Everybody Knows That the Dice Are Loaded

How Can We Block the Nerves That Innervate the Knee Joint Without Blocking the Nerves That Innervate That Joint?

André P. Boezaart, MD, PhD† and Hari K. Parvataneni, MD†*

In arguably the best and most beautifully illustrated account of the innervation of the knee joint to date, a truly international group of very accomplished authors from Denmark, Austria, and Canada—Drs Bendtsen, Moriggl, Chan, and Børglum—asks in this issue whether there is a nerve block or combination of nerve blocks available, discovered or not yet discovered, for the management of postoperative pain associated with total knee arthroplasty (TKA) that does not interfere with the motor function of the leg.[1] The most daring part of their discourse, if one translates the question that they ask, is of course the question itself: How can we block the nerves that innervate the knee without blocking the nerves that innervate the knee? This, as they elegantly demonstrate, is the real challenge, because, with the exception of the saphenous nerve (the infrapatellar branch of which is often transected during the anteromedial approach to TKA), all the nerves that innervate the knee joint and surrounding tissue (the pain generators following TKA) are mixed motor and sensory nerves—and everybody knows we should not block the motor nerves. Until we have a drug that can selectively block the sensory and pain fibers of a nerve, this may well be a bridge too far.

It was not long ago (a few decades) that patients who received total joint replacement were kept in the hospital for weeks and were not mobilized immediately but rehabilitated progressively as the physiologic responses from surgery abated. Since that time, there has been a progressive shift toward anesthetic and surgical optimization to allow for immediate rehabilitation starting even the day of surgery. The focus of all anesthetic work in this regard had been on safer options for anesthesia and pain control. Selective nerve blocks, especially continuous femoral nerve block (CFNB), have become the criterion standard for this. Now, the main focus, including of this article,[1] seems to be on providing optimal analgesia for TKA via nerve blocks while preserving muscle function (especially the quadriceps muscles). And, of course, everybody knows that we have to preserve the quadriceps function with our nerve blocks to optimize the surgical outcome. But does everybody really know this? Is this a scientific fact? Or is it perhaps dogma, folklore, culture, or belief that is not based on solid scientific evidence? Can what we do to block all the nerves that innervate the knee joint for a day or two really have a detrimental effect on the outcome of TKA in the short, medium, and long run? We must confront the question: "What has the most significant and prolonged effect on muscle (especially quadriceps function) during the recovery phase of TKA: pain control modality, the disease itself, or the surgery?"

While we would not be daring enough to venture into politics, this question reminds one a bit of the arguments around global warming. Everybody knows that the earth is warming and that we humans are causing it. Similarly, everybody knows that with continuous nerve blocks (CNBs) we may paralyze one or more of the quadriceps muscles, which worsens the surgical outcome of TKA. But does everybody know that? Over many millions or billions (who knows) of years, the earth has been warming and cooling in cycles lasting millions of years, and now we as humans think we can, in our short time here, influence this. Similarly, can we negatively influence the outcome of TKA with our short-term worsening of the quadriceps muscle function?

Humans compulsively, impulsively, intuitively, and repeatedly feel the need to explain and control (or feel in control of) events that we may not fully understand. Quadriceps (and other muscle) function is clearly weakened by surgery and arthrogenic muscle inhibition (AMI), in addition to any preoperative weakness.[2,3] This persists for weeks and months after surgery and is a variable independent of anesthetic or surgical type (patient factors are a major contributor).[4–7] Why is there pressure on us to address this long-term multifactorial issue simply with nerve block techniques that are a factor for 24 to 48 hours, at the most (if at all)?

The only direct controlling factor related to this subject is pain-related AMI. Optimal pain control that is safe and includes modalities to reduce inflammation via the inflammatory cascade should be the strategy—not muscle-sparing approaches for muscles that have already been weakened by surgery and

From the *Division of Acute and Perioperative Pain Medicine, Department of Anesthesiology, and †Department of Orthopaedics and Rehabilitation, University of Florida College of Medicine, Gainesville, FL.

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Address correspondence to: André P. Boezaart, MD, PhD, Division of Acute and Perioperative Pain Medicine, Department of Anesthesiology, University of Florida College of Medicine, 1600 SW Archer Rd, PO Box 100254, Gainesville, FL 32610 (e-mail: aboezaart@anest.ufl.edu).

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AMI. As we better understand the neural mechanisms of AMI, may CNB even improve muscle function in the long run and enhance rehabilitation?[2,3] Are we chasing a false target—shorter-term blockade with an obsession about motor-sparing options? Should we not be focusing instead on a means of obtaining longer-term pain control via blockade? Perhaps we should be considering continuous postdischarge blockade options to make rehabilitation virtually opioid-free (safety) while improving rehabilitation and range of motion because patients are experiencing less pain (efficacy). If the inflammatory cascade was somehow related to painful stimuli (perhaps via AMI),[2,3] it would confer an additional benefit to this change in philosophy.

