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ORIGINAL RESEARCH ARTICLE

Factors associated with a maternal lower-limb neurological deficit after vaginal delivery

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Abstract

Introduction: The etiology of lower-limb neurological deficit after vaginal delivery remains poorly understood. The objective herein was to identify factors associated with this maternal nerve injury after vaginal delivery.

Material and methods: A single-center, case–control (matching 1:4) study. Cases were women with a lower-limb neurological deficit that appeared immediately after vaginal delivery. Controls were randomly selected women who gave birth vaginally during the same period, without any deficit. Finally, to assess the rates of factors associated with these deficits, we studied them using a randomly selected 5% sample of the population with vaginal deliveries.

Results: During the 30-month study period, 31 cases were identified among 10 333 women who gave birth vaginally (0.3%, 95% CI 0.20–0.43); 124 controls were also included. After logistic regression, the presence of a neurological deficit after delivery was associated with second-stage labor duration (per hour odds ratio [OR] 3.67, 95% CI 2.09–6.44; OR per standard deviation increase 2.73, 95% CI 1.75–4.25, $p < 0.001$) and instrumental delivery (OR = 3.24, 95% CI 1.29–8.14, $p = 0.012$), with no interaction effect ($p = 0.56$). Extrapolation of these factors to a 5% sample of the overall population of women with vaginal births showed that the rate of these deficits would be very low for women with second-stage labor lasting up to 90 min without instrumental delivery (0.05%) but increased to 1.52% when these factors were combined (OR 33.1, 95% CI 9.4–116.9).

Conclusions: Following vaginal delivery, the onset of a neurological deficit is principally associated with the duration of second-stage labor and instrumental delivery.

KEYWORDS
birth, delivery, lower-limb neurological deficit, maternal nerve injury, second-stage labor, vaginal delivery

Abbreviations: CI, confidence interval; OR, odds ratio; VAS, visual analog scale.

1 | INTRODUCTION

Among the potential complications following vaginal delivery, nerve injury involving a lower-limb neurological deficit affects 0.1%–2% of women giving birth vaginally.[1–5] Such deficit may be sensory and/or motor and involve different nerve territories, most frequently those of the lateral femoral cutaneous nerve and the femoral nerve.[1] Most of these injuries are transient and last only several days (usually 1 week to a month).[1,3,4] Sometimes, however, they are still present at discharge and cause discomfort or even disability, representing a significant harm for a woman who is also caring for an infant. When the deficit continues for several months, it can also lead to litigation involving both the obstetrician and the anesthesiologist.[6]

The etiology of such delivery-associated injuries remains poorly understood. Numerous risk factors have been reported: these are a history of neurological problems,[3] nulliparity,[1,2,7] maternal obesity,[2] gestational age >41 weeks,[4] epidural analgesia,[5,7] late epidural initiation,[4] certain prolonged labor positions,[1,8] prolonged labor duration[2] (especially the second stage),[1] instrumental delivery,[1 4,7] large newborn birthweight,[4] and cephalopelvic disproportion.[8] Most are inconsistently found, and a few are controversial, especially epidural analgesia.

After observing several cases of severe neurological deficit at our center, we sought to assess such cases prospectively, over a 3-year period. We then conducted a case–control study to better understand the mechanism by which these sometimes impressive deficits occur. Our objective was to identify the principal factors related to onset of a lower-limb neurological deficit after vaginal delivery, in order to avoid their occurrence if possible.

Key message

Maternal nerve injury is associated with second-stage labor duration and instrumentation. These data may support limiting prolonged second-stage labor.

2 | MATERIAL AND METHODS

2.1 | Study design

This case–control study took place in our Level III university hospital maternity ward. Women with cesarean delivery, multiple pregnancy, in utero death, or pregnancy termination were excluded. The case group, women who spontaneously complained of a neurological deficit during their post-delivery hospitalization, were prospectively collected during the study period. Given the rarity of these deficits and the size of previously published series, we decided to consider a case–control study only when the number of cases was 30 or more, and the case collection lasted 3 years, from 2013 to 2015. We then compared the cases with a matching sample of control women randomly selected and retrospectively identified in the population of vaginal delivery during the 3-year consecutive period.

