# WHICH TYPE OF REPETITIVE MUSCLE CONTRACTIONS INDUCE A GREATER ACUTE IMPAIRMENT OF POSITION SENSE?

SYLVIE FORTIER

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# WHICH TYPE OF REPETITIVE MUSCLE CONTRACTIONS INDUCE A GREATER ACUTE IMPAIRMENT OF POSITION SENSE?

BY

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A THESIS SUBMITTED TO THE SCHOOL OF GRADUATE STUDIES
IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF
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**ABSTRACT**

The objective of this study was to determine which type of repetitive muscle contractions induces a greater acute impairment of elbow position sense. Eleven male subjects participating in the study underwent (i) a fatigue task (FT) consisting of 9 sets of 10 voluntary isometric, concentric, or eccentric contractions randomly performed on three separate sessions, and (ii) pre- and post-treatment maximal voluntary isometric contraction (MVC). Prior to and between sets of FT, a proprioception task (PT) consisting of matching the right arm to the left reference arm was performed at three different target angular positions (70°, 110° and 150°). Each set was immediately followed by 3 PT and 1 minute rest. The statistical analysis revealed that post-treatment MVCs were significantly decreased compared to pre-treatment MVC in all conditions with a greater drop following the eccentric session. Despite this greater drop, position sense was only significantly affected by the concentric exercise session. In addition, matching errors tended to be larger at 110° compared to the other angles. The results showed that concentric muscle contractions impaired position sense in the midrange angle of the elbow joint and this should be taken into consideration when designing proprioception rehabilitation programs.

**Key words**: elbow, muscle contraction, position sense, force, midrange angle, matching.

**ACKNOWLEDGMENTS**

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I also would like to thank my family and friends who have supported me throughout this process in many ways, for being a listening ear when I needed it, and for always giving me the freedom to make my own choices. Lastly, I express a special thanks to my partner, Harold Loveless, for his support and encouragement during my graduate studies.

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| | |
|---|---|
| α | alpha |
| γ | gamma |
| % | percentage |
| ° | degree |
| Ach | acetylcholine |
| ACL | anterior cruciate ligament |
| ADP | adenosine diphosphate |
| AE | absolute error |
| ATP | adenosine triphosphate |
| C | celsius |
| $Ca^{2+}$ | calcium ion |
| CE | constant error |
| CI | confidence interval |
| cm | centimetre |
| CNS | central nervous system |
| Cr | creatine |
| DOMS | delayed onset muscle soreness |
| E-C | excitation-contraction |
| EMG | electromyography |
| FT | fatigue task |
| h | hour |
| $H^+$ | hydrogen ion |

| | |
|---|---|
| $H_2PO_4^-$ | dihydrogen phosphate |
| Hz | hertz |
| iEMG | integrated electromyography |
| iMVC | maximal isometric voluntary contraction |
| $K^+$ | potassium ion |
| Kg | kilogram |
| KHz | kilohertz |
| $Mg^{2+}$ | magnesium ion |
| min | minute |
| mm | millimetre |
| $mmol \bullet L^{-1}$ | millimol per litre |
| ms | millisecond |
| m/s | metre per second |
| mV | millivolt |
| MVC | maximal voluntary contraction |
| N | Newton |
| $Na^+$ | sodium ion |
| $NH_3$ | ammonia |
| NMJ | neuromuscular junction |
| NS | non significant |
| PCL | posterior cruciate ligament |
| PCr | phosphocreatine |
| PFK | phosphofructokinase |

| | |
|---|---|
| Pi | inorganic phosphate |
| PT | proprioception task |
| s | second |
| SE | standard error |
| SI | primary somatosensory cortex |
| SMA | supplementary motor area |
| SR | sarcoplasmic reticulum |
| TAP | target angular position |
| T-tubule | transverse tubule |
| VE | variable error |

# CHAPTER 1 THESIS OVERVIEW

## 1.1 OVERVIEW OF THESIS

Chapter 2 includes a review of literature concerning the debate about the origin of the proprioception senses and to what extent they are derived peripherally (peripheral afferent signals) or centrally (sense of effort). Nevertheless, the center of interest of the review is on the effect of repetitive muscle contractions on position sense. A substantial body of research demonstrated that position sense is negatively affected by isometric, concentric, and eccentric exercise protocols. The different possible mechanisms involved are then discussed. The review also covers a section on sport rehabilitation through proprioception training. Prevention of reinjury and effect of athletic training are also briefly discussed in this section.

Chapter 3 reports the investigation of eleven active male subjects matching three different elbow angles after having gone through an isometric, concentric, and eccentric exercise task performed on three separate sessions. Pre- and post- maximal voluntary contractions were also collected in each session to ensure that a decrement in muscle performance has occurred in the three exercise tasks. The hypothesis was that eccentric exercise would lead to a greater decrease in force and consequently to larger matching errors.

In Chapter 4, the responses to the research hypothesis are answered together with a brief summary of the thesis. In addition, a discussion of the limitations of the methods employed in the study is included.

## 1.2 COLLABORATIONS

The design and identification of my research proposal was a collaborative effort between Dr. Fabien Basset and I. The set-up of the equipment and fine-tuning of the protocol was a collaborative effort between Chris Batten, Randy Thorne, Stephen Sooley, Lise Petrie, Dr. Basset and I. The data collection was completed with the help of Lise Petrie, a group of undergraduate students and I. The experiment was conducted in Dr. Behm's Exercise Physiology Laboratory using his laboratory equipment. The data analysis was a collaborative effort between Dr. Basset and I. For the review of literature and the manuscript included in the thesis, I prepared first drafts of both chapters. Dr. Basset provided the necessary feedback and corrections on both chapters, where Dr. Teasdale, Dr. Billaut and Dr. Behm provided indispensable comments on the manuscript.

# CHAPTER 2 REVIEW OF LITERATURE

## 2.1 INTRODUCTION

In the absence of vision we have an accurate sense of limb position. Thus, in a dark room, we are unerringly able to place our index finger on the tip of our nose. It implies that human being knows the position of the hand at any time during a movement and has an accurate map of the location in space of different body parts. The sense which the body relies on to achieve such an accomplishment is called proprioception. It is generally divided into two distinct elements: static and dynamic proprioception. Static proprioception, also called joint position sense, is the conscious perception of the orientation of different body parts whereas dynamic proprioception, defined as kinaesthesia, is the sense of limb movement. Due to the complexity of the subject, this review focuses primarily on static proprioception.

The control of a movement is highly dependent on the quality of the afferent information originating from the various somatosensory systems, such as interoceptors, involved in proprioception. There has been much debate about the origin of the proprioceptive senses and to what extent they are derived peripherally or centrally. The traditional view is that signals from muscle spindles provide us with our sense of limb position. However, the present-day view is that the sense of effort would play a more important role in joint position sense. Thus, the role of peripheral afferents directly or indirectly involved in proprioception is also illustrated in this work.

Different factors such as injury, cognitive distraction, inactivity, and muscle fatigue have been demonstrated to impair proprioception. The latter is defined as a class of acute effects that impair performance including an increase in the perceived effort and an eventual

inability to produce this force. Its effect on position sense is the center of interest for this review. It is legitimate to question how intense physical activity would perturb the sense of position knowing that repeated muscle contractions modify the peripheral proprioceptive system and thus the central processing of proprioception. In fact, results from independent studies using different protocols confirm that position sense is negatively affected following isometric, concentric, and eccentric type exercise protocols. Alas, none at this point has compared the three muscle contraction types within the same protocols and subjects.

Proprioception has also been a topic of interest in sport rehabilitation because injuries have been found to have a detrimental effect on proprioception through damage of mechanoreceptors in ligaments. In theory, operative techniques could restore proprioception directly through reinnervation of damaged structures; however controversy still exits regarding the return of proprioceptive function after joint reconstruction. Beneficial aspects of proprioception training following injury or surgery have been reported for many years. Accordingly, proprioceptive training has become an integral aspect of functional rehabilitation. Additionally, topics such as the prevention of reinjury and the effect of athletic training on proprioception are briefly discussed in this work. Finally, the last section of this review provides ideas for future studies related to the present subject, proprioception.

## 2.2 MUSCLE PERFORMANCE DECREMENT

### 2.2.1 Voluntary movements

Feedback from afferent neural pathways is provided to the brain by a number of sensors throughout the body. Sensory information includes cutaneous sensations (touch, pressure, vibration, and temperature), special senses (taste, smell, hearing, sight, and equilibrium), and proprioceptive sensations (awareness of body positions). The term proprioceptive is used for sensations pertaining to the musculoskeletal system, that is, muscles, tendons, joint capsules, and ligaments. These are all low-threshold mechanoreceptors and signals from them are conducted centrally in thick, myelinated axons. Whether located in a muscle or in a joint capsule, joint movement is the natural stimulus that leads to activation of such receptors (Brodal, 1998).

Descending control from higher brain centers can produce muscle contraction directly by acting on the alpha ($\alpha$) motoneurons and indirectly by the gamma ($\gamma$) motoneuron causing contraction of muscle fibres. Alpha motoneurons innervate extrafusal (striated) muscle fibres while gamma motoneurons innervate intrafusal muscle fibres, adjusting the sensitivity of the muscle spindle (muscle spindles are sensory receptors in the muscle) so that it will respond appropriately during muscle contraction.

The Ia afferents synapse with the $\alpha$-motoneuron that leads to the contraction. Later on, information from muscle spindles arrives at the cerebral cortex allowing perception of limb position and also passes to the cerebellum where it aids in the coordination of muscle contraction (Gandevia, 1996). The intrafusal fibres are innervated by sensory neurons called annulospiral and flower spray regulating the rate of length change and the change in length, respectively. When the muscle is stretched/shorten as the result of contractions, annulospiral

afferents transmit the signal to the spinal cord. Within the spinal cord the annulospiral afferent fibre bifurcates. One section synapses with the α-motoneuron leading to the agonist muscle. The other section connects with the inhibitory interneuron which then synapses with the antagonist muscle. The α-motoneuron will then leave the spinal cord and synapse with the extrafusal fibres of the agonist muscle. This results in the contraction of the muscle that is equal in force and distance to the original stretch. This is known as the myotatic reflex. The Ia afferents also make synaptic contact in the spinal cord with an inhibitory interneuron that synapses with the α-motoneuron of the antagonist muscle. This is known as reciprocal inhibition and is defined as the reflex relaxation of the antagonist muscle in response to the contraction of the agonist. This facilitates the contraction by preventing the antagonist from resisting the agonist contraction. This allows a higher rate, amplitude and intensity of movement contraction. The Ib afferents and Golgi tendon organ also control contractions by providing the cerebellum with sensory information regarding tension. This allows the muscle adjustments so that only amount of tension needed to complete the movement is produced.

Lower motoneurons (alpha and gamma) send their axons out of the brain stem to innervate the skeletal muscles of the head and body, and are the motoneurons in the ventral horn of the spinal cord. Local circuit neurons receive sensory inputs as well as descending projections from higher centers and provide much of the coordination between different muscles groups essential for organized movement. Each lower motoneuron innervates muscle fibres within a single muscle. The axons of all the motoneurons located in one spinal segment leave the spinal cord through one ventral root and continue in one spinal nerve.

The motoneuron pool is all the lower motoneurons that innervate a single muscle. Motoneuron pools are grouped together into rod-shaped clusters running down the cord for one or more spinal cord segments. Motoneuron pools innervating the arm are located in the cervical enlargement (C5-C7 innervate biceps brachii and brachialis muscles) and those that innervate the legs are located below in the lumbar enlargement (Kahle, Leonhardt, Platzer, & Cabrol, 1979; Rossignol, Dubuc, & Gossard, 2006). Neurons that innervate the axial and proximal musculature are located medially in the cord, while in the lateral cord are motoneuron pools that innervate muscles in the lateral portion of the body (Brodal, 1998).

The afferent pathways consist of three set of neurons: first, second, and third order. First order neurons carry signals from receptors to the brain stem and the spinal cord. Second order neurons carry signals from the spinal cord and brain stem to the thalamus; they decussate to the opposite side of these structures before reaching the thalamus. Third order neurons connect the thalamus to the somatosensory areas of the cortex where sensation is perceived. All of the somatosensory pathways are crossed, so that signals from one side of the body are brought to the cerebral hemisphere of the other side. The actual crossing over takes place at different levels for the various pathways (Brodal, 1998).

*2.2.1.1 Ascending tracts*

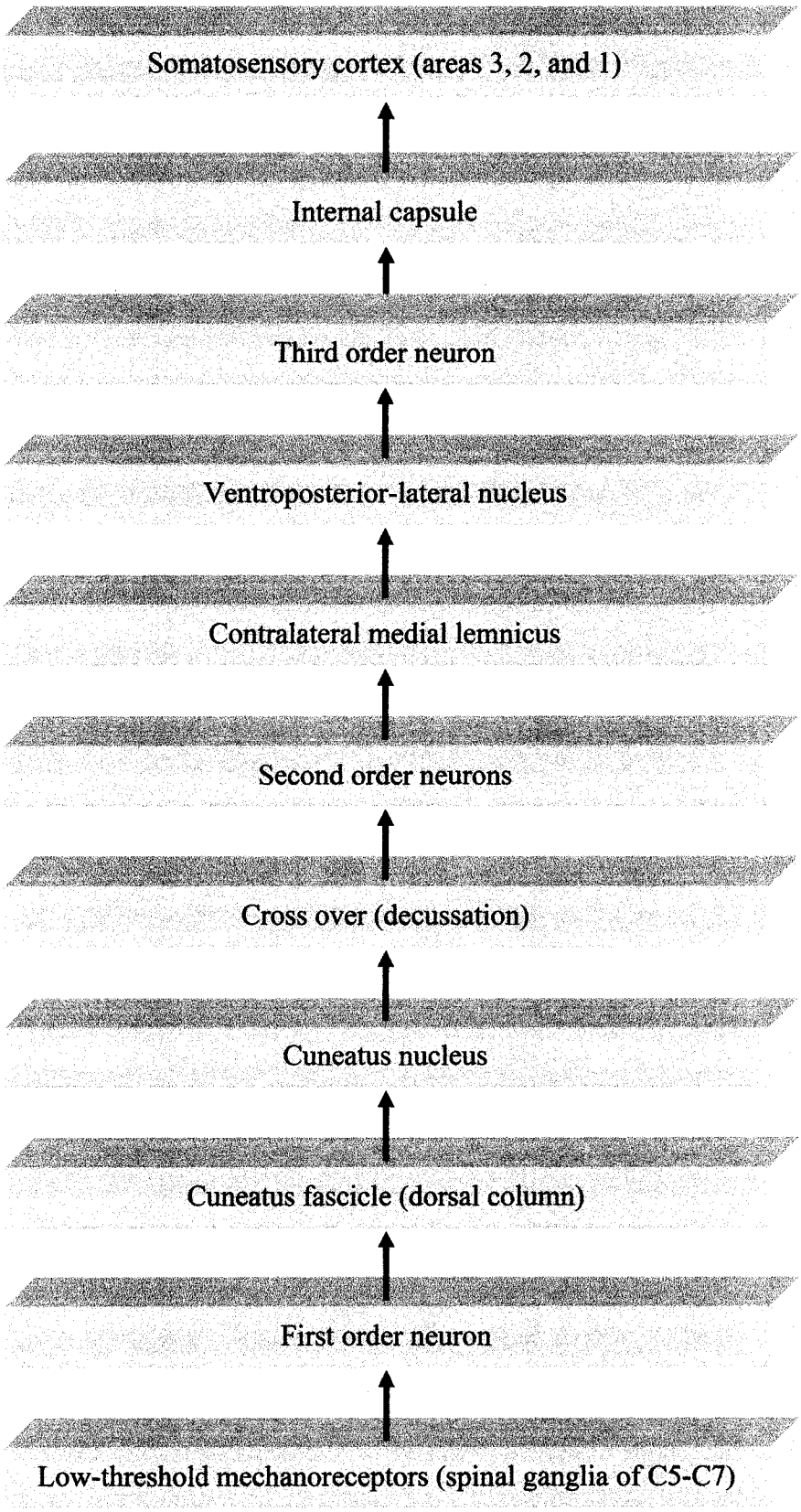
The dorsal column-medial lemniscus pathway is the main pathway for transmission of signals from low-threshold mechanoreceptors. The dorsal columns of the spinal cord carry information from sensory neurons in the spinal ganglia to the brain. The ascending fibres of the dorsal column pathway terminate in the cuneate and gracilis nuclei in the medulla. Afferents from the legs travel in the gracile fascicle and terminate in the gracilis nucleus,

while afferents from the arms travel in the cuneate fascicle and terminate in the cuneate nucleus. From the medulla, these signals travel to the ventroposterior-lateral nucleus of the thalamus via the medial lemniscus. The medial lemniscus crosses the midline of the body, so that signals from the left part of the body are received by the right ventroposterior-lateral nucleus (Latash, 1998). In other words, according to Brodal (1998) the dorsal column-medial lemnicus afferent pathway would look like Figure 2.1.

Almost all pathways conducting sensory information from the receptors to the cerebral cortex are synaptically interrupted in the thalamus or the cerebellum, which then distribute peripheral information to other brain structures. Eventually, a command is generated to the periphery on the basis of sensory information.

All of the neocortex project to the basal ganglia structures. Inputs to the basal ganglia may originate in different cortical areas such as the motor cortex, the premotor cortex, the supplementary motor area, the somatosensory cortex, and the superior parietal cortex with the exception of the primary visual and primary auditory cortices. Signals pass from the motor areas of the cerebral cortex to the pons and then into the cerebellum. In turn, signals from the cerebellum are transmitted back to the motor cortex by way of the ventro-lateral nucleus of the thalamus. This circuit could allow integration between the basal ganglia feedback signals and the feedback signals from the cerebellum (Latash, 1998). The latter sends information primarily to cell groups that give origin to the motor pathways, like the motor cortical areas, the thalamus, and the reticular formation of the brain stem. For instance, the cerebrocerebellum pathway would be involved in planning and initiating of voluntary activity by providing input to cortical motor areas while the spinocerebellum

Figure 2.1 Dorsal column medial lemnicus tract (spinothalamic tract) (Brodal, 1998)



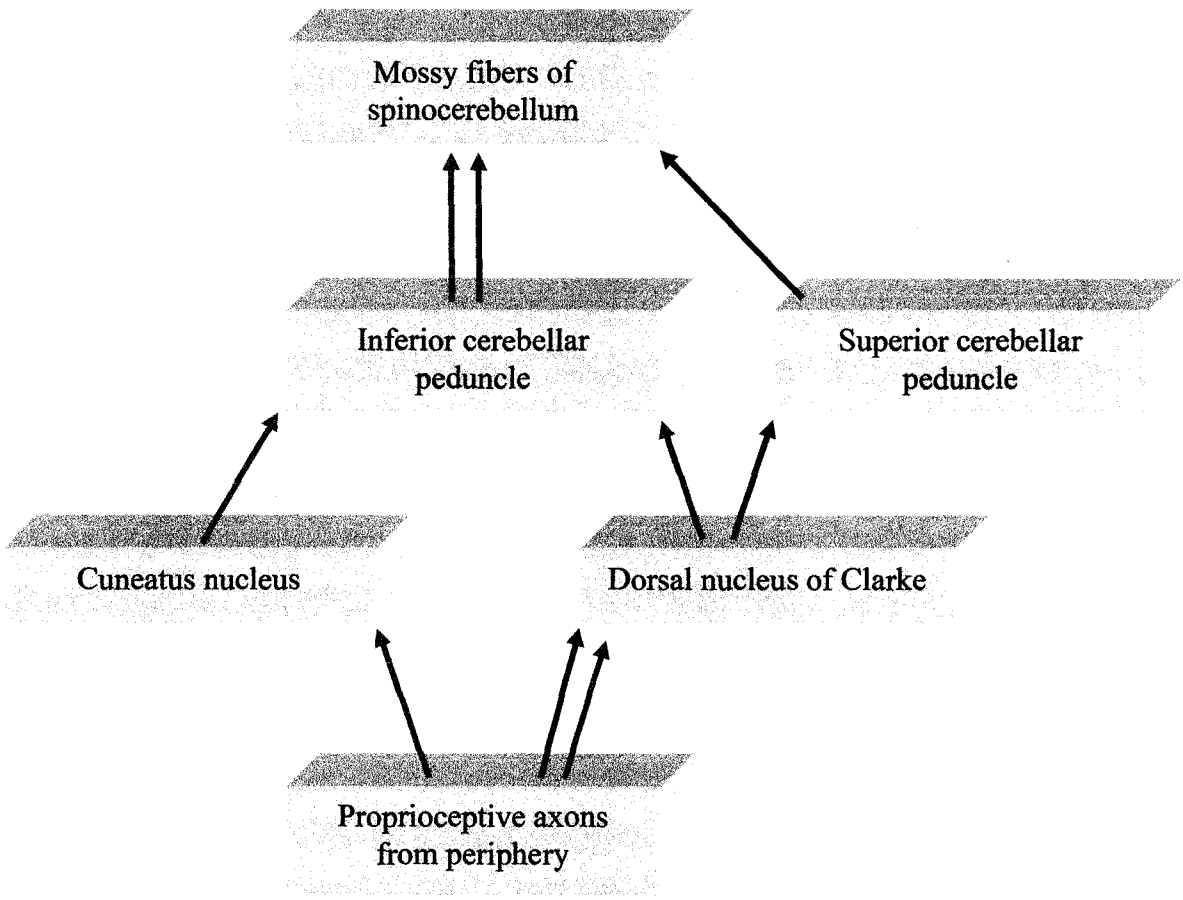
pathway would ensure accurate timing of contractions to coordinate movement involving multiple joints. The cerebellum receives important sensory signals directly from the peripheral parts of the body, which reach the cerebellum by way of the anterior and posterior spinocerebellar tracts which pass ipsilaterally up to the cerebellum. These tracts convey information from muscle spindles, Golgi tendon organs, and cutaneous low-threshold mechanoreceptors which provide the cerebellum with information about the movements produced. All this information keeps the cerebellum constantly appraised of the instantaneous physical status of the body (Guyton, 1981) and can probably lead the command to the desired result. There are three afferent pathways from the spinal cord to the cerebellum described in Figure 2.2.

The spinal cord receives sensory information from its own district, processes this information, and issues orders through motor nerves to muscles to ensure adequate responses. Many of the functional tasks of the spinal cord are under strict control and supervision from higher levels of the central nervous system (CNS). This control is mediated by fibres from the brain stem and cerebral cortex which descend in the white matter of the spinal cord and terminate in the gray matter of the spinal segments that are to be influenced. The information is mediated by long, ascending fibres in the white matter of the cord terminating in the brain stem (Brodal, 1998).

*2.2.1.2 Descending tracts*

The output of the cortical motor areas includes projections to basal ganglia, the cerebellum (mediated by pontine nuclei), the red nucleus, the reticular formation, and the spinal cord (corticospinal tract). Descending motor tracts take the commands to the spinal cord and to

Figure 2.2 (i) Anterior spinocerebellar tract (blue); (ii) Posterior spinocerebellar tract (red); (iii) Cuneocerebellar tract (green) (Brodal, 1998)

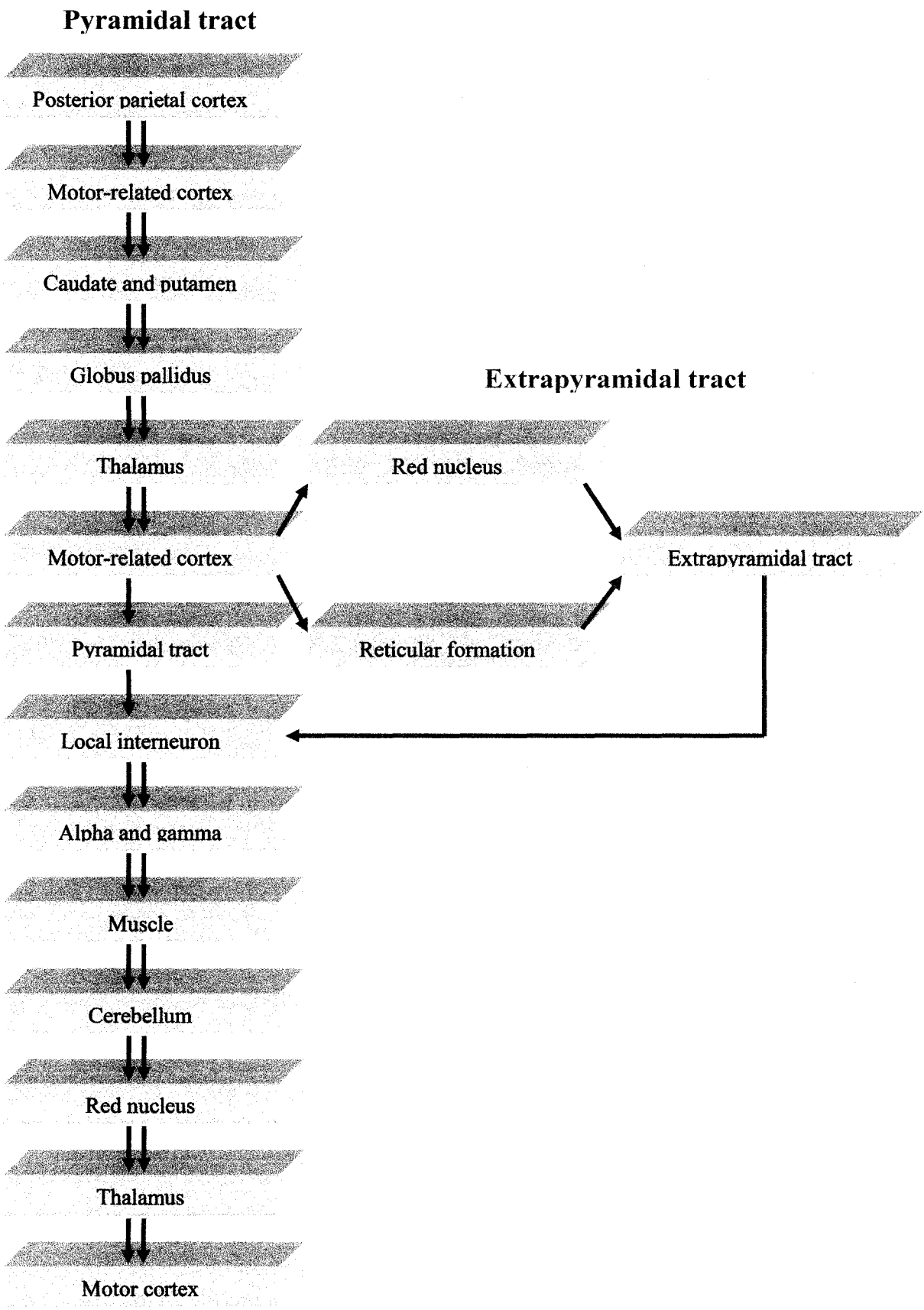


the motoneurons. These tracts originate from the motor regions of the cortex and the brain stem. In the brain stem, tracts originate from the red nucleus, the pontine, medullary reticular formations, and the vestibular nuclei (Guyton, 1981). Overall organizations of voluntary movements would look like Figure 2.3.

