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Neural mechanisms for orienting the fovea toward a visual target

Laurent Goffart

CNRS

Institut de Neurosciences Cognitives de la Méditerranée
Marseille, France

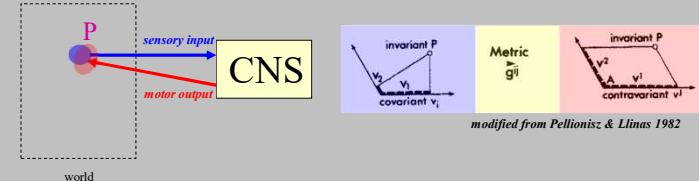


INCM

One simple question

What are the neural mechanisms that ensure the spatial congruence between :

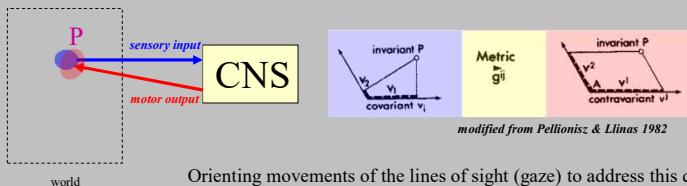
- the source of a sensory stimulus and
- the endpoint of a movement directed toward this source ?



One simple question

What are the neural mechanisms that ensure the spatial congruence between :

- the source of a sensory stimulus and
- the endpoint of a movement directed toward this source ?



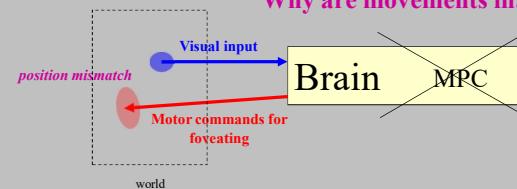
Orienting movements of the lines of sight (gaze) to address this question
 - hodology, anatomy, simplicity (2x6 muscles)
 - accurate measurements in space and time
 - possible extension to 3D

The medio-posterior cerebellum *consequence of its functional perturbation*

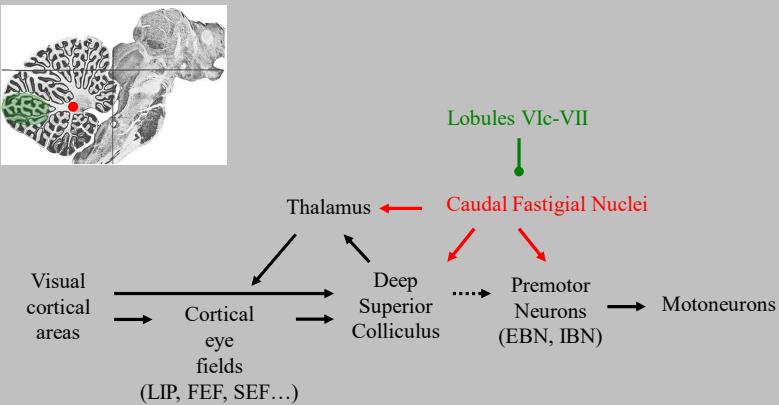
Dysfunction of MPC abolishes the spatial congruence between :

- the location of a visual event
- the endpoint of gaze shifts towards it

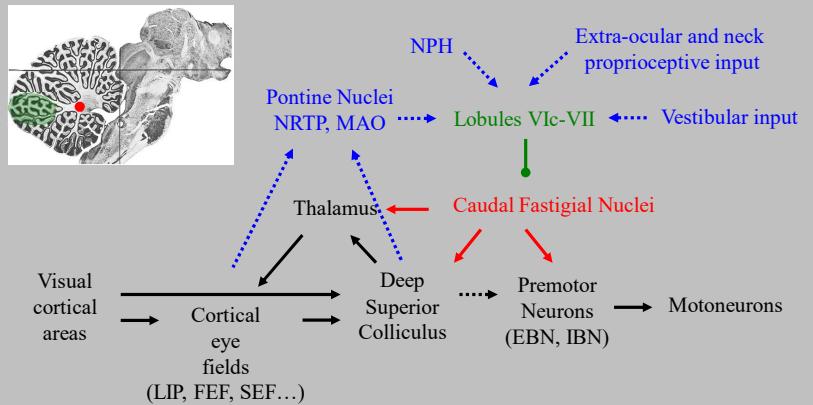
Why are movements inaccurate ?



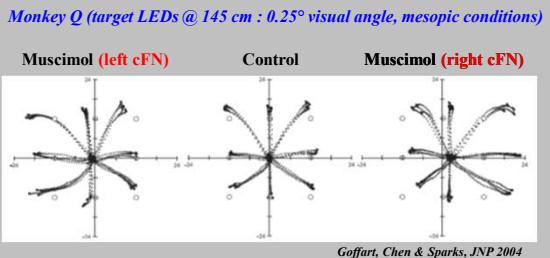
The medio-posterior cerebellum



The medio-posterior cerebellum



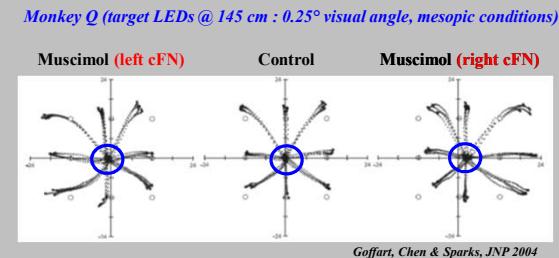
Horizontal dysmetria after cFN inactivation in the head restrained monkey



