Dysfunctional labor and delivery: adverse effects on offspring

Emanuel A. Friedman, MD, Med ScD; Wayne R. Cohen, MD

There is no doubt that parturition can produce fetal and neonatal adversity, but the frequency with which this occurs is uncertain, particularly in modern healthcare settings. Moreover, there is a paucity of recent studies in this area. Substantial challenges impede epidemiologic study of the effect of parturition on offspring. Randomized trials would be ethically fraught. Therefore, large observational samples with detailed data concerning labor and delivery events are needed. Importantly, long-term follow-up of infants is necessary to reach reliable conclusions. Few such data sets exist, and it is difficult, expensive, and time-consuming to create and to study them. Reports of immediate newborn condition in relation to the antecedent labor are helpful, but this evidence is an imperfect predictor of long-term neurologic status. In this review, we endeavor to summarize existing information about the relationship between objectively defined abnormalities of labor progress and long-term disability in offspring. The only data available are from collected experiential information on outcomes stratified according to labor and delivery events. Most studies do not ensure against confounding by the many concurrent conditions that may affect outcome, or use inconsistent criteria to define abnormal labor. According to the best available evidence, dysfunctional labor patterns are potentially associated with poor outcomes for surviving infants. The question of whether these adverse effects can be mitigated by early diagnosis and expeditious management deserves to be answered, but cannot be at this time. In the absence of more conclusive results from well-designed studies, we can conclude that the best interests of offspring are served by adhering to evidence-based paradigms for the prompt identification and treatment of dysfunctional labor patterns.

Key words: arrest disorders, brain damage, dysfunctional labor, labor, National Collaborative Perinatal Project, prolonged latent phase, protraction disorders

Introduction

A question of principal importance in guiding our approach to intrapartum care is whether labor, particularly dysfunctional labor, can affect the long-term health of progeny. If being born after an abnormal labor, especially if prolonged and neglected, does confer risk, then prompt diagnosis and evidence-based management of abnormal labor is of consequence for the fetus and newborn, and discussions about how best to recognize and codify dysfunctional labor are critical and urgently needed. Maternal considerations are obviously important to investigate as well, but we limit ourselves here to the impact of labor on offspring.

There is no doubt that parturition can produce fetal and neonatal adversity; the perinatal injury engendered by prolonged and neglected labor that can occur in the nonindustrialized world, where there is often poor access to medical care, offers convincing testimony to this fact.[1–3] However, whether labor and delivery that occurs in the context of informed surveillance by trained health professionals is associated with enduring fetal injury is another matter, and not one readily resolved. In this review, we use an epidemiologic perspective to examine how exposure to dysfunctional labor may influence short- and long-term outcome in offspring. Our goal is to summarize available information and stimulate the need for further research in this regard.

From the Department of Obstetrics, Gynecology and Reproductive Biology, Harvard Medical School, Boston, MA (Dr Friedman); and Department of Obstetrics and Gynecology, The University of Arizona College of Medicine – Tucson, Tucson, AZ (Dr Cohen).

Received May 12, 2022; accepted Oct. 10, 2022.

The authors report no conflict of interest.

The authors report no funding for this study.

Corresponding author: Wayne R. Cohen, MD. cohenw@arizona.edu

0002-9378/$36.00

https://doi.org/10.1016/j.ajog.2022.10.011

Click Video under article title in Contents at **ajog.org**

Investigative challenges

There are evident and formidable challenges involved in epidemiologic investigation of the effect of the birth process on human offspring. Long-term randomized trials would be difficult because of ethical constraints. Large observational samples with meticulously collected data concerning labor and delivery events and long-term follow-up of infants would be necessary to reach solid conclusions. Few such data sets exist, and it is difficult, expensive, and time-consuming to create and to study them.

Reports of immediate newborn condition in relation to the antecedent labor (mortality, Apgar scores, cord blood pH or base excess, seizures, etc.) are plentiful and helpful; however, these measures are imperfect predictors of long-term neurologic health.[4–7] Even when children or adults are available for evaluation, it is uncertain what the best diagnostic tools are to identify disability with a perinatal source. In addition, many social and environmental factors influence performance on psychometric and neurologic tests, and differences found many years after birth may reflect exposures and interventions that have positively or negatively influenced injuries sustained during labor. Socioeconomic status, educational achievement, race, medical comorbidities, and other factors can influence test performance.