The pyramidal tract (corticospinal tract) passes downward through the brain stem; then it decussates mainly to the opposite side to form the pyramids of the medulla. By far the majority of the pyramidal fibres descends in the lateral corticospinal tracts of the cord and terminates principally on interneurons at the bases of the dorsal horns of the cord gray matter. These fibres control the motion of distal limbs on contralateral side of body. A few fibres, however, do not cross to the opposite side but pass ipsilaterally down the cord in the ventral corticospinal tracts and then mainly decussate to the opposite side further down the cord. The uncrossed fibres innervate mostly axial muscles (rotation of the trunk) (Guyton, 1981; Latash, 1998).

The extrapyramidal tracts are, collectively, all the descending tracts besides the pyramidal tract itself that transmit motor signals from the cortex to the spinal cord. Extrapyramidal pathways follow a more complex route through several structures, including the motor cortex, basal ganglia, thalamus, cerebellum, red nuclei, reticular formation, and nuclei in the brain stem (Guyton, 1981). In the spinal cord, the rubrospinal tract lies lateral and ventral to the lateral corticospinal tract. The rubrospinal tract controls distal hand movements on contralateral side of body. Stimulation of the red nucleus tends to excite flexor muscles of the arm and inhibit extensors (Karamyan, 1969). Based on this, one would deduce that it has

Figure 2.3 (i) Pyramidal tract (blue); (ii) Extrapyramidal tracts (red) (Guyton, 1981)



a role to play in control of reaching movements.

*2.2.1.3 Movement initiation*

Planning and realizing a movement result from an interaction between the sensory and motor systems. The major components of a motor program are believed to be developed not by the motor cortex itself, but by three interconnected cortical regions: the supplementary motor area (SMA), the premotor cortex, and the posterior parietal cortices in the posterior portions of the parietal lobe. The planning is performed by the posterior parietal cortex (Krakauer and Ghez, 2000; Scherberger, Jarvis, & Andersen, 2005). Together these three regions specify the movement toward the item of interest and thus play a crucial role in generating a motor response that is appropriate to the intended goal. The premotor and SMA receive input from the posterior parietal areas (5 and 7) and both project to the motor cortex (Krakauer and Ghez, 2000). The translation of the kinematic plan into activations that are necessary for the muscles relies on the motor cortex. The cerebellum makes a contribution to this translation by compensating for the motions that involve rotations in multiple joints. However, remember that movement initiation does not take place in the motor cortex. The cortex rather translates the motor idea and elaborates a spatio-temporal diagram of the planned action. Descending pathways from areas such as the lateral premotor cortex and the medial premotor cortex are required for the planning, initiation, and direction of voluntary movement. Guyton (1981) also stated that the caudate nucleus and putamen seem to function together to initiate and regulate gross intentional movements of the body. To perform this function, they transmit impulses through two different pathways: 1) into the globus pallidus and substantia nigra by way of the thalamus to the cerebral cortex, and finally downward into the spinal cord through the corticospinal and extracorticospinal

pathways; and 2) downward through the globus pallidus and the substantia nigra by way of short axons into the reticular formation and finally into the spinal cord mainly through the reticulospinal tracts. In contrast to Guyton, Latash (1998) mentioned that an important feature of changes in the firing patterns of cells in the basal ganglia is that they occur rather late; the majority of the cells show changes in their firing after movement initiation. Note that many neurons of the motor cortex change their firing prior to the movement initiation. So it is possible to conclude that neurons of the basal ganglia do not initiate movements and are related more to control of movements that are already under way (DeLong, 2000). Brodal (1998) is in accordance with Latash stating that the basal ganglia and the cerebellum have their main connections with the central motor nuclei and are necessary for the proper execution of movements rather than for their initiation (Ghez and Krakauer, 2000). Moreover, Guyton (1981) also quoted that the reticular formation of the brain stem and much of the thalamus play essential roles in activating all other parts of the brain. Therefore, it is very likely that these areas provide at least part of the initial signals that lead to subsequent activity in the motor cortex, the basal ganglia, and the cerebellum at the onset of voluntary movement. It was also reported by Brodal (1998) that not only the cerebral cortex is activated before a voluntary movement, there is also increased neuronal activity in the cerebellum, the thalamus, and parts of the limbic structures. Thus, many parts of the brain cooperate in deciding and planning movements.

*2.2.1.4 Muscle contraction*

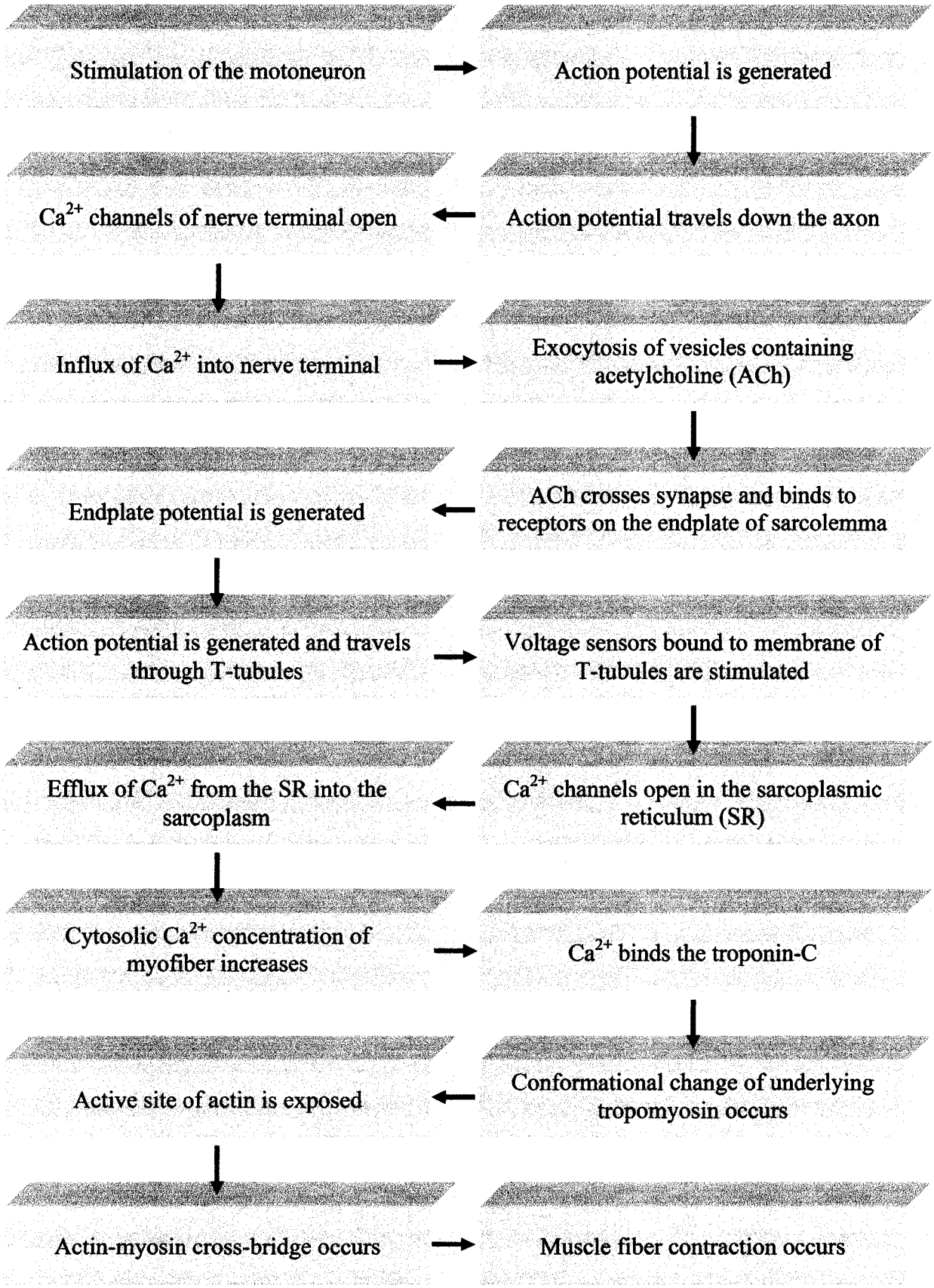
Electrical signals that arise in the CNS are propagated as action potentials to skeletal muscles by lower motoneurons. These somatic efferent nerves have their cell bodies either in the brain stem or spinal cord. The axons of motoneurons are large in diameter and

myelinated and are thus able to propagate action potentials to muscle fibres at high velocity. Each muscle fibre has one neuromuscular junction (NMJ), and yet each motoneuron innervates many muscle fibres within a given motor unit (Brooks, Fahey, White, & Baldwin, 1987). A muscle fibre contracts in response to one or more action potentials propagating along its sarcolemma and through its T-tubule system. Muscle action potentials arise at the NMJ, the synapse between a motoneuron and a skeletal muscle fibre. Because the cells do not physically touch, the action potential from one cell cannot jump the gap to directly excite the next cell. Instead, the first cell communicates with the second indirectly by releasing a chemical called a neurotransmitter. A nerve impulse elicits a muscle contraction in numerous steps illustrated in Figure 2.4.

*2.2.1.5 Pathways involved in a matching task*

During a matching task, for instance, a proprioception task consisting of a flexion-extension arm movement during which the subject best match his right arm (without vision) to his left reference arm, the upper limb proprioceptive ascending tracts and the descending tracts of upper limb proximal muscles are the ones to explore. In summary, the dorsal column-medial lemniscus pathway is the main ascending pathway for transmission of signals from low-threshold mechanoreceptors. The ascending fibres of the dorsal column pathway terminate in the cuneate nucleus in the medulla. From the medulla, these signals travel to the ventroposterior-lateral nucleus of the thalamus via the medial lemniscus of the pons. The medial lemniscus crosses the midline of the body, so that signals from the left part of the body are received by the right ventroposterior-lateral nucleus. An overall organization of the spinothalamic tract for the abovementioned voluntary movement would look like Figure 2.1.

Figure 2.4 Steps involved in muscle contractions



The dorsal spinocerebellar tract also receives contributions from the muscle spindle and Golgi tendon organ afferents. However, note that Clarke's column starts only at the T1 level, which means that the dorsal spinocerebellar tract conveys information only from the hindlimb, not from the forelimbs. The analogue of the dorsal spinocerebellar tract from the forelimb is known as the cuneocerebellar tract. Peripheral afferent fibres (proprioceptors in muscles and tendons) project on the cuneate nucleus, whose neurons send their axons to the cerebellum. Note that the axons do not cross the midline (Latash, 1998). An overall organization of the cuneocerebellar tract for the abovementioned voluntary movement would look like Figure 2.5.

The medial and lateral descending reticulospinal tracts have both excitatory and inhibitory connections with spinal interneurons and motoneurons. Indeed, the reticulospinal fibres act on both the alpha and gamma motoneurons. The reticulospinal tracts are of particular importance for postural mechanisms for the orientation of the head and body toward external stimuli and for voluntary movements of proximal body parts. According to Brodal (1998), an overall organization of the reticulospinal tracts for the abovementioned voluntary movement would look like Figure 2.6.

In conclusion, sensory impulses from nearly all parts of the body are transmitted to the CNS, bringing information about conditions in the various tissues and organs and in our surroundings. The receptors are formed by the terminal branches of an axon (joints, muscles) and transmit the message to the CNS. The receptor translates the stimulus to the language spoken by the nervous system, that is, electrical impulses in the form of action

Figure 2.5 Cuneocerebellar tract (Latash, 1998)

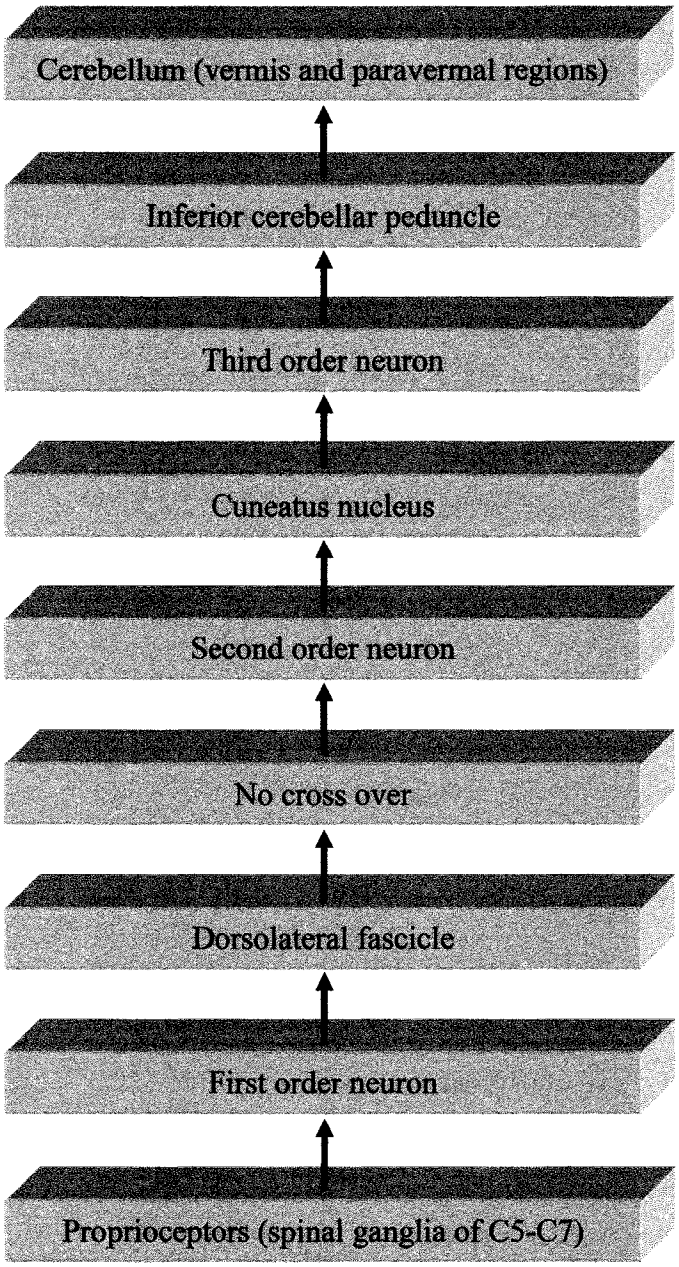
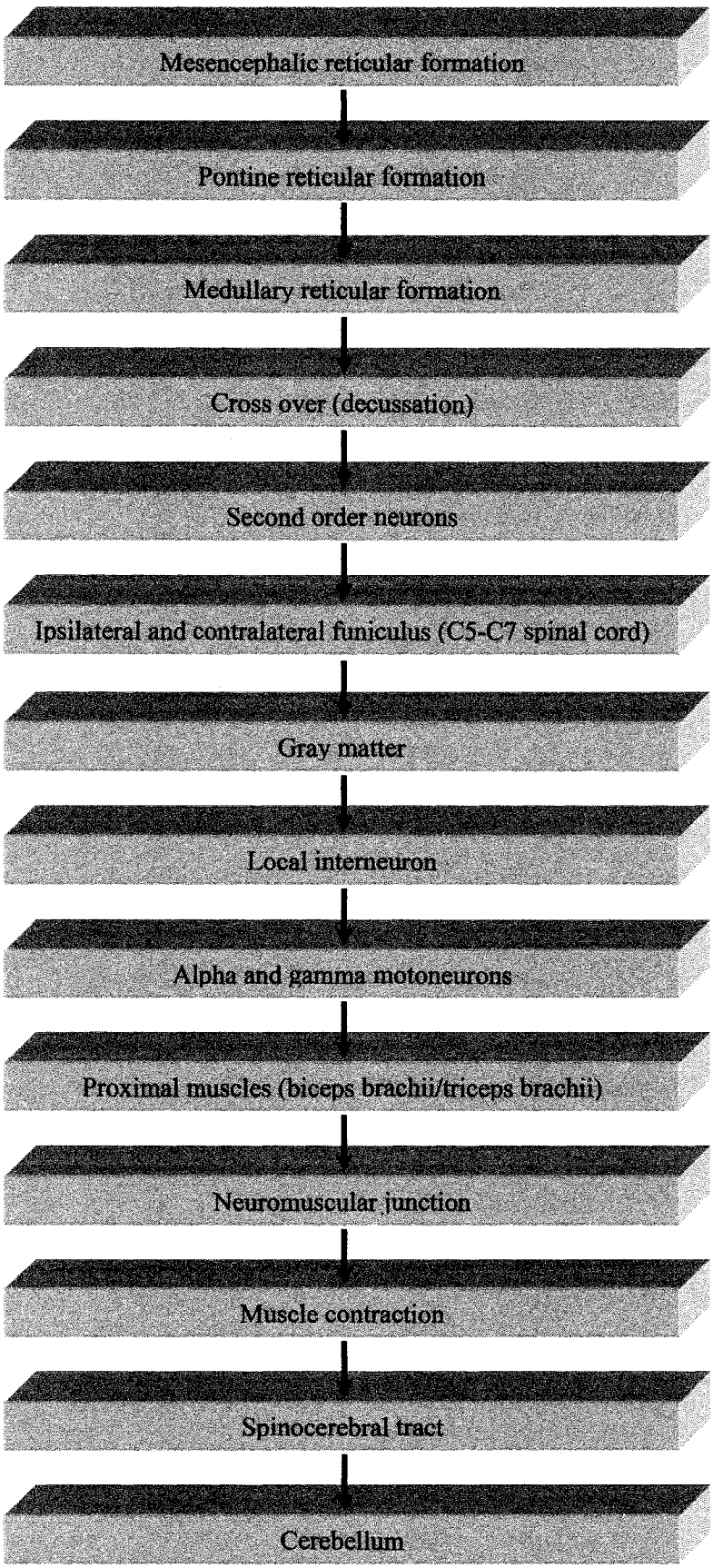


Figure 2.6 Reticulospinal tract (Brodal, 1998)



potential. The tracts in the CNS that control the activity of the skeletal muscles compose the motor systems. The peripheral motoneurons and the central motor pathways are directly involved in mediating the commands from the motor centers to the muscles. These parts of the motor systems are necessary for the initiation of voluntary movements. The basal ganglia and the cerebellum are necessary, however, for the proper execution of movements rather than for their initiation.

The voluntary movement involved in the matching task is a proprioceptive task where a subject has to perform a flexion-extension arm movement during which the subject best match his right arm (without vision) to his left reference arm. This task involves proprioceptors of the biceps brachii and the triceps brachii muscles meaning that the ascending pathways will follow a proprioceptive tract and the descending pathways will follow a proximal muscles tract. In this specific movement, the cerebellum receives information from peripheral sensory receptors by means of the dorsal column-medial lemniscus pathway. It is the main pathway for transmission of signals for low-threshold mechanoreceptors. The dorsal columns of the spinal cord carry information from sensory neurons in the spinal ganglia to the brain. Descending motor tracts take commands from the spinal cord to motoneurons. A ventromedial pathway includes the reticulospinal tract. This tract projects primarily from the reticular formation to the spinal cord and terminates in the ventromedial part of the spinal cord influencing proximal muscles. The medial and lateral reticulospinal tracts have both excitatory and inhibitory connections with spinal interneurons and motoneurons. The lateral reticulospinal tract activates flexor reflexes and inhibits extensor reflexes, whereas the medial reticulospinal tract activates extensor reflexes

and inhibits flexor reflexes of the proximal portions of the limbs. These tracts are, therefore, of particular importance for voluntary movements of proximal body parts.

### 2.2.2 Physiological events involved in muscle performance decrement

Muscle fatigue was recently denoted as a class of acute effects that impair performance, which includes both an increase in the perceived effort necessary to exert a desired force and an eventual inability to produce this force (Enoka and Stuart, 1992). This definition appears to be more appropriate than the traditional definition which does not appear to be an accurate description of the numerous physiological processes involved (Edwards, 1981). However, to avoid any confusion concerning the controversial definition of muscle fatigue, the term muscle performance decrement will be used in this review to denote any decline in exercise performance.

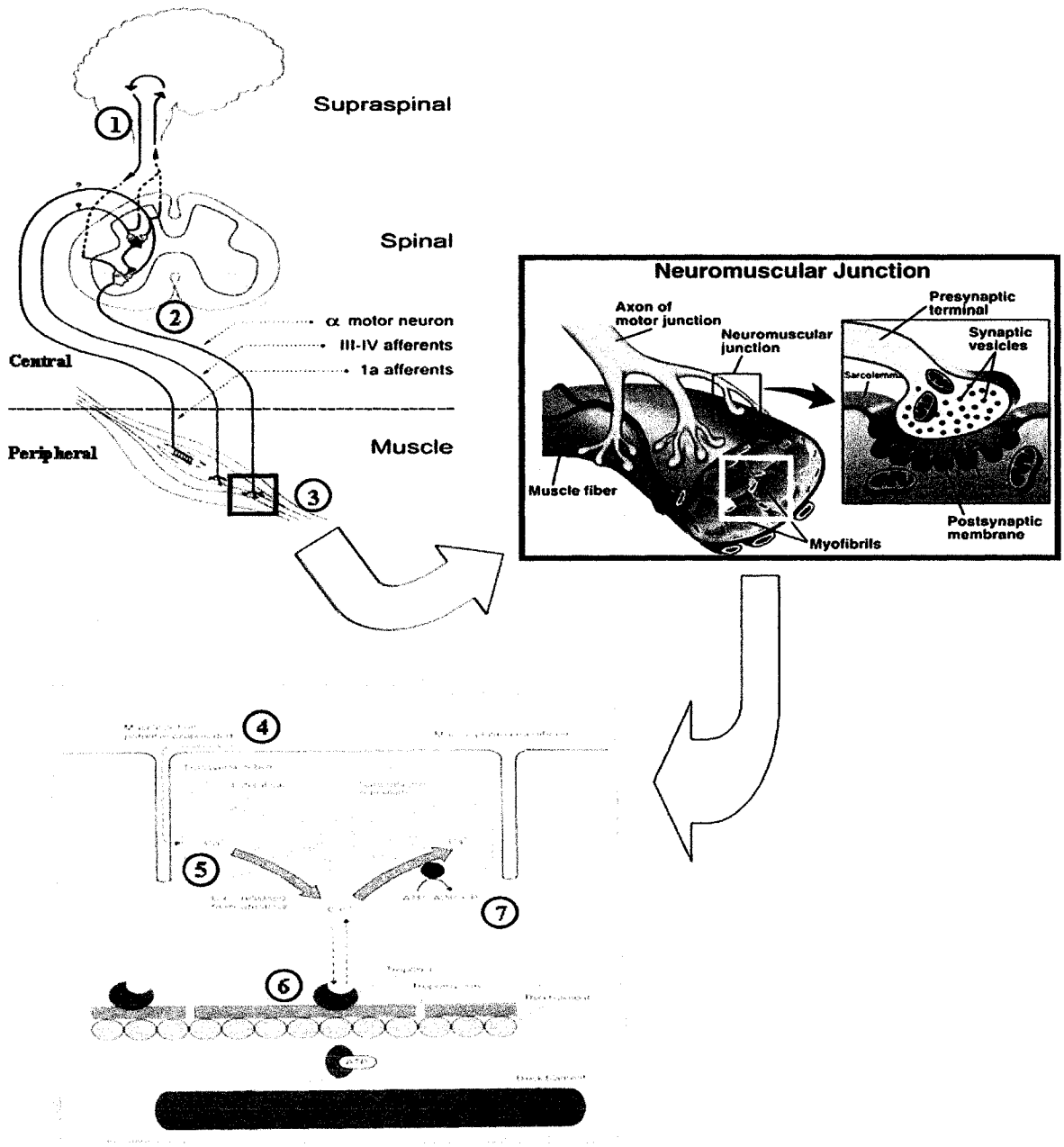
Despite years of investigation, a great deal of controversy surrounding the physiological causes of muscle performance decrement remains. There does not appear to be a single definitive cause, but rather a spectrum of events which occur in a complex chain of central and peripheral events (Fitts, 1994; Westerblad, Lee, Lannergren, & Allen, 1991). Thus, the origin of muscle performance impairment could be within the central nervous system (CNS) down to the actin-myosin cross-bridges. Central fatigue is described as a reduction in neural drive or motor command to the muscle resulting in a decline in force or tension development (Enoka and Stuart, 1992). Classically, central fatigue depends on motivation, CNS command transmission, or motor axons recruitment (Bigard and Guézennec, 1993). On the other hand, peripheral fatigue is defined as a decrease in the force generating

capacity of the skeletal muscle due to action potential failure, excitation-contraction (E-C) coupling failure, or impairment of cross-bridge cycling in the presence of unchanged or increased neural drive (Taylor, Bronks, Smith, & Humphries, 1997; Viitasalo and Komi, 1997). It includes mechanisms situated inside the muscle itself.