For the cases group, midwives managing postpartum care were asked to systematically record every complaint of neurological deficit and to notify the obstetrician in charge of the ward that a neurological examination should be performed to medically confirm the deficit. These cases have previously been described.[9] Briefly, more than 80% of the injuries reported were unilateral, two-thirds were sensory, and they primarily concerned the femoral territory. After neurological examination, the examining physician judged whether additional tests were necessary to exclude a cause other than birth injury. Only four women underwent lumbar imaging by computed tomography ($n = 1$) or magnetic resonance imaging ($n = 3$). Six women underwent electromyography in the first month postpartum, and two had anomalies on this examination. Overall, 42% recovered during the postpartum week, 69% recovered within 6 weeks, and 11% continued to have a neurological deficit 1 year postpartum, including one with a motor impairment that hindered her walking.[9] All women were followed by their general practitioner and all were contacted for the study.

The control group was randomly selected by cumulative sampling among women without neurological deficit who gave birth vaginally during the same period (four controls for each case).

2.2 | Research methods

All data were retrospectively collected among those that are systematically recorded in the medical record for all women giving birth in our center. We collected the following pregnancy and delivery data for the case and control groups: maternal age, parity, preconception body mass index, gestational age at birth, epidural analgesia, and cervical dilation when it was placed, analgesic efficacy scored according to the mean visual analog scale (VAS) on the partograph (coded as 0 = null for VAS 9–10; 1 = poor for VAS 7–8; 2 = average for VAS 4–6; 3 = effective for VAS 2–3; and 4 = very effective for VAS 0–1), duration of the first and second labor stages, longest maintained maternal labor position, and instrumentation (i.e., use of forceps or vacuum extractor). Neonatal data were also collected: arterial pH, birthweight, head circumference, and transfer to the intensive care unit.

2.3 | Statistical analyses

Categorical variables were expressed as numbers (percentages) and quantitative variables were expressed as means ± standard deviations or median (range) in cases of non-normal distribution; normality of the distribution was assessed graphically and by using Shapiro–Wilk test. The cases were compared with the controls with the χ^2 or Fisher's exact test, as appropriate, for the qualitative variables, with Student's t test for the normally distributed quantitative

variables, and with the Mann–Whitney *U* test for non-Gaussian quantitative variables.

To evaluate the factors associated with neurological deficit, we used logistic regression models with Firth's penalized likelihood approach to account for the smaller number of neurological deficit cases ($n = 31$); odds ratios (ORs) of neurological deficit were estimated from penalized logistic regression models as effect sizes with their 95% confidence intervals (CI). For all quantitative variables, we assessed the log-linearity assumption by using restricted cubic spline functions.[10] We then developed a multivariable parsimonious model of neurological deficit by considering all pre-specified candidate factors irrespective of their univariate associations with neurological deficit cases. Before developing the multivariable model, we checked the absence of co-linearity between candidate factors by calculating the variance inflation factors (all of which were <2.6).[11] Missing information in candidates factors (concerning six patients, 3.9%) was handled by single imputation using a regression switching approach (chained equation) under missing at random assumption with predictive matching method for quantitative variables and logistic regression models (binary, ordinal, or polynomial) for categorical variables.[12] To account for the number of candidate factors regarding the small number of cases and limit the risk of over-optimism, the parsimonious model was built using a bootstrap resampling procedure. The procedure consisted of creating bootstrap resamples[13,14] ($n = 200$) from the original data sets and applying in each replicate, a multivariable penalized logistic regression model including all candidate predictors and using an automatic forward selection procedure. For each candidate factor, the proportion of replicates in which that variable was retained in the final model with two-sided *p* value less than 0.20, was determined and the variables with at least 70% of replicates were retained to build the final multivariable model. To acknowledge the drawback of a forward-selection procedure, we repeated the bootstrap selection procedure using a backward selection procedure and found similar variable selection. We then examined the performance of the final multivariable model in terms of calibration using a Loess-based calibration plot and the Hosmer and Lemeshow goodness-of-fit test, and in terms of discrimination by calculating the c-statistic with 95% CI.[15]

