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Femoral Nerve Block Causes Quadriceps Dysfunction

The incidence of ACL disruption is estimated between 8.1-36.9/100,000 persons per year,[1-2] and it is caused by either a contact or non-contact pivoting mechanism. ACL reconstruction (ACLR) is indicated for symptomatic instability, and for those who wish to engage in pivoting activities (e.g., soccer, basketball) but who do not have significant knee arthrosis. ACLR is a common operation – plus or minus 100,000 are performed annually in the United States.[3-4]

ACLR patients are young and healthy. In a recent population study[4] of 70,547 operations, the average age was 31.5 years, approximately two-thirds were male, and 97.4 percent of patients had no medical comorbidity.

A successful femoral nerve block (FNB) is useful regional anesthesia for ACLR, but it may also cause partial or complete motor block. This motor involvement has the potential for post-operative quadriceps dysfunction. Although infrequently measured, we present both direct

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Femoral nerve block (FNB) can provide substantial pain relief after anterior cruciate ligament (ACL) reconstruction.[1] Unfortunately, FNB can cause prolonged quadriceps dysfunction. Perhaps less widely appreciated is the fact that ACL injury and ACL reconstructive surgery can also cause quadriceps dysfunction. The question becomes how frequently do non-anesthetic factors account for postoperative quadriceps dysfunction?

Let us consider first the wider perspective of quadriceps weakness and the ACL-injured patient. Management of ACL ligament injury, particularly for the active individual, has long been a dilemma.[2] Ongoing quadriceps weakness remains an obstacle to the goal of returning the patient, particularly an active high-performance athlete, back to the pre-injury level of sport.

The quadriceps muscle can lack 20 percent or more of its previous strength six months after ACL reconstruction.[3] Strength deficits between the injured and the

and indirect evidence that dysfunction occurs and may carry negative consequence.

Early Dysfunction and Falls

One surrogate for dysfunction that has received significant recent attention in the literature is falls. Most of the data come from studies of FNB after total knee arthroplasty[5-7] (TKA). Although there are numerous other risk factors for falls in this patient population (e.g., age, narcotic use, co-morbidities), the data are concerning.

The rate of falls after FNB for TKA varies between 0.6-2 percent [Table 1, page 10]. The majority of these patients required further operations and extended hospitalization, suggesting that the associated morbidity is significant. Unfortunately, none of these authors included data on falls post-TKA without FNB, and none measured quadriceps (dys)function, yet all hypothesized that it played a role.

More concerning and convincing evidence comes from a report of falls after FNB for ACLR[8] [Table 1, page 10]. These authors retrospectively reviewed their randomized, controlled trial[9] (RCT) for the rate of falls after FNB. Four falls occurred after discharge in 155 patients (2.6 percent) who had received FNBs. No fall resulted in significant morbidity. Although the authors did not measure quadriceps function, none of the 78 patients in their study control group (saline FNB) suffered a fall.

Two studies have specifically documented quadriceps function after FNB. A pilot study of 36 patients receiving one of three different concentrations of ropivicaine FNB examined quadriceps function, mobilization and pain scores after TKA.[10] On the morning of the second post-operative day, they found measurable quadriceps weakness in 30 percent of patients receiving the highest concentration FNB, but weakness in only 11 percent and 16 percent of patients receiving lower concentrations ($p<0.05$). There were no differences at other time-points.

In an RCT of 24-hours FNB versus intra-articular injection following ACLR, therapists assessed 90 patients prior to discharge for the ability to weight bear, perform a quadriceps set and straight leg raise (SLR).[11] Only one patient, from the FNB group, could not bear weight. However, they found a significant ($p=0.004$) three-times-higher rate of inability to perform an SLR in the FNB group (40 percent) compared to the injection group (13 percent).

