protocol throughout the study period. Epidural analgesia was initiated when cervical dilatation reached 1 – 3 cm and was maintained until delivery. An epidural catheter was placed at the L2–L3 interspace; the initial dose was 10 mL of 0.125% ropivacaine with 0.4 µg/mL sufentanil. After a 10-minute observation period, a patient-controlled epidural analgesia (PCEA) pump (0.08% ropivacaine with 0.4 µg/mL sufentanil) was connected, with a background infusion of 1 mL/h, a patient-controlled bolus of 8 mL, a lockout interval of 30 minutes, and maximum hourly volume of 50 mL. An anesthesiologist reviewed each woman hourly, and the catheter was removed after delivery. Because of the retrospective design, individual-level adherence to this protocol, the exact timing of epidural initiation relative to cervical dilatation, and the total duration of neuraxial exposure could not be extracted for every woman; the exposure is therefore characterized at the level of the institutional standard protocol, and the absence of individual initiation times and exposure durations is acknowledged as a limitation (see Limitations).

2.4 Statistical analysis

Per-minute association (aim 1). We regressed each sEMG outcome on second-stage duration (per minute, rescaled per 10 and per 30 minutes for presentation) with mutual adjustment for forceps, perineal grade, epidural use, parity, age, BMI, birth weight, and days postpartum. Because forceps and perineal injury can lie downstream of a prolonged second stage, the primary model including them is complemented by a pre-specified model restricted to pre-labor and baseline covariates (Supplementary Table S5). Macrosomia ($\geq 4,000$ g) was not included in the primary model because it overlaps informationally with continuous birth weight and is partially a downstream consequence of labor; a model replacing birth weight with macrosomia is reported as a sensitivity analysis (Supplementary Table S5). Predicted cumulative differences were computed against a 20-minute second stage, a common duration in spontaneous,

non-instrumented births in our data. Because a dose–response claim should not rest on an assumed shape, we tested the linear specification against restricted cubic splines (knots at 15/30/60/120 min) by likelihood-ratio tests and AIC. To probe the epidural-mediated interpretation, we formally tested the epidural $\times$ second-stage interaction, and to examine whether the association decayed with postpartum recovery we tested the second-stage $\times$ early-assessment (≤ 42 days) interaction. Collinearity was examined with variance inflation factors and pairwise correlations. Sensitivity analyses excluded durations >180 minutes, excluded forceps births, restricted the sample to intact/minor perineal trauma, restricted to testing within 42 days postpartum, and examined the slope separately in nulliparous and multiparous women (Supplementary Table S1a).

Model diagnostics. Residual normality was assessed with the Jarque–Bera test and Q–Q plots, heteroskedasticity with the Breusch–Pagan test, and influential observations with Cook’s distance and DFBETA. Because residuals departed from normality (Section 3.2), all primary models are reported with HC3 heteroskedasticity-robust standard errors (Supplementary Table S7).

Missing data. The complete-case restriction excluded 114 of the 1,241 eligible women (9.2%), driven mainly by missing forceps ($n = 81$) and fast-twitch ($n = 18$) fields. Because missingness could be informative, we did not treat complete-case analysis as sufficient; we compared included and excluded women on all observed characteristics (Supplementary Table S4) and refit the primary model after multiple imputation by chained equations (50 imputed datasets): binary variables (forceps, epidural, parity) were imputed by stochastic regularized logistic regression, continuous variables by linear regression with predictive mean matching, and perineal grade as an ordinal variable with rounding to the nearest level; estimates and standard errors were pooled using Rubin’s rules [19].

Epidural association (aim 2). We estimated the crude epidural association and then adjusted sequentially for second-stage duration and for the remaining covariates, reading the attenuation pattern before fitting any formal decomposition. In an exploratory mediation analysis, second-stage duration was treated as the mediator: the a-path (epidural $\rightarrow$ duration) and the b-path (duration $\rightarrow$ outcome, covariate-adjusted) were estimated by linear regression; the indirect effect was $a \times b$ with a bootstrap 95% CI (1,000 resamples), and the direct effect came from the

fully adjusted model [20]. This decomposition was exploratory and is presented as a statistical decomposition compatible with a duration-mediated pathway, not as causal proof. We decided a priori that if the direct and indirect effects had opposite signs, we would report the two components separately rather than a “proportion mediated,” which is uninterpretable in that situation. Analyses used two-sided $p < 0.05$ in Python 3.12/statsmodels. Unless otherwise stated, all analyses use the complete-case sample ($n = 1,127$); denominators for every table, figure, and subset analysis are reported alongside each estimate.

3. Results

3.1 Cohort

The 1,127 analyzed deliveries (885 with and 242 without epidural analgesia; Table 1) had a mean maternal age of 30.5 ± 3.7 years; 34.7% were multiparous, 78.5% received epidural analgesia, and 3.5% were forceps-assisted. Mean birth weight was $3,294 \pm 396$ g. The median interval from delivery to sEMG assessment was 49 days (IQR 43–61; range 15–195), and 247 women (21.9%) were assessed within 42 days postpartum. The median second stage lasted 51 minutes (IQR 27–89); 492 women (43.7%) had a second stage of 60 minutes or longer and 159 (14.1%) of 120 minutes or longer (distribution in Supplementary Table S2). Mean fast-twitch amplitude was 29.2 ± 14.3 μV . Women with epidural analgesia had substantially longer second stages (median 62 versus 19 minutes), a pattern that motivated the subsequent mediation analysis. Women included in the analytic sample were broadly similar to the 114 women excluded for incomplete data (all standardized mean differences ≤ 0.13 ; Supplementary Table S4).

3.2 The per-minute association with second-stage duration

Each additional minute of second stage was associated with -0.039 μV of fast-twitch amplitude (95% CI -0.059 to -0.019 , $p < 0.001$): -0.39 μV per 10 minutes, or about 4.0% of the cohort mean per 30 minutes (Table 2, Figure 2). Sustained-contraction and endurance amplitudes behaved almost identically (-0.027 and -0.028 $\mu\text{V}/\text{min}$, both $p < 0.001$), which argues against an artifact isolated to one phase of the protocol. Against a 20-minute reference, predicted fast-twitch differences were -1.56 μV at 60 minutes and -3.89 μV at 120 minutes, the latter corresponding to roughly 0.27 SD.

Across all three outcomes, no statistical evidence of departure from linearity was identified within the observed data (spline $P = 0.549, 0.712, \text{ and } 0.914$; linear models yielded lower AIC); the spline fit is shown with its 95% confidence band alongside the duration distribution, which is sparse beyond 180 minutes (Figure 5). The association was robust across all sensitivity analyses (Figure 6, Supplementary Table S1a): excluding durations beyond 180 minutes ($\beta = -1.19 \mu\text{V}/30 \text{ min}$), excluding forceps births (-1.33), and restricting to intact or minor perineal trauma (-1.18 ; all $p \leq 0.002$). Multiple imputation (50 imputations), which readmitted the 114 women with incomplete records, produced a nearly identical pooled estimate ($-1.03 \mu\text{V}$ per 30 min, 95% CI -1.62 to -0.44 , $p < 0.001$), making selection bias from documentation gaps an implausible explanation. In women tested within 42 days of delivery the slope was steeper ($-2.20 \mu\text{V}$ per 30 min, 95% CI -3.54 to -0.86 , $p = 0.001$, $n = 247$) than in women tested later ($-0.88 \mu\text{V}$ per 30 min, 95% CI -1.52 to -0.25 , $p = 0.006$, $n = 880$), and the formal second-stage $\times$ early-assessment interaction was significant ($P = 0.042$; Supplementary Table S3); this subgroup finding should nevertheless be read as hypothesis-generating. Model diagnostics supported the reported inference: heteroskedasticity was not detected (Breusch–Pagan $P = 0.221$) and no observation approached influence thresholds (maximum Cook’s distance 0.085); residuals were non-normal (Jarque–Bera $P < 0.001$), so all primary estimates use HC3 robust standard errors, which changed no conclusion (Supplementary Table S7).