Ipsilesional saccades : horizontal component is hypermetric

Contralesional saccades : horizontal component is hypometric

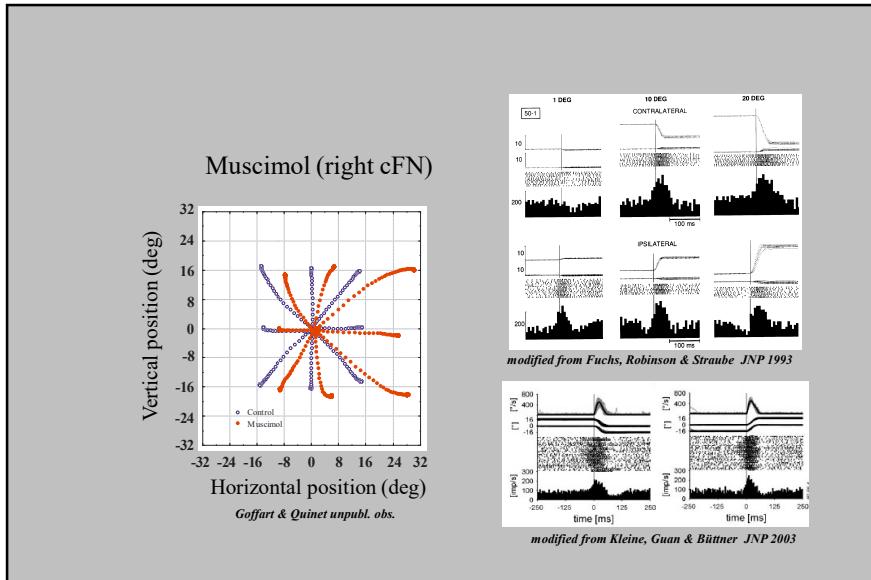
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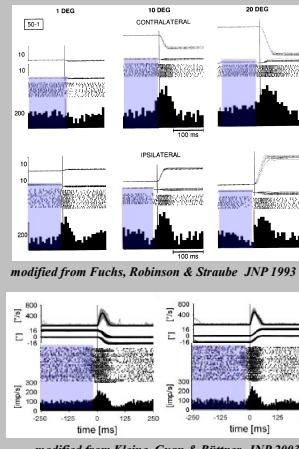
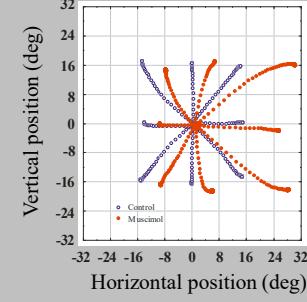
Fixation offset : ipsilesional shift in the scatter of eye position during fixation



What is the time window of the perturbation ?

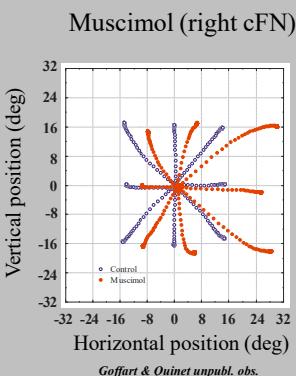
Presaccadic,

Muscimol (right cFN)

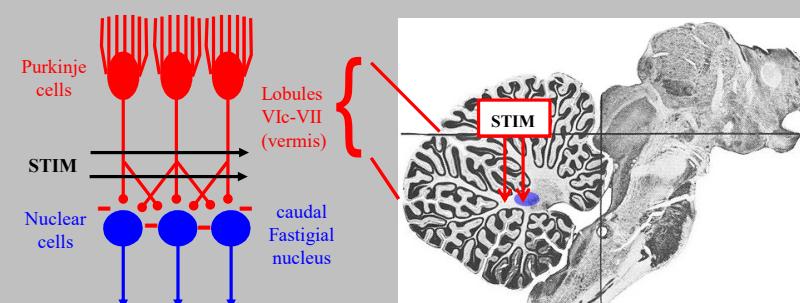


What is the time window of the perturbation ?

Presaccadic, intrasaccadic, both ?

