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Joint Receptor Contributions to Reflexive and Kinesthetic Responses

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This article presents a review of the neuroanatomy and neurophysiology of joint receptors. The role of joint receptors in signaling position and movement has been studied since the early part of this century. Morphological descriptions and reflexive and kinesthetic contributions of articular receptors in the regulation of motor behavior have been identified in studies on anesthetized animals and to a lesser extent in studies on human subjects. Areas for future laboratory and clinical research include articular receptor input in motor learning and relearning tasks as well as standardization of methodology and responses for assessing joint position.

Key Words: *Articular receptors, Motor control, Kinesthesis.*

Since the 1950s, neurophysiological theories of the function of joint receptors have been important factors in the applications of therapeutic exercise. More recently, the importance of the role of joint position and the influence of joint receptor input on the CNS have been addressed in physical therapy orthopedic procedures.[1] Because of a continued and growing interest in joint mechanoreceptors, a review of the neuroanatomy and neurophysiology of this subject is timely.

MORPHOLOGY AND BEHAVIORAL CHARACTERISTICS

Detailed light and electron microscopic studies in the cat and, to a lesser extent, in humans have delineated four types of joint receptors located in and around joints (Table).[2-7] Types I, II, and III are regarded as "true" joint receptors; Type IV is considered a class of pain receptor. Generally, the four types are found in all diarthrodial joints and associated ligaments, and more recently have been identified in the meniscal horns of the knee joints of cats and dogs.[8] The density, location, and morphology of these receptors vary within joints of one or more species.[9-11] In this article, the figures delineating the morphology of these receptors are composite drawings. Unless specified otherwise, the research presented here has been conducted on the knee joint of the cat.

Type I receptors are found primarily in the stratum fibrosum of the joint capsule and on ligaments such as the posterior oblique ligament and medial and lateral collateral ligaments of the knee.[11] The density of their distribution tends to be in the more proximal joints of the extremities and in the cervical region of the vertebral column. More specifically, a high concentration of receptors is found on the anterior and posterior aspects of the capsule.[2, 3] Dee delineated a greater number of joint receptors in the inferior anteroposterior (AP) aspect of the hip capsule than in the superior AP aspect.[2]

The sensory unit for Type I joint receptors consists of a cluster of two to six receptor corpuscles and a myelinated parent axon, 8 to 12 μm in diameter.[3, 11] The corpuscles are about 300 μm wide and 300 to 800 μm long and are sometimes called Ruffini-like (Fig. 1). Ultrastructure studies by Halata have demonstrated a variety of afferent terminal endings in the laminated capsule including loops and disks.[11] Capillaries tend to be in close association with each cluster.

Type I mechanoreceptors subserve both static and dynamic physiologic functions. They have a resting discharge rate of 1 to 20 impulses per second and demonstrate an alteration in rate upon joint movement, muscle contraction, or altered joint pressure gradient. These receptors thus are able to provide constantly the CNS with information in both resting and active conditions. The resting discharge is believed to be caused by either muscle tension from those muscles crossing the joint or by the gradient between internal and external capsular pressure. Type I receptors have a low threshold for activation and are slow to adapt to changes altering their firing frequency.[6]

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TABLE

Summary Characteristics of Joint Receptors[a]

Type	Location	Receptor Appearance	Sensory Unit	Physiologic Function
I	Stratum fibrosum of capsule; ligaments Higher density in proximal joints	Laminated Ruffini-like corpuscle 300 μm wide 300–800 μm long	Myelinated parent and 2–6 corpuscles	Active at rest and during movement Low threshold for activation Slowly adapting
II	Junction of synovial and fibrosum of capsule; intraarticular and extraarticular fat pads Higher density in distal joints	Laminated, pacinian-like, conically shaped corpuscle 150–250 μm long 20–40 μm wide	Myelinated parent axon and 1–5 corpuscles	Active at onset and termination of movement Low threshold for activation Rapidly adapting
III	Collateral ligaments Not found in interspinous ligaments of cervical region	GTO-like corpuscle 800 μm long 100 μm wide	Myelinated parent axon and 1 corpuscle	Active at end of joint range High threshold for activation Slowly adapting
IV	Ligaments, capsule, and articular fat pads Absent in synovial tissue	Free nerve endings or lattice type endings	Thinly myelinated parent axon and terminal endings	Active only to extreme mechanical or chemical irritation High threshold for activation Slowly adapting

