Neuromuscular Prehabilitation to Prevent Osteoarthritis After a Traumatic Joint Injury

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Abstract: Post-traumatic osteoarthritis (PTOA) is a process resulting from direct forces applied to a joint that cause injury and degenerative changes. An estimated 12% of all symptomatic osteoarthritis (OA) of the hip, knee, and ankle can be attributed to a post-traumatic cause. Neuromuscular prehabilitation is the process of improving neuromuscular function to prevent development of PTOA after an initial traumatic joint injury. Prehabilitation strategies include restoration of normative movement patterns that have been altered as the result of traumatic injury, along with neuromuscular exercises and gait retraining to prevent the development of OA after an injury occurs. A review of the current literature shows that no studies have been performed to evaluate methods of neuromuscular prehabilitation to prevent PTOA after a joint injury. Instead, current research has focused on management strategies after knee injuries, the value of exercise in the management of OA, and neuromuscular exercises after total knee arthroplasty. Recent work in gait retraining that alters knee joint loading holds promise for preventing the development of PTOA after joint trauma. Future research should evaluate methods of neuromuscular prehabilitation strategies in relationship to the outcome of PTOA after joint injury.

INTRODUCTION

Post-traumatic osteoarthritis (PTOA) is a process resulting from direct forces applied to a joint that cause injury and degenerative changes. An estimated 12% of all symptomatic osteoarthritis (OA) of the hip, knee, and ankle can be attributed to a post-traumatic cause [1]. Andriacchi et al [2] described the in vivo pathogenesis of knee OA as occurring in 2 steps: an initiation phase and a progression phase. In PTOA, the initiation phase is the change toward an abnormal kinematic loading pattern that occurs after a traumatic injury to the knee joint and the breakdown of cartilage. The progression phase, which occurs after a critical level of cartilage loss, magnifies the effects of increased loads on an abnormal joint.

Degenerative changes after an initial joint injury, which may occur over the course of years to decades, are represented by a range of processes from microscopic (ie, cell death and altered cartilage from inflammation and free-oxygen radicals) to macroscopic joint-level trauma (ie, involving bone, ligaments, and cartilage), all of which lead to altered mechanics [3]. Participation in sports that involve high-impact and torsional forces also may contribute to joint injury and subsequent joint degeneration [4]. The resulting types of cartilage damage from such injuries differ in radiographic characteristics. For example, evenly distributed structural damage of the knee joint has been reported in soccer players who sustained anterior cruciate ligament (ACL) injuries and in whom PTOA subsequently developed, which is in contrast to primary nontraumatic OA, which typically is localized to the medial compartment of the knee [5].

O’Connor and Brandt [6] have suggested that the neuromuscular system has a primary role in the prevention of OA by regulating the forces transmitted to articular and perarticular tissues within the joint. Bennell et al [7,8] have suggested that neuromuscular exercises may help prevent the progression of knee OA, especially in patients with joint instability. A PubMed search for peer-reviewed studies performed in December 2011 that used the specific terms prehabilitation, rehabilitation, osteoarthritis, neuromuscular, and posttraumatic did not identify any longitudinal studies in which the authors assessed the role of neuromuscular prehabilitation to prevent PTOA after an initial traumatic joint injury.
Neuromuscular prehabilitation in the prevention of PTOA. In most of the studies that were identified, researchers reviewed the following topics: (1) the management strategies implemented after a traumatic knee injury, (2) the role of exercise in patients with OA, and (3) the role of neuromuscular rehabilitation after total knee arthroplasty. In this article, we review current evidence regarding how neuromuscular rehabilitation exercises after knee injury may promote a return to normal muscle and joint function and modify gait to reduce additional injury and reduce the risk of PTOA. We hypothesize that neuromuscular prehabilitation may reduce the development of PTOA and provide recommendations for directions of future research.

**REVIEW OF CURRENT LITERATURE**

Neuromuscular rehabilitation refers to the process of restoring neuromuscular control after a joint injury. In contrast, neuromuscular prehabilitation is the concept that the initiation of neuromuscular exercises after joint trauma may restore function and prevent the future development of PTOA. Topp et al [9] presented evidence that exercise prehabilitation, including strength and flexibility training before surgery, may result in better functional outcomes after total knee arthroscopy. Palmieri-Smith and Thomas [10] suggested that arthrogenic muscle inhibition (AMI) contributes to quadriceps weakness and may contribute to the development of PTOA after ACL injury. AMI refers to the inability to fully activate muscles after a joint injury. Consequently, quadriceps weakness, muscle atrophy, and neuromuscular dysfunction may contribute to pathogenesis of PTOA.

Neuromuscular prehabilitation (including re-establishing normal muscle recruitment patterns) that is designed to treat AMI may enable the restoration of joint function and prevent subsequent PTOA. However, the possible effects of AMI have been challenged [11] because some studies have not demonstrated significant muscle inhibition after an ACL injury. Palmieri-Smith et al [12] showed that a neuromuscular electrical stimulation protocol administered in women with mild to moderate OA was not effective in increasing quadriceps strength or activation during a 4-week period, although they did not assess the radiographic progression of OA or changes in pain.

The effects of surgical versus nonoperative treatment in the management of ligament and joint-related injuries have been compared in relationship to the development of knee OA. At 10 to 20 years after injury, up to 50% of athletes with previous ACL or meniscus injuries have been shown to exhibit clinical evidence of OA [13]. In a randomized clinical trial, Frobell et al [14] compared the effects of early ACL repair versus conservative management strategies after an initial ACL injury and reported no difference in functional outcome between subjects who received structured rehabilitation and delayed ACL repair and those who underwent early ACL repair. However, conclusions from that study were limited to young nonprofessional-level athletes (age range, 18-35 years) during a 2-year prospective follow-up. Most subjects in both cohorts required meniscus repair.

