



# The functional role of triceps during human locomotion

Jean-Louis Honeine

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par

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<p><b>Le rôle fonctionnel du triceps sural durant la marche</b></p>
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# List of abbreviations

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AP	Antero-posterior
CNS	Central nervous system
CoM	Centre of mass
CoP	Centre of pressure
CPG	Central pattern generator
EMG	Electromyography
GM	Gastrocnemius Medialis
GL	Gastrocnemius Lateralis
GRF	Ground reaction force
ML	Medio-lateral
SOL	Soleus
TIB	Tibialis Anterior
TS	Triceps Surae
Ver	Vertical

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## i. Sommaire

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L'importance de la marche dans nos activités de tous les jours est évidente. La bipédie est avantageuse dans la mesure où elle libère les bras durant la marche, cependant, elle ajoute une contrainte très importante qui est de lutter contre la gravité pour maintenir l'équilibre tout en générant des forces de propulsion.

Il paraît évident que la marche nécessite l'orchestration des muscles des membres inférieurs qui génèrent ou bien qui contrôlent les couples articulaires de la hanche, genou et cheville. Toutefois, la cheville de la jambe d'appui est la seule articulation qui fait l'interface entre le corps et le sol durant la phase de simple appui. Ceci rend les muscles fléchisseurs plantaires de parfaits candidats pour propulser le corps et maintenir l'équilibre durant la marche. Le rôle postural du triceps sural (TS) dans le maintien de l'équilibre est bien établi (Schieppati et al., 1994; Morasso & Schieppati, 1999; Sozzi et al., 2013). Par contre, la participation du TS dans la génération de force propulsive quand celui-ci est en contraction concentrique durant la phase finale de simple appui est toujours sujet à débat.

Certains auteurs suggèrent que le TS est responsable du « push-off <sup>1</sup> » observé à la fin du simple appui. En d'autres termes, ils postulent que l'augmentation de l'activité du TS à la fin de la phase de simple appui sert à accroître l'accélération du centre de masse (CoM) durant cet intervalle (Winter, 1983 ; Neptune et al., 2001 ; McGowan et al., 2008). D'autres soutiennent que le rôle principal du TS est de résister à la rotation du tibia et donc de maintenir l'équilibre. Ainsi, l'amplification de l'activité du TS durant la phase finale de simple appui résulterait du fait que la force nécessaire pour maintenir l'équilibre augmente considérablement quand le CoM s'éloigne de la jambe d'appui (Simon et al., 1978 ; Murray et al., 1978 ; Sutherland et al., 1980).

L'objectif principal de cette dissertation est de révéler le rôle fonctionnel du TS durant la marche. Plus précisément, dans un premier temps, nous voulions savoir si le TS joue un rôle direct dans la génération des forces de propulsion. Deuxièmement, nous voulions savoir comment le système nerveux central (CNS) module l'activité du TS pour déterminer la

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<sup>1</sup> Le push-off indique l'augmentation de forces antéropostérieures et verticale qui se produit vers la fin de la phase d'appui.

cinématique globale (longueur de pas et cadence) et la cinétique de la marche (force de propulsion et équilibre).

Nos hypothèses de travail sont les suivantes :

- 1) Le TS ne génère pas directement les forces de propulsion durant la marche. Le rôle principal du TS est de maintenir l'équilibre postural durant la marche.
- 2) La modulation de la durée d'activation du TS permet de contrôler la durée de simple appui, donc la cadence. La marche pouvant être modélisée comme une succession de mouvements d'un modèle de pendule inverse, le contrôle de la durée d'activation du TS permet d'ajuster la longueur de pas et la position du CoM par rapport au pied d'appui (représenté par le point d'application des forces ou encore le centre de pression, CoP).
- 3) L'amplification de l'activité du triceps durant la marche rapide est due à une augmentation de la nécessité de support du corps.

Deux études ont été mises en place pour tester nos hypothèses de travail pour un total de trois expérimentations (1 pour la première étude et 2 pour la deuxième étude).

Avant de présenter les deux études, il est important de rappeler quelques éléments essentiels de la marche. Durant la marche, les oscillations de la jambe pendulaire sont quasi-ballistiques et modélisables comme une série de pendules simples (Mochon & McMahon, 1980), tandis que le mouvement du CoM peut être représentés par un pendule inverse (Cavagna & Margaria, 1966). Le CoM tourne donc autour du CoP pour atteindre sa position la plus basse au moment du contact de la jambe pendulaire avec le sol. Durant la marche, et comme dans un système de pendule inverse, l'énergie potentielle et l'énergie cinétique travaillent en antiphase (Cavagna et al., 1976), qui permet à la force verticale<sup>2</sup> de générer un couple articulaire (voir figure II.4). Ce couple articulaire, appelé couple de déséquilibre dans cette dissertation, permet la génération de force propulsive (Cavagna & Franzetti, 1986 ; Cavagna et al., 2000).

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<sup>2</sup> La force verticale est la résultante de deux forces : Le poids et les forces inertielles dues à l'accélération verticale du CoM.



Pour les deux études, les participants devaient se tenir debout pieds nus sur une plateforme de force (0,90 m×1,80 m, Advanced Mechanical Technology, Inc., 176 Waltham Street, Watertown, MA 02472-4800, USA). Il leur était ensuite demandé d'initier la marche après le déclenchement d'un signal sonore. La plateforme de force a été utilisée pour mesurer les trois composantes de la réaction au sol et du moment (ML, Medio latéral ; AP, Antéropostérieur ; Ver, Vertical). La position du centre de pression (CoP) a été reconstruite à partir des données de la plateforme. Les activités électromyographique (EMG) des trois muscles du TS (Soléaire - SOL ; Gastrocnémien Médiale – GM ; Gastrocnémien Latéral – GL) et du Tibiale Antérieur (TA) ont été mesurées. Les signaux EMGs ont été mesurés à l'aide d'un système sans-fil (Zero-wire, Aurion, It). Le protocole européen SENIAM a été respecté pendant la pose des électrodes de surface (Merletti & Hermens, 2000). Les signaux analogiques acquis via la plateforme et l'amplificateur EMG ont été synchronisés et numérisés à une fréquence d'échantillonnage de 1000 Hz. Les données acquises étaient en suite classées sur un ordinateur et traitées hors-ligne à partir d'un logiciel opérant sur Matlab 2008b (Mathworks, Natick, Massachusetts, USA).

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Pour la première étude, nous voulions démontrer que le TS n'était pas directement impliqué dans la génération de force de propulsion durant la marche. Si son rôle n'est pas de générer les forces de propulsion, alors son rôle serait donc de maintenir l'équilibre de la personne durant la phase de simple appui. En conséquence, ceci implique que la propulsion durant la marche est causée par le couple gravitationnel.

Pour tester cette hypothèse, nous avons utilisé le protocole de l'initiation de la marche (GI - Gait Initiation). Un échantillon de 10 volontaires composés de 9 sujets masculins et 1 sujet féminin ont participé à l'expérimentation. Leurs âge, masse et taille était en moyenne de 34 années (fourchette comprise entre 23–54 a), 72 kg (fourchette comprise entre 61–83 kg) et 1,73 m (fourchette comprise entre 1,69–1,83 m), respectivement. Les participants ont été soumis à 4 conditions expérimentales. L'ordre des conditions était aléatoire. Les conditions étaient réparties selon deux critères: 2 vitesses de marche et 2 poids différents ajoutés autour du CoM. En incrémentant le poids des participants et leur demandant de maintenir la vitesse de marche effectuée sans charge, les lois de la mécanique prédisent une augmentation de la force de propulsion. Si l'activité (EMG) du TS augmente également, alors ceci impliquerait

que ce dernier participe activement à la propulsion du corps. A l'inverse, si l'activité EMG ne change pas, alors l'hypothèse « push-off » doit être écartée. La charge que portaient les participants consistait de 2 disques en métal de 10 kg. Les disques étaient attachés au corps au niveau abdominal avec l'aide d'une ceinture (un disque a été placé ventralement et l'autre dorsalement). Le positionnement des disques a été choisi de façon à conserver autant que possible la position initiale du CoM.

Les conditions étaient les suivantes :

- (S) Initier la marche à une vitesse spontanée.
- (S+L) Initier la marche à une vitesse spontanée + charge de 20 kg.
- (F) Initier la marche à une vitesse rapide.
- (F+L) Initier la marche à une vitesse rapide + charge de 20 kg.

Quinze essais ont été effectués pour chaque condition. Une analyse de variance à 2 facteurs (vitesse et poids) a été utilisée pour tester les différences significatives entre conditions des variables suivantes: vitesse AP du CoM au 'foot-contact', longueur du pas, durée de simple appui, amplitude de la composante AP du push-off, amplitude du freinage verticale du CoM, amplitude du couple de déséquilibre au 'foot-contact', distance du CoM au CoP au 'foot-contact' et l'amplitude de l'activité moyenne de l'EMG de SOL, GM et GL. Toutes les analyses statistiques ont été réalisées sur SPSS version 20.0 (International Business Machines Corporation, Armonk, New York, USA).

Le but principal de cette étude était de comparer l'activité EMG du TS durant une marche sans charge à une marche avec charge à vitesse égale. L'analyse de variance n'a montré aucun effet de la charge sur la vitesse AP du CoM au 'foot-contact', sur la longueur du pas et sur la durée de simple appui. Les mêmes résultats ont été obtenus pour les 2 conditions pour la marche rapide (F et F+L). Durant la marche rapide, tous les participants ont augmenté la longueur de pas. Les résultats figurent dans le tableau III.1.

En deuxième lieu, nous avons examiné l'amplitude de l'activité moyenne EMG du SOL, du GM, et du GL durant l'initiation de la marche avec ou sans charge. Nous avons aussi comparé l'amplitude de la composante AP du « push-off » avec ou sans charge. L'analyse de variance a mis en évidence une absence de l'effet de la charge sur l'activité EMG des trois muscles

pour chacune des vitesses de marche (S et F). La force de propulsion, quant à elle, a augmenté de façon significative lorsque les participants ont initié la marche avec charge (voir figure III.3). Ceci démontre que le TS ne participe pas activement dans la génération de force de propulsion.

L'amplitude du freinage vertical pour contrôler la chute du CoM n'a pas varié entre les conditions de charge, pour une même vitesse. L'amplitude du freinage était calculée comme la différence entre la vitesse verticale du CoM au foot-contact et la vitesse verticale minimale. Cependant, l'amplitude de freinage a augmenté quand les participants ont marché plus rapidement. Cette augmentation du freinage du fait d'une chute plus importante requiert alors une augmentation des forces antigravitaires (Liu et al., 2008). En cohérence, l'amplitude des activités moyennes d'EMG des trois muscles du TS a augmenté lorsque les participants marchaient plus rapidement (voir figure III.3). Ceci implique que le rôle principal du TS dans la marche est de freiner activement la chute du CoM, comme décrit par Chong et al. (2009) et Chastan et al. (2010).

Enfin, nous avons examiné le couple de déséquilibre pour comprendre la cause de l'augmentation des forces propulsive durant la marche. Le couple de déséquilibre a été calculé comme le produit du CoM-CoP gap par la force verticale appliquée au CoM. L'analyse de variance a montré une absence d'effet de la charge sur le CoM-CoP gap à l'instant du 'foot-contact'. Par contre, l'amplitude du couple de déséquilibre a augmenté significativement lorsque les sujets marchaient avec le poids supplémentaire (voir figure III.4). L'accroissement du couple de déséquilibre était donc dû au poids de la charge ajoutée. Autrement dit, le poids supplémentaire a augmenté la force verticale ressentie par le CoM, ce qui par conséquent a augmenté le couple de déséquilibre.

D'après Cavagna et al. (1976), le couple de déséquilibre génère les forces de propulsion. Au regard de la figure III.4, la similarité dans le profil des tracés de la force de propulsion et du couple de déséquilibre est remarquable. Les courbes situées dans la figure III.4 suggèrent aussi la cause du push-off antéropostérieur à la fin de la phase d'appui. En effet, durant la phase de simple appui, le CoM accélère dans le plan sagittal. Le CoP qui, au début de la phase de simple d'appui, se situe à l'arrière du pied se met à se déplacer en avant. A la fin de la phase de simple appui, le CoP ne peut plus avancer étant donné qu'il reste coincé sous les orteils du pied d'appui. Entre-temps, le CoM continue à progresser d'une manière

parabolique. Ceci fait croître le CoM-CoP gap brusquement, qui à son tour augmente le couple de déséquilibre et provoque le push-off dans le plan sagittal.

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Au cours de la marche « normale » il nous arrive souvent « d'accélérer » le pas, i.e. d'augmenter la vitesse de progression, ou de marcher plus lentement. De par la relation établie  $V = L \times F$ , ( $V$ ,  $L$ ,  $F$  désignent respectivement la vitesse, la longueur du pas, et la fréquence ou cadence des pas), il apparaît qu'un grand nombre de configurations cinématiques de la marche est possible. Les relations explicitant ces différentes configurations peuvent être retrouvées dans les articles de Alexander (1984), Nilsson et al. (1985), Leurs et al. (2011) et Ivanenko et al. (2011). Cependant, dans ces publications les auteurs n'ont pas examiné ces relations avec l'activité EMG. Dans la marche « normale », la vitesse et la longueur du pas co-varient linéairement, la fréquence apparaît comme un paramètre d'ajustement. Toutefois on peut considérer que les configurations extrêmes de marche sont la marche lente avec un grand pas et la marche rapide avec des petits pas. L'objet de cette seconde étude était de montrer comment le CNS opère par l'intermédiaire de l'activité EMG du TS pour obtenir toutes les conditions de marche.

Il a été déjà montré que l'activité EMG du TS varie en durée et en amplitude avec la vitesse (Pedotti et al., 1977; Winter, 1983 ; Der Otter et al., 2004). Cependant, les résultats de l'étude précédente a clairement montré que l'activité EMG du TS ne générait pas les forces de propulsion, i.e. qu'elle ne contrôle pas la vitesse. Cependant, c'est bien l'activité EMG du TS de la jambe d'appui qui doit être responsable de la modulation des différents paramètres cinématiques de la marche, selon le principe de la mécanique qui stipule que les forces de propulsion ne peuvent être générées que s'il y a un appui. Cette ambiguïté – appui (interface pied-sol) / activité EMG jambe d'appui – n'en est pas une si l'on prend en compte les résultats de l'étude précédente.

Dans la deuxième étude, nous avons postulé qu'étant donné que le TS est responsable du support du corps, en modulant la durée de l'activité du TS, le CNS peut déterminer les deux composantes cinématiques principales qui sont la vitesse et la longueur de pas et la cinétique de la marche (la force de propulsion). En d'autres termes, dû à la vocation que nous pensons antigravitationnelle de la force du TS, en modulant la durée de l'activité du TS de la jambe

d'appui, le CNS peut contrôler l'instant du posé du pied controlatéral. Donc de cette façon, le CNS détermine la cadence ou la durée de simple appui. De plus, au moment du décollement de la jambe pendulaire « foot-off », le CoM voyage déjà à une certaine vitesse antéropostérieure du CoM du (« foot-off »), et puis il continue à accélérer jusqu'au contact du pied avec le sol à cause de l'action du couple de déséquilibre. En conséquence, en contrôlant la durée de la phase de simple appui, le TS détermine donc également la distance que le corps effectue par rapport à la jambe d'appui. En d'autres termes, il détermine à la fois la longueur du pas et le CoM-CoP gap. Ce dernier permet de moduler le couple de déséquilibre et donc la force de propulsion ainsi que la vitesse de marche.

Deux expérimentations complémentaires ont été utilisées afin de tester notre hypothèse. Un échantillon de 19 volontaires composés de 9 sujets féminins et 10 sujets masculins ont participé à l'expérimentation. Leur âge, masse et taille étaient en moyenne de 25 années (fourchette comprise entre 20–29), 72 kg (fourchette comprise entre 48-92) et 1,76 m (fourchette comprise entre 1,61-1,83), respectivement. Onze personnes ont participé à la première expérimentation et onze autres ont été soumises à la deuxième.

- Le protocole de l'initiation de la marche (GI – Gait Initiation) a été choisi pour la première expérimentation qui a pour objectif de démontrer que la modulation temporelle de l'activité du TS détermine la durée de simple appui.
- Le protocole du rattrapage de l'équilibre (BR - Balance Recovery) a été sélectionné pour la deuxième expérimentation afin d'examiner si la modulation temporelle de l'activité du TS détermine également la longueur du pas.

Pour la première expérimentation, les participants ont été soumis à trois conditions expérimentales:

- (Normal) Initiation de la marche à une vitesse spontanée ou normale.
- (Slow) Initiation de la marche à une vitesse lente.
- (Fast) Initiation de la marche à vitesse rapide.

Douze mesures ont été effectuées pour chaque condition. Il était indiqué aux sujets de maintenir une longueur de pas constante pour toute la durée de l'expérimentation. On sait que la vitesse ( $V$ ), la longueur du pas ( $L$ ) et la fréquence des pas ( $F$ ) sont reliées par la relation  $V$

$= L \times F$ , où  $V$  est la vitesse de marche,  $L$  est la longueur de pas et  $F$  est la fréquence de pas. En demandant au sujet d'initier la marche avec une longueur de pas constante tout en modulant la durée de simple appui la vitesse de progression devrait varier. Si notre hypothèse est correcte, la durée de l'activité EMG du TS doit covarier avec la durée de simple appui.

Pour la deuxième expérimentation, les participants ont été soumis à trois conditions expérimentales:

- (Normal) Rattrapage de l'équilibre à une longueur de pas normale.
- (Short) Rattrapage de l'équilibre à une petite longueur de pas.
- (Long) Rattrapage de l'équilibre à un pas très long.

Douze essais ont été effectués pour chaque condition. Le protocole BR consistait à provoquer une chute vers l'avant à partir d'une posture initiale inclinée du participant. La consigne pour le sujet était de marcher pour rattraper la chute-avant. La posture inclinée du participant était obtenue grâce à un dispositif comprenant un système électro-mécanique, un câble en acier et une ceinture abdominale. Le participant, ceinturé au niveau abdominal, est relié au système électro-mécanique par l'intermédiaire du câble. Le système électro-mécanique est composé d'un dynamomètre solidarisé à un électro-aimant pouvant coulisser le long d'une colonne métallique laquelle est fixée au mur. Pour que la force de retenue ne modifie pas le poids du sujet une attention particulière est portée sur l'horizontalité du câble. Le dynamomètre permet alors de calculer l'inclinaison du sujet.

L'avantage du protocole de rattrapage de la chute est que la vitesse AP du CoM au foot-off reste constante quand les participants, effectuent des pas de longueurs différentes, parce que elle ne dépend que de l'inclinaison du corps (Do et al., 1982). Ceci permet d'isoler l'effet de la vitesse antéropostérieur du CoM précédant le foot-off dans la détermination de la longueur de pas. Si notre hypothèse est correcte, en contrôlant la durée de simple appui, le TS doit également moduler la longueur de pas.

Comme dans l'étude précédente, les participants ont effectué les tâches sur une plateforme de force (0,90 m x 1,80 m, Advanced Mechanical Technology, Inc., 176 Waltham Street, Watertown, MA 02472-4800, USA). Pour GI, il leur était demandé d'initier la marche après le déclenchement d'un signal sonore. Pour BR, l'angle d'inclinaison des participants variait

très peu autour de 17°. Les participants n'étaient pas informés de l'instant où le câble était relâché.

Une analyse de variance à 1 facteur (vitesse pour GI et Longueur de pas pour BR) a été utilisée pour tester les différences significatives entre conditions pour les variables suivantes: vitesse AP du CoM au foot-contact, longueur du pas, durée de simple appui, amplitude de la composante AP du push-off, amplitude du freinage verticale du CoM, amplitude du couple de déséquilibre au foot-contact, CoM-CoP gap au foot-contact et amplitude de l'activité moyenne de l'EMG de SOL, GM et GL<sup>3</sup>. La méthode de Pearson de calcul de régression a été utilisée afin de mettre en évidence la relation entre la durée de simple appui et la durée de l'activité du TS dans GI d'une part, et la relation entre la durée de simple appui et la longueur de pas dans BR d'autre part. Toutes les analyses statistiques ont été réalisées sur SPSS version 20.0 (International Business Machines Corporation, Armonk, New York, USA).

Pour GI, la durée de simple appui a augmenté dans la condition 'slow' et a été réduite dans la condition 'fast'. Un coefficient de régression  $r^2=0,63$  a été obtenu entre la durée de simple appui et la durée d'activité du TS<sup>4</sup>. L'analyse de variance a montré que l'amplitude du freinage verticale du CoM ainsi que l'amplitude de l'activité moyenne du TS ont significativement augmenté lorsque la vitesse de marche des participants augmentait. Finalement, l'analyse de variance a également dévoilé une augmentation du CoM-CoP gap et donc du torque de déséquilibre au foot-contact quand les participants marchaient plus rapidement (voir figure IV.1).

Pour BR, la durée de simple appui a diminué dans la condition 'short' et augmenté dans la condition 'long'. Un coefficient de régression  $r^2=0,91$  a été obtenu entre la durée de simple appui et la durée d'activité du TS. En outre, un coefficient de régression  $r^2=0,70$  a été trouvé entre la durée de simple appui et la longueur du pas. L'analyse de variance a montré une augmentation significative du CoM-CoP gap et donc du couple de déséquilibre au foot-contact lorsque les participants marchaient plus rapidement (Voir figure IV.2).

En regroupant les résultats des deux expérimentations, nous pouvons conclure qu'en modulant la durée de l'activité du TS, le CNS peut moduler les deux variables cinématiques

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<sup>3</sup> D'autres variables ont été mesurées mais ne sont pas reportées dans le sommaire.

<sup>4</sup> Le  $r^2$  a été calculé entre la durée de simple appui et la durée d'activité de SOL. Cependant, l'analyse de variance a montré que la durée d'activité de SOL est similaire à celles de GM et GL (ceci est valable pour les deux protocoles GI and BR)

globales de la marche qui sont la longueur de pas et la cadence. Le contrôle temporel du TS permet également de déterminer le CoM-CoP gap, qui à son tour module le couple de déséquilibre, faisant ainsi varier la vitesse de marche. Enfin, un contrôle en amplitude du TS de la part du CNS permet de freiner la chute du CoM à la fin de la phase de simple appui.

En conclusion, les résultats obtenus pendant ce travail montrent comment le SNC à travers une succession de contrôles, contrôle de l'activité du TS, contrôle de la durée du simple, contrôle du gap et contrôle du déséquilibre, « joue » avec la gravité pour marcher « à la demande », c'est-dire adopter toutes les façons de marche.



## ii. Sommario

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Il cammino è una funzione cardinale nell'attività umana. L'andatura bipede è vantaggiosa nella misura in cui libera le braccia durante il marcia. Tuttavia, essa implica la necessità di contrastare l'attrazione della gravità per mantenere l'equilibrio del corpo durante il suo movimento.

Mentre è evidente che la marcia necessita l'opportuna orchestrazione dell'attività dei muscoli dei membri inferiori che generano e controllano i momenti articolari dell'anca, del ginocchio e della caviglia, la caviglia della gamba di appoggio è l'unica articolazione che interfaccia il corpo con il suolo durante la fase di appoggio del piede. Questo fatto rende i muscoli flessori plantari i migliori candidati per la propulsione del corpo ed il mantenimento dell'equilibrio durante il cammino. Il ruolo posturale del tricipite surale (TS) nel mantenimento dell'equilibrio durante la stazione eretta è ben stabilito (Schieppati et al., 1994; Morasso & Schieppati, 1999; Sozzi et al., 2013). Invece, le modalità di partecipazione del tricipite nel controllo dell'equilibrio dinamico del corpo durante la marcia e nella generazione delle forze propulsive è ancora un soggetto di dibattito. Tra l'altro, non è ancora chiarita la funzione del muscolo nella propulsione quando esso è in contrazione concentrica durante la fase finale di singolo appoggio.

Alcuni autori suggeriscono che il TS sia responsabile della spinta ("push-off<sup>5</sup>") che si verifica alla fine della fase di singolo appoggio del piede, poco prima che la gamba pendolare tocchi il suolo. Una ipotesi è che l'aumento dell'attività del TS alla fine della fase di singolo appoggio serva a incrementare l'accelerazione del centro di massa (CoM) (Winter, 1983; Neptune et al., 2001 ; McGowan et al., 2008). Altri autori sostengono che il ruolo principale del TS sia di resistere alla rotazione della tibia sulla articolazione tibio-tarsica, permettendo così di mantenere l'equilibrio. Quindi, l'aumento dell'attività del TS durante la fase finale del singolo appoggio resulterebbe del fatto che la forza necessaria per mantenere l'equilibrio aumenta considerabilmente quando il CoM si allontana dalla gamba di appoggio (Simon et al., 1978; Murray et al., 1978; Sutherland et al., 1980).

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<sup>5</sup> Il push-off indica l'aumento della forze anteroposteriore et verticale che si produce verso la fine della fase d'appoggio.

L'obiettivo principale degli studi descritti in questa tesi è stato rivelare il ruolo funzionale del TS durante il cammino e le modalità attraverso le quali questo ruolo si manifesta. Più precisamente, ci siamo domandati in un primo tempo se il TS partecipi direttamente alla generazione delle forze propulsive; secondariamente, come il Sistema Nervoso Centrale (SNC) moduli la cinematica (lunghezza del passo e cadenza) e la cinetica (forze propulsive e equilibrio) del cammino.

Le nostre ipotesi di lavoro sono state le seguenti:

- 1) Il ruolo principale del TS è di mantenere l'equilibrio posturale durante la marcia. Il TS non genera dunque direttamente le forze propulsive durante il cammino.
- 2) La modulazione della durata dell'attivazione del TS permette di controllare la durata del singolo appoggio, dunque la cadenza. Controllando la fase di singolo appoggio, il TS partecipa alla determinazione della lunghezza del passo e determina la posizione del CoM rispetto al piede di appoggio (rappresentato dal punto di applicazione della forze, ovvero il centro di pressione, CoP)
- 3) L'aumento dell'attività del TS quando si cammina velocemente e per contrastare un aumento nella necessità di frenare la caduta.

Due studi sono stati messi in opera per testare le ipotesi di lavoro per un totale di tre protocolli sperimentali (1 per il primo studio e 2 per il secondo).

Prima di presentare i due studi, è molto importante di mettere in evidenza qualche fatto sul cammino. Durante il cammino, gli oscillazioni della gamba pendolare sono quasi-ballistiche e modellizzabili come una serie di movimenti di pendoli semplici (Mochon & McMahon, 1980). Quindi tutto controllo proveniente dal SNC si esplica attraverso la gamba di appoggio. Quest'ultima e il resto del corpo (il torso e la testa) possono essere rappresentati come un pendolo inverso (Cavagna & Margaria, 1966). Il CoM gira dunque attorno al CoP per raggiungere la sua posizione più bassa al momento del contatto della gamba pendolare col suolo. Durante la marcia, e come in un sistema di pendolo inverso, l'energia potenziale e

cinetica lavorano in anti-fase (Cavagna et al., 1976), questo permette alla forza verticale<sup>6</sup> di generare una coppia articolare, chiamata coppia di disequilibrio del testo. Questa coppia genera le forze propulsive (Cavagna & Franzetti, 1986; Cavagna et al., 2000).

Per i due studi, i partecipanti hanno dovuto mettersi in piedi sulla piattaforma di forza (0,90 m×1,80 m, Advanced Mechanical Technology, Inc., 176 Waltham Street, Watertown, MA 02472-4800, USA). In seguito, è stato chiesto loro di iniziare il cammino, in modo il più possibile spontaneo (non secondo le modalità di un tempo di reazione), dopo un segnale di comando. La piattaforma di forza è stata usata per misurare i tre componenti della reazione del suolo e i tre momenti (ML, Medio-laterale; AP, Antero-posteriore; Ver, Verticale). La posizione del CoP è stata ricostruita a partire dai dati in uscita dalla piattaforma. Le attività EMG dei tre muscoli del TS (Soleo, SOL; Gastrocnemio Mediale, GM; Gastrocnemio Laterale, GL) e del Tibiale Anteriore sono state raccolte con un sistema wireless (Zero-wire, Aurion, It). Il protocollo europeo SENIAM è stato rispettato durante la posa degli elettrodi di superficie (Merletti & Hermens, 2000). I segnali analogici acquisiti dalla piattaforma e gli EMG sono stati sincronizzati e digitalizzati con una frequenza di campionamento di 1000 Hz. I dati raccolti erano poi salvati su un calcolatore e trattati off-line con un programma eseguito su Matlab 2008b (Mathworks, Natick, Massachusetts, USA).

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Nel primo studio, volevamo dimostrare che il TS non è implicato direttamente nella generazione delle forze propulsive durante il cammino. Il suo ruolo principale di mantenere l'equilibrio della persona durante la fase di singolo appoggio. Come conseguenza, questo fatto implicherebbe che la propulsione durante il cammino sia causata dalla coppia di disequilibrio gravitazionale anziché da una spinta attiva del TS.

Per testare questa ipotesi, abbiamo usato il protocollo dell'inizio del cammino (GI, Gait Initiation). Dieci volontari (9 maschi e 1 femmina) hanno partecipato all'esperimento. La loro età, massa e altezza media era di 34 anni (range: 23-54 a), 72 Kg (61-83 Kg) e 1,73 m (1,69-1,83 m), rispettivamente. I volontari hanno partecipato a 4 condizioni sperimentali. Durante il cammino, il loro peso poteva essere aumentato dall'aggiunta di un carico. L'ordine delle

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<sup>6</sup> La forza verticale è la risultante di due forze : il peso e le forze inerziali dovute all'accelerazione verticale del CoM.

condizioni era aleatorio. Le condizioni erano ripartite secondo due criteri: 2 velocità di cammino e 2 pesi diversi. Incrementando il peso dei partecipanti e chiedendo loro di mantenere la velocità di cammino costante, si forza di necessità l'aumento delle forze di propulsione. Se l'attività EMG del TS aumenta in proporzione al carico, allora ciò implica che il TS partecipi attivamente alla propulsione del corpo. Se, al contrario, l'attività EMG rimane la stessa rispetto alla condizione senza carico allora si può pensare che il 'push-off' attivo non sia necessario, il che porta a scartare l'ipotesi che il TS produca il 'push-off'. Il carico che portavano i soggetti consisteva di 2 dischi di metallo. Ognuno pesava 10 chili. I dischi erano fissati al corpo al livello addominale con l'aiuto di una cinghia (un disco era posizionato ventralmente e l'altro dorsalmente). La disposizione dei dischi era stata selezionata in un modo da non cambiare la posizione del CoM.

Le condizioni erano le seguenti:

- (S) Iniziare la marcia con una velocità spontanea.
- (S+L) Iniziare la marcia con una velocità spontanea + carico di 20 kg.
- (F) Iniziare la marcia con una velocità rapida.
- (F+L) Iniziare la marcia con una velocità rapida + carico di 20 kg.

Quindici prove erano state effettuate per ogni condizione. Una analisi di varianza a 2 fattori (velocità e peso) è stata usata per testare la differenza significativa tra le seguenti variabili: Velocità AP del CoM al 'foot-contact', lunghezza del passo, durata di singolo appoggio, ampiezza della componente AP del 'push-off', ampiezza del frenaggio verticale del CoM, ampiezza della coppia di disequilibrio al contatto del piede (foot-contact, FC), distanza del CoM al CoP al FC e durata e ampiezza dell'attività media dell'EMG del SOL, GM e GL. Tutte le analisi statistiche sono state realizzate su SPSS versione 20.0 (IBM Corp, Armon, New York, USA).

L'obiettivo principale di questo studio era di comparare l'attività EMG del tricipite durante un cammino senza e con carico a velocità costante. Prima di proseguire con l'analisi principale, abbiamo verificato che il carico non abbia nessun effetto sulla cinematica globale del cammino. L'analisi di varianza non ha mostrato alcun effetto del carico né sulla velocità AP del CoM al FC, né sulla lunghezza del passo e la durata del singolo appoggio. Lo stesso risultato vale per le due velocità di marcia (spontanea, S) e veloce (fast, F). Durante la marcia

veloce, tutti i partecipanti hanno aumentato la lunghezza del passo. I risultati si trovano nella tabella III.1.