[a] Composite of sources: Freeman and Wyke,[3] Polacek,[9] and Halata.[11]

Type II joint receptors are located at the junction of the synovial membrane and fibrosum (layer) of the joint capsule and intraarticular and extraarticular fat pads.[7, 11] They are more numerous in distal joints (eg, wrist and ankle) than in proximal joints (eg, hip). They have also been identified in the temporomandibular joint (TMJ).[5] In contrast to Type I receptors, these receptors are more highly concentrated on the medial and lateral aspects of the joint capsule.[3]

The sensory unit for this class of receptor is composed of a 9 to 12 μm myelinated parent axon and either a single encapsulated receptor or a cluster of up to five receptors (Fig. 2). These multilaminated, conically shaped structures are about 150 to 250 μm in length and 20 to 40 μm in diameter and are sometimes called pacinian-like.[11] Ultrastructural studies by Halata have demonstrated that the sensory axon splits into unmyelinated processes of varying lengths that terminate as bulbous endings.[11] These endings lie inside a multilaminated sheath of modified Schwann's cells. Each sensory unit is situated near a blood supply.

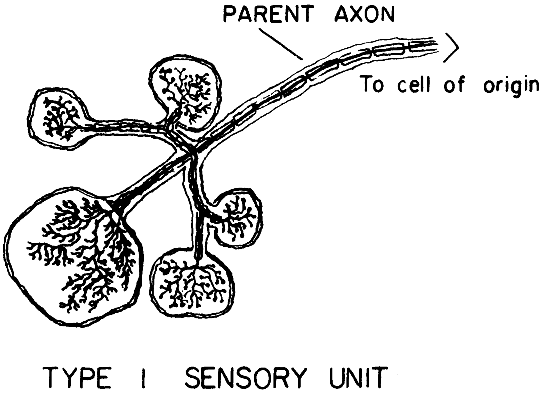

Fig. 1. Composite drawing of Type I receptor and parent axon.

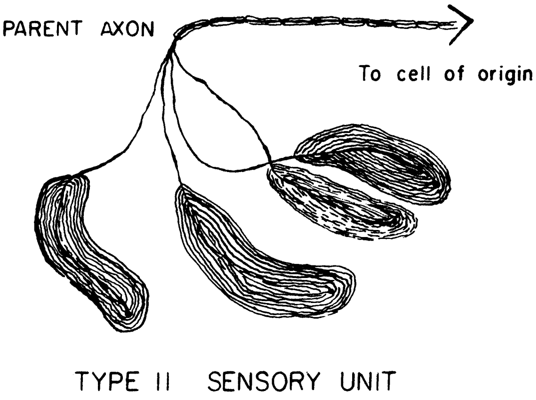

Fig. 2. Composite drawing of Type II receptor and parent axon.

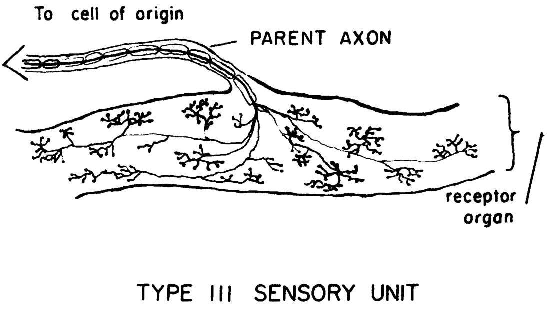

Fig. 3. Composite drawing of Type III receptor and parent axon.

Functionally, like Type I receptors, Type II receptors have a low threshold for activation. These dynamic receptors respond to joint movement by eliciting two bursts of action potentials in the parent axon, each lasting about one-half second and signaling the onset and termination of movement.[7] Type II receptors are thus termed rapidly adapting (RA). The arrangement of the laminated structure and terminal endings may enable them to serve as tension monitors like other encapsulated receptors. Deformation of the capsule produces tension on the layers of the capsule, which, in turn, generates action potentials in the sensory endings.[12]

Type III joint receptors have been found in collateral ligaments of the joints of the extremities, the TMJ, and facet joints of the thoracolumbar part of the spine, and in the longitudinal and interspinous ligaments of the vertebral column.[7] Type III receptors have not been found in the cervical region.[13] To date, electron microscopic studies by Halata indicate these receptors are absent in the knee joint of cats.[11] Morphologically similar to Golgi tendon organs, these are the largest of the articular receptors measuring 600 μm in length and 100 μm in width (Fig. 3). The sensory unit consists of a 13 to 17 μm myelinated parent axon serving one receptor.[7, 11] The sensory axon arborizes into multiple endings. Type III receptors do not exhibit a close relationship with a blood vessel.