To make matters more challenging, the brains of newborns may express great plasticity.[8] Some may have considerable power to heal or to compensate for injury; others may not. As a consequence of these many potential sources of misinterpretation, study conclusions that derive from analyses that did not consider the influence of various confounding factors must be viewed with circumspection.

The proportion of long-term neurologic disability attributable to labor and delivery events is uncertain. Although most disabling fetal injuries probably accrue to the fetus during intrauterine life before labor, a substantial proportion is attributable to problems encountered in labor or at delivery,[9,10] especially hypoxia and physical trauma.[9–13] Other factors, including infections and exposure to toxins can play a role. These provocations could be additive, permissive, or even synergistic.

The commonality in the etiology of most permanent central nervous system injury is probably brain ischemia. This may be the consequence of severe systemic fetal oxygen deprivation that causes hypotension and cardiac dysfunction, or it may occur from excessive mechanical force exerted on the fetal head. Excessive head compression can probably produce intracranial pressure sufficiently high to compromise brain perfusion.[14–16] In addition, it may cause stretches or tears of the vessels and supporting tissues of the brain.[17] The resulting intracranial hemorrhage can be destructive. Unfortunately, we have no objective clinical means to identify potentially deleterious extremes of skull compression or molding during labor.

There has been an understandable paucity of recent studies in this area. Therefore, the only data available are from collected empirical observations on outcomes stratified according to labor and delivery events. The investigations on which most such reports are based were carried out principally without cautionary measures having been taken to ensure against confounding by the many concurrent conditions that prevail among the studied population samples, or they used inconsistent or vague criteria to define abnormal labor. For example, Roemer et al[18] showed lower intelligence quotient (IQ) scores and school performance in children born after long labors compared with their siblings born by cesarean delivery without labor. These findings were disputed by other investigators, who found no effect of delivery route on IQ after adjusting for socioeconomic variables.[19] In fact, the conclusions of almost all of the research in which labor progress or delivery mode have been linked to long-term cognitive and neurologic outcome suffer from these flaws. An exception to these concerns were the data derived from the National Collaborative Perinatal Project (NCPP), which took great pains to deal with myriad potential confounders.[20–22]

The National Collaborative Perinatal Project

The NCPP was sponsored by the National Institutes of Health in the United States. Detailed data were collected prospectively by trained observers from a sample of approximately 58,000 deliveries between 1958 and 1974 at 14 large academic and hospital departments.[20] Pertinent analyses focused on a group of nearly 18,000 term infants whose maternal records contained sufficient documentation to allow reconstruction of a meaningful pattern of cervical dilatation and fetal descent. Labor progress was categorized according to the framework described by Friedman.[22–24] These data have been analyzed exhaustively and include a cohort of children followed for 7 years after birth. The possible influence of hundreds of potentially confounding variables was studied using complex multivariate regression analytics. In that manner it was possible to evaluate the contributions to outcome of maternal demographics, antepartum complications, infections, drug use, maternal comorbidities, and many variables related to intrapartum observations and events. Although essentially unique in the thorough follow-up of children born to study mothers, legitimate questions about the current validity of the NCPP results can be raised, considering the substantial changes in obstetrical and neonatal care that have occurred since the data were collected, including the addition of electronic fetal heart rate monitoring to obstetrical practice. Moreover, the relatively low cesarean delivery rate at the time made it difficult to determine how resorting to cesarean delivery might have altered outcomes. These reservations notwithstanding, the NCPP data demonstrate remarkably uniform relations among several measures of outcome (perinatal mortality; Apgar scores; speech, language, and hearing disabilities at age 3; IQs at 4 and 7 years) vis-à-vis the events of labor and delivery.

