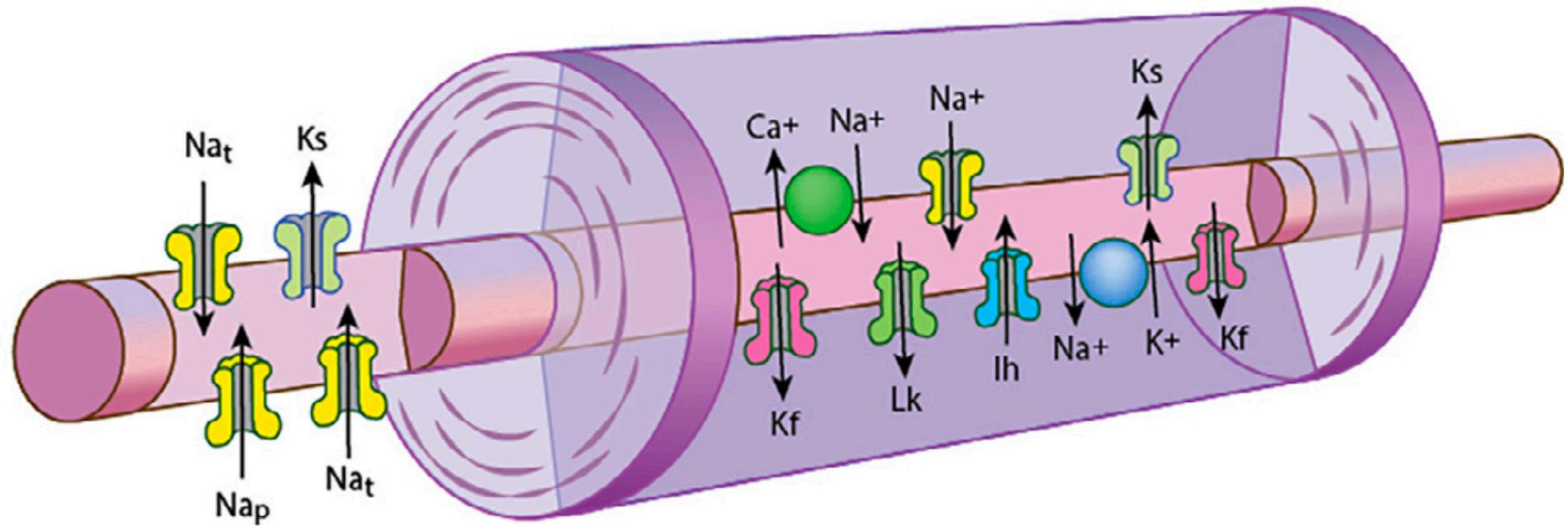
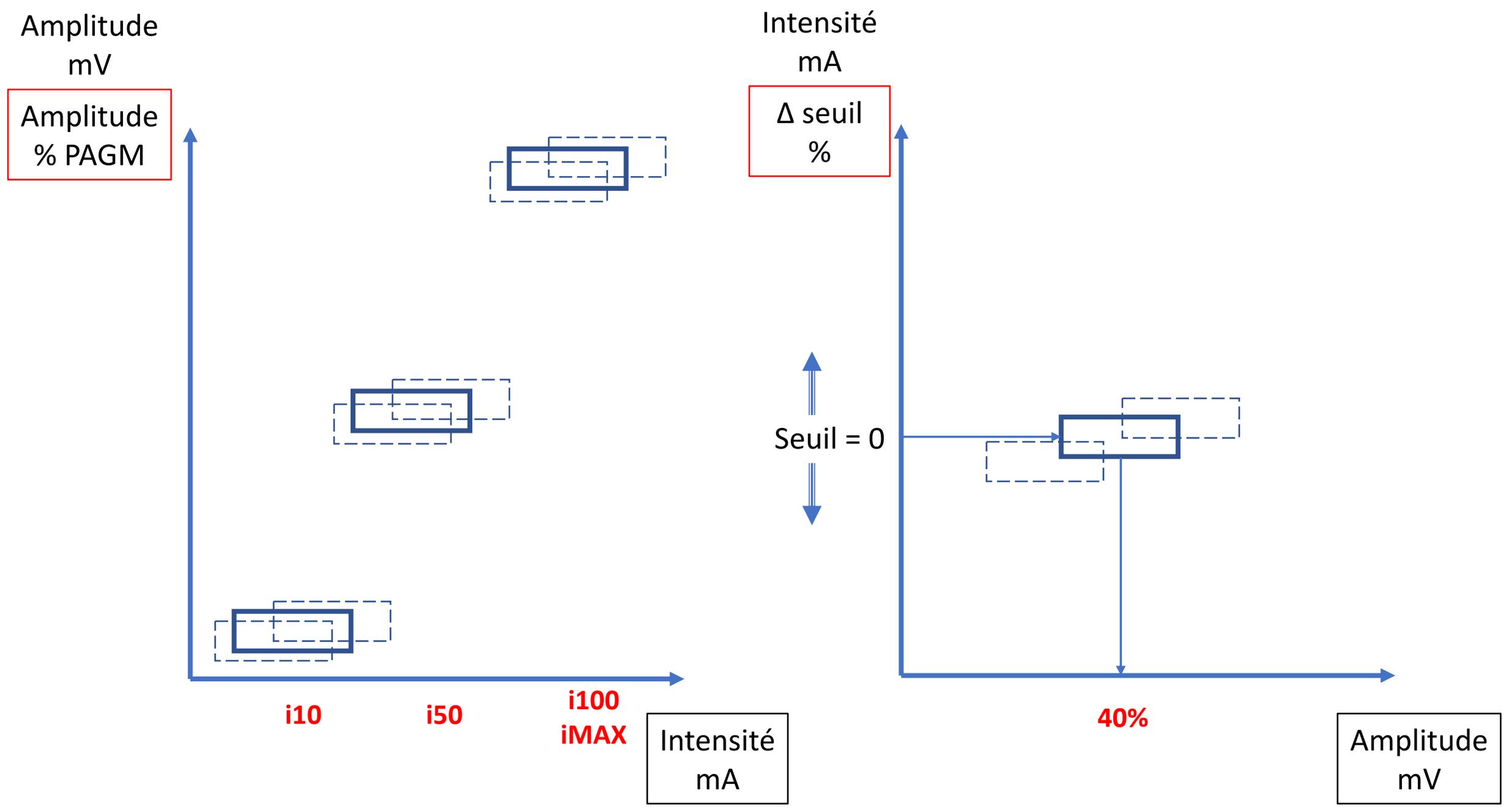