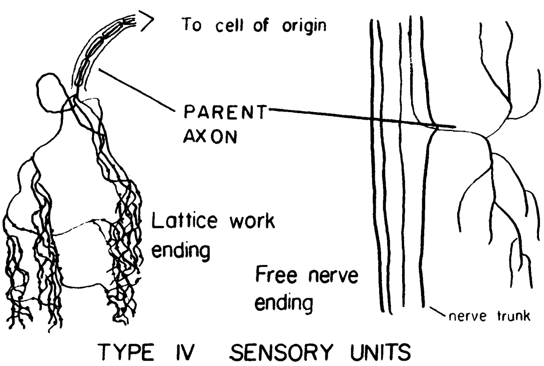

Fig. 4. Composite drawing of Type IV receptor and parent axon.

These dynamic receptors have a high threshold to stimulation and are slowly adapting.[8] Normally they are inactive in immobile joints and are responsive only in the extreme ranges of active or passive joint movement or to large amounts of longitudinal traction.[6]

Type IV receptors possess free nerve or lattice-type endings that have been found in joint ligaments, capsules, and fat pads.[3, 7] Like the other types of joint receptors, they are absent in synovial tissue and meniscal body.[10] The sensory unit of the pain receptor consists of a very thinly myelinated parent axon, 2 to 5 μm in diameter (Fig. 4). These receptors have a high threshold to activation and, like Types I and III, are slowly adapting (SA). They are not normally active, but respond to extreme mechanical deformation of the joint as well as to direct chemical or mechanical irritation.[14] Based on the findings that Type IV endings were stimulated by algesic substances and strong local mechanical stimulation to the joint capsule, Hong suggested they are polymodal nociceptors.[15] Furthermore, he considered them similar to nociceptors found within the skeletal muscle by Mense.[16]

Inasmuch as each of the four types of joint receptors is unique in location and density, hypotheses related to their function based on anatomical evidence have been fostered. Recent light microscopic studies have led to the "sensory hypotheses" for the meniscus.[8] Type I and II receptors were found in the anterior and posterior horns of the meniscus and in the meniscofemoral ligaments of the cat knee joint. No receptor endings were found in the meniscal body. O'Connor and McConnaughey postulated that movement of the knee joint stimulated various groups of receptors thereby signaling exact joint position. Because the knee joint is primarily a gliding joint that relies on ligaments and muscle action for stability, this sensory information from joint receptors in the meniscus could also contribute to the initiation of reflexive muscular responses to maintain the integrity of the knee joint.

Extensive histological studies of joint receptors were first conducted in 1857 by Rudigner and more recently by Polacek.[9] Research investigating the role of articular receptors in reflexive and motor activity has lagged because of the inability to attribute specific motor responses to activation of a particular class of receptors.

REFLEXIVE RESPONSES

Early methods to examine the reflexive responses of articular nerves in animals varied with respect to the type of animal preparation (decerebrate or spinal) as well as the stimulus characteristics. Gardner electrically stimulated (characteristics and preparation not given) the articular nerves in the hind limb and obtained a flexion response.[4, 17] If a stronger stimulus was applied, a crossed extension reflex accompanied the flexor withdrawal. He postulated that both large and small diameter afferent fibers contributed to activation of flexor muscles and inhibition of ipsilateral extensor muscles. Skoglund noted that electrical stimulation of the articular nerve produced a reciprocal relationship of facilitation and inhibition between flexor and extensor muscles, respectively.[6] The muscles facilitated varied with the type of animal preparation. On the other hand, a total muscular inhibition has been noted in the hip joint when the nerve to the ligamentum capitus femoris is electrically stimulated.[18]

