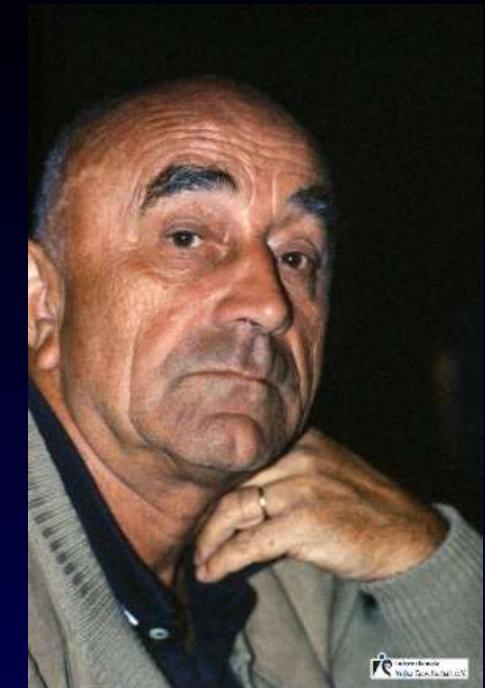


# Guided Self-rehabilitation Contracts in Spastic Paresis



Jean-Michel Gracies  
Henri Mondor University Hospitals, Créteil, France

# Disclosure

JMG received research grants and consultancy fees from Allergan, Ipsen and Merz.

# L'approche éducative du thérapeute dans l'application des Contrats d'Autorééducation Guidée (CAG)

*Kinésithér Scient* 2018,0602:31-43



## RÉSUMÉ | SUMMARY

Le thérapeute tient une place centrale dans le Contrats d'Autorééducation Guidée (CAG), jouant les rôles de prescripteur, éducateur et entraîneur. Grâce à ces trois fonctions assumées au sein des CAG, celui-ci amène le patient à améliorer progressivement la compréhension de son problème, puis, en conséquence, son degré de responsabilité quant à sa propre rééducation, lui permettant à terme d'augmenter l'intensité nécessaire de son travail rééducatif.

Cet article propose quatre fiches éducatives. Les deux premières présentent la survenue des différents phénomènes physiopathologiques de la parésie spastique. La troisième explique leurs intrications progressives et l'approche technique du CAG qui en découle, ciblant spécifiquement les muscles antagonistes. La dernière fiche illustre l'approche psychologique du CAG avec la place centrale occupée par le registre, outil fondamental de responsabilisation et de motivation.

*The therapist plays a central part in the Guided Self-rehabilitation Contract (GSC), with the threefold mission of prescriber, teacher and coach. Successful fulfillment of this triple role will enable to increase the patients levels of knowledge and understanding of their problem, therefore their level of responsibility regarding their own rehabilitation, ultimately allowing to enhance the intensity level of the physical work that they will perform.*

*This article proposes four educational sheets for use by the therapist. The first two present the pathophysiological mechanisms at work in spastic paresis. The third sheet explains the progressive entanglements between them, and the technical approach of the GSC as a result, targeting specifically antagonist muscles. The final sheet illustrates the psychological approach of the GSC, the central component being the diary, fundamental tool of empowerment and motivation.*



## MOTS CLÉS | KEYWORDS

- Kinésithérapeute prescripteur
- Motivation
- Myopathie spastique
- Parésie spastique
- Registre

- Therapist prescriber
- Motivation
- Spastic myopathy
- Spastic paresis
- Diary

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# DU Neurorééducation du Mouvement

## Université Paris-Est Créteil - mai-juillet 2022

### Neurorééducation du Mouvement - 10 journées d'enseignement

#### Jean-Michel Gracies, Professeur

Lieu des cours : Faculté de Médecine de Créteil, 8 rue du Général Sarrail, 94000 Crêteil  
Métro Crêteil l'Echat - Hôpital Henri Mondor

<b>Module 1</b> <u>19-20 mai 2022</u> J1 – Jeudi 19 mai xxxxx	<b>Parésie Spastique Déformante : définition d'un syndrome</b> 9H Physiopathologie Pr J-M GRACIES ( <i>Rééducation Neurolocomotrice, UPEC</i> )  14H Taxonomie Pr J-M GRACIES 16H Evaluations cliniques Pr J-M GRACIES
J2 – Vendredi 20 mai xxxxx	9H Plasticité cérébrale et musculaire Pr J-M GRACIES 15H Techniques neuro-rééducatives validées dans la parésie Pr J-M GRACIES + Maud PRADINES (MCF, UPEC)

## Module 2

2-3 JUIN 2022

J3 – Jeudi 2 juin

XXXXXX

J4 – Vendredi 3 juin

XXXXXX

### Syndromes parkinsoniens

9H Histoire, sémiologie, diagnostic, physiopathologie

Pr J-M GRACIES

11H Traitements médicamenteux et chirurgicaux

Pr J-M GRACIES

14H Imagerie cérébrale et syndromes parkinsoniens+

Pr P REMY (Neurologie, UPEC)

16H Stimulation cérébrale profonde non - Dr T HALBIG (Charité, Berlin)

9H15 Evaluation et Rééducation des Parésies faciales

Dr M BAUDE (Neurorééducation, UPEC)+

11h Traitement neurorééducatif Parkinsonisme

Pr J-M GRACIES

### **11h 10 Ateliers au choix :**

1. Analyse du mouvement (labo Mondor) – E Hutin - M Ghédira+
2. Evaluation parésie/injection – E Savard+
3. Evaluation parésie/prescription Contrat – M. Pradines+
4. Evaluation parkinsonisme – N. Bayle+
5. Autorééducation guidée mb sup parétique – JM Gracies
6. Autorééducation guidée mb inf parétique - C. Colas+
7. Autorééducation guidée Parkinsonisme - Th. Santiago+
8. Evaluation et prescription rééducation Tremblements - D. Motavasseli non
9. Kinovéa – MH Andruet non
10. Yoga thérapeutique – P. Parejo Margallo+
11. Tango et parkinsonisme – R. Delaroche+
12. Parésie faciale – éval clinique et 3D – M. Baude non

# DU Neurorééducation du Mouvement UPEC - mai-juillet 2022

**Module 3****9-10 JUIN 2022**

J5 – Jeudi 9 juin

xxxxxx

J6 – Vendredi 10 juin

Hôpital A. Chenevier

Pavillon Wurtz

**Tremblements – Cervelet - Ataxies – Apraxies – Chorées - Horizontalité****9H Tremblements : sémiologie, physiopathologie, typologie, évaluations**

Pr J-M GRACIES

**11H Tremblements : Neurorééducation et autres traitements**

Pr J-M GRACIES

**14H Ataxies, apraxies, chorées. Sémiolo**

Pr J-M GRACIES

**16H Complications de l'horizontalité**

Pr J-M GRACIES

**9h30-11h Ateliers au choix :**

1. Analyse du mouvement (labo Mondor) – E Hulin- M Ghédira non
2. Evaluation parésie/injection – E Savard+

3. Evaluation parésie/prescription Contrat – M. Pradines+

4. Evaluation parkinsonisme – N. Bayle+

5. Autorééducation guidée mb sup parétique – JM Gracies

6. Autorééducation guidée mb inf parétique - C. Colast+

7. Autorééducation guidée Parkinsonisme - Th. Santiago+

8. Evaluation et prescription rééducation Tremblements - D. Motavasseli+

9. Kinovéa – MH Andruet non

10. Yoga thérapeutique – P. Parejo Margallo+

11. Tango et parkinsonisme – R. Delaroche+

12. Parésie faciale – éval clinique et 3D – M. Baude 9h30+

**14H30 Traitements physiques du parkinsonisme (suite)**

Pr J-M GRACIES

**DU Neurorééducation du  
Mouvement  
UPEC - mai-juillet 2022**

# DU Neurorééducation du Mouvement

## UPEC - mai-juillet 2022

### **Module 4**

**16-17 JUIN 2022**

J7 – Jeudi 16 juin

xxxxxx

J8 – Vendredi 17 juin

Hôpital A. Chenevier  
Pavillon Wurtz

### **Analyse du Mouvement - Parésies faciales - Neurorestauration chez l'enfant**

9H Parésies infantiles – aspects théoriques

Pr J-M GRACIES

14H Equilibre et Chutes chez le sujet âgé

Pr J-M GRACIES

### **Visio plasticité musculaire influence muscle sur cerveau**

14H Biothérapies dans la maladie de Parkinson+

Pr S PALFI (Neurochirurgie, UPEC)

16H Pratique neurorééducative en parkinsonisme (suite)

Th SANTIAGO et Pr J-M GRACIES

14H30 Droit et handicap - Réparation du dommage neurologique

Inclusion sociale et modifications des droits de l'homme

Maître ME AFONSO (Avocat à la Cour, Paris)

# DU Neurorééducation du Mouvement

## UPEC - mai-juillet 2022

### **Module 5**

**23-24 JUIN**

J9 – Jeudi 23 juin  
xxxxx

J10 – Vendredi 24 juin  
xxxxx

#### **Cas concrets - Handicap – Aspects juridiques**

**9H15** Hétérorééducation parentale guidée 0-2 ans+

Dr C AMELON-PETIT (CRF Saint-Fargeau)

**11H** Kinésithérapie neurologique en pratique libérale+

F LAURENT (Kinésithérapeute libéral, Bordeaux)

**14H** Kinésithérapie neurologique en pratique libérale++

M PRADINES (MCF, Université Paris-Est Créteil, Paris)

**16H** *Tai Chi Médical et affections neurologiques du mouvement non*

*Dr L CONDAMINE (Professeur et Championne du Monde de Tai Chi, UPEC)*

**9-10h +++ EXAMEN ECRIT FACULTE DE MEDECINE CRETEIL +++**

**11H** Rôle du kinésithérapeute dans les Contrats d'Autorééducation Guidée

Th SANTIAGO

Cas concrets

Pr J-M GRACIES

### **Informations complémentaires - Conditions d'admission**

Auprès de la Faculté de Médecine de Créteil : DUFMC, 8 rue du Général Sarraïl 94010 Crêteil Cedex

Téléphone : 01 49 81 39 03 ou 37 32, mails : [inscriptions.dufmc@u-pec.fr](mailto:inscriptions.dufmc@u-pec.fr) / [dufmc.fi@u-pec.fr](mailto:dufmc.fi@u-pec.fr) / [dufmc.fc@u-pec.fr](mailto:dufmc.fc@u-pec.fr)





# CAG-unlimited !



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**ABSTRACT:** Spastic paresis follows chronic disruption of the central execution of volitional command. Motor function in patients with spastic paresis is subjected over time to three fundamental insults, of which the last two are avoidable: (1) the neural insult itself, which causes paresis, i.e., reduced voluntary motor unit recruitment; (2) the relative immobilization of the paretic body part, commonly imposed by the current care environment, which causes adaptive shortening of the muscles left in a shortened position and joint contracture; and (3) the chronic disuse of the paretic body part, which is typically self-imposed in most patients. Chronic disuse causes plastic rearrangements in the higher centers that further reduce the ability to voluntarily recruit motor units, i.e., that aggravate baseline paresis. Part I of this review focuses on the pathophysiology of the first two factors causing motor impairment in spastic paresis: the vicious cycle of paresis–disuse–paresis and the contracture in soft tissues.

*Muscle Nerve* 31: 535–551, 2005

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## **PATHOPHYSIOLOGY OF SPASTIC PARESIS. I: PARESIS AND SOFT TISSUE CHANGES**

JEAN-MICHEL GRACIES, MD, PhD

Department of Neurology, Mount Sinai Medical Center, One Gustave L Levy Place,  
Annenberg 2/Box 1052, New York, New York 10029-6574, USA

*Accepted 19 November 2004*

**ABSTRACT:** In the subacute and chronic stages of spastic paresis, stretch-sensitive (spastic) muscle overactivity emerges as a third fundamental mechanism of motor impairment, along with paresis and soft tissue contracture. Part II of this review primarily addresses the pathophysiology of the various forms of spastic overactivity. It is argued that muscle contracture is one of the factors that cause excessive responsiveness to stretch, which in turn aggravates contracture. Excessive responsiveness to stretch also impedes voluntary motor neuron recruitment, a concept termed stretch-sensitive paresis. None of the three mechanisms of impairment (paresis, contracture, and spastic overactivity) is symmetrically distributed between agonists and antagonists, which generates torque imbalance around joints and limb deformities. Thus, each may be best treated focally on an individual muscle-by-muscle basis. Intensive motor training of the less overactive muscles should disrupt the cycle of paresis-disuse-paresis, and concomitant use of aggressive stretch and focal weakening agents in their more overactive and shortened antagonists should break the cycle of overactivity-contracture-overactivity.

*Muscle Nerve* 31: 552–571, 2005

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## **PATHOPHYSIOLOGY OF SPASTIC PARESIS. II: EMERGENCE OF MUSCLE OVERACTIVITY**

JEAN-MICHEL GRACIES, MD, PhD

Department of Neurology, Mount Sinai Medical Center, One Gustave L Levy Place,  
Annenberg 2/Box 1052, New York, New York 10029-6574, USA

Accepted 19 November 2004



## Influence of effort intensity and gastrocnemius stretch on co-contraction and torque production in the healthy and paretic ankle

Maria Vinti<sup>a,b,c,\*</sup>, Annabelle Couillandre<sup>d</sup>, Jérôme Hausselle<sup>a</sup>, Nicolas Bayle<sup>a,b,c</sup>, Aldo Primerano<sup>b,c</sup>, Andrea Merlo<sup>e</sup>, Emilie Hutin<sup>a,b,c</sup>, Jean-Michel Gracies<sup>a,b,c</sup>

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<sup>b</sup> Université Paris Est Créteil (UPEC), Créteil, France

<sup>c</sup> AP-HP, Service de Médecine Physique et de Réadaptation, Unité de Neurorééducation, Groupe Hospitalier Henri Mondor, Créteil 94010, France

<sup>d</sup> EA2931, Centre de Recherches sur le Sport et le Mouvement, UFR STAPS, Université Paris Ouest (UPO), Nanterre, France

<sup>e</sup> Movement Analysis Laboratory, Rehabilitation Department, Reggio Emilia Local Health Unit Correggio, Reggio Emilia, Italy

### ARTICLE INFO

#### Article history:

Accepted 20 August 2012

Available online 10 October 2012

#### Keywords:

Hemiparesis

Spastic co-contraction

Torque

### HIGHLIGHTS

- This study quantifies plantar flexor co-contraction in hemiparetic patients as compared with healthy subjects during graded dorsiflexor efforts in two ankle positions.
- In hemiparesis, gastrocnemius stretch (position knee extended) markedly increased co-contraction as compared to normal, reversing or canceling the torque intended in 26% cases.
- The major dynamometric impact of co-contraction with stretched position of the co-contracting muscle may justify muscle length modifications (aggressive stretch programs) to improve function in spastic paresis.

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# SPASTIC COCONTRACTION IN HEMIPARESIS: EFFECTS OF BOTULINUM TOXIN

MARIA VINTI, PT, MSc,<sup>1,2,3</sup> FILOMENA COSTANTINO, MSc,<sup>1,2</sup> NICOLAS BAYLE, MD,<sup>1,2,3</sup>  
DAVID M. SIMPSON, MD,<sup>4</sup> DONALD J. WEISZ, PhD,<sup>5</sup> and JEAN-MICHEL GRACIES, MD, PhD<sup>1,2,3</sup>

<sup>1</sup> Arts et Métiers ParisTech, Laboratoire de Biomécanique, Paris, France

<sup>2</sup> Université Paris Est Créteil (UPEC), Créteil, France

<sup>3</sup> AP-HP, Service de Médecine Physique et de Réadaptation, Unité de Neurorééducation, Groupe Hospitalier Henri Mondor, 50 avenue du Maréchal de Lattre de Tassigny, Créteil F-94010, France

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<sup>5</sup> Department of Neurosurgery, Mount Sinai School of Medicine, New York, New York, USA

Accepted 23 April 2012

**ABSTRACT:** *Introduction:* In this study of spastic hemiparesis we evaluated cocontraction during sustained agonist/antagonist efforts, before and after botulinum toxin (BoNT) injection in 1 agonist. *Methods:* Nineteen hemiparetic subjects performed maximal isometric elbow flexion/extension efforts with the elbow at 100° (extensors stretched). Using flexor and extensor surface electromyography we calculated agonist recruitment/cocontraction indices from 500-ms peak voluntary agonist recruitment, before and 1 month after onabotulinumtoxinA injection (160 U) into biceps brachii. *Results:* Before injection, agonist recruitment and cocontraction indices were higher in extensors than flexors [0.74 ± 0.15 vs. 0.59 ± 0.10 ( $P < 0.01$ ) and 0.43 ± 0.25 vs. 0.25 ± 0.13 ( $P < 0.05$ ), respectively]. Biceps injection decreased extensor cocontraction index (~35%,  $P < 0.05$ ) while increasing flexor agonist recruitment and cocontraction indices. *Conclusions:* In spastic hemiparesis, stretch may facilitate agonist recruitment and spastic cocontraction. In the non-injected antagonist, cocontraction may be reduced by enhanced reciprocal inhibition from a more relaxed, and therefore stretched, agonist, or through decreased recurrent inhibition from the injected muscle.

*Muscle Nerve* 46: 926–931, 2012

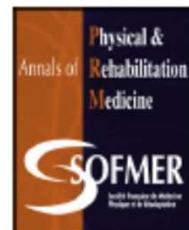
offers opportunities to better stretch the injected muscle and strengthen its antagonist.<sup>5,6</sup>

Although several studies have qualitatively described cocontraction in spastic paresis, few have attempted to quantify the phenomenon.<sup>7–9</sup> Dynamometric measurements have been used, but the mathematical approaches involved<sup>10,11</sup> are difficult to apply in practice and would not have much validity in rheologically modified muscles, as is the case in spastic paresis. Electromyography (EMG) measurements are a preferred choice. The root mean square (RMS) of the EMG signal expresses average flows of electrical signals in a window of time and is considered a valid measure of motor unit behavior.<sup>12</sup> In this study, we used the RMS obtained during a 500-ms interval around the peak voltage as the reference for maximal voluntary muscle recruitment<sup>13,14</sup> to perform agonist recruit-



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Update article

## Coefficients of impairment in deforming spastic paresis



J.-M. Gracies

Service de rééducation neurolocomotrice, laboratoire analyse et restauration du mouvement, université Paris-Est, hôpitaux universitaires Henri-Mondor, AP-HP, 51, avenue du Maréchal-de-Lattre-de-Tassigny, 94010 Créteil, France



### ARTICLE INFO

#### Article history:

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#### Keywords:

Spastic paresis  
 Muscle length  
 Spastic cocontraction  
 Spastic dystonia  
 Spasticity  
 Stepwise quantified assessment  
 Tardieu Scale  
 Fatigability  
 Weakness  
 Coefficients of impairment

### ABSTRACT

This position paper introduces an assessment method using staged calculation of coefficients of impairment in spastic paresis, with its rationale and proposed use. The syndrome of deforming spastic paresis superimposes two disorders around each joint: a neural disorder comprising stretch-sensitive paresis in agonists and antagonist muscle overactivity, and a muscle disorder ("spastic myopathy") combining shortening and loss of extensibility in antagonists. Antagonist muscle overactivity includes spastic cocontraction (misdirected descending command), spastic dystonia (tonic involuntary muscle activation, at rest) and spasticity (increased velocity-dependent reflexes to phasic stretch, at rest). This understanding of various types of antagonist resistance as the key limiting factors in paretic movements prompts a stepwise, quantified, clinical assessment of antagonist resistances, elaborating on the previously developed Tardieu Scale. Step 1 quantifies limb function (e.g. ambulation speed in lower limb, Modified Frenchay Scale in upper limb). The following four steps evaluate various angles X of antagonist resistance, in degrees all measured from 0°, position of minimal stretch of the tested antagonist. Step 2 rates the functional muscle length, termed  $X_{V1}$  ( $V1$ , slowest stretch velocity possible), evaluated as the angle of arrest upon slow and strong passive muscle stretch.  $X_{V1}$  is appreciated with respect to the expected normal passive amplitude,  $X_N$ , and reflects combined muscle contracture and residual spastic dystonia. Step 3 determines the angle of catch upon fast stretch, termed  $X_{V3}$  ( $V3$ , fastest stretch velocity possible), reflecting spasticity. Step 4 measures the maximal active range of motion against the antagonist, termed  $X_A$ , reflecting agonist capacity to overcome passive (stiffness) and active (spastic cocontraction) antagonist resistances over a single movement. Finally, Step 5 rates the residual active amplitude after 15 seconds of maximal amplitude rapid alternating movements,  $X_{A15}$ . Amplitude decrement from  $X_A$  to  $X_{A15}$  reflects fatigability. Coefficients of shortening ( $(X_N - X_{V1})/X_N$ ), spasticity ( $(X_{V1} - X_{V3})/X_{V1}$ ), weakness ( $(X_{V1} - X_A)/X_{V1}$ ) and fatigability ( $(X_A - X_{A15})/X_A$ ) are derived. A high (e.g., >10%) coefficient of shortening prompts aggressive treatment of the muscle disorder – e.g. by stretch programs, such as prolonged stretch postures –, while high coefficients of weakness or fatigability prompt addressing the neural motor command disorder, e.g. using training programs such as repeated alternating movements of maximal amplitude.



# Clinical Neurophysiology

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## Review

# On Denny-Brown's 'spastic dystonia' – What is it and what causes it?



Jakob Lorentzen <sup>a,b,\*</sup>, Maud Pradines <sup>c</sup>, Jean-Michel Gracies <sup>c</sup>, Jens Bo Nielsen <sup>a,b</sup>

<sup>a</sup> Section for Integrative Neuroscience, Center for Neuroscience, University of Copenhagen, Denmark

<sup>b</sup> Elsøss Institute, Holmegårdsvæj 28, 2920 Charlottenlund, Denmark

<sup>c</sup> EA 7377 BIOTN, Université Paris-Est, Hospital Albert Chenevier-Henri Mondor, Service de Rééducation Neurodynamotrice, APHP, Créteil, France

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## ARTICLE INFO

### Article history:

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### Keywords:

Spasticity

Spastic dystonia

Hypertonia

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## HIGHLIGHTS

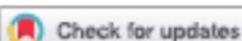
- Stretch and effort-unrelated sustained involuntary muscle activity following central motor lesions may be caused by:
- Plastic changes at a spinal level involving upregulation and sprouting of surviving descending fibres and/or changes in intrinsic properties of motoneurones.
- Re-organization in the motor cortex.
- Lesions in basal ganglia.

**Baude M, Nielsen JB, Gracies JM.**

***The neurophysiology of deforming spastic paresis: A revised taxonomy.***

**Ann Phys Rehabil Med. 2019;62(6):426-430**

This paper revisits the taxonomy of the neurophysiological consequences of a persistent impairment of motor command execution in the classic environment of sensorimotor restriction and muscle hypo-mobilization in short position. Around each joint, the syndrome involves 2 disorders, muscular and neurologic. The muscular disorder is promoted by muscle hypo-mobilization in short position in the context of paresis, in the hours and days after paresis onset: this genetically mediated, evolving myopathy, is called spastic myopathy. The clinician may suspect it by feeling extensibility loss in a resting muscle, although long after the actual onset of the disease. The neurologic disorder, promoted by sensorimotor restriction in the context of paresis and by the muscle disorder itself, comprises 4 main components, mostly affecting antagonists to desired movements: the first is spastic dystonia, an unwanted, involuntary muscle activation at rest, in the absence of stretch or voluntary effort; spastic dystonia superimposes on spastic myopathy to cause visible, gradually increasing body deformities; the second is spastic cocontraction, an unwanted, involuntary antagonist muscle activation during voluntary effort directed to the agonist, aggravated by antagonist stretch; it is primarily due to misdirection of the supraspinal descending drive and contributes to reducing movement amplitude; and the third is spasticity, one form of hyperreflexia, defined by an enhancement of the velocity-dependent responses to phasic stretch, detected and measured at rest (another form of hyperreflexia is "nociceptive spasms", following flexor reflex afferent stimulation, particularly after spinal cord lesions). The 3 main forms of overactivity, spastic dystonia, spastic cocontraction and spasticity, share the same motor neuron hyperexcitability as a contributing factor, all being predominant in the muscles that are more affected by spastic myopathy. The fourth component of the neurologic disorder affects the agonist: it is stretch-sensitive paresis, which is a decreased access of the central command to the agonist, aggravated by antagonist stretch. Improved understanding of the pathophysiology of deforming spastic paresis should help clinicians select meaningful assessments and refined treatments, including the utmost need to preserve muscle tissue integrity as soon as paresis sets in.



## Quantified clinical measures linked to ambulation speed in hemiparesis

Mouna Ghédira <sup>a,b</sup>, Maud Pradines<sup>b</sup>, Valentina Mardale<sup>a</sup>, Jean-Michel Gracies<sup>a,b</sup>, Nicolas Bayle<sup>a,b</sup>, and Emilie Hutin<sup>a,b</sup>

<sup>a</sup>Laboratoire analyse et restauration du Mouvement (ARM, Hôpitaux Universitaires Henri Mondor, Assistance Publique-Hôpitaux de Paris (AP-HP); <sup>b</sup>EA 7377 BIOTN, Université Paris-Est Créteil (UPEC), Créteil, France

### ABSTRACT

**Background:** In spastic paresis, the respective contributions to active function of antagonist hypoextensibility, spasticity, and impaired descending command remain unknown. Objectives: We explored correlations between ambulation speed and coefficients of shortening, spasticity and, weakness for three lower limb extensors.

**Methods:** This retrospective study identified 140 subjects with chronic hemiparesis (>6 months since injury) assessed during a single visit with barefoot 10-meter ambulation at comfortable and fast speed, and measurements of passive range of motion ( $X_{V1}$ ), angle of catch at fast stretch ( $X_{V3}$ ) and active range of motion ( $X_A$ ) against the resistance of gastrocnemius, rectus femoris, and gluteus maximus. Coefficients of shortening ( $C_{SH} = [X_N - X_{V1}] / X_N$ ;  $X_N$ , normal expected amplitude based on anatomical values), spasticity ( $C_{Sp} = [X_{V1} - X_{V3}] / X_{V1}$ ), and weakness ( $C_{Wk} = [X_{V1} - X_A] / X_{V1}$ ) were derived. For each muscle, multivariable analysis explored  $C_{SH}$ ,  $C_{Sp}$ , and  $C_{Wk}$  as potential predictors of ambulation speed.