3.3 Forceps, perineal grade, and birth weight

Forceps delivery carried the largest single association in the mutually adjusted model: $-6.08 \mu\text{V}$ of fast-twitch amplitude (95% CI -10.70 to -1.47 , $p = 0.010$) and $-3.94 \mu\text{V}$ of sustained contraction ($p = 0.016$), essentially unchanged under imputation ($-6.31 \mu\text{V}$, $p = 0.008$). Forceps births were uncommon in this cohort ($n = 39$, 3.5% of the analytic sample), so this estimate is imprecise. Collinearity does not explain these estimates: all variance inflation factors were ≤ 1.63 (Supplementary Table S8), and forceps use correlated only weakly with duration (Spearman $r = 0.15$); epidural use and duration correlated moderately ($r = 0.42$), which is expected if duration genuinely lies on the causal path from epidural to outcome rather than indicating redundant variables. Perineal injury grade showed no independent association ($+0.54 \mu\text{V}$ per grade, $p = 0.438$). The null for perineal grade was not an artifact of lumping episiotomy with minor tears: modeling episiotomy as a separate indicator changed nothing (tear grade $p = 0.721$; episiotomy β

= $-0.26 \mu\text{V}$, $p = 0.887$; Supplementary Table S1a). Replacing continuous birth weight with macrosomia ($\geq 4,000 \text{ g}$) left the second-stage estimate identical ($-1.17 \mu\text{V}$ per 30 min; Supplementary Table S5), and removing the potentially downstream variables (forceps, perineal grade) strengthened it slightly ($-1.38 \mu\text{V}$ per 30 min).

3.4 Epidural analgesia and the mediation decomposition

The crude association between epidural analgesia and fast-twitch amplitude was small and non-significant ($\beta = -0.58 \mu\text{V}$, $p = 0.577$). Adjusting for second-stage duration did not attenuate it toward zero—it reversed the sign to $+1.41 \mu\text{V}$ —and the fully adjusted direct effect was $+0.93 \mu\text{V}$ (95% CI -1.29 to $+3.15$, $p = 0.413$; Table 3, Figure 3). Sustained contraction ($+0.65 \mu\text{V}$, $p = 0.411$) and resting tone ($-0.001 \mu\text{V}$, $p = 0.996$) showed the same pattern, and imputation left the direct estimate unchanged ($+1.03 \mu\text{V}$, $p = 0.353$). The 95% CI of the direct effect spanned -1.29 to $+3.15 \mu\text{V}$; no prespecified clinically important threshold exists for this outcome, so we do not claim that clinically meaningful harm is excluded.

The exploratory mediation analysis (Figure 4) clarifies the pattern. Epidural labors had 20.3-minute longer second stages on average (a-path, $p < 0.001$), and the indirect effect through duration was $-0.79 \mu\text{V}$ (95% CI -1.26 to -0.40)—significant. The covariate-adjusted total effect is $+0.14 \mu\text{V}$ (95% CI -2.06 to $+2.34$, $p = 0.901$), exactly the sum of the direct and indirect components, as the algebra of linear mediation requires. One clarification matters for reading Table 3: the crude association ($-0.58 \mu\text{V}$) is not the total effect of this decomposition. The decomposition is built on covariate-adjusted models, and the $0.72 \mu\text{V}$ gap between the crude association and the adjusted total reflects confounding by baseline covariates (women receiving epidural analgesia were younger and more often nulliparous), not the mediated pathway. The epidural $\times$ second-stage interaction was not significant ($P = 0.131$; Supplementary Table S3), consistent with the no-interaction assumption of the decomposition, although power for interactions is limited. In this setting, the usual ‘proportion mediated’ metric is uninterpretable because the direct effect ($+0.93 \mu\text{V}$, non-significant) and the indirect effect ($-0.79 \mu\text{V}$, significant) have opposite signs; we therefore report the two components separately, as prespecified in our analysis plan. The substantive reading is simpler than the statistics: in exploratory mediation analysis, the lower sEMG amplitudes observed in epidural labors were statistically accounted for by longer second-stage duration, not by any detectable direct

association of epidural analgesia with postpartum muscle function. The direct estimate, descriptively positive and non-significant, supports neither harm nor benefit..

4. Discussion

4.1 Principal findings

This is one of the larger sEMG cohorts with linked intrapartum records, and to our knowledge the first analyzed with continuously modeled second-stage duration and an exploratory mediation decomposition of the epidural association. Second-stage duration showed a linear, per-minute association with early postpartum pelvic floor contractile function—about $-0.39 \mu\text{V}$ of fast-twitch amplitude per 10 minutes. The estimate was stable across imputation, functional-form tests, and a battery of subset analyses, and it was steeper in the early (≤ 42 -day) postpartum window (formal interaction $P = 0.042$). Epidural analgesia showed no direct neuromuscular association for any sEMG outcome; the significant signal ran entirely through its association with longer second-stage duration. Two subsidiary findings were notable: forceps retained a large independent association even after accounting for duration, whereas perineal injury grade—clinically the most visible form of birth trauma—carried no independent association.

4.2 How much does a fraction of a microvolt matter?

Skepticism is warranted here. The per-minute association is small—about 0.08 SD per 30 minutes—and most women recover from an uncomplicated vaginal birth without symptoms [21]. We would caution against reading our dose–response as a map of imminent disease. The more defensible interpretation is that sEMG amplitude indexes the neuromuscular reserve available for continence and organ support, and that early differences in this reserve may compound across subsequent births and age-related physiological decline. That interpretation is not purely speculative: in the only long-term cohort to measure it directly, lower pelvic floor muscle strength roughly a decade after childbirth predicted a higher subsequent incidence of pelvic floor disorders [22], and delivery-mode differences in these disorders remain detectable five to twenty years out [2,4,23]. Whether microvolt-scale differences at six weeks behave the same way is an open empirical question, and we frame it as such. Two features of our data nonetheless argue

against dismissing the association as trivial. The slope was steeper within the early postpartum window, consistent with partially reversible acute injury drawing down reserve before recovery—a trajectory that imaging studies of levator avulsion also describe [24]; and the population distribution of second-stage duration is wide (median 51 minutes, 43% longer than one hour), so a small per-minute slope translates into appreciable between-woman differences at the extremes. Consider two otherwise similar nulliparas screened at six weeks, one delivered after a 40-minute second stage and the other after 100 minutes—both unremarkable durations in our data. The model predicts that the second woman arrives at her screening visit with fast-twitch amplitudes about 2.3 μV lower. Individually modest; at the scale of a delivery ward, systematic.

4.3 A dose, not a threshold—and a tension with the symptom literature

Dichotomous definitions of “prolonged” second stage embed a threshold assumption that our data do not support: no statistical evidence of departure from linearity was identified between 1 and 293 minutes, with no acceleration at 60 or 120 minutes, although we acknowledge that power to detect non-linearity is limited at the tails where observations are sparse. The honest translation for practice is therefore an exchange rate, not a new cutoff, and it should inform—rather than dictate—the balance between expectant and operative management when progress slows, alongside the maternal and neonatal considerations that rightly dominate that decision [7,25].

