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# The Stretch-shortening cycle as a model to study compensatory mechanisms of muscle-tendon deficits

Caroline Nicol

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**Université de la Méditerranée (Aix-Marseille II)**  
Faculté des Sciences du Sport

*Habilitation à Diriger les Recherches*

Spécialité : Sciences du mouvement Humain

**LE CYCLE ETIREMENT-DETENTE :  
un modèle d'étude des mécanismes de compensation  
en cas d'altération du système musculo-tendineux**

*Mémoire de recherche présenté par*

**Caroline Nicol**

Soutenu le 30 janvier 2009 devant la commission d'examen ainsi composée

<b>Eric BERTON</b>	(PU)	Université de la Méditerranée - Marseille
<b>Xavier BIGARD</b>	(PAgVdG)	Centre de Recherche du Service Santé des Armées – La Tronche (Rapporteur)
<b>Jacques DUCHATEAU</b>	(PU)	Université Libre de Bruxelles – Belgique (Rapporteur)
<b>Laurent GRELOT</b>	(PU)	Université de la Méditerranée - Marseille
<b>Guillaume MILLET</b>	(PU)	Université de Saint Etienne – St Etienne (Rapporteur)
<b>Stéphane PERREY</b>	(MCU-HDR)	Université de Montpellier I - Montpellier
<b>Paavo V. KOMI</b>	(PU)	Université de Jyväskylä – Jyväskylä (Invité)

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*La suite de ce sommaire est présentée en langue anglaise pour correspondre aux trois chapitres qui le composent.*

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## Résumé

Dans les formes habituelles de la locomotion, les muscles extenseurs des membres inférieurs sont engagés au cours de chaque appui au sol dans un enchaînement de phases de freinage (excentrique) et de poussée (concentrique). Cette combinaison, appelée *Cycle Étirement-Détente*, est connue pour accroître l'efficacité de la phase finale concentrique, notamment par la restitution de l'énergie élastique préalablement accumulée lors de l'étirement actif du complexe musculo-tendineux. Nos travaux ont contribué à démontrer que l'intervention de la composante de courte latence du réflexe d'étirement musculaire permettait d'accroître la force et la raideur du muscle. L'utilisation récente de l'échographie à haute fréquence révèle l'existence d'un étirement des faisceaux musculaires des muscles mono- et biarticulaires en début de phase de freinage. Cela renforce l'hypothèse d'une contribution non négligeable du réflexe d'étirement à l'efficacité du CED.

Lorsqu'ils sont épuisants ou inhabituels, les exercices de type CED peuvent engendrer des micro-lésions musculaires dont la lente résorption comprend un phénomène inflammatoire ou de remodelage associé à des douleurs musculaires retardées. La période de récupération est généralement de type biphasique, montrant des baisses fonctionnelles immédiates, suivies d'une récupération partielle ou totale à 2h qui précède de nouvelles baisses pouvant perdurer plusieurs jours. L'évolution parallèle des paramètres nerveux et mécaniques souligne l'influence des baisses d'activation sur les pertes de force enregistrées. L'étude des performances maximales et sous-maximales de type CED montre la flexibilité des ajustements neuromusculaires. La présence de facilitation ou d'inhibition de l'activation centrale et/ou réflexes suggère une intégration sensorimotrice des informations relatives à l'état du muscle (avec ou sans inflammation) et des contraintes de la tâche (maximale vs. sous-maximale). L'inhibition spécifique du muscle « lésé » lors de son étirement actif pendant la phase de freinage pourrait ainsi refléter l'intervention d'un mécanisme protecteur. En accord avec de récentes études des mécanismes centraux de la fatigue, ces résultats tendent à conforter l'hypothèse d'une inhibition motoneuronale réflexe via un circuit activé par les afférences de type III et IV.

Nos travaux soulignent également la persistance d'une mauvaise estimation et/ou compensation des déficits fonctionnels (altération des sens de la position et du mouvement) ainsi qu'une absence de compensation contralatérale d'une fatigue musculaire ipsilatérale. Les conséquences de tels déficits perceptivo-moteurs pourraient s'avérer critiques lorsque la douleur disparaît précocement par rapport au retour à l'intégrité structurale et fonctionnelle.

Nos perspectives de recherche visent à approfondir les mécanismes sous-jacents des inhibitions centrales et réflexes qui caractérisent la phase retardée de la récupération des exercices de types CED. Les protocoles de recherche en cours examinent les stratégies neuromusculaires adoptées dans une tâche plus extrême d'impact supramaximal. Les prochains protocoles devront s'enrichir des techniques actuelles d'investigation de la fatigue centrale.

## Avant-propos

Ce document recense les travaux réalisés au sein de laboratoires de la Faculté des Sciences du Sport de l'Université de la Méditerranée à Marseille, tels que « Mouvement et Perception » (UMR – CNRS 6152), « Déterminants Physiologiques de l'Activité Physique » (UPRES EA 3285) et « Institut des Sciences du Mouvement Humain : Etienne-Jules Marey » (UMR – CNRS 6233), et du « Department of Biology of Physical Activity » devenu « Neuromuscular Research Center » de l'Université de Jyväskylä (Finlande).

La majorité des travaux menés s'inscrit depuis toujours dans une logique de compréhension menée de concert avec l'équipe du Professeur Paavo V. Komi sur les thèmes (i) du mode naturel de locomotion humaine, le cycle étirement détente, (ii) de la fatigue qu'il engendre et (iii) des ajustements neuromusculaires qu'il permet de mettre en évidence. Ce mémoire de HDR ne peut dès lors reposer sur la base de mes seuls travaux. Ces derniers seront donc présentés au sein d'un ensemble élargi de travaux réalisés par l'équipe du Pr P.V. Komi en lien avec ceux d'autres équipes internationales. Ce mémoire repose en grande partie sur la base d'une revue récente de la littérature sur la fatigue de type SSC (Nicol et al. 2006) et de deux chapitres écrits avec le Professeur P.V. Komi et récemment publiés dans un ouvrage collectif intitulé « Physiological Bases of Human Performance during Work and Exercise » (N.A.S. Taylor, H. Groeller and P.L. McLennan, 2008). La partie relative aux perspectives de recherche constitue le projet de thèse débuté en 2008 par Mlle Amandine Galindo. Ce travail co-dirigé par le Pr Janne Avela associe deux collègues, Mmes Joëlle Barthélemy et Pascale Chavet, et un autre étudiant de thèse, M. Cédric Morio, sans lesquels ce nouvel axe de recherche ne pourrait être conduit.

L'objectif de l'ensemble des travaux répertoriés dans ce document est triple :

**Le chapitre 1** vise à décrire le mode naturel de locomotion terrestre que constitue le Cycle Etirement-Détente (CED). La présentation du CED reste succincte car elle n'a pour objectif que de faciliter l'approfondissement dans le chapitre 2 de la fatigue neuromusculaire que ce type d'exercice peut engendrer. Nous insisterons néanmoins sur l'intervention et le rôle fonctionnel des réflexes d'étirement qui constituent l'un de nos deux principaux axes de recherche.

**Le chapitre 2** propose le CED en tant que modèle d'étude des mécanismes de compensation en cas d'altération du système musculo-tendineux sur plusieurs jours. Ce chapitre précise les caractéristiques de la fatigue dite de type CED, s'attache à montrer la flexibilité des ajustements neuromusculaires qui l'accompagnent et souligne la pertinence des tests sous-maximaux de type CED dans ce domaine. Les techniques actuelles d'évaluation de la fatigue centrale y sont développées car elles constituent un manque évident à nos propres travaux de recherche; manque que nous espérons combler dans nos travaux à venir.

**Le chapitre 3** présente nos perspectives de recherche, qui viennent de débiter au sein de l'équipe «P2M» de l'ISM, dans le cadre du travail de thèse de Mlle Amandine Galindo. Ce projet vise à approfondir les capacités d'ajustements centraux et réflexes du système neuromusculaire en situation de perturbations internes (épuisement) et/ou externes (amorti impacts élevés). Ce travail sera codirigé par le Pr Janne Avela du Neuromuscular Research Center de Jyväskylä en Finlande.

Afin de respecter notre propre contribution aux 2 premières thématiques, le chapitre 1 s'attardera plus particulièrement sur les techniques expérimentales, tandis que le chapitre 2 n'y fera qu'allusion (celles-ci étant décrites en fin de document dans les publications originales) pour se concentrer sur la discussion des résultats expérimentaux.

Je tiens à remercier tous ceux qui ont permis à ce document d'aboutir.

*Monsieur le Professeur **Eric Berton**, co-Directeur de l'Institut des Sciences du Mouvement (UMR 6233) - Université de la Méditerranée, pour la confiance octroyée en m'acceptant dans son équipe « Performance Motrice et Modélisation » (P2M). L'équipe qu'il a su créer me permet dès aujourd'hui de m'engager avec sérénité et plaisir sur deux axes de recherche alliant tous deux des aspects fondamentaux et appliqués tels que j'ai toujours souhaité le faire. Qu'il soit assuré ici de ma profonde gratitude et de tous mes souhaits de réussite à l'ISM et à la Chaire.*

*Monsieur le Professeur **Xavier Bigard**, Directeur du «Département des Facteurs Humains » - CRSSA de La Tronche, pour avoir accepté d'être non seulement membre du jury mais également l'un des rapporteurs de ce travail. Son travail de recherche dans les domaines de la physiologie de l'effort et du muscle en particulier illustre parfaitement les raisons de mon enthousiasme pour les capacités d'adaptation du système neuromusculaire. Son intérêt pour la course au large l'ayant déjà conduit à Marseille, je tiens à le remercier d'y revenir pour la présente soutenance.*

*Monsieur le Professeur **Jacques Duchateau**, Directeur du «Laboratory of Applied Biology » - Université Libre de Bruxelles, pour le Modèle Scientifique international et Humain qu'il représente pour nous tous et pour l'Amitié sur musique francophone qui nous a liée depuis mes premiers congrès. Malgré le nombre incalculable de présentations orales en parallèle m'empêchant de venir l'écouter, chacune des exceptions n'en a été que plus appréciée. J'aimerais que les jeunes thésards comprennent au travers de son exemple combien l'objectif et les techniques d'un protocole gagnent à être ciblés et combien les sciences appliquées gagnent à s'appuyer sur des connaissances fondamentales. Le livre de Pierrot-Deseilligny qu'il m'a récemment conseillé en est encore l'une des pertinentes illustrations. Je suis très honorée de sa participation à ce jury et lui suis particulièrement reconnaissante d'avoir accepté d'expertiser ce travail malgré un emploi du temps plus que chargé.*

*Monsieur le Professeur **Laurent Grélot**, Doyen de la Faculté des Sciences du Sport de Marseille et ancien directeur du laboratoire des « Déterminants Physiologiques de l'Activité Physique » (UPRES EA3285 - Marseille) pour sa contribution essentielle à mon parcours d'enseignant-chercheur. Dès son arrivée, il m'a permis de poursuivre enfin à Marseille mon travail de recherche sur le Cycle Etirement Détente par l'acquisition pour le laboratoire de l'ergomètre « indispensable », le Sledge, et d'un appareil d'enregistrement électromyographique. Ajoutant à cela sa large contribution scientifique et humaine au travers de la co-direction de deux étudiants de thèse en STAPS, Foued Ftaiti et Sophie Regueme, il n'y aurait pas de HDR aujourd'hui sans son aide. Merci aussi pour sa compréhension à l'arrivée de Marine.*

*Monsieur le Professeur **Guillaume Millet**, Directeur du « Laboratoire de Physiologie de l'Exercice (EA 4338) - Université de St-Etienne, pour le modèle qu'il représente pour ses connaissances dans le domaine de la physiologie de l'exercice et, plus particulièrement, dans celui de la fatigue neuromusculaire. Sa connaissance de la littérature (ancienne et récente), des intérêts et limites des techniques actuelles d'investigation et son investissement ininterrompu auprès de ses étudiants se traduisent par des articles scientifiques qui comblerent les vides que je n'ai su remplir dans mon propre thème de recherche. Je lui suis particulièrement reconnaissante d'avoir accepté d'expertiser le manuscrit et de participer à ce jury.*

*Monsieur le Maître de Conférence-HDR **Stéphane Perret**, Responsable de l'Equipe « Locomotion Humaine » dans le laboratoire Efficience et Déficiency Motrices - Université de Montpellier I, pour sa participation à ce jury et à celui de mon ancienne étudiante de thèse, Mlle Sophie Regueme. Son expertise scientifique étant très complémentaire de la mienne, telle que j'ai eu la chance de l'apprécier lors de la soutenance de thèse de Thomas Rupp, j'espère que ces échanges nous conduiront dans un futur proche à des protocoles communs de recherche.*



# Curriculum Vitae

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## I. FORMATION ET EXPERIENCE PROFESSIONNELLE (\*développés dans ce mémoire)

2007-	<b>Maître de Conférences des Universités</b> (Section CNU 74), Faculté des Sciences du Sport, Université de la Méditerranée (Aix-Marseille II).
2007- *	- Membre de « l'Institut des Sciences du Mouvement », Etienne-Jules Marey, UMR 6233, équipe « Performance Motrice et Modélisation » dirigée par le Professeur E. Berton.
2000-07*	- Membre de l'UPRES EA3285 « Déterminants Physiologiques de l'Activité Physique » dirigée par le Professeur L. Grélot. - <b>2 ans de détachement au CNRS</b> (2000-02) dans la FRE 2099 devenue UMR 6152 « Mouvement et Perception », dirigée par le Professeur M. Laurent.
1995-99*	- Membre de l'UPR 9041 du CNRS dirigée par le Professeur J.F. Marini.
1994-95*	<b>Maître de conférences stagiaire</b> , Faculté des Sciences du Sport, Université Aix-Marseille II. - Membre de l'UPR 9041 du CNRS dirigée par le Professeur J.F. Marini.
1993-94*	<b>Attachée Temporaire d'Enseignement et de Recherche (A.T.E.R.)</b> , Faculté des Sciences du Sport, Université d' Aix-Marseille II. - Membre de l'UPR 9041 du CNRS dirigée par le Professeur J.F. Marini.
1992-93*	<b>Stage post-doctoral</b> dans le « Department of Biology of Physical Activity », Université de Jyväskylä (Finlande). Projet : « Quantification de la contribution des boucles réflexes à la production de force » Sous la direction du Professeur P.V. Komi.
Juin 1992*	<b>Doctorat en Sciences de la Vie</b> , Université Jean Monnet de Saint-Etienne, soutenu le 30 juin 1992. Spécialité Biologie de l'Exercice Musculaire Mention Très Honorable avec les félicitations du jury Thèse intitulée : « <i>Manifestations neuro-musculaires de fatigue engendrées par une course de marathon</i> », sous la direction des Professeurs P.V. Komi (Jyväskylä, Finlande) et A. Geysant (St-Etienne, France).
Juin 1988	<b>Diplôme d'Etudes Approfondies en Sciences et Techniques des Activités Physiques et Sportives (STAPS)</b> , spécialité Physiologie Musculaire, UFR STAPS de Marseille. Mention Bien, Mémoire intitulé : « <i>Effet d'un entraînement à charge élevée sur les paramètres vitesse et puissance musculaires</i> » Sous la direction des Docteurs J.F. Marini et R. Hyacinthe.
Juin 1987	<b>Maîtrise Fondamentale en STAPS</b> , UFR STAPS de l'Université de Nice Spécialité Bioénergétique, Mentions Bien-Très Bien (théorie-mémoire) Mémoire intitulé : « <i>Cinétique des lactates et paramètres terminaux chez des cyclistes au cours d'efforts progressifs et maximaux sur ergocycle</i> » sous la direction du Docteur P. Marconnet.
Juin 1986	<b>Licence STAPS</b> , UEREPS de Nice. Mention Bien, major de promotion.
Juin 1985	<b>D.E.U.G. STAPS</b> , UEREPS de Nice.
Juin 1983	<b>Baccalauréat de l'enseignement du 2nd degré</b> , Acad. de Nice, <b>Série D</b> , mention Assez Bien.

## II. ACTIVITES D'ENSEIGNEMENT

### Etablissement : Université de la Méditerranée, Aix-Marseille II, Faculté des Sciences du Sport

#### *Année 2008-2009 : 241h ETD*

- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD), Méthodes de gestion de la vie physique (CM,TD,TP), Biologie générale (TD), Physiologie et intervention en EPS (CM).
- 2<sup>ème</sup> cycle: L'effort en EPS (CM), techniques d'intervention en APA et santé (CM,TD), Découvertes des institutions dans le domaine des APA (TD), Suivi de stages (TD).
- 3<sup>ème</sup> cycle : Sciences biomédicales (CM), Activité physique et longévité (CM), Modification des attitudes et des comportements (TD), Evolution dans les pratiques éducatives corporelles et prévention (CM), Régimes alimentaires (CM), Evaluation des participants (CM,TD), .

#### *Année 2007-2008 : 199h ETD*

- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD), Méthodes de gestion de la vie physique (CM,TD,TP).
- 3<sup>ème</sup> cycle : Particularismes physiologiques (CM).

#### *Année 2006-2007 : 192h ETD*

- DEUST : Anatomie Fonctionnelle (CM)
- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD), Méthodes de gestion de la vie physique (CM,TP) et Anglais (TD).
- 3<sup>ème</sup> cycle : Particularismes physiologiques (CM).

#### *Année 2005-2006 : 249h ETD*

- DEUST : Anatomie Fonctionnelle (CM)
- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD) et Méthodes de gestion de la vie physique (CM,TP).
- 3<sup>ème</sup> cycle : Mécanismes de la fatigue (CM) et Fonction Neuromusculaire (CM).

#### *Année 2004-2005 : 247h ETD*

- DEUST : Anatomie Fonctionnelle (CM)
- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD), Méthodes de gestion de la vie physique (CM).
- 2<sup>ème</sup> cycle : Approche pluridisciplinaire des APS (CM) et Mécanismes de la Fatigue (CM).
- 3<sup>ème</sup> cycle : Méthodologie de la recherche (CM).

#### *Année 2003-2004 : 247h ETD*

- DEUST : Anatomie Fonctionnelle (CM)
- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD), Anatomie Fonctionnelle (CM) et Bioénergétique (CM,TP).
- 2<sup>ème</sup> cycle : Approche pluridisciplinaire des APS (CM) et Mécanismes de la Fatigue (CM).
- 3<sup>ème</sup> cycle : Méthodologie de la recherche (CM).

#### *Année 2002-2003 : 198h ETD*

- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD), Anatomie Fonctionnelle (CM) et Bioénergétique (CM,TP).
- 2<sup>ème</sup> cycle : Fatigue Neuromusculaire (CM).
- 3<sup>ème</sup> cycle : Méthodologie de la recherche (CM).

*Années 2000-2001 et 2001-2002 : pas d'enseignement pour cause de détachement au CNRS.*

#### *Année 1999-2000 : 217h ETD*

- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD), Anatomie Fonctionnelle (CM) et Bioénergétique (CM,TP).
- 2<sup>ème</sup> cycle : Physiologie (CM), Biomécanique (CM) et Fatigue Neuromusculaire (CM).

#### *Année 1998-1999 : 194h ETD*

- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM,TD), Anatomie Fonctionnelle (CM) et Bioénergétique (CM,TP).
- 2<sup>ème</sup> cycle : Physiologie et Biomécanique (CM).

#### *Année 1997-1998 : 222h ETD*

- 1<sup>er</sup> cycle: Système Neuromusculaire (CM), Anatomie Fonctionnelle (CM) et Bioénergétique (CM).
- 2<sup>ème</sup> cycle : Physiologie et Biomécanique (CM).

#### *Année 1996-1997 : 219h ETD*

- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM), Anatomie Fonctionnelle (CM), Bioénergétique (CM) et Pratique Sportive Voile (TD,TP).
- 2<sup>ème</sup> cycle : Physiologie et Biomécanique (CM) et Méthodologie de la Recherche (TP).

#### *Année 1995-1996 : 192h ETD*

- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM), Anatomie Fonctionnelle (CM et TD) et Pratique Sportive Voile (TD,TP).

- 2<sup>ème</sup> cycle : Biomécanique (CM), Méthodologie de la Recherche (TP) et Physiologie de l'entraînement (TP).

*Année 1994-1995 : 189h ETD*

- 1<sup>er</sup> cycle: Fonction Neuromusculaire (CM) et Bioénergétique (TD) et Pratique Sportive Optionnelle Voile (TD,TP).
- 2<sup>ème</sup> cycle : Biomécanique (CM), Méthodologie de la Recherche (TP) et Pratique Sportive Optionnelle Voile (TP).

*Année 1993-1994 : Demi poste d'ATER (157h ETD)*

- 1<sup>er</sup> cycle: Physiologie Nerveuse et Musculaire (CM), Entraînement et Préparation Bio-Energétique du Sportif (CM,TD), Pratique Sportive Optionnelle Voile (TD,TP).
- 2<sup>ème</sup> cycle : Natation (TP).

### **Enseignements dans d'autres établissements**

- **IUFM (2004-08)** : Préparation au CAPEPS et à l'Agrégation interne  
« Bioénergétique et Système Neuromusculaire chez l'enfant et les adolescents » (1 CM par an).
- **Faculté de Médecine**
  - o *Master Sport Santé* (1CM en 2006 et en 2007), unité d'enseignement *Pathologies aiguës et chroniques induites par les activités physiques et sportives* : « Physiopathologie du tendon, du muscle et courbatures ».
  - o Unité commune à plusieurs *Masters de la Faculté de Médecine* (1 CM depuis 2006), unité d'enseignement *Bases de Physiologie biochimie et microbiologie* : « Le Muscle ».
  - o *Enseignement Post-Universitaire- FPC* - devenu *Formation Médicale Continue- FMC - en Traumatologie du Sport* (1 CM par an depuis 2003).
  - o *Capacité de Médecine et Biologie du Sport* : (1 CM par an depuis 2000) :  
« Physiopathologie du tendon du muscle et DOMS » ; « Physiopathologie du travail statique, concentrique et excentrique », « Travail musculaire excentrique dans le cadre de la rééducation fonctionnelle » « Effet de l'arrêt du sport sur le muscle ».

### **Enseignements à l'étranger et/ou à des étudiants étrangers**

- **Advanced Intensive Course / European Master of Sport Science**, Neuromuscular Research Center, Université de Jyväskylä, Finlande.
  - o 2007 : "Control of SSC and landing during extremely high loads" (C. Nicol, 1h).
  - o 2002 : "Reflex measurements applied to neuromuscular control of movement" (C. Nicol, 2h)
  - o 1999 : "Stretch induced force enhancement in human-muscle tendon complex" (C. Nicol, J. Avela, 7h).
  - o 1998 : "Fatigue induced muscle damage and changes in stretch-reflex excitability" (C. Nicol, 4h).
- **European School Marseilles (ESM)**, Faculté de Médecine, Université de la Méditerranée, Marseille, France.
  - o 2007 : "The functional role of active Ligaments".
  - o 2003-06 : « Active Ligaments & LCA Rupture: a potential vicious loop ».
  - o 2001 : « Muscle Evaluation ».
  - o 2000 : "What could favour or limit a maximal force production?"

### **Encadrement d'étudiants de Maîtrise / Master 1 en STAPS**

#### **Encadrement d'étudiants en Master 1 STAPS – Sciences et Technologies du Mouvement Humain:**

1. M. Thomas MOLEUR (2006-07). *Un impératif de la voile de compétition : la recherche de sponsors.*

#### **Encadrement d'étudiants en Maîtrise STAPS, filière Entraînement Sportif et Performance Motrice:**

1. M. Frédéric GRAPPIN (1999-2000). *Adaptations neuro-musculaires à la fatigue associée à un exercice de rebonds poursuivi jusqu'à épuisement.*
2. M. Gillel NAFATI (1999-2000). *Quantification de la fatigue neuromusculaire immédiate et retardée engendrée par un exercice maximal de type isocinétique excentrique.*
3. M. Philippe VANGEL (1998-99). *Effets de l'hyperthermie sur les caractéristiques neuromusculaires de force et d'endurance de force maximale du membre inférieur.*
4. Mlle Rola SBAT (1998-99). *Intérêts et limites des données isocinétiques et électromyographiques dans l'analyse des déficits fonctionnels de patients opérés du ligament croisé antéro-externe.*
5. M. Nicolas GOUBI (1997-98). *Adaptations neuro-musculaires aux perturbations mécaniques et sensorielles associées à la rupture et à la reconstruction du ligament croisé antéro-externe.*
6. Mlle Cécile GENTY (1997-98). *Mise au point d'un protocole d'évaluation des effets d'une reconstruction du LCAE sur le sens de la position du genou et sur les coordinations inter-musculaires.*
7. M. David IOLA (1996-97). *Evaluation de la fatigue neuromusculaire associée à un entraînement en ski alpin.*
8. Mlle Myriam ANTONIOL (1994-95). *Etude de l'influence de l'entraînement et de la fatigue sur les temps de phases d'un geste technique d'haltérophilie: l'arraché.*
9. M. Olivier MAISETTI (1993-94). *Etude électromyographique et cardiovasculaire du Pumping en laboratoire.*

### **Encadrement d'étudiants d'autres cursus**

#### **Mémoire de Maîtrise de Biologie Cellulaire et Physiologie, Option Neurosciences Approfondies, Aix-Marseille I**

1. Mlle Sophie REGUEME (2000-01). *Conséquences fonctionnelles d'une fatigue unilatérale de type excentrique sur les capacités d'activation et de force maximale : effets immédiats et retardés, ipsi- et controlatéraux.*

#### **Mémoire de Diplôme d'Etudes Spécialisées de Médecine Physique et de Réadaptation :**

1. M. Christian FLIN (1999-00). *Déficit de force musculaire après ligamentoplastie : le rôle du contrôle neuromusculaire.*

### III. ACTIVITES DE RECHERCHE

#### Thèmes de recherche

Liens entre recherche fondamentale et appliquée enrichissant le modèle de compréhension (i) du cycle-étirement détente (CED) dans la locomotion et (ii) des mécanismes d'adaptation du système neuromusculaire dans son ensemble en situations de perturbations internes et/ou externes prolongées.

#### Développement de ces 2 thèmes de recherche depuis 1988

##### I. Compréhension du fonctionnement du Cycle Etirement-Détente (CED) dans la locomotion :

###### **1998-92 Evolution du CED au cours d'un exercice épuisant de locomotion (Thèse) :**

- Quantification des déficiences fonctionnelles en cours d'effort et immédiatement après celui-ci.
- Mise en évidence des capacités d'adaptation de la commande nerveuse, notamment en situation de CED.
- Hypothèse d'une détérioration de la tolérance à l'impact liée à la moindre contribution du réflexe myotatique.

###### **1993- Rôle des boucles réflexes spinales dans la locomotion :**

- Quantification *in vivo* de la contribution du réflexe myotatique à la production de force mesurée au niveau du tendon d'Achille (jauge de contrainte puis fibre optique) suite à un étirement passif (1993-95).
- Résistance à l'étirement de la réponse mécanique réflexe (2003-).

*L'équipe de professeur P.V. Komi a enrichi ces études par la quantification in vivo de l'évolution des forces au niveau du tendon d'Achille et du tendon Patellaire dans de multiples activités locomotrices. L'utilisation récente de l'échographie à haute fréquence a permis de conforter l'hypothèse de l'intervention du réflexe myotatique par la démonstration d'un étirement des faisceaux musculaires des muscles mono- et bi-articulaires dans la phase initiale de freinage du CED.*

##### II. Capacité d'adaptation du système neuromusculaire en situation de perturbations internes prolongées : ajustements centraux et/ou réflexes

Ce thème a été essentiellement développé avec des étudiants de Maîtrise, de DEA et de Thèse dans son application aux domaines suivants :

###### **1997-99 La rééducation fonctionnelle :**

- après ligamentoplastie du ligament croisé antéro-externe du genou.

###### **1999- la locomotion :**

- **en état de déshydratation et d'hyperthermie engendrée par l'effort** (1999).
- **suite un exercice épuisant de type CED : récupération fonctionnelle biphasique** (1999-2006)
  - évolutions au cours du temps de l'activation et de la force maximales volontaires (1999-06).
  - évolution de l'activation au cours des 3 phases (préactivation, freinage, poussée) du CED (1999-02).
  - effets fonctionnels ipsi- vs. controlatéraux (2002-06)
  - influence sur le sens de la position et le sens de la vitesse (2004-06)
  - évolution de la sensibilité des réponses réflexes à l'étirement :
    - réponse réflexe à un étirement passif (1995-96)
    - réponse réflexe en situation de performances maximales de type CED (1995-02)
    - réponse réflexe en situation de performances sous-maximales de type CED (2001-07)

##### III. Mécanismes d'adaptation du système neuromusculaire en situation d'impacts élevés (2004-)

- **Caractérisation des ajustements inter-segmentaires en l'absence de fatigue :**
  - Pendant les phases pré- et post-impact
  - Selon les caractéristiques de l'impact (pic *versus* taux de charge)
  - Selon la tâche post-impact (amorti *versus* rebond)
- **Modifications de ces ajustements en situation de fatigue de type CED**
  - Ajustements centraux *versus* réflexes de l'activation musculaire

### Collaborations internationales

#### Séjours à l'étranger

- **Un séjour de 5 ans au sein du laboratoire du Pr. Paavo V. Komi (1988-93)**, Department of Biology of Physical Activity, Université de Jyväskylä, Finlande. Ce séjour a conduit à la soutenance de la thèse en septembre 1992 suivie d'un stage post-doctoral d'un an dans le même laboratoire.
- **Un séjour de 2 fois 2 mois dans ce même laboratoire (2001-2002)**. Ce séjour a permis de finaliser la rédaction de plusieurs articles portant sur des expérimentations réalisées lors de courts séjours antérieurs.
- **Les projets de recherche réalisés pendant ces 2 séjours ont été financés par :**
  - une bourse de 9 mois du Ministère Français des Affaires Etrangères (C. Nicol 1989)
  - des bourses du Ministère Finlandais de l'Education
    - \* Pr P.V. Komi (1988, 89, 91, 92, 96, 97, 98, 99, 2001, 02)
    - \* C. Nicol (1990)

#### Collaborations subventionnées par d'autres bourses de recherche:

- Avec les professeurs **P.V. Komi et T. Sinkjaer (décembre 1999)** des Universités de Jyväskylä (Finlande) et d'Aalborg (Danemark). Expérimentation relative à la « *Quantification de la contribution des boucles réflexes à la production de force à divers instants du cycle de marche* ».
  - Avec les professeurs **P.V. Komi et V. Strojnik (février 2000)** des Universités de Jyväskylä (Finlande) et de Ljubljana (Slovénie). Expérimentation relative à la « *Fatigue neuromusculaire de type Cycle-Etirement Détente engendrée par une semaine de ski alpin* ».
- Les 2 derniers projets ont été subventionnés par les Ministères de l'Education finlandais, danois et slovène.
- Avec le professeur **P.V. Komi (février 2004)** de l'Université de Jyväskylä (Finlande). Expérimentation relative aux « *Effets de la préactivation musculaire sur les caractéristiques mécaniques et réflexes de l'impact supramaximal* ».
- Projet subventionné par la Commission Européenne dans le cadre du projet HUMOS 2 (G3RD-CT-2002-00803).

### Encadrement d'étudiants en Doctorat et DEA/Master 2

#### Co-direction (en cours) d'étudiants en Doctorat STAPS :

1. Mlle GALINDO Amandine sur le thème « Stratégies inter-segmentaires d'ajustement neuromusculaire à la variation de contraintes externes et internes en situation d'impact élevé. ».
 

Codirection avec le Prof. Janne AVELA de l'Université de Jyväskylä en Finlande. Thèse dans l'attente d'une bourse de financement par le Ministère de l'Education finlandais.
2. Mr MORIO Cédric sur le thème « Effet de la fatigue sur la stabilité dynamique »: application aux principes d'amorti et de stabilité».
 

Thèse subventionnée par une bourse CIFRE.

#### Co-direction d'étudiants en Doctorat STAPS :

1. M. FTAITI Foued sur le thème « *Contribution à l'étude de l'hyperthermie lors d'une course prolongée* » (1996-00). Situation actuelle du diplômé : poste d'Assistant à l'Université de Sfax en Tunisie.

- Mlle REGUEME-LOUVEL Sophie sur le thème « *Conséquences neurophysiologiques de la fatigue musculaire excentrique* » (2002-06). Situation actuelle : poste d'Attaché Temporaire d'Enseignement et de Recherche à la Faculté des Sciences du Sport de Poitiers.

Co-direction d'étudiants inscrits à l'étranger - en Master of Sport Sciences en Finlande :

- Mlle GALINDO Amandine (2007-08): "Neuromuscular control during high impact loads in landing tasks" Neuromuscular Research Center, University of Jyväskylä, Finland.

Mémoire obtenu comme le Master avec la mention "Magna cum laude Approbatur » (très bien).

Encadrement (en cours) d'étudiants en Master 2 « Sciences du Mouvement Humain » en STAPS :

- Mr REY Olivier. *Efforts intermittents courts en natation versus en course à pied.*
- Mr MALFATTI Albéric. *Optimisation du suivi de la préparation physique en planche à voile olympique.*

Encadrement d'étudiants en DEA « Sciences du Mouvement Humain » en STAPS :

- Mlle Valérie GRANVILLE (1993-94). *Fatigue neuro-musculaire associée à un travail dynamique de flexion-extension des bras.*
- Mr Foued FTAITI (1996-97). *Fréquence cardiaque - température corporelle au cours d'un exercice de longue durée.*
- Mr Jean-Marie COUDREUSE (1997-98). *Troubles de la commande neuro-musculaire dans les lésions du ligament croisé antérieur opérées par ligamentoplastie.*
- Mr Nicolas GOUBI (1997-98). *Adaptations neuro-musculaire aux perturbations biomécaniques et sensorielles associées à la rupture et à la reconstruction du ligament croisé antéro-externe du genou.*
- Mlle Céline BEQUET (1998-99). *La reconfiguration des réseaux de neurones spinaux lors d'une fatigue induite par des actions musculaires de type excentrique.*
- Mlle Florie FALCHERO (2001-02). *Ajustements neuromusculaires suite à une fatigue unilatérale de type cycle étirement-détente.*

Articles publiés dans des revues internationales indexées (25 dont 23 après la thèse)

1991

- Nicol C., Komi P.V. & Marconnet P. (1991). Effects of marathon fatigue on running kinematics and economy. *Scand. J. Med. Sci. Sports* 1: 195-204.
- Nicol C., Komi P.V. & Marconnet P. (1991). Fatigue effects of marathon-running on neuromuscular performance. II. Changes in force, integrated electromyographic activity and endurance capacity. *Scand. J. Med. Sci. Sports* 1: 18-24.
- Nicol C., Komi P.V. & Marconnet P. (1991). Fatigue effects of marathon-running on neuromuscular performance. I. Changes in muscle force and stiffness characteristics. *Scand. J. Med. Sci. Sports* 1: 10-17

1996

- Horita T., Komi P.V., Nicol C. & Kyröläinen H. (1996). Stretch-shortening cycle fatigue: interactions among joint stiffness, reflex, and muscle mechanical performance in the drop jump. *Eur. J. Appl. Physiol.* 73(5): 393-403.
- Nicol C., Komi P.V., Horita T., Kyröläinen H. & Takala T.E.S. (1996). Reduced stretch-reflex sensitivity after exhaustive stretch-shortening cycle (SSC) exercise. *Eur. J. Appl. Physiol.* 72: 401-409.

1998

- Nicol C. & Komi P.V. (1998). Significance of passively induced stretch reflexes on Achilles tendon force enhancement. *Muscle and Nerve* 21: 1546-1548.

1999

- Pullinen T., Nicol C., MacDonald E. & Komi P.V. (1999). Plasma catecholamine responses to four resistance exercise tests in men and woman. *Eur. J. Appl. Physiol.* 80(2): 125-131.
- Horita T., Komi P.V., Nicol C. & Kyröläinen H. (1999). Effect of exhausting stretch-shortening cycle exercise on the time course of mechanical behavior in the drop jump: possible role of muscle damage. *Eur. J. Appl. Physiol.* 79(2): 160-167.

- **Nicol C.** & Komi P.V. (1999). Quantification of Achilles tendon force enhancement by passively induced dorsiflexion stretches. *J. Appl. Biom.* 15(3): 221-232.

#### 2001

- Ftaiti F., Dufлот J.C., **Nicol C.** & Grélot L. (2001). Tympanic temperature and heart rate changes in fire-fighters during treadmill runs performed with different fireproof jackets. *Ergonomics* 44(5): 502-512.
- Ftaiti F., Grélot L., Coudreuse J.M. & **Nicol C.** (2001). Combined effect of heat stress, dehydration and exercise on neuromuscular function in humans. *Eur. J. Appl. Physiol.* 84(1-2):87-94.

#### 2002

- Kuitunen S., Avela J., Kyröläinen H., **Nicol C.** & Komi P.V. (2002). Acute and prolonged reduction in joint stiffness after exhaustive SSC exercise. *Eur. J. Appl. Physiol.* 88(1-2): 107-116.
- Horita T., Komi P.V., **Nicol C.** & Kyröläinen H. (2002). Interaction between pre-landing activities and stiffness regulation of knee joint musculoskeletal system in the drop jump: implication to performance. *Eur. J. Appl. Physiol.* 88(1-2): 76-84.

#### 2003

- **Nicol C.**, Kuitunen S., Kyröläinen H., Avela J. & Komi P.V. (2003). Immediate and delayed fatigue effects on reflex EMG and force responses of the muscle-tendon complex. *Eur. J. Appl. Physiol.* 90(5-6): 470-479.
- Linnamo V., Moritani T., **Nicol C.** & Komi P.V. (2003). Motor unit activation during isometric, concentric and eccentric actions at different force levels. *J. Electromyogr. & Kinesiol.* 13(1): 93-101.

#### 2004

- Coudreuse J.M., Dupont P. & Nicol C. (2004). Douleurs musculaires post-effort. *Ann. Réadapt. Med. Phys.* 47 : 290-298.

#### 2005

- Bottas R., Linnamo V., **Nicol C.** & Komi P.V. (2005). Repeated maximal eccentric actions cause long-lasting disturbances in movement control. *Eur. J. Appl. Physiol.* 94: 62-69.
- Regueme S.C., **Nicol C.**, Barthélemy J. & Grélot L. (2005). Acute and delayed neuromuscular adjustments of the triceps surae muscle group to exhaustive stretch-shortening cycle fatigue. *Eur. J. Appl. Physiol.* 93: 398-410.

#### 2006

- **Nicol C.**, Avela J. & Komi P.V. (2006) Stretch-shortening cycle (SSC): a model to study naturally occurring neuromuscular fatigue. *Sports Medicine* 36(11): 977-999. Review.
- Ishikawa M., Dousset E., Avela J., Kyröläinen H., Kallio J., Linnamo V., Kuitunen S., **Nicol C.** & Komi P.V. (2006) Changes in the soleus muscle architecture after exhausting stretch-shortening cycle exercise in humans. *Eur. J. Appl. Physiol.* 97(3): 298-306.
- Ftaiti F., Dantin M.P., **Nicol C.**, Brunet C., Valli M., Komi P.V. & Grélot L. (2006) The effect of desmopressin, a vasopressin analog on endurance performance during a prolonged run in simulated heat conditions. *Appl Physiol Nutr Metab.* 31(2): 135-43.

#### 2007

- Bottas R., **Nicol C.**, Komi P.V & Linnamo V. Adaptive changes in motor control of rhythmic movement after maximal eccentric actions. *J. Electromyogr. Kinesiol.* 2007 Oct 13; [Epub ahead of print].
- Regueme S.C., Barthélemy J., Gauthier G.M. & **Nicol C.** (2007) Changes in illusory ankle movements induced by tendon vibrations during the delayed recovery phase of stretch-shortening cycle fatigue: An indirect study of muscle spindle sensitivity modifications. *Brain Res.* 1185:129-35.
- Regueme S.C., Barthélemy J. & **Nicol C.** (2007) Exhaustive stretch-shortening cycle exercise: no contralateral effects on muscle activity in maximal motor performances. *Scand. J. Med. Sci. Sports.* 17(5):547-55.

#### 2008

- Regueme S.C., Barthélemy J., Gauthier G.M., Blin O. & **Nicol C.** (2008) Delayed influence of stretch-shortening cycle fatigue on large ankle joint position coded with static positional signals. *Scand. J. Med. Sci. Sports.* 18(3):373-82.



### Chapitres d'ouvrages internationaux avec comité de lecture (9 dont 8 après la thèse)

#### 1992

- Komi P.V., **Nicol C.** & Marconnet P. (1992). Neuromuscular fatigue during repeated stretch-shortening cycle exercises. In: *Marconnet P., Komi P.V., Saltin B., Sejersted O.M. (Eds.), Muscle Fatigue Mechanisms in Exercise and Training. Med. Sport Sci., Basel*, vol. 34, pp. 172-181.

#### 1996

- **Nicol C.** & Komi P.V. Neuromuscular fatigue in stretch-shortening cycle exercises. In : *P. Marconnet, B. Saltin, P.V. Komi, and J. Poortmans (Eds.), Human Muscular Function during Dynamic Exercise. Med. Sport Sci. Basel, Karger*, vol. 41, pp. 134-147.

#### 2000

- Komi P.V. & **Nicol C.** Stretch-shortening cycle of muscle function. In: *V. Zatsiorsky (Eds.) Biomechanics in Sport: performance enhancement and injury prevention, The Encyclopedia of Sports Medicine, Blackwell Science*, Chap.5, pp.87-102.
- Komi P.V. & **Nicol C.** Stretch-shortening cycle fatigue. In: *B.M. Nigg, B.R. MacIntosh and Mester J.(Eds.) Biomechanics and Biology of Movement, Human Kinetics, II*, Chap. 20, pp.385-408.

#### 2001

- Strojnik V., **Nicol C.** & Komi P.V. Fatigue during one-week tourist alpine skiing. In: *E. Müller et al. (eds.) Science and Skiing II, Kovač, Hamburg*, Chap. 46, pp. 599-607.

#### 2003

- **Nicol C.** & Komi P.V. Stretch-shortening Cycle Fatigue and its Influence on Force and Power Production. In: *P.V. Komi (Ed.) Strength and Power in Sport. The Encyclopedia of Sports Medicine, Blackwell Science*, Chap.11, pp. 203-228.

#### 2005

- Regueme S., Barthèlémy J., Gauthier G.M. & **Nicol C.** Central nervous system contralateral adjustments after unilateral muscle fatigue. In: *Gantchev (Ed.), From Basic Motor Control to Functional Recovery IV, Marin Drinov Academic Publishing House, Sofia*. pp. 300-307.

#### 2008

- Komi P.V. & **Nicol C.** Neuromuscular adaptation to exercise. In: *Nigel Taylor, H. Groeller and PL McLennan (Eds.) Physiological Bases of Human Performance during Work and Exercise. Churchill Livingstone*, Section 1, Chap.4, pp. 71-89.
- Taylor J., Komi P.V. & **Nicol C.** Central and Neuromuscular fatigue. In: *Nigel Taylor, H. Groeller and PL McLennan (Eds.) Physiological Bases of Human Performance during Work and Exercise. Churchill Livingstone*, Section 1, Chap.5, pp. 91-113..

### Articles de vulgarisation dans des revues à comité de lecture (6)

#### 1994

- Bosco C., Komi P.V., Bosco E., **Nicol C.**, Pulvirenti G., & Caruso I. (1994) Influence of training on mechanical and biomechanical profiles of athlete's muscles. *Coaching & Sport Sci J*, 1(1) : 8-13.

#### 1997

- Benezet P., **Nicol C.**, & Delarque A. (1997). Evaluation de la fatigue musculaire par isocinétisme. *Sport Med'*, 97 : 9-10.

#### 1998

- Ftaiti F., Dufлот J.C., **Nicol C.**, & Micallef G. (1998). Evolution comparée de la fréquence cardiaque et de la température corporelle au cours de l'exercice prolongé. *Médecine du Sport*, 72(1) : 13-18.

#### 2000

- Coudreuse J.M., **Nicol C.**, Gouby N., Viton J.M., Fondarai J., Delarque A., & Brunet C. (2000). Trouble de la commande neuro-musculaire dans les lésions du ligament croisé antérieur opérées par ligamentoplastie: intérêt du couplage isocinétisme-électromyographie. *J Traumatol Sport*, 17:132-148.

#### 2001

- **Nicol C.**, Gouby N., Coudreuse J.M., Flin C., Viton J.M., Delarque A., Brunet C., & Komi P.V. (2001). Activation and torque deficits in ACL-reconstructed patients 4 months post-operatively. *Eur J Sports Sci*, 1(2). (Electronic journal).

#### 2005

- Pithioux M., Chavet P., St-Onge N. & Nicol C. (2005) Influence of muscle preactivation of the lower limb on impact dynamics in case of frontal collision. *International Journal of Crashworthiness*, Vol. 10(6): 1-9.

### Conférences invitées internationales

#### *Congrès Internationaux (13)*

##### 1995

- Nicol C. & Komi P.V. (1995). Neuromuscular fatigue induced by short and long-lasting stretch-shortening cycle exercises. *5th International Symposium on Exercise and Sport Biology*. Nice, February 9-11, France.

##### 1996

- Nicol C., Komi P.V. & Avela J. (1996). Fatigue effects of exhaustive SSC exercise on reflex excitability. *1st Congress of the European College of Sport Sciences*. Nice, May 28-31, France.

##### 1997

- Nicol C. & Komi P.V. (1997). Surface EMG can be used to demonstrate that stretch-reflexes play an important role in normal locomotion, and vary in case of fatigue. *2nd General Workshop of SENIAM (Surface EMG for the Non Invasive Assessment of muscles)*, Var Gard, Saltsjöbaden, June 12-15, Sweden.

##### 1998

- Nicol C. & Komi P.V. (1998). Contribution of short-latency reflexes to force production during passive stretches. *The 3rd World Congress of Biomechanics (WCB'98)*. Hokkaido University, Sapporo, August 2-8, Japan.
- Nicol C., Komi P.V., Finni T., & Lepola V. (1998). Sarcomere kinetics and stretch induced force enhancement in human skeletal muscle. *4th Scandinavian Congress on Medicine and Science in Sports*, Lahti, 5-8 November, Finland.
- Nicol C., Komi P.V., Finni T., & Lepola V. (1998). Contribution of short-latency reflexes to force production during passive stretches. *4th Scandinavian Congress on Medicine and Science in Sports*, Lahti, 5-8 November, Finland.

##### 1999

- Nicol C. & Komi P.V. (1999). Mechanism of neuromuscular fatigue. *4th Annual Congress of the European College of Sport Sciences*. Rome, July 14-17, Italy.

##### 2000

- Nicol C. & Komi P.V. (2000). Stretch-induced force enhancement in human. *Neuromuscular control in human movements, satellite work shop from the Osaka Biomechanics and Physiology Joint Meeting 2000*. Osaka, July 27-28, Japan.
- Nicol C. & Komi P.V. (2000). Characteristics of SSC type fatigue. *46th Annual Meeting of the American College of Sports Medicine*. Seattle, June 2-5, USA.

##### 2001

- Nicol C., Komi P.V., & Avela J. (2001). Immediate and delayed deficiencies in force and stiffness regulation after fatiguing SSC exercise. *5th Annual Congress of the European College of Sport Sciences*. Köln, July 28-31, Germany.

##### 2005

- Nicol C., Regueme S., Dousset E., Avela J., Komi P. (2005). "Markers" of neuromuscular strategies to reveal short-term overloading of intensive SSC exercise". *10th Annual Congress of the European College of Sport Sciences*. Belgrade, July 13-16, Serbia-Montenegro.

##### 2006

- Nicol C., Ishikawa M., Cronin N., Avela J., Komi P. (2006). Influence of exhaustive fatigue on stretch response of the muscle-tendon unit. *11th Annual Congress of the European College of Sport Sciences*. Lausanne, July 4-9, Suisse.

##### 2007

- Nicol C., Avela J., Ishikawa M., Komi P.V. (2007) Neuromuscular fatigue caused by exhaustive SSC exercise. *12th Annual Congress of the European College of Sport Sciences*. Jyväskylä, July 11-14, Finland.

#### *Autres conférences invitées (18)*

##### 1997

- **Nicol C.**, Komi P.V., Benezet P., Viton J.M., Coudreuse J.M., & Delarque A. (1997). Les différents aspects de la fatigue neuromusculaire. *34<sup>èmes</sup> Journées Médicales de la Fédération Française de Basket Ball*, Marseille, 12-14 septembre, France.
- **Nicol C.** & Komi P.V. (1997). Effets de la fatigue neuromusculaire sur la tolérance des impacts au sol. *Les Journées de Médecine Physique et de Réadaptation de Marseille 97*, Marseille, 26-27 septembre, France.

#### 1998

- **Nicol C.** & Komi P.V. (1998). Biomécanique tendineuse. *1<sup>ère</sup> Rencontre de Traumatologie du Sport de Marseille*, Marseille, 16 Mai, France.
- **Nicol C.**, Coudreuse J.M., & Delarque A. (1998). Evaluation fonctionnelle après lésion du ligament croisé antéro-externe chez le footballeur. *Médecine Physique et Football*, Toulouse, 20 juin, France.
- **Nicol C.** & Komi P.V. (1998). Effet de la fatigue neuro-musculaire sur la tolérance des impacts au sol. *Le Pied du Sportif*, Toulouse, 16-17 octobre, France.
- **Nicol C.** (1998). Intérêt du couplage de l'enregistrement électromyographique aux évaluations isocinétiques. *Les Journées de Médecine Physique et de Réadaptation de Marseille 98*, Marseille, 23-24 Octobre, France.
- **Nicol C.** & Komi P.V. (1998). Stretch induced force enhancement in single and in successive passive stretches of human skeletal muscles. *Aalborg University, Center for Sensory-Motor Interaction*, 14 December, Denmark.

#### 2003

- **Nicol C.**, Regueme S., Dousset E., & Barthélémy-Montfort J. (2003). Ajustements de la commande motrice à la fatigue musculaire d'origines chimique et mécanique. *5<sup>ème</sup> Réunion du Club du Motoneurone : Le motoneurone dans tous ses états...*, 26-28 février, Marseille.
- **Nicol C.** (2003). Ligaments Actifs & Rupture du Ligament Croisé Antéro-externe: un cercle vicieux potentiel. *Les Premières Rencontres Sport et Santé de l'Olympique de Marseille*, Marseilles, 24 mai, France.
- **Nicol C.** (2003). Ajustement du système nerveux en cas de courbatures musculaires. *Neuroméditerranée V. Ouarzazate*, 10-11 Octobre, Maroc.

#### 2004

- **Nicol C.** (2004). Dysfonctions neuromusculaires avant et après rupture du LCAE. *Séminaire inter-facultés : Le genou du sportif*, Marseille, 28-29 janvier, France.
- **Nicol C.** (2004). Perturbations de l'activation musculaire après rupture du pivot central. *Les Journées de Rééducation du Genou*, Marseille, 15 mai, France.

#### 2005

- Coudreuse J.M., Dupont P., **Nicol C.**, Viton J.M., Delarque A. (2005). Douleurs musculaires post-effort. *20th Congrès National de la SOFMER*, Dijon, 13-15 octobre, France (Annales de Réadaptation et de Médecine Physique. 48(7) : 421-580).

#### 2006

- **Nicol C.** (2006). Qualités neuromusculaires du sportif vieillissant. *9<sup>ème</sup> rencontres de traumatologie du sport : spécificité du sportif après 40 ans*, Marseille, 11-13 mars, France.

