

The fastigial oculomotor region and the cerebellar control of gaze orientation

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DYVA

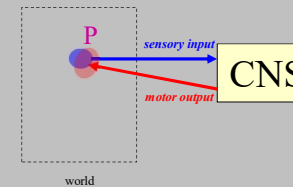
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Visuomotor spatial congruence

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

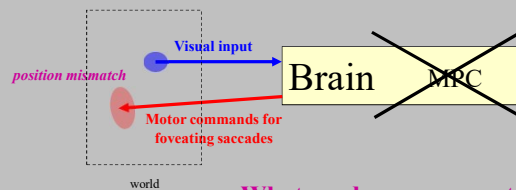
- the source of a sensory stimulus and
- the endpoint of a movement directed toward this source ?



The medio-posterior cerebellum and dysmetria

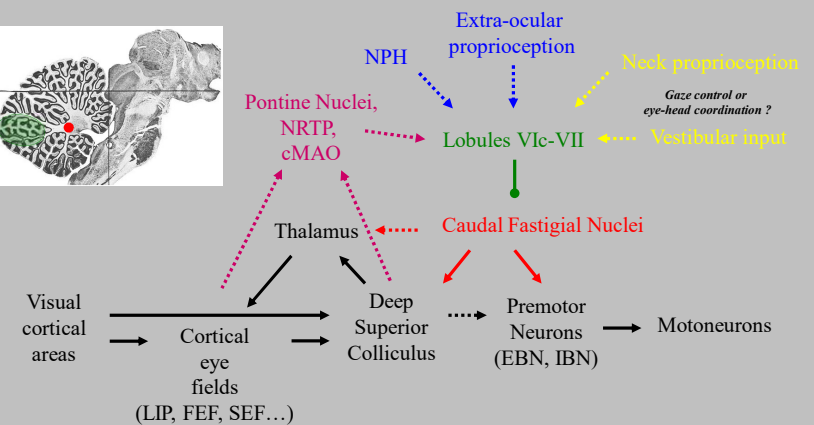
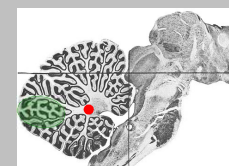
Dysfunction of MPC leads to gaze **dysmetria** i.e. it alters the spatial congruence between :

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



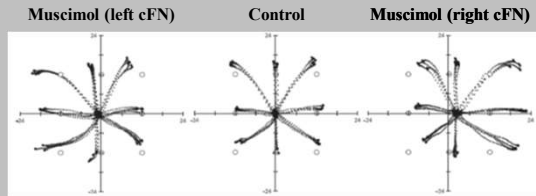
What renders movements inaccurate ?

The medio-posterior cerebellum



Horizontal dysmetria after cFN inactivation in the head restrained monkey

target LEDs @ 145 cm : 0.25° visual angle, mesopic conditions



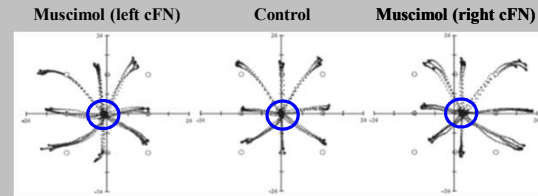
Goffart, Chen & Sparks, JNP 2004

Ipsilesional saccades : horizontal component is **hypermetric**

Contralesional saccades : horizontal component is **hypometric**

Horizontal dysmetria after cFN inactivation in the head restrained monkey

target LEDs @ 145 cm : 0.25° visual angle, mesopic conditions



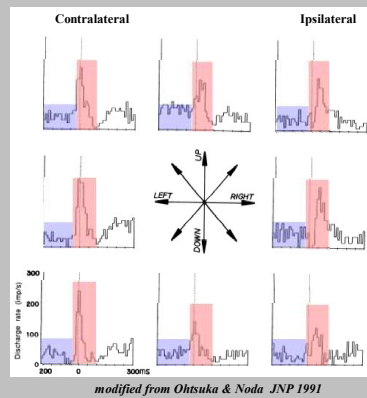
Goffart, Chen & Sparks, JNP 2004

Ipsilesional saccades : horizontal component is **hypermetric**

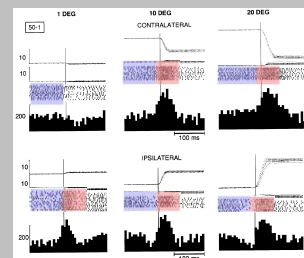
Contralesional saccades : horizontal component is **hypometric**