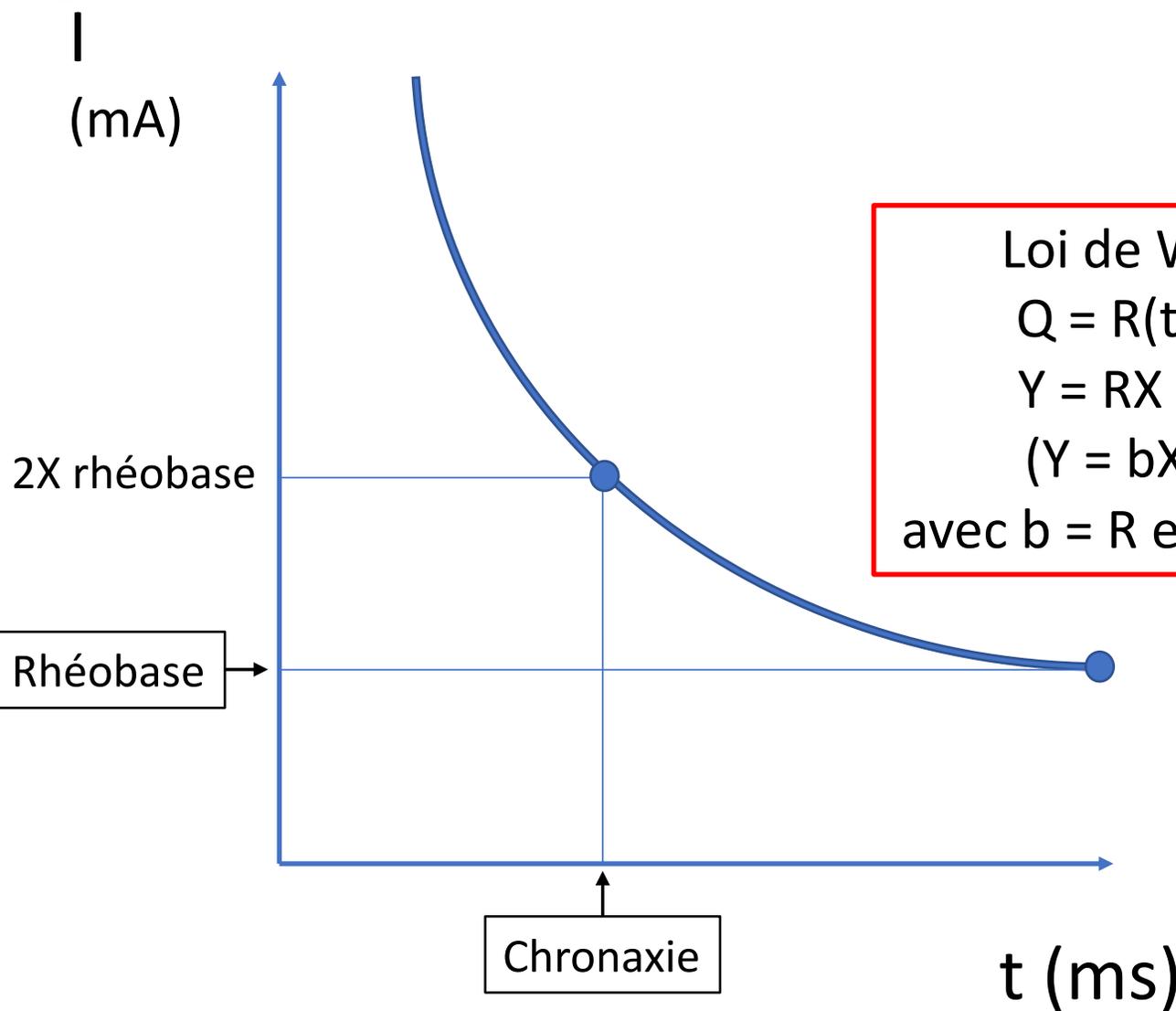
L'excitabilité nerveuse en pratique

F Wang

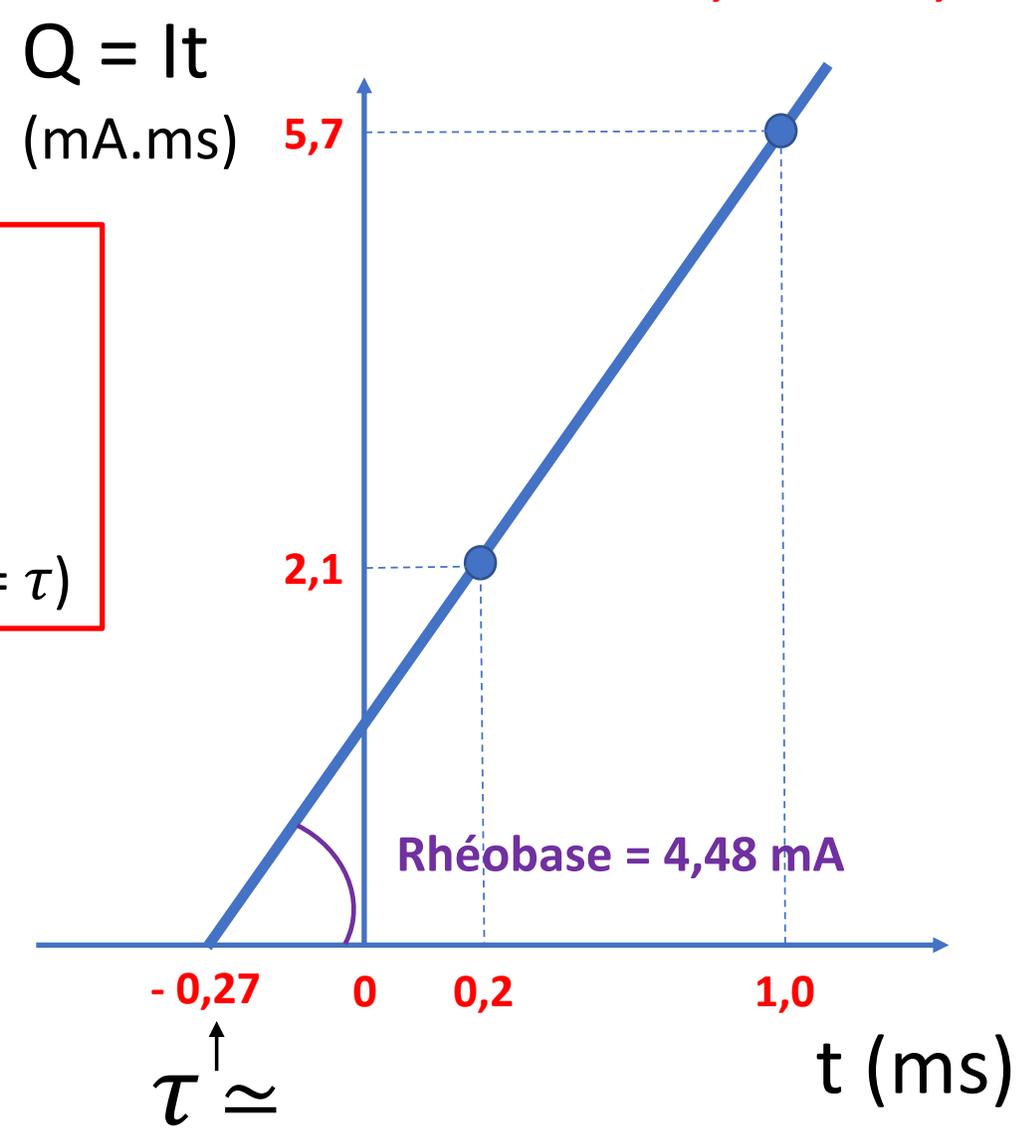




A



B



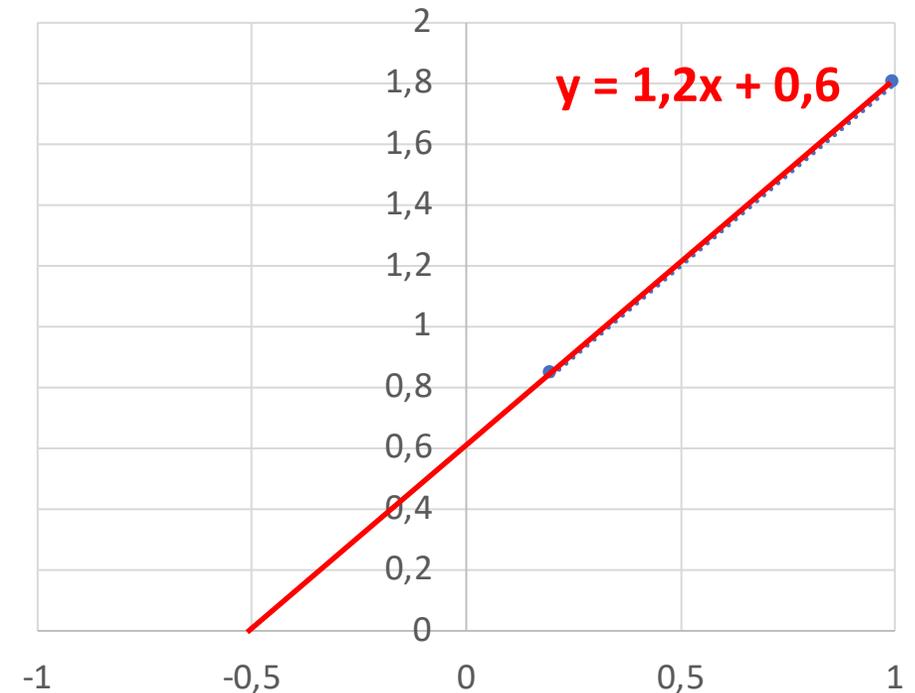
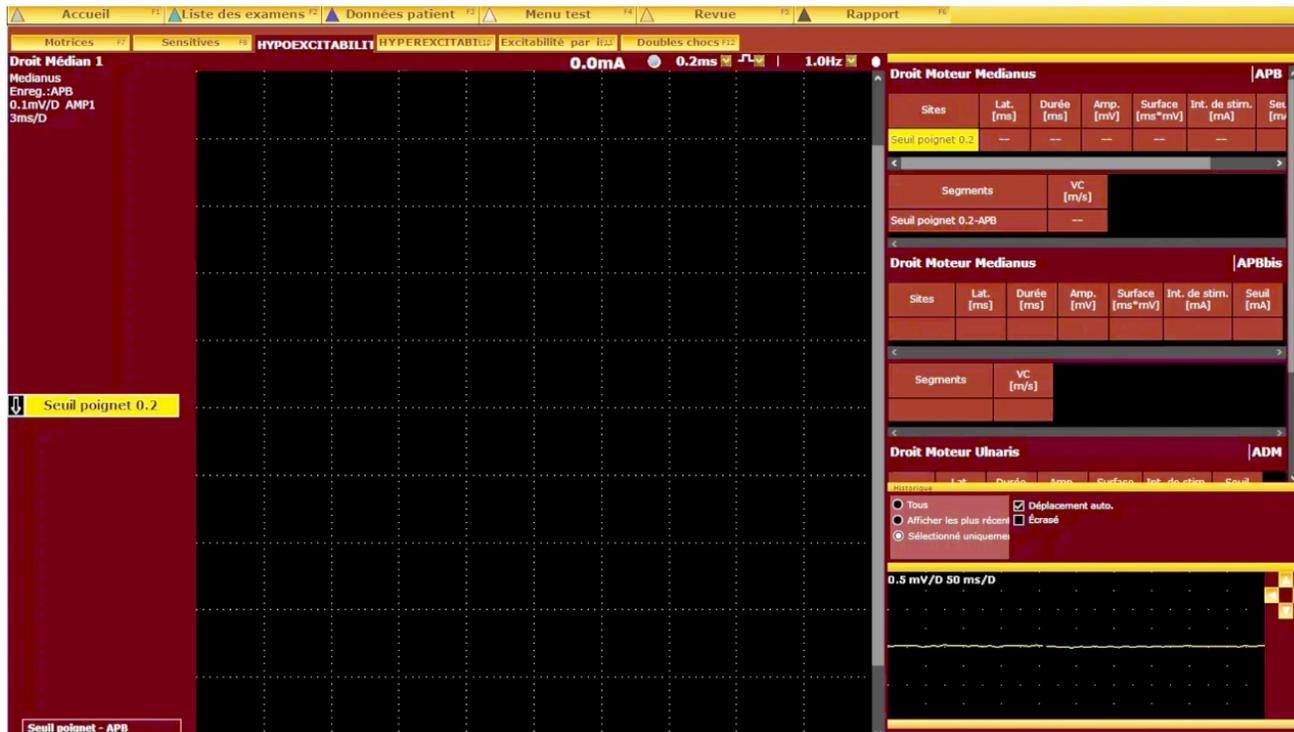
Loi de Weiss
 $Q = R(t + \tau)$
 $Y = RX + R\tau$
 $(Y = bX + a$
 avec $b = R$ et $a/b = \tau)$

Chronaxie : $1,23/4,48 = 0,27$ ms

- Intensité nécessaire pour obtenir une réponse motrice de **100 μ V**
 - durée de stimulus = **0,2 ms**
 - durée de stimulus = **1,0 ms**

$$Q = It$$

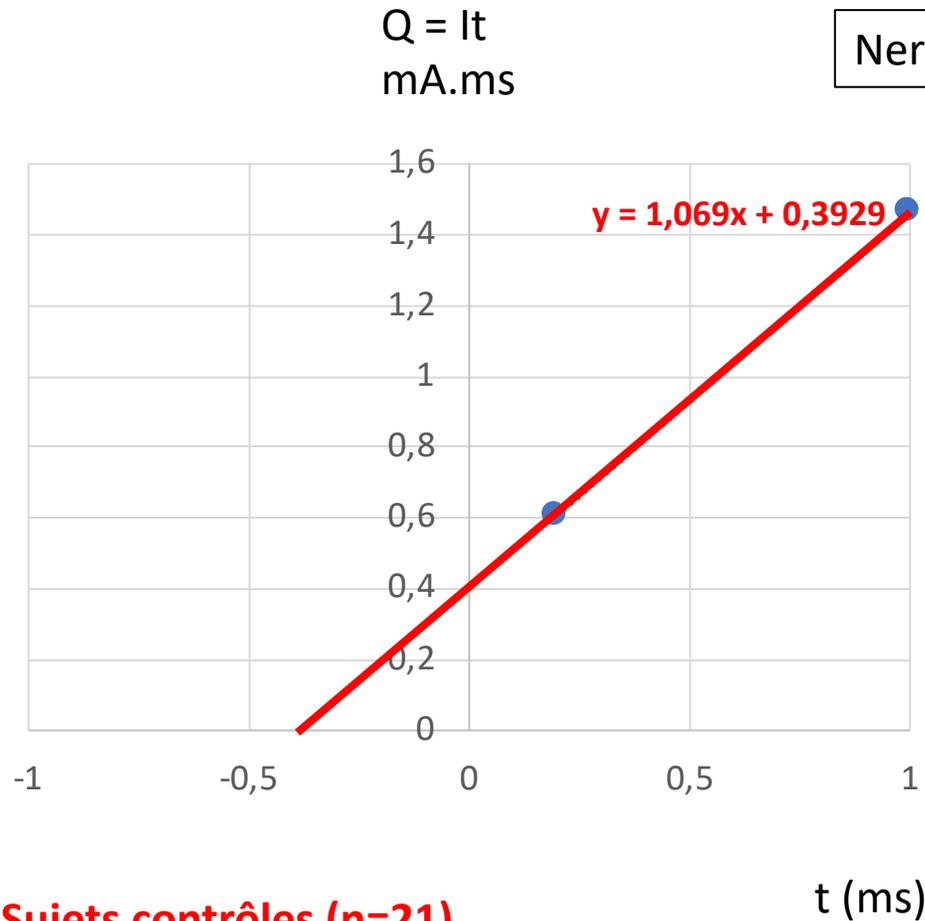
mA.ms



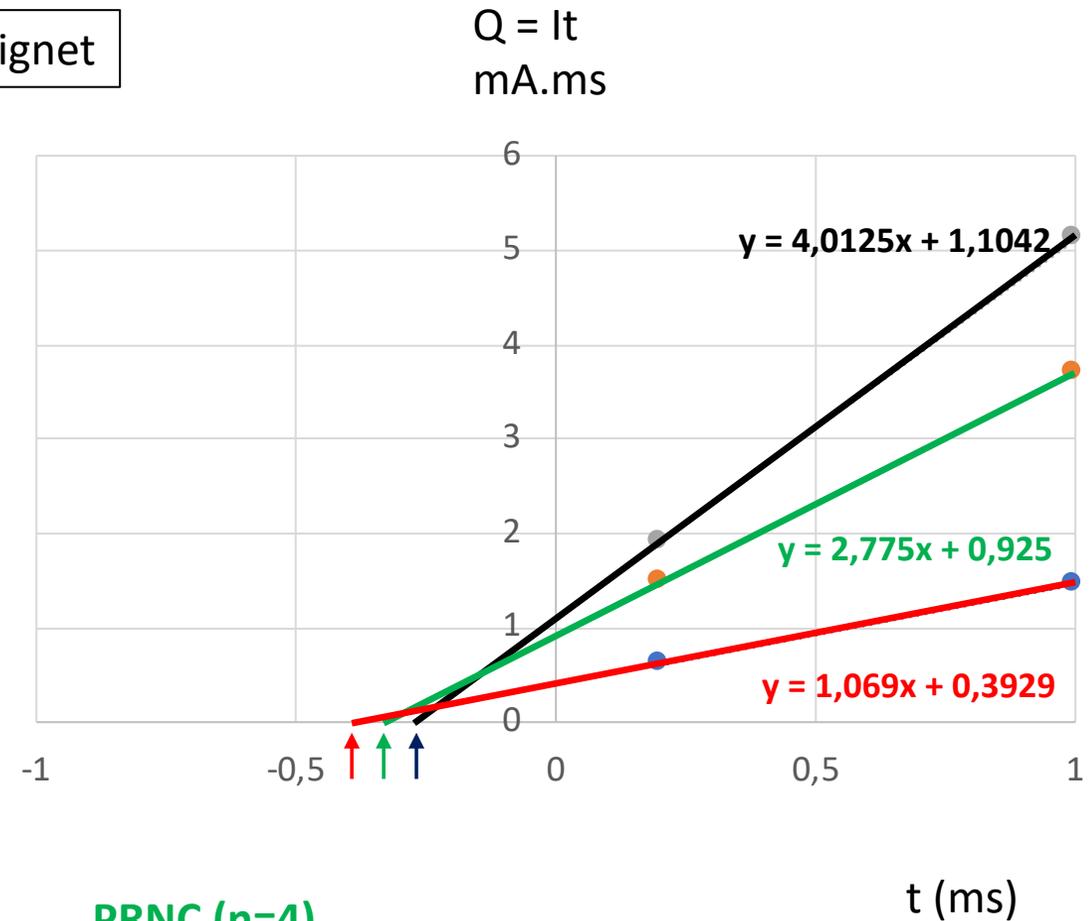
Rhéobase = 1,2 mA
Chronaxie = 0,5 ms

t (ms)

- Intensité nécessaire pour obtenir une réponse motrice de **100 μV**
 - durée de stimulus = **0,2 ms**
 - durée de stimulus = **1,0 ms**



Sujets contrôles (n=21)



PRNC (n=4)