Our continuous signal sits in some tension with the questionnaire literature, which has generally found weak or absent associations between second-stage duration and self-reported pelvic floor symptoms except at extremes [26–28]. O’Leary and Keane, for instance, found that only second stages beyond 120 minutes carried more bladder symptoms at three months [26]; Rogers and colleagues, prospectively following nulliparas, attributed only a modest share of postpartum symptoms to the second stage [29]; and in the Swedish population cohort, among women already past three hours, further duration added little unless delivery was completed by vacuum [28]. Trial data on how the second stage is managed point the same way: delaying pushing lengthens the stage without improving delivery outcomes [30], and instrumental success, not the clock, dominates its morbidity [31]. Older epidemiology was equally inconsistent [32].

We do not think these findings contradict ours. Symptoms require that physiological damage cross a perceptual and functional threshold; sEMG catches the dose accumulating below it. The two instruments are measuring different points on the same pathway, and our results may help explain why symptom studies see signal only at the tail—the tail is where the accumulated dose finally crosses into dysfunction. The large independent forceps association (≈ 0.4 SD) fits comfortably with the imaging literature, in which instrumental—especially forceps—delivery is the dominant obstetric determinant of levator ani avulsion [33–35]. The null association for perineal grade, robust to however we handled episiotomy, is harder to reconcile with intuition but echoes studies that failed to link tear severity or episiotomy to later muscle function [36–38]; one plausible reading is that tear grade records the anatomy of an achieved vaginal birth more than it measures injury to the muscle itself, and that repair quality matters more than the label. Importantly, because no third- or fourth-degree tears occurred in this cohort, this null finding cannot be extrapolated to severe perineal trauma: the effective contrast in our data is among intact/minor trauma, episiotomy, and second-degree tears.

4.4 Epidural analgesia: an answer to a narrower question

Prior electromyographic work was small ($n \approx 124$) and, crucially, unadjusted for labor duration [12]. With a substantially larger sample, mutual adjustment, and imputation-consistent results, we found no direct epidural association with any contractile or resting outcome. Absence of evidence is not evidence of absence: the confidence bounds (-1.29 to $+3.15$ μV) are compatible with small effects in either direction, and no validated clinically important difference exists for sEMG amplitude. Given how firmly epidural lengthens the second stage—by 20.3 minutes in our data, squarely within the 15–30 minutes reported by trials and meta-analyses [9–10,39]—an unadjusted comparison is almost guaranteed to mislead. Our exploratory decomposition suggests that unadjusted or partially adjusted estimates may attribute to the block a decrement that is statistically accounted for by longer labor.

This should not be read as a blank check. The same analysis says the longer labors that accompany neuraxial analgesia carry a measurable per-minute association with lower sEMG amplitudes, which makes time-aware second-stage management in epidural labors—timely assessment of descent, avoidance of passive expectancy—a hypothesis worth prospective testing, rather than any hesitation about offering the block. Whether modifying second-stage

management improves pelvic floor outcomes requires prospective evaluation, and any such intervention must be weighed against operative delivery and neonatal risks. As neuraxial analgesia use continues to rise in China [40], distinguishing the association of the anesthetic from the management of the labor it accompanies becomes increasingly important.

4.5 Strengths and limitations

The study's strengths include its size—one of the larger sEMG series with linked intrapartum records—and its minute-level exposure data. We also performed mutual adjustment across intrapartum factors, formally tested linearity and the key interactions, verified robustness by 50-dataset imputation, and conducted a prespecified exploratory mediation analysis that reported direct and indirect effects separately when their signs disagreed.

Several limitations should be acknowledged. This is a single-center retrospective study of a self-referred screening population, and selection into screening could bias absolute amplitudes even if the dose–response slope survives. Per-unit associations are modest. sEMG measures function, not structure; we cannot see levator avulsion, and our forceps estimate presumably blends muscle and nerve injury mechanisms that imaging would separate [6,41]. Perineal grade and analgesia type came from routine records, with the usual risk of misclassification. We could not retrieve individual-level details of neuraxial analgesia—exact initiation time relative to cervical dilatation, total exposure duration, and protocol adherence—so the exposure was defined by the institutional standard protocol in place throughout the study period, and residual exposure misclassification cannot be excluded. Repeat-visit linkage relied on names and telephone numbers in routine records; although we retained only the first examination per woman, imperfect identifier quality means residual duplicate participation cannot be fully excluded. Two further gaps matter more. We lacked data on fetal station and position at the onset of the second stage and maternal pushing technique, both plausible confounders of the duration–injury association; and we had no measure of pre-pregnancy or antenatal pelvic floor function, so if women with lower baseline function both labor longer and test lower postpartum, our reading of the slope would be overstated—residual confounding by baseline fitness cannot be excluded. Mediation analysis in observational data rests on no-unmeasured-confounding assumptions that our design cannot verify, and we present this exploratory decomposition as explanatory, not as causal proof. Finally, our outcomes are early postpartum muscle physiology (median 49 days),

not incontinence or prolapse; whether this physiological decrement matures into clinical disease requires the longitudinal follow-up we are now undertaking.

5. Conclusions

Second-stage duration was linearly associated with early postpartum pelvic floor muscle electrophysiology—about $-0.4 \mu\text{V}$ of fast-twitch amplitude per 10 minutes—with no threshold at conventional cutoffs. No direct association between epidural analgesia and postpartum muscle function was detected; the lower sEMG amplitudes observed in epidural labors were statistically accounted for by their longer second-stage duration in exploratory mediation analysis. Because the outcome is an early physiological surrogate and the design observational, these findings are hypothesis-generating: whether time-aware second-stage management improves pelvic floor outcomes requires prospective evaluation, and any such intervention must be weighed against operative delivery and neonatal risks.

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 committee waived the requirement for informed consent because of the retrospective study design and the exclusive use of anonymized routine clinical data. All procedures were conducted in accordance with the Declaration of Helsinki and the Measures for Ethical Review of Biomedical Research Involving Humans (China).

Consent for publication

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

Availability of data and materials

The de-identified analytic dataset and the complete scripted analysis pipeline are available from the corresponding author on reasonable request and with appropriate institutional approval; the raw clinical records are not publicly available due to institutional privacy policies.

Competing interests

The authors declare that they have no competing interests.

Funding

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

Authors' contributions

Jiaping Lu was responsible for data collection, database sorting, preliminary statistical analysis and manuscript drafting. Qiulian Xu participated in clinical data screening, case inclusion and exclusion judgment, and revision of the results section. Heng Xu 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.

Acknowledgments

The authors gratefully acknowledge the contributions of all obstetricians, midwives, and nursing staff whose clinical work made this study possible.

References

1. Nygaard I, Barber MD, Burgio KL, et al. Prevalence of symptomatic pelvic floor disorders in US women. *JAMA*. 2008;300(11):1311-1316.
2. Blomquist JL, Muñoz A, Carroll M, Handa VL. Association of delivery mode with pelvic floor disorders after childbirth. *JAMA*. 2018;320(23):2438-2447.
3. Rortveit G, Daltveit AK, Hannestad YS, Hunskaar S. Urinary incontinence after vaginal delivery or cesarean section. *N Engl J Med*. 2003;348(10):900-907.
4. Gyhagen M, Bullarbo M, Nielsen TF, Milsom I. The prevalence of urinary incontinence 20 years after childbirth: a national cohort study in singleton primiparae after vaginal or caesarean delivery. *BJOG*. 2013;120(2):144-151.
5. Lien KC, Mooney B, DeLancey JOL, Ashton-Miller JA. Levator ani muscle stretch induced by simulated vaginal birth. *Obstet Gynecol*. 2004;103(1):31-40.

