

Letters to the Editor

Block of the Posterior Femoral Cutaneous Nerve

To the Editor:

The article by Barbero et al. (1) provided interesting data complementing our original description of an anterior approach to the sciatic nerve (2). However, their interpretation of our previously published data deserves the following comments: 1) the authors' representation of our published landmarks was incorrect. Our landmarks are based on the use of the inferior border of the anterosuperior iliac spine (AIS) and the superior angle of the pubic symphysis tubercle. These are the same as those described by the authors. Therefore the AIS-TS lines presented in Figure 1 in their article should be changed. 2) The posterior femoral cutaneous nerve (PFCN) is a branch of the sacral plexus, not a branch of the sciatic nerve. More importantly, it is a sensory nerve (3). Contrary to the authors' belief, the PFCN does not innervate the gluteal muscle! Therefore, the PFCN block while performing a sciatic nerve block should be considered a consequence of the local anesthetic diffusion toward the PFCN and does not reflect the intensity of the sciatic block. 3) In the Introduction of their article, the authors indicate that with our technique the block of the PFCN was inadequate (?) in 45% of patients, while in the Discussion they refer to a 50% success rate! Does this 5% difference refer to the difference between inadequacy and failure? 4) In our original description (2), we did not report on the extension of this block to the PFCN. After our original publication, a technical description was indeed published (4). In this technical paper, we reported a complete block of PFCN in 9 of 16 patients, and a partial block of PFCN in 6 of 16 (37%). The failure to block the PFCN in the study by Barbero et al. (1) and our studies led to the need for supplemental anesthesia. As indicated in our technical description (4), there is no doubt that the block of the PFCN is important for surgery in the posterior aspect of the thigh, but evidence supporting the need for a complete PFCN block for surgery at the knee and below without and without a tourniquet remains to be established. Thus, in 60 patients undergoing foot surgery with the use of a tourniquet under a femoral and a posterior popliteal block, a supplemental dose of sufentanil (5–10 μ g) was required only in one patient (5).

Further studies are required to determine the conditions in which the block of PFCN is required in the presence of a tourniquet for surgery at the knee or below.

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In Response:

We would like to thank Drs. Chelly and Delaunay for their comments. There is in fact an error in the abstract of our paper in

describing the anatomic landmarks: the inguinal line described is identical to that of Chelly and Delaunay (1). The correct description is developed in the Methods, locating the medial limit of the inguinal line on the superior border of the pubic symphysis, and the puncture site is in a more medial position than that described by Chelly and Delaunay (1).

Studies will perhaps soon demonstrate that this nerve is not necessary to tolerate a thigh tourniquet but, as described in Figure 3 in our article, posterior femoral cutaneous nerve (PFCN) anesthesia is necessary for surgery of the thigh, the knee, and the superior posterior part of the leg.

The PFCN is effectively a purely sensitive nerve, but it is often joined to the inferior gluteal nerve (60% of cases) from the sacral roots to the gluteal area (2). When stimulating the PFCN, a motor response can therefore be found by stimulating the associated inferior gluteal nerve. Setting aside the trivial slip that allowed a 5% difference to appear in the success rate of a study on 16 patients, our technique of including the patient's height as a variable to determine the landmarks seems to be useful to get a correct anesthesia of the PFCN (92% success rate for 53 patients studied).

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Anaphylaxis Is Not a Dose/Response Effect

To the Editor:

The case report (1) on latex anaphylaxis illustrates well the problem of identifying the trigger agent when there are two or more coincidental potential triggers such as, in this report, the administration of an antibiotic and the beginning of surgery.

However, it is important to understand that the anaphylactic reaction is the result of a specific IgE immunoglobulin binding into a receptor site and triggering a cascade. This releases many active agents, including tyrosine kinases, histamine, tryptases, proteoglycans, and leukotrienes (2,3). This process consumes both the IgE and the active agents, which is why at least 4 weeks delay is recommended before the provocative testing of suspected triggering agents (4).

It was therefore disturbing to read that the authors considered that it was "important to convert to a latex-free environment during the resuscitation." This is illogical, as the initial event has almost certainly depleted both the triggering antibody and the vasoactive agents. Any attempt to guess and remove the possible triggers is futile and may interfere with the appropriate treatment.