**Results:** Ambulation speed was  $0.62 \pm 0.28$ m/s (mean  $\pm$  SD, comfortable) and  $0.84 \pm 0.38$ m/s (fast) and was correlated with  $C_{SH}$  and  $C_{Wk}$  against gastrocnemius ( $C_{SH}$ , comfortable, ns; fast,  $\beta = -0.20$ ,  $p = .03$ ;  $C_{Wk}$ , comfortable,  $\beta = -0.21$ ,  $p = .010$ ; fast,  $\beta = -0.21$ ,  $p = .012$ ), rectus femoris ( $C_{SH}$ , comfortable,  $\beta = -0.41$ ,  $p = 6E^{-7}$ ; fast,  $\beta = -0.43$ ,  $p = 5E^{-7}$ ;  $C_{Wk}$ , comfortable,  $\beta = -0.36$ ,  $p = 5E^{-5}$ ; fast,  $\beta = -0.33$ ,  $p = .0003$ ) and gluteus maximus ( $C_{SH}$ , comfortable,  $\beta = -0.19$ ,  $p = .02$ ; fast,  $\beta = -0.26$ ,  $p = .002$ ;  $C_{Wk}$ , comfortable,  $\beta = -0.26$ ,  $p = .002$ ; fast,  $\beta = -0.22$ ,  $p = .010$ ). Ambulation speed was not correlated with  $C_{Sp}$ .

**Conclusions:** In chronic hemiparesis, ambulation speed correlates with coefficients of shortening and of weakness in lower limb extensors, but not with their spasticity level. This may encourage therapists to focus treatment primarily on muscle shortening by stretching programs and on impaired descending command by active training.

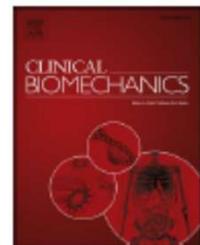
### ARTICLE HISTORY

Received 18 January 2021

Accepted 5 June 2021

### KEYWORDS

Paresis; contracture; spasticity; walking; stroke



## Agonist and antagonist activation at the ankle monitored along the swing phase in hemiparetic gait

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### ARTICLE INFO

**Keywords:**  
Electromyography  
Muscle spasticity  
Paresis  
Dorsiflexion  
Stretching  
Antagonist activation

### ABSTRACT

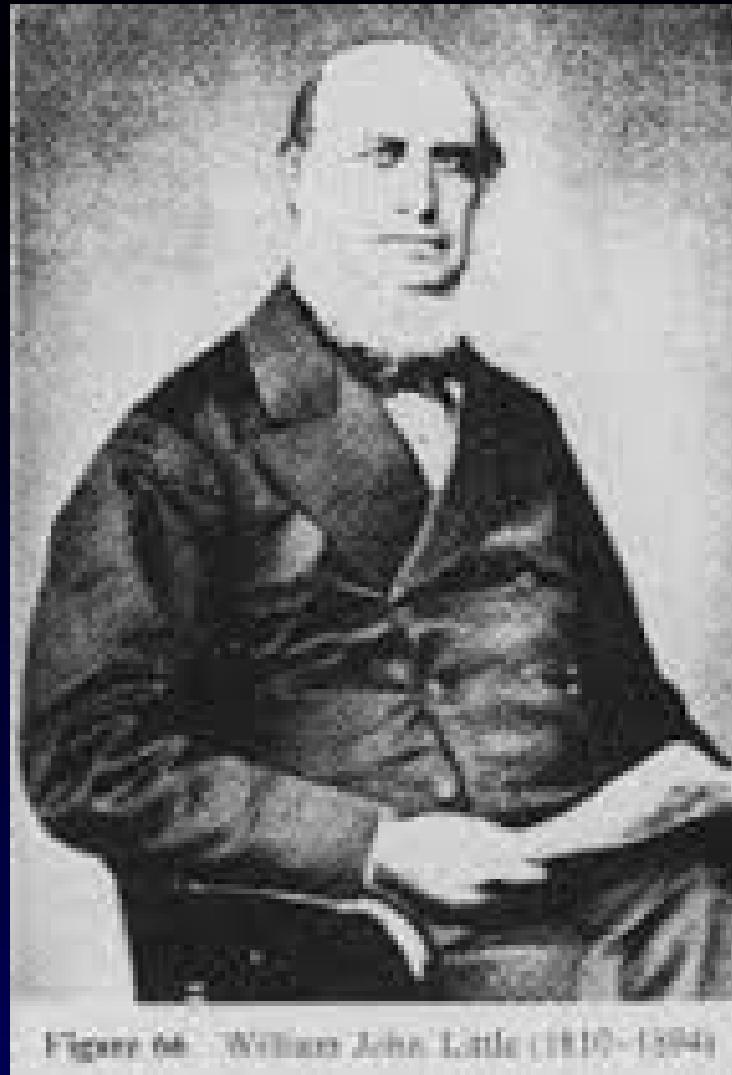
**Background:** Descending command in hemiparesis is reduced to agonists and misdirected to antagonists. We monitored agonist and antagonist activation along the swing phase of gait, comparing paretic and non-paretic legs.

**Methods:** Forty-two adults with chronic hemiparesis underwent gait analysis with bilateral EMG from tibialis anterior, soleus and gastrocnemius medialis. We monitored ankle and knee positions, and coefficients of agonist activation in tibialis anterior and of antagonist activation in soleus and gastrocnemius medialis over the three thirds of swing phase. These coefficients were defined as the ratio of the root-mean-square EMG from one muscle over any period to the root-mean-square EMG from the same muscle over 100 ms of its maximal voluntary isometric contraction.

**Findings:** As against the non-paretic side, the paretic side showed lesser ankle dorsiflexion and knee flexion ( $P < 1.E^{-5}$ ), with higher coefficients of agonist activation in tibialis anterior ( $+100 \pm 28\%$ ,  $P < 0.05$ ), and of antagonist activation in soleus ( $+224 \pm 41\%$ ,  $P < 0.05$ ) and gastrocnemius medialis ( $+276 \pm 49\%$ ,  $P < 0.05$ ). On the paretic side, coefficient of agonist activation in tibialis anterior decreased from mid-swing on; coefficients of antagonist activation in soleus and gastrocnemius medialis increased and ankle dorsiflexion decreased in late swing ( $P < 0.05$ ).

**Interpretation:** During the swing phase in hemiparesis, normalized tibialis anterior recruitment is higher on the paretic than on the non-paretic leg, failing to compensate for a marked increase in plantar flexor activation (cocontraction). The situation deteriorates along swing with a decrease in tibialis anterior recruitment in parallel with an increase in plantar flexor activation, both likely related to gastrocnemius stretch during knee re-extension.

# *Deforming Spastic Paresis?*



Little WJ. “Deformities of the human frame”, 1843

# Which factors impede movements?

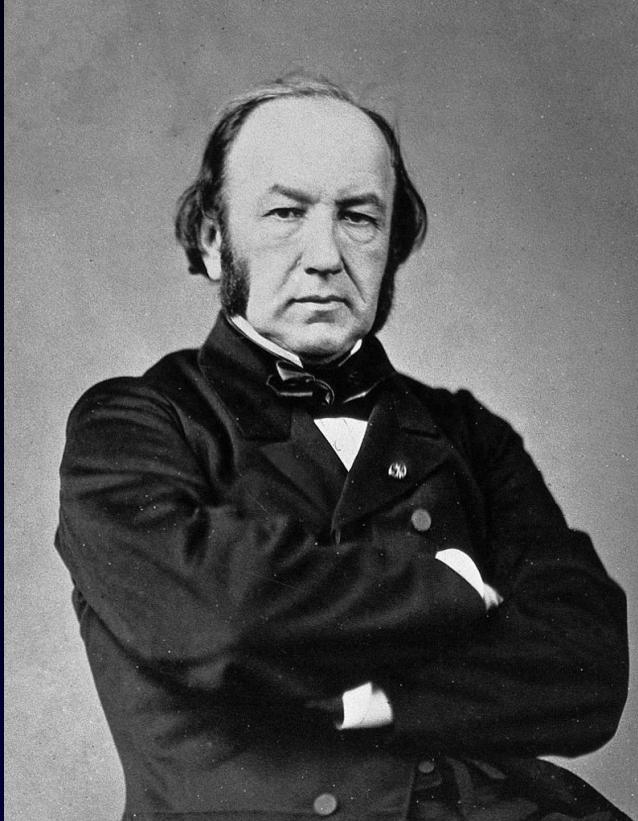




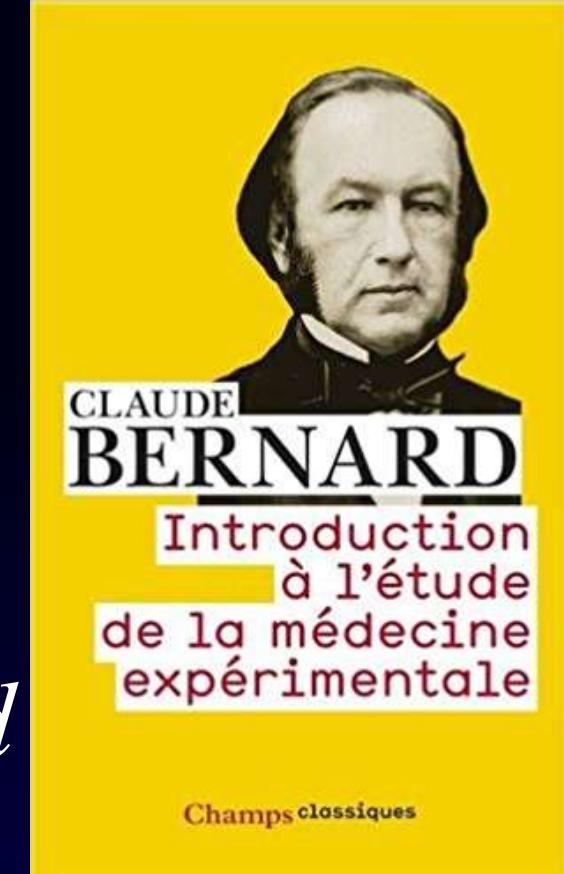
**Deforming spastic paresis is first a problem with the antagonist.**

*Tardieu, throughout his career*

**But which problem?**



**Physiology** must be  
able to explain life's  
phenomena, provided  
it remains built upon  
the knowledge of  
**histology**.



*Claude Bernard, c 1845*

The Syndrome of  
Deforming Spastic Paresis

=

*Spastic Myopathy*

+ ...

RES

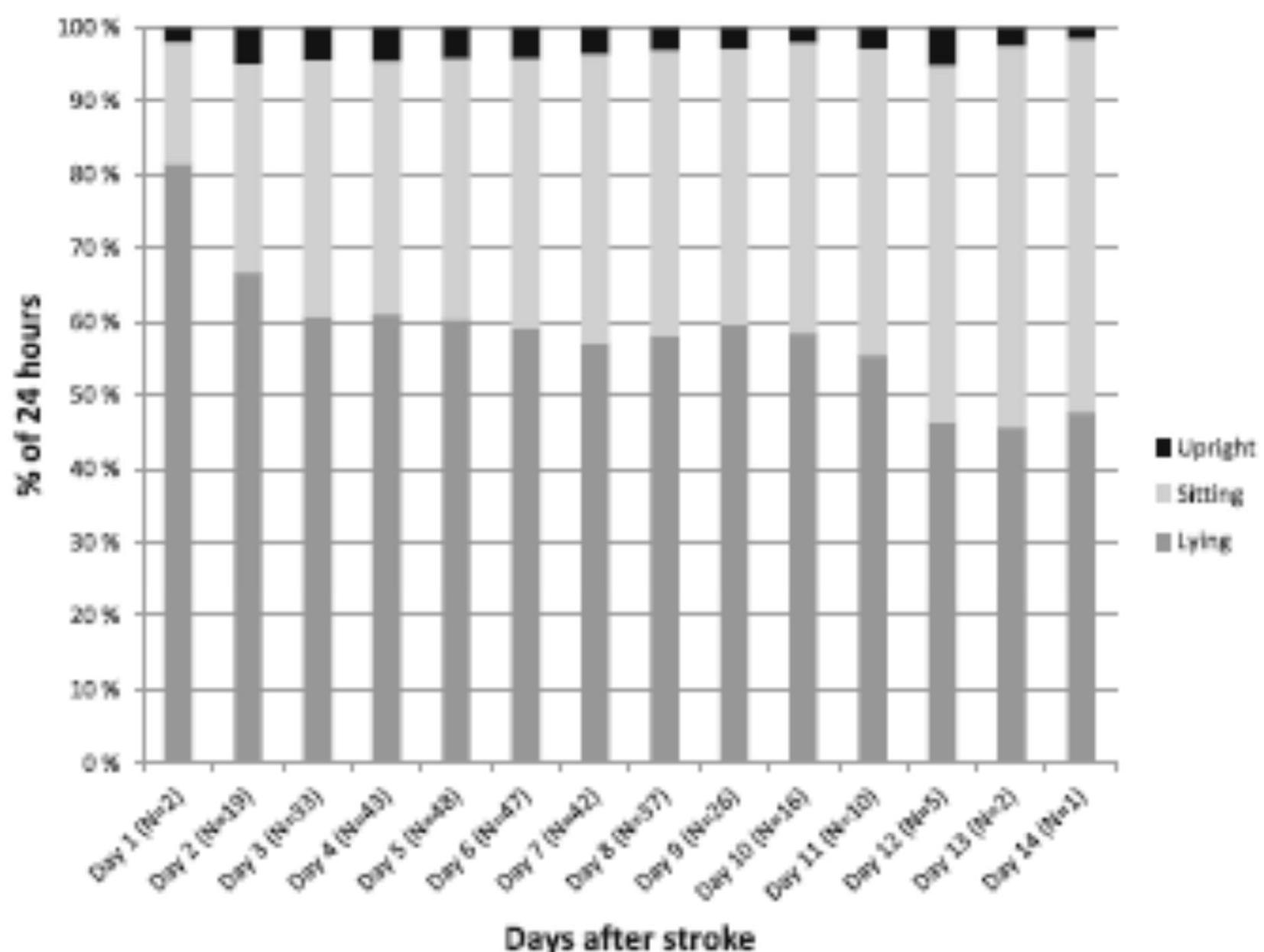
Tim  
dur  
a p

Ole Pett  
and Tor

Access



davik<sup>1,3</sup>



**Fig. 2** Percentage of time spent in different positions within every 24 h

# Time of arm positions in healthy subjects

- Arm movements observed for >5h/day in homes and local community of 21 older people age 73 (SD 7).
- Duration (min/hr) of arm positions  $> 90^\circ$  elevation + purpose (manipulating, holding, reaching, pulling/pushing, or gesturing) recorded.
- Participants' arms spent 0.6 min/hr at  $> 90^\circ$  elevation

**~ 6-12 min / waking day !**

*Schurr K, Ada L. Observation of arm behaviour in healthy elderly people: implications for contracture prevention after stroke.  
Aust J Physiother. 2006;52(2):129-33*

**Immobilization  
or hypo-  
mobilization in  
short position**

=

**Muscle aggression  
worse than stroke**

*(Jalal et al, 2019)*



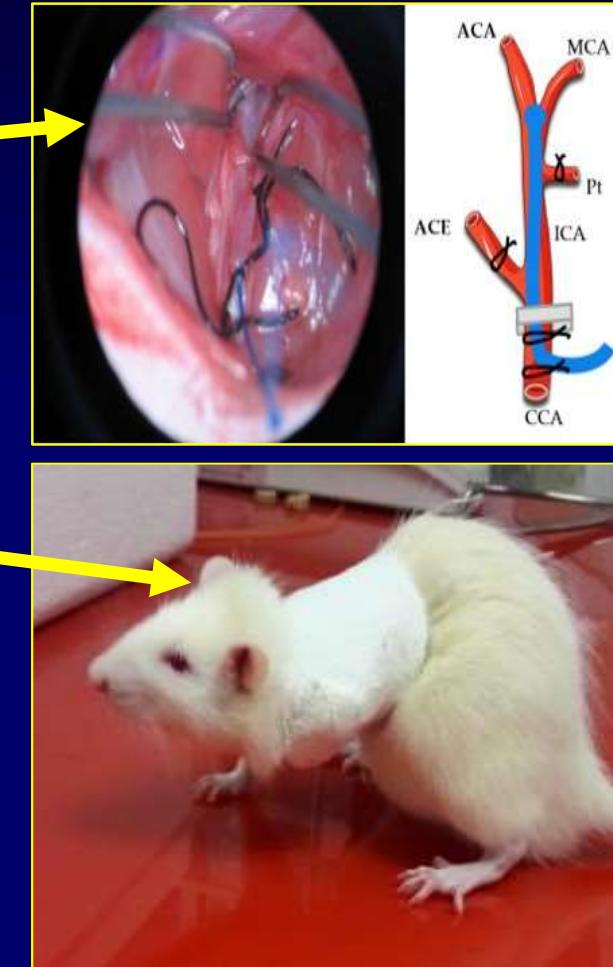
*Singer et al, 2002*

# Muscle structure: Role of Immobilization vs Stroke?

Four groups :

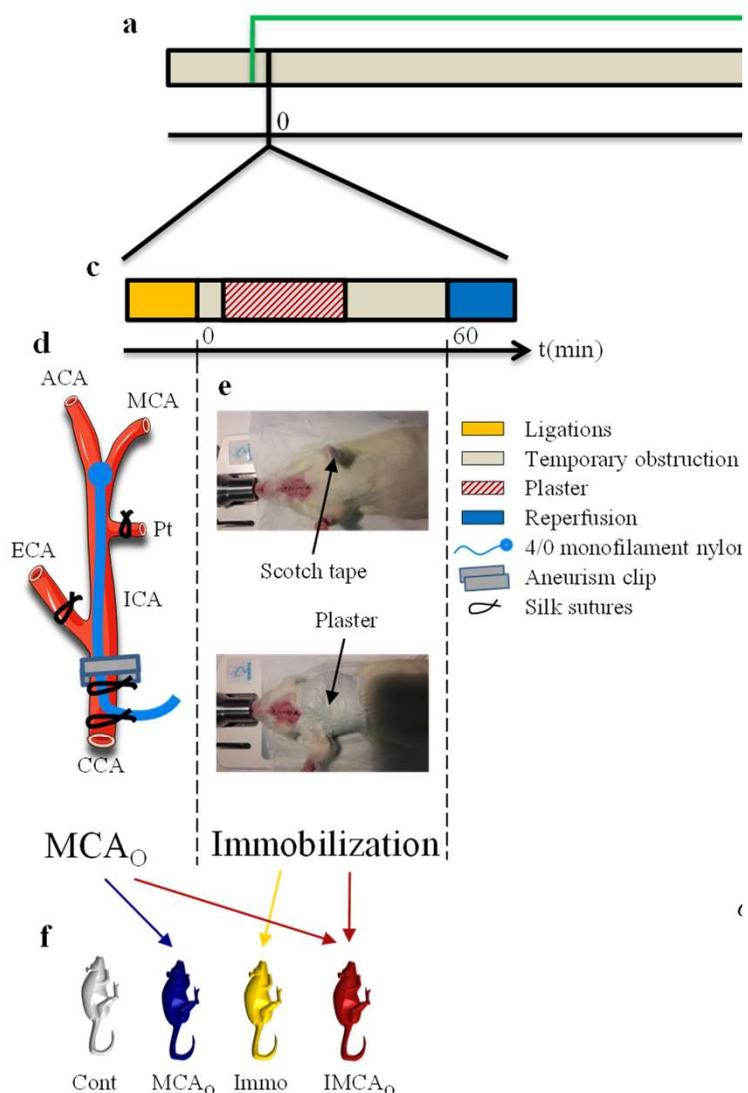
- S: “Stroke” without immobilization
- I: "Immobilization" without stroke
- S+I: “Stroke+Immobilization”
- C: “Sham” (failed strokes)

Duration : 14 days



*Jalal N, Gracies JM, Zidi M. Mechanical and microstructural changes of skeletal muscle following immobilization and/or stroke.  
Biomech Model Mechanobiol. 2020;19(1):61-80*

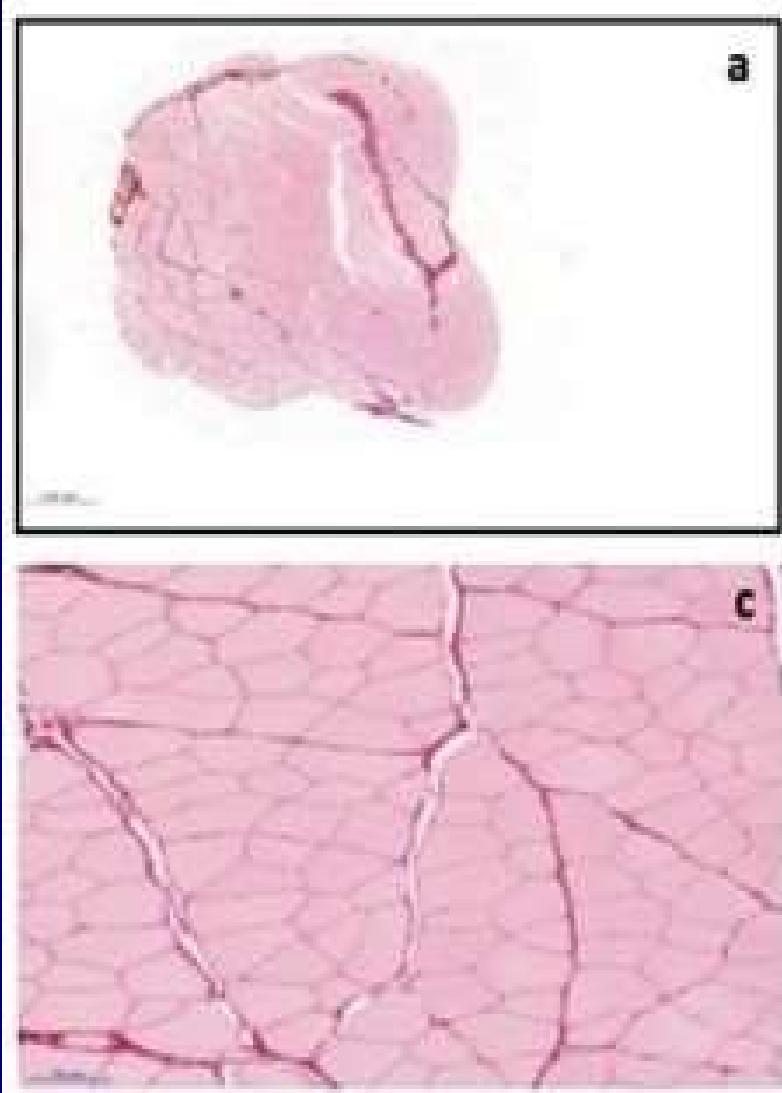
# Methods



*Jalal N, Gracies JM, Zidi M. Mechanical and microstructural changes of skeletal muscle following immobilization and/or stroke. Biomech Model Mechanobiol. 2020;19(1):61-80*

# Histology of flexor carpi ulnaris

## Red Sirius: quantification of collagen



Stroke w/o  
Immobilization

Immobilization w/o  
stroke

*Jalal N, Gracies JM, Zidi M. Mechanical and microstructural changes of skeletal muscle following immobilization and/or stroke. Biomech Model Mechanobiol. 2020;19(1):61-80*



# Do Muscle Changes Contribute to the Neurological Disorder in Spastic Paresis?

Maud Pradines<sup>1,2\*</sup>, Mouna Ghédira<sup>1,2</sup>, Blaise Bignami<sup>2</sup>, Jordan Vielotte<sup>2</sup>, Nicolas Bayle<sup>1,2</sup>, Christina Marciniaik<sup>3,4</sup>, David Burke<sup>5</sup>, Emilie Hutin<sup>1,2</sup> and Jean-Michel Gracies<sup>1,2</sup>

<sup>1</sup> UR 7377 BIOTN, Laboratoire Analyse et Restauration du Mouvement, Université Paris Est Crétal (UPEC), Crétal, France

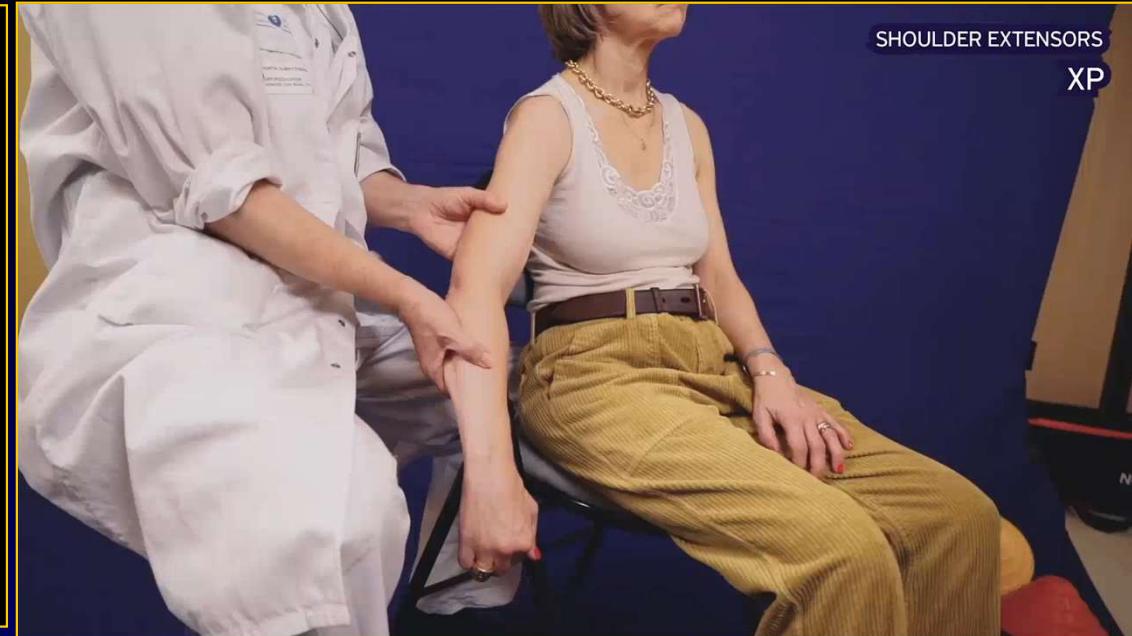
<sup>2</sup> AP-HP, Service de Rééducation Neurolocomotrice, Unité de Neuroéducation, Hôpitaux Universitaires Henri Mondor,