The long and detailed follow-up of the study infants makes the NCPP unlikely to be replicated or improved upon in the foreseeable future. Sophisticated statistical techniques applied to the study data allowed assessment of the specific effects of numerous obstetrical practices and their individual (and collective) contributions to outcomes and the impact of the labor process itself. The findings with reference to the association of specific labor disorders with outcome, at minimum, should still be considered relevant to current practice.

Prolonged latent phase

After a labor complicated by a prolonged latent phase (PLP) (defined by Friedman as >20 hours in a nullipara or >14 hours in a multipara[22–24]), most infants do well, and this disorder was initially thought to be innocuous.[25,26] However, there is a growing body of evidence indicating that there is a significantly worse perinatal outcome after labors complicated by a PLP than after a normal labor pattern when controlled for potentially confounding factors.[27] Data from the NCPP subjected to multivariate analysis showed significantly increased relative risks (RR) for stillbirth (RR, 2.43 for multiparas and 3.00 for nulliparas; $P<.05$), neonatal depression (RR, 3.70 and 6.43; $P<.001$), abnormal speech language and hearing at 3 years (RR, 1.28; $P<.05$), and low IQ scores at 7 years of age (RR, 1.27 and 1.34; $P<.05$).[20] Similarly troubling results were encountered by Chelmow et al,[28] although they studied only short-term

neonatal outcomes. They found a high frequency of active-phase labor disorders among parturients with PLP (43% vs 16% in controls; $P<.05$). In addition, significantly higher rates of neonatal depression were encountered in this group (RR, 1.97; 95% confidence interval, 1.23−3.16). Furthermore, the more the latent phase is prolonged, the greater the risk of these abnormal outcomes. Analogous results were also found by other investigators. For example, Maghoma and Buchmann[29] found significantly higher rates of oxytocin administration (62% vs 17%; $P<.0001$), cesarean delivery (29% vs 6%; $P<.0001$), and neonatal intensive care unit admission (22% vs 1%; $P<.0001$) in a group of 150 cases with a long latent phase compared with controls with a shorter one.

Should this knowledge about the possible adversity conferred by a PLP alter our management of labors complicated by it? The assumption that earlier intervention to shorten the latent phase would negate its potentially adverse effects has never been studied. Moreover, the mechanism by which a PLP would result in neonatal adversity is not at all clear. It may be that prolongation of this portion of labor is a manifestation of some defect in prelabor or early-labor cervical remodeling or in the generation of uterine contractility that continues to hamper dilatation. That might explain the propensity of these labors to develop subsequent protraction or arrest disorders in their active phase, but it does not shed light on why they seem to be associated with short- and long-term neonatal problems. Given our considerable lack of relevant knowledge about the causes and consequences of PLP and the absence of data from prospective trials, we recommend adhering to the conservative management regimen described previously.[22−24]

This involves choosing between oxytocin administration and providing therapeutic rest with a dose of a narcotic or ataractic agent. The former approach is used most commonly today.[27]

Protraction disorders

In a protraction disorder, dilatation or descent proceeds steadily, but at a rate below the limit of normal, as defined by the fifth percentile of the distribution of active-phase dilatation rates.[22−24] Several kinds of analyses support the view that both protracted active phase dilatation and protracted descent are associated with adverse fetal and neonatal outcomes. Early studies found associated perinatal mortality rates to be 3-fold higher compared with normal labor, and poor long-term outcomes to be more than twice as frequent.[26] The latter included abnormalities of speech, language, or hearing at age 3 and lower IQ scores at the age of 7 years. To minimize confounding and interactional effects on these observations, complex logistic regression analyses of the NCPP database were done, and the results support these conclusions. The RR for composite perinatal morbidity and mortality was 2.12 ($P=.005$).[20] Unfortunately, no recent studies addressing this issue have been published to provide further insights.