Secondariamente, abbiamo osservato l'ampiezza dell'attività media dell'EMG di SOL, GM, GL durante l'inizio del cammino con e senza carico. Ugualmente, abbiamo comparato l'ampiezza della componente AP del 'push-off' con e senza carico. L'analisi della varianza non ha mostrato alcun effetto del carico sull'attività EMG dei tre muscoli durante entrambe le condizioni di velocità (S e F). Contrariamente, la forza di propulsione aumentava in modo significativo quando i partecipanti iniziavano la marcia con il carico (vedi figura III.3). Questo dimostra che il tricipite non partecipa attivamente nella generazione di forza propulsiva.

L'ampiezza della forza verticale registrata durante la caduta del CoM (forza di frenata) non cambiava tra le condizioni di carico, per la stessa velocità. Tuttavia, l'ampiezza della forza aumentava quando i partecipanti variavano la velocità del cammino. È stato già mostrato che camminare a velocità elevate necessita un aumento nella generazione di forze antigravitazionali (Liu et al., 2008). Coerentemente, l'ampiezza dell'attività media dell'EMG dei tre muscoli del TS aumentava quando i partecipanti camminavano più rapidamente (vedi la figura III.3). Questo implica che il ruolo principale del TS durante il cammino è di frenare attivamente la caduta del CoM, come inizialmente prospettato da Chong et al. (2009) e Chastan et al. (2010).

Inoltre, abbiamo esaminato la coppia di disequilibrio per capire la causa dell'aumento delle forze propulsive durante il cammino. La coppia di disequilibrio è stata calcolata come il prodotto di CoM-CoP e la forza verticale applicata al CoM. L'analisi della varianza non ha mostrato alcun effetto del carico su CoM-CoP all'istante del FC. Invece, l'ampiezza della coppia di disequilibrio aumentava significativamente quando i soggetti camminavano con il peso aggiunto (Vedi la figura III.4). La causa dell'aumento della coppia di disequilibrio è dunque dovuta al peso del carico che è stato aggiunto. Semplicemente, il peso supplementare ha aumentato la forza verticale risentita dal CoM e di conseguenza ha aumentato la coppia di disequilibrio.

Secondo Cavagna et al. (1976), la coppia di disequilibrio genera delle forze di propulsione. Guardando la figura III.4, è possibile notare che la somiglianza tra il profilo della curva delle

forze propulsive e la coppia di disequilibrio. Le due curve situate nella figura III.4 suggeriscono anche la causa del ‘push-off’ in senso postero-anteriore alla fine della fase di singolo appoggio. Durante questo periodo, il CoM sta accelerando nel piano sagittale. Il CoP, che all’inizio della fase di singolo appoggio è situato nella parte posteriore del piede (verso il tallone), inizia ad avanzare verso la punta del piede. Alla fine della fase di singolo appoggio, il CoP non può più avanzare perché si trova bloccato sotto le dita del piede, mentre il CoM può proseguire la sua corsa incrementando la variabile CoM-CoP.

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Durante il cammino normale capita spesso di accelerare il passo, i.e. di aumentare la velocità di progressione oppure di camminare più lentamente. Dalla relazione  $V = L \times F$ , (dove V, L e F riflettono rispettivamente la velocità, la lunghezza del passo e la frequenza oppure cadenza del passo), sembra che esistano infinite possibili configurazioni cinematiche del cammino. Le relazioni esplicitante queste differenti configurazioni possono essere trovati negli articoli di Alexandre (1984), Nilsson et al. (1985), Leurs et al. (2011) e Ivanenko et al. (2011). In queste pubblicazioni gli autori non hanno esaminato le relazioni cinematiche con l’attività EMG. Durante la marcia normale, la velocità e la lunghezza del passo co-variano linearmente, la frequenza appare come un parametro di aggiustamento. Tuttavia, si può considerare che le configurazioni estreme del cammino siano la marcia lenta a grandi passi e la marcia rapida a piccoli passi. L’obiettivo di questo secondo studio è di mostrare come il CNS opera attraverso la modulazione dell’attività EMG del TS per ottenere tutte le condizioni del cammino.

È stato già dimostrato che la durata e l’ampiezza dell’attività EMG del TS aumenta progressivamente quando si cammina a velocità elevata (Winter, 1983; Der Otter et al., 2004). Tuttavia i risultati dello studio precedente hanno chiaramente mostrato che il TS non genera direttamente la forza di propulsione durante il cammino, cioè la forza che esso produce non si trasferisce direttamente nella la velocità del cammino. Nonostante questo, l’attività del TS della gamba d’appoggio non può che essere responsabile della modulazione delle parametri cinematici della marcia, secondo i principi della meccanica che stabiliscono che le forze di propulsione non possono essere generate se non c’è un appoggio. Questa ambiguità - appoggio (interfaccia piede-suolo)/ attività EMG gamba di appoggio - non esiste se si tengono in considerazione i risultati dello studio precedente. In effetti, le forze propulsive sono

generate attraverso la coppia di disequilibrio causata dalla caduta del CoM durante la fase di singolo appoggio.

Nello secondo studio, abbiamo quindi postulato che siccome il tricipite è responsabile del sostegno del peso del corpo, allora la modulazione della durata dell'attività del tricipite permette al SNC di determinare le due componenti cinematiche principali che sono la velocità e la lunghezza del passo, oltre alla cinetica (le forze di propulsione). In altri termini, crediamo che siccome la forza generata dal tricipite è antigravitazionale, allora modulando la durata dell'attività del TS dovrebbe permettere al SNC di controllare il momento al quale la gamba controlaterale arriva a toccare la terra. Dunque, in questa maniera si potrebbe controllare la cadenza, ovvero la durata dello singolo appoggio. Inoltre, il CoM viaggia con una certa velocità antero-posteriore al momento del FC e continua ad accelerare fino al contatto con la terra sotto l'azione della coppia di disequilibrio. Di conseguenza, controllando la durata della fase di singolo appoggio dovrebbe permettere al tricipite di determinare anche la distanza trascorsa dal centro di massa del corpo rispetto al punto di appoggio (il piede a terra durante fase di appoggio). In altri termini, ciò determinerebbe nello stesso tempo la lunghezza del passo e il CoM-CoP. Quest'ultimo permetterebbe di modulare la coppia di disequilibrio e dunque la forza di propulsione e con ciò la velocità di progressione.

Due esperimenti complementari sono stati eseguiti al fine di testare questa ipotesi. Diciannove volontari composti di 9 femmine e 10 maschi hanno partecipato a questo esperimento. La loro età, massa e altezza media era di 25 anni (range: 20-29), 72 kg (48-92) e 1,73 m (1,61-1,83), rispettivamente. Undici volontari hanno partecipato al primo esperimento ed altri undici al secondo.

- Il protocollo dell'inizio del cammino (GI, Gait Initiation) è stato scelto per il primo esperimento, che aveva per obiettivo la dimostrazione che la modulazione temporale dell'attività del TS determina la durata del singolo appoggio.

- Il protocollo del recupero dell'equilibrio (BR, Balance Recovery) è stato scelto per il secondo esperimento, che aveva per obiettivo di esaminare se la modulazione temporale dell'attività del TS determina la lunghezza del passo.

Per il primo esperimento, i partecipanti hanno eseguito tre condizioni sperimentali:

- (Normal) Inizio del cammino con una velocità spontanea o normale.
- (Slow) Inizio del cammino con una velocità lenta.
- (Fast) Inizio del cammino con una velocità rapida.

Dodici misure sono state effettuate per ogni condizione. È stato indicato ai partecipanti di mantenere una lunghezza del passo sempre costante. Secondo la formula  $V = L \times F$ , (dove V, L e F riflettono rispettivamente la velocità, la lunghezza del passo e la frequenza oppure la cadenza del passo), chiedendo ai partecipanti di mantenere costante la lunghezza del passo, li si obbliga a modulare la durata di singolo appoggio per poter variare la velocità di progressione. Se nostra ipotesi è valida, allora la durata dell'attività EMG del TS deve co-variare con la durata di singolo appoggio.

Per il secondo esperimento, i partecipanti hanno eseguito tre condizioni sperimentali:

- (Normal) Recupero dell'equilibrio con passi normale.
- (Short) Recupero dell'equilibrio con passi piccoli.
- (Long) Recupero dell'equilibrio con passi lunghi.

Dodici prove sono state compiute per ogni condizione. Il protocollo BR consisteva nel provocare una caduta in avanti a partire da una postura iniziale inclinata del soggetto. L'istruzione impartita era di riprendere l'equilibrio evitando la caduta ed eseguendo un passo e di continuare a camminare. La postura inclinata del partecipante era ottenuta con l'aiuto di un dispositivo composto da un sistema elettro-meccanico, un cavo di metallo e una cinghia addominale. Il partecipante, agganciato a livello addominale, era legato al sistema elettro-meccanico per mezzo del cavo. Il sistema elettro-meccanico è composto da un dinamometro solidale ad un elettromagnete che può scorrere lungo una colonna metallica fissata al muro. Per fare sì che la forza di trazione del cavo non modificasse il peso del soggetto, una attenzione particolare è stata data all'orizzontalità del cavo. Il dinamometro permette allora di calcolare l'inclinazione del soggetto.

Il vantaggio del protocollo di BR è che la velocità AP del CoM al FO rimane costante quando i partecipanti eseguono passi di lunghezze diverse, perché essa dipende solamente dalla inclinazione del corpo (Do et al., 1982). Questo protocollo permette allora di isolare l'effetto della velocità del CoM che precede il FO nella determinazione della lunghezza del



passo. Se la nostra ipotesi è corretta, allora controllando la durata del singolo appoggio, il tricipite partecipa ugualmente alla determinazione della lunghezza del passo.

Come nello studio precedente, i partecipanti hanno effettuato i loro compiti su una piattaforma di forza (0,90 m x 1,80 m, Advanced Mechanical Technology, Inc., 176 Waltham Street, Watertown, MA 02472-4800, USA). Per GI, era stato loro chiesto d'iniziare la marcia spontanea. Per BR, l'angolo di inclinazione dei partecipanti variava attorno a 17°; i partecipanti non erano informati di quando il cavo sarebbe stato rilasciato.

Un'analisi della varianza ad un fattore (velocità per GI e lunghezza del passo per BR) è stata usata per testare le differenze significative tra condizioni (lunghezza del passo o velocità della marcia) per le variabili seguenti: velocità AP del CoM al FC, lunghezza del passo, durata di singolo appoggio, ampiezza della componente AP del 'push-off', ampiezza della frenata verticale del CoM, ampiezza della coppia di disequilibrio al FC, CoM-CoP gap al FC e ampiezza media dell'attività EMG di SOL, GM e GL<sup>7</sup>. Il metodo di Pearson del calcolo di regressione è stato usato per permettere di mettere in evidenza la relazione tra la durata di singolo appoggio e la durata del TS in GI in una parte e la relazione tra la durata di singolo appoggio e la lunghezza del passo in BR da un'altra. Tutte le analisi statistiche sono state fatte con SPSS versione 20.0 (International Business Machines Corporation, Armonk, New York, USA).

Per GI, la durata di singolo appoggio aumentava nella condizione 'Slow' e diminuiva nella condizione 'Fast'. Il coefficiente di determinazione tra la durata del singolo appoggio e la durata dell'attività del tricipite era  $r^2=0,63^8$ . L'analisi della varianza ha mostrato che l'ampiezza della frenata verticale del CoM e l'ampiezza dell'attività media del TS aumentavano significativamente quando la velocità del cammino aumentava. Finalmente, l'analisi di varianza ha ugualmente rivelato un aumento del CoM-CoP e dunque del torque di disequilibrio al FC quando i partecipanti camminavano più rapidamente (Vedi la figura IV.1).

Per BR, la durata del singolo appoggio diminuiva nella condizione 'short' e aumentava nella condizione 'long'. Un coefficiente di determinazione di  $r^2=0,91$  è stato ottenuto tra la durata di singolo appoggio e la durata dell'attività di TS. Inoltre, un coefficiente di determinazione di

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<sup>7</sup> Altri variabili sono state misurate ma non sono riportate nel sommario.

<sup>8</sup> Il  $r^2$  è stato calcolato tra la durata di singolo appoggio e la durata di attività di SOL. Tuttavia, l'analisi di varianza ha mostrato che la durata di attività di SOL è uguale a quella di GM et GL (questo è vero per i due protocolli GI and BR).

$r^2=0,70$  è stato trovato tra la durata del singolo appoggio e la lunghezza del passo. L'analisi di varianza mostra un aumento significativo del CoM-CoP gap e dunque della coppia di disequilibrio al foot-contact quando i partecipanti camminavano rapidamente (Vedi Figura IV.2).

Raggruppando i risultati dei due esperimenti, si può concludere che modulando la durata dell'attività del tricipite, il CNS può modulare le due variabili cinematiche globali del cammino che sono la lunghezza del passo e la cadenza. Il controllo temporale del TS permette pure di modulare la coppia di disequilibrio, facendo in conseguenza variare la velocità del cammino. Infine, il controllo in ampiezza del TS da parte del CNS permette di frenare la caduta del CoM alla fine della fase di singolo appoggio.

In conclusione, i risultati ottenuti durante il lavoro mostrano che l'Uomo si è evoluto fisiologicamente in una maniera a sfruttare la forza di gravità per propulsarsi. Durante il cammino, il TS produce forze antigravitazionali per determinare la cinematica e cinetica della marcia nel piano sagittale.

# I. Introduction

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Bipedal walking is one of the cornerstones of human evolution. Fossil evidence shows that it goes back many thousands of years before the appearance of *Homo sapiens* (Raichlen et al., 2010). Bipedalism allowed hominids to become very efficient hunter-gatherers by means of extended-limb bipedalism that is more energetically economical than ape-like bipedalism, liberates the arms during locomotion for picking and transporting objects and raises the field of vision for locating food and danger.

Locomotion is by definition the process of transporting a body from one position to another. To do so Newton's laws of motion require a propulsive force to be applied to a body. Bipedal walking also requires the central nervous system (CNS) to control body equilibrium in order to avoid that the body collapses under the effect of gravity. Furthermore, both tasks of propelling the body and maintaining equilibrium have to be done at low metabolic cost. Otherwise, we would not be able to drag and lift our bodies throughout the day as we do so often.

Human walking is undoubtedly the result of intricate orchestration of motor commands that flow continuously through a complex redundant neural network that maintains posture and drives muscles across the ankle, knee and hip joint. Control over the ankle joint is undoubtedly the most important since it is the only articulation that interfaces between the body and the ground. This makes ankle plantarflexors excellent candidates for being propellers and regulators of equilibrium during gait. Thus, comprehending the functional role of plantarflexors is fundamental for understanding human gait in its globality. The plantarflexors muscles in humans are many: triceps surae (TS), flexor hallucis longus, flexor digitorum longus, tibialis posterior and plantaris. However, TS is certainly the most dominant of the plantarflexors. For this reason, TS is very well documented in literature regarding the biomechanics and the neuroscience of human gait.

Nonetheless, the role of TS in human gait is very hard to study. Gait is a complicated and coordinated movement where many variables are intertwined together. Current technology does not provide instruments capable of directly measuring muscle force in-vivo non-

invasively during gait in humans (see Proske et al., 1984, in the cat). Scientists have thus long debated about whether TS propels the body in late stance other than maintaining balance.

The aim of this dissertation is to uncover the functional role of the TS during gait.

When studying gait it is important to keep in mind the wise words of Bernstein (1976), who stated that locomotion is not purely due to neural activity, but also subject to biomechanical constraint and the interaction of the body with the environment. This triggered the creation of a joint project between the University of Paris-Sud and the University of Pavia. The purpose of the collaboration was to put into practice protocols that can allow both the neuroscience and biomechanics disciplines to put gait under the microscope and dissect out variables that could suggest the physiological function of TS. The project was created and guided by Professor Manh-Cuong Do and codirected by Professor Marco Schieppati.

Consequently, two studies have been conducted. In the first study, we examined whether TS played an active role in propelling the body forward. We also investigated how propulsion is generated in gait. In the second, we explored how the central nervous system (CNS) modulates TS activity in order to determine global kinetics and kinematics of gait.

- The first study is entitled: The functional role of the triceps surae muscle during human locomotion. This study was published in the journal PloS One.

- The second study is entitled: By counteracting gravity triceps surae sets both kinematics and kinetics of gait. This study has been submitted to the Journal of Physiology and is under review.

In the course of the thesis, our team has managed to publish another paper that dealt with the role of tibialis anterior, soleus and peroneus longus in balance control during tandem stance. This study puts forward the balance control role of soleus in maintaining balance. The study has been excluded from this dissertation. Nonetheless, interested readers could refer to (Sozzi et al., 2013) and explore the article.

Hence, this dissertation is composed of six main chapters:

- The second<sup>9</sup> is the state of the art chapter in which a review of the literature concerning TS and gait is provided. This chapter is composed of four subsections where the anatomy of TS is briefly considered, and is followed by a short outline of neural physiology and the main circuitries that are involved in controlling the TS. Following is a section that goes through a brief description of gait biomechanics. Then a short review of the main literature debating the role of TS in gait is provided. Finally the chapter funnels into the main hypotheses that were tested in this dissertation.
- Study one is found in the third chapter. It is composed of five subsections: introduction, material and methods, results and discussion and summary.
- Study two is found in the fourth chapter. It is composed of five subsections: introduction, material and methods, results and discussion and summary.
- The fifth chapter is the general discussion. In this chapter, the results of both studies are restated and discussed in terms of strengths and weaknesses in order to provide the reader with a conclusive answer on the function of TS during gait.
- The dissertation is concluded in the sixth chapter where the perspectives of the research and its clinical applications are discussed.

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<sup>9</sup> The first is the current chapter.

## II. State of the art

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The purpose of this chapter is to introduce the work postulates. To do so properly, one has to know what is triceps surae, how it is controlled, what is the existing literature concerning its function in gait and finally what is the biomechanics involved in gait. Thus, the chapter will go through all of these issues one at a time and conclude with the work hypotheses.

### II.1 Physiology and motor command of triceps-surae

Triceps surae is composed of the soleus and the gastrocnemius lateralis and gastrocnemius medialis muscles. Proximally, the soleus arises from the head and upper dorsal surface of the fibula and from the tendinous arch between the head of the fibula and the tibia. The gastrocnemius is composed of two heads. It arises proximally to the medial femoral condyle with a medial head and with a lateral head proximal to the lateral femoral condyle. Both head of the gastrocnemii join the tendon of the soleus and are inserted into the tuber calcanei via their aponeuroses to ultimately insert on the calcaneus (Platzer, 2008).

The most straightforward difference between the two muscles is that soleus is a mono-articular joint while the gastrocnemii are bi-articular around the knee. At the ankle level, the combined forces of the three heads of the TS are transmitted to the Achilles tendon in order to plantarflex the foot. The range of amplitude of the force, of healthy young adults, applied to the Achilles tendon during gait, has been estimated to be around 1400-2600 N (Finni et al., 1998; Giddings et al., 2000). The gastrocnemius also flexes the knee (Li et al., 2002).

In mammals, the predominant muscle fibres in soleus are of type I or slow twitch fibres and on average constitute 80% of muscle fibres (range: 64 to 100%). In contrast, the gastrocnemii are composed only of 54% slow twitch fibres (range: 34 to 82%) (Gollnick et al., 1974). The tibial nerve innervates the TS. The efferent neurons composing the tibial nerve and innervating the TS originate from the roots of L2 to S2 vertebrae. Mapping of neuronal activity in the spinal cord can be found in Capellini et al. (2010)<sup>10</sup>.

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<sup>10</sup> That paper showed that in gait biomechanical mechanisms are tightly correlated with specific modes of progression of motor pool activity situated in the spinal cord.

Motorneurones (MNs) is the term given to efferent neurons that are located inside the CNS with axons projecting outside innervating the muscles. Lower motorneurones<sup>11</sup> (MNs) exist in three main categories: alpha, gamma and beta.

- Alpha motorneurones ( $\alpha$ -MNs) are the largest of MNs. They innervate skeletal (extrafusal) motor units. Activation of  $\alpha$ -MNs directly causes muscle contraction.  $\alpha$ -MNs also have the highest conduction velocity between all MNs.
- Gamma motorneurones ( $\gamma$ -MNs) innervate muscle spindles. Their co-activation keeps the equatorial part of the muscle spindles under stress, which allows  $\alpha$ -MNs to continue muscle contraction in spite of muscle shortening.  $\gamma$ -MNs inhibition is also adjusted to muscle spindle sensitivity (Burke et al., 1979).
- Beta motorneurones ( $\beta$ -MNs) innervate both the intrafusal fibres of muscle spindles and extrafusal fibers (Manuel & Zytnicki, 2011).

Muscle fibres largely outnumber MNs, so individual motor axons of a single  $\alpha$ -MN synapse on many fibres across one muscle to form a motor unit (Sherrington, 1929). This means that the smallest unit of force generated by a muscle is due to a single action potential coming from a single  $\alpha$ -MN. To increase muscle force, CNS has to simply activate more  $\alpha$ -MNs and/or to increase the frequency of activation of motoneurons (Matthews 1933; Rothwell, 1994). Motor units vary in size depending on the type of fibres it innervates. Small motor units innervate slow fibres while larger  $\alpha$ -MN innervate the larger and faster fibres. The changes of motor unit types allow the CNS to cope with different movements during diverse circumstances. Small, slow, non-fatigable motor units have lower activation thresholds, which allows for tonic activity over long periods of time such as in quiet stance. Larger and faster motor units have higher activation thresholds and allow for rapid movements requiring great force are made, such as jumping. The soleus muscle is innervated mainly via slow motor units that have an average innervation ratio of 180 fibre/ $\alpha$ -MN. In contrast, the gastrocnemius muscle includes both small and larger motor units. It has an innervation ratio of 1000–2000 fibre/ $\alpha$ -MN. Subtle variations are present in athletes on different training regimens such as marathon runners versus sprinters (Purves et al., 2001).

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<sup>11</sup> Lower motor neurons is a term sometimes used to designate MNs located in the brainstem and in the spinal cord that project to muscle fibres.

### *Organisation of the circuitry commanding triceps surae*

Bernstein (1967) stated in his classical study that neural control based solely on feed-forward commands is not sufficient to deal with the changing contingencies of the natural environment. Since then, a common consensus had united neuroscientists investigating the neural circuitry involved in human walking. This can be summed up by the following quote stated by Chiel et al. (2009): *“In gait, control may be shared between the nervous system and the periphery, that neural activity organizes degrees of freedom into biomechanically meaningful subsets, that mechanics alone may play crucial roles in enforcing gait patterns, and that mechanics of sensors is crucial for their function.”*

Indeed, during gait the nervous system groups together degrees of freedom (Bernstein, 1967). This must be achieved in interaction with the complex biomechanics of the body and an irregular and changing environment. Thus, the CNS must organize the degrees of freedom in a biomechanically and ecologically efficient way. In addition, the CNS has to rely on afferents coming from mechanical sensors in order to orchestrate the firing rate of  $\alpha$ -MNs.

It is commonly believed that the main global circuits involved in gait are:

1. Rhythmic neural networks commonly known as central pattern generators (CPG) (Dietz, 2003).
2. Descending control signals from supraspinal structures (Barthélemy et al., 2011).
3. Ascending tracts from the periphery (Nielsen & Sinkjaer, 2002).

1. Central pattern generator is the term given to a neural network that is organised at the spinal level and capable of commanding  $\alpha$ -MN in order to produce rhythmic movements. Sherrington (1910) firstly hinted at the evidence of CPGs when he demonstrated that cats, made decerebrate by cutting the spinal cord at the level of the brain stem, could perform rudimentary stepping movements. A year later, Brown (1911) made similar observations, using decerebrate cats that also had undergone transection of the spinal cord at T12 and deafferentation by cutting the afferent nerves from the hind-limb muscles. Grillner (1975) successfully mapped the CPG in the lamprey. Later, Rossignol et al. (1996) proved the existence of CPGs in lower vertebrates and cats.

However, locomotion has never been observed in humans following complete spinal cord transection. Nonetheless, indirect evidence of its existence in humans was shown in patients



suffering spinal cord injuries and infants (Calancie et al., 1994; Dimitrijevic et al., 1998; Yang et al., 1998). Finally, a case-study published by Harkema et al., (2011) seems to confirm the presence of CPGs in human. In their case study, they showed that it is at least possible for a patient suffering from an injury at the C7/T1 level to regain the ability to successively stand and step by means of chronically stimulating the spinal cord at the L1-S1 level by using a 16-electrode array. Conclusively, even in the case of the lack of direct evidence, neuroscientists generally agree on the existence of CPGs in the human spinal cord (Dietz, 2003).

2. Supraspinal control makes common sense since effective locomotion in a natural environment requires slowing down, accelerating, stopping and initiating gait again. Thus, CPGs require a central command in order to change the pattern, frequency and amplitude of extensor/flexor activity. Furthermore, in the occurrence of injury to one or both limbs, the CPG system should be modified or by-passed in special cases. We also don't always walk in a straight line, which requires supraspinal control to exploit CPG networks, (Courtine & Schieppati, 2004). Indeed, descending pathways from the brain can influence locomotor performance by acting either directly (i.e. mono- or oligosynaptically) on  $\alpha$ -MN or indirectly via an influence on the CPGs. (Armstrong 1988; Eidelberg et al., 1981; Fedirchuk et al., 1998; Cheron et al., 2011; Le Ray et al., 2011; Haefeli et al., 2011; Petersen et al., 2012).

Studying supraspinal control is a very difficult task since traditional cortex imaging methods such as functional magnetic resonance imaging and magnetoencephalography cannot be used (Jahn & Zwergal, 2010). To overcome these problems others methods have been developed such as positron emission tomography and single photon emission tomography, which estimates the flow of blood in certain regions of the brain (Fukuyama et al., 1997) and EEG (Cheron et al., 2011). A review explaining the roles of the brainstem, cerebellum and motor cortex in locomotion can be found in Armstrong (1988). In this dissertation, I shall not go into details about the supraspinal control, since it goes beyond the scope of my researches.

3. It is well established that CNS relies on sensory information gathered mainly from the vestibular, visual and somatosensory systems in gait (Duysens et al., 2000; Bent et al., 2004; Clarac, 2008; Ruget et al., 2008; Sinkjaer et al., 2010). The labyrinth of the inner ear detects head motion and spatial orientation and relays that information to the vestibular nuclei in the brainstem and the cerebellum. It plays an important role in erect posture (Chester, 1991; Horak, 2010). It has been shown that signals coming down from vestibular apparatus might play a role in step making (Bent et al., 2002) and gait (Bent et al., 2000). Vision is for obvious

reasons involved in orientation during walking. It has been shown that visual input helps stabilize subjects in erect posture (Collins & De Luca, 1995). However, the roles of vision and vestibular input in controlling TS in gait is not yet very well understood (see Rougier et al., 2003; Jahn et al., 2004). Thus, this section mainly deals with the afferent signals originating from the somatosensory system and more precisely proprioception.

The literal meaning of proprioception is “sense of self”. It is commonly used to indicate the capability of the CNS to sense the position and orientation of body segments in space (Prochazka, 1996; Proske & Gandevia, 2012). The main sensors responsible for proprioception are muscle spindles, Golgi tendon organs, joint receptors and stretch sensitive skin receptors.

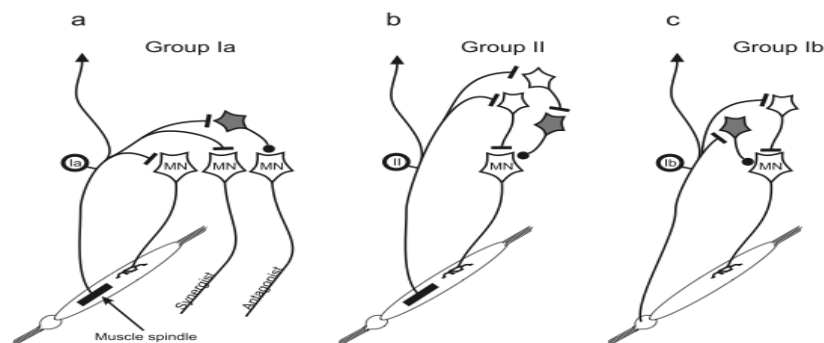
Muscle spindles are located amongst the muscle fibres. Many small intrafusal fibers are found within muscle fibres. When the muscle lengthens and the muscle spindle is stretched, this opens mechanically-gated ion channels in the sensory dendrites, leading to a receptor potential that triggers action potentials in the muscle spindle afferent. The afferents are the primary (group Ia) and secondary (group II) myelinated sensory axons (Prochazka, 1996). Group Ia afferents are fast-conducting fibres, mostly velocity sensitive, whereas group II afferents are slow-conducting and more sensitive to length changes (Purves et al., 2001).

Golgi tendon organs are the primary muscle force sensors in the body. They are located in the musculo-tendinous junction or in the tendinous inscription in series with muscle fibres (Jami, 1992). When muscles contract, collagen fibrils inside the tendon are pulled tight, and activate group Ib afferents. Houk & Henneman (1967) showed that group Ib afferents modulate their firing rates more when the load changes than for the static load. The Golgi tendon organs have an autogenic disynaptic inhibitory pathway to the motoneurone and since they have a high threshold of activity to impose muscle stretches in relaxed muscles, it was first thought that Golgi tendon organs worked as overload sensors.

In the human skin there exists a variety of different mechanoreceptors (Macefield, 2005). The main receptors are Meissner’s corpuscles, Merkel disks, Follicle endings, Ruffini endings, Pacinian corpuscles and nerve endings. This proprioceptive role of skin afferents also translates to the human ankle joint; where the population vector (Georgopoulos et al., 1986) of in vivo recorded cutaneous afferents was correlated with ankle position (Aimonetti et al., 2000). Though there is increasing evidence involving skin afferents in human proprioception,

the involvement of cutaneous feedback in the motor control of the lower leg muscles in walking human is still not fully understood (Sayenko et al., 2009). However, it has been shown that in locomotion as compared to standing the detection threshold of cutaneous input increases by 30%. Furthermore, ipsi and contralateral decreases in sensitivity of cutaneous input (Duysens et al., 1995). Proprioceptive feedback has been implicated in switching between phases (Duysens & Pearson, 1980), enabling rapid corrective response to mechanical perturbations (Dietz et al., 1984; Yang et al., 1991; Sinkjaer et al., 1996), contributing to the locomotor activity (Sinkjaer et al., 2000; Grey et al., 2004; Grey et al., 2007), and modulating the locomotor activity (Schieppati & Nardone, 1999; Mazzaro et al., 2006; Stephens & Yang 1999; Bachmann et al., 2008; Nardone et al., 2013). Finally, Altenmüller et al. (1995) showed that a modulatory effect is produced by locomotion on somatosensory input at a cortical level. More precisely, the excitability of the somatosensory cortex is increased during the middle and late swing phases and decreased during the support phase.

The afferent neurons connect with the efferent MN through mono- and oligosynaptic connections in the spinal cord. A schematic view of main features of the spindle and Golgi tendon organ pathways is presented in Fig II.1. It is highly possible that the afferent-efferent interconnections interact with the rhythm generating network or the remnant of it in humans. However, proving it could require a complete mapping of the very complex and rich spinal cord network. Nonetheless, extensive work is being done in order to map the major afferent projections involved in afferent feedback (Jankowska, 1992; Rossignol et al., 2006).