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

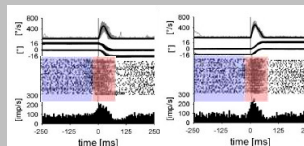
'Omniburst' neuron with sustained presaccadic activity



modified from Ohtsuka & Noda JNP 1991

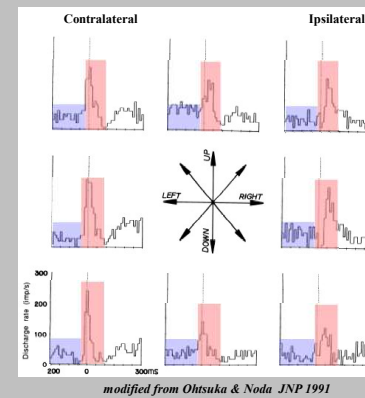


modified from Fuchs, Robinson & Straube JNP 1993

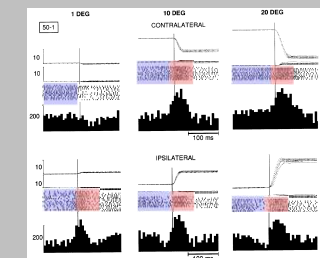


modified from Kleine, Guan & Bättner JNP 2003

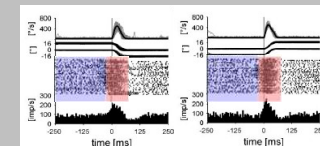
Dysmetria = consequence of perturbing presaccadic or intrasaccadic activity ?



modified from Ohtsuka & Noda JNP 1991

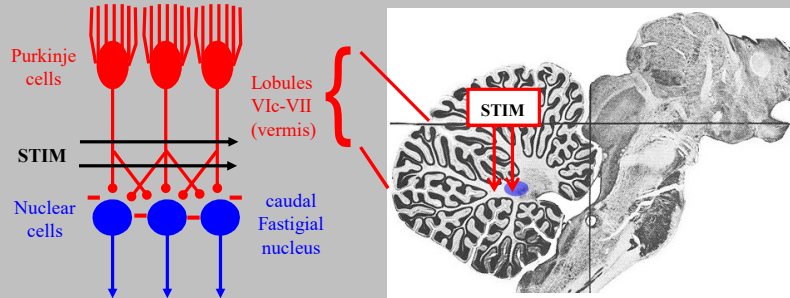


modified from Fuchs, Robinson & Straube JNP 1993



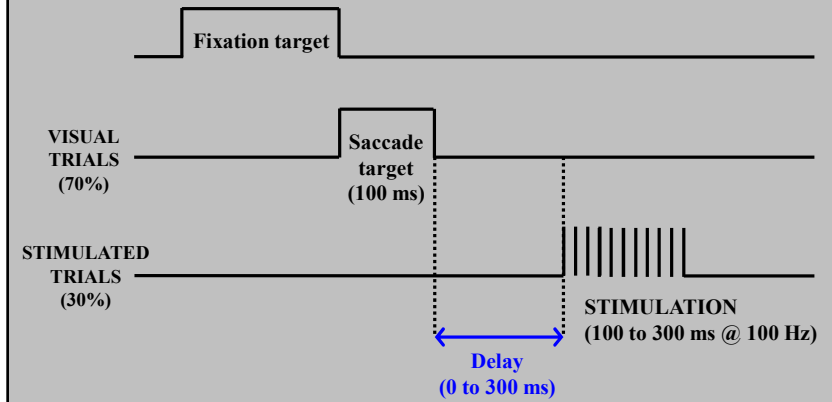
modified from Kleine, Guan & Bättner JNP 2003

Transient inhibition of cFN activity with electrical microstimulation



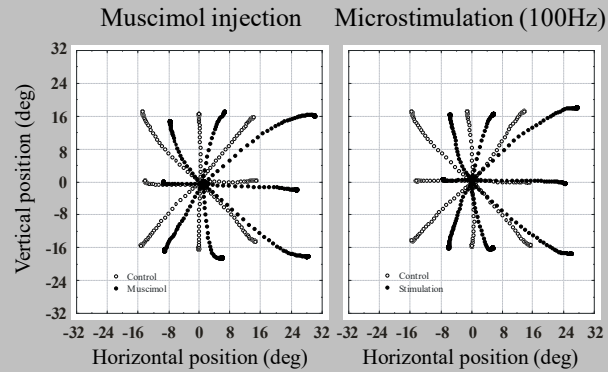
One should be able to mimic, with a microstimulation train, the effects of muscimol on saccade accuracy.