Finally, to obtain a better picture of the risks of neurological deficit according to the risk factors ascertained in this case–control study, we randomly selected 5% of the women with vaginal deliveries who were eligible for inclusion in analyses, to extrapolate their results to the entire population of women with vaginal deliveries (Figure 1). For this 5% sample, we collected only factors that were selected to be associated with nerve deficit in multivariable analysis and determined, for each sub-category of women (after categorizing the second stage duration into group ≤90 min vs >90 min), the absolute risk of nerve deficit with 95% exact CI; we also reported the relative risk estimates using women with no instrumental delivery and second stage of 90 min or less

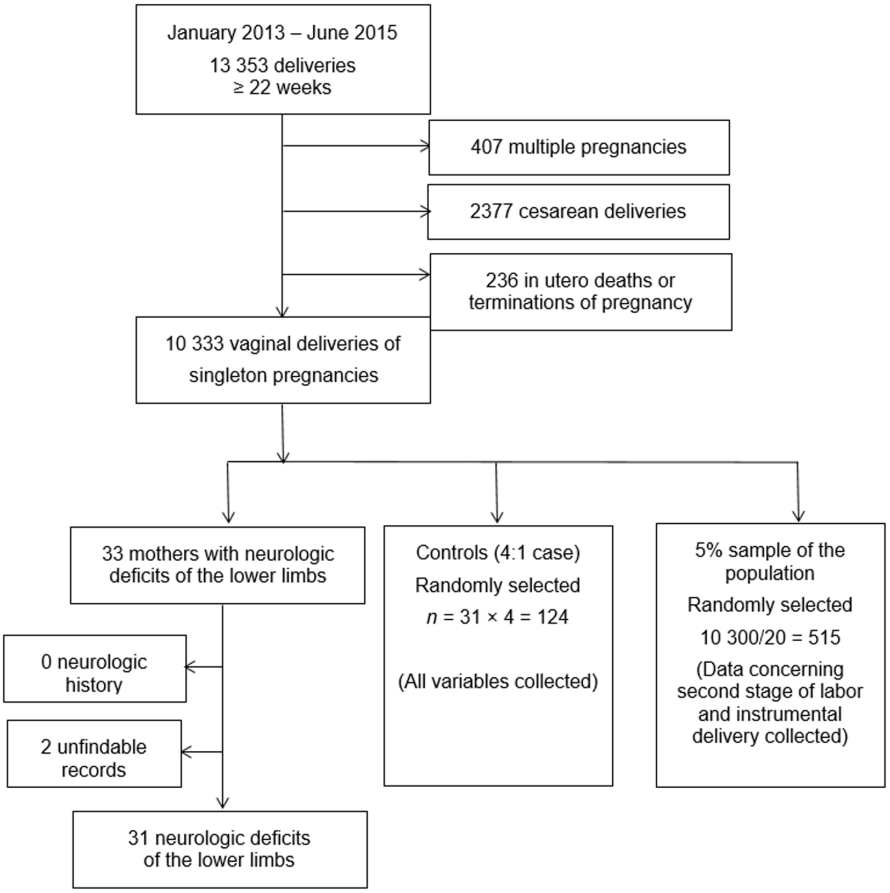

FIGURE 1 Study flow chart.

as reference. Data were analyzed using the SAS software version 9.4 (SAS Institute, Cary, NC, USA).

