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Special Issue: Wrist

Rehabilitation strategies for wrist sensorimotor control impairment: From theory to practice

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ABSTRACT

This clinical review discusses the organization, neuroanatomy, assessment, clinical relevance, and rehabilitation of sensorimotor (SM) control impairment after wrist trauma. The wrist SM control system encompasses complex SM pathways that control normal wrist active range of motion and mediate wrist joint neuromuscular stability for maintaining joint function. Among various known assessment methods of wrist SM control impairment, the active wrist joint position sense test is determined to be a clinically meaningful and responsive measure for wrist SM control impairment after wrist fracture. Wrist trauma may involve significant soft tissue injury (ie, skin, ligament, muscle), which could disrupt the generation and transmission of adequate proprioceptive input from wrist mechanoreceptors, thus leading to significant joint SM impairment. Various clinical examples of wrist trauma (eg, distal radius fracture, scapholunate joint injury) along with known prognostic factors (eg, pain) that may influence wrist SM control impairment recovery are discussed to illustrate this point. This article proposes promising rehabilitation strategies toward restoring wrist joint conscious and unconscious SM control impairments, integrating current research evidence with clinical practice. These strategies require more rigorous evaluation in clinical trials.

Level of evidence: 5.

Introduction

The wrist sensorimotor (SM) control system encompasses 3 complex physiological processes: (1) sensory feedback input, (2) central processing, and (3) motor feedforward output. Sensory feedback information is imported from peripheral (ie, cutaneous, joint, and musculoskeletal) neuroreceptors.[1] Afferent input is then centrally integrated, resulting in efferent neuromuscular responses toward joint stability and motor control[2] during functional activity (Fig. 1). Proprioception is one constituent of a complex SM control process. Proprioception requires the reception and central integration of incoming afferent signals. Afferent input is conveyed to upper central nervous system (CNS) centers (ie, cerebral cortex and cerebellum) via ascending spinal cord and brain stem pathways for central integration and interpretation.[3,4] This proprioceptive input is centrally integrated and processed to provide bodily awareness of all joint motions and positions. Central processing of sensory input elicits descending supraspinal pathways that exert constant feedforward efferent influences at the spinal cord, providing automatic neuromuscular joint control for various functional activity demands or conditions.[5] Trauma may lead to significant soft tissue injury (ie, skin, ligament, muscle), which could disrupt the generation and transmission of adequate proprioceptive input from wrist mechanoreceptors, thus leading to significant joint SM impairment. Pain arising from trauma may lead to neuroplastic changes in CNS centers,[6-9] ultimately altering the processing of incoming proprioceptive sensory information and leading to joint SM control deficit.[10-12]

Limited research exists on the prevalence, assessment, and treatment of SM impairment after wrist trauma. Wrist SM control deficit has been identified during the initial 3-month period after distal radius fractures (DRFs) and has been linked to clinically significant functional deficits.[13] Various sensory (ie, sensibility and proprioception) and motor (ie, muscle recruitment, force production, and endurance) deficits have been correlated with significant functional impairment after wrist trauma. Among them, diminished active wrist joint position sense (JPS) has been determined to be the most clinically meaningful indicator related to functional loss.[13]

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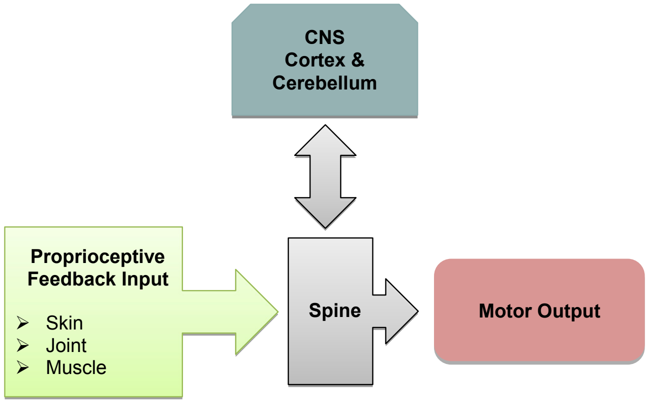

Fig. 1. Sensorimotor control system integration of afferent peripheral input toward motor output.

Emerging scientific evidence has encouraged stronger clinician awareness regarding the role of SM control system for maintaining wrist joint control during upper extremity function. Hand therapists should recognize known patient-specific prognostic factors that may influence wrist SM control impairment and recovery, which may lead to greater functional impairment. Clinical knowledge regarding conscious and unconscious wrist SM control deficits is essential for developing efficacious rehabilitation paradigms with an ultimate goal to reinstate optimal function after wrist trauma. The primary aims of this review are to discuss the organization, neuroanatomy, clinical relevance, and rehabilitation of SM control impairment after wrist trauma, thus bridging theory to clinical practice.