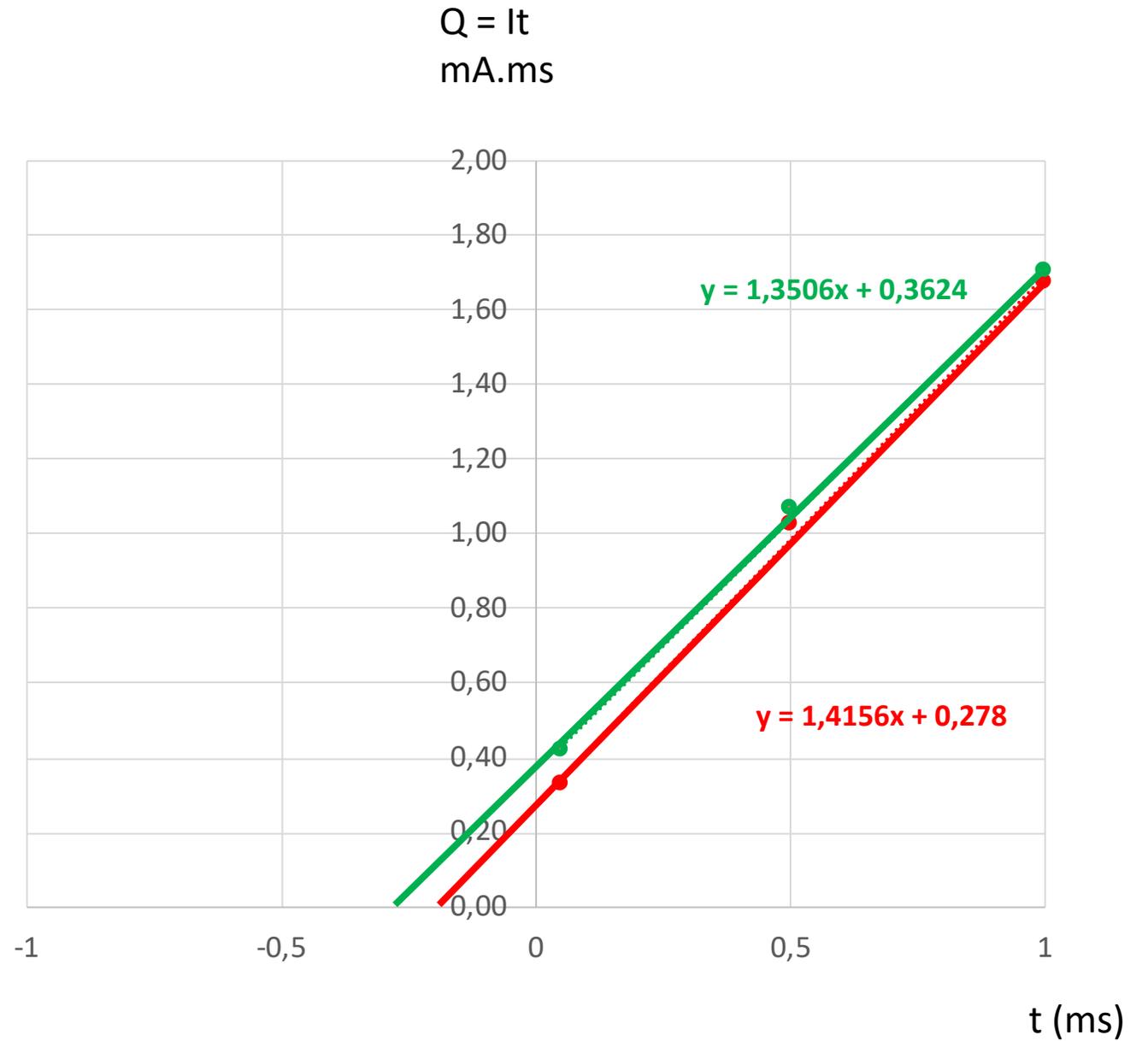
CMT1a (n=6)

- durée de stimulus = 0,05 ms
- durée de stimulus = 0,5 ms
- durée de stimulus = 1,0 ms

Nerf médian au poignet

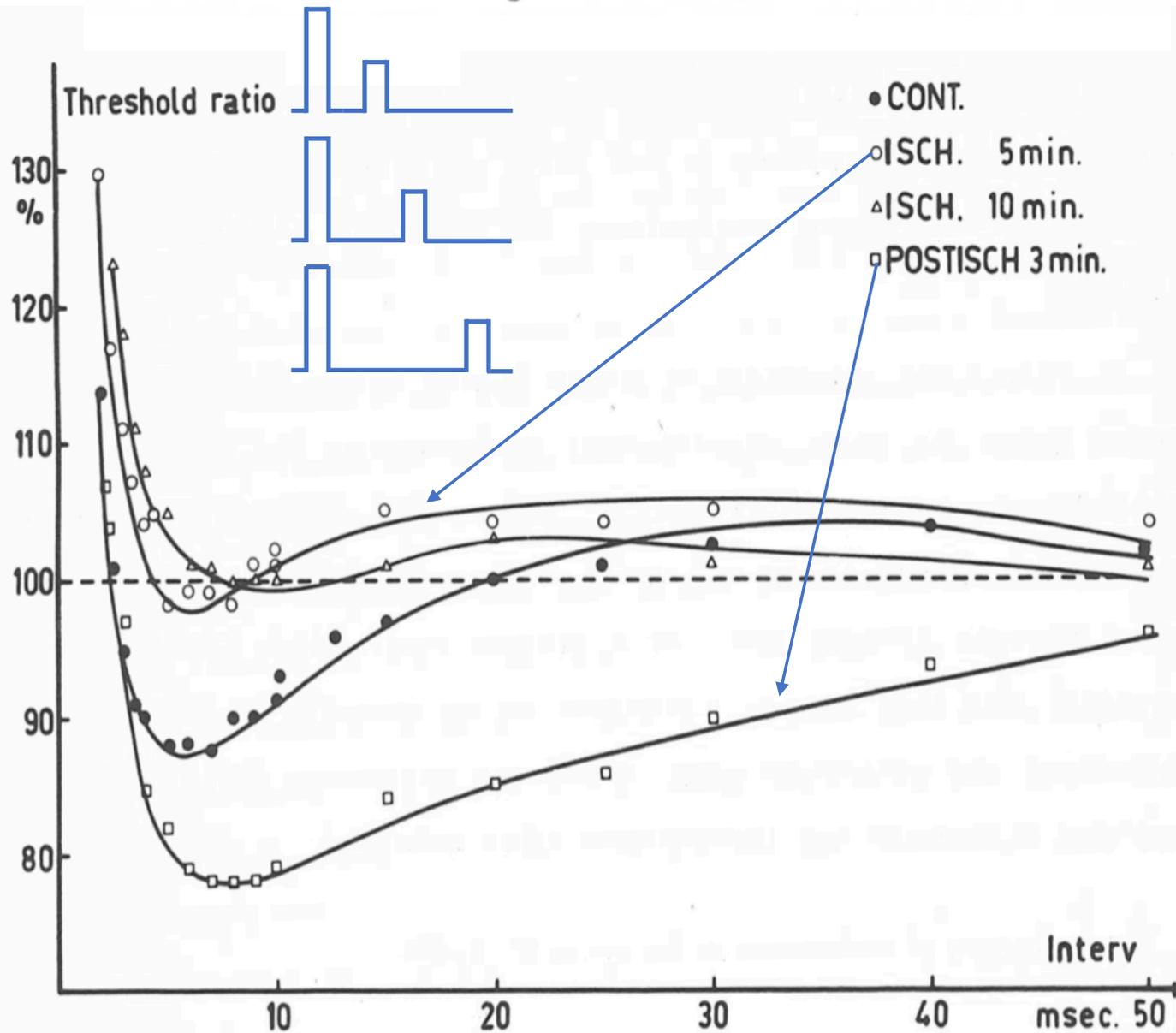
Sujets contrôles (n=8)

SLA (n=10)

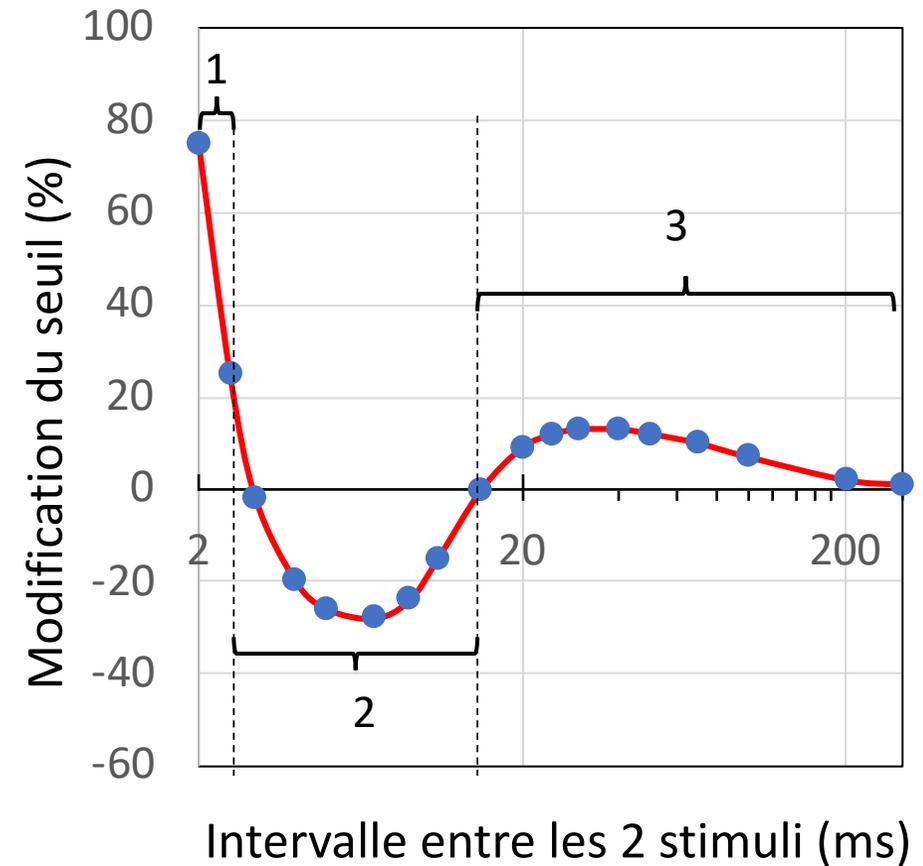


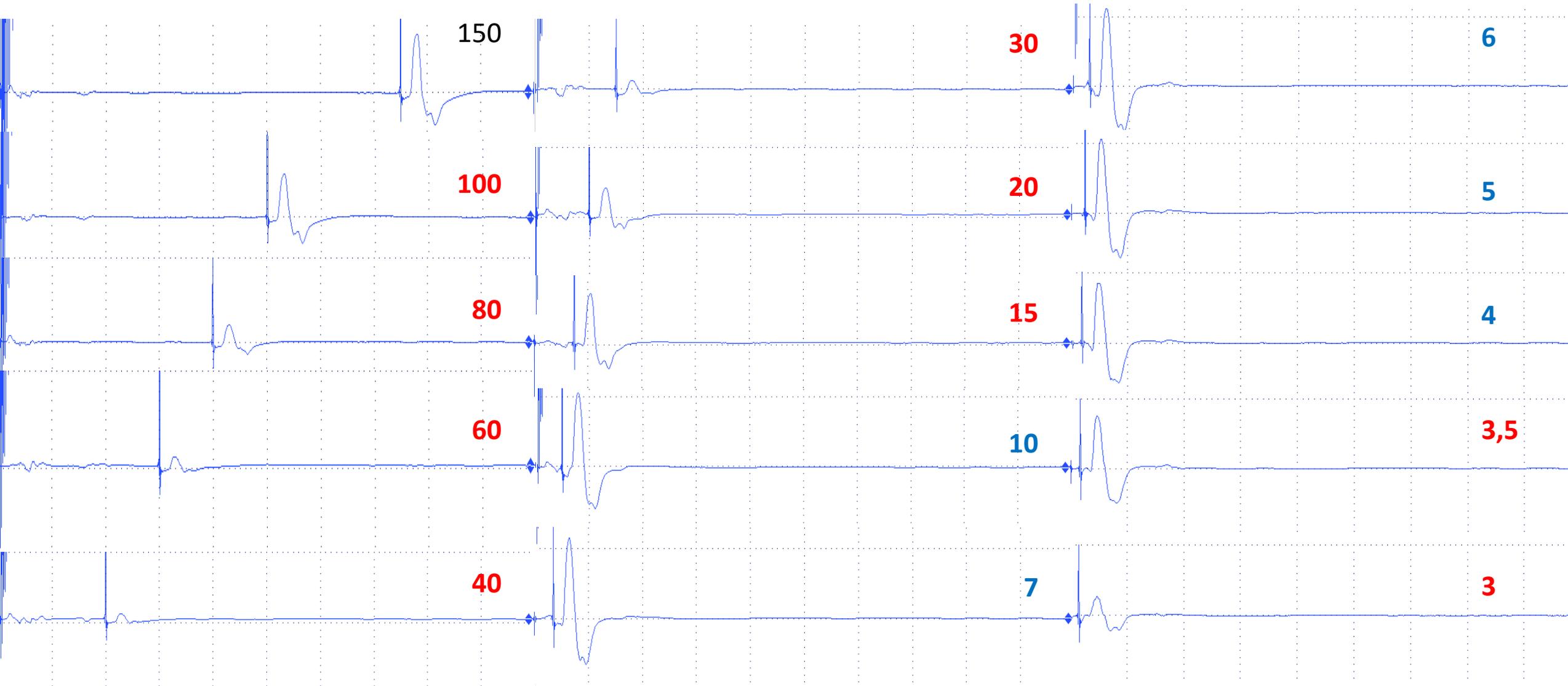
A

Figure 55. The influence of ischaemia on the recovery after single activation.