Microstimulation during saccade generation



Inhibitory perturbation

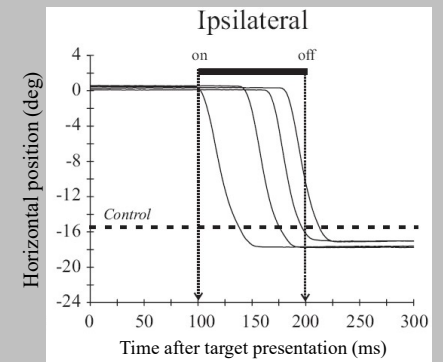
Right cFN



Goffart & Quinet unpubl. obs.

Time window of the perturbation

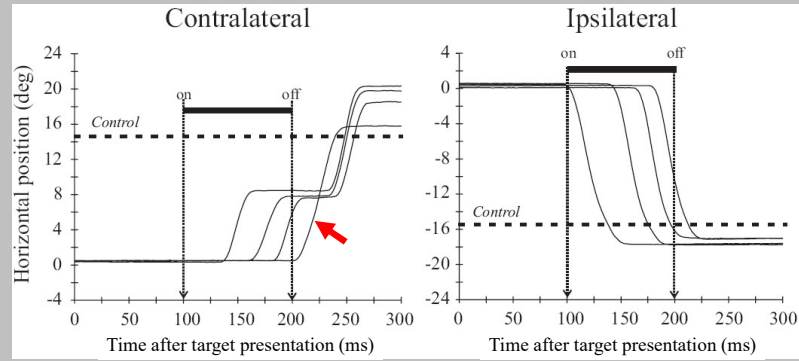
Horizontal saccades



Goffart & Quinet unpubl. obs.

Time window of the perturbation

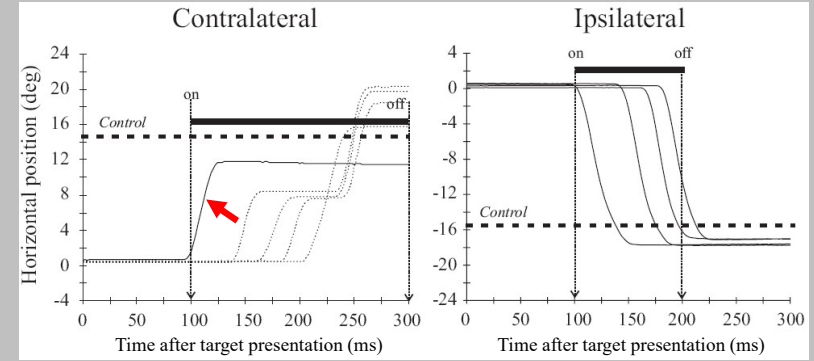
Horizontal saccades



Goffart & Quinet unpubl. obs.

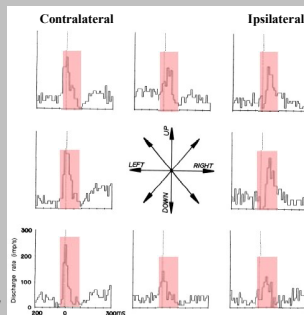
Time window of the perturbation

Horizontal saccades



Goffart & Quinet unpubl. obs.

Dysmetria is due to an imbalance in **intrasaccadic** activity



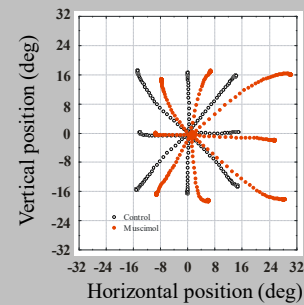
modified from Ohtsuka & Noda JNP 1991

Suppression of the burst in one FOR leads to
- ipsilesional horizontal hypermetria
- contralesional horizontal hypometria



