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Generalized Vibratory Deficits in Osteoarthritis of the Hip

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Abstract

Objective—Lower extremity sensory deficits, including reduced proprioception, joint kinesthesia, and, recently, vibratory sense, have been described in subjects with osteoarthritis (OA) of the knee. However, comparable deficits in OA of the hip have not previously been evaluated. Vibratory perception threshold (VPT) is a reliable measure used to assess sensory deficits and is amenable to testing multiple body sites. This study examined VPT at the upper and lower extremities of subjects with hip OA compared with subjects without hip OA.

Methods—Fourteen subjects with symptomatic and radiographic hip OA were compared with 13 age-matched controls without hip OA. VPT was assessed using a biothesiometer. Five sites in the lower extremity and 1 site in the upper extremity (radial head) were evaluated and compared between OA and control subjects.

Results—VPT was significantly reduced at all 6 testing sites of the OA subjects compared with controls ($P < 0.05$ for all sites). VPT scores (mean \pm SEM volts) for OA subjects and controls were as follows: first metatarsophalangeal joint (13.5 ± 1.4 versus 7.4 ± 0.7), medial malleolus (18.1 ± 2.6 versus 11.2 ± 1.7), lateral malleolus (20.9 ± 2.4 versus 10.6 ± 1.5), medial femoral condyle (22.8 ± 2.9 versus 12.6 ± 1.3), lateral femoral condyle (26.7 ± 2.6 versus 16.2 ± 1.9), and radial head (10.2 ± 0.8 versus 7.5 ± 0.6).

Conclusion—To our knowledge, this is the first study to evaluate sensory deficits in hip OA and to demonstrate that there is vibratory sense loss at both the upper and lower extremities in these subjects compared with controls. The noted generalized deficits may have significant implications in the neuromechanical pathophysiology of OA.

INTRODUCTION

Patients with osteoarthritis (OA) of the knee have been shown to have lower extremity sensory deficits. These deficits, which are described as proprioceptive loss, have been measured by balance, joint position sense, and kinesthesia testing (1-3). The role of these deficits in the pathophysiology of knee OA is not clear. However, it has been hypothesized that either OA-mediated joint destruction can lead to these deficits or, conversely,

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AUTHOR CONTRIBUTIONS

Dr. Shakoor had full access to all of the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Study design. Shakoor, Block.

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preexisting sensory deficits can increase the risk for developing knee OA. The premise of the latter is that diminished sensory input may impair or reduce protective muscular reflexes around the joint, leading to increased mechanical load on the joint and subsequent damage (4,5). In either case, the nature and pathophysiologic role of sensory deficits in OA are subjects of active investigation.

Methodology to assess sensory dysfunction in OA has tended to be joint specific and limited to the knee, thus restricting investigation of sensory deficits in OA of other joints. Standard methods for testing proprioception in knee OA are subject to a variety of biases: they require movement of the arthritic knee and may therefore be confounded by disease severity and pain independent of any true lower extremity sensory deficits; they depend on patients' reaction times, and may be confounded by delayed reflexes; and they may be further confounded by their requirement for subject comprehension, concentration, and memory. Thus, the reliability and precision of these procedures has not been ideal (6). Furthermore, these methods are not adaptable to the hip, which is a physiologically more complicated joint. Therefore, the potential significance of sensory deficits in hip OA has not been evaluated; furthermore, it remains unclear whether the sensory deficits characteristic of patients with lower extremity OA are limited to the legs or whether they represent a more widespread systemic process.

We recently described deficits in another separate but closely related sensory pathway, vibratory sense, in subjects with knee OA (7). In addition to the knee, however, we observed that subjects with knee OA also had significant vibratory sensory loss in their feet and ankles compared with age-matched healthy subjects without OA (7). The methodology used to assess vibratory sense is technically much simpler to perform and involves substantially smaller test–retest variability compared with proprioception testing methodology (6-9). As a result, it permits a broader application, and is amenable to evaluating multiple sites on the body. The aim of the present investigation was to quantitatively evaluate vibratory sense in the lower and upper extremities of subjects with OA of the hip. Our hypotheses were that subjects with hip OA would have impaired vibratory sense at the lower extremity compared with subjects without hip OA, in a manner comparable with the deficits observed among patients with knee OA (7), and that this sensory deficit would be systemic and therefore detectable in the upper extremity as well.