The development of the temporary loss of force caused by exercise is a complex process and results from the failure of a number of processes. In practical terms, however, we cannot know what actually leads to a decline in function. Bigland-Ritchie (1984) classified the potential events involved in muscle performance decrement into several sites (Figure 2.7): (1) excitatory drive to lower motoneurons; (2) motoneuron excitability; (3) neuromuscular transmission; (4) sarcolemma excitability; (5) E-C coupling; (6) contractile mechanism, and (7) metabolite accumulation.

Considerable controversy exists regarding the role of these sites, in particular, the relative importance of central (sites 1-2) versus peripheral (sites 3-7) mechanisms in the etiology of a decline in muscle performance. The relative importance of each factor will vary depending on the muscle contraction type (see section 2.2.4) and the intensity of the exercise. In fact, muscle performance diminution during tasks involving heavily loaded contractions such as weight lifting will differ likely from that produced during relatively unloaded movement (running, swimming). High-intensity exercise is frequently associated with a reduced neural drive and α-motoneuron activation frequency; however, rather than precipitate diminished performance, this change is thought to protect against its development (Bigland-Ritchie, Jones, & Woods, 1979; Bigland-Ritchie, Dawson, Johansson, & Lippold, 1986; Noakes and St Clair Gibson, 2004).

Figure 2.7 Potential events involved in muscle performance decrement (top panel was adapted from "Anatomy & physiology," by R. R. Seeley, T. D. Stephens, and P. Tate, 2006, p. 287. Copyright 2006 by The McGraw-Hill Companies, Inc.; bottom panel was adapted from "Human physiology: the mechanisms of body function," by A. J. Vander, J. H. Sherman, and D. S. Luciano, 1975, p.201 Copyright 1975 by McGraw-Hill, Inc.)



Factors eliciting muscle performance decrement during short-duration high-intensity exercise are clearly different from those involved during submaximal prolonged work. It involves a high contraction frequency and a high degree of anaerobic metabolism (Fitts, 1992). As a consequence of the high activation frequency, disturbances in E-C coupling, such as a conduction block of the action potential or an inhibition in SR $Ca^{2+}$ release, are more likely to occur. However, the high level of anaerobic metabolism will lead to an increase in intracellular hydrogen ion ($H^+$) and inorganic phosphate ($P_i$), factors known to inhibit peak force (Fabiato and Fabiato, 1978; Fitts, 1992; Robergs, Ghiasvand, & Parker, 2004; Thompson and Fitts, 1992). Thus, potential physiological sites involved in muscle performance decrement are briefly presented in this work.

*2.2.2.1 Central events*

There are several possible candidate mechanisms, working either individually or in combination, that could explain the decrease in central activation with exercise. They include: (1) decreased excitability of the motoneurons from supraspinal sources, (2) decreased excitatory influence from peripheral sources, and (3) increased inhibitory influences on motoneurons (Gardiner, 2001). Both supraspinal and spinal excitation may be inhibited by peripheral afferents. The decrease in net excitation appears to be due to a decreased excitatory influence from muscle spindles and an increased inhibitory influence from muscle receptors (Gardiner, 2001). In fact, Woods, Furbush, & Bigland-Ritchie (1987) and Garland, Garner, & McComas (1988) have previously found that spinal reflex arising from group III and IV afferents caused the decline in motoneuron firing rates. These afferents can be stimulated by chemicals such as metabolites and acidity (Basset and Boulay, 2002; Behm, 2004; Mateika and Duffin, 1995) and can provide both inhibitory and

facilitatory influences on motoneurons (Gandevia, Allen, & McKenzie, 1995). Chemosensitive afferent inhibition of the motoneuron should ensure that the activation of muscle fibres is down-regulated to diminish the chances of muscle damage (Behm, 2004; Haouzi, Hill, Lewis, & Kaufman, 1999). Some authors reported that the reafferences from the exercised muscle can give information about the level of muscle exhaustion, thus allowing the regulation of the motoneurons firing rate (Bigland-Ritchie et al., 1986; Garland and McComas, 1990). Thus, compensatory mechanisms at various levels of the neuromuscular system may act to delay the effects of exercise, this prolonging the accuracy of the motor activity (Enoka and Stuart, 1992). In addition, an increase in recruitment of new motor units has been reported during exercise as a result of a decrease in conduction velocity (Rozzi, Yuktanandana, Pincivero, & Lephart, 2000). This is to compensate for a decreased firing rate of the already recruited units in order to maintain constant tension (Rozzi et al., 2000). This change to lower frequencies for the motoneuron means less action potential generation per unit time, thus less stimulus and less stress on the neuromuscular junction (NMJ) and the sarcolemma propagation mechanisms. Perhaps central drive is limited because continued drive to the muscle would put the NMJ or more likely the intracellular events accompanying E-C coupling and actin-myosin interactions into a catastrophic status, one from which recovery would be delayed or impossible (Gandevia, 2001). Further work is needed to extract those supraspinal changes that cause the changes in voluntary activation and cause the changes in motor cortex excitability, and to correlate these central changes with indices of performance in different subject groups and experimental tasks.

*2.2.2.2 Peripheral events*

Neuromuscular transmission failure during muscle contractions refers to a failure of a nervous impulse to be translated into an impulse on the sarcolemma immediately beneath the motoneuron terminal. There are three main possibilities: 1) failure of propagation of the action potential into axon branches (branch-block failure), 2) neurotransmitter (ACh) depletion, and 3) post-synaptic membrane failure (Gardiner, 2001). In fact, axon branch-block failure occurs when the action potential generated in the axon is not propagated into all of the branches extending to the muscle fibres and the amplitude of end-plate potentials decrease due to lowered number of vesicles, ACh per vesicle, or both (McComas, 1996; Reid, Slater, & Bewick, 1999; Wu and Betz, 1998). Moreover, Na+/K+ pump located at the sarcolemma level is also considered as a possible cause of muscle performance impairment. It is apparent from the increase in K+ and Na+ that the pump capacity is insufficient to maintain the ionic gradients for K+ and Na+ essential for the maintenance of the cell excitability (Lindinger and Sjogaard, 1991; Sjogaard, 1991). The pump density may not be high enough to fully compensate for the ionic fluxes during the action potentials. The general theory is that K+ efflux and inhibition of the Na+/K+ pump (or its inability to keep pace with K+ efflux and Na+ influx) causes cell depolarization, a reduced action potential amplitude, and in some cells, complete inactivation (Fitts, 1994). Altered concentrations of ions (decreased K+, increased Na+) may disturb the membrane potential sufficiently to decrease the excitability of the sarcolemma (Sjogaard, 1986). In contrast, the sarcolemma action potentials recorded from surface electrodes did not diminish in amplitude during exercise, leading Merton (1954) to conclude that muscle impairment was not caused by NMJ or sarcolemma failure, but rather by events within the muscle cell.

The frequency-dependent decline in force-generating capacity indicates that muscle performance decrement was associated with E-C coupling impairment (Saugen, Vollestad, Gibson, Martin, & Edwards, 1997). Similarly, Roberts and Smith (1989) concluded that the early phase of muscle performance decrement was probably due to E-C coupling failure as a result of impaired transverse tubule (T-tubule) function with incomplete membrane repolarisation and possible changes in the ionic composition of the T-tubule fluid. While elevated T-tubular $Ca^{2+}$ might impair muscle performance by blocking conduction of the action potential into the axial core of the fibre, low $Ca^{2+}$ could directly reduce intramembranous T-tubular charge movement leading to a reduced $Ca^{2+}$ release and force production (Fitts, 1994). Further, Garcia, Gonzalez-Serratos, Morgan, Perreault, & Rozycka (1991) also concluded that the decline in muscle performance was caused by either a failure of the tubular action potential or the conduction signal between the T-tubular system and the terminal cisternae. Additionally, the combination of a high K+ concentration in the T-tubules due to slow diffusion, and only modest electrogenic Na+/K+ pumping would be expected to result in sustained depolarization of the tubular membrane and block off the local action potential mechanism (Jones, 1996; Sjogaard and McComas, 1995; Westerblad et al., 1991). In fact, failure of inward spread of the action potential is suggested to diminish signal entering the fibre from the T-tubules and then less $Ca^{2+}$ would be released from the SR by the ryanodine receptors (McComas, 1996; Rios, Ma, & Gonzalez, 1991). On the other hand, Westerblad and Allen (1991) agreed that a decrease in muscle performance may occur because of the inhibition of the release of $Ca^{2+}$ from the SR. They found a reduction in $Ca^{2+}$ release from the SR during exercise in single muscle fibres of mice. They also found that the reduction in $Ca^{2+}$ release from the SR and the depression of force were lessened by the addition of caffeine, which activates $Ca^{2+}$ release channels in the SR. The action of

caffeine suggests that the SR $Ca^{2+}$ release channel is the site responsible for the reduction in $Ca^{2+}$ release seen during exercise. Likewise, Gyorke (1993) concluded that the reduced $Ca^{2+}$ transient was caused by a direct inhibition of the SR $Ca^{2+}$ release channel and not by a disturbance in T-tubular action potential. Metabolic end products such as $H^{+}$, lactate, and $Mg^{2+}$ have been shown to reduce $Ca^{2+}$ release from SR vesicles (Favero, Zable, Bowman, Thompson, & Abramson, 1995). Thus, the metabolic alterations resulting from high-intensity exercise may have impaired SR function and contributed to the reduced exercise performance. Swollen SR vesicles and a decrease in SR ATPase uptake of $Ca^{2+}$ have also been observed following endurance exercise in the presence of excess $H^{+}$ (Inesi and Hill, 1983). In fact, a decreased intracellular pH is known to inhibit $Ca^{2+}$ pumps and myofibrillar ATPase activities, leading to a reduced rate of $Ca^{2+}$ uptake by the SR and detachment rate of the cross-bridges, consequently slowing muscle relaxation (Allen, Westerblad, & Lannergren, 1995; Fitts and Metzger, 1988; Westerblad, Allen, Bruton, Andrade, & Lannergren, 1998). However, metabolic changes alone cannot explain the decrease in force. Indeed, a maximal torque deficit was still present after a 30-min rest period, a time during which metabolic changes should normally be restored (Jones, 1996; Miller et al., 1987; Westerblad et al., 1991).

The role of $Ca^{2+}$ in E-C coupling suggests several sites at which $Ca^{2+}$ flux may be altered. The transmission of an action potential down the T-tubules results in the release of $Ca^{2+}$ from the terminal cisternae of the SR. The increased level of cytoplasmic $Ca^{2+}$ leads to increased binding of $Ca^{2+}$ to troponin-C and the subsequent events resulting in mechanical contraction (Roberts and Smith, 1989). However, Lee, Westerblad, & Allen (1991) and McComas (1996) also observed another factor that would contribute to E-C uncoupling,

that is, a reduced sensitivity of the contractile elements (troponin-C) to $Ca^{2+}$. In fact, competitive inhibition by $H^+$ with $Ca^{2+}$ for troponin-C binding has been demonstrated by Fabiato and Fabiato (1978) and Inesi and Hill (1983). Furthermore, Roberts and Smith (1989) concluded that muscle performance decrement was rather related to a decrease in the number of cross-bridges formed. The most commonly observed alterations include Z-line streaming, A-band lesions, and lengthened and nonuniform sarcomeres. Decline in muscle performance can also be caused by a myofibrils inability to use efficiently the available $Ca^{2+}$ for a muscle contraction or the inability to use the calcium signal to effectuate mechanical work.

*2.2.2.3 Metabolite accumulation*

A decline in muscle performance can begin immediately with the start of activity. In the first few seconds of a muscle contraction, there are increases in a range of metabolic by-products such as ADP, inorganic phosphate ($P_i$), creatine (Cr), and hydrogen ion ($H^+$) as a consequence of the increases in ATP and phosphocreatine (PCr) utilization (Sahlin, Tonkonogi, & Soderlund, 1998). One early candidate for the prominent role in muscle performance decrement was lactic acid. However, the consensus later on appears to be that an elevation of $H^+$ concentration was more critical than lactate or the undissociated lactic acid (Fitts and Metzger, 1988). Although little evidence exits to explain why a drop in pH would reduce force, one hypothesis suggests that a decrease in pH would reverse the equilibrium of the ATP-hydrolysis step, thereby limiting the binding of actin and myosin (Stackhouse, Reisman, & Binder-Macleod, 2001). In other words, a reduction in the amount of hydrolyzed ATP due to acidification would reduce the number of myosin heads undergoing a power stroke and therefore produce a lower amount of force (McLester, 1997;

Pate, Bhimani, Franks-Skiba, & Cooke, 1995). A decline in pH may reduce muscle force by: (1) decreasing $Ca^{2+}$ release from the SR, (2) decreasing the binding sensitivity of troponin-C to $Ca^{2+}$, (3) interfering with cross-bridge cycling, (4) decreasing in the frequency and duration of channel openings, (5) decreasing in maximal velocity of shortening, (6) prolonging SR $Ca^{2+}$ reuptake and relaxation time, (7) inhibiting ATPase, and (8) inhibiting PFK enzyme activity and thereby slowing glycolysis. All those factors may have a direct impact on power output production (Allen et al., 1995; Hargreaves et al., 1998). In fact, Stackhouse et al. (2001) mentioned that lowering pH reduced the affinity of $Ca^{2+}$ binding to troponin-C. They concluded that the mechanism behind this decreased sensitivity is unknown but evidence suggests that the increase in $H^+$ concentration during prolonged high-intensity activities directly interfere with the contractile machinery of muscle by competing for the $Ca^{2+}$ binding sites on troponin-C. A change in the ability of troponin-C to bind $Ca^{2+}$ could therefore reduce force generation. Moreover, acidic pH has also been shown to depress SR $Ca^{2+}$ reuptake presumably by inhibiting both the formation and cleavage of the phosphorylated enzyme (Fitts, 1994). However, this interaction does not appear to be a major mechanism underlying the decline in force. Additionally, the clear asynchronous dissociation between metabolic response and changes in force-generating capacity support the hypothesis that muscle performance impairment is unrelated to metabolite accumulation (Saugen et al., 1997). Exercise could, in spite of nearly depleted PCr levels, be continued without further pH changes and exhaustion was not associated with any sudden final changes in high-energy phosphates or pH. Hence, exhaustion was apparently not caused by lack of substrates for ATP resynthesis. It has also been found that, during the initial phase of recovery from exercise, pH either remains stable or continues to drop, whereas maximal voluntary contraction (MVC) steadily increases toward control

levels (Degroot et al., 1993). Thus, the major argument against a role for $H^+$ in the etiology of muscle performance decrement is the observation that pH recovers at a different rate than force following exercise (Adams, Fisher, & Meyer, 1991; Degroot et al., 1993; Pate et al., 1995; Westerblad, Bruton, & Lannergren, 1997; Wiseman, Beck, & Chase, 1996). Furthermore, Pate et al. (1995) used temperature jump techniques that allow testing of skinned fibres at temperatures above 15°C. They found a significant reduction (53%) of maximum isometric force and shortening speed by acidosis at 10°C temperature. In contrast, at 30°C temperature, they found only a minimal effect of acidosis, that is, a drop of only 18% in maximum tension. They concluded that at temperatures only slightly below physiological for mammalian skeletal muscle, pH plays a much less important role in the process of muscle performance decrement than has been suggested by data obtained at physiologically unrealistic temperatures. These experiments, therefore, demonstrate that when muscle is studied at temperatures that are closer to the normal body temperatures of living organisms, the effect of a decreasing pH on maximum isometric force and shortening speed is greatly reduce. Thus, because of the limited effect of pH when muscles are studied at temperatures similar to those in living organisms, the role of pH as a major causative factor in fatigue has been questioned.

Inorganic phosphate ($P_i$) is a by-product of the hydrolysis of ATP and its concentration also increases during intense skeletal muscle activity mainly due to breakdown of PCr (Westerblad, Allen, & Lannergren, 2002). There are various sites by which $P_i$ may affect muscle function during exercise. Increased $P_i$ may act directly on the myofibrils and decrease cross-bridges force production and myofibrillar $Ca^{2+}$ sensitivity. By acting on SR $Ca^{2+}$ handling, increased $P_i$ may also increase tetanic $Ca^{2+}$ concentration by stimulating the

SR $Ca^{2+}$ release channels; inhibit the ATP-driven SR $Ca^{2+}$ uptake; and reduce tetanic $Ca^{2+}$ concentration by entering the SR precipitating with $Ca^{2+}$ and thereby decreasing the $Ca^{2+}$ available for release. Thus, on the basis of recent findings, increased $P_i$ rather than acidosis appears to be the most important cause of muscle performance decrement during high-intensity exercise (Dahlstedt, Katz, & Westerblad, 2001; Dahlstedt, Katz, Wieringa, & Westerblad, 2000; Fryer, Owen, Lamb, & Stephenson, 1995). In fact, the results from Degroot et al. (1993) indicated that $H_2PO_4^-$ and $P_i$ demonstrate closer correlations to MVC than $H^+$ suggesting that $H_2PO_4^-$ and $P_i$ are more likely mediators of the decline in muscle performance although this does not establish causality.

Other metabolites such as magnesium, ammonia, potassium, and high-energy phosphate have also been reported to have a possible detrimental effect on muscle performance. However, it is still uncertain as to whether they are causative or merely coincidental. To sum up, among the metabolic changes that occur during exercise, only acidosis and increased $P_i$ are likely to have a negative effect of the depressed cross-bridge force early during muscle contractions. Giving that muscle performance impairment is unrelated to $H^+$ accumulation (Saugen et al., 1997), then by exclusion the depression would be due to accumulation of $P_i$ (Westerblad et al., 1998). However, it must be remembered that impairment in muscle performance is a complex phenomenon that does not appear to have a single definitive cause and that these causes may differ according to the different types of muscle contraction.

*2.2.2.4 Mechanisms of muscle performance decrement in regard to isometric, concentric, and eccentric contractions*

At first, it must be elucidated that the decline in muscle performance seen following all forms of exercise (isometric, concentric, eccentric) can come from a disturbance in any of the seven sites described earlier (see Figure 2.7). The relative importance of each factor and the time course may vary depending on the muscle contraction type. Nevertheless, it is well accepted that a failure of some steps in E-C coupling are impaired with isometric (Vollestad, Sejersted, Bahr, Woods, & Bigland-Ritchie, 1988), concentric (Pasquet, Carpentier, Duchateau, & Hainaut, 2000), and eccentric (Allen, 2001) contractions and would explain the force loss. That, in turn, leads to an uncontrolled release of $Ca^{2+}$ into the sarcoplasm and development of a local injury contracture (Morgan and Allen, 1999).

It also appears from the literature that the origin of a decline in muscle performance depends on the movement angular velocity (Linnamo, Hakkinen, & Komi, 1998) and muscle contraction mode (Kay, St Clair Gibson, Mitchell, Lambert, & Noakes, 2000). For example, using the twitch interpolation technique, Gandevia, Herbert, & Leeper (1998) argued that fatigue during a 30°/s concentric exercise might primarily be peripheral in origin, whereas Pasquet et al. (2000) obtained central fatigue (EMG activity decreases) during faster concentric contractions. Babault, Desbrosses, Fabre, Michaut, & Pousson (2006) also revealed different central and peripheral profiles when performing concentric and isometric exercises. During concentric exercise, decline in muscle performance was peripheral in origin and then central. The opposite profile was obtained when performing the isometric procedure since decline in muscle performance was firstly central, then peripherally mediated. The difference in central fatigue development observed between

concentric and isometric may originate from either or both spinal and supraspinal factors. These differences could also be partly the consequences of increased metabolite concentrations (de Haan, 1990). Likewise, Kay et al. (2000) reported that neuromuscular impairment profiles were different for isometric, concentric and eccentric muscle activities. These authors mentioned that whereas eccentric activity was largely fatigue resistant, isometric and concentric contractions displayed different neuromuscular impairment profiles. Moreover, isometric and concentric actions appear to be distinct regarding muscular mechanical properties and motor unit activation (Babault et al., 2006). In fact, in contrast to the findings with isometric contractions, integrated EMG output during concentric contractions was maintained or increased, while force output decreased. This indicates that neural drive to peripheral muscle was maintained during concentric but not during isometric contractions (Taylor et al., 1997). The implication is that, due to the dynamic nature of the concentric exercise, afferent information may be different and thus their effects on motoneuron firing rate during muscle activity may also be different. Supraspinal phenomena, such as suboptimal cortical output (Gandevia, Allen, Butler, & Taylor, 1996; Taylor, Butler, Allen, & Gandevia, 1996), might also account for the differences between isometric and concentric contractions. However, it was suggested that failures in motor cortex excitability may not explain the different central fatigue obtained during concentric and isometric, since an increase in cortical excitability has already been observed during isometric and concentric fatiguing contractions (Loscher and Nordlund, 2002; Taylor et al., 1996). We shall have to see more experiments involving dynamic types of submaximal contractions to resolve this issue.

Most studies of neuromuscular activity and muscle performance decrement have evaluated constant-load isometric contractions for an obvious reason: intramuscular electrode stability during the contractions allows unequivocal identification of the same motor unit over a prolonged period of time (Gardiner, 2001). Results showed that the limitation in blood flow appears to be specific to the isometric contraction (Humphreys and Lind, 1963). Accordingly, metabolite concentration might be higher during the isometric procedure compared with the concentric and eccentric and would increase the inhibitory effect of small-diameter chemosensitive afferents (Babault et al., 2006). Thus, due to the restriction of blood flow (ischemia) during isometric contraction, exhaustion from sustained activity is associated with the elevated intramuscular pressure or an increase concentration of metabolites associated with muscle activity such as $H^+$, K+, $P_i$ ions (Harris, Sahlin, & Hultman, 1977; Vollestad and Sejersted, 1988) or $NH_3$ (Mutch and Banister, 1983) and possibly also with a decline in the free energy of ATP hydrolysis (Dawson, Gadian, & Wilkie, 1980). In contrast with the continuous isometric contraction, the intermittent nature of the concentric procedure (muscular actions followed by a passive movement) may favour blood flow during the passive phase of the movement and, therefore, the evacuation of metabolic by-products (Laaksonen et al., 2003).

Accumulating evidence suggests that the CNS uses different control strategies for eccentric (lengthening) contractions and concentric (shortening) contractions (Enoka, 1996). This is commonly observed, indirectly, as a general inability for the CNS to fully activate the motoneuron pool during maximum voluntary eccentric contraction (Eloranta and Komi, 1980; Tesch, Dudley, Duvoisin, Hather, & Harris, 1990; Westing, Cresswell, & Thorstensson, 1991) and would therefore involve different levels of muscle activation rather

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than a selective activation of motor units. Moreover, compared to a concentric MVC, an eccentric MVC is usually associated with a lower activation level as measured by surface EMG. In fact, the EMG activity is significantly lower during eccentric compared to concentric and the differences in EMG are further reflected by differences in the excitability of the involved spinal motoneurons (Pinniger, Nordlund, Steele, & Cresswell, 2001). Thus, the observation that the recruitment order of motor units is altered during eccentric contractions provides evidence that the neural commands for these contractions are unique compared to other contraction types (Enoka, 1996; Hanon, Thepaut-Mathieu, Hausswirth, & Le Chevalier, 1998). However, some authors have reported that under some conditions, activation levels during concentric and eccentric contractions are not different (Komi, Linnamo, Silventoinen, & Sillanpaa, 2000).

