170-175 *Clinical rheumatology*, 1993, **12**, N° 2

Proprioceptive Sensibility in Women with Normal and Osteoarthritic Knee Joints

R. MARKS, H.A. QUINNEY*, J. WESSEL**

Summary **The objective of this study was to quantify the effect of degenerative joint change on the proprioceptive acuity of women with osteoarthritis (OA) of the knee. Middle-aged women with OA of the knee, age-matched healthy women, and younger healthy women were studied. Tests examined the ability of subjects to correctly reproduce knee angles in weightbearing and non-weightbearing situations. The knee angles were photographed and measured in degrees. The absolute error (AE) of each matching test was calculated and the mean AE of two tests was used as the criterion variable in a three-way analysis of variance (ANOVA). The relationship between the reproduction error and the osteoarthritic subjects' self-paced walking speed was also examined. Under both test conditions the AE of the osteoarthritic subjects was greater than that of the healthy subjects ($p<.05$). There was, however, no significant relationship between the AE measurements of the patients and their walking speed. These data indicate that while women with OA may have poorer proprioception than healthy controls, this impairment may not affect their walking ability.**

Key words **Proprioception, Knee Joint, Osteoarthritis.**

INTRODUCTION

Anatomical studies have verified the existence of sensory receptors in the capsular and pericapsular tissues of synovial joints (1-4). These receptors project information to many levels of the CNS (5), and are thought to mediate information on joint motion and joint position (6,7). The same receptors have been linked to the postural reflexes of muscles acting on these joints, and hence to control of muscle tone (1,8).

In rheumatic diseases such as osteoarthritis (OA), however, localized degenerative joint changes could affect the function of those proprioceptors located within the capsular and pericapsular tissues, thereby altering sensory input. As a result of altered sensory input, the conscious appreciation of limb position (9-11) as well as limb function might be compromised (12,13).

Skinner et al. (14) demonstrated that persons with OA of the knee had greater difficulty matching knee angles in sitting in response to passive displacement cues than healthy age-matched controls. The proprioceptive deficit was attributed, in part, to the disease-related destruction of joint sensory receptors. Since the magnitude of the impairment varied inversely with the subjects' cadence, the investigators inferred that the position sense loss had resulted in slower locomotion.

Another body of literature has stressed the importance of muscle receptors (15-19) as well as the role of active muscle contraction in the calibration of judgments of the position of a limb in space (23). On this basis, the ability to reproduce specific joint positions in an individual with knee OA might not be impaired when subjects contract their muscles or carry out functional activities. On the other hand, muscle pathology (20) or factors such as articular pain sensation, leading to disease-related changes in reflex activity (8), might alter muscle receptor input, and thereby, the appreciation of limb position (14) and limb function (21).

By failing to include active displacement cues in their tests of position sensitivity, however, the findings of previous investigators (14,22,47) were largely limited to evaluating the effects of OA on the function of the proprioceptive contribution of the joint sensory receptors. In addition, by performing their tests solely in a nonfunctional situation, the inferred relationship between the

St. Michael's Hospital, Toronto; Department of Physical Education and Sports Studies, The University of Alberta, Edmonton, Canada; *The Department of Physical Education and Sports Studies. The University of Alberta, Edmonton, Canada. **Department of Physical Therapy, The University of Alberta, Edmonton, Canada.

Table I: *Description of study subjects*

Group		Age yrs	Weight kg	Height cm
Young	X	20.57*	56.50*	165.00
	SD	2.05	6.64	4.52
Old	X	48.20	64.16**	162.20
	SD	6.97	8.55	7.45
OA	X	54.60	79.47	162.40
	SD	9.87	16.34	6.11

* significantly different ($p<.01$) from old and OA groups; ** significantly different ($p<.01$) from OA group.

perceptual deficit found in those with knee OA and their unsteadiness during gait was not conclusive.