SUBJECTS AND METHODS

Subjects

This research was in compliance with the Declaration of Helsinki. The study was approved through Rush University Medical Center's review board for studies involving human subjects, and written informed consent was obtained from all subjects. This was a cross-sectional study comparing sensory testing data from 2 groups of subjects: those with unilateral hip OA and age-matched controls without hip OA. The hip OA subjects were part of an ongoing study evaluating biomechanical factors in the pathophysiology of OA. Control subjects were recruited from other ongoing studies and by pamphlets in the local community. Inclusion criteria for the hip OA group included the presence of symptomatic OA of the hip, which was defined by the American College of Rheumatology clinical criteria for classification and reporting of hip OA (10) and by the presence of at least 30 mm of pain (on a 100-mm scale) while walking (corresponding to question 1 of the visual analog format of the knee-directed Western Ontario and McMaster Universities Osteoarthritis Index [WOMAC]) (11), and radiographic OA of the symptomatic hip, documented within the preceding 6 months, of grade 1 or higher out of a maximum of 4 as defined by the modified Kellgren/Lawrence (K/L) grading scale (12). Subjects in the OA group were excluded if they had symptomatic knee OA (presence of at least 20 mm of pain on question 1 of the

WOMAC visual analog scale). For the comparator group, age-matched subjects were recruited and were excluded if they had pain or symptoms of arthritis in either lower extremity (site-directed WOMAC visual analog scale score ≥ 10 mm). The exclusion criteria for all subjects included the presence of diabetes mellitus, systemic inflammatory arthropathy, history of knee or hip replacement, or evidence by history or physical examination of neurologic disease.

Testing procedures

All subjects underwent clinical evaluation and testing for vibratory perception threshold (VPT) using a biothesiometer (Bio-Medical Instrument, Newberry, OH) according to previously published methods (7,8). The biothesiometer has a vibrating tip that oscillates at a constant frequency of 120 Hz. A manual dial on the instrument is used to adjust the voltage; the amplitude of the vibration from the tip is proportional to the square of the applied voltage. The tip was applied with uniform pressure to the bony prominences at 6 sites: first metatarsophalangeal (MTP) joint, medial malleolus, lateral malleolus, medial femoral condyle, lateral femoral condyle, and the radial head. Uniform pressure was obtained by using the weight of the testing device itself as the sole source of pressure of the biothesiometer tip against the skin. This was facilitated by having the subjects in a dependent position during testing. For example, for testing the MTP joint, the plantar aspect of the subjects' feet was against the examining table. Likewise, for testing the left medial malleolus and left medial femoral condyle, subjects laid on their left side, and for the left lateral malleolus and lateral femoral condyle, on their right side. Prior to testing, subjects were given a demonstration of the effect of the biothesiometer on their hand, with an initial demonstration of pressure from the weight of the machine only with the voltage set at zero, and then a demonstration of the sensation of vibration with the voltage increased from zero to moderate and then high levels. At each of the 6 testing sites, the voltage on the biothesiometer was initially set at zero, and then the voltage was increased by 1 volt per second. The subjects remained supine and did not move the extremity during the testing procedure. Subjects were instructed to comment on their first sensation of vibration, and this was noted as the VPT. A higher VPT threshold represents a greater sensory deficit. The VPT measurement was then obtained a second time by resetting the voltage to zero and again increasing the voltage by 1 volt per second, and the second VPT measurement was averaged with the first VPT reading at each site to give the final VPT measurement reading. Previous studies using the biothesiometer to evaluate VPT, including our previous study (7), have demonstrated high reproducibility and reliability with this method (8,9,13).

Statistical analysis

Statistical analysis was performed using SPSS software (SPSS, Chicago, IL). An independent samples *t*-test was used to compare VPT of OA subjects with controls. Linear regression was used to evaluate the effects of age, body mass index (BMI), and sex on the results.

A power analysis was conducted using estimated effect sizes based on difference in VPT values for the 2 groups (14). The effect size was *d*, which is the difference between means divided by the pooled standard deviation. The effect sizes were large ($d = 0.87-1.94$) and yielded the following power estimates: assuming an effect size of 1.0, a sample size of 13 subjects with OA of the hip and 13 control subjects resulted in a power of 0.80 to detect a significant difference at the 0.05 level for both groups.