**Figure II.1 Schematic view of the main features of group Ia, II and Ib projections on the homonymous motoneuron (af Klint, 2009).**

a) The group Ia pathway originates in the primary endings of the spindle. The Ia fibre, sensitive to velocity of spindle lengthening, has an excitatory monosynaptic projection to the homonymous MN and synergists MN. It also inhibits antagonist MN. Group Ia is these under control from supraspinal centres. b) The group II pathway also originates from the muscle spindles but the secondary endings are mostly length sensitive. The group II fibres projects on the homonymous MN through excitatory disynaptic pathway and a trisynaptic inhibitory pathway. c) The group Ib pathway originates from the Golgi tendon organ. During walking the group Ib pathway has a predominant excitatory disynaptic connection to the homonymous MN. It has been suggested that also indirect excitatory connections exist through the CPG (Gossard et al. 1994).

Understanding the role of afferent feedback in locomotion is very difficult due to the fact that non-invasive direct measurement is not possible to perform on humans using the actual technology. Still, the concerned literature is rich with scientific articles providing indirect knowledge obtained via animal (notably cats) and human experiments. Forssberg et al. (1980) showed that kittens with complete transection of the spinal cord could adapt the phase transition to the speed of a treadmill. Other experiments performed on cat with complete spinal cord transection suggested that group I afferent from leg extensors (Duysens & Pearson, 1980; Conway et al., 1987; Whelan et al., 1995a; Whelan et al., 1995b) and length sensitive afferents coming from hip-flexors (Grillner & Rossignol, 1978; Hiebert et al., 1996) are implicated in phase transition.

Different techniques were used to give a clearer idea on the role of afferent feedback on ankle extensors during human locomotion. Crenna & Frigo (1987) compared the excitability of the Hoffman-reflex with respect to the length of the soleus muscle during treadmill walking. They found that the H-reflex excitability was low during early and late-stance and high during middle-stance. Their result suggested that active afferent regulation based on soleus length affects the transmission in the soleus myotatic arc during gait. Capaday & Stein (1986) also showed that the H-reflex was highly modulated during the step cycle. The highest excitability values were recorded during the stance phase.

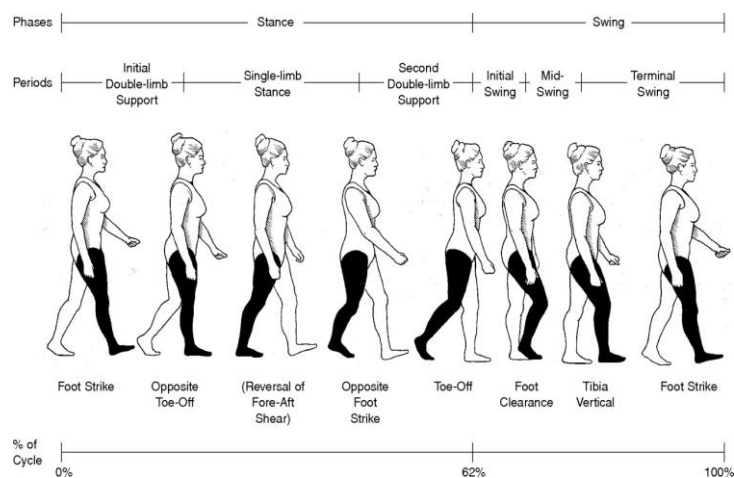
Dietz et al. (1984) applied mechanical perturbations to the subjects by applying short impulses to the treadmill, which either accelerated or decelerated the treadmill belt on which they were walking. The perturbation elicited reflex activity in the plantar flexor muscles. This clearly shows that the afferent information is taken in consideration during human locomotor activity. Faist et al. (2006) examined the group Ib pathway from gastrocnemius medialis to the soleus motoneurons. They did so by investigating the H-reflex in the Ib group afferent during sitting, lying supine, lying supine with 300 N pressure applied to the foot sole, standing, and rhythmic loaded and unloaded gait. They found that the Ib inhibition is only present when the leg is not loaded. They also suggested that the group Ib pathway may become opened during gait. Klint et al. (2010) altered bodyweight and ankle angle during treadmill walking. Their result suggested that force-related afferent feedback contributes both to the background locomotor activity and to the medium latency stretch reflex.

Conclusively, these three circuitries are all connected together to create a complex and redundant network. This allows the CNS to gain the plasticity it needs to deal with the requirements of interacting with the natural environment.

## II.2 The biomechanics of gait

Human gait can be described as a cyclic locomotive movement during which at least one contact with the ground is maintained at all time. Conventionally, a gait cycle is divided into two periods: stance and swing.

Stance designates the entire period during which the foot touches the ground. The stance phase itself is divided in two phases: single stance and double stance. Single stance is the phase where one foot is in contact with the ground. Double stance is the phase where both feet are in contact with the ground. Swing applies to the time the leading leg is in the air.



**Figure II.2 The normal gait cycle (Sutherland et al., 1994)**

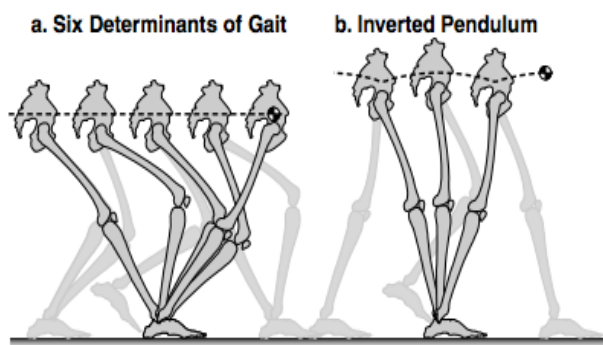
When walking spontaneously, stance phase lasts about 60% of the cycle while the swing phase lasts about 40%. Single stance usually

comprises 40% of the entire cycle and double support about 10% (Fig II.2). These proportions vary with the speed of walking (Otis & Burstein, 1981). In level walking, humans usually adopt a preferred cadence and step length (Cavagna & Franzetti, 1986).

Two contradictory gait model theories have been widely debated over by biomechanists and physiologists for the last five decades: The six determinants of gait and the inverted pendulum analogy (Fig II.3)

- The six determinants of gait theory was first introduced by Saunders et al. (1953). It is based on the principle that vertical and horizontal centre of mass (CoM) displacements are energetically costly. It proposes a set of kinematic features that help reduce the vertical displacement of body CoM that are: pelvic rotation, pelvic tilt, knee and hip flexion, knee and ankle interaction, and lateral pelvic displacement. In this theory, flexion of the knee during the stance phase and rotations of the pelvis are coordinated in order to reduce displacements of the CoM.

- The inverted pendulum theory (Cavagna et al., 1963; Cavagna and Margaria, 1966) proposes that it is energetically less costly for the stance leg to act like an inverted pendulum. During single stance, the stance leg and upper body rotate around the ankle/forefoot complex so that CoM would prescribe a falling compass trajectory. During double stance the CoM is redirected upward as the trailing leg is lifted into swing (Kuo, 2007).

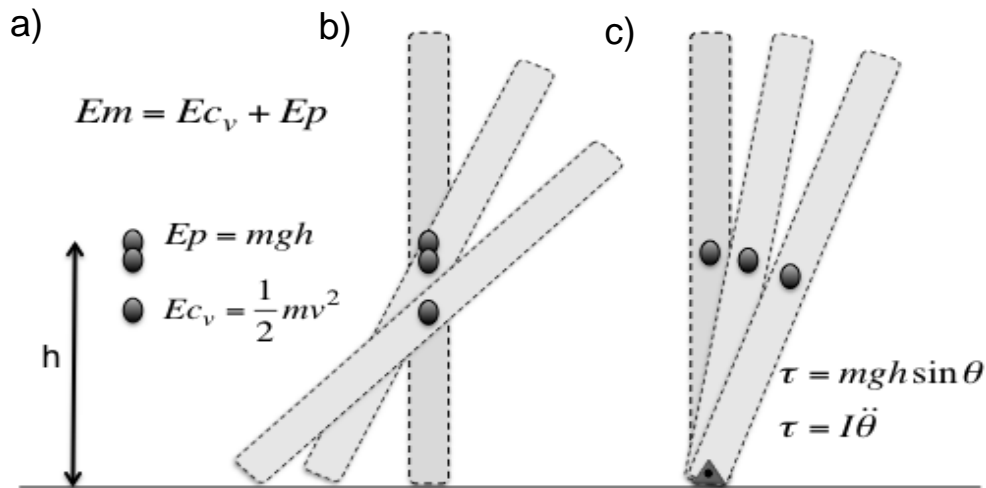


**Figure II.3 The main two theories of gait (Kuo, 2007).**

a) The six determinants of gait states that the body reduces vertical and lateral CoM displacement. E.g. knee flexion can help flatten the CoM trajectory.

b) The inverted pendulum analogy states that the stance leg is kept straight during single support, functioning like an inverted pendulum. This causes the CoM to rotate around the stance foot.

More recent studies have shown evidence in disfavour of the six determinants of gait. Three of the determinants, that are hip tilting (Gard & Childress, 1997), knee flexion during stance (Gard & Childress, 1999) and pelvis rotation about the vertical axis (Kerrigan et al., 2001), actually contribute far less than previously thought in reducing the vertical displacement of the CoM. It was also demonstrated that when humans voluntarily reduce vertical displacement of the CoM, they spend more metabolic energy when compared to their normal gait (Gordon et al., 2003; Ortega and Farley, 2005). Hence, the inverted pendulum model has become the most widely accepted gait model.



**Figure II.4 Gravity torque of an inverted pendulum**

a) When a point mass object is raised from the ground, it acquires potential energy. When it falls the potential energy is transformed gradually into kinetic energy.

b) If a rod is put vertical on a perfect frictionless ground: When perturbed the potential energy of the rod's CoM is transformed into a kinetic energy as in part a. Since the rod can slip, the CoM falling trajectory is a straight line.

c) Finally the rod is attached to the ground by means of a hinge (as stance leg is attached to the ground by means of the ankle). When perturbed the potential energy is transformed into a torque rotating around the hinge. The torque is expressed as the product of weight of the rod and the distance of the rod's CoM from the vertical. The lever arm is expressed here as  $h \sin \theta$

One of the major advantages of the inverted pendulum is that its potential energy can be transformed into forward kinetic energy. The process is explained in Fig II.4. Any point-object positioned and maintained at a certain height has a potential energy. In other terms, it has the potential to fall down. The equation of the potential energy ( $E_p$ ) is very simple:  $E_p = mgh$ , where  $m$  is mass,  $g$  is the earth gravitational constant and  $h$  is the height of the object with respect to a reference. According to the principle of conservation of energy, if the object is allowed to fall, the potential energy of the object is then transformed into vertical kinetic energy (Part A in Fig II.4). If you replace the point object by a rod and position it vertically on a very slippery surface, so that friction is negligible, it might stay initially in equilibrium. When equilibrium is perturbed, the rod will slide backwards and its CoM will exhibit the same behaviour as in the point mass explained previously (Part B in Fig II.4). Finally, if the base of the rod is fixed on a hinge, it would behave like an inverted pendulum. Again when positioned vertically, the rod will stay in equilibrium, when a gap is created between the CoM and the base of the pendulum, a disequilibrium torque is created (Part C in figure). Cavagna &

Margaria (1963) showed that, within a certain range<sup>12</sup>, the changes of CoM potential and kinetic energy of the CoM are conveniently in opposite phases. They, then, showed that the fall of CoM during single stance in human gait is capable of transforming potential energy into forward velocity (Cavagna et al., 1976; Cavagna & Fanzetti 1986; Cavagna et al., 2000).

It is important to understand the human body is not just a simple pendulum. It is composed of many segments, each linked together by a complex system of ligaments, tendons and muscles that have non-linear mechanical properties. Furthermore, the ankle/foot<sup>13</sup> system is far more complicated than a single hinge attached to the ground so the point of rotation of the pendulum is not fixed but moves along the stance foot (Wright et al., 2012). However, since the knee and hip are well extended during gait then the inverted pendulum could be fairly used to describe gait.

Finally, the swing leg acts rather like a ballistic pendulum (Mochon & McMahon, 1980). In other terms, given an initial momentum in early swing, the leading leg moves through the remainder of swing phase under the action of gravity. It is also to note that electromyographic (EMG) measurements of swing leg muscle show very little activity during gait at normal, i.e., spontaneous walking velocity (Basmajian, 1976), except for the beginning and ending of swing leg to secure soft take-off and landing of the foot and reduce metabolic cost of gait. From a mechanical point of view, it is expected that CNS controls gait mainly by modulating muscle activity only through muscles active during the stance phase since the stance leg is the limb that is interacting with the ground, i.e. generating the ground reaction forces.

McGeer et al. (1990) showed that ballistic walking based only on inverse pendulum dynamics, i.e. without motor control, could be done indeed. They demonstrated that a robot could go down a gentle slope driven by gravity alone. However, it is trivial that the human inverted pendulum requires some sort of control by the CNS. Firstly, a correct posture has to be maintained throughout gait so that the body behaves as an inverted pendulum. Secondly, if the inverted pendulum is entirely passive, i.e., is driven by the pull of gravity alone, then its

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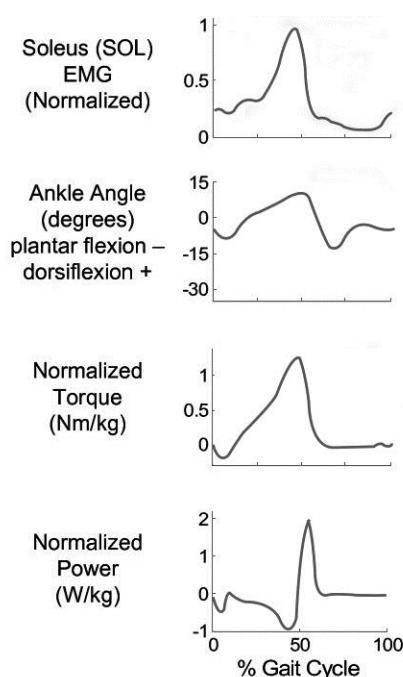
<sup>12</sup> Cavagna et al. (1976) state that the conversion of potential energy to forward kinetic energy occurs at intermediate speeds of walking and that is no longer tenable above 7 km/hr.

<sup>13</sup> The foot alone is composed of 26 bones and 33 joints



angular velocity can be only controlled by means of regulating the stiffness of its lower joint or in other terms the ankle joint. Eccentric plantarflexors contraction increases ankle stiffness by resisting the rotation of the tibia around the tarsus (Houtz & Walsh, 1959).

Stance phase is dominated by TS activity. In about 80% of stance phase, the ankle is in dorsiflexion, thus TS is in eccentric contraction. In late stance, the ankle plantarflexes and thus TS works concentrically (Rose & Gamble, 1994). The ankle torque increases and peaks in late stance and corresponds roughly with the peak of TS electromyographic (EMG) signal. The ankle power calculated as a product of the torque and the angular velocity peaks in double stance (Fig II.5). In late stance, the ground reaction force (GRF) increases in both the vertical (Ver) and anteroposterior (AP) direction (Jarrett et al., 1980). The increase in GRF in late stance is commonly referred to as push-off. Walking at higher velocities leads to an increase in ankle torque, ankle power and TS EMG activity. This led many scientists to believe that plantarflexor activity is the main cause of ankle torque and power (Winter, 1980; Winter, 1989; Müller et al., 1995). Others disagreed, which led to a long lasting debate on the functional role of the TS in gait.



**Figure II.5 Ankle angle, torque and power in addition to soleus EMG during gait (Cain et al., 2007 - Figure modified)**

It can be seen from the figure that the ankle joint is dorsiflexed from the beginning till late in the stance phase. (Stance phase lasts 60% of cycle). In late stance, ankle is then plantar flexed.

The ankle torque increases throughout the entire single stance phase. The ankle torque is negative and becomes positive only in late stance where it peaks to very high value. The high peak in angle power has been described as “push-off”.

The soleus EMG is highest in late stance. It can be seen that soleus EMG peak occurs while the ankle is still in dorsiflexion. It has been debated whether the increase in soleus activity in late stance causes push off.

### II.3 The functional role of triceps surae

Starting the early 1950s, physiologists initiated what would turn to be a long ongoing debate about whether ankle flexors provided an upward thrust or push-off during late stance. While authors such as Eberharht et al. (1954), Duboh et al. (1976) and Brandeblet al. (1977) agreed that push-off occurred after heel-off, others such as (Bresslerb & Berry, 1951; Inman, 1966, Mann et al., 1974) questioned the entire push-off concept. In addition, Perry (1974), in her book about kinesiology, advised dropping the term push-off and postulated that the late floor-reaction peak is the result of leverage exerted by body alignment rather than an active downward thrust.

Authors questioning the push-off hypothesis offered several alternative explanations. According to Houtz & Walsh (1959), the soleus, tibialis posterior, and peroneus brevis muscles stabilize the ankle by adjusting the tibia on the tarsus. Furthermore, Sutherland (1966) emphasized on the importance of the knee-ankle stability linkage, noting that the ankle plantarflexors decelerate the stance-phase forward rotation of the tibia on the talus, providing selective, more rapid extension at the knee joint. Mann et al. (1974) concluded that the plantarflexors do not actively push or propel the body forward. They stated that plantarflexors control the momentum of the gravity torque.

This explanation, while denying the validity of the push-off theory, acknowledges the contribution of the plantarflexors to increase step length. At that time, there seemed to be little dispute over the decelerative or stabilizing function of the calf muscles during forward movement of the tibia on the talus, but controversy still surrounded their accelerative function during heel-off. Part of the problem resides in the simplicity of the studied models and their difficulties in separating the contributions of individual muscle force, gravity, and kinetic energy in any analysis of gait. This problem remains, as yet, unresolved.

Pedotti et al. (1977) accentuated this problem by finding good correlation between EMG activity of ankle flexors, kinetics and kinematics during gait. Sutherland et al. (1980) explained that other factors to the already existing problem were added when attempts were made to draw conclusions from patients with paralysis of the plantarflexors. Motor paralysis from poliomyelitis or myelodysplasia frequently produced alteration of the length and weight of the shank segment and of the shape, weight, and function of the foot. Vertical alignment of

the calcaneus in the foot with a calcaneus deformity changed the axis of the subtalar joint. Constant stress on the posterior soft tissues of the ankle produces elongation of the capsule, tendons, and ligaments. Also, compensatory muscle activity partially masked the primary gait deficit. The study of patients with below-the-knee prostheses to determine the function of the ankle plantarflexor muscles had similar objections (Michel & Do, 2002). In addition to the differences in the weight of the shank, the suspension of the prosthesis may affect knee movements. Proprioception and sensation are absent and motion of the ankle joint is usually excluded by prosthetic design.

From the late 70's till late 90's, the biomechanical community was divided between three hypotheses that stated that the role of ankle flexors during normal gait is to:

1. Provide a controlled roll-off.
2. Accelerate the leg into swing.
3. Actively provide forward progression or push-off.

1. The term controlled roll-off was proposed by Perry (1992) and describes forward progression during single-leg stance as a controlled fall. Thus, the proposed primary action of the ankle plantarflexors during the controlled roll-off is to decelerate tibia rotation and prevent knee flexion as the body rotates over the stance leg. Forward progression is then the result of a passive mechanism as the body moves forward as a result of momentum and inertia. Supporting evidence for the controlled roll-off theory is found in the work of Simon et al. (1978) and Sutherland et al. (1980). They both performed tibial nerve block by injecting an anesthetic agent (six to fifteen milliliters of 1 per cent lidocaine, mepivacaine, or mepivacaine hydrochloride) into the sheath of the tibial nerve in the popliteal fossa. This process produces surface anesthesia of the sole of the foot as well as muscle paralysis. Both studies found that in the absence of normal plantar flexor activity walking velocity increased, leading them to conclude that the plantar flexors limit forward momentum rather than propel the body forward by contributing to knee stability, providing ankle stability, restraining the forward rotation of the tibia on the talus during stance phase, and thus conserving energy by minimizing vertical oscillation of the body centre of mass. To do so, plantar flexors first provide active resistance to forward rotation of the tibia via eccentric contraction, while providing increasing resistance that eventually checks further forward rotation of the tibia. They then progressively shorten (concentric contraction), reducing ankle dorsiflexion, adjusting limb length, and restraining the drop of the body centre of mass.

Furthermore, Murray et al. (1978) published a paper documenting a case study of a person who had a surgical excision of the gastrocnemius and soleus muscles. After the operation, this particular patient was able to maintain the duration of a normal stride, but walked unevenly. The step produced when the operated leg was the stance leg was shorter. She also showed an increase in dorsiflexion and diminished plantarflexion during gait. The patient compensated for the gait abnormalities by excessive lateral pelvic tilt and prolonged quadriceps activity. They argued that the results obtained in their case study were in line with those of Sutherland et al. (1980). In other terms, they explained that the excessive dorsiflexion is caused by the absence of the TS that prevents tibial rotation. However, tibial nerve blocks and excision of TS, in all three studies that were stated previously, altered walking cinematic variables such as step length, step time and joint angles. Furthermore, paralysis also affected the plantaris, tibialis posterior, flexor digitorum longus, and flexor hallucis longus muscles and altered EMG activity in other muscles. Thus, it is possible that subjects relied on a different motor program after nerve block, which makes comparisons with unaltered plantar flexor function difficult.

2. Meinders et al. (1998) performed inverse dynamics and mechanical energy analyses to show that, although the net ankle moment generated the majority of the mechanical work during the so-called push-off phase, only a small portion of this mechanical energy was transmitted to the trunk segment. Instead, their data showed that during late stance, ankle flexors generated on average 23.1J of energy that was stored in the leg and that only 4.2J were transferred directly to the trunk. This suggests that plantarflexors are not involved in push-off since their energy is not transferred to the CoM. Similarly, Hof et al. (1993) examined correlations between changes in body segment mechanical energy and work of TS group. An EMG to force processing algorithm was used to compute the work of the TS. Their results show that in normal walking ankle plantar flexors provide the major part of positive work for the initiation of swing at the push-off phase. Moreover, they found that a minor contribution to trunk forward acceleration would be supplied by ankle flexors only when a person performs large steps. Therefore, Meinders et al. (1998) and Hof et al. (1993) both concluded that the ankle plantar flexors' work is to primarily accelerate the leg into swing and that most of this energy is translated into the trunk at the end of the swing phase. However, Dillingham et al. (1992), using the inverse dynamics method as well, stated that the swing leg is controlled in its motion, serving to store and return energy to the body during the swing phase. They agreed that the swing leg was the major factor propelling the body forward, but

that push-off should not be discarded, even if its contribution is much smaller.

3. The active push-off hypothesis states that the energy generated by the plantar flexor group during late stance is transferred to the trunk to provide support and forward progression. The hypothesis gained popularity when Winter (1983), while studying the power output of the net ankle and knee joint moments during normal gait, found that the ankle moment was the primary source of positive work, and that plantar flexor activity coincided with the second peak of the vertical ground reaction force. He concluded that an active plantar flexor push-off, rather than a passive roll-off, provides forward progression. Later on, other studies identified as well strong correlations between the net ankle moment and power produced by the ankle plantar flexors and gait performance in several patient populations (Müller et al., 1995; Nadeau et al., 1999; Olney et al., 1990; Olney et al., 1994; Winter et al., 1990).

Kepple et al. (1997) performed a study based on inverse dynamics obtaining results contradicting those of Hof et al. (1993) and Meinders et al. (1998). Their results showed that plantar flexor moment was the primary contributor to the accelerations of the head–arms–trunk segment in both the horizontal (considered analogous to forward progression) and vertical (considered analogous to support) directions during the second-half of the single leg stance phase. This contradiction may be due to the fact that biomechanical analyses based on net ankle (and knee) joint moments cannot elucidate the potentially different mechanical contributions of individual uniarticular and biarticular plantar flexor muscles to the overall gait performance (e.g. support and forward progression).

Sadeghi et al. (2001) looked at the problem from another angle and performed principal component analysis on the hip and ankle moment. They found that the first two principal components contained over 70% and 85%, respectively, of the information in the ankle and hip moment curves. They interpreted their statistical analysis as follows: The first principal component reveals that the main role of the ankle and hip is to keep the body from collapsing. The second principal component is associated with the functional contribution of both ankle plantar flexors and hip flexors during the propulsion phase (50–60% of the gait cycle). A high coordination ( $r^2=0.67$ ) between the ankle and hip moments was observed. Their results however suggested that maintaining body support against gravity is the first functional task of the ankle plantar flexors and hip extensors, while contribution to propulsion would most probably be the second major role for the ankle plantar flexors and hip flexors.

In order to have a smarter understanding of the role of each individual ankle flexor muscle, scientists resorted to mathematical modelling and simulation resorting to minimization algorithm to solve a very complex mechanical problem with many unknown variables, degrees of freedoms and redundant equations. If the model is able to reproduce crucial gait parameters, such as gait speed, muscle time activation and work around the joints etc. then the functional role of all the muscles in the lower body could be faithfully predicted. However, many approximations are required in order to be able to solve these equations. Firstly, the muscle tendon complex will be modelled using Hill's equations (Hill, 1938). Furthermore, in order to reduce the required computational power to solve the problem the ankle, knee and hip articulation would be considered as normal hinges (so only flexion and extension movements are studied), friction between different segments will be considered null, ligaments would be modelled as springs, all the segments will be considered as rigid, the CoM of each segment will be determined using anthropometric data and the very complex foot architecture and the non-homogeneous surface of contact between the sole and the floor should be much simplified.

Still, Anderson & Pandy (2001) were able to describe a muscle's contribution to support by its contribution to the time history of the vertical force exerted by the ground. The analysis was based on a three-dimensional, muscle-actuated model of the body (54 muscles) and a dynamic optimization solution for normal walking. Their results showed that, in early stance, before the foot was placed flat on the ground, support was provided mainly by the ankle dorsiflexors. After foot-flat, but before contralateral toe-off, support was generated primarily by gluteus maximus, vasti, and posterior gluteus medius/minimus; these muscles were responsible for the first peak seen in the vertical ground-reaction force. The majority of support in midstance was provided by gluteus medius/minimus, with gravity assisting significantly as well. The ankle plantarflexors generated nearly all support in late stance and were responsible for the second peak in the vertical ground-reaction force.

However, Neptune et al. (2001) argued that solving the force-sharing problem alone (algorithm performed by Anderson & Pandy, 2001), like inverse dynamics-based analyses, does not provide insight into causal relationships between muscle activity and task performance. On the other hand, they suggested that acceleration and power analyses (Fregly & Zajac, 1996) of forward dynamics simulations of walking, that are driven by individual muscles, can be faithfully used to determine the functional role of the gastrocnemius and

soleus muscles in providing body support and propulsion<sup>14</sup>. In order to do so, they developed a musculoskeletal model and optimization framework to generate a forward dynamics simulation of normal walking at 1.5 m/s. At any instant in the gait cycle, the contribution of a muscle to support and forward progression was defined by its contribution to trunk vertical and horizontal acceleration, respectively, and its contribution to swing initiation by the mechanical energy it delivers to the leg in pre-swing (i.e., double-leg stance prior to toe-off). Their results show that the gastrocnemii and the soleus provide trunk support during single-leg stance and pre-swing. In early single-leg stance, undergoing eccentric and isometric activity, all three heads of the TS were shown to accelerate the trunk vertically but decelerate forward trunk progression. In mid stance, while isometric, the gastrocnemii would deliver energy to the leg while the soleus decelerates it, and the soleus would deliver energy to the trunk while the gastrocnemii decelerate it. In late single-leg stance through pre-swing, though the gastrocnemii and soleus both undergo concentric activity and accelerate the trunk forward while decelerating the downward motion of the trunk (i.e., providing forward progression and support), they execute different energetic functions. The energy produced by the soleus accelerates the trunk forward, whereas the gastrocnemii would deliver almost all its energy to accelerate the leg to initiate swing.

In summary, the results coming out of the forward dynamics model show that throughout single-leg stance both the soleus and the gastrocnemii provide vertical support. However in mid single-leg stance they have opposite energetic effects on the leg and trunk to ensure support and forward progression of both the leg and trunk, and in pre-swing only the gastrocnemius was found to contribute to swing initiation.

Liu et al. (2006) also studied the contributions of individual muscles to forward progression and vertical support during walking using a similar model. However, they systematically perturbed the forces in 54 muscles during a three-dimensional simulation of walking, and then computed the changes in fore-aft and vertical accelerations of the body mass centre due to the altered muscle forces during the stance phase. They found that the muscles that provided most of the vertical acceleration (i.e., support) also decreased the forward speed of the centre of mass during the first half of stance (vasti and gluteus maximus). Moreover, they also state that muscles that supported the body (soleus and gastrocnemius) also propelled it forward during the second half of stance.

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<sup>14</sup> The reader is referred to the article of Zajac *et al.* (2002) for better understanding of the forward dynamics technique and the interpretation and significance of results obtained by such a technique.

The musculoskeletal models just cited above, all try to solve complex yet indeterminate mathematical problems<sup>15</sup>. The indetermination is caused by the fact that many muscles cross the same joint and can perform agonist or antagonist work. Consequently, the number of equations is higher than the number of variables to solve. Thus, the models have to rely on minimization algorithms that try to best guess the force in each of the muscles required to perform a certain movement. Hence, these minimization algorithms remain very subjective. Furthermore, these algorithms fail to incorporate antagonist muscle contractions, since they do not seem logical from a minimal metabolic expenditure point of view. This led scientists to resort to more traditional experimental protocols in order to provide evidence that TS is responsible for push-off.

Stephens & Yang (1999) had the idea of comparing gait between unloaded and loaded subjects and monitor EMG activity. Subjects were loaded by adding a mass and unloaded by means of winch and cable system anchored over their heads. When loaded, an augmentation of level and duration of SOL activity was recorded, whereas unloading affected EMG duration only but not amplitude. Gottschall & Kram (2003) tried another setup. They used an aiding horizontal pulling apparatus connected at the waist of the subjects in order to decrease propulsion forces. When subjects were pulled by the cable, GM EMG activity decreased but SOL activity remained unchanged. They reasoned that GM is involved in the generation of the propulsion force while SOL only provides body support. McCowan et al. (2008) argued that the horizontal force exerted by the pull may have changed body posture inclination with respect to the feet and by this altered ankle torque. Therefore, McGowan et al. (2008) adopted a protocol similar to that of Stephen and Yang (1999), but allowed the vertical apparatus to slide over the subjects' head. They also tested the effect of body mass to increase inertia by adding a load and pulling vertically in order to unload the subjects. Only SOL activity was affected by the change in body inertia, while both GM and SOL activity increased when body weight increased. Strikingly, GM and SOL activity decreased when subjects were unloaded, in opposition to what Stephens and Yang found with almost the same protocol. However, Lewek (2011) used a similar apparatus for unloading subjects on a treadmill. In contradiction to McGowan et al. (2008) he stated that SOL, GM and GL activity was unaffected when subjects were unloaded. This unchanged activity of TS occurred for different walking velocities. What these experiments have in common is that all subjects were attached by a

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<sup>15</sup> An indeterminate problem refers to a mathematical problem that allows for an infinite number of solutions.



cable, which pulls on them. This unavoidably adds up another external force, which is the tension in the cable. Consequently, muscles are unloaded differently and all joint torques are modified slightly. Thus, these results cannot be extrapolated to normal gait where only two external forces are applied to the body: bodyweight and friction under the foot. Furthermore, the different experimental setups in these studies should explain the partly contradicting results that were reported by the authors.

## II.4 Working Hypotheses

The main debate about the role of TS during single stance is whether it functions solely as a body support or provides a forward thrust in single stance as well. In quiet standing, the body-support role of TS, notably of soleus, is well established and accepted (Schieppati et al., 1994; Morasso & Schieppati, 1999, Van Doornik et al., 2011). All the scientists that have been previously cited in the previous section unanimously agree that when TS is in eccentric contraction its sole role is to support the body during single stance. The debate is on its function during late stance. In single stance, CoM is falling and has thus a negative vertical velocity. However, Chong et al. (2009) and Chastan et al. (2010) showed that at around middle stance, the gradient of the vertical velocity is reversed. In mechanical terms, CoM is accelerating upwards. This can only mean that the fall of the CoM is being actively braked. We assumed that the increase in TS activity in middle to late stance is to produce that braking action.