In the light of the above considerations, the objectives of the present study were the following: 1) to compare the ability of persons with and without OA of the knee joint to extract information on knee position in weightbearing (WB) and nonweightbearing (NWB) situations using active joint motion as the sensory cue; 2) to evaluate the relationship of the angle reproduction error measurement of the osteoarthritic subjects and their self-paced walking speed.

SUBJECTS

The subjects were ten women, aged 37-64, with radiographic evidence of OA of the knee, ten healthy control women matched for age and activity, and ten healthy younger women, aged 18-23 (Table I). OA subjects also met at least three of the following six criteria (24): 1) age> 50 years; 2) morning stiffness < 30 minutes; 3) crepitus on movement of the knee joint; 4) bony tenderness at the knee joint margins; 5) bony enlargement palpable or visible; 6) no palpable warmth.

Subjects with neurological conditions or any musculoskeletal disease other than OA were excluded from the study. Informed consent was obtained from all subjects.

METHODS

Each subject participated in two test sessions spaced one week apart but occurring at approximately the same time of the day. At the first session, age, height and weight were recorded (Table I) and position sense was tested. The OA subjects also performed a self-paced walking test. Position sense and ambulation were assessed in exactly the same manner at the second session. All tests were performed twice at each test session. In three cases, OA subjects were not able to complete the tests performed at the second session due to a flare up in their disease.

Measurement of joint position sense

Joint sense was recorded as the error occurring when the subject tried to actively reproduce a criterion position of the knee. Control subjects had their dominant knee tested (25), while OA subjects had the more affected joint measured. All tests were performed with the subject standing barefoot on a smooth, firm, level surface using a hand support when required. For the WB test, the subjects stood on the test limb and tried to reproduce a criterion angle between 20° and 40° of knee flexion. For the NWB test, the subjects stood on the other limb and bent the test knee to a criterion angle between 70° and 90° (Figs. 1 and 2).

For each test, the subjects maintained the criterion position for 5 seconds and then returned the leg to the starting position of knee extension. After 7 seconds, they attempted to actively reproduce the criterion position. The order of testing was randomized, and the combinations were balanced among the groups.

Both the criterion angle and the reproduction angle were recorded photographically using a Polaroid SX 70 Land Camera, Model #2. The camera was mounted on a tripod 77 cm from the floor and 185 cm from the test limb. It was set on automatic focus. To facilitate the identification of anatomical landmarks on the photographs, the proctor placed reflective markers, 2 cm diameter, mounted on a contrasting background, 2.5 square cm, on the greater trochanter, lateral epicondyle and lateral malleolus of the subjects' test limb. Subjects were photographed in the sagittal plane with the test limb facing the camera.

Knee joint angles were determined directly from the photographic prints by measuring the angle formed by lines connecting the centres of the markers. The absolute error (AE) (i.e. the absolute difference between the criterion and reproduction angles) was used as the measure of position sense.

Gait measurements

OA subjects were instructed to walk at their preferred speed over a 13 meter walkway. Walking time was recorded in seconds using photocells placed at the beginning and end of the walkway. Subjects walked a short distance at their preferred speed before actual walking time was recorded. Gait velocity was calculated from the walking time scores.

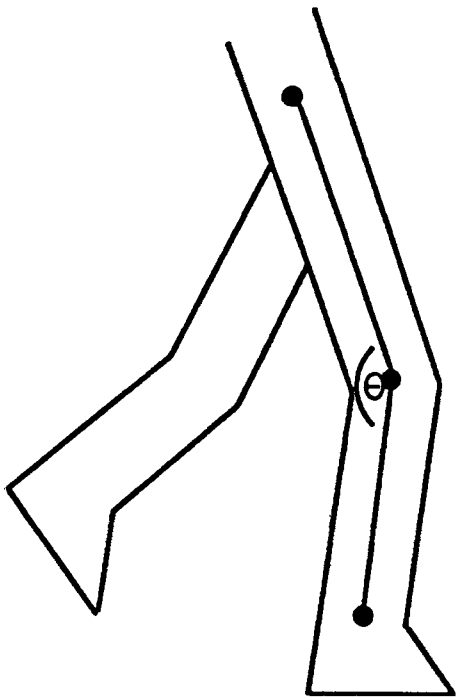

Fig. 1: Schematic representation of the reproduction tests performed in weightbearing position.