2.4 | Ethics statement

The collection and use of these data were reported to the National Commission for Data Protection and Liberties (no. DEC16-273) on 30 October 2017. Informed verbal consent was obtained from all study participants.

3 | RESULTS

During the study period, 13 353 women gave birth in our center; 10 333 of these were vaginal deliveries of live singletons (Figure 1). Among the latter, 33 women (0.3% of those with vaginal singleton deliveries) had lower-limb neurological deficit during the immediate postpartum period. None of these women had any neurological history. Records on two of these women could not be located, leaving a sample of 31 women with neurological deficit of the lower limbs for analysis. The control group included $n = 124$ (i.e., 31×4) women who gave birth during the same period. Finally, the 5% sample of the 10 300 women who gave birth vaginally to a live singleton and had no deficit was n = 515 (see Material and methods).

Compared with the control women, cases were more often nulliparous, gave birth at a later gestational age (by 1 week), had longer first and second labor stages, were more often positioned in genupectoral during labor, and had more instrumental deliveries (Table 1, Figure 2). Infants born to mothers in the case group had larger head circumferences.

Results of bootstrapping selection procedures to identify the independent risk factors of lower-limb nerve injury after vaginal delivery among the 10 candidate factors regarding the potential causal relation are shown in the Supporting Information (Table S1). Second labor stage duration and the use of instrumental delivery were selected in multivariable regression analysis in more than 70% of bootstrap samples. In the parsimonious multivariable penalized logistic regression model, prolonged second-stage labor (OR per standard deviation increase 2.73, 95% CI 1.75–4.25, $p < 0.001$) and instrumental delivery (OR 3.24, 95% CI 1.29–8.14, $p = 0.012$) were both associated with lower-limb nerve injury after vaginal delivery (Table 2) without interaction effect between these factors ($p = 0.56$). The selected multivariable model had a good calibration (Figure S1) and discrimination (c-index, 0.837; 95% CI 0.764–0.910).

Next, we estimated the proportion of women with a second-stage labor duration longer than 90 min and those undergoing instrumental delivery among the 5% sample (Table 3). The rates of neurological deficit were: 0.05% among women with second-stage labor lasting up to 90 min and without instrumental delivery (64.5% of sample); 0.43% for second-stage labor lasting up to 90 min and with instrumental delivery (20.4% of sample, risk ratio 9.5, 95% CI 2.5–35.1); 0.89% for second-stage labor longer than 90 min and without instrumental delivery (7.6% of sample, risk ratio 19.7, 95% CI 5.1–76.1); and 1.52% for second-stage labor lasting longer than 90 min and with instrumental delivery (7.6% of sample, OR 33.6, 95% CI 9.4–118.7). Figure 3 depicts the observed rates of lower-limb deficit as a function of these factors.

TABLE 1 Characteristics of mothers, labor, and neonates, by study group

	Case group ($n = 31$)	Control group ($n = 124$)
Maternal age (years)	29.9 ± 4.7	29.6 ± 5.1
Nulliparous[a]	22 (70.8)	56 (45.2)
BMI (kg/m^2)[a]	23 [18–36]	23 [16–50]
Gestational age at delivery (weeks)[a]	40 [34–41]	39 [27–42]
Epidural analgesia[a]		
None	3 (9.7)	14 (11.7)
Ineffective	5 (16.1)	29 (24.2)
Effective or very effective[b]	23 (74.2)	77 (64.2)
Cervical dilation at analgesia placement (cm)	3.5 [2–8]	3 [2–10]
Duration of the first stage of labor (h)[a]	5.5 [1.0–10.0]	3.0 [0.0–11.0]
Duration of the second stage of labor (min)[a]	94 [13–224]	27 [2–174]
> 90 min	19 (61.3)	16 (12.9)
Descent (min)	80 [1–190]	15 [0–148]
Expulsion (min)	22 [5–53]	9 [1–54]
Position held the longest time[a]		
Supine or lateral decubitus	18 (58.1)	103 (83.1)
Seated	5 (16.1)	15 (12.1)
Genupectoral	8 (25.8)	6 (4.8)
Instrumental delivery[a]	21 (67.7)	39 (31.5)
Forceps	14 (45.2)	29 (23.4)
Vacuum extractor	9 (29.0)	13 (10.5)
Neonatal arterial pH	7.23 ± 0.07	7.24 ± 0.08
Birthweight (g)	3581 ± 446	3297 ± 551
Macrosomia (>3800 g)[a]	6 (19.4)	19 (15.3)
Head circumference (cm)[a]	36 [32–38]	35 [28–38]
Transfer to intensive care unit	0 (0.0)	5 (4.1)