In a review of the therapeutic management of ACL injuries, Delince and Ghafil [15] concluded that in the management of complete-tear ACL injuries, no evidence-based research favors systematic surgical intervention over conservative treatment strategies such as neuromuscular rehabilitation. However, those authors recognized that current evidence is difficult to apply as a generalization because of multiple factors, including differences among the populations studied, therapeutic modalities, timing from injury to rehabilitation, and lack of consensus on the interpretation of clinical and radiographic measures of joint function to assess the outcome of interventions.

Neuman et al [16] reviewed the progression of OA after complete ACL injury in 100 consecutive patients during a 15-year period. In the population studied, 13 of 35 subjects who had undergone menisectomy exhibited radiographic evidence of OA. In contrast, radiographic evidence of OA was found in none of the remaining subjects’ knees. The authors concluded that meniscus integrity was the primary predictor of the progression of degenerative joint changes, although neuromuscular rehabilitation and early activity modification may have contributed to the prevention of knee OA.

Gait retraining is an additional form of neuromuscular prehabilitation that has been suggested as a method for shifting musculoskeletal loading away from joint contact areas prone to the development of OA [17]. External knee adduction moment has been linked to the presence, severity, and rate of progression of OA of the knee [18-20]. A modified gait that reduces the external knee adduction moment may be effective in preventing medial compartment knee OA, which is common in persons with a traumatic knee injury. The mediolateral knee position, foot progression angle, and trunk sway angle have been altered through gait modification in separate studies to successfully shift mechanical loads from the medial to the lateral tibiofemoral knee compartment [21-24]. Shull et al [25] trained patients in simultaneous upper and lower body gait modifications that were tailored for each patient and demonstrated precise knee compartment loading shifts. Wheeler et al [26] used real-time biofeedback knee loading measurements to enable individuals to self-select preferred gait modifications for altering knee loads. In that study, the subjects were young and healthy and thus good candidates for preventative rehabilitation.

**FUTURE DIRECTIONS**

A review of the existing literature reveals areas for future research with regard to the use of neuromuscular prehabilitation strategies in the management of post-traumatic joint injury to prevent PTOA. One of the authors (P.B.S.) is cur-
Currently using gait-modification techniques to retrain walking patterns in patients with both early-stage radiographic evidence and mild symptoms of knee OA. Qualifying patients are in good overall health and can walk for at least 25 continuous minutes without assistance. To date, most of the qualifying subjects have experienced a traumatic knee injury. All study subjects are trained to reduce medial compartment loading by increasing the tibia angle (which moves the knee position medially) and/or by increasing trunk sway (which moves knee loads laterally) depending on each patient's preference.

To train the subjects in new gait patterns, kinematic performance feedback is presented via small haptic vibration motors that are placed on each subject's back and previously injured leg, using a method similar to that described previously [25]. Training sessions in the laboratory occur once weekly for 7 consecutive weeks, and patients are strongly encouraged to practice walking with the new gait patterns on their own throughout the week. The initial results after the completion of testing in 7 patients revealed decreased knee pain and increased knee function after 6 weeks of gait retraining as quantified by a comparison of Knee Injury and Osteoarthritis Outcome Score surveys between the first and last testing sessions. For validation, those preliminary results require testing of a larger number of patients but are nonetheless an encouraging step forward in the examination of gait retraining as a conservative treatment for knee OA.

Future research should examine the muscle memory and learning retention associated with acquiring new walking patterns, which are necessary for long-term effectiveness. In addition, long-term changes in pain, function, and radiographic OA progression have not been studied with respect to movement retraining strategies for healthy individuals or those with early stage OA.

Additional studies should be encouraged to explore mechanisms for continued joint degeneration after injury, including AMI as it relates to knee PTOA. The limitations of neuromuscular prehabilitation with respect to the severity and types of joint injuries in the prevention of PTOA also remain an important area of future research. To our knowledge, no longitudinal studies have been published in which the authors conclusively demonstrate the role of neuromuscular prehabilitation in the prevention of PTOA. Given the natural history of OA, future studies should select a sample from a large population of subjects at risk for traumatic joint injuries and provide long-term surveillance spanning years. We recommend documenting the radiographic and clinical evidence of degenerative joint changes and detailing the methods of neuromuscular prehabilitation over time. Descriptive studies that document neuromuscular prehabilitation techniques and quantify changes in function may be more feasible to perform in the near term and would add information to the limited reports on neuromuscular rehabilitation that are currently available [27].

CONCLUSIONS
Neuromuscular prehabilitation may be important in restoring and preserving joint function by strengthening appropriate muscle groups, improving joint kinematics, and retraining gait patterns to prevent development of PTOA. We present preliminary evidence regarding how gait retraining as a form of neuromuscular prehabilitation may be effective in the management of knee injuries. Gait retraining that deviates from preinjury joint function should be considered in the context of each patient's age and activities. From a practical standpoint, gait retraining may be more appropriate in older individuals and in a nonathletic population that would tolerate changes in gait patterns. Gait retraining may be an appropriate preventative treatment for a select population at risk for OA. Future research in neuromuscular prehabilitation may help advance treatments to restore function and prevent future joint injury and disease for patients who have experienced a traumatic joint injury.

REFERENCES