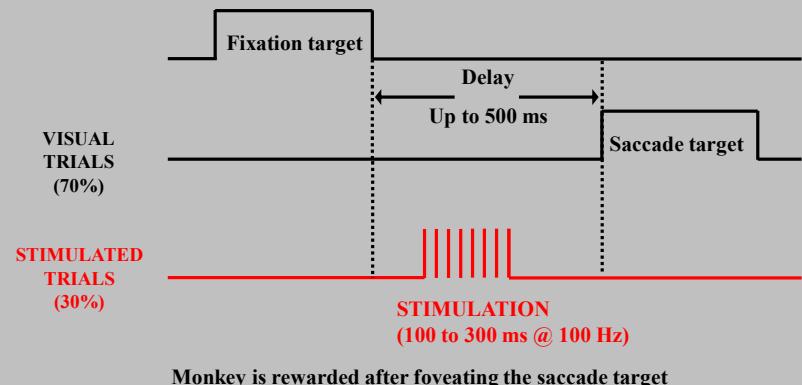


Transient inhibition of cFN activity

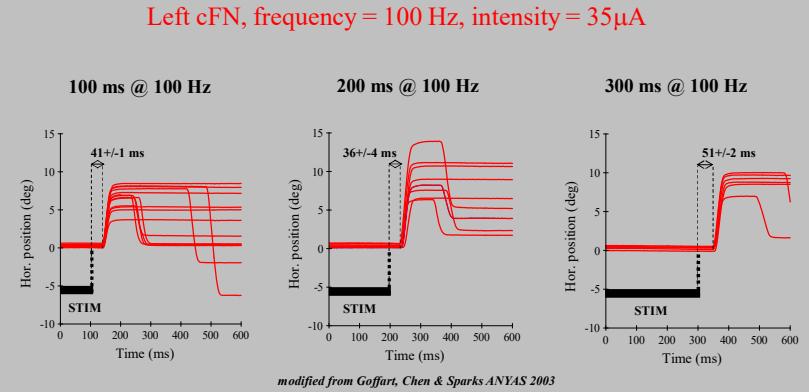


If this is true, one should be able to mimic, with a microstimulation train the effects of muscimol on saccade accuracy.

Stimulation only (control)

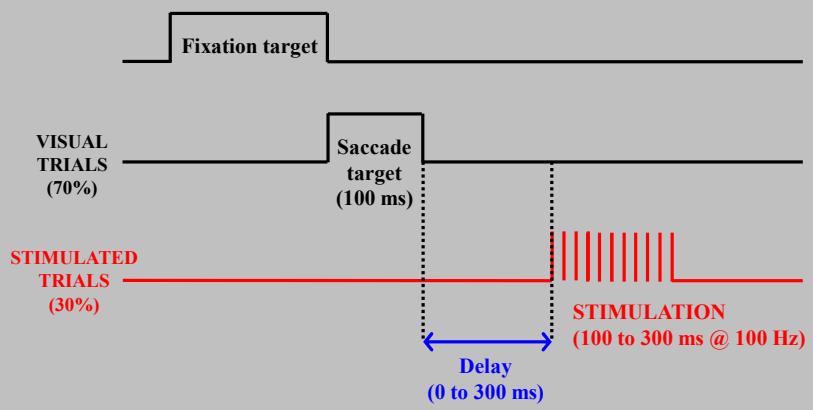


Saccades evoked BY the stimulation OFFset



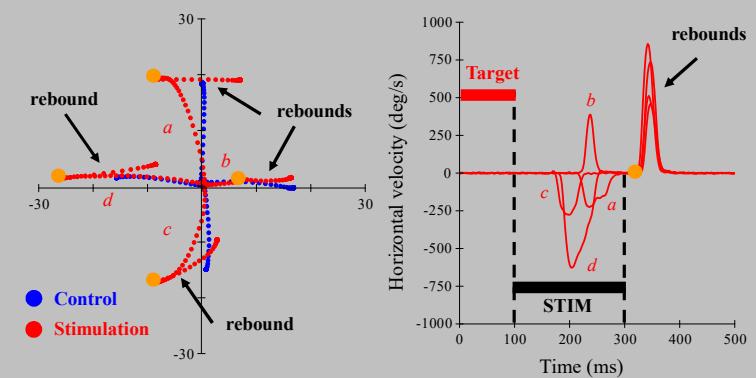
i30oct12

Microstimulation during saccade generation



Microstimulation during a visual saccade

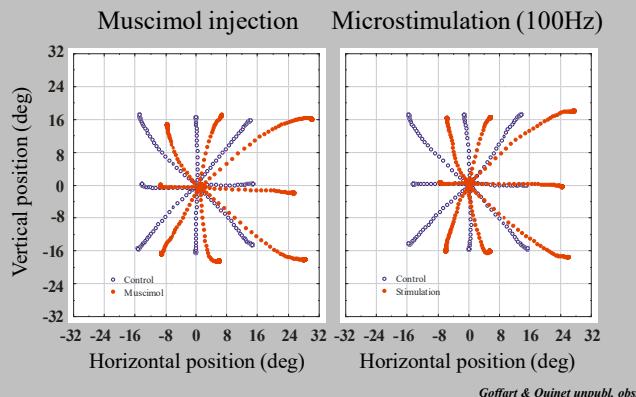
(Left cFN, 200 ms @ 100Hz)



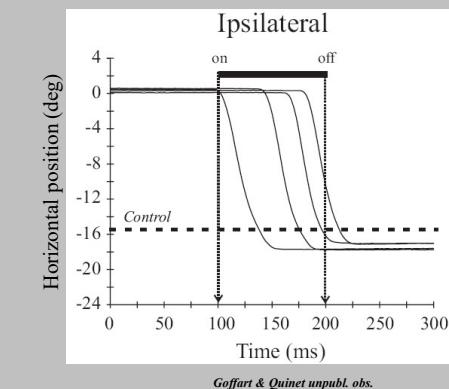
i30oct3

Inhibitory perturbation

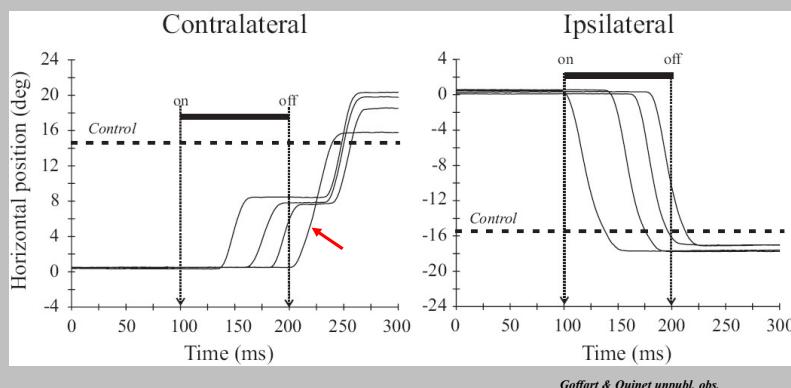
Right cFN (after removing rebounds)