**B**

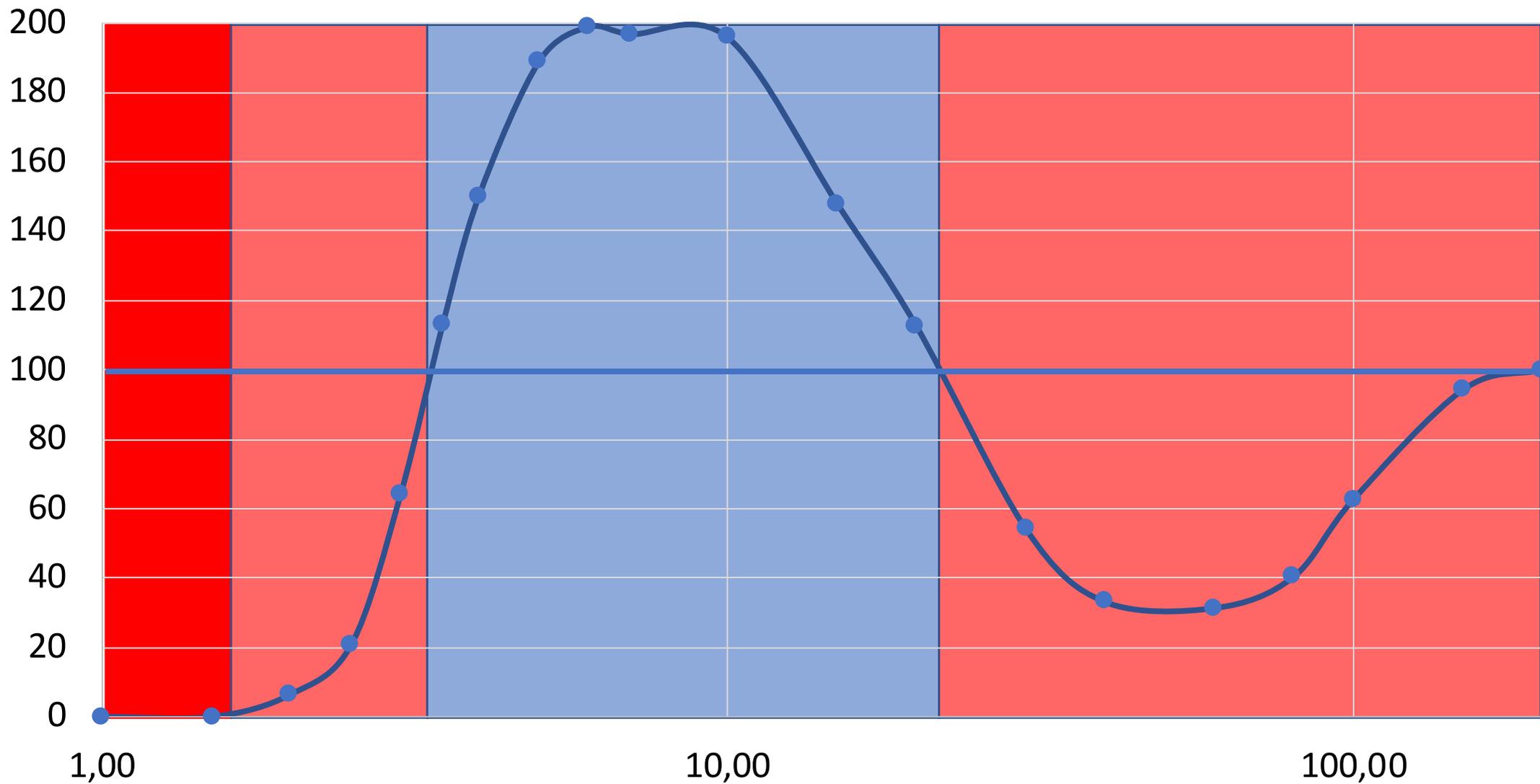
- 1 Période réfractaire relative
- 2 Période supernormale
- 3 Période sous-normale tardive

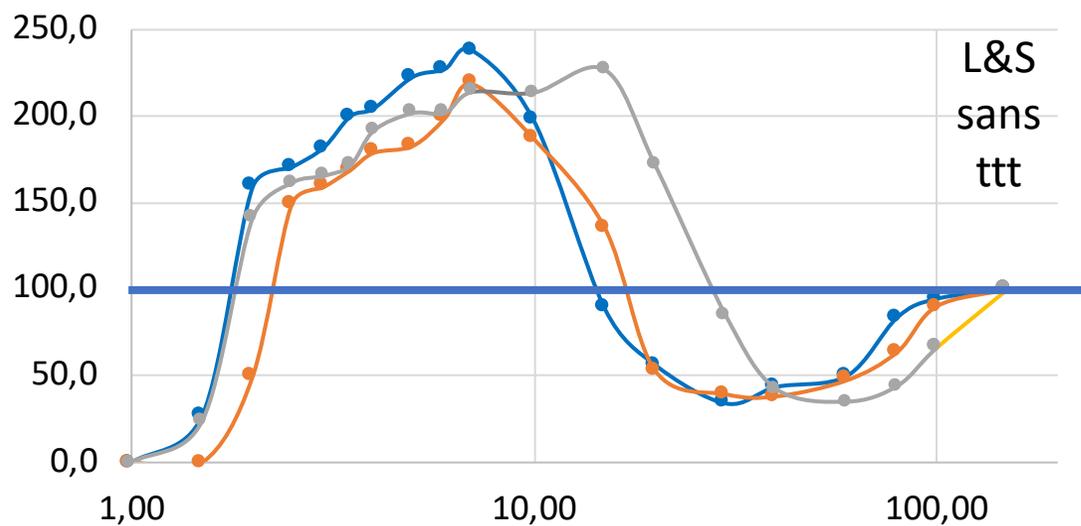
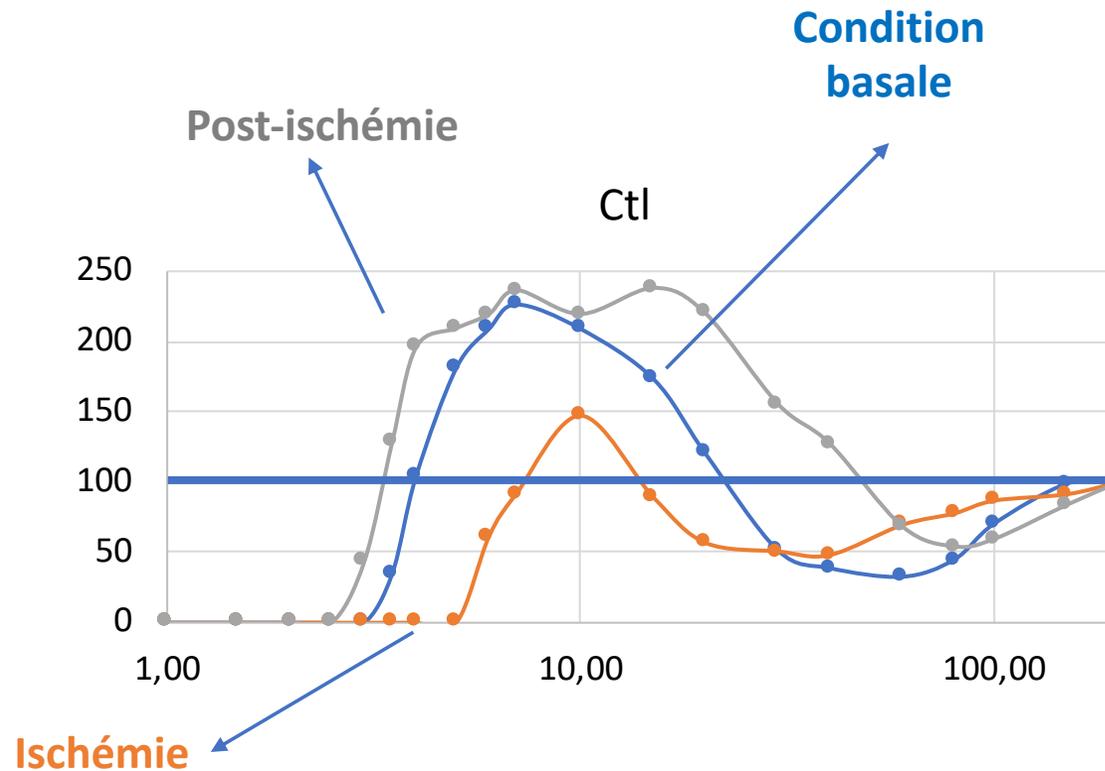
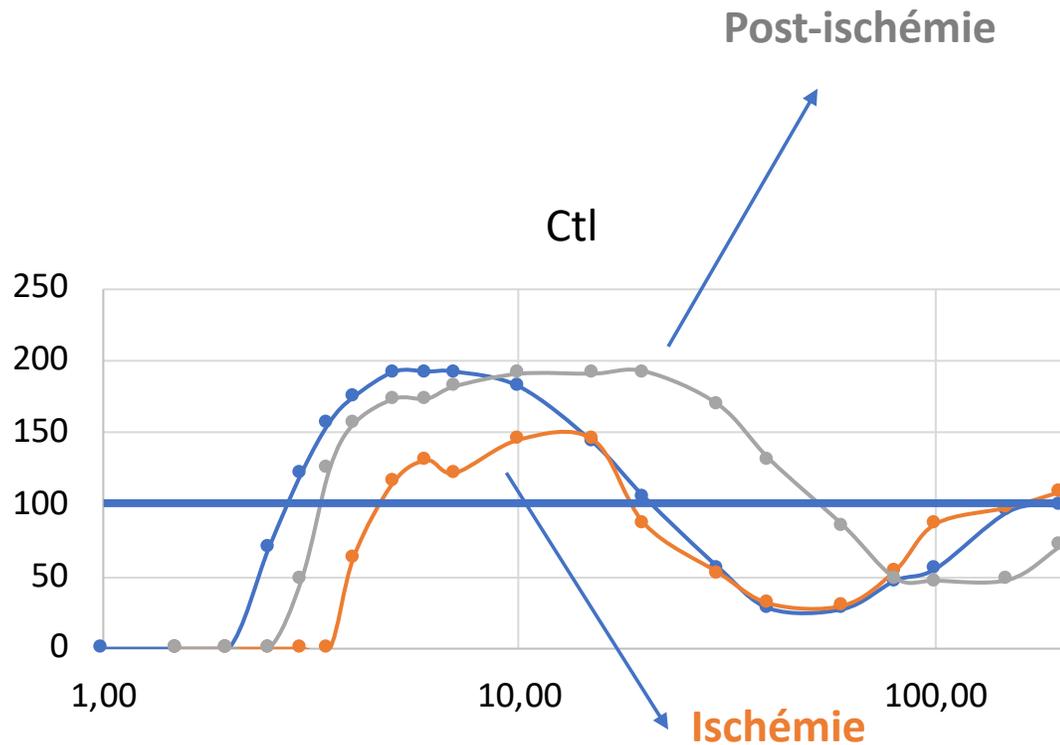


