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# NEURAL MECHANISMS OF KINESTHESIA

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Our primary interest is in where the signals for position and movement of the limbs originate and how the information is encoded. Humans can independently sense the positions and movements of their limbs in the absence of vision, and these senses are derived from mechanoreceptors located in the limbs. Receptors in muscles, skin and joints are potential sources of position and movement signals, but how (and whether) each of these groups contributes to kinesthesia remains uncertain. Recent evidence indicates that a sense of static-position derives from length receptors in muscle, whereas receptors in the skin, and possible though not likely the joints, can signal movement of a limb but not its static position.

We distinguish this static-position sense from a movement sense on the basis of whether a subject's ability to sense a small displacement (3 to 5 deg) of a joint varies with the rate of joint displacement. Therefore, if subjects possess an awareness of the static-position of a joint, slow rates of rotation should not seriously degrade their ability to sense displacements. In the absence of a static-position sense, subjects would rely on movement signals that do depend on the rate of rotation and their ability to detect displacements should decrement with decreasing rate. This is roughly analogous to distinguishing a low-pass filter (a static-position sense) from a high-pass filter ) a movement sense only) by their frequency response characteristics.

The interphalangeal (IP) joints of the fingers (and probably the toes) appear to lack a static-position sense. Subjects were unable to detect large displacements of the IP joints if the displacements were slow enough (our tests used rates less than 1 deg/min). In contrast, small displacements of the metacarpophalangeal (MCP) joint of the finger could be detected at the slowest rates we used. This is a surprising finding given that people seem quite unaware they have this kinesthetic deficit at the fingers.

At present, we are examining the consequences of not having a static-position sense at the fingers apart from an inability to detect very slow displacements. Using a more demanding test, we have found no substantial difference in the accuracy of reproducing target positions passively imposed on the MCP joint and the proximal interphalangeal (PIP) joint of the index finger (the MCP has and the PIP lacks a static-position sense). Our working hypothesis is that subjects normally use movement sense mechanisms to detect displacements, probably because they are faster, but how (or whether) subjects manage to obtain an absolute position reference with only a movement sense available remains unknown and is an issue under investigation.

We have also shown that subjects can reproduce target positions of a joint from memory about as accurately as they can match the position of the joint to its corresponding joint on the opposite limb. Reproducing targets from memory remains remarkably accurate even after 24 hours. The existence of a stable, long-term memory for limb position is an important component of a model for motor

control we are investigating. Irrespective of the mechanism, the accuracy of this memory complicates the study of position and movement senses. For example, one cannot study a static-position sense by moving a joint to some position and waiting until all movement signals have faded away. With a memory, one could not distinguish a true static-position mechanism from a mechanism that derived position from a velocity signal and stored the result in a memory.