### Hypothesis 1:

Triceps surae does not provide a propulsive push in late stance but brakes the fall of CoM.

We believed that the CoM momentum generated solely by the disequilibrium torque should be sufficient for propelling the body. We wanted to investigate how this torque is modified by TS activity. The torque can be calculated as the product of gap between the CoM and CoP and the vertical force. Due to the body geometry and segment kinematics during stepping, CoM position and step-length are highly linked together. Thus by varying step length the gap is modulated accordingly, hence torque and walking velocity. However, it is possible to vary walking speed without changing step length, i.e. by varying cadence. To solve this problem,

we thought about the CoM momentum entering the single stance phase. The CoM should have a lower momentum for slower cadences and higher momentum for fast cadences. A lower momentum should cause lesser displacement of CoM and thus a smaller CoM-CoP gap. Meanwhile a higher momentum should cause an ampler CoM-CoP gap.

Hypothesis 2 & 3:

Modulation of the duration of triceps surae activity should set kinematics of gait by determining step length and cadence and the kinetics of gait by tuning the CoM-CoP gap, i.e. the disequilibrium torque.

When walking faster, the amplitude of triceps surae rises in order to counter the increasing requirement of body support.

### III. Study One

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Honeine JL, Schieppati M, Gagey O, Do MC.

The functional role of the triceps surae muscle during human locomotion.

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### III.1 Abstract

**Aim** Despite numerous studies addressing the issue, it remains unclear whether the triceps surae muscle group generates forward propulsive force during gait, commonly identified as ‘push-off’. In order to challenge the push-off postulate, one must probe the effect of varying the propulsive force while annulling the effect of the progression velocity. This can be obtained by adding a load to the subject while maintaining the same progression velocity.

**Methods** Ten healthy subjects initiated gait in both unloaded and loaded conditions (about 30% of body weight attached at abdominal level), for two walking velocities, spontaneous and fast. Ground reaction force and EMG activity of soleus and gastrocnemius medialis and lateralis muscles of the stance leg were recorded. Centre of mass velocity and position, centre of pressure position, and disequilibrium torque were calculated.

**Results** At spontaneous velocity, adding the load increased disequilibrium torque and propulsive force. However, load had no effect on the vertical braking force or amplitude of triceps activity. At fast progression velocity, disequilibrium torque, vertical braking force and triceps EMG increased with respect to spontaneous velocity. Still, adding the load did not further increase braking force or EMG.

**Conclusions** Triceps surae is not responsible for the generation of propulsive force but is merely supporting the body during walking and restraining it from falling. By controlling the disequilibrium torque, however, triceps can affect the propulsive force through the exchange of potential into kinetic energy.

*Keywords:* body support, body propulsion, electromyography, disequilibrium torque, gait, triceps surae.

## III.2 Introduction

Putting in motion any material system requires the application of external forces. Bipedal locomotion is no exception. In human walking, forces are necessary for propelling the body forward. At the same time, due to the gravitational attraction exerted by the Earth, upward-directed forces are obligatory for keeping the body in equilibrium and preventing it from falling. Plantar flexor muscles are good candidates for generating both forces, since they exert their action at the interface between the human body and the ground. There is no general consensus as to whether the triceps surae muscle, the major plantar flexor group, contributes to the propulsive action (active thrust or ‘push-off’) or to the support action, or both.

Debate has been surrounding the functional role of ankle flexors for some time. Originally, based on the existing co-variation between triceps surae electromyographic (EMG) activity and velocity of progression, Winter (1983) suggested that ankle plantar flexors provide the active push-off during the late part of the single stance phase. In contrast, Perry (1974) had advised dropping the term push-off and postulated that the peak of the ground-reaction force in late stance phase is the result of the leverage put forth by the body alignment with respect to Earth-vertical axis rather than of an active downward thrust. An introduction to these issues can be found in an influential paper by Sutherland et al., 1980. At the other temporal edge, Bogey et al., (2010) have provided an extensive review of more recent reports, to which the reader is referred.

The push-off hypothesis has been endorsed by a series of articles modelling the net ankle moment (Kepple et al., 1997; Neptune et al., 2001; Sadeghi et al., 2001; Zajak et al., 2002; Liu et al., 2006). However, other experiments have provided results that undermine this hypothesis. Tibial nerve block (with paralysis of triceps surae muscle along with plantaris, tibialis posterior, flexor hallucis longus, and flexor digitorum longus) resulted in an *increase* in forward velocity of the centre of mass (CoM) during the late stance phase of gait, which was interpreted as defective control of CoM fall (Sutherland et al., 1980; Simon et al., 1978). Replacement of one lower limb by a prosthesis did not affect the speed of progression, regardless of whether the stance limb was prosthetic or not (Michel & Do, 2002). These findings support Perry’s (1974) statement and the conclusion of a seminal paper by Cavagna & Franzetti (1986), who posited that the fall of the CoM during the single stance phase of gait

is enough for transforming the potential energy into forward kinetic energy for progression during normal level walking. Therefore, triceps surae would not provide the propelling thrust by pushing off the ground. Hence, the contribution of the plantar flexors to whole-body forward displacement during normal walking would primarily consist in restraining forward tibial rotation, thereby stabilising the knee joint Sutherland et al., (1980), and in controlling the braking of the fall of the CoM during the single support phase of gait, as recently suggested by Chastan et al., (2010).

The role of the ankle flexors in body support during gait has been agreed upon in the literature. The postural role of soleus in quiet standing is amplified during locomotion (Schieppati et al., 1994; Morasso & Schieppati, 1999), which imposes more body support as ankle torque increases (Winter et al., 1998; Ivanenko et al., 2011). Anderson & Pandy (2001) studied the muscle contribution to body support using mathematical modelling based on a force-sharing problem algorithm. They found that the ankle flexors generated nearly all the body support in early and late stance, in addition of being responsible for the second peak typically observed in the vertical ground reaction force. Neptune et al., (2001) used a more elaborate model based on forward dynamics, and stated that the gastrocnemii and soleus provide trunk support during single stance and pre-swing. They stressed however the fact that in late stance phase the energy produced by the soleus accelerates the trunk forward, while the gastrocnemii would deliver most of their energy to accelerate leg into swing. On the other hand, Liu et al., (2008) used mathematical modelling to simulate locomotion at varying walking velocities, and concluded that an increase in gastrocnemii and soleus activity accompanies body support as walking speed increases.

Our aim was to unravel the functional role of ankle plantar flexors during human locomotion. We set out to see whether the triceps surae provides forward thrust by pushing off the ground or it controls body dynamic equilibrium, or does both at the same time. The problem here lies in the fact that the coordination and synergy produced by the walking body make that several gait parameters are highly correlated. Propulsive force, forward velocity and triceps surae EMG during the stance phase are one example (Winter, 1983; Pedotti et al., 1977). We posited that in order to properly assess the role of ankle plantar flexors in gait, one should *increase* propulsive forces for a *constant* forward velocity. This can be done by adding an extra load to a subject and instruct him/her to maintain constant walking velocity. Since adding a load requires greater external force to move the body, then an increase in plantar

flexor EMG activity for the greater antero-posterior force at the same walking velocity would verify the push-off hypothesis. Conversely, the absence of load-related increase in plantar flexor activity would discard the push-off hypothesis and support a functional role of these muscles in balance control.

The gait initiation paradigm was used (Carlsöö, 1966; Brenière et al., 1981; Crenna & Frigo, 1991). This choice was based on the fact that subjects initiating walking from a static upright position, when the initial forward-directed velocity is null, would actively produce the whole of the propulsive force according to the push-off concept. The push off might be less necessary if gait was already in the stationary state, when the body speed itself would produce part of the propulsive force. So, failure to support the push-off hypothesis during gait initiation would reinforce with stronger reason the alternative hypothesis. We concurrently analysed the contribution of gravity in creating propulsive force by measuring the displacement of the CoM away from the support axis, which generates the forward disequilibrium torque. Two walking velocities were tested, in order to generalize the main conclusion to different progression speeds, in which neural mechanisms such as altered fusimotor drive, reduced pre-synaptic inhibition and/or increased descending excitatory input may undergo subtle changes (Cronin et al., 2009).

### III.3 Materials and Methods

#### *Subjects*

Ten healthy volunteers (one female and nine males) took part in the experiment. Their mean age, body mass and height were 34 years (range 23-54), 72 kg (61-83) and 1.73 m (1.69-1.83), respectively. Written informed consent was obtained, as required by the Declaration of Helsinki and by the EA 4532 local Ethics Committee of University Paris-Sud, who specifically approved this study.

#### *Experimental set-up*

A large force platform (0.90 m x 1.80 m, AMTI, USA) was used to record ground reaction force and moments. The force platform was embedded in the ground. The walkway was long

enough (7 meters) to allow subjects to carry out at least 6 steps, hence avoiding the interference of gait termination with the gait initiation motor programme (Crenna et al., 2001).

The overall mass of the added load was 20 kg and consisted of 2 weight-lifting disks of equal mass and size (29 cm diameter and 3 cm thickness) put in two backpacks carried by the subject at the abdominal and lumbar back level, i.e. roughly around the centre of mass (CoM) position. The backpacks were firmly wrapped to the body to avoid unwanted displacement during stepping. For a subject weighting 83 kg, a total mass of 30 kg load was used instead. So the added load increased the body weight by a proportion ranging from 25% to 33%, depending on the subject.

Surface EMG activity was recorded using bipolar Ag-AgCl electrodes (8 mm diameter, 20 mm inter-electrode distance). Electrode sites were prepared by cleansing and shaving the skin for optimal myoelectric impedance. EMG activity was collected from right and left tibialis anterior (TA), soleus (SOL), gastrocnemius medialis (GM) and gastrocnemius lateralis (GL) muscles by preamplified wireless electrodes (Zero-wire, Aurion, Milan, Italy). GM and GL were recorded from only 7 of the 10 subjects. EMG signals were amplified ( $\times 1000$ ) and band-pass filtered (10-500 Hz). Electrodes were placed according to the SENIAM protocol (Merletti & Hermens, 2000). Force platform and EMG data were digitised at a sampling frequency of 1000 Hz on the same A/D converter card and saved on a PC for off-line analysis.

### *Experimental Protocol*

Before recording, we determined the preferential starting foot of the subjects. Subjects were asked to stand still eyes closed, and a small thrust was applied to their back forcing them to make a step forward. This was repeated 3 times. Then, subjects were instructed to initiate gait with the stepping leg that was used during this test. In order to obtain a good reproducibility of the progression velocity during the experiment, subjects executed several blank trials to determine the steps lengths corresponding to their spontaneous (S) and fast (F) speed walking conditions, and two landmarks representing the step length for the S and F condition were then drawn on the force platform for each subject. The average walking velocity proved to be about  $1.1 \text{ ms}^{-1}$  at spontaneous and  $1.5 \text{ ms}^{-1}$  at fast velocity (see Results).



Subjects stood still, barefoot on the force platform, looking straight ahead, and initiated gait following a verbal go-signal. They were instructed not to start walking in a reaction-time mode, but to start when they felt ready. This usually occurred within 2s from the go-signal. Both S and F conditions were repeated with and without the added load (L). The four experimental conditions are termed S, S+L, F and F+L in the text. All subjects started by performing the spontaneous unloaded series, following which the other conditions were performed in a pseudo-random order. Fifteen trials were acquired in each experimental condition.

### *Ground reaction force and disequilibrium torque*

While walking, the body exerts a force on the ground, which in return applies on the subject an opposite force, the ground reaction force (GRF) that is measured by the force platform. This force was divided into 3 components (antero-posterior, AP; medio-lateral, ML; vertical, Ver). In addition, the force platform gave two moments with respect to the AP and ML axis of the platform. The coordinates of centre of pressure (CoP) were obtained by dividing these moments by the vertical GRF. The CoP instantaneous position was used to establish the instant of foot off (FO) and foot contact (FC). FO was the point at which the ML CoP position shifts under the stance foot, and FC the point at which the AP CoP position suddenly increases as the swing foot touches the ground (see Figure III.1, 7<sup>th</sup> & 8<sup>th</sup> trace). CoM velocity was obtained by integration of the CoM acceleration. CoM forward acceleration was obtained as  $AP\ GRF/BM$ , and CoM vertical acceleration as  $(Ver\ GRF-BW)/BM$ , where BW and BM are body weight and body mass, respectively. The instantaneous position of the CoM was obtained by double integration of the CoM acceleration with respect to time (Bernière & Do, 1991; Chastan et al., 2010).

During gait, CoM oscillates vertically while rotating around the CoP in the sagittal plane, and the CoM fall is braked during the single stance phase of gait. The displacement of the CoM generates a disequilibrium torque, driven by gravity, as CoM moves beyond the CoP. The braking of the CoM fall was evaluated as the variation of the vertical GRF between its minimum and maximum value within the single stance phase (points 4 and 5 in Figure III.1). The disequilibrium torque was calculated as  $Ver\ GRF \cdot (CoM-CoP)$ . The difference (CoM-CoP) represents the instantaneous distance (hereafter called gap) between the AP position of the CoP and the corresponding position of the ground projection of the CoM.

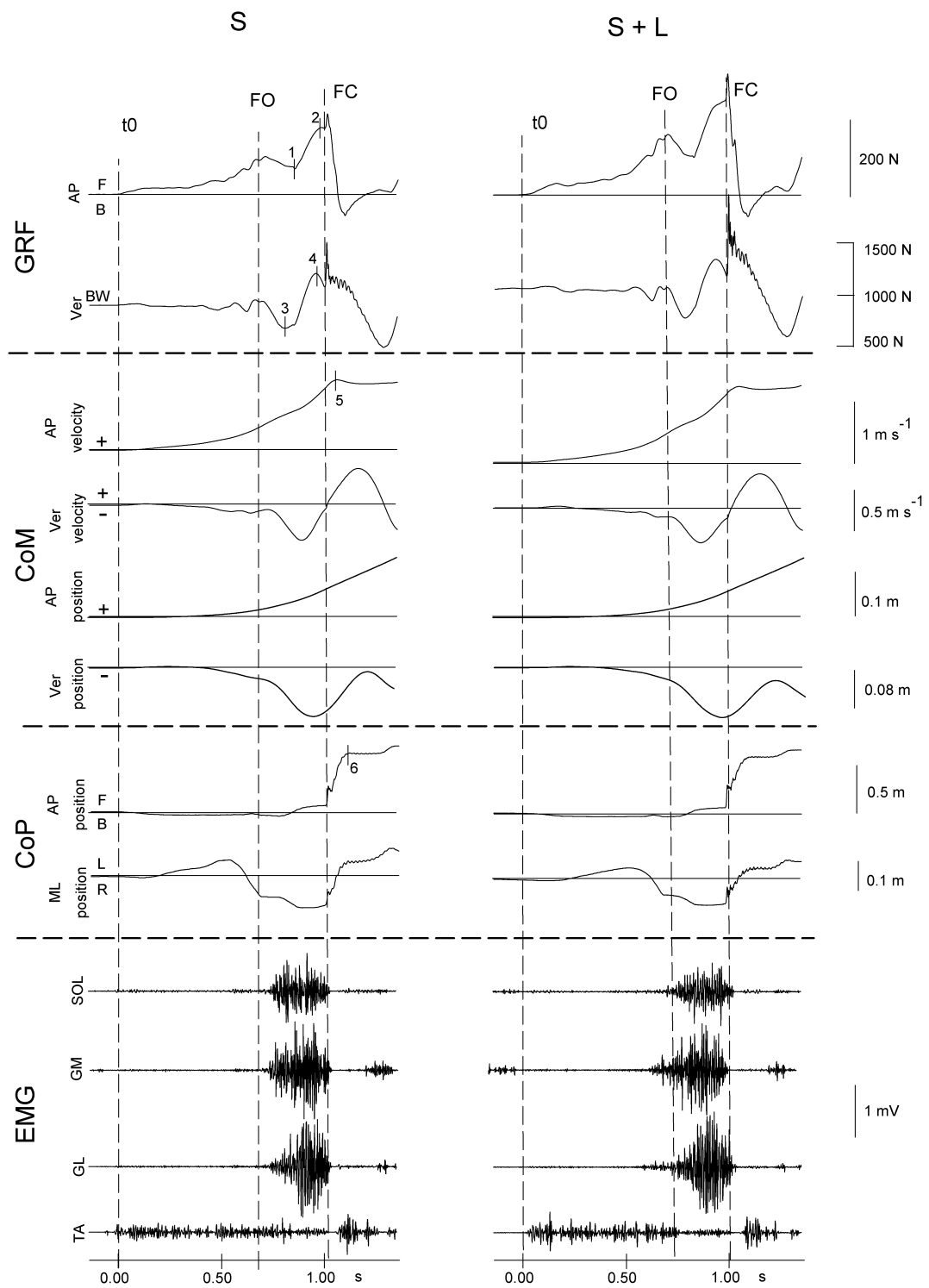
### *EMG Analysis*

The EMG activity of SOL, GM and GL was rectified after removing the baseline offset and was time-integrated. EMG activity of each muscle was calculated for three partly overlapping time windows:  $W_{tot}$  corresponds to the total duration of the burst, between the onset and termination of the EMG activity;  $W_b$  is the braking action phase and  $W_p$  is the propulsive phase. The braking action goes from the instant when  $Ver$  GRF reverts and goes upwards until the time of foot contact (FC), and the propulsive phase corresponds to the time interval initiating at the instant when AP GRF increases steeply and terminating just prior to FC (points 1 and 2 in Figure III.1). The integrated EMG activity of each trial was then divided by the time duration of each window in order to get the mean activity level. For graphical representation (Figure III.3), EMG activity was expressed as a percentage relative to the mean value of the unloaded spontaneous velocity condition for each muscle and for each of the three time-windows.

### *Statistical Analysis*

The focus of the present study was the effect of load on muscle activity for a same walking velocity as opposed to the effect of velocity itself. However, velocity was selected as independent variable in order to check the statistical difference between the velocities. Two-way ANOVA was used to compare each of the measured or calculated biomechanical variables. Categorical factors were velocity and load. The mechanics-related variables were: forward velocity, step length, instants of occurrence of FO (foot-off of the swinging leg) and FC (foot contact of the same leg), amplitude of the sudden increase of the propulsive force (the 'push-off') and amplitude of vertical braking action. The EMG-related variables were the instants of onset and offset of the activity of each of the muscles with respect to time of gait initiation, and the mean level of the EMG surface of each muscle for each of the time windows ( $W_{tot}$ ,  $W_p$  and  $W_b$ ). Paired t-test was used to test the effect of load on the delay between the onset of SOL and the onset of the braking action. The level of significance was set at  $p < 0.05$  for all tests.

### III.4 Results



**Figure III.1. Time-course of gait initiation variables in a representative subject.**

All mechanical and EMG traces refer to walking at spontaneous (S) velocity. The left panel shows the control condition (no added load), the right panel shows the loaded (L) condition. The traces are assembled in four panels according to the type of recording. From top to bottom: Ground Reaction Forces (antero-posterior and vertical GRF), Centre of Mass (antero-posterior and vertical velocity and position of CoM), Centre of Foot Pressure (antero-posterior and medio-lateral CoP), EMG activity of the triceps surae muscles of the stance leg (soleus, SOL; gastrocnemius medialis, GM; gastrocnemius lateralis, GL; tibialis anterior, TA). The direction of the changes in the antero-posterior (AP) and medio-lateral (ML) position of GRF and CoP is indicated (forwards, F; backwards, B; left, L; right, R). The signs + and – in the CoM traces refer to positive and negative values of CoM velocity and position in the AP and vertical (Ver) direction. All traces start at time 0, corresponding to the onset of the anticipatory adjustment preceding the production of the first step, based on the (magnified) trace of the ML CoP position. The vertical dotted lines are set at the instant of foot off (FO) and foot contact (FC) of the swinging leg. The period between FO and FC is the single stance phase of gait. The triceps muscles are active during this phase, starting shortly after FO and terminating around FC. The numbers and ticks on selected traces of the left panel indicate critical points for the analysis: 1-2, onset and offset of the propulsive force increase; 3-4, onset and offset of the braking action; 5, peak of CoM AP velocity; 6, AP CoP position used to determine step length. Adding the load (right panel) increases Ver GRF, the value of which corresponds to the body weight (BW) in the period from  $t_0$  to FO. The load also increases the difference (2 – 1) of the AP component of the GRF, but has negligible effect on the other variables. Notably, adding the load has no effect on the ‘braking action’, or the difference (4 – 3) in the Ver component of the GRF, and in the pattern of triceps activity.

In Figure III.1, mechanical and EMG traces for the S condition (spontaneous velocity, unloaded condition) are compared with the S+L (spontaneous velocity, loaded condition). The overall kinematics and lower limb muscle activity of the gait initiation process have been described in detail elsewhere (Carlsöö 1966, Crenna & Frigo 1991, Brenière & Do, 1991). Briefly, the gait initiation process includes two phases. The former is an anticipatory postural adjustment (APA), which starts at the onset of the ground reaction force (GRF) variation ( $t_0$  in Figure III.1) and lasts until the first foot-off (FO). The APA prepares the step execution by means of a motor programme involving a deactivation of SOL background EMG activity, followed by a bilateral TA activation. Both events produce the backward displacement of the CoP. This produces a gap between the CoP position and the vertical projection of the CoM, causing a forward disequilibrium torque (Brenière & Do, 1991; Lepers & Brenière, 1995). The latter phase is the step execution that follows the APA. It goes from the time of FO of the swinging leg until the foot-contact (FC) of the same leg.

**Table III.1 Kinematic variables:** Grand Mean and standard deviation of the instant of foot off (FO) and foot contact (FC) of the swing leg with respect to the onset of movement t0, the peak antero-posterior CoM velocity (AP Vel) reached shortly after FC, and the step length calculated as the difference of the AP position of CoP between the end and the beginning of the stance phase. F and p values are reported to show that load had no significant effect on any of these kinematic variables for both velocity conditions.

		FO (s)	FC (s)	AP Vel (m·s <sup>-1</sup> )	Step length (m)
S	mean ± SD	0.571 ± 0.05	0.955 ± 0.06	1.09 ± 0.07	0.59 ± 0.03
S+L	mean ± SD	0.605 ± 0.05	0.957 ± 0.06	1.09 ± 0.07	0.61 ± 0.03
	F (1,9); P	2.7; 0.13	0.003; 0.95	0.007; 0.93	3.3; 0.10
F	mean ± SD	0.597 ± 0.06	0.968 ± 0.05	1.56 ± 0.07	0.84 ± 0.03
F+L	mean ± SD	0.609 ± 0.07	0.988 ± 0.06	1.517 ± 0.08	0.85 ± 0.03
	F (1,9); P	0.2; 0.63	0.6; 0.44	1.1; 0.31	0.2; 0.69

Kinematics results (Table III.1) show that all subjects faithfully executed the experimental instruction, and thus maintained their walking velocity and step length regardless of the added load, thereby producing the same velocity condition. For the progression velocity, the grand mean value was almost identical without and with load for the spontaneous velocity. The load had no effect on the progression velocity for the fast velocity, either. Adding the load had no effect on step length, for either the spontaneous or fast velocity conditions. The instants of FO and FC with respect to t0 did not change with added load, either, and this was true within the spontaneous and the fast velocity conditions (Table III.1).

#### *Spontaneous velocity, unloaded and loaded condition*

During single stance (FO to FC in Figure III.1), the AP GRF (top trace) had a two-phase time-course, describing an early small variation followed by a steep increase (the propulsive phase). The onset of this increase (point 1) was set at the minimal value of AP GRF during the single stance phase that occurred usually around mid-stance. The magnitude of the increase is the difference in AP GRF between points 1 and 2.

Ver GRF trace was valley-shaped. Shortly after FO, Ver GRF started decreasing below subject's body weight, reflecting CoM downward acceleration. After attaining a minimum value (point 3), it increased again and reached a value well beyond body weight (point 4). The variation of Ver GRF between points 3 and 4 is the braking action, or the vertical force opposing the CoM fall seen in the vertical CoM velocity trace.

Visual inspection of the two columns of Figure III.1 (left, S; right, S+L) shows that the difference in the amplitude of AP GRF between point 1 and 2 increased with the added load. Conversely, adding the load did not obviously change the braking action amplitude.

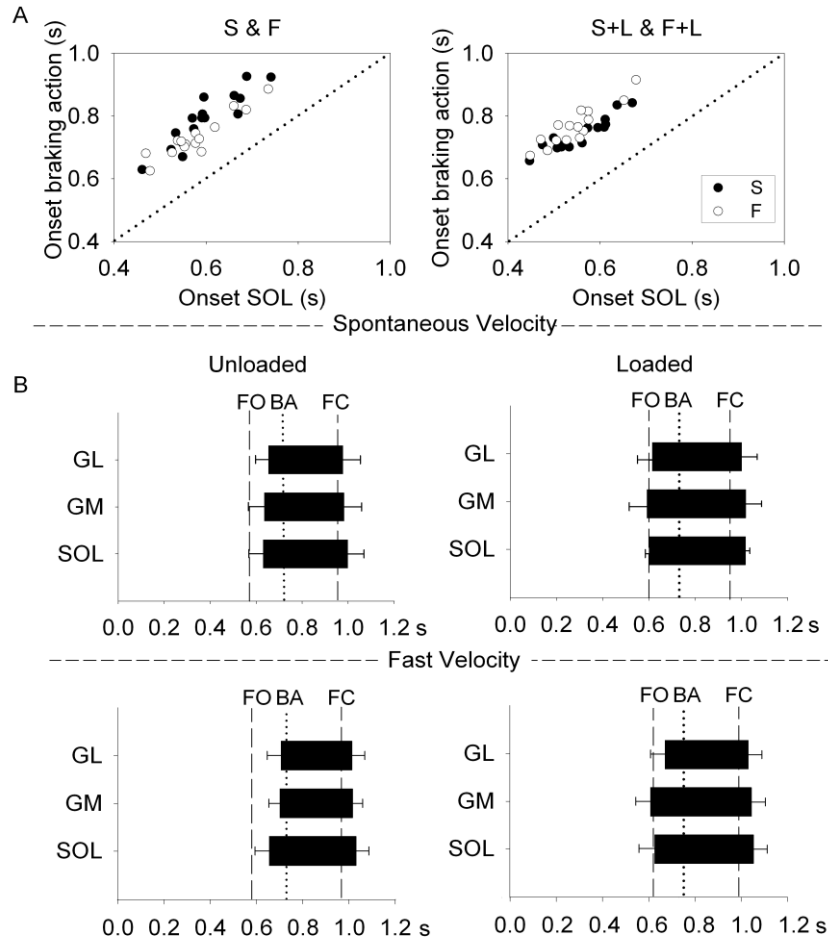
The onset of the increase in Ver GRF always preceded the onset of the steep increase in AP GRF, while the duration of the braking action and that of the propulsive phase overlapped for a while during the single stance period (from FO to FC). More precisely, the time window of the braking action phase accounted for  $66\% \pm 10$ ,  $64\% \pm 4$ ,  $66\% \pm 5$  and  $63\% \pm 6$  of the entire single-stance duration made equal to 100% for S, S+L, F and F+L conditions, respectively. The time window of the propulsive phase accounted for  $41\% \pm 14$ ,  $43\% \pm 8$ ,  $49\% \pm 7$  and  $55\% \pm 8$  of the single stance duration for S, S+L, F and F+L conditions, respectively.

Velocity and position of the CoM are shown in Figure III.1, below the GRF traces. The peak of CoM AP velocity was reached after FC (3<sup>rd</sup> trace from top, point 5). The negative value of the vertical CoM velocity until FC indicates that the CoM was always falling during the whole single support period, but with two phases, the first accelerating downward, the second decelerating, when the fall of CoM was being restrained. The Ver CoM instantaneous position (6<sup>th</sup> trace from top) fell below its initial value during the single stance, stabilised slightly around foot contact and moved up again during the double support phase while the triceps surae muscles were being deactivated. The CoM curve matches well in profile and maximum value the curve calculated with an independent method (a motion capture system) by Jian et al., (1995). Multiplying the gap between the CoM and CoP by the vertical force acting on the CoM (Ver GRF) gave the disequilibrium torque acting around the CoP (reported in Figure III.4, 4<sup>th</sup> trace). The third panel from top shows the CoP traces. The CoP underwent a displacement from the rear to the fore foot, where its forward movement was obviously halted due to foot length limitation, while the CoM continued to advance on the sagittal plane in a parabolic manner (5<sup>th</sup> trace from top in Figure III.1).

Figure III.1 (bottom traces) also illustrates the SOL, GM, GL and TA EMG activity of the stance leg. Triceps surae muscles were silent during the postural adjustment phase, while their synchronous bursts preceded shortly the vertical braking action and ended at around FC. This was true under both unloaded and loaded conditions. The onset of the burst occurred in the interval between FO and the onset of the braking action and varied slightly between subjects

and repeated measures. One source of variability between trials likely depended on the force platform registering the global resultant forces and not the local forces produced by the individual muscles. This variation also affected the onset and termination of the muscle bursts. Another source of variability depended on the way the onset of the braking action was identified, since this was conservatively set at the lowermost point of the Ver GRF trace, in a region where the profile is not particularly sharp.

In spite of these sources of variations, consistent findings were observed in the EMG pattern. On the average, all muscles initiated their activity around FO, and ahead of point 4 (the onset of the braking action), and terminated at or shortly after FC. Figure III.2A shows the onset of the braking action plotted versus the onset of the SOL activity, both measured with respect to FO, under both unloaded and loaded conditions for one subject. In this example, but also for all muscles and subjects, the braking action never anticipated the EMG onset. The onset of SOL activity with respect to FO was significantly anticipated in the loaded trials ( $F(1,9)=5.183$ ,  $p<0.05$ ); however velocity *per se* had no significant effect ( $F(1,9)=0.01$ ,  $p=0.92$ ). The grand mean and standard deviation of the time-interval between onset of SOL and onset of braking action were  $0.124 \pm 0.06$  s for the unloaded trials and  $0.199 \pm 0.05$  s for the loaded trials. A paired t-test showed that load significantly increased the delay between the onset of SOL and that of braking action ( $p<0.001$ ).



**Figure III.2. Temporal pattern of activity of the triceps surae muscles (SOL, GL, GM) during the stance phase of gait initiation.**

(a). the onset of the braking action has been plotted as a function of the onset of the SOL EMG activity for the unloaded (left) and loaded condition. The individual data points corresponding to spontaneous and fast velocities are superimposed in each plot. The braking action regularly lags the onset of SOL activity, so that the data points identify a line parallel to the identity line (dotted diagonal). In this subject, the points for spontaneous and fast velocity are almost confounded, and the points for the loaded condition indicate a small delay of the onset of the braking action with respect to the onset of the muscle activity. Such behaviour is only in part reflected in the other subjects, so that the mean intercept of the best fit lines are not significantly different between unloaded and loaded conditions. (b). The grand mean values ( $\pm$  SD) of onset and termination of the bursts of activity are reported for the three triceps muscles, referred to time 0 for all subjects and conditions of load and velocity. On the same time scale, the mean instants of foot-off (FO) and foot contact (FC) of the swing leg are indicated by vertical dashed lines. The vertical dotted lines refer to the mean onset of the braking action. The two top graphs refer to spontaneous (S) walking velocity, unloaded (left) and loaded (right). The data of the fast (F) velocity conditions are reported in the bottom graphs. There is no clear-cut difference neither in the overall time pattern of the activity across muscles, nor between S and F or between S+L and F+L. However, for both velocities, load increased the duration of the bursts, chiefly by anticipating the onset of their activity with respect to FO.