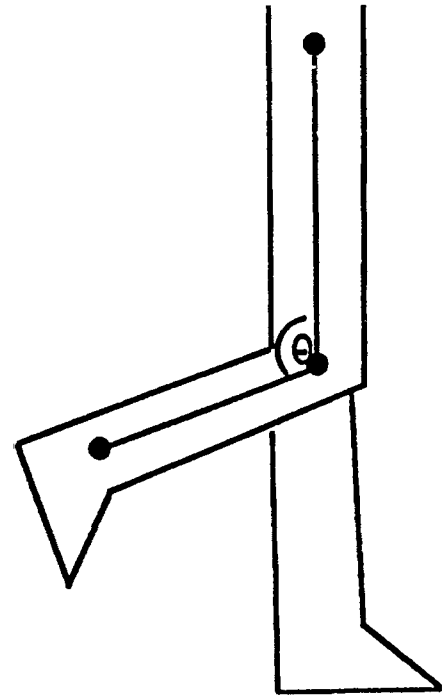

Fig. 2: Schematic representation of the reproduction tests performed in nonweightbearing position.

Data reduction and analyses

Anthropometric and age differences among the three groups were examined using a one-way analysis of variance (ANOVA). Measurement precision within and between sessions was assessed by the standard error of measurement (SEM). Group differences in mean AE across tests and sessions were determined using a three-way ANOVA with repeated measures on two factors (group vs. time vs. test position). The Newman-Keul post hoc test was performed to examine the significant ($p<.05$) main and interaction effects. Intraclass correlation coefficients (ICC's) were calculated to examine the reliability of the walking tests. The relationship between the gait velocity of the osteoarthritic subjects and their angle reproduction error scores recorded during session one was evaluated using linear regression.

RESULTS

The descriptive characteristics of the study subjects are presented in Table I. As indicated, the older controls were significantly heavier then the young controls, and the osteoarthritic subjects were heavier than the age-matched controls ($p<.05$).

The results of the WB and NWB tests are presented in Tables II and III. As revealed by ANOVA there was a statistically significant group-position interaction (see Table III). The SEM of the tests is displayed in Table IV.

Table II: *Weightbearing (WB) and nonweightbearing (NWB) absolute error measurements (AEs) over trials and test sessions*

Group		Session 1				Session 2			
		WB		NWB		WB		NWB	
		1	2	1	2	1	2	1	2
Young	$\bar{X}$	2.5	2.6	3.2	3.1	1.7	2.1	1.7	3.3
	SD	1.7	1.8	2.5	1.8	0.7	1.5	1.4	2.3
Old	$\bar{X}$	2.9	2.3	3.8	2.9	2.3	2.4	5.5	4.4
	SD	1.9	1.4	2.2	1.8	1.3	1.6	4.1	2.9
OA	$\bar{X}$	3.7	4.6	8.7	4.2	3.9	3.7	7.8	4.2
	SD	2.8	3.2	3.4	3.5	1.9	2.6	5.7	1.5

Table III: *Mean absolute error (AE) of trials recorded over two test sessions*

Group		Session 1		Session 2	
		WB	NWB	WB	NWB
Young	X	2.55	3.25	2.55	2.80
	SD	1.69	1.90	1.69	1.62
Old	X	2.60	3.35	2.35	4.95**
	SD	1.67	1.75	1.42	3.50
OA	X	4.10*	6.80**	3.78*	6.07**
	SD	2.78	3.45	2.24	3.60

*significantly different ($p < .05$) from young and old groups; *significantly different ($p < .05$) from WB.

