Cerebral Palsy (CP) is a known complication of preterm birth. However, approximately three quarters of patients diagnosed with CP were delivered after 36wks estimated gestational age (EGA). There is little information on the relation of CP risk to EGA when term and postterm. The population used in the study was the Medical Birth Registry of Norway. Births between 1967 (the start of the registry) and 2001 (the last year that would still allow the study a four year follow-up for the diagnosis of CP in the patients) were used. Only singleton live births with an EGA of 37-44 wks by LMP were used. Exclusion criteria from the study included: birth weight  3 SD of the mean EGA, babies with anomalies, a baby that died prior to 4 year of age, any baby in which the EGA information was missing. Initially 2,024,215 participants were identified. However, after the previously mentioned criteria were evaluated in the group, 1,682,441 participants were used in the final data collection. In calculating the relative risk (RR) for each group, a log-binomial regression was used. Adjustments were made for year of birth, baby’s sex, maternal age, maternal and paternal education level, maternal marital status, and immigration status of the parents. The study found a U-shaped distribution of CP prevalence with the highest being at 37 wks and >42 wks EGA and the lowest being at 40 wks EGA. The RR, when compared to 40 wks EGA, at 37 wks EGA was 1.9 and at >42 wks EGA was 1.5. Children with CP more likely to have single mothers (P=.03), mother’s with lower maternal education (P<.001), complications of labor (p<.001), lower mean BW (p<.001), and smaller head circumference (p<.001). However, once adjustments were made for these factors, there was identical RR by EGA. Dating by ultrasound (US) was available for births from 1998-2001. When compared to dating by LMP, dating by US showed the same U-shaped distribution of prevalence. However RR was stronger. When compared to 40 wks EGA, at 37 wks EGA the RR was 3.7 and at >42 wks EGA it was 2.4. Also, in looking at various time period intervals within the date, the prevalence of CP decreased while the association with EGA remained the same.

The article addresses an important issue in the practice of obstetrics. CP is a condition we know is more prevalent with prematurity, but is there also a continuous association between CP and gestational age at term and post-term? While there is no specific hypothesis stated per se, their research objectives were clear and unambiguous.

The retrospective population-based cohort study design is an appropriate method to address the question; however, for this study to have a true impact on our practice (for example, induction of all patients at 40 wga), a RCT showing a true difference would be needed. This study generated Level II-2 evidence and has very strong internal validity; after all, they did include the entire population. The external validity, however, could be called into question because our patient population and our current practice methods (such as timing of interventions) likely differ rather significantly from this study population in Norway. Inclusion and exclusion criteria overall were appropriate; however, some argue that those who died prior to age 4 should have been included in the study and considered to be “positive” for CP (this brings up the idea of “competing risk”). The authors seemed to adequately address and control for potential confounders, such as parental demographics, dating criteria, and historical time period, but one could argue that the presence or absence of labor and delivery complications should have been included. Their argument (a valid one) was that these complications may be part of the causal pathway of CP and should not be included in the analysis. Additionally, a strength of this study is that it avoids the problems of selective participation, recall bias, and loss to follow-up, but observation bias may play a role. Were the patients correctly identified and accurately diagnosed with CP? Some patients with mild CP were missed; however they refer to a validation study which showed that registered CP diagnoses correspond well with medical records, which did address this issue.

Statistically, the data was analyzed with binomial regression, generating a relative risk of CP with each week of gestational age (37-44 wga). P-values for different variables were reported and a 5% significance level was used. Sample size calculation and power was not necessary (the entire population was studied). Given that, in this case, the null hypothesis was rejected, there is a potential for Type I error (alpha), which by definition is stating there is a relationship between two variables when in fact there is not.

The conclusions stated by the authors seemed reasonable, in that an association is present between CP and all gestational ages, with the lowest risk at 40wga; however, this does not prove causation. “If the time of delivery affects CP risk, then intervention at 40 weeks might reduce CP risk, while elective delivery at 37 or 38 weeks might increase it. If infants prone to CP are disrupted in their delivery times, the prevalence of CP would be unchanged regardless of time of delivery. A definitive answer would require a randomized clinical trial...an impractical option, given the very low prevalence of CP.”