Understanding the mechanisms of force reduction following eccentric exercise has made considerable progress in the last decade. It is now clear that the decline in muscle performance in eccentric work results from mechanical damage rather than the chemical processes of muscular contractions (Newham, McPhail, Mills, & Edwards, 1983). In fact, eccentric contractions have a lower metabolic cost than concentric contractions and thus the tension generated across the reduced number of recruited fibers is larger than for concentric contractions. Because of the greater force exerted by individual cross-bridges during eccentric contractions, the neural input required to produce a given force is much lower during eccentric than concentric contractions (Enoka, 1996; Westing et al., 1991). Thus, when a contraction changes from the shortening (concentric) of active muscle to lengthening (eccentric), there can be a change in the motor units that contribute to the muscle force. During eccentric contractions, individual sarcomeres elongate excessively

during the stretch but that most of these sarcomeres return to normal during relaxation with the thick and thin filaments reinterdigitated. With repeated stretch, it is probable that these sarcomeres gradually become damaged and then fail to reinterdigitate (Allen, 2001). The precise details of the sarcomere disruption process following eccentric contractions remain the subject of speculation. They may involve the elastic filament titin which anchors thick filaments to Z discs or the structural protein desmin (Allen, 2001). However, it is difficult to determine whether loss of desmin is a cause or a consequence of sarcomere disruption. Abnormalities due to repeated eccentric contractions may also include: dilation of the T-tubule system, distortion of myofibrillar components, fragmentation of the SR, lesions of the plasma membrane, cytoskeletal damage, changes in the extracellular myofiber matrix, swollen mitochondria, mechanical disruption of actin-myosin bonds, widening of A and I bands, and displacement of organelles (Friden and Lieber, 1992; Friden, Sjostrom, & Ekblom, 1981, 1983; McNeil and Khakee, 1992). Consequently, there are at least four mechanisms of force reduction involved in fibres damaged by repeated eccentric stretches: (1) weakened or overstretched sarcomeres cause a shift in the peak of the force-length curve to longer lengths; (2) changes in E-C coupling lead to reduced $Ca^{2+}$ release and reduced force; (3) there are fibres which are clearly degenerating and would be unexcitable; and (4) it seems likely that there are sarcomeres which are still close to their normal length but give less force, for instance, because their thick and thin filaments do not reinterdigitate (Allen, 2001). This is a category of damage for which there is little specific evidence. More work is needed to determine the generality of these observations, the mechanisms underlying the variability in motor unit recruitment, and the susceptibility of these mechanisms to the effects of muscle performance decrement.

To sum up, when the results from the various paradigms and protocols are considered together, it is apparent that the decline in force after exercise can be caused by many different mechanisms. Bigland-Ritchie classified in 1984 the major potential sites of muscle performance decrement into seven sites. A decline in muscle performance can come from a disturbance in any of these sites (see Figure 2.7). The relative importance of each factor and the time course for the onset of the decrease in muscle performance will vary depending on the muscle contraction type involved and the intensity of the exercise. Disturbances in E-C coupling could be mediated by an altered sarcolemma or T-tubular excitability, a depressed T-tubular charge sensor, or inhibition of the SR $Ca^{2+}$ release channel. Feedback of the declining peripheral performance is available via the full array of intramuscular afferent receptors. At a spinal level, these produce competing excitatory and inhibitory influences on the motoneurons pool, many of which could contribute to the decline in motor unit firing rate observed during MVC. Although there are undoubtedly significant peripheral sites involved, central sites may add substantially to the decline in muscle performance, even under optimal experimental conditions. Some of this reflects a failure of supraspinal drive to motoneurons that will act to protect the muscle from further decline in performance from peripheral sites, but at the expense of truly maximal performance. Input from small-diameter muscle afferents, particularly the group IV muscle afferents transmitting nociceptive input, reduces voluntary drive through a supraspinal action. The actions of group III and IV muscle afferents are complex and not exerted at only one point in the pathways responsible for force production.

### 2.2.3 Overview of EMG

Electromyography (EMG) is a technique to study muscle functions through analysis of the electrical signals emanated during muscular contractions. EMG measures the electrical signal associated with the voluntary or involuntary activation of the muscle. The EMG activity of voluntary muscle contractions is related to tension. The functional unit of the muscle contraction is a motor unit, which is comprised of a single α-motoneuron and all the fibres it enervates. Muscle fibres contract when the action potential (impulse) of the motoneuron reaches a depolarization threshold. The depolarization generates an electromagnetic field which spreads along the membrane of the muscle. The resulted muscle action potential is measured as a voltage. The EMG signal is thus the algebraic summation of the motor unit action potentials within the pick-up area of the electrode being used. However, EMG data cannot tell us how strong the muscle is, if one muscle is stronger than another muscle, if the contraction is a concentric or eccentric contraction, or if the activity is under voluntary control by the individual.

The features of the EMG signal depend on many factors. Some of them are not intuitive and vary with experimental conditions (Farina, Merletti, & Enoka, 2004). Many investigators assume that a crosstalk signal has a lower frequency spectrum because it originates further away and will be subject to additional low-pass filtering due to spatial filtering (De Luca, 1997). According to this rationale, high-pass filtering should reduce crosstalk; however, this is not a general finding (Farina et al., 2004). Moreover, EMG amplitude is influenced by such factors as electrode location, thickness of the subcutaneous tissues, distribution of motor unit conduction velocities, and the detection system used to obtain the recording.

Although some of these can be reduced by appropriate placement of the electrodes, there remains a mismatch between the output of the spinal cord and the EMG amplitude (Farina et al., 2004). Many other factors influence the relation between EMG amplitude and force. When muscles and subjects are compared, these factors include the thickness of subcutaneous tissue, the recruitment strategy, and the peak discharge rates of the different motor units. Because of the many factors that can influence this relation, there is no reason to expect that a specific EMG amplitude-force relation should have general validity (Farina et al., 2004). Other factors that influence surface EMG are for instance the shortening of muscle fibres during a dynamic contraction, distribution of the motor unit territories in the muscle, skin-electrode contact (impedance, noise), interelectrode distance, and so on (Farina et al., 2004). These limitations could lead to erroneous interpretation of the results and conflicting reports in the literature. Therefore, it is of major importance to be mindful and taking action to minimise their effect.

There are two main types of electrodes used for EMG: surface and fine wire. The surface electrodes are also divided into two groups. The first is active electrodes, which have built-in amplifiers at the electrode site to improve the impedance. The other is a passive electrode, which detect the EMG signal without a built-in amplifier, making important to reduce as much as possible all possible skin resistance. The advantages of surface electrodes are that there is minimal pain with application, they are easy to apply, and they are good for movement applications. However, they have more potential for cross talk from adjacent muscles since they have a large pick-up area. Surface EMG has been used for years as an overall index of muscle electrical activity, in as many domains as fatigue analysis, training assessment, muscle action in sport movements, pattern classification, pathology

identification, and many other areas where surface recordings were required owing to their non-invasive characteristic. The form of processing the EMG signal depends on the domain studied. The most common forms are: 1) full-wave rectification (absolute value of the entire signal); 2) linear envelope (low-pass filtering of the full-wave rectified signal); 3) root mean square (basically square the signal, take the mean of a timed determinant window, then take the square root); 4) integrated EMG (area under the rectified curve can be determined for the entire activity or for pre-set time or amplitude values); and 5) frequency analysis (typically determined via Fast Fourier analysis and looking at the power density spectrum). Between-subject comparisons can be made after normalizing the EMG activity, because the reference activity for a given subject is compared with the relative amount of activity for that subject and is therefore dependent on each individual's own proportion of maximal activity. Comparing a specific EMG muscle activity with a reference EMG value and expressing the activity of the muscle as a percentage of this reference value can establish its relative muscle activity (Hunter, St Clair Gibson, Lambert, & Noakes, 2002). Probably the normalization method the most widely used is to standardize to the maximum voluntary isometric contraction (iMVC) for the specific muscle being used. However, it is debatable if one can really ever obtain a true iMVC. Furthermore, EMG normalization also allows for slight changes in variables such as electrode placement and skin impedance (Mirka, 1991).

There is a relationship of EMG to many biomechanical variables. With respect to isometric contractions, there is a positive relationship in the increase of tension within the muscle with regards to the amplitude of the EMG signal recorded. One must be careful when trying to estimate force production from the EMG signal, as there is questionable validity of the relationship of force to amplitude when many muscles are crossing the same joint (De Luca,

1997). Moreover, it tends to have a relationship between EMG and velocity of the movement. In fact, there is an inverse relationship of strength production with concentric contractions and the speed of movement, while there is a positive relationship of strength production with eccentric contractions and the speed of movement.

Integrated EMG (iEMG) is probably one of the oldest forms of surface EMG activity tracking. It has been computed in several ways depending on the calculating procedure, analog or digital. This first step is always a signal rectification and the second an integration process (Duchene and Goubel, 1993). The iEMG analysis allows for determination of motor unit recruitment (muscle excitation), while EMG signal frequency spectrum analysis is generally a reliable indicator of signal conduction velocity (Enoka and Stuart, 1992; Sadoyama, Masuda, & Miyano, 1983). Both are considered as indicators of neuromuscular fatigue. When a subject is asked to generate a submaximal contraction until exhaustion, muscle EMG increases gradually to maintain the force through a combination of additional motor units recruited and increases in firing rates (Beck et al., 2006; Garland, Enoka, Serrano, & Robinson, 1994; Pincivero, Gandhi, Timmons, & Coelho, 2006). However, there is a loss of the high-frequency component of the signal with repeated muscle contractions which can be seen by a decrease in the median frequency of the muscle signal (Garland et al., 1994), suggesting changes in the muscle fibre conduction velocity and therefore possible derecruitment of some of the fast units (Komi et al., 2000). In the initial portions of a contraction, increased firing frequencies can be used to increase or maintain force, whereas in the later portion the decreased firing frequency can serve as an energy saving (Behm, 2004). It must be remembered that, in a voluntary situation, there is always some variation in the forces due to, for instance, motivation. Therefore, some changes in EMG are to be

expected for this reason. It has also been suggested that the EMG is reproducible in static situations as well as dynamic. In dynamic actions, it is possible that the electrodes may change its location in relation to the innervation zone, which may affect EMG power spectrum (De Luca, 1997; Roy, De Luca, & Schneider, 1986). However, the possible effects caused by electrodes movement should be similar among experiment sessions. Moreover, it has been reported that the average EMG was lower during the eccentric than the concentric in the middle part of the motion (Eloranta and Komi, 1980; Pincivero et al., 2006; Tesch et al., 1990; Westing et al., 1991). This reduced EMG suggests an incomplete activation of the motoneurons that innervate the muscle. This might take the form of a lower level of activation, distributed across the entire population of motoneurons, or the activation only of a subset of the entire population (Enoka, 1996). Because of the technical limitations of conventional electrophysiological methods, it has not been possible to distinguish between these possibilities.

## 2.3 PROPRIOCEPTION

### 2.3.1 Overview of proprioception

When we close our eyes, we are able to touch the tip of our nose with remarkable accuracy. The sense which we rely on to achieve such an accomplishment is called proprioception. Scaliger used the term "motion sense" for the first time in 1557. In 1880, Bastian ( 1880) preferred using the term kinaesthesia, because he felt that perception was based on muscles, tendons, extremities and skin. Proprioception was originally defined by Sherrington ( 1906) as, "the position of joint and body movement as well as position of the body, or body

segments, in space". Later, Paillard and Brouchon ( 1974) defined proprioception as the conscious experience informing us about the position and motion of our limbs, permitting us to reproduce positions and movements.

Proprioception is one of the somatic senses. Somatic senses are functions of the nervous system that collect sensory information from the body, to the exclusion of the special senses (sight, hearing, taste, touch, smell, and vestibular). In the literature, three somatic senses are described: pain sense, thermoreceptive sense, and mechanoreceptive sense, the latter of which includes position sense. Hence, proprioception could also be defined as the cumulative neural input to the central nervous system (CNS) from specialized nerve endings call mechanoreceptors.

Proprioception is generally divided into two distinct elements: static and dynamic proprioception. Static proprioception, usually defined as the joint position sense, is the conscious perception of the orientation of different parts of the body with respect to another whereas dynamic proprioception, defined as kinaesthesia, is the sense of limb movement. There are evidences that static and dynamic proprioception are mediated by separate line of information.

Proprioception is crucial for coordinated movement. When there is a deficit in proprioception, controlled movements are impossible without continuous visual guidance (Ghez, Gordon, & Ghilardi, 1995), maintenance of force or position is severely impaired, and a tremor develops (Marsden, Rothwell, & Day, 1984). Different factors such as injury, cognitive distraction, muscle exercise, and inactivity have been demonstrated to impair

proprioception. Goodwin, McCloskey, & Matthews (1972) commented that proprioception may also be altered if subjects are not voluntarily in control of their limb, that is, if their arm is moved by the experimenter without the need for subjects to direct themselves to the target angle. Therefore, attention must be paid when choosing a protocol to test proprioception. A summary of the most common protocols is described next.

### 2.3.2 Experimental protocols in evaluation of proprioception

There are different ways in which to evaluate proprioceptive capabilities: histological, neurophysiological and clinical. In the clinical setup, most authors apply the threshold to detection of passive motion to evaluating kinaesthesia and the angle reproduction capability for measuring joint position sense. Other clinical protocols include the reproduction of target torques or velocities. All these tests are conventionally performed while limiting external stimuli such as visual cues and most of these test both limbs to compare bilateral ability.

Joint kinaesthesia is determined clinically by establishing the threshold to detection of passive motion, that is, an assessment of the ability to detect relatively slow passive joint motion. In order to minimize the contribution of musculotendinous mechanoreceptors in providing the CNS with information regarding limb position and movement, the threshold to detection of passive movement is conducted at slow angular velocities (0.5° to 2° per second). Without warning of initiation, the experimenter moves the joint in a randomized sequence of velocities. Subjects indicate when they first detect motion of the joint and at that point the motion is stopped. Most studies use a mechanical switch or verbal cue by the

subject to identify the initiation of passive motion. Another method used to measure kinaesthetic acuity is to impose limb movements and ask subjects to report the direction of movement. This method examines kinaesthesia when the muscles acting on the target limb are relaxed.

Protocols for measurement of joint position sense greatly vary among studies: (1) passive positioning with unsupported reference limb, (2) passive positioning with supported reference limb, and (3) active positioning of the reference limb. Passive means the experimenter moves the subjects' limb while active means that the limb is voluntary placed at the target position by the subjects.

Two methods of passive positioning with unsupported limb are commonly used: active reproduction of passive positioning and co-contraction positioning. The active reproduction of passive positioning required the subject to remain relaxed while the experimenter moves their reference limb at one of the test positions. The blindfolded subjects are then asked to hold it, unsupported, and match its position by voluntary placement of their contralateral limb. Subjects indicate verbally when they think both limbs are matched. The co-contraction condition is similar but it also requires subjects to maintain a 15% of maximal voluntary contraction (MVC) co-contraction of the agonist and antagonist muscles while it is being positioned, and then continue the co-contraction throughout the duration of the trial. Other investigators preferred testing the unilateral ability to passive positioning (Marks and Quinney, 1993; Sterner, Pincivero, & Lephart, 1998). It requires subjects to relax as the experimenter slowly moves their limb to the test angle. Subjects are then given 10 s to concentrate on the test angle before their limb is passively returned to the initial position.

With visual cues eliminated, subjects are instructed to actively attempt to reproduce the test angle.

The second method of testing joint position sense through passive positioning is used to test whether the peripheral afferents are involved in proprioception by supporting the reference limb. In this protocol, the experimenter positions the reference limb at a set angle where it is maintained by means of a support. Subjects therefore do not need to generate any effort to maintain the position of the reference limb. Once it has been placed on the support, subjects are instructed to relax and match the reference limb position by voluntary placement of their contralateral limb. A similar protocol was used by Walsh, Allen, Gandevia, & Proske (2006) but this time with both elbows on supports in the horizontal plane where there is no gravitational cue. In this case, the horizontal movement is almost frictionless and requires no effort to maintain a given elbow angle. Again in this protocol, the experimenter slowly positions the reference arm to the test angle and subjects are instructed to actively attempt to reproduce the test angle with their indicator arm.

The active reproduction of active positioning of the reference limb is evaluated similarly to the previously described protocols, but in this case subjects actively orientate themselves to the test angle. From the starting position, subjects actively move their reference limb until a command to stop is given. They are then asked to match it by similar placement of their indicator limb. After each match, both limbs are returned to the starting position. For the studies using the unilateral active positioning, a rig is locked after subjects have actively reached the target angle and the limb remains at this target position for 5 s. Then they

actively return to the starting position. Finally, they actively move the same limb in an attempt to match the target position.

Force-matching task is another frequently used protocol to test proprioception. In these experiments, subjects are asked to generate, under visual control, a given target force with their reference limb. Once they have satisfactorily achieved the reference force for more than 2 s, they are asked to match it with the contralateral limb, the indicator, whose force output is not displayed. In some studies, pain is induced in the indicator arm by injection into the muscle of small amounts of hypertonic (5%) saline (Bennell, Wee, Crossley, Stillman, & Hodges, 2005; Proske et al., 2004; Proske et al., 2003; Weerakkody, Percival, Canny, Morgan, & Proske, 2003b). Once the muscle is sore, the force-matching task is repeated. However, many subjects find it difficult to align isometric tensions, so a more familiar form of contraction has been used in many otherwise similar tests. In these tests, subjects are asked to lift a weight by contracting a particular muscle group on the reference side and to match it by selecting a weight on the indicator side such that both feel the same.

Detection of specific speeds of motion is also a method used to measure proprioception. For the movement-matching task, a range of movements is selected which includes speeds normally used by subjects in a simple pointing task. The experimenter moves the reference limb from a flexed into an extension position at one of the angular velocities and asks the blindfolded subjects to track the movement as accurately as possible with their indicator limb. One study has added a vibration task during the movement-matching task (Allen and Proske, 2006). Generally, in vibration experiments the movement-matching task is carried out while the limb's muscles are vibrated, using 80 Hz vibration. Vibration starts 0.5 s

before the beginning of each movement and is turned off once the indicator limb has reached full extension. The velocities between limbs are then compared.

### 2.3.3 Role of peripheral afferent signals

The control of a movement is highly dependent on the quality of the afferent information originating from the various somatosensory systems involved in proprioception. Receptors can sense information that is generated from within the organism (interoception) or sense information that arises from external stimulation (exteroception). Proprioception, in the classic sense, refers to position sense and movement sense arising from interoceptors (Hiemstra, Lo, & Fowler, 2001). Afferent nerves, also referred to as mechanoreceptors, are located within the skin, in the musculotendinous unit and within the bone, joint ligaments, and joint capsule. Over the years, many different views have been put forward on the origin of proprioception. The dominant view in the 19$^{th}$ century was that our kinaesthetic sense was a consequence of the effort we make to move and arises within the CNS. Subsequently, during much of the 20$^{th}$ century, it was believed that proprioception was dependent on peripheral afferent signals, but they were thought to be largely of nonmuscular origin, arising in the joints (Skoglund, 1973). However, because articular cartilage contains no neural elements, it was less likely that the actual joint surfaces played a major role in proprioceptive sense. It was the experiments of Goodwin et al. (1972) that provided the first direct evidence that signals from muscle spindles generated sensations of limb position and movement. More recently, Nicol and Komi (2003) reported that a potential reflex inhibition could be involved in proprioception via the sensitization of group III and IV receptors by the mechanical, thermal and chemical changes associated to the inflammatory phase of the

muscle regeneration process. The present-day view on peripheral afferents is that muscle spindles are responsible for the sense of position and movement, Golgi tendon organs provide the sense of tension, and that the sense of effort is generated within the CNS, as reviewed by Gandevia (1996).

*2.3.3.1 Muscle spindles*

Muscle spindle receptors are an encapsulated group of fine specialized intrafusal muscle fibres. The muscle spindles are 4-7 mm long and 80-200 microns wide. Their locations are in deep muscle tissue and they are arranged in parallel with the muscle fibres. The location and attachment of the muscle spindles make them sensitive to changes in length of the muscle. There are two types of intrafusal muscle fibres: nuclear bag and nuclear chain. The nuclear bag fibres are longer and thicker and they have multiple nuclei arranged centrally. The nuclear chain fibres are shorter and thinner and they have fewer nuclei. The nuclear bag fibres are involved in slow contraction and the chain fibres in fast contraction. The intrafusal fibres receive innervation from fusimotor axons (γ-motoneurons). The afferent terminals consist of primary (group Ia) and secondary (group II) endings. Since the muscle spindle is connected in parallel to the surrounding skeletal muscle mass, stretch of the muscle as a whole will excite the spindle sensory endings and afferents (group Ia, II). The primary endings are sensitive both to the length of the muscle and to the rate of stretch of the spindles while the secondary endings are mainly sensitive to the length of the muscle (Willis and Coggeshall, 1991).

The importance of muscle receptors in proprioception has been demonstrated by experiment in which vibration of a muscle produces illusions of position and movement of a joint. As

mentioned earlier, it was the experiments of Goodwin et al. (1972) on the sensory effects of muscle vibration that provided the evidence for a role of muscle spindles in conscious sensation. They believed that the vibration of the biceps tendon would produce a rapid alternating stretch and release of that tendon and in turn elicit distorted signals from the muscle spindles located in the vibrating biceps muscle. If the muscle spindle input is important in limb proprioception, then the distorted muscle spindle signals should result in impaired proprioception, which did occur in their investigation. Other studies recorded similar findings supporting the notion that muscle spindles may be the prime determinant in the perception of limb position (Bullen and Brunt, 1986; Capaday and Cooke, 1981). In the simplest view, muscle spindles are able to provide a position signal because they are stretch receptors. As the muscle lengthens, the spindle discharge increases, in direct proportion to the length of the muscle. The monotonic relationship between maintained spindle firing rate and muscle length could be used by the CNS to derive information about the length of the muscle and therefore the position of the limb. However, this brings with it the complication that spindle firing rates can be altered without changing muscle length, by means of fusimotor activity. In spite of that, it is generally accepted that muscle spindle afferents are mainly responsible for the sense of position (Gandevia, 1996; Matthews, 1988; McCloskey, 1978; Proske, Wise, & Gregory, 2000) and kinaesthesia, the perception of limb movement (Allen and Proske, 2006; Proske, Wise, & Gregory, 1999). The primary endings of muscle spindle would be concerned with signalling position and movement, while the secondary endings of muscle spindles are largely signalling position.

Many studies have looked at the effect of eccentric contractions on position sense performance (see section 3.5). It was speculated that if ordinary muscle fibres were

damaged by eccentric exercise, it was conceivable that the intrafusal fibres of muscle spindles, too, could become damaged. This would lead to a disturbance of spindle function and consequently errors in position sense. However, recent animal observations have shown that there is no evidence of a change in spindle responsiveness to stretch and to fusimotor stimulation after the damage from a severe eccentric exercise (Gregory, Morgan, & Proske, 2004). It was then argued that the class of muscle receptors to which the kinaesthetic sense is currently assigned, the muscle spindles is not well suited to signalling positional information. Other difficulties with muscle spindles as position sensors that need to be kept in mind are that the response of the passive spindle is dependent on the muscle's previous history of contraction and length changes (Gregory, Morgan, & Proske, 1988; Proske, Morgan, & Gregory, 1993) and that position sense becomes much more precise if active placement is used compared to when the target is achieved by placement of the passive limb (Allen and Proske, 2006; Goodwin et al., 1972). However, it is not intended to imply that muscle receptors are not involved in proprioception. Obviously, muscle spindles contribute to proprioception based on the illusions evoked by muscle vibration. Moreover, in the absence of an effort cue, muscle spindles provide a position signal, but not a very accurate one (Allen and Proske, 2006). The current working hypothesis of active researchers is that we receive positional cues both from signals of central origin (effort) and peripheral origin (muscle spindles).

*2.3.3.2 Golgi tendon organs*

The Golgi tendon organs are a slender capsule approximately 1 mm long and 0.1 mm in diameter located in tendons and aponeuroses. They are large fusiform-shaped receptors enclosed in a fine connective tissue capsule. These receptors are high-threshold, slowly

adapting mechanoreceptors that are stimulated only at extreme angles of joint displacement (Gandevia, 1996). Activation of the Golgi tendon organs requires an active or passive force by any of the attached muscle fibres to the receptor (Fukami, 1981). Binder (1981) has shown that the Golgi tendon organs can respond to forces < 0.2 N. They are sensitive to muscle stretch and contraction and are innervated by group Ib axons (Willis and Coggeshall, 1991). Contraction of muscle fibres stretches the tendon strands and that, in turn, stretches the nerve endings (Fukami and Wilkinson, 1977). So Golgi tendon organs, like muscle spindles, are stretch receptors. Stretch threshold for Golgi tendon organs is much higher than for muscle spindles and tendon organs typically do not exhibit background activity. They are presently regarded as sensitive detectors of muscle tension in a localized portion of a muscle (Fukami, 1981; Gregory, Brockett, Morgan, Whitehead, & Proske, 2002). However, if human Golgi tendon organs are similar to those in the cat in showing little change in their responses after eccentric exercise, the implication of these results for proprioception is the view that Golgi tendon organs do not play a dominant role in the perception of muscle tension. If Golgi tendon organs would continue to signal muscle force accurately, it seems unlikely that they would make a large contribution to force perception in circumstances where perturbations of force sense are seen.

*2.3.3.3 Group III and IV muscle afferents*

The afferents fibres are classified as large myelinated (group I), small myelinated (group II), smaller myelinated (group III), and unmyelinated (group IV). Group Ia fibres are associated with muscle spindles and are large and fast conducting. Group Ib fibres are associated with the Golgi tendon organs and are little smaller and slower conducting than the group Ias.