Table IV: *The standard error (SEM) of repeated absolute error (AE) measurements, in degrees, over trials and test sessions*

Group	WB Test			NWB Test		
	Session		Inter-session	Session		Inter-session
	1	2	1X2	1	2	1X2
Young	.57	.34	.33	.66	.50	.64
Old	.31	.36	.95	.48	.70	.86
OA	.33	.68	.63	.58	.29	.76

The within and between session ICC's of the walking tests performed by osteoarthritic subjects were 0.97 and 0.78, respectively. The Pearson correlation coefficients for the variables of AE and gait velocity were -0.18 ($p = .60$) (WB) and 0.40 ($p = .24$) (NWB).

DISCUSSION

The present study comparing the ability of healthy women and women with knee OA to reproduce knee angles in WB and NWB situations demonstrated that irrespective of age, the mean AE of the healthy subjects was significantly lower than that of the osteoarthritic subjects under both test conditions (Table III). This result concurred with the findings of Skinner et al. (14) who showed that the ability to reproduce knee angles in sitting was significantly impaired in persons with knee OA compared to normal healthy age-matched and younger controls. The results also accorded with those of Glenross and Thornton (26) who found significant differences in angle reproduction error between sprained ankles and unsprained ankles. They were also in agreement with those of Barrack et al. (27) who recorded a similar proprioceptive loss when ballet dancers with knee joint laxity were compared with normal healthy controls.

In their study, Skinner and co-workers (14) attributed the proprioceptive deficit of those with osteoarthritis, primarily, to disease-related destruction of the joint afferents. Despite an attempt, however, to eliminate cutaneous and muscle contraction cues as sources of sensory input in their experiment, there was no indication that these were silent during passive positioning of the limb prior to rematching. Similarly, Barrack et al. (27) were not able to ascribe the poorer position sensitivity of ballet dancers to either joint or muscle receptors. Clark et al. (29), however, found only a modest decrease in the appreciation of limb position when the joint and skin around the normal knee joint were anaesthetized which suggested that muscle receptors were the primary determinants of knee position sense.

The more accurate appreciation of joint position occurring following active movement has provided additional support for the view that muscle receptors play a primary role in mediating positional information (30). The role of muscle receptors as a source of positional information is also supported by the observation that joint receptors fire predominantly at the extremes of range (29) and not in the working range of the joint. Thus, the finding that the present angle rematching tests were still carried with greater error by the osteoarthritic subjects than the controls might be interpreted as being due to abnormal input from muscles. This could occur due to weakness (48) or impaired fusimotor input (49).

Because joint as well as muscle receptors have been found sensitive to muscle tension (32-34) and because deficient input to the muscle receptors could, in turn, result directly from damage to the joint receptors (8), the present findings could not be attributed specifically to either impaired muscle or joint receptor input. In addition, because the ability to recognise position may involve central (5,49) as well as peripheral mechanisms (32,40,44,48), it was not possible to ascribe the present deficiency specifically to a peripheral afferent mechanism.

Proprioception might also be differentially influenced by alterations in input from pressure receptors in other joints as well such as load receptors in the leg extensors which signal changes of the projection of the body's centre of mass with respect to the feet (46). Moreover, muscle weakness or obesity could alter the speed of limb movement and thereby the velocity of stretch applied to the peripheral afferents (38,39), eliciting an inadequate proprioceptive response. Conceivably, stretch applied to muscles of different sizes might also impact differentially on proprioceptive sensitivity (28,35).

The ability to integrate spatial information as well could be impaired by other sensory events which accompany the disease, such as pain. Skinner et al. (14), however, found no relationship between the reproduction error measurements of persons with knee OA and

their pain. We also observed a poor correlation between WB pain and the present angle reproduction measurements in this group. Nevertheless, OA subjects on medication for pain may well suffer depression of the functioning of cortical neurons, thereby shrinking their receptive fields, reducing neuronal excitability, and extinguishing responsiveness of higher order feature detecting neurons, as reported to occur for anaesthetics (45). Pain might also interfere with the central processing capacity of short-term memory received from visual or proprioceptive inputs, compounding the difficulty experienced by persons with diseased knees to appreciate body postures accurately after initial presentation of the standard.