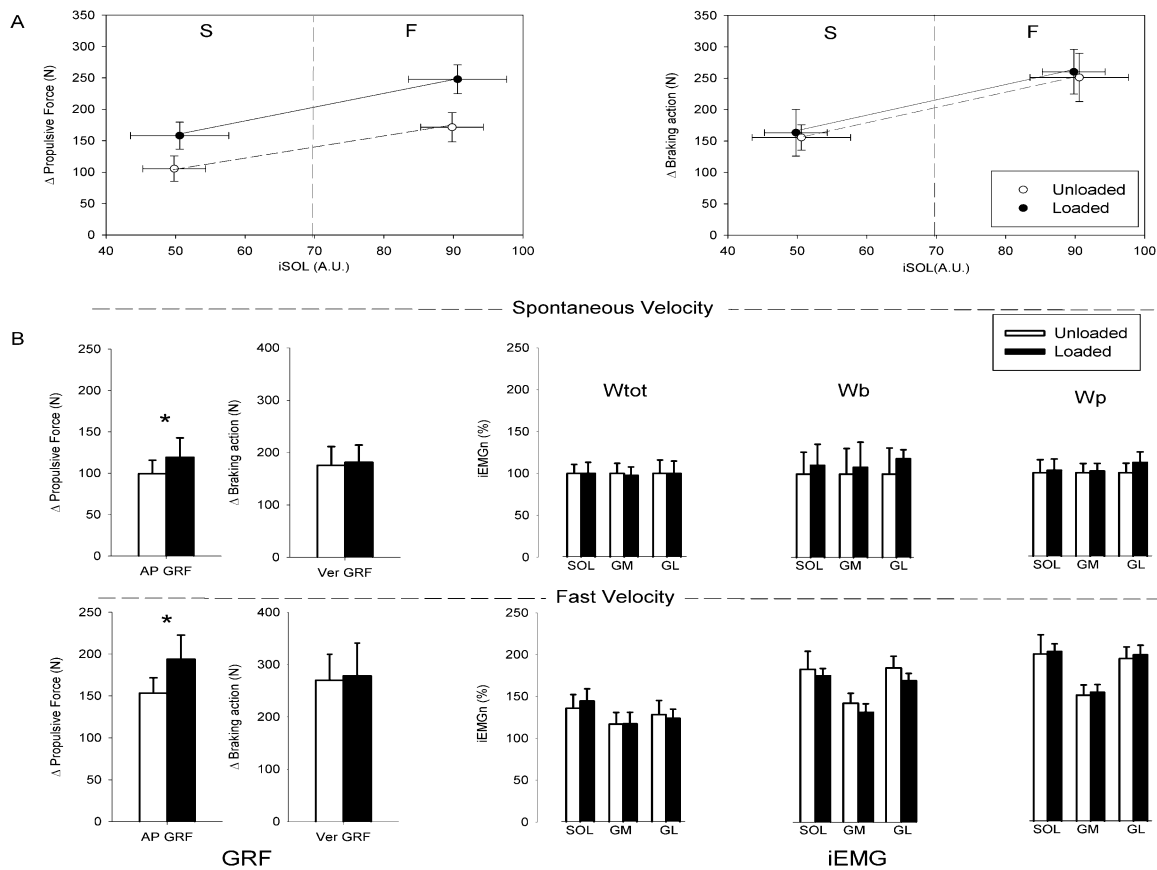
The histogram bars of Figure III.2B show the time-course of the EMG activity of the soleus and gastrocnemii muscles of the stance leg (with reference to FO and FC, respectively), for spontaneous and fast velocity, and for unloaded and loaded condition. The time-intervals between onset of EMG and onset of braking action (BA, dotted line) are broadly superimposed for the different muscles, with some anticipation for the SOL, inconsistent across trials and subjects. To note is an advancement of the onset of the EMG bursts with load for both velocities, in the face of a substantial similarity in the time distribution of the EMGs between spontaneous and fast velocity. ANOVA showed no significant difference in the time of onset across the three muscle bursts (SOL, GM and GL) ( $F(2,6)=0.96$ ,  $p=0.40$ ), indicating a concurrent recruitment and a common action of the triceps surae group aimed to brake the fall of the body during the single stance phase.

Figure III.3A shows the increase of the propulsive force (left panel) and of the braking action measured from the Ver GRF trace (right panel) plotted versus the mean SOL EMG level for one subject, for spontaneous and fast velocity. The values pertain to the time-window  $W_{tot}$ , from the onset to the termination of the EMG. The left panel indicates that the subject generated two different propulsive forces during the unloaded and loaded trials, while the corresponding mean EMG values were essentially the same for the same walking velocity. The right panel shows that neither the amplitude of the braking action nor the EMG activity were affected by load within each velocity. Hence, the extra load enhanced the AP GRF selectively. This is summarized in Figure III.3B (left panel). The propulsive force increased on average by about 36% for the spontaneous velocity ( $F(1,9)=101.1$ ,  $p<0.001$ ), and by about 29% for the fast velocity ( $F(1,9)=8.1$ ,  $p<0.05$ ), showing that a significant increase in AP propulsive thrust was generated during the late stance by the added load. In contrast, the Ver GRF remained almost unchanged with the added load ( $F(1,9)=0.11$ ,  $p=0.92$ ) but increased significantly with velocity ( $F(1,9)=5.7$ ,  $p<0.05$ ).

The right panel of Figure III.3B shows the mean amplitude of the EMG activity of SOL, GM and GL recorded during the three time-windows ( $W_{tot}$ : entire burst;  $W_b$ : from onset of braking action until point 4 (as in Figure III.1);  $W_p$ : from onset of AP GRF increase until point 2). The graph has been built on the basis of the EMG data from the seven subjects in which we recorded SOL, GM and GL. Notably, also in the three subjects in which Sol was the sole muscle recorded from, SOL EMG behaved very much as depicted in this Figure. For the fast velocity condition, the amplitude of AP GRF, of the braking action and of the EMG

increased compared to spontaneous velocity. However, within the same velocity, even when the amplitude of the propulsive force increased significantly as an effect of load, the grand mean of the EMG activity remained unchanged for SOL, GM and GL, regardless of the three time-windows used. Worth noting is that, when the subjects performed the fast walking trials, EMG activity increased concurrently with the increase in the braking action at fast velocity, but again there was no increase in EMG activity when the load was added. Remarkably, there were no changes with load even in the Wp interval corresponding to the propulsive phase of the stance period, in any muscle and for either velocity, i.e. when the active ‘push-off’ would be expected.

Therefore, it is fit to conclude that active recruitment of SOL, GM and GL muscle activity was not responsible for the increase in propulsive force required by the added load, but only for the increase in the braking action occurring with the increase in step length.



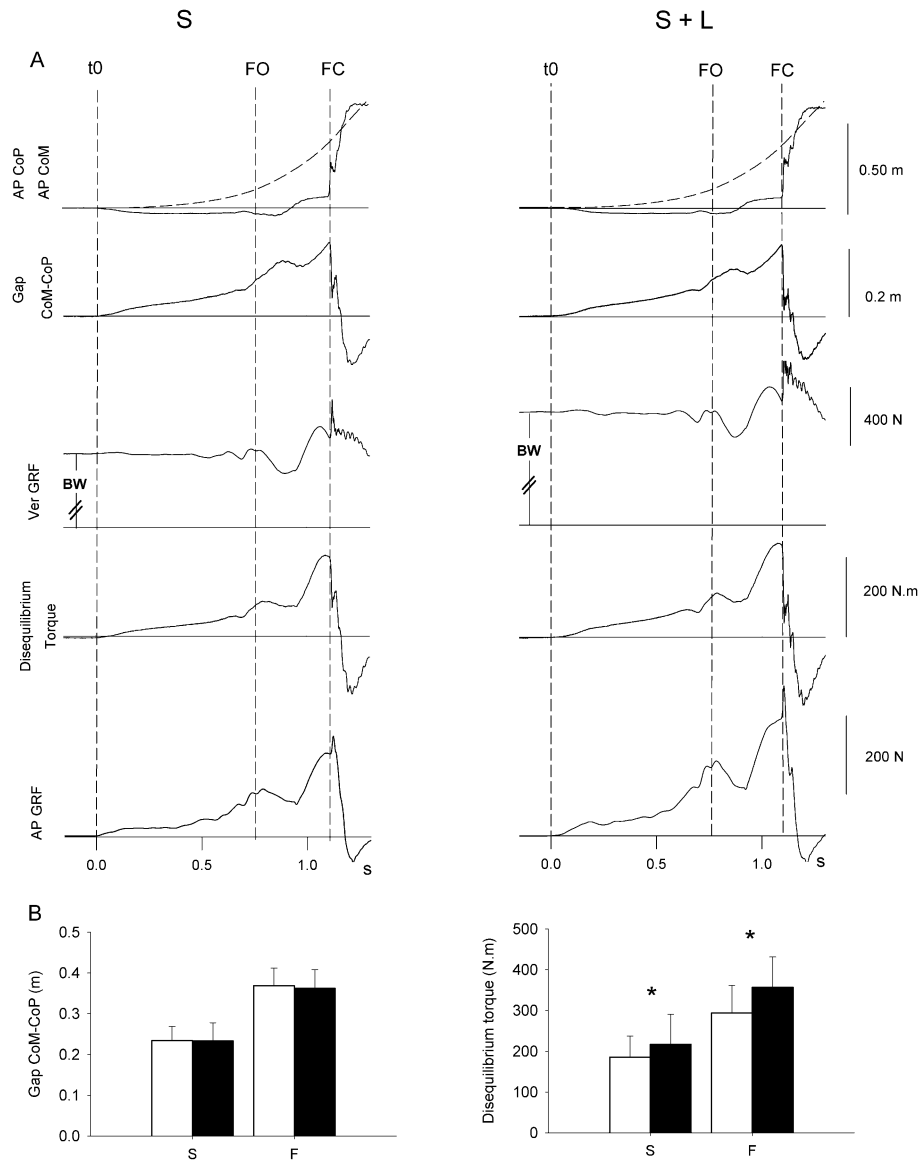
**Figure III.3 Divergent effects of load and walking velocity on propulsive force, braking action and triceps activity.**

The upper part of the Figure (a) contains two graphs, reporting the mean data from all trials of a representative subject. The left part of the left graph (spontaneous velocity, S) shows that AP GRF (the propulsive force) is larger when load is added (filled circle) compared to no-load (open circle). Notably, this increase occurs without changes in the SOL activity (measured in Wtot). A similar pattern is shown in the right part of the same graph (fast velocity, F). Note that F velocity is associated with an increase in SOL activity with respect to S velocity (abscissa): adding the load increases the propulsive force but does not further increase the amplitude of the burst. The lower part (b) contains two composite panels that summarize the results from all subjects. The left panel shows the mean and standard deviation of AP and Ver GRF, for the spontaneous (top) and fast velocity (bottom). Open bars refer to no-load, filled bars to added-load condition. The right panel shows the muscle activities for spontaneous (top) and fast velocity (bottom) conditions, unload and loaded, calculated within each time window (Wtot: entire burst, Wb: braking action, Wp: propulsive force). Asterisks indicate  $p < 0.05$ . There is an effect of velocity on braking action, propulsive force and muscle activity (all bars are higher in the bottom graphs), but no effect of load on any variable, except propulsive force (at both velocities).

### *Disequilibrium torque*

Figure III.4A compares, in a representative subject, the time-course of the instantaneous antero-posterior positions of CoP (continuous trace) and of CoM (dashed trace), the CoM-CoP gap, the vertical GRF, the disequilibrium torque (the product of the gap by the vertical GRF) and the AP GRF, for the unloaded and loaded condition (left and right panels), at spontaneous velocity. Worth noting is the striking similarity of the traces of the disequilibrium torque and AP GRF between unloaded and loaded conditions. This similarity verifies the fact that the energy generated by CoM rotation around CoP is transformed into forward propulsive force. Visual inspection of the individual traces in Figure III.4 shows that load did not affect the CoM-CoP gap. However, both disequilibrium torque and AP GRF increased concurrently with load. To better understand this, the CoM-CoP gap and the disequilibrium torque at the instant of foot contact were calculated. The grand mean values and standard deviation of the gap and disequilibrium torque are reported in Figure III.4B, in which gaps and torques are compared at spontaneous and fast velocities, unloaded and loaded conditions. The grand mean value of the gap measured at FC was the same between no-load and load conditions, at both spontaneous ( $F(1,9)=0.0002$ ,  $p=0.98$ ) and fast velocity ( $F(1,9)=1.74$ ,  $p=0.22$ ). At foot contact, gap was found to be around  $49\% \pm 4$  of the step length and  $48\% \pm 4$  for the S and S+L conditions, and  $52\% \pm 4$  and  $51\% \pm 5$  for the F and F+L conditions, respectively. The grand mean value of the torque was significantly larger under the loaded condition, both for the spontaneous ( $F(1,9)=9.92$ ,  $p<0.05$ ) and for the fast velocity ( $F(1,9)=163.98$ ,  $p<0.001$ ).

The disequilibrium torque depends on both CoM-CoP gap and Ver GRF. Since the CoM-CoP gap remained unchanged, the increase in torque solely depended on the changes in Ver GRF. In turn, the Ver GRF is composed of body weight and variations with respect to it due to the vertical acceleration of CoM. Since these variations (CoM fall and braking action) remained constant, then the increase in the disequilibrium torque was due to the absolute increase in body weight (4<sup>th</sup> trace in Figure III.4A, left & right) when subjects were loaded.



**Figure III.4** Disequilibrium torque calculation and its dependence on AP GRF.

The upper traces in (a) show, from top to bottom, the time-course of the instantaneous AP position of CoP (the position of CoM is superimposed, dashed line), the CoM-CoP gap, the vertical GRF, the disequilibrium torque (calculated as the product of the gap by the vertical GRF), and the AP GRF. The traces are from one representative subject, during unloaded (left) and loaded condition (right) at spontaneous walking velocity. The bottom histograms (b) show the grand mean values ( $\pm$  SD) of gap (left graph) and torque (right graph), computed at FC for spontaneous (S) and fast velocity (F), unloaded (open bars) and loaded (filled bars) conditions. The gap increases with velocity, but does not change with load. Conversely, the torque increases with both velocity and load. Asterisks indicate  $p < 0.05$ .

### III.5 Discussion

Our findings are not in keeping with the notion that the triceps surae is responsible for the generation of propulsive forces for walking. They rather show that the triceps supports the body while it translates over the ankle joint, restraining it from falling. Indirectly, though, triceps surae activity controls step length and walking speed.

#### *Rationale of the investigation*

To challenge the push-off hypothesis we have used the gait initiation paradigm and compared the condition, in which the subject was loaded, to that without load while maintaining the same progression velocity between experimental conditions. Since it has been repeatedly postulated that the triceps surae muscle is responsible for the ‘push-off’ action in the second part of the single stance phase of waking, we measured both biomechanical variables and the activity of soleus (SOL), gastrocnemius medialis (GM) and lateralis (GL) during the whole single stance period, to check whether any of these muscles, or all together, are accountable for the push-off.

The gait initiation paradigm should militate against our initial hypothesis (no active ‘push-off’) because the subject is initially motionless. A thrust to the ground support would seem necessary to generate a forward-propulsive force to propel the body forward, and even more so with the added load. This push-off would necessarily come from ankle plantar-flexor force, because plantar flexors are suitably arranged and are active during single stance (Crenna & Frigo, 1991; Anderson & Pandy, 2001; Franz & Kram, 2012). Since laws of motion dictate that more force is required to propel a heavier object, we added a weight to the subject to induce an increase in the propulsive force. The significant increase of antero-posterior ground reaction force (AP GRF) that we observed during the loaded trials was indeed in accordance with the predicted effect of load.

Because of the linear relationships between progression velocity and triceps surae EMG activity, and between progression velocity and propulsive force (Saunders, 1953; Winter, 1983), the effect of velocity had to be isolated in order to unambiguously assert that triceps activity is or is not responsible of AP propulsive force generation. Thus, recording the triceps

activity while imposing the same progression velocity between unloaded and loaded conditions permitted to extricate the postural from the propulsive role of the triceps.

Further, the effect of load was tested at two velocities. Replicating the effect of load at different velocity conditions corroborated the strength of the results. Our findings were consistent within the two velocity conditions, where GRF, kinematics and EMG are modified by the effect of speed. Therefore, we feel confident that our conclusions can be generalized to a range of walking velocities.

*The role of the triceps surae in push-off can be rejected.*

GRF data alone undermine the push-off hypothesis. If the subjects were to increase their push off the ground to propel themselves when a load was added, then basically both the AP and Ver components of the force vector measured by the force platform should increase concurrently. However, only the amplitude of the rise in AP GRF, but not the amplitude of the braking action, increased when load was added.

Adding the load increased significantly the AP propulsive force throughout the entire gait initiation process including the preparatory and single-support stance phases, in the same proportion as the added load with respect to the body weight. In contrast, EMG activity of the triceps of the stance leg during the whole single-support phase, as well as within the time-interval corresponding to the vertical braking phase, did not change when the load was added. Therefore, the increase in the propulsive force, when a load was added to the walking subject, was not accompanied by any increase in triceps activity. In other words, the triceps does not participate in the augmentation of the AP component of the GRF (the propulsive force) connected with the increased body weight. Thus, our subjects need not push-off the ground to propel themselves forward.

The onset of the braking action was the first measurable mechanical event picked up by the force platform shortly after the onset of the triceps surae EMG activity. The time-interval between onset of EMG and onset of braking action was independent of the exact time of the braking action onset with respect to FO. This suggests a strong relationship between triceps surae contraction and braking action. AP GRF variations is instead linked to the CoM-CoP gap that builds up during the single-stance phase. Notably, the EMG did not even change

within the time-window corresponding to the propulsive phase (Wp). These data therefore show that the triceps surae is responsible for the braking action seen in Ver GRF and that propulsive force is produced by the disequilibrium torque that is generated as CoM-CoP gap becomes more prominent during stance phase. This interpretation is in line with Simon et al., 1978 and Sutherland et al., 1980, who found that speed at late stance increased when ankle plantar-flexors were paralysed. Moreover, this behaviour is common to each of the three head of the triceps muscle. Neither SOL nor GM nor GL activity augmented their pattern of activity, commonly or independently, on increasing body weight. Therefore, in spite of their potentially independent activation during postural tasks (Nardone et al., 1990), we consider safe to deduce that these three muscles are driven by a single motor program during gait, and all of them are devoted to body support.

In contrast, the triceps can increase its activity, and it did so at higher walking speed, in keeping with the data in the literature showing a relationship between velocity and triceps EMG (Winter, 1983). When our subjects passed from the spontaneous to the fast speed, the GRF along both AP and Ver axes was enhanced along with the triceps surae EMG activity, but again EMG activity did not further increase on adding the load, in any examined time-window. On increasing velocity, EMG activity was slightly higher for SOL and GL than for GM. This simple observation lessens the risk that cross-talk between muscles can have blurred differences in their activation pattern; however it raises the question whether GM has an activation ceiling during locomotion.

Others have conducted experiments on a treadmill where an external force was applied to the subjects in order to manipulate the propulsive force. Stephens & Yang (1999) loaded the subjects by adding a mass and unloaded them by means of winch and cable system anchored over their heads. They reported an augmentation of level and duration of SOL activity in the loaded condition, whereas unloading affected EMG duration only but not amplitude. Gottschall & Kram (2003) used an aiding horizontal pulling apparatus connected at the waist of the subjects in order to decrease propulsion forces. They reported a decrease in GM but not in SOL activity and claimed that GM is involved in the generation of the propulsion force while SOL only provides body support. On the other hand, the horizontal force exerted by the pull may have changed body posture inclination with respect to the feet, as may happen with walking up a slope. This probably alters triceps surae function more into propulsion. Furthermore, McGowan et al. (2008) argued that a horizontal pulling apparatus would disturb



the ankle torque and adopted a protocol similar to that of Stephen & Yang (1999), but allowed the vertical apparatus to slide over the subjects' head. They also tested the effect of body mass to increase inertia by adding a load and pulling vertically in order to unload the subjects. Only SOL activity was affected by the change in body inertia, while both GM and SOL activity increased when body weight increased. Strikingly, GM and SOL activity decreased when subjects were unloaded, in opposition to what Stephens & Yang (1999) found with almost the same protocol. Lewek (2011) unloaded subjects as well at different speeds and found that SOL, GM and GL activity was affected by walking velocity but not by the unloading factor. The reasons behind the discrepancy between our experiment and those mentioned previously could be the non-negligible differences in kinematic and kinetic variables between over ground gait and treadmill walking (Zanetti & Schieppati, 2007; Lee & Hidler, 2008; Decker et al., 2012). This issue requires further investigation. Furthermore, the friction force (even qualified as low friction) and/or the inertia of the pulling apparatus used during those experiments could have altered the sensori-motor organization, in turn modifying EMG activity (Huang et al., 2009; Bastianne et al., 2000). Also, adding a load can alter the body CoM position depending on the placement of the added mass, and therefore the pattern of muscle activity. Since in our case the added load was positioned both anterior and posterior to the CoM position, the changes in body CoM position should be negligible. McGowan et al. (2008) pointed out that a certain external force would alter joint moments when a lever arm is created between the force and the joint at hand. Therefore, adding an external force, in the experiments where a pulling apparatus was used, could have altered the behaviour of the triceps surae working across the ankle. More importantly, it is hard to keep the vertical pulling apparatus perfectly vertical at all times: the varying tension in the cable could affect Ver GRF, which we believe is responsible for generating disequilibrium torque, and slightly alter ankle torque and consequently normal triceps surae activity. On the contrary, Lewek (2011) found with a similar protocol (treadmill walking and pulling apparatus) that SOL activity did not change when reducing ankle torque by applying an upward vertical force to the subject. This is contradictory to the results obtained by McGowan et al. (2008) on the basis of which SOL activity was expected to decrease. Lewek's (2011) findings are instead clearly complementary to ours, in that the change in ankle torque was only affected by gravity both when subjects were unloaded and when they were loaded, while in both cases the EMG activity remained constant.

### *Source of AP propulsive force*

Two postulates have been used for explaining the process of generation of the AP propulsive forces during free walking: i) the triceps surae ‘pushing off’ the ground or ii) the transformation of the potential energy into the forward kinetic energy (Cavagna & Franzetti, 1986, Cavagna et al., 2000). The first postulate is discarded by our data. Our results are instead in line with Cavagna & Franzetti (1986) and Cavagna et al., (2002), who showed that the AP propulsive force comes from the transformation of the kinetic energy of the fall of the CoM during the single support phase into propulsive energy, and give further insight into the nature of the parameter that is controlled in order to produce propulsive force according to the demand.

The body is in equilibrium when the vertical ground projection of the CoM and the CoP are confounded (Morasso & Schieppati, 1999, Winter et al., 1998). During locomotion, the CoM and the CoP in the sagittal plane must be separated in order to create a lever arm and thus a disequilibrium torque driven by the force of gravity. In gait initiation, this is done initially by means of a backward shift of the CoP (Brenière et al., 1981; Crenna & Frigo, 1991; Jian et al., 1993). Then, during the single stance phase the body starts rotating around the ankle forcing the CoP to move forward. At late stance, the forward shift of the CoP is stopped as it is constrained within the anterior geometrical limit of the stance foot. Meanwhile, the CoM maintains its forward momentum and thus the lever arm between CoM and CoP cannot but sharply increase (Figure III.4).

Examination of the traces of CoM-CoP gap and of disequilibrium torque explains how the AP GRF is created. While the time courses of the AP GRF, CoM AP and CoP displacements are different, the time courses of the gap between CoM and CoP and of the disequilibrium torque are quite superimposed to that of AP GRF. In other words, AP propulsive force is modified by the unique control of the CoP position and the GRF vector. Similar results were obtained by Gruben and Boehm (2012a, 2012b), who showed that during level walking CoP is shifted such that the GRF vector always points to a specific reference in close proximity of CoM. In our case, when the subjects were loaded, the sole increase of their body weight was accountable for the increase in the disequilibrium torque that appears on Figure III.4, since gap or EMG activity were not affected by the load. This increase in torque was transformed into AP GRF according to Cavagna & Franzetti (1986).

The lever-arm was found to be around 48.5% and 51.5% of step length for spontaneous and fast steps respectively. Therefore, in order to increase CoM progression velocity (in the fast speed condition), one must increase step length, which will in turn increase CoM-CoP gap because a longer step implies an ampler displacement of CoM. Since CoM-CoP gap increases with a lengthier step then so will the disequilibrium torque along with the propulsive force. However, to obtain a larger CoM-CoP gap, forward momentum of the CoM should increase, while the CoM vertical position has to be kept from descending beyond a certain threshold, or else lifting up CoM again to perform the second step would not be energy efficient. To do so, subject have to increase the braking action, which requires more force and motor units to be recruited in the three muscles SOL, GM and GL. This increase of EMG activity to provide a stronger body support at higher velocity is in line with the results provided by Liu et al., (2008).

#### *The braking action of the triceps surae*

EMG activity of the triceps surae remained unchanged when increasing propulsive force (as needed by adding an extra load to body weight) while maintaining the same velocity. This opens the question of the motor strategy used to control balance during gait. Some authors used different modelling techniques and agreed that the triceps surae does play a role in body support, a term broadly used to designate the control of balance (Neptune et al., 2001; Anderson & Pandy, 2001; Liu et al., 2008, Wright et al., 2012). Body support was defined as a vertical force applied by the triceps surae to resist gravity or, in other terms, to brake the fall of the CoM. Vertical braking of the CoM has been previously investigated in elderly people and in subjects suffering from motor disorders such as Parkinsonian patients. Interestingly, both populations showed either insufficient or no braking of the CoM when they initiated gait (Chastan et al., 2010; Holt et al., 2003). Here, the braking of CoM fall is seen clearly in the Ver GRF profile, since GRF increased beyond body weight when the downward velocity of CoM reversed, resulting in actively deceleration of the CoM before foot contact (Figure III.1). Furthermore, when the load was added, the Ver GRF recorded throughout the trial increased systematically by around 200 N, as predicted. However, for the same velocity, the vertical braking action (the distance 3 to 4 in the Ver GRF traces of Figure III.1) was not affected by load and remained constant (bars in Figure III.3B, left panel). This can be explained by ideally considering the body as an inverted pendulum, where the body is a point mass positioned at CoM and rotates around CoP. Newton's law of motion  $\tau_{\text{ext}} = I \cdot \alpha$  (where  $\tau_{\text{ext}}$  is the

net external torque,  $I$  is the moment of inertia and  $\alpha$  is the angular acceleration) can be then applied. In our case, the equation is expressed as  $mg \cdot (\text{CoM-CoP gap}) = m \cdot r^2 \alpha$  (where  $m$  is body mass,  $g$  is the gravitational acceleration and  $r$  is the distance between CoM and CoP). By dividing both parts of the equation by  $m$  results in the downward acceleration of the CoM being only affected by the CoM-CoP gap, which should be constant for the same step length according to Gruben and Boehm (2012a, 2012b), regardless of the load.

Thus, by controlling CoM-CoP gap, the triceps muscle exerts the activity necessary for keeping the body upright in spite of the added load. Even under standing condition, a torque is exerted by triceps surae in order to counteract the gravity torque, since the CoM projection lies just in front of the ankle joint (Schieppati et al., 1994; Morasso & Schieppati, 1999); the added load requires a minor but definite increase in the antigravity activity. At gait initiation, in the loaded compared to unloaded condition, subjects anticipated the onset of the burst in the three triceps muscles (SOL, GM and GL). This low-level, early activation of the triceps surae muscle, performing eccentric contraction, would help stiffening and stabilising the ankle joint as a result of the increased bodyweight, right at the critical time when the double support turns into the single support, and when the foot arch flattens under the effect of the new load on the single supporting foot and of the forward tilt of the tibia (Jonkers et al., 2003).

On the other hand, when subjects performed fast walking, the braking action augmented, since more force was required to counter the larger CoM vertical displacement. The increase in vertical action was accompanied by an increase in triceps EMG activity. The reason behind the increased EMG during the braking action phase is to prevent the CoM from falling beyond a certain level, where rising it up again for the second step would be metabolically costly as energy transfer would be less effective. On the other hand, velocity *per se* had no significant effect on the time of onset of triceps muscles' burst. Our results are complementary to those of Holt (2003), who found that the vertical displacement of CoM was increased with faster walking velocity but was not affected by load. Interestingly, triceps activity is maintained for some time after FC and is silenced briefly prior to lifting of the lagging leg into swing as the tibialis anterior of that leg becomes active. Kuo (2007) has explained that in double stance CoM does not require to be propelled and lifted, but redirected due to the net action of both legs exerting positive (lagging leg) and negative (leading leg) work, since CoM ends single support with an appropriate height, momentum and energy. However, it is also possible that

the work performed by the triceps surae of the lagging leg during double stance contributes to lifting that leg into swing along with hip flexor activity (Neptune et al., 2001).

Other evidences from remote lines of investigation point to the triceps as a major controller of the effects of gravity on body mass. Sinkjaer et al., 2000 applied slow-velocity enhancements and reductions to the natural ankle dorsiflexion during the stance phase of walking, thus mimicking variations potential changes in the ankle dorsiflexion trajectory connected to uneven ground during walking, and found that dorsiflexion enhancements generated gradual increments in the soleus EMG. Afferent feedback from large- and medium-diameter spindle sensory fibres was shown to contribute to the background SOL activity (Dietz & Duysens, 2000; Mazzaro et al., 2005). Such mechanism may be at least in part responsible for the gradual build-up in the SOL EMG burst during the stance phase of gait initiation and contribute to the control of the braking action, as well as for the increase in triceps activity during fast walking (accompanied by larger ankle dorsiflexion). On a different vein, it is pertinent to recall here that by merely controlling the braking action through triceps activity, the CNS can modulate walking velocity, and parsimoniously produce the difference in step length between legs necessary for producing steering of the walking trajectory (Courtine & Schieppati, 2003; Courtine et al., 2006).

### III.6 Summary

Triceps surae EMG activity did not change when propulsive forward force increased due to the effect of adding a load. This was true both for spontaneous and for fast velocity condition. Therefore the hypothesis stating that the triceps pushes-off the ground to generate propulsive force is discarded. The triceps is instead responsible for balance control by braking CoM vertical displacement. In this light, the ‘push-off’ term itself is more a confounding misnomer than a short designation of the real increase in propulsive torque occurring in the second part of the stance phase. The forward progression of the body is only due to the transformation of the potential energy of the CoM fall into forward kinetic energy (Cavagna et al., 2000). EMG activity increases only when the vertical braking action of CoM augments as a result of increasing body support demand while walking quickly. By controlling body support, triceps modulates the antero-posterior distance between the centre of mass (CoM) and the centre of foot pressure (CoP), thereby indirectly modulating walking velocity.

## IV. Study Two

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By counteracting gravity, triceps surae sets both kinematics and kinetics of gait.

(To be submitted to the Journal of Physiology).

### Keypoints:

- During single stance of gait, the human body behaves like an inverted pendulum, which rotates around stance foot.
- The rate of rotation of the pendulum is determined by gravity and initial velocity prior to single stance.
- Triceps surae activity decelerates the fall of the centre of mass by resisting the rotation of the body, thereby setting step length and cadence.
- Triceps surae also contributes to avoiding the swing leg to impact the floor abruptly and allows for a smoother step-to-step transition.
- These results explain how the central nervous system sets global gait kinematics via modulation of triceps-surae activity.

## IV.1 Abstract

**Aim:** In the single-stance phase of gait, the human body behaves like an inverted pendulum. Gravity acting on the centre of mass (CoM) causes a disequilibrium torque, which generates propulsive force. Triceps-surae activity resists gravity by restraining forward tibial rotation and is capable of tuning CoM momentum. We hypothesised that time- and amplitude modulation of triceps surae activity determines the kinematics (step length and cadence) and kinetics of gait.

**Methods:** Nineteen young subjects participated in two experiments. In the Gait Initiation (GI) protocol, subjects deliberately initiated walking at different velocities for the same step length. In the Balance Recovery (BR) protocol, subjects executed steps of different length after being unexpectedly released from an inclined posture. Ground reaction force was recorded by a large force platform and EMG of soleus, gastrocnemius medialis and lateralis, and tibialis anterior muscles was collected by wireless surface electrodes.