Group II fibres are associated with muscle spindles but are slower conducting and smaller that the group Ias and group Ibs. The muscle is also innervated by group III and group IV muscle afferent fibres. Group III fibres are smaller than group Is and group IIs and are only lightly myelinated and relatively slow conducting. Such fibres are associated with cooling and first pain and can be activated by mechanical stimulation of the muscle. These afferent fibres have different types of receptors. Some respond to the pressure applied at the junction between the muscle and the tendon; others are activated when the pressure is on the belly of the muscle; some are activated to muscle stretch, and others respond to manipulation of the space between muscles. Group IV afferents fibres are unmyelinated and have a role in muscle pain during ischemia because they respond to noxious chemical stimulation (Adreani and Kaufman, 1998; Mateika and Duffin, 1995; Willis and Coggeshall, 1991). Group III afferents are responsive mainly to mechanical stimuli, while group IV afferent are primarily nociceptors but also include mechanoreceptors (Hayward, Wesselmann, & Rymer, 1991). Group III and IV afferents exert strong inhibitory effects on γ-motoneurons innervating the muscle of origin of the afferents and their synergists by chemical changes (Nicol and Komi, 2003) and could provide information about the disposition of the limbs.

An alternative classification related to the sizes of axons is A and C fibres. A fibres (group III) convey sensory information for proprioception and touch while C fibres (group IV) represent unmyelinated fibres that primarily convey dull aching pain. C fibres are the most common element in peripheral nerves and almost all C fibres are nociceptors (Torebjork, 1974). Fine muscle afferents in group III and IV ranges also appear to serve as nociceptors. Histologically, the nociceptors are free nerve endings with conduction velocities less than 30 m/s and their typical location in the skeletal muscle is the wall of arterioles and the

surrounding connective tissue. Considerable evidence has been collected about the roles of A and C nociceptors in pain perception. Myelinated A axons have lower thresholds for electrical stimulation and are stimulated at lower stimulus levels than are C fibres. When peripheral nerves are stimulated in this way in humans, excitation of A fibres alone produces a tingling sensation (Collins, Nulsen, & Randt, 1960). When the stimulus is increased to also excite C fibres, a persistent burning sensation is produced.

It has been shown that afferent neuromuscular pathways are modulated via reflexes originating from small-diameter muscle group III and IV afferents (Bigland-Ritchie et al., 1986; Gandevia, 1998). There is a possibility that excitability of motor cortex is reduced as a result of the group III and IV input during muscle contractions (Taylor, Petersen, Butler, & Gandevia, 2000). These reflexes originating from the muscle and generated in response to metabolic changes that accompany muscle exercise could modify the central processing of proprioception by an inhibitory influence via group III and IV muscle afferents (Bigland-Ritchie et al., 1986; Nicol, Komi, Horita, Kyrolainen, & Takala, 1996). A further detailing of the mechanism may reside in the accumulation of metabolites within the muscle during activity to exhaustion (Basset and Boulay, 2002). Receptors of group IV afferents increase their firing rates in the presence of a number of substances that might increase in an exercised muscle, including bradykinin, potassium chloride, lactic acid, serotonin, and arachidonic acid (Djupsjobacka, Johansson, Bergenheim, & Wenngren, 1995; Nicol and Komi, 2003; Sinoway, Hill, Pickar, & Kaufman, 1993). In fact, metabolites have previously been shown to elicit increased muscle spindle static and/or dynamic sensitivity via reflex-mediated pathways from chemosensitive group III and IV afferents onto $\gamma$-motoneurons (Johansson, Djupsjobacka, & Sjolander, 1993; Pedersen, Sjolander, Wenngren, &

Johansson, 1997). Another hypothesis refers to a potential reflex inhibition via the group III and IV receptors by chemical changes associated to the inflammatory phase of the muscle regeneration process (Nicol and Komi, 2003). It is believed that the tissue-breakdown products associated with the inflammation sensitizes muscle nociceptors and elicits an increase in muscle spindle sensitivity (Smith, 1991). Therefore, in the acute phase following eccentric exercise, the observed changes in performance is suggested to be related to metabolic changes via group III and IV muscle afferents. This mechanism, however, does not explain the persistence of the matching errors over several days, at a time when all metabolites would have been removed. The delayed recovery, therefore, is suggested to be related to problems in the proprioceptive feedback caused by muscle damage and inflammation (Bottas, Linnamo, Nicol, & Komi, 2005).

The sense of muscle pain is believed to be mediated by group III and IV muscle afferents (Mense, 1996) becoming active during a contraction. Hypertonic saline is the commonly used model of muscle pain and it is known to excite a majority of A and C fibre afferents in the muscle (Kumazawa and Mizumura, 1977; Mense, 1996). Previous studies suggest the hypothesis that pain at the time of position sense testing may interfere with the perception of the position of the painful limb. There is evidence that stimulation of nociceptors may interfere with proprioception at the point of convergence of afferent inputs in the dorsal horn (Capra and Ro, 2000). One suggested mechanism is that a large proportion of free nerve endings are sensitised by peripheral release of pain modulating substances produced during the pain response. Neuroplastic changes in the integration of inputs from group III and IV afferents in the spinal cord may lead to abnormal drive of muscle spindles in the affected region and therefore abnormal sense of joint position (Hellstrom et al., 2000;

Johansson et al., 1993; Schaible and Grubb, 1993). Recently, a significant relationship between force-matching errors and pain levels was found (Proske et al., 2004; Proske et al., 2003). Matching torque levels with a sore muscle led to a pattern of errors similar to that seen after exercise; as soon as pain had set in, matching errors increased dramatically. When the indicator biceps was sore, the matching torques was significantly below the reference level and when the reference biceps was sore, the indicator arm matched with higher torque levels than given by the reference. There was a correlation between the size of the matching errors and the level of pain (Weerakkody et al., 2003b). It could be argued that the presence of pain simply distracted subjects from their matching task, leading to larger than normal errors. Perhaps subjects were simply favouring their sore muscles. However, factors such as distraction and unintentional sparing of the muscle during contraction would not have been expected to lead to such a close correlation between the size of the errors and the level of perceived pain. It was then concluded that muscle soreness can interfere with a subjects' ability to match forces, perhaps as a result of a reduced excitability of motor cortex (Proske et al., 2003). Likewise, it was found that heating the skin did interfere with matching performance in a manner similar to that seen following the saline injections (Proske et al., 2003; Weerakkody et al., 2003b). On the other hand, Bennell et al. (2005; 2003) revealed no association between proprioception and pain. Other studies also indicated that simple nociceptive stimulation does not induce a deficit in proprioception (Baker, Bennell, Stillman, Cowan, & Crossley, 2002; Sharma, Pai, Holtkamp, & Rymer, 1997). Matre, Arendt-Neilsen, & Knardahl (2002) found that pain did not affect ankle joint position sense but did affect ankle movement detection threshold. Yet, the relationship between pain/heat and proprioception is complex with human studies failing to find a clear link between the two.

### 2.3.4 Role of the central nervous system

The central nervous system (CNS) receives input from three main subsystems: the somatosensory system; the vestibular system; and the visual system (Tyldesley and Grieve, 1989). The somatosensory system provides information to three distinct levels of motor control: the spine, the brain stem, and the higher centers (basal ganglia and motor cortex) (Burgess, Wei, Clark, & Simon, 1982; Griffin et al., 2000; Lephart and Fu, 1995; Lephart and Henry, 1995; "Principles of Neural Science," 2000). The sensory receptors for proprioception are found in the skin, muscles, and joints as well as in ligaments and tendons and they all provide input to the CNS regarding tissue deformation. They also provide the CNS with knowledge of the shape, size and mass of body segments and allow the CNS to determine the orientation, position and velocity of our body and limbs during movement as well as muscle tension (Gandevia, 1996; Kerr and Marshall, 1995; McCloskey, 1978). It has been shown in animal (monkey) experiments that the primary somatosensory cortex (SI) is one of the cortical targets of the afferent inputs signalling limb movements. Neurons in area 3a (Huerta and Pons, 1990) and area 2 (Costanzo and Gardner, 1981) in nonhuman primates respond to passive or active limb movements by receiving the afferent inputs. Our understanding on how proprioception is used by the CNS is yet far from complete. Nevertheless, recent evidences are provided in support of the centrally generated sense of effort having the main contributory role, rather than muscle receptors, to limb position sense.

*2.3.4.1 Sense of effort*

There has been much debate about the origin of the proprioceptive senses and to what extent they are derived peripherally or centrally. The traditional view is that signals from muscle spindles provide us with our sense of limb position (Gandevia, 1996; McCloskey, 1978). However, the present-day view is that the sense of effort plays a major role in joint position sense (Allen and Proske, 2006; Walsh et al., 2006; Walsh, Hesse, Morgan, & Proske, 2004; Winter, Allen, & Proske, 2005) and is generated centrally as a result of interactions between sensory and motor cortex (Gandevia, 2001). The sense of effort has been referred to as deriving from a corollary discharge or efference copy of the motor command (Matthews, 1988; McCloskey, 1981). Whenever we carry out a voluntary contraction, it is postulated that a copy of the motor command reaching the motor cortex is sent to sensory areas to generate the effort sensation. For a review of this subject see McCloskey, Gandevia, Potter, & Colebatch (1983). When subjects hold their reference arm at the test angle, this would be signalled in part by the effort sensation accompanying the force generated in elbow flexors to support the weight of the arm against gravity. Thus, the effort required to maintain position of the arm against the force of gravity provides us with information about its location in space. Furthermore, in studies using force-matching tasks, it was suggested that subjects were using their sense of effort to match forces, not a peripherally derived sense of tension (Carson, Riek, & Shahbazpour, 2002; Weerakkody, Percival, Morgan, Gregory, & Proske, 2003a). A peripheral signal would have indicated the true level of tension and, therefore, would not have led to matching errors. Given that the sense of effort contribute to the position sense and sense of torque, a recent study by Allen and Proske (2006) has explored the possibility that the sense of effort could also contribute to movement sensation. It was found that exercising elbow flexors led to significant position

matching errors but subjects were still able to accurately carry out a movement tracking task. It was then concluded that the sense of effort did not contribute to movement sensation and that this sense was generated entirely by signals from muscle spindles. It is presumable that whenever we carry out movements against the force of gravity, we are provided with effort cues. This proposition would help account for the proprioceptive disturbances known to occur in high-gravity (Lackner and Graybiel, 1981) or in low-gravity environments hence the difficulties encountered by astronauts in outer space carrying out motor tasks in the absence of vision (Young et al., 1993). Up to this point, because most of the studies had involved forearm position matching in the vertical plane, it was assumed that effort only played a role in position sense under circumstances where the gravity vector was acting (Walsh et al., 2004). However, the experiment of Walsh et al. (2006) has explored the possibility that a sense of effort may also contribute to position sense when the force of gravity is not able to provide positional cues. In summary, it is suggested that muscle spindles contribute, in part, to position sense and an additional cue is provided by the amount of effort required to maintain the position. The current working hypothesis is that as soon as spindles are activated through the fusimotor system, they no longer contribute to position sense. At the same time, the effort signal generated by the motor command provides additional positional information (Winter et al., 2005). It will be a challenge for the future to define more precisely the central site of origin of the effort sensation and how it combines with afferent information from the periphery to give us our proprioception sense.

### 2.3.5 Decrement of position sense performance induced by different muscle contraction types

A restricted number of studies have used an isometric type exercise protocol to measure its effect on proprioception. Nevertheless, the results are in favour with a deterioration of the sense of velocity (Jaric, Radovanovic, Milanovic, Ljubisavljevic, & Anastasijevic, 1997) and position sense (Forestier, Teasdale, & Nougier, 2002a; Jaric et al., 1999; Winter et al., 2005) due to an increase in fusimotor activity with muscle exercise. In general, results demonstrate that the indicator limb undershoots the target position (Forestier et al., 2002a; Jaric et al., 1999). However, other findings reported that progressive isometric exercise does not systematically alter the joint position sense (McCloskey, 1973; Sharpe and Miles, 1993).

Although only few investigations have used a concentric protocol to test position sense at the elbow after exercise (Allen and Proske, 2006; Brockett, Warren, Gregory, Morgan, & Proske, 1997; Walsh et al., 2004), these have been of a major importance in changing the traditionally view of the origin of proprioception, the muscle spindles. Originally, it was speculated that if ordinary muscle fibres were damaged by eccentric exercise, it was conceivable that the intrafusal fibres of muscle spindle, too, could become damaged. This would lead to a disturbance of spindle function and consequently errors in position sense. Such observations would not normally be expected by other types of muscle contraction since muscle damage is only induced after eccentric contractions. However, it has recently been shown that a drop in maximum voluntary force after a concentric exercise was also accompanied by a significant position-matching error (Allen and Proske, 2006). Likewise,

Walsh et al. (2004) reported a correlation between matching error and MVC decline with concentric exercise. Given that eccentric and concentric exercises produced matching errors, it implied that the muscle damage associated with eccentric exercise was not a contributing factor to proprioception, other than by its effect on muscle force. Therefore, what is responsible for the position-matching errors after exercise? It was then argued that in a situation where a muscle is exercised while being required to maintain a certain level of force, centrally driven activation rates of motoneurons increase to maintain the level of force, and in doing so lead to a perceived increase in effort. It was therefore suggested that the effort required to maintain the arm against the force of gravity provides a positional cue (Allen and Proske, 2006; Semmler and Miles, 2006; Walsh et al., 2004). Many studies revealed that the errors were in the direction predicted by the effort:position matching hypothesis, that is, when the exercised arm was the reference, the indicator adopted a more extended position immediately after the concentric exercise. The explanation is that the reference effort signal was larger than normal, because, as a result of exercise, the tension generated by a given effort was less. Because of the larger reference effort, the unexercised matching arm adopted a position where more effort would be required to maintain its position. When the unexercised arm was the reference, the indicator adopted a more flexed position. Again, the interpretation is that the exercise had disturbed the relationship between force and effort so that more effort was now required to maintain position of the arm at a given forearm angle. On the other hand, the results from Sterner et al. (1998) did not demonstrate a detrimental effect of muscle exercise on the ability to passively reproduce a target angle. The difference in exercise protocol may have accounted for this opposing finding. Furthermore, contrary to the sense of position, the sense of movement does not seem to be affected by concentric exercise (Allen and Proske, 2006). The data supports the

view that muscle spindles would be responsible for the sense of movement, and that this sense would not be prone to the disturbance from exercise.

It is a common experience to feel clumsy and awkward after a period of intense exercise and to have difficulty in performing finely skilled movements. Such anecdotal observations have led to the suggestion that exercise, particularly eccentric exercise, can disturb proprioception. Given that the sense of position is believed to be provided by afferent signals coming from muscle spindles, investigators considered the possibility that eccentric exercise not only damaged the ordinary muscle fibres, but also disturbed the function of muscle receptors explaining the disturbance to human proprioception (Proske and Allen, 2005). However, it has recently been shown that eccentric contractions, which lead to clear evidence of muscle damage, do not appear to have any effect on spindles (Gregory et al., 2004). The same seems to apply also to tendon organs (Gregory et al., 2002). Nevertheless, many studies demonstrated clearly that position sense acuity at the elbow joint is decreased after a period of eccentric exercise (Brockett et al., 1997; McCloskey, Cross, Honner, & Potter, 1983; Saxton et al., 1995; Walsh et al., 2006; Walsh et al., 2004). A similar decline in position sense acuity is usually observed for both males and females, although females generally displayed an overall poorer acuity than males (Pederson, Lonn, Hellstrom, Djupsjobacka, & Johansson, 1999). The sizes of the matching errors correlated with the fall in force, suggesting that the drop in MVC was responsible for matching errors. However, other studies for the knee (Marks and Quinney, 1993), elbow (Sharpe and Miles, 1993) and shoulder joint (Sterner et al., 1998) revealed no such effects using other types of muscle contractions. The effect of exercise on position sense can differ as a function of which muscle is exercised. For example, Jaric et al. (1999) showed that exercising the agonist

muscles is associated with final error position, while exercising the antagonist muscles has no effect. In addition, studies demonstrated that eccentric exercise impairs joint position sensibility while having no apparent effect on kinaesthesia (Skinner, Wyatt, Hodgdon, Conard, & Barrack, 1986). Similarly, vibration at low frequency can induce errors of position without inducing illusions of movement (McCloskey, 1973). It therefore suggests that position sense and sense of movement have separate lines of information. In the joint position-matching test performed without vision, the exercised indicator arm was either flexed more (Saxton et al., 1995) or less (Brockett et al., 1997) as compared to the non-exercised reference arm. These authors explained the matching errors by the exercise-induced changes in muscle afferent discharge. Recent studies at the elbow joint have found the same results, which are, when the exercised arm was the indicator, it tended to adopt a more flexed position than the unexercised reference and if elbow flexors in the reference arm were exercised, the indicator arm adopted a more extended position to match it (Gregory et al., 2004; Walsh et al., 2006; Walsh et al., 2004). The currently accepted interpretation of the observed disturbance in proprioception after eccentric exercise is not the result of dysfunction of muscle sense organs. Rather, the observations lead to conclude that, in the unsupported arm position-matching task, the sense of effort plays a major role (Walsh et al., 2006; Walsh et al., 2004). During active placement of our limbs, we use, as a positional cue, the sense of effort required to maintain limb position against the force of gravity. To hold the arm at a set angle against the force of gravity requires a certain amount of effort. The perceived effort increases significantly if arm muscles are damaged from eccentric exercise. If subjects match efforts to align their arms they will place the exercised arm more nearly vertically where less force is required to support it leading to position matching errors. Adoption of a more vertical position requires less effort, for two reasons.

First, the moment of the force of gravity on the arm is less. Second, a more vertical position is closer to the elbow flexors' optimum length for active tension. Thus, in matching efforts, subjects placed the exercised arm in a position where the force generated by its elbow flexors was sufficient to bear its weight. Another consistent trend was the finding that at small forearm angles, that is, with the forearm more nearly horizontal, errors tended to be larger than when the arm was closer to the vertical. For example, position errors at a test angle of 30° from the horizontal tended to be larger than at 60° of flexion (Walsh et al., 2004). Here again it was speculated that, as the arm was placed more nearly horizontally, a larger vector of the force of gravity would be acting on it. Therefore, we can conclude that a period of eccentric exercise leads to a change in the effort:force relationship and this produces the position matching errors in the vertical plane, where gravity cue is available. It has been observed that when subjects carry out a forearm position matching task in the horizontal plane or by mean of a support, they become more erratic in their performance (Walsh et al., 2006; Walsh et al., 2004). It was proposed that these effects are due to withdrawal of a positional cue normally available to subjects when matching the unsupported arms in the vertical plane, the force of gravity, and that position sense with this posture arises primarily from muscle spindles. The current working hypothesis is that, during development, we learn to routinely associate effort sensations with movements as signalled by proprioceptive feedback from the moving muscles. Eventually we begin to use the sense of effort as a proprioceptive signal in its own right.

Similarly to position sense, there are large force-matching errors after a series of eccentric contractions (Brockett et al., 1997; Proske et al., 2004; Proske et al., 2003; Saxton et al., 1995; Weerakkody et al., 2003a). These can be due to three possible causes. One, the

greater effort required to achieve a given force, as a result of exercise and muscle damage. Secondly, there is an increase in central neural drive, presumably accompanied by an increase in effort, which lasts for up to 48h. Finally, there is the influence of delayed-onset muscle soreness (DOMS) which does not begin to exert a significant effect until 24h after the exercise and which lasts for at least four days suggesting that here short-term effects, such as accumulation of metabolites, is unlikely to be responsible. Another hypothesis considered for these findings is that contractures in the muscle fibres damaged by the eccentric contractions are responsible for the reduced elbow angle and could activate some tendon organs. If the tendon organs output of the muscle rises because of the higher resting tension, this may lead to the perception of a higher level of force in the muscle than was actually generated and that would produce the observed tension mismatch (Brockett et al., 1997). However, Proske (2005) mentioned in his review that force matching errors after eccentric exercise were not the result of a disturbance of function in tendon organs. The explanation that best fits is that subjects were matching forces not by matching the torque level but by matching the amount of effort required to achieve a given level of force (Carson et al., 2002; Proske et al., 2004; Proske et al., 2003; Weerakkody et al., 2003a), not a peripherally derived sense of tension. Proske et al. (2004) suggested that sense of effort is the dominant sense in a force matching task. Thus, for example, if the contracting muscles on the reference side are weakened by the exercise, the target tension is achieved only by an unusually large motor command and effort. When this is perceived, it influences the matching process as indicated by a larger-than-normal matching contraction made with the unaffected indicator muscles on the opposite side (McCloskey, Ebeling, & Goodwin, 1974).

In summary, the most important evidence in support of a sense of effort contributing to position sense is that after exercise, subjects make position matching errors. A role for muscle spindles in kinaesthesia still remains undisputed. But, because of the fusimotor innervation of spindles, there are difficulties in assigning to them a major role in static position sense. Recent studies have shown that since the damaged muscle produced less force, it required more effort to maintain a given arm position against the force of gravity leading to propose that subjects were matching their efforts to align their arms. So the weaker elbow flexors of the exercised arm would match the effort required to hold the unexercised arm by adopting a more vertical position where the force of gravity was less. That, in turn, would produce the matching errors. An important consequence of this assertion is that all forms of exercise that produced a decrease in muscle performance should lead to position-matching errors, no just eccentric exercise. The current working hypothesis is that when the arm is moved into position, the movement information comes largely from muscle spindles, with additional contributions from skin and joint receptors. Once the arm is in position, the effort required to keep it there provides detailed positional information (Walsh et al., 2004).

## 2.4 SPORT REHABILITATION

### 2.4.1 Role of proprioception in rehabilitation and prevention of injury

Conscious proprioception is essential for proper joint function in sports and occupational tasks. Unfortunately, injuries are very common in sport and have been found to have a detrimental effect on proprioception through the damage of mechanoreceptors in ligaments.

Statistically, ankle sprains are the most frequent type of sports injuries, damaging predominantly (85-90%) the lateral ankle ligaments and corresponding structures. As a result, chronic functional instability may develop (Hertel, 2000; Karlsson, Bergsten, Lansinger, & Peterson, 1988; Peters, Trevino, & Renstrom, 1991) leading to a loss of proprioception that may have a profound effect on neuromuscular control and activities of daily living. The proper management of athletic-related injuries and orthopaedic lesions can be complex in the sports medicine setting. One of the most challenging aspects to the clinician is to understand the role of proprioceptively mediated neuromuscular control after joint injury and its restoration through rehabilitation. This understanding, coupled with a base of knowledge regarding the current research on proprioception, is necessary for sports medicine practitioners to optimize treatment programs for athletes. Previous studies have shown that women participating in jumping and cutting sports have a fourfold to sixfold higher incidence of knee injury than males (Arendt and Dick, 1995; Hewett, Stroupe, Nance, & Noyes, 1996; Zelisko, Noble, & Porter, 1982). One possibility for these differences with respect to knee injuries may be attributable to deficits in proprioception and neuromuscular control in female athletes (Hewett, Paterno, & Noyes, 1999). These observations have to be interpreted with caution, but if generalized they might imply that women are more prone to sport injuries than men. Interestingly, some studies lend some support to such speculations, as they suggest that women are more susceptible to sport injuries than men (Arendt and Dick, 1995; de Loes, 1995; Gray et al., 1985; Jones, Bovee, Harris, & Cowan, 1993). Therefore, rehabilitation and preventive programs are crucial in both genders but especially in women.

Glencross and Thornton (1981) were the first group to quantify ankle proprioception by using a standard goniometer. They found greater errors in active repositioning of plantar flexion in the injured ankle, as compared with the uninjured ankle. Freeman, Dean, & Hanham (1965) postulated that during an ankle sprain, the nerve fibres of the mechanoreceptors located at the torn ligament could also be damaged. As a result, proprioceptive inputs from the ankle joint could be reduced. These authors further suggested that a reduction in the ankle sprain could be a predisposing factor for postural instability. Later studies indeed demonstrated that both joint position and movement sense were found to be reduced in players with sprained ankles when compared with healthy players (Forkin, Koczur, Battle, & Newton, 1996; Garn and Newton, 1988; Konradsen, Olesen, & Hansen, 1998; Lentell et al., 1995). Similarly, it was reported that proprioception was significantly worse in the injured joint than in the noninjured shoulder (Smith and Brunolli, 1989). Decreased position sense and kinaesthesia have also been demonstrated in the ACL-deficient knee (Barrack, Skinner, & Buckley, 1989; Barrett, 1991; Beard, Dodd, Trundle, & Simpson, 1994a; Beard, Kyberd, Fergusson, & Dodd, 1993; Beard, Kyberd, O'Connor, Fergusson, & Dodd, 1994b; Beynnon et al., 1999; Borsa, Lephart, Irrgang, Safran, & Fu, 1997; Corrigan, Cashman, & Brady, 1992; Fischer-Rasmussen and Jensen, 2000; Fremerey et al., 2000; Friden, Roberts, Zatterstrom, Lindstrand, & Moritz, 1996, 1999; Friden, Zatterstrom, Lindstrand, & Moritz, 1989; Lattanzio and Petrella, 1998; Lephart, Kocher, Fu, Borsa, & Harner, 1992; MacDonald, Hedden, Pacin, & Sutherland, 1996; Mizuta, Shiraishi, Kubota, Kai, & Takagi, 1992; Ochi, Iwasa, Uchio, Adachi, & Sumen, 1999; Pap, Machner, Nebelung, & Awiszus, 1999; Roberts, Friden, Stomberg, Lindstrand, & Moritz, 2000), the ACL-reconstructed knee (Barrett, 1991; Co, Skinner, & Cannon, 1993; Friden, Roberts, Zatterstrom, Lindstrand, & Moritz, 1997; Ochi et al., 1999),

and the PCL-deficient knee (Clark, MacDonald, & Sutherland, 1996; *Proprioception and neuromuscular control in joint stability*, 2000; Safran et al., 1999). In the fibres of the ACL, the presence of specific proprioceptive mechanoreceptors has been shown (Friden et al., 1989; Johansson, Sjolander, & Sojka, 1991; Schultz, Miller, Kerr, & Micheli, 1984; Schutte, Dabezies, Zimny, & Happel, 1987). It was hypothesized that ACL disruption and meniscal injury damage articular structures containing mechanoreceptors and, therefore, result in deficits of kinaesthesia and joint position sense (Lephart, Pincivero, & Rozzi, 1998). However, when both sense of movement and joint position sense were measured in the same subjects, there was no relationship between the sensations. Sense of movement was impaired in ACL deficient knees compared with the healthy control (Friden et al., 1997) while joint position sense was unaffected even in the presence of instability (Roberts, Friden, Zatterstrom, Lindstrand, & Moritz, 1999). Thus, in the ACL deficient knee, there appears to have a deficit in sense of movement but not joint position sense. These findings support McCloskey's hypothesis that signals are processed differently for position sense and sense of movement. Moreover, other authors have failed to find a significant difference when comparing patients to external controls or the uninjured limb (Friden, Roberts, Movin, & Wredmark, 1998; Pap et al., 1999). Proprioceptive deficits also have been documented in the non-involved limb (Corrigan et al., 1992; Reider et al., 2003). The results of Hewett et al. (1999) are consistent with this finding and raised an interesting question regarding the use of the contralateral limb as a control during proprioceptive testing. These results indicated that clinicians should not use the contralateral limb as a control when assessing proprioceptive parameters, because deficits similar to the involved limb are seen on the non-involved side.