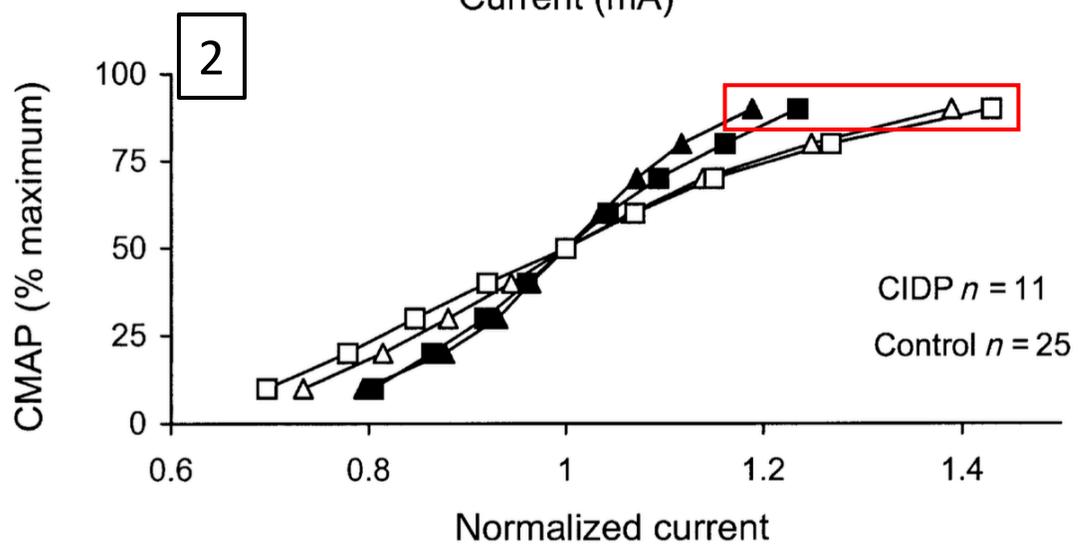
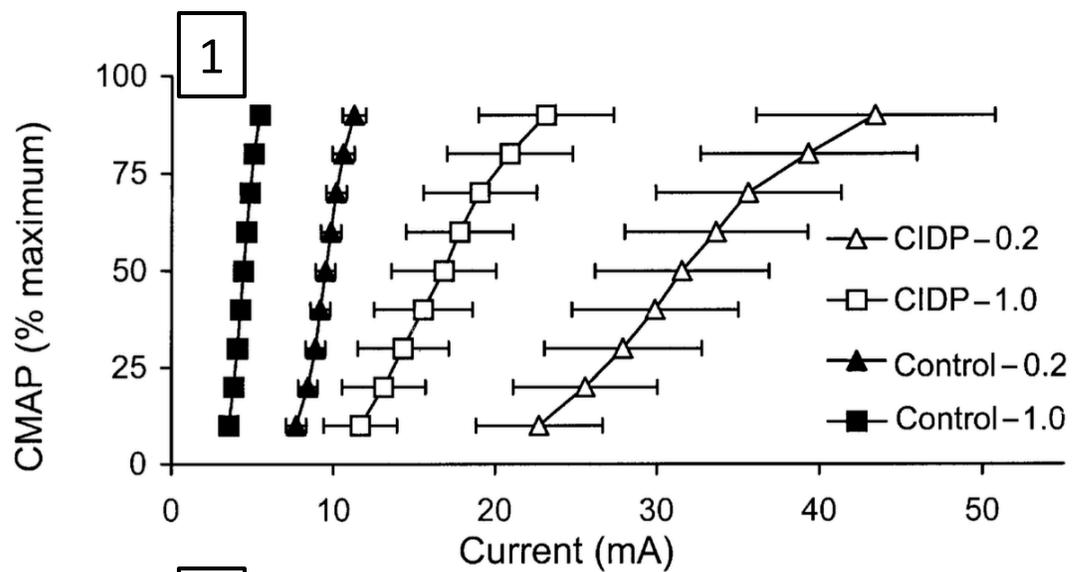
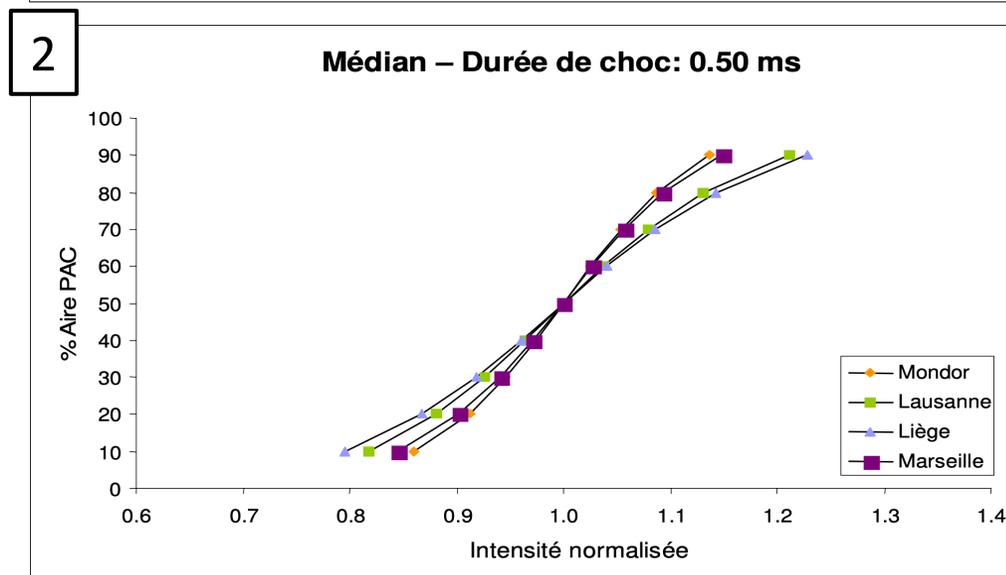
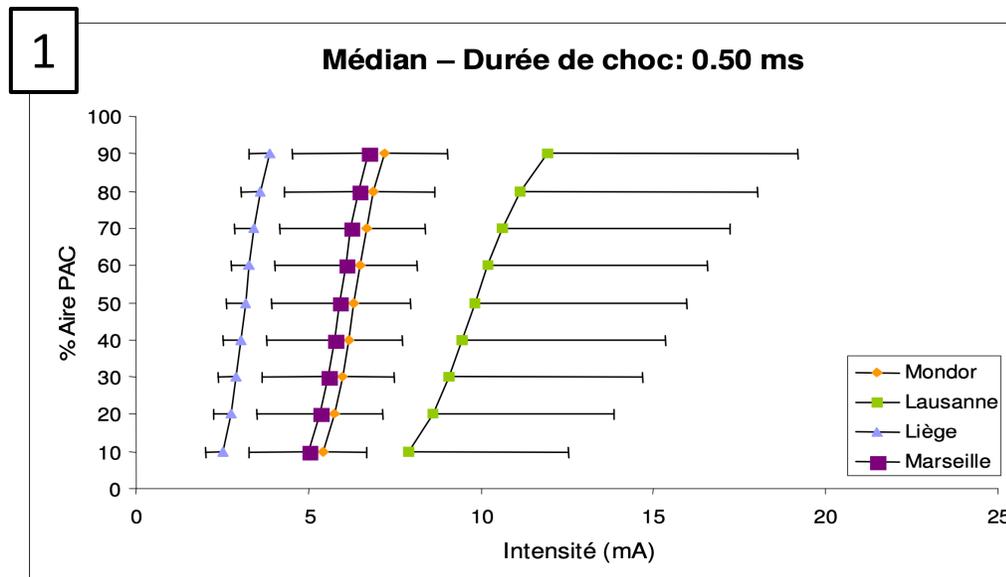


Intensité du choc conditionnant = **supramaximale** ; Intensité du choc test : **i40**

Sujets contrôles (n=11)

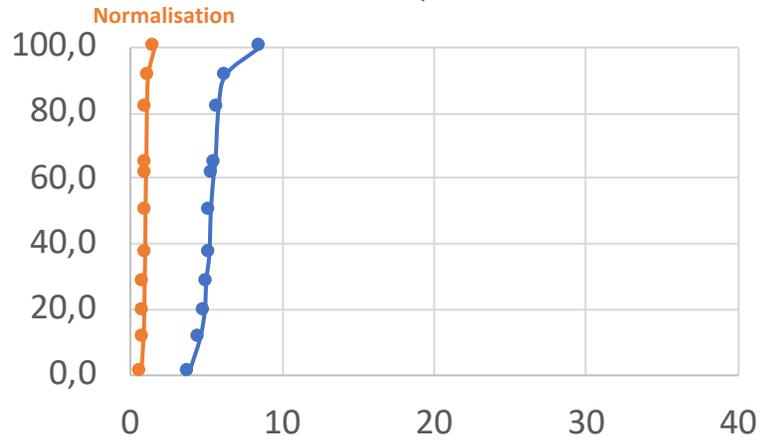




A**B**

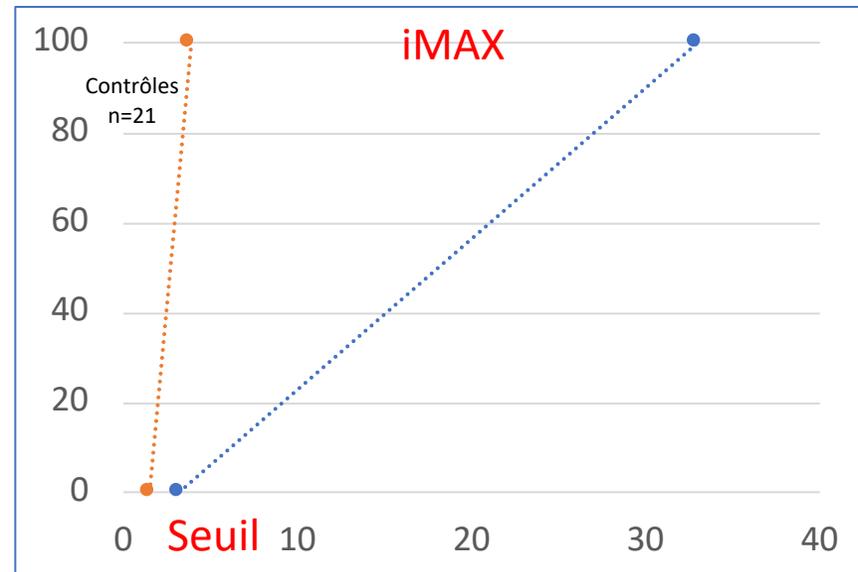
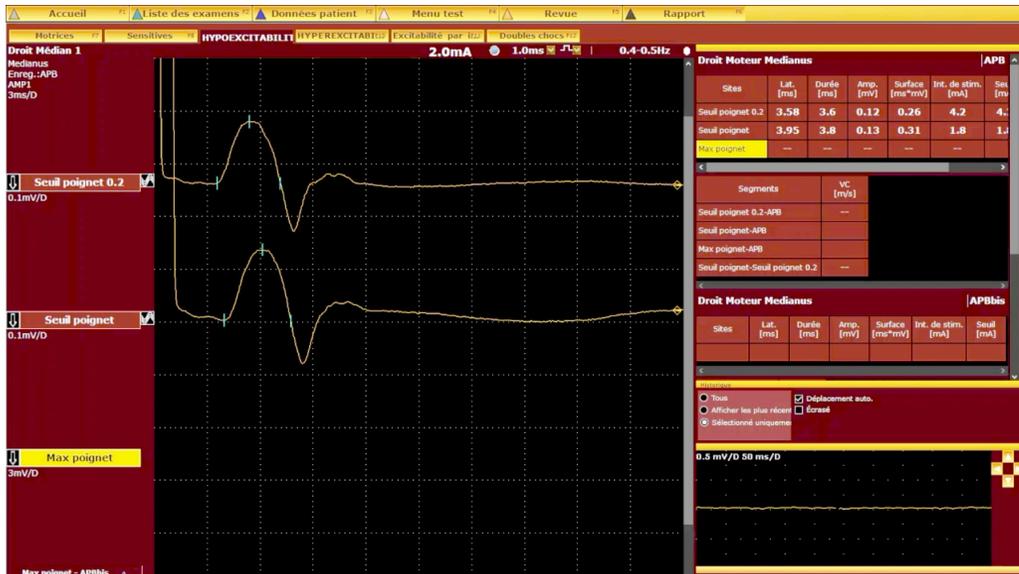
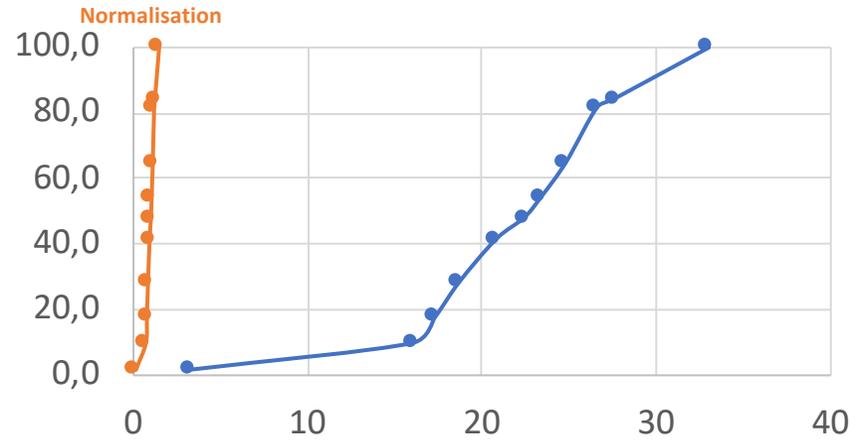
Sujet contrôle