RESULTS

Fourteen OA subjects and 13 control subjects completed the study. The OA group included 4 (29%) women and 10 (71%) men, and the control group included 7 (54%) women and 6 (46%) men ($P = 0.252$). The mean \pm SD age of the OA and control subjects was not significantly different (64 ± 10 years and 59 ± 10 years, respectively; $P = 0.173$). The mean \pm SD BMI was similar in the OA group compared with the control group (27.1 ± 4.5 kg/m² and 27.7 ± 4.6 kg/m², respectively; $P = 0.517$). The OA group included 3 subjects with K/L grade 2, 5 subjects with K/L grade 3, and 5 subjects with K/L grade 4 radiographic hip OA.

Within each of the 2 groups, VPT assessment revealed an increase in VPT from distal testing sites to more proximal sites ($P = 0.026$), as expected among other populations (15,16). In addition, however, the OA subjects had significantly higher VPTs at each of the 6 test sites compared with the controls. Importantly, the subjects with hip OA had elevated VPTs not only throughout their lower extremities but also at their radial heads. Large effect sizes were observed at each site despite the small sample size (Table 1).

Secondary analyses using linear regression were also performed to evaluate the effects of age, BMI, and sex on the results. After adjustment for these variables, the results did not substantially change. Because there were differences (not statistically significant) in sex between the OA and control groups, a subgroup of the original study population that was closely matched for sex was also evaluated, and the primary results did not change.

DISCUSSION

This study demonstrated that there are significant sensory deficits associated with OA of the hip, and that these deficits are generalized and involve both the upper and lower extremities. These observations may have significant implications for both the future assessment of sensory deficits in OA and our understanding of the neuromechanical pathophysiology of hip OA.

Previous studies have reported sensory deficits in lower extremity OA, but these studies have primarily been limited to assessing deficits in proprioceptive acuity and balance in knee OA (1-3). Proprioceptive sense is believed to propagate from peripheral mechanoreceptors through large-fiber (specifically, $A\alpha\beta$) afferent nerves to the dorsal root ganglia of the dorsal columns of the spinal cord (17). Vibratory sense also travels through similar peripheral neurologic pathways (17). We recently demonstrated that vibratory sense was significantly diminished in subjects with OA of the knee compared with age-matched healthy subjects (7). In the present study, we observed impairments in vibratory sense in subjects with hip OA that were quantitatively comparable with those that we previously reported in subjects with knee OA (7).

In addition to deficits at the lower extremity, the finding of significant deficits in vibration at the upper extremity of hip OA subjects compared with controls is intriguing. The prior observation that the unaffected contralateral knee of subjects with unilateral knee OA had proprioceptive loss was originally explained by either 1) an OA-initiated phenomenon in which sensory loss from localized, OA-associated degeneration and damage to mechanoreceptors at the affected knee was propagated through neurologic feedback mechanisms to produce similar deficits at the non-diseased contralateral knee, or 2) an inherent generalized neurologic deficit, which was a risk factor for the development of OA (18). The premise of the second hypothesis is that diminished sensory input may interfere with or diminish protective muscular reflexes around the joint, leading to increased mechanical load on the joint and subsequent damage to cartilage (4,5). The fact that both upper and lower extremity deficits were observed in the current investigation tends to favor

the latter explanation and suggests that indeed these subjects may have a generalized deficit in sensation, which includes the upper extremity as well.

It should be recognized that neither this study nor previous studies have established a causal relationship between the sensory deficits and the development of knee OA. Therefore, it is not clear whether these deficits precede or follow the development of knee OA. Second, although proprioception and vibration sense travel through similar neurologic pathways, future studies should evaluate correlations between these sensory modalities. Finally, when evaluating for sensory deficits at the upper extremity, the presence or absence of hand OA should also be noted in future investigations.

To our knowledge, this is the first study to evaluate VPT or any sensory modality in subjects with OA of the hip and to demonstrate that there is vibratory sense loss at both the upper and lower extremity of these subjects compared with controls. Vibration deficits have now been observed in OA of both the knee and hip. VPT should be considered a useful technique to evaluate sensory loss in OA, and the pathophysiologic role of vibratory deficits in lower extremity OA warrants further evaluation.