6. DeLancey JOL, Kearney R, Chou Q, Speights S, Binno S. The appearance of levator ani muscle abnormalities in magnetic resonance images after vaginal delivery. *Obstet Gynecol.* 2003;101(1):46-53.
7. American College of Obstetricians and Gynecologists; Society for Maternal-Fetal Medicine. Obstetric Care Consensus No. 1: Safe prevention of the primary cesarean delivery. *Obstet Gynecol.* 2014;123(3):693-711.
8. Gimovsky AC, Berghella V. Randomized controlled trial of prolonged second stage: extending the time limit vs usual guidelines. *Am J Obstet Gynecol.* 2016;214(3):361.e1-361.e6.
9. Anim-Somuah M, Smyth RMD, Cyna AM, Cuthbert A. Epidural versus non-epidural or no analgesia for pain management in labour. *Cochrane Database Syst Rev.* 2018;(5):CD000331.
10. Cheng YW, Shaffer BL, Nicholson JM, Caughey AB. Second stage of labor and epidural use: a larger effect than previously suggested. *Obstet Gynecol.* 2014;123(3):527-535.
11. Chi X, Yu S, Zhu K, Chen Y, Chu Y, Chen X. Influence of different obstetric factors on early postpartum pelvic floor function in primiparas after vaginal delivery. *Int J Womens Health.* 2023;15:81-90.
12. Jiang Q, Jin Z, Wang W, Ji Q, Qi C. Retrospective study to assess the effect of epidural analgesia on labor progress and women's pelvic floor muscle from the perspective of electromyography. *J Matern Fetal Neonatal Med.* 2023;36(1):221-228.
13. Glazer HI, Romanzi L, Polaneczky M. Pelvic floor muscle surface electromyography. Reliability and clinical predictive validity. *J Reprod Med.* 1999;44(9):779-782.
14. Frawley H, Shelly B, Morin M, et al. An International Continence Society (ICS) report on the terminology for pelvic floor muscle assessment. *Neurourol Urodyn.* 2021;40(5):1217-1260.
15. Haylen BT, de Ridder D, Freeman RM, et al. An International Urogynecological Association (IUGA)/International Continence Society (ICS) joint report on the terminology for female pelvic floor dysfunction. *Neurourol Urodyn.* 2010;29(1):4-20.
16. Van Geelen H, Ostergard D, Sand P. A review of the impact of pregnancy and childbirth on pelvic floor function as assessed by objective measurement techniques. *Int Urogynecol J.* 2018;29(3):327-338.

17. Botelho S, Riccetto C, Herrmann V, Pereira LC, Amorim C, Palma P. Impact of delivery mode on electromyographic activity of pelvic floor: comparative prospective study. *Neurourol Urodyn*. 2010;29(7):1258-1261.
18. Zhao Y, Zou L, Xiao M, Tang W, Niu HY, Qiao FY. Effect of different delivery modes on the short-term strength of the pelvic floor muscle in Chinese primipara. *BMC Pregnancy Childbirth*. 2018;18(1):275.
19. Rubin DB. *Multiple Imputation for Nonresponse in Surveys*. New York: John Wiley & Sons; 1987.
20. VanderWeele TJ. Mediation analysis: a practitioner's guide. *Annu Rev Public Health*. 2016;37:17-32.
21. Thom DH, Rortveit G. Prevalence of postpartum urinary incontinence: a systematic review. *Acta Obstet Gynecol Scand*. 2010;89(12):1511-1522.
22. Blomquist JL, Carroll M, Muñoz A, Handa VL. Pelvic floor muscle strength and the incidence of pelvic floor disorders after vaginal and cesarean delivery. *Am J Obstet Gynecol*. 2020;222(1):62.e1-62.e8.
23. Handa VL, Blomquist JL, Knoop LR, Hoskey KA, McDermott KC, Muñoz A. Pelvic floor disorders 5-10 years after vaginal or cesarean childbirth. *Obstet Gynecol*. 2011;118(4):777-784.
24. van Delft KW, Thakar R, Sultan AH, IntHout J, Kluivers KB. The natural history of levator avulsion one year following childbirth: a prospective study. *BJOG*. 2015;122(9):1266-1273.
25. Cheng YW, Hopkins LM, Caughey AB. How long is too long: does a prolonged second stage of labor in nulliparous women affect maternal and neonatal outcomes? *Am J Obstet Gynecol*. 2004;191(3):933-938.
26. O'Leary BD, Keane DP. Effect of the length of the second stage of labor on pelvic floor dysfunction. *Am J Obstet Gynecol MFM*. 2023;5(2):100795.
27. McKenzie CM, Woolfolk CL, Rieger MM, et al. Impact of the duration of the second stage of labor on postpartum pelvic floor symptoms. *Urogynecology (Phila)*. 2024;30(3):381-387.
28. Bergendahl S, Sandström A, Zhao H, Snowden JM, Brismar Wendel S. Pelvic floor dysfunction after intervention, compared with expectant management, in prolonged

second stage of labour: a population-based questionnaire and cohort study. *BJOG*. 2024;131(9):1279-1289.

29. 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):1145-1153.
30. Cahill AG, Srinivas SK, Tita AT, et al. Effect of immediate vs delayed pushing on rates of spontaneous vaginal delivery among nulliparous women receiving neuraxial analgesia: a randomized clinical trial. *JAMA*. 2018;320(14):1444-1454.
31. Aiken CE, Aiken AR, Brockelsby JC, Scott JG. Factors influencing the likelihood of instrumental delivery success. *Obstet Gynecol*. 2014;123(4):796-803.
32. Casey BM, Schaffer JI, Bloom SL, et al. Obstetric antecedents for postpartum pelvic floor dysfunction. *Am J Obstet Gynecol*. 2005;192(5):1655-1662.
33. Kearney R, Fitzpatrick M, Brennan S, et al. Levator ani injury in primiparous women with forceps delivery for fetal distress, forceps for second stage arrest, and spontaneous delivery. *Int J Gynaecol Obstet*. 2010;111(1):19-22.
34. Friedman T, Eslick GD, Dietz HP. Delivery mode and the risk of levator muscle avulsion: a meta-analysis. *Int Urogynecol J*. 2019;30(6):901-907.
35. Dietz HP, Lanzarone V. Levator trauma after vaginal delivery. *Obstet Gynecol*. 2005;106(4):707-712.
36. Sartore A, De Seta F, Maso G, Pregazzi R, Grimaldi E, Guaschino S. The effects of mediolateral episiotomy on pelvic floor function after vaginal delivery. *Obstet Gynecol*. 2004;103(4):669-673.
37. Leeman L, Rogers R, Borders N, et al. The effect of perineal lacerations on pelvic floor function and anatomy at 6 months postpartum in a prospective cohort of nulliparous women. *Birth*. 2016;43(4):293-302.
38. Bø K, Hilde G, Tennfjord MK, Engh ME. Does episiotomy influence vaginal resting pressure, pelvic floor muscle strength and endurance, and prevalence of urinary incontinence 6 weeks postpartum? *Neurourol Urodyn*. 2017;36(3):683-686.
39. Comparative Obstetric Mobile Epidural Trial (COMET) Study Group UK. Effect of low-dose mobile versus traditional epidural techniques on mode of delivery: a randomised controlled trial. *Lancet*. 2001;358(9275):19-23.