SM system organization

Historically, the SM control system has been a focus of inquiry for neuroscientists since the 19th century,[1] and knowledge about its organization and function has been evolving and not yet fully defined. Its theoretical framework was first introduced by Von Helmholtz in 1867[14] and Bastian in1888[15] who coined the existence of proprioceptive senses. Research findings of Sherrington in 1900[16,17] and Goodwin et al in 1972[18] helped to advance knowledge regarding the physiological basis of human SM control system to its current levels.

The human body SM control system is organized in 2 distinct physiological senses: unconscious and conscious (Fig. 2).[2,19] The unconscious sense represents the body's involuntary function to maintain dynamic joint stability and equilibrium during human kinesis.[19-21] This sense depends on afferent input mainly from joint and muscle receptors[19,22,23] and is linked to the feedforward anticipatory muscle control system that is heavily regulated by the cerebellum.[24] Furthermore, the conscious sense embodies the body's willful recognition of joint motion and position.[19] This sense depends mainly on cutaneous and muscle receptor sensory input, which ascends to the somatosensory cortex to produce the conscious senses of kinesthesia and joint position.[1,2,19]

Kinesthesia is the human perception of joint motion.[1,10] It is mediated by muscle spindle signals[25,26] throughout the joint range of motion (ROM) and cutaneous receptor input mostly toward the end range of limb physiological movement.[1,2] At the wrist, primary kinesthetic input is derived from skin receptors over the joint and muscle receptors adjacent to the joint.[19] Wrist mobility depends on extrinsic muscles located at the forearm.[27] JPS is the human perception of precise joint angle or position.[1,19] It is mediated by combined muscle spindle proprioceptive afferent information and

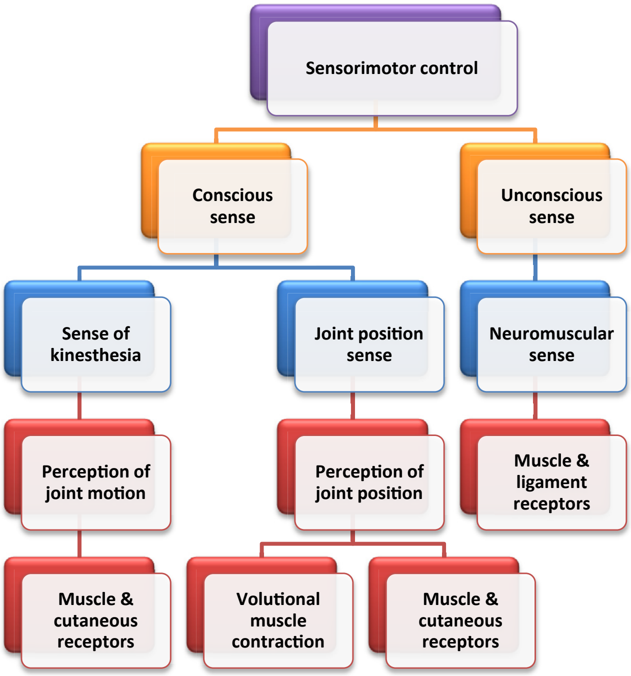

Fig. 2. Sensorimotor control system organization of conscious and unconscious senses and their responsible peripheral mechanoreceptors.

volitional muscle contraction,[28,29] which elicits an additional afferent signal toward joint position recognition.[28] Muscle contraction can be adversely influenced by muscle deconditioning or fatigue.[30] Significant JPS errors of 14.4° and 11.6° in active elbow flexion and extension motions, respectively, have been reported as a result of muscle fatigue.[31] Cutaneous receptors also influence JPS perception mostly at the end range of joint motion.[2]

Neuroanatomy of wrist SM system

The physiological function of the SM control system is to convey and centrally interpret peripheral proprioceptive information toward eliciting automatic feedforward efferent responses for optimal joint dynamic control. This complex neurophysiological process requires precise contribution from various peripheral and CNS structures. These structures consist of peripheral mechanoreceptors, proprioceptive ascending pathways, supraspinal processing centers, descending motor control pathways, and spinal cord reflex mechanisms.

Peripheral mechanoreceptors

Mechanoreceptors are specialized sensory nerve endings that sense and convey afferent signals from peripheral body regions. They are imbedded within static (ie, skin, joint capsule, ligaments) and dynamic (ie, musculotendinous) tissues[32] and respond to various types of mechanical deformation applied to their host membrane. Exteroceptive mechanoreceptors respond exclusively to cutaneous tactile and vibration stimuli.[27] Deep proprioceptive mechanoreceptors convey sensations from static and dynamic tensile tissue changes within articular and musculotendinous tissues.[25] On mechanical stimulation, they transmit specialized neural signals proportional to the degree of their deformation.[26] These signals travel through peripheral nerve fibers to the spinal cord and higher CNS centers for specialized processing and interpretation.[3,4]