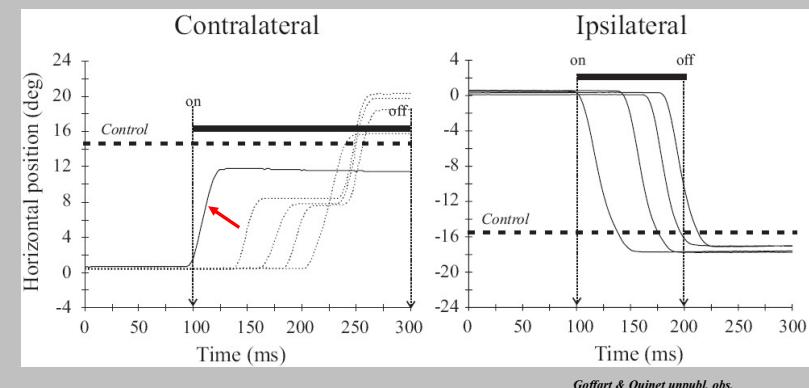
Time window of the perturbation



Time window of the perturbation

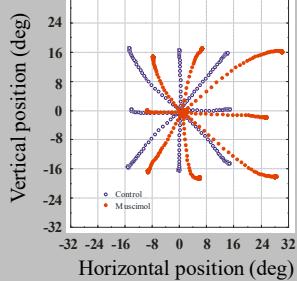


Time window of the perturbation

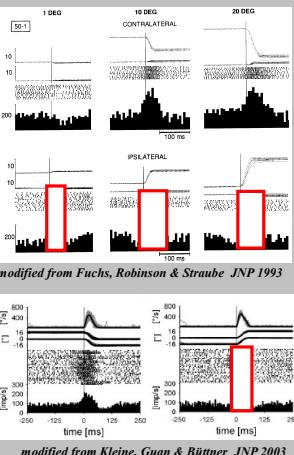


Dysmetria is due to the suppression of the intrasaccadic burst

Muscimol (right cFN)



- Suppression of the burst in one FOR leads to
- ipsilesional horizontal hypermetria
- contralateral horizontal hypometria (possible alternative)



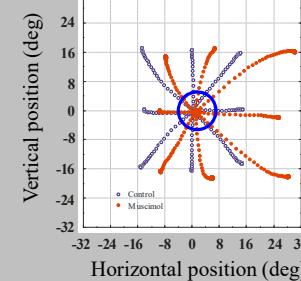
modified from Fuchs, Robinson & Straube JNP 1993

modified from Kleine, Guan & Büttner JNP 2003

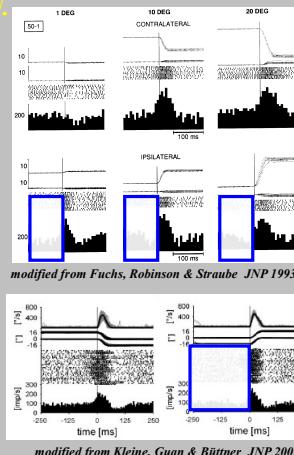
What causes the fixation offset ?

> poster Guerrasio L., Goffart L. & Büttner U.

Muscimol (right cFN)



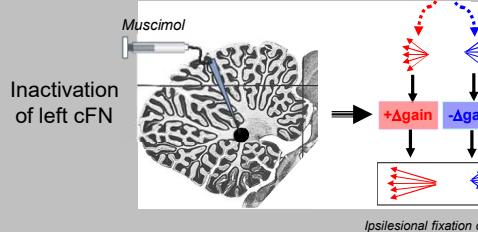
- Suppression of sustained presaccadic activity is responsible for the fixation offset



