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Fewer Failed Spinal Anesthetics with the Sprotte Needle

To the Editor:—In a recent letter,[1] Crone and Vogel suggested that there is an increase in the failure rate of spinal anesthetics using the Sprotte needle. We do not agree with the authors. We have indeed published results of the controlled prospective study[2] that Crone and Vogel say is necessary, but unfortunately the paper is written in German. We studied 500 patients undergoing operations on the lower extremities who received spinal anesthesia using either the 24-G Sprotte needle or the 25-G Quincke needle. Puncture characteristics were evaluated by a four-point scale (1 = easy, 2 = difficult, 3 = very difficult, 4 = impossible). A "failed technique" was defined as the lack of acceptable anesthesia for the proposed surgical procedure, following the injection of local anesthetic after free-flow cerebrospinal fluid was identified, as mentioned in the letter by Crone and Vogel. In addition, the incidence of post–dural puncture headache was evaluated in a double-blind fashion.

There were no differences between the two groups concerning age, sex, and the type of local anesthetic agent used. The puncture characteristics were assessed to be significantly better using the Sprotte needle ($P < 0.005$, Mantel-Haenszel test). In 243 patients (Sprotte needle) and 244 patients (Quincke needle), injections of local anesthetic agent could be performed after free-flow of cerebrospinal fluid was identified. Using the Sprotte needle, 4 of 243 (1.6%) anesthetics had to be classified as a failure compared to 19 of 244 (7.8%) using the Quincke needle ($P < 0.005$, chi-square test). Taking the type of local anesthetic agent used into account, the relationship remained the same: mepivacaine 4% hyperbaric 3 of 135 (Sprotte) *versus* 14 of 127 (Quincke), bupivacaine 0.5% hyperbaric 1 of 92 (Sprotte) *versus* 5 of 90 (Quincke). One reason for the higher incidence of failure rate in the Quincke needle group might be the deflection of a beveled needle away from the midline.[3,4] In our study we always entered the dura with the bevel parallel to its fibers, which could lead to an unequal distribution of the anesthetic. With respect to the incidence of post–dural puncture headache, we did not find any difference between the two types of needles (Sprotte 8.2% *vs.* Quincke 7.8%).

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In Reply:—Buettner *et al.* address pertinent issues with regard to the use and benefits of the Sprotte spinal needle. The advantage of this needle is clearly related to the decreased incidence of post–dural puncture headache. However, their studies do not support our suggestion of an increased incidence of failed spinal anesthesia. Identification of free-flow cerebrospinal fluid, a prerequesite of our study design, ensured proper placement of the needle. The reason for the discrepancy in the results of our two studies is unclear. Cesarini *et al.*'s[1] approach is to advance the needle 1–2 mm following identification of cerebrospinal fluid. It would be of interest to document the incidence, if any, of paresthesias experienced with "needle advancement" once cerebrospinal fluid has been identified, which was not documented in either of the above studies. A controlled prospective study is now in progress at our institution to assess the incidence of paresthesias, failed spinal anesthesia, needle damage, and post–dural puncture headaches.

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Adverse Outcomes and the Multicenter Study of General Anesthesia: I

To the Editor:—Forrest and colleagues[1] should be congratulated for their large, randomized, prospective clinical study in which they evaluated multiple independent predictors of severe perioperative adverse outcomes. However, there seems to be discrepancy between the text and the logistic coefficients presented in table 1. The article reports that obesity, smoking, and male gender all were predictors for severe

respiratory outcomes. Review of the data in table A3 seems to confirm this finding. Nevertheless, the model and the logistic coefficients from table 1 suggest that these factors are protective for severe respiratory outcomes. In fact, across all outcomes in which these factors were reported as significant predictors, in both table 1 and table A3, they seem to be protective. For example, take the hypothetical case used by the authors: a 60-y-old person with a smoking history and a history of high blood pressure. They report a 0.5% probability of this patient developing severe perioperative hypertension. If this patient were a nonsmoker, his risk of developing severe perioperative hypertension increases to 0.7%. Similarly, an obese male smoker has a computed risk of 0.5% of developing any severe respiratory outcome. His thin, female, nonsmoking counterpart's computed risk is 1.9%. Is this due to a typographic error? If not, can the authors explain this data?