Theoretically, operative techniques can restore proprioception directly through reinnervation of damaged structures or indirectly through restoration of appropriate tension in capsuloligamentous structures. Indeed, the return to preinjury value of proprioceptive function after ACL reconstruction has been reported (Barrack et al., 1989; Barrett, 1991; Co et al., 1993; Harrison, Duenkel, Dunlop, & Russell, 1994; Swanik, Lephart, & Rubash, 2004). However, several studies have shown that proprioceptive deficits that exist in ACL-deficient knees can only be partially restored by surgical reconstruction (Barrack et al., 1989; Barrett, 1991; Beard, Dodd, & Simpson, 1996; Co, Skinner, & Cannon, 1991; Lephart, Warner, Borsa, & Fu, 1994) and the improved scores following total knee reconstruction could be attributed to the reduction in pain, swelling and deformity (Swanik et al., 2004). Barrack, Skinner, Cook, & Haddad (1983) and Barrett, Cobb, & Bentley (1991) also investigated the effect of total knee replacement on knee proprioception. The insertion of a total knee replacement resulted in the removal of most joint receptors located in the knee. They found in both studies that there was no significant difference in knee proprioception between the operated and non-operated knee. Similarly, no significant decrease in joint proprioception was observed after joint replacement surgery in the hip (Grigg, Finerman, & Riley, 1973). A study by MacDonald et al. (1996) using threshold to perception of passive motion, also found no improvement in proprioceptive function after the joint reconstruction. It was reported that after ACL reconstruction, patients continue to have deficits in proprioception and neuromuscular joint control at least 6 months to 1 year postoperatively and in some cases beyond 1 year when compared with a control group (Hewett, Paterno, & Myer, 2002). In addition, Lephart et al. (1992) found significantly worse joint kinaesthesia than in the contralateral healthy joint after ACL reconstruction.

Therefore, controversy exits regarding the return of proprioceptive function of the joint reconstruction and further research on this topic is required.

Although evidence regarding the effects of rehabilitation on proprioception in regaining neuromuscular control has yet to be verified, it is suggested that proprioceptive exercises may have a beneficial role on the sensory afferent and efferent motor responses (Lephart et al., 1998). Regaining neuromuscular control after injury or surgery is a necessary prerequisite for athletes wishing to return to competition. Rehabilitation of athletics injuries requires the prescription of sport-specific exercise and activities that challenge the recovering tendons, ligaments, bones, and muscle fibres without overstressing them. The goal of rehabilitation is to return an athlete to the same or higher level of competition as before the injury. Methods to improve proprioception after injury or surgery could improve function and decrease the risk of reinjury. As mentioned earlier, afferent input is altered after joint injury and may remain altered after joint reconstruction. Proprioceptive rehabilitation, however, may allow the patient to retrain altered afferent pathways resulting in enhanced sensation of joint movement. Therefore, proprioceptive training has become an integral aspect of functional rehabilitation (Lephart and Borsa, 1993). A theoretical rationale exists that proprioceptive and balance training may improve the nervous system's ability to synchronize muscular activity around a joint improving dynamic joint stability, thereby improving proprioception in people following injury or surgery (Cooper, Taylor, & Feller, 2005). Proprioceptive training and rehabilitation techniques are used extensively to prevent injury and also to provide optimal functional restoration during rehabilitation. Caraffa, Cerulli, Projetti, Aisa, & Rizzo (1996) reported that the frequency of injuries in the proprioceptively trained group showed a sevenfold reduction over the control group. These

results indicate that a program of proprioceptive training can reduce the number of ACL injuries in soccer. However, some authors reported limited improvements in joint position sense, muscle strength, and hop testing following proprioceptive and balance exercise when compared with traditional strengthening exercises in ACL-deficient populations (Ageberg, Zatterstrom, Moritz, & Friden, 2001; Beard et al., 1994a; Fitzgerald, Axe, & Snyder-Mackler, 2000; Zatterstrom, Friden, Lindstrand, & Moritz, 2000). In summary, there are numerous studies supporting the positive effect of the proprioceptive training on balance (Bernier and Perrin, 1998; Gauffin, Tropp, & Odenrick, 1988; Hoffman and Payne, 1995; Kynsburg, Halasi, Tallay, & Berkes, 2006; Lephart, Pincivero, Giraldo, & Fu, 1997; Matsusaka, Yokoyama, Tsurusaki, Inokuchi, & Okita, 2001; Paterno, Myer, Ford, & Hewett, 2004; Rozzi, Lephart, Sterner, & Kuligowski, 1999; Tropp, Askling, & Gillquist, 1985a; Tropp, Ekstrand, & Gillquist, 1984a, 1984b; Tropp, Odenrick, & Gillquist, 1985b), but little is known about its effect on joint position sense.

Rehabilitation programs should be designed to include a proprioceptive component that addresses the following three level of motor control: spinal reflexes, cognitive programming, and brainstem activity. Such a program is highly recommended to promote dynamic joint and functional stability. Proprioceptive and balance exercises appear to be a safe form of rehabilitation, with no study reporting increased passive joint laxity or decrease in strength when compared with standard rehabilitation programs. As a result, it has been suggested that proprioceptive training should become standard in preseason training as well as during the actual playing season. Functional rehabilitation is an integral part of any rehabilitation program. It incorporates not only the traditional elements of physical therapy, such as strength and flexibility, but also activities to enhance agility, proprioception, and

neuromuscular control. Agility and proprioceptive training are incorporated to restore neuromuscular mechanism responsible for joint kinematics, enabling the individual to return to preinjury levels of activity while also reducing the risk of reinjury (Lephart and Borsa, 1993; Markey, 1991). Once proprioceptive deficiencies have been identified, subsequent to either an acute or chronic musculoskeletal injury, a rehabilitation programme should be developed and implemented. Initially, patients concentrate on the rehabilitation task being performed in order to facilitate and maximise sensory input. As the patient progresses, the activities incorporate cognitive or psychomotor aspects, which ultimately aid in converting conscious joint stabilization and control to unconscious motor programming (Lephart and Fu, 1995; Voight and Cook, 1996). To maximally restore proprioception and neuromuscular control, it is recommended that the following progression of activities be conducted to allow the return of an athlete to functional levels: 1) joint position sense and kinaesthesia, 2) dynamic joint stabilization, 3) reactive neuromuscular control, and 4) functionally specific activities. Such a progression allows the rehabilitation program to address the integration of spinal reflex, cognitive, and brainstem pathways to focus on joint stabilization (Lephart and Henry, 1996). However, it is important to remember that in the acute phase, the focus should be on controlling inflammation, re-establishing full range of motion and weight bearing strength. Once pain-free range of motion and weight bearing have been established, balance-training exercises can be incorporated to normalize neuromuscular control. Once the athlete has reached the functional stage of rehabilitation, the objectives of proprioception training are to refine joint position awareness which initiates muscle reflex stabilization to prevent reinjury. Moreover, there is evidence that additional improvement in quadriceps and hamstring muscle strength can be obtained using proprioceptive and balance training for both ACL-deficient (Zatterstrom et al., 2000) and

ACL-reconstructed individuals when compared with a standard rehabilitation program (Liu-Ambrose, Taunton, MacIntyre, McConkey, & Khan, 2003). This is a contrary finding since, according to the guidelines of the American College of Sports Medicine (2002), proprioceptive and balance training would not be expected to provide sufficient stimulus to increase muscle strength. It could be speculated that proprioceptive and balanced training might enhance neuromotor recruitment, this enhancing muscle strength.

A rehabilitation program to address proprioceptive deficits should integrate all subsystems of proprioception in addition to the 3 levels of motor control. To enhance motor function at the brainstem level, balance and postural maintenance activities should be employed. These equilibrium-promoting activities should be performed both with and without visual system input, be implemented following a standardised progression, and be specific to the type of activities and skills the patient will require. Once implemented, these activities should follow the progression from static balance activities to dynamic skill activities. For static balance activities, patients should progress from bilateral to unilateral activities, from activities with eyes open to those with eyes closed and those performed on a stable surface to those performed on an unstable surface (Guskiewicz and Perrin, 1996; Lephart and Fu, 1995; Lephart et al., 1997; Nyland, Brosky, Currier, Nitz, & Caborn, 1994; Voight and Cook, 1996). To stimulate reflex joint stabilization, which emanates from the spinal cord and improves the neuromuscular mechanism, activities should focus on sudden alterations in joint positioning that necessitate reflex muscular stabilization. In the first week of phase I of rehabilitation, simple weight shifting can initiate this process. Progression to weight bearing can be taught with the aid of a bathroom scale. The proprioceptive exercises concentrate on balance and dynamic joint stabilization that will permit the athlete to

progress to directional changes in the next phase of the rehabilitation program (Lephart and Henry, 1995). Proprioception training continues in phase II of the program with the initiation of balance board (Heckmann, Noyes, & Barber-Westin, 2000). The proprioceptive activities in this phase involve more dynamic movements and encourage changing directions rapidly, landing, and performing balancing manoeuvres while involved in sports-specific activities. As the patient achieves full weight bearing without pain, proprioceptive training is initiated for the recovery of balance and postural control. The simplest device for proprioceptive training is the wobble board, a small discoid platform attached to a hemispheric base (Hintermann, 1999). The exercise can be progressed by having the patient use different-sized hemispheres and by varying visual input. Moreover, proprioception challenge can be improved by uneven surfaces (wobble board, foam cushions), crossing the arms, closing the eyes, and external forces (therapist, resistance tubing). All exercises should be performed at various positions throughout the full range of motion because of the difference in the afferent response that has been observed at different joint positions (Lephart et al., 1997). Proprioception training begins with such simple tasks as balance training and joint repositioning and becomes increasingly more difficult as the patient progresses. A common mistake when performing proprioception and balance exercises is the lack of variability in speed and intensity.

The principle of specificity must be incorporated into the functional rehabilitation program. The specificity of the training principle involves matching the neuromuscular and physiologic demands during rehabilitation with those that the athlete will be returning to. Specificity during functional rehabilitation restores reflex muscle activity (Lephart and Henry, 1995) and help to fine tune the afferent-efferent arcs. Exercises should include

consciously mediated movement sequences, sudden alterations of joint position to initiate reflex subconscious muscle contraction (Hewett et al., 1996; Hoffman and Payne, 1995), and specific drills that are similar to movement patterns used in the sport. Closed kinetic chain exercises are more effective for challenging the dynamic and reflexive aspects of proprioception for the lower extremities. The lower extremities are used in a closed chain manner during sports and daily life activities, and training and testing in this fashion facilitates the proper neuromuscular engrams (Laskowski, Newcomer-Aney, & Smith, 2000). Plyometric exercises also fit nicely into the specificity of training principle. These exercises should be implemented into the advanced phase II of the functional rehabilitation program to begin development of explosive contractile strength (Lephart and Borsa, 1993; Markey, 1991). Swanik et al. (2002) suggested that plyometric activities facilitate neural adaptations that enhance proprioception and kinaesthesia. In fact, the plyometric group improved significantly more than the control group in the position-matching and in the detection of passive motion tests. These differences suggest that peripheral and central neural adaptations were induced by plyometric training, resulting in improved joint position sense and detection of joint motion. Rehabilitation is said to be incomplete unless manoeuvres specific to the sport and the athlete's position can be performed maximally and without pain or loss of function. Testing of these skills before return to competition should always be performed.

Proprioceptive deficits may predispose an athlete to reinjury through decrements in the neuromuscular pathways as a result of a decrease in sensory input from joint receptors. This would then lead to abnormal body positioning and diminished postural reflex responses (Gross, 1987). Tropp et al. (1985) were the first to examine the effects of an ankle disk

training program in 65 male soccer players with previous ankle sprains. Results from their study indicated an 80% decrease in the frequency of repeat sprains over a 6-month period in the exercise group when compared with the non-exercise group. They concluded that completion of the progressive neuromuscular control rehabilitation program minimizes the risk of reinjury and promotes a greater change of successful return to competition (Lephart et al., 1997). Moreover, proprioception has been shown to play an important role not only in rehabilitation, but also in the prevention of injuries (Zatterstrom, Friden, Lindstrand, & Moritz, 1992). Once the final stage of rehabilitation is reached, regaining joint sense awareness to initiate muscular reflex stabilization to prevent reinjury should be the primary objective (Lephart et al., 1997). According to recent studies, proper proprioceptive sensorimotor control is the key to the prevention of injuries and chronic functional instability (Konradsen, Beynnon, & Renstrom, 2000a, 2000b). Caraffa et al. (1996) showed in a prospective controlled study that preventive neuromuscular training of high level male soccer players significantly decreased the incidence of ACL injury. After a progressive five-phase training program on balance board, injury incidence decreased more than sevenfold in these male athletes. Likewise, Hewett et al. (2002) reported that female athletes trained with the prevention program were three to four times less likely to suffer from serious knee injuries. The results observed with this program justify the alteration of current protocols for preparing females for participation in high-risk sports. Such training, if effectively implemented on a widespread basis, could help to significantly decrease the number of athletes injured each year. Therefore, noninjured athletes can benefit from incorporation of proprioceptive exercises into their training program and the effectiveness of the rehabilitation/prevention program is crucial since it often determines the success of future function and athletic performance.

### 2.4.2 Effect of athletic training background and bracing

Although a restricted number of studies exist on the topic, results have demonstrated that extensive athletic training influences knee proprioception (Barrack, Skinner, Brunet, & Cook, 1983a, 1984a; Skinner et al., 1986). Knee kinaesthesia has been shown to be significantly better in trained collegiate gymnasts compared to age-matched controls, suggesting that extensive training has a positive effect on some measures of proprioception (Lephart, Conners, Fu, Irrgang, & Borsa, 1991; Lephart, Giraldo, Borsa, & Fu, 1996). Enhanced kinaesthesia was also found in trained dancers (Barrack et al., 1984a) and in the dominant radiocarpal joint of elite table tennis players (Jerosch and Prymka, 1996b). According to these findings, highly trained athletes seem to possess enhanced neurosensory pathways. Such pathways appear to improve proprioception through enhanced central and peripheral neural mechanisms and may be developed as a result of long-term athletic training. However, another possible explanation is that athletes may have genetically determined superior kinaesthetic responses. Barrack et al. (1983) studied highly conditioned professional ballet dancers to determine the effect of training on their proprioception. Paradoxically, they found that these dancers performed significantly better in their threshold to their detection of movement and significantly worse in their ability to reproduce a joint position than an age-matched control group. The authors believed that athletic training can affect joint proprioception and that possibly these two tests for proprioception depend on different neural mechanisms (Barrack et al., 1984a; Barrack, Skinner, & Cook, 1984b; Carpenter, Blasier, & Pellizzon, 1998). They also believed that the hypermobility of joints of ballet dancers is due to their training, which results in stretched joint capsules and ligaments and therefore impair their proprioception. Likewise, other investigators have

shown that subjects with increased laxity have poorer proprioception (Barrack et al., 1983a; Barrack et al., 1984b; Rozzi, Lephart, Gear, & Fu, 1999). Nevertheless, it is still unknown whether athletic training can actually change a person's proprioception sensitivity or change the way position sense is affected by muscle fatigue effects. The effects of training on proprioception need to be further studied in controlled trials.

Adjunctive use of bracing or taping during the rehabilitation process is common and in some cases may offer additional protection for the athlete. Some studies reported that knee angle reproduction was improved with elastic bandage in normal individuals, ACL deficient, and after posttraumatic pattela dislocations (Jerosch and Prymka, 1996a; Lattanzio, Petrella, Sproule, & Fowler, 1997). Part of this beneficial effect is thought to be mediated by improved proprioception from the injured area, favourably influencing protective neuromuscular arcs. The effect of elastic bandages on knee proprioception demonstrated significant improvements immediately after bandage application and after one hour of bandage wear (Barrett, 1991; Lephart et al., 1992). Loss of benefit was documented after removal of the bandage (Perlau, Frank, & Fick, 1995). In the ankle joint, different studies have also proved the positive effect of bracing on the proprioceptive ability (Baier and Hopf, 1998; Jerosch, Hoffstetter, Bork, & Bischof, 1995; Konradsen, Ravn, & Sorensen, 1993; Laskowski et al., 2000; Lephart and Fu, 1995; Lephart et al., 1997). On the other hand, there are studies which demonstrated no positive influence in the ACL-reconstructed knee (Jerosch and Prymka, 1996a) or in patients suffering from a medial lesion of the meniscus (Jerosch, Prymka, & Castro, 1996). Still, the use of taping and bracing causes minimal to no performance decrements and seems to have proprioceptive, mechanical, and injury-protection benefits (Bocchinfuso, Sitler, & Kimura, 1994;

Macpherson, Sitler, Kimura, & Horodyski, 1995; Pienkowski, McMorrow, Shapiro, Caborn, & Stayton, 1995).

## 2.5 FUTURE DIRECTIONS

Although many years of research working on the origin and mechanisms of proprioceptive afferents have been completed, the role of muscle and joint receptors are still unclear. Given that the sense of effort is gaining popularity as a factor involved in position sense, mechanisms are still vague and indefinite. It would be appealing to develop testing protocols where subjects are not influenced by the sense of gravity, such as in the water or space environments. A lot of work still needs to be done on that topic. First and foremost, it is primordial to agree on the major physiological mechanisms involved in position sense to facilitate testing, rehabilitation and training of proprioception. Afterwards, it would be interesting to conceive a portable instrument to assess, diagnose, or pre-screen for functional instability in patients and athletes. Moreover, more studies should be done to clarify why females appear to have a poorer performance than males when tested for positional acuity. Is there a physiological, hormonal, or psychological aspect that could explain such a difference?

It seems clear that proprioceptive deficits exist after an injury. Nevertheless, further research to elucidate how these deficits can be remediated or compensated for to improve function, prevent reinjury, and restore proprioception by surgical procedure, is needed. Moreover, does the incidence of sport related injuries correlate with decreased proprioception, or does injury lead to a generalized decrease in proprioception? It is also

apparent from the literature that a large number of studies have focused on favourable aspects of proprioceptive treatment in rehabilitation after injuries and joint reconstruction. However, little is known about the effectiveness of proprioceptive training as a preventive tool for injuries. Can a preventive proprioception program really help to avoid injuries in athletic setting? This question is still unanswerable at the moment and should be of an interest for future studies.

Another fascinating area for future research would be to measure proprioception in the athletic setting to determine whether proprioception impairment is related to sport performance and whether proprioception is enhanced with years of athletic training. Only few and unconvincing studies have been done on the latter. Similarly, it would be fascinating to verify whether proprioception is improved with intense weight training, as it is the case with bodybuilders. In addition, the literature is still not persuasive whether proprioception is trainable or it is a gift from life. Can everyone benefit from a proprioceptive training program or only those who are naturally underprivileged? Also, how traditional weight training programs can be changed to incorporate a proprioception training knowing that it is negatively affected by dynamic muscle contractions during exercise? Although researchers are starting to understand what proprioception is, a lot of unanswered questions still remain and accordingly future studies are indispensable.

# CHAPTER 3 MANUSCRIPT

# WHICH TYPE OF REPETITIVE MUSCLE CONTRACTIONS INDUCE A GREATER ACUTE IMPAIRMENT OF POSITION SENSE?

Sylvie Fortier[1]
Normand Teasdale[2]
David Behm[1]
and
Fabien A. Basset[1]

[1]School of Human Kinetics and Recreation
Memorial University of Newfoundland
St. John's, NL
Canada A1C 5S7

[2]Groupe de Recherche en Analyse du Mouvement et Ergonomie
Division de Kinésiologie
Département de Médecine Sociale et Préventive
Faculté de Médecine
Université Laval, Québec (Qc)
Canada G1K 7P4

✉
Fabien A. Basset, Ph.D.
School of Human Kinetics and Recreation
Memorial University of Newfoundland
St. John's, NL
Canada A1C 5S7
Telephone: (709) 737-6132
Fax: (709) 737-3979
E-mail: fbasset@mun.ca

Running head: **Muscle contraction and position sense**

## 3.1 INTRODUCTION

A complex combination of many different signal sources, such as those from the tendon organs, muscle spindles, joint receptors, and cutaneous receptors, result in very sensitive and unambiguous sensations about our movement and limb position in space (McCloskey, 1978). In fact, in the absence of visual input, we have an accurate sense of position implying that we know the position of our limbs at any time during a movement. Attaining the aimed final position of either a limb or a particular end-point of the locomotor apparatus is an important motor task of our everyday behaviour; it can be crucial for success in various professional or sporting activities. Indeed, it has been suggested that muscle fatigue could predispose a joint to injury and thus an eventual decrease in athletic performance (Skinner et al., 1986). Proprioception has also been a topic of interest in sport rehabilitation because injuries have been found to have a detrimental effect on proprioception through the damage of mechanoreceptors in ligaments.

Fatigue studies have demonstrated that changes induced by muscle fatigue may well be influenced by whether the contraction is static (Bigland-Ritchie, Johansson, Lippold, & Woods, 1983) or dynamic (Colliander, Dudley, & Tesch, 1988). Likewise, it is also well known that strenuous eccentric exercise induces greater acute damage to muscle fibres than concentric and isometric exercises (Komi and Rusko, 1974). The main difference between the three muscle contractions is that in an isometric contraction the muscle length remains constant while the contracting muscle is forcibly lengthened during an eccentric contraction and shortens during a concentric contraction. Therefore, comparing the three types of

muscle contractions on a matching task becomes appealing to find out whether the type of contraction could have an effect on proprioception.

As a matter of fact, it has been recently shown that proprioception is weakened following repetitive muscle contractions. The size of the matching errors was correlated with the drop in force (Saxton et al., 1995; Walsh et al., 2006). It was then concluded that the fall in force led to an increase in the effort required to maintain position of the limb against the force of gravity and that this increase in effort led to the matching errors (Walsh et al., 2004). Moreover, the direction of the matching errors suggested that we make use of signals of both peripheral and central origin in determining the position of our limbs in space by reliance, in part, on the amount of effort required to maintain limb position against gravity (Walsh et al., 2006).

Muscle fatigue and proprioception has been the focus of many studies in which the majority of them have sought the effect of either isometric, concentric, or eccentric exercise on position sense (Allen and Proske, 2006; Forestier, Teasdale, & Nougier, 2002b; Walsh et al., 2006; Winter et al., 2005). Other authors have compared the effect of concentric versus eccentric exercise on position sense (Brockett et al., 1997; Walsh et al., 2004). However, little is known about which type of muscle contractions would affect proprioception to the utmost level. Therefore, the aim of the study was to determine which type of repetitive muscle contraction would express the greater acute impairment of elbow position sense. Knowing that eccentric exercise exhibits a large acute drop in force in the exercised muscles, the purpose of the present study was to test the hypothesis that eccentric

contractions would lead to the greater decrease of force and would therefore impair proprioception to a greater degree than concentric and isometric contractions.

## 3.2 METHODS

### 3.2.1 Subjects

This study was conducted on 11 right handed male subjects (mean age 28.9 ± 10.6 years; mean height 173.8 ± 6.2 cm; mean weight 80.4 ± 11 kg), without any previous history of upper limb musculoskeletal problems (Table 3.1). They were all aerobically active at least three times a week and 7 subjects also included weight training in their fitness program. All subjects gave their written inform consent in compliance with Memorial University of Newfoundland Human Investigation Committee regulations, were all instructed regarding the procedure of the experiment, and filled in a Physical Activity Readiness Questionnaire (PAR-Q) (Canadian Society for Exercise Physiology, 2003).