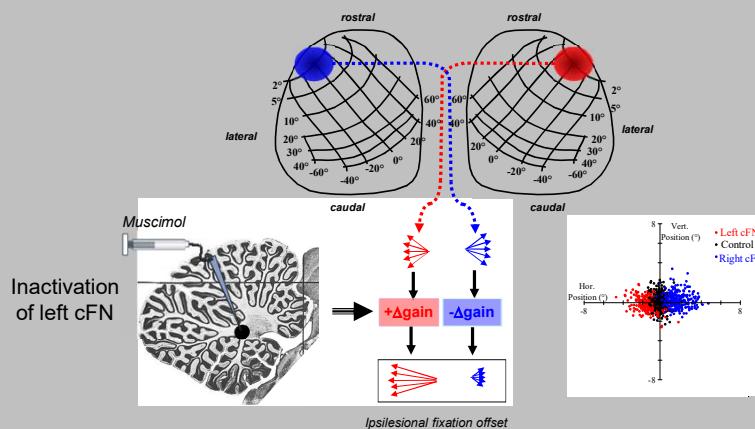
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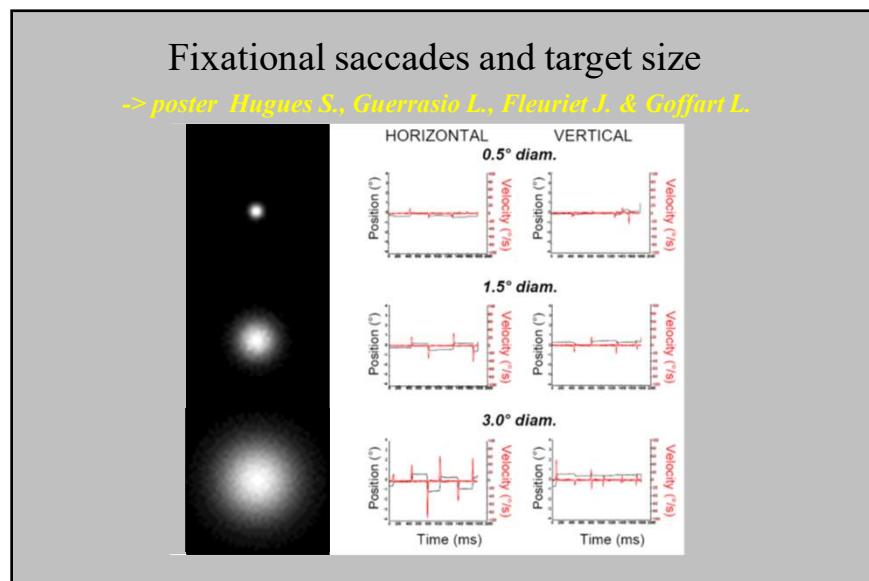
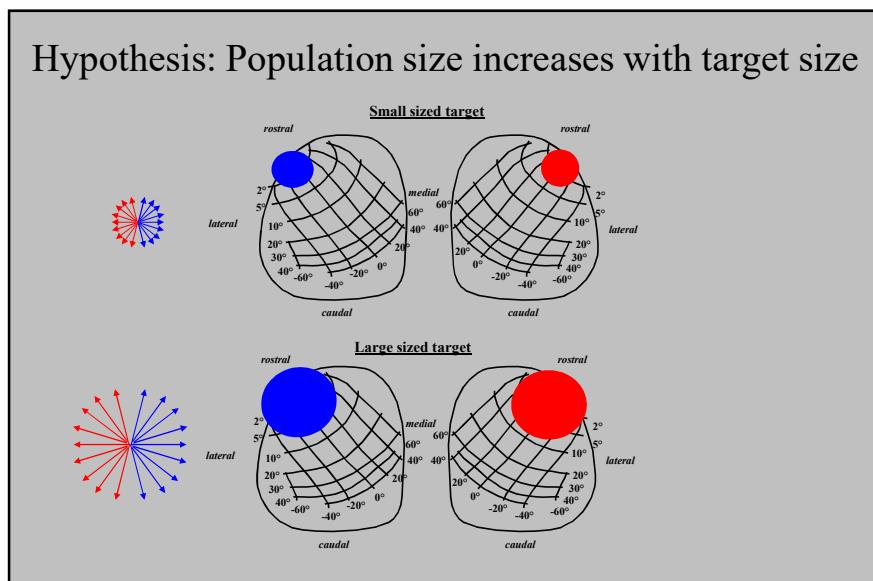
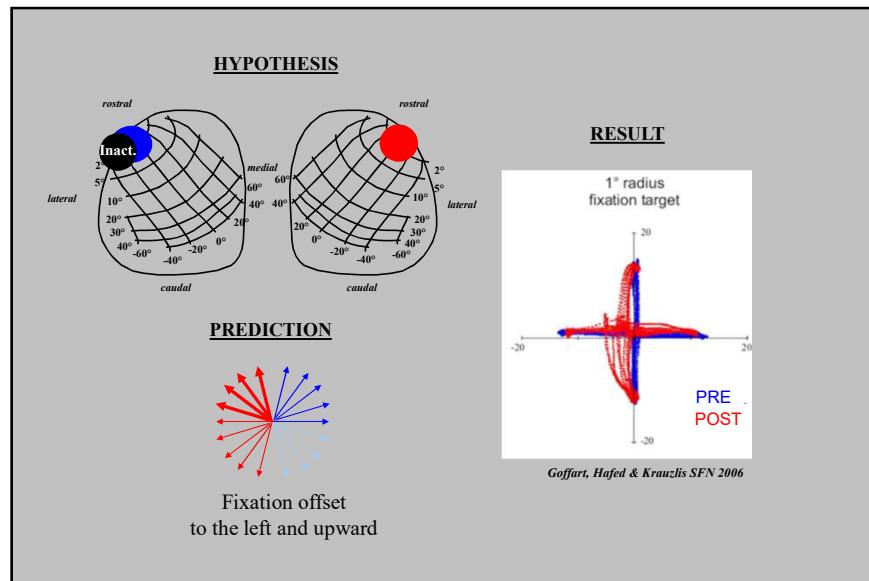
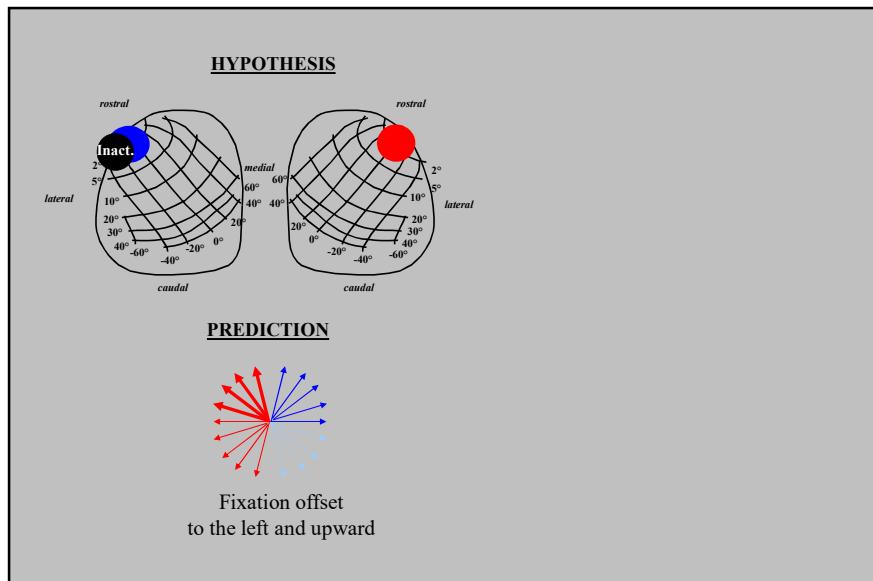
Ipsilesional fixation offset : a problem in matching gaze and target directions

