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Letters

Position sense in a damaged knee

Sir; Until recently, it was generally thought that position sense, a routine and essential part of the clinical examination of the nervous system, was mediated by joint receptors. However, experiments in man[1 2] have suggested that the sense of position at the elbow and at the finger joints is mediated by muscle receptors particularly the primary and secondary muscle spindle afferents and perhaps also by the Golgi tendon organs. A role for joint receptors in joint position sense is thus now often doubted.

Following open surgery to my left knee I have experienced abnormalities of the sense of position and movement at this joint during standing and walking, both in the dark and light, which indicate the complexity of these sensations in motor control, and which suggest that these complex sensory experiences are derived at least in part, from joint receptors.

After a torsional injury an audible click on passive and active movement developed in the left knee. During the next few weeks joint pain persisted and an intra-articular effusion developed. Activity was restricted by these symptoms, and by brief locking of the joint during running. Six months later, after an arthrogram, the left knee was explored through a 4 cm crescentic lateral incision. The lateral cartilage, which was torn and separated from its anterior attachment, was partially removed. No abnormality of the medial cartilage or of the cruciate ligaments was noted. After 2 weeks immobilisation in extension in a plaster cast active rehabilitation was pursued and after a period of several weeks of persistent joint effusion, reduced range of movement and mild quadriceps weakness, physical activity was resumed, However, several unexpected abnormalities have persisted in the four years since the operation.

From the time, a few months after the operation, when mobility became relatively unrestricted, and near-full flexion of the knee became possible, an abnormality of sensation, apparently in the joint itself, became evident. This was, and remains, most evident when walking in darkness, for example in a bedroom at night. The previously normal sense of placement of the left leg on the floor is altered so that unexpected displacements occur leading to near-falls. Minor obstructions, for example a dressing gown left lying on the floor, cause particular difficulty. If the left foot is placed on such an object, which is soft and compressible under body weight, it is difficult to prevent falling. There is insecurity of the sensation of the position and rate of change of position of the knee and thigh during the small displacement of the soft object, so that inaccurate, unconscious, corrective movements of the leg are made. No such difficulties are experienced with the right leg.

Walking on a level surface in the dark is more difficult than it was before knee surgery. Ther is a subjective sensation that errors of control of force and displacement at the knee occur, leading to instabiity of gait, or even ataxia. In daylight unexpectedly encountered irregularities in contour of the ground lead to uncompensated displacements of the left leg which, on the right, would be readily and unconsciously compensated. Walking is thus a more conscious effort that it used to be. Running is similarly affected on rough ground. Walking across the inclined slope of a hillside is difficult to manage with the left leg, especially if the angle of the slope varies from step to step. Balancing on the ball of the left foot, as when spinning through 45° or 90° on one foot is poorly controlled on the left but easily managed on the right. Running downstairs is similarly less well performed because of the altered sense of position and movement in the left knee.

There is no formal abnormality of position sense and sense of movement at the knee joint or in other joints, or of vibration sense, light touch, temperature and pinprick in either leg. The tendon reflexes and plantar resxponses are normal. Strength, estimated by hopping on either leg, is now equal in both quadriceps, although the left quadriceps is 2 cm smaller in circumference than the right. The knee joint itself is stable. There is an oval 2 × 3 cm area of distorted sensation in the region of the crescentic incision on the lateral side of the left patello-femoral joint. Sensory and motor nerve conduction velocities in the left lateral popliteal nerve are normal.

In the usual clinical test for *position sense* the patient is asked to indicate, either verbally or by analogy, the static position of a limb or digit following small, applied displacements about a particular joint. In a variation of this test the patient may be asked to indicate the moment of perception of the passively-applied movement itself; this represents a form of movement sensation. Position sense thus differs from *proprioception,* a term which is properly applied to the sense of awareness of a limb in space. However, the term proprioception is also commonly used to indicate sensory input utilised in orienting reflexes, and in the control of voluntary movement, not necessarily consciously perceived. In clinical practice this distinction is not useful since unconscious sensory input cannot be assessed.

Kinaesthesis, the sensation of movement of a limb (or joint) is a sensation derived from sensory input during active movement of a limb. Sherrington[4] concluded that this probably depended on the integration of sensory input from muscle and tendon receptors, from joint receptors and perhaps also from skin receptors adjacent to the joint or muscle. Matthews[5] has reviewed the evidence for this view, and suggested that the primary spindle endings contribute to movement information, the spindle secondaries to positional information (referred to the relevant joint and not to the muscle itself), and the tendon organs to the perception of force exerted. However, there is clinical evidence that Matthews' suggestion cannot represent a complete explanation of these sensations. For example, in motor neuron disease no abnormality of these sensations has been described but in this disease the infrafusal muscle fibres lose both their gamma and alpha motor nerve supply,[6] leaving sensory input from the unaffected primary and secondary sensory endings unmodulated by the CNS. In this situation one might expect there to be an abnormality of any perceived sensation derived from these receptors but none has been recognised.