The question of whether these adverse effects can be mitigated by early diagnosis and expeditious management deserves to be answered, but cannot be at this time. Furthermore, it is not yet known if the risks of adversity conferred by a protraction disorder are proportional to its duration, that is, if slower rates of cervical dilatation in the active phase or of fetal descent in late labor are associated with higher or long-term fetal and neonatal morbidity and mortality. We can only surmise that intervention might succeed in diminishing these deleterious effects, but because many of these cases are associated with cephalopelvic disproportion and other factors,[24] some of the poor outcomes may reflect those associations and related management decisions rather than the protraction disorder itself. It is regrettable that there have been no more recent studies undertaken to verify these findings in an era when obstetrics is practiced with greater attention to continuous fetal surveillance and more expeditious intervention to effect delivery than was the case when the NCPP data were generated.

Arrest disorders

An arrest disorder occurs when, once labor has entered the active phase, dilatation or descent ceases for 2 hours or ≥1 hour among nulliparas and multiparas, respectively. Arrest patterns of labor are useful clinical markers of cephalopelvic disproportion.[20−24] When an arrest disorder is encountered, the probability of a safe vaginal delivery is reduced by approximately a half because of the high likelihood that disproportion exists.[22] Accordingly, it is not unexpected that these labor patterns would be associated with adverse outcomes. This applies especially to situations in which vaginal delivery is attempted without the precaution of ensuring that the cephalopelvic relations are favorable and the prospects for safe vaginal delivery are good. Thorough clinical cephalopelvimetry is especially important in this regard.[24]

Increased rates of perinatal deaths and morbidity in association with arrest disorders were documented in the NCPP.[25,26,30] The RR of perinatal deaths found in association with arrest disorders of labor was 7.5, based on an absolute rate of 36.6 per 1000, as contrasted with 4.9 per 1000 after normal labor progress. Correcting for the type of delivery more than doubled the RR to 15.7, although absolute mortality was reduced by this correction from 36.6 to 16.0 per 1000, and the perinatal mortality after normal labor is reduced even more to 1.5 per 1000.[20,21] A later study[26] showed even higher perinatal losses, approximately 4 times more frequent than those occurring after normal labor patterns.

In parallel with the mortality risks, several investigators found a strong relationship between arrest patterns and reduced performance on childhood neurologic and cognitive testing.[20,25,26] Nelson, who has opined that intrapartum events have little or no adverse effect on the fetus, has acknowledged that arrest disorders are exceptions.[31,32] A large case−control study found nearly 3 times the frequency of arrest of labor progress among developmentally abnormal children. Not unexpectedly, a significantly increased frequency of forceps procedures and cesarean delivery, low Apgar scores, and need for intensive

care and long-term neurologic follow-up have also been reported following arrest of dilatation.[33,34]

Of particular interest in this last cited report is that stratification by delivery method showed that vaginal delivery after an arrest disorder magnified the poor infant results, whereas cesarean delivery diminished the impact attributable to the labor disorder. These observations contradict the notion that avoiding cesarean delivery in the presence of an arrest disorder by undertaking a potentially difficult vaginal delivery is reasonable. However, they are not the last word on the subject.

Contradictory results were reported by Rosen et al.[34,35] They found that the frequency of neurologic abnormalities did not differ significantly between children born after arrest disorders and controls after an average follow-up of 5 to 6 years. The cases came from a single institution with standardized protocols for labor management. Although these findings seem reassuring, cases and controls were not strictly matched for factors that could contribute to subsequent injury. The most recent observational study to address this issue found no association with the immediate neonatal outcome in cases complicated by arrest of dilatation compared with controls. Unfortunately, no data on long-term outcome were presented, and the authors used a definition of arrest disorder different from that in most of the related literature.[36]

When logistic analytical methods were used to assess the impact of arrest disorders while other factors were taken into account simultaneously, the adverse perinatal effect largely disappeared.[20] On the basis of these findings, it could be surmised that much of the fetal danger associated with arrest disorders is subsumed by other factors acting simultaneously. These probably include uterotonic stimulation, the presence of cephalopelvic disproportion, and the timing and type of delivery intervention taken to address the labor problem. In addition, the duration of the arrest pattern may play a critical role in this regard, as evidenced by the correlation between longer arrest periods and more intense and prolonged adverse effects. For example, neonatal death rates increase progressively with duration of arrest of descent in nulliparas.