Crétal, France, <sup>3</sup> Department of Physical Medicine and Rehabilitation, Northwestern University and the Shirley Ryan AbilityLab, Chicago, IL, United States, <sup>4</sup> Department of Neurology, Northwestern University and the Shirley Ryan AbilityLab, Chicago, IL, United States, <sup>5</sup> Department of Neurology, Royal Prince Alfred Hospital and the University of Sydney, Sydney, NSW, Australia



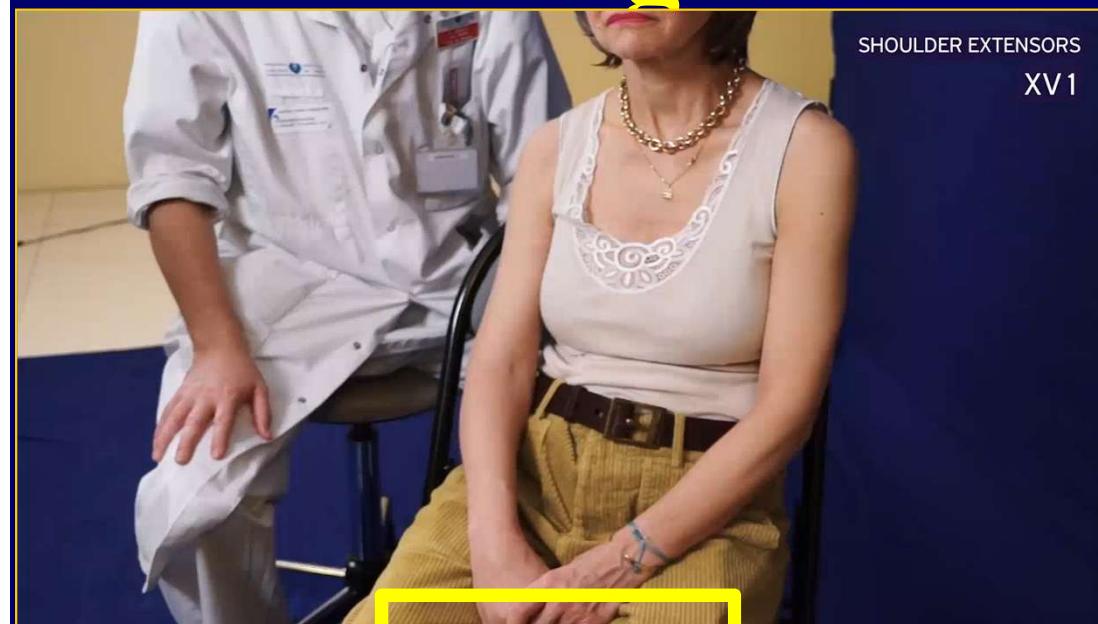
**Finger flexor angles**

INTRO



## 1. Angle

SHOULDER EXTENSORS  
XV1



## 2. XP

SHOULDER EXTENSORS  
XV3



## 3. X<sub>V1</sub>

Five Step Assessment – Shoulder extensors

## 4. X<sub>V3</sub>

# Five Step Assessment – Shoulder extensors

4. X<sub>A</sub>



SHOULDER EXTENSORS  
XA

5. X<sub>A15</sub>



SHOULDER EXTENSORS  
XA 15

# Step 2 => “Coefficient of Shortening” = Muscle disorder

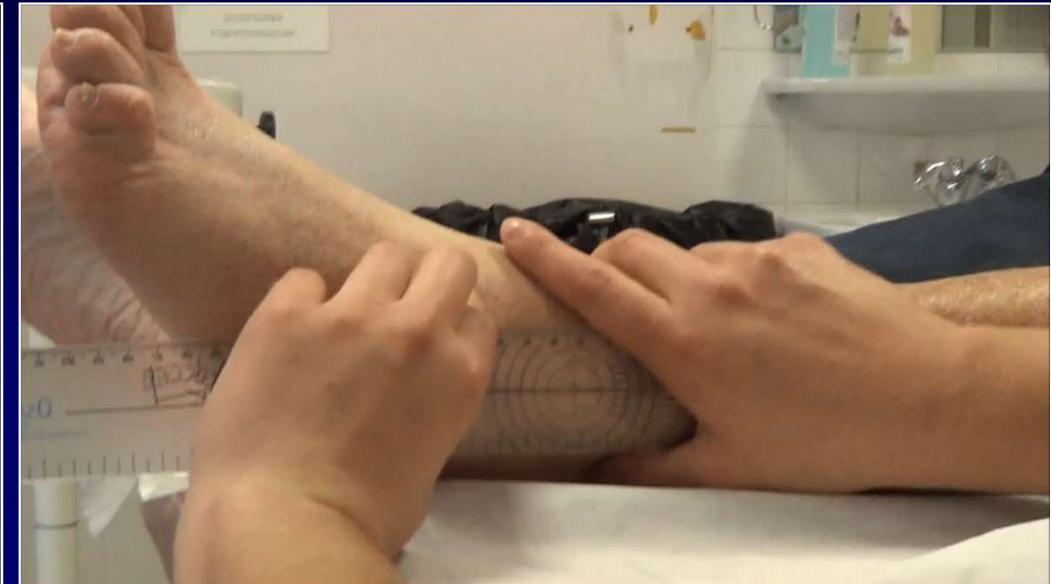


Maximal passive range of motion  $X_{V1}$   
Expected physiological value:  $X_N$

$$\text{Coefficient of Shortening} = (X_N - X_{V1})/X_N$$

*Gracies. Coefficients if impairment in spastic paresis.  
Ann Phys Med Rehabil 2015*

# Steps 4+2 => “Coefficient of Weakness” = Neurological disorder



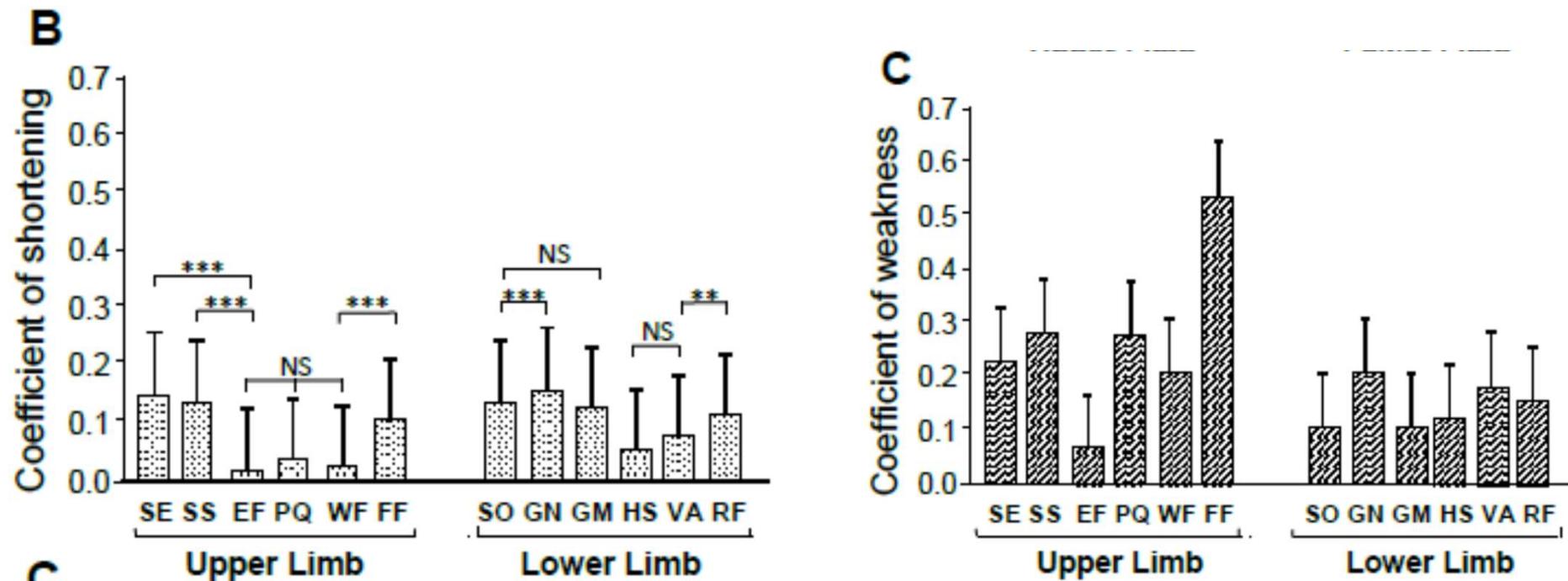
Maximal active range of motion  $X_A$   
Maximal passive range of motion  $X_{V1}$

$$\text{Coefficient of Weakness} = (X_{V1} - X_A) / X_{V1}$$

*Gracies. Coefficients of Impairment in Spastic Paresis.  
Ann Phys Med Rehabil 2015*

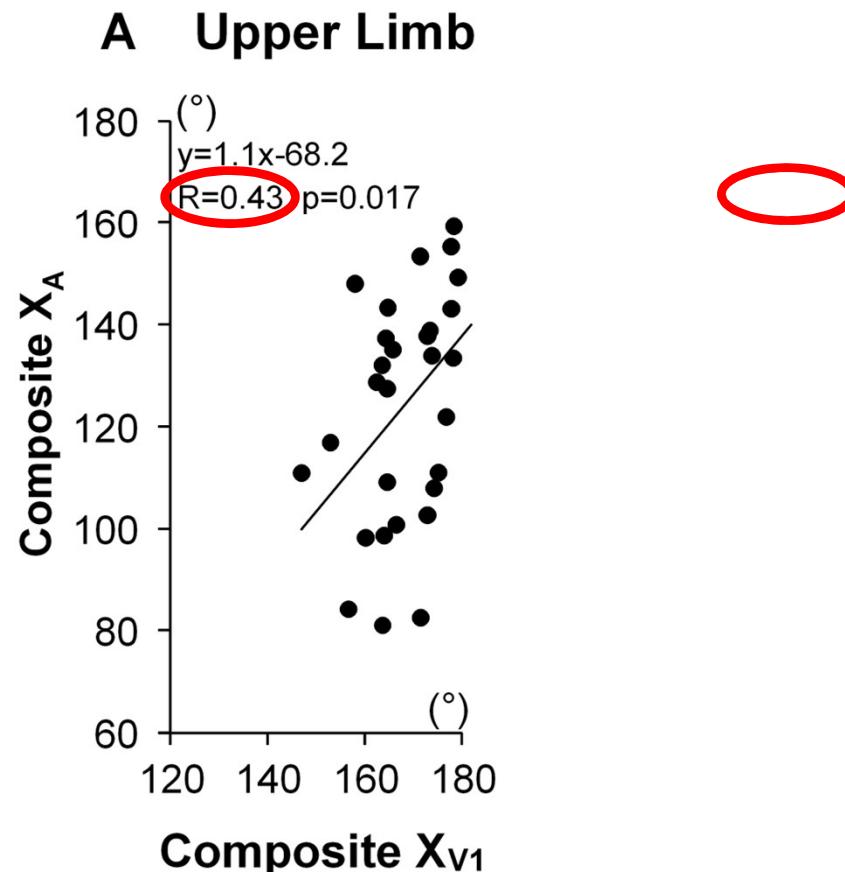
Figure 3

# Paretic upper and lower limb are two different beasts



*Pradines et al, Does the muscle disorder contribute to the neurological disorder in spastic paresis? Front Neurol 2022, in press*

# Role of histological muscle changes in active movements



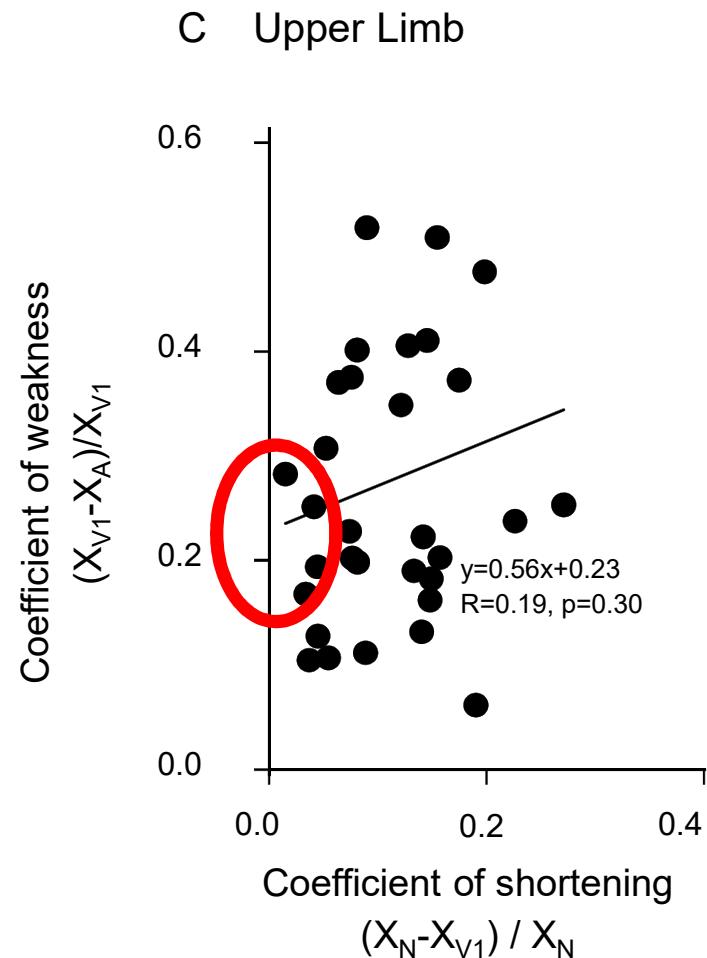
*Pradines et al, Does the muscle disorder contribute to the neurological disorder in spastic paresis? Front Neurol 2022, in press*

In words..

In the paretic lower limb (~50%)  
and to a lesser degree in the paretic  
upper limb (~16%?),

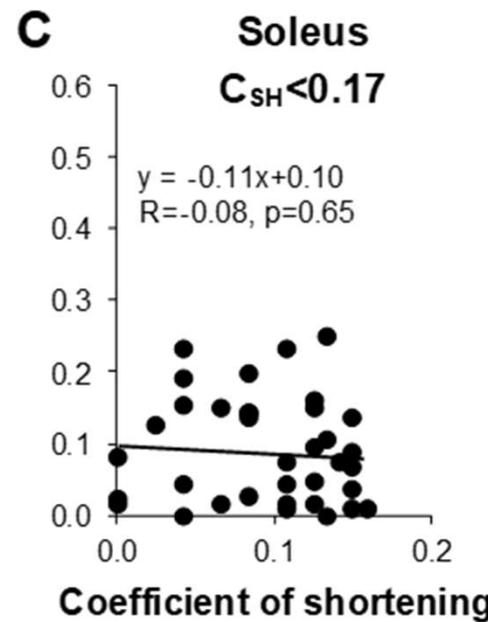
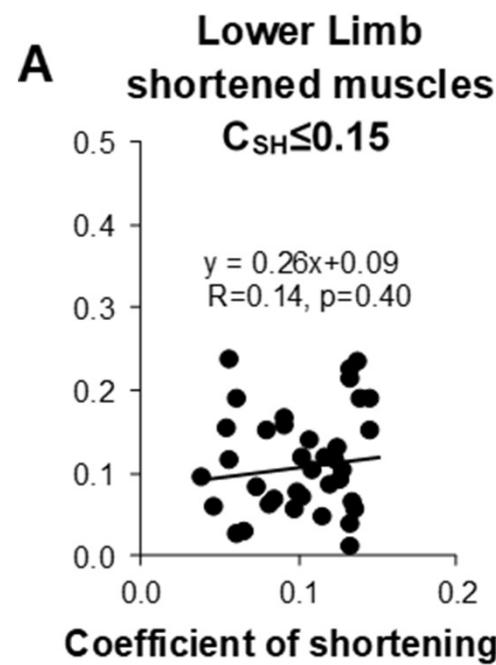
**active movement capacities  
*are determined by*  
passive movement capacities.**

# Paretic upper and lower limb are two different beasts



*Pradines et al, Does the muscle disorder contribute to the neurological disorder in spastic paresis? Front Neurol 2022, in press*

# Role of histological muscle changes in increasing cocontractions?



*Pradines et al, Does the muscle disorder contribute to the neurological disorder in spastic paresis? Front Neurol 2022, in press*

# In two Double nature of ‘coefficients of shortening’ and ‘of weakness’?

- $X_{V1}$  = a mostly histological measure:  $X_{V1}$  measurements after lidocaine blocks still remain far from expected physiological values (*Winston et al, 2019*)
- $X_A$  = a mostly neurological measure, of cocontractions:  $X_A$  markedly increased after lidocaine blocks (*Winston et al, 2019*)

*Winston P, Mills PB, Reebye R, Vincent D. Cryoneurootomy as a Percutaneous Mini-invasive Therapy for the Treatment of the Spastic Limb: Case Presentation, Review of the Literature, and Proposed Approach for Use. Arch Rehabil Res Clin Transl. 2019 Oct 17;1(3-4):100030*

The Syndrome of  
Deforming Spastic Paresis

=

*Spastic Myopathy*  
+ *Spastic Cocontraction*

# Spastic cocontraction recording during gait



# Agonist and antagonist activation around the ankle during the swing phase of gait in hemiparesis

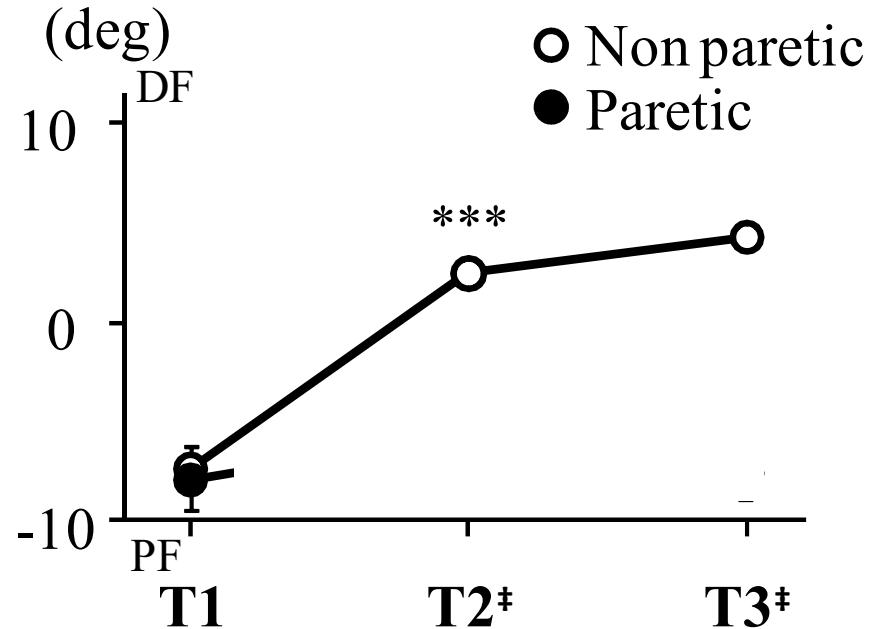
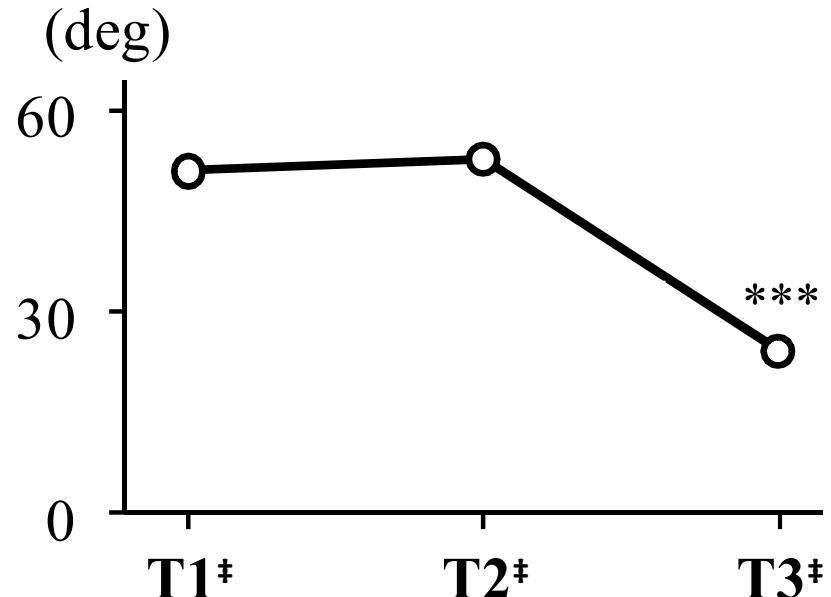
Subjects (n)	42
Age (y)	50 ± 15
Time since paresis onset (y)	7 ± 7
<i>Gender</i>	
Female (n)	14
Male (n)	28
<i>Paretic side</i>	
Left (n)	28
Right (n)	14
<i>Cause</i>	
Ischemic stroke (n)	21
Hemorrhagic stroke (n)	10
Non-evolutive tumor (n)	6
Traumatic brain injury (n)	5

## *Comfortable gait*

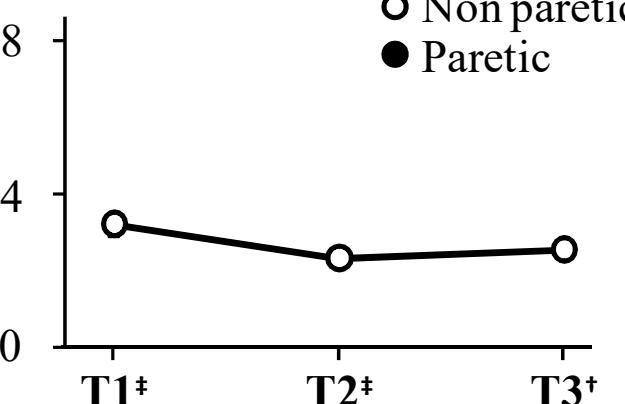
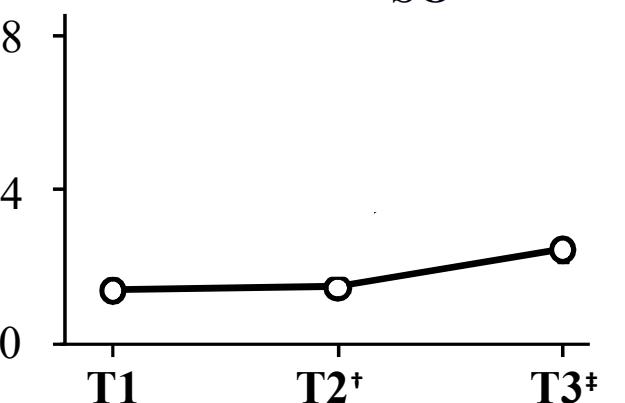
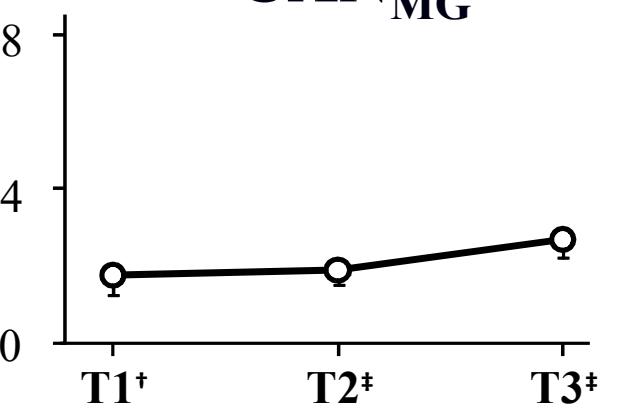
Speed (m/s)	0.66 ± 0.26
Paretic step length (m)	0.47 ± 0.12
Non paretic step length (m)	0.41 ± 0.16
Cadence (step/s)	1.47 ± 0.27

Ghédira M, Albertsen IM, Mardale V, Loche CM, Vinti M, Gracies JM, Bayle N, Hulin E. Agonist and antagonist activation at the ankle monitored along the swing phase in hemiparetic gait.