#### 2007

- **Nicol C.** & Komi P.V. (2007). Muscle et Sport. *17<sup>ème</sup> Congrès de la Société Française de Traumatologie du Sport*. Toulouse, 29-31 mars, France.
- Coudreuse J.M., **Nicol C.**, Viton JM, Delarque A (2007). Place des étirements chez le sportif. *22<sup>ème</sup> Congrès National de la SOFMER*, Saint-Malo, 4-6 octobre, France.

#### 2008

- **Nicol C.** & Coudreuse J.M. (2008). Raideur musculo-tendineuse. *11<sup>èmes</sup> rencontres de Traumatologie du Sport de Marseille « Facteurs de risque de blessure du sportif : idées reçues et réalité scientifique »*, Marseille, 8 mars, France.
- **Nicol C.** (2008). Raideur musculo-tendineuse. *Les Rencontres du Comité régional Olympique et Sportif « Préparation Physique et Prévention des Blessures*», Aix-en-Provence, 7 juin, France.
- **Nicol C.** (2008). Force et Activation musculaires. *1<sup>ère</sup> Journée de Kinésithérapie du Sport de Marseille*, Marseille 4 décembre, France.

**Communications affichées en congrès internationaux avec actes impliquant  
des étudiants co-encadrés en DEA ou en Thèse**

**2000**

- Ftaiti F., Nicol C. & Grélot L. (2000). Combined effect of heat stress, dehydration and exercise on neuromuscular function. *5<sup>th</sup> Annual Congress of the European College of Sport Sciences*. Jyväskylä, July 19-23, Finland.

**2001**

- Ftaiti F., Dantin M.P., Tabka Z., Nicol C., & Grélot L. (2001). The effect of desmopressin, a vasopressin analogue, on performance during a prolonged run performed in simulated heat conditions. *6<sup>th</sup> Annual Congress of the European College of Sport Sciences*. Köln, July 19-23, Germany.

**2003**

- Regueme S.C., Barthélémy J., Grélot L., & Nicol C. (2003). Neuromuscular adjustments during and after an exhaustive stretch-shortening cycle exercise. *8<sup>th</sup> Annual Congress of the European College of Sport Sciences*, Salzburg, July 9-12, Austria.

**2005**

- Regueme S.C., Barthélémy J., & Nicol C. (2005). Task-dependent and Time-dependent contralateral neural adjustments after a unilateral SSC fatiguing exercise. *10<sup>th</sup> Annual Congress of the European College of Sport Sciences*, Belgrade, July 13-16, Serbia-Montenegro.

**Autres communications affichées en congrès internationaux avec actes (29)**

**1990**

- Nicol C., Komi P.V. & Marconnet P. (1990). Marathon running induces dramatic reductions of the neural activation and force production of the knee extensor muscles. *4<sup>ème</sup> Symposium International de Biologie de l'Exercice et de l'Entraînement Physique*, Nice, 1-4 Novembre, France.

**1991**

- Nicol C., Komi P.V. & Marconnet P. (1991). Combined effects of marathon race on running kinematics and neuromuscular function. *XIII<sup>th</sup> International Congress on Biomechanics (ISB)*, Perth, 9-13 December, Western Australia.

**1992**

- Nicol C., Komi P.V. & Marconnet P. (1992). Effect of a marathon induced muscular impairment on SSC muscle action. *Malaga Olympic Scientific Congress*, Benalmadena, 14-19 July, Spain.

**1993**

- Nicol C., Komi P.V., Horita T., Kyröläinen H. & Takala T.E.S. (1993). SSC fatigue effects on stretch-reflex sensitivity. *XIV<sup>th</sup> International Congress on Biomechanics (ISB)*. Paris, 4-8 Décembre, France.

**Cette présentation a reçu « Young Investigator Award for the Best Podium Presentation ».**

**1994**

- Nicol C., Komi P.V., Gollhofer A. & Jarvinen M. (1994). Reflex contribution to Achilles tendon force during controlled stretches. *American College of Sports Medicine (A.C.S.M.) Annual Meeting*, Indianapolis, 1-4 June, Indiana, USA.
- Nicol C., Avela J. & Komi P.V. (1994). Reduced reflex sensitivity after exhaustive stretch-shortening cycle exercise. *Symposium on the Neural and Neuromuscular Aspects of Muscle Fatigue*, Miami, 10-13 November, Florida, USA.
- Horita T., Komi P.V. & Nicol C. & Kyröläinen H. (1994). Interaction between stiffness and stretch reflex in SSC fatigue *Symposium on the Neural and Neuromuscular Aspects of Muscle Fatigue*, Miami, 10-13 November, Florida, USA.

**1995**

- Nicol C., Komi P.V., Belli A., Huttunen V., & Partio E. (1995). Reflex contribution to Achilles tendon forces: in vivo-measurements with the optic fiber technique. *XV<sup>th</sup> International Congress on Biomechanics (ISB)*. Jyväskylä, July 2-6, Finland.
- Gollhofer A., Komi P.V., Voigt M., & Nicol C. (1995). Mechanical contribution of isolated stretch evoked EMG-responses. *XV<sup>th</sup> International Congress on Biomechanics (ISB)*. Jyväskylä, July 2-6, Finland.

- Horita T., Komi P.V. & **Nicol C.** (1995). SSC fatigue ; interaction between joint stiffness regulation and muscle elasticity during drop jump. *XVth International Congress on Biomechanics (ISB)*. Jyväskylä, July 2-6, Finland.

#### 1996

- **Nicol C.**, Avela J. & Komi P.V. (1996). Stretch-shortening cycle fatigue reduces stretch reflex response. *The 1996 International Pre-Olympic Scientific Congress*, Dallas, July 10-14, USA.
- Komi P.V., **Nicol C.** & Avela J. (1996). Fatigue effects of exhaustive SSC exercise on stretch reflex response. *First General SENIAM Workshop*, Torino, 6-7 Septembre, Italy.

#### 1997

- **Nicol C.** & Komi P.V. (1997). Effects of repeated exhaustive stretch-shortening cycle exercise (SSC) on short latency reflex responses. *The 1997 ACSM Annual Meeting*, Denver, May 28-31, USA.
- **Nicol C.**, Bonnard M. & Pailhous J. (1997). An alternative agonist-antagonist relationship in the absence of proprioceptive informations. *Second Congress of the European College of Sport Sciences*. Copenhagen, August 20-23, Denmark.
- **Nicol C.** & Komi P.V. (1997). Combined stretch and reflex potentiations depend on their respective timing. *XVIIth International Congress on Biomechanics (ISB)*. Tokyo, August 25-29, Japon.

#### 1998

- Komi P.V., Avela J. & **Nicol C.** (1998). Neuromuscular control and exercise induced muscle soreness. *The third World Congress of Biomechanics (WCB'98)*. Hokkaido University, Sapporo, August 2-8, Japan.

#### 1999

- Voigt M., Komi P.V., **Nicol C.**, Andersen J.B., Haase J. & Sinkjaer T. (1999). Stretch reflex contribution to the Achilles tendon force during sitting and walking. *Neural Control of Movement (CNM)*. Hawaii, April 11-16, USA.
- **Nicol C.**, Komi P.V, Finni T. & Lepola V. (1999). Stretch induced force enhancement in human skeletal muscle. *ACSM's 46th Annual Meeting*, Seattle, June 2-5, USA.
- **Nicol C.**, Finni T. & Komi P.V. (1999). Prolonged deficiencies in force and stiffness regulation after exhaustive SSC exercise. *Fourth Annual Congress of the European College of Sport Sciences*, Rome, July 14-17, Italie.
- **Nicol C.**, Finni T., Kyröläinen H., Avela J & Komi P.V. (1999). Immediate and delayed fatigue effects on reflex-induced force response of the muscle-tendon complex. *Satellite Symposium of the XVIIth ISB Congress on Skeletal Muscle Mechanics*, Canmore, August 6-7, Alberta, Canada.

#### 2001

- Sares F., Merhi O., Bresciani J.P., Barthèlemy J., **Nicol C.**, Bourdin C., Gauthier G.M. (2001). Control of posture in a changing gravito-inertial field. *Symposium Espace et Action*, 22-24 Mars 2001, Bron, France.
- Sares F., Merhi O., Gauthier G.M., **Nicol C.**, Barthèlemy J. (2001). Contrôle de la posture en champ gravito-inertiel modifié. *IX ième Congrès de l'ACAPS*, 1-3 Novembre 2001, Grenoble, France.

#### 2004

- Chavet P, Pithioux M, St-Onge N & **Nicol C.** (2004) Influence of muscular preactivation on the impact mechanical characteristics. *Congrès International de la Société Française de Biomécanique*, Septembre 4-7, Paris, France.

#### 2006

- Ishikawa M., Vincent M., Kanervo M., Peltonen J., **Nicol C.**, Chavet P., Komi P.V. (2006). Repetitive hopping exercise alters the skeletal muscle architecture and the fascicle and tendon interaction. *11th Annual Congress of the European College of Sport Sciences*. Lausanne, July 4-9, Suisse.
- Avela J., Ishikawa M., **Nicol C.**, Chavet P., Peltonen J. & Komi P.V. (2006) Neuromuscular control in extreme high impact loads. *5th World Congress of Biomechanics*. Zurich, July 29 – August 4, Germany.

#### 2007

- Chavet P., Barthèlemy J., **Nicol C.**, Gautrand E., Ishikawa M., Avela J., Komi P.V. (2007) Modulation of centrally programmed activation in (crash type) landing. *12th Annual Congress of the European College of Sport Sciences*. Jyväskylä, July 11-14, Finland.
- Avela J., **Nicol C.**, Linnamo V., Komi P.V. (2007) Reflex adaptation and increased muscle compliance during stretch-shortening cycle (SSC) exercise. *12th Annual Congress of the European College of Sport Sciences*. Jyväskylä, July 11-14, Finland.

- Cronin N., Ishikawa M., **Nicol C.**, Komi P.V., Avela J. (2007) Passive stretch-induced fascicle behaviour in the human soleus muscle. *12th Annual Congress of the European College of Sport Sciences*. Jyväskylä, July 11-14, Finland.

#### **Membre de Sociétés Savantes**

- 1992-1996: International Society of Biomechanics (I.S.B.)  
 1998-1999 : Association des Chercheurs en Activités Physiques et Sportives (A.C.A.P.S.)  
 1993-2006: American College of Sports Medicine (A.C.S.M.)  
 1996- : European College of Sport Sciences (E.C.S.S.)  
**1999- : Membre du Comité Scientifique du Collège Européen des Sciences du Sport (E.C.S.S.)**  
 (préparation du programme scientifique, gestion de symposiums et jugement du Young Investigation Award lors des congrès annuels).

#### **Expertises de manuscrits**

Une vingtaine d'expertises pour BJSM, EJAP, IJSM, SJMS, JAB, MSSE.

#### **Membre de jurys**

##### *Thèses étrangères*

1. Expertise du manuscrit et membre du jury de la Thèse d'Etat de Mr Janne Avela sur le thème: Stretch-Reflex Adaptation in Man: interaction between load, fatigue and muscle stiffness. Faculty of Sport and Health Sciences, University of Jyväskylä, December 1998, Jyväskylä, Finland. Jury: Dyrhe-Poulsen P. (Danemark), Nicol C. (France).
2. Expertise du manuscrit de Thèse de Mlle KA Sharwood sur le thème: The effects of endurance training on neuromuscular characteristics in masters runners. University of Cape Town, 12/2003, South Africa.

##### *Thèses françaises*

1. Membre du jury de la Thèse en Motricité Humaine et Handicap de l'Université de St Etienne, soutenue le 7 décembre 2007 par Mr Karim Zameziati sur le thème: « Facteurs biomécaniques de la performance lors de différents modes de contraction : influence de l'efficacité de pédalage et du temps de couplage sur le rendement mécanique ». Jury: Pr Antonutto G., Pr Belli A., Pr Minetti A.E., Pr di Prampero P.E., Dr Hautier C., Dr Cloutier-Hintzy F., Dr Nicol C.
2. Membre du jury de la Thèse en Sciences du Mouvement Humain de l'Université Montpellier I, soutenue le 25 septembre 2008 par Mr Thomas Rupp sur le thème: « Responsabilité de la commande motrice centrale dans la fatigue neuromusculaire ». Jury: Pr Kayser B., Pr Millet G., Pr Grebe R., Pr Perrey S., Dr Nicol C.

##### *Autres jurys :*

- Membre du jury à l'évaluation finale des stagiaires de la Formation au Brevet d'Etat « Aptitude à l'Enseignement de la Culture Physique et de Culturisme » (B.E. A.E.C.P.C. , Jeunesse et Sports).
- Membre du jury au Concours Interne d'Educateur Territorial.
- Membre du jury à l'évaluation finale des stagiaires de la Formation au B.E. des Métiers de la Forme, D.D.J.S. du Vaucluse.
- Membre du jury de mémoires de DEUST, de Licence Professionnelle et de Maîtrise en STAPS.

#### **Coopérations industrielles et valorisation (Contrats, dépôts de brevets, logiciels)**

1999: *Subvention du Conseil Général des Bouches-du-Rhône* : « Modifications de la commande motrice consécutives à des traumatismes de l'appareil moteur »

- Responsables scientifiques : C. Nicol et A. Delarque (170 KF).
- 2000: *Participation à l'obtention d'une Convention DGA-DSP 00.34.029.00.470.75.01. « Commande et régulation motoneuronale »*  
- Responsables scientifiques : JP Vedel, D. Zytnicki, L. Grélot  
1200 KF/ 2 ans répartis sur 3 unités de recherche (soit 400 KF/2 ans par unité).
- 2000: *Participation à l'obtention d'une subvention du Conseil Général des Bouches-du-Rhône : « Régulations thermique et hydrominérale au cours d'une course prolongée en ambiance thermique chaude : modifications hormonales et actions de la desmopressine »*  
- Responsable scientifique : L. Grélot (50 KF).
- 2002 : *Participation à un Contrat Européen: Human Model for Safety Two (HUMOS2). « Contribution au développement d'un modèle biomécanique permettant d'étudier les réactions d'un être humain assis dans une voiture lors de collisions »*  
- Projet de type GROW ; Responsable scientifique : C. Brunet (enveloppe globale 83KEuros)  
- Partenaires: INRETS, Chalmers Univ. of Technology AB, Technical Univ. of Eindhoven, Engineering System International GmbH, Faurecia, Univ. of Heidelberg, Sociétés d'Etudes et de recherches de l'ENSAM Mecalog Sarl Univ. de la Méditerranée, Institute für Rechtsmedizin der Univ. München, Peugeot Citroën, Automobiles, Regienov-Renault Recherche Innovation Netherlands Organisation for Applied Research, Volvo Car Corporation, Volkswagen AG.
- 2008 : *Participation à l'obtention d'une Chaire d'Excellence entre Décathlon-Performance Motrice et Modélisation.*  
- Responsables scientifiques : P. Freychat et E. Berton. (300 K€/ 3 ans).

#### IV. ACTIVITES ADMINISTRATIVES : FONCTIONS D'INTERET COLLECTIF

- 1995 : Création d'un « Module de Préparation Physique » au sein de l'établissement visant, par la pratique et la réflexion sur celle-ci, à doter l'étudiant d'outils lui permettant de comprendre les principes élémentaires du développement de la condition physique.
- 1998 : Dans le cadre de la réforme du DEUG mise en place à la rentrée universitaire 1998, ce module a été transformé en un enseignement relatif aux « Méthodes de gestion de la vie physique et de la santé de l'étudiant ». Visant à permettre à l'étudiant d'acquérir une condition physique suffisante pour entamer des études en STAPS en toute sécurité.
- 2002-08: **Co-directrice du DEUST STAPS** intitulé «Agent de Développement Local Socio-Sportif » de la Faculté des Sciences du Sport de l'Université de la Méditerranée.
- 2002-08: **Membre nommé de la Commission de Spécialistes de l'établissement** (CSE, 74<sup>ème</sup> section).
- 2008- : **Membre élu du Conseil de Laboratoire.**
- 2008-12: **Co-responsable d'une Licence Professionnelle en STAPS** intitulée «Gestion et développement des organisations, des Services sportifs et de loisirs » - Métiers du littoral - à la Faculté des Sciences du Sport de l'Université de la Méditerranée.

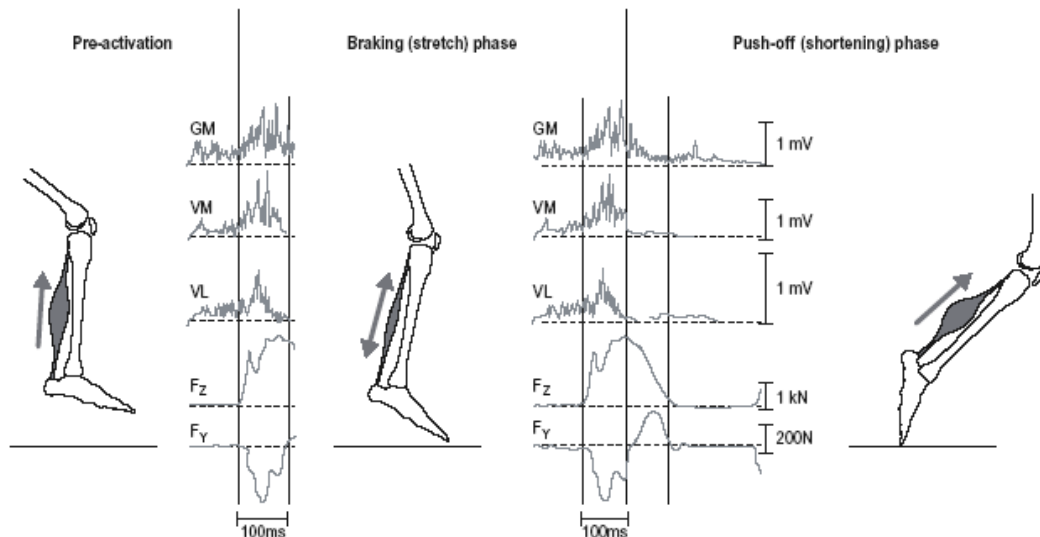
#### V. AUTRES DIPLOMES

- 1984 : **Brevet National de Secourisme et de Ranimation**
- 1984 : **Moniteur Fédéral de Voile (planche à voile).**
- 1987 : **Brevet National de Sécurité et de Sauvetage Aquatique**

## **1. Stretch-shortening cycle (SSC): a natural form of locomotion and power production**

### **1.1. The nature of the stretch-shortening cycle**

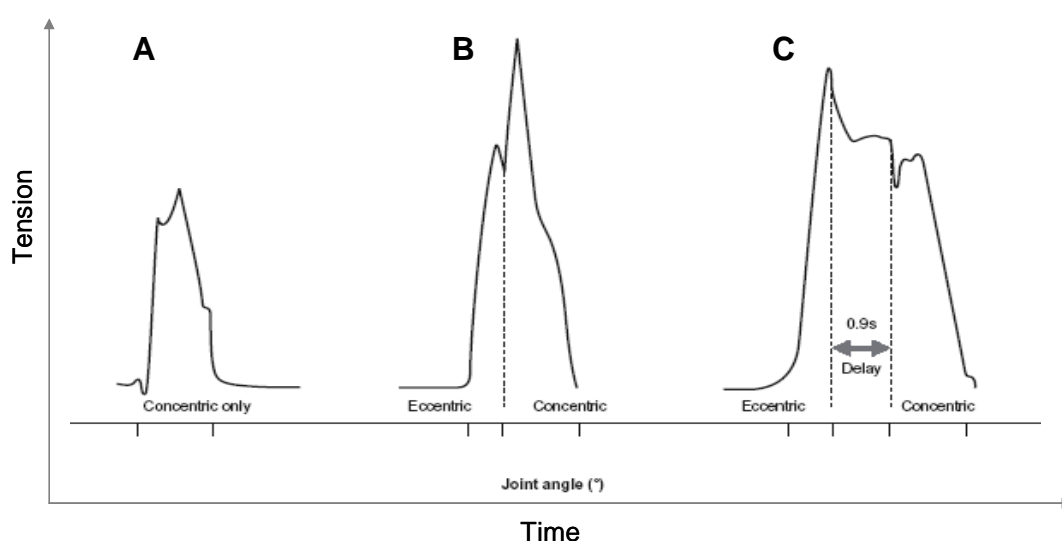
As illustrated in Fig. 1. SSC refers, by definition, to a series of consecutive muscle actions starting with preactivation (100-200 ms prior to ground contact). The *preactivation* amplitude is a function of both the expected impact load, with a positive relationship observed between gastrocnemius preactivation and running velocity or impact load (Komi et al. 1987a). The greatest EMG activity is measured during the *braking phase*, with most of the lower limb extensor muscles experiencing eccentric action when the muscle-tendon unit lengthens. This active braking (eccentric) phase is followed, without delay, by the *push-off phase*, usually performed through concentric (shortening) muscle action, but with very low EMG activity. The nature of this final push-off phase can be illustrated as a recoil action resulting in low metabolic activity, but high mechanical efficiency. Consequently, SSC is important for locomotion, since it takes up the unnecessary delays in the force-time relationship by bringing the pre-activated force up to the level necessary to meet the expected impact loading. It also helps the concentric action (push-off) produce peak power (maximal efforts) or to generate force more economically (submaximal efforts). Running, jumping and hopping are typical examples of SSC actions.



**Fig. 1. The stretch-shortening cycle (SSC) during the functional contact phase of running.** The drawings illustrate simultaneously recorded vertical ( $F_z$ ) and horizontal ( $F_y$ ) ground reaction forces, together with EMG activities of the gastrocnemius medialis (GM), vastus medialis (VM) and lateralis (VL) muscles. The vertical lines denote beginning of ground contact, end of braking phase and end of push-off phase. Note that especially gastrocnemius medialis (GM) shows high preactivation, with all muscles showing low activity during push-off. (Adapted from Komi 2000).



Cavagna and colleagues were among the first ones to describe the mechanisms of this performance potentiation in experiments on isolated frog *sartorius* muscle (Cavagna et al. 1965), and later in human forearm flexors (Cavagna et al. 1968). Fig. 2 illustrates knee extensor tension (Komi 1983), and shows that the time delay (coupling time) between stretch (eccentric) and shortening (concentric) has influences on force and power output in the concentric phase of the cycle. That is, a short coupling time enhances performance. In addition, a shorter coupling time also improves economy (Aura and Komi 1986). The mechanisms of this potentiation will involve the entire muscle-tendon unit: fascicles and tendinous tissue.



**Fig. 2. Three force-time traces for knee extension (100-175°); all performed with maximal voluntary activation. (A)** A pure maximal concentric action. **(B)** Concentric activation is preceded by an eccentric (stretching) activation, but no delay is allowed between these actions; note the force enhancement. **(C)** An identical action, but with a time delay between stretching and shortening; concentric force potentiation is reduced. (Adapted from Komi 1983)

## 1.2. Indirect estimation of the force-length changes

For understanding muscle function during locomotion, it is important to know how muscles are producing force and power as a function of muscle length and velocity of shortening and lengthening, for example. Until recently, it was very difficult to measure much more than the force-angle relationship *in situ*, forcing the estimation of force-length changes. The indirect approach can refer to methods such as the mathematic solution of the muscle force in the indeterminate musculoskeletal system. This requires the grouping of muscles to reduce the number of unknowns for the appropriate equations of motion. An example of this type of approach can be taken from the study of Scott and Winter (1990) who described an Achilles

tendon model and its use to estimate Achilles tendon loading during the stance phase of running. In this “equivalent muscle” approach, the electromyography (EMG) was used to estimate loading of individual human skeletal muscles. The method may sound promising, especially because a linear or slightly curvilinear relationship can be established between EMG activity and muscular force (Komi and Buskirk 1972; Bouisset 1973), but unfortunately EMG is a very poor predictor of continuous force record, because it is very sensitive to different types of muscle action (isometric, eccentric, concentric) and to the velocity of action in dynamic situations (Komi 1983). The list of problems increases when one considers that EMG is very adaptable to training and detraining (Häkkinen and Komi 1983) as well as to fatigue (Komi 1983). In certain dynamic situations such as running and hopping, EMG may also be momentarily silent while considerable force can be recorded from the Achilles tendon (AT) during the same time period (Fig. 1). Considering that indirect estimates are often also time consuming, it is understandable that researchers became interested in developing methods to directly record in vivo human tendon and ligament forces during dynamic activities.

### **1.3. The instantaneous force-velocity relationship during SSC**

It has been a long time wish and challenge among researchers to be able to capture exactly and continuously the loading of the various musculoskeletal structures in the human body. Especially relevant in this regard has been a need to record continuously the loading of an individual tissue (e.g. tendon or ligament) in its natural environment, i.e. when it is functioning together with other similar or different (e.g. muscle) tissues. It is very likely that the loading (and function) of a tissue may be very different when one compares the “natural” setting with that of an isolated tissue loading. Direct application of the force-velocity relationship of isolated muscle to natural locomotion such as the SSC is difficult, indeed, since isolated preparations use constant electrical stimulation. The natural setting involves not only different structural elements, but also interference from the nervous system (e.g. sensory input with various connections, variable central command to the muscles). For instance, the neural control of a given braking phase after impact is well known to differ when the task is performed with the instruction to either rebound (SSC) or to simply land (pure eccentric action). This makes necessary to measure the instantaneous force-velocity relationship in human, using temporarily *in-vivo* techniques such as the buckle transducer or the fine optic fiber set around or through major tendons.

Tendons are of particular interest because, besides being an important part of the muscle–tendon unit (MTU), their chief function is to transfer force produced by the contractile component to the joint and/or bone connected in series. The human Achilles tendon (AT) represents an important part of lower extremity function. Because of their anatomic position, the AT and its muscles (soleus and gastrocnemius) are the first structures to take up the impact loads in many activities. Of particular importance is the ability of these tissues to store energy when deformed (stretched) by external force and to recoil after being stretched (Huijing 1992; Komi 2000). This gives the MTU efficient possibilities for force production during locomotion. To evaluate the behaviour of the MTU during human movements, it is useful to quantify in-vivo the imposed loads (forces) as well as the length changes within the MTU. Both the buckle and the optic fiber techniques have been shown to be appropriate as they are very sensitive to small variations in tensile force during normal activities.

### ***1.3.1. In-vivo buckle-transducer technique***

In humans the basic methodology has involved a buckle-transducer that has been implanted under local anaesthesia around the Achilles tendon (AT). This tendon is an ideal one because of the large space between the tendon and bony structures within the Karger triangle. Several experiments have been performed successfully with this method (Komi et al. 1992). In the first experiment with a human subject (Pr P.V. Komi himself), the E-form transducer was implanted around the AT and kept in situ for 7 days before measurements were performed during slow walking (Komi et al. 1984). This type of transducer did not, however, satisfy the requirements of pain-free, natural locomotion, and the transducer design was changed to Salmons' original buckle type (Salmons 1969) (see Fig. 3). In brief, it consists of a main buckle frame, two strain gauges, and a centre bar placed across the frame (Komi 1990). To provide normal proprioception during movements, lidocaine is carefully injected around the tendon. An incision of approximately 50 mm in length is made on the lateral side just anterior to the tendon to avoid damage to small saphenous vein and the sural nerve. The size of the buckle is matched with that of the tendon. The correct-sized cross-bar is then placed under the tendon into the slots of the frame. This causes a small bend in the tendon. If the bending is excessive, the tendon structures may suffer damage. The cable containing the wires from the strain gauges is threaded under the skin and brought outside approximately 10 cm above the transducer. After the cut is sutured and carefully covered

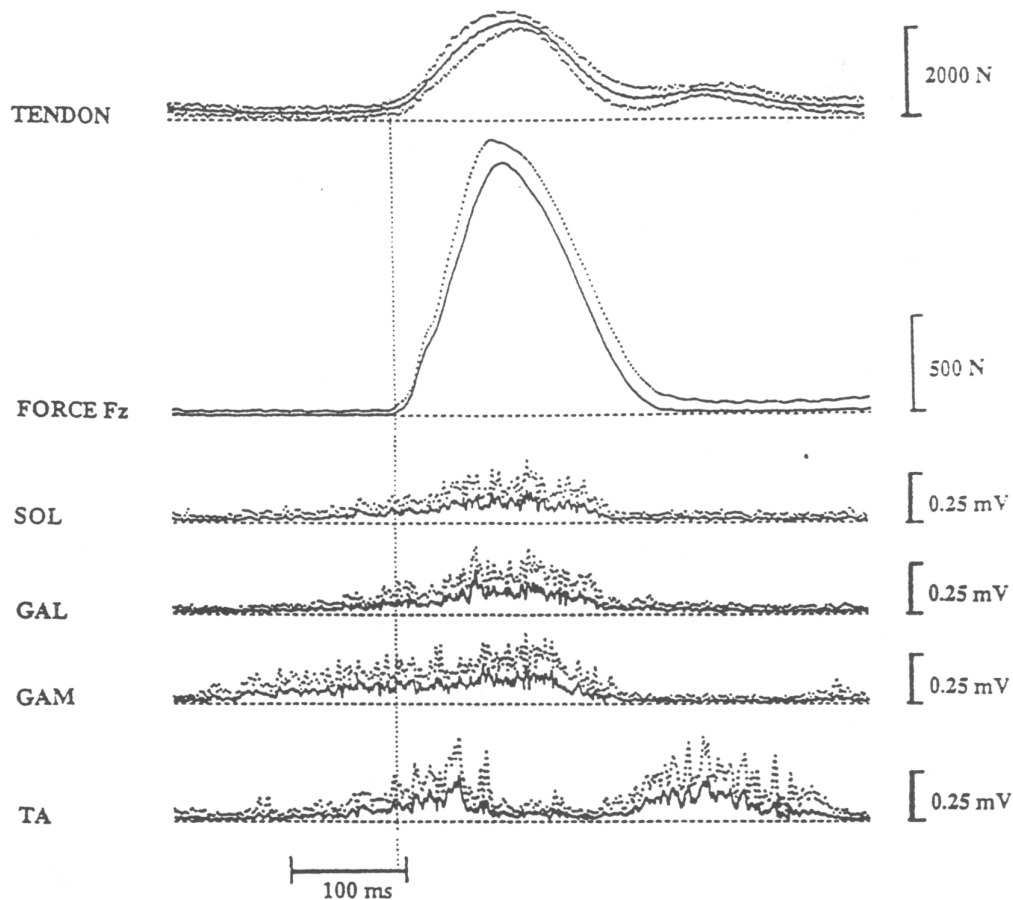
with sterile tapes, the cable of the transducer is connected to an amplifying unit for immediate check-up.

It must be admitted, however, that the buckle transducer method is quite invasive, and may receive objections for use by the ethical committee in question. Other restrictions in the use of this technique lie in the critical aspects of the calibration procedure (Komi 1990; Komi et al. 1987b) and in the problems of its application when long-term and repeated implantation may be of interest. The buckle transducer method cannot isolate the forces of the contractile tissue from the tendon tissues. This method can therefore be used to demonstrate the loading characteristics of the entire MTU only.



**Fig. 3 Lateral radiographic view of the buckle transducer in situ after its surgical implantation.**

Nevertheless, the buckle transducer is the original method which revealed that the largest AT forces occur in submaximal hopping (Fig. 4) and not in maximal squat jump or counter-movement jump ( Komi et al. 1992; Fukashiro et al. 1995b). As illustrated on this figure, the mean ATF peak value recorded in hopping is about twice higher than the vertical ground reaction force (Fukashiro et al. 1993; Nicol and Komi 1998). This technique revealed also that *gastrocnemius* and *soleus* demonstrate a small, but meaningful stretch-shortening cycle action during cycling (Gregor et al. 1991). Traditionally, cycling was considered an almost pure form of positive (concentric) work. Finally, this technique allowed the first in-vivo quantification of the mechanical stretch-reflex response in passive stretch condition (Nicol and Komi 1998, 1999). This will be further discussed in section 1.5.1.



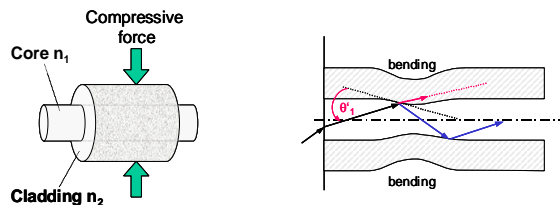
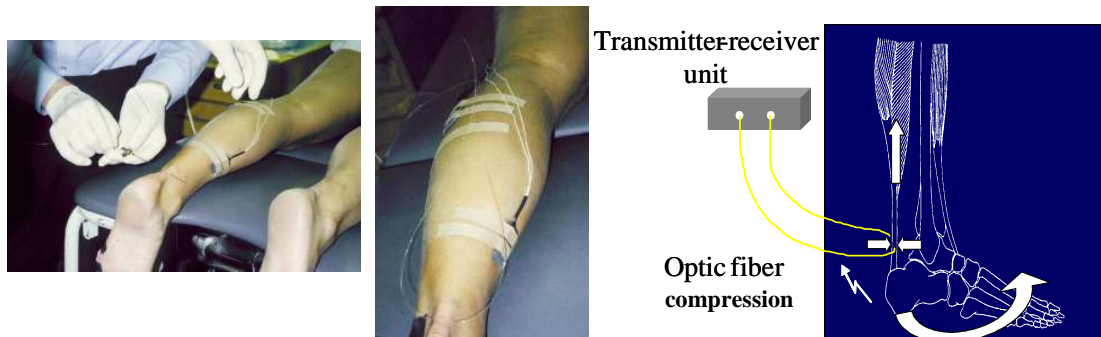
**Fig. 4 Average curves of the different parameters while a subject was hopping on a force plate submaximally at preferred frequency of 2.5 Hz.** In addition to the Achilles tendon force and the vertical ground reaction force ( $F_z$ ), the figure shows the rectified EMGs for the following muscles: soleus (SOL), lateralis and medialis gastrocnemius (GAL and GAM, respectively) as well as tibialis anterior (TA). The dashed line denotes the beginning of the foot contact on the force plate. The records are averages of 12 consecutive hops. The  $F_z$  and EMG records include mean + SD, and the Achilles tendon force mean  $\pm$  SD. (From Nicol and Komi 1998)

### ***1.3.2. In-vivo optic fiber technique***

Further technical developments for direct tendon force measurements resulted in a new a less invasive approach, the optic fiber technique (Komi et al. 1996). In this technique (Fig. 5A), an optic fiber ( $\varnothing$  0.5 mm) is inserted through the tendon to serve as a transducer of tendon stress. The principle of this method is as follows: when a thin optic fiber is bent or compressed the light can be reduced linearly with pressure and the sensitivity depends on fiber index, fiber stiffness and /or bending radius characteristics. Fig. 5B characterizes the principle of the light modulation in the two layer (cladding and core) fiber when the fiber

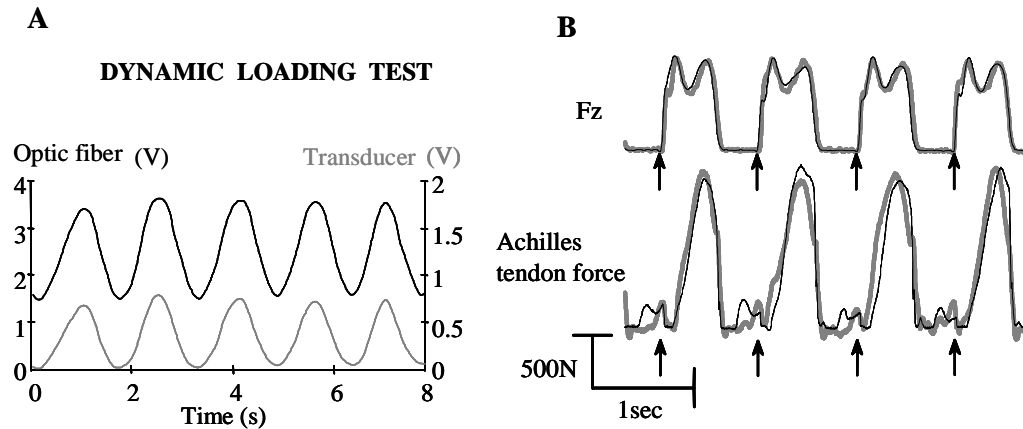
diameter is compressed by external force. The core and cladding will be deformed and a certain amount of light is transferred through the core-cladding interface. In order to avoid the pure effect of bending of the fiber, the fiber when inserted through the tendon must have a loop large enough to exceed the so called critical bending radius. It is also recommended to insert the optic fiber in a global limb position close to the one tested (i.e. with extended knee joint when subsequently used during locomotion).

**Fig. 5 (A)** Demonstration of the insertion of the optic fiber through the tendon. A hollow 19 gauge needle is first passed through the tendon (left). The sterile optic fiber is then passed through the needle; the needle is removed and the fiber remains in situ (right).



**(B)** Basic principle to demonstrate how the compression on the optic fiber (left) causes microbending (right) and less of light through the core-cladding interface (From Ishikawa and Komi 2008).

Although the optic fiber method may not be more accurate than the buckle transducer method, it has several unique advantages. First of all it is much less invasive and can be reapplied to the same tendon at a few day intervals (Nicol et al. 2003). In addition, almost any tendon can be studied provided that critical bending radius is not exceeded. This method is also quite reliable. As shown in Fig 6A the dynamic testing shows good match between the optic fiber and external force records (Komi et al. 1995). This has been confirmed by our own recordings of the mechanical stretch-reflex response induced by passive stretches of the triceps surae muscle group (Nicol et al. 2003) and will be further developed in section 1.5.3. Figure 6B is another excellent example of the similarity of ATF patterns when the subject walked at the same speed during a 4-month interval (Komi et al. 1995). Please note how the two records follow each other.



**Fig. 6. Optic fiber recordings in dynamic testing conditions.**

(A) Records of optic fiber on external force transducer during quick dynamic loading and unloading of the knee extension movement. (Adapted from Komi et al. 1995)

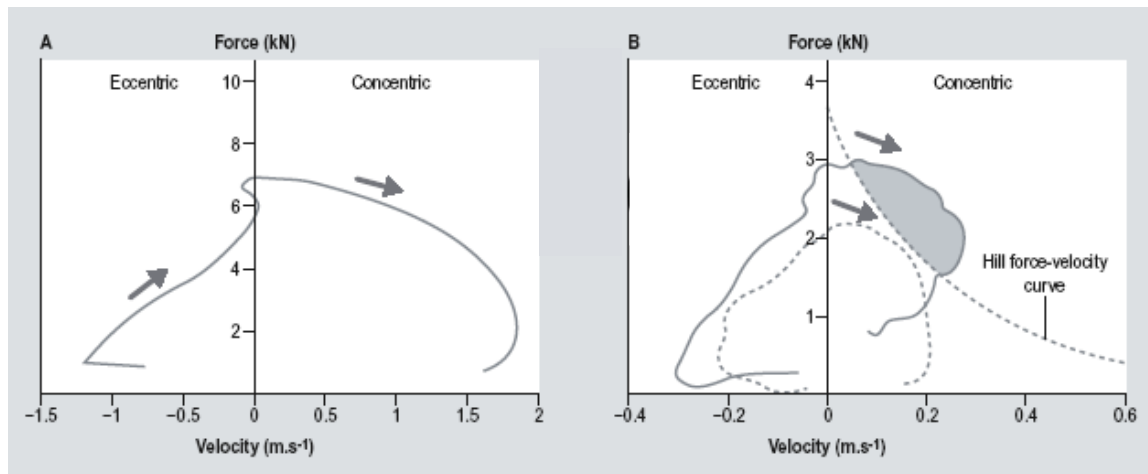
(B) Achilles tendon (optic fiber) and vertical ground reaction forces (Fz) measured for one subject while walking at same speed at 4 month interval. Black line (1<sup>st</sup> measurement); grey line (2<sup>nd</sup> measurement). The arrows represent the first point of heel contact with the ground. (From Ishikawa and Komi 2008)

### 1.3.3. The instantaneous force-velocity curves obtained by in-vivo techniques

A characteristic of the instantaneous force-velocity curves is the considerable force potentiation, as seen during the push-off (concentric) phase of running (Fig. 7A). Furthermore, force-velocity measurements during the contact phase of running revealed a curve that was completely different from the classical Hill relationship. Fig. 7B, obtained simultaneously for the patella and Achilles tendons (optic fibre technique), demonstrates two important aspects of human skeletal muscle function. First, in short-contact hopping actions, *triceps surae* behaves like a bouncing ball. Second, when the hopping intensity is increased or changed to another type of movement, the contribution of the patella tendon force increases, and that of the Achilles may decrease (Finni et al. 2001). The Hill curve, obtained during a constant maximal (concentric) activation is superimposed, and the shaded area denotes remarkable performance potentiation, even though the hopping effort was submaximal. Animal experiments have obtained similar results (Gregor et al. 1988).

Such a difference between the instantaneous and classical curves is partly due to differences in muscle activation between the two conditions. Isolated preparations primarily quantify the shortening properties of the contractile elements, whilst natural locomotion uses the whole SSC, and involves the controlled release of elastic forces from the muscle-tendon unit that were generated during the eccentric phase. Some of energy recovery occurs during the shortening phase, resulting in performance potentiation. Thus, natural locomotion may produce more efficient muscle function that differs markedly from that observed in isolated

preparations. In addition, during SSC activity performed without fatigue, electromyographic activity usually peaks before the eccentric phase ends, thereby confirming the importance of the eccentric phase.

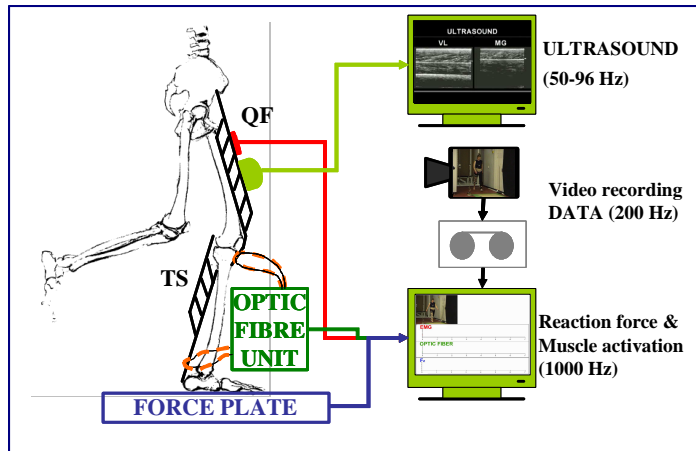


**Fig. 7. Instantaneous force-velocity (F-V) curves.** F-V curves measured with a buckle transducer on the Achilles tendon during running (A:  $9 \text{ m.s}^{-1}$ ), and with an optic fibre for the Achilles (solid) and patella tendons (dashed) during hopping (B). Note the force potentiation during the concentric phase of the stretch-shortening cycle (shaded area). (Adapted from Komi 1992 (A) and Finni et al. 1998 (B))

#### 1.4. Fascicle-tendon interactions in natural human movement

By definition, the SSC involves the entire muscle-tendon unit (MTU), although the contractile tissue, aponeurosis and tendons may experience different changes in length. In the contractile element, bundles of fibers are called muscle fascicles and they play also an important role in the function of the entire MTU. As the fascicles are controlled both by external stretch and internal activation, it is important to study the differences in length changes of fascicles and tendons in various muscles during SSC exercises. Furthermore, synergists might experience different length changes especially when comparing mono- and biarticular ones. Therefore, it appears clearly that the muscle function in locomotion cannot be studied from measurements of muscle-tendon unit (MTU) only. Both the buckle transducer and optic fiber techniques, although they produce very valuable information, are limited in this regard. With the development of real-time ultrasonography, it is now possible to non-invasively examine the *in-vivo* behavior of muscle fascicles and tendinous tissues (aponeuroses and tendon in series) during exercise. Figure 8 shows the combined use of the optic fiber and ultrasonography techniques during normal human locomotion to capture the tensile forces and muscle architectural changes, respectively, in combination with simultaneous application of EMG recording.





**Fig. 8 . Schematic presentation how the optic fiber and ultrasound measurements can be combined with each other and together with other instruments. (From Ishikawa and Komi 2008)**

#### ***1.4.1. The ultrasonography (U.S.) technique***

This method is a relatively useful tool to apply simultaneously with other biomechanical parameters in movements covering a large range of intensities. Ultrasound has been defined as the sound wave which is higher than human being can hear ( $> 20$  kHz). The sound waves are propagated at a speed of approximately 1540 m/sec in soft tissues (e.g. bone 3360, muscle 1570, water 1520, brain 1510, and fat 1440 m/sec, respectively). The thickness, size and location of various soft tissue structures, such as skin, fat, muscle and tendinous tissues, in relation to the origin of the ultrasound beam are calculated at any point in time. The strength of the reflected sound wave depends on the difference in the acoustic impedance between adjacent structures. The acoustic impedance of a tissue is related to its density; the greater the difference in acoustic impedance between two adjacent tissues the more reflective will be their boundary.

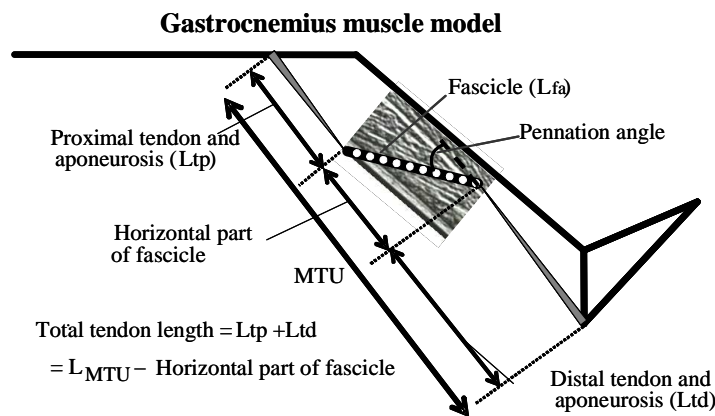
In the biomechanical research area, 1-10 MHz frequency ultrasound transducers (probes) are commonly used. The resolution of US is depending not only on the transmitted material but also on the frequency of the ultrasound. In ultrasound physics, the wavelength ( $\lambda$ ; lateral resolution) equals the speed of the sound transmission ( $v$ ; 1540 m/s) divided by the frequency of the ultrasound ( $f$ ; 1-10 MHz) ( $\lambda = v / f$ ). Therefore, the resolution of ultrasound scanning image in the human body can be theoretically achieved with accuracy from 0.15 to 1.54 mm range. The precision, linearity and reproducibility of US have been confirmed in several studies (Henriksson-Larsen et al. 1992; Kawakami et al. 1993; Kawakami et al. 2000; Rutherford and Jones 1992). Techniques of the US scanning have developed considerably and now it is possible to monitor the lengthening and shortening of fascicles and tendinous structures during in vivo human movements (Herbert and Gandevia 1995; Fukashiro et al.

1995a; Fukunaga et al. 1996, 1997, 2001, 2002; Maganaris et al. 1998, 2003; Maganaris 2003; Finni et al. 2003a; Ishikawa and Komi 2004, 2005a, 2005b, 2007; Narici et al. 1996). Especially, there are particular advantages in evaluating muscle-tendon behaviour for better understanding of the muscle mechanics during human locomotion as compared to the data derived from cadavers (Grieve et al. 1978).

The instantaneous lengths of fascicle and tendinous structures can be measured on the basis of a geometric MTU models (Zajac 1989; Allinger and Herzog 1992). As shown in Fig 9, the total tendon length (TT) is defined as the sum of the proximal and distal tendinous structures and aponeuroses (Fukunaga et al. 2001; Muraoka et al. 2001; Kurokawa et al. 2001, 2003). The length changes in TT are then calculated by subtracting the horizontal part of fascicle in the direction to the aponeurosis from the MTU length, thus assuming that changes in one fascicle reflect the changes in the entire muscle compartment.

$$L_{TT} = L_{MTU} - L_{fa} \cdot \cos \alpha,$$

where  $L_{TT}$  is the TT length,  $L_{MTU}$  is the muscle-tendon unit length,  $L_{fa}$  is the fascicle length and  $\alpha$  is the fascicle angle in each muscle.



**Fig. 9. Schematic models of gastrocnemius muscle.** The method requires that the total MTU length is recorded continuously, e.g. kinematically, during locomotion. The rest of the measurements are based on the continuous ultrasound records (Adapted from Zajac 1989; Kubo et al. 2000; Fukunaga et al. 2001; Kurokawa et al. 2001)

#### ***1.4.2. Methodological problems and possible errors in U.S. measurements during SSC***

To apply U.S. in dynamic movements, the appropriate probe frequency should be selected according to the width of the muscle region of interest. While probes producing higher frequencies have less penetrating ability, probes producing lower frequencies will provide greater depth of penetration but with less well-defined images. In most cases with human muscles, scanning is usually performed using the 5.0–7.5 MHz probes, although the evaluation of superficial muscle and tendons requires the use of the 7.5–10 MHz probes. In

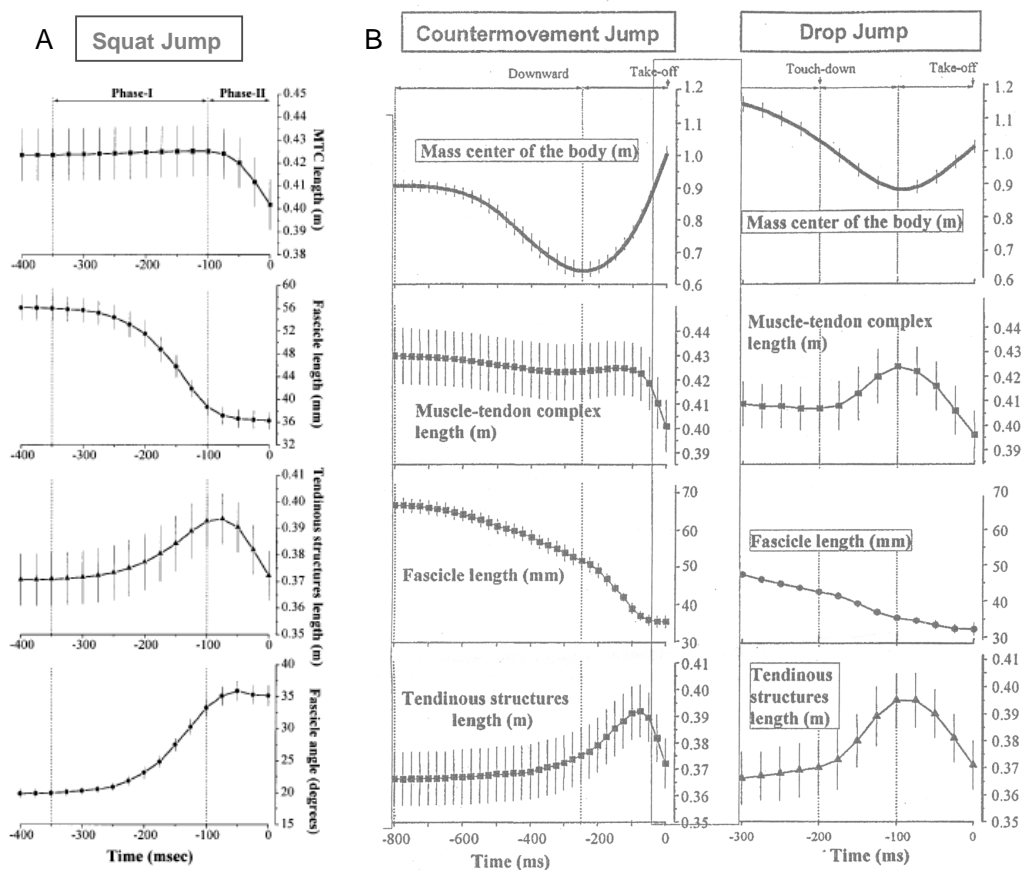
addition, dynamic movements require an appropriately selected time resolution of U.S. scan sampling (Ishikawa et al. 2005a, 2005b). After the probe has been selected, it is then placed over the mid-belly of the selected muscle. The soundhead is tipped slightly and medially or laterally to find the best images, which coincides with the plane of the muscle fascicle (Herbert and Gandevia 1995; Narici et al. 1996). The investigator visually confirms the echoes reflected from aponeuroses and interspaces in order to avoid echoes of the reverberation artifacts and pitfalls of vascular origin. It must be noted, however, that in many cases with adult human superficial muscles, the probe length (e.g. 60 mm) does not cover the entire length of the fascicle. For this reason, appropriate approximations have been adapted for obtaining correct estimates of changes in the tensile structure lengths (Finni et al. 2001, Finni and Komi 2002; Ishikawa et al. 2003).