These findings indicate that delayed diagnosis of an arrest disorder is detrimental to the fetus. The benefit of early diagnosis presumably arose because some form of evaluation was done to rule out disproportion in cases in which labor was allowed to continue. Alternatively, disproportion was presumed to exist by virtue of the presence of an arrest disorder with or without other demonstrable signs, and cesarean delivery was then pursued. Operative delivery may have averted any adverse consequences from continuing the labor. Although these data are strongly suggestive, the notion of mitigating adverse effects of arrest disorders by early diagnosis and aggressive intervention is unsubstantiated, lacking supportive data derived from a reliable recent source, such as a well-designed clinical study.

Other lines of evidence support the association of abnormal labor with adverse outcomes. In 1999, Towner et al[37] examined the frequency of neonatal intracranial hemorrhage according to delivery type in a sample of more than half a million cases. They found that instrumental delivery and cesarean delivery done during labor were associated with higher rates of neonatal intracranial hemorrhage compared with spontaneous vaginal delivery. The rate among infants delivered by cesarean delivery before labor was, however, not elevated. Their analysis strongly implicated abnormal labor—the reason for most intrapartum operative deliveries—as a common risk factor for neonatal intracranial injury.

Associations

A proportion of demonstrable fetal adversity associated with dysfunctional labor is attributable to the mode of delivery and to factors that caused the abnormal labor and the need for operative delivery. Midforceps operations confer special risk to the fetus, especially when used following protraction or arrest disorders.[20,25,26] Although the inference that instrument-assisted vaginal delivery could be related to direct traumatic brain injury is understandable, it is less evident how a dysfunctional labor itself could be detrimental, especially if it eventuates in a spontaneous delivery. A plausible mechanism is that excessive compression of the head against an unyielding birth canal can cause direct intracranial trauma or can reduce brain perfusion, resulting in ischemic damage.[11] The direct relationship between the duration of arrest disorders and the risk of neurologic or cognitive disability supports this hypothesis and emphasizes the virtues of prompt recognition and management of abnormal labor progress. This is, however, a hypothesis that requires careful testing.

Conclusions

Dysfunctional labor patterns, objectively defined, are potentially associated with poor outcomes for surviving infants. This adversity is influenced substantially by the delivery method. Certain kinds of potentially traumatic instrumentation, such as difficult midforceps procedures, will diminish the potential for a good outcome. By contrast, timely intervention by cesarean delivery, when indicated, may be beneficial by reducing the deleterious effect of further abnormal labor progression. The obstetrical care provider should be alert to the need to recognize dysfunctional labor patterns early, and to evaluate for associated or etiologic conditions that threaten fetal well-being. If such conditions are found, atraumatic delivery should be expedited.

It is not likely that the controversy over the potential adverse effects of labor will be resolved to everyone's complete satisfaction in the near future. The issues are complex, and the inability to isolate labor's specific contributions to outcome hinders interpretation of most observational studies. Moreover, it is possible that dysfunctional labor may act as an adversary of

good outcome only when other factors are introduced (such as instrumental delivery, prolonged and unrecognized arrest or protraction disorder, incautious use of oxytocin). It may be that when complex or difficult instrumental delivery is eschewed and dysfunctional labor is managed according to uniform and validated protocols, as is increasingly the case today, risks are minimized. This change in practice patterns may explain why some contemporary data make dysfunctional labor seem less alarming than did earlier work. Widespread use of labor curves to monitor progress may have reduced the number of dysfunctional labors that persist unrecognized and ignored, and that ultimately lead to fetal trauma. The failure to recognize an aberrant labor pattern and allowing it to persist, or undertaking a potentially deleterious type of delivery ostensibly to prevent harm, may paradoxically result in fetal trauma.

There are many more questions than there are satisfying answers available in this regard. One thing is certain: if we are ever to answer compelling questions about abnormal labor and its effects on delivery outcome and long-term health, it can only be done in the context of our profession adopting a uniform system of diagnosing and a common lexicon for describing abnormal labor.[23,24] Universal use of a standardized form of graphic labor analysis and an agreed-upon lexicon for defining abnormalities would be a good step in better understanding the processes and consequences of human labor and birth. We are in urgent need of well-designed controlled trials of diagnosis and treatment.