Ekholm and colleagues observed flexor muscle facilitation and extensor muscle inhibition by increasing the intraarticular pressure of the cat knee joint. The same response was noted when the anterior region of the knee was probed or tension of the medial collateral ligament was increased.[19]

Distension of joints and subsequent muscle inhibition has also been noted in humans.[20, 21] Eyring and Murray examined the relationship between limb position and alterations of intraarticular pressure in both fresh cadavers and living subjects.[21] All joints studied were infiltrated with saline. Minimal intraarticular pressure was measured by manometer, when the joint was placed in a neutral to slightly flexed and relatively abducted position. For example, the position for the cadaver hip joint ranged from 30 to 65 degrees of flexion and 15 degrees of both abduction and external rotation. Similar limb positions were noted in normal infused joints. De Andrade and associates examined the relation of the knee joint infusion and quadriceps femoris muscle inhibition in three healthy subjects and 14 patients with Charcot-Marie-Tooth atrophy.[20] He noted that the position of slight knee joint flexion produced minimal pressure and pain. Electromyographic recordings showed a decrease in the amplitude of quadriceps femoris muscle activity during knee straightening from a starting position of 10 degrees of flexion. These findings demonstrate that muscle inhibition is reflexive and may be the mechanism that prevails after knee surgery and in joint disease.

Boyd and Roberts postulated that articular contribution to quiet standing originated in the ankle joint.[22] Freeman and Wyke examined reflex responses at the ankle joint.[7] Passive dorsiflexion of the tenotomized and skinned ankle of the cat produced an increased reflex response in the gastrocnemius muscle and a reciprocal inhibition in the anterior tibialis muscle. Similarly, when the ankle was positioned in plantar flexion, the anterior tibialis muscle was facilitated with a concomitant inhibition of the gastrocnemius muscle. Onset of the reflex responses occurred about one second after initiation of the movement, indicating a relatively long reflex latency. Burgess and Clark demonstrated that some joint receptors were active at both ends of the range.[23] These double-ended receptors, as well as those active at only one end of the range, could contribute to the facilitation of muscle activity during plantar flexion and dorsiflexion.

Freeman and Wyke also analyzed the effects of an ankle joint neurectomy on locomotor behavior and noted that the cat had moderate impairment in standing, locomotion, jumping, and bar walking.[7] When the cat stood, the operated limb assumed a flexed and externally rotated position, and when the cat walked, the limb was circumducted. From these results they postulated that the joint afferent fibers in the posterior region of the capsule contributed to both static and dynamic postural reflex activity. Lindstrom and Norrsell repeated these studies.[24] They noted that sectioning the posterior articular nerve alone or in combination with the medial articular nerve did not produce deficits in standing or locomotion as described by the earlier studies previously mentioned. Differences may be caused by the varied descriptions of locomotor behavior as well as by surgical techniques.

Lundberg and colleagues demonstrated that an electrical stimulation volley to the posterior nerve of the knee joint altered the reflexive responses of the Ib afferents of the Golgi tendon organs (GTOs).[25] By intracellular recordings in alpha motoneurons of the hind limb musculature, these researchers were able to detect alterations of the disynaptic and trisynaptic inhibitory and facilitory pathways originating at the GTOs. They postulated that this regulatory mechanism could be brought about by joint receptors, thus demonstrating their influence on locomotion. In addition, they confirmed that the posterior articular nerve is involved in facilitation of flexors with concurrent inhibition of extensors. Alteration of posture and locomotion would suggest that the gamma efferents as well as alpha motoneurons are influenced by joint receptor input through multisynaptic interneuronal connections.

Articular receptor influence upon gamma motoneurons was indirectly measured by recording impulses from triceps surae muscle spindle afferents.[26] Application of pressure to the contralateral knee joint capsule to mimic muscle contraction increased excitation of the afferents. Infusion of the capsule with local anesthetic abolished the response. Based on findings of activation of articular receptors by muscle contraction these authors also postulated that joint afferents may be involved in the coordination of bilateral activity such as locomotion.