Note: Values are expressed as number (percentage), means ± standard deviations or medians [range].

[a]Considered as candidate factors associated with neurological deficit after vaginal delivery.

[b]Defined as analgesia effectiveness score ≥3.

4 | DISCUSSION

After logistic regression analysis, our study showed that the principal factors related to lower-limb nerve injuries were second-stage

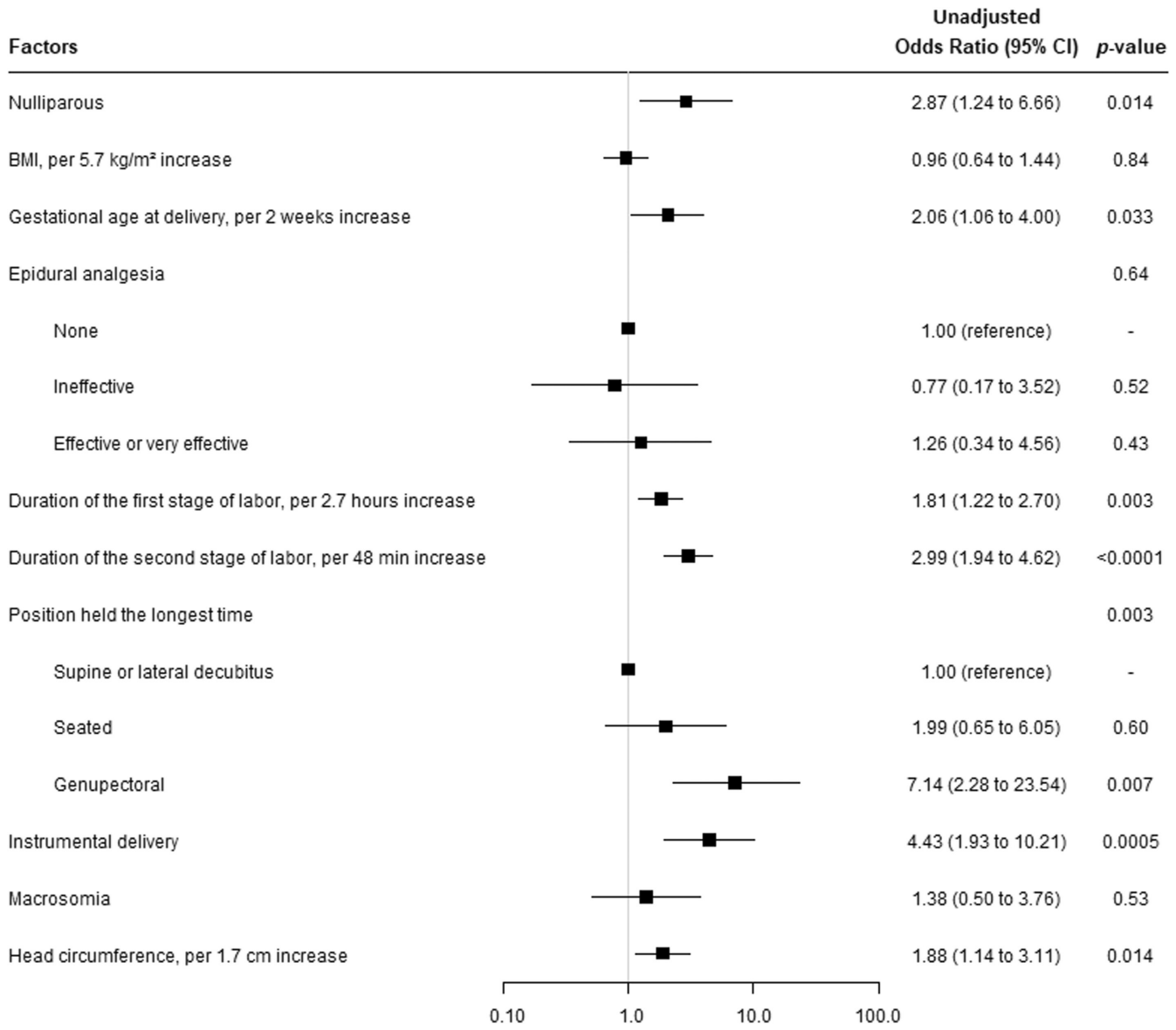

FIGURE 2 Unadjusted odds ratio of neurological deficit for pre-specified patient characteristics.

labor duration and instrumental delivery. Each additional hour of second-stage labor, or use of an instrument, approximately tripled the risk of later lower-limb neurological deficit. In our sample, symptomatic lower-limb nerve injury occurred in 0.3% of singleton vaginal deliveries, which is consistent with the literature. Previously reported injury rates have ranged from 0.1% in retrospective studies to 2.0% in prospective studies.[1–5] Although most injuries were sensory and unilateral,[6] they could be disabling. Our data, like those published to date,[2,4–6] indicate that approximately 30% of women remain symptomatic 6 weeks postpartum, and 10% remain so until 1 year postpartum.[9]

All factors identified in our univariate analysis have been previously linked to risk of neurological deficit after vaginal birth: nulliparity,[1,2,7] advanced gestational age,[4] longer labor duration,[1,2] longer genupectoral labor position,[1,8] instrumental delivery,[1,4,7] larger head circumference.