40. Meng X, Ye J, Qiao P, Ren T, Luo Q, Hu LQ, Zhang J. Labor neuraxial analgesia and its association with perinatal outcomes in China in 2015-2016: a propensity score-matched analysis. *Anesth Analg*. 2023;137(5):1047-1055.
41. Shek KL, Dietz HP. Intrapartum risk factors for levator trauma. *BJOG*. 2010;117(12):1485-1492.

Figure legends

Figure 1. Study flow diagram. Of 1,810 women screened, 31 twin pregnancies and 506 cesarean births were excluded, leaving 1,273 singleton vaginal deliveries; 25 lacked a parseable second-stage duration, and 7 repeat sEMG records from women already in the cohort were removed under the one-record-per-woman rule, leaving 1,241 eligible women; 114 had incomplete covariate data (predominantly the forceps field, $n = 81$), yielding the 1,127-woman complete-case analytic sample.

Figure 2. The per-minute association: observed binned means and adjusted linear fit of fast-twitch amplitude against second-stage duration ($n = 1,127$).

Figure 3. Attenuation sequence for the epidural association with fast-twitch amplitude ($n = 1,127$), from the crude estimate to the fully adjusted direct effect, with 95% CIs.

Figure 4. Mediation analysis with all path coefficients ($n = 1,127$): $a = +20.3$ min ($P < 0.001$); $b = -0.039$ $\mu\text{V}/\text{min}$ ($P < 0.001$); indirect = -0.79 μV (95% CI -1.26 to -0.40 , significant, solid arrows); direct $c' = +0.93$ μV (95% CI -1.29 to $+3.15$, n.s., dashed gray arrow); covariate-adjusted total $c = +0.14$ μV (95% CI -2.06 to $+2.34$, n.s.), which equals $c' + a \times b$ exactly, as required in linear models. Direct and indirect effects of opposite sign; no proportion mediated is reported. The crude unadjusted association (-0.58 μV) appears in Table 3a and is not the mediation total.

Figure 5. Restricted cubic spline (with 95% confidence band) versus linear fits for three sEMG outcomes, with the distribution of second-stage durations annotated (linearity verification; $n = 1,127$).

Figure 6. Sensitivity analyses of the second-stage association with fast-twitch amplitude, including 50-dataset multiple imputation; analytic n and 95% CI shown for each row.

Figure S1. Directed acyclic graph (DAG) of the assumed causal structure. Unmeasured variables (baseline pelvic floor function, fetal station/position, pushing technique, oxytocin use) are shown as red dashed nodes.

Tables

Table 1. Baseline and sEMG characteristics by epidural use in the complete-case analytic sample (n = 1,127), with standardized mean differences (SMD).

Variable	Epidural (n=885)	No epidural (n=242)	SMD	P
Age, years	30.0 ± 3.5	32.2 ± 3.9	−0.59	<0.001
BMI, kg/m ²	22.7 ± 3.5	22.8 ± 2.9	−0.02	0.798
Parity, median (IQR)	1 (1–1)	2 (1–2)	−0.93	<0.001
Multiparous, %	25.0%	70.2%	−1.02	<0.001
Birth weight, g	3290.8 ± 383.8	3304.3 ± 437.2	−0.03	0.637
Days from delivery to sEMG, median (IQR)	49 (43–61)	49 (43–65)	−0.03	0.943
Second-stage duration, min, median (IQR)	62 (37–99)	19 (11–41)	0.96	<0.001
Forceps, %	4.1%	1.2%	0.18	0.053
Fast-twitch max, μV	29.0 ± 14.1	29.6 ± 15.1	−0.04	0.577
Sustained mean, μV	18.9 ± 9.8	19.2 ± 10.6	−0.02	0.773
Endurance mean, μV	16.1 ± 8.9	16.5 ± 9.7	−0.04	0.581
Pre-resting tone, μV, median (IQR)	3 (2–6)	3 (2–5)	0.06	0.780

Table 2. Mutually adjusted associations of intrapartum exposures with three sEMG outcomes (HC3 robust standard errors), with predicted cumulative differences for a 60- and 120-minute second stage versus a 20-minute reference. The primary model adjusts for forceps,

perineal grade, epidural use, parity, age, BMI, birth weight, and days postpartum (macrosomia-free; see Supplementary Table S5).

Outcome	n	Exposure	β (μ V)	95% CI	P
Fast-twitch max	1127	Second stage, per 10 min	−0.39	−0.59 to −0.19	<0.001
Fast-twitch max	1127	Second stage, per 30 min	−1.17	−1.77 to −0.56	<0.001
Fast-twitch max	1127	Forceps delivery	−6.08	−10.70 to −1.47	0.010
Fast-twitch max	1127	Perineal injury, per grade	0.54	−0.83 to 1.92	0.438
Fast-twitch max	1127	Epidural analgesia	0.93	−1.29 to 3.15	0.413
Fast-twitch max	1127	Predicted loss at 60 min vs 20 min	−1.56	−2.36 to −0.75	—
Fast-twitch max	1127	Predicted loss at 120 min vs 20 min	−3.89	−5.90 to −1.88	—
Sustained mean	1120	Second stage, per 10 min	−0.27	−0.41 to −0.13	<0.001
Sustained mean	1120	Second stage, per 30 min	−0.81	−1.23 to −0.38	<0.001
Sustained mean	1120	Forceps delivery	−3.94	−7.16 to −0.72	0.016
Sustained mean	1120	Perineal injury, per grade	0.31	−0.65 to 1.27	0.531
Sustained mean	1120	Epidural analgesia	0.65	−0.90 to 2.20	0.411
Endurance mean	1122	Second stage, per 10 min	−0.28	−0.41 to −0.15	<0.001
Endurance mean	1122	Second stage, per 30 min	−0.85	−1.23 to −0.46	<0.001
Endurance mean	1122	Forceps delivery	−3.97	−6.92 to −1.03	0.008
Endurance	1122	Perineal injury, per grade	−0.11	−0.99 to 0.77	0.801

Outcome	n	Exposure	β (μV)	95% CI	P
mean					
Endurance mean	1122	Epidural analgesia	0.61	−0.81 to 2.03	0.399

Table 3. Epidural analyses (n = 1,127): (a) attenuation sequence for the association with fast-twitch amplitude; (b) mediation decomposition via second-stage duration (bootstrap, 1,000 resamples).

(a) Attenuation sequence for the epidural association with fast-twitch amplitude (μV)

Model	n	β (μV)	95% CI	P
Crude (unadjusted)	1127	−0.58	−2.61 to 1.46	0.577
Adjusted for second-stage duration	1127	1.41	−0.72 to 3.55	0.194
Fully adjusted (direct effect)	1127	0.93	−1.29 to 3.15	0.413
Total effect (covariate-adjusted)	1127	0.14	−2.06 to 2.34	0.901
Fully adjusted: sustained mean	1120	0.65	−0.90 to 2.20	0.411
Fully adjusted: endurance mean	1122	0.61	−0.81 to 2.03	0.399
Fully adjusted: pre-resting tone	1121	−0.001	−0.47 to 0.47	0.996

(b) Mediation decomposition via second-stage duration (bootstrap, 1,000 resamples)

Component	Estimate	95% CI	P
a-path: epidural → second stage	+20.3 min	—	<0.001
b-path: second stage → fast-twitch	−0.039 $\mu\text{V}/\text{min}$	—	<0.001
Indirect (mediated) effect	−0.79 μV	−1.26 to −0.40	bootstrap

Component	Estimate	95% CI	P
Direct effect	+0.93 μ V	−1.29 to +3.15	0.413
Total effect (covariate-adjusted; equals direct + indirect exactly in linear models)	+0.14 μ V	−2.06 to +2.34	0.901

Supplementary Material

Supplementary Table S1a. Sensitivity analyses of the second-stage association with fast-twitch amplitude (seven pre-specified subset analyses, each with analytic n). **Supplementary Table S1b.** Multiple-imputation pooled estimates (50 imputations) for the primary model. **Supplementary Table S2.** Distribution of second-stage duration in the analytic sample. **Supplementary Table S3.** Formal interaction tests: epidural $\times$ second-stage duration; second-stage duration $\times$ assessment within 42 days postpartum. **Supplementary Table S4.** Comparison of included versus excluded women, with standardized mean differences. **Supplementary Table S5.** Covariate-strategy analyses: primary model; model without downstream variables (forceps, perineal grade); model with macrosomia replacing continuous birth weight. **Supplementary Table S6.** Raw perineal-status categories as recorded, with the analysis coding. **Supplementary Table S7.** Model diagnostics for the primary model (residual normality, heteroskedasticity, influence statistics, robust versus classical standard errors). **Supplementary Table S8.** Variance inflation factors for the primary model. **Figure S1.** Directed acyclic graph of the assumed causal structure.