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REFERENCES

1. Hurley MV, Scott DL, Rees J, Newham DJ. Sensorimotor changes and functional performance in patients with knee osteoarthritis. *Ann Rheum Dis.* 1997; 56:641–8. [PubMed: 9462165]
2. Barrett DS, Cobb AG, Bentley G. Joint proprioception in normal, osteoarthritic and replaced knees. *J Bone Joint Surg Br.* 1991; 73:53–6. [PubMed: 1991775]
3. Pai YC, Rymer WZ, Chang RW, Sharma L. Effect of age and osteoarthritis on knee proprioception. *Arthritis Rheum.* 1997; 40:2260–5. [PubMed: 9416866]
4. Sharma L. Proprioceptive impairment in knee osteoarthritis. *Rheum Dis Clin North Am.* 1999; 25:299–314. [PubMed: 10356419]
5. Shakoor N, Moio K. A biomechanical approach to musculoskeletal disease. *Best Pract Res Clin Rheumatol.* 2004; 18:173–86. [PubMed: 15121038]
6. Lephart, SM.; Fu, FH. Proprioception and neuromuscular control in joint stability. Human Kinetics; Champaign (IL): 2000.
7. Shakoor N, Agrawal A, Block JA. Reduced lower extremity vibratory perception in osteoarthritis of the knee. *Arthritis Rheum.* 2008; 59:117–21. [PubMed: 18163397]
8. Frenette B, Mergler D, Ferraris J. Measurement precision of a portable instrument to assess vibrotactile perception threshold. *Eur J Appl Physiol Occup Physiol.* 1990; 61:386–91. [PubMed: 2079057]
9. Van Deursen RW, Sanchez MM, Derr JA, Becker MB, Ulbrecht JS, Cavanagh PR. Vibration perception threshold testing in patients with diabetic neuropathy: ceiling effects and reliability. *Diabet Med.* 2001; 18:469–75. [PubMed: 11472466]
10. Altman R, Alarcon G, Appelrouth D, Bloch D, Borenstein D, Brandt K, et al. The American College of Rheumatology criteria for the classification and reporting of osteoarthritis of the hip. *Arthritis Rheum.* 1991; 34:505–14. [PubMed: 2025304]
11. Bellamy N, Buchanan WW, Goldsmith CH, Campbell J, Stitt LW. Validation study of WOMAC: a health status instrument for measuring clinically important patient relevant outcomes to antirheumatic drug therapy in patients with osteoarthritis of the hip or knee. *J Rheumatol.* 1988; 15:1833–40. [PubMed: 3068365]
12. Kellgren JH, Lawrence JS. Radiological assessment of osteoarthrosis. *Ann Rheum Dis.* 1957; 16:494–502. [PubMed: 13498604]

13. Davis EA, Jones TW, Walsh P, Byrne GC. The use of biothesiometry to detect neuropathy in children and adolescents with IDDM. *Diabetes Care*. 1997; 20:1448–53. [PubMed: 9283795]
14. Cohen, J. *Statistical power analysis for the behavioral sciences*. Lawrence Erlbaum Associates; Hillsdale (NJ): 1988.
15. Halar EM, Hammond MC, LaCava EC, Camann C, Ward J. Sensory perception threshold measurement: an evaluation of semiobjective testing devices. *Arch Phys Med Rehabil*. 1987; 68:499–507. [PubMed: 3619613]
16. Inami K, Chiba K, Toyama Y. Determination of reference intervals for vibratory perception thresholds of the lower extremities in normal subjects. *J Orthop Sci*. 2005; 10:291–7. [PubMed: 15928892]
17. Goetz, CG. *Textbook of clinical neurology*. 2nd ed.. WB Saunders; Philadelphia: 2003.
18. Hurley MV. The role of muscle weakness in the pathogenesis of osteoarthritis. *Rheum Dis Clin North Am*. 1999; 25:283–98. [PubMed: 10356418]

Table 1

Vibration perception thresholds in subjects with hip OA compared with control subjects*

	1st MTP	Medial malleolus	Lateral malleolus	Medial femoral condyle	Lateral femoral condyle	Radial head
OA VPT, mean \pm SEM	13.5 \pm 1.4	18.1 \pm 2.6	20.9 \pm 2.4	22.8 \pm 2.9	26.7 \pm 2.6	10.2 \pm 0.8
Control VPT, mean \pm SEM	7.4 \pm 0.7	11.2 \pm 1.7	10.6 \pm 1.5	12.6 \pm 1.3	16.2 \pm 1.9	7.5 \pm 0.6
t(df)	3.7 (20)	2.2 (25)	3.5 (25)	3.2 (18)	3.2 (25)	2.5 (20)
P value	0.001	0.040	0.002	0.005	0.004	0.022
Adjusted P value [†]	0.030	0.050	0.022	0.034	0.010	0.029
Effect size	1.94	0.87	1.43	1.32	1.25	1.15

* OA = osteoarthritis; MTP = metatarsophalangeal joint; VPT = vibratory perception threshold.

[†] Adjusted for age, sex, and body mass index.