[4] After multivariate analysis, only second-stage labor duration and use of an instrument during childbirth remained associated with injury in our sample. Our data indicate that the risk of neurological deficit is minimal for second-stage labor lasting up to 90 min and delivery without instrumentation (0.05%), which account for almost two-thirds of deliveries at our center. In contrast, instrument use and second-stage labor longer than 90 min each raised the risk of neurological deficit substantially, multiplying risk by a factor of approximately 30 for the combination.

Both the descent and expulsion phases of the second labor stage were included in our analyses. Compared with the control group, the median duration of second-stage labor was more than 1 h longer among case women. Consequently, 61.3% of the women in the case group had a second stage longer than 90 min, compared with only 12.9% of controls. Like others,[16,17] we hypothesize that the longer each of the descent and expulsion phases lasts, the stronger and more harmful pelvic nerve compression might be. Stretching

of nerves and disorders of their vascularization can intensify their damage.[16] However, the second-stage labor duration in our case group was not excessive according to current standards of practice. A widely influential consensus statement from the USA, aimed at reducing first cesarean deliveries, recommends considering second-stage labor duration prolonged when it is beyond 3 h in a nulliparous woman with epidural analgesia (2 h without epidural) and after 2 h in a primiparous or multiparous woman with epidural analgesia (1 h without epidural).[18,19] Herein, only three of the 31 women in our case group (9.7%), and none in the control group, exceeded these limits.