Enrolled postpartum women
N = 1,810



Excluded multiple gestation (n = 31)
N = 1,779



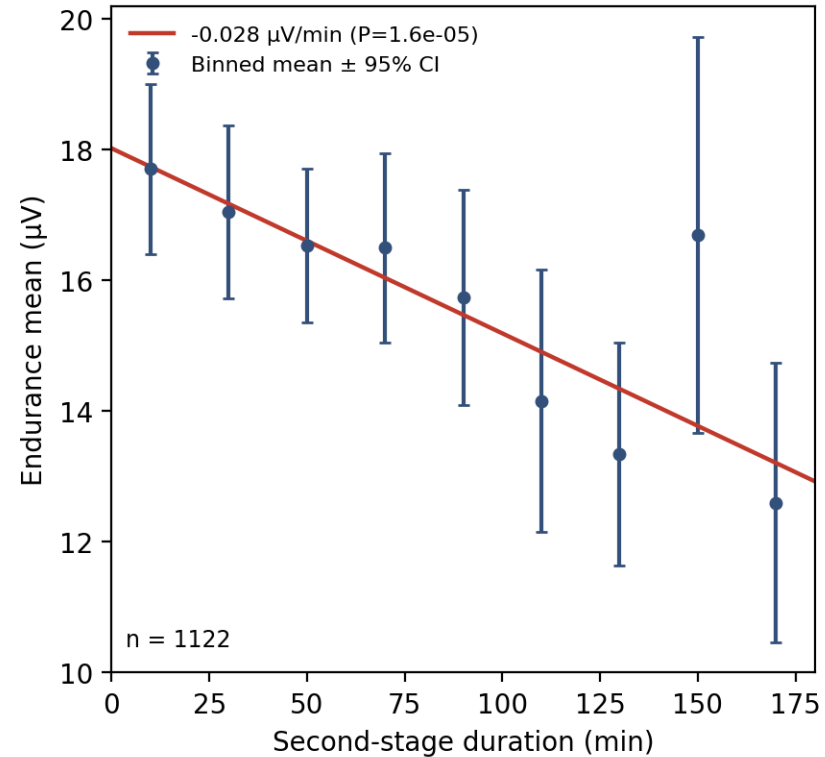
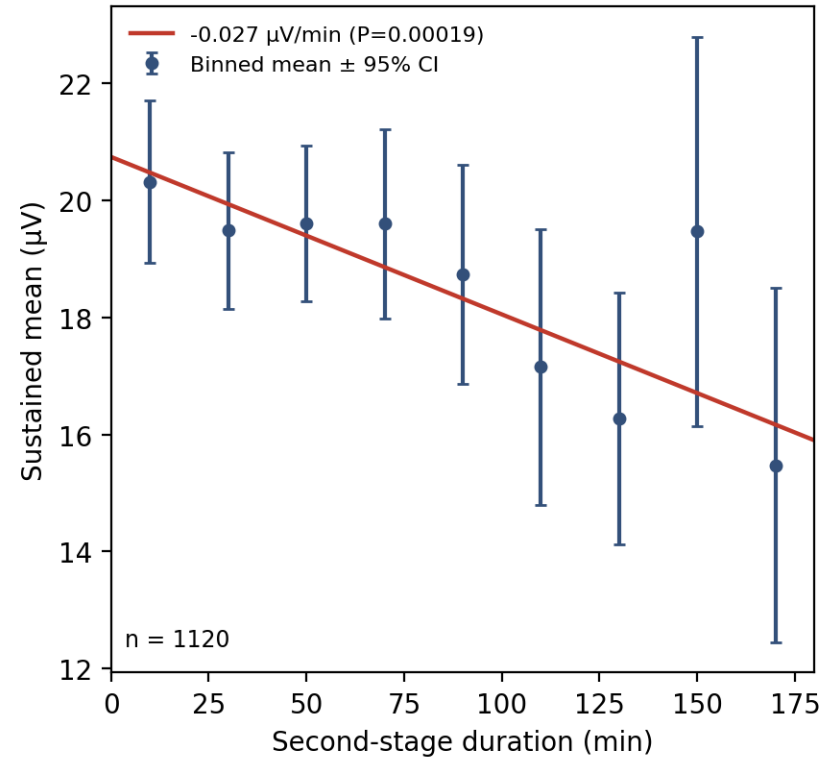
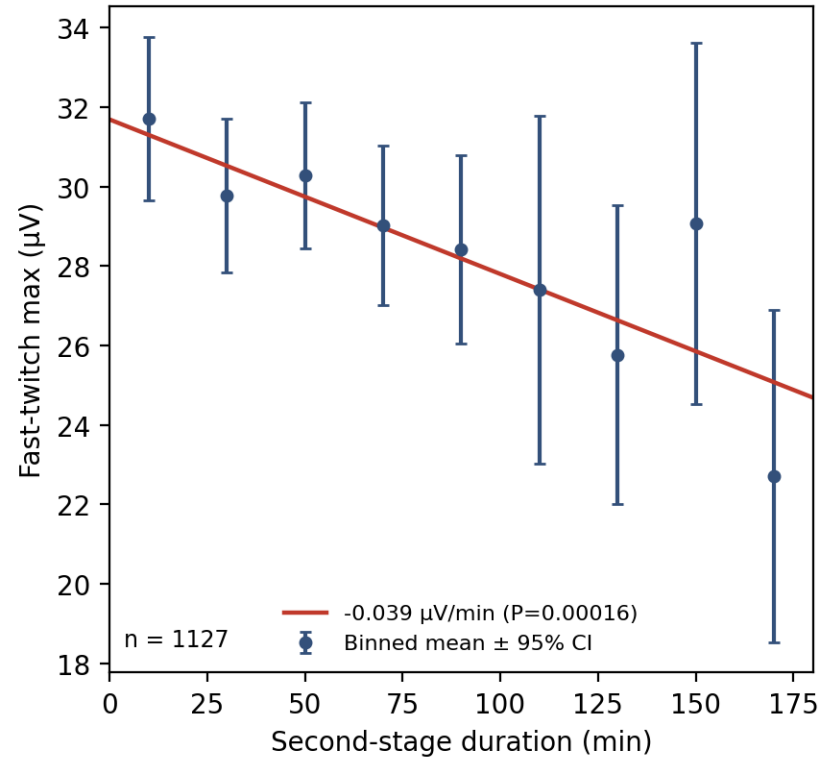
Vaginal deliveries only
N = 1,273