Stimulus/Réponse
durée = 0,2 ms

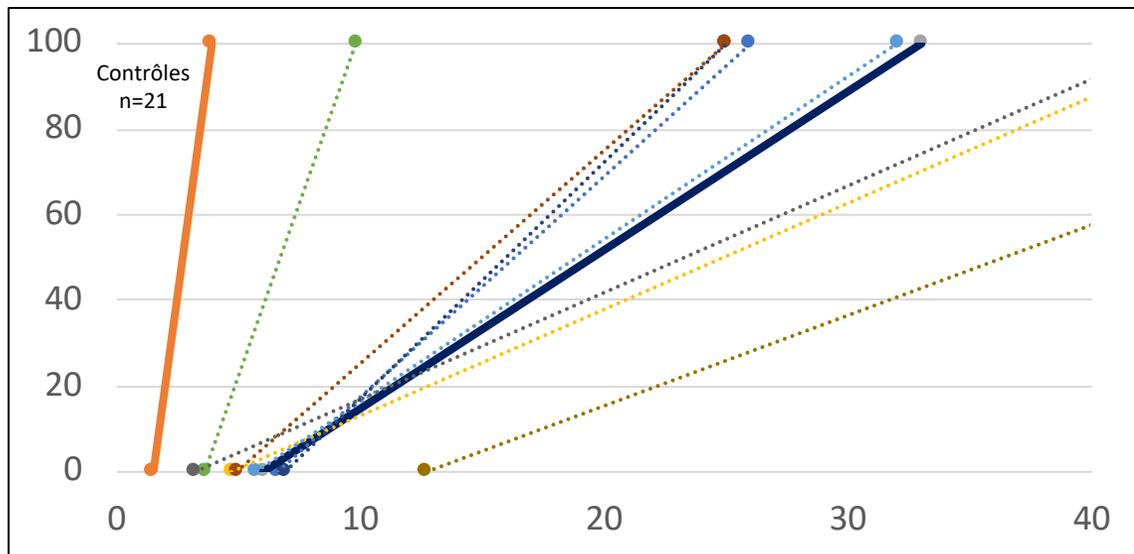


CMT

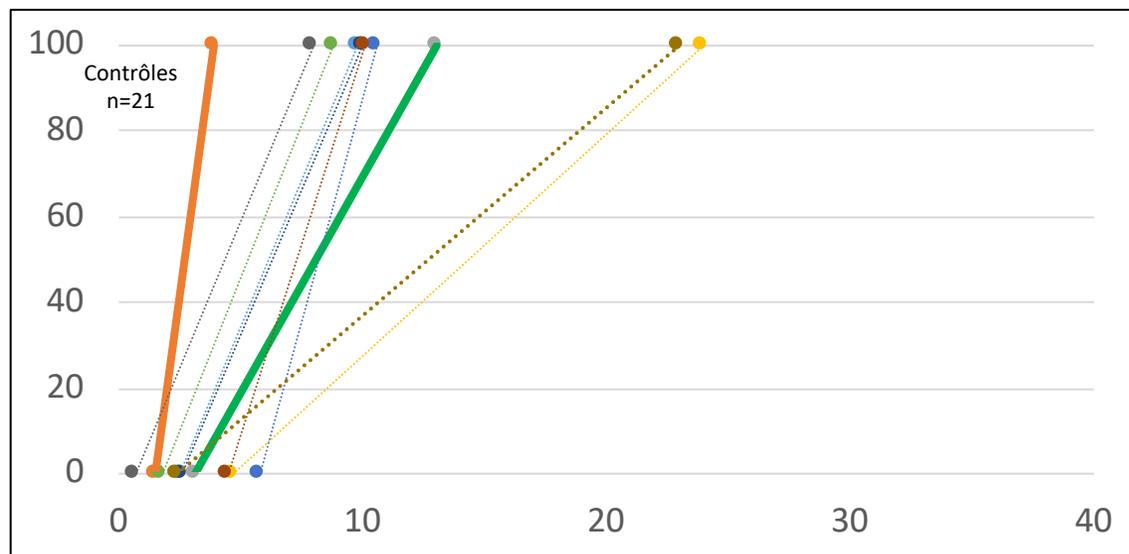
Stimulus/Réponse
durée = 1 ms



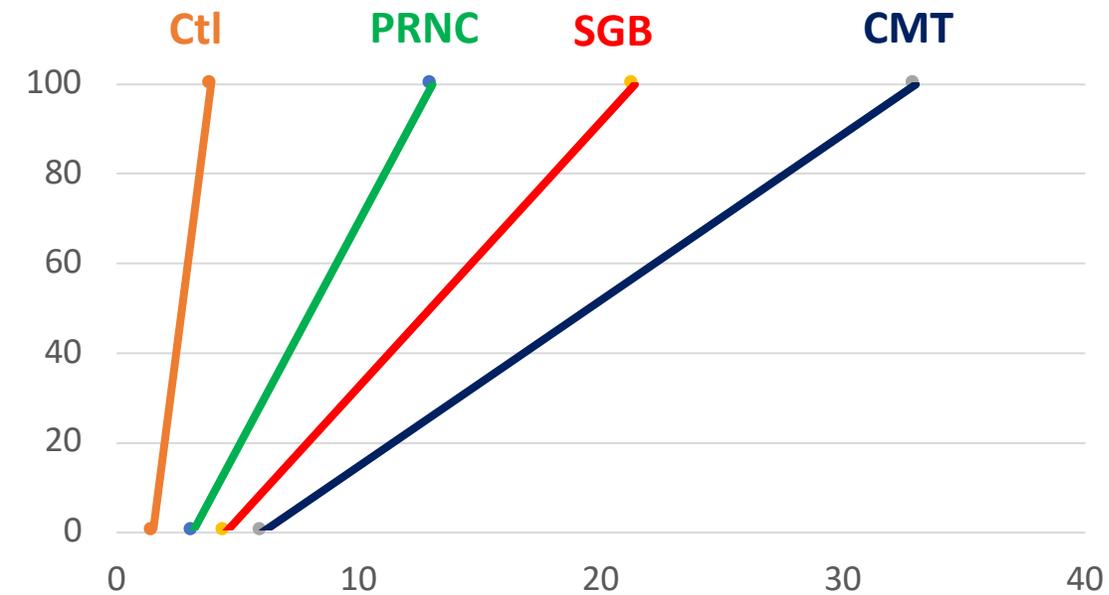
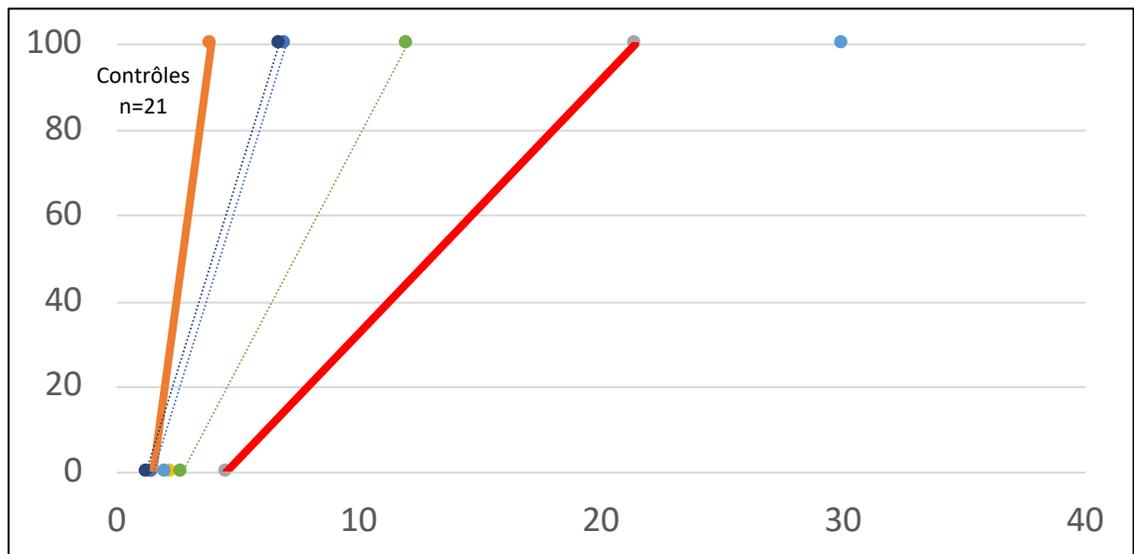
CMT (n=8)

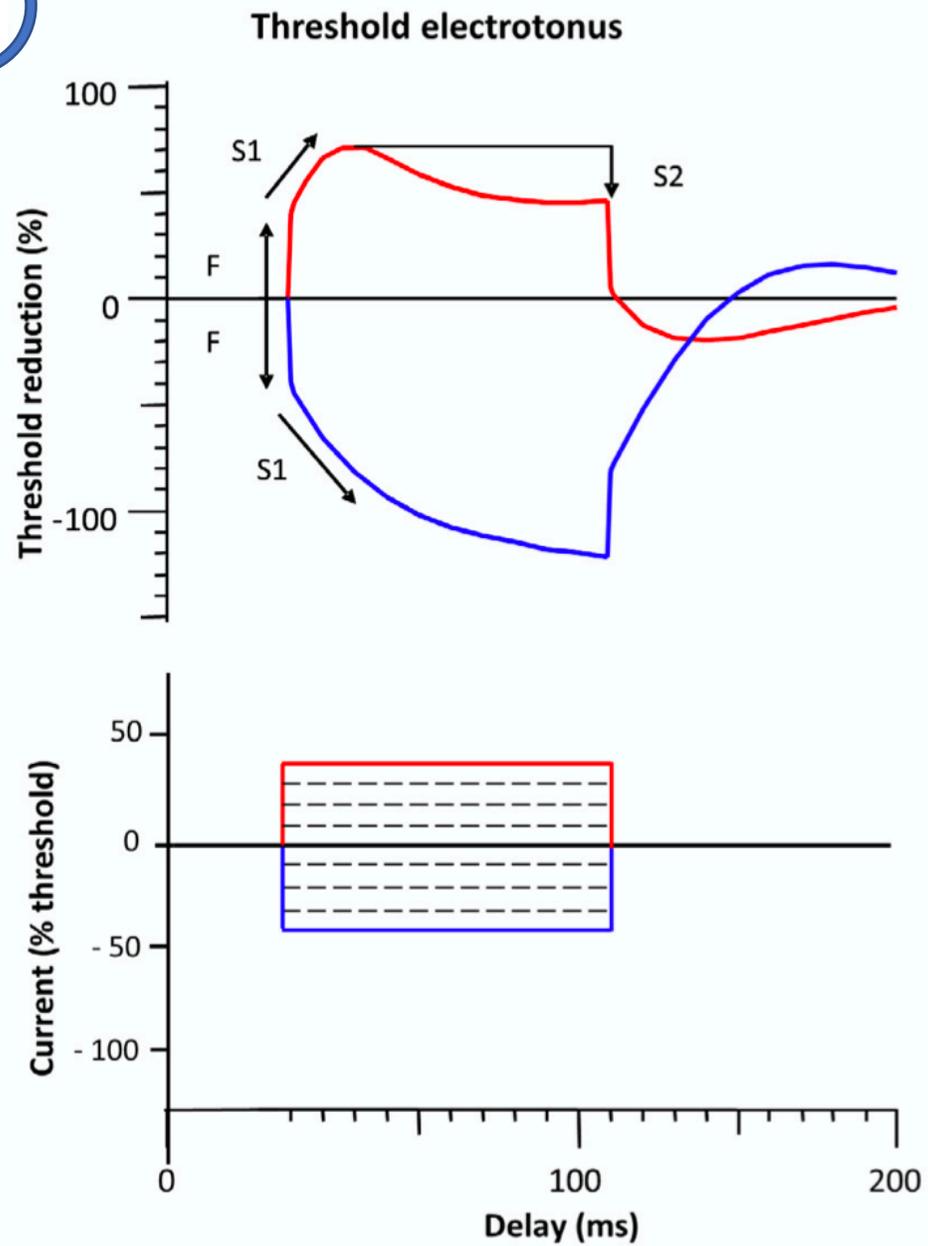
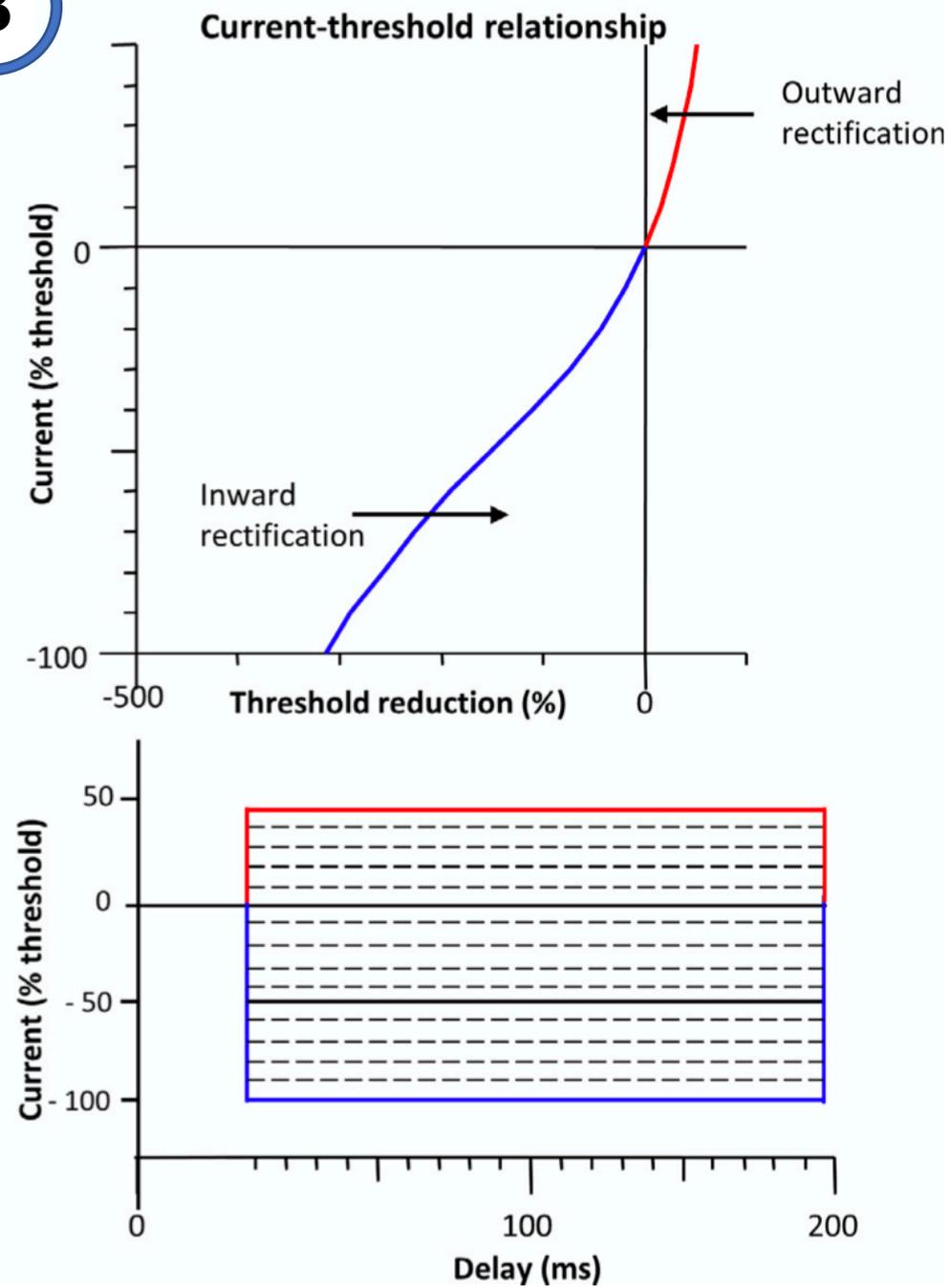