**Results:** In both protocols, the duration of triceps activity was highly correlated with single-stance duration (GI,  $R^2=0.68$ ; BR,  $R^2=0.91$ ). Step length was highly correlated with single stance duration (BR,  $R^2 = 0.70$ ). Control of CoM momentum was obtained by vertically decelerating the CoM fall via modulation of amplitude of triceps activity. Doing so allowed the CNS to control the position of CoM with respect to the centre of pressure (CoP). The CoM-CoP gap in the sagittal plane was determinant for setting the disequilibrium torque and thus walking velocity. Thus, by controlling the gap, CNS modified walking velocity (GI,  $R^2=0.86$ ; BR,  $R^2=0.92$ ).

**Discussion:** This study is the first to highlight that by merely counteracting gravity, triceps activity sets the kinematics and kinetics of gait. It also provides evidence that the surge in triceps activity during fast walking is due to the increased requirement of braking the fall of CoM in late stance in order to perform a smoother step-to-step transition.

**Keywords:** Gait, step duration, step length, triceps surae, EMG, kinematics, kinetics, CoM, CoP, gravity torque.

## IV.2 Introduction

In daily life, we walk at different velocities. Walking necessitates proper orchestration of lower limb muscle activity by the central nervous system (CNS) in order to set specific kinetic and kinematic parameters. Kinetically, locomotion requires the application of a propulsive force to accelerate the body and achieve a certain walking velocity. Kinematic variables are strictly linked together by the equation:  $V = L \times C$ , where  $V$  is walking velocity,  $L$  is step length and  $C$  is cadence (Nilsson et al., 1985; Brenière, 1996; Bertram & Ruina, 2001). Many studies have investigated the relationship between the three variables (Alexander, 1984; Nilsson et al., 1985; Stoquart et al., 2008; Leurs et al., 2011; Ivanenko et al., 2011). These studies, mostly conducted on a treadmill, proved that all combinations between walking velocity, step length and cadence are possible.

Human bipedal gait is a particular form of locomotion, characterised by a succession of single- and double-stance phases. During single stance, gravity causes the body's centre of mass (CoM) to fall and rotate around the stance foot where the ground reaction force is applied. Cavagna et al. (1976) showed that the downward kinetic energy of the CoM is transformed into forward propulsive energy during walking. In a previous study, we showed how gravity acting on the CoM generates a disequilibrium torque, which is responsible for propelling the body (Honeine et al., 2013). The disequilibrium torque was the product of the antero-posterior distance between CoM and centre of foot pressure (CoP) by the vertical force acting on the CoM. In that study, we also provided evidence, by monitoring the triceps EMG activity when subjects initiated gait with and without a load, that triceps surae does not directly contribute to body propulsion. For the same walking velocity, loading the subjects necessarily increased propulsive force but did not alter the amplitude of the triceps EMG. We concluded that the role of the triceps surae during gait is to support the body during single stance. Nonetheless, the amplitude of triceps surae activity of the stance leg covaried with walking velocity during single stance (Winter, 1983; Hof et al., 2002; Den Otter et al., 2004). In the present study, we investigated the mode through which the time course of triceps activity can ultimately produce different walking velocities. Furthermore, in order to advance knowledge of the mechanisms underpinning the covariation between walking speed and triceps amplitude, we investigated whether the rise in triceps activity in fast walking is imposed by the need of additional braking action to counteract the fall of CoM.



We took advantage of the inverted pendulum mechanics in order to understand how modulation of the amplitude and duration of triceps surae activity sets walking velocity. In a passive inverted pendulum, CoM momentum is generated solely by the pull of gravity. During the single-stance phase of human gait, the CNS controls CoM momentum (Popovic et al., 2004; Herr & Popovic, 2008; Neptune & McGowan, 2011), since triceps surae restrains tibial rotation (Sutherland et al., 1980). We hypothesized that modulation in amplitude and duration of triceps activity determines both kinematics and kinetics of gait. More precisely, we postulated that control over the amplitude of triceps activity allows the CNS to adjust CoM momentum, while the duration of triceps activity determines the duration of the single stance. By controlling CoM momentum and single-stance duration, the CNS should set how far the body travels away from the stance foot before the foot-contact of the swing leg, i.e., it would determine step length. In the process, the distance of CoM with respect to CoP, or the CoM-CoP gap, will be set. This in turn determines the amplitude of the disequilibrium torque, which generates the propulsive force and adjusts walking velocity.

In order to test our hypothesis, we exploited two complementary protocols, in which certain variables can be isolated. In the first experiment, we investigated how triceps controls walking velocity when subjects walk with a fixed step length. In the second experiment, we examined how triceps defines step length. Combining the results of both experiments should allow understanding how the pattern of triceps surae activity sets velocity in all the kinematic combinations of gait.

In the first experiment, we opted for the gait initiation protocol (GI). In GI, when subjects start walking spontaneously, they perform each time a step of a rather constant length (Brenière & Do, 1991; MacDougall et al., 2005). According to the equation  $V = L \times C$ , if subjects are instructed to vary walking velocity without changing step length then the duration of the single stance has to be modulated. We expect that to walk slowly, the CoM momentum should decrease while the duration of single stance should increase. The opposite should occur when walking faster. In the preparatory phase of GI, the amplitude of the CoM momentum is controlled by the tibialis anterior activity (Nardone & Schieppati, 1988; Lepers & Brenière, 1995). During the following single-stance phase, the amplitude of the triceps activity should be modulated in order to fine tune the CoM momentum and react to the CoM fall.

In the second experiment, designed to test how step length is determined, we resorted to the balance recovery protocol (BR). In BR, subjects are positioned in an inclined position then released. When released, the fall provokes an automatic step, the length of which is determined by the angle of inclination (Do et al., 1982; Pai & Patton, 1997; Aftab et al., 2012). Asking subjects to vary step length, without changing the initial inclination, should modulate the duration of triceps surae activity accordingly. Reducing the duration of triceps activity should cause earlier contact with the ground resulting in a short step. The opposite should occur when performing a longer step. Notably, the CoM momentum of the body does not change prior to single stance when subjects are asked to recover balance with different step-lengths (Do et al., 1982). This should allow us to investigate how triceps modifies CoM momentum without the influence of events occurring before single stance.

### IV.3 Material and Methods

Nineteen healthy volunteers (9 females and 10 males) took part in the experiments after giving written informed consent as required by the Helsinki Declaration and the local Ethics committee. Their mean age, body mass and height were 25 yrs (range 20-29), 72 kg (48-92) and 1.76 m (1.61-1.83), respectively. Eleven subjects participated in the GI protocol. Eleven subjects performed the BR experiment. Three subjects performed both experiments. Two different groups were used for each of the two experiments because the results of each protocol were not to be directly confronted.

#### *Experimental set-up*

A large force platform (0.90 m wide, 1.80 m long, AMTI, USA) was used to record ground reaction force (GRF) and moments. The platform was embedded in the ground and placed at 0.5 m from the wall. Subjects stood on the platform so that the ground reaction force of the first step could be recorded. The entire walkway was long enough for the subjects to carry out several steps. Surface EMG activity was recorded from the soleus (SOL), gastrocnemius medialis (GM), gastrocnemius lateralis (GL) and tibialis anterior (TA) muscles of both legs using bipolar Ag-AgCl electrodes (8 mm diameter, 20 mm inter-electrode distance). Electrodes were placed according to the SENIAM protocol (Merletti & Hermens, 2000) after preparing the skin to minimize impedance. EMG activity was on-site pre-amplified (x1000,

Zero-Wire, Aurion, Milan, Italy), wirelessly sent to a PC and band-pass filtered (10-500 Hz). Force platform and EMG data were digitized at a sampling frequency of 1000 Hz on the same A/D converter card and saved for off-line analysis. A Matlab 2008b (Mathworks, MA, USA) routine was used for processing the data.

For the ‘balance recovery’ (BR) protocol, an electro-mechanical device composed of a load cell (S-Beam, Vishay Celtron, USA) coupled to an electromagnet was attached via a sliding joint to a vertical shaft fixed to the wall (Do et al., 1982). An abdominal belt was used to attach the subject to the device by means of a steel cable. This allowed subjects to stand quietly in a forward-inclined position. The sliding joint permitted to set the cable horizontally. The cable was released by operating on the electromagnet. The load cell was used to identify the time of the release of the cable. It also helped measure the body inclination of the subjects, where  $\text{inclination} = \tan^{-1}(\text{force of load cell} / \text{Ver GRF})$ . To ensure that the cable was kept horizontal, the subjects’ bodyweight was measured under quiet stance. A change in this measure while inclined indicated that the tension in the cable had a vertical component (i.e. the cable was not horizontal).

### *Protocols*

Subjects stood barefoot on the platform, looking straight ahead. The contour of the feet was drawn with a chalk on the platform so that the subjects always stood in the same position throughout the experiment.

For ‘gait initiation’ (GI), the subjects stood erect on the platform until they were instructed to initiate gait following a verbal go-signal. Then, they continued walking until the end of the walkway. They were told not to start walking in a reaction-time mode, but when they felt ready. This usually occurred within 2 s from the go-signal. The subjects performed three sets of walking tasks composed of 10 trials each. In the first task, the subjects started walking at their usual velocity (normal condition). Then, they were asked to walk slow or to walk fast. The slow and fast tasks were performed in random order across the subjects.

For ‘balance recovery’ (BR), the subjects were inclined forward and stood as relaxed as possible. Body inclination with respect to the vertical was kept at 15° and monitored by means of the feedback from the force platform and the force transducer on the cable. Upon

release, the subjects continued walking until the end of the walkway. The time of release was unknown to them. The subjects performed three sets of walking tasks composed of 10 trials each. In the first task, subjects were given no instruction about step length. Then, they performed short and long steps. The order of the short-step and long-step tasks was randomized across the subjects.

Subjects initially performed 6 blank trials for both GI and BR to get accustomed to the protocols. During the blank tests, the preferential leading foot of each subject was detected so that subjects would later initiate gait or recover from fall using the same leading leg throughout the experiment. On average, each of the experiments (GI or BR) lasted 45 to 60 minutes.

### *Ground reaction force*

The platform measured the ground reaction forces (GRF) and moments in the antero-posterior (AP), medio-lateral (ML) and vertical (Ver) directions. From these, the CoP coordinates were computed, according to an established procedure (Brenière & Do, 1991; McIlroy & Maki, 1999). The time of foot-off, i.e. the onset of the single-stance phase of gait, was the instant when the ML CoP moved under the stance foot. The time of foot contact was the instant when the AP CoP position shifted abruptly forward. The procedure of timing the foot-off and foot-contact by means of the force platform output was previously validated with respect to foot-switch data (Caderby et al., 2013). The CoM momentum is calculated as:  $\text{Momentum} = m \times v$ , where  $m$  is the mass of the subject and  $v$  is the velocity of CoM. CoM velocity was obtained by time-integrating CoM acceleration. CoM AP acceleration was calculated as  $\text{AP GRF}/\text{BM}$ , and CoM vertical acceleration as  $(\text{Ver GRF} - \text{BW})/\text{BM}$ , where BW and BM are body weight and body mass, respectively. BW and BM were measured by the force platform when subjects were standing still in upright posture. CoM position was obtained by time-integrating CoM velocity. In GI, the initial position of CoM in the AP direction was considered to be equal to that of the CoP (Winter, 1995). In BR, the subjects were initially inclined, so that the CoM was positioned away from the CoP. To estimate the initial position of CoM, we asked subjects to stand in bipedal erect stance. The horizontal distance between their anterior superior iliac spine and the wall behind them was measured ( $d_1$ ). This was compared with the same distance ( $d_2$ ) measured when subjects were inclined for the task. Hence, the CoM initial AP position was ( $d_2 - d_1$ ).

### *Disequilibrium torque*

During gait, CoM moves up and down while rotating around the CoP in the sagittal plane, and the CoM fall is braked during the single stance phase of gait. The displacement of the CoM generates a disequilibrium torque, driven by gravity, as CoM moves beyond the CoP. The disequilibrium torque was calculated as  $\text{Ver GRF} \times (\text{CoM-CoP})$ . The difference (CoM-CoP) represents the instantaneous distance (hereafter called the gap) between the AP position of the CoP and the corresponding position of the ground projection of the CoM.

### *EMG analysis*

To better display the EMG activity (Figs 1 and 2), the envelopes of SOL, GM, GL and TA were calculated. To do so, the EMG signals were rectified and low-pass filtered with a Butterworth 3<sup>rd</sup> order low-pass zero-lag filter. The cut-off frequency was set at 30 Hz. To quantify the duration of triceps surae activity, the onset and end of the SOL, GM and GL EMG were detected. These time instants were carefully identified visually on the raw EMG traces of the three muscles and expressed relative to the time, at which gait (GI) or balance recovery (BR) initiated (at  $t_0=0.0s$  in the Figures). We also wanted to understand how the activity of the triceps surae was modulated in amplitude during single stance. The triceps surae activity was quantified based on the rectified SOL, GM and GL EMG signals, which were time-integrated from foot-off (FO) until foot-contact (FC). The integrals were then divided by the duration of the muscle activity to obtain the mean level of activity. In GI, the amplitude of TA activity was calculated by integrating the rectified EMG from  $t_0$  until FO. To further specify triceps EMG activity, we divided the single-stance period in four equal-duration intervals: early (0-25%), early-mid (25-50%), mid-late (50-75%) and late (75-100%) stance. Mean levels of SOL, GM and GL EMG were then computed in each interval. The average vertical acceleration was also computed in each time-window. A positive acceleration indicates that the CoM fall is being actively braked. Acceleration was therefore used in order to quantify the vertical braking action of CoM.

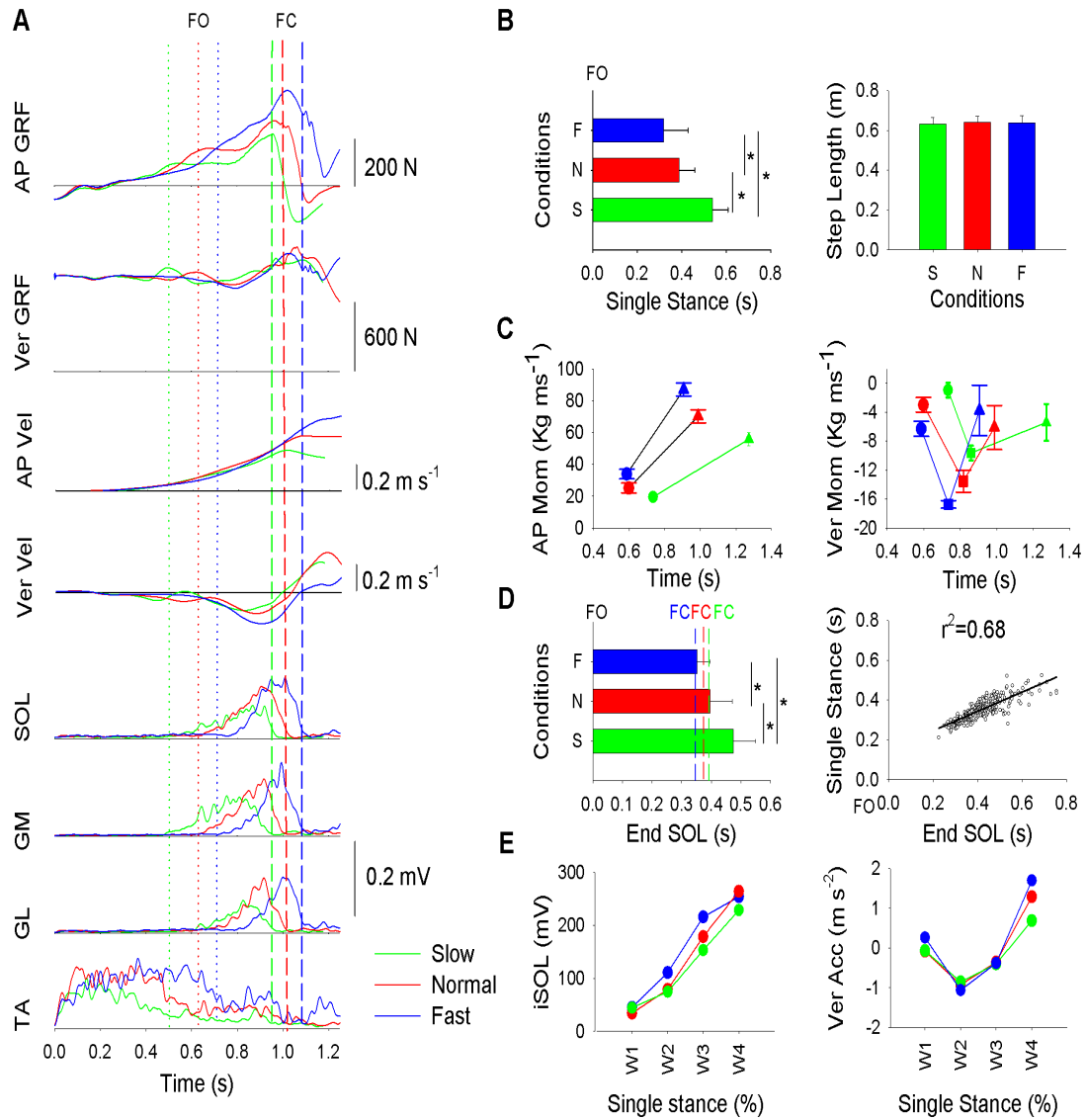
## *Statistics*

Analyses were performed with the SPSS software (SPSS 20.0, Chicago, IL). One-way repeated-measure ANOVA was performed across step-length conditions in BR and across walking velocities in GI for each of the outcome variables: step length, CoM AP position at foot-contact, CoM AP & Ver momentum at foot-off and foot-contact, average EMG amplitude of SOL, GM and GL during the single stance, the average EMG amplitude of TA prior to single stance in GI and finally the CoM-CoP gap and disequilibrium torque at foot-contact. Two-way ANOVA separately assessed the differences in the end of the EMG activity of SOL, GM, and GL. The first categorical factor was step length in BR and walking velocity in GI. The second factor was muscle (SOL, GM and GL). Repeated-measure ANOVA was used to assess differences in the average SOL, GM and GL activity in addition to the average vertical acceleration of CoM in the four time-windows, in which the single stance was divided. As in the one-way ANOVA, the categorical factor was step length in BR and walking velocity in GI. The repeated-measures were the four time-windows. Post-hoc tests were performed using the Bonferroni corrected paired t-tests. The p value was set at 0.05. The linear relationship between the CoM-CoP gap and CoM AP velocity was assessed using Pearson's  $r^2$  coefficient of determination.

## IV.4 Results

This section is divided into three main parts. In the first two parts, we provide the results describing the triceps surae activity in relation to the two global kinematic variables of gait: cadence for gait initiation (GI) and step length for balance recovery (BR). In the last part, we describe how propulsion is determined by modulating the disequilibrium torque via control of triceps activity in both the GI and BR protocol. In each part, qualitative data of a representative subject are followed by quantitative data, i.e. grand means and standard deviations from all subjects. The result section only reports data measured during the execution of the first step, i.e. from  $t_0$  till foot-contact.

## Gait Initiation: the duration of triceps activity determines cadence



**Figure IV.1** Gait initiation.

Green, red and blue indicate slow, normal and fast walking velocity, respectively. (A) shows the time-course of gait initiation variables in a representative subject (each trace is the average of 12 trials). Mechanical variables (AP & Ver GRF and CoM velocity) and the EMG envelopes of SOL, GM, GL and TA are shown from top to bottom. Vertical dotted lines indicate the instant of foot-off (FO), dashed lines the instant of foot-contact (FC). In (B, left) the horizontal bars indicate the grand mean ± SD of duration of single stance. In (B, right) the bars depicts the grand mean ± SD of step length. In (C, left) the plot depicts the grand mean ± SD of CoM AP momentum at FO (circles) and FC (triangles). In (C, right) the plot depicts the grand mean ± SD of CoM Ver momentum at FO (circles), minimum value (square) and FC (triangles). In (D, left) the horizontal bars are the grand mean ± SD of the end of activity of the EMG activity of SOL with respect to FO ( $t=0$ ). The dashed lines indicate the grand mean of the time of FC. In (D, right) the time instant of the end of soleus activity with respect to FO is plotted against the single stance duration (all trials collapsed). There is a strict correspondence between the duration of muscle activity and duration of single stance. The linear equation is:  $y=0.49x + 0.14$  (E) shows the average values of the level of EMG activity of SOL (left) and the average CoM vertical acceleration (right) measured in the 4 equal time-windows, in which the single stance was divided. The mean level of EMG increases with the progression of the single stance and increases in parallel with the acceleration.

Subjects were asked to initiate gait at different speeds without changing step length. For a fixed step length, cadence (proportional to the duration of single stance) must increase when walking faster and decrease when walking slower. So when walking slowly, we expected subjects to increase the duration of triceps activity, which would in turn increase single stance duration. The reverse should occur when walking fast. Figure 1A shows the time profiles of GRF and CoM velocity in AP and Ver directions and the envelopes of the EMG of SOL, GM, GL and TA of the stance leg of a representative subject (each trace is the mean of 12 trials) under all GI conditions (green, red and blue indicate slow, normal and fast walking, respectively). GRF traces are divided in double- (from t0 to FO) and single-stance phase (from FO to FC). The disequilibrium torque is initiated by SOL silence and TA activation, both shifting the CoP backwards (Carlsöö, 1966; Nardone & Schieppati, 1988; Lepers & Brenière, 1995; Crenna & Frigo, 1991; Jian et al., 1993). It has been previously reported that TA activity increases when subjects initiate gait faster, in order to increase the momentum of the CoM prior to single stance (Lepers & Brenière, 1995). In this experiment as well, TA increased its activity as subjects walked faster (Fig. 1A, bottom traces).

In the single-stance phase, AP GRF increased and reached its maximum value slightly after foot-contact. In the slow and normal condition, AP GRF showed a tendency to plateau before increasing towards late stance. In the fast condition, it continued rising until mid-stance and increased further in late stance. At foot-contact, the AP GRF peak was lowest when the subject walked slowly and highest when subjects walked faster. In all conditions, Ver GRF fell below bodyweight, reached a minimum value around mid-stance and increased beyond bodyweight in late stance. Ver GRF value below bodyweight indicates that CoM is accelerating downwards, while Ver GRF above bodyweight reflects an upward CoM acceleration, or, in other terms, it indicates that the fall of CoM is being braked. AP CoM velocity increased in a parabolic fashion for all velocities and no difference was present in the shape of the traces across the three tasks before each respective foot-contact. Ver velocity curves show that following foot-off CoM falls until mid-stance, it then increases to reach a value around zero at foot-contact. The minimum value of Ver CoM velocity is lowest during slow walking and highest during fast walking.

#### *Duration of single stance and walking velocity*

There was an effect of walking velocity on the duration of the single stance (one-way



ANOVA,  $F(2,30) = 15.7$ ,  $p < 0.001$ ). The duration of the single stance decreased when walking faster and increased in slow walking (post-hoc,  $p < 0.05$  for all comparisons). In Figure 1B (left) the grand mean and standard deviations of the duration of the single-stance (the length of the bars) is represented with respect to the grand mean of foot-off (origin of the abscissa). Figure 1B (right) shows the grand means and standard deviations of step length. As expected, walking velocity had no effect on step length (one-way ANOVA,  $F(2,30) = 0.18$ ,  $p = 0.8$ ).

#### *AP and Ver momentum of CoM*

In Figure 1C (left), the grand means and standard deviations of AP momentum of CoM at the time instant of foot-off (FO, circles) and of foot-contact (FC, triangles) are shown. At foot off, there was a difference in CoM AP momentum (one-way ANOVA,  $F(2,30) = 9.2$ ,  $p < 0.001$ ). Momentum was not different between slow and normal walking (post-hoc,  $p = 0.41$ ), but increased in fast walking with respect to normal and slow speed ( $p < 0.05$ , for both comparisons). ANOVA showed a significant effect of walking velocity on TA activity just prior to single stance, i.e., from t0 to FO ( $F(2,30)=8.32$ ,  $p < 0.01$ ). The post-hoc test showed that the average TA EMG amplitude prior to foot-off was highest during fast walking ( $p < 0.05$ ). One-way ANOVA showed that the instant of foot-off was also slightly different between the three conditions. Foot-off occurred earlier at normal speed than at fast and at slow walking momentum (post-hoc,  $p < 0.05$ ). At foot-contact, there was a significant difference in CoM AP momentum between conditions (one-way ANOVA,  $F(2,30) = 19.2$ ,  $p < 0.001$ ). At foot-contact, CoM AP momentum was highest for the fast condition and lowest during the slow condition (post-hoc,  $p < 0.05$ ).

In Figure 1C (right), the grand means and standard deviations of Ver momentum of CoM at the moment of foot-off, at its minimum value and at foot-contact are shown. The abscissa in the graph indicates the instant of foot-off (circles), of minimum value of Ver momentum (squares) and of foot-contact (triangles). At foot off, there was a difference in CoM AP momentum (one-way ANOVA,  $F(2,30) = 10.5$ ,  $p < 0.001$ ). Momentum was not different between slow and normal walking (post-hoc,  $p = 0.41$ ), but increased in fast walking with respect to normal and slow speed ( $p < 0.05$ , for both comparisons). For the minimum value of CoM Ver momentum, ANOVA showed a difference between all conditions ( $F(2,30) = 10.2$ ,  $p < 0.001$ ). The minimum value was lowest for slow walking and highest for fast walking.

Finally, the vertical momentum of CoM at foot-contact was not different between walking velocity conditions (one-way ANOVA,  $F(2, 30) = 0.8$ ,  $p = 0.4$ ).

#### *Duration of single stance and time-course of triceps activity*

Single stance was dominated by triceps surae activity. The duration of the activity and the duration of the single stance increased in the slow- and decreased in the fast-velocity condition. For each velocity, two-way ANOVA showed that the end of EMG activity was not different across the individual muscles ( $F(2,30) = 0.12$ ,  $p = 0.88$ ), but revealed a significant effect of walking speed on the end of EMG activity ( $F(2,60) = 69.4$ ,  $p < 0.001$ ). Grand means and standard deviations with respect to foot-off are shown in Fig. 1D (left). The post-hoc test indicated that the end of triceps surae EMG activity was anticipated for fast walking and was delayed for slow walking with respect to normal condition ( $p < 0.001$ , for all comparisons). Since the temporal behaviour of the three muscles was similar, then for clarity only grand means and standard deviations of SOL are provided in Fig. 1D (left). The linear relationship between the end of SOL activity and the duration of the single stance had a coefficient of determination ( $r^2=0.68$ ) indicating a strong association between the two variables (Fig. 2D, right). The obtained linear equation was  $y=0.49x + 0.14$ .

#### *EMG activity and vertical braking action of CoM*

To further investigate the modulation of the level of triceps activity in the control of Ver acceleration, we calculated the average level of triceps EMG and Ver acceleration in four equal-duration time-windows of single stance (W1 to W4 in Fig 1E). Repeated-measures ANOVA showed a difference in the EMG level of each muscle for the three walking velocities (SOL,  $F(2,30) = 4.6$ ,  $p < 0.05$ ; GM,  $F(2,30) = 9.7$ ,  $p < 0.05$ ; GL,  $F(2,30) = 12.4$ ,  $p < 0.001$ ). Post-hoc test showed an increase in EMG level when walking faster ( $p < 0.01$ , for all comparisons). The post-hoc test indicated that triceps activity increased progressively from one time-window to the next in each of the three muscles for each of the walking velocity conditions ( $p < 0.05$ ). In W1, the post-hoc showed no effect of walking velocity on triceps activity. In W2, triceps EMG level increased only in the fast condition ( $p < 0.01$ ). In mid- to late-stance, triceps EMG level was highest in fast and lowest in slow walking in W3 ( $p < 0.05$ ), while in W4 EMG level was lower in slow walking with respect to the normal condition and highest in the fast condition ( $p < 0.05$ ). Since the results obtained for each of

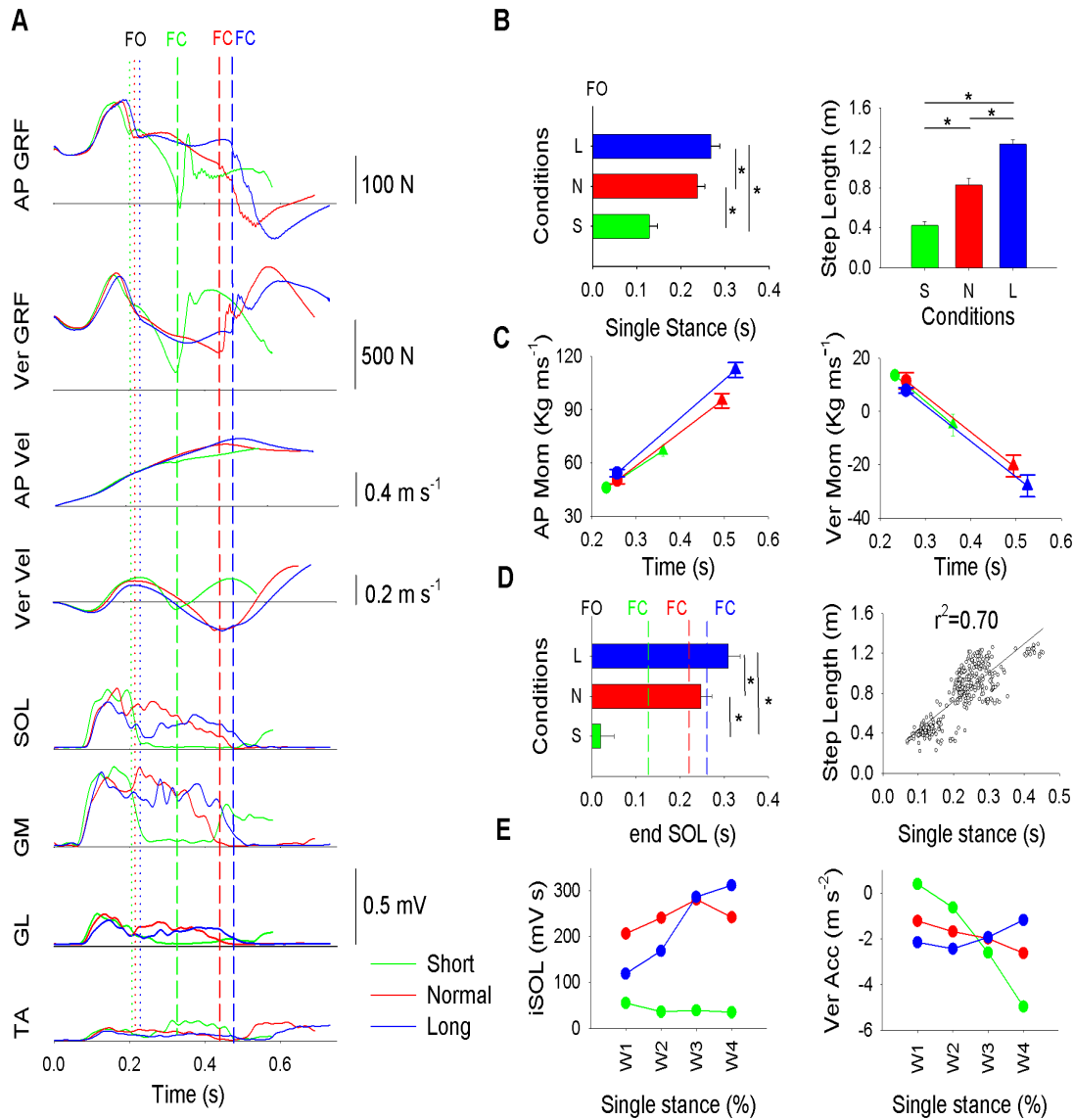
muscle were superimposable, then for clarity only data of SOL EMG are shown in Fig. 1E (left panel).