#### ***1.4.3. Major findings from U.S. measurements in SSC tasks***

U.S. measurements performed at rest (Kawakami et al. 1993) and in isometric actions (Narici et al. 1996; Fukunaga et al. 1997; Maganaris et al. 1998; Maganaris 2003) have shown that the muscle-tendon architecture undergoes remarkable changes. As illustrated in Fig. 10A, these changes have been clearly demonstrated in a squat jump condition during the upward movement of the body mass centre (phase I) preceding the final push-off (Kurokawa et al. 2001). Some studies have succeeded in exploring these changes during different intensities of locomotion: in walking (Fukunaga et al. 2001; Ishikawa et al. 2005a), running (Ishikawa et al. 2007b) and in jumping (Kawakami et al. 2002; Kurokawa et al. 2003; Ishikawa et al. 2003, 2005b; Ishikawa and Komi 2004;). Their findings have dramatically advanced our understanding of the specificity of muscle-tendon interaction during normal locomotion.

As already discussed, the natural way of muscle function is SSC, in which muscles are actively stretched and subsequently shortened. This concept refers to the entire muscle-tendon unit (MTU). Now it is of interest to examine how the separate compartments (contractile and elastic components) obey this SSC concept. Several animal studies have reported that the muscle fibers shorten during the braking phase, even if the MTU is stretching (Griffiths 1991; Roberts et al. 1997). Fukunaga and colleagues (Fukunaga et al. 2002) recently summarized that in human movements such as walking and jumping, the gastrocnemius medialis fascicles behave almost isometrically, whereas the tendon performs a SSC. They suggested that the human MTU is designed to match the capacity of muscle to generate force and the elasticity of the tendon to generate efficient movement performance. As illustrated in Fig. 10B (left

graphs) for a counter movement jump, the rapid shortening of MTU towards the end of the push-off phase would depend on the shortening of the tendinous structure (Kurokawa et al. 2003). This mechanism has been called a catapult action (Hof et al. 1983). In a more intense drop-jump condition (Fig. 10B, right graphs), the SSC behaviour of the MTU complex during the braking and push-off phase was also found to be largely dependent on the behaviour of the tendinous structure whereas the gastrocnemius medialis fascicles shortened only slightly during the whole movement.

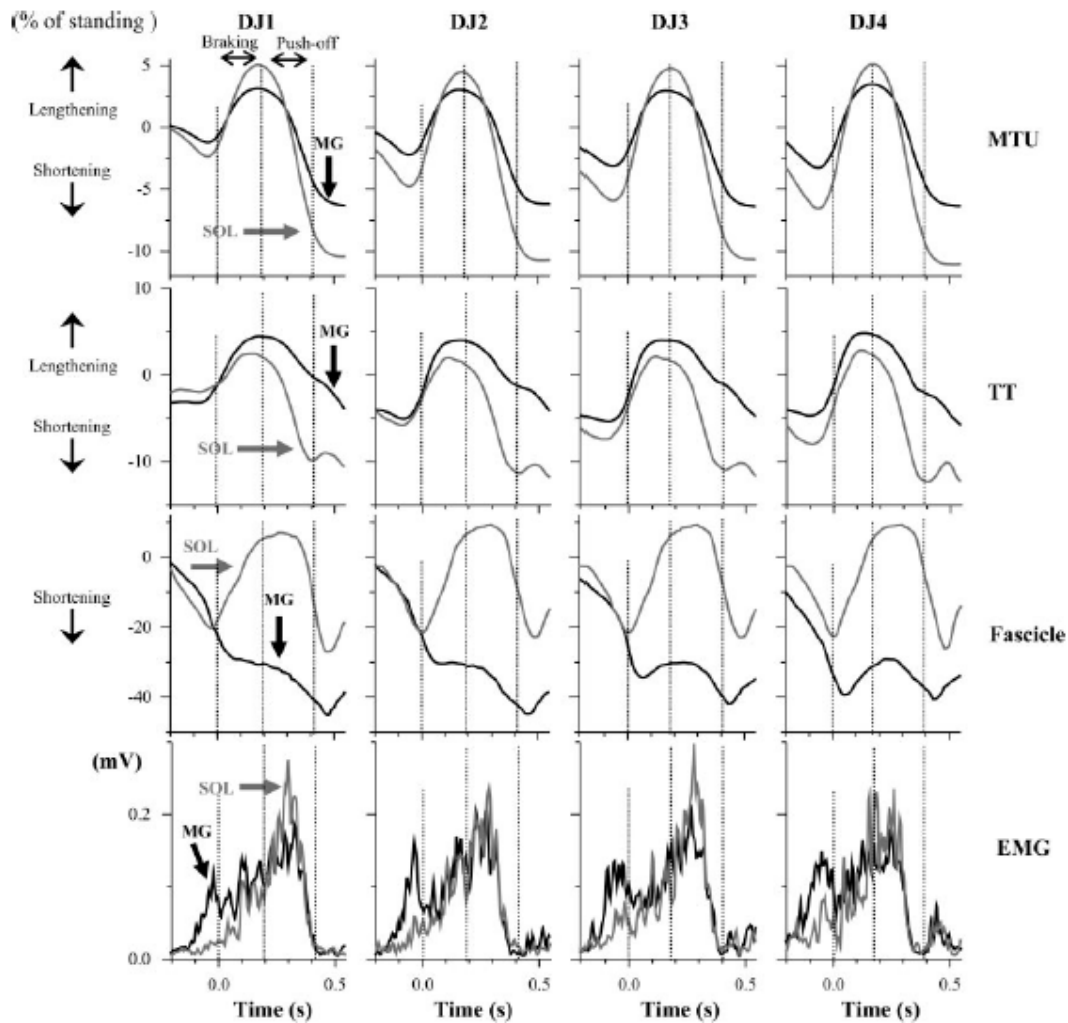


**Fig. 10. Mean time histories of the structural changes of the MTU unit in different jumping conditions.** (A) Changes observed during the time course of a squat jump in the muscle-tendon complex length, fascicle length, tendinous structure length, and fascicle angle. Left vertical dotted lines represent the onset of push-off, i.e., start of upward movement of mass center of the body; right vertical dotted lines represent the onset, from which the MTC begins to become remarkably shortened. Time is expressed relative to the instant of toe-off. (B) Mean time history of the vertical displacement of mass center of body (MCB) and respective lengths of the muscle-tendon complex, gastrocnemius medialis fascicle and Achilles tendon structures. Left vertical dotted lines represent the start of the downward movement of the MCB. Middle vertical dotted lines represent the start of its upward movement. Right vertical dotted lines indicate the start of the rapid shortening of the muscle-tendon complex. Thin vertical bars indicate mean standard error for 8 subjects. (Adapted from (A) Fukashiro et al. 2006 and (B) Kurokawa et al. 2003)

However, although human leg muscles are commonly accepted as being synergists, they do not have similar mechanical behaviour within the fascicles during locomotion. For instance, in extremely high impact loaded drop jumps, Ishikawa and colleagues (Ishikawa et al. 2003; Ishikawa and Komi 2004) demonstrated stretching and subsequent shortening of the vastus lateralis fascicles similarly to those of the MTU.

Furthermore, many of these studies used U.S. scanning rates of 25-50 Hz that may not be precise enough to characterize the movement of the fascicles. This may have caused misleading generalizations. In particular, some caution should be used when comparing mono- and biarticular muscles. Based on modern application of high-speed (96 Hz) ultrasound scanning to the study of fascicle behaviour, Ishikawa and Komi present the hypotheses that changes in length of the fascicles and tendon, when acting together, depend on the movement task, movement intensity, and muscles involved (Ishikawa and Komi 2008). In particular, the fascicle behaviour of two different muscles (medial *gastrocnemius* and *soleus*) was measured simultaneously during stretch-shortening cycle movements: drop jumps, hopping and running (Ishikawa et al. 2005a, 2005b; Ishikawa and Komi 2007). The mono-articular *soleus* fascicles did behave in a SSC manner whereas the biarticular medial *gastrocnemius* fascicles kept a constant length. In drop jumps with maximal rebounds (Sousa et al. 2007), however, the gastrocnemius fascicle length was found to be either shortened, or constant or even initially lengthened depending on the dropping height that varied from a low (50%: DJ1) to a supramaximal (120%: DJ4) level as compared to the optimal one (Fig. 11). These differences in fascicle behaviour supports the concept of the previous studies (Elftman 1939; Alexander 1974), which suggested that biarticular muscle (*e.g.* medial *gastrocnemius*) could play a greater energy-conservation role during human locomotion, relative to mono-articular muscle (*e.g.* *soleus*).

We must therefore consider that the SSC behaviour inside given MTU may differ across different types of SSC exercise, and also across different muscles. This emphasizes the interest to use such a technique to examine the effects of fatiguing SSC exercises on different types of SSC testing tasks (maximal versus submaximal) and different mono- and biarticular muscles. Early attempts have been performed (Ishikawa et al. 2007a) that need to be repeated. This will be further developed in chapter 2.



**Fig. 11. Representative example in drop jumps (DJ) from increasing dropping heights of the time course data of the lengths of the MG and Sol muscle-tendon units (MTU), tendinous tissue (TT), and fascicle, together with the EMG activity of the MG and Sol muscles.** Subjects performed the DJ with maximal rebounds from four individually predetermined dropping heights: 50, 75, 100, and 120% of the optimal drop height (DJ1, DJ2, DJ3, and DJ4, respectively), in a random order. Vertical dotted lines show the points of the contact, end of the braking phase, and take-off. The “% of standing” in the y-axis for the first 3 rows shows the relative changes from the length in the upright position. (From Sousa et al. 2007)

### 1.5. Role of stretch reflexes in stretch-shortening cycle exercise

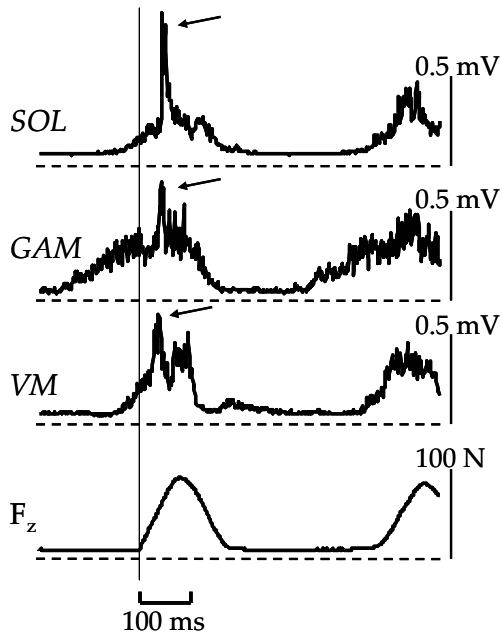
As will be discussed later in chapter 2, the stretch reflexes have been found to be quite sensitive to exhaustive SSC fatigue. In order to understand this in terms of functional significance, it is of interest to demonstrate what the possibilities are for stretch reflexes to be operative and functionally meaningful in SSC activities. Short contact SSC exercises such as hopping and running activities are often used as suitable models of human SSC for possible

intervention of stretch-reflexes. As illustrated earlier in Fig. 1, SSC activities are very effective due to the following fundamental conditions (Komi and Gollhofer 1997): 1) the muscles are preactivated before touch down (and the braking phase); 2) the eccentric (lengthening phase) is short and fast, and 3) there is an immediate transition (short delay) between stretch (eccentric) and shortening (concentric) phases. Stretch reflexes are known as increasing muscle stiffness regulation per same operating force than in an areflexive muscle (Hoffer and Andreassen 1981). According to Nichols and Stein (Stein 1982; Nichols 1987), it is the stretch reflex system that provides high linearity in muscular stiffness. This is considered as a logical consequence of how muscle spindles and Golgi tendon organs are operating in the control of muscle length and tension (Houk and Rymer 1981). In SSC, stretch reflexes may thus be expected to make a net contribution to muscle stiffness already during the eccentric phase and this could be a prerequisite for an efficient storage of elastic energy. Their intervention and contribution to SSC muscle actions have been, however, source of several debates in the literature.

#### ***1.5.1. Demonstration of stretch-reflex intervention in SSC***

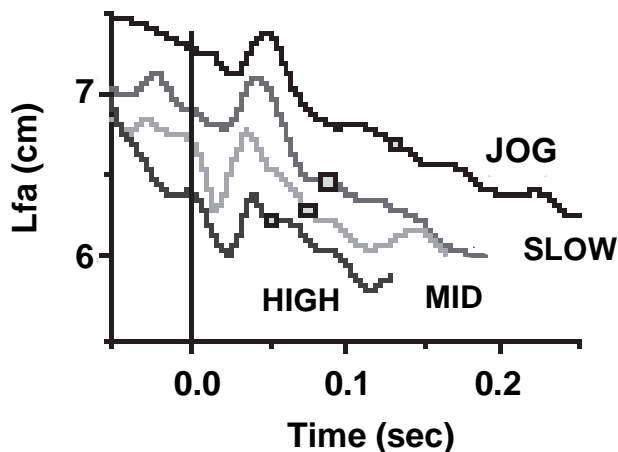
First evidence for the short-latency stretch reflex component in *gastrocnemius* came from the study of Dietz et al. (1979) later confirmed by Fellows et al. (1993), who used the ischemic blocking method to isolate the Ia afferent information acting on spinal pathways during fast running. During ischemic blocking, *gastrocnemius* electromyographic activity was dramatically reduced during the contact phase, but there was no change in pre-activation. The control (non-ischemic) runs demonstrated that *gastrocnemius* had a clear stretch reflex component during the contact phase, with the average electromyographic activity being 2-3 times higher than the activity during maximal voluntary isometric plantar flexion.

Since then, other studies supported the intervention of Ia afferents in SSC activities in the commonly observed EMG burst (identified by an arrow in Fig 12) in running and hopping activities. The short-latency component, appearing about 40 ms after the ground contact, is visible in all examined muscles and is especially strong in the soleus muscle. These records were obtained by averaging the rectified EMGs over several trials involving two leg hops with short contact times. Appearance of these reflex components is a very common and repeatable observation (Komi and Gollhofer 1997). It is true, however, that in normal movements with high EMG activity, the magnitude and net contribution of reflex regulation of muscle force is methodologically difficult to assess. This has led to much controversy about their functional contribution to the produced force and power output.



**Fig 12. Short contact SSC type hopping introduces clear bursts in rectified EMG records.** This representative example is from a drop jumps performed from 60 cm height. Timing of the sharp EMG reflex peak occurs within 40-45 ms after the initial ground contact corresponding to a short latency component (SLC) in Lee & Tatton classification (1982). The reflex EMG peak (indicated by an arrow) in these jumps is usually very clear in the soleus (SOL) muscles, but can be identified for the gastrocnemius (GAM) and vastus (VM) medialis muscles as well. Fz signifies the vertical ground reaction force. (From Komi and Gollhofer 1997)

In order to be fully convinced of the stretch reflex intervention in SSC muscle action, it must be demonstrated that the fascicles are being stretched during the braking phase of the cycle. Based on the ultrasonography measurements of relatively low (40-50 Hz) image frequency, fascicle stretching during ground contact was found as much greater in *soleus* than medial *gastrocnemius* muscle, which was even thought to demonstrate only fascicle shortening during the braking phase (Fukunaga et al. 2001). More recently, however, Ishikawa and Komi (2007) used higher (96 Hz) frequency and observed an early appearance of the fascicle lengthening immediately upon ground contact (Fig. 13). This can be taken as an important observation and proof that there is enough fascicle stretching to activate the stretch reflex component, even in the biarticular medial *gastrocnemius*.

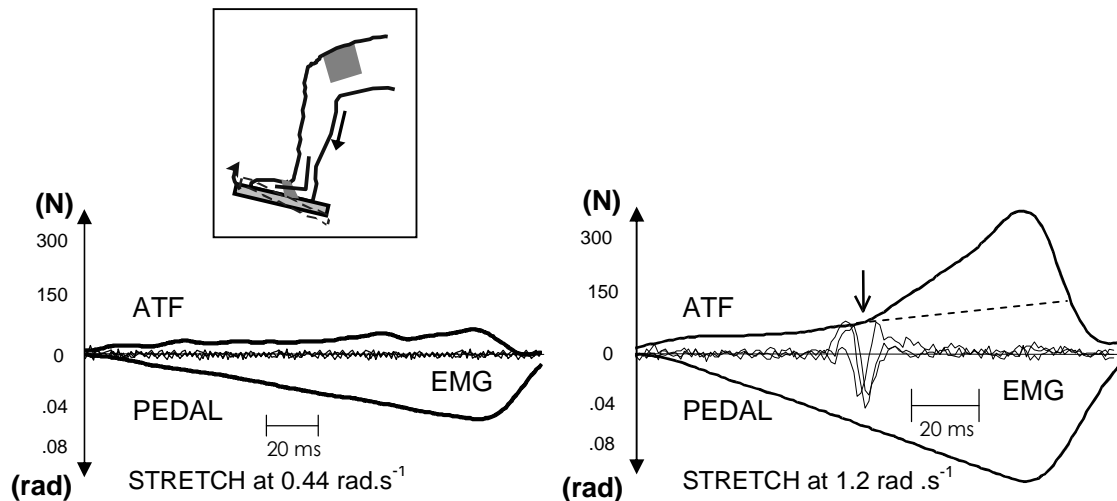


**Fig. 13. Examples of fascicle length (Lfa) of the MG muscle during running.** Lfa of the right leg during the ground contact are shown for 4 different running speeds. 0 represents the moment of ground contact. □: end of the MTU stretching. (Modified from Ishikawa and Komi 2007)



### 1.5.2. Do stretch-reflexes have time to be operative in SSC?

Their possible role has been questioned (van Ingen-Schenau et al. 1984) by an argument that the time constraints limit their possibility to have any functional meaning during the stance phase of for example running. A first answer to this question came from our studies (Nicol and Komi 1998, 1999) in which the Achilles tendon force was quantified *in-vivo* using the buckle transducer technique during pure passive dorsiflexions (0.06 – 0.12 rad of amplitude and 0.44 – 1.9 rad.s<sup>-1</sup> of mean velocity) induced by a powerful ankle ergometer (fig. 14). This passive stretch situation made easier the respective identification of the stretch reflex EMG- and mechanical response. The use of the buckle transducer (Nicol and Komi 1998, 1999) and, later on, of the optic fiber technique (Nicol et al. 2003) gave similar results. During the fastest stretch, the ATF started to increase clearly 13–15 ms after the onset of the reflex EMG response (Fig. 14), showing stretch reflex-induced force enhancement between 200 to 500 % over the pure passive stretch response (without a reflex EMG response).



**Fig. 14. Demonstration of passively induced stretch reflexes on the Achilles tendon force (ATF).**

**Left:** passive dorsiflexion at slow stretch caused no reflex EMG response and led to a small and rather linear increase of the ATF (pure passive response). **Right:** in case of faster and larger stretches the reflex contribution to ATF corresponds to the additional ATF response above the pure passive influence represented by the dashed line. The vertical arrow indicates the beginning of the reflex-induced mechanical response (From Nicol and Komi 1998).

This electromechanical delay (EMD) value gives then a rational basis for calculating the possibilities for stretch reflexes to have time for operating during SSC activities. Considering the duration of a monosynaptic stretch reflex loop of 40 ms, the maximum delay between initial stretch and subsequent force potentiation would be around 50 to 55 ms. When referred to a running situation, the first contact on the ground indicates the point of initial stretch. The contact time depends naturally on running speed, being in marathon run around

250 ms, but in sprinting as low as 90-100 ms (Mero and Komi 1986). The duration of these two important phases of the contact (braking and push-off) decrease in parallel with the running speed. Consequently, in slow running the net reflex contribution will occur at the end of the eccentric (braking) phase of the cycle, and in fast sprinting it may be partly extended also to the push-off (concentric) phase of SSC. These time calculations confirm that the stretch reflexes of short latency could have ample time to be instrumental in force and power enhancement during SSC. During short contact SSC activities such as hopping, activity of longer latency stretch reflex components should not assist the resistance to muscle stretch, but would rather contribute to the push-off phase (Voigt et al. 1998). Thus, there are no time constraints for short latency reflexes to be operative in stiffness regulation during a SSC.

### ***1.5.3. What is the functional significance of stretch-reflexes in SSC?***

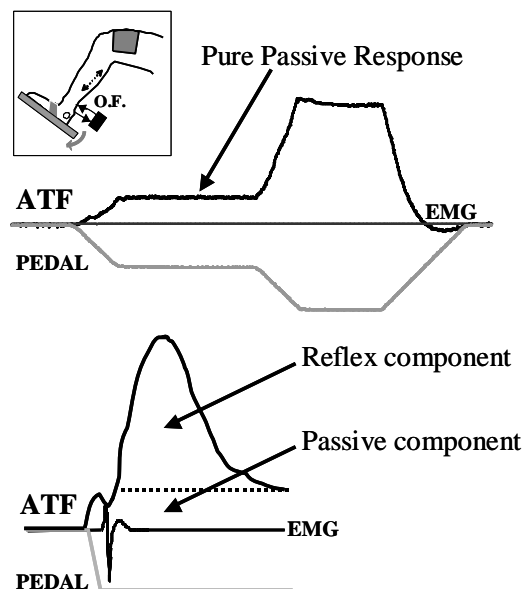
Despite the possibility to record in-vivo the muscle-tendon force in a reliable way, magnitude of the reflex contribution to stiffness and force enhancement of tendon-muscle complex is largely unknown in normal locomotion. This is mostly attributed to the difficulty to differentiate the reflex-mediated force response from the resistive force of the intrinsic contractile components and from the passive tissues and relaxed muscle fibers.

A partial answer came from the comparison in a given subject of direct ATF measurements (with the buckle) in pure passive stretch conditions and in hopping (Nicol and Komi 1998). This study revealed that the highest reflex-induced Achilles tendon force recorded on the ergometer corresponded to 6.7 % of peak ATF ( $2750 \pm 51$  N) recorded in hopping. In normal running and hopping, even when performed submaximally, the reflex contribution should be more substantial due to the great number of motor units receiving Ia afferent stimuli from the condition of relatively high stretch velocities ( $10 - 12 \text{ rad.s}^{-1}$ ) at the ankle joint.

The stretching velocity effect was not demonstrated within this slow range of velocities ( $1.2 - 1.9 \text{ rad.s}^{-1}$ ), but this has been subsequently shown at slightly higher velocities ( $2.0 - 4.0 \text{ rad.s}^{-1}$ ) (Gollhofer and Rapp 1993; Gollhofer et al. 1995) that were still 5 to 10 times lower than those occurring in natural forms of locomotion.

As shown in Fig. 15, the subsequent data obtained with the optic fiber technique while introducing a long plateau duration in the pedal movement in between the two successive stretches revealed that the ATF continues to rise until about 100 ms (Nicol and Komi 1998; Nicol et al. 2003). This is in line with the twitch contraction time earlier reported for the soleus and gastrocnemius muscles (Buller et al. 1959; McComas and Thomas 1968; Sale et al.

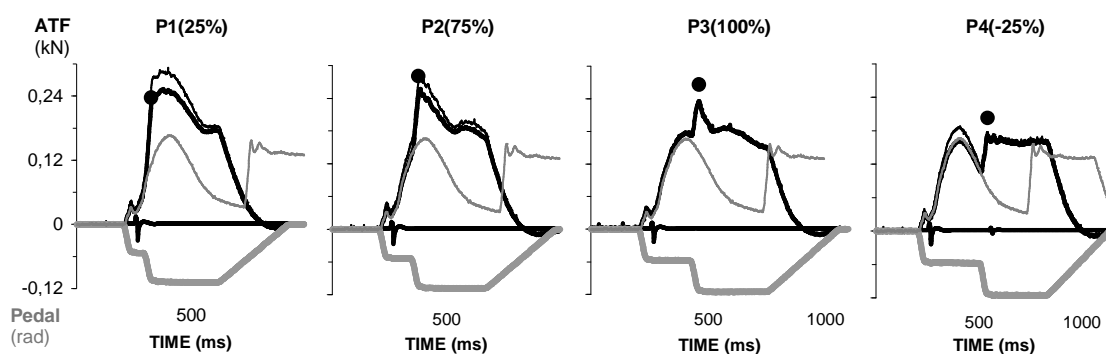
1982). In the absence of reflex EMG response at slow stretching velocity (upper graph, Fig. 15), the force-stretch curves reflected the pedal movement, but demonstrated a steeper ATF rise during the second stretch as compared to the first one. Although the 2 mechanically induced stretches were very similar, the second one started from a 3% more dorsiflexed position, emphasizing the potential influence of the tendon compliance on the recorded ATF. This confirms also the sensitivity of the optic fiber technique.



**Fig. 15. Passive dorsiflexions were induced by the ankle ergometer while the Achilles tendon force (ATF) was recorded *in-vivo* using the optic fibre (OF) technique.** At low stretching velocity (upper graph), the absence of reflex response resulted in quantification of the “pure passive ATF response to stretch” during the plateau phase. This was used at higher stretching velocities to isolate the “pure reflex component” (lower graph). (Adapted from Nicol et al. 1999).

If we consider the fact that during various forms of locomotion the stretch-reflex mechanical response takes place while the muscle is actively stretched, it is of interest to examine the resistance to stretch of the mechanical reflex response (Nicol and Komi 1998; Nicol et al. 1999). For this purpose, the timing of the 2<sup>nd</sup> stretch was varied by adjusting the plateau duration so that the 2<sup>nd</sup> stretch started at different force levels, both during the rising and the decreasing phases of the twitch (Fig. 16) (Nicol et al. 1999). It seems important to differentiate these two phases because, as this mechanical reflex response results from a single burst of action potentials, the rising phase of the twitch response should correspond to the attachment phase of most of the involved cross-bridges, whereas the decreasing phase should correspond to the detachment of most of them. As shown on this figure, the results show that when the 2<sup>nd</sup> stretch occurred during the rising phase of the first stretch-induced

twitch contraction, it led to clear enhancement of the rate and peak of ATF (Fig. 16A). Whereas, if the stretch occurred at the onset of the decreasing phase of the mechanical reflex response, the combined effect was reduced in all subjects as compared to the expected summation of the reflex and stretch effects (Fig. 16B). Referring to the work of Edman et al. on isolated sarcomeres, it is suggested that the combined stretch and reflex potentiation is very likely to depend on the sarcomere kinetics (Edman et al. 1978; Edman 1980). Our data of combined stretch and reflex potentiation demonstrate that the resistance to stretch is particularly efficient during the early part of the reflex response. This is in agreement with the short range elastic stiffness hypothesis according to which the cross-bridge resistance to stretch is particularly efficient during the early part of the cross-bridge attachment (Edman 1980). Therefore, the rapid reflex-induced cross-link formation could play a substantial role in the force generation during stretch.

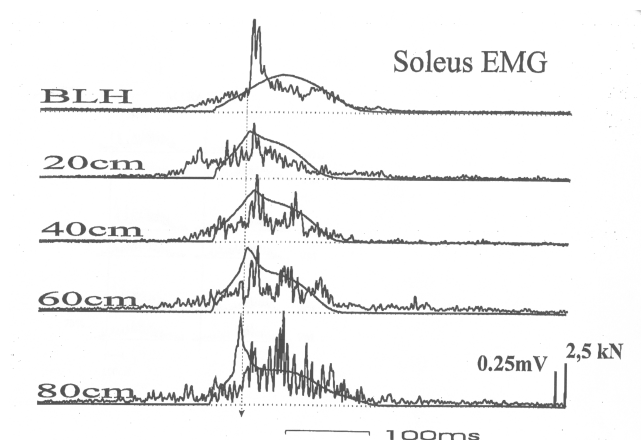


**Fig. 16. ATF potentiation when the 2<sup>nd</sup> stretch occurs during the rising phase (P1 & P2) or during the decreasing phase (P3 & P4) of the reflex induced mechanical response.** Individual ATF records are presented as mean + SD of seven trials. The exact summation of the reflex and stretch effects are represented by dots that can be compared to the actual combined effects (Adapted from Nicol et al. 1999).

Thus, evidence exists that stretches in the early contact phase of SSC actions such as running and jumping are powerful enough to induce sufficient muscle spindle afferent activation. This would also mean that stretch reflexes are contributing to the efficacy of motor output by making it more powerful. According to Voigt et al. (1998), the combination of the “pre-reflex” background activation and the following reflex activation might represent a scenario that supports yield compensation and fast rate of force development. The concept of elastic storage favours also the existence of reflex activation since high muscular activation during the braking phase of SSC is a prerequisite for efficient storage of elastic energy. All these aspects may contribute to the observation that mechanical efficiency in natural SSC is higher than in pure concentric exercise (Aura and Komi 1986; Kyrolainen et al. 1990).

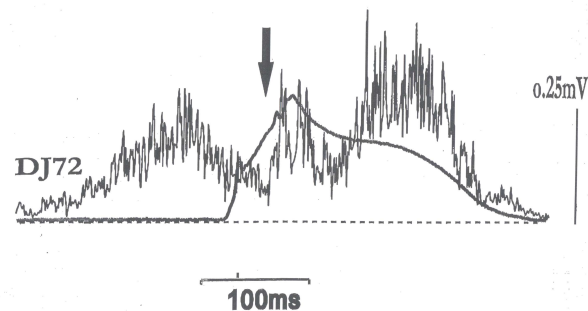
#### 1.5.4. Task dependent modulation of the reflex gain

A common finding of many drop jump studies is that, as the height of the drop preceding the rebound is increased, performance can initially improve (Asmussen and Bonde-Petersen 1974; Bosco et al. 1981), but eventually will decrease (Komi and Bosco 1978). In drop-jumps (Fig. 17), the short-latency reflex (SLR) response showed higher amplitude with increased drop height from 20 to 40 and 60 cm, respectively (Komi and Gollhofer 1997). However, in jumps from excessive heights (80 cm), the SLR was diminished. Despite larger impact loads and higher stretch velocities, the SLR was decreased suggesting decreased facilitation from muscle spindles and/or increased inhibitory drive from various sources such as Golgi tendon organs or voluntary protection mechanisms (Komi and Gollhofer 1997). Changes in excitability of the Ia afferent pathway could occur at the spinal level or could be induced by an altered fusimotor drive. To our knowledge, there is no study which investigated fusimotor drive during jumping, but the excitability at the spinal level has been recently examined (Leukel et al. 2008). In this study, H-reflex excitability during the SLR component was compared in the landing phase of drop-jumps from excessive and normal heights. H-reflex excitability and H/M ratio were found to be reduced at SLR at excessive heights as compared to normal heights. H-reflex excitability was not different at the time of ground impact. Their study supported the earlier hypothesis of a “prevention strategy” to reduce eccentric stress on the tendomuscular system (Schmidtbleicher and Gollhofer 1982; Komi and Gollhofer 1997). Presynaptic inhibition of Ia afferents was thought as most likely responsible for the change in H-reflex excitability between the two jump conditions (Leukel et al. 2008).



**Fig. 17. Rectified and averaged EMG pattern of the soleus muscle and vertical ground reaction force in both leg hopping (BLG) and in various drop jumps from different dropping heights (20 - 80 cm).** The figure illustrates the modulation in the EMG pattern and in the force record with increasing stretch load. The broken vertical line indicates the initiation of the phasic activation with a latency of 40 ms after ground impact. (Adapted from Komi and Gollhofer 1997)

In this line, in drop jumps with high stretch loads, neuromuscular inhibition is often observed prior to reflex activation (Fig. 18) (Gollhofer et al. 1992). Similar reflex behaviour could be observed in cats by (Prochazka et al. 1977). The authors discussed these reductions as a strategy of the neuromuscular system to reset the motoneurons in order to get a triggered and synchronized activation of all motor units, simultaneously with the ground impact, i.e. with the stretching of the extensor muscles. Similar EMG patterns have been recently observed in a landing task from supra-maximal dropping height (Galindo et al. in press). This has been further confirmed by the recent comparison of landing and rebound tasks from a given maximal dropping height on the sledge ergometer (Galindo et al. Unpublished data). This will be further discussed in chapter III.



**Fig. 18. Rectified and averaged EMG pattern of the gastrocnemius muscle and vertical ground reaction force of a drop jump from 72 cm height.** Note that the EMG profile shows a clear reduction in amplitude already prior to contact, which is consistent until the mid-phase of contact. The phase of reduction is dispersed by a phasic EMG contribution (see vertical arrow) occurring 43 ms after the ground impact. (Adapted from Gollhofer et al. 1992)

Additionally, ultrasonography results demonstrate that the fascicle-tendinous tissues interaction can be modified by the prestretch and rebound intensity efforts (Ishikawa and Komi 2004; Ishikawa et al. 2006b). Their studies demonstrate that during SSC exercises not only the stretch intensity, but also the rebound intensity can have considerable influence on the activation pattern and consequently on the elastic energy storage and recoil via fascicle length modification. It is worth noting that, for a specific muscle, there may be critical prestretch intensities that can be tolerated, in a sense that if this stretch speed is exceeded (*e.g.* very-high drop jump), the fascicles may lose their ability to resist the stretch. This is clearly observed in supra-maximal landing conditions (Galindo et al. in press). In SSC, the attached cross-bridges may thus be broken so that the subsequent rebound height in the concentric push off phase is decreased (Ishikawa and Komi 2004). This phenomenon may explain why, after a certain drop height, jumping performance decreases (Asmussen and Bonde-Petersen 1974; Komi and Bosco 1978).

## **1.6. Concluding comments**

These overall observations demonstrate the richness of the neuromuscular adjustments that are taking place in a natural SSC muscle action and give, I hope, some support to my fascination for this model. It is suggested that in the non fatigued state a proper activation of the muscles allow the muscle-tendon system to damp the impact and to resist the imposed stretch in order to favour the storage and subsequent recoil of elastic energy. Stretch-reflexes appear to play a major role in this process, but their intervention should be considered within the overall pre- and post-impact activation pattern. Similarly, the muscle-tendon unit should be examined more precisely for each of its component.

Examination of SSC fatigue might have been the natural follow-up of our SSC studies, but this has been in fact conducted in parallel, with a clear influence on the protocols developed in each of these 2 research lines. In particular, this made us examining the SSC fatigue effects in different (maximal and submaximal) testing tasks and repeatedly along the several days of the neuromuscular recovery period, with special emphasis on the stretch-reflex response. Similarly, this has led us to investigate more recently the neuromuscular adjustments in the stressful situation of supra-maximal landings.

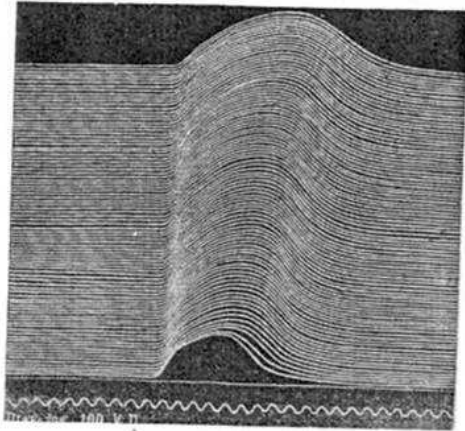
## **2. Neuromuscular fatigue of exhaustive SSC exercise**

This second half focuses on neuromuscular fatigue during exercise involving the stretch-shortening cycle, which occurs during natural, but exhaustive activities such as short and intensive or prolonged running and jumping exercises. There is no doubt that much before the true SSC studies had started, muscle fatigue had been widely explored for decades. These studies have introduced vast amount of information and several mechanisms have been proposed and proven to explain the various fatigue events (for a review, Gandevia 2001). This information has, however, come primarily from studies utilising isolated forms of muscle action (isometric, concentric or eccentric) in human and animal models. Many of these fundamental fatigue mechanisms may not be transferred directly to SSC fatigue without considering the special features of SSC in general and the nature of SSC fatigue loading in particular. Although SSC fatigue is a relatively young object of research, it has now been explored enough so that its peculiarities can be presented in a review form. Further investigations are needed, however, to understand further the underlying fatigue mechanisms.

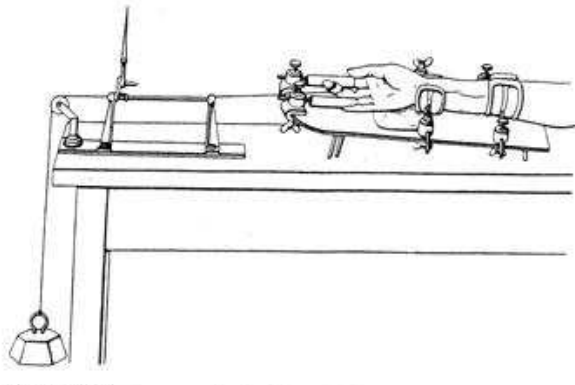
### **2.1. Major historical developments to characterize fatigue**

As a member of the IFR Etienne-Jules Marey, it is for me a privilege to cite him as one of the first scientists who brought some valuable information about the functional effects of fatigue. Fig. 19 is from Marey (1868) and serves as a demonstration on a frog muscle of the fatigue-induced changes in the mechanical responses to stimulation. The first scientist who studied then objectively “Muscle Fatigue” in humans was probably Mosso who developed an ergograph to record on a smoked drum the effects of given loads on repetitive movements performed by the middle finger of the hand (Fig. 20) (Mosso 1892). At that time already, Mosso was able to demonstrate the high individuality of the fatigue response (Fig. 21A) as well as the positive effects of the increased nervous arousal (Fig. 21B). Although the concept of “central fatigue” dates back to the work of Mosso (1892), the distinction between “central” and “peripheral” fatigue has emerged only slowly in the 1950s and it is still the subject of considerable controversy. By the present historical summary we would like to acknowledge the studies of Asmussen and the pertinence of his review on “Muscle fatigue” (Asmussen 1979). Today, more precise information can be found in the review of Gandevia (2001) who presented in particular the list of conceptual and technical advances that have been highly relevant to examine the contributions of the central nervous system to human muscle fatigue.

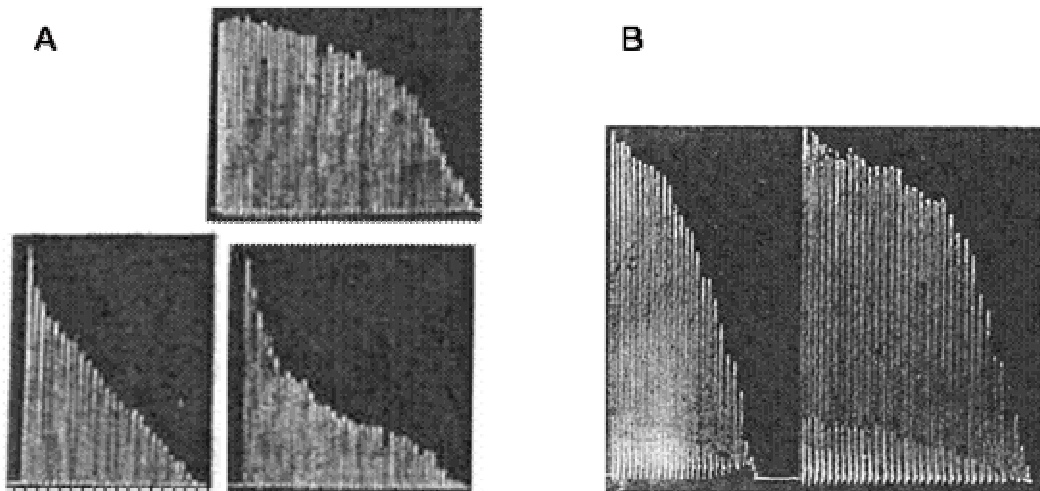




**Fig. 19 Superimposed contractions of a frog muscle.** First contraction at bottom. (From Marey 1868).



**Fig. 20 The classical finger ergograph developed by Mosso (1892)**



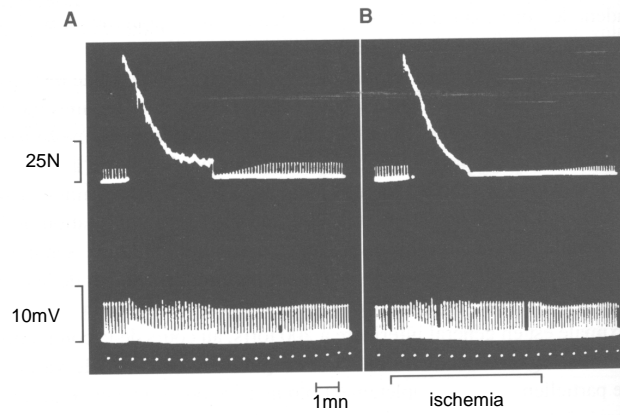
**Fig. 21 (A) Three individual fatigue curves.** (Adapted from Mosso 1892)  
**(B) Two fatigue curves illustrating the central arousal effect.** From a young colleague of Mosso, the left recorded before his inaugural lecture and the right recorded directly after lecturing. (Adapted from Mosso 1892)

One of the first major steps in the evaluation of the “*central*” versus *peripheral*” components of fatigue is the development by Merton (Merton 1954) of the *twitch interpolation technique*. This allowed to assess in humans maximal voluntary activation and to predict maximal evocable force. The increment in force so produced (superimposed or interpolated twitch) is inversely related to the strength of the activation (Merton 1954). No force increment appears when voluntary force approached maximal values, so that it has been used to measure the excitation level of motoneurons (referred to as “voluntary activation” or “neural drive”). Measurements of *voluntary activation* use the principle that those motor units, which are not engaged during a voluntary effort, can be activated by electrical stimulation of the nerve or muscle. When the added electrical stimulation does not increase the measured voluntary force curve, the level of voluntary activation is considered as 100%. In the absence of fatigue, full activation of the adductor pollicis was demonstrated by Merton (1954). This technique remained then unused until the 1980s when Belanger and McComas (1981) reassessed its assumptions and applicability for the limb muscles. They suggested that the level of voluntary activation could vary among muscles. Since then, voluntary activation has been reported as 85-95 % for the knee extensors and >95% for the plantarflexors (Taylor et al. 2008).

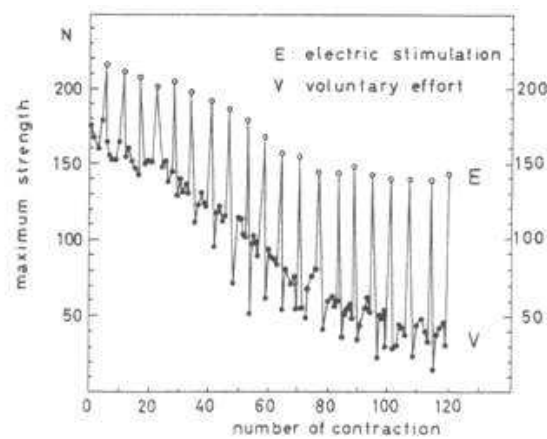
Merton applied this technique also to fatigue conditions (Merton 1954). During the course of a maintained isometric effort with the adductor pollicis muscle, he found no superimposed twitch to single nerve shocks but a decrease of the resting twitch, and suggested a purely peripheral site of the muscle fatigue (Fig. 22A). This study provided additional insight that there was no recovery of voluntary strength until the blood flow to the muscles was restored (Fig. 22B). In contrast, Ikai et al. (1967) found relatively increasing contractions as responses to short bursts of tetanizing stimuli applied repeatedly during a series of maximal voluntary contractions (Fig. 23) that supported the hypothesis of a central component of muscle fatigue. Bigland-Ritchie et al. (1978) confirmed these findings in the bigger muscle group of the quadriceps femoris (Fig. 24), but this was found in only some of their subjects.

In terms of methodology, Asmussen (1979) already indicated that “central fatigue should show itself as a decrease in the EMG signals from “centrally fatigued” muscles. A parallel fall in EMG and mechanical output in fatigue should then be expected but, as a fall in EMG would also be the results of peripheral transmission fatigue, the crucial experiment should include not only recordings of parallel changes in mechanical output and EMG but also recordings of muscle action potentials elicited by electrical stimulation distal to the spinal motor neurons. Such experiments have to my knowledge not yet been performed”. Now it

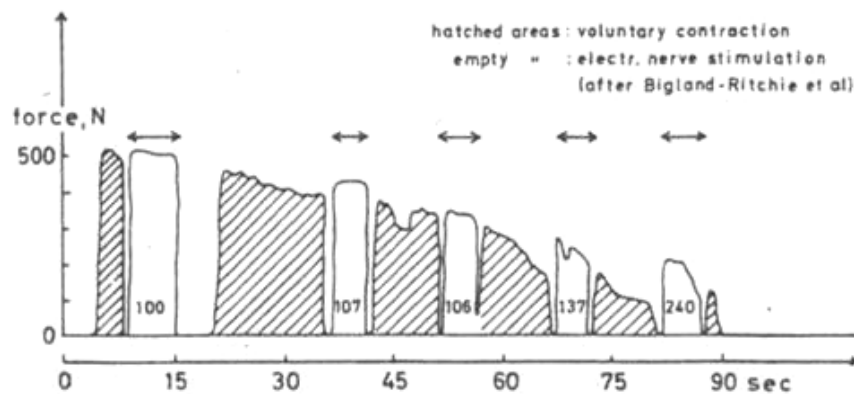
seems quite obvious that those comments of Asmussen should have been more widely followed in SSC fatigue studies.



**Fig. 22.** (A) A maximal voluntary effort persisted until severely fatigued. A series of single twitches precede and follow. Lower trace, the corresponding action potentials. Time markers ½ min. (B). The same but with the circulation occluded for the period indicated by a line beneath the record. (From Merton 1954)



**Fig. 23.** Maximal voluntary isometric contractions of the thumb muscles (black dots) interrupted every five seconds by electrical stimulation of the ulnar nerve. (From Ikai et al. 1967)

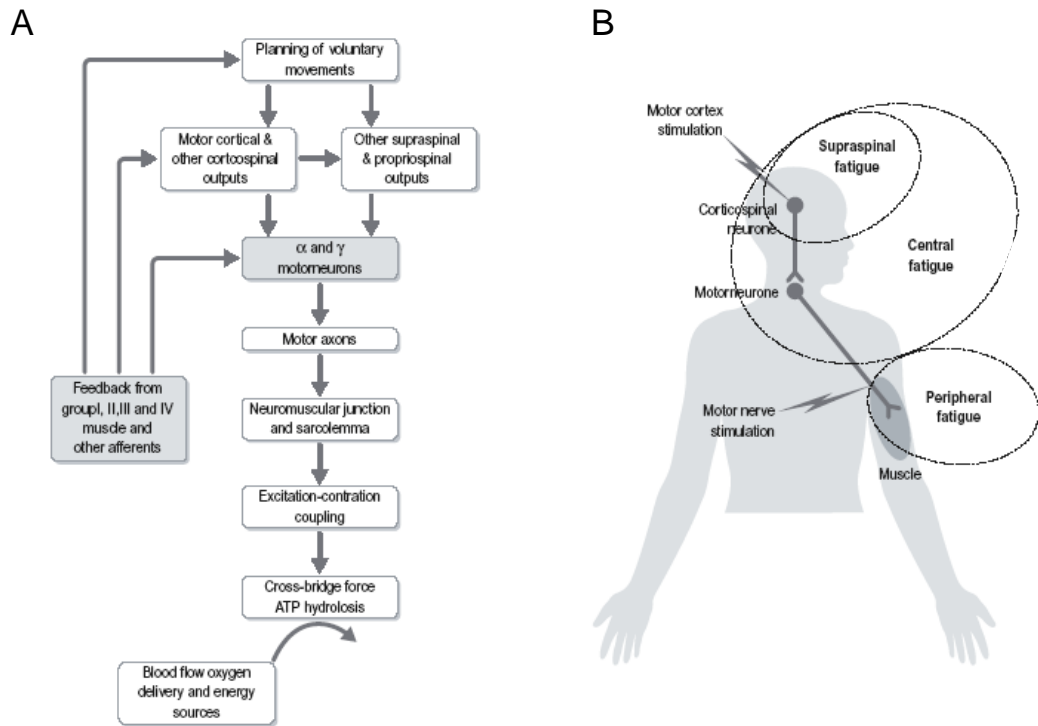


**Fig. 24.** Maximal voluntary isometric contractions of the quadriceps muscle group (hatched areas) interrupted by periods of electrical tetanizing stimulations (arrows) of femoral nerve. The tetanic forces (white areas) are expressed in percent of the force recorded immediately before stimulation (from Bigland-Ritchie et al. 1978 in Asmussen 1979).

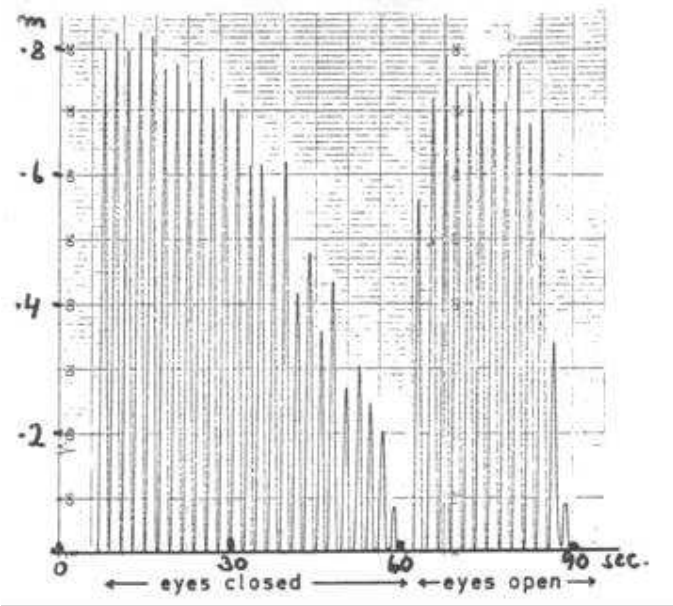
As early as in 1979, Asmussen concluded that ““*central*” and “*peripheral*” *fatigue* may appear separately or combined, depending on the specific situation”. The potential sites of impairments were introduced as “any one link in the *long chain* from the voluntary motor centers in the brain to the contractile filaments in the single muscle fibers may be the weaker and thus the most direct cause of muscle fatigue”. This is close to the traditional model for considering muscle performance traces a causative “chain” from high levels within the central nervous system (CNS) via descending paths to the motoneurons and then via motor axons to the neuromuscular junction, the sarcolemma, t-tubules, and ultimately the actin and myosin interactions (Fig. 25A). More recently, supraspinal fatigue has been identified as a component of central fatigue (Fig. 25B) (Gandevia et al. 1996; Taylor et al. 2000a; 2006). Asmussen (1979) mentioned also the potential influence of “pain-producing catabolites” and suggested that ““central fatigue” is caused by an inhibition elicited by nervous impulses from receptors (probably some kind of chemoreceptors) in the fatigued muscles. The inhibition may act on the motor pathways anywhere from the voluntary centers in the brain to the spinal motor neurons.” This assumption is in complete agreement with the *feedback from muscle afferents* shown in Fig. 25A as going to the motoneurons, the motor cortex and premotor areas (Gandevia 2001).