Clin Biomech (Bristol, Avon). 2021 Oct;89:105459

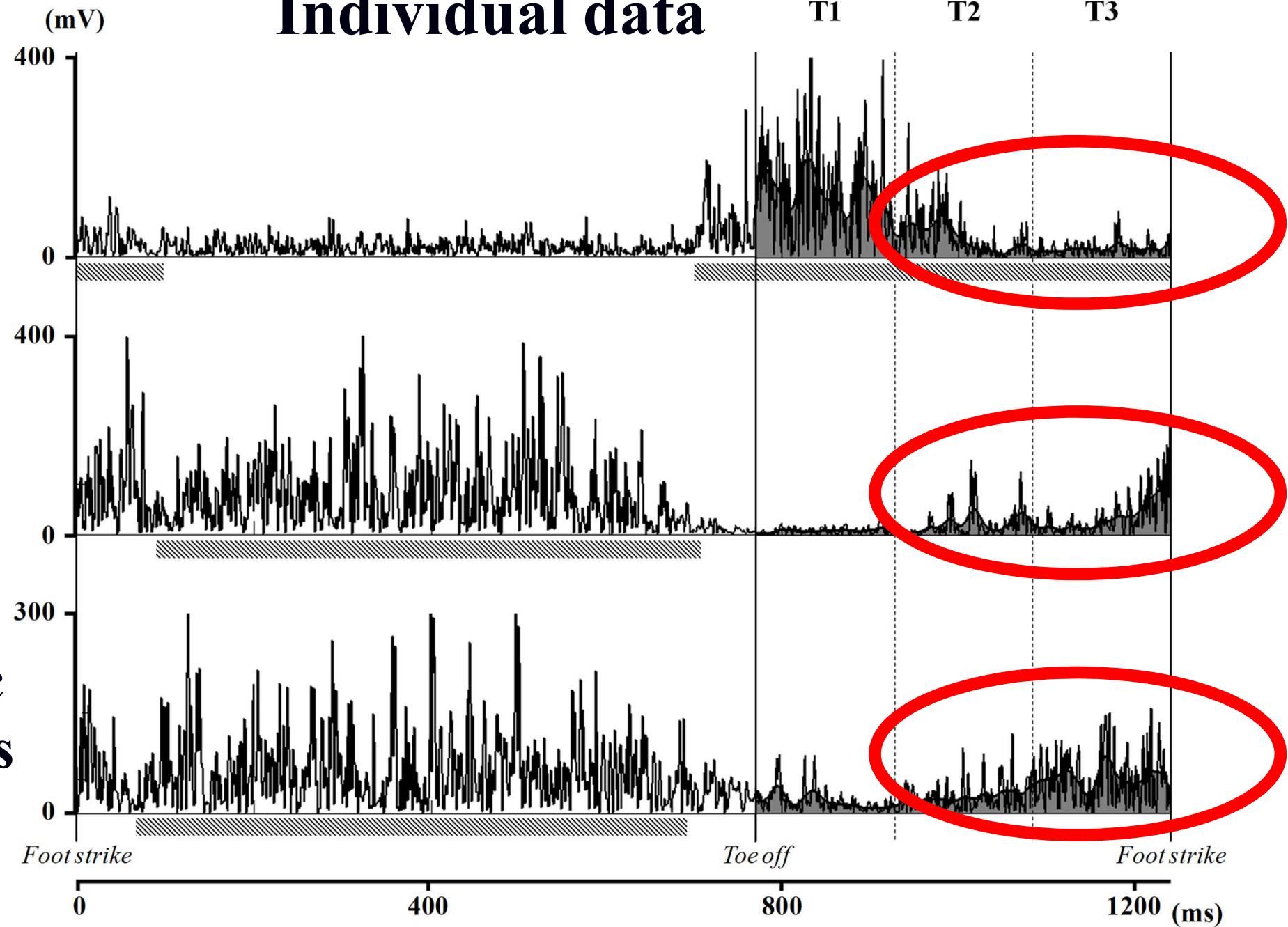
**A****Ankle position****B****Knee position****A****CAG<sub>TA</sub>**

- Non paretic
- Paretic

**Coeff agonist tib ant activation****A****B****CAN<sub>SO</sub>****Coeff antagonist soleus activation****B****C****CAN<sub>MG</sub>****Coeff antagonist gastroc activation****C**

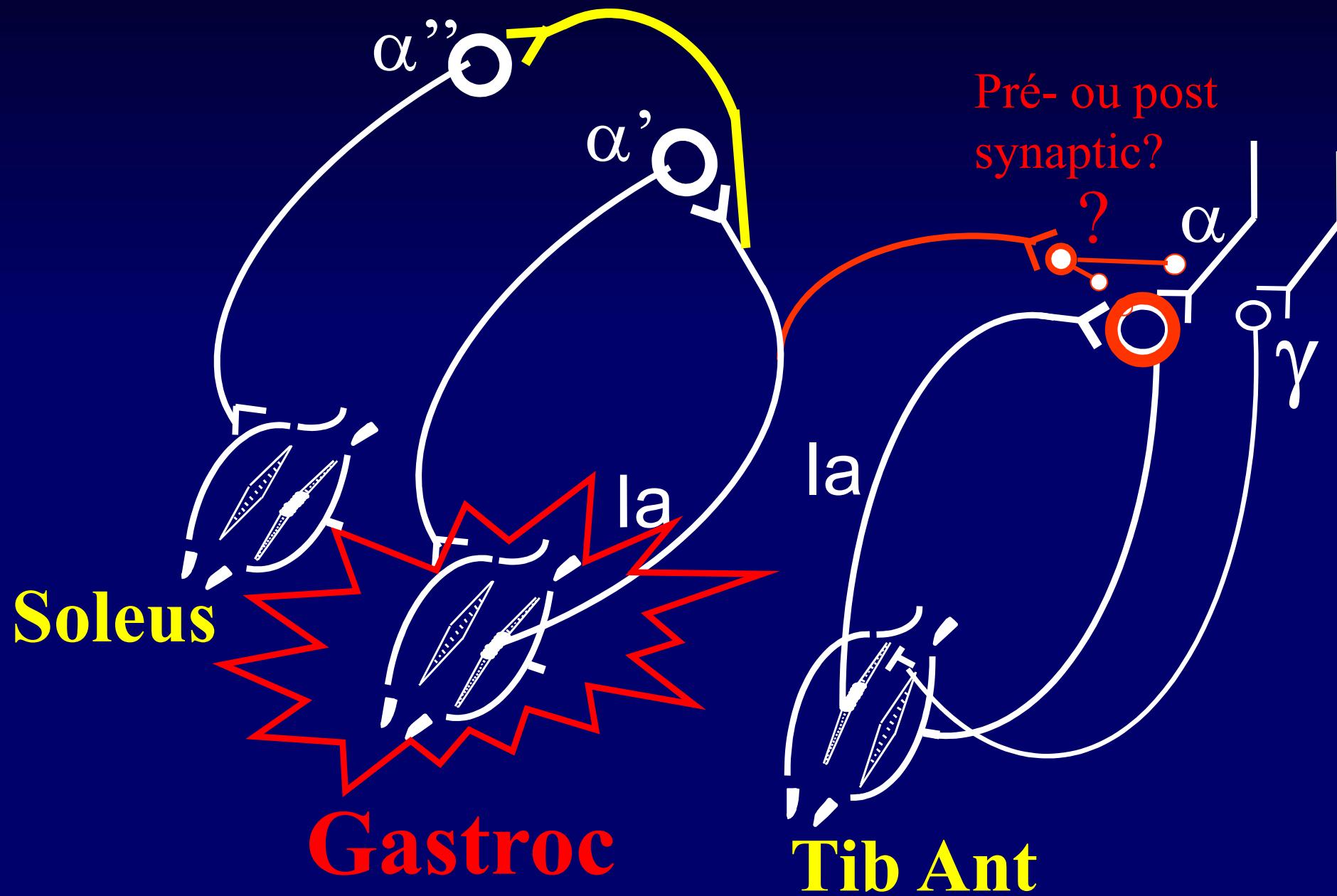
Tibialis  
anterior

# Individual data



Ghédira M, Albertsen IM, Mardale V, Loche CM, Vinti M, Gracies JM, Bayle N, Hutin E. Agonist and antagonist activation at the ankle monitored along the swing phase in hemiparetic gait. Clin Biomech (Bristol, Avon). 2021 Oct;89:105459

# Hypothesis for stretch-sensitive paresis and for spastic cocontraction



The Syndrome of  
Deforming Spastic Paresis  
=

*Spastic Myopathy*  
+ Spastic Cocontraction !

- Botulinum toxins ~ effective but crude way to take care of excessive muscle activations (only)
- Comprehensive approach? → Let us start by trying to take care of the *sick muscle*



*“It is often said, and rightly so, that science advances in fits, according to successes in the methodological field. ... This is why our most urgent task was the development of a Method.”*

*Ivan Petrovitch PAVLOV, 1897*  
*Conferences on the activity of the main digestive glands.*  
*I. Pavlov. Selected works, ed. Kh. Kochtoianz, Moscow, 1954. p.92*

2nd step

=

Maximal extensibility?

=

X<sub>V1</sub>

=

Slow and strong passive movement by clinician

# Five Step Assessment → Muscle disease?

Rectus femoris



Passive range of motion: X<sub>V1</sub>  
Expected value: X<sub>N</sub>

$$\text{Coefficient of Shortening} = (X_N - X_{V1})/X_N$$

*Gracies. Coefficients if impairment in spastic paresis.  
Ann Phys Med Rehabil 2015*

4th step

=

What can the  
agonist achieve  
against the tested  
antagonist?

=

X<sub>A</sub>

=

Active Movement  
by patient

## Five Step Assessment →Neurological disease?

Rectus  
femoris



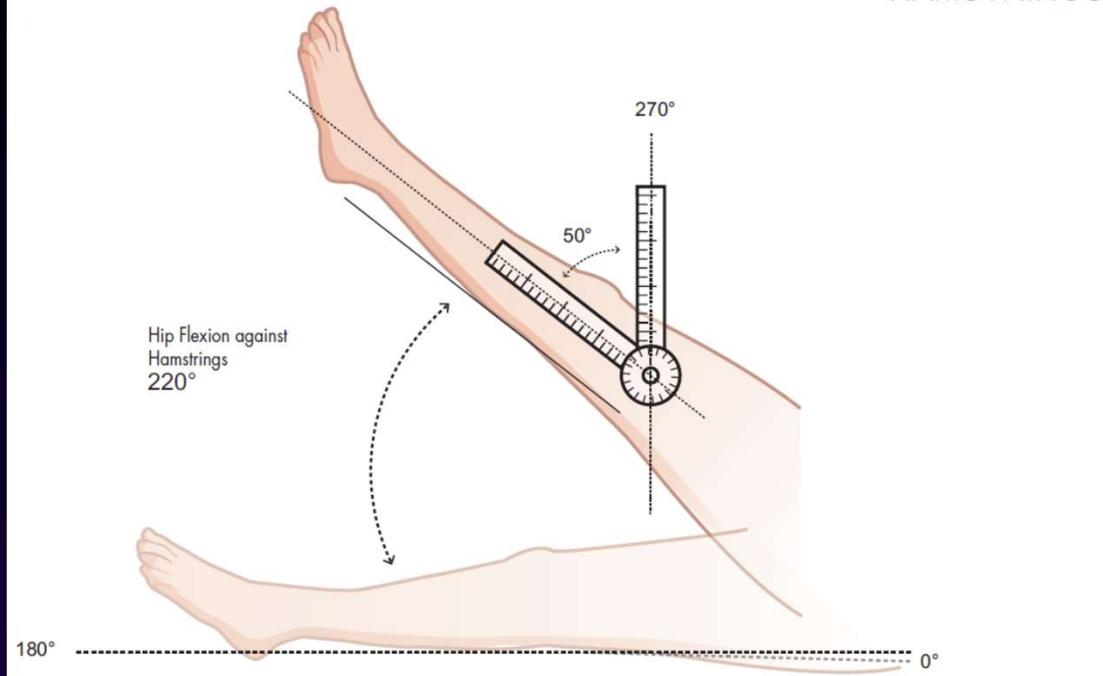
Maximal active ROM X<sub>A</sub>  
Maximal passive ROM X<sub>V1</sub>

$$\text{Coefficient of Weakness} = (X_{V1} - X_A) / X_{V1}$$

*Gracies. Coefficients of Impairment in Spastic Paresis.  
Ann Phys Med Rehabil 2015*

HAMSTRINGS

# Hamstrings X<sub>A</sub>



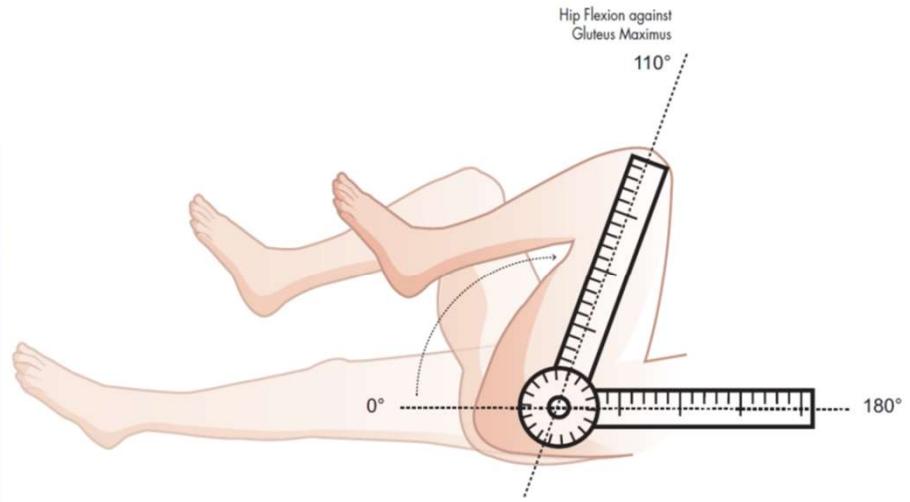
*Five Step  
Assessment  
→neurological  
disorder?*



# Rectus femoris X<sub>V1</sub>, X<sub>A</sub>



GLUTEUS MAXIMUS



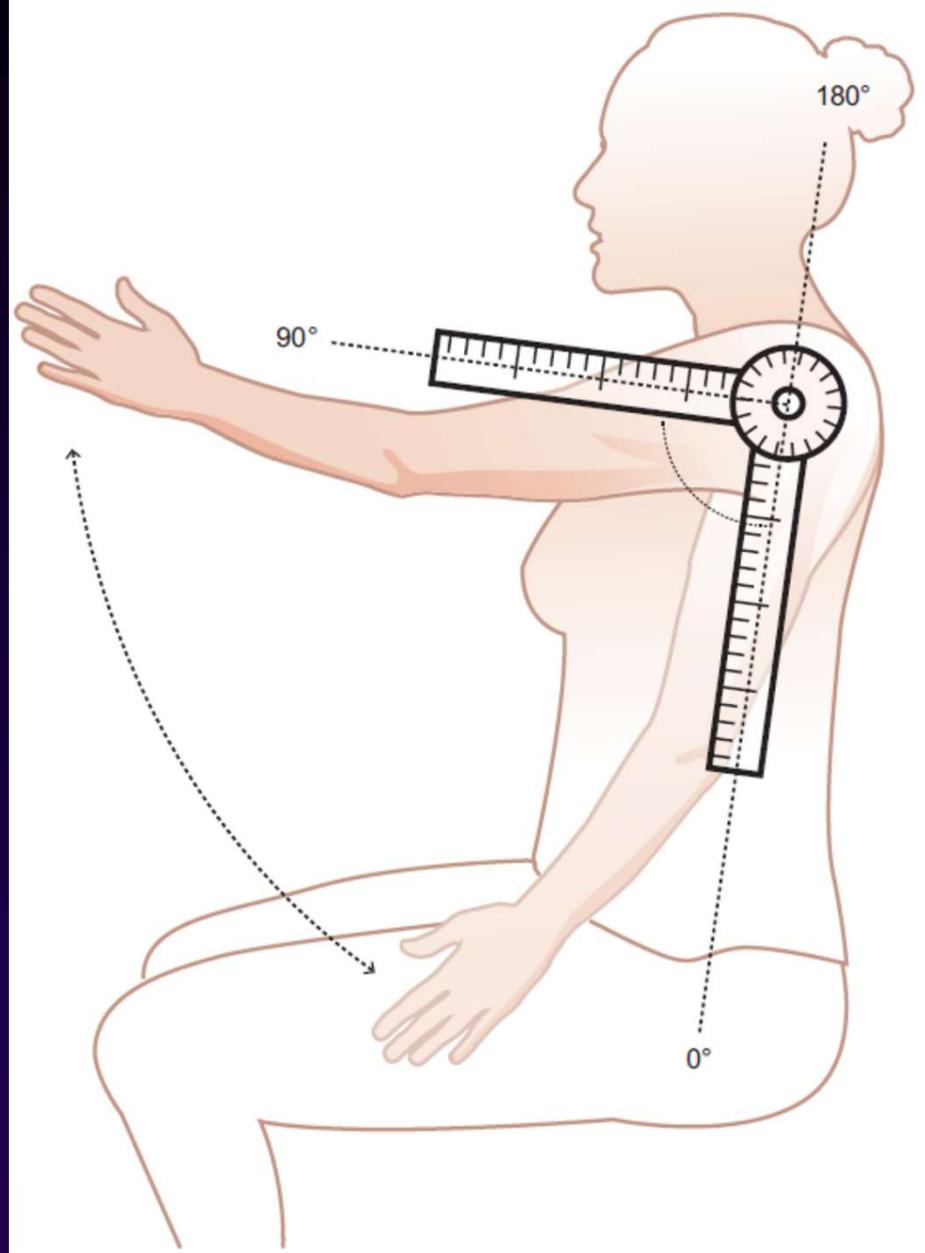
# Gluteus maximus, Gluteus maximus, $X_{V1}$ , $X_A$

## Five Step Assessment



$$\text{Coefficient of Weakness} = (X_{V1} - X_A) / X_{V1}$$

## SHOULDER EXTENSORS

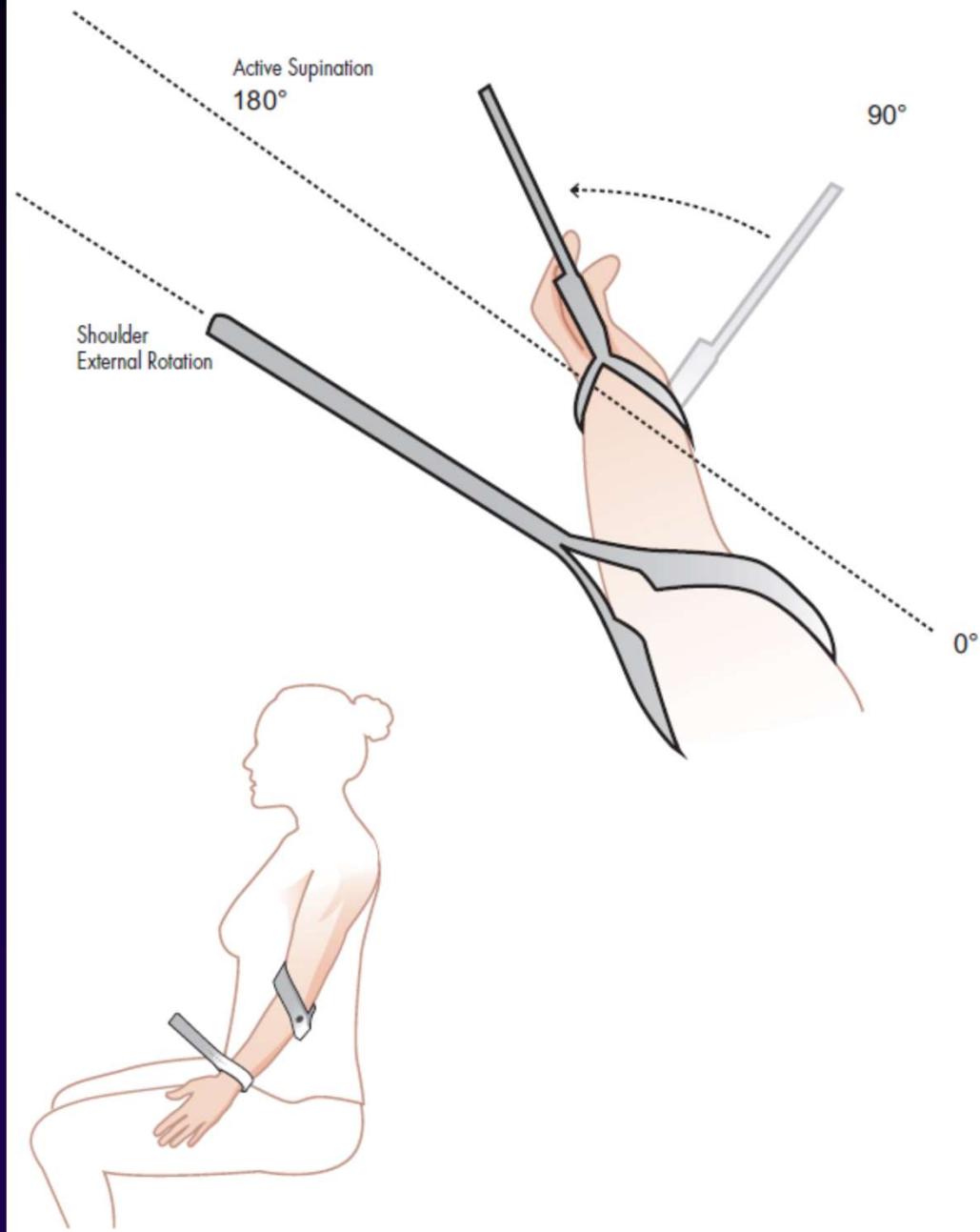


# Shoulder extensors



$$\text{Coefficient of Fatigability} = (X_A - X_{A15})/X_A$$

## PRONATOR TERES



# Pronator teres



# Influence maladie du muscle sur commande?

## Objectif :

Etudier relation extensibilité musculaire et association parésie/cocontraction

## Hypothèses :

1. Au-delà d'un certain seuil de perte d'extensibilité, influence myopathie spastique *sur* commande descendante
2. Au-delà d'un certain seuil de perte d'extensibilité, influence myopathie spastique sur fonction active

# Méthodes

## Critères de sélection

- Unique AVC,  $\geq 1$  an
- Pas de toxine < 3 mois
- 6 muscles testés
- Marche autonome

## Muscles évalués

6 Mb Sup - Inf

## Critères d'évaluation

$X_{V1}$  extensibilité

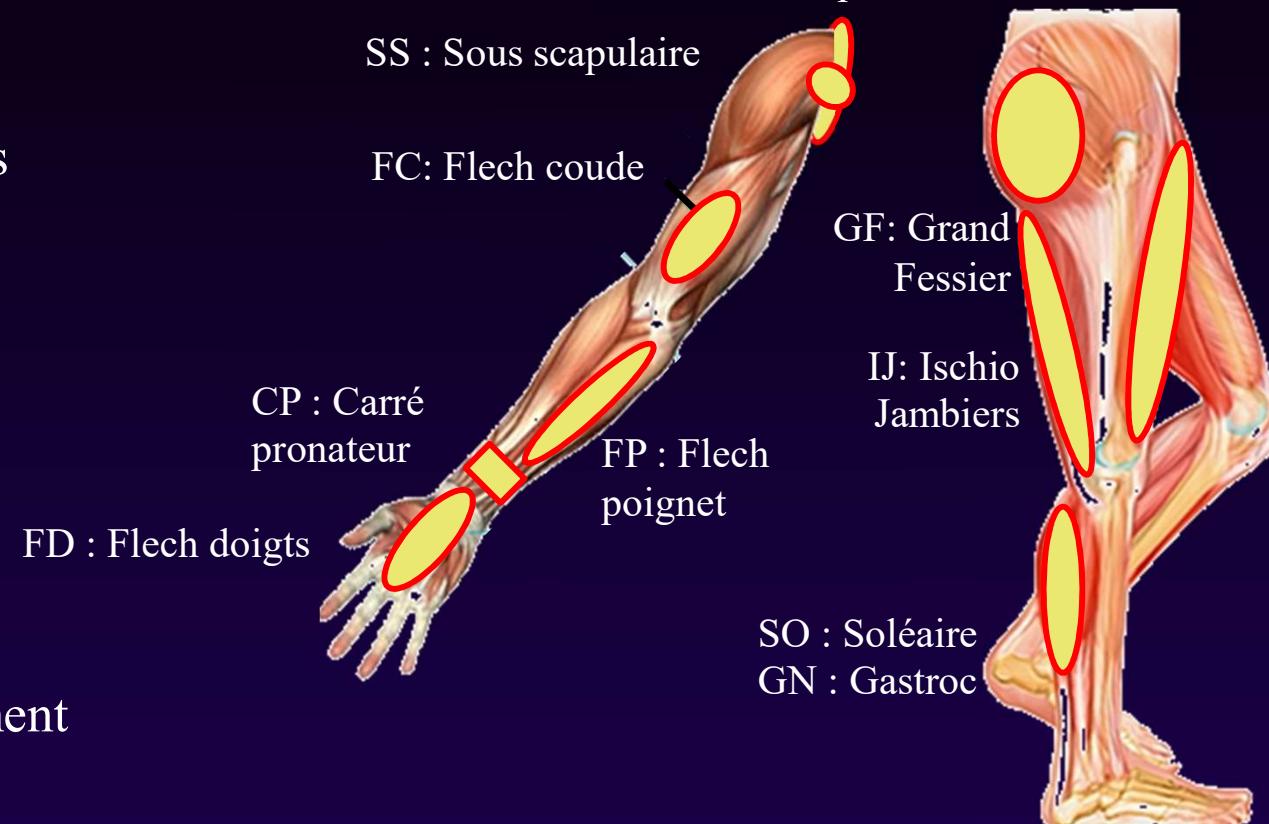
$X_A$  actif

CR coeff raccourcissement

CF coeff faiblesse

MFS (Frenchay)

Vitesse marche



## Traitement données Analyse statistique : sur 6 muscles et composite

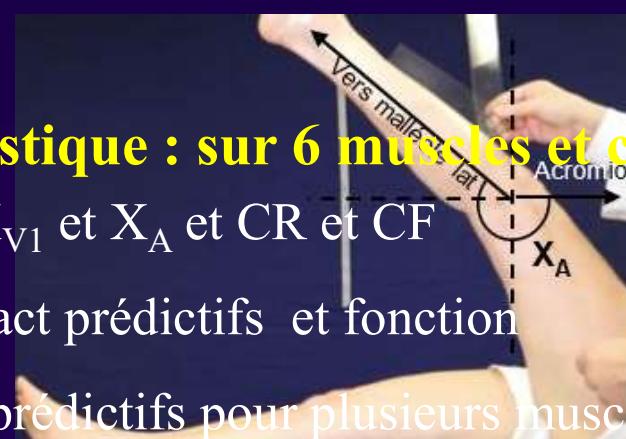
$X_N \rightarrow 180^\circ$

$X_{V1} X_A$  CR CF normalisés

1. Régression  $X_{V1}$  et  $X_A$  et CR et CF

2. CR et CF = fact prédictifs et fonction

3. CR ou CF = prédictifs pour plusieurs muscles : multivariable



## Clinical parameters

<b>Lower limb</b>	Soleus	Gastroc	Glut Max	Hamst	Vastus	Rect Fem	Comp score
X <sub>V1</sub>	153±9	149±6	155±15	168±9	137±7	156±12	153±7
X <sub>A</sub>	137±13	118±21	139±19	148±16	112±16	132±19	131±13