Highlights

- There is no doubt that parturition can produce fetal and neonatal adversity, but the frequency with which this occurs is uncertain, particularly in modern healthcare settings.
- Dysfunctional labor patterns, objectively defined, are potentially associated with poor outcomes for surviving infants. This adversity is influenced substantially by the delivery method.
- The proportion of long-term neurologic disability attributable to labor and delivery is as yet uncertain. Most handicapping fetal injuries probably accrue during intrauterine life before labor, but a substantial proportion are attributable to problems encountered in labor or at delivery.
- Most infants born after a prolonged latent phase do well, but this disorder is associated with significantly worse perinatal outcome compared with normal labor patterns.
- Both protracted active phase dilatation and protracted descent are associated with adverse fetal and neonatal outcomes. Whether these unfavorable effects can be mitigated by early diagnosis and expeditious management is not known.
- Increased rates of perinatal deaths and childhood neurologic and cognitive disability in association with arrest disorders have been documented.
- The direct relationship between the duration of arrest disorders and the risk of neurologic or cognitive disability indicates the importance of prompt recognition and safe management of abnormal labor progress.

Research Questions

Further meaningful research in this area is of great importance, but faces daunting challenges related to the need for large sample sizes, a uniform system of data collection and analysis, and long-term neurocognitive follow-up of children. A contemporary version of the NCPP would be of enormous value, but might prove prohibitively expensive. Some questions could be answered with narrowly directed randomized trials, but these would also require many years of follow-up evaluation. Well-designed case-control studies addressing pregnancy, labor, and delivery events in children with and without certain disabilities could prove useful, but few available data sets provide sufficiently robust and accurate data on intrapartum progress to be of value. It would be a great benefit if some neonatal biomarker (biochemical or imaging), would be developed to reduce the need for long-term evaluation. ■

REFERENCES

1. Neonatal and perinatal mortality: country, regional and global estimates. Geneva, Switzerland: World Health Organization; 2006.
2. Akombi BJ, Renzaho AM. Perinatal mortality in sub-Saharan Africa: a meta-analysis of demographic and health surveys. Ann Glob Health 2019;85:106.
3. Wonde TE, Mihretie A. Maternofetal outcomes of obstructed labor among women who gave birth at general hospital in Ethiopia. BMC Res Notes 2019;12:128.
4. Yıldız EP, Tatlı B, Ekici B, et al. Evaluation of etiologic and prognostic factors in neonatal convulsions. Pediatr Neurol 2012;47:186–92.
5. Ehrenstein V. Association of Apgar scores with death and neurologic disability. Clin Epidemiol 2009;1:45–53.
6. McKinney JR, Allshouse AA, Heyborne KD. Duration of labor and maternal and neonatal morbidity. Am J Obstet Gynecol MFM 2019;1:100032.
7. Blankenship SA, Raghuraman N, Delhi A, et al. Association of abnormal first stage of labor duration and maternal and neonatal morbidity. Am J Obstet Gynecol 2020;223:445. e1–15.
8. Takesian AE, Hensch TK. Balancing plasticity/stability across brain development. Prog Brain Res 2013;207:3–34.
9. Nelson KB. Can we prevent cerebral palsy? N Engl J Med 2003;349:1765–9.
10. Paneth N. Birth and the origins of cerebral palsy. N Engl J Med 1986;315:124–6.
11. Kawakami MD, Sanudo A, Teixeira MLP, et al. Neonatal mortality associated with perinatal asphyxia: a population-based study in a middle-income country. BMC Pregnancy Childbirth 2021;21:169.
12. Novak CM, Ozen M, Burd I. Perinatal brain injury: mechanisms, prevention, and outcomes. Clin Perinatol 2018;45:357–75.
13. Prayer D, Brugger PC, Kasprian G, et al. MRI of fetal acquired brain lesions. Eur J Radiol 2006;57:233–49.
14. Amiel-Tison C, Sureau C, Shnider SM. Cerebral handicap in full-term neonates related to the mechanical forces of labour. Baillieres Clin Obstet Gynaecol 1988;2:145–65.
15. Vlasyuk VV. Compression circulatory hypoxia of the brain as a type of intrapartum hypoxia. J Gynecol 2019;4:000173.
16. Schifrin BS, Deymier P, Cohen WR. Fetal neurological injury related to mechanical forces of labor and delivery. In: Zhang L, Longo L, eds. Stress and developmental programming in health and disease: Beyond phenomenology. New York, NY: Nova Biomedical; 2014. p. 651–88.
17. Hong HS, Lee JY. Intracranial hemorrhage in term neonates. Childs Nerv Syst 2018;34:1135–43.
18. Roemer FJ, Rowland DY, Nuamah IF. Retrospective study of fetal effects of prolonged labor before cesarean delivery. Obstet Gynecol 1991;77:653–8.
19. Khadem N, Khadivzadeh T. The intelligence quotient of school aged children delivered by