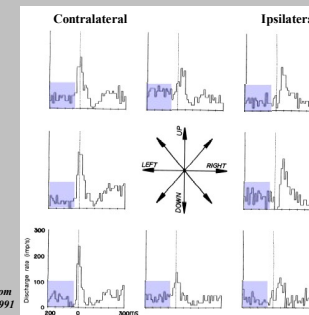
hypometria can as well be due to the unaffected FOR
what matters is the balance between left and right cFN (see Goffart, Chen & Sparks 2004)

Muscimol (right cFN)



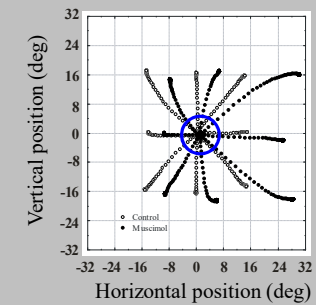
Goffart & Quinet unpubl. obs.

Fixation offset is due to an imbalance in **presaccadic** activity



modified from Ohtsuka & Noda JNP 1991

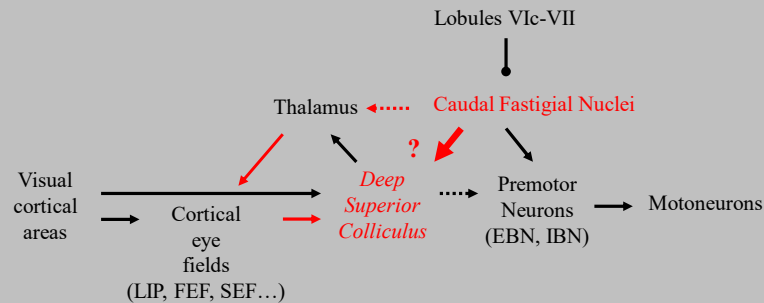
Muscimol (right cFN)



Goffart & Quinet unpubl. obs.

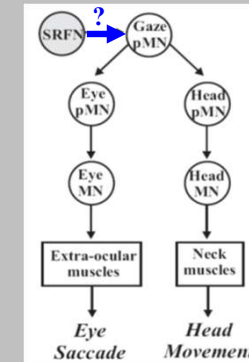
Question

Are the **fixation offset** / **horizontal dysmetria** due to a change in the topology of active neurones on the collicular map ?



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 ?

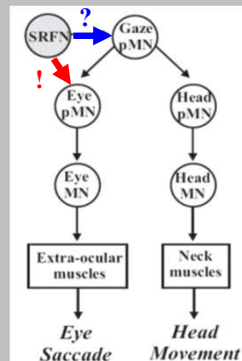


Is the **horizontal dysmetria** due to a change in the topology of active neurones on the collicular map ?

Is the dysmetria a disorder in the control of :
 - orienting the visual axis in space (gaze) ?
 > dysmetria eye + head ? **NO**
 > eye-head coupling unchanged ?

- moving the eyes in the orbit **YES**
 > eye dysmetria only !
 > eye-head coupling changed !
 > modifications affecting the eye and not the head !

Quinet & Goffart JNP 2005

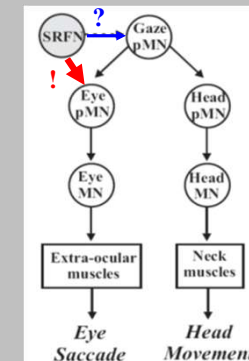


Is the **horizontal dysmetria** due to a change in the topology of active neurones on the collicular map ?

Is the dysmetria a disorder in the control of :
 - orienting the visual axis in space (gaze) ?
 > dysmetria eye + head ? **NO**
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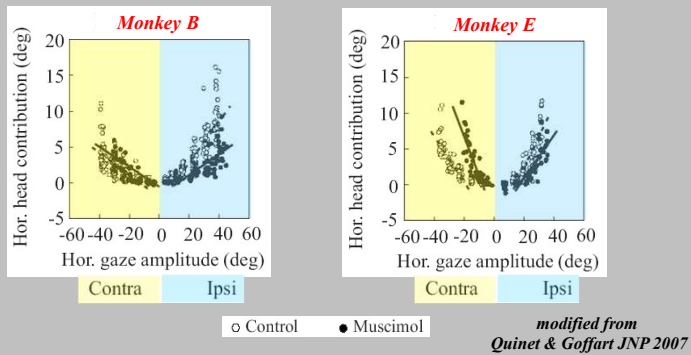
- moving the eyes in the orbit **YES**
 > eye dysmetria only ! **YES**
 > eye-head coupling changed ! **YES**
 > modifications affecting the eye and not the head ! **YES**