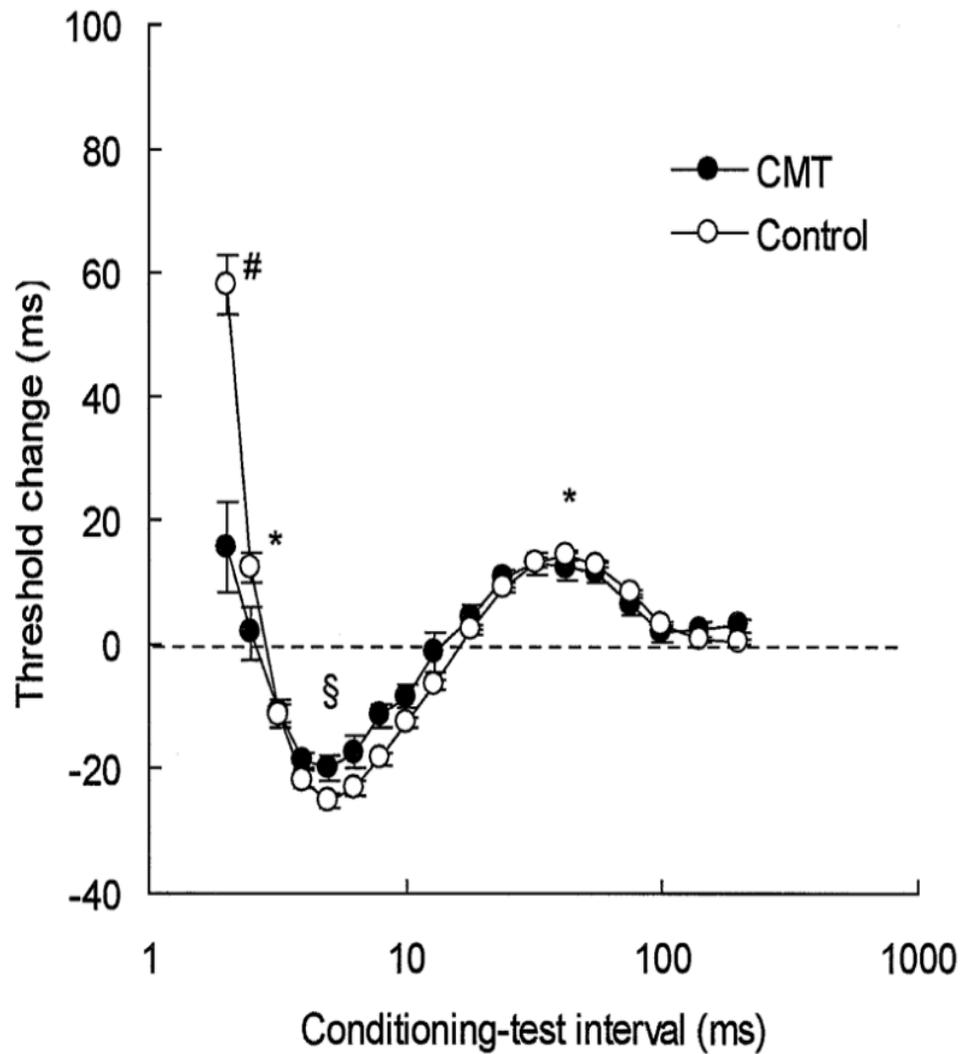
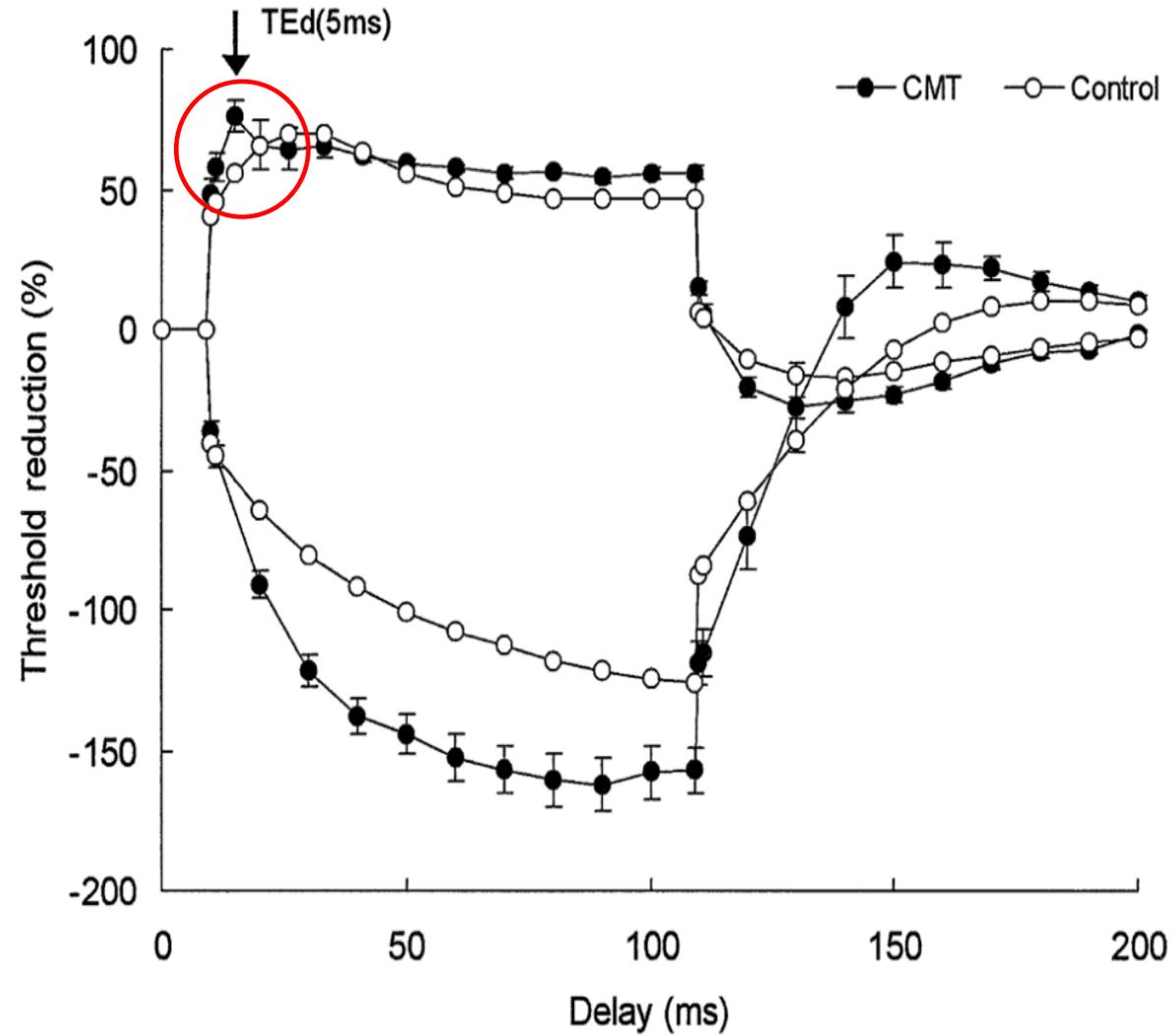
PRNC (n=8)



SGB (n=5)



A**B**

A**B**

Diagnosis and treatment of axonal excitability: Consensus guidelines

Matthew C. Kiernan^{a,*}, Hugh Bostock^b, Susanna B. Park^a, Ryuji Kaji^c, Christian Krarup^d, Arun V. Krishnan^e, Satoshi Kuwabara^f, Cindy Shin-Yi Lin^a, Sonoko Misawa^f, Mihai Moldovan^d, Jiaying Sung^g, Steve Vucic^h, Brian J. Waingerⁱ, Stephen Waxman^{j,k}, David Burke^a

^aBrain and Mind Centre, University of Sydney and Institute of Clinical Neurosciences, Royal Prince Alfred Hospital, Sydney 2006, Australia

^bUCL Queen Square Institute of Neurology, London WC1N 3BG, United Kingdom

^cNational Utano Hospital, 8-Narutaki Ondoyamacho, Ukyoku, Kyoto 616-8255, Japan

^dDepartment of Neuroscience, University of Copenhagen and Department of Clinical Neurophysiology, Rigshospitalet, Blegdamsvej 9, 2100 Copenhagen, Denmark

^ePrince of Wales Clinical School, University of New South Wales, Sydney, Australia

^fDepartment of Neurology, Graduate School of Medicine, Chiba University, Japan

^gTaipei Medical University, Wanfang Hospital, Taipei, Taiwan

^hDepartment of Neurology, Westmead Hospital, Western Clinical School, University of Sydney, Australia

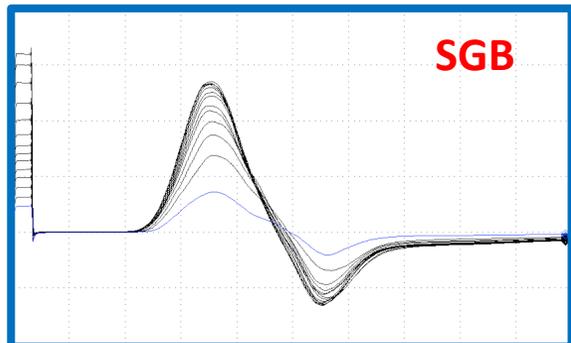
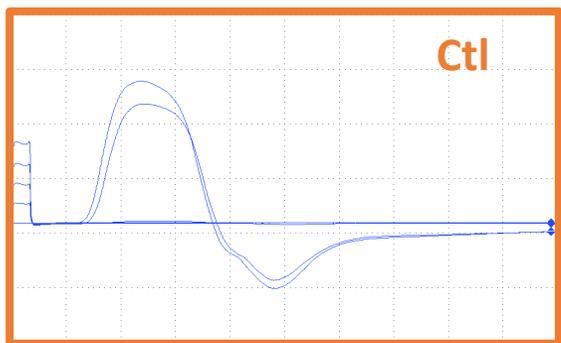
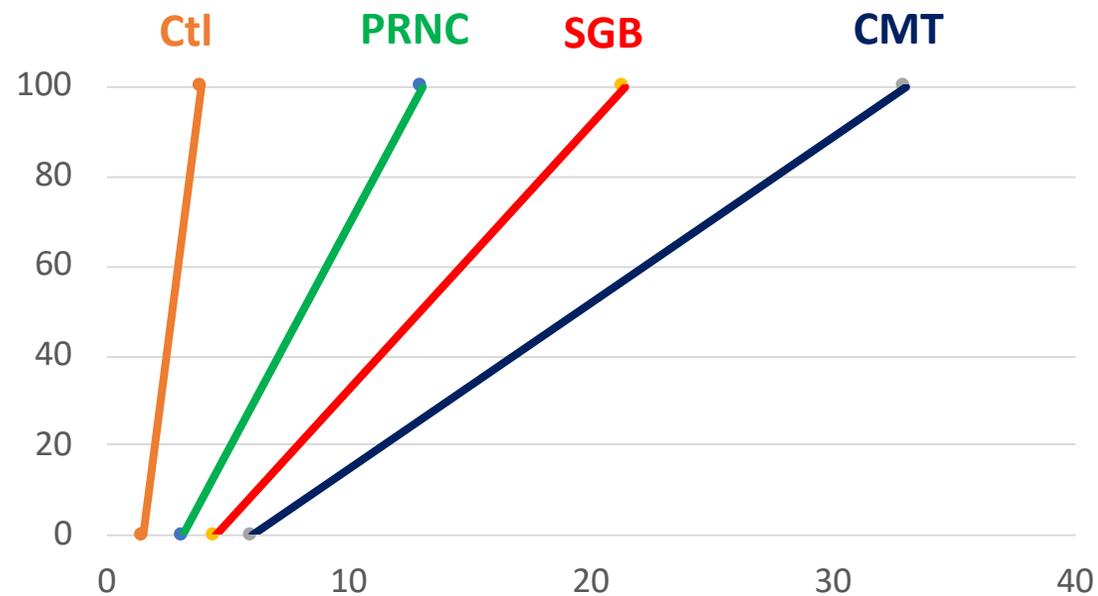
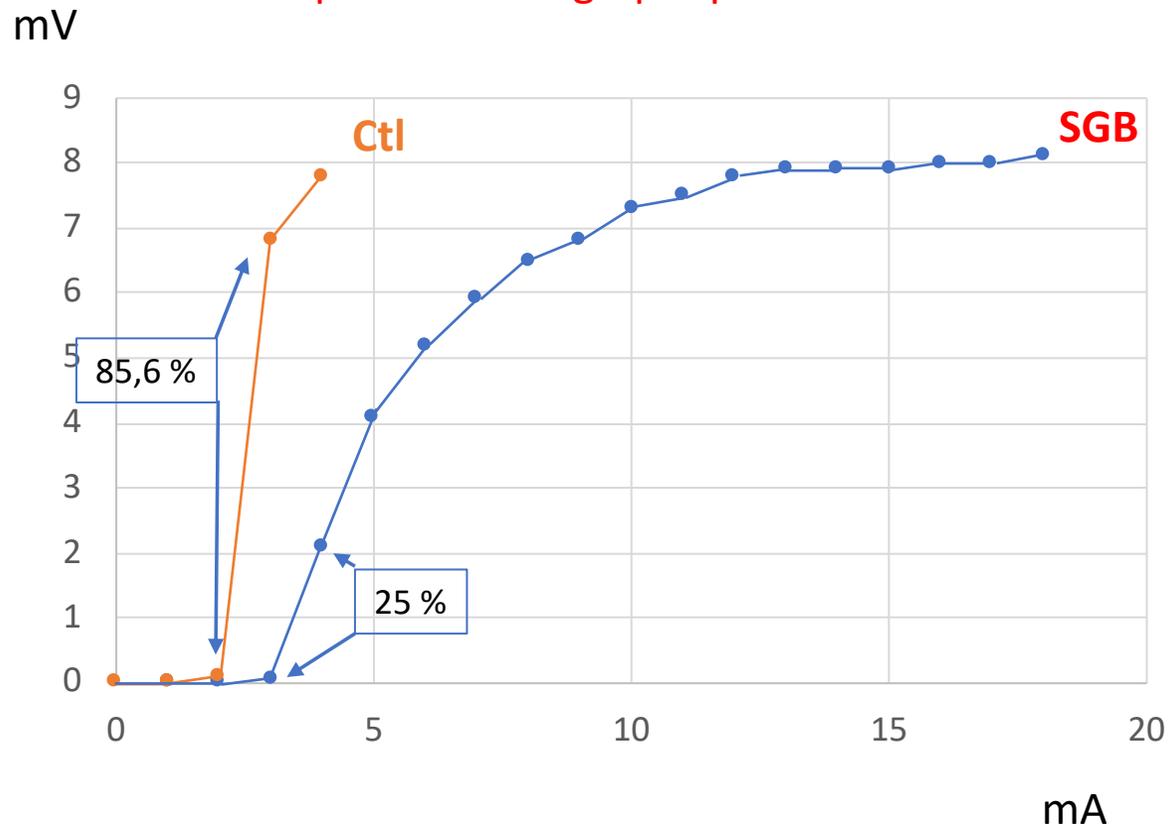
ⁱDepartment of Neurology and Anesthesiology, Critical Care & Pain Medicine, Massachusetts General Hospital, Boston, MA 02114, USA