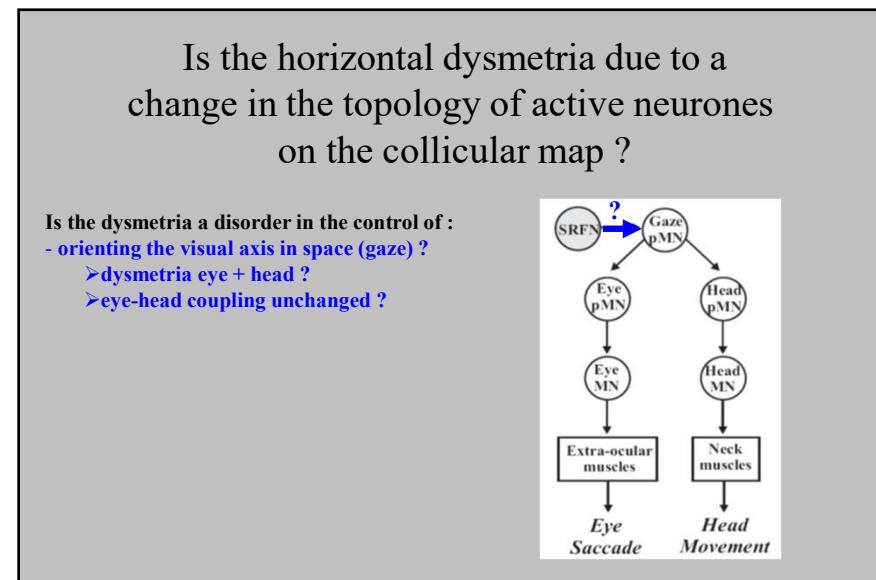
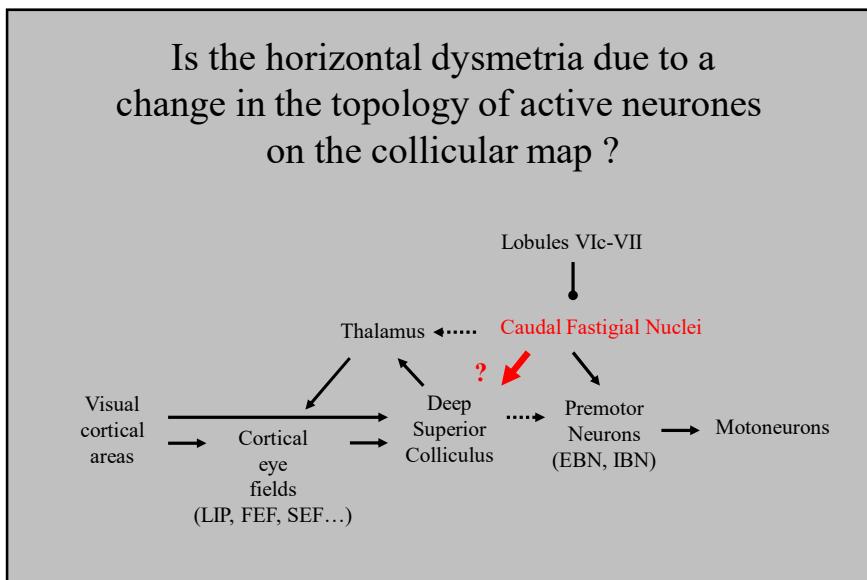
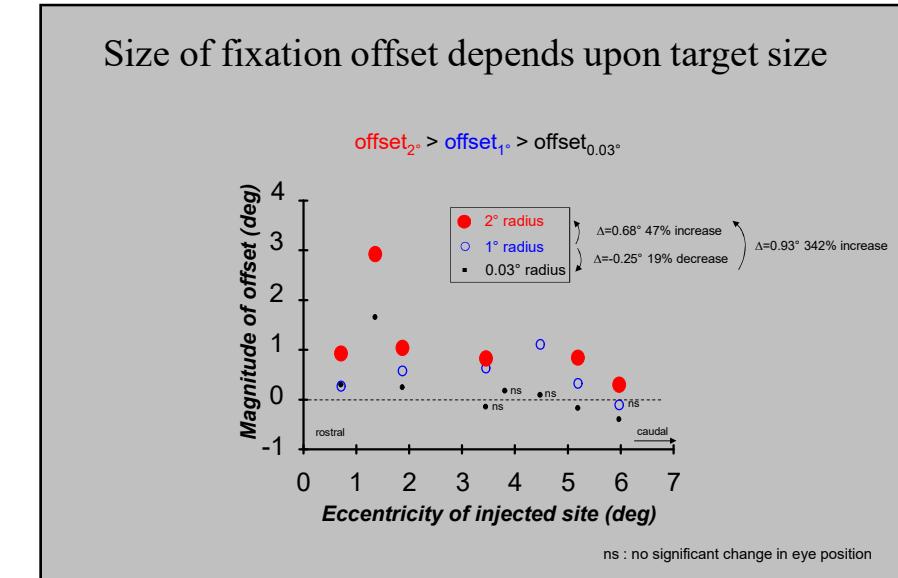
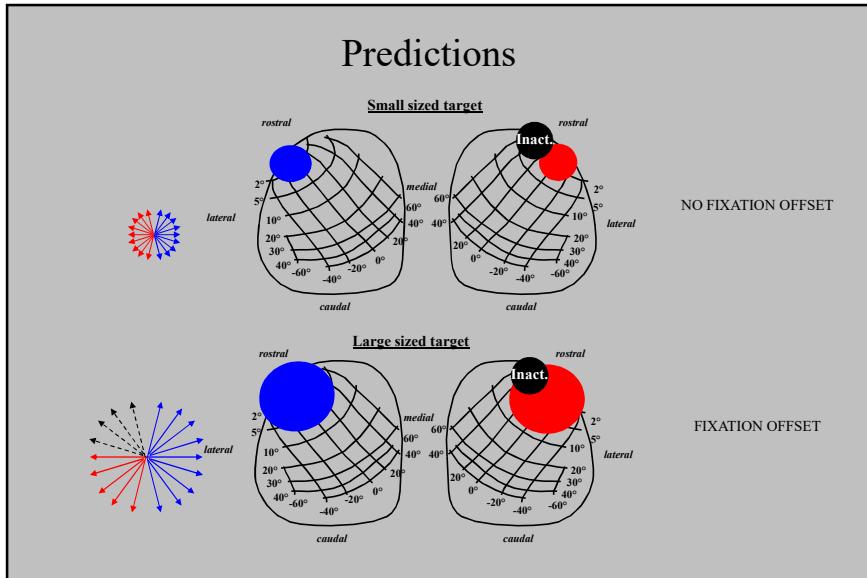
Commands
for orienting the fovea
toward a central target