---

Ambul speed 0.88±0.39

---

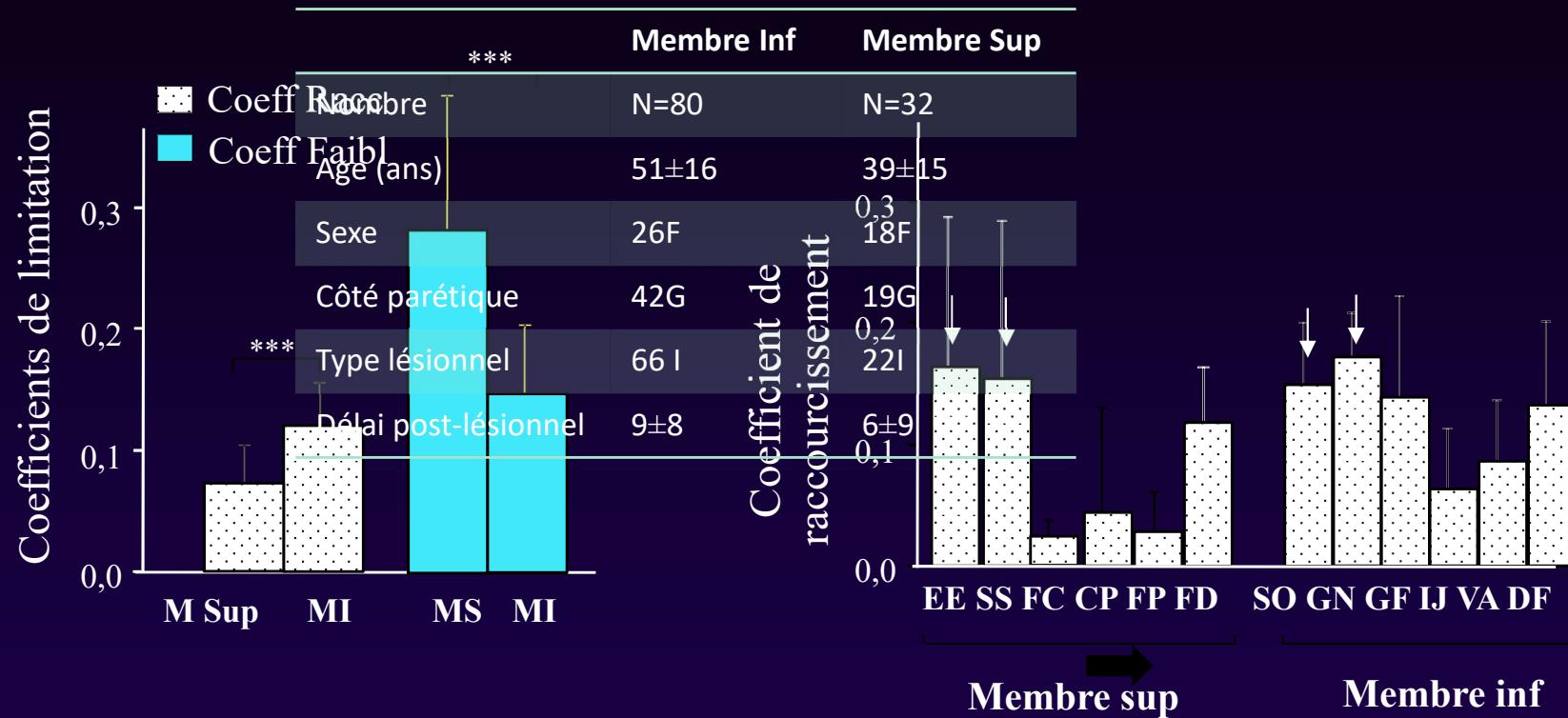
<b>Upper limb</b>	Sh Ext	Subscap	Elbow Flex	Pron Quad	Wrist Flex	Finger Flex	Comp score
X <sub>V1</sub>	150±22	152±23	177±4	177±16	175±6	176±5	169±7
X <sub>A</sub>	116±33	111±37	165±13	128±44	138±21	80±36	125±19

---

MFS 5.5±1.1

---

# Résultats descriptifs



- **Raccourcissement :**  
 $Mb\ Sup < Mb\ Inf$
- **Faiblesse :**  $Mb\ Sup > Mb\ Inf$

- **Raccourcissement :**
  - Extenseurs d'épaule
  - Sous-scapulaire
  - Gastrocnémiens
  - Soléaire

# Spastic Myopathy post Immobilisation = THERAPEUTIC OPTIONS

---

- Electrical stimulation, vibration, US, shock/short waves?
- Nutrition? → leucine (*Baptista et al, 2010*)?
- Pharmacological?

→ tétracyclines (effects on muscle and bone..)

→ water saturated with hydrogen (antioxydant)



Minimisation of immobilisation? Stretching?  
**Kelleher, 2015 = genetic reversibility of spastic myopathy**

STUDY PROTOCOL

Open Access



# Guided Self-rehabilitation Contract vs conventional therapy in chronic stroke-induced hemiparesis: NEURORESTORE, a multicenter randomized controlled trial

Jean-Michel Gracies<sup>1,2</sup>, Maud Pradines<sup>1,2</sup>, Mouna Ghédira<sup>1,2</sup>, Catherine-Marie Lache<sup>2</sup>, Valentina Mardale<sup>2</sup>, Catherine Hennegrave<sup>2</sup>, Caroline Gault-Colas<sup>2</sup>, Etienne Audureau<sup>3,4</sup>, Emille Hutin<sup>1,2</sup>, Marjolaine Baude<sup>1,2</sup>, Nicolas Bayle<sup>1,2</sup> and the Neurorestore Study Group

# Ultrasound Structural Changes in Triceps Surae After a 1-Year Daily Self-stretch Program: A Prospective Randomized Controlled Trial in Chronic Hemiparesis

Neurorehabilitation and Neural Repair  
1–15  
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[journals.sagepub.com/home/nnr](http://journals.sagepub.com/home/nnr)



Maud Pradines, PT, PhD<sup>1,2</sup>, Mouna Ghedira, PT, PhD<sup>1,2</sup>, Raphaël Portero, PhD<sup>1</sup>, Ingrid Masson, PhD<sup>1</sup>, Christina Marciak, MD<sup>3</sup>, Dawn Hicklin, PT<sup>4</sup>, Emilie Hutin, PhD<sup>1,2</sup>, Pierre Portero, PhD<sup>1</sup>, Jean-Michel Gracies, MD, PhD<sup>1,2</sup>, and Nicolas Bayle, MD<sup>1,2</sup>

# Self-stretching and structural muscle changes

Objectives : Assess structural changes and passive extensibility in triceps surae + function, following a guided self-stretching program

Inclusion : 1<sup>st</sup> stroke > 1 year ; comfortable barefoot walking speed >0.1 et <1.2m/sec



Ancillary to *Neurorestore*

# Etude prospective randomisée contrôlée en simple insu – ancillaire PHRC Neurorestore

Recrutement préliminaire: sujets adultes atteints d'hémiplégie chronique observés au centre Hospitalier Mondor (n=31)

Patients non inclus (n=8):  
- >1 AVC ou hémiplégie sur autre cause (n=5)  
- Troubles cognitifs sévères (n=3)

J1: Evaluation biomécanique/clinique + Randomisation (n=23)

Rééducation conventionnelle (CONV)  
(n=11)

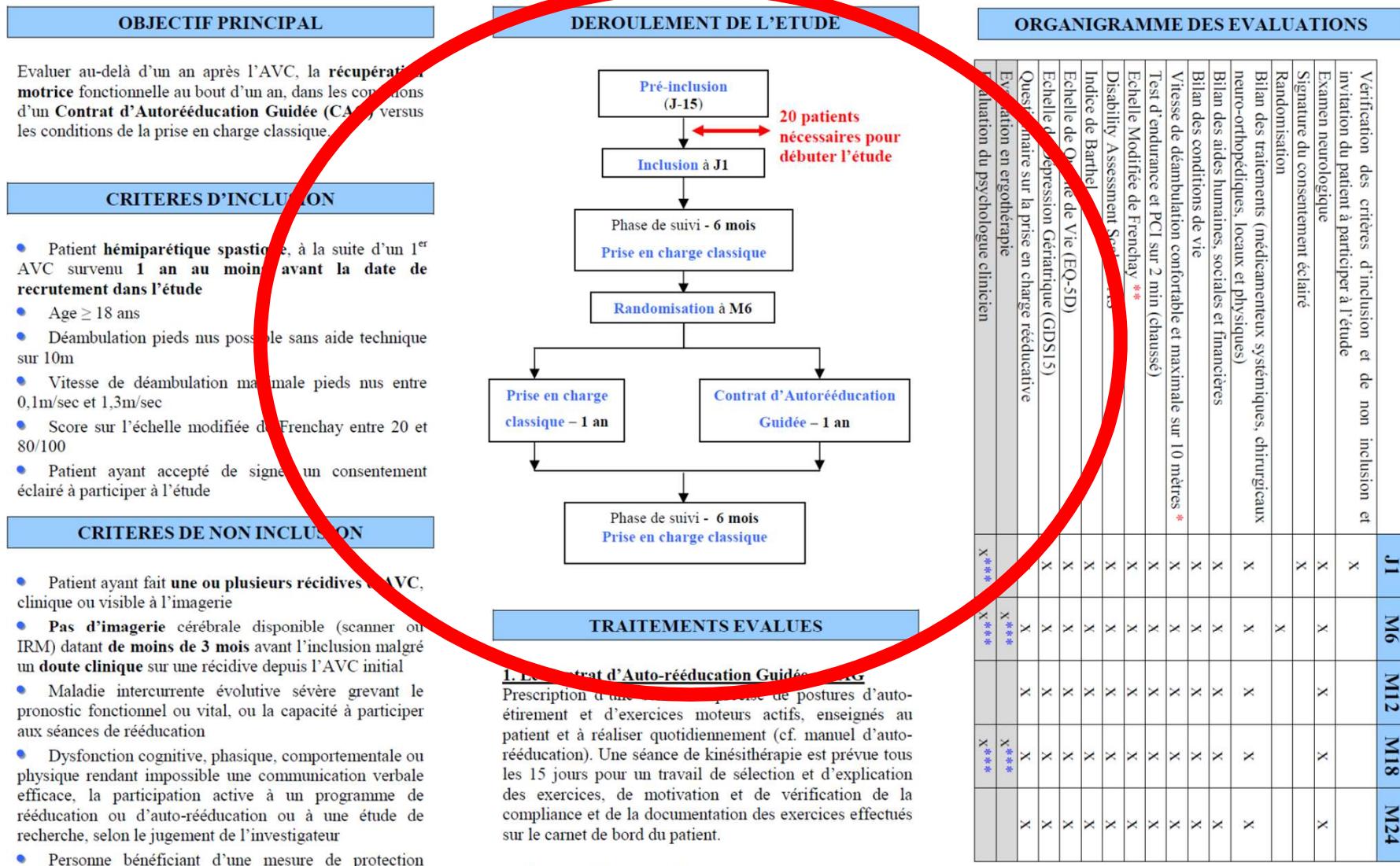
Contrat d'Autoreéducation Guidée (CAG) + Réeduc conv. (n=12)

M12: Analyse biomécanique / Evaluation clinique (n=23)

Toxine botulique autorisée

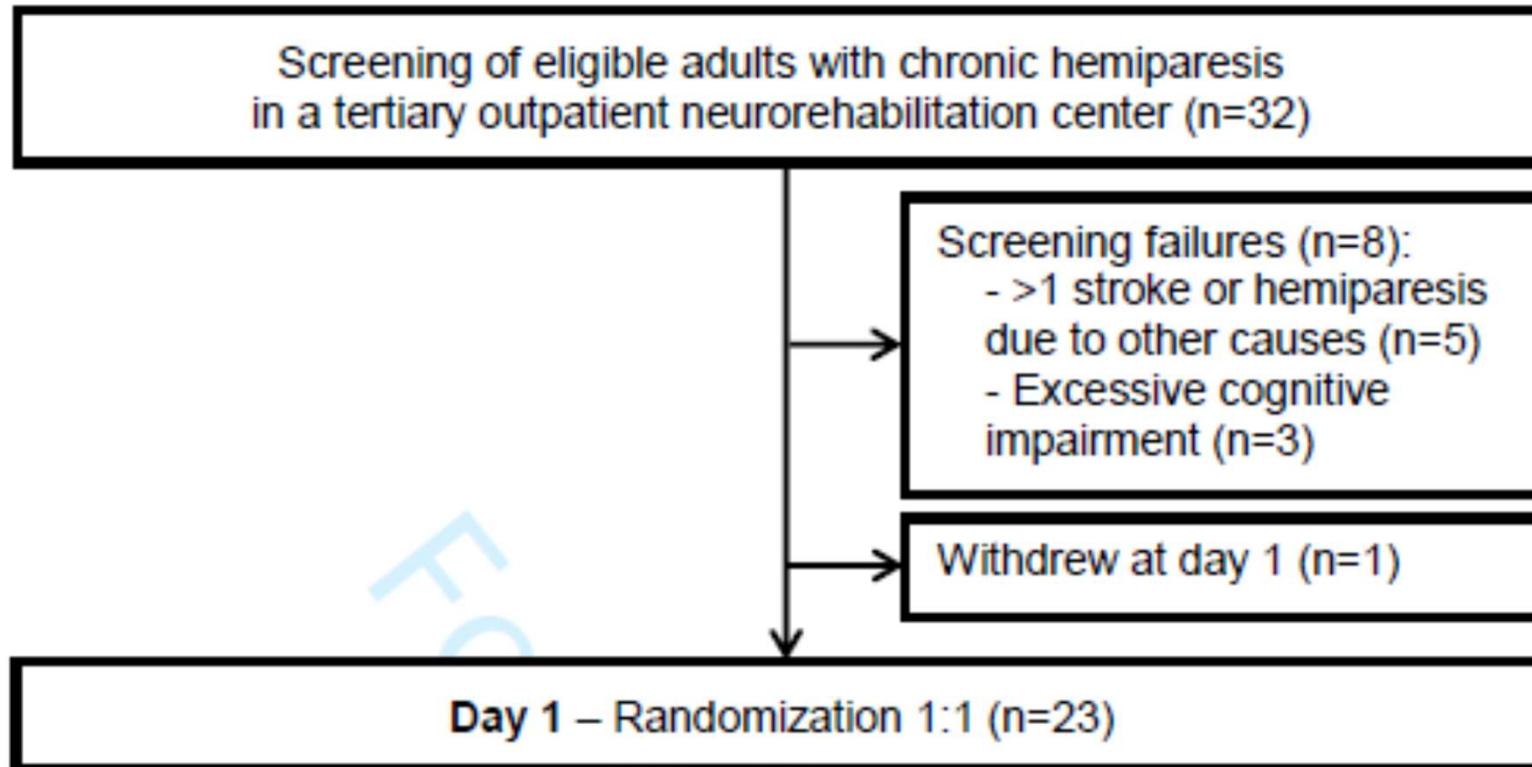
# Publicly funded project *Neurorestore*

## HU Mondor



\* Critère d'évaluation principal pour le membre inférieur  
\*\* Critère d'évaluation principal pour le membre supérieur  
\*\*\* Seulement pour les patients H. Mondor et H. Valdarni

# Publicly funded *Neurorestore* project - Flow chart



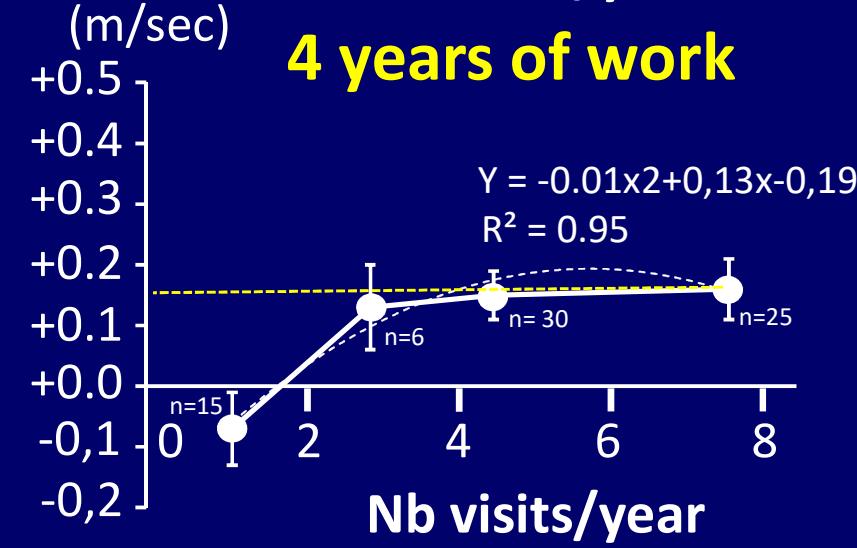
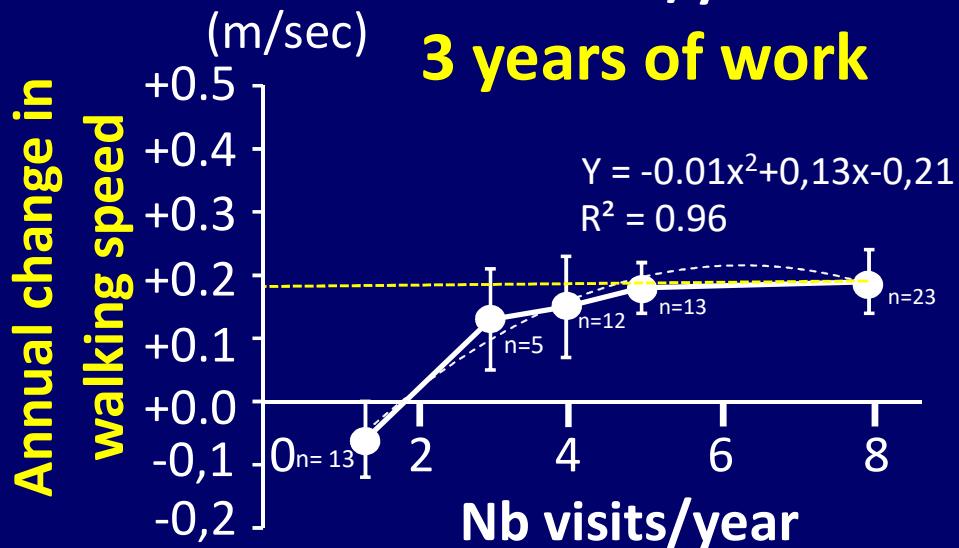
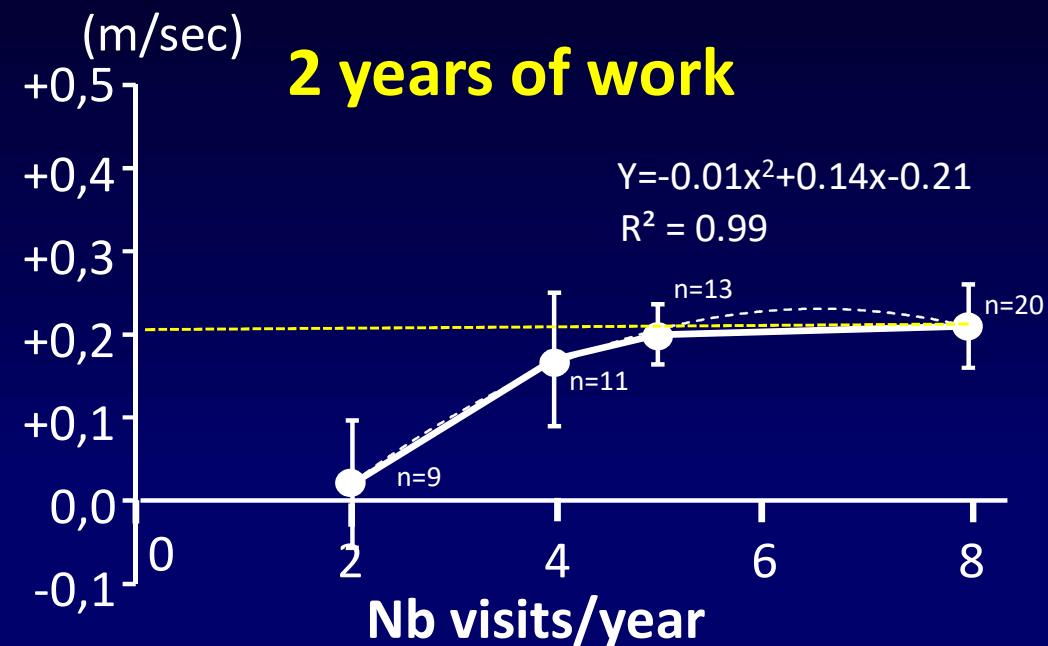
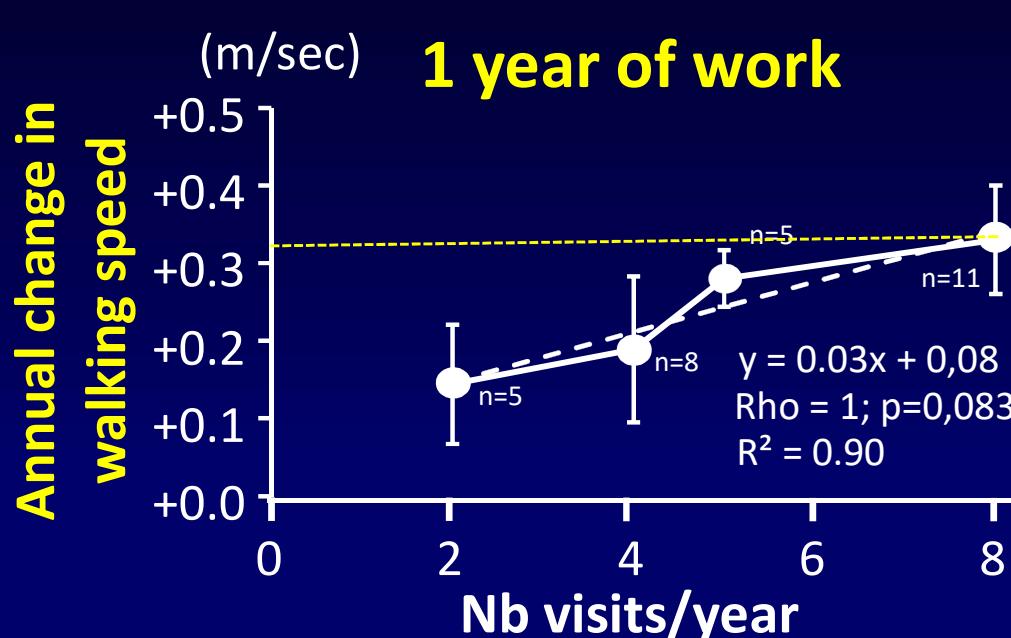
*Pradines et al.*  
*Neurorehabil*  
*Neural Repair.*  
2019;33(4):245  
-259

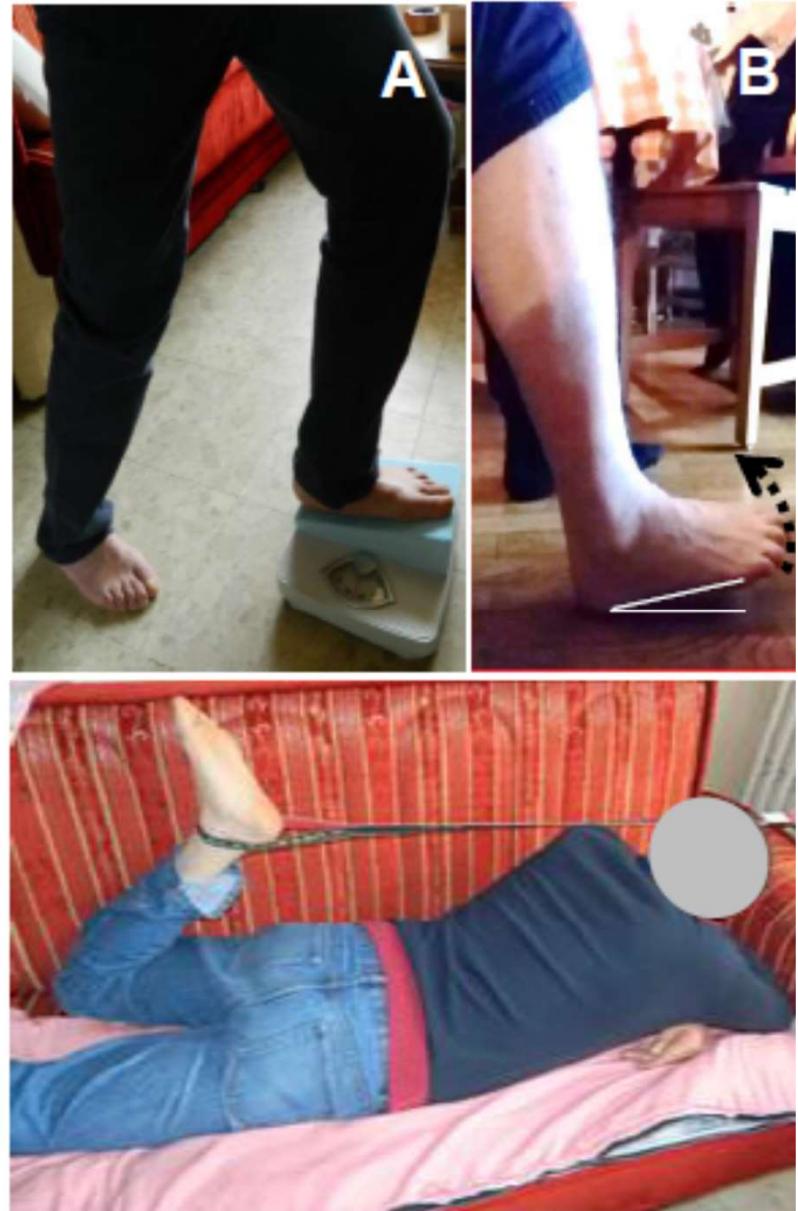
# Description of the two groups

- **CONV group**: conventional community-based therapy sessions, based upon prescription by physician and patient requests
- **GSC group**: conventional sessions allowed, plus one visit every other week by study therapist to prescribe the program, teach self-stretching techniques on specific muscles and verify diaries
- **In both groups**: local treatment with BoNT (+/- systemic) allowed

