

as parity, fetal risk score, use of oxytocin, etc., that are strongly associated with the use of epidural anesthesia and also are plausible causes of nonspontaneous delivery.

The authors elect to control for these confounding variables by using stratified analysis. While stratified analysis is useful for evaluating the *individual* contribution of each confounding variable examined in isolation, this method is inadequate for analyzing the *simultaneous* contribution of all confounding variables. Stratified analysis does not, therefore, provide an odds ratio adjusted for the *cumulative* effect of all identified confounding variables.²

The authors cite Blake's article on stratified analysis to support their methodology,³ but Blake himself cautions in the conclusion to his article that "multiple regression is often superior to stratified analysis when there is a need to assess conjoint confounding by two or more variables." (p 225)

We are confused why the authors' presentation of their regression analysis is so cursory. While their report devotes considerable text and seven figures to stratified analysis, their presentation of multifactorial analysis is limited to the comment that "regression analysis for all these factors failed to eliminate the increased odds ratios for patients who received epidural anesthesia." (p 241)

Exactly *which* factors were included in the regression analysis? Was the analysis limited to only six variables described as showing effect modification on stratified analysis (an entirely different phenomenon than confounding), or were all variables associated with epidural use included in the regression analysis? Why are no actual numerical values provided for the adjusted odds ratio and *P* value calculated by regression analysis? And lastly, why does the methods section make no mention of the statistical model and instruments used for the regression analysis?

The decision by a woman and her physician to use epidural anesthesia in labor is often a difficult one. The published research on the impact of epidural anesthesia on labor outcomes remains clouded by conflicting conclusions, widely variant obstetrical practice patterns, and poor study designs. There has never been—and for ethical reasons, is unlikely ever to be—a randomized controlled trial of epidural anesthesia in labor that could accurately evaluate the independent contribution of this intervention to labor outcomes.

Unfortunately, because of the many methodological problems we have discussed, we believe the study by Niehaus and colleagues cannot meaningfully contribute to clarifying the risks and benefits of epidural anesthesia. It would be unfortunate if family physicians seeking guidance in this area interpreted this study as a compelling reason to withhold epidural anesthesia in instances in which its use might prove advantageous.

Kevin Grumbach, M.D.
Ya'aqov Abrams, M.D.
San Francisco General Hospital
University of California, San Francisco

References

1. Niehaus LS, Chaska BW, Nesse RE. The effects of epidural anesthesia on type of delivery. *J Am Bd Fam Pract* 1988; 1:238-44.
2. Newman TB, Browner WS, Hulley SB. Enhancing causal inference in observational studies. In: Hulley SB, Cummings SR, eds. *Designing clinical research: an epidemiologic approach*. Baltimore: Williams & Wilkins, 1988:98-109.
3. Blake RL. The use of stratified analysis to detect confounding and interaction in primary care research. *Fam Pract Res J* 1985; 4:219-25.

The above letter was referred to the authors of the article in question, who offer the following reply.

To the Editor: We are pleased by the continuing interest in our article, "The Effects of Epidural Anesthesia on the Type of Delivery." I believe some of the concerns raised by Doctors Grumbach and Abrams's letter have been answered in our response to letters published in a previous issue of this journal (April-June 1989). However, these correspondents raise two issues that need clarification.

Doctors Grumbach and Abrams's major concern is that the epidural blocks performed in our study were given because of the need to provide anesthesia for a planned procedure such as forceps delivery or Cesarean section. In the methods and study design section of our paper, we reported that the low-risk obstetrics patients studied received an epidural block electively for pain relief only. Those patients in which the epidural block was medically indicated were eliminated from the study.

The correspondents also expressed concern about the use of stratified analysis to identify effect modifiers. We compared obstetrical characteristics and demographics between low-risk patients who received an epidural block and those who did not. In examining the differences, we found effect modification present for 10 variables overall and found significant effect modification for six variables. We used regression analysis to examine these variables individually and in combination, and certain combinations (notably, nulliparous women who were not given a continuous epidural block) did decrease the odds ratio for instrumental delivery between patients receiving epidural block and those who did not. However, no combination of variables studied eliminated the use of epidural block as an independent risk factor for instrumental delivery.

We agree with Doctors Grumbach and Abrams that research on epidural block to date has not provided a comprehensive and clear answer to their question of whether epidural blocks *cause* instrumental deliveries. I suspect that no prudent authors doing retrospective work will be willing or able to provide them with this answer. However, our work does show that in low-risk obstetrics patients, the use of an epidural block is associated with an increased frequency of instrumental delivery. In addition, our work shows this increase is not

explained by simple differences in patient demographics or labor characteristics, based on the effect modifiers that we studied. Family physicians seeking guidance in this area should consider these issues and reserve the use of epidural block for those labors in which it is clearly indicated or advantageous to the patient. In addition, the patient should be provided with informed consent regarding the effects and risks of the procedure.