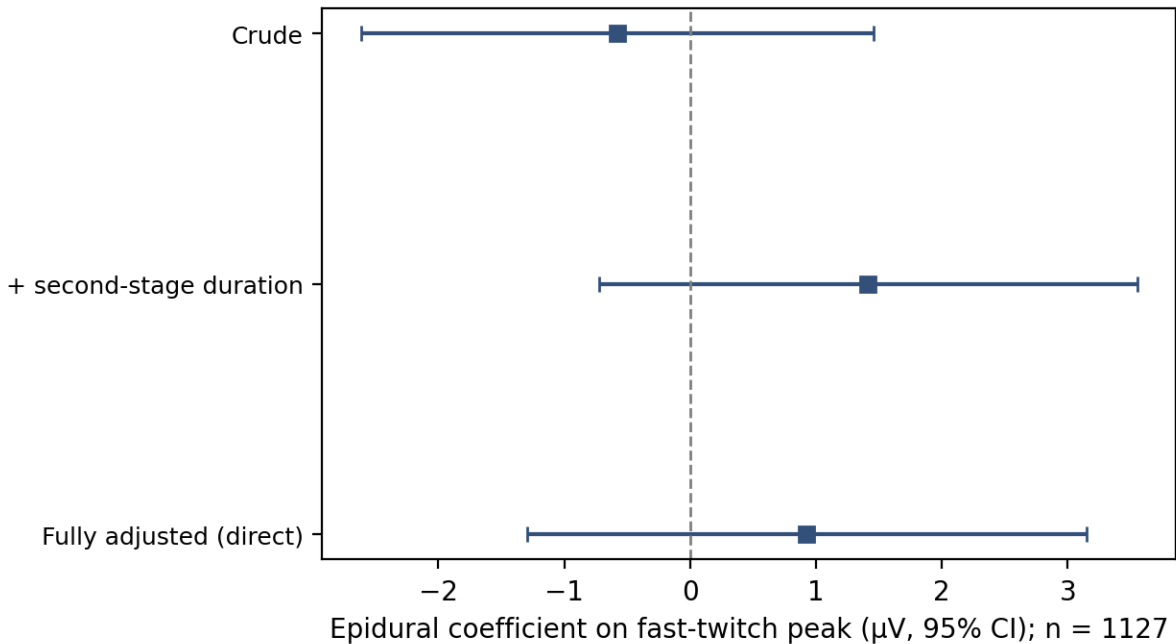


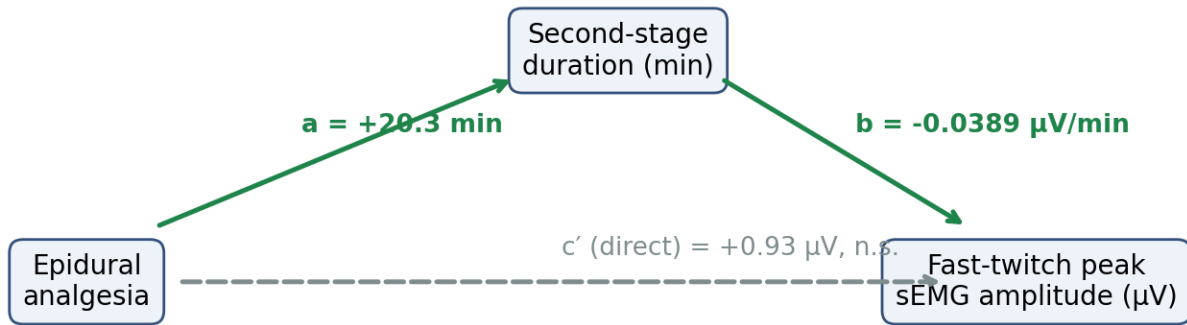
Valid second-stage duration
N = 1,248



Complete-case analytic sample
n = 1127 (MICE: N = 1,248)