RECEPTORS AS DETECTORS OF JOINT ANGLE

The influence of articular receptors on the detection of joint position has been noted since the early 1900s. Evidence that joint afferent fibers could be involved in the perception of position as well as in higher center regulation of locomotion is found in the presence of ascending projections from knee joint afferents to both the cortex and cerebellum.[27, 28] In addition, cells located in the nucleus gracilis, cuneate nucleus, and somatosensory thalamus have been shown to respond to joint angles by altering their discharge rate.[11, 19, 29, 30]

Research on the identification of activation angles of articular receptors and the function of these receptors in movement has and is currently being conducted. Electrophysiological studies classify afferent fibers and associated articular receptors according to the discharge rate and adaptation of fiber action potentials as well as to direction of joint movement. Boyd and Roberts considered studying SA receptors for precise information concerning joint angles.[22] These SA receptors were similar to Wyke's Type I Ruffini-like and Type III GTO-like receptors. Andrew considered that the function of the GTO-like receptor was to measure tension of the ligament.[6] The SA receptors were best adapted to measure static joint position and were capable of detecting direction of motion.[6]

The RA receptors were considered phasic receptors corresponding to the pacinian-like or Type II receptors of Wyke.[7] The RA receptors produce a short burst of action potentials during movement, and are thereby capable of signaling motion. Using 278 single dorsal root fiber recordings of the posterior articular nerve of the knee joint of the cat, Burgess and Clark also categorized afferent fibers as SA or RA.[23] An SA flexion-extension afferent fiber was identified as a fiber that responded with a quick burst of up to 250 impulses per second when a specific angle of activation was reached. A steady state followed with a frequency rate between 10 and 150 impulses per second. The RA phasic fibers responded to joint movement with a firing burst and were capable of a resting firing rate of less than 10 impulses per second.

These researchers further divided SA receptors into three groups according to direction of movement: those that monitored 1) flexion, 2) extension, or 3) both flexion and extension. Of the 278 fiber recordings, 140 (50.4%) responded to 10 to 15 degrees of movement before the end range of flexion and extension; 47 (15.9%) responded only to flexion, and 12 (4.3%) only to extension. Of the remaining total recordings, 44 (15.8%) were RA and responded to joint movement in any direction. This lack of directionality was confirmed by afferent recordings of the medial articular nerve of the cat knee joint.[31] Most of the afferent fibers responded at the extremes of knee joint range either in flexion-extension or adduction-abduction. A forceful flexion was needed to activate the receptors; however, they responded dramatically to gentle external pressure. Burgess and Clark proposed that these receptors contribute to deep pressure sensation rather than to the detection of joint position.

Carli and colleagues examined SA afferent fiber discharges in the posterior articular nerve of the hip joint during a variety of movements.[32] They noted an optimal discharge frequency at extremes of the range. Most neurons were activated by a combination of internal rotation with adduction or external rotation with abduction. They concluded that these receptors were not absolute detectors of position.

Millar confirmed this finding by noting an apparent hysteresis loop in receptor firing between flexion and extension movements.[33] If the elbow was extended in 5 to 10 degree increments from neutral, the afferent fiber would display a phasic burst then maintain a steady firing frequency. If the elbow was flexed in the same increments through the same range, the fiber would first cease firing, then return to a resting discharge level. This resting level was notably lower than when the angle was produced in extension. Although the author did not discuss the significance of such a finding, one could postulate that the difference in firing frequency could be a mechanism for determining direction of limb movement. McCall and associates examined this apparent hysteresis loop and emphasized the need to examine the effects of differences in receptor firing frequencies at higher levels of sensory integration in the unanesthetized animal.[34]

The absence or small number of fibers activated in intermediate joint ranges has been confirmed in the knee joint and has been observed in the elbow, wrist, and costovertebral joints of the cat and monkey.[33, 35–37] Those afferent fibers discharging in the intermediate ranges, and not discharging with the knee joint in full flexion or extension, were postulated to be muscle spindle fibers originating from the popliteus muscle and not from other muscles such as the soleus or gastrocnemius.[31, 38] Ferrell performed similar studies on the knee joint and noted that removal of the popliteus muscle did not eliminate the intermediate range discharge.[39] This fact, however, did not rule out the possibility that spindle fibers from other muscles contributed to the midrange detection. Because muscle spindle pathways to the cerebral cortex have been identified, the fibers could contribute information for the detection of joint position.[17]