Matching gaze and target directions during fixation



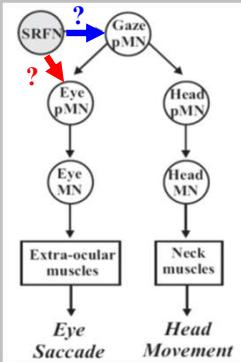




Is the horizontal dysmetria due to a change in the topology of active neurones on the collicular map ?

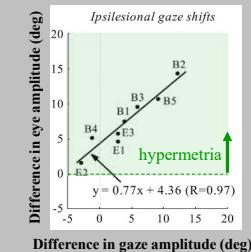
Is the dysmetria a disorder in the control of :

- orienting the visual axis in space (gaze) ?
 - dysmetria eye + head ?
 - eye-head coupling unchanged ?
- moving the eyes in the orbit
 - eye dysmetria only ?
 - eye-head coupling changed ?
 - modifications affecting the eye and not the head ?



cFN inactivation in the head unrestrained monkey

Ipsilesional movements :
- amplitude of eye component increased

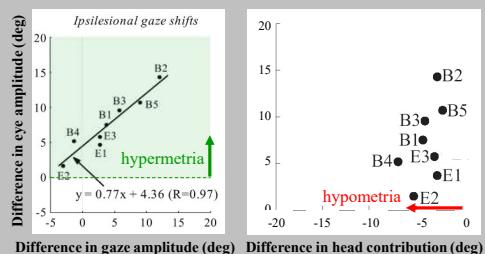


modified from Quinet & Goffart JNP 2005

cFN inactivation in the head unrestrained monkey

Ipsilesional movements :
- amplitude of eye component increased
- amplitude head component reduced

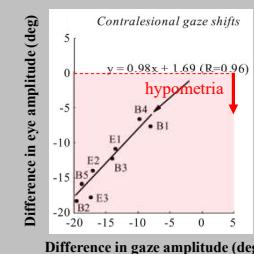
uncorrelated changes



modified from Quinet & Goffart JNP 2005

cFN inactivation in the head unrestrained monkey

Contralesional movements :
- amplitude of eye component reduced
- amplitude of head component reduced



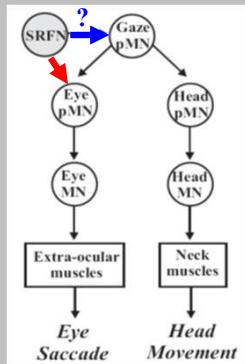
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Is the dysmetria a disorder in the control of :

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 - > dysmetria eye + head ? **NO**
 - > eye-head coupling unchanged ?

