Letters to the Editor

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EXPECTED DATE OF CONFINEMENT

To the Editor:

Physicians want to be great prognosticators, to know and tell the future, to be the Delphic oracle. This is particularly true for physicians who deliver babies. For over a century, physicians have used Nägele's calculation[1] to predict the most likely day a pregnant woman will give birth. Generations of obstetricians have tried to improve on this method of determining the expected day of confinement (EDC). By using various other measurements, such as ultrasound examinations, uterine growth to the umbilical level, and detection of fetal heart tones, they always come up with a specific date on which expectant mothers expect to be confined in anticipation of the great event.

It is beyond our ability to estimate how much misery the EDC has brought, not only to pregnant women but to everyone around them as well. Despite all assertions that the EDC is not an accurate prediction of the specific day the baby will be born, psychologically, it becomes the target date for the end of the pregnancy. How many mothers and mothers-in-law of pregnant women have arranged their lives around the EDC only to be disappointed or inconvenienced? Employers also make arrangements for pregnant woman. Then if the infant is born 2 weeks before the EDC, the child earns the label *premature.* Even worse for the expecting mother, attending physician, and prospective grandmothers is the fear that the baby will be overdue and that if nothing is done, the infant will be born with the concocted "disease" called *postmaturity.* It is anybody's guess how often labor has been induced or cesarean sections performed because the EDC was reached or passed.

The truth about EDCs is obvious: there is no good way of predicting the day of delivery—but the solution to this dilemma is simple. We can and should establish the predicted time frame during which delivery should occur in most pregnancies. Delivery should not be expected to occur exactly 280 days after the first day of the last menstrual period: only 4% of deliveries occur precisely on the EDC.[2] Rather than attempting to pinpoint the time of delivery to a single day, we should use a range of time during which the happy event is most likely to occur. This range might be the 5th to the 95th percentile of the distribution of days after the first day of the last menstrual period.

Less specific terminology, such as "month of expected delivery," should also be introduced. This range of expected delivery dates would cure the many ills stemming from pseudoaccuracy of the EDC, such as an unnecessarily extended visit from a mother-in-law who based her arrival on the EDC. In any case, revising our method of predicting deliveries would most certainly reduce unnecessary induction of labor and cesarean sections, and babies would no longer be arbitrarily labeled premature or postmature. No doubt, mothers would prefer a specific date to a range, but physicians should stand firm, advise about the great range of normal pregnancy durations, and hope that it will not take the span of another generation before society and the medical community prefer the EMD (expected month of delivery) to the EDC.

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CAROTIDYNIA

To the Editor:

It is so good to see a description of carotidynia and review of the literature in *The Journal of Family Practice* (*Hill LM, Hastings G. Carotidynia: A Pain Syndrome. J Fam Pract 1994; 39:71–5*). I became an advocate of looking for this syndrome in atypical facial pain, ear ache without infection, etc, after having had an episode myself. An astute otolaryngologist made the diagnosis, and since I was also a migraineur, treated it with ergotrates. Thankfully, I have had no further episodes.

I have been trying to teach family practice residents to look for carotidynia as a cause for head and neck pain without obvious etiology since I learned of the syndrome in the early 1980s. The frequency of occurrence of carotidynia in an unselected family practice population would take a large multicenter prospective surveillance of all patients with compatible complaints. Is anyone interested?

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ACQUIRED STUTTERING

To the Editor:

The term *acquired stuttering* has been widely used in the literature to refer to stuttering that presents in individuals with no childhood history of stuttering, in association with stroke, degenerative disease of the central nervous system, head trauma, brain tumor, and the use of certain drugs.

The literature on stuttering indicates that cases of acquired stuttering are relatively rare. The etiology of acquired stuttering and developmental stuttering is not well understood. Information from the literature suggests that stuttering may result from neurologic and neuropsychologic anomalies in interhemispheric processing.[1] The speech and nonspeech characteristics that differentiate acquired stuttering from the more common developmental stuttering have been described in the literature.[2,3]

It has been documented in the literature that certain drugs (tricyclic antidepressants, phenytoin, and phenothiazine derivatives) affect neurologic function, resulting in stuttering or stuttering-like behaviors.[4–6] In most cases, the effects were temporary, and normal speech returned after the drug was discontinued.

The following case study describes an individual who developed severe stut-

tering while being treated for depression with fluoxetine. In the literature, there is only one similar documented case involving fluoxetine.[7] The cause of this stuttering disorder in terms of serotonergic inhibition has been further discussed.[7,8]

A 27-year-old single white woman was admitted to hospital in February 1991 following a 3-month history of sad mood, withdrawal from regular activities, poor memory, decreased concentration, anhedonia, increased anxiety, and feelings of depolarization and derealization. Psychiatric evaluation revealed unresolved family issues dealing with grief and sexual abuse. Before the present hospital admission, the patient had been receiving psychological counseling to help her cope. Her psychiatric history included admission to hospital in 1984 for a depressive episode during which she attempted suicide.

On February 7, fluoxetine (20 mg po daily) was begun. By February 19, her speech had become "clumsy," based on self-report. By February 28, she presented with severe stuttering. She later reported that she had been unable to utter a single word and that her communication was reduced to writing on a notepad. Fluoxetine administration was discontinued on that date.

A speech pathology assessment was conducted in March. She reported that while she did not have a childhood history of stuttering, she had experienced a 3-month period of stuttering upon moving from one city to another in 1989. She had attributed her problem to "nerves." She reported that her brother had stuttered while in elementary school.

During the speech assessment, she showed a high frequency of stuttering in both conversation and reading. She stuttered on 38% of syllables in conversation, and on 19% of syllables while reading. These values placed her stuttering in the severe to profound range. Primary stuttering behaviors included sound and word repetitions and sound prolongations without effort or pressure. Physical concomitants included lip tremor and eye blinking during more pronounced instances of stuttering.

Between March and April, she attended four speech therapy sessions. With the discontinuation of fluoxetine and the practice of speech therapy techniques, the patient was able to speak slowly and with minimal stuttering. By April, her stuttering was less than 1% in the contexts of reading and conversation.

In the present case, stuttering was acquired soon after the initial introduction of treating the patient with fluoxetine. The patient's speech patterns were characteristic of acquired stuttering, as described in the literature.[2,3]

Frequency, duration, and severity of stuttering patterns decreased when fluoxetine was discontinued. The association between fluoxetine and stuttering levels does not provide definitive evidence that fluoxetine was a direct cause of her stuttering; however, it does appear that her stuttering was triggered by the medication.

The literature has documented several instances of acquired stuttering that were drug-induced.[5,6] In a similar case in which fluoxetine was associated with stuttering, a woman developed stuttering symptoms 3 weeks after being treated with fluoxetine for depression.[7] The stuttering resolved with discontinuation of fluoxetine. The authors speculated whether the stuttering symptoms, which they theorized were caused by serotonergic inhibition of dopamine function, and the symptoms could be associated with other potent serotonin reuptake inhibitors.[7–9]

While an increase in serotonin may directly affect speech mechanisms, information from the literature is still inconclusive at this time regarding the etiology of stuttering and the mechanisms involved. The literature indicates that similar acting drugs may influence stuttering either positively or negatively. Therefore, further research involving controlled drug trials is needed to determine more precise neuropharmacology to maintain fluent speech.

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