A mechanism for stimulating joint receptors in the midrange may originate with the capsular tension that results from stretch or contraction of muscles crossing the joint. Grigg noted that a tetanic electrical stimulus to the gastrocnemius muscle of the cat was sufficient to excite 16 of 24 extensor axons of the posterior articular nerve, providing the knee was positioned within five degrees of the activation angle for that afferent fiber.[40] With intact nerve root recordings, discharges were noted in response to electrical stimulation in the intermediate range. In a similar study using monkeys, Grigg and Greenspan noted that electrical stimulation to the gastrocnemius and quadriceps muscles also produced afferent fiber activation in the posterior articular nerve, as evidenced in the dorsal root recordings.[36] Additionally, a heavy load (mean of 2450 gmcm) was needed to stretch the muscle enough to activate the articular receptors.

One could postulate that contraction of muscles crossing the joint would activate certain joint receptors and that those receptors on the nonstressed side would not be activated. In this case, because of the pattern of muscle activation, joint position as well as direction and velocity of the movement would be indicated.

In summary, Type I Ruffini-like receptors could detect speed and direction of movement. Type II pacinian-like receptors could detect small movement as well as accelerating movements (because of their RA characteristic). Finally, the Type III GTO-like receptors could detect position and direction of movement. Probable functions of the receptors are based on test results obtained from studies on anesthetized animals. Articular receptor functions of natural movements have not yet been studied in detail.

In order to examine receptor functions in locomotion and movement in general, research needs to be conducted on neurologically intact, unanesthetized, and unrestrained animals. Loeb and associates have made progress toward solving this problem in one area by developing a technique to stabilize recording electrodes in dorsal roots of unrestrained cats.[41]

A second area that needs to be addressed is the formulation of criteria for identification of joint receptor input at the cortical level. Lemon and Porter recorded electrical activity of precentral cortical neurons while a monkey performed a motor task.[42] These researchers were unable to differentiate precisely between muscle, joint, and ligamentous receptor input.

KINESTHESIA

Joint and muscle afferent contributions to kinesthesia have been conducted in humans under a variety of conditions including joint anesthesia, infiltration, joint replacement, passive movement, and limb-matching studies. Kinesthesia may be defined as the ability to discriminate joint position; relative weight of body parts; and joint movement including direction, amplitude, and speed. In 1889, Goldschneider determined that small, passive movements, in the range of 0.5 to 0.7 degrees, were detected if the speed of movement was 1.0 to 2.0°/sec.[43] Active joint movement was detected at angles of less than 5.0 degrees.

In a classical work, Browne and colleagues postulated that there are two sensory mechanisms; one for detecting passive movement and one for detecting active movements.[44] The average detection angle for passive movements of the metatarsophalangeal joint of the great toe was 4.4 degrees at speeds of 1.0 and 2.0°/sec. Of 82 subjects, 10 (12%) detected movement sensation at angles of 15 degrees or more. Active movement sensation in these 10 subjects was not impaired. A second series involved anesthetizing (8–12 ml procaine hydrochloride, 1% solution) the joint capsule and pericapsular structures. Eight of the nine subjects tested lost both joint position sense and movement sensation. When the muscles were tense, however, the subjects could detect movement. Browne postulated that passive movement and position sense were detected by changes in tension on the joint capsule, and that active movement was detected by muscle afferents as well as by joint afferents (the latter because muscle contraction deformed the joint capsule).

Provins examined the metacarpophalangeal joint of the index finger under conditions similar to Browne's.[45] He noted that anesthesia (15 ml lidocaine hydrochloride, 0.9% solution) impaired the "appreciation" of both active and passive movement and that tensing muscles did not increase awareness of joint movement. When both skin and capsule were anesthetized, kinesthesia was eliminated. Passive movements of 40 degrees were not detected by five subjects. He postulated that movement was detected by deformation of those nonanesthetized structures such as skin. Gelfan and Carter also failed to produce joint or muscle sensations when exposed tendons were pulled.[17 (p 130)]

Contrary to the above findings, Goodwin and colleagues support the idea of muscle spindle input contributing to kinesthesia.[43] With an anesthetic applied proximal to the interphalangeal joint, they noted that subjects could detect 10 to 20 degree movements at velocities of 5 to 10°/sec. Detecting movements at a speed of below 1°/sec was difficult if not impossible, however, even when the muscles surrounding the joint were tense. Gandevia and McCloskey also concluded that both joint and cutaneous input subserved joint position sense and that muscle receptor input would further enhance position sense.[46]

Vallbo, while recording finger flexor spindle afferents in the median nerve, noted an increased firing frequency when the metacarpophalangeal joint was passively moved.[47] Although evidence showed a low sensitivity (0.18 impulses per second per degree as compared to 1.0 impulses per second per degree for cat ankle extensors) for perception of joint position, these receptors could contribute to kinesthesia.