- moving the eyes in the orbit
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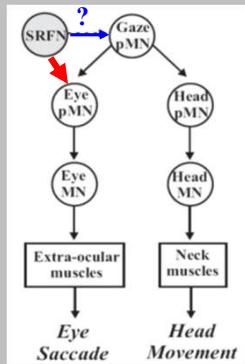


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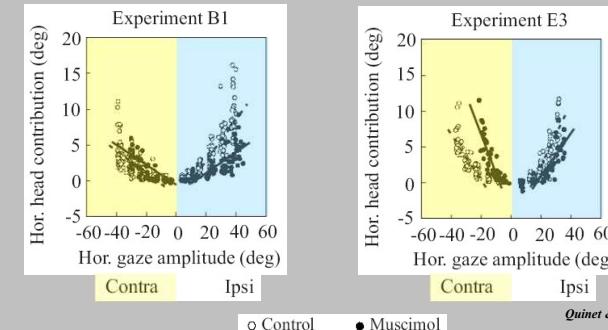
- moving the eyes in the orbit
 - > eye dysmetria only ? **YES**
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Eye-head coupling is modified

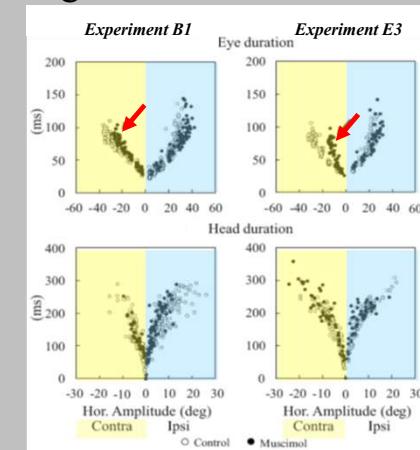
Ipsi : head contributes less

Contra : head contributes more



Quinet & Goffart JNP 2007

Slowing of contralateral saccades
No change in head movement velocity



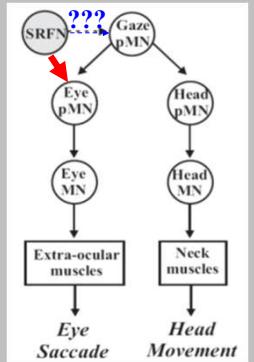
Quinet & Goffart JNP 2007

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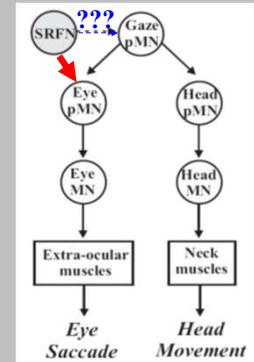
Eye-in-space or eye-in-head ?

Does the stimulation elicit head movements?

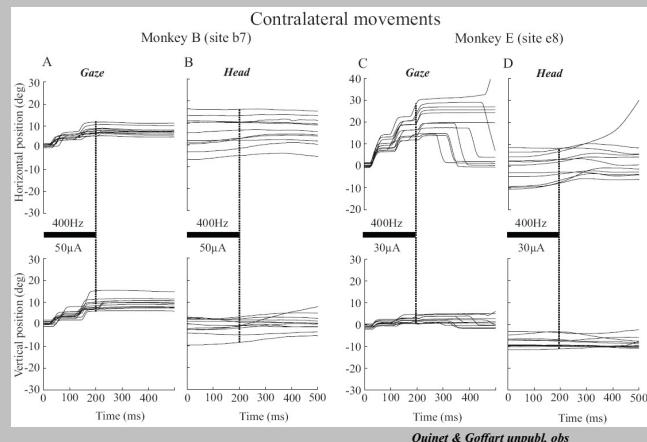
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 - > modifications affecting the eye and not the head ? YES



Microstimulation in the head unrestrained monkey



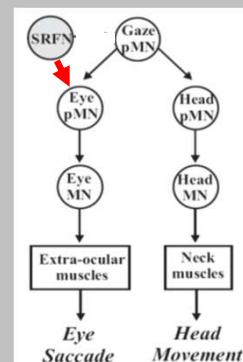
Fastigial oculomotor region (FOR)

Pharmacological inactivation

Electrical microstimulation

Anatomical studies

Robinson, Phillips & Fuchs *J.Comp.Neurol.* 1994



Conclusions

Contribution of an output nucleus from the medio-posterior cerebellum (Fastigial Oculomotor Region) in orienting gaze toward a stationnary visual target

- Control of the amplitude of the horizontal component
- Intrasaccadic critical period
- Influence restricted to the oculomotor system (studies in the head-unrestrained monkey)
- Participation to the mechanisms determining the horizontal direction of gaze during fixation (fixation offset)
- Bilateral influence at different levels of the oculomotor system (studies of the coupling between H and V components)

Acknowledgments

Work on the caudal Fastigial Nucleus:

Julie Quinet, Eye-head coordination lab, Rochester, USA
David L. Sparks, Division of Neuroscience, Houston, USA

Work on the rostral Superior Colliculus:

Rich J. Krauzlis, Salk Institute for Biological Studies, La Jolla, USA
Ziad Hafed, Salk Institute for Biological Studies