Repeated-measure ANOVA also showed that Ver CoM acceleration was different in the four time-windows ( $F(3,40) = 24.5$ ,  $p < 0.001$ , respectively). The CoM accelerated downwards during early and mid-stance. It reverted upwards in mid- to late stance and became positive, i.e. vertical velocity decreased in late stance. In W1 and W2, Ver CoM acceleration was highest in fast walking ( $p < 0.05$  for both time-windows), but did not change between normal and slow walking. Ver CoM acceleration did not change in W3. In W4, Ver CoM acceleration was always positive, indicating that CoM vertical velocity is decreasing. The braking action on CoM was highest for fast and lowest for low walking ( $p < 0.01$ ). This suggests that when CoM vertical velocity increases as a result of fast walking, the triceps activity in late stance augments in order to increase the braking action. The increase of the braking action decreases vertical momentum so that at foot-contact CoM Ver velocity is unchanged between walking velocity conditions.

### ***Balance Recovery: triceps surae activity determines step length***

Figure 2A shows the mean (12 trials) time profiles of GRF and CoM velocity in AP and Ver directions, and the EMG of SOL, GM, GL and TA envelopes of the stance leg of one representative subject in all balance-recovery conditions (green, red and blue indicate short, normal and long steps, respectively). GRF traces are divided in double- (from t0 to FO) and single-stance phase (from t0 to FC).

For a brief period (less than 250 ms) following the release of the cable, both feet were in contact with the floor. CoM was initially positioned largely ahead of the CoP due to the inclination of the body, and the release of the cable caused the gravitational torque to have a high value due to the presence of a large CoM-CoP gap and the body weight of the subject. The torque caused CoM to fall rapidly for around 50 ms. The fall was followed by a reaction phase characterized by upward and forward increase in GRF and short-latency activation of triceps surae (Do et al., 1990). The upward surge in GRF caused the CoM to accelerate upwards, during which period subjects recovered a more vertical orientation of the body. In this time period, no major difference in GRF and EMG can be observed between all step-length conditions during double-stance, as previously shown by Do et al. (1982).



**Figure IV.2 Balance recovery.**

(A) shows the time-course of balance recovery variables in a representative subject (each trace is the average of 12 trials). Same layout as in Fig. 1. In (B, left) the horizontal bars indicate the grand mean  $\pm$  SD of duration of single stance. In (B, right) the bars depicts the grand mean  $\pm$  SD of step length. In (C, left) the plot depicts the grand mean  $\pm$  SD of CoM AP momentum at FO (circles) and FC (triangles). In (C, right) the plot depicts the grand mean  $\pm$  SD of CoM Ver momentum at FO (circles), and FC (triangles). In (D, left) the horizontal bars are the grand mean  $\pm$  SD of the end of activity of the EMG activity of SOL with respect to FO ( $t=0$ ). The dashed lines indicate the grand mean of the time of FC. In (D, right) step length is plotted against the single stance duration (all trials collapsed). There is a strict correspondence between step length and the duration of single stance. The linear equation is:  $y = 2.87x + 0.14$ . (E) shows the average values of the level of EMG activity of SOL (left) and the average CoM vertical acceleration (right) measured in the 4 equal time-windows, in which the single stance phase was divided. In BR, SOL EMG activity was very low during single stance for short steps, which caused CoM to accelerate quickly downwards. In normal steps, SOL EMG activity was high throughout single stance, which decelerated the CoM fall with respect to short steps where SOL EMG level was low. In long steps, SOL EMG was highest in late stance, which decelerates further CoM fall during this period.

Single-stance features were instead clearly different between short-, normal- and long-step conditions. In the normal condition, i.e. when subjects had no instruction about step length, both AP and Ver GRF decreased until foot-contact. EMG activity in all three heads of the triceps was large in early stance and decreased throughout single-stance until ending at about the instant of foot-contact. When executing short steps, GRF decreased rapidly reflecting a high CoM downward acceleration. This caused the swing foot to touch the floor early resulting in the execution of the short step. Triceps activity ended just slightly after foot-off. The lack of triceps EMG activity in the stance phase of short steps reduces dramatically the body-support, causing the high downward acceleration of CoM. In normal steps, triceps EMG was present throughout single stance, causing CoM to reduce downward acceleration, as reflected by the decrease in Ver GRF, with respect to the short-step condition. For long steps, the subject prolonged the duration of the triceps EMG burst, thus delaying the instant of foot-contact. The EMG amplitude was lower in early stance and increased toward mid-late stance. GRF also decreased in early stance and increased in late stance. During single-stance, AP velocity increased in an almost linear fashion. The time profile of AP velocity was similar during each step-length condition until foot-contact that mechanically braked forward progression. CoM Ver velocity decreased after foot-off. In short step, Ver velocity reached a slightly negative value, since duration of single stance was short. During normal step, Ver velocity decreased throughout single-stance. In long step, Ver velocity decreased too. However, CoM accelerated faster downwards in middle stance with respect to normal step, but then decelerated so that the instantaneous value of the CoM Ver velocity at foot-contact did not change between the normal- and the long-step condition.

#### *Duration of single stance and step length*

Grand means and standard deviations of the duration of single stance are shown in Fig. 2B (left). Step length had a significant effect on the duration of the single stance (one-way ANOVA,  $F(2,30) = 107.8$ ,  $p < 0.001$ ). The post-hoc test indicated that subjects varied the single-stance duration in order to execute steps of different lengths ( $p < 0.001$ , for all comparisons). In the right panel of Fig. 2B, the means and standard deviation of step length at foot-contact are shown. As expected, step length was different across conditions ( $F(2,30) = 143.2$ ,  $p < 0.001$ ) (for all paired comparisons,  $p < 0.001$ ).

### *CoM AP and Ver momentum*

Figure 2C (left) shows the AP momentum of CoM at both foot-off and foot-contact. The abscissa indicates the instant when foot-off (circles) and foot-contact (triangles) occurred. At foot-off, there was no significant effect of step length on CoM AP momentum (one-way ANOVA,  $F(2,30) = 1.9$ ,  $p = 0.16$ ). The mean CoM AP momentum (all trials collapsed) was around  $50 \text{ Kg.m.s}^{-1}$  at foot-off, which is much higher than that obtained normally in GI, since the disequilibrium torque due to the initial CoM-CoP gap linked to body inclination was responsible for the propulsion in the period preceding the step execution. At foot-contact, ANOVA showed a significant effect of step-length on CoM AP momentum ( $F(2,30) = 42.4$ ,  $p < 0.001$ ). CoM AP momentum was highest for long steps and lowest for short steps (post-hoc,  $p < 0.001$ )

Figure 2C (right) shows the Ver momentum of CoM at both foot-off and foot-contact. At foot-off, there was no significant effect of step length on CoM Ver momentum ( $F(2,30) = 1.9$ ,  $p = 0.16$ ). The mean CoM Ver momentum at foot-off was around  $10.5 \text{ Kg.m.s}^{-1}$  (all trials collapsed). At foot-contact, ANOVA showed a significant effect of step-length on CoM Ver momentum ( $F(2,30) = 29.44$ ,  $p < 0.001$ ). Post-hoc test showed that CoM Ver momentum at foot contact was lower in the short-steps compared to normal- and long steps ( $p < 0.001$ , for both comparisons). However, Ver CoM momentum did not change between normal and long steps ( $p = 0.17$ )

### *Triceps temporal modulation and step length*

Two-way ANOVA showed that the end of EMG activity was not different across muscles ( $F(2,30) = 0.21$ ,  $p = 0.97$ ), but there was a significant effect of step-length on end of EMG ( $F(2,60) = 800.4$ ,  $p < 0.001$ ). The end of triceps EMG activity was anticipated for short steps and delayed for long steps with respect to normal condition ( $p < 0.001$ , for all comparisons). Since the temporal behaviour of the three muscles was similar, only the grand means and standard deviations of the end of SOL activity are provided in Fig. 2D (left). The end of triceps activity occurred just shortly after foot-off in short steps, slightly following foot-contact in the normal condition and well after foot-contact for long steps.

A high coefficient of determination was found between the end of SOL activity and the duration of single stance in normal and long steps ( $r^2 = 0.91$ ) (not shown in Fig. 2). The obtained linear equation was:  $y = 2.1x - 0.01$ . In the short-step condition, the initial inclination of the subjects forced them to react quickly to stop the EMG activity as early as possible in order to allow premature contact with the floor. A high coefficient of determination between step-length and single stance duration was obtained ( $r^2 = 0.70$ ) (Fig. 2D right, all trials collapsed). The obtained equation was:  $y = 2.87x + 0.14$ .

#### *EMG activity and vertical acceleration of CoM*

In the BR experiment, the CoM is initially placed away from the CoP and at a lower position than in erect posture. During the balance-recovery step, this condition constrains the CNS to compensate for the imposed gap and check the vertical force by triceps surae activity. To investigate how this is done, we examined the triceps EMG level and Ver CoM acceleration in the four time-windows of single stance. Repeated measures ANOVA showed a difference in the EMG area of each muscle for the different step lengths (SOL,  $F(2,30) = 36.2$ ,  $p < 0.001$ ; GM,  $F(2,30) = 9.4$ ,  $p < 0.01$ ; GL,  $F(2,30) = 37.2$ ,  $p < 0.001$ ). For short steps, SOL activity decreased rapidly, which caused a rapid downward acceleration of CoM. For the normal steps, SOL activity increased until mid-late stance and then decreased in late stance. CoM downward acceleration increased but at much lower rate than in short steps. For long steps, triceps EMG level was low in early to mid-stance and increased in mid- to late stance. Post-hoc test showed that EMG level was always significantly different ( $p < 0.001$ ) in all time windows, except in W3 between normal and long steps ( $p < 0.01$ ). For short steps, triceps EMG activity was down to base level during single stance. Otherwise, triceps EMG level were highest in normal steps in early to mid stance with respect to long steps. However, In W4, triceps EMG level was highest in long steps with respect to normal steps. Since the results obtained for each of muscle are similar, then for clarity only results of SOL EMG level are shown in Fig. 1E (left panel).

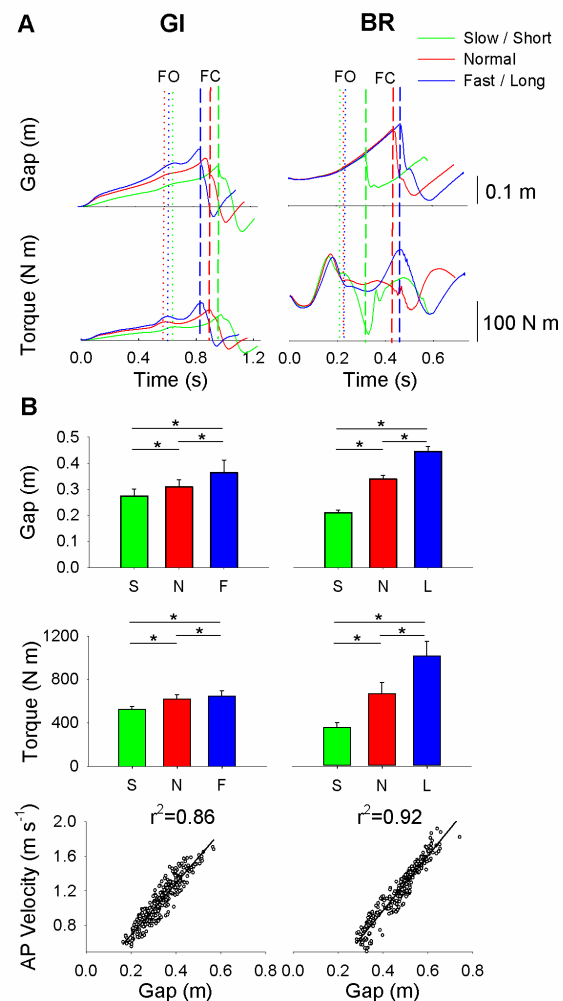
Repeated-measure ANOVA also showed that Ver acceleration was different in the four time-windows ( $F(3,40) = 103.5$ ,  $p < 0.001$ ). Ver acceleration was always negative in all time-windows and for all step-length conditions. Post-hoc test showed that Ver acceleration was always different ( $p < 0.001$ ) in all time windows, except in W3 between normal and long steps ( $p < 0.01$ ). In short steps, the lack of triceps activity during single stance, cause CoM to

quickly accelerate downwards and establish early contact with the ground. In normal steps, the presence of triceps activity decreased downward acceleration with respect to short steps where no activity was present. In long step, the surge of triceps activity in late stance decreased further the downward acceleration of CoM.

### *Disequilibrium torque in GI and BR*

As shown previously in Fig. 1A, the preparatory phase (from  $t_0$  to FO) of gait initiation (GI) was marked by SOL silence and TA activation. This shifted the CoP backwards creating a gap between the CoM and CoP in the sagittal plane that initiated the disequilibrium torque. As stated earlier, TA activity increased in GI when walking faster, thereby increasing the backward shift of CoP, so that gap and torque reached greater instantaneous amplitudes at foot-off. In the single-stance phase, CoM travelled further away from CoP causing gap and torque to build up. In late stance, the gap and torque showed a steep increase (Figure 3A, left). CoM-CoP gap at foot-contact was different between the three walking velocity conditions (one-way ANOVA,  $F(2,30) = 8.9$ ,  $p < 0.001$ ).

It was smallest in slow steps and highest in fast steps ( $p < 0.05$ , for all comparisons). Mean values of the gap are reported in Fig. 3B (upper left). At foot-contact, Ver GRF (that is the other component of the torque) was not different between the three walking velocities ( $F(2,30) = 0.05$ ,  $p = 0.9$ ) (not shown). Mean and standard deviation of disequilibrium



**Figure 3. Disequilibrium torque.**

This figure shows the results obtained during GI (left) and BR (right). In (A) the mean (average of 12 trials) time-courses of CoM-CoP gap and of disequilibrium torque of a representative subject are shown. Same layout as Fig. 1 In (B, upper panel) the bars show the grand mean  $\pm$  SD of the CoM-CoP gap at FC. In (B, middle panel) the bars show the grand mean  $\pm$  SD of the disequilibrium torque at FC. (B, lower panel) shows the position of the CoM in the sagittal



torque are shown in Fig. 3B (middle left). The disequilibrium torque at foot-contact was different between walking velocity conditions ( $F(2,30) = 31.8$ ,  $p < 0.001$ ). It was smaller in short steps and higher in long steps ( $p < 0.01$ , for all comparisons). Figure 3B (bottom left) shows a scatter plot of the CoM-CoP gap against the CoM velocity at foot-contact (all trials of all subjects collapsed). Gap and walking velocity at FC had a strong linear relationship ( $y = 2.97x + 0.1$ ;  $r^2 = 0.86$ ).

In BR, prior to the release of the cable, the AP position of CoM is located around 17 cm forward to that of CoP. This caused the disequilibrium torque to increase very rapidly at the time of the release. During single stance, the CoM-CoP gap increased in a similar fashion in all three step-length conditions, because in BR the CoM AP velocity was unchanged at foot-off. However, the time-profile of Ver GRF changed considerably between conditions, which caused considerable changes in the time-profile of the disequilibrium torque (Fig. 3A, right panel). One-way ANOVA showed that CoM-CoP gap at foot-contact was different between the three step-length conditions ( $F(2,30) = 29.6$ ,  $p < 0.001$ ). Mean values of the gap are shown in Fig. 3B (upper right panel). The post-hoc test showed that gap was smaller in short and higher in long steps, respectively ( $p < 0.01$ , for all comparisons). One-way ANOVA showed that at foot contact, Ver GRF was significantly different between the three walking velocity conditions ( $F(2,30) = 17.9$ ,  $p < 0.001$ ) (not shown), its amplitude at foot-contact being lowest in short steps and highest in long steps. Finally, one-way ANOVA showed that disequilibrium torque at foot-contact was different between walking velocity conditions ( $F(2,30) = 27.8$ ,  $p < 0.001$ ). The post-hoc test showed that torque was smaller in short steps and higher in long steps ( $p < 0.01$ , for all comparisons). Grand mean and standard deviation of disequilibrium torque are shown in Fig. 3B (middle right). Gap and walking velocity at foot-contact had a strong linear relationship ( $r^2 = 0.92$ ) (bottom right). The obtained linear equation was  $y = 2.95x - 0.13$ .

## IV.5 Discussion

### *Rationale of the experiment*

It has been proven long time ago that the changes in potential and kinetic energy during human gait are conveniently in opposite phase ‘as in a rolling egg’ (Cavagna & Margaria, 1966). This allows the pull of gravity to generate a disequilibrium torque, which in turn

produces propulsive force (Cavagna et al., 1976; Cavagna & Franzetti, 1986, Honeine et al., 2013) that ultimately determines the velocity of progression.

In our daily life, we walk at different velocities and are capable of exercising all combinations of step length and cadence to produce a certain velocity (Alexander, 1984; Nilsson et al., 1985; Stoquart et al., 2008; Leurs et al., 2011, Ivanenko et al., 2011). As yet, little insight is available on how the constant pull of gravity is exploited as to produce different walking velocities. Obviously, modulation of walking velocity occurs through control over lower limb muscle activity. Indeed, progression speed and triceps surae activity have been shown to covary (Winter, 1983; Hof et al., 2002; Den Otter et al., 2004; Courtine et al., 2006). However, in a previous paper, we provided evidence, by adding a load to the body, that the main function of triceps surae during walking is to decelerate the CoM fall and not to propel it forwards (Honeine et al., 2013). In the present study, we hypothesised that, during single stance, triceps surae can set global gait kinematics and kinetics by appropriately tailoring its decelerating action on the pull of gravity.

In gait, the two global kinematic variables, step length and duration of single stance, are strongly linked (Murray et al., 1964; Öberg et al., 1993). In this study, we needed to devise an experiment that allowed testing how triceps modulation can generate different combinations of step length and cadence to obtain a wide range of walking velocities. We decided to use two experimental protocols. In the first protocol (gait initiation, GI), we studied how triceps surae activation can modulate single stance duration. In the second (balance recovery, BR), we studied how triceps surae activation varies step length. Combining both results obtained in GI and BR allowed us to get insight into the link between muscle activation and walking determinants.

The GI and BR protocols are well documented in the literature (Carlsöö, 1966; Brenière et al., 1981; Do et al., 1982, 1990; Brenière & Do, 1991; Pai & Patton, 1997; Aftab et al., 2012). In GI, we asked subjects to initiate gait keeping step length constant (Brenière & Do, 1991; MacDougall et al., 2005) in different trials performed at slow, spontaneous or fast velocity. Thus, according to the formula  $V = L \times C$ , subjects modified cadence (an index of which was the duration of the single stance). In BR, subjects were initially inclined by means of a cable, the release of which provoked an automatic step at a constant initial progression velocity of

the CoM. Here, we asked the subjects to recover balance by executing short, normal and long steps.

### *Modulation of triceps activity determines the global kinematics of gait*

In human gait, muscle activity in the swing leg is present only at foot-off and foot-contact (Basmajian, 1976), i.e. the swing leg moves under the effect of gravity and body's kinetic energy in a quasi-ballistic mode (Mochon & McMahon, 1980). Consequently, the CNS sets step length and duration of single stance by controlling the CoM momentum of the body by means of modulating the muscle activity of the triceps surae of the stance leg.

In both protocols, the duration of the single stance was highly correlated with the duration of triceps activity. More precisely, the duration of the triceps activity was shortened in order to reduce the duration of the single stance, and lengthened in order to increase it. Hence, by controlling the duration of triceps activity, the CNS determines the duration of the single stance and thus cadence. On the other hand, by modulating the amplitude of the CoM momentum during the single stance, the CNS can control the distance travelled by the falling body before the swing leg establishes contact with the floor. In other terms, a particular amount of CoM momentum occurring during a definite duration of single stance should result in a specific step length. The BR protocol provided insight into how duration of single stance and CoM momentum are modulated by triceps activity to set step length. Firstly, the duration of the single stance was highly correlated with step length. Secondly, since the CoM velocity at foot-off was equal across step-length conditions, CoM momentum was solely modulated by means of varying the level activity of triceps surae EMG. In short steps, triceps surae was almost silent during single stance. In this special condition, the inverted pendulum is driven solely by gravity. CoM accelerates quickly downwards and the swing leg establishes premature contact with the floor. In normal-length steps, triceps surae activity builds up and cushions the fall of CoM. This reduces the downward acceleration of CoM and at the same time allows it to travel further away from the stance foot before foot-contact. In long steps, triceps activity is initially low in early and in mid-stance. This allows the CoM to accelerate quickly forwards and downwards. In mid- to late-stance, the triceps surae activity increases and reduces the CoM downward acceleration. Consequently, the foot contact is delayed, while the instantaneous Ver velocity of CoM at foot-contact is unchanged between normal and long steps.

### *Disequilibrium torque and propulsive force are modulated via the CoM-CoP gap*

From a mechanical point of view, walking velocity is the result of applying propulsive force against the ground via the stance foot. In Honeine et al. (2013), we showed that propulsive force is the result of the disequilibrium torque. The torque is the product of the CoM-CoP gap and the vertical force acting on the CoM. The present study shows that by controlling the gap, triceps activity modulates the disequilibrium torque and so the propulsive force and walking velocity. Controlling the duration of the single stance and CoM momentum allows the CNS to determine the position of the CoM relative to CoP, i.e., the CoM-CoP gap. In the fast-walking condition of GI, the high momentum of CoM at foot-off allows it to travel further away from CoP than in the normal condition. The opposite occurs during slow walking. In BR, where CoM position and step length are highly linked together due to biomechanical constraints, CoM-CoP gap increases progressively as step length increases. Controlling CoM-CoP gap modulates the disequilibrium torque, thus propulsive force and walking velocity. Indeed, the CoM-CoP gap was highly correlated with walking velocity at foot-contact in both protocols. Interestingly, the slopes of the linear equations of walking velocity plotted as a function of the gap were very similar in the two different protocols (2.97 for GI and 2.95 for BR). This strongly suggests that the amount of increase in the CoM-CoP gap when velocity increases is the same regardless of which kinematic variable is being modulated (i.e., cadence or step length). We would like to underline the fact that two different populations performed the GI and BR experiments. Therefore, the difference in bodyweight that is a factor in determining the disequilibrium torque should have resulted in the difference in the intercept of the two linear equations obtained in the two experiments. We, thus, believe that if the same population performed both experiments, a certain CoM-CoP gap would have always resulted in the same walking velocity in both protocols. However, further experimentation is required to ascertain this fact.

### *Braking action of CoM and push-off.*

In gait, triceps EMG increases alongside the AP and Ver GRF in late stance. The rise in GRF is termed 'push-off'. Many authors have associated the increase of the triceps surae EMG in late stance with the push-off (Winter, 1983; Neptune et al, 2001; Anderson & Pandy, 2001). However, in Honeine et al. (2013), we found that triceps EMG does not increase when subjects initiate gait with and without a load at the same walking velocity, in spite of the

increase in propulsive force due to the increase in bodyweight. The vertical braking action of CoM did not change, either, when the load was added. The braking action has been previously reported by Chong et al. (2009) and Chastan et al. (2010) and is quantified by the amount by which Ver velocity of CoM is reduced in late stance. However, when subject walked faster both the braking action and triceps activity increased. In this study, we provide proof that the reason of the surge in triceps activity in faster walking is due to the increased requirement of the CoM vertical braking action.

In the GI experiment, CoM vertical momentum increased as a function of walking speed (Fig 1C, left panel). In GI, the increase is caused by a rise in TA activity prior to single stance (Nardone & Schieppati, 1988; Lepers & Brenière, 1995). Therefore, around middle stance the downward velocity of CoM is highest in fast walking. Thus, a higher vertical momentum requires the application of a stronger upward force in late stance to brake the CoM fall. The increase in upward force is done by rising triceps activity. Consequently, the CoM Ver velocity at foot-contact is unchanged regardless of the walking speed condition. Verdini et al. (2006) suggested that reducing vertical acceleration of the falling CoM allows for a softer touchdown of the swing leg with the floor for a smoother step-to step transition in the subsequent double stance phase. Kuo (2007) explained that the step-to-step transition is dependent upon the vertical force applied by the stance leg and by the swing leg as it touches the ground. He inferred that the forces provided by both legs help redirect the CoM upward during double stance. We suggest that, by braking the fall of CoM, the CNS should also modulate the force produced by the swing leg as it impacts the floor and thus control the redirection of the CoM during step-to-step transition.

#### *General considerations and limitations*

Donelan & Kram (1997), Cavagna et al. (2000) and Sylos-Labini et al. (2013) all had previously shown that walking in a reduced gravity environment changes significantly gait kinematics. In other terms, kinematics of walking rely strongly on the vertical force acting on CoM. In this study, under constant gravitation attraction, the means by which the vertical force is modulated to produce different gait kinematics is by triceps surae activation, which counteracts the downward pull of gravity. During single stance, humans are always in a state of disequilibrium and it is the torque caused by the disequilibrium that generates propulsion. By opposing the pull of gravity, CNS is capable of modulating the disequilibrium torque and

of producing all combinations of step length and cadence merely through control of triceps activity of the stance leg. Recent findings have given evidence that motor cortex and cortico-spinal tract contribute directly to the muscle activity observed in steady-state treadmill walking (Petersen et al., 2012). The CNS would therefore be able to select and implement each combination of cadence and step length to produce a given walking velocity. In this study, we used the gait initiation and balance recovery protocols to prove our hypothesis. In a sense, in our case, deliberate intervention of the higher brain centres was unavoidable and warranted, thereby engaging cortico-spinal control. On the other hand, a reflex contribution to this process should be acknowledged because sensory feedback plays a critical role exactly in the adaptation of activation of extensor motoneurons during locomotion (Sinkjaer et al., 2000; Tokuno et al., 2007; van Doornik et al., 2011). In addition to the inflow from primary and secondary muscle spindle fibres (Mazzaro et al., 2005), a relevant feedback would come from the load receptors present in plantar-flexors, which are known to exert a major action in human locomotion (Dietz & Duysens, 2000). Indeed, the fall of the CoM during single stance partly unloads the Golgi tendon organs in the Achilles tendon. Thus, the feedback signal via the group Ib afferent fibres would decrease alpha-motoneurone inhibition and increase the firing rate of the triceps motor units during the braking action, together with the enhanced facilitatory spindle input, therefore ultimately opposing the fall of the CoM.

In spite of the obvious differences between GI or BR and spontaneous steady-state walking, we would note that in GI and BR *and* steady state gait the body acts as an inverted pendulum for a considerable range of walking velocities (Cavagna & Franzetti, 1986). We believe that, within that range, the conclusions obtained in our experiments from the concurrent analysis of the spatio-temporal EMG and pendulum mechanical constraints can be largely extrapolated to steady-state walking. It is also to be noted here that when executing very long steps, the body stops acting as an inverted pendulum (Cavagna & Franzetti, 1986). Indeed, the speed at which our subjects performed the long steps in BR was similar to that at the transition from walking to running (Cavagna & Kaneko, 1977; Thorstensson & Roberthson, 1987; Diedrich & Warren, 1995; Hreljac et al., 2005). Thus, the results discussed in this study are valid within a wide range of walking velocities, yet far from those occurring close to transition to running.

One limitation of this study is that we are not in the position of comparing the force contribution of each of the individual muscles. The three muscles plantar-flex the ankle, but they can have antagonistic behaviour across the knee (van Ingen Schenau et al., 1987). During

responses to postural perturbation by displacement of the support base, the three heads may behave differently (Nardone et al., 1990). However, even though authors do not agree on the exact role of each individual muscle, everybody agrees that all three heads of triceps contribute in body-support (Neptune et al., 2001; Anderson & Pandy, 2001; Francis et al., 2013). In our hands, no significant differences between the onset and end of activity of the three heads of the triceps surae were found. They seem to obey the same motor command that has been traced to their motor pools in the spinal cord (Cappellini et al., 2010). Notably, the central pattern generator (CPG) for locomotion commonly recruits the three components of the triceps (Dimitrijevic et al., 1998; Lacquaniti et al., 2012).

#### IV.6 Conclusion

Humans have evolved to take advantage of the torque driven by gravity to propel themselves. When we learn to walk, the spinal circuits ultimately driving the triceps motoneurons are gradually conquered by the developing descending tracts (Musienko et al., 2011), which learn ‘to talk to the spinal cord in a language that it can understand, determined by its pre-existing circuits’ (Matthews, 1995). Such process makes bipedal walking possible by making representation of gravity part of our brain activity (Papaxanthis et al., 2003; Ivanenko et al., 2006) and by exploiting it to produce propulsive force (Cavagna et al., 1976; Honeine et al., 2013). In this work, we showed that modulation of triceps surae in amplitude and duration allows the CNS to perform all the possible combinations of step length and cadence. In the process, the disequilibrium is set and consequently walking velocity.

We believe that such capacity worsens when the interaction between brain, central pattern generator, interneurons and motoneurons and sensory feedback fails. Gait is abnormal in peripheral neuropathy (e.g. Casasnovas et al., 2008; Wrobel & Najafi, 2010; Nardone et al., 2013). CNS problems also produce dramatic effects, notably in stroke patients showing inconsistent step-length asymmetries (Roerdink & Beek, 2011), in Cerebral Palsy patients, where changes in muscle fibre properties together with altered reflex excitability (Berger et al., 1982) modify the stance phase of gait, and in Parkinsonian patients, who are unable to produce long steps, relying instead on short and frequent steps (De Nunzio et al., 2010; Nanhoe-Mahabier et al., 2011). Interestingly, replacement of one lower limb by a prosthesis does not affect the speed of progression, regardless of whether the stance limb was prosthetic

or not. Most likely, other muscles take over the control of the duration of the stance phase of gait and of the body mass CoM momentum (Michel & Do, 2002; Michel & Chong, 2004; Wentink et al., 2013). Whether these subjects can still manage to select different stepping frequencies or step lengths has not received much attention to date (Roerdink et al., 2012).



## V. General discussion

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Bipedal stance requires strict control over the ankle joint in order to assure proper balance control and propulsion. In humans, this control is done through activation of ankle extensors, mainly the triceps surae. A lot of studies investigated the role of TS, notably soleus, in bipedal quiet stance at various foot positioning (Schieppati et al. 1994; Van Doornik et al., 2011; Sozzi et al., 2013). All studies agree that TS stiffens the ankle joint and supports the ankle by preventing tibial rotation.

Human gait is a succession of single and double stances. During single stance, gravity pulls the CoM so that it rotates around the ankle/forefoot system. This causes the body to be in disequilibrium throughout single stance. Thus, bipedal walking necessitates balance control to prevent fall during single stance. Balance control can be achieved by activation of the anti-gravitational muscles such as TS. Indeed, TS activity dominates single stance.

Kinematics studies of gait showed that, throughout almost 75% of single stance, the ankle joint is in dorsiflexion. Thus, during this period, TS works eccentrically. It is widely agreed upon that when in eccentric contraction, TS functions mainly to support the body (Sutherland et al., 1980). In late stance, the foot is lifted, thus the ankle is in plantarflexion and TS starts working concentrically. Kinetics analyses show that during plantarflexion, the GRF vector increases in both the vertical and anteroposterior direction. The increase in GRF in late stance has been termed push-off in the literature. Late stance is also characterized with the highest level of TS EMG activity during single stance. Hence, in late stance, increase in CoM AP acceleration actually corresponds with an increase in TS EMG contraction.

The level of the TS EMG activity also covaries with walking velocity (Winter, 1983; Den Otter et al., 2004). This led many scientists to consider that TS activity causes push-off in single stance. Others disagreed, sticking to the idea that TS primarily role is to support the body during single stance. This led to a long lasting debate about the real physiological function of TS in gait. The reason behind the debate is that no one was really able to empirically prove or disprove causality between TS activity in late stance.