The *sense of effort* itself is probably related to corollary discharges in the motor system and does not depend on afferent input from sensory receptors.[3] Moberg[7] has presented an argument, based on clinical experience from hand surgery, that the sensations of position and kinaesthesis are derived predominantly from cutaneous afferents rather than from joint or muscle receptors.

The abnormality of sense of movement and of motor control I have experienced after partial lateral meniscectomy is referred to the damaged knee joint, and is heightened by increasing levels of difficulty in the motor task, for example walking on an unexpectedly uneven surface, especially in darkness, or walking on an inclined slope. The sensory disturbance is thus an abnormality of movement sensation, static position sense being normal when tested formally in the knee and in the other joints of the left leg. There is no evidence of

neuromuscular disorder or of central nervous system lesion. A disturbance of corollary discharge or of efference copy can thus be ruled out. Since no abnormality of sensation of position or of movement was evident before operation, the abnormality seems directly attributable to the lateral meniscectomy. The small area of altered cutaneous sensation in the region of the scar seems unlikely to be relevant since it is so slight. McCloskey *et al*[2] have discussed the possible errors in Moberg's interpretation that the relatively extensive cutaneous sensory disturbance noted in his patients was important in their altered position sense.

In the cat there is evidence that both muscle receptors and joint afferents are necessary to achieve entrainment, that is correct phase relationships, between limb movement and muscle activity in locomotion.[8] This results from neuronal activity at various levels in the nervous system, probably including cord and cortex, at which convergence of input from joint, skin and muscle receptors occurs.[9 10]

There seem to be two possible explanations for my experience. Firstly, it can be suggested that joint receptors play a role in movement sensation and that this sensation is more complex than static position sense itself. The latter is perhaps only part of the substrate of the former. Movement sensation may thus depend on input from several classes of receptors of which joint receptors are only one component. The abnormality of motor control I have experienced might thus result from disturbance of sensory function. Alternatively, this disturbance of motor control might result from inappropriate reflex corrections occurring after limb displacement. On this view the perceived abnormality would represent out of phase movements causing inappropriate displacement of the limb.

Persistent minor degrees of impairment of function are widely recognised after knee surgery of various types but no systematic evaluation of them seems to have been made. A study of the effect of total knee arthroplasty revealed no change in joint position sense, although abnormalities of this sensation were noted in control knees damaged by degenerative joint disease,[11] an observation that might indicate that sensory abnormality resulted from mismatch between disturbed sensory input and motor output in the damaged knees, an abnormality that was removed when this joint was surgically excised and replaced with a prosthesis. It may thus be of particular interest to investigate joint sense and kinaesthesis before and after joint surgery in previously healthy young people, as well as in an older population in whom surgery has been performed for long standing degenerative joint disease.

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Transient paraparesis—a manifestation of ischaemic episodes in the anterior cerebral artery territory

Sir; Transient paraparesis or sudden unexpected falling due to leg weakness without loss of consciousness ("drop attack") occurs in about 15% of patients with vertebro-basilar insufficiency.[1] It is usually associated with symptoms due to brainstem or occipital ischaemia and rarely occurs as an isolated event. Ischaemic episodes in the anterior cerebral territory due to emboli from the internal carotid artery are uncommon, but might give rise to transient paraparesis if multiple emboli affected both anterior cerebral arteries simultaneously. We describe a patient presenting with this symptom in whom angiography showed severe right internal carotid stenosis, both anterior cerebral arteries being supplied from the right internal carotid.

A 49-year-old man was admitted for investigation of episodes of transient neurological dysfunction which began six months previously. These were described as follows: (1) Two episodes of sudden weakness of the right lower limb lasting about ten minutes with complete recovery. (2) Four episodes of sudden weakness of both lower limbs causing him to fall. Strength gradually returned over a few minutes and he was able to walk normally again. In one of the attacks, the patient became very "dizzy" and lost consciousness for some minutes. (3) Ten episodes of "weakness" and numbness in the left hand lasting up to one hour; several occasions these were associated with numbness in the upper and lower lips on the left.

Relevant past history was a blind right eye since birth from congenital cataract. He smoked 56 g of tobacco each week. On examination he was jovial and his behaviour rather disinhibited. He was fully orientated and higher mental functions were normal on simple testing. Cranial nerves were normal; however, the right fundus could not be seen because of severe cataract. In the motor system there was a mild spasticity with some drifting of the outstretched left arm. There was mild weakness of left wrist extension, finger extension and finger abduction; plantar responses were both flexor. There was some impairment of joint position sense, stereognosis and two point discrimination in the left hand. In the cardiovascular system, the radial pulse was 68 per minute and regular. Blood pressure was 165/95 mm Hg and equal in both arms. There were no