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Adverse Outcomes and the Multicenter Study of General Anesthesia: II

To the Editor:—As a follow-up to the 1990 publication of the results of "Multicenter Study of General Anesthesia"[1,2] (a randomized, controlled clinical trial of four general anesthetics), a subset of the authors have published a secondary analysis of the original data, seeking predictors of severe perioperative adverse outcomes by a multistage method (univariate contingency table analysis followed by stepwise logistic regression analysis).[3] The general approach and statistical methods were up to date and received the approbation of a statistician editorialist, although he did specify the need for prospective validation of the predictors from this trial data set in a new data set.[4] Various demographics, disease states, operative procedures, and anesthetics were related to certain individual severe adverse outcomes and supersets of severe adverse outcomes, particularly changes in hemodynamic variables. These outcomes—hypotension, hypertension, tachycardia, and bradycardia—were defined as a 20% change from preinduction values. An adverse outcome was declared severe if a therapeutic intervention of a significant degree was required; one example was given—administration of an antiarrhythmic drug to treat ventricular arrhythmia. Observations of these hemodynamic variables continued for up to 7 days postoperatively. It is not specified what proportion of these hemodynamic events occurred during each of the perioperative and postoperative periods.

I wish to challenge some of these results, particularly those concerning anesthetic choice and adverse hemodynamic outcome. In their original report, it was explicitly stated that the low incidence of death, myocardial infarction, and stroke and the study size (46 events in 16,023 patients completing the protocol) prevented definitive comparisons between anesthetics for morbidity and mortality; there were 10 patients in each anesthetic group with one or more of these outcomes. The original report clearly showed different patterns of hemodynamic alterations among the anesthetics, more tachycardia with isoflurane, and more hypertension with fentanyl. In this new exploratory analysis, halothane, isoflurane, and fentanyl, as contrasted to enflurane, were classified as increasing the risk of "any severe cardiovascular outcome" (the superset of hemodynamic changes plus arrhythmias).

It is unreasonable at the present time to categorize hemodynamic variables as true outcome variables such as death, myocardial infarction, and stroke. Blood pressure and heart rate changes during anesthesia are a reflection of the dynamic interaction of patient disease, surgical procedure, anesthetic drugs, and clinical care. Such variables are considered process or intermediate variables. It is clearly plausible that changes in blood pressure and heart rate might be part of the process of producing real morbidity and mortality. If this correlation between process and outcome were statistically well established, then hemodynamic changes might be considered surrogate endpoints for the endpoints of interest.[5] Such might not be the case in these patients. Mangano reviewed and summarized an enormous body of literature, concluding that hypotension and tachycardia were predictors of perioperative cardiac morbidity; hypertension and arrhythmias were indeterminate as predictors.[6] Many of the studies considered important concerning hypotension and tachycardia were in patient populations with a high prior probability of ischemic heart disease. Moreover, the definitions of hypotension and tachycardia tended to be more stringent in the reports reviewed by Mangano than in the definitions by Forrest *et al.* Considering the generally healthy patients in the Multicenter Study (more than 90% ASA physical status 1 and 2), the rather modest vital sign deviations defined as hemodynamic abnormalities, the unreported specific meaning of significant therapy, and the lack of blinding as to anesthetic agent, allowing a bias in the willingness or attentiveness for treating arrhythmias and hemodynamic changes, I would argue that hemodynamic changes should be treated only as process variables in this study.

Forrest *et al.* do mention some appropriate reservations about the generalizability of their results. However, the loudest messages of their paper are that anesthetic choice alters the risk of hemodynamic changes and that hemodynamic changes are important outcome variables; thus anesthetic choice is an important determinant of outcome. Though not advocating a sloppy inattentiveness to hemodynamics during anesthesia, I believe a more conservative interpretation of their data is to be preferred for healthy patients: 1) the current level of research effort can not distinguish mortality and serious morbidity between the most common anesthetic agents, and 2) the clear differences in hemodynamic patterns among these anesthetic agents has an unknown, perhaps nonexistent, relationship with mortality and serious morbidity.

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