# *Therapeutic frequency in neurorehabilitation*

## Annual gain in walking speed vs number of clinic visits/year





*Pradines et al. Neurorehabil Neural Repair. 2019;33(4):245-259*



# Quantified Diary++++

Catherine		20 mn Etirement Grand Fessier	Flexion/mn hanche genou plié	20 mn Etirement Ischio jambier	Flexion/mn hanche genou droit	15 mn Etirement Droit antérieur	Flexion/mn genou fesse	Soléaire	Moment	Divers	relevés de Pied
jeudi	26/01/12	20	3X21	20	3X23	10	3X26				
vendredi	27/01/12										
samedi	28/01/12	20	3X21	20	3X24	10	3X27				
dimanche	29/01/12										
lundi	30/01/12										3X17
mardi	31/01/12	20	3X20	20	3X25	10	3X26				3X19
mercredi	01/02/12										
jeudi	02/02/12	20	3X21	20	3X24	10	3X26				3X19
vendredi	03/02/12										
samedi	04/02/12	20	3x20	20	3X23	10	3X25				3X19
dimanche	05/02/12										4X17
lundi	06/02/12										3X17
mardi	07/02/12	20	3X21	20	3X24	10	3X26				3X17
mercredi	08/02/12										3X17
jeudi	09/02/12	20	3X21	20	3X24	10	3X26				3X17
vendredi	10/02/12										
samedi	11/02/12	20	3X21	20	3X23	10	3X26				3X17
dimanche	12/02/12										3X17
lundi	13/02/12										
mardi	14/02/12	20	3X19	20	3X23	10	3X26				3X15
mercredi	15/02/12										3X18
jeudi	16/02/12	20	3X21	20	3X23	10	3X26				3X17
vendredi	17/02/12										3X18
samedi	18/02/12	20	3X19	20	3X23	10	3X26				3X18
dimanche	19/02/12	20	3X21	20	3X24	10	3X26				3X17
lundi	20/02/12										3X19
mardi	21/02/12	20	3X19	20	3X24	10	3X26				3X19
mercredi	22/02/12										3X20
jeudi	23/02/12	20	3X20	20	3X24	10	3X26				3X19
vendredi	24/02/12										3X20
samedi	25/02/12										3X20
dimanche	26/02/12	20	3X21	20	3X23	10	3X26				3X21

Muscles	Dates	Nombre de minutes d'étirements					
		20/08	21/08	22/08	23/08	24/08	25/08
Grand pectoral (éche 17)	<i>E</i> <i>X+</i>	3m-3m 3m-2m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-5m 3m-3m 3m-3m 3m
Grand dorsal et long chef du triceps (éche 18)	<i>E</i> <i>X+</i>	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m
Sous-scapulaire (éche 19)	<i>E</i> <i>S+</i>	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m
Fléchisseurs du coude (éche 20)	<i>E</i> <i>X+</i>	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m
Carré pronateur (éche 21)							
Rond pronateur (éche 21)	<i>E</i> <i>X+</i>	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m	3m-3m 3m-3m 3m-3m 3m-3m 3m
Fléchisseurs du poignet (éche 22)							
Fléchisseurs du pouce (éche 23)	<i>E</i> <i>S+</i>	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m
Fléchisseurs des II <sup>e</sup> et III <sup>e</sup> doigts (éche 24)	<i>E</i> <i>X+</i> <i>m</i>	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m
Fléchisseurs des IV <sup>e</sup> et V <sup>e</sup> doigts (éche 25)	<i>E</i> <i>X+</i> <i>m</i>	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m	5m-5m 5m
Interosseux (éche 26)							

**Etirement  
Fléchisseurs du coude**

(1)

**Objectif :** Mieux tendre le bras.



**Posture d'étirement** Assis les jambes croisées, placer le coude sur la cuisse, en se penchant un peu en avant. Saisir le poignet avec l'autre main, paume vers le bas, pour tendre le coude le plus possible.

**Attention :** Bien placer le coude sur la cuisse.

**Sensation** Tension non douloureuse dans le bras, le coude et l'avant-bras.

**Fréquence** Maintenir l'étirement

> 5 minutes, 2 fois par jour.

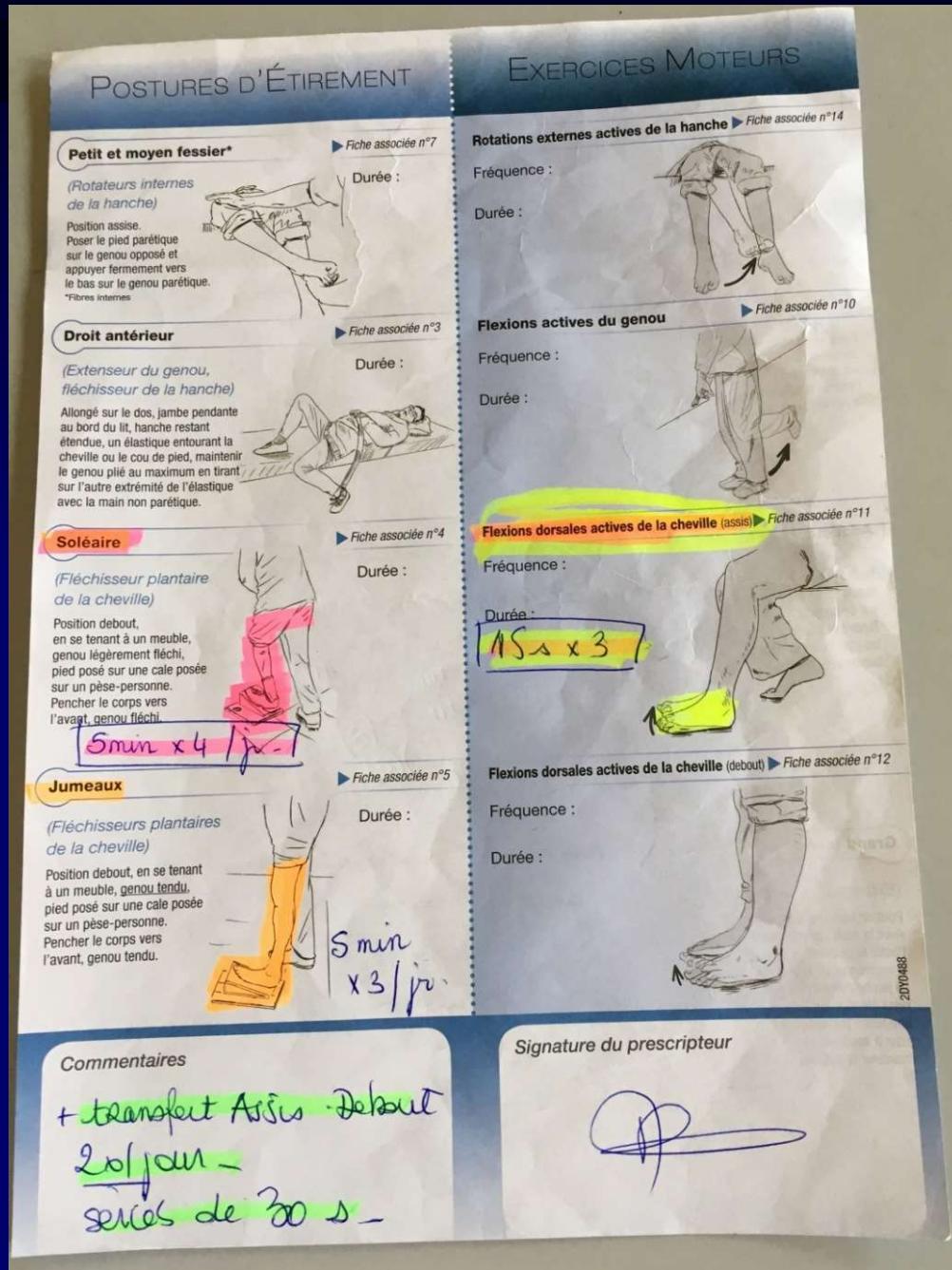
5 dakita Günde 2 defa



Equipe de rééducateurs du Service de MPR du Pr Gracies  
Hôpital Albert Chenevier - Créteil (94)

Version 1 - Questions et remarques : eleonore.lamour@ach.aphp.fr

Tarih	dakika	Günde kaç defa
18. 6. 15	S - Min	2
19. 6. 15	S	2
20. 6. 15	S	2
21. 6. 15	S	3
22. 6. 15	S	3
23. 6. 15	S	3
24. 6. 15	S	3
25. 6. 15	S	3
26. 6. 15	S	3
27. 6. 15	S	4
28. 6. 15	S	4
29. 6. 15	S	4
30. 6. 15	S	2
01. 7. 15	S	2
02. 7. 15	S	2
03. 7. 15	S	3
04. 7. 15	S	4
05. 7. 15	S	4
06. 7. 15	S	1
07. 7. 15	S	3
08. 7. 15	S	3
09. 7. 15	S	3
10. 7. 15	S	4
11. 7. 15	S	4
12. 7. 15	S	2
13. 7. 15	S	3
14. 7. 15	S	3
15. 7. 15	S	2
16. 7. 15	S	3
17. 7. 15	S	2
18. 7. 15	S	2
19. 7. 15	S	2
20. 7. 15	S	2
21. 7. 15	S	2
22. 7. 15	S	2
23. 7. 15	S	2
24. 7. 15	S	4



**Programme Membre Inférieur**

Nombre de minutes d'étirement	
Ischio-jambiers	20/10/11/12/13/14/15/16/17/18/19/110/111
Adducteurs de hanche	
Grand fessier	
Droit antérieur	
<b>Soleaire (indiquer aussi nb de kgs)</b> 40kg (5)	11/11/11/11/11/11/11/11/11/11/11/11/11/11
<b>Jumeaux (indiquer aussi nb de kgs)</b> 20kg (5)	11/11/11/11/11/11/11/11/11/11/11/11/11/11
Nombre maximal de mouvements en 1 minute (étirements avant et après)	
Flexions hanche genou fléchi (sol -main)	
Flexions hanche genou tendu (sol -main)	
Abductions hanche (sol -main)	
Flexions genou en arrière (talon - fesse)	
<b>Flexions dorsales cheville assis (relevés de pied)</b>	11/11/11/11/11/11/11/11/11/11/11/11/11
Flexions dorsales cheville debout (relevés de pied)	
Assis - debout	11/11/11/11/11/11/11/11/11/11/11/11/11
Comptage des pas sur distance spécifique	

Service de MPR, Neuroéducation - Hôpital Albert Chenevier - Henni Mondor

2D70488



# Biomechanical assessment

*Ultrasound  
diagnostic  
scanner model  
EZU-MT24-S1  
(Hitachi)*

Calibrated image  
Frequency 13Mhz  
Depth 50mm

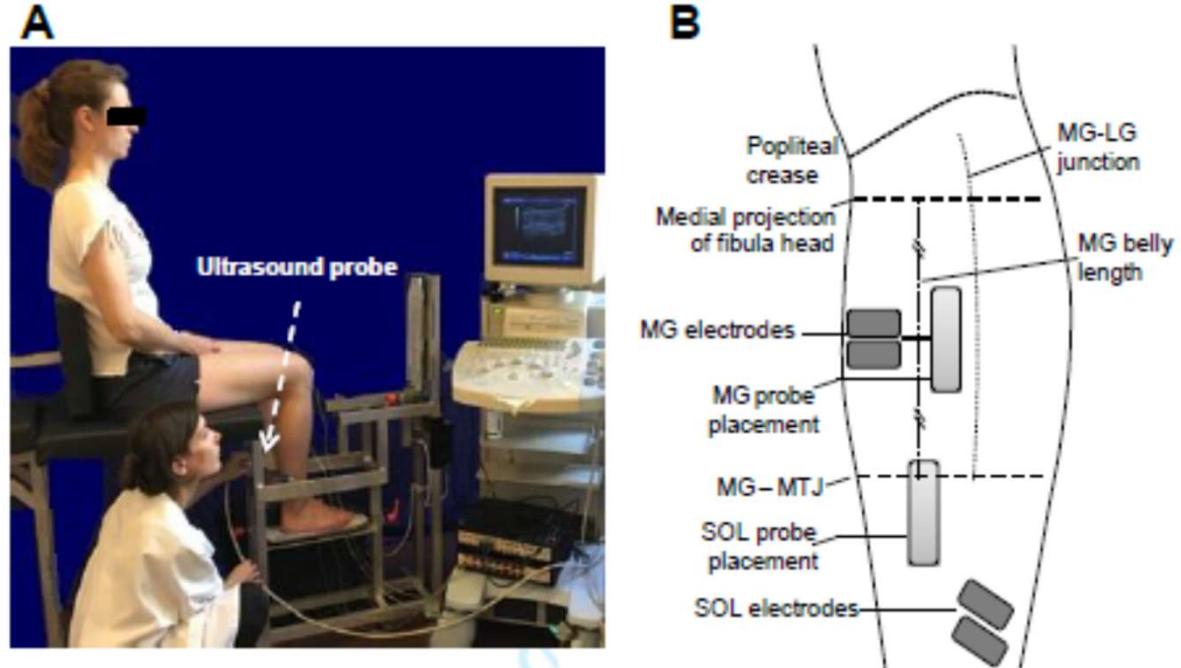


g-BS amp g-tech  
Surface  
EMG

Ankle ergometer with two force platforms (*Techno Concept*)

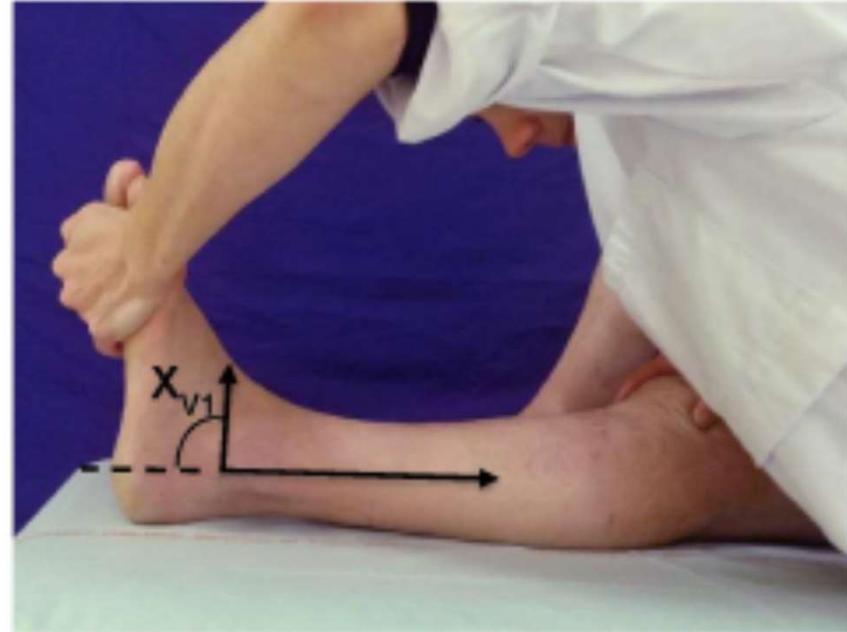
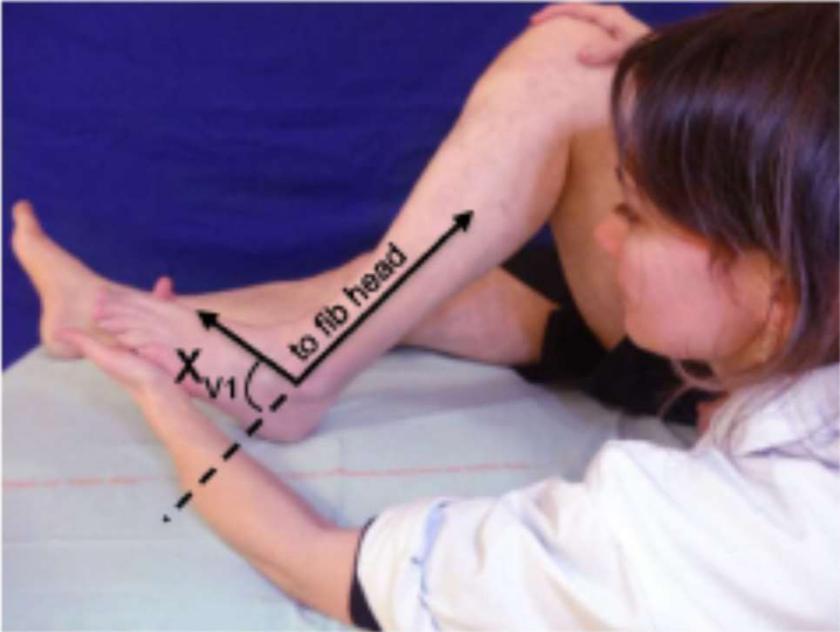
*Pradines et al. Neurorehabil Neural Repair. 2019;33(4):245-259*

# Methods



*Pradines et al.  
Neurorehabil Neural  
Repair:  
2019;33(4):245-259*

# Clinical assessment



$$X_{v1} \text{ Composite} = (X_{v1\text{GAS}} + X_{v1\text{SOL}} + X_{v1\text{GM}} + X_{v1\text{RF}}) / 4$$

# At baseline..

<b><i>Subject characteristics</i></b>	<b>CONV (n=11)</b>	<b>GSC (n=12)</b>
Age (years)	55±13	57±11
Time since lesion (years)	8±5	10±9
Gender	8M	5M
Paretic side	6R	6R
Lesion type	8I	8I

**Healthy subjects**  
43 mm  
40 mm  
15.5 mm  
17 mm

*Gao et al, Arch Phys Med Rehabil 2009; Zhao, Appl Physiol 2015; Simpson CL et al, Scand J Med Sci Sports. 2017; Maganaris et al, J; Bolsterlee B... Gandevia SC, Herbert RD. J Biomech. 2015;48(6):1133-40*

**A****SOLEUS**

CONV					GSC				
n	X <sub>V1</sub>	X <sub>V3</sub>	X	Y	n	X <sub>V1</sub>	X <sub>V3</sub>	X	Y
1	95	80	15	2	1	97	85	12	2
2	100	90	10	2	2	105	90	15	2
3	95	90	5	2	3	100	90	10	2
4	90	80	10	2	4	108	90	18	2
5	98	75	23	3	5	100	80	20	3
6	110	85	25	3	6	95	90	5	2
7	110	85	25	2	7	95	85	10	2
8	95	85	10	2	8	100	90	10	2
9	97	90	7	2	9	110	80	30	2
10	105	90	15	2	10	110	90	20	2
11	105	85	20	2	11	90	80	10	2
					12	95	75	20	3
Mean	100.0	85.0	15.0		Mean	100.4	85.4	15.0	
SD	6.6	5.0	7.3		SD	6.5	5.4	6.9	

		Initial (D1)			Final (M12)			
Group	n	Muscle injected	Toxin	Dose	Delay inj-assessment	Toxin	Dose	Delay inj-assessment
CONV	1	Gastroc.	abo	300U	1 day	abo	300U	1.5 month
		Soleus	abo	200U	1 day	abo	200U	
	2	Gastroc.				abo	300U	3 months
	3	Rect Fem				abo	300U	8 months
GSC	1	Gastroc.				abo	200U	6 months
		Soleus				abo	300U	
	2	Gastroc.				mcg	25U	7.5 months
		Tib.Post.				mcg	75U	
		Soleus				mcg	100U	
	3	Gastroc.				abo	200U	7 months
		Soleus				abo	100U	
	4	Rect Fem	abo	300U	3 months	abo	300U	0.5 month

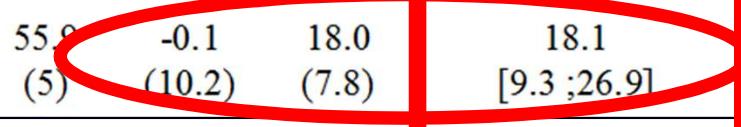
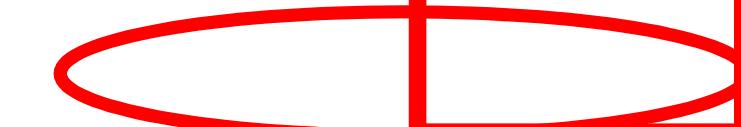


# **Descriptive results: treatments**

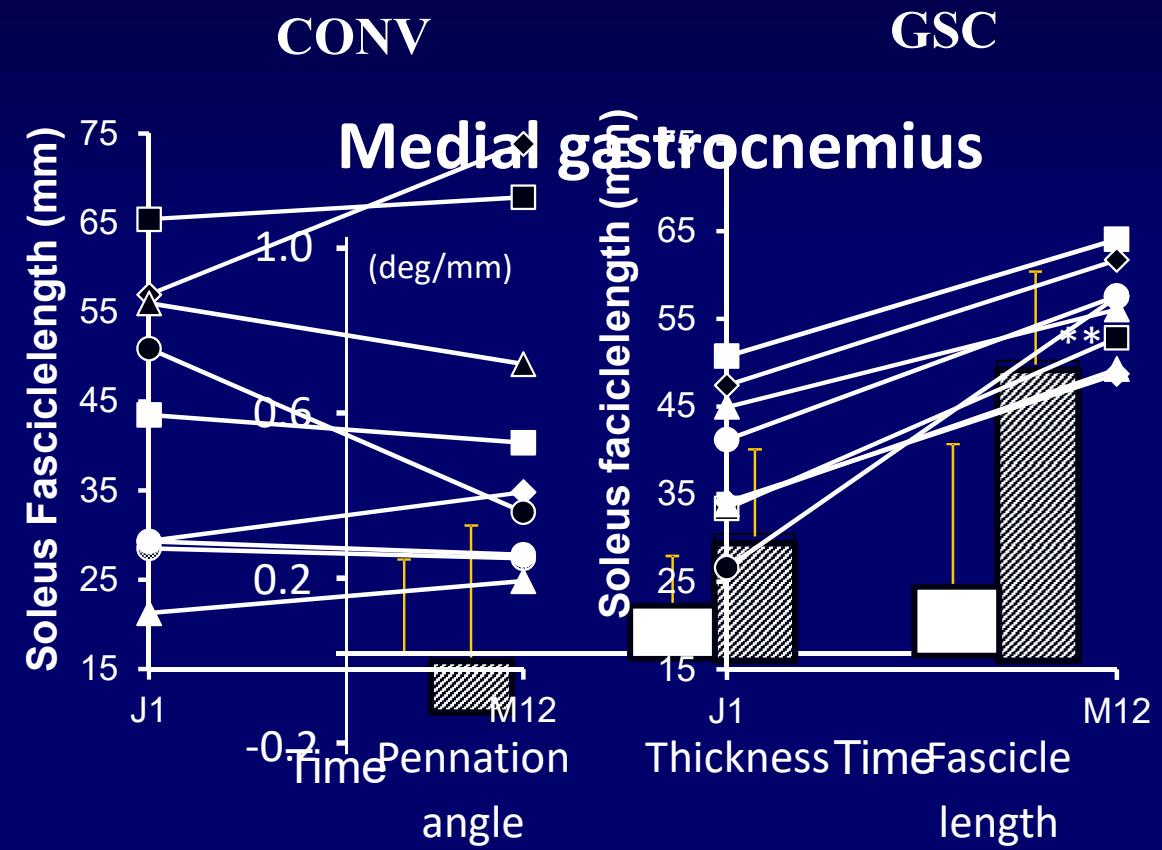
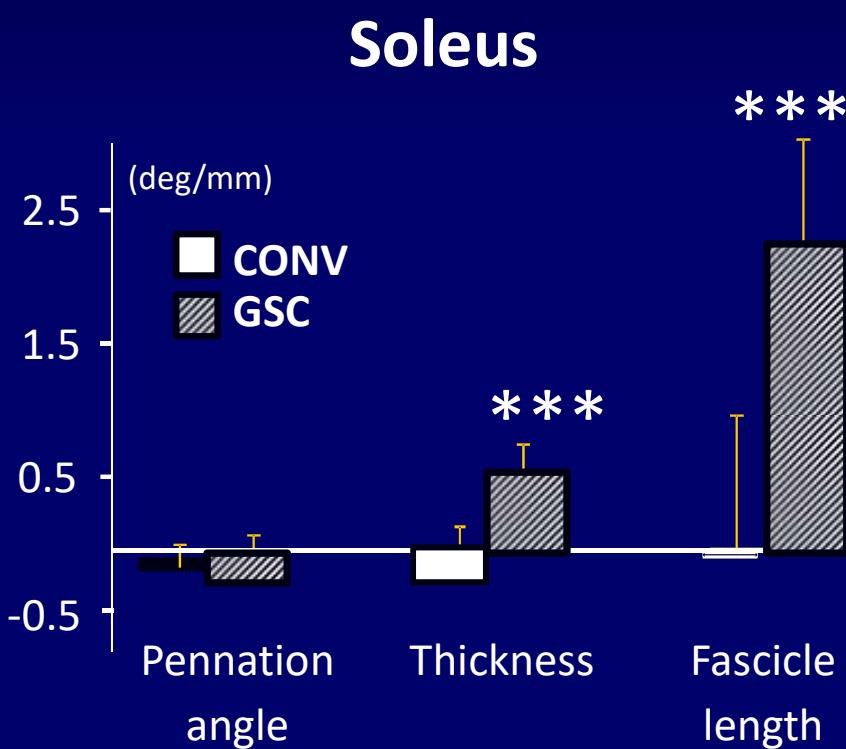
- **Mean time of conventional therapy:**
  - CONV:  $81.8 \pm 55$  min (1h30)/week = 12 min/d
  - GSC:  $57.8 \pm 37.5$  min (1h)/week = 8 min/d
- **GSC: mean reported daily time of self-stretch/muscle:**
  - Soleus:  $5.0 \pm 3.3$  min/d
  - Gastrocs:  $5.0 \pm 2.1$  min/d
  - Glut Max:  $6.3 \pm 3.2$  min/d
  - Rectus fem:  $8.4 \pm 3.9$  min/d
- **BoNT injections:**
  - Triceps surae: CONV n=2, GSC n=3
  - Rect Fem: CONV n=1, GSC n=1