Asmussen early works (Asmussen 1934 cited in Asmussen 1979) revealed the different levels of susceptibility to fatigue of two different peripheral sites: the “transmission mechanism” (neuro-muscular junction, muscle membrane and sarcoplasmic reticulum) and the “contractile mechanism” (muscle filaments) (Fig. 25). These observations are still considered as a major principle to emphasize the *task dependency of the site of impairments* (Enoka and Duchateau 2008). Figure 26 is another elegant demonstration of the task dependency. The classical ergograph task that consisted of lifting weights was performed with eyes closed until exhaustion. The simple instruction to open the eyes allowed the subjects to perform immediately 20-30 % more extra work (Asmussen and Mazin 1978). The reversed strategy (closing the eyes after reaching exhaustion with eyes opened) had no effect.

These observations emphasize the complexity of the SSC-induced impairments as this type of exercise involves a large number of muscles, different forms of muscle actions, different intensities and time durations (short and intensive versus prolonged exercises) and combined interventions of central and reflex neural components. Furthermore its recovery process is characterized by the delayed occurrence of muscle pain sensation, termed delayed onset muscle soreness (D.O.M.S.), which is known as suggested by Asmussen to influence muscle activation.



**Fig. 25 (A) The chain of processes leading to voluntary movement.** Feedback from muscle afferents is shown going to the motoneurons, the motor cortex and premotor areas. **(B) Division of muscle fatigue into peripheral and central fatigue by motor nerve stimulation.** Supraspinal fatigue is a subset of central fatigue. (Modified from Taylor et al. 2008).



**Fig. 26 Illustration of the “Task dependency” from arm ergograph recordings.** The lifting task started with subject’s eyes closed till exhaustion, at which time he opened his eyes and continued to exercise (From Asmussen & Mazin 1978).

## **2.2. Definitions of fatigue as they apply to repeated SSC loading**

At the influential Ciba Foundation symposium on human muscle fatigue held in London in 1980, however, “fatigue” applied to muscular exercise was defined as “failure to maintain the required or expected force” (Edwards 1981). This definition is close to the one earlier proposed by Asmussen (1979) in which fatigue referred to “the transient decrease in performance capacity of muscles when they have been active for a certain time, usually evidenced by a failure to maintain or develop a certain expected force or power”. As reviewed by Gandevia (2001) and Enoka and Duchateau (2008), however, such definitions may be criticised for their lack of precision and for the fact that fatigue really begins almost at the onset of exercise and develops progressively before the task failure. This may explain why many researchers adapted the definition of fatigue given by Bigland-Ritchie and Woods (1984): “*Any exercise-induced reduction in the ability to exert muscle force or power, regardless of whether or not the task can be sustained*”. Accordingly, the inability to continue a task is often termed “exhaustion” and considered as the culmination of these ongoing fatigue processes. This time is clearly dependent on the exercise intensity and on its mode (continuous or intermittent). In a submaximal exercise, fatigue may then occur without any decrement in the task performance as other motor units or muscles are recruited to compensate for those that are fatiguing.

With regard to the SSC task specificity defined in section 1, one can easily see that the loading characteristics are different from isolated isometric, concentric or eccentric actions. SSC is a natural but complex activity, which in itself is often difficult to understand in its all features. First, SSC is characterised by high impact forces that are often repeated over long durations (*e.g.* during a marathon run). In fatiguing SSC exercises, the impact loads are repeated over time, stressing the metabolic, mechanical and neural components. Intensive and/or unaccustomed SSC type exercises may thus result in reversible ultrastructural muscle damage and delayed onset muscle soreness. Secondly, the impact loads and the nature of stretches involved in the active braking phase of SSC are usually very fast, of short duration and controlled simultaneously by reflex and central neural pathways. The SSC fatigue model is of particular interest as it causes disturbances in stretch-reflex activation and, thus, provides an excellent basis for studying neuromuscular adaptation to exhaustive exercise. Finally, the magnitude of metabolic stress is dependent on the velocity of stretch and on the coupling time between stretch and shortening. Extending then the traditional fatigue thoughts to the SSC fatigue requires rearranging of the thinking of the possible mechanisms involved. It requires also complementary techniques of investigation.

### 2.3 Short- and long-term SSC fatiguing protocols

In the experiments to be reviewed in the following paragraphs, the impact loads in the SSC exercises were carefully controlled, but varied in terms of intensity and duration. In most of these studies, kinematics and kinetic techniques were combined with surface electromyographic (EMG) recordings to examine the changes during the course of the exercise as well as during the subsequent days of recovery. Our recent review (Nicol et al. 2006) included 45 SSC fatigue studies performed during the last 20 years on human subjects to which have been added 10 others.

Our own fatiguing protocols shown in Fig. 27 and indicated by an asterisk in Tables 1 and 2 consist of either prolonged running (30min-6h) or shorter but more intensive rebound series on a specific sledge apparatus developed in professor Komi's laboratory (Kaneko et al. 1984; Komi et al. 1987c). The rebound exercises consist either of continuous or intermittent series until exhaustion. The rebound height is set at either 70 or 80% of the maximal rebound height. The lower knee flexion reached at impact is also pre-set at 90 to 110 degrees, and checked all along the exercise by the use of visual feedback on the lower height. The net duration of the rebound exercise varies from 1-5 min in the continuous protocol to 6-20 min for the intermittent one. Fatigue effects of a leisure week of alpine skiing (Strojnik et al. 2001) have been tested as well.

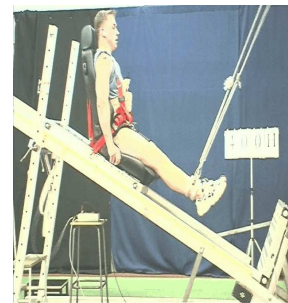
**Experimental marathon**



**Treadmill**



**Intensive bilateral rebounds**



**Unilateral rebound series (continuous or intermittent series of 30-40 rebounds until**



**Fig. 27 Presentation of the different fatiguing SSC protocols reported in our papers, with the exception of the alpine skiing one.**

More generally, and as shown in Tables 1 and 2, the prolonged SSC protocols included different duration-intensity combinations of endurance running and skiing exercises. The short and intensive SSC exercises included series of either rebound or running exercises that were for some of them of fixed duration, but in most cases the exercises were performed until exhaustion.

The long-lasting SSC performances serve for the purpose to characterize more specifically the effects of a great number of repeated eccentric muscle actions on various aspects of the neuromuscular function. As shown in Table 1, the running protocols varied from a 10-km run by non endurance runners (Nicol et al. 2003) to a 65-km ultramarathon race 65 km with an altitude difference of 2500 m ( $\approx$  8 h 30 min) by well-trained runners (Millet et al. 2002). The cross-country skiing protocols varied similarly, from a skiing marathon ( $\approx$  2 h 30 min) (Millet et al. 2003b) to an 85-km skiing race ( $\approx$  4 to 10 h) (Viitasalo et al. 1982). The braking (or impact) phases are naturally different depending on the activity. Cross-country skiing (X-C) is a typical form of locomotion, in which the braking phase is long and relatively smooth in both free and traditional techniques. In the latter technique the vertical ski forces seldom exceed 1.5 times body weight (Komi and Norman 1987). Consequently the repeated SSC actions in this event will have much smaller functional, structural and damaging effects on muscle if compared to running, for example. In this line, Takashima et al. (2007) reported an early (24h) MVC recovery after a 50-km cross-country skiing that was found to result in very limited muscle damage. Except perhaps for ultra long performances, the impact peak in running is much higher and the duration of the braking (“eccentric” phase) short (50-120 ms). The repeated loading will consequently have greater stretch-induced effects than in X-C (Millet and Lepers 2004). These two events represent a good comparison, especially regarding the long-term SSC fatigue effects. It is not possible for a runner to repeat the marathon race in one week intervals due to the typical SSC fatigue induced structural damage. In X-C, even when performed with the same intensity and duration as the marathon run race, the recovery processes from possible muscle damage take place much faster, and the athletes are usually ready to repeat the 50 km race after a few days only. This emphasizes that SSC, when repeated long enough and with high intensity causes reversible neural, structural, and functional disturbances, severity and duration of which are dependent on the nature of SSC task.



**Table 1: Long-lasting SSC exercises reported in the literature.** This list that includes running and skiing (indicated in *italic*) protocols may not be exhaustive. Their classification is based on the exercise time duration. Our own studies are indicated by an asterisk.

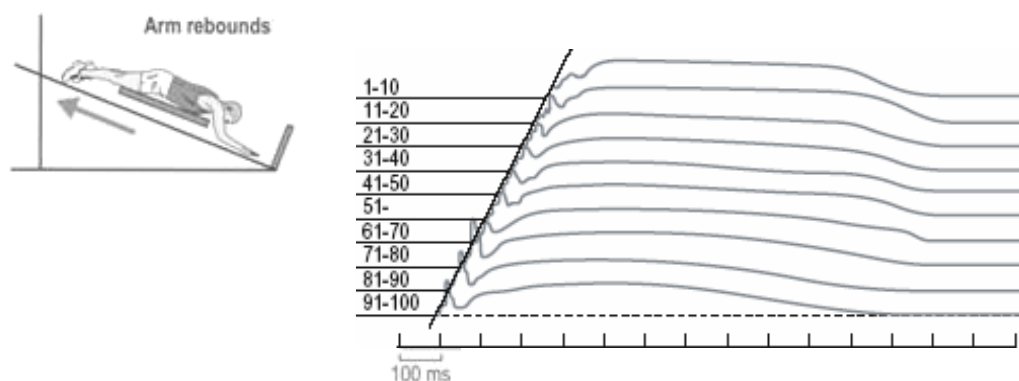
<b>Long-lasting fatiguing SSC exercise</b>		
Verin et al.	2004	Incremental treadmill running test ( $\approx$ 18 min)
Martin et al. (b)	2004	30min downhill treadmill run
Millet et al.	2004	30 min downhill treadmill run
Ftaiti et al.*	2001	45 min treadmill run at 65% MAV with heat stress and dehydration
Petersen et al.	2001	90 min downhill run at 75%, -5% slope
Davies & White	1982	1h treadmill run at 80%
Paavolainen et al.	1999	10-km ( $\approx$ 36-39min) run
Nicol et al.*	2003	10 km run
Racinais et al.	2007	90 min running (continuous vs intermittent)
Lepers et al.	2000	2h run at 75% MAV
Saldanha et al.	2007	2h treadmill running test at 75% VO <sub>2</sub> max
Millet et al. (a)	2003	30-km ( $\approx$ 2h 30min) running race
Lepers & Hausswirth	1984	Marathon run vs. Triathlon vs 45 min run
Sherman et al.	1984	Marathon race
Hyvärinen et al.	1985	Marathon run
Nicol et al. (a) (b) (c) *	1991	Marathon run ( $\approx$ 2-3h)
Pullinen et al.	1997	Marathon run
Avela and Komi (a) (b)	1998	Marathon run
Avela et al.	1999	Marathon run
Ross et al.	2007	Marathon treadmill run ( $\approx$ 3-4h)
Girard et al.	2007	3h of tennis match at 75% FCmax
Davies & Thompson	1986	4h treadmill run
Place et al.	2004	5h treadmill run at 55% MAV ( $\Leftrightarrow$ 52km)
Millet et al. (a) (b)	2002	65-km ( $\approx$ 6-8h) ultramarathon run
<i>Millet et al. (b)</i>	<i>2003</i>	<i>Ski skating marathon</i>
<i>Takashima et al.</i>	<i>2007</i>	<i>50km cross-country skiing</i>
<i>Viitasalo et al.</i>	<i>1982</i>	<i>65 km (<math>\approx</math> 4-10h) cross-country skiing race</i>
<i>Forsberg et al.</i>	<i>1979</i>	<i>60miles (<math>\approx</math> 4-6h) cross-country skiing race</i>
<i>Strojnik et al.*</i>	<i>2001</i>	<i>1 week of leisure alpine skiing</i>

The short-term SSC exercise protocols (Table 2) consist usually of intensive and exhaustive series of rebounds performed on the sledge apparatus. By adjusting the subject's position on the gliding sledge, fatigue can be induced selectively in the lower- (Fig. 27) or in the upper-limb muscles (Fig. 28). The upper-limb protocol used by (Gollhofer et al. 1987a, 1987b) includes 100 submaximal SSC rebounds with both arms. In the basic fatiguing protocol of the lower-limb extensor muscles, the exercise is performed in a sitting position by rebounding as long as possible to a given submaximal rising height (70-80 % of the maximal rebound height). Exhaustion is usually reached after 100-400 consecutive submaximal rebounds ( Horita et al. 1996, 1999; Nicol et al. 1996a, 1996b; Avela and Komi 1998), for a total of 1-5 min of intensive work.. The severity of the fatigue protocol can be increased by adding 100 maximal drop-jumps prior to the exhaustive exercise (Kuitunen et al. 2002, 2004, 2007; Nicol et al. 2003) However, facing the premature stop of some of the subjects possibly

due to central fatigue and/or rapid metabolic fatigue, the continuous rebound exercise has been recently replaced by an intermittent rebound exercise that consisted of series of 30 to 40 successive rebounds with intermediate recovery periods of 3 min (Regueme et al. 2005b, 2005c, 2007a, 2007b, 2008). This resulted in a systematic increase of the net SSC exercise duration (6-20 min) that secured in most subjects the occurrence of delayed fatigue effects.

**Table 2: Short and intensive SSC exercises reported in the literature.** This list that includes jumping, running and skiing (indicated in *italic*) protocols may not be exhaustive. Our own studies are indicated by an asterisk.

Short and intense fatiguing SSC exercise		
Gollhofer et al. (a) (b)	1987	100 submax intermittent arm rebounds
Hortobagyi et al.	1991	50 maximal Drop-Jumps
Skurvydas et al.	2002	100 maximal drop jumps
Skurvydas et al.	2000	100 intermittent or continuous Drop Jumps
Martin et al. (a)	2004	15*1min one-legged downhill treadmill run
Strojnik and Komi	1998	Exhaustive maximal leg rebounds ( $\approx$ 1min )
Nicol et al. (a) (b)*	1996	Exhaustive submax leg rebounds ( $\approx$ 3 min)
Horita et al.*	1996	Exhaustive submax leg rebounds
Horita et al.*	1999	Exhaustive submax leg rebounds ( $\approx$ 3 min)
Strojnik and Komi	2000	Exhaustive submax leg rebounds
Horita et al.*	2002	Exhaustive submax leg rebounds
Kuitunen et al.*	2002	100 max and exhaustive submax leg rebounds
Nicol et al.*	2003	100 max and exhaustive submax leg rebounds
Kuitunen et al.	2004	100 max and exhaustive submax leg rebounds
Ishikawa et al.*	2006	100 max and exhaustive submax leg rebounds
Dousset et al.	2007	100 max and exhaustive submax leg rebounds
Regueme et al.*	2005	Exhaustive intermittent submax leg rebounds ( $\approx$ 15 min)
Regueme et al. (b)*	2007	Exhaustive intermittent submax leg rebounds ( $\approx$ 9 min)
Regueme et al. (a)*	2007	Exhaustive intermittent submax leg rebounds ( $\approx$ 11 min)
Regueme et al.*	2008	Exhaustive intermittent submax leg rebounds ( $\approx$ 6 min)



**Fig. 28. Progression of SSC fatigue induced by sledge rebound exercise performed with the upper limbs (Gollhofer et al. 1987b).** The force plate records (averaged over 10 successive contacts) show progressive increases in the impact peak and contact time.

It is noteworthy that the first SSC fatigue studies mentioned in Table 2 include fatiguing protocols of fixed and rather short duration. Based on the large inter-individual variability in exhaustion time systematically observed in our own experiments, it is suggested that a short term SSC exercise of fixed duration may not lead each subject to exhaustion. This may not apply, however, to the 100 submaximal rebound exercise performed with the upper-limbs on the sledge (Gollhofer et al. 1987a, 1987b). The large and systematic changes observed in the normal component of the reaction force (Fig. 28) are likely to indicate that all subjects had reached exhaustion.

## **2.4. How is SSC fatigue revealed through various testing protocols?**

### ***2.4.1. Common protocols***

Only a few SSC studies investigated the fatigue response during the time course of the exercise. In most studies, the exercise-induced impairments were examined by performing repeated static and dynamic strength tests immediately before the SSC exercise and during the acute recovery phase (< 2h). In some studies, the tests were repeated at 2h and along the delayed recovery period (at 2 days as well as 4-5 and 7-10 days later). In the present review we will combine their outcomes for three given time periods: (1) during the course of the exhaustive SSC exercise and along its subsequent (2) acute and (3) delayed recovery phases.

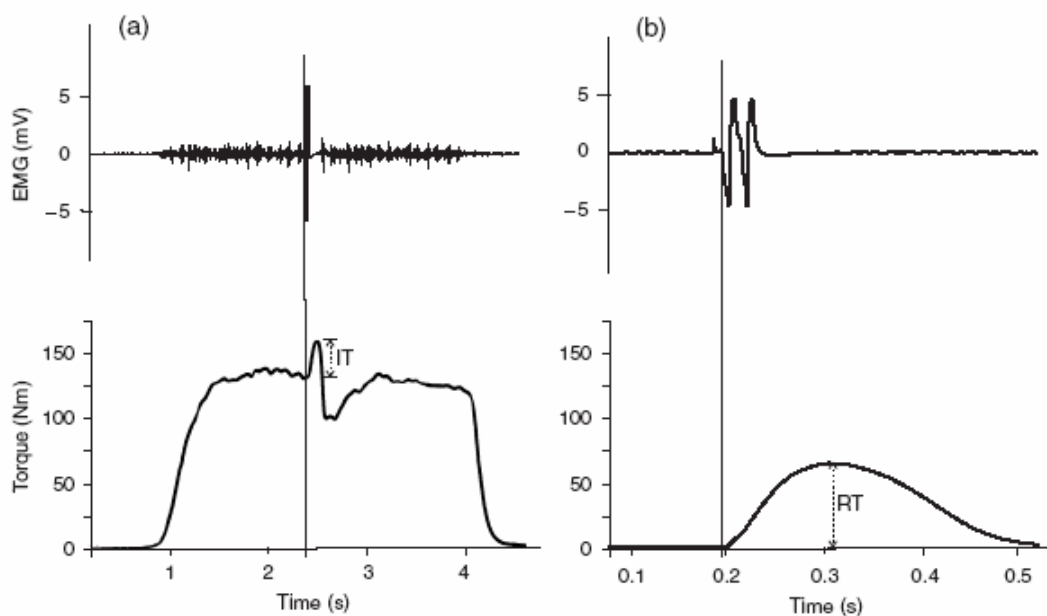
In terms of methodology, we will not concentrate on our own testing protocols as more details can be found in the enclosed original papers. Without being exhaustive, we will rather concentrate on the techniques used to investigate the central aspects of fatigue because of their clear lack of use in the SSC literature. We do intend also to use more such techniques in our future investigations.

### ***2.4.2. Measures of “voluntary activation”***

As previously shown in Fig. 26B, muscle fatigue can be divided into two aspects: “peripheral fatigue”, which includes processes at or distal to the neuromuscular junction and “central fatigue”, which includes more proximal processes and is defined as a progressive reduction in voluntary activation of muscle during exercise (Gandevia 2001). That is, despite maximal voluntary effort, the motor units are not driven fast enough to generate maximal force. Different techniques have been developed to quantify the fatigue induced fall in “voluntary activation”. It should be considered, however, that alterations in intrinsic motoneurone

properties, sensory feedback or descending drive can contribute to central fatigue. We will thus present some of the recent techniques that intend to isolate these different components.

*The twitch superposition (or interpolation) technique with peripheral nerve or muscle stimulation* (Merton 1954) commonly used in fatiguing isometric tasks (e.g. Bigland-Ritchie et al. 1983a, 1983b; McKenzie et al. 1992) has only been recently applied to SSC fatigue, and mostly used in the acute (< 30min) recovery phase (Millet et al. 2002, 2003a, 2003b; Martin et al. 2004a; Place et al. 2004; Racinais et al. 2007; Saldanha et al. 2008). Some authors have used double maximal shocks superimposed to maximal contraction (Strojnik and Komi 1998; Strojnik et al. 2001; Avela et al. 2001, 2004). In the studies of Martin et al. (2004a, 2004b), transcutaneous stimulation was found to be less painful than nerve stimulation. The capacity to fully activate a muscle is measured by comparing the twitch superimposed to a MVC to the twitch evoked on the relaxed muscle (Merton 1954) (Fig.29).



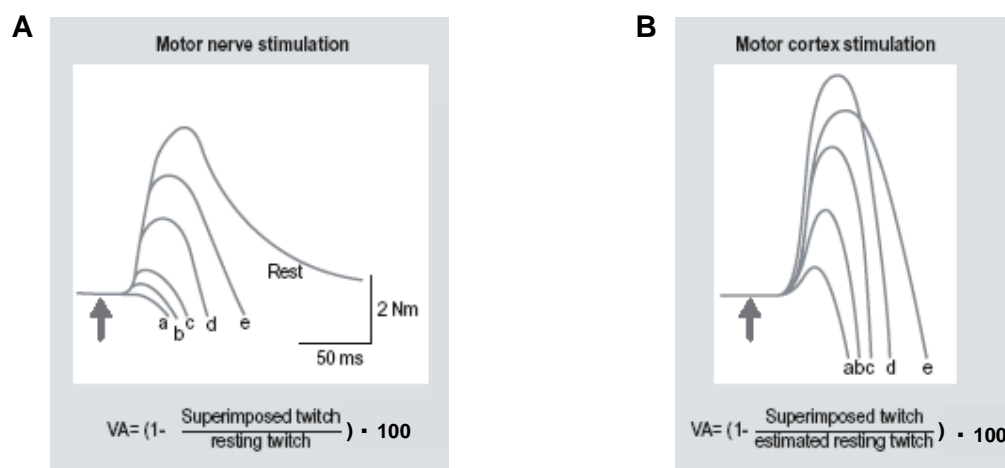
**Fig. 29. Example of the electromyogram (EMG, upper traces) and torque (lower traces) responses to the twitch interpolation stimulus.**

(a) Maximal voluntary isometric contraction (MVC) with electrical stimulation applied to the tibial nerve (above) and torque produced during MVC with interpolated twitch (IT) caused by the electrical stimulation (below).

(b) EMG from the electrical stimulation applied at rest (above) and torque produced by the electrical stimulation applied at rest (resting twitch, RT) (below). The vertical lines in (a) and in (b) indicate the deflection in the M-wave (EMG) taken as the reference for the timing of the twitch measurements. Note the differences in time scales between (a) and (b).

(From Saldanha et al. 2008)

As indicated in Fig. 30A, the percentage of voluntary activation (VA) is thus calculated as follows:  $VA (\%) = (1 - \text{Super-imposed twitch} / \text{resting twitch}) \times 100$  (Allen et al. 1995).



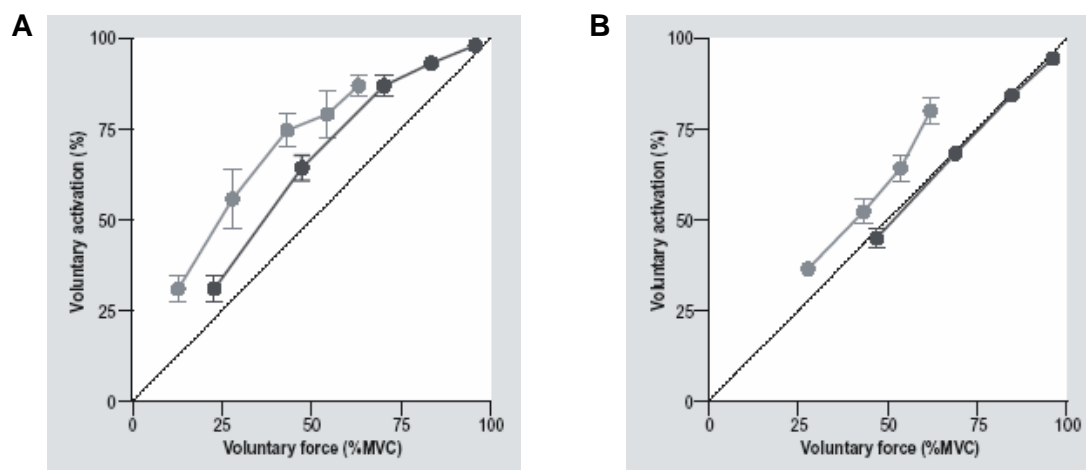
**Fig. 30. Voluntary activation measured with motor nerve stimulation or with motor cortex stimulation.** The top panels show superimposed twitches evoked during brief isometric voluntary elbow flexions of 100% (a), 90% (b), 75% (c), 50% (d) and 25% MVC (e) in one subject. The background forces are offset to allow comparison of the twitches. Arrows indicate the timing of stimulation.

**(A)** Responses to motor nerve stimulation of biceps and brachialis. These include a twitch evoked from the resting muscle (rest). To calculate voluntary activation (VA) the size of the superimposed twitch is compared to the size of the resting twitch.

**(B)** Responses evoked from all the elbow flexors by transcranial magnetic stimulation of the motor cortex. To calculate voluntary activation (VA), the size of a resting twitch is estimated by extrapolation of a linear regression of superimposed twitch size against voluntary force for contractions >50% MVC. The superimposed twitch is then compared to the estimated resting twitch. (From Todd et al. 2003b)

It is now clearly established that complete or maximal VA is the exception rather than the rule during attempted MVC, even in the absence of fatigue, in well-motivated subjects using feedback of force. For instance, Belanger and McComas (1981) found that ankle plantarflexors could not be activated as fully as ankle dorsiflexors in MVC. This difficulty applies in particular to the soleus muscle and has been confirmed by others (Bawa et al. 2002; Bigland-Ritchie et al. 1986). At the end of prolonged (90 to 180 s) MVC tasks, voluntary activation is reported to fall by 10-15 % (Gandevia et al. 1996; Rattey et al. 2006). Notwithstanding, this technique presents some limitations. As shown in Fig. 31A, the amplitude of the interpolated twitch is nonlinearly related to “excitation” of the motoneurons pool, so that at near-maximal contraction intensities (> 90% MVC) the accuracy of VA is low as larger increases in force are observed for smaller changes in activation of a non fatigued muscle (Allen et al. 1998; Behm et al. 2001; Todd et al. 2003). Another limitation is that the

influence of synergists on the variability in VA and force production is unknown (Allen et al. 1998).

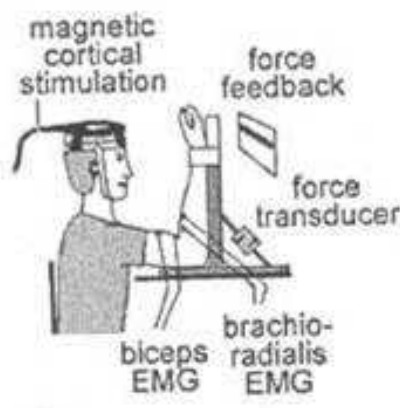


**Fig. 31. Voluntary activation measured in fresh and fatigued muscle conditions.** Voluntary activation was measured over a range of contraction strengths for a group of subjects when the muscle was fresh (black circles) or when it was fatigued (grey circles) such that maximal voluntary force had fallen by 40%. **(A)** Voluntary activation measured with motor nerve stimulation shows a curvilinear relationship to voluntary force. **(B)** Voluntary activation measured with motor cortex stimulation shows a linear relationship to voluntary force both when the muscle is fresh and when it is fatigued. (From Todd et al. 2003b)

Finally, one should also be aware that the magnitude of the resting twitch, often used also as an indicator of peripheral fatigue, is the net result of fatigue phenomena causing a decrease of the resting twitch, and post-activation potentiation causing an increase (Rassier and Macintosh 2000). Higher peak twitch may thus be found in the fatigued state with depressed tetanus force (Rassier and Macintosh 2000). Furthermore, post-activation potentiation seems to be greater in endurance-trained people than in sedentary subjects and “habitual weight trainers” (Hamada et al. 2000).

Voluntary activation can also be measured using the “*central activation ratio*” (Kent-Braun and Le Blanc 1996; Behm et al. 2001). In this technique, a train of stimuli rather than single or double stimuli is delivered during a MVC contraction. The voluntary force prior to stimulation is compared to the total force (voluntary plus evoked) during stimulation. This technique shows similar falls in voluntary activation close to 20% when measured on ankle dorsiflexors during a 4 min MVC (Kent-Braun 1999). The major drawback is that the comparison is likely to overestimate voluntary activation unless all synergists can be stimulated maximally. Trains of stimuli can also evoke inhibitory reflexes between synergists and reduce the voluntary contribution to force (Naito et al. 1996).

More recently, the contribution of descending drive has been explored with *transcranial magnetic stimulation (TMS) over the motor cortex* during maximal voluntary efforts (Fig. 32). The elicited a superimposed twitch that gives a measure of VA (Gandevia et al. 1996; Todd et al. 2003b) (Fig. 30B). This technique identifies the *supraspinal fatigue* component of central fatigue (Gandevia et al. 1996; Taylor et al. 1996), which is defined as fatigue produced by failure to generate output from the motor cortex. This means that during fatiguing exercise, motor cortical output becomes less able to drive the motoneurons and muscles maximally although there is additional output available which is not employed voluntarily but can be elicited with TMS. The advantage of the supraspinal fatigue process remains controversial, but supraspinal fatigue has been proposed to be a protective mechanism to prevent irreversible exercise-induced muscle damage and/or homeostatic failure (Sacco et al. 1997).



**Fig. 32. Experimental arrangement for magnetic cortical stimulation (TMS).** Subject seated with the right arm on a myograph with force feedback provided and EMGs from arm muscles recorded. Stimuli are delivered to the cortex via a magnetic coil.

As shown in Fig. 30B, calculation of VA is similar to the one used with the twitch interpolation technique, except that in the TMS technique the twitch evoked response at rest is estimated rather than measured directly (Todd et al. 2003b, 2004). This is because motor cortical neurones and motoneurons are less excitable with the muscle at rest than during voluntary activity. In this line, it is theoretically impossible for VA measured with TMS to be worse than that measured with motor nerve stimulation, since impairments in voluntary activation shown by the former method are a subset of the latter one (Fig. 25B). After eccentric exercise of the elbow flexor muscles, Prasartwuth et al. (2005) showed impairment in voluntary activation when estimated by nerve stimulation but not by TMS. As previously mentioned, however, the relationship of VA to contraction strength is curvilinear for

peripheral nerve stimulation (Fig. 31A). In contrast, it is linear for TMS (Fig. 31B), so that the values derived by the two techniques are not easily compared (Taylor et al. 2006; Todd et al. 2003b). In addition, the relationship between voluntary activation measured by TMS and voluntary force remains linear with fatigue for medium to high contraction strengths, so that it is possible to estimate the proportion of force loss attributable to supraspinal fatigue (Todd et al. 2003b; Taylor et al. 2006). This varies from about 25% during a 2-min MVC to about 40% during a 40 min 15% MVC. The TMS technique has been successfully used to demonstrate supraspinal fatigue during sustained and intermittent isometric MVCs of the elbow flexors, as well as during bouts of maximal concentric and eccentric contractions (Gandevia et al. 1996; Taylor et al. 2000a; Löscher and Nordlund 2002). Sustained submaximal isometric contraction also results in progressive supraspinal fatigue tested during occasional brief MVCs (Søgaard et al. 2006).

To our knowledge, only one SSC study (Ross et al. 2007) investigated with TMS supraspinal fatigue of the lower limb muscles after prolonged, whole body exercise. It is noticeable; however, that the investigated muscle was the tibialis anterior (TA) muscle rather than one of the lower limb extensor muscles that should be more fatigued by the experimental marathon run. The TA choice was based on the fact that stronger TA corticomotoneuronal connections (exclusively facilitated responses of consistent magnitudes) have been observed when compared with those onto soleus or gastrocnemius motoneurons (Brouwer and Qiao 1995; Bawa et al. 2002). A near-maximal excitatory response in the ankle flexors may thus be achieved with only a small response in the antagonist ankle extensors. This is essential as the lack of precision in the stimulated muscle constitutes a clear limitation of this technique.

In terms of our research perspectives, it is noteworthy that Brouwer and Qiao (1995) reported that the occurrence of strong facilitatory and inhibitory responses or an absence of a response within a given gastrocnemius medialis motoneuron pool was associated with coefficients of variation about 13 times that of TA and 4 times that of soleus. They suggested that the degree of non-homogeneity within a motoneuron pool reflects the ability of the cortex to modulate other inputs. Stimulation of the triceps surae muscle group remains therefore of interest for the examination of the central SSC fatigue effects.

#### ***2.4.3. Focal changes in cortical excitability***

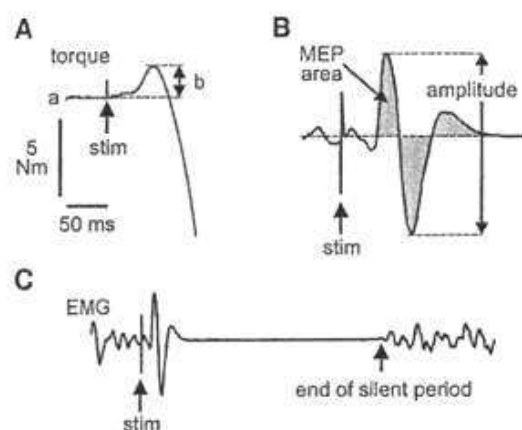
TMS evokes a short-latency excitatory response in most muscles in humans. The motor response is seen in the EMG as motor evoked potential (MEP) (Fig. 33). As reviewed by



Gandevia (2001), different studies using TMS have revealed lability in the motor cortical response to exercise. Not only is the drive to corticospinal cells suboptimal, but the apparent “excitability” of cortical circuit also changes.

The characteristics of the MEP response are well known to be sensitive to fatigue both during the exercise and after it. However, the changes in the response to TMS that occur during and after a fatiguing task are complex.

During the fatiguing effort itself, both excitatory and inhibitory mechanisms take place. As illustrated in Fig. 34 during a sustained MVC, the MEP grows in size and the subsequent profound inhibition of ongoing EMG, known as the silent period, is prolonged (Taylor et al. 2000b). The silent period is usually attributed to an interruption of the corticomotor output and its duration is often used as an indicator of the level of inhibition in the motor cortex (Roick et al. 1993; Inghilleri et al. 1993; Di Lazzaro et al. 2002). According to Gandevia (2001) more weight must be put on changes in the cortical silent period. The MEP amplitude is indeed variably influenced by changes in the excitability of the spinal motoneurons (Morita et al. 2000) and at the muscle fiber membrane. To limit the latter influence, the changes in the MEP amplitude are usually normalized to the compound muscle action potentials (M-wave) measured during the same contraction (Nielsen et al. 1995). Recording of the (M-wave) is one of the common methods to assess the potential failure of peripheral factors involved in electrical propagation along the neuromuscular pathway.



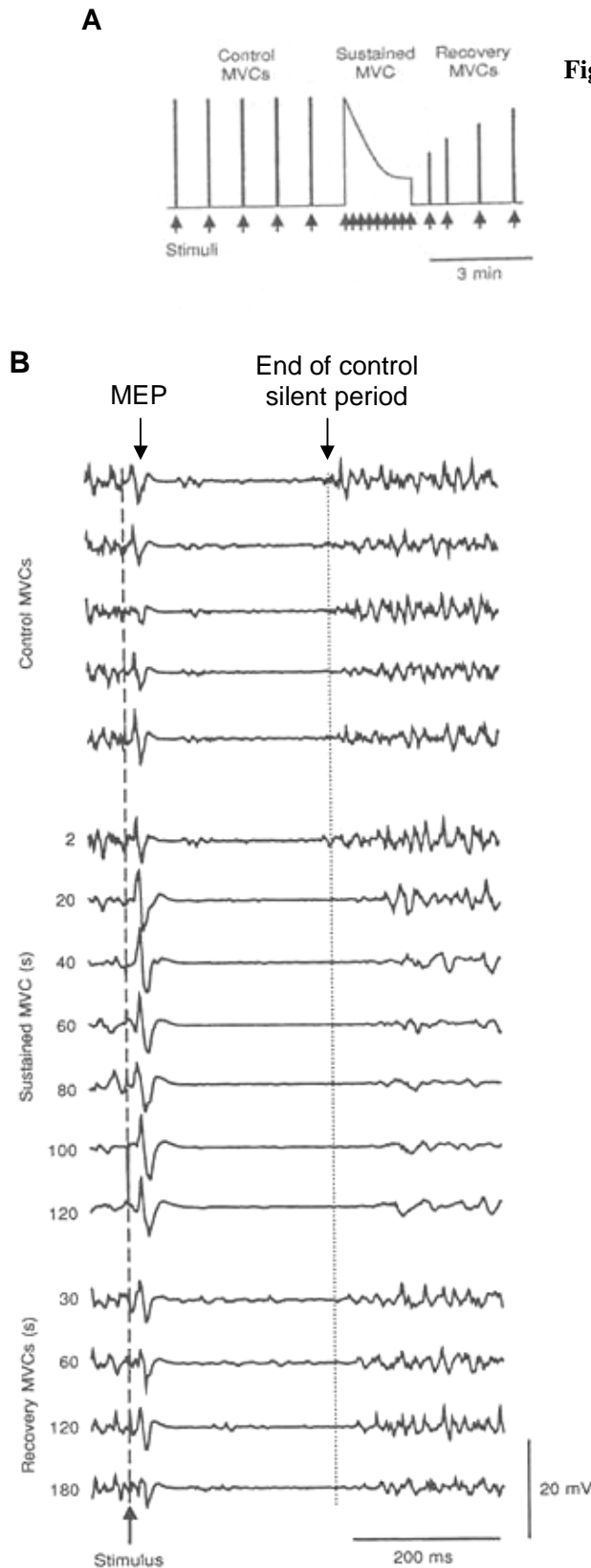
**Fig. 33. Schematic representations of torque measurements and EMG responses after TMS.**

Arrows on each trace point to the stimulus artefact.

(A) The torque trace shows the prestimulus level of ongoing maximal torque (a) and the increment (b) evoked by the stimulus.

(B) This EMG trace shows a motor evoked potential (MEP). The measured area of the MEP is shaded. Amplitude is measured peak to peak.

(C) EMG shows the MEP and subsequent silent period measured from the stimulus artefact to the return of EMG, as indicated by the arrow. (From Taylor et al. 2000a)



**Fig. 34. (A) Example of testing protocol.**

Subjects initially perform brief maximal voluntary contractions (control MVCs of 2-3 s). During each MVC a stimulus is given (arrows). Subjects then perform a sustained MVC (1.5-2 min durations). Stimuli are delivered every 10-15 s. Additional brief MVCs with superimposed stimuli are performed during the recovery period. (Adapted from Taylor et al. 1996)

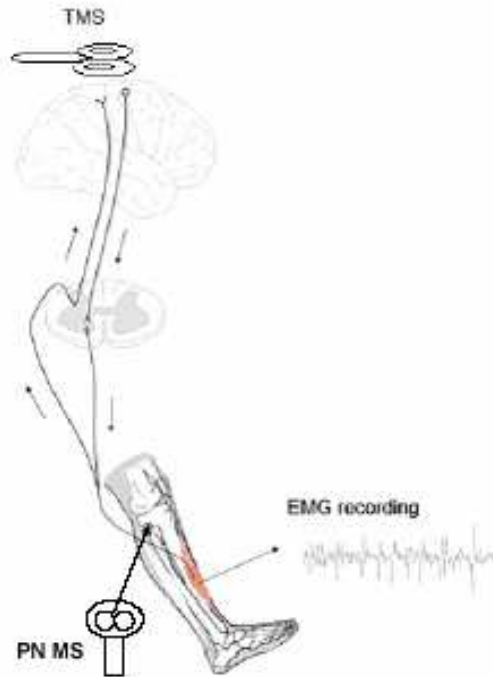
**(B) Individual EMG traces following TMS during MVCs.**

Upper five traces from the biceps brachii during the brief control MVCs, middle seven traces during the sustained 2 min MVC and lower four traces during recovery.

The TMS stimulus (left vertical line) is followed by a short latency excitatory response (MEP) followed by a period of small-amplitude variable EMG activity (silent period) before voluntary EMG resumes.

The silent period lengthens during the sustained MVC and quickly recovers. (Adapted from Taylor et al. 1996)

Additionally, Ross et al. ( 2007) completed the use of TMS over the motor complex by *peripheral magnetic stimulation* of the peroneal nerve (PN MS) (Fig. 35). This allows comparison of the motor evoked potentials (MEPs) evoked via TMS from those evoked by PN MS. The outcome of this study will be developed in the results sections 2.6.2.3. and 2.6.2.4.



**Fig. 35. Transcranial and peripheral magnetic stimulation.**

Electromyogram was recorded by surface electrodes over the tibialis anterior muscle. Transcranial magnetic stimulation (TMS) was delivered over the motor cortex and peripheral nerve magnetic stimulation (PN MS) was applied to the peripheral nerve. (Adapted from Ross et al. 2007).

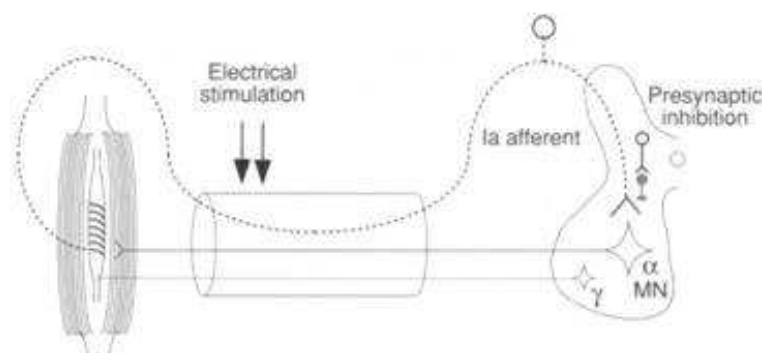
The post-exercise period may demonstrate different responses. While it is usually characterized by facilitation after non fatiguing tasks (Balbi et al. 2002; Samii et al. 1997), prolonged fatiguing tasks induce post-exercise depression (Sacco et al. 2000; Verin et al. 2004), although this is not constantly observed (McKay et al. 1995) and may depend on individual variations of the baseline cortical excitability (Zijdewind et al. 2000). Verin's et al. (2004) data revealed significant depression of both diaphragm and quadriceps MEP after incremental treadmill exercise, suggesting a role for central fatigue in task failure with the conventional Bruce protocol (Mead 1979) of progressive and maximal treadmill run. Hollge et al. (1997) are seemingly the only investigators to have studied the response to TMS of various muscles after different types of whole body exercise. They showed that predominantly anaerobic exercises (press-ups, 400-m runs) induced a marked post-exercise depression in MEP without changes in latency and without changes in peripheral responses to stimulation. Conversely, predominantly aerobic prolonged exercises, such as jogging, were not associated

with post-exercise changes in the MEP amplitude, and resulted in shortened MEP latency. These observations support the concept of central fatigue as a protective mechanism (Gandevia 2001) in intense short bouts of exercise-generating high-mechanical forces.

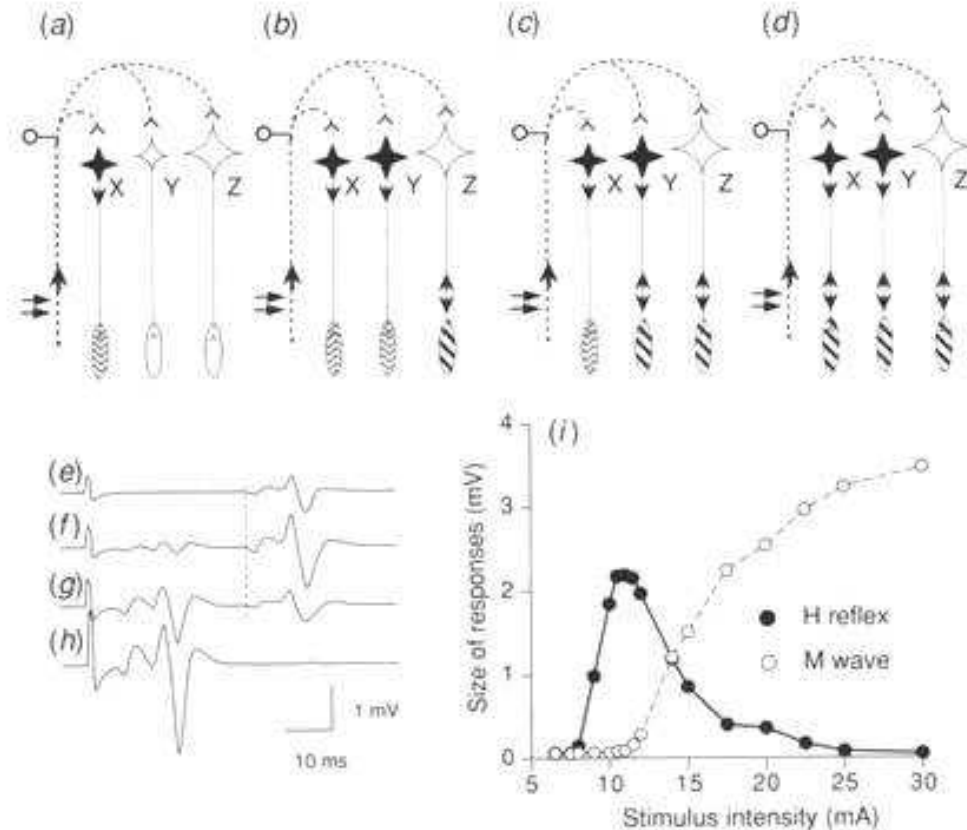
#### **2.4.4. Estimations of changes in motoneuron “excitability”**

As reviewed by Taylor and Gandevia (Taylor and Gandevia 2001), several techniques can estimate changes in motoneuron “excitability”: the *F-wave*, *H-reflex*, *transcranial electrical stimulation* and *cervicomedullary stimulation*. However, none of these techniques seems to be ideal. Each of them will be introduced shortly with special emphasis on their advantages and disadvantages (for complete information, see Gandevia 2001; Pierrot-Deseilligny and Burke 2005; Taylor et al. 2008; Taylor and Gandevia 2008).

Percutaneous electrical stimulation of the posterior tibial nerve produces a synchronized response in the soleus muscle (Hoffman 1919). This became known as the Hoffmann reflex or H-reflex, which tests the oligosynaptic reflex pathway from Ia afferents. As shown in Fig. 36, H-reflex is produced by electrical stimulation of Ia afferents, which have a lower electrical threshold than alpha motor axons. The electrically induced afferent volley bypasses muscle spindles and produces a single synchronous volley in group Ia and Ib afferents. Paillard was one of the pioneer scientists who developed this technique detailed in Fig. 37.



**Fig. 36. Sketch of the pathways of the monosynaptic reflex.** Ia afferents from muscle spindle primary endings have monosynaptic projections to alpha motoneurons (MNs) innervating the corresponding muscle (homonymous MNs). The H reflex is produced by electrical stimulation of Ia afferents, and bypasses muscle spindles. The pathway of presynaptic inhibition of Ia terminals is represented. (Modified from Pierrot-Deseilligny and Burke 2005).



**Fig. 37. Recruitment curve of the H and M-waves in the soleus.** Sample EMG responses ((e)-(h)) and sketches of the corresponding volleys of Ia afferents and motor axons ((a)-(d)) when the stimulus intensity is progressively increased. MNs discharging by the Ia volley are black, muscle fibers activated by the H reflex are speckled and those activated by the M-wave are hatched.

As the intensity of the electrical stimulus is increased, there is a progressive increase in size of H reflex due to stronger Ia afferent volley that cause both MNs “X and Y” to fire ((a)-(b) and (e)-(f)). It also elicits a motor volley in the axon of the largest motoneurons (e.g. MN “Z”) and a short-latency direct motor response (M-wave) appears in the EMG ((b) and (f)). Because the largest motoneurons have a high threshold for recruitment into the H reflex, the MN “Z” does not contribute to the reflex. Therefore the antidromic volley in MN “Z” (upper arrow) does not collide with and interfere with the H reflex response.

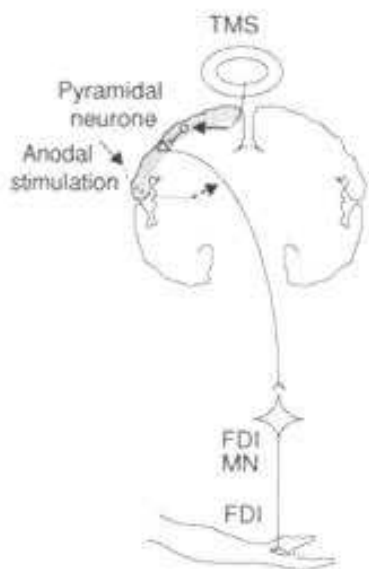
Further increases in the intensity of the test stimulus ((c) and (g)) elicit a motor volley in the axons of MNs “Z” and “Y”. This causes the M-wave to increase, but the H-reflex to decrease due to the antidromic volley in the axon of MN “Y”.

Finally, stronger stimulation  $M_{max}$  ((d) and (h)), but the H-reflex is completely suppressed.

The vertical dashed line in (e) and (g) indicates the latency of the H-reflex and (i) its amplitude. (Adapted from Pierrot-Deseilligny and Burke 2005)

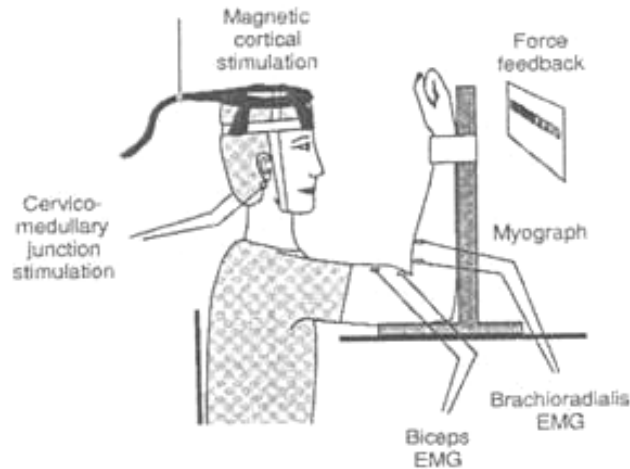
The impetus for *transcranial electrical stimulation* came from the studies of Merton and Morton (Merton and Morton 1980). They used a single high voltage transcranial capacitive discharge and showed that stimulation over the motor cortex could produce a twitch of contralateral limb muscles (Fig. 38). The shorter response latency to the electrical stimulus as compared to the magnetic one suggests that the electrical stimulus excites the

corticospinal axons directly (Day et al. 1987), but can also be affected by changes in cortical excitability. The major problem of this technique is that only a small fraction of the current flows into the brain. Much of the current flows between the electrodes on the scalp and produces strong discomfort and local pain (Pierrot-Deseilligny and Burke 2005).



**Fig. 38. Sketch of the presumed pathways involved in the electrical and in the magnetic transcranial stimulations.** A pyramidal neurone projecting to a first dorsal interosseus motoneuron (FDI MN) is activated at the level of its axon (dashed arrow) by anodal electrical stimulation, transynaptically (continuous arrow) by transcranial magnetic stimulation (TMS). (Adapted from Rothwell 1997).