Although no data indicate that shortening the second stage of labor decreases the risk of lower-limb deficit after childbirth, our data suggest that the duration of both phases of second-stage labor (i.e., descent and expulsion) should be reconsidered. A recent trial found that delayed pushing exposes nulliparous women to higher risks of infection, hemorrhage, and neonatal acidemia but does not increase rates of vaginal birth.[20] A new American College of Obstetricians and Gynecologists recommendation supports pushing at the start of the second stage of labor for nulliparous women with neuraxial analgesia.[21] Immediate—compared with delayed—pushing shortens second-stage labor by around 1 h.[22,23] Our data indicate that this reduction, beneficial for some newborns, might also limit the risk of maternal neurological deficit.

Our study also shows that instrumental delivery is a risk factor for postpartum neurological deficit. This observation is consistent with previous findings[1,4,7] and may be explained by the rapid stretching and additional compression to pelvic nerves from instrumentation.[16] These are, however, hypotheses that would need to be tested. The absence of statistical interaction between instrumental extraction and the long duration of the second stage of labor argues in favor of a specific and additional role of these two factors, although it is not possible to affirm this. Paradoxically, it is sometimes necessary to use an instrument to reduce the duration of the second stage of labor.

Our study failed to show associations with several factors previously indicated as risk factors for nerve injury, such as nulliparity[1,2,7] and epidural analgesia.[5,7] Given our relatively small sample size, we cannot exclude the possibility that low statistical power prevented us from showing these associations. The high rate of epidural analgesia use in our center limited our ability to explore its role, including

TABLE 2 Multivariable model for factors associated with neurological deficit

Factors	aOR (95% CI)[a]	*p* value[a]
Duration of the second stage of labor per standard deviation increase	2.73 (1.75 to 4.25)	<0.0001
Instrumental delivery	3.24 (1.29 to 8.14)	0.012
C-statistic, 95% CI	0.837 (0.764 to 0.910)	

[a]Estimated using a penalized logistic regression model by including the selected factors from the bootstrap selection procedure (see Table S1) considering the following candidate factors: nulliparous, body mass index, gestational age at delivery, epidural analgesia, duration of first and second stages of labor, position held the longest time, instrumental delivery, macrosomia, and head circumference.

TABLE 3 Estimation of the gross risks of neurological deficit of the lower limbs over the entire study population (of vaginal births) (the data of the 5% sample of the non-case vaginal births were multiplied by 20)

	Controls	Cases	Risk of deficit [95% CI] (*n*/*N*)	Relative risk of deficit (RR, 95% CI)
No instrumental delivery and second stage ≤90 min	64.5% (6640/10 300)	9.7% (3/31)	0.05% [0.01–0.13] (3/6643)	1 (ref)
Instrumental, ≤90 min	20.4% (2100/10 300)	29.0% (9/31)	0.43% [0.20–0.81] (9/2109)	9.45 [2.56–35.08]
No instrumental delivery >90 min	7.6% (780/10 300)	22.6% (7/31)	0.89% [0.36–1.82] (7/787)	19.70 [5.10–76.02]
Instrumental delivery, >90 min	7.6% (780/10 300)	38.7% (12/31)	1.52% [0.79–2.63] (12/792)	33.55 [9.48–118.64]

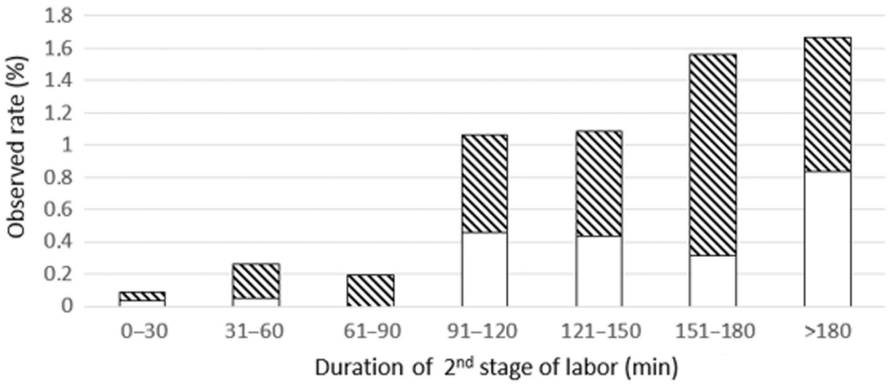

FIGURE 3 Observed rate of lower-limb deficit (%) depending on the duration of the second phase of labor (minutes). The proportion of instrumental extractions is represented in hatched form (vacuum extractor or forceps).