In addition, although head position was stabilized throughout by having subjects focus on a wall marker at eye level, and no movements other than those of the test knee were allowed, subtle group differences in visual or vestibular input due to weakness of the stabilizing musculature might have distorted the processing of sensory signals from the osteoarthritic limb. Finally, fatigue due to stress on the muscles could result in inconsistent movements and the use of different muscle groups to assist in the matching movements.

In both cases, however, the finding that the algebraic error of the osteoarthritic cohorts fell beyond two standard deviations of those recorded by the healthy subjects suggested that both tests were sufficiently sensitive to discriminate between subjects with and those without knee joint pathology. Theoretically, however, due to the differential inputs, the NWB test, as applied in the present study, probably provided the more sensitive indicator of proprioception per se than the WB test, since the relative contribution of the vestibular, visual and composite afferent signals from joints, muscle and skin could not be differentiated in the one-legged standing test. Furthermore, because the sensation of larger movements (as was the case with the NWB test) requires activation of a greater number of receptors (41,42), this test might be the more sensitive one for detecting proprioceptive abnormalities than the WB test since the smaller amplitudes of movement used would probably be perceived more accurately (26).

Of significant concern in the present study was the question concerning the extent to which a deficit in proprioceptive coding might affect weightbearing function. Skinner et al. (14) observed that persons with advanced knee OA who had poor position sense walked more slowly than those with better judgement. This suggested that persons with reduced position sensitivity compensated with slower, safer locomotion. In the present study, however, there was no relationship between the proprioceptive deficit of the patients and their walking speed. This suggests that individuals with mild to moderate joint disease might receive sufficient compensatory input from receptors in muscle, tendon, skin or capsular receptors of other joints to maintain limb control when walking at relatively slow speeds. Unsteadiness during gait might also be more closely related to other criteria, such as, variability in AE or variable error, disease severity, muscle weakness or instability.

In summary, the present study has shown the following: a) cognitive perception of limb position as measured by the ability to reproduce knee angles in WB and NWB situations is significantly impaired in women with OA of the knee as compared to healthy subgroups; b) the impairment may be related, in part, to age and to peripheral and/or central proprioceptive factors other than vision and vestibular mechanisms which were normal, and therefore constant in all groups; c) the impairment does not necessarily affect the normal walking speed of persons with mild to moderate knee OA; d) knee position sense accuracy as recorded in the present study decreases with age.

Further study focusing on the specific factors which contribute to a decline in cognitive perception of limb position in knee OA should be undertaken. Additional research is also required to examine the validity and sensitivity of the present measurements. Further studies of persons with more advanced disease tested under different WB conditions will provide more information on whether abnormal gait in OA is produced in an effort to maximise proprioceptive input.

Although the present measurement error of the exprimental protocol used in the present study was small (Table IV) and there were no significant AE measurement differences within and across sessions ($p = .62$), test accuracy in future studies might be improved by using fixed criterion angles and by paying careful attention to head, body, arm and support limb position. Likewise, to prevent subjects from duplicating joint angles based on time, a metronome could ensure the limb is repositioned at a different speed (44). In addition, because subjects might be able to reproduce joint angle, but might not be able to hold the position for the photo, an alternate recording system such as video imaging might be more appropriate.

Acknowledgements: The study was supported by a grant from The Small Faculties Committee, The University of Alberta, Edmonton, Canada. The editorial assistance of Dr. J. Yang and the assistance of Mr. E. Rogers with the artwork is also gratefully acknowledged.