According to Taylor and Gandevia (2001), the *corticospinal tract stimulation* would be the best test of the motoneurons responsiveness. Similarly to the transcranial electrical stimulation, however, its use is limited by the associated discomfort (Ugawa et al. 1991). As illustrated in Fig. 39, stimuli are delivered to the cervicomedullary junction through electrodes placed over the mastoids. This stimulation evokes large, short-latency responses (cervicomedullary motor evoked potentials: CMEPs) (Ugawa et al. 1991; Taylor and Gandevia 2004), characteristics of which are changing with fatigue (Taylor et al. 1996; Butler et al. 2003; Martin et al. 2006;). As it has a large monosynaptic component and is free in humans from traditional presynaptic inhibition, the size of the CMEP reflects motoneurons excitability (Ugawa et al. 1991; Nielsen and Petersen 1994; Petersen et al. 2002). The CMEP responses to cervicomedullary stimulation are also normalized to M-max (Taylor et al. 1999).



**Fig. 39. Experimental arrangement for magnetic cortical and corticospinal tract stimulations.** Subject seated with the right arm on a myograph with force feedback provided and EMGs from arm muscles recorded. Stimuli are delivered to the cortex via a magnetic coil and to the cervicomedullary junction through electrodes over the mastoids. In this experiment the two types of stimuli were delivered alternatively every 10-15 s during a sustained MVC. (Modified from Taylor et al. 1996)

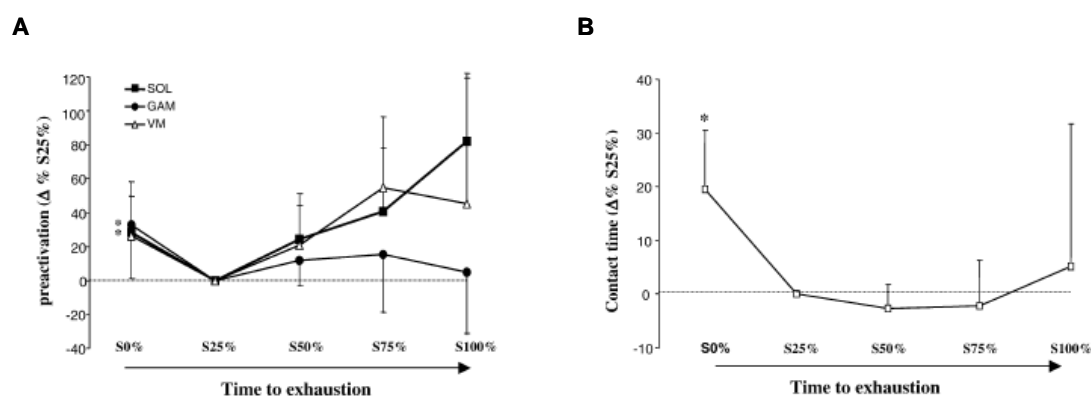
## 2.5. Fatigue during the course of the exercise

The critical feature consists in revealing the progressive development of fatigue during the time course of the SSC task. This fatiguing progress is indeed clearly individual and exercise dependent, both in its timing and amplitude. Even though the development of contractile failure can be reflected by progressive kinetic, kinematic and electromyographic (EMG) changes, it is noteworthy that marathon running kinematics and running economy are not interrelated when fatigue progresses (Nicol et al. 1991a; Williams et al. 1987; Kyröläinen et al. 2000).

As suggested by Place et al. (2004), part of the changes in the SSC pattern might reflect adjustments to fatigue rather than any real failure to compensate for it. Supporting this hypothesis, when subjects performed a fatiguing series of 100 drop jumps from a 0.4 m dropping height (Skurvydas et al. 2002), intermediate maximal jumping tests (squat jump, counter-movement jump, drop jump from 0.9 and 1.35 m) performed after 50 drop jumps were 5 to 10 % lower than the pre-fatigue performances. No further reductions were observed when the exercise continued until 100 jumps. Importantly, this observation applies to untrained, sprinters and elite long-distance runners.

### 2.5.1. Follow up of the changes affecting the exercise task

Since the SSC technique is a complex motor task, it is frequently improving by learning along the repetitions. Based on our own studies (Ftaïti et al. 2001; Regueme et al. 2005c) it is suggested that special care should be taken with the so-called “pre-fatigue” SSC pattern. During an exhaustive treadmill run, the running pattern was found to get stabilized in most of the recorded muscles after the first 10 minutes (Ftaïti et al. 2001). Similarly the middle and lower graphs of Fig. 40 show, in an intermittent rebound exercise on the sledge, 20% longer contact time and 30% higher preactivation of lower limb extensor muscles during the first rebound series (S0%) as compared to the third or fourth ones (S25%) (Regueme et al. 2005c). The S25% series was therefore chosen instead of S0% as the “PRE-fatigue reference”. Preactivation was indeed at its lowest level at S25% before increasing again in all recorded muscles and the contact time remained then stable for a few series before rising again towards the end of the exhaustive exercise.



**Fig. 40. Relative group-average changes in the submaximal rebound characteristics along an exhaustive intermittent SSC exercise.** For each subject, the 5 selected rebound series correspond to the first one (S0%) and to 4 others corresponding to every 25% of the exercise duration (S25%, S50%, S75% and S100%).

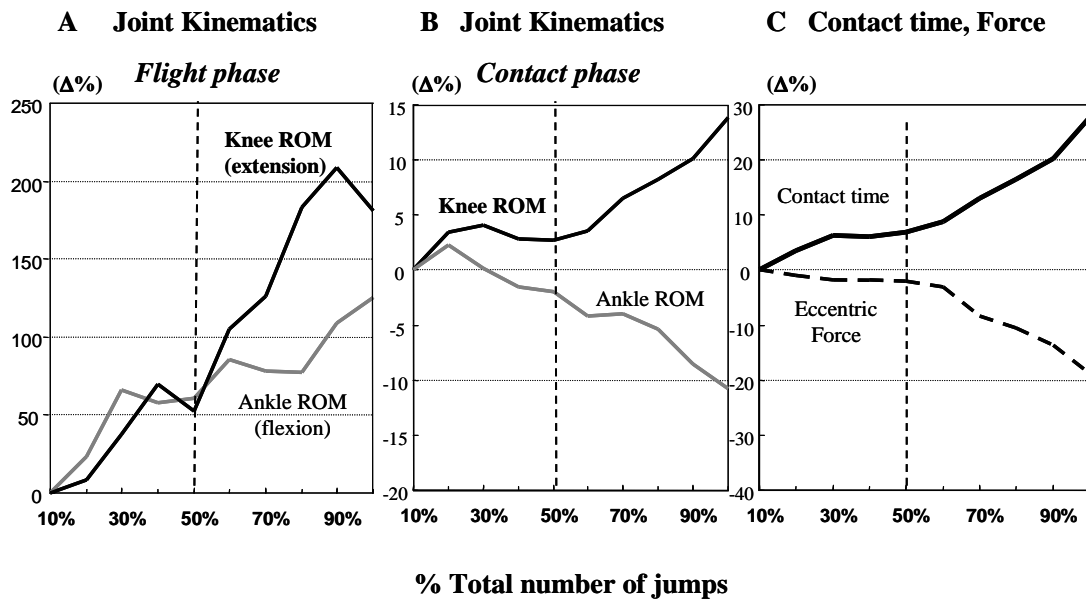
**(A) Preactivation** in the soleus (SOL), gastrocnemius medialis (GAM) and vastus medialis (VM) muscles.

**(B) Contact time on the force plate.** \* P < 0.05 from the reference S25%.

(From Regueme et al. 2005)

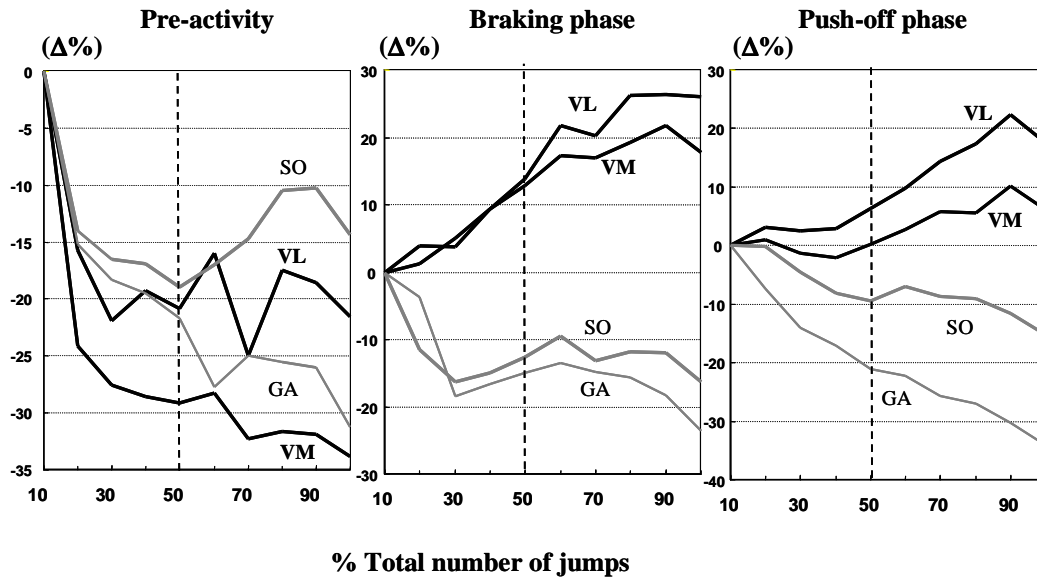
During the time course of the exercise, different compensatory neural strategies may then emerge within the fatigued joint as well as across joints in order to maintain the same global power output as long as possible (Bonnard et al. 1994). Horita's finding (2000) of a clear turning point in the neural adjustments to fatigue after the middle stage of an exhaustive leg rebound exercise is in agreement with this thought. The first half of the exercise presented rather limited kinetic and kinematic changes during the contact phase (Fig. 41B and 41C), but large kinematic alterations during the flight phase (Fig. 41A).



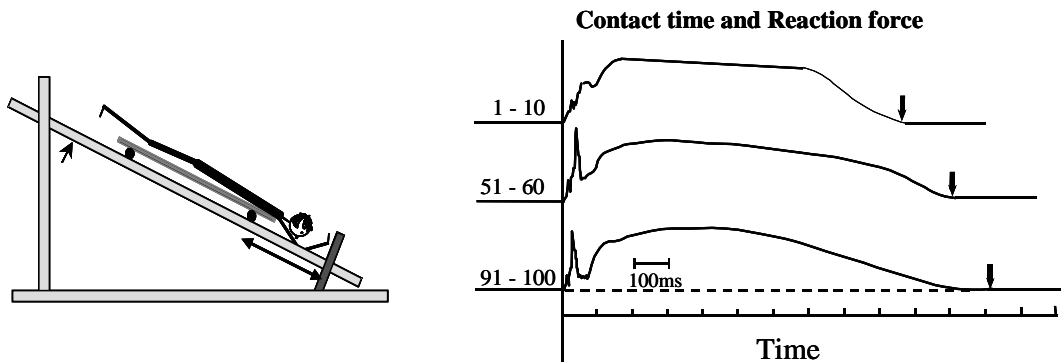


**Fig. 41. Relative fatigue-induced changes in kinematics (A & B) and in kinetics (C) during the time course of SSC rebound exercise with the lower limbs. (Data from Horita 2000)**

This was accompanied by clear EMG adjustments during the preactivation phase with a linearly increased activation of the knee extensors in the braking phase (Fig. 42). The associated stable values of range of knee joint motion (ROM) and contact time demonstrated the effectiveness of this adaptation to counteract the loss of force of the knee extensors (Fig. 41B & 41C). It was concluded that the braking phase of the SSC cycle operated effectively until the middle stage of the exercise. In the second half of the rebound series, however, the pre-landing joint kinematics changed dramatically (Fig. 41A) and influenced significantly the subsequent post-landing stiffness regulation (Fig. 41B) and contact time (Fig. 41C). The deterioration of the SSC efficacy during the braking phase is further confirmed by the associated increases of the extensor EMG activity and EMG/force ratio during the subsequent push-off phase. This increased work allowed the submaximal jumping task to be continued. Finally, the observations of opposite trends of changes in the knee and ankle range (Fig. 41B) of motion suggest attempts of compensation among the different segments that may not remain sufficient, however, as indicated by the dramatic increase of the contact time towards the end of the exercise. In such a submaximal rebound height, the increased contact time can indeed compensate effectively for the reduced force output, and thus contribute to the maintained external work output. This is in line with the progressive increase (up to 30%) in contact time reported by Gollhofer et al. (1987b) (Fig. 43) at the end of short and intensive SSC rebound exercises performed with the upper-limbs.



**Fig. 42. Relative group averaged EMG changes along exhaustive SSC rebound exercise.** EMG activity of (VL) vastus lateralis, vastus medialis (VM), gastrocnemius (GA) and soleus (SO) during a submaximal SSC rebound exercise with the lower limbs. (Data from Horita 2000)

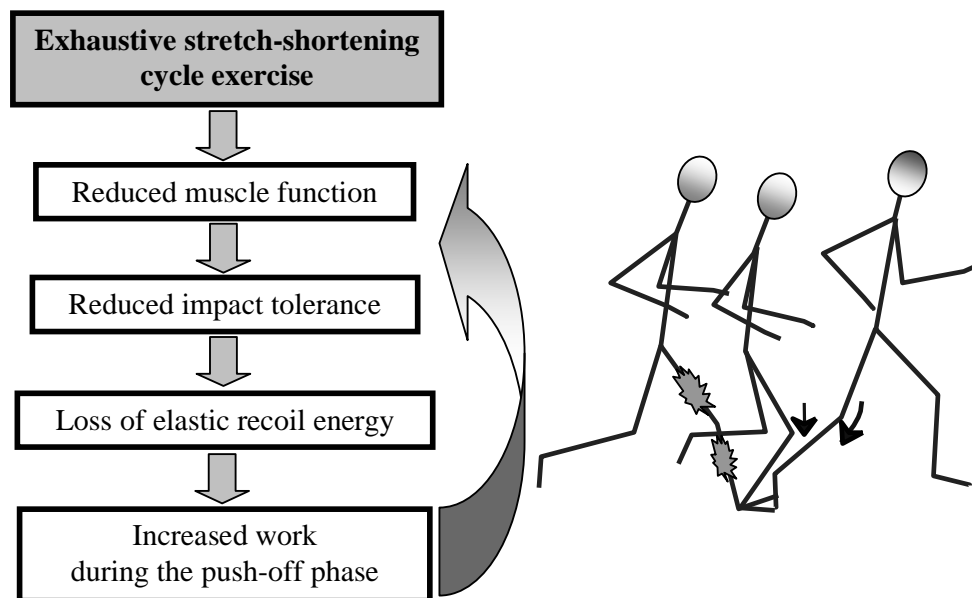


**Fig. 43. Relative changes in kinetics (contact time and reaction force) during the time course of 100 submaximal rebounds with the upper limbs.** (Data from Gollhofer et al. 1987b)

The marathon run model has also shown similar increases in the ground contact duration (Komi et al. 1986). The fatigue state is characterized by a drop in the force after the impact that is likely to be related to the observed faster and longer flexion movement (Nicol et al. 1991b; Horita et al. 1996). In case of the arm exercises the dramatic increase in the impact peak (Fig. 43) resulted, most likely, from increased preactivation of the arm extensor muscles (Gollhofer et al. 1987a, 1987b). The increase in preactivation is, indeed, usually associated with more extended limbs and increased muscle-tendon stiffness prior to impact. The subsequent drop in force after impact is nowadays considered as the important indicator of reduction in tolerance to repeated stretch loads as fatigue progresses (Nicol et al. 2006). A

logical consequence of this is that in order to maintain the same SSC performance, for example at a constant marathon speed or a constant submaximal rebound height, the subject must perform greater work during the push off phases leading to even a faster progression of fatigue. Depending upon exercise intensity, this represents a vicious circle leading to a progressive reduction of the capacity to maintain the task. Fig. 44 represents in more practical terms the sequence of events expected to take place during fatiguing SSC exercise.

These overall findings emphasize the pertinence of the SSC model to study the neuromuscular adjustments to the contractile failure and to reveal potential neural limitations. It is suggested that in the non fatigued state the muscles are able to damp the impact in SSC by a smooth force increase and smooth joint motion. During submaximal SSC exercises, the exhaustion time is delayed by the intervention of different neural strategies that take place both during the pre- and the post-impact phases.



**Fig. 44. Schematic representation of the sequence of events expected to take place during fatiguing SSC exercise.** (Modified from Nicol and Komi 2000)

### *2.5.2. Intermediate maximal and submaximal testing tasks*

As previously mentioned in the section 2.1., the common protocol used to quantify the development of muscle fatigue is to interrupt the on-going exercise with brief maximal contractions (voluntary or electrically evoked) to estimate the decline in maximal force

capacity and voluntary activation (Merton 1954). It is only recently, however, that SSC fatigue studies used such tests during the time course of the exercise.

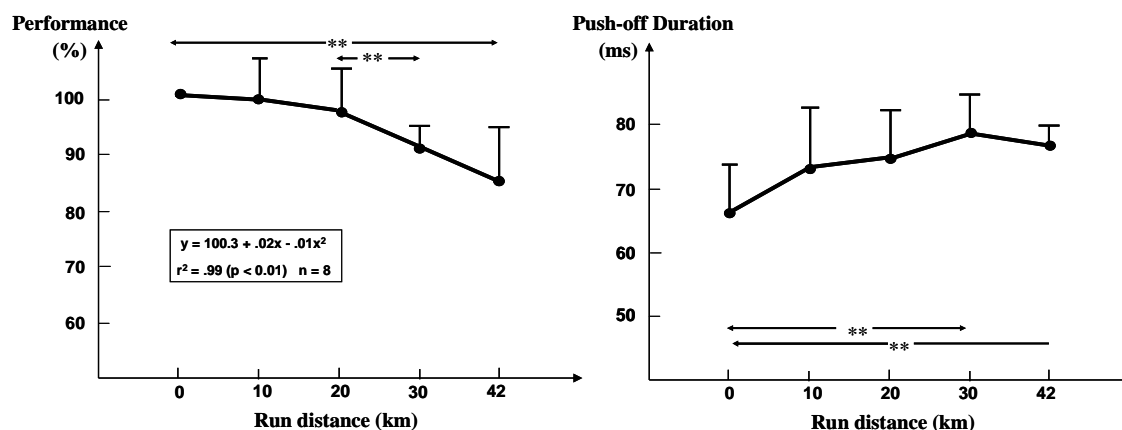
Place et al. (2004) introduced brief tests every hour during a 5h running exercise performed at moderate velocity. Their testing protocol examined the changes in voluntary and electrically induced contractions of the quadriceps muscle group. Their findings suggested that maximal voluntary force capability of the knee extensor muscles was depressed in the final stages of a 5-h running exercise, and clear evidence existed for central activation failure as well as for alterations in muscle action potential transmission.

Subsequently, Millet and Lepers (2004) compared these results with those obtained during a cycling exercise (Lepers et al. 2002). This comparison revealed a similar time course of MVC reduction throughout the first 3 hours, but differences occurred after the 4th hour where MVC declined by 14 % in running and by only 6 % in cycling. As expected, the 5h running exercise ended up with a greater (-28 %) loss of maximal knee extension strength capacity than the 5h cycling exercise (-18 %).

To our knowledge, only Girard et al. (2007) examined the time course of impairment in neural and contractile processes every 30 min during a prolonged (3h) high intensity intermittent exercise (prolonged tennis playing). Voluntary activation was assessed through 3 methods: the normalized EMG response (RMS/M-wave), the superimposed twitch on MVC and the 80 Hz tetani. The latter technique was applied only before and after the tennis match and to those subjects who could tolerate it. Their results revealed progressive but moderate reductions in the maximal voluntary knee extension torque (-9%). Central activation failure (decreased RMS/M) and alterations in excitation–contraction coupling (decreased P20/P80) are suggested as the main mechanisms contributing to the observed loss in torque.

The progressive development of fatigue may also be investigated by repeating submaximal and/or maximal SSC tests during the time course of the exhaustive exercise. For example, our initial marathon protocol (Nicol et al. 1991a, b, c) included submaximal and maximal running tests performed on the track every 10 km of the marathon run. Kinetic analysis of the track tests did not reveal any change in the submaximal run performed at the individually fixed initial marathon running velocity (Nicol et al. 1991a). In contrast, the maximal running test revealed a parabolic decrease of the sprinting velocity after the first 20 km (Fig. 45, left panel) with associated decrement in the resistance to the impact load and subsequent increased work during the push-off phase (Fig. 45, right panel) (Nicol et al. 1991a). The tests of higher loading level (sprint run) may thus reveal a more homogeneous

deterioration of the muscle function than the submaximal ones, but they should be complemented by voluntary and electrically-induced maximal contractions.



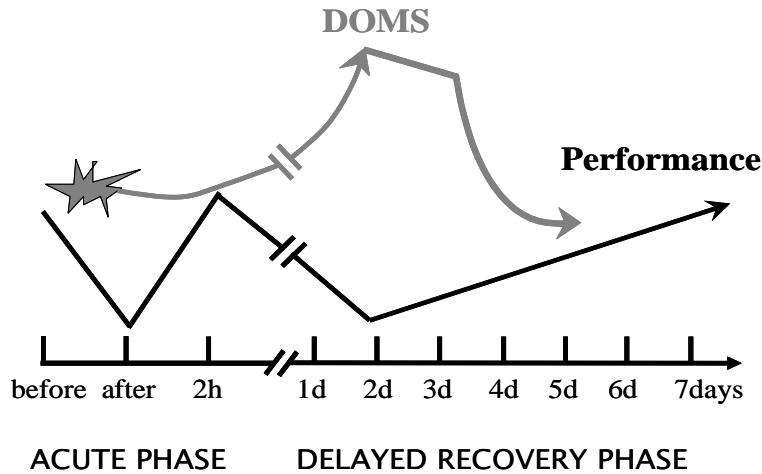
**Fig. 45. Progressive changes in a sprint performance performed every 10 km during the course of a marathon run.**

**Left panel:** Relative change (mean + SD) of the maximal sprint velocity (100 % = before marathon value). **Right panel:** Duration of the push-off phase of the sprint runs along the marathon. (From Nicol et al. 1991c)

## 2.6. Bimodal response of neuro-mechanical parameters?

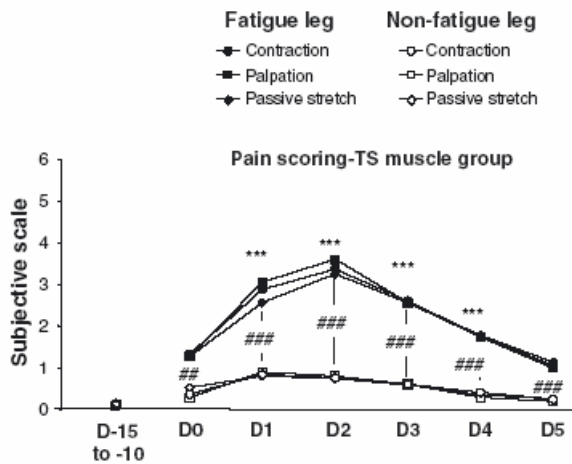
### 2.6.1. Basic pattern of bimodality

Intensive and/or unaccustomed SSC exercises induce various impairments of the neuromuscular function that are usually bimodal in nature (Fig. 46, 48, 59). This pattern is quite similar to the *bimodal recovery concept* of Faulkner et al. (1993) after eccentric type exercise. This bimodality is first characterised by large acute changes in muscle mechanics and neuromuscular activation that may significantly influence the regulation of joint and muscle stiffness. This is followed after a few hours by a short-term “acute” recovery, which is in turn followed by a “delayed” functional reduction with a slow recovery. This bimodality concept is not always valid, especially if the SSC fatigue has not been exhaustive enough. The delayed recovery phase is typically associated with delayed-onset muscle soreness (D.O.M.S.) sensation (Fig. 46, 47).



**Fig. 46. Schematic representation of the general trend of changes in performance and delayed onset muscle soreness (D.O.M.S.) sensation after exhaustive SSC exercise.**

In brief, the D.O.M.S. sensation is characterised by a sensation of dull pain and discomfort, increasing in intensity during the first 2 days, remaining symptomatic for 1-2 days and usually disappearing 5-7 days after exercise (Fig. 46, 47). Soreness is not constant, being mostly felt when the exercised limbs are extended or fully flexed, or when the muscles are palpated deeply (Howell et al. 1993). Sore muscles are often stiff and tender, and their ability to produce force is reduced for several days or weeks (Asmussen 1956; Komi and Rusko 1974; Sherman et al. 1984; Nicol et al. 1996a, 2003; Murayama et al. 2000). Particularly important in terms of injury prevention is the timing of DOMS disappearance, as it occurs prior to complete structural and functional recoveries (Fig. 47). It cannot therefore be used to reflect detailed recovery processes. This will be further discussed in sections 2.7.1.1. and 2.8.2.



**Fig. 47. Pain scoring Group-averaged values (-SD) of subjective pain [on a six-level scale – from no pain (0) to extremely sore muscle (6)] of the triceps surae (TS) muscle group.** There were three testing conditions (palpation, contraction, and passive stretch). \*\*\*  $P < 0.001$ , significantly different from the prefatigue (PRE) absolute values; ##  $P < 0.01$ , ###  $P < 0.001$ , significant inter-leg difference. (From (Regueme et al. 2007b))

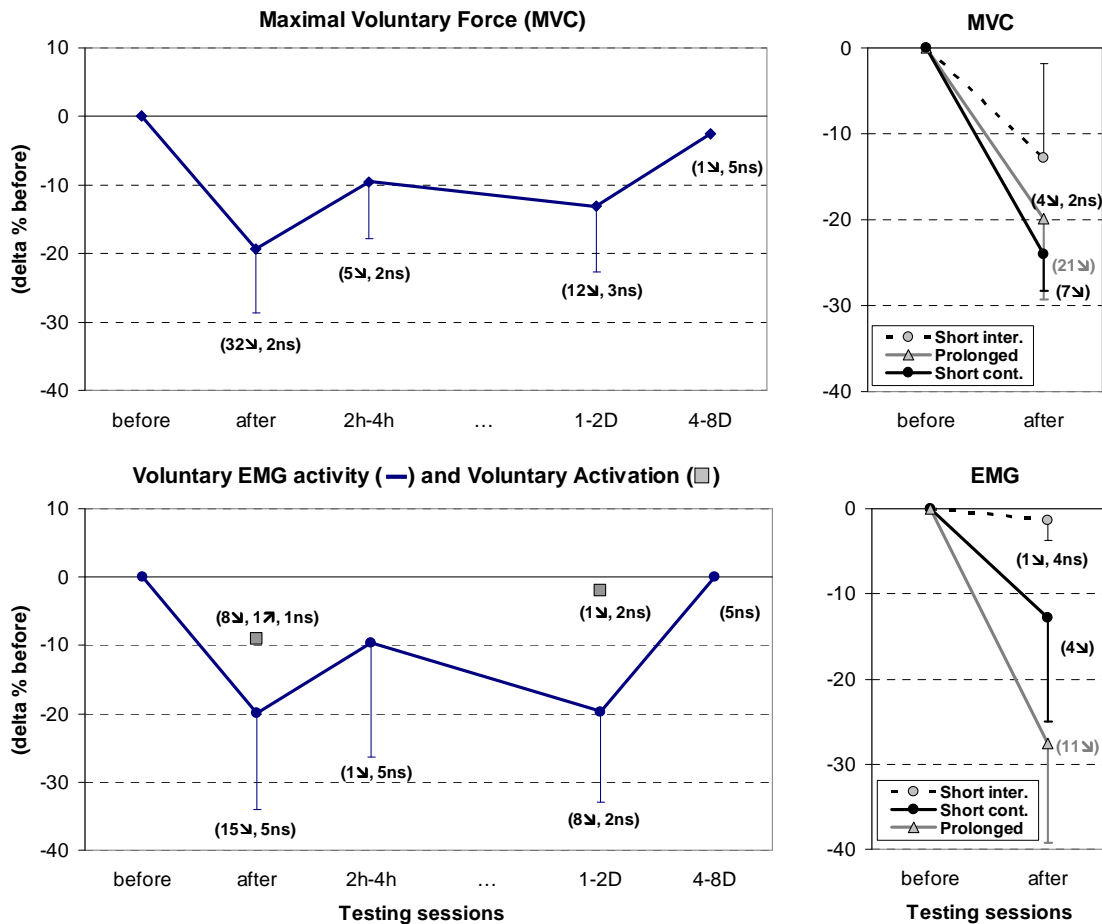
Based on our recent (Nicol et al. 2006) and present literature reviews of SSC fatigue studies, it appears that approximately 60 % (31/55) of the studies concentrated on the acute recovery phase (< 2 hours post-exercise), whereas the 24 other studies extended their investigation to the delayed recovery phase (up to 8 days). Fig. 48 shows their combined results in terms of both acute and delayed changes in maximal voluntary force (MVC), voluntary EMG activity and voluntary activation (by twitch interpolation technique).

### ***2.6.2. “Time-dependent” effects: a parallelism between neural and mechanical changes***

The concept of “time-dependent” effect is based on the general observation of a bimodal recovery pattern along the several days of the recovery period (Fig. 46). The overall results are in agreement with Asmussen (1979), who was among the first ones to suggest that fatigue-induced changes occur in parallel between mechanical and neural factors. In SSC fatigue situations, there is enough evidence to consider this interaction as neural mechanisms that would compensate for contractile failure or protect fatigued muscles (Nicol et al. 2006).

#### ***2.6.2.1. Maximal voluntary electromyographic activity and force production***

SSC-induced fatigue has mostly been quantified by using isometric MVC tests, with simultaneous recordings of surface electromyographic activity (EMG). Most of the SSC fatigue studies (34/36) revealed an immediate ( $21 \pm 8$  %) drop in maximal voluntary isometric contraction (MVC). The mean value presented in Fig. 48 includes the 2 non-significant considered as null values. When re-examined 2h post-exercise a partial or complete recovery of MVC is frequently observed ( $-10 \pm 8$  %). From these 36 SSC fatigue studies, 15/20 reported an immediate drop in both force and voluntary EMG activity and 8/10 secondary reductions on Day 2. On the average, a parallelism is observed between activation and force changes along the 8 days of the recovery period. Please note, however, that the fatiguing protocol was for 9 of these 10 studies of short and intensive SSC type, consisting of exhaustive rebounds on the sledge or downhill treadmill run. It is unfortunate that only (Avela et al. 1999b) examined the two factors during the delayed recovery period of prolonged (marathon) SSC exercise. Thus, one may conclude that the acute drop in MVC is quite independent of the fatiguing protocol duration (prolonged *versus* short and intensive that may vary from 3 min to a few hours).



**Fig. 48. Relative acute and delayed decrements in maximal voluntary isometric force (MVC) and electromyographic (EMG) activity (RMS or IEMG) following SSC exercises.**

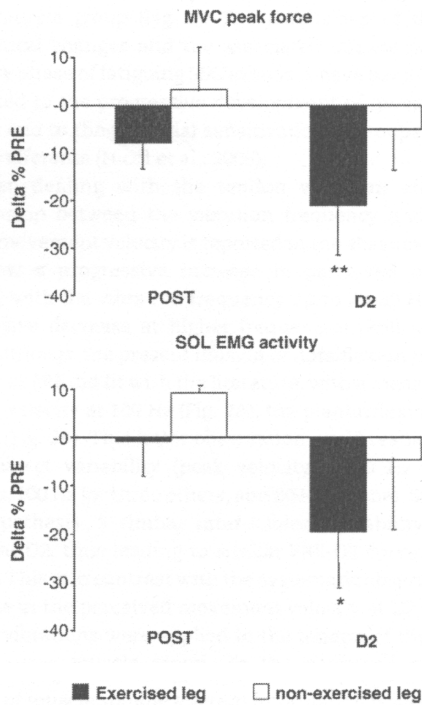
**Upper panels:** MVC data compiled from 31 published articles using either knee extensor or plantarflexor muscles and reporting either decreased ( $\nabla$ ) or non significant (ns) changes. The upper right panel differentiates the immediate post-exercise (after) changes of the three major types of SSC protocols: either prolonged (running and skiing exercises, N = 21 studies) or short and intensive ones (mostly rebound exercises, N = 13 studies), the latter condition being subdivided into its continuous and intermittent modes. For the mean value calculation, the non significant (ns) values have been replaced by the value zero.

**Lower panel:** corresponding changes reported by some of these studies in voluntary electromyographic activity measured by surface EMG electrodes and in electrically evoked voluntary activation.

On the other hand, the use of intermittent fatiguing SSC protocols (Regueme et al. 2005a, 2005b, 2007a, 2007b, 2008) emphasizes the neural influence on the drop in force by showing systematically small immediate drops in MVC associated with non significant changes in voluntary EMG activity (Fig. 48 right panel), followed on day 2 by large secondary declines in both of these parameters. This is illustrated in Fig. 49 for one of these studies. Furthermore, this particularity of the short intermittent SSC exercise is in agreement with the relatively limited 9% drop in MVC reported by Girard et al. (2007) after a prolonged

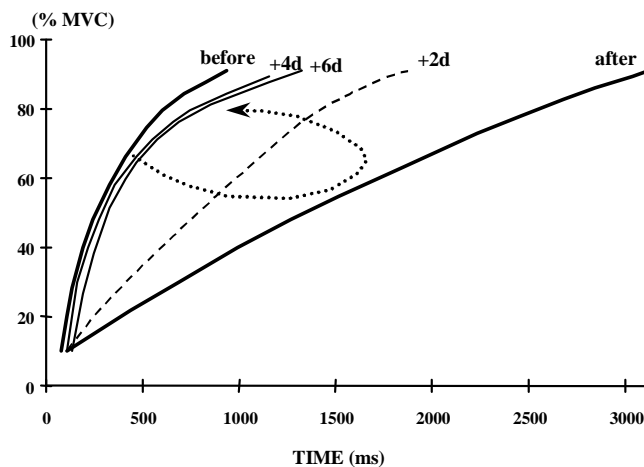


(3h), but intermittent tennis match. This study emphasizes also the necessity to consider the results of a given protocol in the light of the expertise level of the subjects. Consequently, it is suggested that one should differentiate the continuous mode of SSC fatiguing protocols from the intermittent one.



**Fig. 49.** Group averaged relative changes (mean  $\pm$  SD) of maximal voluntary contraction (MVC) and soleus (SOL) electromyographic (EMG) activity immediately after exhaustive intermittent rebound exercise (POST) and 2 days later (D2). The means are expressed in percentage of the pre-fatigue (PRE) absolute values for the exercised (■) and non-exercised (□) leg. \*  $P < 0.05$ , \*\*  $P < 0.01$ : significantly different from the PRE values. (Modified from (Regueme et al. 2007b))

The rate of force development is also reported to be dramatically reduced and accompanied by a slow recovery (Pullinen et al. 1999) (Fig. 50). In accordance again with the concept of parallel neural and mechanical changes with SSC fatigue, recovery of both maximal EMG and rate of force development remained incomplete until the 6th day post-exercise (Pullinen et al. 1999).



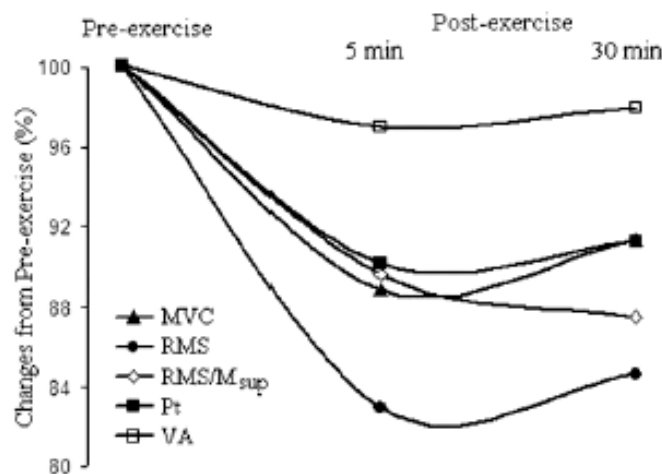
**Fig. 50:** Isometric force-time curves of the maximal knee extension measured immediately before and after a marathon run as well as 2, 4 and 6 days later. Note the large and immediate decrease in the rate of force development and its slow recovery towards the 4th day post-marathon. (Data from Pullinen et al. 1999)

#### 2.6.2.2. *Maximal voluntary EMG normalized to maximal compound action potential amplitude*

Although the voluntary EMG data demonstrate persisting reductions for a few days after exhaustive SSC exercises, caution should be exercised when interpreting surface EMG changes during muscle fatigue. These changes do not only reflect the number and firing rates of the recruited motor units but can also be influenced by changes in the size of the surface-detected motor unit action potentials (Chan et al. 1998). Therefore, it cannot be concluded from the sole root mean square (RMS) or integrated EMG data that the measured decreases result entirely from the occurrence of central fatigue. In agreement with the early methodological comment of Asmussen (1979) and with the more recent review of Gandevia (2001), impairments in neuromuscular transmission should also be considered. To take into account this possibility, one of the common techniques consists in using the *maximal compound action potential amplitude* “*maximal M-wave*” as an index of neuromuscular transmission and action potential propagation in muscle fibers.

However, even though the ratio of the voluntary EMG activity (root mean square – RMS- or integrated EMG) divided by the M-wave has been commonly used in the literature; it has been introduced only recently in the SSC testing protocols (Strojnik and Komi 2000; Millet et al. 2002, 2003a, 2003b; Kuitunen et al. 2004; Place et al. 2004; Dousset et al. 2007; Girard et al. 2007; Racinais et al. 2007, 2008). This constitutes one of the major limitations of our own measurements mentioned in Tables 1 and 2. Immediately after a ski skating marathon (Millet et al. 2003b), RMS was thus found to be decreased but RMS/M was unchanged. This is similar to the influence of prolonged cycling exercise involving mostly concentric muscle actions (Lepers et al. 2000). Minimizing, however, the role of a reduced M-wave in the acute recovery phase, both RMS and RMS/M parameters were significantly reduced immediately after prolonged running (Millet et al. 2003a; Place et al. 2004; Racinais et al. 2007) (Fig. 51). Furthermore, the extended follow-up of the recovery period (over 6-8 days) after a marathon run (Avela et al. 1999b) as well as after our common exhaustive rebound protocol on the sledge (Kuitunen et al. 2004; Dousset et al. 2007) revealed very limited and non significant changes in maximal M-wave. Bimodal decrements were observed, however, in maximal RMS (Kuitunen et al. 2004; Dousset et al. 2007), in voluntary activation level (Dousset et al. 2007) as well in both stretch reflex and H reflex amplitudes (Avela et al. 1999b). Finally, it is noteworthy that the reported changes in the M-wave amplitude are quite variable among SSC studies and between subjects ( $-29 \pm 24$  % after a 30 km running race *versus* a non significant  $1 \pm 36$  % after a 65 km ultramarathon and a  $-9 \pm 18$  % after a ski-skating marathon). From

these observations it can only be concluded that the M-wave characteristics are not always the major cause of the acute drop in maximal voluntary EMG activity after prolonged SSC exercises.



**Fig. 51:** Changes in plantar flexors maximal isometric voluntary contraction (MVC), peak twitch torque (Pt), EMG activity of the soleus muscle recorded during the MVC expressed in absolute units (RMS) and normalized by the M-wave amplitude (RMS/M<sub>sup</sub>), and voluntary activation (VA) level estimated by the twitch interpolated method. Data are expressed as percentage from pre-exercise (90 min run) values. All the data displayed a significant effect of the exercise ( $P < 0.05$ ) and without difference between the data recorded 5 or 30 min after the exercise. (Modified from Racinais et al. 2007)

### 2.6.2.3. Voluntary activation

In the SSC literature, it is only recently that some studies (including only one of my own, Strojnik et al. 2001) intended to assess the “central” versus the “peripheral” origins of the decreases in voluntary EMG activity and force production through the estimation of “Voluntary Activation” (Douset et al. 2007; Kuitunen et al. 2004; Martin et al. 2004a; Millet et al. 2002, 2003a, 2003b; Place et al. 2004; Racinais et al. 2007; Ross et al. 2007). Their combined results are presented in Fig. 48.

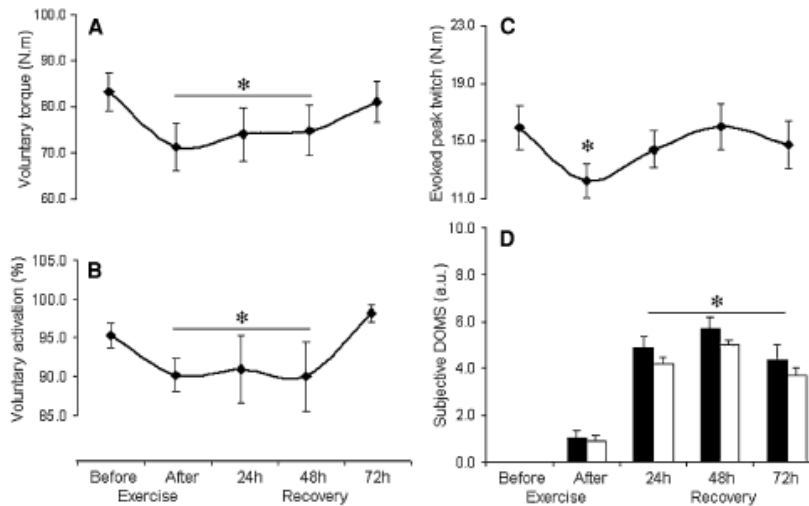
*In the acute SSC recovery phase*, the use of the twitch interpolation technique has revealed non significant change after continuous and intermittent 90 min runs (Racinais et al. 2007), but an activation deficit was reported after a 30-km race (2h30min) and three times larger one (-23 versus -8%) after a 65-km (6 to 8h) one (Millet et al. 2002, 2003a). Similarly, intermediate 16 and 19% deficits in voluntary activation are reported for 2h treadmill run durations (Place et al. 2004; Saldanha et al. 2008). Supporting our introductory comments on the running versus X-C skiing comparison, no deficit in voluntary activation was reported after a free style ski-skating marathon despite of a similar drop in force than after a 30-km run of same duration (Millet et al. 2003a). Voluntary activation may also be estimated by

comparing the forces achieved with voluntary- (MVC) and electrically-evoked (80Hz) contractions (Duchateau and Enoka 2002). To our knowledge, only a few SSC fatigue studies have used this method (Millet et al. 2003a; Martin et al. 2004b). In these studies, fatigue was induced either by a prolonged 30 km running race or by a 30-min downhill treadmill run. In agreement with the findings obtained with the twitch interpolation technique, a significant acute decrease of the MVC/80Hz ratio was found after the 30-km run (Millet et al. 2003a). In the other study, both of these parameters decreased on the average by 10%, but the MVC/80Hz ratio was not reported. On the other hand, their parallel investigation of “low frequency fatigue” (LFF), i.e. the preferential loss of force at low frequencies of electrical stimulation (Jones DA 1996), revealed relative decrements in low-to-high frequency (20/80Hz) ratios. Peripheral fatigue was therefore suggested to explain mostly, but not totally, the drop in force.

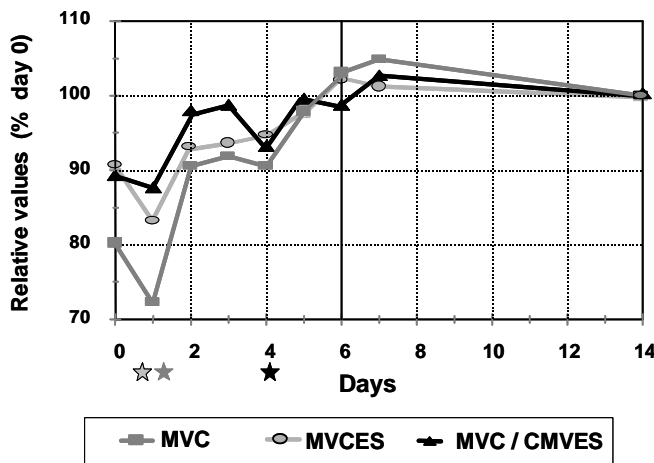
*For the delayed recovery phase*, the lack of estimation of voluntary activation is more critical as only four SSC fatigue studies are reported (Kuitunen et al. 2004; Martin et al. 2004a; Dousset et al. 2007; Ross et al. 2007). At the present stage, only the rebound study (Dousset et al. 2007) reported a significant (6%) 2 day drop in voluntary activation whereas no change was found in the other reports (Fig. 48). Although being closer from a pure eccentric exercise than from an SSC one, the treadmill backward walking study of Racinais et al. (2008) is of particular interest. As shown in Fig. 52, acute and 2 days delayed decrements were found, both in maximal voluntary torque (MVT) and voluntary muscle activation (VA) of the plantar flexor muscles, whereas the measure of the (peripheral) contractile properties (electrically evoked twitch) had recovered significantly within the first 24 h post-exercise. As shown in this figure, the delayed recovery period was associated with DOMS sensation. This was attributed to the occurrence of a supraspinal modulation of muscle activation during this period when muscle contractile properties had fully recovered following the exercise. Based on the persistence of the decrement for several days it was suggested that the delayed modulation would be related to the concurrent presence of muscle soreness. This possibility will be further developed in section 2.8.2.2.

Our own study examined the effects of a leisure alpine skiing week on maximal voluntary contractions performed without (MVC) and with additional electrostimulation (MVCES) (Strojnik et al. 2001). As shown in Fig. 53, both MVC and MVCES forces declined after the first skiing day, but on the 4<sup>th</sup> day only MVC/MVCES was significantly reduced, thus suggesting a central adjustment to the contractile fatigue. Interestingly, the subsequent day (day 5) was then characterized by a significant self reduction by the skiers of

their freely chosen skiing distance. Although no conclusion can be drawn yet, these observations give additional support to the “Time-dependent” neural adjustments along the recovery phase.



**Fig. 52: Changes Evolution of voluntary torque (a), voluntary activation (b), electrically evoked peak twitch (c) and subjective delayed onset muscle soreness (d, black rectangle: analogic visual scale, white rectangle: Lickert scale) across the experimental sessions.** Data in mean  $\pm$  SEM, Asterisk indicates value or group of values significantly different from the other values of the graph ( $P < 0.05$ ). (From Racinais et al. 2008)

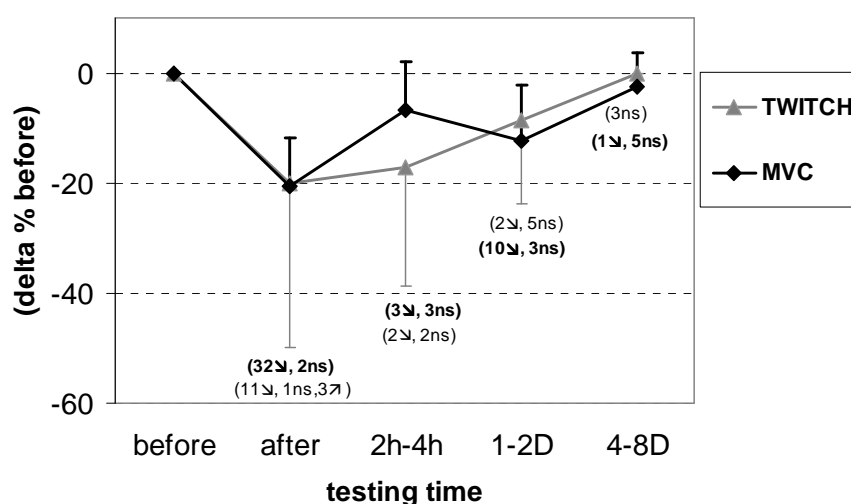


**Fig. 53: Effects of a leisure week of alpine skiing.** Relative values of maximal voluntary contractions performed, without (MVC) and with additional electro-stimulation (MVCEs), before and during a leisure week of alpine skiing and repeated 7 days later (d14). (Adapted from Strojnik et al. 2001)

#### 2.6.2.4. Electrically and magnetically evoked twitch EMG and force responses

The electrically evoked twitch response in passive testing condition is considered as one index of peripheral excitation-contraction coupling and/or contraction failure (Edwards et al. 1977). For 11 out of the 15 SSC fatigue studies that examined this parameter (Fig. 54), the acute recovery phase was characterized by reductions in the peak twitch torque that varied

from -8 to -70 %, whereas large increases (+8 and +18 %) were reported after ski skating marathon and ultra-long (5 to 8.5 h) running performances, respectively. Four of the 7 studies that examined the delayed fatigue effects observed a complete recovery already 1-2 days post-exercise ( Nicol et al. 2003; Kuitunen et al. 2004; Martin et al. 2004a; Ross et al. 2007). Only the study of Skurvydas et al. (2000) reported both large acute (-50 to -70 %) and delayed (-34 and -27%) reductions in the peak twitch. The fatiguing protocol consisted of a fixed number of either 100 maximal intermittent drop jumps or 100 continuous counter movement jumps. The authors suggested that mechanical factors (related to the imposed ground impacts) would cause larger acute and prolonged contractile failure than metabolic factors.

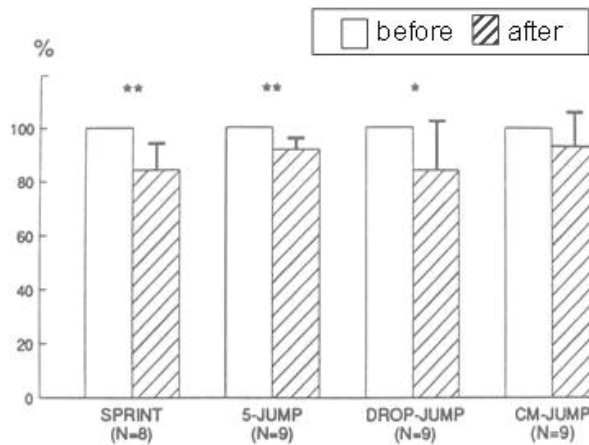


**Fig. 54. Relative acute and delayed decrements in electrically induced twitch amplitude (TWITCH) and in maximal isometric voluntary contraction (MVC).** MVC data are compiled from 29 published articles using either knee extensor or plantarflexor muscles and reporting either decreased ( $\Delta$ ) or non significant (ns) changes. The corresponding twitch changes are reported for 11 of these studies. For the mean value calculation, the non significant (ns) values have been replaced by the value zero.

#### 2.6.2.5. Maximal and submaximal SSC type performances

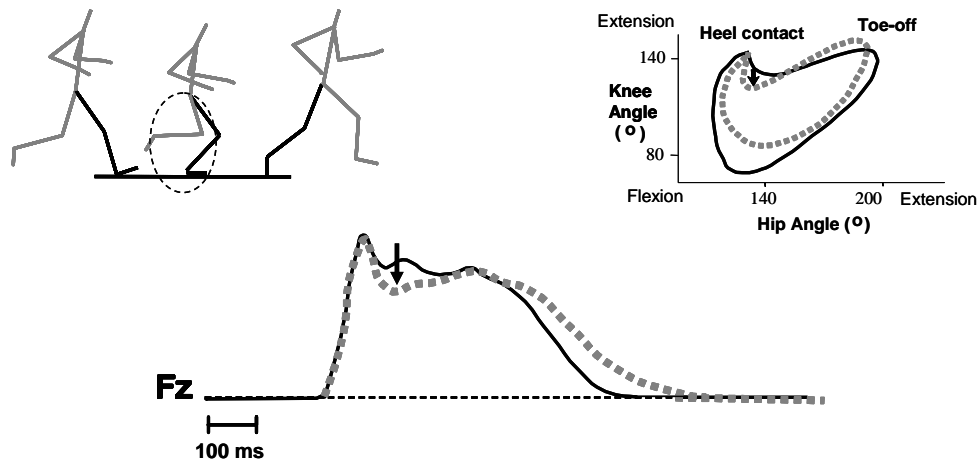
Although maximal isometric tests often reveal the effects of SSC fatigue, maximal dynamic tests seem more meaningful mechanistically. In dynamic tests involving slight or no ground impact such as in counter-movement and squat-jumps, no significant change is usually observed after SSC fatigue. However, when those tests are maximal and involve high ground impact peaks (drop-jump, five-jumps and sprint), significant acute (Nicol et al. 1991b) (Fig. 55) and delayed (Nicol et al. 1996b; Horita et al. 1996, 1999, 2003) reductions in performance have been reported after exhausting SSC exercise. After fixed fatiguing rebound protocols,

however, (Skurvydas et al. 2002) reported similar acute reductions in squat jump, counter-movement jump and drop-jump performances.