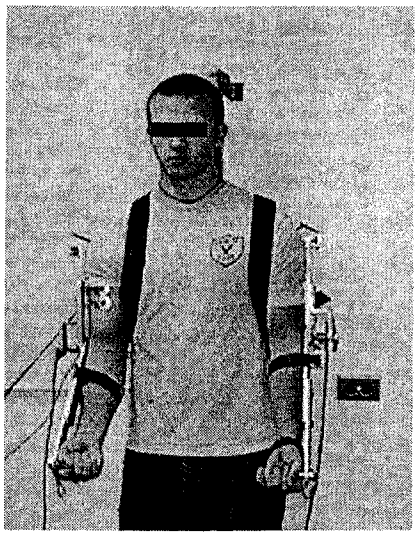
### 3.2.2 Apparatus

As displayed in Figure 3.1, a supporting frame with two handles fully adjustable in the horizontal and vertical planes was instrumented with linear potentiometers (Model K/RV4, Precision Electronic Components Ltd, Weston, Ont.) and a strain gauge (Omega Engineering Inc. LCCA-250, Laval, Qc). The signals from potentiometers and strain gauge were amplified and sampled (Biopac Systems Inc., Santa Barbara, CA) along with surface electromyographic (EMG) signals at a rate of 1 kHz (12-bit A/D). The EMG activity was sampled at 2000 Hz, with a Blackman -61 dB band-pass filter between 10-500 Hz, amplified (bi-polar differential amplifier, input impedance = 2M, common mode rejection

Table 3. 1 Participants' physical characteristics

| Subjects | Age (yrs) | Height (cm) | Body Mass (kg) |
|---|---|---|---|
| 1 | 23 | 172 | 80 |
| 2 | 49 | 175 | 99 |
| 3 | 43 | 163 | 71 |
| 4 | 43 | 163 | 67 |
| 5 | 24 | 173 | 67 |
| 6 | 25 | 171 | 86 |
| 7 | 23 | 185 | 82 |
| 8 | 21 | 175 | 76 |
| 9 | 22 | 175 | 84 |
| 10 | 20 | 180 | 74 |
| 11 | 25 | 180 | 98 |
| Mean (±SD) | 29 (±10) | 174 (±7) | 80.4 (±11.0) |

Figure 3.1 Position sense apparatus consisting of a supporting frame with two handles fully adjustable in the horizontal and vertical planes instrumented with linear potentiometers. The axis of rotation of the subject's elbow was aligned with the axis of rotation of the handle where the potentiometer was positioned. Shoulders and waist were tightly strapped with safety belts



ratio ≥ 110 dB min (50/60 Hz), gain x 2000, noise ≥ 5 μV). A voltage pulse (0-5 Volts) was also collected to mark temporally the matching between the reference and indicator arms. Accuracy of angular displacements and force output were ≤0.05% (0-5 Volts) and ≤0.03% with a full scale ranged from 0° to 314°and from 0 to 1000 N, respectively. The axis of rotation of the subject's elbow (lateral epicondyle) was aligned with the axis of rotation of the handle where the potentiometer was positioned. Shoulders and waist were tightly strapped with safety belts to limit trunk motion. The subjects' arms were kept next to the body with their hands holding the handles at different angles. For the exercise tasks, the resistance was attached to the right handle by a strain gauge.

### 3.2.3 Experimental procedure

Subjects first attended a familiarization session in which anthropometric measurements were obtained to fit the subjects to the manipulandum. They also produced 3 maximal voluntary isometric contractions (MVC) with their right dominant arm with a 5 min rest interval between MVCs. The maximum value was used to determine the exercise resistance for the subsequent sessions. All subjects then participated to 3 different sessions separated by a minimum interval of 7 days. For each session, testing was conducted at the same time of day to nullify possible differences attributed to diurnal rhythms. The three experimental sessions (randomly presented) were differentiated by the nature of the exercise tasks which consisted of isometric contractions, concentric contractions – concentric phase only, or eccentric contractions – eccentric phase only. Each session included three tasks: an exercise task (isometric, concentric or eccentric), a proprioception task (PT), and a MVC. They all started by collecting the resting blood lactate level (Accutrend® Lactate Analyzer,

Mannheim, Germany) followed by 3 pre-exercise PT at each target angular position (TAP) (see Figure 3.2). The PT consisted of matching angular position of the right indicator arm to that of the left reference arm. Target angular positions were set at 70°, 110° and 150° of elbow flexion. During the PT the subjects voluntarily positioned their reference arm at the TAP using visual feedback provided through the computer screen. They were asked to maintain, unsupported, this reference position, to close their eyes, and then, to match with their indicator arm. No feedback was given about their performance. When the subject indicated that both arms matched, a voltage pulse was triggered by the experimenter to identify the matching (Figure 3.3). Before each TAP, subjects returned both arms to 90° using visual feedback. Thereafter, subjects performed the first of nine sets of the exercise task. Each set consisted of 10 voluntary contractions of 4 seconds each with a 2 seconds rest between contractions. For the isometric condition, the elbow was flexed at 90 degrees and the torque output was provided on the computer screen. For the concentric condition, subjects were asked to extend their elbow starting from an elbow angle of 180° and to flex the arm until the arm was fully flexed, keeping their shoulder stable. At this point, they were asked to relax their arm (indicator arm) while the experimenter returned the weight to its initial position. Subjects were required to lift 75% of their maximal concentric contraction (isometric MVC minus 20%). The resistance for concentric and eccentric contractions was applied by means of weight plates attached to the right handle of the supporting frame. Similar procedures were adopted for the eccentric condition; subjects were asked to do series of lifts from full flexion to full extension at 75% of their maximal eccentric voluntary contraction (isometric MVC plus 20%). Each set of 10 contractions was separated by approximately 2 minutes (about 1-min to complete the 3 PT and 1-min of complete rest). This procedure was completed 9 times with 3 PT at the same TAP and

Figure 3.2 Design of the familiarization session and an experimental session

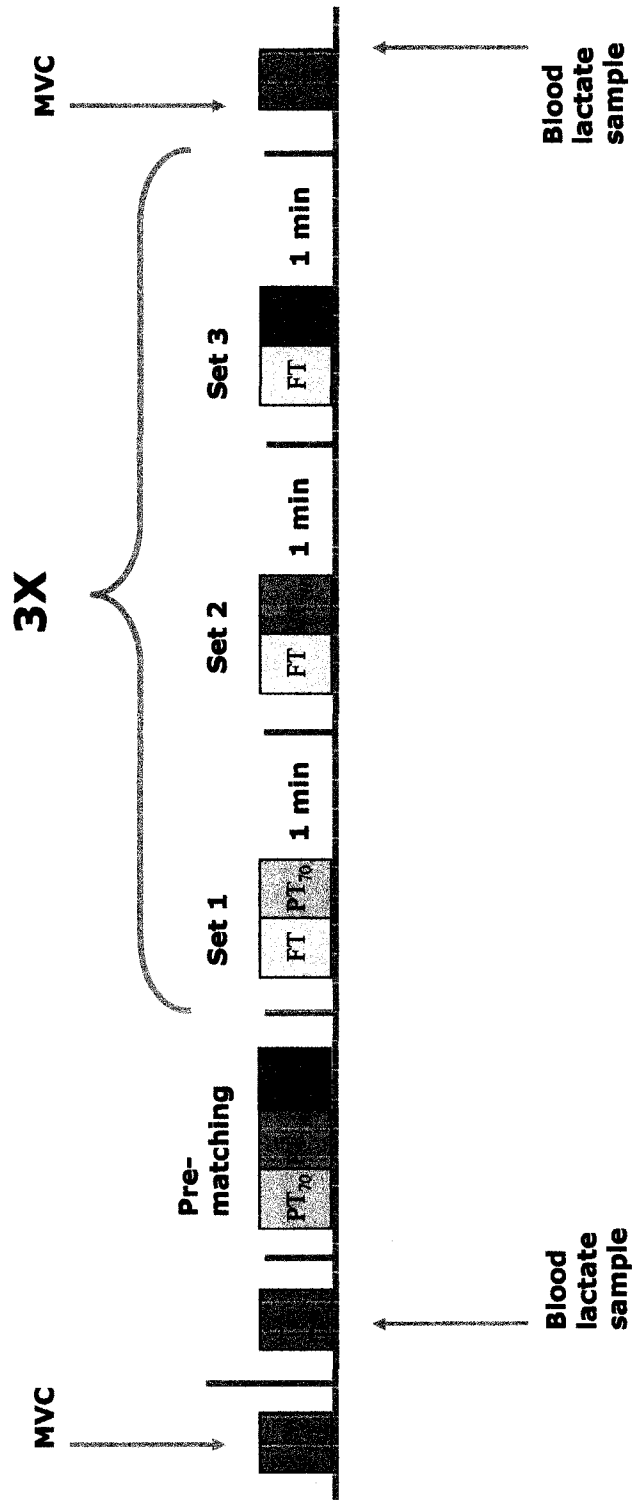
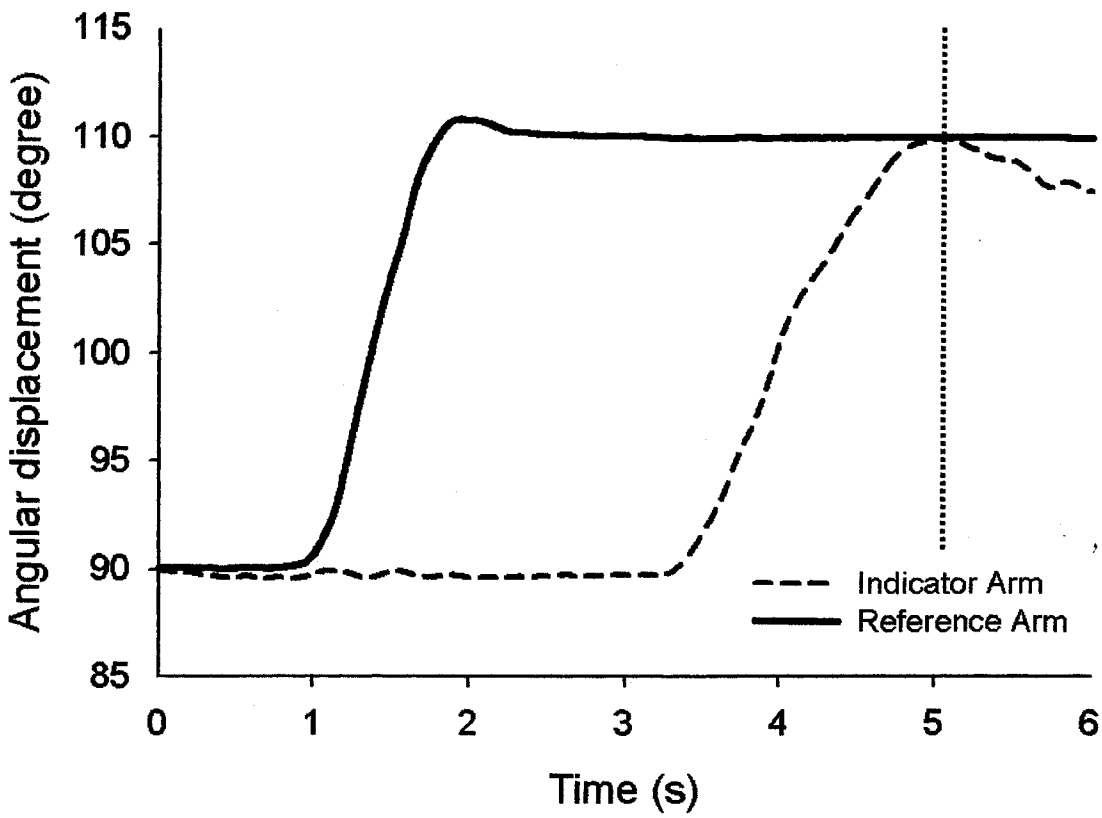


Figure 3.3 A typical set of kinematic data. The solid line represents the angular displacement of the reference arm and the dash line represents angular displacement of the indicator arm. The starting position of both arms was always at 90° of elbow flexion and when both arms were matched, a voltage pulse was triggered and is represented by the dotted vertical line in this figure



1-min rest between each set. The TAP differed from set to set. Once subjects could no longer maintain the required exercise task for two consecutive contractions, the ongoing set was stopped and immediately followed by a post-exercise MVC and blood lactate level collection. Surface EMG recording electrodes (MediTrace, Tyco Healthcare Group, Mansfield, MA) were placed over the belly of the *biceps brachii* and *triceps brachii* muscles in a bipolar fashion and approximately 3 cm apart. Electrodes were placed along the estimated direction of the muscle fibres. The ground electrode was placed on the clavical shaft. Skin preparation for all electrodes included removal of body hair with a razor and cleaning dead epithelial cells using an isopropyl alcohol swab.

### 3.2.4 Data analysis

All kinetic and kinematic parameters were analyzed using custom made software (MATLAB, MathWorks Inc., Natick, MA). From these parameters, the following variables were determined: (a) maximal voluntary contraction (MVC) force: the force curve was filtered (second-order low-pass Butterworth filter with a 7 Hz cutoff frequency) and the MVC was determined by the maximal torque output of the curve; (b) position matching errors: angular position signals were smoothed with a moving window average (101-ms window) and position matching error was calculated as:

angle (reference arm) – angle (indicator arm),

where 90° = horizontal forearm and 180° = vertical forearm. For each trial, matching accuracy was determined using the constant (CE), absolute (AE), and variable (VE) errors. Constant error is the mathematical operation difference between the position of the right

arm and the position of the left arm. By convention, a negative value refers to a more extended indicator arm whereas a positive value refers to a more flexed indicator arm. Absolute error is the absolute deviation between the position of the indicator arm and the reference position and represents the overall accuracy in performance. Finally, variable error represents the amount in variability in the response regardless of the target; (c) total matching time: defined as the time between the first movement of the right arm from the starting position and the onset of the voltage pulse; (d) movement time: defined as the time between the start and the end of a contraction performed during the exercise task. The start and the end of a contraction was determined by using the right potentiometer for the concentric and eccentric conditions while using the force output for the isometric condition; and (e) integrated EMG: the EMG signals from the *biceps brachii* were full-wave rectified and integrated. The EMG activity was integrated between the start and the end of a contraction and normalized by the time. The integrated EMG (iEMG) values for submaximal contractions during the exercise task were normalized relative to pre-exercise iEMG value obtained from the MVC.

### 3.2.5 Statistical analysis

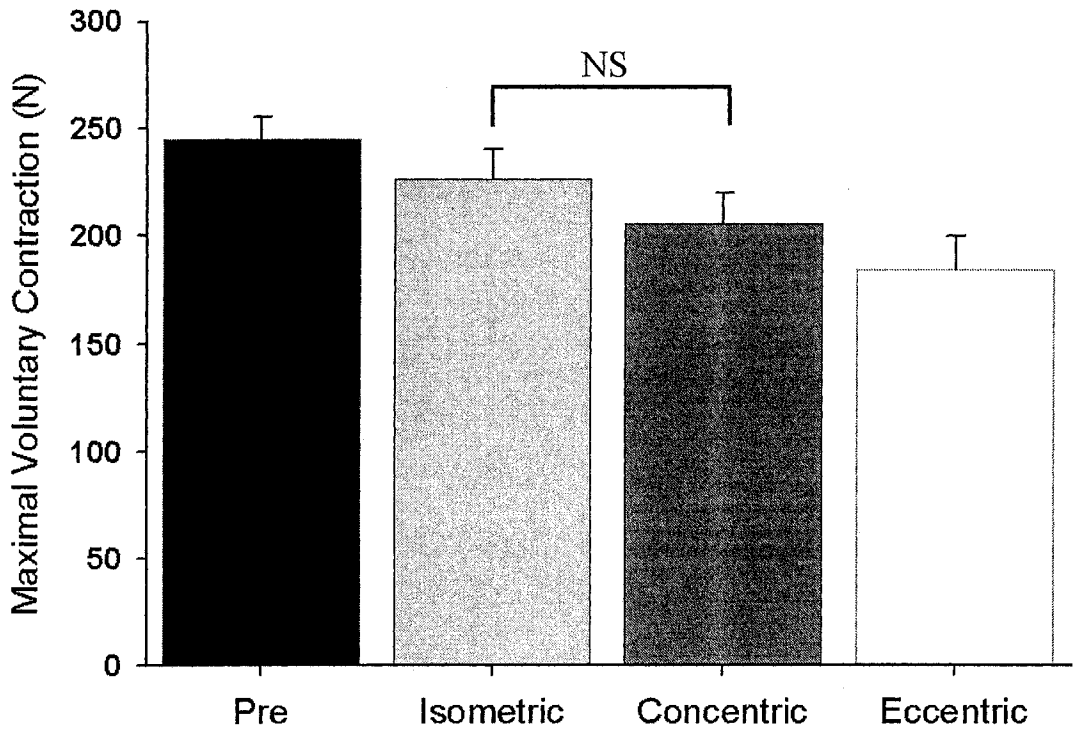
All variables are presented as mean (±SE) and 95% confidence intervals unless otherwise mentioned. Repeated measures analysis of variance followed by post hoc Bonferroni test was used for statistical comparisons of means. Levene tests for equality of variances were performed and, if significant, square root adjustments were made. First, a one-way analysis of variance with repeated measures on the factor condition (Pre-exercise, Isometric, Concentric, and Eccentric) was performed on the MVC parameters. Second, a two-way analysis of variance [2 data acquisition time (Pre - Post) x 3 exercise tasks (Isometric,

Concentric, and Eccentric)] with repeated measures on both factors was computed on blood lactate values. Third, a one-way analysis of variance with repeated measure (Isometric, Concentric, and Eccentric) was performed on iEMG data and movement time, and finally a two-way analysis of variance [4 exercise tasks (Pre-exercise, Isometric, Concentric, and Eccentric) x 3 PT (70°, 110°, and 150°)] with repeated measures was computed on CE, AE, and VE. The level of significance was set at a $p$-value of 0.05. For all statistical tests, SPSS 14.0 for Windows was used (SPSS inc., Chicago, IL).

## 3.3 RESULTS

The pre-exercise mean value (±SE) for MVC was 244 (±10) N [95% CI= 221–268]. The values recorded at the end of the post-exercise (i.e., approximately 1 min after completing the last set of the exercise task) for isometric, concentric, and eccentric conditions were 226 (±14) N [95% CI= 195–258], 205 (±14) N [95% CI= 172–238], and 184 (±15) N [95% CI= 150–218], respectively (Figure 3.4). Bonferroni post hoc analysis showed that post-exercise MVCs decreased significantly ($p < 0.05$) compared to the pre-exercise with a greater drop in force for the eccentric contractions. Nevertheless, the post-exercise isometric and post-exercise concentric MVCs were not significantly different from each other. The pre-exercise mean values (±SE) of blood lactate concentration for isometric, concentric, and eccentric conditions were 1.8 (±0.2) $mmol \cdot L^{-1}$ [95% CI= 1.4–2.2], 1.8 (±0.1) $mmol \cdot L^{-1}$ [95% CI= 1.5–2.2], and 1.8 (±0.3) $mmol \cdot L^{-1}$ [95% CI= 1.2–2.5], respectively. Blood lactate concentrations recorded at the end of the isometric, concentric, and eccentric exercises were 3.6 (±0.3) $mmol \cdot L^{-1}$ [95% CI= 2.8–4.2], 3.7 (±0.2) $mmol \cdot L^{-1}$ [95% CI= 3.2–4.3], and

Figure 3.4 Mean (n=11) maximal isometric voluntary contraction (MVC) for pre-exercise, post-exercise isometric, post-exercise concentric, and post-exercise eccentric (NS, Non Significant; Error bars +SE)



3.6 (±0.5) mmol•$L^{-1}$ [95% CI= 2.5–4.8], respectively (Figure 3.5). For all three exercises, post-exercise blood lactate concentrations increased significantly ($p < 0.05$). However, the pre- and post-exercise values did not differ among conditions and the analysis revealed no interaction. In the same way, results from a pilot study revealed that blood lactate concentrations did not significantly differ at 5-min as compared to immediately after the end of the exercise.

The mean value (±SE) of iEMG during the isometric condition was 0.197 (±0.023) mV•$s^{-1}$ [95% CI= 0.145–0.249], 0.228 (±0.049) mV•$s^{-1}$ [95% CI= 0.115–0.340] during the concentric condition, and 0.166 (±0.013) mV•$s^{-1}$ [95% CI= 0.137–0.196] during the eccentric condition. The analysis reveals that the iEMG was not significantly different ($p < 0.05$) between conditions.

Figure 3.6 shows that subjects were accurate before the exercise with a mean CE of -0.1° (±1.1) [95% CI= -2.4–2.4] at 70°, -1.6° (±0.9) [95% CI= -3.6–0.3] at 110°, and 0.1° (±1.2) [95% CI= -2.7–2.8] at 150°. After the isometric exercise, the indicator arm matched the reference arm with a CE of 0.6° (±1.5) [95% CI= -2.9–4.0] at 70°, -3.3° (±1.1) [95% CI= -5.9–-0.8] at 110°, and 0.2° (±1.1) [95% CI= -2.2–2.6] at 150°. With the concentric exercise, the CE was -0.8° (±1.1) [95% CI= -3.3–1.7] at 70°, -4.9° (±1.4) [95% CI= -8.1–-1.7] at 110°, and -2.2° (±1.2) [95% CI= -5.0–0.5] at 150°. Finally, the eccentric exercise led to a CE of 0.4° (±1.2) [95% CI= -2.4–3.0] at 70°, -3.6° (±1.7) [95% CI= -7.5–0.3] at 110°, and -2.2° (±1.1) [95% CI= -4.8–0.4] at 150°. Negative values correspond to a more extended indicator arm position. Results show that the CE was only decreased after the concentric

Figure 3.5 Mean (n=11) blood lactate concentration for pre- and post-exercise isometric, pre- and post-exercise concentric, and pre- and post-exercise eccentric (* $p < 0.05$; Error bars +SE)

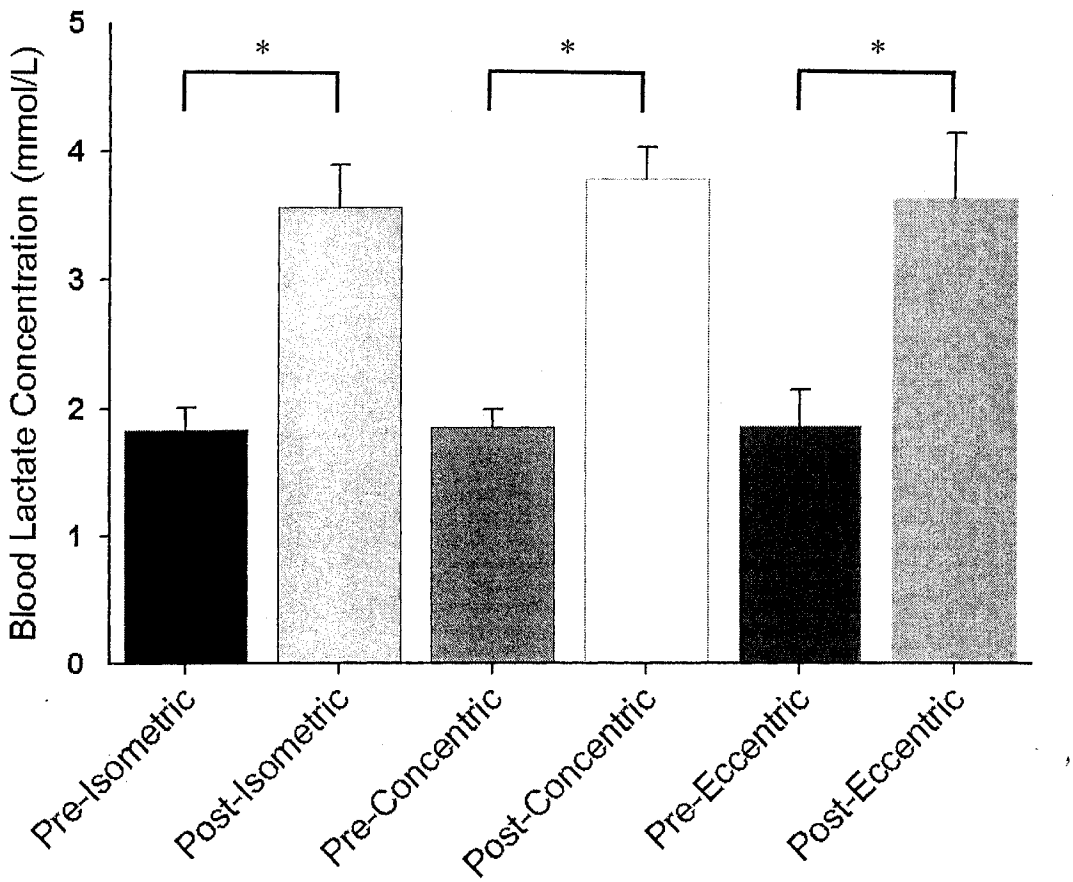
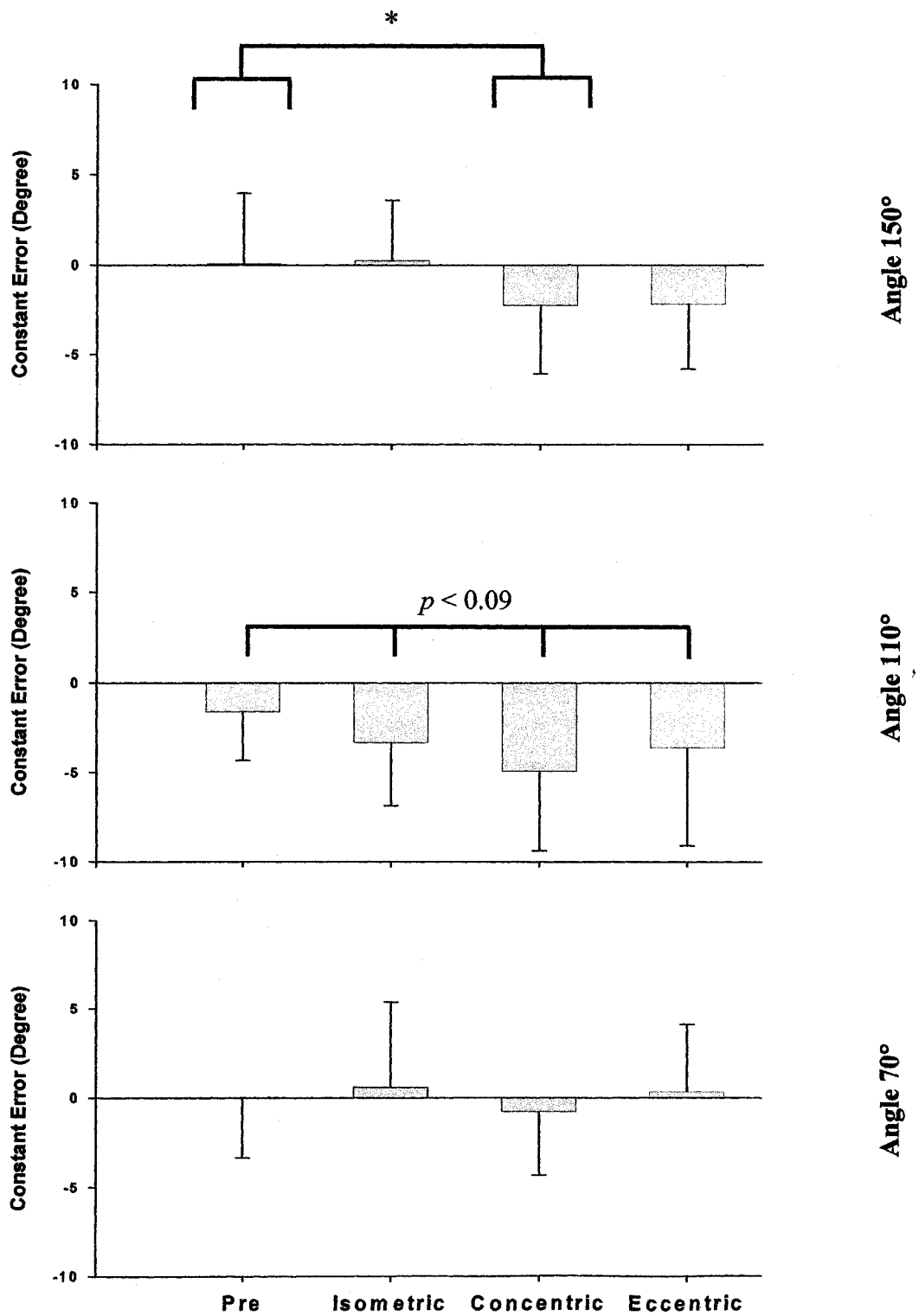