If we accept, in an even more daring discourse, that increased postoperative quadriceps muscle dysfunction caused by nerve blockade does not negatively affect the surgical outcome in the long run, thus removing this issue from the equation, then providing effective analgesia to the knee would be uncomplicated and very effective. All we have to do would be to obey Hilton's Law of Anatomy (1862), which states that any nerve that innervates a group of muscles that moves a joint also innervates that joint and the skin that overlies the joint.[8,9] The work of Hébert-Blouin and colleagues[10] in 2013 that challenged this law and this soon-to-become classic account of the innervation of the knee joint by Bendtsen and colleagues[1] clearly illustrate that Hilton's Law is alive and well, with the nerve to the rectus femoris muscle the only glaring exception, as it supplies sensory innervation to the hip joint only and not the knee joint, while it does innervate a muscle that moves it. (In the defense of Hilton, though, his law states "group" of muscles and not individual muscles.)

From that, it should be abundantly clear that to effectively block pain generated by TKA, or any other major lower-extremity joint surgery for that matter, we have to block all the nerves that originate from the entire lumbosacral plexus (LSP), similar to having to block the entire brachial plexus to provide analgesia for major surgery to any major upper-extremity joint. Because CFNB is a more complete nerve block as outlined in the article by Bendtsen et al,[1] and perhaps blocking the afferent neural pathways of AMI, may there be any basis for blockade prior to surgical trauma to reduce the postoperative AMI and inflammatory response? If so, CFNB would be better than any of the lesser blocks in terms of coverage. Only a continuous LSP block would be superior.

Most of the research done to compare one approach to block one part of the LSP with another (adductor canal block with CFNB, for example) has unfortunately been lacking scientific validity. In almost every one of the studies, patients were divided into 2 groups: one group with the one block and the other group with the other block (and universally without a control group with no block "for ethical reasons"). Pain and quadriceps motor function—both extremely inaccurate and subjective measurements—were the major metrics, while all the patients in both groups received very effective multimodal analgesia—"for ethical reasons." In 1 study, all patients in both groups even received effective epidural analgesia, with the authors reporting no difference in pain but a difference in quadriceps function.[11] From this, they concluded that adductor canal block is superior to CFNB for pain associated with TKA. None of the studies reports on unwanted opioid adverse effects—the very reason we do CNBs in the first place. Meanwhile, Memtsoudis and colleagues[12] basically ended the debate on an increased risk of falling specifically due to peripheral nerve blockade.

Our research and clinical focus should therefore not be on the possible (and mostly irrelevant) negative influence of any short-term muscle inhibition, but on the vast positive effects of nerve blockade, possibly even decreasing AMI. Peripheral nerve blockade, especially CFNB, has had tremendous efficacy, improved safety, and reduced adverse effects after major joint replacement surgery, and our efforts going forward should focus on improved multimodal pain control, as well as tempering the local effects of surgery (other than pain) that result in AMI—including inflammation, swelling, and other injury cascades.[2] Furthermore, we should focus on early postoperative ambulation, early improvement of range of motion, early discharge from the hospital with or without longer-term ambulatory CNBs, and preoperative and postoperative rehabilitation of the quadriceps dysfunction caused by the disease and, much more importantly, by the surgery. We seem to be (mis)focused on selective, short-term blocks for TKA, as affirmed by this Bendtsen article. That's how it goes, ...everybody knows... (with apologies to Leonard Cohen).

REFERENCES

1. Bendtsen TF, Moriggl B, Chan V, Børglum J. The optimal analgesic block for total knee arthroplasty. *Reg Anesth Pain Med*. 2016;41:711–719.
2. Rice DA, McNair PJ. Quadriceps arthrogenic muscle inhibition: neural mechanisms and treatment perspectives. *Semin Arthritis Rheum*. 2010;40:250–266.
3. Ewell M, Griffin C, Hull J. The use of focal knee joint cryotherapy to improve functional outcomes after total knee arthroplasty: review article. *PM R*. 2014;6:729–738.
4. McKay C, Pravessis H, Doherty T. The effect of a prehabilitation exercise program on quadriceps strength for patients undergoing total knee arthroplasty: a randomized controlled pilot study. *PM R*. 2012;4:647–656.
5. Topp R, Swank AM, Quesada PM, Nyland J, Malkani A. The effect of prehabilitation exercise on strength and functioning after total knee arthroplasty. *PM R*. 2009;1:729–735.
6. Pua YH. The time course of knee swelling post total knee arthroplasty and its associations with quadriceps strength and gait speed. *J Arthroplasty*. 2015;30:1215–1219.
7. Mizner RL, Petterson SC, Snyder-Mackler L. Quadriceps strength and the time course of functional recovery after total knee arthroplasty. *J Orthop Sports Phys Ther*. 2005;35:424–436.
8. Carmichael SW, Spinner RJ. A glimpse of the past: John Hilton 1804–1878. *Clin Anat*. 2006;19:89–90.
9. Gibson A. John Hilton: "rest and pain." *Can Med Assoc J*. 1955;73:569–572.
10. Hébert-Blouin MN, Tubbs RS, Carmichael SW, Spinner RJ. Hilton's Law revisited. *Clin Anat*. 2014;27:548–555.
11. Kim DH, Lin Y, Goytizolo EA, et al. Adductor canal block versus femoral nerve block for total knee arthroplasty. *Anesthesiology*. 2014;120:540–550.
12. Memtsoudis SG, Danninger T, Rasul R, et al. Inpatient falls after total knee arthroplasty: the role of anesthesia type and peripheral nerve blocks. *Anesthesiology*. 2014;120:551–563.