**Fig. 55: Relative changes in the before-after marathon of the maximal running, jumping and isometric performances.** SPRINT: 10 m sprint, 5-jump: 5 jump series, Drop-jump: drop-jump from a 50 cm height, CM-jump: counter-movement jump. These subjects present a mean 22% reduction in maximal isometric contraction. \*, \*\* P<0.05 and P<0.01 in pre-post marathon comparison. (Modified from Nicol et al. 1991b)

In these maximal situations, where stretch loads are high and muscle stiffness must be well regulated to meet the external loads, the decline in performance is typically characterized by a large peak force reduction after impact. These findings led to the hypothesis that the observed loss of tolerance to ground impact could result from a reduced contribution of the monosynaptic stretch-reflex to the leg stiffness (Nicol et al. 1991a, 1991b) (Fig.56).

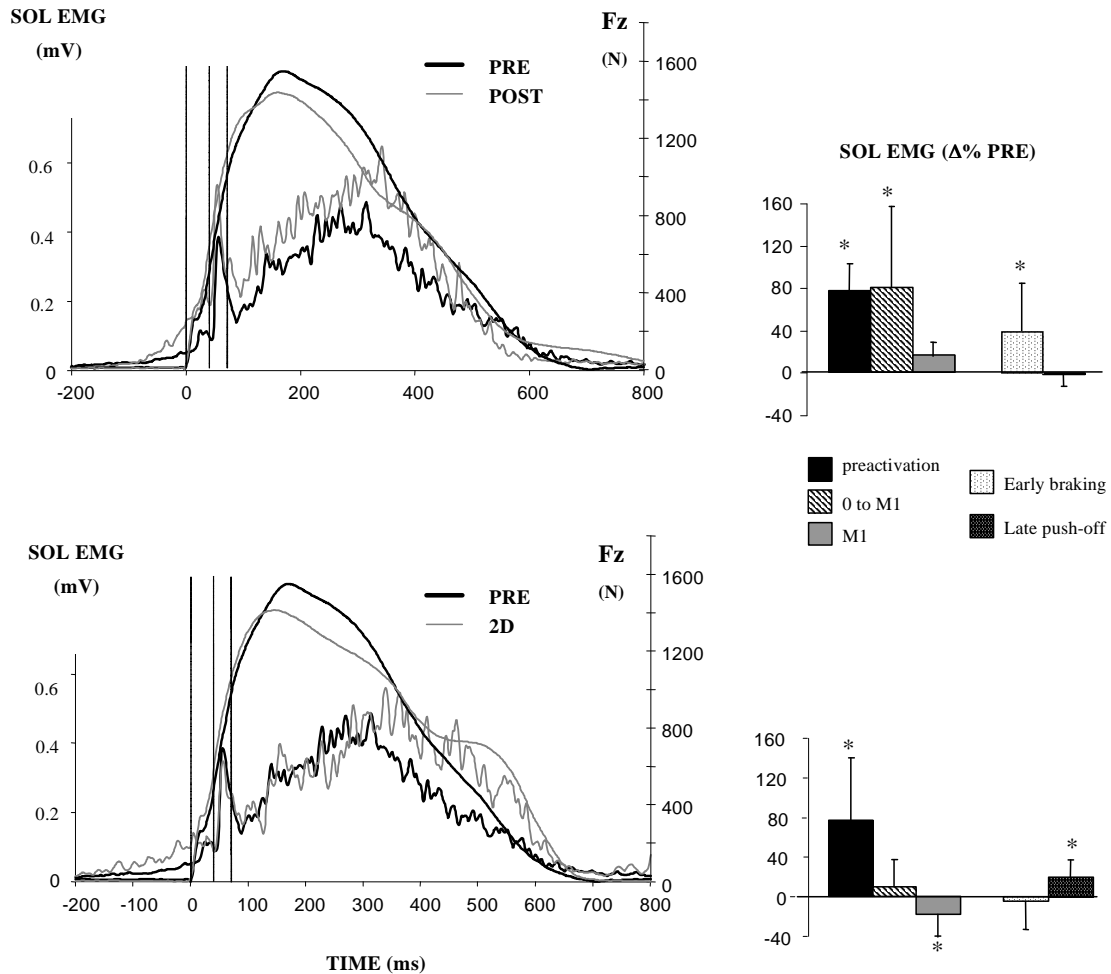


**Hypothesis: Reduced contribution of monosynaptic stretch-reflex (M1) ?**

**Fig. 56: Schematic representation of the hypothesis that the fatigue-induced loss of resistance (tolerance) to impact might result partly from a reduced contribution of the short-latency stretch reflex (M1) during the early braking phase.** (Modified from Nicol et al. 1991a, 1991b)

In SSC type performances, both central and reflex adjustments are indeed operative and expected to contribute to the observed changes in the post-landing stiffness along the

recovery period. The submaximal SSC tests emphasize the flexibility of the neuromuscular adjustments between the acute (POST) and the delayed (2D) recovery phases. In a submaximal rebound test, Regueme et al. (2005c) demonstrated varying neural changes immediately after (POST) and 2 days after (2D) an exhaustive SSC exercise (Fig. 57).



**Fig. 57** The left figures show group averaged records of vertical force (Fz) and soleus (SOL) electromyographic (EMG) activity compared at three measurement points after exhaustive stretch-shortening cycle exercise: pre-exercise (Pre), immediately after (Post) and 2 days post-exercise (2d). The figures on the right present the relative EMG changes (histograms) for the preactivation, 0–M1 and M1 periods, the early braking (first 200ms) and late push-off (last 200ms) of the contact phase. \*  $p < 0.05$ , significantly different from the Pre absolute values. (From Regueme et al. 2005c)

In the POST testing session, large increases in SOL activation occurred both during the preactivation and the early braking phase. Differing from these POST exercise observations, the increased SOL preactivation at 2D was not associated with an increased activation during the early braking phase, but during the late push-off phase. This shift of muscle activation

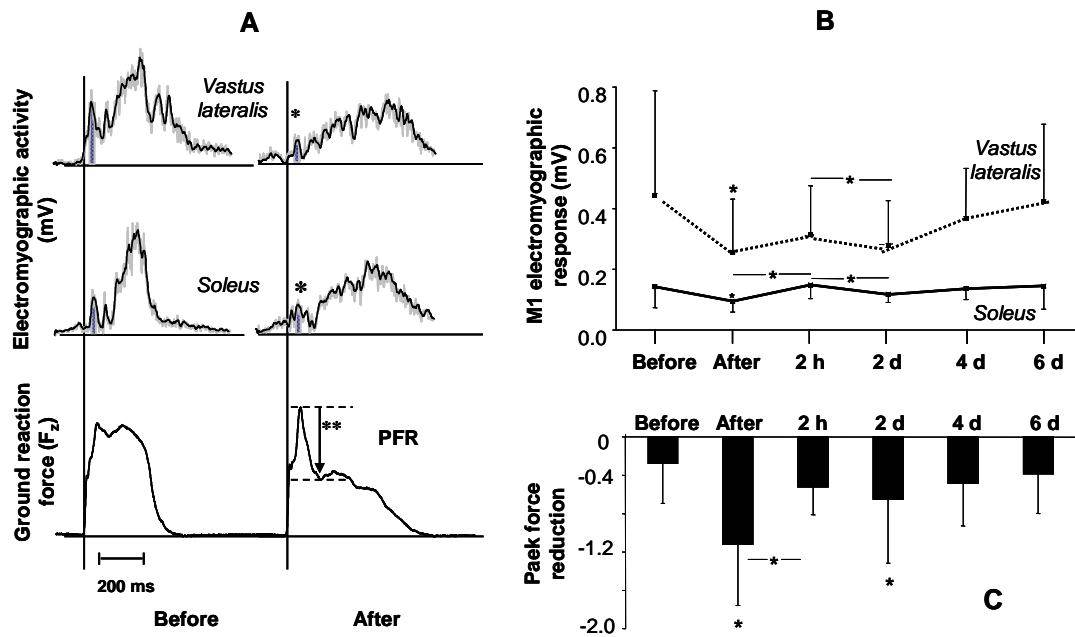


may thus be considered to reflect a neural strategy to protect the recovering muscles at 2D point from the stressful stretching phase, while securing the rebound performance by increased work during the push-off phase. Differing from the increase in central activation (preactivation and 0-M1 periods) in both POST and 2D, the M1 reflex response of SOL did not change in the acute phase and decreased at 2D (Fig. 57). These data support the hypothesis of a bimodal reduction of the stretch-reflex response. Furthermore, the 2D test revealed a 36 % increase in GAM muscle preactivation. These results are supported by the earlier mentioned fatigue study of Horita (2000) who demonstrated, during the time course of a rebound exercise, progressive inter-muscular compensations for the exercise-induced contractile failure (Fig 42). Finally, even though the EMG analysis of submaximal SSC tasks is shown to reveal the existence of a contractile failure, this may not necessarily be detected based through the kinematic and kinetic analysis of the movement (Finni et al. 2003b; Kyröläinen et al. 2000)

#### ***2.6.2.6. Stretch- and H-reflex responses***

As it is not always easy to isolate the stretch-reflex EMG response from the global EMG recordings, potential effect of SSC fatigue on the EMG reflex response have been examined, at first, in *passive reflex testing conditions* (Nicol et al. 1996b). As previously described in section 1.5.2, a powerful engine was used to induce passive stretches of the shank muscles at slow and intermediate angular velocities ( $0.44 - 1.9 \text{ rad}\cdot\text{s}^{-1}$ ). Intensive SSC rebound exercises were found to result in a bimodal trend of decline in the peak-to-peak EMG reflex response to passive stretches after a marathon run (Avela et al. 1999a) as well as after a very intensive rebound exercise on the sledge (Nicol et al. 1996b).

In subsequent SSC fatigue studies, the stretch-reflex EMG response has also been measured in active stretching conditions, while performing maximal rebound tests (Horita et al. 1996; Avela et al. 1999b; Dousset et al. 2007; Regueme et al. 2007b) on the sledge ergometer. Supporting the functional role of a prolonged reduction in M1, Avela et al. (1999b) showed a parallelism in the bimodal trends of reduction observed in M1 and in the tolerance to high ground impact loads (Fig. 58). This was associated with clear EMG reductions during both centrally programmed preactivation and the subsequent braking phase. In SSC type performances, both central and reflex adjustments are operative and they are expected to contribute to the observed changes in the post-landing stiffness along the recovery period.



**Fig. 58. SSC fatigue effect on the active short-latency stretch-reflex (M1) response and associated changes in the rebound kinetics.**

(A) Bimodal recovery of electromyographic activity and vertical ground reaction force over time, during 10 successive sledge rebounds performed before and after marathon running.

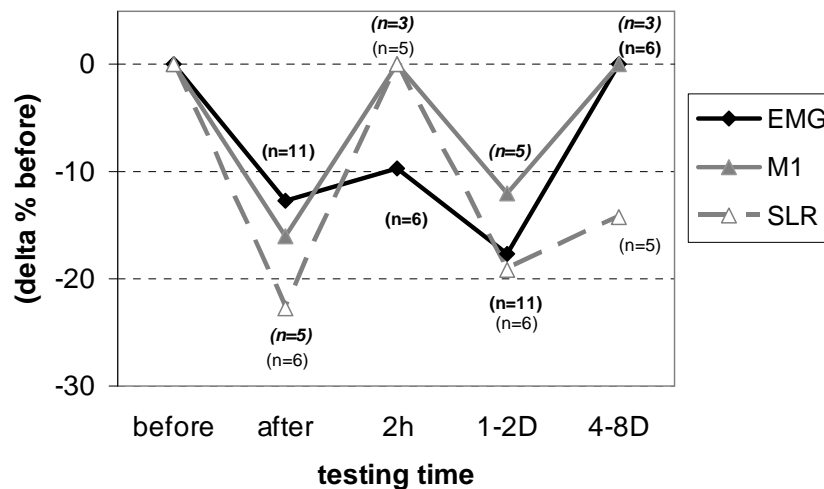
(B) Corresponding active short-latency reflex component (M1) of electromyographic activity.

(C) Postlanding stiffness regulation as reflected by peak force reduction (PFR) measured from the normal component of the ground reaction forces (Adapted from Avela et al 1999b)

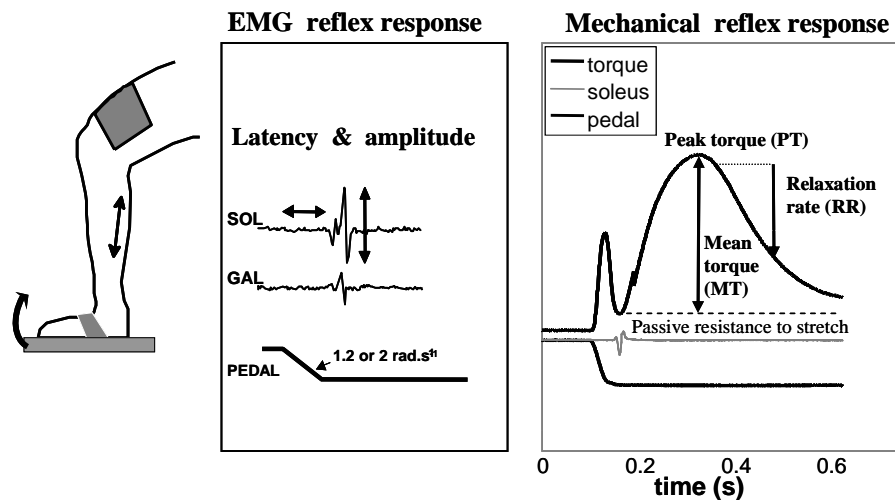
As shown in Fig. 59, the passive short-latency EMG reflex response (SR) and the active one (M1) measured in maximal drop-jump test on the sledge follow on the average the bimodal trend of recovery. The inter-individual variability may deserve, however, some comments as reflex facilitation has been reported for a few subjects in the acute recovery phase (after - 2h), in both passive and active stretch reflex responses (Kuitunen et al. 2004; Nicol et al. 2003). Depending on the study, this reflex facilitation was followed either by a complete recovery (Nicol et al. 2003) or by a secondary inhibition on day 2 (Kuitunen et al. 2004). Based on these observations, it is suggested that the bimodal shape of recovery should not be generalized to all subjects as “fast-recovering” subjects have also been observed in SSC fatigue studies (i.e. Nicol et al. 2003).

More recently, the SSC fatigue effects on the mechanical stretch-reflex response have been quantified along a 7 day recovery period (Nicol et al. 2003). The tests consisted of passive single stretches of the triceps surae muscle group. As shown in Fig 60, the pedal torque signal was used to quantify the passive stretch effect as well as the reflex-induced peak torque (PT), mean torque (MT) and relaxation rate (RR). This study revealed a bimodal

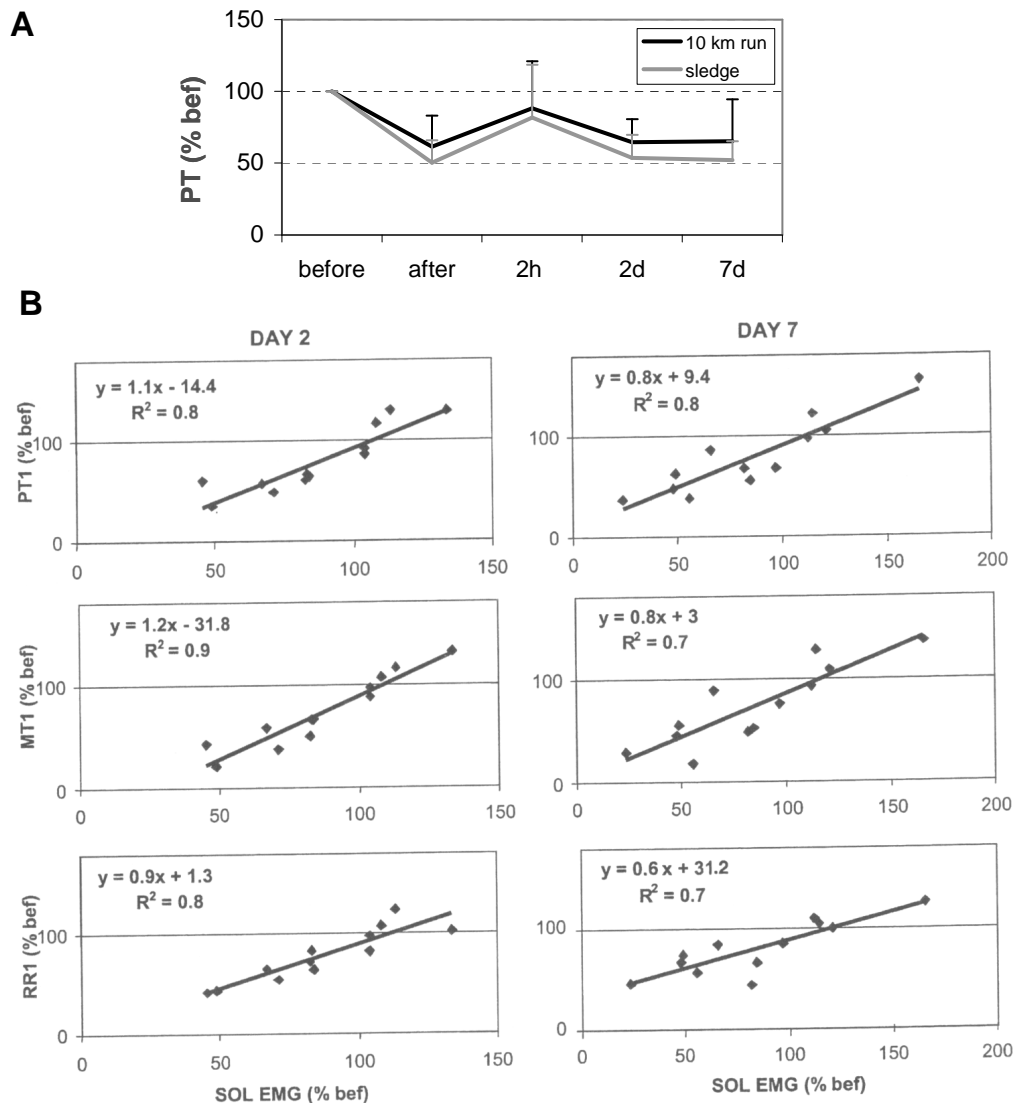
recovery of the mechanical reflex response after short-term (sledge exercise) as well as after long-term (10-km run) fatiguing exercises (Nicol et al. 2003). The relative changes in the EMG amplitude of the reflex response were found to be positively related at day 2 to each of the measured mechanical response during the delayed recovery phase (Fig. 61). This implies that the EMG/force ratio could be maintained to a large degree during the 2-7 day recovery, thus emphasizing once again the parallelism of the neural and mechanical changes along the recovery period.



**Fig. 59. Relative acute and delayed decrements following SSC exercises in maximal voluntary EMG activity (EMG), in the passive short-latency stretch-reflex response (SLR) and in the active (M1) one.** Data compiled from 14 published articles testing either knee extensors or ankle plantarflexors. For the group mean calculation (n = number of studies), the non significant (ns) values have been replaced by the null value.



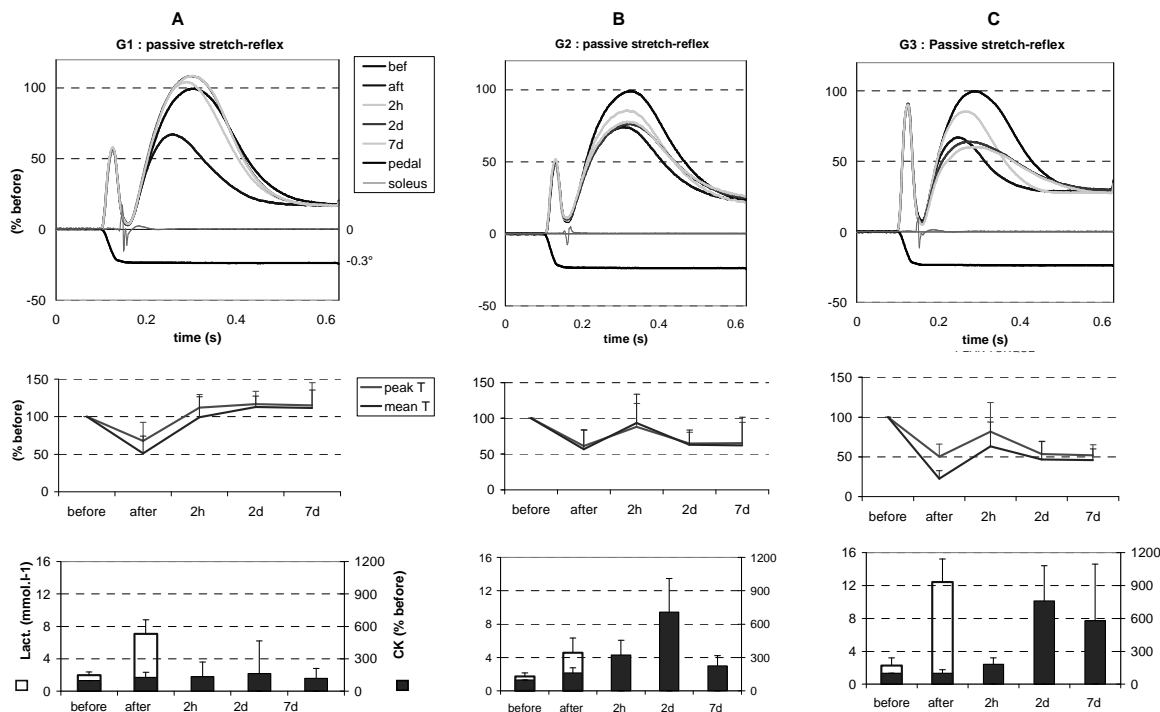
**Fig 60. Schematic presentation of the stretch-reflex test with the analysis of the associated EMG (left) and mechanical (right) reflex responses.** (Modified from Nicol et al. 2003)



**Fig 61. (A) Acute and delayed fatigue effects on the mean torque parameter in the 10 km and sledge subgroups. (B) Relationship on days 2 and 7 between the respective relative changes in SOL EMG and twitch reflex parameters. PT1, MT1 and RR1 correspond to the respective peak, mean torque, and relative relaxation rate of the twitch response. All correlations were statistically significant ( $P < 0.001$ ). (From Nicol et al. 2003)**

The preceding presentation may give an impression that the fatigue response and subsequent recovery is a generalized pattern. This is not at all true in all cases, because SSC fatigue responses can be very individual in nature. Fig. 62 gives examples of these varying responses. In this study (Nicol et al. 2003), the pure mechanical reflex response of 12 subjects were divided into 3 subgroups: G1 presents only acute changes (Fig. 62A), G2 follows the expected bimodal trend (Fig. 62B), and G3 presents an additional slower rate of relaxation on

the 2nd and 4th days post-exercise (Fig. 62C). The delayed mechanical changes varied in accordance with the respective changes in EMG reflex response and in creatine kinase (CK) activity, an indirect marker of muscle damage. As shown by the limited CK changes, the first subgroup included the less fatigued subjects.

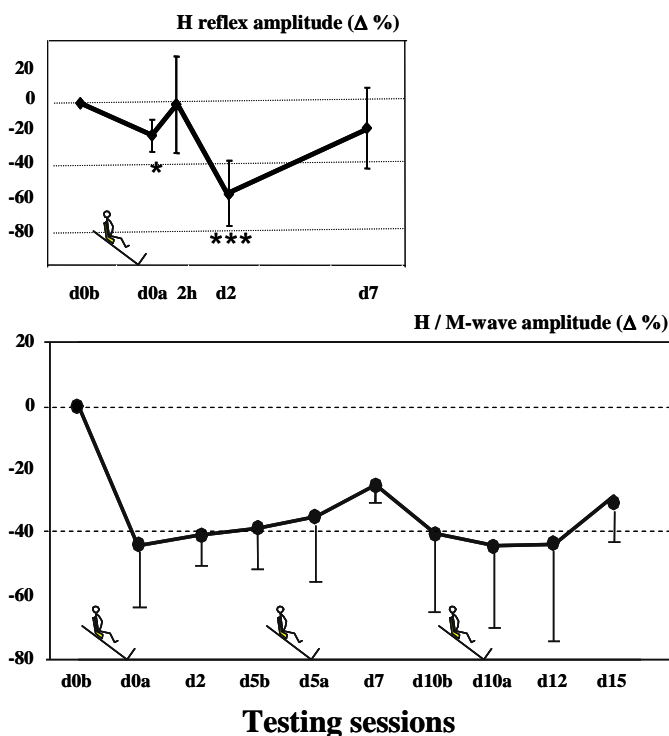


**Fig 62. Effects of fatiguing SSC exercises on the passive mechanical reflex response (torque response) (upper 2 lines of graphs) with the respective changes in serum lactate concentration and creatine kinase (CK) activity.** The data are presented for 12 subjects divided into 3 subgroups according to the peak and mean torque (T) reflex responses: G1 with only acute changes (A), G2 with a bimodal trend (B), and G3 with additional slower rate of relaxation on the 2<sup>nd</sup> and 7<sup>th</sup> days post exercise (C). (From Nicol et al. 2003)

These overall results demonstrate that muscle function and stiffness regulation may be disturbed in a delayed, but individual manner after exhaustive type SSC exercise. On the other hand, a clear parallelism exists between the respective changes in performance, in neural (central and peripheral) activation, and in indirect indicators of either metabolic or structural source of fatigue. This implies the existence of potential coupling between the contractile type of failure and the central and peripheral adjustments that take place along its recovery.

Finally, to detect potential changes in the spinal excitability level, the *Hoffmann-reflex test (H-reflex)* of the soleus muscle have also been also recorded. After intensive rebound exercise on the sledge (Nicol et al. 1996b), this was associated with a bimodal decrease of the H-reflex response. Similarly, both acute and delayed reductions in H/M ratio have been

observed after each of 3 successive exhaustive rebound exercises performed on the sledge at a 5 day interval (Nicol et al. 1996a) (Fig. 63). In two other marathon running studies, however, the acute reduction in the H/M-wave ratio was not followed by any significant secondary decline (Avela et al. 1999b; Kuitunen et al. 2004). However, emphasizing the neural influence on the bimodal stretch-reflex changes, Dousset et al (2007) recently reported parallel changes in both passive (SR) and active (M1) EMG responses as well as in the normalized H (H/M) reflex response.



**Fig 63. Upper graph:** Relative changes (mean  $\pm$  S.D.) in H-reflex amplitude after 75 min of electrical stimulation combined with mechanically induced stretches. (Unpublished observations)  
**Lower graph:** Relative changes (mean - S.D.) in the H/M-wave ratio in the course of a 15-day follow-up during which an exhaustive short-term SSC exercise was repeated on days 0, 5 and 10. In both experiments the changes are expressed as a percentage of the pre-fatigue value (d0b: day 0 before). (Data from Nicol et al. 1996a)

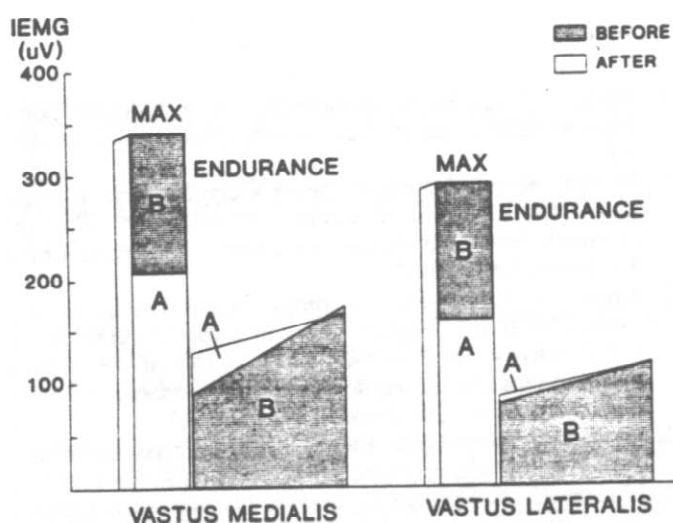
### 2.6.3. “Testing –task” influence on the activation pattern

If the “time-dependency” is a factor to take into consideration in SSC fatigue studies, the “testing task-dependent” effect should be equally considered.

#### 2.6.3.1. Maximal versus endurance type isometric tests

According to our knowledge, only one study (Nicol et al. 1991b) examined the influence of an exhaustive SSC exercise (marathon run) on both maximal and submaximal

voluntary isometric tasks. In sustained submaximal isometric contractions, EMG amplitude is reported to increase during the course of the exercise, and to remain less than maximal at the limit of endurance (Gandevia 2001). In the present study, the post-marathon test was found to start at a higher EMG activity level and to end at a similar terminal value than the pre-marathon one (Fig. 64). Although this led to an earlier stop of the exercise, the initial increase of the neural drive is likely to reflect the recruitment of additional motor units in order to compensate for fatigue of active muscle fibres. As shown in Fig. 64, this contrasted clearly with the parallel and systematic drops in maximal force and voluntary activation observed in the MVC test. The submaximal testing condition may thus reveal a “central attempt of compensation” of the contractile failure.

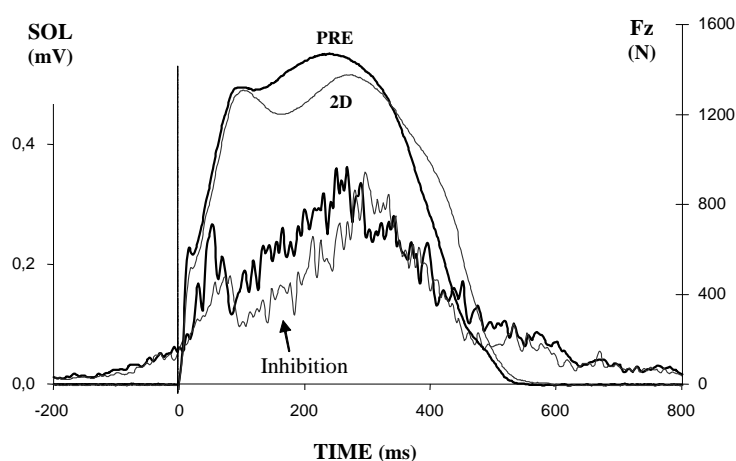


**Fig 64. Before and after-marathon levels of the average integrated (IEMG) activity in the maximal (MAX) and endurance tests for vastus medialis and vastus lateralis muscles:** (From Nicol et al. 1991b)

### ***2.6.3.2. Influence on maximal vs. submaximal SSC dynamic contractions***

The hypothesis of “testing task dependent” effects is supported by the different trends of neural adjustments to fatigue reported in submaximal (Regueme et al. 2005c) as compared to maximal (Avela et al. 1999b; Regueme et al. 2005a) rebound tests. In maximal drop-jump performances that require an appropriate muscle stiffness regulation to meet the external loads, the decline in performance is typically characterized by a loss of resistance to impact (PFR: peak force reduction after impact) (Fig. 58A, C). As illustrated in Fig. 65, this is associated with clear EMG reductions during both the centrally programmed preactivation

phase, and the subsequent braking phase. Simultaneously observed reductions in the short latency stretch-reflex response (M1) suggest that the observed EMG changes in SSC tasks are both central and reflex in nature. These observations support the existence of neural attempts to protect the fatigued muscles in maximal SSC (e.g. drop-jump) conditions. They also support the findings of Gollhofer et al. (1987a, 1987b) in arm muscles that protective neural mechanisms (reduced short, medium and long latency components of the stretch reflex) only take place in the most extreme testing conditions (falls on the floor performed with arm muscles). Supporting the functional role of a prolonged reduction in M1, Avela et al (1999b) showed a parallelism in the bimodal changes between M1 and the ability to withstand high ground impact loads (Fig. 58B, C).



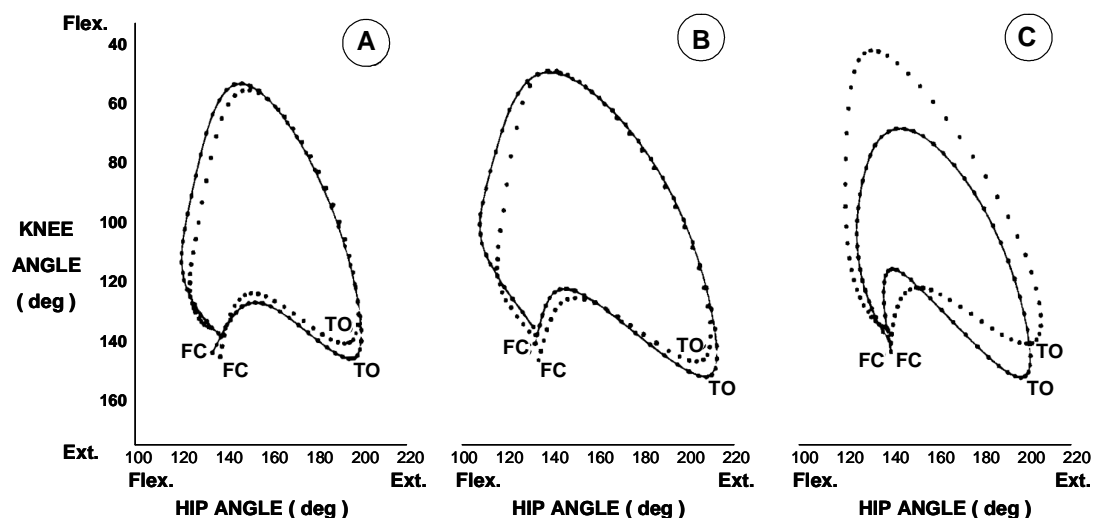
**Fig. 65.** Group ( $n = 10$ ) averaged records of vertical reaction force ( $F_z$ ) and soleus (SOL) electromyographic activity recorded in maximal drop jump and compared at two measurement points after exhaustive stretch-shortening cycle exercise: just before exercise (Pre) and 2 days after the exercise (2d) (From Regueme et al. 2007b).

The neural changes with fatigue may differ in submaximal SSC rebound and running tests in which the neuromuscular system may adjust in a more flexible way to the acute and delayed contractile impairments. For example, Regueme et al. (2005c) demonstrated in a submaximal SSC rebound task large acute (+ 74 %) and delayed (+ 79 %) increases in SOL preactivation (Fig 57). It is well known that a link exists between the increases in preactivation and impact peak (Avela et al. 1999b; Gollhofer et al. 1987a, 1987b; Komi et al. 1987a). In the SSC fatiguing condition, a loss of tolerance to ground impact occurs frequently (Gollhofer et al. 1987a, 1987b; Nicol et al. 1991c; Avela et al. 1999b). In such a case, an increased preactivation should be functionally useful in submaximal rebound conditions (Bonnard et al. 1994), but detrimental in maximal rebound jumps. In this line, the only SSC fatigue study that reported post-fatigue increases in muscle preactivation in maximal DJ was



characterized by large decreases in performance (Horita et al. 1999). Deviating slightly from this “general rule” of testing task dependency, Avela and Komi (Avela and Komi 1998) revealed large, but similar fatigue-induced reductions in preactivation in SSC performances performed from submaximal (70 %) and supramaximal (130 %) dropping heights, but in both cases with maximal rebound effort. This would suggest that exhaustive SSC exercise leads to different variations in neural adjustment depending on the type of the drop jump test. When the rebound height is varied (sub- vs. maximal) the variability is greater as compared to the test in which the dropping height is varied (sub- vs. supra-maximal one).

In our initial marathon study (Nicol et al. 1991a), even though the immediate post-marathon treadmill test revealed unchanged running kinematics at the three pre-set submaximal velocities, EMG activity of the gastrocnemius muscle was significantly increased at the two fastest velocities during both braking and push-off phases. The higher the testing velocity, the larger was the increase in gastrocnemius activation (Nicol et al. 1991a). Clear modification in the running kinematics was observed only in the subject (C in Fig. 66) whose maximal and endurance force productions by the knee extensors were reduced at the post-marathon test by 40 and 56%, respectively. In this subject fatigue was found to result in a larger and faster knee flexion after impact. This is likely to indicate a deterioration of the musculo-tendinous tolerance to impact with a consequent loss in its recoil characteristics. To maintain the imposed running velocity, this would explain also the needs for an increased work during the push-off phase and for a shorter and faster return during the flight phase.



**Fig. 66.** Examples (subjects A, B and C) of hip-knee angle cyclograms measured before (cut line) and after (black line) the marathon at the fast ( $4.5\text{-}6\text{ m}\cdot\text{s}^{-1}$ ) treadmill running speed. (Adapted from Nicol et al. 1991a)

In conclusion, our analysis of the relevant SSC fatigue literature emphasises the testing time and task dependency of the neural adjustments to meet the functional requirement of the peripheral system. Centrally programmed and reflex activation changes may be opposite in a given task (central facilitation vs. peripheral inhibition). They may also vary depending on the task intensity (maximal vs. submaximal) and recover at different pace (acute vs. delayed). It is very likely that in order to observe true changes in these parameters one may need to fatigue the subjects more exhaustively as this was the case (see Fig. 66) for the most fatigued runner of our marathon study (Nicol et al. 1991a).

#### ***2.6.4. Delayed influence on the sense of position and velocity***

Surprisingly, the literature presents no clear agreement related to the influence of eccentric- and SSC-type muscle fatigue on the position sense acuity. As reviewed by Proske and Morgan (Proske and Morgan 2001), no effect as well as opposite proprioceptive disturbances have been reported. These conflicting results may have different origins. Firstly, perception of active limb position may arise as a consequence of the effort required to accomplish the task (Gandevia et al. 2006; Proske 2006). Secondly, position coding is known to depend on the sensory information available during the positioning phase of the limb toward the target. Healthy human subjects are indeed aware of the positions of their limbs while the limbs are moving (dynamic information) as well as when they remain stationary (static information) (Cordo et al. 1994). It is noteworthy that sensitization of small diameter muscle afferents by intramuscular injections of inflammatory substances has led to the following trends: an increase of the static intrafusal fibre sensitivity (Djupsjobacka et al. 1995; Thunberg et al. 2002; Masri et al. 2005) and a decrease of the dynamic one (Pedersen et al. 1998).

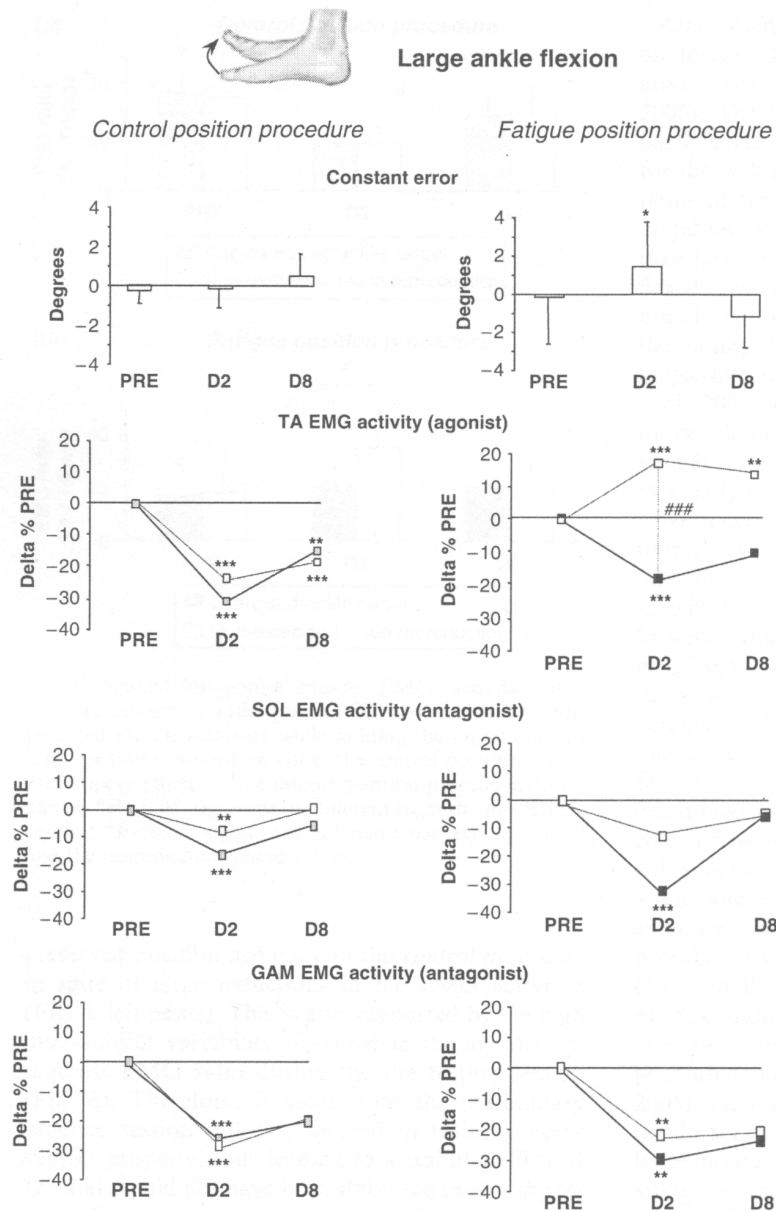
The recent study of Regueme et al. (2008) investigated the influence of an exhaustive SSC exercise on the static position sense in an attempt to give information about its effects on the static sensitivity of the muscle spindle afferents. The position sense test was carried out before an exhaustive SSC exercise, and was repeated at the expected times of major inflammation and pain (day 2), and at complete recovery (day 8). A protocol making dynamic information unavailable in position coding was considered as an appropriate method to assess the SSC fatigue influence on the static sensitivity of the muscle spindle afferents (Regueme et al. 2008). The test consisted of actively reproducing, with the non-exercised ankle, target dorsiflexed positions previously maintained with either the non-exercised (control procedure) or the exercised ankle (fatigue procedure). Based on the main contribution of the stretched

muscle to the position sense (Roll et al. 1989; Ribot-Ciscar et al. 2003) and on the linear relationship between muscle spindle discharge and muscle stretch (Vallbo 1974), a position sense test using ankle dorsiflexion was indeed expected to reveal potential changes in the muscle spindle afferent sensitivity of the fatigued (stretched) triceps surae muscle group. The use of the non-exercised ankle for the active reproduction was based on the fact that SSC muscle fatigue may affect both the ascending (proprioceptive) and the descending (motor) neural pathways of the exercised ankle (Nicol et al. 2006). The time between the target phase with the exercised ankle and the reproduction phase with the non-exercised ankle was chosen to be long enough to prohibit any memory storage of the efferent copy (3s).

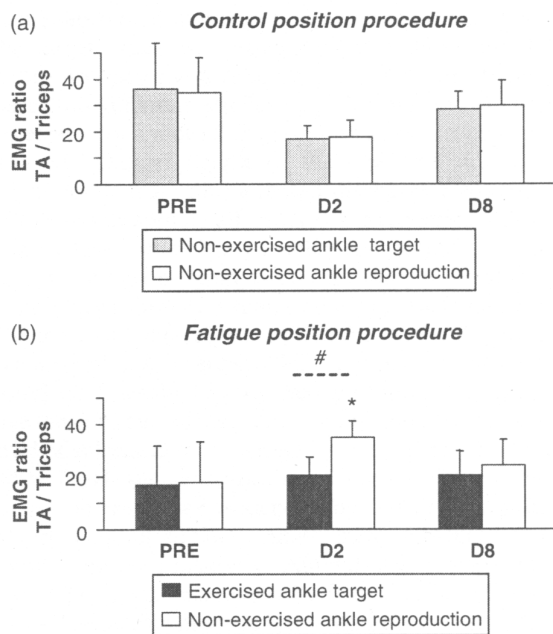
Testing of the fatigued leg supported the hypothesis that the proprioceptive information and especially the position sense might be modified during the recovery period following an exhaustive SSC exercise (Regueme et al. 2008). As previously observed after such a fatiguing protocol (Regueme et al. 2005c, 2007b), the exercised leg presented significant reductions in both maximal plantarflexion force and voluntary SOL muscle activity at day 2 (D2). In addition, all subjects reported major muscle pain in the fatigued triceps surae muscle group, with a peak at D2. As expected, no fatigue effect was revealed in the MVC test of the contralateral (non-exercised) leg. In the position sense test, no error in the ankle dorsiflexed position coding was found when the non-exercised ankle was used both in the target and reproduction phases (control procedure) (Fig. 67 upper left histograms). In contrast, the fatigue procedure revealed positive and constant coding errors at D2 (Fig. 67 upper right histograms) when the non-exercised ankle reproduced the large dorsiflexed target position held by the exercised ankle, thus reflecting a systematic overestimation of the target dorsiflexed position held with the exercised ankle.

The parallel EMG analysis revealed, however, large reductions in both agonist and antagonist EMG activities in the control procedure (Fig. 67 left panel). Interestingly, when the exercised ankle was used in the target phase of the fatigue position procedure (Fig. 67 right panel, black squares), similar trends of changes were observed. Based on the high inter-subject variability observed in the agonist/antagonist EMG ratio during the pre-fatigue (PRE) testing session (Fig. 68), these EMG changes might reflect a learning effect. The respective analysis of the fatigue position procedure revealed a significant reduction in gastrocnemius medialis (GAM) but not in soleus (SOL) muscle activity at D2 that contrasted with an increased TA muscle activity up to day 8 (D8). This resulted at D2 in a significant increase of the agonist/antagonist EMG ratio as compared with the Pre testing session (Fig. 68b).

In agreement with the antagonist (stretched) muscle influence on the position sense, the overestimation of large dorsiflexed position is mostly attributed to potential SSC fatigue effects on ascending proprioceptive afferents issued from the exercised/inflamed antagonist muscles.



**Fig. 67. Major results of the large dorsiflexed position, when using either the control position procedure (left panel) or the fatigue one (right panel) in the tests performed before (Pre), 2 (D2) and 8 days (D8) after the unilateral exhaustive SSC exercise.** *In the control procedure, the non-exercised ankle was used in both the target (■) and reproduction (□) phases, whereas in the fatigue procedure the target was held with the exercised ankle (■), and the reproduction with the non-exercised one (□). The constant error is represented by its group-averaged values (and SD). The relative changes in the recorded muscle activities are expressed in percentage of their values recorded during the position sense test in the Pre testing session (delta % Pre). \*P<0.05, \*\*P<0.01, \*\*\*P<0.001: significantly different from the Pre absolute values; ###P<0.001: reproduction phase significantly different from the target phase. (From Regueme et al. 2008)*



**Fig. 68. Agonist/Antagonist muscle EMG activity ratio.** Agonist/antagonist ratio (mean+SD) calculated from the recorded muscle activities while holding the target and the reproduction positions, in either the *control position procedure* (upper panel) or the *fatigue position procedure* (lower panel). \*P<0.05: significantly different from the Pre-fatigue session; # P<0.05: significant difference between the target and the reproduction phase values. (From Regueme et al. 2008)

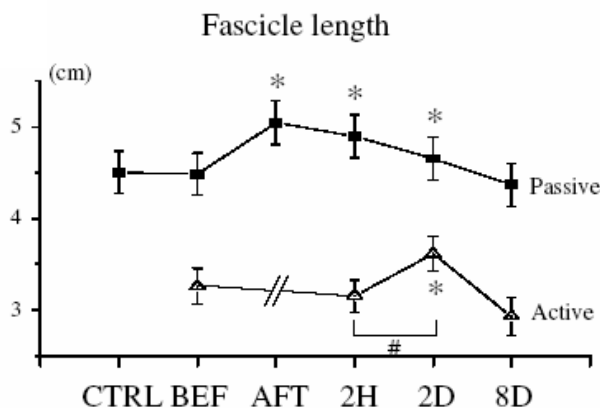
In another study of Regueme et al. (2007a), the perceived movement velocity induced by tendon vibrations was examined during the delayed recovery phase of a SSC-type exercise characterized by 2 to 4 days of neuromuscular and proprioceptive impairments. SSC fatigue effects were quantified for the exercised and non-exercised legs through a maximal voluntary plantarflexion test (MVC) performed immediately before (PRE) and after the SSC exercise, and repeated two days later (D2). At PRE and D2, mechanical vibrations at 40, 60, 80, 100, and 120Hz were randomly applied to distal tendons of the exercised ankle. For each vibration, the subjects had to reproduce the perceived movement velocity with the non-exercised ankle. In agreement with previous studies (Regueme et al. 2005c, 2007b), MVC data showed significant decreases in maximal force and mean soleus muscle activity at D2 restricted to the exercised leg. As compared to the PRE test and in all subjects, the vibrations applied at D2 to the tendon of the fatigued ankle extensor muscles led to significant decreases in the perceived movement velocity at 80 and 100 Hz, but to an increased one at 40 Hz. In contrast, vibrations applied to the tendon of the non-fatigued ankle flexor muscle did not result in any significant change.

These results suggest that SSC fatigue can result in proprioceptive disturbances lasting for a few days. Similarly to what was observed in animal (Pedersen et al. 1998) and in Human (Paschalis et al. 2007) studies, the present proprioception impairments suggest a decrease in sensitivity of the primary endings during the delayed phase of the SSC recovery period.

### 2.6.5. Muscle specificity

Differing from the “eccentric type exercise”, SSC leads to a more complex mechanical loading of the fascicles and tendinon-aponeurosis tissues. In the SSC description it has been shown that the interaction between contractile tissue (fascicles) and tendinous tissue may be a complex one. The recent works of Ishikawa and collaborators (Sousa et al. 2007; Ishikawa and Komi 2008) confirm the possibility that the contractile tissue does not always follow the basic concept of SSC of the whole muscle-tendon-unit MTU. The general observation was that the gastrocnemius muscle usually demonstrates fascicle shortening during the braking phase at low impact conditions of the drop jumps. With very high dropping heights the fascicles may become dramatically lengthened during the braking phase. This was interpreted to indicate reduced resistance to stretch through possible detachment of cross-bridges. The soleus and vastus lateralis muscles, on the other hand, showed usual SSC behaviour in the fascicles. The fascicle length changes were thus found to be clearly “muscle specific” (GA vs. SOL) and “intensity specific”. The tendinous tissue (aponeurosis and outer tendon) may behave even more differently from the basic MTU SSC concept (Fig. 11).

If the questions raised above deal with the non-fatigue situation, the understanding of the changes in fascicle length and orientation during exhaustive SSC fatigue is even more open. In our recent study (Ishikawa et al. 2006a), the fascicles were found to be lengthened in a passive (resting) situation immediately after the exercise and they remained lengthened until 2 days post-exercise (fig. 69). During maximal isometric action, the final fascicle length measured at peak force was also longer at day 2 post-exercise, and recovered completely to the original length at day 8 post-exercise. It is too early to interpret these findings in terms of functional mechanisms. More studies are needed with the ultrasound method that should also take into consideration the entire fascicle-tendon interaction.