its currently debated influence on prolonging the second stage of labor.[24–29] In particular, we cannot rule out the possibility that epidural analgesia, by limiting sensations of impending nerve injury (sensory block) and reducing the potential for repositioning (motor block), may promote the occurrence of neuropathies by allowing prolonged nerve compression. The current incidence of motor block during labor epidural analgesia is approximately 15%.[30] Notwithstanding very rare cases in which epidural analgesia can itself cause nerve injury,[31] our data do not suggest that its use in vaginal deliveries plays a direct role in neurological deficits.

Likewise, the genupectoral (knee–chest) position has been previously described as a risk factor for neurological deficit, particularly in the femoral and lateral femoral-cutaneous territories, via compression under the inguinal ligament.[1,6,8,16,17] Although several cases of neurological deficit from this position have been reported—even outside the childbirth context[32,33]—the disappearance of this factor after logistic regression analysis suggests that any compression linked to a prolonged position may be from its duration rather than the position type. As such, frequent position changes during labor have been recommended.[1,6]

Our study is limited by its retrospective nature, the small number of events (n = 31 cases), and its low statistical power with limited possibilities to highlight several variables that would be simultaneously related to the occurrence of a neurological deficit after logistic regression. However, most studies have encountered this methodological problem in analyzing these low-frequency injuries, with four of the five series (either prospective or retrospective) to date including fewer than 25 women with such deficits.[2–5] Only Wong et al. were able to evaluate 56 women with a nerve injury in their prospective series.[1] In addition, to account that the ratio of number of events per variable into multivariate logistic analysis is low (events per variable <5), we use a multivariable penalized logistic regression model with a forward stepwise selection procedure combined with bootstrap resampling to build the predictive multivariable model. This method allows us to increase the robustness of variables selection despite low events per variable, although we caution that estimates of selected variables could be over- or under-estimated.

Further larger series are warranted. Considering a frequency of neurological deficits of about 0.3% of deliveries, these series should certainly consider a number of deliveries of several tens of thousands. This could be achieved through multicenter and prospective collection of cases of neurological deficits after birth. Studies systematically looking for neurological deficits after delivery have shown rates of up to 2%, which could reduce the length of inclusion of cases.[1]

5 | CONCLUSION

Obstetricians must remain mindful that vaginal delivery involves a low risk of neurological deficit (about 1/10 000 deliveries), and represents a rare but sometimes disabling complication (symptoms persist after 1 year in approximately 1/10 000 deliveries). Having shown that they are mainly associated with second-stage labor duration and instrumental delivery does not mean that they are avoidable. Our ability to avoid these complications has never been discussed and could be the subject of interventional studies. The most important appears to recognize these deficits as childbirth complications, and reassure women who suffer from them as they heal in most cases. Finally, our finding that the injury rate may reach 1.5% when these two risk factors are combined leads us to reconsider the costs and benefits of prolonging the second stage of labor by delaying pushing, especially if this delay is accompanied by other disadvantages.[20,34]

AUTHOR CONTRIBUTIONS

All authors contributed to the study conception and design. The study was supervised and designed by DS. Material preparation and data collection were performed by ACD and AT. Analysis was performed by JL and ED. The first draft of the manuscript was written by AT and DS. All authors commented on previous versions of the manuscript and gave their expertise including FC, MG, and CG. All authors read and approved the final manuscript.

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CONFLICT OF INTEREST

None.

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SUPPORTING INFORMATION

Additional supporting information can be found online in the Supporting Information section at the end of this article.

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