Other factors play a role in post-operative quadriceps weakness, including pre-operative function. Studies have demonstrated that ACL-deficient athletes are weaker than matched controls,[12] and pre-operative strength predicts post-operative strength in ACLR.[13] In the acute post-operative period, pain inhibition may also play a role, but this effect is difficult to quantify. We know that surgical factors do not influence short- or long-term post-operative weakness, including the

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Following Anterior Cruciate Ligament Reconstruction

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uninjured limb may persist for years following reconstruction.[4] Rates of arthrogenic muscle inhibition after ACL injury vary from 0-40 percent.[5] Arthrogenic muscle inhibition, or voluntary activation failure, may result from loss of mechanoreceptors from the ACL. Loss of mechanoreceptors disrupts the ligamentous-muscular reflex between the ACL and the quadriceps so that the muscle cannot recruit high-threshold motor units during voluntary quadriceps contraction. When a patient is asked to make a maximal effort contraction of the quadriceps, the muscle itself, though capable of a stronger contraction, is unable to produce one because of a neural to mechanical breakdown. In one series,[5] the involved side quadriceps was weaker than the uninvolved side for the entire sample of 100 patients following rupture. Thirty percent of these patients demonstrated voluntary activation failure. An additional study[6] confirmed that every ACL patient had weaker quadriceps on the involved side. The presence of voluntary muscle inactivation, along with associated muscle atrophy, accounted for 60 percent of the weakness exhibited. Studies such as these suggest that an important neurosensory connection between the ACL and the quadriceps muscle is disrupted when the ACL is ruptured. This contributes to the quadriceps weakness. Furthermore, ACL reconstruction surgery is associated with persistent quadriceps weakness that has been shown by EMG studies to be caused by inhibition of the femoral nerve.[7]

Given that this complex neuromuscular interplay reduces quadriceps strength following ACL rupture and repair, the FNB cannot be singled out as the cause for prolonged weakness, except in cases of actual neuropraxia. What are the risks of this occurring?

A recent review[8] indicates a risk of neuropraxia after surgery and FNB to be 3.4 out of 1,000 (95 percent CI: 0.4 to 28.1/1,000). This is based on data from four studies involving 13,378 patients (Auroy[9], Capdevila[10], Cuvillon[11], Fanelli[12]). Only one patient (out of more than 13,000) had a permanent neuropathy after FNB. It is notable that the Fanelli study used a multiple-injection technique, i.e., three separate muscular responses were sought for each FNB. It is conceivable that multiple injections increase the possibility of needle injury to nerves. It is also important that the Capdevila and Cuvillon studies reflect rates of neuropraxia after femoral nerve catheters, which may have higher rates of neuropraxia than single-injection FNB. Out of 683 femoral catheters, three patients had femoral nerve symptoms. CT studies ruled-out hematoma, while EMG studies found decreases in latency and amplitude in two patients. The patient with normal EMGs had complete resolution in 36 hours. The two patients with abnormal EMGs had complete resolution at eight weeks and 10 weeks, respectively. The Auroy series observed three

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use of a tourniquet[14,15] and the type of graft selected (patella tendon autograft versus allograft[16] or versus hamstrings tendon autograft[17,18]).

Late Dysfunction After FNB

Beyond one to two days after cessation of FNB, quadriceps dysfunction can occur if there is a femoral nerve palsy — an acknowledged complication of FNB. A recent review of 16 studies demonstrated a mean femoral palsy rate of 0.34 percent (range 0.04-2.81 percent), but only one case of permanent dysfunction.[19] However, three series published since have demonstrated similar palsy rates but with three cases of permanent dysfunction[6,7,20] [Table 2, page 11].

A permanent motor nerve injury would be devastating in an ACLR patient. However, even a transient motor palsy with a lengthy resolution has potentially negative consequences, as post-operative rehabilitation, including strengthening, is critical to successful ACLR. To our knowledge, this has not been discussed in the literature.

Conclusion

What we present is evidence that FNB produces post-operative quadriceps dysfunction. Although rarely considered, two studies suggest it is measurable in approximately one-third of patients. A more comprehensive and sophisticated study is certainly warranted to clarify the basal rate with and without FNB and possible dose or threshold effect of anesthetic.

