ciated with reductions in the risk for death at 2 yr ($P = 0.025$). The primary end-point of the atenolol long-term study[4] was the effect of atenolol on 2-yr mortality. In patients who have or are at risk for coronary artery disease who must undergo noncardiac surgery, treatment with atenolol during hospitalization reduced mortality ($P = 0.019$) and the incidence of cardiovascular complications for as long as 2 yr after surgery ($P = 0.008$). These two studies provide clear and statistically significant evidence to support the claims that perioperative atenolol reduced incidence of morbidity and mortality and improves survival in patients at risk for cardiac morbidity.

Dr. Leung does not understand the interpretation of univariable and multivariable models presented in table 2 of the Mangano *et al.* manuscript.[4] Once statistical significance is established, it is acceptable to do a secondary analysis of the data to look for other predictors of outcomes. We demonstrated clearly that atenolol reduced the risk of death. Diabetes mellitus and ischemia increased the risk of death. In the multivariable model we demonstrated that diabetes was statistically significant, but once patients with diabetes were excluded there was not sufficient power to demonstrate the effect of atenolol. Thirty-one percent of the patients in this trial had diabetes. If one excludes 31% of the patients in a trial, power is lost. That in no way brings the results into question; it simply implies that if one excludes 31% of the patients, one also loses power to see the effect. Furthermore, Dr. Leung is incorrect in stating that $P = 0.06$ (6% type I error) is not significant when $P = 0.05$ (5% type I error) is suddenly significant. It is simply the difference between a 5% and 6% chance of a type I error. Perioperative administration of atenolol reduces the incidence of perioperative myocardial ischemia, postoperative mortality, and cardiovascular complications.

Dr. Leung's statement that the use of perioperative beta blockade is based on misinformation is incorrect. It is important to understand statistics and study design before making such a statement. Physicians should clearly evaluate any therapy. Many trials of beta-blocker use demonstrate benefits in nonsurgical patients, including the ISIS-1 (International Study of Infarct Survival),[5] the MIAMI (Metoprolol in Acute Myocardial Infarction),[6] the MAPHY (Metoprolol *versus* Thiazide Diuretics in Hypertension),[7] and the ASIST (Atenolol Silent Ischemia Study)[8] trials. We added to this abundant literature and have clearly demonstrated that perioperative beta blockade improves survival in surgical patients at risk for cardiac morbidity.

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Catheter Tip Position and Baricity of the Local Anesthetic Have an Impact on Maldistribution in Microcatheter CSA

To the Editor:—We read with interest the study of Biboulet *et al.*, which shows that the orientation of the catheter tip has a major impact on the spread of local anesthetics (LA) irrespective of their baricity.[1]

These results are consistent with data from other studies using 28-gauge microcatheters and isobaric bupivacaine[2,3] and show convincingly that caudally positioned catheter tips are associated with a higher requirement of LA to reach the respective sensory level than cranially directed catheter tips. The results suggest that caudally extended catheter tip levels cause maldistribution of the LA and that cranially oriented catheter tips should be obtained in CSA.

However, the authors give no information how a cranial catheter tip position can be obtained in CSA. They do not mention the respective literature that relates to this issue. Data from a study of Ata *et al.* suggest that the paramedian lumbar approach may facilitate the spinal

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catheter passage into the subarachnoid space and possibly may provide more cranially directed catheters.* Yurino *et al.* described a preformed, coiled-tipped microcatheter that could easily be placed in the subarachnoid space within 2 min and provide adequate blocks in 94% of patients, suggesting that these catheters remained at the level of the puncture site or took a cranial direction.[4] In one study we investigated the impact of different needle designs, directional and nondirectional, on the intrathecal catheter tip position.[5] The use of a nondirectional Quincke needle was associated with a 35% incidence of caudad catheter tip positions, and the directional Sprotte needle provided no caudally positioned tips of the 28-gauge catheters.

The results of the three studies suggest that technical modifications can help to increase the percentage of spinal catheters with the intended cranial tip position.

Second, we would like to stress the different injection speeds of micro- and macrocatheters. The fact that maldistribution was not significantly related to the baricity of the applied LA in Biboulet's study may give the impression that hyperbaric LAs can be used in combination with microspinal catheters in the same way.

Rigler and Drasner clearly demonstrated in 1991 that a low injection speed produces inadequate distribution of dye-colored hyperbaric lidocaine.[6] The mean injection time of 1.0 ml is 50 s for 32-gauge and 28 s for 28-gauge microcatheters compared with 3 s for 20-gauge, large-bore catheters.

Therefore, the reduced injection speed through microcatheters appears to be an additional factor, besides the orientation of the catheter tip, which may significantly enhance the risk for maldistribution of hyperbaric LA. The combination of the reduced injection speed through microcatheters, high doses of hyperbaric LA, and caudally oriented catheter tips have produced significant maldistribution in models of the spinal canal and probably have caused the well-known cauda equina syndrome as a result of toxic concentrations of the hyperbaric LA at the dorsal sacral spinal nerves.[7–9]

Although the results of the spinal canal models cannot completely be transferred on patients in whom lower concentrations of hyperbaric LA may occur because of diffusion and vascular uptake, and although neurologic sequelae have also been reported after macrocatheter CSA, the conclusion in Biboulet's article that "hyperbaric solutions do not appear to be a clinical factor in the development of limited block" should be taken with caution. In our opinion, this conclusion is only justified for the use of large-bore catheters and must not be transferred to microcatheter CSA.

As a consequence we would like to recommend techniques that facilitate cranial catheter tip placement and advise against the combination of microcatheters and hyperbaric LA for CSA. In light of these aspects we agree with the statement of the authors that we should not discourage the use of microcatheters for CSA.

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In Reply:—Dr. Standl is right in highlighting the fact that several clinical studies have described techniques destined to decrease the incidence of caudally directed spinal catheters. However, it is important to note that these studies were performed when the role of the catheter's sacral direction in the occurrence of maldistribution was but an experimental hypothesis.[1,2] The point of our work was to objectively identify the clinical causes of maldistribution. Using 19-gauge, end-holed catheters, the study showed that the caudal orientation of the catheter tip is a factor of maldistribution rather than the caudal direction of the catheter. As such, the sacral flow of local anesthetics seems to be the most important factor of maldistribution; a cranially directed catheter can have a distally oriented catheter tip if a loop is created during catheter insertion, leading to a distal flow of local anesthetics.

Second, the role of injection speed, lower when local anesthetics are administered *via* microcatheters and experimentally evoked as being

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