REFERENCES

1. Skoglund, S. Anatomical and physiological studies of knee joint innervation in the cat. Acta Physiol Scand 1956, 124 (Suppl), 1-101.
2. O'Connor, B.L., McConnaughey, J.S. The structure and innervation of cat knee menisci, and their relation to a "sensory hypothesis" of meniscal function. Am J Anat 1978, 153, 431-432.
3. Burgess, P.R., Clark, F.J. Characteristics of knee joint receptors in the cat. J Physiol (Lond) 1969, 203, 317-335.
4. Schultz, R.A., Miller, D.C., Kerr, C.S., Micheli, L. Mechanoreceptors in human cruciate ligaments. J Bone Joint Surg 1984, 66A, 1072-1076.
5. Mountecastle, V., Poggio, G., Werner, G. The relation of thalamic cell response to peripheral stimuli varied over an intense continuum. J Neurophysiol 1963, 26, 775-806.
6. Boyd, I.A., Roberts, T.D.M. Proprioceptive discharges from stretch-receptors in the knee joint of the cat. J Physiol (Lond) 1953, 122, 38-58.
7. Cohen, L.A. Activity of knee joint proprioceptors recorded from the posterior articular nerve. Yale J Biol Med 1955, 28, 225-232.
8. Freeman, M.A.R., Wyke, B. Articular contributions to limb muscle reflexes. Br J Surg 1966, 53, 61-69.
9. Threlkeld, J., Currier, D.P. Osteoarthritis. Effects on synovial joint tissues. Phys Ther 1988, 68, 364-370.
10. Wyke, B. Neurology of the cervical spinal joint. Phys Ther 1979, 65, 72-79.
11. Adams, J.A. Feedback theory of how joint receptors regulate the timing and positioning of a limb. Psychol Rev 1977, 84, 504-523.
12. Khodeddah, S. Quantitative approach to osteoarthritic gait assessment. N Engl J Med 1987, 16, 9-14.
13. Stauffer, R.N., Chao, E.Y.S., Gyory, R. Biomechanical gait analysis of the diseased knee joint. Clin Orthop Rel Res 1977, 126, 246-255.
14. Skinner, H.B., Barrack, R.L., Cook, S.D., Haddad, R.J. Jr. Joint position sense in total knee arthroplasty. Clin Orthop Rel Res 1984, 1, 276-283.
15. Kelso, J.A. Joint receptors do not provide a satisfactory basis for motor timing and positioning. Psychol Rev 1978, 85, 474-481.
16. Gandevia, S.C., McCloskey, D.I. Joint position sense, muscle sense and their combination as position sense measured at the distal interphalangeal joint of the middle finger. J Physiol (Lond) 1976, 260, 387-407.
17. Grigg, P., Finerman, G.A., Riley, L.K. Joint position sense after total hip replacement. J Bone Joint Surg 1973, 55A, 1016-1025.
18. Cross, M.J., McCloskey, D.I. Position sense following surgical removal of joints in man. Brain Res 1973, 55, 443-445.
19. Clark, F.J., Burgess, R.C., Chapin, J.W., Lipscomb, W.T. Role of intramuscular receptors in the awareness of limb position. J Neurophysiol 1985, 54, 1529-1540.
20. Glasberg, M.R., Glasberg, J.R., Jones, R.E. Muscle pathology in total knee replacement for severe osteoarthritis. A histochemical and morphometric study. Henry Ford Hosp Med J 1986, 34, 37-40.
21. Andriacchi, T.P., Galante, J.O., Ferrer, R.W. Influence of total knee replacement design on walking and stair climbing. J Bone Joint Surg 1982, 64A, 1328-1335.
22. Barrack, R.L., Skinner, H.B., Cook, S.D., Haddad, R.J. Jr. Effect of articular disease and total knee arthroplasty on knee position sense. J Neurophysiol 1983, 50, 684-687.
23. Paillard, J., Brouchon, M. A proprioceptive contribution to the spatial encoding of position cues for ballistic movements. Brain Res 1974, 71, 273-284.
24. Altman, R.D., Bloch, D.A., Bole, G.G. Jr., Brandt, K.D., Cooke, D.V., Grenwald, R.A., Hochberg, M.C., Howell, D.S., Kaplan, D., Koopman, W.J., et al. Development of clinical criteria for osteoarthritis. J Rheumatol 1987, 5, 3-6.