Figure 3.6 Subjects (n=11) mean constant error, that is the sign difference between the reference arm and the indicator arm, for the pre-exercise matching, the isometric condition, the concentric condition, and the eccentric condition at different target angle positions (* $p < 0.05$; Error bars +SE)



exercise as compared to pre-exercise ($p < 0.05$) with no interaction. The statistical analysis also reveals that CE tended to be larger at 110° than at 70° and 150° of elbow flexion ($p < 0.09$). Despite this non significant $p$-value, analysis of confidence intervals most likely reveals a real angle effect. In fact, 74% and 64% of the range of the true value at 110° did not overlap the ones at 70° and at 150°, respectively. Note that 110° displayed a wider range of true value as a consequence of a lesser matching accuracy [95% CI= -5.8–-0.9] compared to 70° [95% CI= -2.3–2.4] and 150° [95% CI= -2.8–0.7]. The AE, which has been square root adjusted because of non-normal distribution, did not reach the significant difference. The VE was 1.2 (±0.2) [95% CI= 0.8–1.7] in pre-exercise as compared to 0.8 (±0.1) [95% CI= 0.6–1.0] after isometric, 0.8 (±0.1) [95% CI= 0.5–1.0] after concentric, and 0.9 (±0.1) [95% CI= 0.7–1.1] after eccentric exercises. The analysis reveals that VE was significantly higher in pre-exercise as compared to the isometric condition. However, this result was mainly due to the inconsistent responses from two subjects, their values (3.6 and 4.3) being substantially outside the 95% confidence intervals for this variable [95% CI= 0.8–1.7]. It is, therefore, almost impossible to interpret this parameter owing to the violation of the normal distribution.

The movement time was 3.65 (±0.03) s [95%CI= 3.59–3.70] for isometric, 3.71 (±0.03) s [95%CI= 3.66–3.78] for concentric, and 4.00 (±0.04) s [95%CI= 3.93–4.07] for eccentric contractions. The analysis reveals that the movement time was significantly higher for the eccentric contractions as compared to the isometric and concentric contractions. Despite the statistical significance, 0.350 s difference in movement times between the eccentric and

isometric contractions is not believed to have considerably affected the aforementioned results. Hence, the parameter will not be further discussed.

## 3.4 DISCUSSION

The objective of this study was to determine which type of muscle contractions would impair position sense to the greatest degree. Our results partly confirmed our hypothesis by revealing that eccentric exercise induced a greater force loss compared to isometric and concentric exercises. Nevertheless, the new insight from this study was that, despite the larger drop in force output seen with eccentric contractions, proprioception was only significantly affected with the concentric exercise task. Matching errors always resulted in the indicator arm adopting a more extended position as compared to the reference arm, independent of the matching angles. We are confident that these outcomes could be generalized to the population at large because our subjects represented a wide age spectrum of the general population (Table 3.1).

The results of the present study showed that post-exercise MVCs were significantly reduced in all conditions suggesting that fatigue occurred with all types of muscle contraction. There was an 8%, 16%, and 25% decline in isometric, concentric, and eccentric elbow force capacity, respectively (Figure 3.4). Similar force reductions were previously observed by Marks and Quinney (1993). The greater loss of force seen after eccentric muscle contractions of the elbow flexors was also observed by Komi and Rusko (1974) and Newham et al. (1983). Indeed, development of fatigue may be specific to activity, duration, and type of contraction suggesting that possible causes of the muscle performance decline

may be different according to the type of contraction. The reduction in force output seen in this study during isometric contractions could result from ischemia due to elevated intramuscular pressure, increasing concentrations of metabolites, and/or limitations in blood flow (Babault et al., 2006; Humphreys and Lind, 1963). Compared with the continuous isometric contraction, the intermittent nature of the concentric procedure (muscular actions followed by a passive movement) may have increased blood flow and therefore the evacuation of metabolic by-products (Laaksonen et al., 2003). The force loss induced by concentric contractions would then be explained by the intracellular excitation-contraction coupling processes as proposed by Pasquet et al. (2000). It is also known that when a muscle lengthens (eccentric) during activation the energy requirement and mechanical response differ from those of shortening (concentric) contractions (Clarkson and Newham, 1995; Enoka, 1996). In fact, Enoka (1996) reported that eccentric contractions require unique activation and control strategies by the nervous system compared to other types of contraction. Given that the actin-myosin bonds should mainly be disrupted mechanically in eccentric contractions, the crossbridge cycling interaction can occur with less ATP hydrolysis than in concentric contractions. Eccentric contractions used in the present study are thus associated with a lower energy cost, but with a higher tension output that may well be the cause of damage to the muscle-tendon system (Clarkson and Newham, 1995; Friden and Lieber, 1992; Lieber, Thornell, & Friden, 1996) which would lead to a greater force loss with eccentric compared to isometric and concentric contractions.

Understanding of the mechanisms of force reduction following eccentric exercise has made considerable progress in the last decade. In eccentric exercise, much evidence exists that the initial local damage results from mechanical rather than metabolic mechanisms (Brooks,

Zerba, & Faulkner, 1995; Lieber and Friden, 1993; Warren, Hayes, Lowe, & Armstrong, 1993). It is clear that some individual sarcomeres elongate excessively during the stretch but that most of these sarcomeres return to normal during relaxation with the thick and thin filaments reinterdigitated. With repeated stretch used during our eccentric exercise task, it is probable that these sarcomeres gradually become damaged and then fail to reinterdigitate (Allen, 2001). Histological studies have reported direct evidence of extensive disorganization and even disruption of the myofibrillar structures and intermediate filaments, leading to the classically observed Z-line streaming (Friden, Kjorell, & Thornell, 1984; Friden et al., 1981). Indices of sarcolemmal disruption, swelling and disruption of the sarcotubular system (Armstrong, 1990; Friden and Lieber, 1996), swollen mitochondria (Stauber, 1989; Warhol, Siegel, Evans, & Silverman, 1985) as well as extracellular matrix injury (Han et al., 1999; Koskinen et al., 2001; Myllyla, Salminen, Peltonen, Takala, & Vihko, 1986) also could explain the drop in force with eccentric exercise. The precise details of the sarcomere disruption process following eccentric contractions remain the subject of speculation. They may also involve the elastic filament titin, which anchors thick filaments to Z disks, or the structural protein desmin, which links adjacent Z discs (Allen, 2001). In addition, inactivation of some sarcomeres from damage to T-tubules may play a part. Whatever the precise details, there is evidence of overextended sarcomeres in muscle which has undergone eccentric contractions which could be responsible for the marked decline in force after exercise as seen in the current study. However, it must be remembered that different eccentric protocols and different muscles can exhibit different patterns of eccentric damage.

In addition to the force output, blood lactate was also collected pre- and post-exercise. It has been reported in the literature that the blood lactate production from exercising skeletal muscle is an indicator of the metabolic rate (Brooks, 1986). More specifically, lactate production reflects the metabolic demand and is an indicator of glycolysis efficiency during exercise particularly in high intensity bouts as used in the current study. In the present experiment, the blood lactate was increased after each condition but was not different between them suggesting that the fatigue protocol used in this study induced the same metabolic stress in all conditions (Figure 3.5). Consequently, the different results observed between the types of contraction were not due to different metabolic stress induced by the exercise task.

Proprioception is crucial for coordinated movement. When there is a deficit in proprioception, controlled movements are impossible without continuous visual guidance (Ghez et al., 1995). Moreover, when the state of the muscular system is modified by muscular contractions, matching experiments have yielded various and somewhat conflicting results (Allen and Proske, 2006; Brockett et al., 1997; Forestier and Bonnetblanc, 2006; Marks and Quinney, 1993; Pederson et al., 1999; Saxton et al., 1995; Skinner et al., 1986; Sterner et al., 1998; Walsh et al., 2006; Walsh et al., 2004; Winter et al., 2005). Nevertheless, it is generally accepted that fatigue negatively affects joint proprioception mainly through the alteration in either muscle afferents or sense of effort. Results of the present study revealed that subjects were variable but quite accurate in position matching task before the exercise. Immediately after the concentric exercise, subjects made systematic position matching errors (-2.6 degrees) by adopting a more extended position with the indicator arm compared to the reference one (Figure 3.6). Recent

investigations found that the exercised elbow flexors adopted a more flexed position in matching the position of the unexercised reference arm (Allen and Proske, 2006; Saxton et al., 1995; Walsh et al., 2004); in other words, the opposite of our finding. In some cases, the size of the matching errors correlated with the decrement in force suggesting that the drop in MVC was responsible for matching errors. Again, this is contrary to our results since the greater drop in MVC was observed after the eccentric exercise and significant matching errors were made after the concentric exercise. However, others authors suggested that agonist muscle fatigue was associated with a more extended final arm position due to a diminution of the contractile capacity of the muscles (Winter et al., 2005). Such contradictory findings might come from dissimilarity in procedure. An important aspect of our design was to reproduce the same metabolic fatigue in all conditions for further comparisons. Indeed, the weight lifted by subjects was express as a percentage of the torque recorded during an isometric MVC. The maximal torque produced in the eccentric and concentric contractions was equivalent to 120% and 80% of the isometric MVC, respectively (see Methods). A similar design was used by Pasquet et al. (2000). In addition, the exercise task was designed to reflect as closely as possible a typical weight training session. It was also important for the matching task to replicate a normal/athletic situation, that is, where subjects are standing up with both arms unsupported on both sides. Moreover, because the same matching angles were used in all conditions, a direct comparison between these three types of muscle contractions was possible. Our design was thus different from other studies which have examined the same muscles. Therefore, comparison of the present results with those of earlier reports cannot be applied directly. However, in the following section we shall highlight mechanisms responsible for the discrepancy between our results and the aforementioned studies.

It is generally accepted that sense of limb position is provided by signals from skin, joint and most importantly muscle receptors (Brockett et al., 1997; Gandevia, Proske, & Stuart, 2002). The current view is that the level of resting activity from muscle spindles signals the length of the muscle and, accordingly, the position of the elbow. Indeed, the experiments on muscle vibration have implicated muscle spindles in the sense of position (Goodwin et al., 1972). The subject whose muscle is vibrated experiences an illusion of movement at the joint about which the vibrated muscle operates and this illusory movement occurs in the direction that normally would stretch the vibrated muscle. Consequently, it was concluded that it was not the physical stretch of the muscles but rather the excitation of the afferents that was most important for the brain to perceive limb movements (Naito, 2004). One explanation is that muscle vibration would cause spindle firing to increase from its pre-existing level to some constant level (McCloskey, 1981). It has also been hypothesized that, with continuous muscular work, an accumulation of metabolic by-products in the muscles could alter group III and IV muscle afferents that could in turn activate γ-motoneurons thus leading to subsequent rises in muscle spindle sensitivity (Nicol and Komi, 2003). Certainly, spindle discharge rates in actively contracting muscles are likely to be higher and more irregular than in the passive muscle, as a result of co-activated static fusimotor activity. An increase in spindle afferent activity in the elbow flexors at a given limb position would give the illusion that the indicator arm is more extended than it really is. One would then expect the subject to place the exercised limb consistently into a more flexed position when it is being used to match the position of the unexercised limb. Therefore, the hypothesis of increased spindle discharge rate in fatigue condition cannot elucidate the present results.

In contrast, some studies performed on humans have indicated that the activity of muscle spindle activity declines during sustained voluntary contractions (Macefield, Hagbarth, Gorman, Gandevia, & Burke, 1991; Vallbo, 1970). The mechanism for the reduction in spindle discharge has been proposed to involve a reflex inhibition of fusimotor neurons by small muscle afferents excited by the metabolic products of the exercise (Bigland-Ritchie et al., 1986; Brockett et al., 1997; Garland, 1991; Jeannerod, Michel, & Prablanc, 1984). Bongiovanni, Hagbarth, & Stjernberg (1990) presented indirect evidence that the fusimotor mediated spindle discharge progressively declines during MVC, and this decline was attributed to muscle spindle 'fatigue', that is, a progressive withdrawal of spindle-mediated fusimotor support to α-motoneurons. It has also been stated that the spindle afferents' role is different with concentric contractions because fusimotor drive may be insufficient to overcome muscle shortening and spindle afferents in the contracting muscle may fall silent (Hayward et al., 1991). Therefore, this reduced stretch sensibility of spindles after a voluntary contraction represents one explanation for the increase in the threshold for muscle spindle discharge. This would be matched by the indicator arm adopting a more extended position, where the muscle and its spindles were subjected to a greater degree of stretch, and thus leading to a decline in position sense after voluntary contractions. Indeed, the present results suggest that the signal from muscle spindles had decreased as a result of the exercise. To match the level of proprioceptive signal (i.e., spindle discharge rates), the exercised muscle had to be stretched more than the control muscle. Therefore, it could be argued that the pattern of positional errors observed in this study is the result of exercise-related changes in the response of muscle spindles.

There has always been some reluctance to accept the concept of muscle spindles as proprioceptors. In fact, the results from Marks and Quinney (1993) did not affirm the importance often attributed to muscle spindles in mediating sense of position. It was then suggested that other sources of afferent or efferent activity operating at other levels of the nervous system might by involved in mediating position sense. Results from Walsh et al. (2004), Winter et al. (2005), and Allen and Proske (2006) have been interpreted as evidence in opposition to muscle spindles as proprioceptors and in favour of an effort-based signal contributing to position sense during limb placement. They proposed that the effort required to maintain position of the arm against the force of gravity provides us with information about its location in space. To hold the arm at a set angle against the force of gravity, unsupported, requires a certain amount of effort. During exercise, as muscle force declines, the central nervous system compensates by increasing activation of motoneurons, leading to a progressive increase in the perceived effort (Proske, 2005). If subjects match efforts to align their arms they will place the fatigued arm more nearly vertically where less force is required to support it and where the same effort would be required to maintain its position. Adoption of a more vertical position requires less effort for two reasons. First, the moment of the force of gravity on the arm is less. Second, a more vertical position is closer to the elbow flexors' optimum length for active tension (Walsh et al., 2004). Indeed, all conditions combined, our subjects adopted a more flexed arm position with the angle 70° and a more extended arm position with the angles 110° and 150° because of the smaller moment of the force of gravity acting on the latter. It was also reported that when subjects carry out a forearm position matching task, they become more erratic in their performance if the task was carried out in the horizontal plane, using a counterweight (Gooey, Bradfield, Talbot, Morgan, & Proske, 2000) or by supporting the arm (Paillard and Brouchon, 1968). It was

then proposed that these effects are due to withdrawal of a positional cue normally available to subjects when matching the unsupported arms in the vertical plane where the gravity plays a major role. These observations suggested that if muscle spindles contributed to position sense, an additional cue was provided by the amount of effort required to maintain the position. Hence, the most recent hypothesis in the literature is that accurate placement of forearms is achieved by a combination of effort-related signals and muscle spindle signals (Allen and Proske, 2006). How these two sources of kinaesthetic information combine to give us our normal positional acuity need further investigations.

It could also be argued that the present results may be explained by muscle thixotropy. The latter depends on the preceding muscle conditioning and the length at which the muscle is held immediately after conditioning. It was suggested that with a human forearm position sense experiment, the position adopted by the indicator arm was always dependent on the form of conditioning that preceded it (Gregory et al., 1990; Gregory et al., 1988; Proske et al., 1993; Winter et al., 2005; Wise, Gregory, & Proske, 1996). Muscle thixotropy acts, in large part, by altering the mechanical state of the intrafusal fibres of muscle spindles (Gregory, Wise, Wood, Prochazka, & Proske, 1998). If the contraction is carried out at a long muscle length, once the muscle has relaxed, the intrafusal fibres at rest remain long. If the muscle is kept still immediately after the contraction, new cross bridges may form at that particular length. Once new stable cross bridges have formed, if the muscle remains passive and undisturbed, they remain intact at that length for a long period. If the passive muscle is then shortened (flexed), such long intrafusal fibres fall slack. Alternatively, if the voluntary contraction is carried out at a short muscle length, intrafusal length will be short and no slack will be present. However, an important requirement for the generation of

muscle conditioning changes in spindle resting activity is that the spindles remain passive since cross bridges can be detached by stretch or by a contraction that can eliminate slack fibres and make all actual fibres of equal length (Proske et al., 1993). Therefore, the effects of muscle thixotropy on the steady state of muscle receptors and their stretch sensitivity would not be expected in the present experiment since active positioning of the subject's forearm in the starting position should have effectively removed the after-effects of previous events. Thus, muscle thixotropy cannot explain the diverse results observed in this experiment with different types of contraction.

Still, it remains difficult to explain the trend for having greater matching errors with the 110° compared to the two other angles (Figure 3.6). Despite having not reached a statistical significant *p*-value, in reality there is a good chance that the treatment did have a real effect on subjects' response. In fact, the analysis of confidence intervals, which represent the likely range of the true effect of treatment on the average subject, revealed a real angle effect (see Results). This statistical approach would then be one of the simplest and the best way to interpret this outcome (Hopkins, 2000). It has been claimed that in many joints, proprioceptive activity was most intense at the near-terminal range of motion of the flexion-extension range (Ferrell, 1980; Lephart et al., 1992; McCloskey, 1978; Skoglund, 1973). A true position sense would then be heightened towards the full flexion or full extension when more joint, cutaneous, and muscle afferents discharge. In fact, Jerosch and Prymka (1996) declared that angle reproduction tests in normal subjects showed significantly worse angle reproduction capability in the midrange. Therefore, the inability to reproduce a passively positioned angle at the midrange of motion, that is 110° in the present experiment, was due to a desensitization of the peripheral afferents. Accordingly, we interpret the smaller

matching errors seen with the 70° and 150° angles as due to the more precise information subjects received about the position of their forearm over this range.

## 3.5 CONCLUSION

The hypothesis of this experiment was that eccentric exercise would lead to a bigger decrease in force and consequently would lead to a greater impairment of position sense. Only the first part of this hypothesis was confirmed by the present study. Actually, it was the concentric rather than eccentric exercise that impaired position sense to a greater level. An important consequence of all of this from a practical point of view is that fatigue from concentric exercise is expected to be accompanied by a disturbance in proprioception. That conclusion has implications for the competing athletes predisposing to an eventual decrease in athletic performance and to sport injuries. In fact, attaining the aimed final position of a limb is an important motor task of our everyday behaviour and can be also crucial for success in various professional or sporting activities. Moreover, knowing that proprioceptive training has beneficial aspects in rehabilitation following injury or surgery, rehabilitation program should be redesigned to include a proprioceptive component while excluding the concentric phase in proprioceptive exercises.

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# CHAPTER 4 CONCLUSION

## 4.1 RESPONSES TO THE RESEARCH HYPOTHESIS

The research hypothesis stated that eccentric contraction would lead to the greater decrease of force and would therefore impair proprioception at a higher level than concentric and isometric contractions. The first half of this hypothesis was accepted since post-exercise MVC reached lowest value after the eccentric exercise task compared to those for isometric and concentric exercises. However, the second half was not confirmed since matching errors were larger in the concentric exercise session.

## 4.2 SUMMARY

The literature supports that muscle performance is reduced after repetitive muscle contractions as shown by a decrease in post-exercise maximal voluntary contraction as compared to pre-exercise. This reduction is more apparent with an eccentric exercise protocol. It is also generally accepted in the literature that position sense is impaired in the acute phase by isometric, concentric, and eccentric repetitive muscle contractions. However, no study at this point has compared proprioception following the three types of muscle contraction. The aim of this thesis was therefore to compare the three types of muscle contraction on position sense using the same protocol and the same subjects.

The study included in the thesis showed that the eccentric exercise task lead to a greater decline in force as compared to isometric and concentric exercise. Results from this study also revealed that, although there was a greater drop in force output seen with the eccentric exercise task, position sense was significantly affected only by the concentric exercise task.

In addition, matching errors were always consistent with the indicator adopting a more extended arm position than the reference arm. Without disallowing the sense of effort as a possible mechanism involved in position sense, our results suggested that a decrease in muscle spindle discharge rate owing to repetitive muscle contractions would more likely be responsible for the matching errors observed following the concentric exercise.

From a practical point of view, fatigue from concentric exercise is thus expected to be accompanied by a disturbance to proprioception. This could in turn have implications for the performance of competing athletes and for sport injuries. In fact, proprioceptive training has become an important aspect of sport rehabilitation after injury or surgery and also in prevention of reinjury. It would then be a good idea in the future to consider the results from this study to modify, if necessary, proprioceptive training program in rehabilitation.

## 4.3 LIMITATIONS OF THE STUDY

(i) Subjects' profile

As displayed in table 3.1, the group of subjects participating in the study was not homogeneous as a result of the age difference. One could argue that results of the study could have been falsified owing to the age effect. For that reason, we ran a statistical analysis with only subjects (n= 8) between 20 and 29 years of age. No difference was observed in this outcome as compared to the analysis with all subjects (n= 11).

(ii) Statistical variance

As mentioned earlier, subjects were quite accurate but variable during the proprioception task. This variance could certainly have affected the final results of the study. In fact, by looking at the Figure 3.6, it seems evident that the angle 110° was negatively affected to a greater degree than the two other angles. Likewise, it could also be argued from this figure that position sense was also affected by the eccentric exercise task. It is, therefore, plausible that the eccentric condition did not reach the significant level as a result of the large variance among subjects.

(iii) Measurements

The degree of muscle fatigue was assessed by two different indicators, the post-exercise MVC along with the blood lactate concentration. One could argue that both are indirect measurements and thus do not give a precise indication of the real fatigue induced by the exercise tasks. Notwithstanding, these two indirect indicators of muscle fatigue are generally accepted in the literature.

The discussion section of the thesis mentioned that the results could be explained by the force of gravity (sense of effort), by the length-tension relationship of the muscle, and by the muscle spindle discharge rate. Unfortunately, none of these variables were directly measured in the study. The discussion is then based on conceivable assumptions from the literature on related subjects.

(iv) Experimental design

The proprioception task used in the study was performed in the vertical plane where gravity may have had a direct influence on the outcome. It would have been interesting to add to the experimental design a condition where subjects would have been in a microgravity environment. Proprioception tasks performed in the horizontal plane or in the water or by using a counterweight are only few ideas that could have been used.

# CHAPTER 5 OVERALL REFERENCES

OVERALL REFERENCES

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