Studies on perception of static joint position have also been conducted. Horch and colleagues examined the accuracy of replication of knee joint angle several minutes after passive placement of the limb.[48] Earlier studies examined accuracy of matching shoulder joint position, but responses were recorded several seconds after movement ceased. Horch noted that subjects were aware of knee joint angle changes when movement velocity was less than 1 degree per minute. In addition, subjects were able to match the passively placed joint position with the other leg even after a three-minute wait. Other sensory cues as well as "kinesthetic" memory could play a role in limb position matching. In a subsequent study, Clark and colleagues noted that subjects could match knee joint position after a 5 degree change with 85 percent accuracy using the opposite leg.[49] Joint or skin anesthesia did not alter this ability to detect joint position. Thus, movement detection does not depend entirely on cutaneous or joint input but also relies on muscle afferents and memory.

Cross and McCloskey[50] and Grigg and colleagues[51] have examined joint position sense after surgical removal of the metacarpal heads and the hip joint, respectively. In both studies, a total capsulectomy was performed. Cross and McCloskey noted that all six patients could detect 10 degree movements at a velocity of 3°/sec or greater. The joint was gently grasped and moved by the experimenter, which added cutaneous input. In the hip, the ability to detect passive movements of greater than 5 degrees was not eliminated by the surgical procedures; however, a tendency to overestimate abduction angles on the affected side was noted. The subjects were able to produce equal abduction angles in their hips.

Accurate monitoring of joint position is needed in all aspects of motor behavior. Adams, in his closed-loop theory of motor learning, postulated the need for joint receptor input for control of both timing and direction of limb movements.[52] This peripheral control theory holds that joint receptor input is a prerequisite to the sequencing of movements; input is processed before the next phase of movement occurs. Use of receptor information in this way requires time and may not be efficient in skill development.[53]

FUTURE STUDY

Four areas of study need to be expanded. First, additional quantitative studies on joint detection have to be completed. In several studies already completed, the authors also served as subjects.[46, 48] In future

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studies, techniques such as Luce's choice theory that uses multiple trials and a mathematical model should be used to eliminate subject bias. Choice theory was applied in a study of joint angle detection ability.[54]

A second area of study is the standardization of the test response. A yes-no response for detection of movement, as well as descriptions of the movement, have both been used. A yes-no response is a low perceptual level response, whereas a description requires more complex integrative processes including memory. The complexity of clinical tests of "sensation" to assess kinesthesia is often the source of inaccurate and misleading information.

A third consideration is to determine whether active positioning or passive movement is a better test for judging joint position.[55] Finally, the influence of articular receptor responses on the development of skills or relearning of movements needs to be investigated.

SUMMARY

Descriptions of the morphological, behavioral, and functional characteristics of joint receptors have been presented. Research delineating articular receptor function has been conducted on animals with a variety of preparations and in both healthy subjects and a variety of patients. The complexity of receptor function under static and dynamic conditions is evidenced by these receptors' influence on GTO reflexive responses, gamma motoneurons, and higher centers. A variety of stimuli including direct mechanical stimulation of the capsule, alteration of capsular pressure, muscle contraction, and passive limb movement have been used in these studies. One or all of these stimuli may contribute to receptor activation in the neurologically intact, unanesthetized, and unrestrained animal.

Cutaneous and muscle receptors contribute to detection of joint position and movement. Clinical tests and techniques using joint movement also use the integration of input from a variety of receptors. The complexity of the nervous system precludes attributing motor responses to a single class of sensory receptors. The need for defining terminology and levels of patient responses and for standardizing methodology in laboratory research as well as in clinical testing and research has been presented.

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