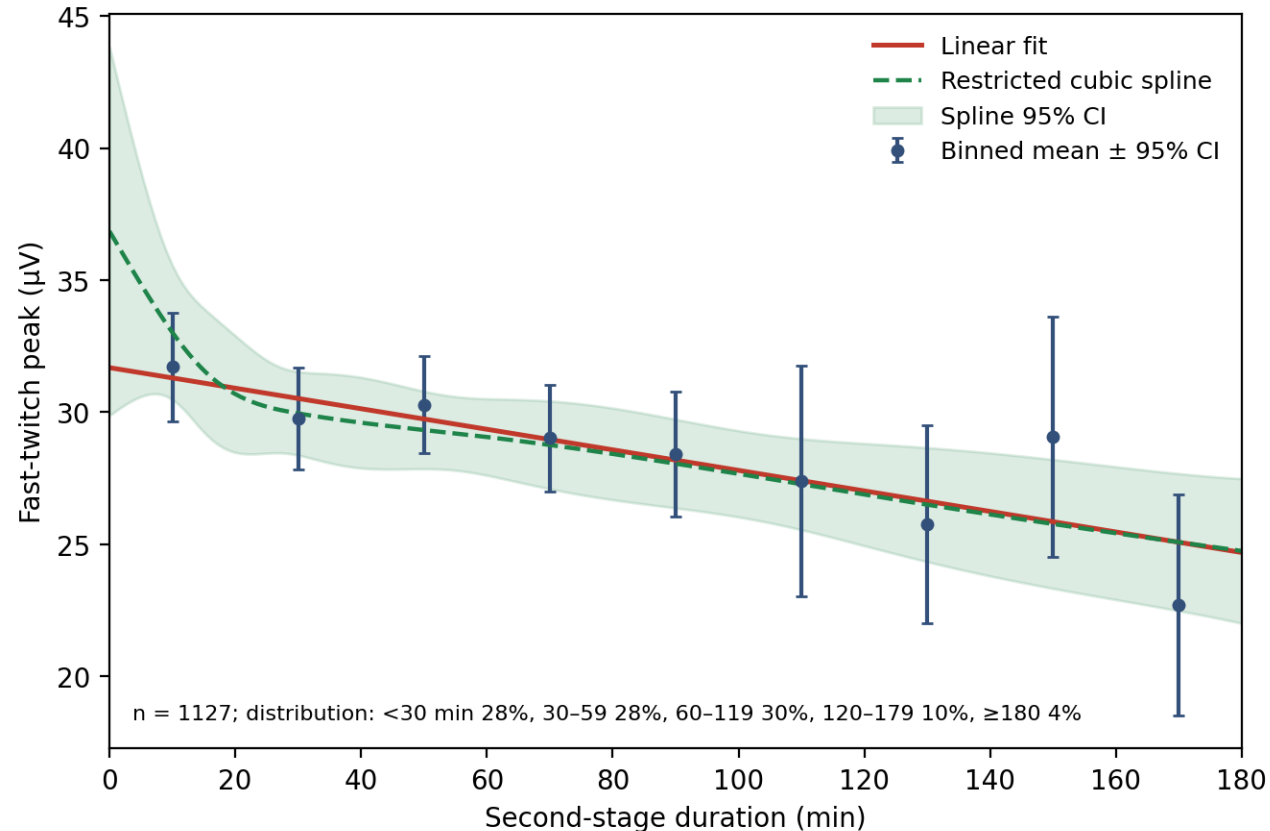


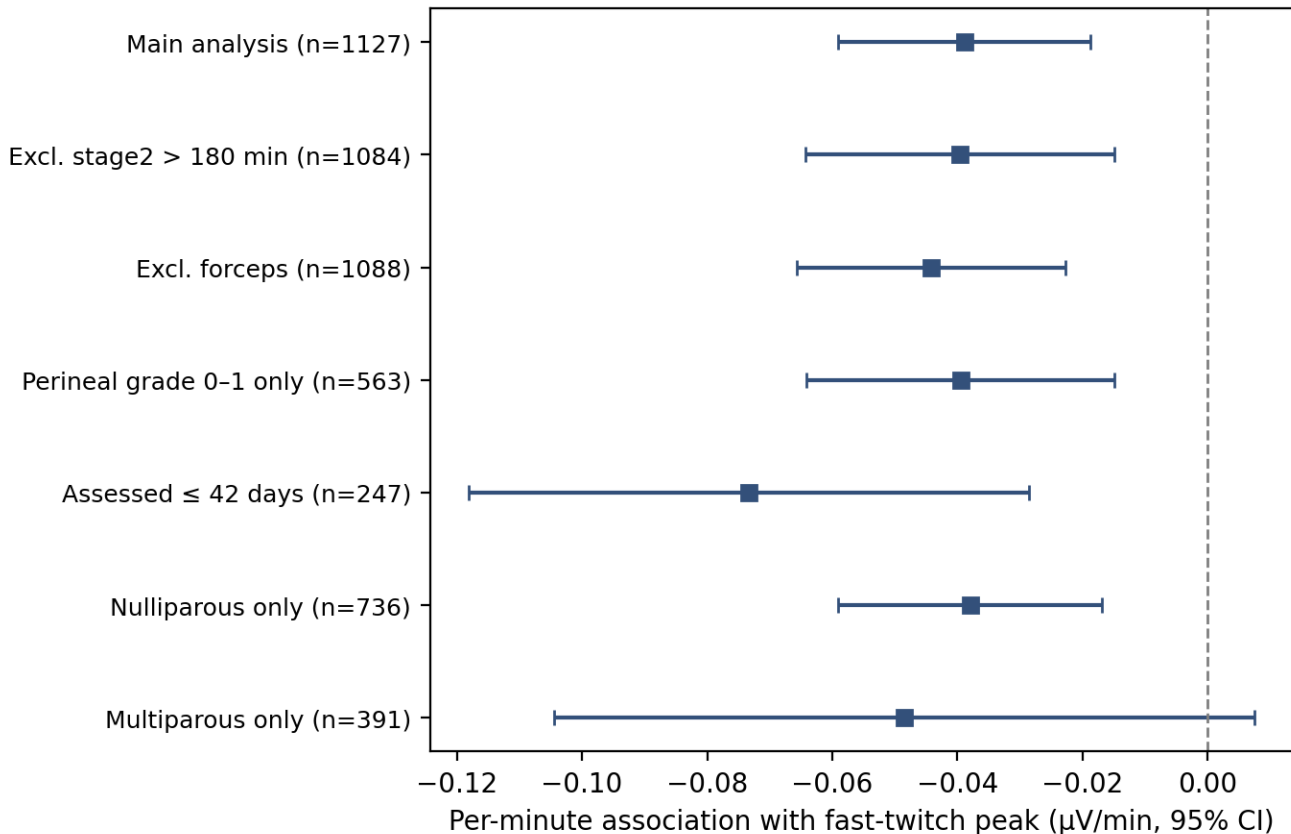


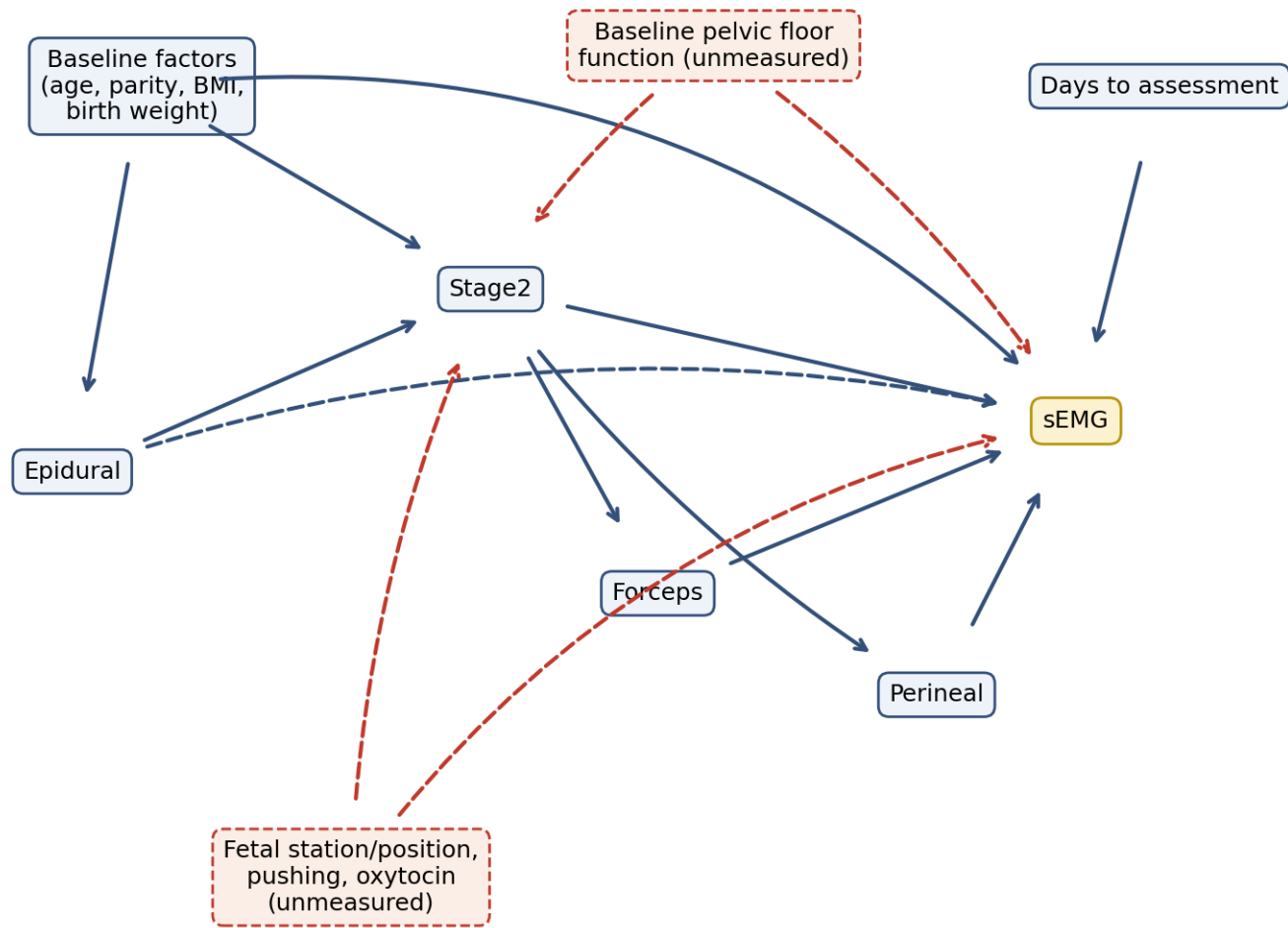
Indirect ($a \times b$) = -0.79 μV , 95% CI -1.26 to -0.40

Total effect (adjusted) $c = +0.14 \text{ μV}$, 95% CI -2.06 to 2.34 , $P = 0.90$, n.s.

$c = c' + a \times b$ exactly (linear models); $n = 1127$; exploratory decomposition







Dashed red: unmeasured (residual confounding cannot be excluded). Forceps and perineal trauma occur during/after the second stage and may lie on the causal pathway.

Supplementary Files

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

- [03SupplementaryMaterial.docx](#)