**Fig. 69. Fascicle length changes in passive and active conditions during the eight day followed-up period (CTRL: control day, BEF: before the fatiguing exercise, AFT: immediately after it, 2H: 2hours later; 2D: 2days and 8D: 8days after the exercise. (Modified from Ishikawa et al. 2006a)**

### ***2.6.6. Any contralateral influence?***

The preceding paragraphs have included material with the assumption that the SSC fatigue effects are primarily unilateral in case the exercise has been performed with one limb only. There is, however, information in the literature that could imply contralateral effects as well. The general existence of “contralateral irradiation” of unilateral activation has been known for a century (e.g. Sherrington 1906). Since then, growing evidence exists that contralateral improvements in motor skill and strength rely on “cross-over effects” of unilateral motor and sensory activity (e.g. Hortobagyi et al. 2003). As muscle fatigue is typically associated with motor and sensory changes, fatiguing voluntary efforts on one side may thus be expected to alter voluntary activation of homologous muscles on the other side of the body.

This hypothesis has been examined by some studies, but mostly after maximal isometric contractions (Zijdewind et al. 1998; Todd et al. 2003a; Rattey et al. 2006). Their results have revealed minor crossover effects on the voluntary activation of upper limb muscles (Zijdewind et al. 1998; Todd et al. 2003a), but more significant ones are expected in the lower limbs (Rattey et al. 2006). Transcranial magnetic stimulation has shown only some evidence of changes in the ipsilateral cortex following fatiguing exercise, with effects confined to the muscle homologous to the fatigued one (Humphry et al. 2004). Within a limb, exercise of the biceps muscle that produced altered responses to transcranial stimulation did not produce similar changes in hand muscles (Taylor et al. 1996; Humphry et al. 2004).

Our interest is, however, to discuss the hypothesis of potential cross-over effects as it is related to the exhaustive SSC exercise, in particular to the delayed recovery period (after day 2). This is due to the fact that it lasts for a few days, usually associated with muscle pain and attributed to the natural time course of inflammatory and/or remodelling processes related to the exercise-induced muscle damage (for a review, see Nicol and Komi 2003). Muscle inflammation and pain have repeatedly been shown to affect the contralateral side in case of chronic (more than three weeks) unilateral perturbations (e.g. Johansson and Sojka 1991; Lund et al. 1991; Radhakrishnan et al. 2003). The SSC recovery is also very likely to result in an increased effort perception for a given task that should favor the occurrence of cross-over effects (Dettmers et al. 1995; Zijdewind et al. 1998). These observations raise the question as to whether contralateral changes that are known to result from long-term adaptations in the human behavior could occur in case of a few days of musculoskeletal disorders. Leading to short-term muscle disorders, with reversible muscle damage, inflammation and pain, exhaustive SSC exercises are thus of particular interest.

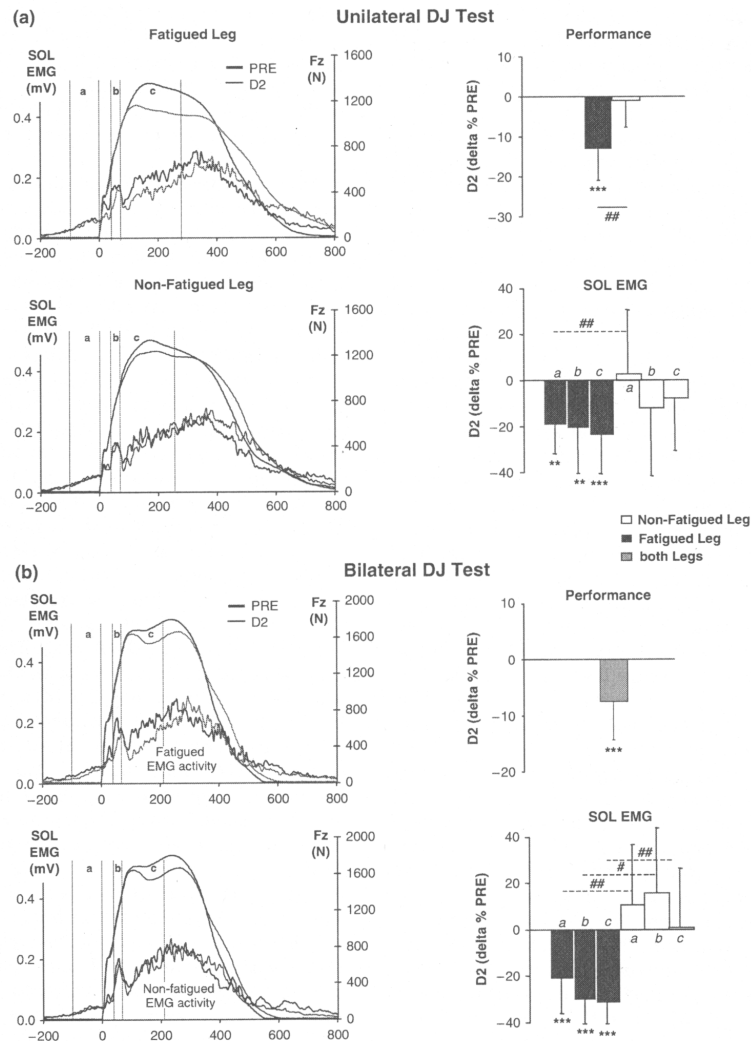
For this purpose, Regueme et al. (2007b) recently examined the possibility of contralateral effects of unilateral SSC fatigue of the triceps surae muscle group in unilateral as well as in bilateral testing conditions. Ipsilateral versus contralateral fatigue effects were compared in a single-joint task of maximal isometric plantarflexion (MVC), and in multi-joint SSC tasks that included 10 intermittent maximal drop jumps (DJ). The latter SSC testing task was chosen to examine fatigue-induced neural adjustments and more specifically those occurring during the preactivation phase and during the intervention of the short-latency M1 stretch-reflex response. Uni- and bilateral tests were carried out before and after the fatiguing exercise and were repeated two days later, at the time of expected peak of damage and pain in the exercising muscles, as well as eight days later. When such a stressful maximal DJ task is performed with both legs, two additional and opposite trends of compensatory contralateral strategies may be expected to occur: (i) an increased activity of the contralateral leg muscles prior to impact and during the subsequent braking phase in order to limit the risk of further injury of the fatigued/damaged muscles or (ii) a decreased contralateral muscle activity in order to either reduce the inter-leg imbalance (McCabe et al. 1972) or to ensure maintained coordination (Rathey et al. 2006).

In all testing conditions, however, the overall fatigue effects occurred at day 2 only, and were restricted to the exercised leg in both uni- and bilateral tests (Fig. 70). Three non exclusive reasons may be considered to explain why we did not observe any clear contralateral neural change. First, it is suggested that the present SSC fatiguing exercise did not lead to sufficient inflammation and pain to affect the contralateral side clearly. Owing to the expected right shift of the force-length relationship after fatiguing eccentric muscle actions (Morgan et al. 2002, McHugh and Tetro 2003), the large 20% drops in isometric force at day 2 might have been overestimated. Secondly, as the DJ test was performed on the sledge, the subjects landed in a well-secured position that did not require any high bipedal balance control. Finally, the duration of the MVC and DJ tests, 3 and 0.6 s respectively, might have been too short for contralateral changes to take place. As suggested by Zijdwind and Kernell (2001) and recently confirmed by Rathey et al. (2006), it seems that “cross-over” of central fatigue is more likely to occur in moderate but sustained contractions than in maximal but brief motor task. Supporting this hypothesis, a continuous series of 15 sub-maximal rebound task used by Regueme et al. (2007b) revealed positive correlations between the fatigue-induced changes in muscle activity of the exercised and non-exercised legs. This task lasted 15 sec and required a high visual attention to remain within the upper and lower rebound targets via visual feedbacks. This precise rebound task, which required high visual



attention and effort levels for at least 15 s, might have favored the occurrence of contralateral neural adjustments. Finally, the observation of restricted contralateral effects at day 2 is likely to support the involvement of central structures associated with pain (Ploghaus et al. 1999).

Based on these results it seems that acute muscle disorders induced by SSC exercises do not lead to contralateral changes in performance in either single or intermittent maximal motor tasks. Some evidence of cross-over of central fatigue exists (Regueme et al. 2007b), however, thus emphasizing the needs to be explored further.



**Fig. 70. Maximal drop jump test. Left panel:** D2 versus PRE group mean absolute Fz and SOL EMG recordings from the fatigued and non-fatigued legs in the unilateral (a) and bilateral (b) DJ tests. **Right panel:** For each testing condition, the D2 relative changes (delta % PRE) ( $\pm$  SD) of the maximal rebound performance. The D2 relative EMG changes (delta % PRE) are presented successively for the preactivation (a), stretch–reflex M1 response (b), and the braking phase (c). (■, fatigued leg; □, non-fatigued leg; and ▒ bilateral data). \*\* $P < 0.01$ , \*\*\* $P < 0.001$ , significantly different from the pre-fatigue (PRE) normalized EMG values (% MVC PRE). # $P < 0.05$ , ## $P < 0.01$ , ### $P < 0.001$ , significant inter-leg difference. SOL, soleus; DJ, maximal drop jump; MVC, maximal isometric voluntary contraction. (From Regueme et al. 2007b)

## **2.7. Potential mechanisms related to SSC-induced neuromuscular changes**

The bimodal nature of fatigue recovery and its associated DOMS sensation can be considered as characteristics of exhaustive SSC exercise. However, the challenge lies in finding the origins and underlying mechanisms of this process. As it has been discussed in the previous paragraphs, SSC exercises load the neuromuscular system in a more complex way than any isolated form of muscle actions.

Including both eccentric and concentric muscle actions, it is evident that the SSC exercise stresses the involved skeletal muscles metabolically, but the magnitude of the metabolic loading is dependent on the velocity of stretch (eccentric), and the coupling time between stretch and shortening (concentric phase). In the present review, we would like to emphasise that, in addition to metabolic effects SSC fatigue includes structural perturbations of the muscle-tendon complex that require several days to recover. SSC is indeed characterised by high impact forces that are often repeated over long durations. These impact loads (braking forces) are accompanied by loading of the reflexes that are involved in both length-feedback (muscle spindles) and force-feedback (Golgi tendon organs) control of muscular forces. Finally, the present review of the SSC literature demonstrates that exhaustive SSC exercises have large functional effects that are both “testing time” and “testing task” dependent.

SSC fatigue may thus be considered as a model to examine the potential neural adjustments to short-term and reversible structural changes of the muscle-tendon complex. As in the case of isolated forms of muscle action we are dealing with both central and peripheral neural adjustments to fatigue that may be thought to differ between the acute (< 2 h) and the delayed recovery phases. The delayed and perhaps the more relevant recovery phase is, in particular, usually related to the natural time course of inflammatory and/or remodelling processes related to muscle damage. This phase usually lasts for a few days and is associated with delayed-onset muscle soreness (DOMS). This may explain why DOMS has been repeatedly considered as an indirect marker of either muscle damage and/or inflammation. It is noticeable; however, that muscle soreness disappears before functional recovery is complete. This observation, in particular, raises interest regarding the potential parameters that could interact during the delayed recovery period. On the other hand, the entire succession of events may be considered accordingly to the “remodelling model” recently proposed by Malm et al. (2000). This model questions the necessity for an inflammatory process in the regeneration of the damaged muscle, but this is still not clearly established in the literature.

As the bimodality concept particularly applies to exercise-induced changes in stretch-reflex response, we will then concentrate on the potential “alteration in the fusimotor-muscle spindle function”. Considering the combined testing time and testing task effects on the neural strategy, special emphasis will be put on the potential influence of sensory inputs at spinal and motor cortex levels.

### ***2.7.1. Metabolic loading***

From the bimodal nature of SSC fatigue recovery curve (Fig. 46), it is obvious that the immediate reduction of performance deals with the metabolic fatigue. That is, it is thought to be caused by the accumulation or depletion of metabolites or ions, either intra- or extracellularly, potentially combined with the effects of heat stress and dehydration.

This type of fatigue has been discussed extensively as a basic fatigue concept for isolated forms of muscle function (usually either isometric or concentric) (for references, see Vandenberg 2004, Allen et al. 2008). For example, in fatiguing exercise, conduction velocity slows, and the amplitude of the action potential can decrease, with depolarisation of the muscle fibre membrane through the accumulation of extracellular potassium (Sjøgaard et al. 1996). After exhaustive dynamic exercise, Marcos and Ribas (1995) reported potassium recovery as a very short-lasting (about 5 min) phenomenon. Interestingly, it has been recently reported that the extracellular accumulation of potassium would be of lesser importance than indicated by previous fatigue studies on isolated muscles (Nielsen et al. 2001, Nielsen and Paoli 2007). However, as the concentration of Na<sup>+</sup>-K<sup>+</sup> pumps is reported to increase with training (Nielsen and Clausen 2000), this parameter could remain of importance in untrained subjects (Millet and Lepers 2004). Although metabolic end products may thus explain part of the acute neuromuscular changes after SSC exercises (see for instance the comparison between the acute effects of intermittent versus continuous forms of SSC exercises in Fig. 48), they cannot be used to explain those observed during the delayed recovery phase. Furthermore, the potential delayed influence of low glycogen stores and blood glucose levels after prolonged SSC exercises are not likely to explain the similar functional defects reported after short and intensive SSC exercises (Nicol et al. 2003).

When considering its potential central influence (for a review, see Nybo and Secher 2004), a deficit in voluntary activation has been reported to be exacerbated by conditions such as a decline in the levels of blood glucose and exercise in hot environments (Nybo and Nielsen 2001; Nybo 2003; Todd et al. 2005). In our own study (Ftaiti et al. 2001), in which hyperthermia ( $40 \pm 0.3$  °C) and dehydration ( $2 \pm 0.5\%$  of body mass loss) was induced by a

40 min treadmill run at 65% of their maximal aerobic velocity while wearing a tracksuit covered with an impermeable jacket and pants, clear decrements were observed in maximal knee extension torque and EMG activity both in isometric and at slow isokinetic velocity ( $60^{\circ} \cdot s^{-1}$ ). However, no differences in these parameters were found at  $240^{\circ} \cdot s^{-1}$ . The EMG patterns remained also remarkable stable during the treadmill run. This would suggest that high internal body temperature per se could limit the production of high force levels more than the SSC performance. Additional evidence for a limited metabolic influence comes from the study of Millet and Lepers (2004) who compared the acute effects of a marathon skiing and a 30 km running of similar duration. The prolonged run led indeed to a large drop in MVC as well as to a significant reduction in activation, but this was not the case after skiing.

### ***2.7.2. Structural changes***

The overall process may be considered on the one hand as following the classical “Armstrong’s model” (Armstrong 1990, 1991) that differentiated four subsequent phases for exercise-induced muscle damage and ensuing inflammation: the initial-, the autogenetic-, the phagocytic- and the regenerative phase. As the exercise-induced ultrastructural muscle damage is reversible, these phases include the whole process from initial damage to repair. It is rather obvious that these different phases can have some acute and long-lasting consequences directly on the contractile properties of the muscle itself and, on the other hand, on its neural control mechanisms. This obviously may be thought to contribute to the SSC-induced functional impairments. In the next few paragraphs, we will attempt to describe the delayed onset muscle soreness (DOMS) sensation and the four phases of Armstrong’s model.

#### *2.7.2.1. D.O.M.S. as an indirect indicator of muscle damage*

Exercise induced muscle soreness has been suggested, as early as in 1902 by Hough (Hough 1902), to result in its delayed phase from "some sort of rupture within the muscle". Hill (1951) suggested that soreness is due to mechanical injury, distributed microscopically throughout the muscle. As referred to by Asmussen (1956), Bøje (1955) was inclined to believe that the pains are located in the intramuscular connective tissues. Supporting the mechanical source of peripheral fatigue, intense or long-lasting SSC exercise is typically associated with D.O.M.S. This delayed type of muscle soreness has been extensively studied in case of eccentric fatigue (for a review, see Cheung 2003), which has helped development of

several mechanistic explanations for the SSC type fatigue and damage has been evidenced directly from histological measurements (see 2.7.1.2.).

At present it is known, however, that neither the degree nor the timing of ultrastructural damage correlates well with the respective changes in D.O.M.S. sensation (Howell et al. 1993; Newham et al. 1983b). Similarly, even though muscle stiffness has been reported to increase with a delayed time course that parallels that of muscle soreness (Jones et al. 1987), stiffness may peak earlier and it often lasts much longer (Dousset et al. 2007; Howell et al. 1993). In case of prolonged SSC exercises, D.O.M.S. may also be considered as attributable to reactive oxygen species (ROS). As recently reported by Close et al. (2006), however, the use of ascorbic acid supplementation to attenuate ROS did not affect D.O.M.S. and was even found to delay the recovery of muscle function.

It is noteworthy that experimental muscle pain induced by intramuscular injections of algogenic substances such as bradykinin, serotonin and substance P did not reveal either any dose-relation with the induced pain intensity (Babenko et al. 1999). This has been attributed to the fact that when peripheral tissues are damaged, the sensation of pain in response to a given stimulus is enhanced. This phenomenon, termed "hyperalgesia", may involve lowering of threshold of nociceptors by the presence of locally released chemicals such as substance P (for a review see Jessel and Kelly 1991, Millan 1999). Hyperalgesia occurs indeed first at the site of tissue damage before spreading throughout other compartments (Bobbert et al. 1986, Fields 1987, Howell et al. 1993). Finally, Malm et al. (2004) recently questioned the use of D.O.M.S. as an indirect marker of skeletal muscle inflammation. Exercise was thought to induce D.O.M.S. by activating inflammatory factors (T cells and/or neutrophils) present in the epimysium before exercise.

It is widely accepted also that D.O.M.S. will only occur after the first few bouts of an exercise programme and therefore training acts in a preventive fashion to reduce muscle soreness. When a given exercise is indeed repeated after complete recovery of the initial one, e.g. one to three weeks later, the subsequent exercise-induced D.O.M.S. and functional impairments are usually reduced (e.g. Byrnes et al. 1985; Brockett et al. 2001). This adaptative process, known as the « repeated bout effect » (e.g. Ebbeling and Clarkson 1990; Nosaka et al. 1995), would rely on both structural and neural adaptations (for a review, see McHugh 2003). As illustrated by the lower graph of Fig. 63, however, it is noteworthy that in most studies with short interval time (3-5 days) the performance parameters recorded at the initiation of a given exercise bout were still close to their lowest values from the previous one (Ebbeling and Clarkson 1990; Nosaka and Clarkson 1995; Chen 2003). Similarly, the DOMS

markers were still very high. One may therefore question the validity of comparing the different bout effects. The reported absence of delaying effect on the recovering process (Chen 2003) might be questioned as well. In the study shown in Fig. 63, an exhaustive SSC rebound exercise was repeated three times at a 5 day interval. The normalized H reflex response (H/M) did not recover for any of the subjects along the 15-day follow-up period (Nicol et al. 1996a). Similarly, Ebbeling and Clarkson (1990) reported a slower recovery of the maximal isometric strength when the second bout was repeated at a 5 *versus* 14 day interval. These observations differ from the on-going recovering process repeated for eccentric exercises repeated at a 3 days interval (Nosaka and Clarkson 1995; Chen 2003)

More generally, our own studies emphasize the danger associated to the D.O.M.S. disappearance one to several days prior to the complete functional recovery (for a review, see Nicol et al. 2006). As illustrated by our alpine skiing study (Strojnik and Komi 2000), subjects are becoming more cautious when D.O.M.S. develops, but they rapidly come back to their original behaviour (e.g. daily skiing distance) when D.O.M.S. disappears. At that time, however, functional tests are still revealing deficits of 20% in maximal force and voluntary activation.

#### 2.7.2.2. *Extra- and intrafusal muscle fibre damage*

In Armstrong's model (Armstrong 1990; Armstrong et al. 1991), the "*initial stage*" includes events that trigger the whole process of further muscle damage and repair. Damage has been evidenced directly from histological measurements as well as indirectly from reductions in strength and range of motion, as increased plasma concentration of soluble muscle proteins (for reviews, see e.g. Clarkson and Newham 1995; Fridén and Lieber 2001a). Sorichter et al. (1999) have summarized the literature on the identification and usefulness of indirect markers of muscle injury and muscle membrane integrity. The most commonly reported indirect indicators include myoglobin (Mb), lactate dehydrogenase (LDH) and creatine kinase (CK). Assays using myofibrillar-bound proteins, such as myosin heavy chain (MHC) fragments of slow-twitch skeletal muscle myosin and calcium regulatory proteins such as the troponins (sTnI) have also been investigated as markers of the disruptions within the contractile unit (Mair et al. 1992; Sorichter et al. 1997, 2001). However, these indirect indicators of muscle damage do not accurately reflect the magnitude of muscle damage. This is particularly true for serum creatine kinase activity (Evans and Cannon 1991; Fielding et al. 1993; Komulainen et al. 1995; Fridén and Lieber 2001b).

In pure eccentric work, much evidence exists that the initial local damage results from mechanical rather than from metabolic mechanisms (Lieber and Fridén 1993; Warren et al. 1993; Brooks et al. 1995). One of the mechanisms proposed for the initial event is based on the “popping sarcomere hypothesis” (Morgan 1990). Briefly, the basis mechanism is the region of instability on the descending limb of the sarcomere length-tension curve. It is suggested that when the myofibrils of a muscle fiber are stretched while contracting, some sarcomere resist the stretch more than others. As a consequence, weaker sarcomeres take up most of the stretch. It is likely that repeated lengthening actions lead to increased number of disrupted sarcomeres in which the myofilaments fail to re-interdigitate. This theory has got recent support through its direct demonstration in a population of living muscle fibers (Patel et al. 2004). Several other mechanical factors have been reported by animal studies to affect the magnitude of contraction-induced injury, such as peak force (Gosselin and Burton 2002), average force (Brooks et al. 1995), work during the stretch (Hunter and Faulkner 1997), strain defined as the relative change in length (Lieber and Fridén 1993, 1999), and fiber length (Hunter and Faulkner 1997; Cutlip et al. 2004; Gosselin and Burton 2002). In an alternative proposal for the initial stage of fatigue, Warren et al. (2001) supported the hypothesis of an excitation-contraction (E-C) uncoupling. Following strenuous exercises, gross focal dilation of sarcoplasmic reticulum would be accompanied by depression in the rate of  $\text{Ca}^{2+}$  uptake and diminished  $\text{Ca}^{2+}$  release, resulting in an increase of intracellular free calcium concentration  $[\text{Ca}^{2+}]_i$  (Byrd 1992). The supporting evidence for this hypothesis was based on intracellular  $\text{Ca}^{2+}$  measurements, but it has been argued since then that the increase in  $\text{Ca}^{2+}$  is secondary to mechanical changes in the fiber (Yeung and Allen 2004). Nowadays, it is suggested that the associated stresses applied to membranous structures lead to the opening of stretch-activated cation channels and produce an influx of  $\text{Na}^+$  and  $\text{Ca}^{2+}$  ions (Proske and Allen 2005). Nevertheless, an excitation-contraction uncoupling mechanism does not readily explain the unique post-eccentric shift of the length-tension relation of the muscle (Morgan et al. 2004; Proske and Allen 2005). The expected consequences of the intracellular increase in  $\text{Ca}^{2+}$  will be therefore presented in the subsequent stages of muscle degradation, and the excitation-contraction uncoupling hypothesis will be discussed in the neural aspects of SSC fatigue.

After SSC exercises, histological studies have reported direct evidence of extensive disorganisation and even disruption of the myofibrillar structures and intermediate filaments, leading to the classically observed Z-line streaming (Fridén et al. 1983, 1984; Waterman-Storer 1991). Indices of sarcolemmal disruption (Hikida et al. 1983; McBride et al. 2000) swelling and disruption of the sarcotubular system (Armstrong 1990; Fridén and Lieber 1996)

and swollen mitochondria (Warhol et al. 1985; Stauber 1989) as well as extracellular matrix injury (Myllyla et al. 1986; Han et al. 1999; Koskinen et al. 2001) have also been reported. In the case of prolonged SSC exercises, influence of free radicals has been suggested to occur as well (Margaritis et al. 1997), but their consequences on exercise-induced oxidative damage indices appear to be linked to subjects' adaptation state. Considering also the absence of decrements in peak twitch reported after very prolonged running (Millet et al. 2002, Place et al. 2004) and skiing exercises (Millet et al. 2003a), this will not be discussed in more details. With regard to the ultrastructural changes along the SSC recovery period, it is noteworthy that cytoskeletal and myofibrillar abnormalities are lower immediately after exercise, relative to those observed 2-3 d later (Newham et al. 1983a). At that time, normal muscle architecture is also reported as being lost from the areas adjacent to the damaged sarcomeres and fibres (Fridén et al. 1983; Newham et al. 1983a). After a marathon run, Hikida et al. (1983) demonstrated significant ultrastructural changes that peaked on days one and three, with some lasting until day seven. These observations give support to by the rapid functional recovery, usually observed 1-2 h after exercise, and followed by a secondary decline 1-2 d later. The resulting sarcomere and muscle fiber disruptions would then contribute to the common shift of the length-tension relationship towards longer muscle lengths after eccentric exercise (Komi and Rusko 1974, Wood et al. 1993, Jones et al. 1997). This shift is now considered as one of the most reliable indicators of muscle damage (Whitehead et al. 2001) and reported to contribute to the decline in force at the original optimum length (Wood et al. 1993, Brockett et al. 2001). There is no reason to doubt that this would also occur in the case of stretch-shortening cycle fatigue (Ishikawa et al. 2006a).

Evidence has also been presented that eccentric exercise is differently severe in muscles with different architectures (Lieber and Fridén 2000; Fridén and Lieber 2001a) and fiber type specific so that fast-twitch fibers would be more sensitive than slow-twitch fibers to eccentric-induced muscle damage (Fridén et al. 1983, 1988; Fridén and Lieber 2001a; Vijayan et al. 2001 Brockett et al. 2002). In addition to their structural differences, fast-twitch fibers possess a reduced oxidative capacity, which is believed to result during intensive exercises in failure of cross-bridges detachment, thus leading to an inhomogeneous resistance to stretch among sarcomeres and muscle fibers (Fridén and Lieber 1992). This would imply that intensive SSC exercise could favour the rapid development of sarcomere inhomogeneity, and enhance the damaging effect of the repeated impact loads. Our own findings using different fatiguing protocols give some support to the additional influence of metabolic failure. Considering the similarity of the functional failures induced by either 3 min of intensive

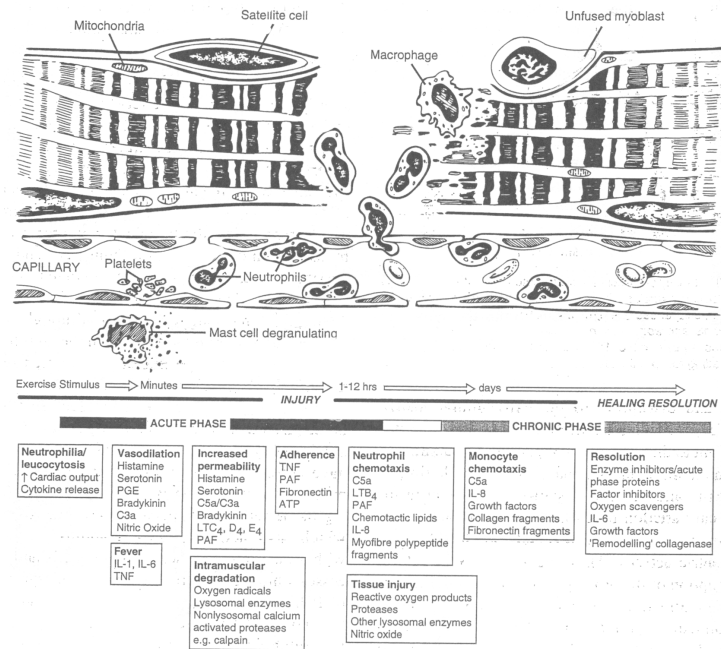


rebound exercise or a prolonged marathon run (Fig. 61), it is suggested that high exercise intensity (reflected by 10 vs. 4 mmol.l<sup>-1</sup> of blood lactate concentration) might favour the rapid development of inhomogeneous resistance to stretch and muscle damage in connection to the more intensive exercise (Nicol et al. 2003). This is expected to have enhanced the damaging effect of the high impact loads experienced during this rebound exercise. As illustrated by the protective effect of previous eccentric work (Komi and Buskirk 1972; Clarkson and Tremblay 1988; Nosaka et al. 2005), the training status of the subjects may also be considered as playing a role in the large individual variability observed in terms of exercise-induced muscle damage.

Finally, provided that mechanical coupling does exist between extra- and intrafusal fibres, this may give support to the possibility of disturbances in the intrafusal fibres themselves (Komi and Nicol 2000). Leading to reduced muscle spindle sensitivity, this could contribute to the immediate reductions of both stretch-reflex sensitivity and the electromyographic response (Nicol et al. 1996a). According to the animal study of Gregory et al. (2004), however, intrafusal fibres are not prone to damage of the kind seen in extrafusal fibres after eccentric work. After eccentric fatigue, however, Trappe et al. (2002) were able to show disruption of titin and nebulin structures in extrafusal fibers. Provided that the mechanical coupling truly exists between extra- and intrafusal fibers, this finding may provide evidence to keep the hypothesis of intrafusal fiber damage alive. Whether this notion applies to stretch-shortening cycle fatigue that involves much higher impact (stretch) loads than pure eccentric exercise remains to be seen.

#### *2.7.2.3. Muscle inflammation and/or remodelling*

In Armstrong's model (Armstrong 1990; Armstrong et al. 1991), the “*autogenetic stage*” corresponds to the first 3-4 h following the injury and marks the beginning of the degrading process of the membrane structures. The “*phagocytic stage*” is characterized by a typical inflammatory response in the tissues which may last for 2-4 days or more, with a peak around the 3<sup>rd</sup> day post-exercise. The “*regenerative stage*” begins on days 4-6 and reflects the regeneration of muscle fibers. In this line, muscular adaptation to exercises involving intense and/or unaccustomed eccentric muscle actions are usually explained by the classical “damage-inflammation-repair” pathway (MacIntyre et al. 1995; Tidball 1995; Chambers and McDermott 1996; Clarkson and Sayers 1999). As shown in Fig. 71, the inflammatory process may be sub-classified into acute and chronic phases.



**Fig. 71. Possible sequence of events involving inflammation in muscle injury.**

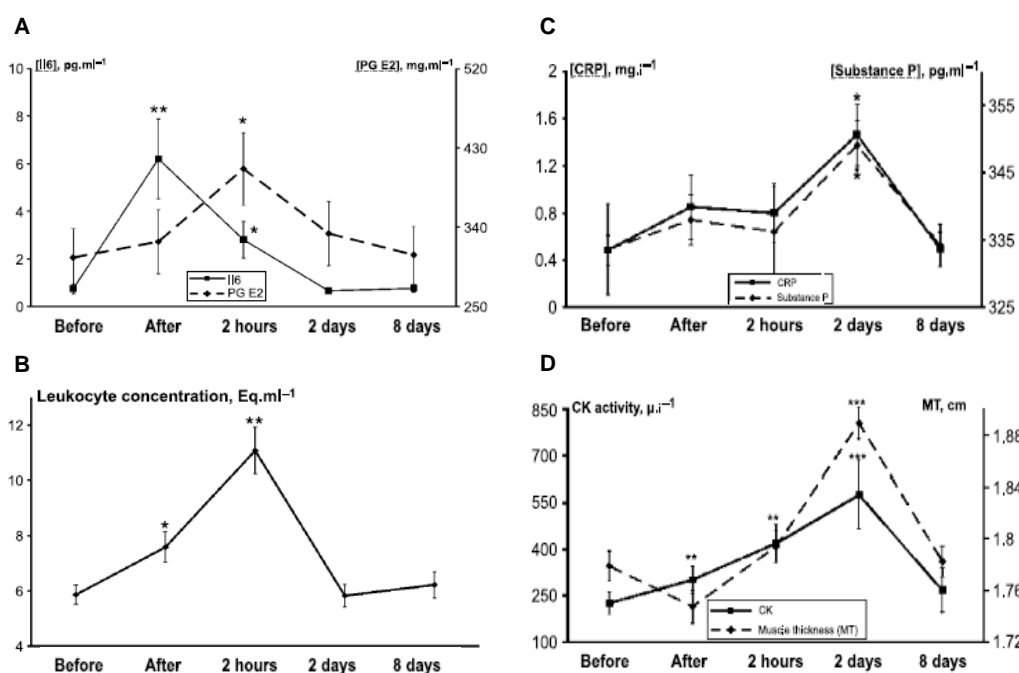
ATP = adenosine triphosphate; IL = interleukin; LT = leukotriene; PAF = platelet activating factor; PGE = prostaglandin E; TNF = tumour necrosis factor. (From MacIntyre et al. 1995)

The acute response begins with changes in the vascular wall structure, leading to structural and functional alterations of the basement membrane, migration of neutrophils to the site of injury, later followed by an accumulation of monocytes by chemotaxis and activation of satellite cells (Evans and Cannon 1991; Fantone 1993; Tidball 1995). Mobilization of neutrophils has been reported to be greater after eccentric- than after concentric-type exercises performed by the same subjects at similar levels of oxygen consumption (Smith et al. 1989). During inflammation, monocytes undergo morphological and functional differentiation, becoming macrophages. Once activated, neutrophils, monocytes and macrophages are capable of phagocytosis, provide a fresh supply of cytokine mediators and cytotoxic factors which may be partly responsible for amplifying and delaying the inflammation (Toumi and Best 2003; Toumi et al. 2006). Macrophages are indeed the prime sources of cytokines such as interleukin-1 (IL-1) and tumour necrosis factor (TNF) that alter endothelial permeability, leading to leucocyte infiltration and oedema.

On the other hand and as reviewed by Evans and Cannon (1991), macrophages secrete fibronectin and proteoglycans that help to stabilize the extracellular matrix, promote cell adhesion, and stimulate fibroblast proliferation and collagen synthesis by means of IL-1 $\beta$ . Elevated levels of IL-1 $\beta$  immunoreactivity in muscle tissue are reported up to 5 days after downhill running (Cannon et al. 1989; Fielding et al. 1993). Non-phagocytic macrophages

(ED2+) and fibroblasts are thought to produce IL-6 later (Cannon and St. Pierre 1998), which is probably related to repair of the tissue. Toumi et al. (2006) recently suggested that reducing neutrophil infiltration would not result only in reduced collateral damage, but also in an attenuated repair response. In this line, there is strong evidence that following exercise-induced muscle injury regeneration proceeds no further in the absence of macrophages (for a review see Carlson and Faulkner 1983; MacIntyre et al. 1995). In the study of Mishra et al. (1995), non-steroidal anti-inflammatory medication after muscle injury was also reported to result in short-term improvement but subsequent loss of muscle function.

Only a few SSC fatigue studies measured functional changes as well as indirect markers of the development of an inflammatory reaction (e.g. Dousset et al. 2007). The results of this study indicated an immediate increase only in interleukin 6 (IL-6) and 2h peaks of prostaglandin E2 (PG E2) and leucocyte concentrations (Fig. 72A and B), with delayed changes in increases in C reactive protein (CRP), substance P concentrations and serum creatine kinase activity (S-CK), and also in muscle thickness (Fig. 72C and D). These changes occurred concomitantly with bimodal decrements in maximal voluntary force, voluntary activation, H reflex/M-wave ratio and stretch reflex EMG amplitude.



**Fig. 72.** Changes in blood concentrations of indirect markers of muscle damage and inflammation before exhaustive SSC exercise and along 8 days of its recovery period. (A) Interleukin 6 (IL-6, solid line) and prostaglandin E2 (PG E2, dashed line). (B) Leukocyte concentration (C) reactive protein (CRP), substance P concentrations (D) Serum creatine kinase activity (S-CK) and muscle thickness (MT)  
\*P < 0.05; \*\*P < 0.01: Significant changes between pre-trial value and other values.

The validity of the inflammatory model after eccentric muscle actions has recently been challenged, however, as recent immunological studies have produced evidence for a remodelling theory without preceding muscle inflammation (Pedersen and Toft 2000; Malm et al. 2000; Malm et al. 2004; Yu et al. 2002, 2003, 2004). This theory argues that exercise-induced disruption of the cytoskeleton is a sign of remodelling. For instance, the frequently reported Z-band streaming (e.g. Fielding et al. 1993) is considered as a desmin re-synthesis (Yu et al. 2002). The results of Malm et al. (2000) demonstrated that with respect to infiltrating neutrophils and macrophages, satellite cell activation and IL-1 $\beta$  detection, eccentric cycling exercise and multiple biopsies cause similar changes in adult human skeletal muscle. According to these authors, if the presence of leukocytes in human skeletal muscle after exercise is established, it could reflect a functional role of leukocytes in muscle adaptation rather than degeneration. As the question remains open, we will now concentrate on the way the different phases of Armstrong's model may apply to the SSC type fatigue.

#### *The “autogenetic stage”*

The additional structural degradation associated to the “*autogenetic stage*” is well in agreement with the secondary functional decline reported after exhaustive SSC exercises. Armstrong's model predicts that exercise-induced damage of the sarcolemma, T-tubule system and sarcoplasmic reticulum (SR) would give rise to a loss of calcium ( $\text{Ca}^{2+}$ ) homeostasis. Following strenuous exercises, gross focal dilation of SR have been reported to be accompanied by depression in the rate of  $\text{Ca}^{2+}$  uptake and diminished  $\text{Ca}^{2+}$  release, resulting in an increase of intracellular free calcium concentration  $[\text{Ca}^{2+}]_i$  (Byrd, 1992). According to the reviews of Ebbeling and Clarkson (1989) and Armstrong (1990), alteration of resting  $[\text{Ca}^{2+}]_i$  in the injured fibers could result in enhanced calcium-activated endogenous proteases (e.g. calpain) causing further muscle injury. Calpain leads to specific hydrolysis of cytoskeletal proteins such as desmin, but not actin and myosin, whereas other calcium-stimulated proteases act directly on the Z-lines (Ebbeling and Clarkson 1989). Interestingly, Belcastro (1993) demonstrated an increased affinity of calpain for calcium after exercise, suggesting that a given amount of damage could then occur at a lower calcium concentration. Quantitative measurements of resting  $[\text{Ca}^{2+}]_i$  in skeletal muscles following acute or long-term downhill running exercise have been performed in rats (Lynch et al. 1997). The results indicated a significant increase in resting  $[\text{Ca}^{2+}]_i$  which coincided with the functional decrement. Further confirmation of the role of the calcium-calpain pathway in the acute cytoskeletal damage and functional deficits after eccentric actions has been recently obtained

by reducing extracellular calcium to zero as well as by applying calpain inhibitor leupeptin (Zhang et al., 2008).

It should be mentioned, however, that Lowe et al. (1994) reported no immediate changes in  $[Ca^{2+}]_i$  and that Lynch et al. (1997) observed changes in both  $[Ca^{2+}]_i$  and muscle function 48 h, but not 24 h post exercise. According to Lynch et al. (1997), these combined results would support the hypothesis that muscles subjected to exercises involving eccentric muscle actions could buffer changes in  $[Ca^{2+}]_i$  until the phagocytic stage of the inflammatory process. In this line, a loss of force generating proteins (myosin heavy chain and actin) by mouse EDL muscle injured by eccentric contractions was not found to contribute to the initial strength loss, but to explain the prolonged time for recovery in muscle strength (Ingalls et al. 1998).

### ***The phagocytic stage***

Supporting the "***phagocytic stage***" occurrence, several studies have reported greater myofibrillar damage 2 days following eccentric exercise than immediately post-exercise in both humans (Fridén et al. 1981; Newham et al. 1983a) and animals (Geronilla et al. 2003). At that time, normal muscle architecture is also reported as being lost from the areas adjacent to the damaged sarcomeres and fibres. After a marathon run, Hikida et al. (1983) demonstrated the presence of significant ultrastructural changes that were found to peak on days 1 and 3, some of them persisting on day 7. These observations give support to the secondary functional deficits observed 1-2 days after exhaustive SSC exercise. Myofiber degeneration and necrosis accompanied with interstitial oedema and infiltration of inflammatory cells have been repeatedly reported by animal studies on day one to three after eccentric (McCully and Faulkner 1986; Lowe et al. 1995; Lieber et al. 2002) or SSC exercise (Geronilla et al. 2003). In the SSC animal study of Geronilla et al. (2003), the pathology scores for myofiber degeneration and necrosis were demonstrated to increase with increasing numbers of SSC and to be significantly greater at 48h than at 30 min post exercise.

Among indirect indicators of muscle inflammation, the relative changes in serum creatine kinase have been frequently used by SSC fatigue studies, including most of our own, to determine the existence (but not the importance and exact timing) of tissue inflammation. According to Malm (2000), however, serum creatine kinase would not even be related to muscle inflammation. On the other hand, serum creatine kinase activity appears to be an indicator of either fast- or slow-recovering subjects (Nicol et al. 2003). This is in line with our earlier findings relative to neuromuscular changes (Kyröläinen et al. 1998; Avela et al. 1999b; Horita et al. 1999; Nicol et al. 2003). Other indirect indicators of exercise-induced

inflammation include delayed changes in muscle stiffness and thickness (e.g. Fig. 73), as well as delayed-onset muscle soreness (Fig. 47).

### *The “regenerative stage”*

This stage is usually characterized by a functional recovery starting on days 4–6 after exhaustive SSC exercises. In the animal study of Lowe et al. (1995), protein synthesis rates was found to increase approximately 48 h post injury, remaining elevated by 83 % five days post-injury. By days 10–14, muscle protein degradation and synthesis rates had returned to normal, and phagocytic infiltration was not detected. However, muscle mass, protein content, and absolute force production were still lower than before. Based on the review of Warren et al. (2001), contractile protein content and strength would recover in parallel towards the end of the recovery (14–28<sup>th</sup> days). Similar delays of recovery (Hikida et al. 1983), but also much longer ones (Warhol et al. 1985; Howell et al. 1993) have been reported in humans following eccentric-induced muscle damage.

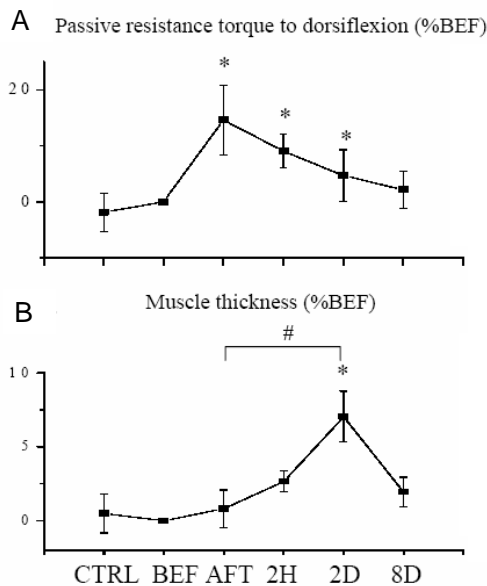
According to the classical “damage-inflammation-repair” theory, regeneration of mature skeletal muscle recapitulates closely foetal myogenesis (Fantone 1993; Tidball 1995). A key element in the initiation of the events leading to recovery of muscle fibers to injuries is the activation of satellite cells. Once activated, satellite cells divide mitotically to form myoblasts, with subsequent fusion of the myoblasts to form myotubes (Carlson and Faulkner 1983; Darr and Schultz 1987). The myotubes become immature fibers and ultimately differentiated muscle fibers. According to Rantanen et al. (1995), the stem satellite cells would follow the entire sequence of events whereas the committed satellite cells would be ready for immediate differentiation. Subsequent proliferation of satellite cells has been reported for instance in rats after downhill running (Darr and Schultz 1987). The satellite cells are reported as self-renewing, so that a residual pool of these cells is re-established after each discrete episode of muscle injury, and therefore capable of supporting additional rounds of regeneration (Hawke 2005). According to Kadi et al. (2004), however, the pool of satellite cells would decrease with ageing.

According to Malm et al. (2000, 2004), however, muscle regeneration after eccentric exercise would not involve satellite cell activation. In both of these studies, expression of CD56<sup>+</sup> was used as a marker for activated satellite cells and regenerating/degenerating muscle fibers in humans (Illa et al. 1992). These markers were found to increase within 24h after the trauma inflicted by the needle biopsy, but not after the eccentric exercise.

#### 2.7.2.4. *Changes in the muscle-tendon unit compliance*

In addition to possible muscle damage, repeated SSC muscle action can also modify the mechanical properties of the muscle tendon unit. It is well known that, with pure eccentric fatigue, muscle volume increases after exercise (Howell et al. 1993, Murayama et al. 2000). This can be caused, among other things, by increased internal fluid pressure due to swelling (Crenshaw et al. 1994). However, swelling and stiffness do not change in parallel (Chleboun et al. 1998), and Ishikawa et al. (2006a) demonstrated this using ultrasound scanning following fatiguing exercise (Fig. 73). In this study peaks for passive resistance to stretch and muscle thickness occurred immediately after, and 2 days after exercise, respectively. Note that the passive resistance to stretch remained elevated, even 2 days after exercise.

This modification and its direct effects on the contractile machinery are difficult to identify in active fatigue conditions. Therefore, passive stretching protocols have frequently been used for this purpose. Several experiments have demonstrated with a variety of protocols that passive dynamic or static stretching of a human skeletal muscle can impair its force generating capacity (Taylor et al. 1990; Avela et al. 1999b, 2001; Fowles et al. 2000). The cause of this contractile impairment has often been attributed to the increased compliance of the muscle tendon unit induced by the stretch. According to Taylor et al. (1990) the mechanical changes of the muscle-tendon unit are time and history dependent because of the viscoelastic nature of the tissue. Two different responses in this regard have been suggested. The first one is stress relaxation (Magnusson et al. 1995; Taylor et al. 1997), which is purely mechanical in nature and is mainly responsible for the reduced passive tension of the muscle-tendon unit over time. The second one is called plastic deformation, which means reorientation of the supporting connective and soft tissue-supporting tissue into more parallel arrays (Purslow 1989). This response is suggested to be more time dependent of the maintained tissue strain. Independently on the exact mechanism, an increased compliance of the muscle-tendon unit could then result in impaired force transfer from the muscle fibres via the tendon to the bone (Lieber et al. 1991), occurring very likely during the course of the SSC exercise and immediately after (early acute phase). This is illustrated in Fig. 74 by the reduction in passive stretch-resisting torque reported by Avela et al. (2004) immediately after 1h of continuous passive stretching. This is opposite to the delayed SSC recovery phase effect, which is characterized by an increased stiffness of the muscle-tendon unit (Avela et al. 1999b; Ishikawa et al. 2006a) (Fig. 73).

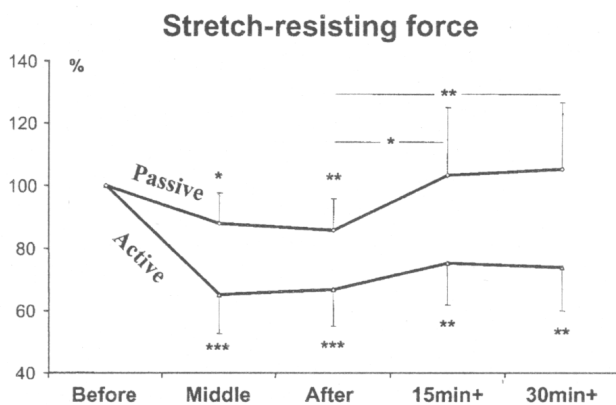


**Fig. 73. Relative changes during the 8-day follow-up period after exhaustive SSC exercise.**

**(A) Passive resistance torque of plantarflexors to dorsiflexion movement (relative to before).**

**(B) Changes in triceps surae muscle thickness.**

(CTRL: control day, BEF: before fatigue, AFT: immediately after fatigue, 2H: 2 h after fatigue; 2D: 2 days after fatigue, 8D: 8 day after fatigue). Statistical significance of the difference from BEF (asterisk symbol) and between the testing points (hash symbol) is expressed as  $P < 0.05$ . (Adapted from Ishikawa et al. 2006a)



**Fig. 74. The relative values of passive and active stretch-resisting forces between different conditions (mean and SD).**

\*\*\*  $P < 0.001$ , \*\*  $P < 0.01$  and \*  $P < 0.05$  refer to the statistical differences before, during and after stimulation or within the post-stimulation period, as marked by bars. (From Avela et al. 2004)

In the case of damaged elbow flexors due to eccentric exercise, delayed changes in stiffness have frequently been reported in association with a reduced ability to fully flex the joint (Clarkson et al. 1992). According to Whitehead et al. (2001), the rise in passive tension results from development of injury contractures in the damaged muscles. Ebbeling and Clarkson (1989) suggested there may be an abnormal accumulation of calcium inside the muscle cell, due either to a loss of sarcolemmal integrity or dysfunction of the sarcoplasmic reticulum. Increased muscle stiffness might be expected as well to result in an increased intrafusal passive tension, and subsequently in an increased resting level of spindle activity (Gregory et al. 1988). Increased stiffness might result also in increased response to stretch of the muscle spindle. This could have contributed to the overestimation reported at day 2 by Regueme et al. (2008) in a large dorsiflexed position when the fatigued muscles were stretched while holding the position (Fig. 67). Similarly, this could explain why some authors



(Kuitunen et al. 2004) demonstrated at 2h post exercise reflex amplitudes that were in some subjects higher than in the before fatigue measurements.

However, this possibility is not in agreement with the secondary reductions repeatedly observed in both EMG and mechanical reflex responses after SSC exercise (e.g. Nicol et al. 1996b; Horita et al. 1999; Avela et al. 1999b; Dousset et al. 2007). This emphasizes the need when discussing of the SSC fatigue effects on the stretch reflex response to take into consideration the influence of other afferent input. As reflected in Fig. 72 and Fig. 73B by the delayed increases in blood markers of inflammation (Dousset et al. 2007) and in muscle thickness (Ishikawa et al. 2006a), the nature of the delayed SSC recovery phase should favour the intervention of reflex adjustments of the neural activation.

### ***2.7.3. Potential sources of neural changes***

As suggested by the complexity of the SSC fatigue effects, the possible neural mechanisms involved in SSC-type fatiguing exercises are great in number. During the time course of fatiguing SSC exercise, a significant interaction has been demonstrated between pre-landing activation and kinematics, and the resulting post-landing stiffness adjustment (see 2.6.2.5.). These results emphasise the plasticity, as well as the efficacy of neural adjustments during the early parts of fatiguing exercise. They may also explain why, in some studies that used very moderate-intensity exercise, the fatigue effects were minimal. As shown in sections 2.6.2.1. and 2.6.2.2., the parallelism between activation and force changes may persist during the recovery period. This would indicate the existence of neural failure or adjustments to contractile deterioration that could operate at different levels of the activation pathways (Fig. 25). Modified from Bigland-Ritchie (1981) and Löscher (1996), these potential sites include the following: 1) excitatory input to supraspinal motor centers, 2) excitatory drive to  $\alpha$ -motoneurons, 3) modulation of interneuronal circuits, 4) motoneuron excitability, 5) peripheral reflex activity from small diameter afferents, and 6) muscle spindle activity. The potential sources of neural changes with SSC fatigue may be discussed in terms of “central” and “peripheral”, with “acute” and “delayed” consequences.

First, we would like to emphasize that the bimodality concept particularly applies to exercise-induced changes in stretch-reflex response. As this is one of the major topics of our own research work since 1991, we will thus concentrate on the potential “alteration in the fusimotor-muscle spindle function”.