# Outcomes

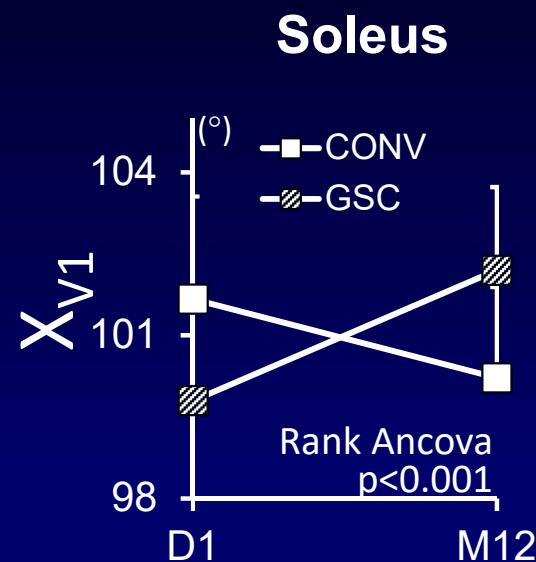
	Sample size CONV GSC	Groups		Within groups		Differences		Effect size Cohen's (d)
		Week 0		Week 52		Week 52 - Week 0	Week 52 - Week 0	
		CONV	GSC	CONV	GSC	CONV	GSC	GSC-CONV
<b>Muscle architecture (mm)</b>								
Fascicle length - soleus	n=9 n=8	40 (16)	37.9 (9.7)	39.9 (18.4)	55.9 (5)	-0.1 (10.2)	18.0 (7.8)	18.1 [9.3 ;26.9] [1.54;2.46]

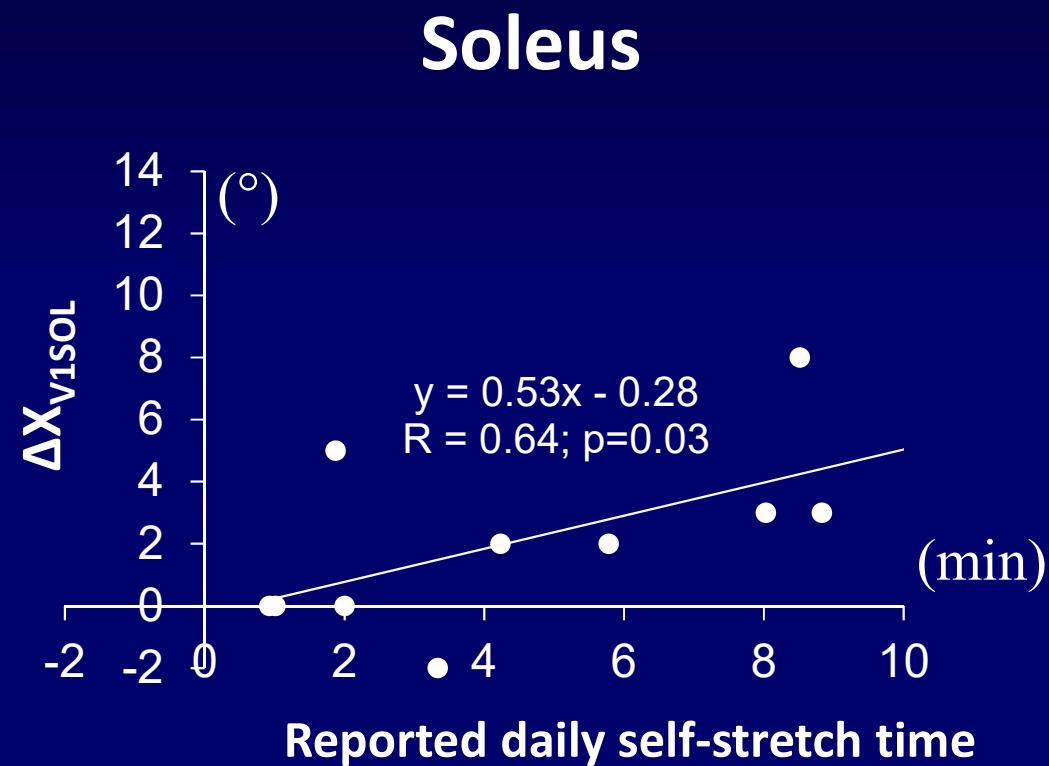
# Outcomes: muscle ultrasonography



# Clinical outcomes

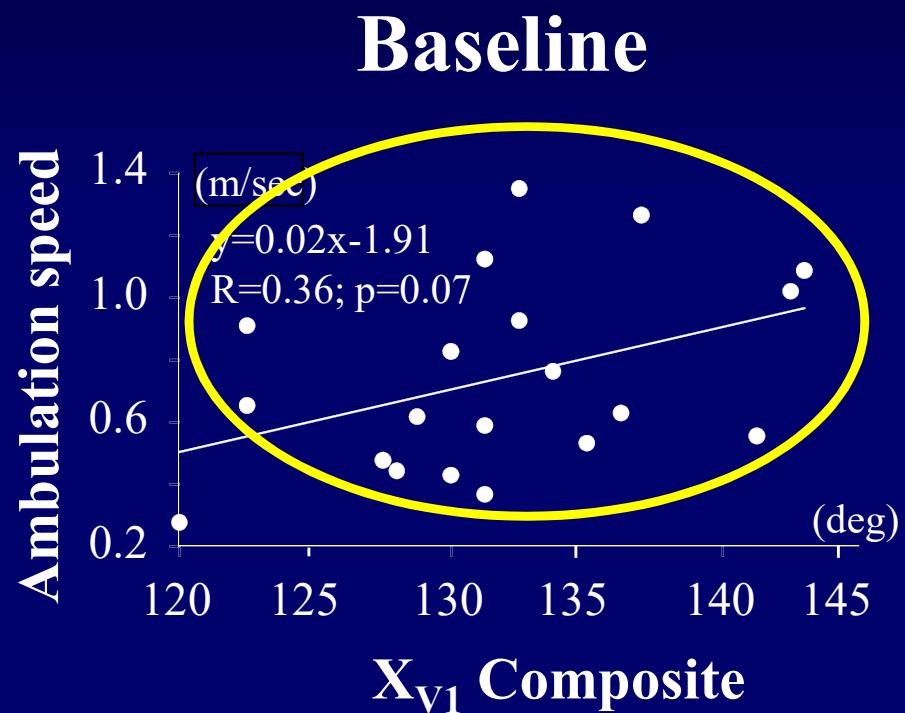


# Relation self-stretch time – extensibility changes



$\Delta X_{V1GF} \rightarrow \text{NS}$   
 $\Delta X_{V1DA} \rightarrow \text{NS}$

# Relation extensibility – function



# Discussion

## Limitations

- Sample size / visibility of fascicles in paretic subjects
- Position in ergometer (soleus pre-tension, not GM)
- Two types of stretching techniques

**Conclusion:** One year prospective randomized controlled trial in chronic spastic paresis: a daily self-stretching program within Guided Self-rehabilitation Contracts (GSC):

- fascicle length
  - muscle extensibility
  - ambulation speed
- } Compared with conventional therapy

# In practice

Jean-Michel Gracies

Guided Self-  
Rehabilitation  
Contract in  
Spastic Paresis

△ Adis

Jean-Michel Gracies

Contrat  
d'autorééducation  
guidée dans la  
Paresie Spastique

△ Adis

Springer, 2016

# A prescription !

**Patienten-Arzt-Vertrag zur Rehabilitation – Rezept**

Name, Vorname: \_\_\_\_\_ Datum: \_\_\_\_\_

**DEHNUNGEN**

Bitte dehnen Sie die Muskeln der Ihnen verordneten Dehnübungen jeden Tag mindestens 1x für jeweils 10 Minuten. Notieren Sie in Ihrem Dehnungsprotokoll den Erfolg.

**Großer Brustmuskel (Schulteradduktor)** ✓ **5x2 Min**

Im Sitzen: Paretischen Arm zur Seite ausstrecken und auf einer Unterlage ablegen. Kopf und Kinn in die entgegengesetzte Richtung drehen.



**Breiter Rückenmuskel und langer Trizepskopf (Schulteradduktor)** ✓ **5x2 Min**

Im Sitzen: Ellbogen so erhöht wie möglich auf einer Unterlage ablegen.  
Im Stehen, an einer Wand: Ellenbogen so erhöht wie möglich gegen die Wand lehnen.



**Bizeps und Musculus Brachialis (Ellenbogenbeuger)** ✓ **5x2 Min**

Im Sitzen: Ellenbogen des ausgestreckten Armes auf dem Knie ablegen. Mit der anderen Hand das Handgelenk des abgelegten Armes ergreifen, um die innere Handfläche nach unten zu dehnen.



**Viereckiger Einwärtsdreher (Einwärtsdreher des Ellenbogens)** ✓ **5x2 Min**

Im Sitzen, gebogter Ellenbogen: Mit der ganzen Hand des anderen Arms von unten das Handgelenk greifen, um die Handfläche nach oben zu ziehen.



**Runder Einwärtsdreher (Beuger und Einwärtsdreher des Ellenbogens)**

Im Sitzen, gestreckter Arm: Mit der ganzen Hand des anderen Arms von unten das Handgelenk greifen, um die Handfläche nach oben zu ziehen.



**BEWEGUNGEN**

Bitte trainieren Sie die Muskeln der Ihnen verordneten Bewegungsübungen. Wiederholen Sie die Übungen so schnell und so oft wie möglich. Notieren Sie in Ihrem Trainingstagebuch den Erfolg.

**Aktive Abduktion der Schulter** ✓ **4x15 Sek**

Im Stehen: Seitlich an die Wand stellen. Markieren wie hoch der Ellenbogen seitwärts nach oben gebracht werden kann. Arm von unten (Hüfte) nach oben zur Markierung führen.



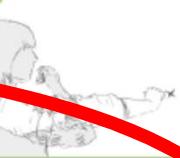
**Aktive Flexion der Schulter** ✓ **4x15 Sek**

Im Stehen: An der Wand markieren wie weit der ausgestreckte Arm nach oben kommt. Arm von unten (Hüfte) nach oben zur Markierung führen.



**Aktive Extension des Ellenbogens**

Im Stehen: An der Wand markieren wie weit die Hand bei ausgestrecktem Arm nach oben kommt. Die Hand von der Nase zur Markierung führen.



**Aktive Supination des gebogenen Ellenbogens** ✓ **4x15 Sek**

Am Tisch sitzen: Ellenbogen entspannt nahe am Körper auf den Tisch legen. Handinnenfläche nach unten auf den Tisch. Handinnenflächen nach oben und zurück nach unten drehen.



**Aktive Supination des gestreckten Ellenbogens**

Am Tisch sitzen: Arm entspannt und gestreckt mit den Handinnenflächen nach unten auf den Tisch legen. Daumen so weit wie möglich von der Hand abspreizen. Handinnenflächen nach oben und zurück nach unten drehen.





**GSC**  
Guided Self-Rehabilitation Contract  
LOWER LIMB

Doctor stamp

Last Name: \_\_\_\_\_  
First Name: \_\_\_\_\_



**GSC**  
Guided Self-Rehabilitation Contract  
UPPER LIMB

# A prescription!

## PASSIVE STRETCH



✓ 2 Minx5

Gluteus maximus

✓ 15 Secx4

## ACTIVE TRAINING



Active hip flexion,  
knee extended (1)

Hamstrings



Passive stretch (3)



Active hip flexion,  
knee extended (4)

## HIP



Passive stretch (5)  
Hip flexor - adductors

Hip adductors

Passive stretch (6)  
Hip extensor-adductors

Active hip abduction (7)



## HIP



Passive stretch (8)

Hip internal rotators

Active hip external rotation (9)



## KNEE



Passive stretch (10)

Rectus femoris

Active knee flexion,  
knee extended (11)



## PASSIVE STRETCH

THUMB



Passive stretch (42)

min times/day

Long thumb flexor



Active long thumb extension (43)



THUMB



Passive stretch (44)

min times/day

Short thumb flexor



Active short thumb flexor (45)



THUMB



Passive stretch (46)

min times/day



Active thumb deopposition (48)



THUMB



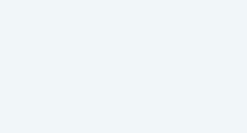
Passive stretch (47)

min times/day

Adductor pollicis



Active short thumb abduction (50)



THUMB



Passive stretch (49)

min times/day

Active short thumb abduction (50)



# **Guided Self-rehab = Contract**

- 1. Patient works and documents**
- 2. Therapist teaches and coaches**

Woman 38 years old

Right MCA stroke Oct 2013

PT twice a week until Feb 2015

Status Feb 2015

000109105701

AU

T2 FLAIR BLADE TRA

CHU DDON

F  
049Y

A10079199187

22/03/2019-16:11

Se:9

Im:2.6

Loc: S 674

RAS

LAS

ET: 28

TR: 8000,00 ms

TE: 140,00 ms

Thk: 4,00 mm

Z: 2,9 X

L: 632 W: 1392

PRS

Blanchon, Laurence (Mrs),8004489600

Acc : 30029980212

Descr. Examen : IRM cérébrale fonctionnelle (fct motrices)

Descr. Série : Pouce-index Multilabel [1]

1004 - 69

Avec perte (1:18)

06/03/2020 14:11:32

HENRI MONDOR

LT : 0,90 mm

C :476 W :1040

Zoom : 316%

R



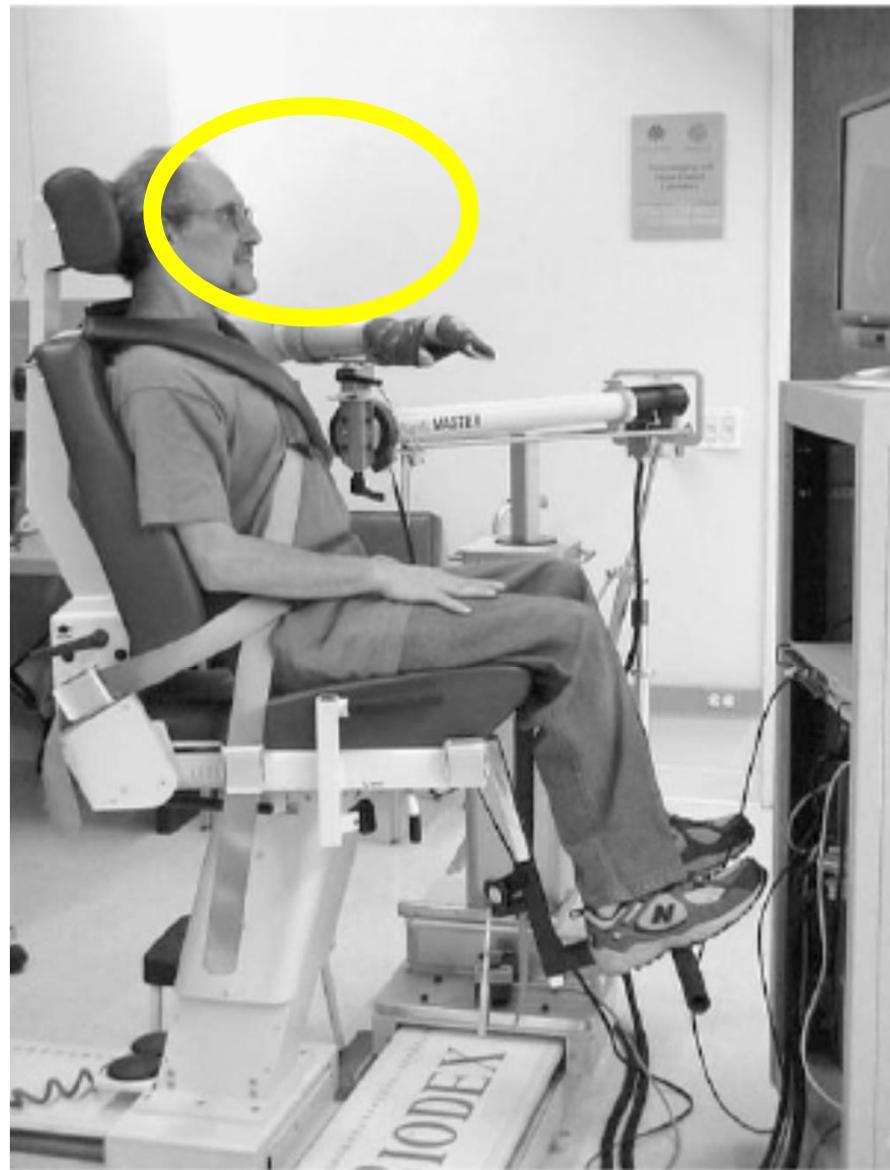
P

ies, 2003



**Feb 2015  
M17 post stroke**

# Shoulder adductors/extensors



**Fig. 2.**  
Subject seated in the ACT<sup>3D</sup> system. His trunk is secured by straps and the arm is attached to the HM with the lightweight forearm-hand orthosis. He is looking at the computer monitor for visual feedback, shown in Fig. 3

*Sukal TM, Ellis MD, Dewald JP. Shoulder abduction-induced reductions in reaching work area following hemiparetic stroke: neuroscientific implications. Exp Brain Res. 2007;183(2):215-23*

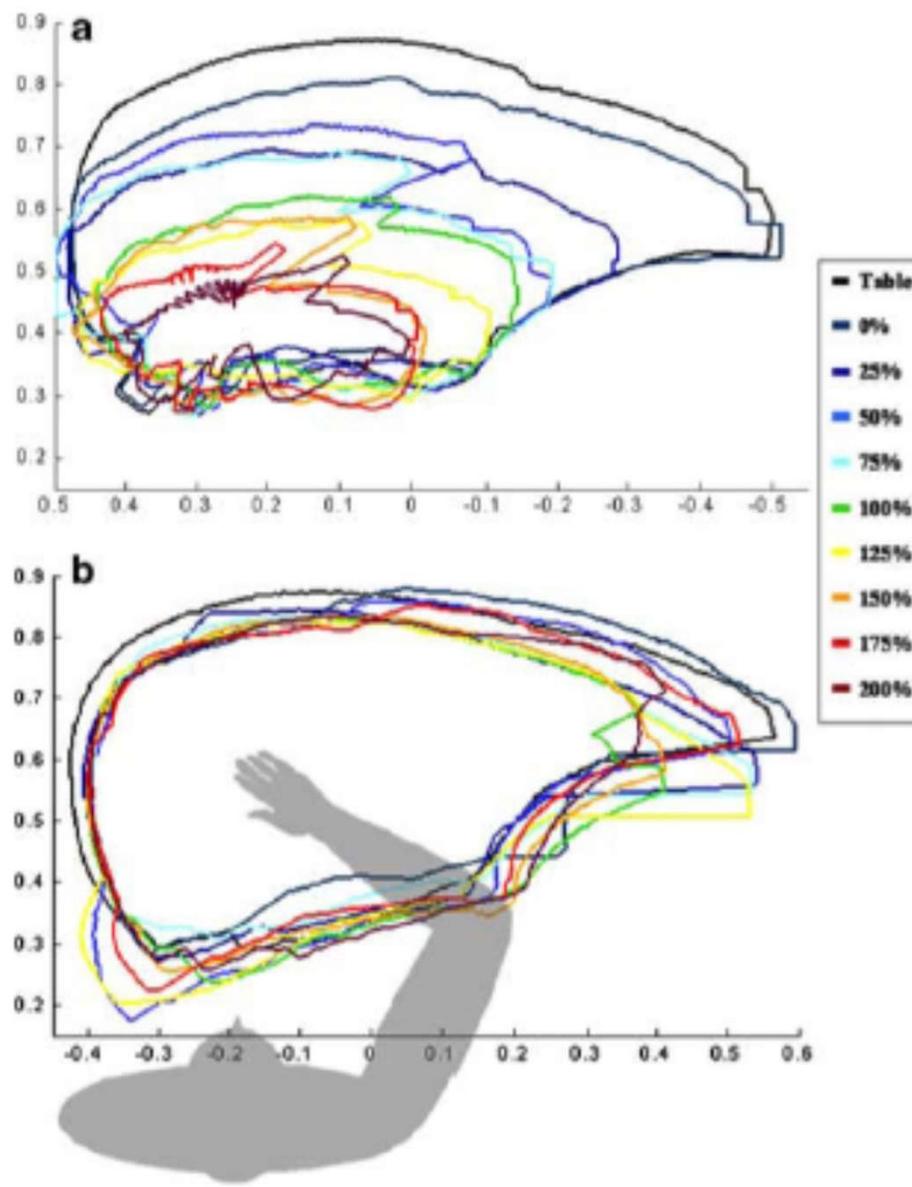
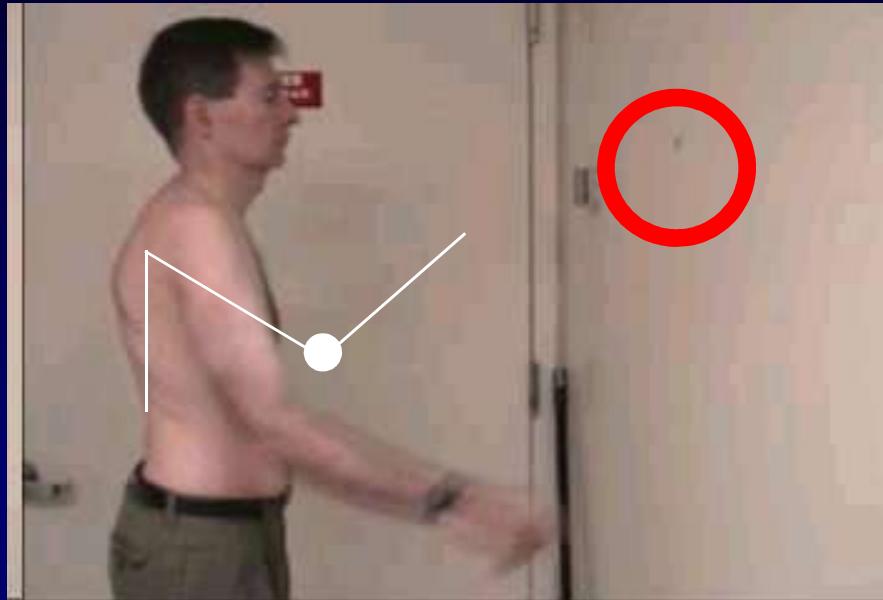


Fig. 4.  
Envelope abilities during various levels of limb support in the left, paretic limb (a) of a single subject, inverted for comparison to the non-paretic limb shown in (b). Axes units are in meters, and an individual's outline is provided in the non-paretic (right) side for reference.