More concerning, however, is the relationship between falls and FNB. It is an established concern in TKA patients, yet these patients have a lengthy hospital or rehab admission with nurses and therapists to assist mobilization. In contrast, ACLR patients are typically sent home within 24 hours of surgery with no trained support. And because ACLR patients are young and high functioning, they are more likely to be very active in the post-operative period, further increasing their risk of falling.

Finally, we note that the current level 1 evidence to support the analgesic efficacy of FNB for ACLR is equivocal — two positive studies,[9,21] and two negative studies.[11,22] Therefore, as surgeons and anesthetists, we should question whether a technique that may not even provide analgesic benefit for ACLR — but carries as high as a 2.8-percent transient nerve palsy risk (with the potential for complete injury) and a risk of falling as high as 2.6 percent, should be used in young, healthy, elective patients.

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Table 1. Falls & FNBs

Reference	N	Technique	Surgery	Falls
5	250	Bolus or bolus + infusion	TKA	5 (2%)
6	709	Bolus	TKA	12 (1.7%)
	261	No FNB	TKA	1 (0.4%)
7	469	Bolus + infusion	87% TKA	4 (0.9%)
	721	Bolus + <12 hours infusion	87% TKA	4 (0.6%)
8	76	Bolus + infusion	ACLR	3 (3.9%)
	79	Bolus only	ACLR	1 (1.3%)
	78	Placebo bolus	ACLR	0

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neuropraxias after 10,309 single-injection nerve blocks; this is the number we routinely quote to patients if they inquire about rate of neuropraxia after FNB.

Other case series confirm low rates of nerve injury after FNB. Watts and Sharma found one patient out of 261 to have a long-term complication after surgery and femoral-sciatic blockade. This patient had numbness of a toe that resolved at nine months — probably not related to the femoral block at all.[13] Swenson, reporting on 206 continuous nerve catheters, found no complications. Here the needle was placed with ultrasound guidance, 1 cm lateral to the nerve.[14] This series brings up several interesting points. Does ultrasound protect against nerve injury? Perhaps visualization of the needle and local anesthetic should reduce nerve injury, but a recent systematic review (Liu et al[15]) failed to show much evidence in support of that view. Similarly, if the nerve block needle is the source of postoperative nerve dysfunction, then avoiding the nerve should avoid neuropraxia. However, there is a case report to the contrary,[16] suggesting that the needle is not the culprit. A fascia iliaca block was placed for analgesia after hip arthroplasty, using a "double-pop" technique. The patient had quadriceps weakness that resolved in eight days. The authors proposed three mechanisms of nerve injury: incorrect identification of needle entry site, anatomic variation leading to needle-nerve contact, or a surgical cause of nerve injury. Unfortunately, the literature regarding the efficacy of FNB in the setting of ACL repair is heteregeneous with respect to patient population, actual type of reconstruction, and postoperative pain regimens. Young and middle-aged patients are averaged, the severity of the injury (presence or absence of meniscal injury) is sometimes overlooked, length of time from injury to operation differs, tourniquet times (a known source of muscle dysfunction[17]) are not consistently factored in, and types of rehabilitation are not uniform. FNB may not be as helpful for pain after harvesting a hamstring autograft (sciatic innervation), and use of an allograft removes the graft site as a source of pain. Additionally, the patient's psychological profile plays a role in the performance outcome.[18] Clearly more research is needed to define patients at risk for prolonged weakness and, in this individual, and perhaps any high-performance athlete where additional risk of any kind should be avoided, the FNB should not be used. Until then, for the patient suffering from severe pain despite multimodal analgesia, FNB is an intervention that may facilitate the transition to motivated rehabilitation.

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Table 2: Femoral Palsy From FNB

Reference	n	Technique	Surgery	Nerve palsies*
6	709	Bolus	TKA	5 (0.7%)
20	628	Bolus + infusion	87% TKA	4 (0.7%) (1 permanent)
7	1190	Bolus + infusion	TKA	9 (0.8%) (2 permanent)

*dysesthesia +/- weakness

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