25. Harris, A.J. Harris Test of Lateral Dominance (3rd ed). New York, Psychology Corporation, 1974.
26. Glenross, D., Thornton, E. Position sense following joint injury. J Sports Med Phys Fitness 1981, 21, 23-27.
27. Barrack, R.L., Skinner, H.B., Brunet, M.E., Cook, S.D. Joint kinesthesia in the highly trained knee. J Sports Med Phys Fitness 1984, 24, 18-20.
28. Hulliger, M., Nordh, E., Vallbo, A.B. The absence of position response in spindle afferent units from human finger muscles during accurate position holding. J Physiol 1982, 322, 167-179.
29. Clark, F.J., Horch, K.W., Bach, S.M., Larson, G.F. Contributions of cutaneous and joint receptors to static knee-position sense in man. J Neurophysiol 1979, 42, 877-888.
30. Eklund, G. Position sense and the state of contraction; the effects of vibration. J Neurol Neurosurg Psychiatry 1972, 35, 606-611.
31. Rymer, W.Z., D'Almeida, A. Joint position sense: the effects of muscle contraction. Brain 1980, 103, 1-22.
32. Newton, R.A. Joint receptor contributions to reflexive and kinesthetic responses. Phys Ther 1982, 62, 22-29.
33. Miller, J. Joint afferent fibres responding to muscle stretch, vibration and contraction. Brain Res 1973, 63, 380-383.
34. Grigg, P. Response of joint afferent neurons in cat medial articular nerve to active and passive movements of the knee. Brain Res 1976, 118, 482-485.
35. McCall, Jr., W.D., Farias, M.C., Williams, W.J., BeMent, S.L. Static and dynamic responses of slowly adapting joint receptors. Brain Res 1974, 70, 221-243.
38. Colley, A., Colley, M. Reproduction of end-location and distance of movement in early and later blinded subjects. J Motor Behav 1981, 13, 102-109.
39. Nade, S., Newbold, P.J., Straface, S.F. The effects of direction and acceleration of movement of the knee joint of the dog on medial articular nerve discharge. J Physiol 1987, 387, 505-519.
40. Karanjia, P.N. Passive joint position sense after total hip replacement surgery. Ann Neurol 1983, 13, 654-657.
41. Marteniuk, R.G. An informational analysis of active kinesthesis as measured by the amplitude of movement. J Motor Behav 1971, 3, 69-77.
42. Neufeld, S.D. Reproducing movement in the lower extremity using kinesthetic cues of distance and location. Phys Ther 1981, 61, 1147-1151.
43. Winer, B.J. Statistical Principles in Experimental Design. 2nd ed. New York, McGraw-Hill, 1971.
44. Smith, R.L., Brunoli, J. Shoulder kinaesthesia after glenohumeral dislocation. Phys Ther 1989, 69, 106-112.
45. Collins, J.G., Roppolo, J.R. A comparison of human tactile stimulus velocity discrimination with the ability of S-I cortical neurons in awake rhesus monkeys to signal the same velocity differences before and after non-anesthetic doses of pentobarbital. Brain Res 1980, 198, 307-321.
46. Dietz, V. Human neuronal control of automatic functional movements: Interaction between central and afferent input. Physiol Rev 1992, 72, 33-69.
47. Barrett, D.S., Cobb, A.G., Bentley, G. Joint proprioception in normal, osteoarthritic and replaced knees. J Bone Joint Surg 1991, 73B, 53-56.
48. Corrigan, J.P., Cashman, W.F., Brady, M.P. Proprioception in the cruciate deficient knee. J Bone Joint Surg 1992, 72B, 247-250.
49. Matthews, P.B.C. Proprioceptors and their contribution to somatosensory mapping: complex messages require complex processing. Can J Physiol Pharmacol 1988, 66, 430-438.

Received: 14 November 1992; Revision-accepted: 22 September 1992
Correspondence to: R. MARKS, Clinical Associate.
The Department of Physiotherapy. St. Michael's Hospital, 30 Bond Street, Toronto, Ontario M5B 1W8, Canada.