Current technology does not permit direct in vivo non-invasive measurement of muscle force during gait. Inverse dynamics alone can be used to give a good estimate of joint moments, at least at the ankle level. However, since many muscle cross the same joint muscle force cannot be extrapolated from joint torques (Neptune et al., 2001). Musculoskeletal models have been used to attempt to solve the problem by utilising different minimization algorithms (Lloyd & Besier, 2003; Dubowsky et al., 2008). Nonetheless, no model has been so far validated, thus the obtained results are still very subjective and depend on which and how algorithms are used (Lund et al., 2012). In addition, the output force of the model is usually correlated with the trunk segment in order to investigate if TS propels the body. However, the activity of any skeletal muscle has been proven to accelerate all joints in the body (Zajac & Gordon, 1989; Nott et al., 2010). So covariation between body acceleration and muscle activity cannot be considered to be fit for determining causality. Others tried to vary propulsion force by pulling or pushing the subject with a rope on a treadmill (Stephens & Yang, 1999; Gottschall & Kram, 2003; McGowan et al., 2008; Lewek, 2011). The tension of the rope adds another external force to the body, which alters all body joints. These experiments hint on how the body reacts to such an external force, but are reliable enough to reveal the functionality of TS in late stance.

On the other hand, the main experiments performed to demonstrate that TS is only responsible for equilibrium control, all altered significantly body kinematics (Simon et al., 1978; Murray et al., 1978; Sutherland et al., 1980). This suggests that it is quite possible that another motor scheme had been used during the experiments, which could falsify the results. In this dissertation, we wanted to find ways to test whether TS activity when walking at different velocities.

### *Study One*

To test if TS activity is responsible for push-off in late stance we opted for the well-established gait initiation protocol (Carlsöö, 1966; Brenière et al., 1981; Crenna & Frigo, 1991). A very large platform was used to measure ground reaction force throughout the entire single stance. We asked subjects to perform gait initiation at two different speeds, normal and fast. Then subjects had to perform gait initiation at the same speeds but while carrying a load evenly distributed around the waist, in the region where CoM is usually situated. Arrelano et al. (2009) showed that adding a load up to 30% on the waist does not affect kinematic

stability in the sagittal plane. For each walking velocity, load increased propulsion force but did not alter TS EMG level. We concluded that it is not TS activity that accelerates the body in late stance. To better understand the cause of the increase in AP forces, we considered the body as an inverted pendulum passing through CoM and CoP and rotating around the latter. In other terms, the body was considered to be the GRF vector rotating around its point of application that is the CoP<sup>16</sup>. The reason is that the CoM is the point where bodyweight acts on the body while CoP is the point where GRF act on the body. The CoP was also considered to be a good approximation of the centre of rotation beneath the foot.

We found that the torque produced by the vertical force acting on CoM was responsible for generating the increase in propulsion force. We termed it disequilibrium torque. During quiet standing, the AP position of CoM coincides with that of the CoP (Winter et al., 1995). When initiating gait, tibialis anterior is activated in order to shift CoP backwards. This creates a gap between CoM and CoP, which allows for the disequilibrium torque to build up, i.e. the CoM starts rotating around the CoP. The AP posterior trajectory of CoM is a parabola since CoM is accelerating forward. Meanwhile CoP starts increasing slightly towards the forefoot. When the ankle is plantarflexed in late stance, CoP AP position is shifted quickly forward and plateaus under the forefoot. This increases sharply CoM and CoP gap. Meanwhile, we found that around middle stance TS action brakes the fall of the CoM. The braking of the CoM results in an upward acceleration of CoM even though its velocity remains negative, i.e. the CoM is still falling. Both the braking action of CoM and the increase in gap boosts the disequilibrium torque. According to Cavagna et al. (1976), the gravity torque acting on an inverse pendulum is capable of transforming vertical kinetic energy into forward kinetic energy. Interestingly, if one changes the point of view of the problem and considers CoM as the centre of rotation, then dividing the torque by the instantaneous vertical position of CoM<sup>17</sup> will result in a very accurate estimation of the AP propulsive force measured by the platform (see figure III.4).

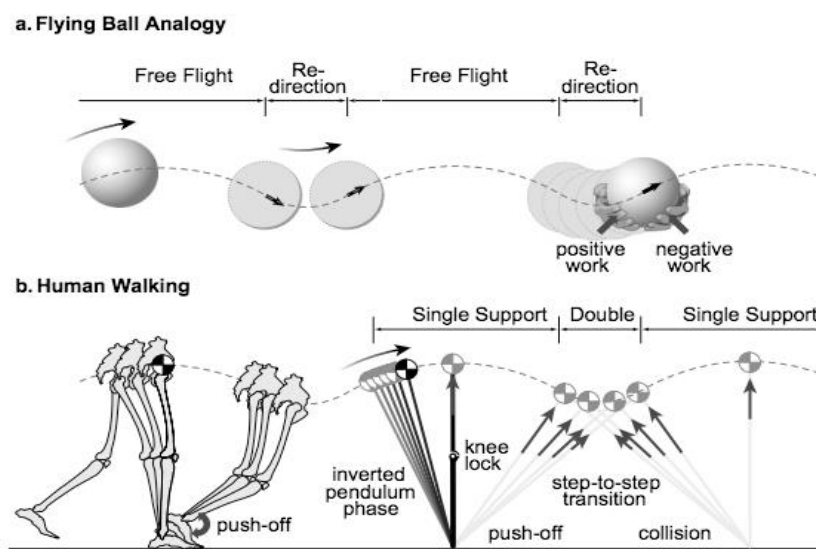
So far, I have just discussed the role of TS during single stance. However, it had been also suggested, that the push-off provided by the TS, during the double stance is required for lifting and pushing forward the centre of mass. Thus, at this point, the function of TS in

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<sup>16</sup> In 2D, the mediolateral dimension was not taken in consideration

<sup>17</sup> Initial vertical CoM position was calculated using the de Leva anthropometric tables (de Leva, 1996). E.g. a 1.75 m tall subject would have CoM positioned at 1.02 m.

double stance needs to be discussed. It is true that at foot-contact, the CoM is at its lowest position. During double stance, the CoM vertical position rises in order to allow for the proper execution of the subsequent step. However, Kuo (2007) in his review about the inverted pendulum model discussed that during double stance the CoM requires theoretically neither propulsion nor lifting. He explains that it is true that push-off accelerates marginally the CoM. However, at foot-contact the gain is negated by the antero-posterior braking caused by the action of the swinging leg. This can also be seen in our first study. When the swing leg contacts the floor, the CoP is shifted abruptly forward. This causes the gap to become negative, which causes CoM AP velocity to decrease slightly. Nonetheless, Kuo (2007) clarifies that, during the step-to-step transition<sup>18</sup>, the negative work caused by the landing of the swing leg is of equal importance as positive work performed by the stance leg. Thus, the combined vertical force of both legs helps redirect the CoM upward. Meanwhile, the momentum of the CoM in the sagittal plane allows it to continue travelling forward during



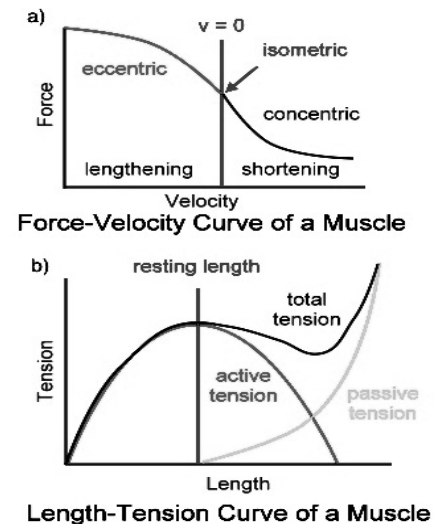
**Figure V.1 The flying ball analogy to explain step-to step transition in human walking (Kuo, 2007)**

a) To explain step-step transition, Kuo (2007) uses the analogy of ball that is redirected during flight. The ball will then be considered to be the CoM during gait in the sagittal plane. To redirect the ball that is falling parabolically, the vertical velocity of the CoM must be reversed into an upward velocity. The redirection of the CoM can be done by placing two hands under the ball when it is falling down. Each hand applies a perpendicular force to the ball's surface. During the redirection, one hand performs positive work on the ball, and the other negative work. This propels and lifts the ball.

b) In single stance phase, the COM acquires vertical momentum due to the gravity torque, similar to when the ball's flight phase. During double support, the COM velocity must be redirected upwards. This should be done by performing active and passive work in the vertical direction (as seen previously). Positive work is performed by the trailing leg and negative work by the leading leg. The net work of both legs during double support accomplishes the redirection of CoM.

<sup>18</sup> Step-to-step transition is the term that Kuo uses to describe double stance

Obviously, Kuo's interpretation is theoretical and is aimed at explaining step-to-step transition in a dynamic inverted pendulum. In vivo, double stance is under control by the CNS via muscle control of the trailing and leading leg. According to our results and the existing literature, we conclude the following. At the end of the stance phase, TS brakes the fall of the centre of mass, so that the swing leg does not hit the ground very strongly. The braking action of CoM should allow the CNS to control the amount of negative work performed by the swinging leg. During double stance, the redirection of the CoM is 'guided' by muscle activity occurring in both legs (Ivanenko et al., 2004a; Lacquaniti et al. (2012), e.g. quadriceps activity in the leading leg and of the ilio-psoas activity in the trailing leg. Furthermore, in all our experiments, TS activity persisted in double stance. It lasted for around 100 ms in double stance, when subjects initiated gait with a spontaneous walking speed. TS activity ended around the instant of foot-contact when subjects walked slowly. However, the end of the TS activity occurred always at around 100 before the foot-off of the trailing leg. This suggests that TS activity is participating alongside psoas activity in lifting the stance leg. Plantarflexing the foot before lifting the leg into swing raises the foot. Thus, when the leg is lifted the foot has enough clearance and does not strike the ground when tibialis anterior is activated to dorsiflex the foot during swing.



**Figure V.2 Mechanical properties of muscles (Hall, 2003).**

- a) The force-velocity curve shows that more force is produced during eccentric contraction.
- b) The length-tension curve shows that when in eccentric contraction, passive force is produced which increase the force generated by the muscle.

Conclusively, in the first study, we demonstrated that the main function of TS is to support the body during single stance. More precisely, TS brakes the fall of CoM. Propulsion force is generated instead by disequilibrium caused by the gravity-driven torque. It might be interesting to note that during eccentric contraction less motor units need to be active with respect to the number active during concentric contraction, because the muscle itself exerts more force during eccentric than concentric contraction other things being equal. The reason is that lengthening of the muscle increases the passive force that it generates. Therefore, when the TS passes from its eccentric to its concentric contraction-phase, the CNS must increase the

MN recruitment to produce the same level of force, hence the increase in the triceps EMG in the late stance.

### *Study Two*

As it often occurs in science, answering one question opens the door for another one. If propulsion is driven by gravity and the latter is constant, then how do humans manage to walk with different speeds? It seemed obvious that the downward acceleration of the CoM that is caused by gravity had to be modified via modulation of lower limb muscle activity. Since TS activity decelerates the fall of the CoM then it is a great candidate for modifying body kinetics in order to generate different velocities.

However, due to biomechanical constraints, walking at a certain speed requires the CNS to set global kinematics and kinetics parameters. We hypothesized that TS plays an important role in determining both parameters. More precisely, we postulated that, since TS supports the body during its fall in single stance, then temporal modulation of TS should determine how long the body would stay in the air before touch down of the swing leg. In other terms it should determine the duration of single stance or cadence of gait. Furthermore, during single stance the swing leg is quasi-ballistic (Mochon & McMahon, 1980) and the momentum of CoM is increasing due to the torque. Thus, by controlling single stance duration, the CNS should also determine where the swing foot would land. In other terms, CNS can set step-length. From a kinetic point of view, TS has to control propulsion, i.e. the disequilibrium torque, and has to maintain balance during walking. Due to body geometry, the CoM position in the sagittal plane covaries with the length of the step. Thus, the same mechanism involved in determining step-length could be put in action to set the CoM at foot contact. More precisely, we hypothesised that by controlling the duration of TS activity, the CNS sets the CoM-CoP gap and consequently modulate the disequilibrium torque.

By controlling the force generated by TS, the CNS can vary the braking action in order to decelerate the fall of centre of mass especially during late stance in order to avoid a fall. Vertical braking of CoM also prevents it from reaching a very low position, so that redirecting it upwards in the subsequent double stance would not be very metabolically costly.

In the second study, we found that the duration of TS EMG activity is highly correlated with the duration of single stance. For the same CoM AP velocity at foot-off, the duration of single stance is in turn highly correlated with step length. The disequilibrium torque was controlled mainly via the CoM-CoP gap. The latter decreases when walking slowly and increases in fast walking, regardless of whether the kinematics strategy is to vary cadence or step length. The amplitude of TS EMG revealed to be sensitive to the vertical momentum of the centre of mass. It increased to counteract a fast CoM and decreased when the CoM momentum was low. Since TS afferent fibres are very sensible to changes in load (Sinkjaer et al., 2000; Duysens et al., 2000; Grey et al., 2007), we suggested that when CoM decelerates downward, it unloads the Golgi tendon organs: this in turn reduces  $\alpha$ -motoneurons inhibition and thereby increases the firing rate of TS motor units.

Therefore, at this point, it is possible to conclude that propulsion is generated by the disequilibrium torque that is driven by gravity. To vary walking velocity, the CNS modulates the TS surae activity. By counteracting the effects of gravity, TS is able to set global kinematics and kinetic.

### *General Discussion*

After having discussed the results of the two studies, I would like to take time and discuss about some general issues concerning the experiments that were performed in the course of this dissertation. The main purpose of the study was to uncover the functional role of the TS in gait. However, to test our hypotheses, we asked healthy volunteers to perform either gait initiation or recovery from fall. Thus, in order to interpret the results in terms of gait one has to extrapolate data and assume that the results are also plausible during steady-state walking.

The kinematics and kinetics of the single stance phase of gait initiation has many similarities to those measured during steady-state. Brenière & Do (1986) had questioned about when is steady-state achieved starting from an upright posture. However, their results show that at foot-contact of the first step, the CoM AP velocity does not differ from that obtained at the end of the second step. They also reported that the duration of single stance obtained during gait initiation does not differ from that reported during steady-state walking. They concluded that: “the aim of the gait initiation process is to place the subject in steady-state gait within the first step, in an invariant time which is dependent only on the body segment parameters of

each subject.” Jian et al. (1993) later confirmed that about 90% of steady-state velocity was achieved during the first step and 100% by the second step. Other ‘small’ differences in kinetics and kinematics have been reported in Park et al. (2008). Furthermore, in gait initiation, during both balance recovery and steady-state walking, the human body behaves as an inverted pendulum, thus it is fair to assume that the same mechanisms should be used by the CNS to control gait kinematics and kinetics. However, the results obtained here apply only to walking within a certain range of velocity on a flat surface. In other forms of locomotion such as running, or going uphill or upstairs the body does not behave as an inverted pendulum. In these conditions, the CoM vertical position rises during single stance. Furthermore, in these conditions, TS contraction is concentric opposedly to the eccentric contraction seen in normal gait. Thus, triceps surae activity in these cases should be to accelerate the CoM upward in order to rise it. Other muscles of the stance leg should also be involved in rising the CoM such as the quadriceps muscle group.

Nonetheless, the gait initiation protocol has its advantages. First, in gait initiation, the initial conditions are all known. This allowed us to take advantage of the large platform that we have in our laboratory to measure GRFs and CoP displacement throughout the whole interval in which the first step was executed. This also allowed us to easily compute the CoM velocity and displacement in the AP and Ver by means of integration and double integration, respectively. It is true that to do such a calculation one has to agree that the body during gait initiation is considered to be just a point mass. In other terms, inertial components of the different body segments are lost. However, it is also true that CoM kinematics computed via optoelectronic data, even if widely accepted, is approximated since it relies mostly on anthropometric data. Anyhow, Jian et al. (1993) studied gait initiation and reported CoM velocities calculated using optoelectronic data. Their mean velocities at foot-contact and the curve trajectories were very similar to those we obtained in our experiment. Second, gait initiation is very reproducible and has been well documented (Carlsöö, 1966; Brenière & Do, 1981; Crenna & Frigo, 1991; Brenière & Do, 1991). Furthermore, the laboratory of sport science has substantial knowledge about the protocol, which allowed me to gain the insight needed to be able to design the experiments and know what to expect from them. The repeatability of the experiments, that can be seen in the low standard deviation obtained in CoM velocity at foot-off and foot-contact, made also the results less noisy and thus interpretation much easier and clearer. Finally, I would like also to discuss one advantage of gait initiation over steady-state gait during the first study. In this study, we knew from data



collected beforehand that the amplitude of the GRF vector in late stance is ampler than that measured during steady-state gait. So push-off in gait initiation, if anything should be stronger than in steady-state gait. In addition, the TS activity during single stance of the first step was always ampler than that measured in the following steps. So, if TS was not responsible for push-off then in gait-initiation then it is highly improbable that it does so in steady-state gait.

In the second experiment, we needed to perform very controlled experiments in order to properly test our hypothesis. The gait initiation and balance recovery protocols allowed us to put gait under the microscope. We were then able to isolate the desired kinematics variables and dissect out the role of TS in determining walking velocities under different conditions. By asking subjects to maintain the same step length and alter walking speed in gait initiation, we were able to study triceps' role in determining cadence. The balance recovery paradigm allowed us to obtain the same CoM AP velocity at foot-off when subjects performed different steps. Only by having an identical CoM momentum at the start of single stance, we were able to understand how can TS play a role in determining step-length.

In balance recovery, the step execution is automatic. Initially when subjects are released from the cable they react by increasing GRF in both the AP and Ver direction. During the reaction phase, the TS is activated and shows a strong EMG burst. We believe that TS activity is due to a supraspinal loop going through the downward tracks. For more information about the reaction phases, the readers may refer to Do et al. (1982), Do et al. (1990), Do & Chong (2008). It is also obvious that balance recovery is much more constraining than gait initiation. The duration of step execution for example is almost half of what is measured for the same step-length in gait initiation. Furthermore, the CoM is positioned at a lower position than it would have if subjects started walking from an upright position. Initially, the CoM-CoP gap is also considerable so that the disequilibrium torque is strong throughout the reaction and the step-execution phase. However, during single stance, TS is still activated to counteract gravity. Conclusively, we believe that, in our study, the balance-recovery protocol amplified the effects that would have been seen during normal gait.

Finally, in the two studies, we only considered the GRF and CoM displacement, velocity and acceleration in the sagittal plane. In other terms, the medio-lateral component was not taken in consideration. It is true however, that the ML displacement of the CoM and the ML component of the GRF are substantial during gait. It should also be true that the

disequilibrium torque has a medio-lateral component. Since the CoM during gait is shifted mediolaterally, it could move away from the CoP. The product of vertical force and the ML distance between CoM-CoP will result in the ML disequilibrium torque. However, in this study we were concerned about the role of TS, that plantarflexes the ankle. Plantarflexion occurs in the sagittal plane. Furthermore, in Sozzi et al. (2013), we found that when subjects stand in tandem stance, balance control in the frontal plane was done via ankle invertor (tibialis anterior) and evertor (peroneus longus). Tandem stance is a very challenging posture, where ML sway increases considerably. Soleus did not participate in medio-lateral balance control but helped in keeping the body upright. GM and GL activity were not measured. If SOL did not participate in ML balance in stance, then it shouldn't do so in gait. When walking, ML balance control, at the ankle level, could be performed by evertors such as peroneus longus. More research is needed to confirm this fact. However, by supporting the body, the TS can modify the downward acceleration of CoM, which results in variation of the vertical force acting on CoM. In turn, this should affect the ML disequilibrium pattern. Thus, future research would be performed in order to model a more complex three-dimensional multi-joint (ankle - knee - hip) disequilibrium torque. Muscles activity across the three lower limb joints of the stance leg should be also measured in order to gather a global understanding about how the CNS controls lower limb muscles to counteract gravity and modulate walking.

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To our current knowledge, the attraction force provoked by gravity is omnipresent. On planet Earth and more particularly on land, gravity pulls all animals towards the ground<sup>19</sup>. As a general rule, to move about, animals need to produce a propulsive force. This force has to overcome the drag that is created by the contact between the body and the ground through friction. As a result, most land animals<sup>20</sup> developed limbs in order to raise the body from the ground. By reducing the contact surface with the ground, the drag caused by gravity decreases considerably. This is efficient since it reduces the metabolic cost of locomotion. However, raising the body from the ground adds up equilibrium as another requirement of gait. Equilibrium requires animals to maintain posture and orientation and control balance to prevent the body from falling. One solution is to have many limbs in order to maintain at least

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<sup>19</sup> This forces the CNS to take into account the pull of gravity while performing any specific movement (Massion et al., 1998; Pedrocchi et al., 2002).

<sup>20</sup> Except for serpent, legless lizards, molluscs such as slugs and snails, leeches and earthworms.

two contacts, such as in quadrupeds, or more contacts such as in insects and arthropods. However, some animals, other than humans, have evolved to be bipeds. These are birds<sup>21</sup>, some primates (e.g. gibbons and lemurs), some macropods (e.g. wallabies and kangaroos), rodents as well (e.g. spring hares, gerbils and kangaroo rats) and occasionally apes (e.g. chimpanzees, gorillas, bonobos, gibbons, japanese macacques, etc.).

Alexander (2004) showed that human bipedalism is much more evolved differently than other bipedal animals. What this dissertation shows is that the human bipedal walking<sup>22</sup> have evolved that takes advantage of the disequilibrium caused by gravity. By controlling the fall of their CoM, humans create friction under the feet, which in turn pushes them forward. They then use the momentum of CoM to redirect it upward and forward in order to execute a subsequent step.

It is important to keep in mind that for human bipedalism to work the way it does, it requires the CNS to control very tightly many aspects of gait. The circuitry involved in gait is thus very redundant and complex. One proof of the complexity of the neural network involved in human bipedal walking is the fact that 'locomotor-like' activity is rarely expressed without descending input from the brain (Yang et al., 1998) and sensory input present (Yang & Gorassini, 2006). These sensory inputs are mainly proprioceptive, but could also rely on afferent from the vestibular system in order to insure balance.

One good way for understanding how the neural network involved in gait is put together is to study human infants. Interestingly, from birth human infants exhibit the capability of stepping (Andre-Thomas & Autgarden, 1966). However, unlike other animals, humans require much more time to learn walking. One of the reasons could be that at birth the human brain is not fully formed (Richardson, 1982). Following birth, the motor cortex (Amunts et al., 1995) and cerebellum (Kinney et al., 1988) experience considerable change as afferent and efferent networks are established. The cortico-spinal tract (Brody et al., 1987), the middle cerebellar peduncle (Yakovlev & Lecours, 1967) and the reticulospinal and vestibulospinal pathways (Richardson, 1982) are also modified for two years following birth.

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<sup>21</sup> When on land.

<sup>22</sup> Probably unlike other bipedal animals

The supraspinal changes obviously affect the descending tracts that interact with spinal cord motoneurons. This should allow the brain to properly command the CPG (Musienko et al., 2011). It is possible that while the brain is forming it learns to incorporate a representation of gravity as part of its neural activity. This explains how by trial and errors babies learn to counteract activity while stepping in order to go forward. During this process, dorsiflexors and plantarflexors are alternately activated and start resembling adult muscle activity. This is followed by gradual changes in coordination that work at different joint (Cheron et al., 2001a & 2001b; Ivanenko et al., 2004b, 2005; Lacquaniti et al., 2013) with different aspects of coordination maturing at different rates.

It is possible that in that way, humans learn that by controlling the duration of plantarflexor activity they can modulate both step length and cadence. To control balance plantarflexors vary the amplitude of the muscle activity in order to brake the fall of the CoM. It is also important to note that all three heads of the TS are also controlled simultaneously. This suggests that in gait, SOL, GM and GL probably receive the same motor command and are part of a single motor scheme. It is also probable that extensors working across the knee and hip joint obey to the same motor command as suggested by Ivanenko et al. (2004a) and Lacquaniti et al. (2012b).

Finally, in this work the protocols used were gait initiation and balance recovery. It is hard to tell whether in gait initiation efferent signals arrive directly from CPG motoneurons or from a supraspinal command. However, Duysens & Van de Crommert (1998) proposes that to initiate gait supraspinal commands follow the downward tracts to *kick start* the CPG. He suggested that ‘in gait initiation, afferents deliver movement-related information to spinal and supraspinal levels. Some of this feedback acts directly on the CPG to aid the phase transitions during the step cycle thus providing the possible induction of variations to meet the environmental demands’. However, the afferent feedback connects directly to spinal motoneurons through various reflex pathways. CPG controls largely these patterns (Duysens & Van de Crommert, 1998). Thus, it is possible that the CNS operates in such a way as to activate the TS at appropriate time during the step cycle (Duysens et al., 1998).

## VI. Perspectives

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This dissertation could not have been achieved without the science that was acquired through the years at the Sports-Science faculty of the University of Paris-Sud. In the four years in which this dissertation was conducted, I had the chance to acquire the knowledge that was required for putting in place the experimentation that permitted proving the work postulates.

To our current day, much needs to be done to improve rehabilitation methods and to develop better cures to help people with gait disorders. This necessitates understanding the fundamentals of how the CNS operates to command lower limb muscles and the functional role of each of these muscles during normal gait. The knowledge obtained from healthy subjects provides the clinicians with a reference that they can base the rehabilitation of the patients upon.

This study points out that under the surveillance of supraspinal commands the sensory-dependent CPG networks alternate ankle extensor/flexor activity at a certain frequency to set gait kinematics by means of determining step-length and cadence and kinetics by varying the disequilibrium torque by tuning CoM-CoP gap. Modulation of the amplitude of ankle extensors activity brakes the fall of the CoM. This reduces the impact of the swing foot with the floor at foot-contact and prevents long-term injury.

One important result obtained in this study is that, during gait, modulation of global kinetics and kinematics is mainly done via vertical control of the CoM fall in the sagittal plane. In hemiplegic or stroke patients, control over the CoM fall when stance leg muscles are dysfunctional is very difficult (Yavuzer et al., 2006). To walk these patients often rely on different motor schemes, which allow them to produce repeatable gait kinematics (Yavuzer et al., 2008). One solution could be to develop an orthosis that is capable of maintaining the affected leg extended when in the stance phase while developing upward vertical force (Zancan et al., 2004). Other, more complicated automated orthosis might be conceived, whereby the subjects themselves could control the force generated by the orthosis. By modulating the vertical force, subjects could thus decelerate the fall of the CoM and hopefully redevelop normal gait patterns. EMG signals from the upper limbs (Cheron et al., 2001a) or

the ipsilateral limb (Ivanenko et al., 2006) could be acquired so that an integrated microprocessor could compute the duration of the force to determine global kinematics and kinetics and to modulate the amplitude of the force to allow for proper landing of the swing foot.

It is also interesting to note that it has been shown that patients with progressive supranuclear palsy (Welter et al., 2007), elderly people (Chong et al., 2009) and Parkinsonians (Chastan et al., 2009) are unable to brake the CoM fall in late single-stance. The lack of active braking leads the CoM to accelerate quickly downwards (Fig. VI.2 - part E). Reducing step length should decrease the vertical velocity at which these patients impact the ground at foot-contact. This misleads the patients into walking with short steps. Nonetheless, it has been proven that walking with short steps reduces stability and increases the risk of falling (Ness et al., 2003; Cromwell et al., 2004). Chastan et al. (2010) showed that somatosensory input was involved in modulating the braking action. Future research could investigate the implication of supraspinal control in tuning the amplitude of the TS activity in order to brake the CoM fall. The results obtained here could help understand why Parkinsonians and progressive supranuclear palsy patients reduce step length while walking. Analogous researches are being conducted in our laboratory in order to also study TS activity in patients suffering from bilateral vestibular dysfunction and cerebral palsy. Future results should hopefully help clinicians with the rehabilitation of such patients.

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## VIII. Annexes:

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The aim of the section is to provide the readers with additional information about the material and methods that were used during the three experiments conducted in study1 and 2. In both studies, biomechanical data were collected from a force platform and electromyographic data of SOL, GM, GL of the stance leg and TA of the swing leg were collected using surface electrodes. Analog data were then synchronized and digitized using a NI analog to digital converter (National Instruments, Austin, USA) at a sampling frequency of 1000hz. Data was then stored on a personal computer for offline analyses using a Matlab routine specifically made to treat the data.

### VIII.1 Force platform

Ground reaction force and moment were measured in the anteroposterior, mediolateral and vertical direction using an AMTI platform (Advanced Mechanical Technology, Waltham Street, Watertown, USA). The dimensions of the platform were: 1.80 m in length and 0.90 m in width. Table VIII.1 shows the technical specifications of the platform.

**Table VIII.1 Force platform technical specifications:** The technical specifications were provided by AMTI (x, y and z indicate ML, AP and Ver respectively).

Sensing Elements	Strain gage bride
Fz Capacity (N)	2000 (9000)
Fx, Fy Capacity (N)	1000 (4500)
Mz Capacity (Nm)	18000 (2000)
Mx Capacity (Nm)	72000 (8000)
My Capacity (Nm)	36000 (4000)
Fz Natural Frequency (Hz)	170
Fx, Fy Natural Frequency (Hz)	140
Fz sensitivity ( $\mu\text{V}/\text{V}\times\text{N}$ )	0.38 (0.08)
Fx, Fy sensitivity ( $\mu\text{V}/\text{V}\times\text{N}$ )	1.5 (0.34)
Mz Sensitivity ( $\mu\text{V}/\text{V}\times\text{Nm}$ )	0.059 (0.53)
Mx Sensitivity ( $\mu\text{V}/\text{V}\times\text{Nm}$ )	0.025 (0.22)
My Sensitivity ( $\mu\text{V}/\text{V}\times\text{Nm}$ )	0.036 (0.32)



The center of pressure coordinates were calculated using the following equations:

$$CoP_{ML} = -\frac{M_{AP}}{GRF_{Ver}}$$

$$CoP_{AP} = \frac{M_{ML}}{GRF_{Ver}}$$

If the body is considered as a point mass located at the CoM then Newton's equation of motion can be used to compute the center of mass acceleration, velocity and displacement in the AP and Ver direction in the following manner:

$$AP: GRF_{AP} = ma_{AP}$$

$$Thus, a_{AP} = \frac{GRF_{AP}}{m}$$

$$v_{AP} = \int a_{AP} dt$$

$$x_{AP} = \int v_{AP} dt$$
  

$$Ver: GRF_{Ver} = m(a_{Ver} + g)$$

$$Thus, a_{Ver} = \frac{(GRF_{Ver} - mg)}{m}$$

$$v_{Ver} = \int a_{Ver} dt$$

$$x_{Ver} = \int v_{Ver} dt$$

Where, m is the mass, a is the acceleration of CoM, g is the gravitational acceleration, v is velocity of CoM and x is the displacement of CoM. It is to be noted that the calculation is possible in gait initiation and balance recovery because the initial acceleration and velocity of CoM are known to be null.

Finally, the disequilibrium torque was calculated as follows:

$$t = (x_{CoM} - x_{CoP}) \cdot GRF_{Ver}$$

## VIII.2 Electromyography

Surface electrodes were placed according to the SENIAM (Surface electromyography for the non-invasive assessment of muscles protocol (Merletti & Hermens, 2000)). The electrodes were then amplified, and band-pass filtered onsite then packetized and transmitted to a receptor using WIFI bandwidth (Zero-wire, Aurion, It). The receptor was then connected to the NI A/D converter. The system was thoroughly tested and a constant 13 ms due to packetizing and transmitting the signal was discovered. The 13 ms delay was then corrected during the offline analysis.

The onset and end of muscle activity was detected manually in order to quantify the temporal behaviour of muscle EMG. Level activity was used to quantify amplitude modulation of the muscles and was always computed as follows:

$$IEMG = \frac{\int_{t_1}^{t_2} |e(t)| dt}{(t_2 - t_1)}$$

Where, *IEMG* is level EMG activity, the absolute value of *e(t)* is the rectified EMG signal and *t*<sub>1</sub> and *t*<sub>2</sub> are the beginning and the end of the time-window in which the level activity was calculated. Different time-windows were chosen according to need in the three experiments.