Robert E. Nesse, M.D.
Mayo Clinic
Rochester, MN

NSAIDs and GI Bleeding

To the Editor: I enjoyed reading the review by Jaydev Varma, M.D., about nonsteroidal anti-inflammatory drugs in lower gastrointestinal bleeding (April–June 1989). I would simply like to add that the clinical experience of my practice is very similar to what he has stated. I have one patient who on three occasions has had lower gastrointestinal bleeding precipitated by the use of Indocin™. This patient has severe gout and ultimately was diagnosed as having angiodysplastic lesions of the colon. Another patient with known diverticular disease developed significant diverticular bleeding after use of a nonsteroidal agent.

Both of these patients were in the geriatric-aged group. I would be curious if the risks of lower gastrointestinal bleeding have been shown to be greater in geriatric patients similar to the increased risk that has been documented of upper gastrointestinal bleeding.

Karl B. Fields, M.D.
The Moses H. Cone Memorial Hospital
Greensboro, NC

The above letter was referred to the author of the article in question, who offers the following reply.

To the Editor: I have read Dr. Field's letter concerning lower gastrointestinal bleeding in the elderly. I find it reassuring that he has had a similar experience with the use of nonsteroidal anti-inflammatory drugs in the elderly.

In response to his question, whether the risks of lower gastrointestinal bleeding has been shown to be greater in geriatric patients similar to the documented risks of upper gastrointestinal bleeding, there are limited published reports in the medical literature. However, the geriatric-aged group is more vulnerable to gastrointestinal side effects of drugs in general and NSAIDs in particular. There are numerous studies relating to NSAIDs and upper gastrointestinal bleeding. However, to my knowledge there are no studies relating NSAIDs to lower gastrointestinal bleeding. My references in the article, "Do Nonsteroidal Anti-Inflammatory Drugs Cause Lower Gastrointestinal Bleeding? A Brief Review," contain the handful of published reports in this regard. A randomized, controlled study may be a reasonable approach to this problem.

Suggested reading:

1. Brocklehurst FC. The gastrointestinal system—the large bowel. In: Brocklehurst FC. Textbook of geriatric medicine and gerontology. New York: Churchill Livingstone, 1985:534-56.
2. Jones JK. Drugs and the elderly. In: Reichel W, ed. Clinical aspects of aging. Baltimore: Williams & Wilkins, 1989:41-60.
3. Carson JL, Strom BL, Morse ML, et al. The relative gastrointestinal toxicity of the nonsteroidal anti-inflammatory drugs. Arch Intern Med 1987; 147:1054-9.
4. Bahrt KM, Korman LY, Nashel DJ. Significance of a positive test for occult blood in stools of patients taking anti-inflammatory drugs. Arch Intern Med 1984; 144:519-21.
5. Patmas MA, Wilborn SL, Shankel SW. Acute multisystem toxicity associated with the use of nonsteroidal anti-inflammatory drugs. Arch Intern Med 1984; 144:519-21.
6. Agarwal AK, Eisenbeis CH Jr. Therapeutic guidelines for use of nonsteroidal anti-inflammatory drugs for rheumatic disorders: nonsalicylates. Fam Pract Recertification 1988; 10:49-70.
7. Amadio P Jr, Cummings DM. Nonsteroidal anti-inflammatory agents: an update. Am Fam Physician 1986; 34:147-54.

Jaydev Varma, M.D.
Medical College of Georgia
Augusta, GA

Thromboembolic Disorders

To the Editor: In the article "Diagnosis and Evaluation of Thromboembolic Disorders" (April–June 1989), I was disturbed by the lack of importance given to the arterial blood gases (ABGs) in the initial workup of suspected pulmonary embolism. While Dr. Brunader states the facts on ABGs in laboratory data, he fails to use these facts later on. In Figure 1, "Approach to Diagnosis of Suspected Pulmonary Embolism," the ABG is especially absent in the initial workup consisting of H+P, EKG and CXR. The ABG, if it shows a PaO₂ > 90 percent, is an approximately 95 percent negative predictor of pulmonary embolism (PE), i.e., highly sensitive to rule out PE. Both the EKG and CXR are in most cases not very helpful in ruling out a PE, especially in the young healthy patient, and, certainly, they do not compare with a 95 percent negative predictor like the ABG. Moreover, in the large subset of patients who fit into the "slightly more than minimal risk" category (my own category), I find the ABG to be invaluable.

For example, consider a 20-year-old white woman with no significant medical history or family history. She was started on birth control pills 2 months ago but stopped them 3 weeks ago because of persistent daily