*Sukal TM, Ellis MD, Dewald JP. Shoulder abduction-induced reductions in reaching work area following hemiparetic stroke: neuroscientific implications.*  
*Exp Brain Res. 2007;183(2):215-23*

# Decrease in finger flexor cocontraction when fight ↓ against shouder extensors



Pre

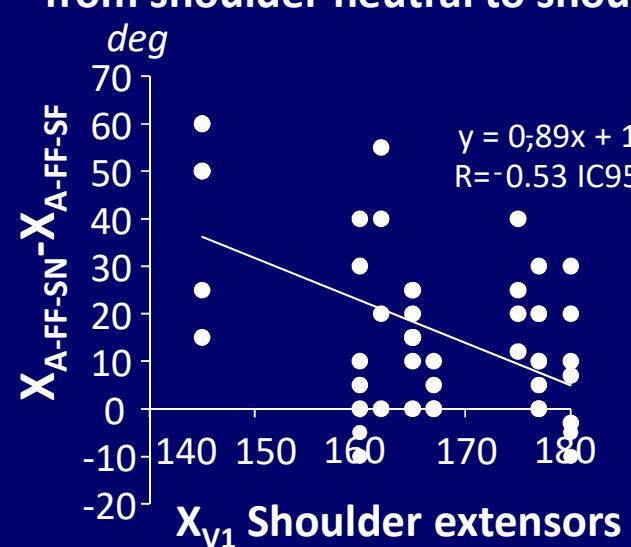
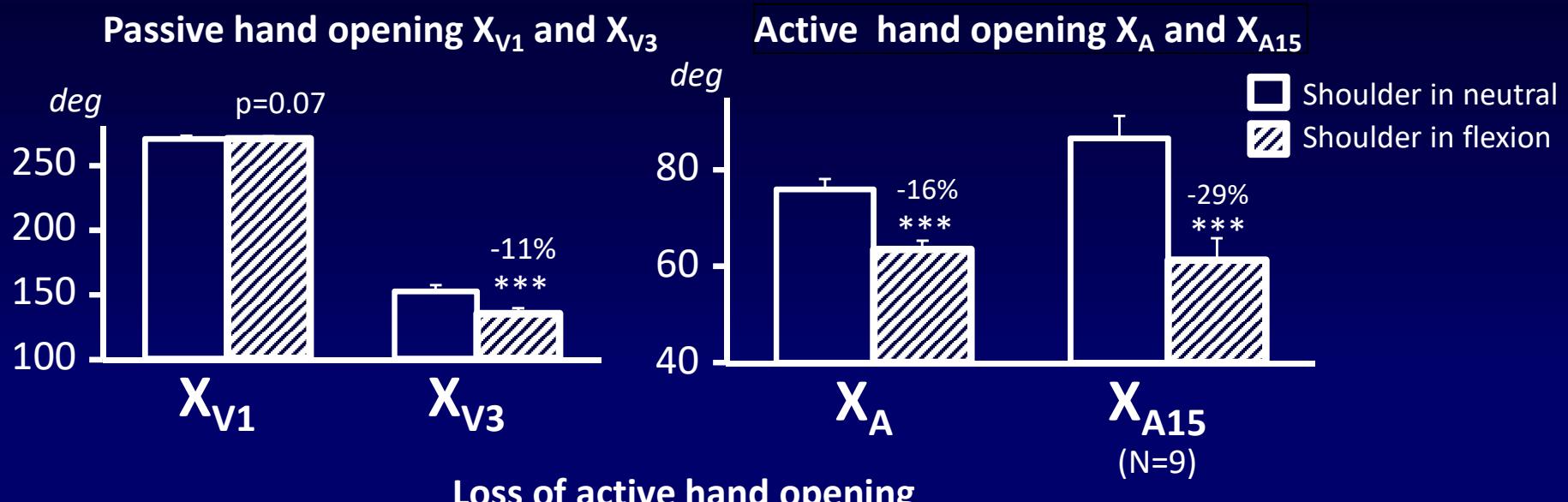
Injection lidocaine 2%



5 minutes post

Pectoralis Major:	4 cc
Long head of triceps:	2 cc
Teres major:	1 cc
Latissimus dorsi:	1 cc
Rhomboïds (maj + min)	2 cc

# *Increase in shoulder extensor extensibility associated with greater hand opening in reaching efforts - n=16*



# M13 post stroke



24 nov 2014

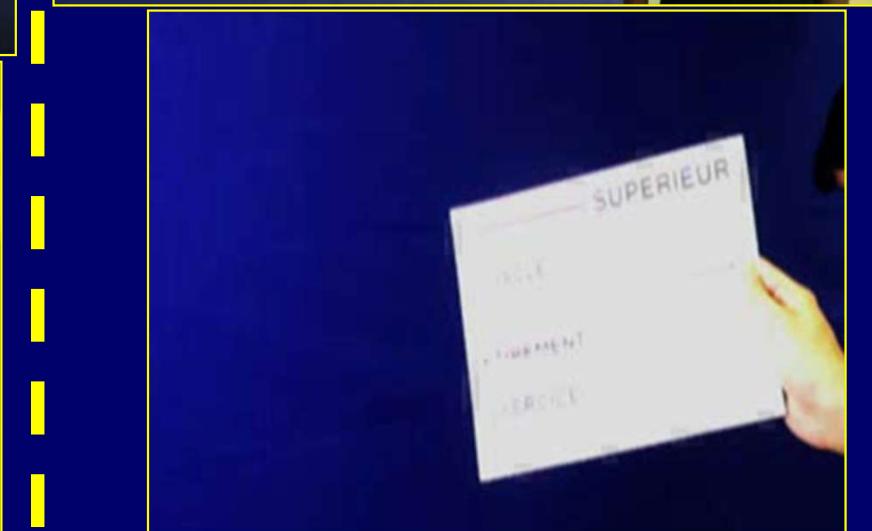
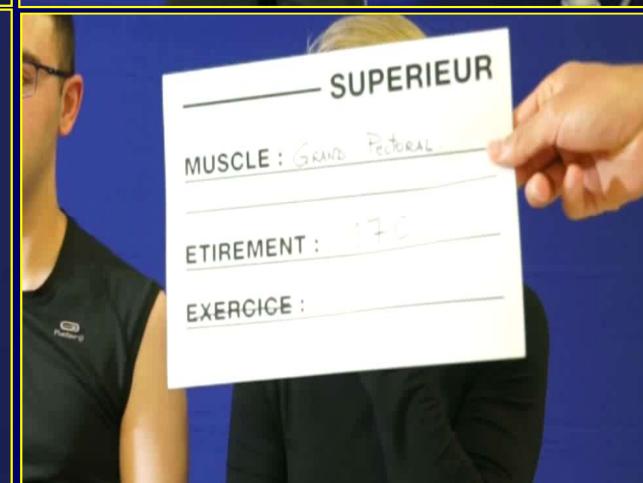
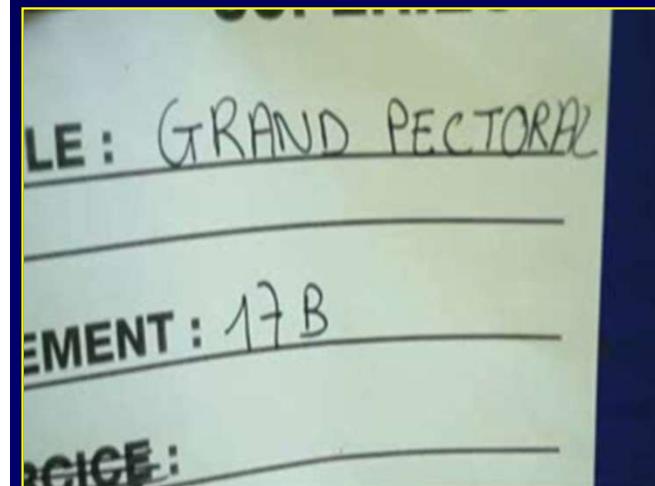
*Lang CE, Wagner JM, Bastian AJ, Hu Q, Edwards DF, Sahrmann SA, Dromerick AW. Deficits in grasp versus reach during acute hemiparesis. Exp Brain Res. 2005;166(1):126-36*

# Against pectoralis major

Treatment of **myopathy**  
= Stretching postures

Treatment of  
cocontraction

- | Rapid Alternating
- | Movements of
- | maximal amplitude,  
| unassisted



# 4 years of Self-Rehabilitation Contract

---



Nov 14 – M13 post stroke



Jul 15 – M21



Mar 16 – 2,5 yrs post



Mar 17 – 3,5 years post stroke

# 4 years of Self-Rehabilitation Contract



Nov 14 – M13 post stroke



Jul 16 – 2,5 years



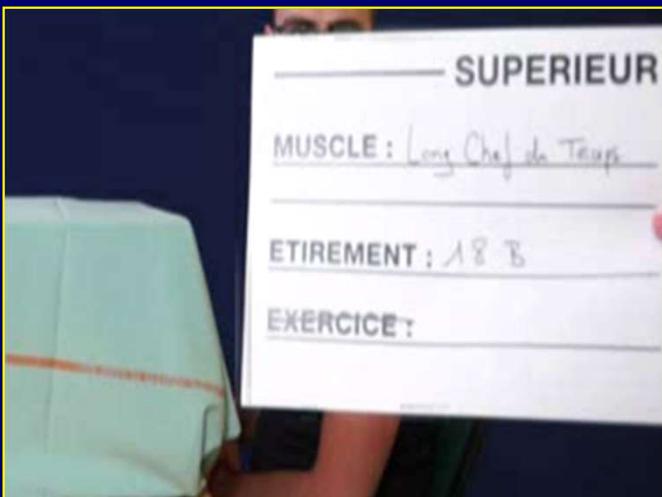
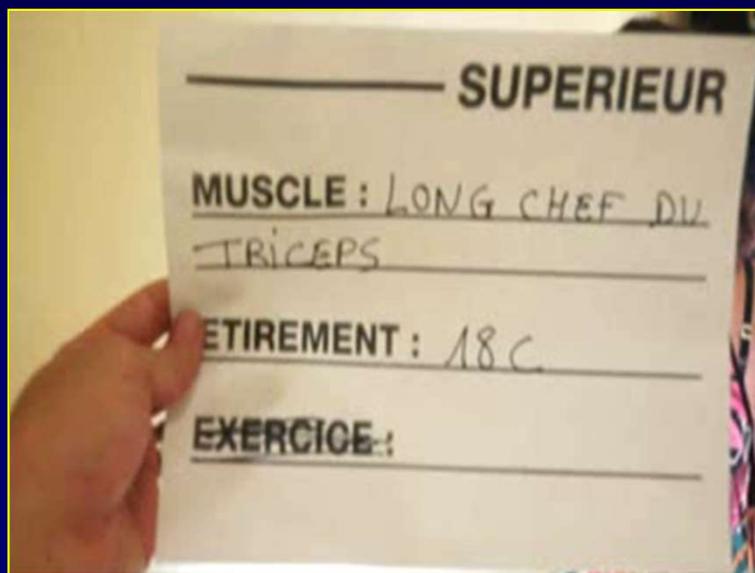
Mar 17 – 3,5 years post stroke



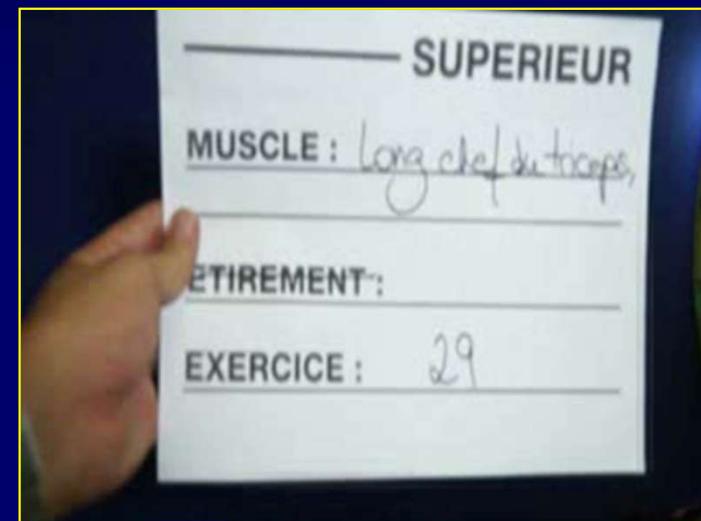
Sept 17 – 4 years post stroke

# Against long head of triceps

Stretching postures  
→ Muscle disorder



Rapid Alternating Movements of maximal amplitude, unassisted  
→ Neurological disorder

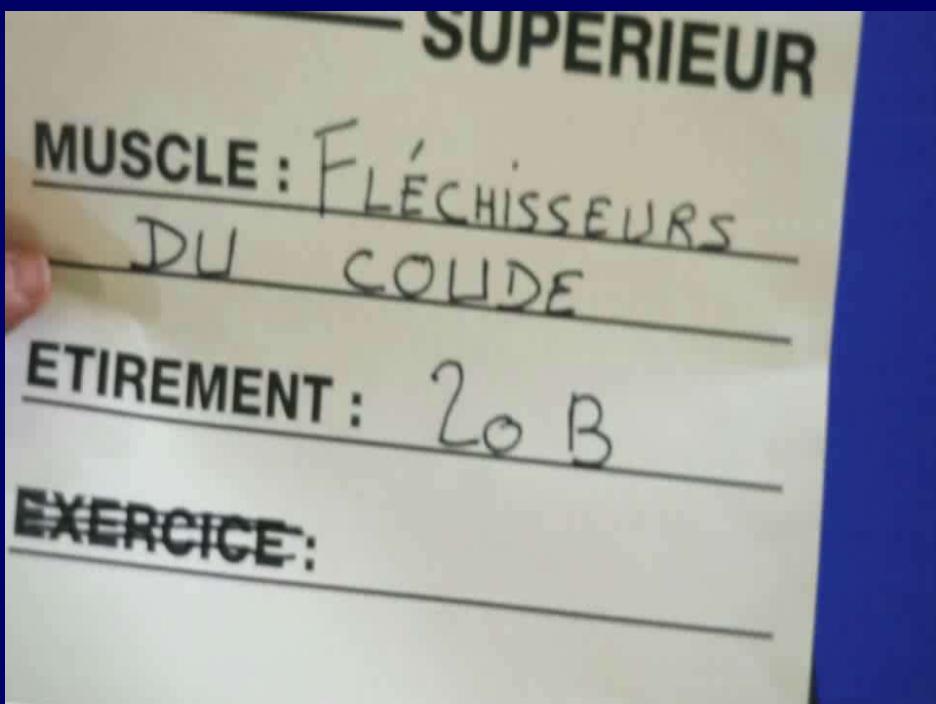


# Work against long head of triceps

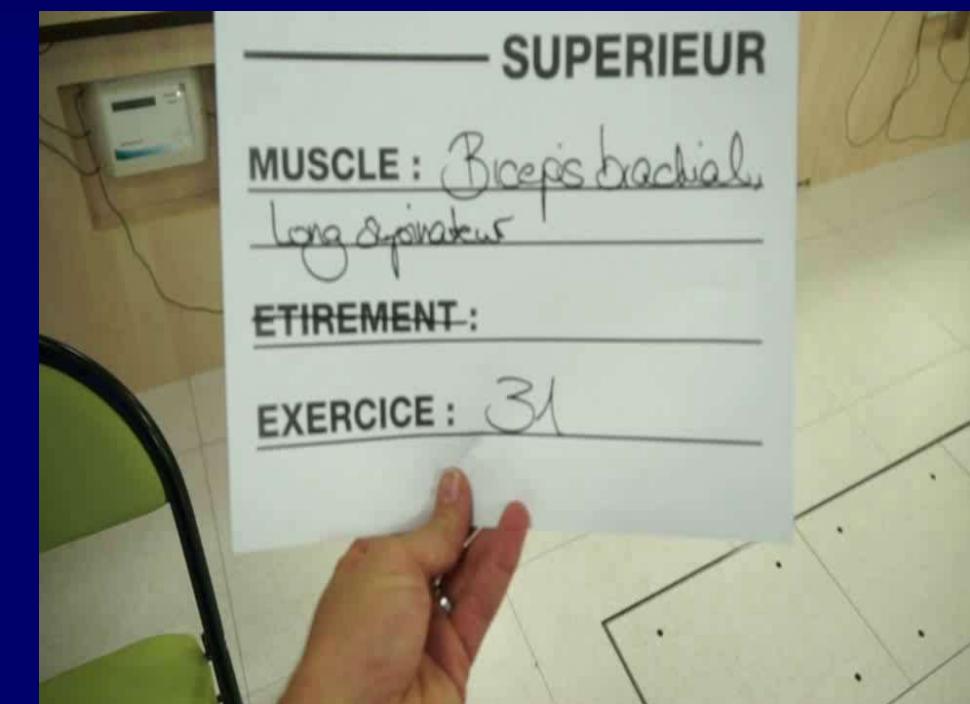


# Against elbow flexors

Stretching postures  
→ Muscle disorder



Rapid Alternating Movements of maximal amplitude, unassisted  
→ Neurological disorder



# 4 years of Self-rehabilitation Contract



Nov 14 – M13



Nov 14 - downwards

Dec 16 – M38



# Against pronator quadratus

## Group workshop



# 4 years of Self-rehabilitation Contract

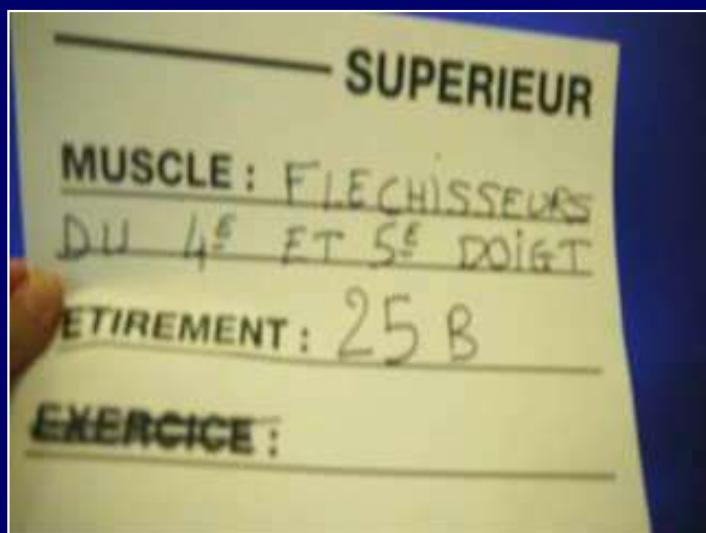
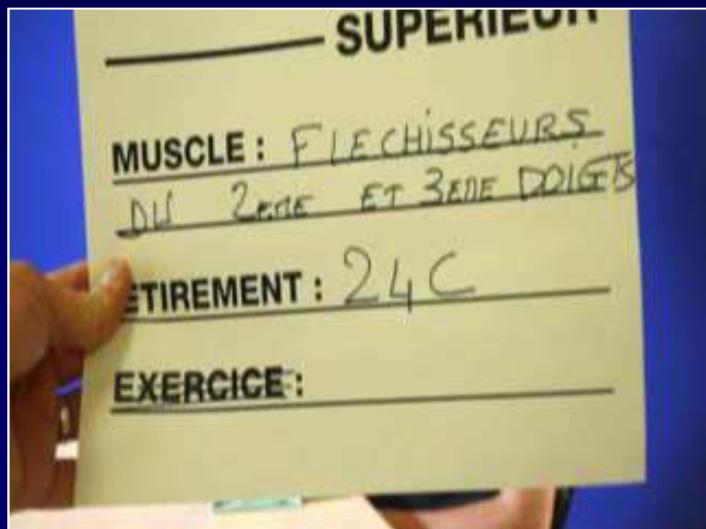


Jan 18 – 4.5 years post stroke

Elbow large supinations vs pronator teres

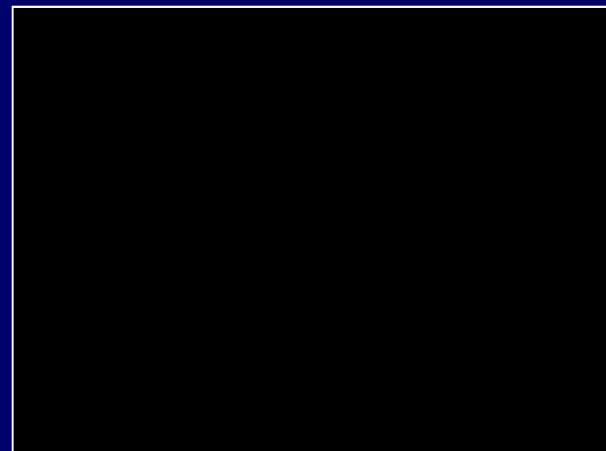
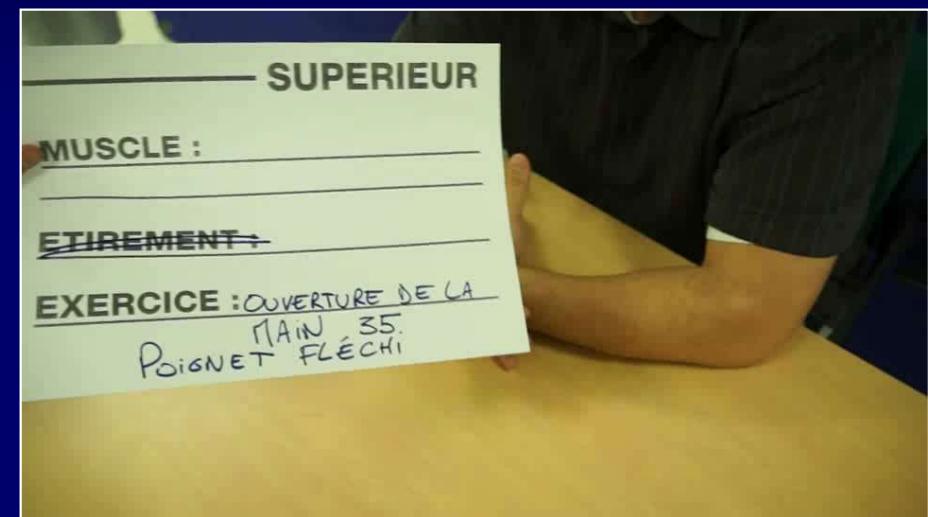
# Against finger flexors

Stretching postures  
→ Muscle disorder



Rapid Alternating  
Movements of maximal  
amplitude, unassisted  
→ Neurological disorder

- |
- |
- | Wrist flexed
- |
- | Wrist in neutral
- |



# Work against cocontraction of extrinsic finger flexors



05/12/17 – Y4



19/06/18 – Y4,5



16/01/18 – Y4,2



13/09/18 – Y4,9



21/03/18 – Y4,5



11/12/18 – Y5

# What we need to function is individuation, more than amount of agonist recruitment

Quantification of 2 critical aspects of hand function, ‘strength’, and independent control of fingers (individuation).

n = 54 patients with hemiparesis over first year after stroke.

→ Most recovery of strength and individuation occurred within the first 3 mo.

→ Recovery of ‘strength’ and individuation tightly correlated up to a ‘strength’ level of ~60% of estimated premorbid strength; beyond this threshold, strength improvement was not accompanied by further improvement in individuation.

*Separable systems for recovery of finger strength and control after stroke*

Xu J, Ejaz N, Hertler B, Branscheidt M, Widmer M, Faria AV, Harran MD, Cortes JC, Kim N, Celnik PA, Kitago T, Luft AR, Krakauer JW, Diedrichsen J. *J Neurophysiol.* 2017;118(2):1151-1163

# Work against cocontraction of FDS-FDP II



Oct 17 – M44



Dec 17 – M46



Ext II-V Dec 17 – M46



Ext II Mar 18 – M49



Ext II Dec 18 – M58

*Lang CE, Schieber MH. Reduced muscle selectivity during individuated finger movements in humans after damage to the motor cortex or corticospinal tract. J Neurophysiol. 2004;91(4):1722-33*

# Work against cocontraction of FDS-FDP III



Oct 17 – M44



Ext III post II preT  
Dec 17 – M46



Ext III Mar 18 – M49



Ext III pre T-  
Jun 18 – M52



Ext III  
post T-  
Sep 18 –  
M55



Ext III  
post T-  
Dec 18 –  
M58

# Work against cocontraction of Palmar IO



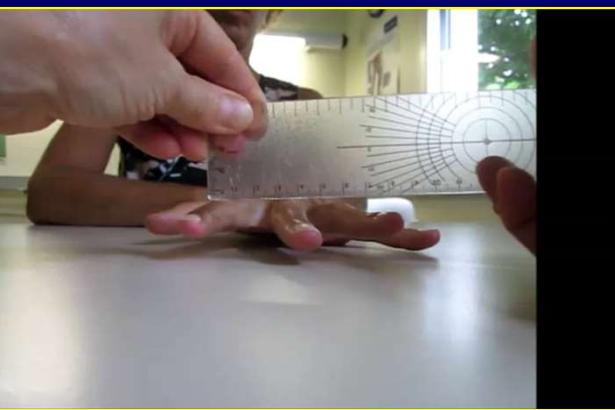
Dec 16 – 3 yrs



Jan 18 – M47



Mar 18 – M49



Jun 18 pre – M52



Sep 18 post – M55



Dec 18 – 5 yrs



AUGUST  
OSAGE COUNTY

Escape 2002



**Mar 15 – M17**



**Dec 18 – Y5**



**22/03/15 – M18**

**22/03/16 – M30**

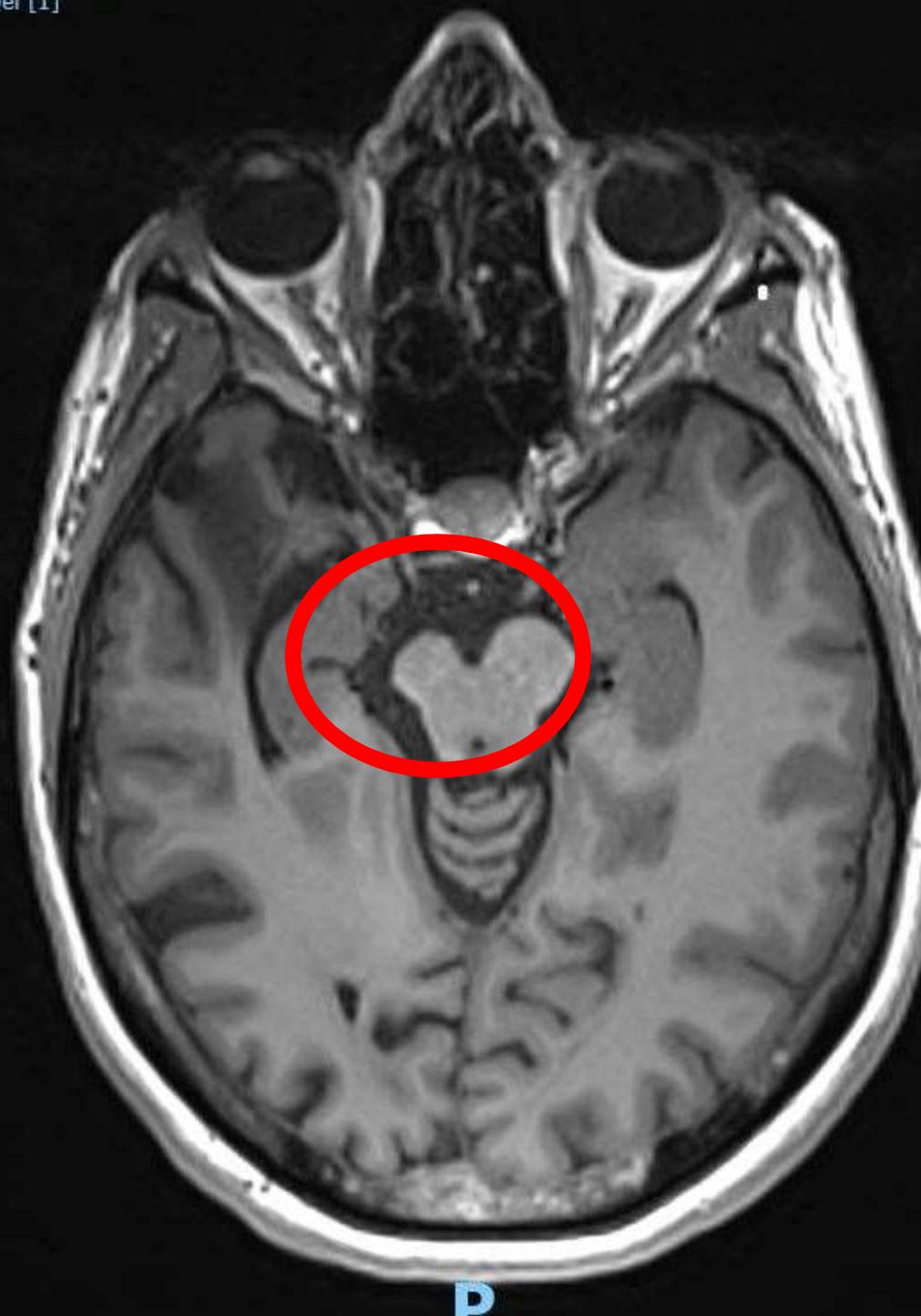


**15/01/18 – Y4 Thank you ! 11/12/18 – Y5**

© Dr C Gault-Colas

Blanchon, Laurence (Mrs),8004489600  
Acc : 30029980212  
Descr. Examen : IRM cérébrale fonctionnelle (fct motrices)  
Descr. Série : Pouce-index Multilabel [1]  
1004 - 69  
Avec perte (1:18)

06/03/2020 14:11:32  
HENRI MONDOR  
LT : 0,90 mm  
C : 476 W : 1040  
Zoom : 316%



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