^jDepartment of Neurology, Yale Medical School, New Haven, CT 06510, USA

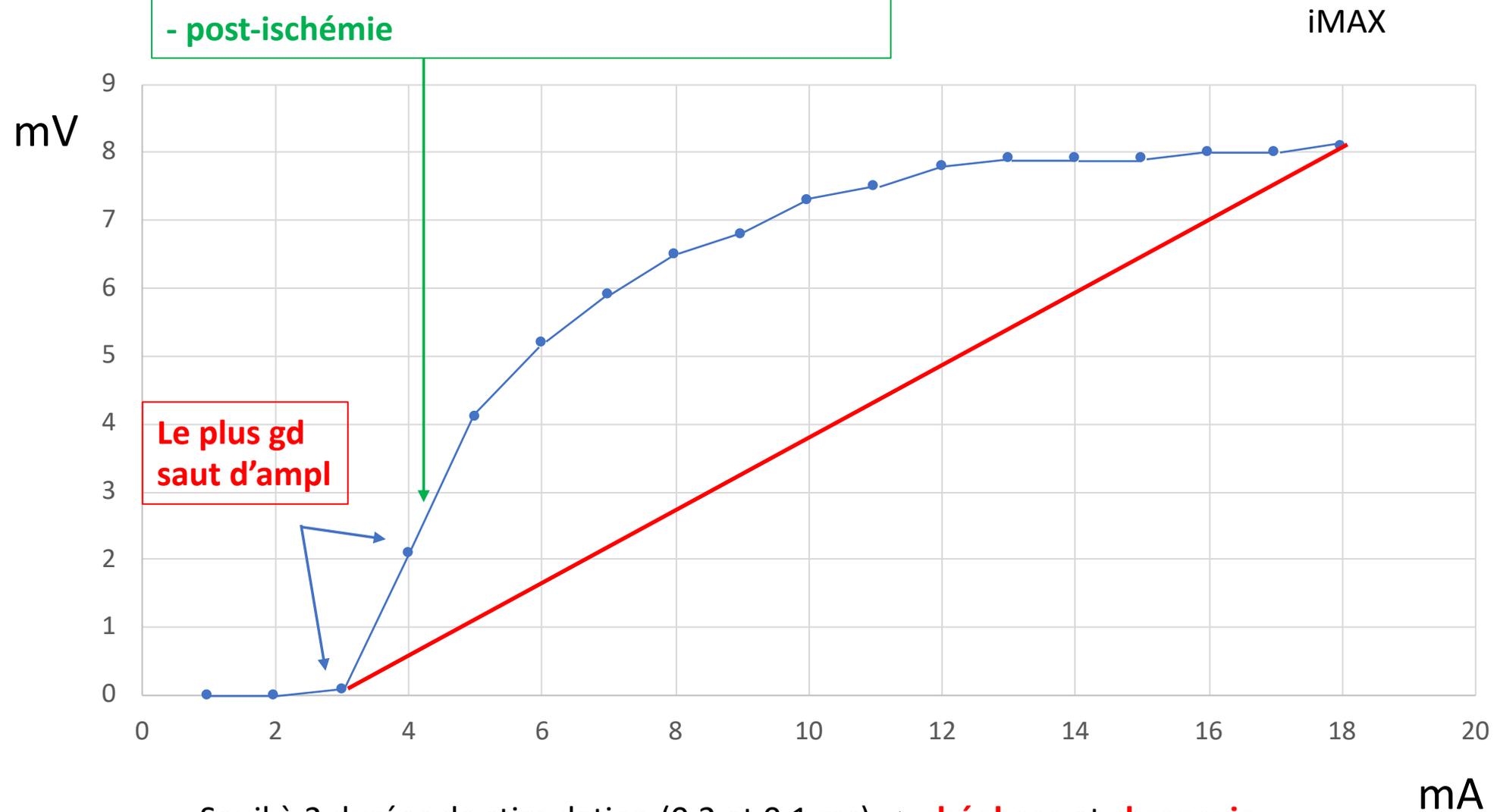
^kNeurorehabilitation Research Center, Veterans Affairs Hospital, West Haven, CT 06516, USA

GBS is currently classified as acute inflammatory demyelinating polyneuropathy (AIDP), the classical demyelinating form, or acute motor (and sensory) axonal neuropathy (AMAN/AMSAN), an axonal variant. The pathology in the two subtypes is different, and so too are the excitability changes. Three studies have compared excitability properties in AIDP and AMAN/AMSAN (Kuwabara et al., 2002a, 2003; Pyun et al., 2017). All excitability indices (τ_{SD} , threshold electrotonus, recovery cycle, and current/threshold relationship) were normal, findings explained by demyelinating lesions selectively affecting the nerve terminals and roots, sparing the wrist portion where testing was performed.

Représentation graphique de l'iUP

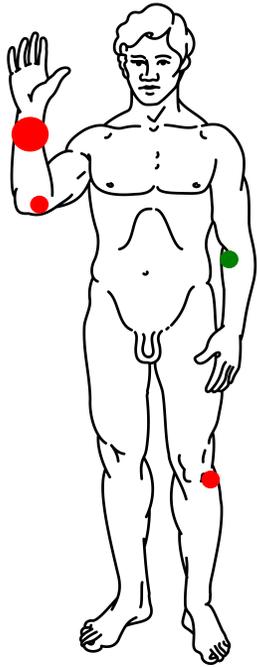


Cycle de récupération de l'excitabilité nerveuse
- condition basale
- 5' d'ischémie
- post-ischémie

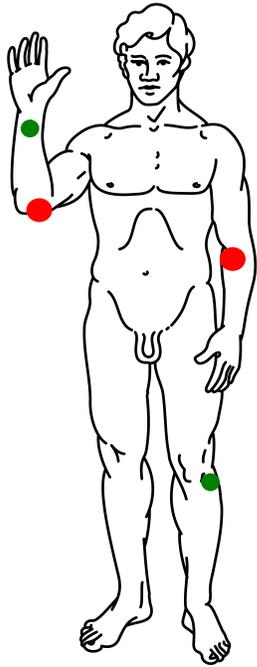


Seuil à 2 durées de stimulation (0,2 et 0,1 ms) => **rhéobase** et **chronaxie**

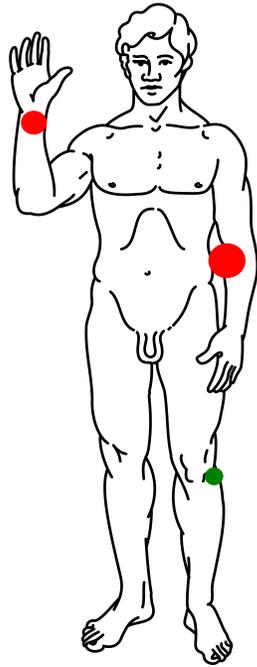
mA



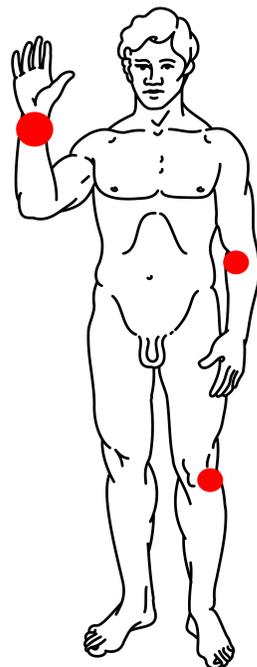
SGB



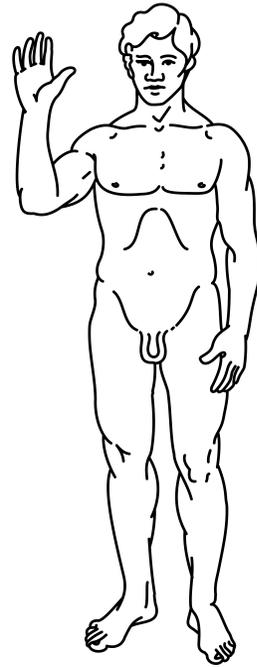
PRNC



MMN

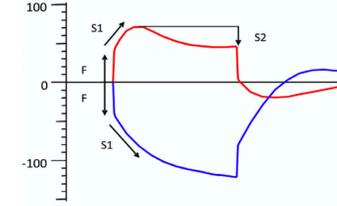
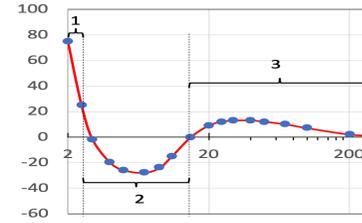
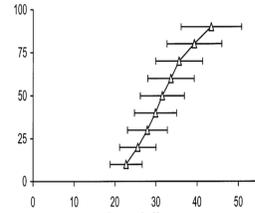
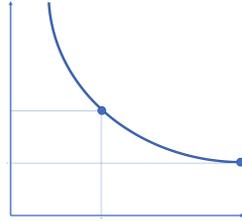


CMT1a



SLA ?

SYNTHESE



Mécanismes	Na_p nodaux L'augmentation de la conductance => augmentation de la chronaxie	Démyélinisation, œdème endoneural, altération des conductances nodales => déplacement -> droite et souvent pente réduite	Na_t nodaux (inactivation) => PR absolue Ks nodaux et paranodaux => période sous-normale tardive (NB : La période supernormale est liée à la décharge du condensateur internodal)	TEd : S1 limité par Kf paranodaux (canaux qui limitent la réexcitation) et S2 traduit l'activation des Ks nodaux TEh : inactivation des Na_p et des Ks et activation des I_H internodaux
Chaleur Exercice Post-ischémie	Chronaxie réduite par diminution de la conductance des Na_p	Augmentation des seuils	Hyperpolarisation => fermeture des Kf => cycle vers le bas => PR réduite et période supernormale augmentée	TEd et TEh : variation des seuils accrue (<i>fanning out</i>)
Froid Ischémie	Chronaxie accrue par augmentation de la conductance des Na_p	Diminution des seuils	Dépolarisation => ouverture des Kf => cycle vers le haut => PR accrue et période supernormale réduite	TEd et TEh : variation des seuils réduite (<i>fanning in</i>)
Sclérose latérale amyotrophique	Augmentation de la chronaxie (Na _p nodaux) versus défaillance globale de tous les canaux ioniques			
CMT1a	Chronaxie normale Rhéobase accrue	Augmentation des seuils => déplacement -> droite Réduction de la pente	Réduction d'amplitude de la PR relative et de la période supernormale (traduit les propriétés passives de la membrane)	TEd et TEh : variation des seuils accrue (réduction de la résistance internodale) et accommodation plus rapide au TEd (Kf)
Syndrome de Guillain-Barré	Chronaxie normale	i50 augmenté, réduction de la pente, iMAX augmenté	AMAN : allongement de la PR (Na_t nodaux) et période supernormale moins ample	TEd et TEh : absence d'anomalie
Polyradiculonévrite chronique	Chronaxie réduite Rhéobase accrue	Augmentation des seuils => déplacement -> droite Réduction de la pente	Réduction d'amplitude de la PR relative et de la période supernormale (traduit les propriétés passives de la membrane)	TEh : variation des seuils accrue

Na_p : canaux sodiques persistants ; Na_t : canaux sodiques voltage-dépendants ; Kf : canaux potassiques rapides ; Ks : canaux potassiques lents ; TEd : électrotonus dépolarisant ; TEh : électrotonus hyperpolarisant ; PR : période réfractaire