Secondly, the manifestation and the contribution of the central and reflex neural adjustments to SSC fatigue are clearly shown to vary depending on the testing task (see section 2.5.3.). The variation in neural adjustments has been classically addressed to several mechanisms; 1) to compensate for the contractile failure (Gollhofer et al. 1987b), 2) to optimize the neural drive in proportion to the contractile failure (also known as “muscle wisdom”) (Bigland-Ritchie et al. 1983a) and 3) to induce contractile failure with inadequate neural drive (Gandevia et al. 1995). It is not very well known why neural drive itself should be a limiting factor for the force output of the muscle, but this has been repeatedly observed after intense or prolonged running and jumping exercises (see section 2.6.2.3.).

In such situations, inadequate neural drive can be considered as an attempt of the neuromuscular system to protect the muscle-tendon unit from additional damage (Horita 2000, Strojnik and Komi 2000, Strojnik et al. 2001, Verin et al. 2004). If the result of these neural adjustments is reduced neural drive, regardless of the mechanism, development of central fatigue exists. In the case of SSC fatigue, the recent use of transcranial magnetic stimulation and corticospinal tract stimulation emphasizes the potential influence of fatigue-sensitive muscle afferents (namely group III and IV) at spinal and motor cortex levels (Martin et al. 2006; Taylor and Gandevia 2001). The characteristics and expected influence of these sensory afferents will thus be developed as this theory might be of particular interest for improving the understanding of the delayed SSC recovery phase.

#### ***2.7.3.1. Specific alteration in the fusimotor-muscle spindle function***

The monosynaptic reflex loop originating from muscle spindle is well known to be under fusimotor control ( $\gamma$ -loop) (Burke et al. 1979). In case of muscle fatigue, various spinal and /or peripheral feedback mechanisms may be suggested to occur (Gandevia 2001). In general, these mechanisms can be related to either presynaptic or postsynaptic inhibitory effects coming from decreased net facilitation (disfacilitation) of the Ia-afferents originating from the muscle spindle or from different feedback sources. Some direct evidence comes from microneurographic recordings that reflex facilitatory influence provided by muscle afferents indeed declines as fatigue develops (Bongiovanni and Hagbarth 1990). Even though the hypothesis of Ia afferent supported reduction in motor unit firing rates have been lately challenged (Gandevia 2001), it is likely that since especially the eccentric (braking) part of the SSC muscle action induces a powerful loading of the muscle spindles, continuous SSC

loading can modify the  $\gamma$ -loop system. Unfortunately, owing to technical difficulties no direct microneurographic recordings have so far been in humans during SSC fatigue.

Although the exact mechanism inducing the reduced Ia-afferent activity has not yet been thoroughly explained, three major possibilities have been presented: 1) withdrawal of the fusimotor support to the muscle spindles (Bongiovanni and Hagbarth 1990; Hagbarth et al. 1986) and/or 2) intrafusal fibre fatigue itself (Emonet-Dénand and Laporte 1974) and/or 3) mechanical unloading of the muscle spindle (Fowles et al. 2000; Avela et al. 2004). Methodologically it is very difficult to separate the effect of fusimotor support to muscle spindles from the effects of muscle spindles themselves in fatigue. In some SSC fatigue experiments listed in Table 2, large acute and/or delayed reductions in stretch-reflex responses have been measured in both passive and active conditions (e.g. Nicol et al. 1996b; Avela et al. 1999a, 1999b; Kuitunen et al. 2004; Regueme et al. 2005a, 2005c, 2007b; Dousset et al. 2007). At present, however, it can only be hypothesized that both direct and indirect fatigue effects on the fusimotor-muscle spindle system exist.

Direct influences include both metabolic and mechanical factors. Bongiovanni and Hagbarth (1990) were able to show that metabolic fatigue can influence the fusimotor support to the muscle spindles. In addition, they suggested also that there is a possibility for some more direct fatigue effects on the intrafusal fibres themselves. The theory of intrafusal fatigue relies on a fatigue-induced decline in intrafusal contraction force, which could then reduce the afferent discharge. However, direct fatigue effects on the intrafusal fibres are scarce. Some signs of metabolic fatigue in intrafusal fibres have been observed following prolonged stimulation of static  $\gamma$ -axons in cat (Decorte et al. 1984), through the effect of  $\text{NH}_3$  and  $\text{CO}_2$  on the sensory ending of cat muscle spindles (Fukami 1988) or during prolonged swimming of mice (Yoshimura et al. 1996).

With regard to the possibility of mechanical changes in the intrafusal fibres, one can assume that the SSC fatigue can potentially influence the intrafusal fibre force. As previously mentioned, the hypothesis of direct intrafusal damage is still questionable (see 2.7.1.2.) whereas there is some evidence of fatigue-induced changes in the muscle-tendon compliance (see 2.7.1.4.). Some signs of the latter influence on the muscle spindle response have been reported by Avela et al. (2004) who showed clear indications of reduced stretch-reflex EMG responses immediately after 1h of continuous passive stretching. Based on the simultaneously measured reduction in fascicle length, their final interpretation was that increased compliance of the aponeurosis-tendon system could have resulted in reduced mechanical response of the muscle spindles, thus leading to reduced Ia-afferent activity and, finally, to disfacilitation of

the  $\alpha$ -motoneuron pool. In this line, Ishikawa et al. (2006a) demonstrated an increased compliance of the tendinous tissue when measured in an MVC test performed 2h after an exhaustive rebound exercise. The passive fascicle length was, however, found to be longer immediately after the SSC exercise as well as 2h and 2 days later. As reflected by the associated delayed peak of muscle stiffness at day 2 (Fig. 73A), increased internal fluid pressure due to swelling may be suggested to have occurred (Crenshaw et al. 1994; Nosaka and Clarkson 1996). This gives some support to the potential influence of mechanical and metabo-sensitive muscle afferents.

### ***2.7.3.2. Reflex adjustments of the neural activation: special emphasis on group III and IV afferents***

It was originally suggested by Asmussen and Mazin (1978) and later supported by Bigland-Ritchie et al. (1986) that reduction in the neural activation could depend on some reflex response from the contracting muscle itself. This hypothesis has been later challenged by Löscher et al. (1996) for sustained isometric contractions. The nature of SSC fatigue could, however, favour the intervention of such mechanisms. During the delayed phase of the recovery, structural and chemical changes associated with induced damage and inflammation should, in particular, have logical consequences on the afferent sensory pathways, and consequently on the efferent activities.

#### *Characteristics of group III and IV afferents*

The afferents most likely to change during fatiguing exercise, and thus contribute to central fatigue, are those in the muscle. These include muscle spindle afferents (group Ia and II), Golgi tendon organ afferents (group Ib), group II and III afferents (mechanically sensitive), and groups III and IV afferents (sensitive to non-noxious and noxious chemical and metabolic events). It is of interest for the present discussion on the functional effects of SSC-induced muscle damage that small diameter, group III and IV, muscle afferents could be likely candidates for such influences. As previously mentioned also (see section 2.7.1.1.), part of these receptors is of nociceptive type and very likely to contribute to the D.O.M.S. sensation.

These free nerve endings are particularly dense in the regions of connective tissues, but also between intra- and extrafusal muscle fibers as well as near blood vessels, in the Golgi tendon organs and at the myotendinous junction (Stacey, 1969; Kaufman et al., 1987). These muscle afferents are known to be mostly polymodal (Kaufman and Rybicki 1987), and

sensitive to several parameters associated with either metabolic fatigue or muscle injury (Kniffki et al. 1978; Rotto and Kaufmann 1988). In the acute phase of SSC fatigue, extracellular increases in potassium, phosphate, and lactic acid may constitute stimuli of the muscle metaboceptors (for a review see Decherchi and Dousset 2003). According to the review of McMahon and Koltzenburg (1990), unmyelinated primary sensory neurones are particularly responsive to the long-lasting changes that can occur with slow tissue perturbations such as inflammation. In case of muscle damage, group III and/or IV metaboceptors are indeed activated by the released biochemical substances, such as bradykinin (Mense and Meyer 1988) and products of cyclooxygenase activation (Kniffki et al. 1978; Rotto and Kaufman 1988, Kaufman et al. 2002). Additional stimuli, such as increases in the intramuscular pressure may also take place during the inflammatory process (Myatani et al. 2000, 2004; Ge anf Khalsa 2003). Once activated, the nociceptors release also neuropeptides, which cause vasodilatation, oedema and release of histamine and substance P (Cuesta et al. 1999). These processes lead then to a further and long-lasting activation of some of the sensory endings (Fields, 1987). In humans, the mean conduction velocity for group III and IV fibers have been reported by Simone et al. (1994) to vary within the following ranges: (3.1 – 13.5) and (0.6 – 1.2) m.s<sup>-1</sup>.

Only part of these receptors is of nociceptive type (Mense and Meyer 1985), but they are very likely to contribute to the D.O.M.S. sensation. Group III and IV afferents, however, have non-homogeneous response characteristics: most group IV afferents are nociceptors, whereas group III afferents also respond to non-noxious mechanical stimuli, such as muscle contraction and stretch (Mense and Meyer 1985; Kaufman and Rybicki 1987; Hayward et al. 1991). Thus, during contractions, mechanically-sensitive group III afferents fire initially whereas the group IV afferents fire later as metabolites accumulate (Kaufman and Rybicki 1987; Kaufman et al. 2002). Finally, myelinated group III (A-delta) carries sharp, localized pain, whereas unmyelinated group IV (C) muscle afferents carry dull and diffuse pain (Mense 1977; Kniffki et al. 1978). Group IV fibers have been suggested by Armstrong (1984) to be primarily responsible for the sensation of D.O.M.S., but group III may be expected to be involved as well when the muscle becomes swollen during the delayed SSC recovery phase.

*Can sensitization of small diameter afferents contribute to the bimodal SSC recovery?*

The exact influence of small diameter muscle afferents on the neural activation is not clearly established in fatigue conditions. In terms of their influence on the neural activation

they can act via spinal as well as via supraspinal loops, but the literature presents two major and contradictory trends in case of pain and damage.

As early as 1942, Travell and collaborators proposed the hypothesis further defined by Johansson and Sojka (1991) of a "vicious circle" in which group III and IV muscle afferents are believed to activate  $\gamma$ -motoneurons and thereby to increase the background firing of the muscle spindles and/or their sensitivity to stretch. This model is mostly supported by animal studies performed on decerebrate cats. Among them, Ellaway et al. (1982) demonstrated a tightly coupled excitation of  $\gamma$ -motoneurons by group III afferents in response to low threshold mechanical stimuli. In the case of SSC fatigue, this mechanism has been suggested to play a role in the large stretch reflex facilitation observed after repeated and prolonged passive muscle stretching (Avela et al. 1999a). In another cat study (Jovanović et al. 1990), intra-arterial injection of algescic agents (bradykinin, potassium chloride, histamine, 5-hydroxytryptamine) and lactic acid were found to induce increase of fusimotor discharge rate, which was mainly attributed to the activity of group III and IV muscle afferents. At the end of fatiguing contractions, evidence exists also for a reflexively-induced increase of the fusimotor discharge rate to the contracting muscle and to its close synergists (e.g. Ljubisavljević et al. 1992, 1995). Other muscles may be influenced as well (for a review, see Ljubisavljević and Anastasijević 1996). In this line, increased arterial concentration of bradykinin and 5-hydroxytryptamine are reported to excite primary and secondary muscle spindle afferents from homonymous as well as heteronymous muscles, including contralateral ones (Djupsjöbacka et al. 1995). Back to the original study of Appelberg et al. (1983), however, sensitization of group III muscle afferents would lead predominantly to inhibition of the dynamic  $\gamma$ -cells and excitation of the static  $\gamma$ -cells. Similar trends have been reported in anesthetized cats after injection of KCl and lactic acid (Johansson et al. 1993). Understanding of this facilitation and of the functional effect of small muscle afferents becomes more complicated, since it is known from the animal study of Cleland et al. (1982) that these small muscle afferents make a powerful input to inhibitory interneurons which could inhibit the Ia-afferent terminals (Duchateau and Hainaut 1993). Additional complication comes from the recent experimental muscle pain studies conducted in humans by Matre and collaborators (1998, 1999). In these studies, the infusion of hypertonic saline resulted in a stretch-reflex facilitation at rest, but had no effect when the muscle was functionally active (during sitting and walking).

The opposite hypothesis refers to the sparing and protective effects of the fatigued muscle (Bigland-Ritchie et al. 1986; Garland 1991; Enoka and Stuart 1992; Jammes and Balzamo 1992; Garland and Kaufman 1995). The hypothesis that small-diameter muscle afferents inhibit motoneurons during fatigue is supported by human studies, in which muscles were maintained ischemic at the end of a fatiguing contraction (Woods et al. 1987). In this condition, motor unit firing rates during MVCs remain low. This is likely to indicate that feedback from the muscle is responsible for low motor unit firing rates in fatigue, and the most likely candidates are the small-diameter afferents. Some convincing, although indirect evidence exists in favour of a presynaptic inhibitory effect on  $\alpha$ -motoneurons (Garland and McComas 1990; Garland 1991). For example Garland and McComas (1990) showed that the H-reflex was significantly depressed after electrically induced fatigue of human ankle plantarflexors under ischemia, and they failed to observe similar reduction in H-reflex with ischemia alone and with electrical stimulation without ischemia. Similarly, Duchateau and Hainaut (1993) observed that the decrement in H-reflex (normalised to the M-wave amplitude) did not recover as long as the fatigue-induced metabolic accumulation was maintained by ischemia. Avela et al. (2001) were able to show the same phenomenon after long-lasting SSC fatigue. Additional support to the involvement of group III and IV muscle afferents comes from the finding that selective damage of these afferents (by application of capsaicin) abolishes the depression of the monosynaptic reflex induced by muscle fatigue (Brunetti et al. 2003).

These overall observations give some support to the hypothesis of a potential functional effect of group III and IV muscle afferents, especially during the inflammatory process that characterizes the delayed SSC recovery period. This is in line with the recent findings of Dousset et al. (2007) that the delayed changes in reflexes and isometric force occurred concomitantly with an increase in muscle thickness, in C reactive protein, and in substance P concentration as well as in serum creatine kinase activity. With regard to the slow conducting velocity of small muscle afferents ( $0.3 - 13 \text{ m}\cdot\text{s}^{-1}$ ), their continuous activation by these polymodal stimuli gives support to their potential role in the delayed and prolonged SSC recovery phase. Thus, it is likely that presynaptic inhibition triggered by the group III and IV muscle afferents may contribute to the bimodal pattern observed along the recovery period.

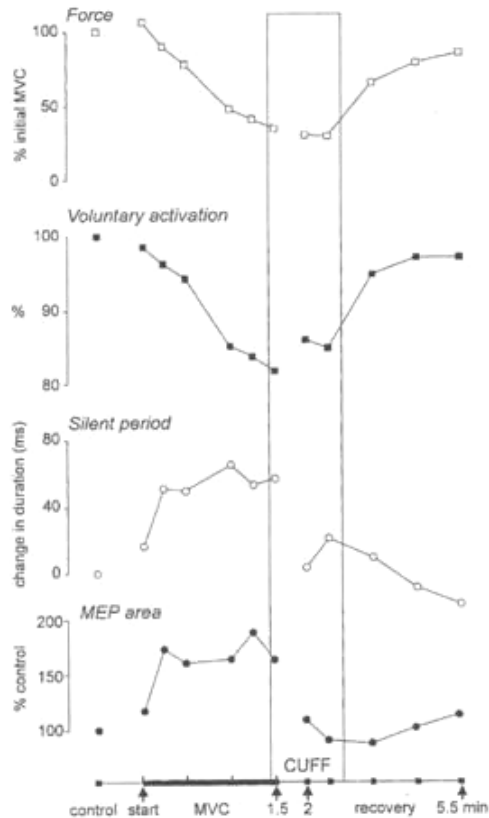
#### *Influence of sensory input at motor cortex levels*

As delayed-onset muscle soreness is characteristic to the delayed SSC recovery phase, one would expect that pain research would bring additional information on the central

influence of nociceptive group III and IV muscle afferents. Although pain influence has not been measured directly in connection with SSC fatigue, modifications in motor control strategies have been demonstrated using experimental pain protocols. With regard of the inflammation effects, Besson et al. (1975) demonstrated inhibitory influence of mesencephalic areas on neurons located in the lamina V from the dorsal horn when they were previously sensitized to bradykinin. Andersen et al. (1995) reported that a central summation of nociceptive and non-nociceptive afferent activity can occur once secondary hyperalgesia is present. The introduction in man of a new method of evoking tonic pain discharge through intramuscular injection of hypertonic saline may bring new insights into the comprehension of the immediate and delayed pain effects (Rossi and Decchi 1995; Rossi et al. 1998, 1999; Andersen et al. 2000; Sohn et al. 2000; Le Pera et al. 2001; Farina et al. 2005; Qerama et al. 2005). This technique is considered as a model for exciting nociceptive afferents without affecting muscle fiber electrophysiological properties (Farina et al. 2005).

For instance, Le Pera et al. (2001) demonstrated that tonic muscle pain can induce an inhibition of the primary motor cortex as reflected by a reduction in motor evoked potentials (MEPs) in the resting muscle. During the peak pain, the absence of effects on the H-reflex response suggested that the observed reduction in size of the motor-evoked potentials (MEPs) was probably due to decreased excitability of the motor cortex. Twenty minutes after pain, the MEP amplitude was found to be further depressed and the H-reflex amplitude was also reduced suggesting an inhibition of the spinal motoneurons, possibly overlapping the cortical inhibitory processes. Furthermore, when the firing of small-diameter muscle afferents is maintained by muscle ischemia at the end of a fatiguing contraction, force and voluntary activation remain reduced, although MEP and the silent period recover (Gandevia et al. 1996) (Fig. 75). Thus, it is likely that an increase in voluntary drive rather than changes in afferent inputs caused the observed MEP changes. On the other hand, the absence of voluntary activation recovery as long as the muscles were held ischemic suggests that small-diameter muscle afferents have a role in supraspinal fatigue.





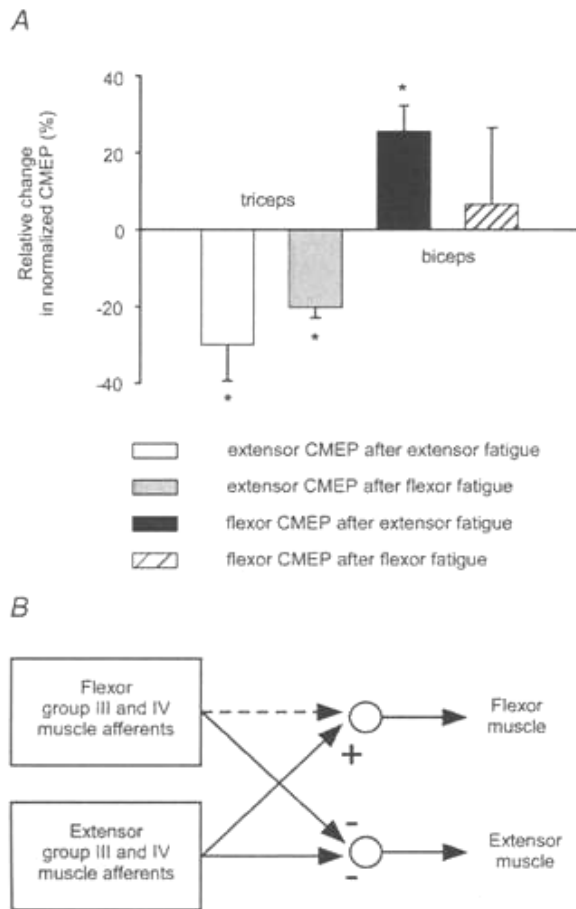
**Fig. 75. Effect of a maintained ischemia after sustained MVC on maximal voluntary force, voluntary activation and MEP characteristics (MEP area and silent period).**

The upper two panels show maximal voluntary force and voluntary activation (estimated by the force increment elicited by TMS). Both of them fell during the sustained MVC, did not recover during the ischemic period, but recovered when blood flow resumed.

The bottom two panels show changes in the TMS elicited EMG responses. Both the silent period duration and the MEP area increased during the sustained MVC. Unlike voluntary force and activation, they recovered despite muscle ischemia. (Adapted from Taylor & Gandevia 2001)

Furthermore and confirming animal studies (Martin et al. 2006) provided evidence that motoneurons of extensor and flexor muscles of the human elbow are not uniformly affected by inputs from group III and IV muscle afferents (Fig. 76A). As shown in Fig. 76B, afferent inputs from homonymous and antagonist muscles inhibit extensor motoneurons, whereas motoneurons innervating flexors are facilitated. This suggests that extensor muscles might require greater cortical output to generate a given force during fatigue (Martin et al. 2006). Thus, small-diameter muscle afferents may contribute to the inhibition of motoneurons in some muscles, but not in others.

Based on these overall observations, it is suggested that group III and IV muscle afferents are likely to contribute to the bimodal pattern observed along the SSC recovery period. On the other hand, both animal and human studies show mixed effects of group III and IV muscle afferents on motoneurons. It is suggested that the spinal and supraspinal effects of the sensitization of small muscle afferents could vary depending on the level of the imposed musculo-tendinous fatigue. Variation in receptor types and in the ability to modulate pain at multiple levels in the nervous system could explain part of the inter-subject variability in soreness perception.



**Fig. 76. Changes in single responses to corticospinal tract stimulation (CMEPs) of elbow flexor and extensor muscles produced by maintained ischemia after sustained MVC.**

(A) Change in CMEP in triceps or biceps during maintained ischemia after a 2 min MVC of the elbow extensors or flexors. The difference in CMEP area is expressed as a percentage of its control size prior to MVC. That is the percentage of facilitation or depression of the CMEP is shown. CMEPs in triceps were always evoked during brief extension MVCs and CMEPs in biceps during brief flexion MVCs. (\*, significantly different from the pre-MVC reference).

(B) Schematic diagram showing apparent effects of firing of group III and IV muscle afferents on the flexor and extensor motoneuron pools. The present findings indicate facilitation of flexor afferents and inhibition of extensors by flexor or extensor afferents. A previous study (Butler et al. 2003) showed nonsignificant facilitation of the flexors by flexors afferents (dashed line). (From Martin et al. 2006).

## 2.8. Concluding comments

This chapter made an attempt to demonstrate that naturally occurring, but exhaustive SSC exercise induces often dramatic reduction in force and power production. Although these effects are in some cases somewhat similar to those occurring after intensive eccentric exercise, the SSC fatigue is much more complex, because its more comprehensive way of loading the neuromuscular system mechanically, metabolically and neurally. The complex features of SSC loading is represented in the bimodal nature of performance deterioration and recovery.

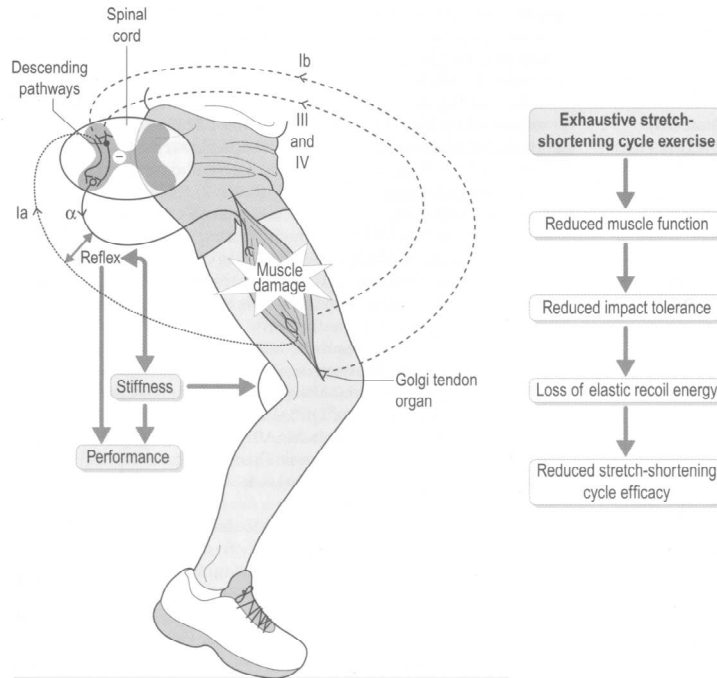
When sufficiently exhaustive, SSC exercises may lead to reversible muscle damage, with subsequent inflammation/remodelling processes associated with delayed-onset muscle

soreness. These events have considerable influence on neuromuscular function including such indices as muscle structure, muscle mechanics, and joint and muscle stiffness. In connection with these variables, the neural modifications are of considerable mechanistic interest. This is demonstrated by the parallelism between the neural and mechanical changes during the SSC exercise and along its subsequent recovery.

In the acute recovery phase, the monosynaptic stretch reflex is one of the most dramatically reduced parameters immediately after SSC exercise. The immediate reduction is thought to result from the short term effects of metabolic fatigue and from the decrease in muscle-tendon compliance.

The delayed recovery is more complex in nature. It is suggested that the delayed recovery results from the influences of structural damage/remodelling of the muscle-tendon tissues. Structural and functional recovery is long lasting, and prevents an individual from performing normal exercise routines for several days. In some severe cases, the recovery period may exceed 10 days. After fatiguing SSC exercise, inadequate neural drive may be considered as an attempt of the neuromuscular system to protect the muscle-tendon unit from additional damage. The many studies quoted in this manuscript suggest the influence of exhaustive SSC fatigue on the fusimotor-muscle spindle function as well as at the supraspinal level. Activation of small (III and IV) afferents is proposed as an attractive factor to cause presynaptic inhibition with subsequent reduction in the stretch reflex response, but also inhibition and/or facilitation at the supraspinal level. The functional neural consequences and their coupling with muscle damage/remodelling induced by SSC exercise are illustrated in Figure 77.

Finally, based on the reported SSC fatigue studies using maximal isometric and/or SSC testing tasks, it is emphasized that the latter ones may allow distinct examination of the central and stretch reflex EMG changes with fatigue. For instance, the maximal SSC tests confirmed the functional role of the stretch-reflex EMG response (discussed in Chapter 1) as their reduction with fatigue was found to result in large decrease in SSC performance. Even more meaningful are the submaximal SSC testing tasks as they may reveal the panel of changes (adjustments) of the central and reflex EMG components along the recovering period. As the SSC tasks involve several joints they favour also the examination of inter-muscular compensations. These remarks apply particularly to SSC exercises performed on the sledge exercise as they are characterized by a high reproducibility.



**Fig. 77. Schematic representation of the possible interaction between neural pathways and the events of mechanical failure during the delayed recovery phase from exhaustive SSC exercise.** Presynaptic inhibition pathway is not yet represented on this graph but should be added to it. When the muscle deteriorates, it is characterised by reduced tolerance to repeated stretch loads, by a deterioration of elastic recoil and increased work during the push-off phase, so that the same functional outcome can be maintained. (Modified from Horita et al. 2000).

### **3. Conclusion and perspectives**

The present review on our own research work within the larger research published on “SSC fatigue” demonstrates the interest of such a model to investigate the neuromuscular adjustments in case of alteration of the musculo-tendinous system. This review emphasizes, however, the need for further investigation of the neural mechanisms (from peripheral to supraspinal levels) that could explain the characteristics of the secondary decrease in performance after SSC exercise.

To examine further the potential mechanisms leading to neural inhibition and their functional consequences within the muscle-tendon unit complex, we recently got interested in maximal and supramaximal landing performances as potential testing tasks. Several experiments have been performed, but up to now in the absence of neuromuscular fatigue. As presented in the coming research plan, SSC fatigue should be examined within one or two years.

The present research plan entitled “Inter-segmental and neuromuscular control of high impact loads” constitutes the PhD project of Amandine GALINDO and will be co-supervised by Pr Janne AVELA from the Neuromuscular Research Center of the University of Jyväskylä (Finland).

#### **3.1. Research perspectives**

Although ground impact peak is usually attenuated by the pre-impact neuromuscular adjustments, intense and/or rapid ground impacts are reportedly associated with considerable risks of injury (Dufek and Bates 1990). It has been speculated that the natural protective neural strategy would be a continuum from the pre- to the post-impact phase (Santello 2005). In particular, the post-impact neural strategy is considered as a way to absorb energy (Dyhre-Poulsen et al. 1991) and to protect the musculo-tendinous system (Leukel et al. 2008a). This phase is characterized by a short-latency EMG burst, which has been recently suggested for the Soleus muscle to be down regulated by supraspinal centers (Leukel et al. 2008a). The pre-impact muscle activity may, thus, be expected to influence the post-impact central and reflex activation. The present project aims to investigate the origin, the flexibility and the functionality of neuromuscular strategies when landings are performed as single leg exercise including very high impact loads. To be optimal, neuromuscular adjustments during the pre- and post-impact landing phases are expected to take into account the external (impact

characteristics) task constraints as well as the internal ones, which are related to the capacity of the musculo-tendinous system to resist to the imposed stretch.

This project will examine in a complementary fashion (1) the influence of the pre-impact force level on the stretch reflex response during the post-impact landing phase, (2) the adjustments of the neuromuscular strategy to the task specificity (expected ground impact peak and required rebound vs. landing task), and (3) the modulation of the corticospinal and spinal excitabilities in case of prolonged, but reversible muscle damage and inflammatory process.

Each of these experiments will involve 12 to 16 normal male/female volunteer subjects. High impact landings will be performed on one leg on a sledge apparatus (Kaneko et al. 1984). A lower limb guiding device will be fixed to the front part of the sledge seat to allow the subjects to sustain given pre-set force levels. Evaluation of the neuromuscular and inter-segmental strategies will be performed using kinematics, kinetics and electromyographic recordings and complemented by the use of H-reflex test and transcranial magnetic stimulation. Functional efficacy of the chosen neural strategies will be examined further through high-frequency ultrasonography recordings on Soleus, Gastrocnemii and Vastii muscles. The study will be conducted according to the declaration of Helsinki after the approval from the ethics committee of the University of Jyväskylä.

### ***3.1. 1. Background and significance***

In vigorous forms of sport activities, the maximal impact peak can reach high or even extreme values that can exceed the mechanical tolerance of the muscle-tendon system. Landing in gymnastics and parachute jumps are typical activities, where the landing motion plays an important role. These activities are also examples, where the ground impact forces can reach impact loads as high as 11 or 18 times body weight (McNitt-Gray 1993; Whitting et al. 2007). Although ground impact peak is usually attenuated by the pre-impact neuromuscular adjustments, intense and/or rapid ground impacts are reportedly associated with considerable risks of injury (Dufek and Bates 1990). Poor landing techniques are identified as a major cause of serious injuries to the muscle-tendon complex as well as to the skeletal and joint structures (Teh et al. 2003). To cope with high impact landings, athletes and paratroopers are properly trained and their multijoint pre- and post-landing strategies are specifically developed to allow effective attenuation of ground impact loads (McNitt-Gray 1993; Whitting et al. 2007). Controlling an expected collision involves a predictive component such as the

estimation of the amplitude and time of the foreseen ground reaction force. Understanding how the central nervous system (CNS) controls impact absorption remains essential to determine the mechanisms of fall-related injuries and to define the pre- and post-impact strategies used to control the movement.

### ***3.1.1.1. Central and reflex neural adjustments in landing***

As recently reviewed by Santello (2005), a safe and smooth preparation for impact requires a precise control of muscle activation by the motor system. Pre-impact modulation of the muscular activity is well known as pre-programmed (Jones and Watt 1971). To attain optimal limb stiffness at impact, both timing and amplitude of pre-landing muscle activity (preactivation) need to be controlled. According to Santello (2005), preactivation modulates muscle forces using a “general strategy” that would be independent on the mechanical action (flexors versus extensors) and anatomical characteristics (uni- versus bi-articular muscles) of the individual muscles. On the other hand, this general strategy requires the integration of both external and internal information such as the drop height, the training level or the performed task. Thus, muscle preactivation is clearly modulated by the dropping height in landing tasks (Santello et al. 2001). In terms of training effect, untrained subjects are usually unaware of their actual functional capacity to resist incoming ground impacts, and this is resulting in a delayed stabilization in landing (McKinley and Pedotti 1992). For trained subjects, viewing the drop height before self initiating the fall seem to allow accurate predetermination of the impact time (Liebermann and Goodman 1991; Santello et al. 2001; Liebermann and Hoffman 2005), and thus the timing of the mechanical stretch (McDonagh and Duncan 2002).

On the other hand, the post-impact phase is characterized by a short-latency EMG burst, which exact origin and modulation are still matter of debate and uncertainty. Both the pre-programmed (Jones and Watt 1971; Dyhre-Poulsen and Laursen 1984; Dyhre-Poulsen et al. 1991) and the reflex (Dietz and Noth 1978; Dietz et al. 1981; Santello and McDonagh 1998; Duncan and McDonagh 2000; Kamibayashi and Muro 2006) hypothesis have received support. More recently, however, the use of false force platform (Duncan and McDonagh 2000; McDonagh and Duncan 2002) revealed robust evidence for short latency stretch reflexes (SLR) in the Gastrocnemius Medialis and Soleus muscles as well as for a potential reflex gain modulation by signals in descending pathways triggered by the false platform. For the quadriceps muscle group, the post-impact EMG burst would be mainly pre-programmed (Duncan and McDonagh 2000), but there is a lack of data for this muscle group. Further

support for a Soleus SLR modulation by supraspinal centers comes from the observation by Leukel et al. (2008a) of a reduced H-reflex sensitivity at the time of the SLR response in landing as compared to drop-jump. The additional observation of lower background EMG activities at SLR in the landing task reinforces the hypothesis of a task-dependent neural strategy. From a functional point of view, this strategy can be considered as a way to absorb energy (Dyhre-Poulsen et al. 1991) as well as a prevention strategy to protect the musculo-tendinous system from the high stretching load. Supporting the latter hypothesis, similar reductions in both stretch- and H-reflex responses have been reported for the Soleus muscle with increasing dropping height in drop-jumps (Leukel et al. 2008b; Taube et al. 2008).

Based on these observations, it is thought that in the case of landing from a high dropping height, the natural protective neural strategy should be considered as a continuum from pre- to post-impact phases. According to Santello (2005), pre-landing muscle activity is essential to prepare the muscle tendon complex for a rapid, forceful stretch occurring after foot contact and throughout the subsequent joint rotations. Pre-landing muscle activity may, thus, be expected to influence the post-impact SLR. However, if the literature is quite rich on the influence of the background EMG activity on the stretch reflex response in the isometric testing condition (Gottlieb and Agarwal 1979; Matthews 1986; Sinkjaer et al. 1988; Toft et al. 1991; Ogiso et al. 2002; Mrachacz-Kersting and Sinkjaer 2003), this relationship has not been tested in the stressful situation of landing.

#### ***3.1.1.2. Dynamic stiffness regulation in landing***

The dynamic relation between joint position and the torque acting about it defines the mechanical behaviour of a joint, called the dynamic joint stiffness (Kearney and Hunter 1990). Dynamic joint stiffness may be decomposed into two components:

1. *An intrinsic component* arising from the mechanical properties of the joint, passive tissue, and active muscle fibers,
2. *A reflex component* arising from changes in muscle activation due to sensory responses to stretch.

The overall dynamic stiffness, and perhaps the relative contributions of the intrinsic and reflex components, can be modulated by the central nervous system to determine the displacements due to the external forces as well as the forces needed to generate voluntary movements (Mirbagheri et al. 2000). In landing, Dyhre-Poulsen et al. (1991) suggested that the dynamic stiffness in the ankle joint increases prior to impact and then becomes negative at post-impact, thus leading to a loss of mechanical energy during post-impact phase. This phase of negative



stiffness plays an important role in motor control of landing as high muscle stiffness might be dangerous for the muscle-tendon integrity in overload conditions. These observations demonstrate that one of the major roles of the muscle-tendon system is to protect the body from injury coming from shockwaves, bone bending stress and joint instability. On the other hand, if excessive muscle stiffness can be detrimental to muscle and tendon; a low level of stiffness may also be considered as inappropriate to stabilize the joint rotations for a safe landing (Mizrahi and Susak 1982).

As previously mentioned, if a stretch reflex intervention during the post-impact landing phase is nowadays accepted, its functional role has not yet been examined. The recent use, however, of high-frequency ultrasonography to reveal a brief stretch of various lower limb muscles during the braking phase in rebounds (Ishikawa and Komi 2007; Sousa et al. 2007) demonstrates its potential use in landing situation.

#### ***3.1.1.3. Influence of muscle fatigue on the central and reflex neural adjustments in landing***

To be optimal, neuromuscular adjustments during the pre- and post-impact landing phases are expected to take into account the external (impact characteristics) task constraints as well as the internal ones, which are related to the capacity of the musculo-tendinous system to resist to the imposed stretch. The latter topic has not been much studied and the landing literature includes only a few studies on either trained (McKinley and Pedotti 1992; McNitt-Gray 1993; Whitting et al. 2007) or fatigued (Coventry et al. 2006; Madigan and Pidcoe 2003) subjects. As recently reported by Coventry et al. (2006), when fatigue progresses along the repetition of submaximal landings, subjects are able to alter their lower limb joint kinematics at impact in a way to maintain the same level of shock attenuation. Although different inter-segmental compensatory strategies have been reported depending to the type of fatiguing protocol (Coventry et al. 2006; Madigan and Pidcoe 2003), the same level of shock attenuation was indeed preserved. It is unfortunate that none of these studies has examined the associated neural adjustments.

Fatigue induced by repeated landings should, however, provide an excellent basis for studying neuromuscular adaptations to prolonged contractile failure. In repeated landings, each ground impact requires from the lower limb extensor muscles to resist actively to the induced stretch. When intensive and/or unaccustomed, this eccentric type of muscle action is well known to induce reversible muscle damage. This is associated with delayed onset muscle

soreness (DOMS), and with proprioceptive and neuromuscular impairments that may last over several days (Cheung et al. 2003; Faulkner et al. 1993).

Interestingly, natural forms of ground locomotion such as running and jumping, which include also repeated impacts and subsequent active braking phase, have been much studied in terms of their delayed structural and functional effects (for a review see Nicol et al. 2006). Recovery from a so-called stretch shortening cycle (SSC) type exercise is a long-term process. The largest functional deficits, including maximal force, activation and reflex (stretch and H-reflex) responses, are usually recorded on the 2<sup>nd</sup> day post-exercise when the symptoms of muscle soreness/damage are also greatest. In the delayed recovery phase, the pain usually disappears one to several days prior to the complete structural and functional recovery. The overall results suggest that fatigue-induced changes occur in parallel between mechanical and neural factors. There is enough evidence also to consider this interaction as neural mechanisms that compensate for contractile failure or protect fatigued muscles. Furthermore, a clear "testing task-dependent effect" is observed that emphasizes the plasticity of the neural adjustments. Clear trends are observed in SSC tests of either facilitation in tasks with submaximal effort level or inhibition in more stressful maximal testing conditions. In the latter case, significant relationships have been found between the fatigue-induced changes in the preactivation and in the post-landing stiffness (Avela and Komi 1998). Importantly also, both central and reflex adjustments were found to vary along the recovery period, and differently in submaximal and maximal rebound tasks.

These overall observations emphasize the interest to use SSC fatigue to examine the hierarchy of the neural adjustments, including the motor reflex adjustments via activation of group III and IV muscle afferents. The delayed recovery phase after SSC exercise is indeed characterized by an inflammatory process associated with intramuscular chemical, mechanical, and thermic changes that are reported to stimulate group III–IV muscle afferents and, thus, to contribute to the DOMS sensation. The landing test imposing high impact loads, it requires optimal neuromuscular strategies and inter-segmental compensations.

### **3.2. Specific aims and unique features of the project**

The aim of this PhD project will be to investigate the origin, the flexibility and the functionality of neuromuscular strategies when landings are performed as single leg exercise including very high impact loads. The investigation of risky activities such as the high impact landings will be chosen because it is expected that the control mechanisms are robust, automatic, and thus susceptible to affect the stretch-reflex response of the post-impact phase.

Specific emphasis will be put on:

- (1) the influence of the pre-impact force level on the post-impact central and stretch reflex responses in a vigorous landing task.
- (2) the adjustments of the neuromuscular landing strategy to the task specificity, with special emphasis on the inter-segmental compensations.
- (3) the central and reflex neural adjustments during the delayed recovery phase of fatiguing SSC exercise inducing reversible muscle damage.

The basic neuromuscular strategy will be thus examined from a given dropping height, and its adaptability will be subsequently tested by varying either the external constraint (dropping height and task) or the internal one (muscle fatigue).

This project follows the initial collaboration work between Prof. Paavo Komi (Neuromuscular Research Center, Department of Biology of Physical Activity, Univ. of Jyväskylä) and Dr Caroline Nicol (Institute of Movement Sciences, Faculty of Sport Sciences, Univ. of the Mediterranean)

The detailed objectives of the project are as follows:

### ***3.2.1. Objective I:***

Influence of the pre-impact force level on the short latency stretch reflex (SLR) response in a vigorous landing task.

#### **Hypothesis I:**

Isometric studies have provided evidence that a linear relationship exists at low force levels between SLR amplitude and background EMG activity. This phenomenon is referred to as “automatic gain control” (Gottlieb and Agarwal 1979; Matthews 1986; Sinkjaer et al. 1988). At higher force levels this relationship has become non linear (Toft et al. 1991; Ogiso et al. 2002; Mrachacz-Kersting and Sinkjaer 2003). Similarly, a linear increase in the H-reflex amplitude (Burke et al. 1989; Butler et al. 1993; Toft and Sinkjaer 1993; Oya et al. 2008) has been reported with increasing plantar flexion torque production up to 50 % of the maximal voluntary contraction, but not above this force level (Taube et al. 2008). Although the pre-impact phase of high impact landings might be quite static up to the impact-induced stretch, this stressful situation is expected to affect the linear relationship reported between low background force level and the SLR response amplitude. In the case of high landing loads, it is thought that an increase in the pre-impact force level up to 50% of MVC might not lead to

an increased SLR response as a neural protective (inhibitory) strategy is likely to occur during the post-impact phase.

**Abstract of the paper entitled “Neuromuscular control in landing from supra-maximal dropping height” Galindo et al. in press (J. Appl. Physiol.) – see Original publications**

The present study utilized high impact supra-maximal landings to examine the influence of the pre-impact force level on the post-impact surface electromyographic (EMG) activity and, in particular, on the short latency EMG reflex (SLR) component. Unilateral-leg landings were performed in a sitting position on a sledge apparatus after release from high, but individually constant dropping height. A lower limb guiding device fixed to the front of the sledge seat allowed the subjects to sustain a given pre-set force level up to impact. This force level was either freely chosen or set at 20, 35 and 50% of maximal isometric plantarflexion force. EMG activity was recorded from eight major lower limb muscles. It was expected that the increase in the pre-impact force level would require the intervention of a protective neural strategy during the post-impact phase that would attenuate the SLR amplitude. Confirmation of the existence of SLR response was confirmed for the SOL muscle by the mechanical stretch revealed by ultrasonography. The main finding was the similarity across all tested conditions of the impact peak force and post-impact EMG activity, including the SLR response. Both of these observations are mostly attributed to the similar EMG levels and close force levels reached towards impact. The instruction to maintain a given pre-set force level was indeed overruled when getting close to impact. It is suggested that in the present supra-maximal landing condition, a protective central neural strategy did occur systematically, but in an adjusted way, that secured similar impact loads.

**3.2.2. Objective II:**

Adjustments of the neuromuscular landing strategy to the task specificity.

**Hypothesis II:**

Influence of the ground impact intensity on the post-impact central and reflex EMG responses has been investigated in rebound conditions (Adams and Hamilton 1988; Avela et al. 1996; Leukel et al. 2008a), but not yet in landings. In the latter study, the H-reflex over the maximal mass compound action potential (M-wave) ratio (H/M) was found to progressively decrease at the SLR timing from low to excessive dropping heights. This was suggested to prevent the musculo-tendinous system from excessive loading. Additionally, this study reported a lower H reflex response at SLR in a landing as compared to a rebound task from a same low

dropping height. It can be hypothesised that in landings, higher dropping height might result in smaller H-reflex and SLR responses highlighting a protective strategy. Furthermore, different inter-segmental strategies might be expected as well to attempt to protect particularly the more distal segment.

### **Research methods II:**

The basic landing protocol previously defined in “research methods I” has been used with a similar number of subjects, but two dropping heights (supramaximal and maximal) have been tested. By changing the dropping height we can affect indirectly (through visuo-vestibular inputs) and directly the impact peak intensity. For the maximal dropping height, a rebound test has been performed as well to allow comparison with the actual literature. In this case, the instruction given to the subject for the post-impact phase intended to rebound instead of landing. *This part of the research plan is under analysis and the results should be presented at the next ECSS Congress (Oslo, Norway June 2009).*

The evaluation of the neuromuscular and inter-segmental strategies will be complemented by the use of H-reflex test. For each subject, three stimulations will be timed, so that the H-reflex peak occurred either at 100ms before impact, at ground impact, or at the SLR peak. H-reflex will be elicited in the Soleus muscle by stimulating the posterior tibial nerve. Moreover, to compare modulation of spinal and corticospinal excitability at distinct phases, motor-evoked potentials (MEPs) will be induced by transcranial magnetic stimulation (TMS). After an optimal coil position on the left motor cortex and an accurate set up of the stimulus intensity for each subject, the MEPs at the same three timings than the ones used for the H-reflex. *This second part of the project will be performed in Jyväskylä, under the co-supervision of Pr J. Avela, in collaboration of Pr M. Gruber and A.G. Cresswell.*

### **3.2.3. Objective III:**

Central and reflex neural adjustments in the delayed recovery phase lasting for a few days after fatiguing SSC exercise inducing reversible muscle damage.

### **Hypotheses III:**

SSC exercise including intense and unaccustomed eccentric muscle actions can induce potential ultrastructural muscle damage that requires several days to recover. As previously mentioned in section 2.7.2.2., group III–IV muscle afferents have been proposed to favour a presynaptic inhibitory effect on  $\alpha$ -motoneurons ( Garland and McComas 1990; Garland

1991; Duchateau and Hainaut 1993; Avela et al. 2001; Brunetti et al. 2003). In addition, the concept of supraspinal fatigue via group III-IV afferents is supported by the reported effects of muscle pain at supraspinal and spinal levels (Le Pera et al. 2001; Qerama et al. 2005). When dealing with fatiguing SSC exercise, the second day is known to be associated in most of the subjects with the largest values of DOMS and functional decrements. At that time, a high landing test may be expected to favour the occurrence of both supraspinal and spinal reflex-induced inhibitions. During the subsequent days, however, the precocity of the DOMS disappearance as compared to the functional neuromuscular recovery (Nicol et al. 2006) is expected to be associated with an earlier recovery of the cortical as compared to the spinal excitability.

### **Research methods III:**

The basic landing protocol previously defined in “research methods I” will be used with a similar number of subjects as test before and after fatigue protocol. The fatigue protocol will consist of intensive and exhaustive series of rebounds on the sledge apparatus. Supramaximal and maximal landing tasks will be tested after, two day, four days and eight days after the exhausted SSC tasks in the way to follow the entire recovery phase. Thus, precise functional evaluation of the adopted central and reflex neural strategies in landing task will be performed by combining the same kinetic, kinematics and ultrasonographic recordings similarly to one described in “research methods I”. Furthermore, the investigation of the inter-segmental strategy will be done by adjusting the SSC fatiguing protocol on the muscle group chosen (Triceps Surae or Quadriceps). Central fatigue can be measured by comparing the twitch superimposed to a Maximal Voluntary Contraction (MVC) and the twitch evoked on the relaxed muscle. The twitch superposition is the most popular method in the literature to reveal an activation deficit (Merton 1954). The inconvenient of this method is that it is not possible to determine if the central fatigue originates from a supraspinal site and/or from spinal level. The change in spinal and corticospinal excitability during the landing after impact along the entire recovery period will be examined by peripheral nerve stimulation (H-reflexes) and transcranial magnetic stimulation (TMS) (motor-evoked potentials, MEPs). This protocol will use these measurement methods to localise as precisely as possible the site of reduced activation (corticospinal, spinal or peripheral) along a recovery period after SSC exhausted exercise.

**The combination of the successive phases of this research plan should reveal some of neural mechanisms involved in the adaptability of the lower limb activation pattern to both external and internal constraints in case of high impact loads. Evaluation of its functional efficacy should allow us to evaluate the pertinence of the chosen neural strategy.**

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## PUBLICATIONS ORIGINALES

### 15 Articles publiés et 1 sous presse

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**Université de la Méditerranée (Aix-Marseille II)**  
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**LE CYCLE ETIREMENT-DETENTE :**  
**un modèle d'étude des mécanismes de compensation**  
**en cas d'altération du système musculo-tendineux**

Mémoire de recherche présenté par **Caroline Nicol**

**Résumé**

Dans les formes habituelles de la locomotion, les muscles extenseurs des membres inférieurs sont engagés au cours de chaque appui au sol dans un enchaînement de phases de freinage (excentrique) et de poussée (concentrique). Cette combinaison, appelée *Cycle Etirement-Détente*, est connue pour accroître l'efficacité de la phase finale concentrique, notamment par la restitution de l'énergie élastique préalablement accumulée lors de l'étirement actif du complexe musculo-tendineux. Nos travaux ont contribué à démontrer que l'intervention de la composante de courte latence du réflexe d'étirement musculaire permettait d'accroître la force et la raideur du muscle. L'utilisation récente de l'échographie à haute fréquence révèle l'existence d'un étirement des faisceaux musculaires des muscles mono- et biarticulaires en début de phase de freinage. Cela renforce l'hypothèse d'une contribution non négligeable du réflexe d'étirement à l'efficacité du CED.

Lorsqu'ils sont épuisants ou inhabituels, les exercices de type CED peuvent engendrer des micro-lésions musculaires dont la lente résorption comprend un phénomène inflammatoire ou de remodelage associé à des douleurs musculaires retardées. La période de récupération est généralement de type biphasique, montrant des baisses fonctionnelles immédiates, suivies d'une récupération partielle ou totale à 2h qui précède de nouvelles baisses pouvant perdurer plusieurs jours. L'évolution parallèle des paramètres nerveux et mécaniques souligne l'influence des baisses d'activation sur les pertes de force enregistrées. L'étude des performances maximales et sous-maximales de type CED montre la flexibilité des ajustements neuromusculaires. La présence de facilitation ou d'inhibition de l'activation centrale et/ou réflexes suggère une intégration sensorimotrice des informations relatives à l'état du muscle (avec ou sans inflammation) et des contraintes de la tâche (maximale vs. sous-maximale). L'inhibition spécifique du muscle « lésé » lors de son étirement actif pendant la phase de freinage pourrait ainsi refléter l'intervention d'un mécanisme protecteur. En accord avec de récentes études des mécanismes centraux de la fatigue, ces résultats tendent à conforter l'hypothèse d'une inhibition motoneuronale réflexe via un circuit activé par les afférences de type III et IV.

Nos travaux soulignent également la persistance d'une mauvaise estimation et/ou compensation des déficits fonctionnels (altération des sens de la position et du mouvement) ainsi qu'une absence de compensation contralatérale d'une fatigue musculaire ipsilatérale. Les conséquences de tels déficits perceptivo-moteurs pourraient s'avérer critiques lorsque la douleur disparaît précocement par rapport au retour à l'intégrité structurale et fonctionnelle.

Nos perspectives de recherche visent à approfondir les mécanismes sous-jacents des inhibitions centrales et réflexes qui caractérisent la phase retardée de la récupération des exercices de types CED. Les protocoles de recherche en cours examinent les stratégies neuromusculaires adoptées dans une tâche plus extrême d'impact supramaximal. Les prochains protocoles devront s'enrichir des techniques actuelles d'investigation de la fatigue centrale.