If a major anaphylaxis occurs, prompt active resuscitation is required. Removing the possible triggers is unnecessary, and even giving more of the triggering agent is not contraindicated, as this is not a phenomenon with a therapeutic dose/response curve, and the associated depletion of the IgE and mast cells means that there will not be any progression of the anaphylactic reaction if additional agent is given. This is a crucial difference from an adverse response with a dose relationship.

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In Response:

We would like to thank Dr. Russell for his interest in our recent case report addressing unrecognized latex anaphylaxis in a patient undergoing revision of total elbow arthroplasty (1). In his Letter to the Editor, Dr. Russell accurately discussed the physiologic foundations of anaphylaxis. However, we respectfully disagree with his contention of not having to remove and/or discontinue the suspected triggering agent(s) as a component of the resuscitative efforts. Anaphylaxis is indeed an IgE-mediated phenomenon based on mast cell degranulation and the release of potent vasoactive substances. However, one has to keep in mind that these reactions are far from uniform and represent a rather wide spectrum of pathology ranging from very mild to potentially lethal. In other words, despite the well-known fact that "anaphylaxis is not a dose-response effect," there is some correlation between the severity of anaphylaxis and the magnitude of antigen exposure—albeit not linear. There is also a strong correlation between serum tryptase levels and the reported severity of anaphylaxis (2). This suggests that in some patients, a large proportion of mast cells undergo activation and subsequent degranulation, while in other patients they do not. Although it is true that once a single mast cell has been activated by an allergen, it becomes desensitized for a period of 24 hours. However, the prompt removal of a triggering allergen would further diminish the chance of activating those mast cells not yet sensitized (personal communication, 2004, Lawrence B. Schwartz, MD, Richmond, VA). This is the physiologic basis—and current foundation of medical practice—to promptly discontinue all offending medication and/or agents believed to be triggering an anaphylactic reaction.

In brief, we strongly believe that most clinicians would agree that removal of an offending allergen during an anaphylactic reaction is prudent medical practice. Most would not rely on the hypothesis that the entire body has been acutely desensitized—thus making removal of offending agents unnecessary. Unfortunately, there is no *definitive* evidence within the literature to support our recommendations. However, these recommendations are considered "current medical practice" by many of our allergy colleagues.

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Differential Effects of Morphine and Codeine on Pupil Size: Dosing Issues

To the Editor:

The article by Knaggs et al. (1) showing that different opioids have different profiles and durations of effect on the measure of pupil size is interesting, in that tramadol had a delayed effect relative to morphine and codeine. However, there appears to be a problem in the design of the study. The authors found that morphine had a more marked effect on miosis than did codeine, and posited that this was due to codeine's weaker affinity than morphine for mu-opioid receptors. However, equianalgesic doses of the two drugs were not tested, and this certainly could be an alternative explanation for the results obtained. The dose of morphine used in the study was 0.125 mg/kg, and according to analgesic potency tables (2), an equianalgesic dose of codeine would be 1.63 mg/kg. Rather, a codeine dose of 1 mg/kg was tested. Therefore, it is not surprising that morphine generated a larger magnitude of effect. The authors also made a comment that there is limited data on the duration of opioid-induced miosis in the literature. In fact, the Knaggs et al. (1) study is preceded by many studies, some conducted over 25 years ago, examining the time course (up to 6 hours and longer) and magnitude of miotic effects of different opioids, both in opioid abusers (3) and in volunteers with a limited history of recreational drug usage (4).

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In Response:

We thank James Zacny for his letter pointing out a potential problem in the design of the study. We agree that an equianalgesic dose of codeine was not tested. Rather, we decided to use doses of opioid that approximated those actually used in clinical practice. Although this may have underestimated the magnitude of effect of codeine on pupil size, our aim was to provide information that may be of practical value. We apologize that this was not made clear in our paper.

We are also grateful for the two additional references that add to the relevant literature.

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Radiologic-Assisted Endotracheal Intubation

To the Editor:

The recently published case report by Reier and Reier (1) describes an elegant use of radiologic-assisted tracheal intubation in a patient with a compromised cervical spine. Successful tracheal intubation