cesarean section and vaginal delivery. Iran J Nurs Midwifery Res 2010;15:135–40.
20. Friedman EA, Neff RK. Labor and delivery: impact on offspring. Littleton, MA: PSG publishing; 1987.
21. Friedman EA. Effects of labor and delivery on the fetus. In: Kurjak A, Chervenak FA, eds. Textbook of perinatal medicine, 2nd ed. United Kingdom of Great Britain and Northern Ireland: Informa Healthcare; 2006. p. 1989–97.
22. Friedman EA. Labor: clinical evaluation and management, 2nd ed. New York, NY: Appleton-Century-Crofts; 1978.
23. Cohen WR, Friedman EA. Clinical evaluation of labor: an evidence- and experience-based approach. J Perinat Med 2020;49:241–53.
24. Cohen WR, Friedman EA. Labor and delivery care: a practical guide. Oxford, United Kingdom: John Wiley & Sons, Ltd; 2011.
25. Friedman EA, Niswander KR, Sachtleben MR. Dysfunctional labor. XI. Neurologic and developmental effects on surviving infants. Obstet Gynecol 1969;33:785–91.
26. Friedman EA, Sachtleben MR, Bresky PA. Dysfunctional labor XII. Long-term effects on infant. Am J Obstet Gynecol 1977;127:779–83.
27. Cohen WR, Friedman EA. The latent phase of labor. Am J Obstet Gynecol 2023.
28. Chelmow D, Kilpatrick SJ, Laros RK Jr. Maternal and neonatal outcomes after prolonged latent phase. Obstet Gynecol 1993;81:486–91.
29. Maghoma J, Buchmann EJ. Maternal and fetal risks associated with prolonged latent phase of labour. J Obstet Gynaecol 2002;22:16–9.
30. Friedman EA. Patterns of labor as indicators of risk. Clin Obstet Gynecol 1973;16:172–83.
31. Nelson KB, Broman SH. Perinatal risk factors in children with serious motor and mental handicaps. Ann Neurol 1977;2:371–7.
32. Nelson KB, Ellenberg JH. Antecedents of cerebral palsy. Multivariate analysis of risk. N Engl J Med 1986;315:81–6.
33. Bottoms SF, Hirsch VJ, Sokol RJ. Medical management of arrest disorders of labor: a current overview. Am J Obstet Gynecol 1987;156:935–9.
34. Rosen MG, Debanne SM, Thompson K. Arrest disorders and infant brain damage. Obstet Gynecol 1989;74:321–4.
35. Rosen MG, Debanne SM, Thompson K, et al. Abnormal labor and infant brain damage. Obstet Gynecol 1992;80:961–5.
36. Henry DEM, Cheng YW, Shaffer BL, et al. Perinatal outcomes in the setting of active phase arrest of labor. Obstet Gynecol 2008;112:1109–15.
37. Towner D, Castro MA, Eby-Wilkens E, et al. Effect of mode of delivery in nulliparous women on neonatal intracranial injury. N Engl J Med 1999;341:1709–14.