Quinet & Goffart JNP 2005
 Quinet & Goffart JNP 2007

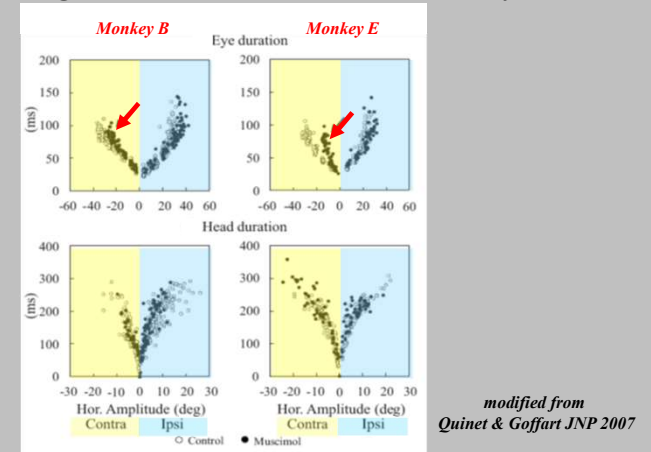


Eye-head coupling is modified

Ipsi : head contributes less
 Contra : head contributes more



Slowing of contralesional saccades No change in head movement velocity



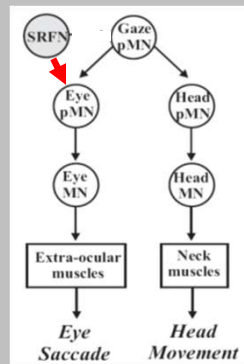
The saccade-related neurons in cFN exert an oculomotor function in the primate

Results in the head unrestrained monkey

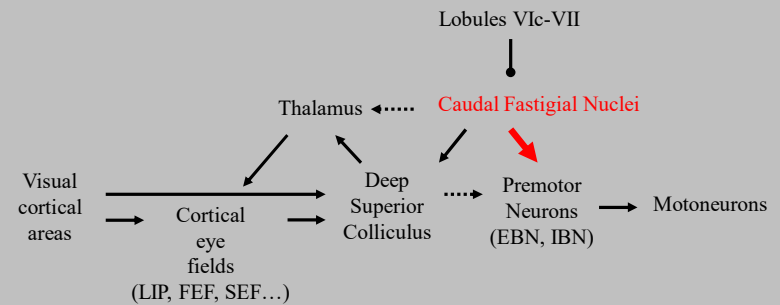
Pharmacological inactivation
 Quinet & Goffart JNP 2005, 2007

Electrical microstimulation
 Quinet & Goffart in revision
 see poster at Barany meeting 2008

Anatomical studies
 Robinson, Phillips & Fuchs JCN 1994

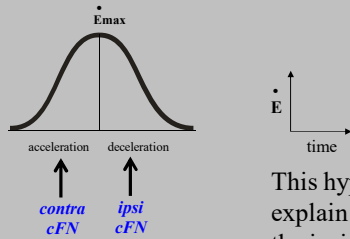


The gaze dysmetria is not due to a change in the topology of active neurones on the collicular map



How does cFN inactivation alter saccade amplitude ?

« push-pull » hypothesis



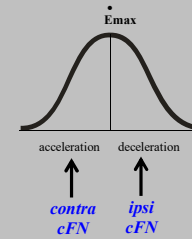
Robinson, Straube & Fuchs *Journal of Neurophysiology* 1993
Robinson & Fuchs *Annual Review of Neuroscience* 2001

This hypothesis does not explain the process leading to the ipsipulsion of vertical saccades (ipsipulsion increases with target eccentricity).

Moreover, perturbing the cFN can affect the « acceleration » of ipsilesional saccades or the « deceleration » of contralesional ones.

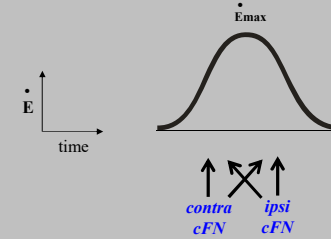
How does cFN inactivation alter saccade amplitude ?

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Robinson, Straube & Fuchs *Journal of Neurophysiology* 1993
Robinson & Fuchs *Annual Review of Neuroscience* 2001

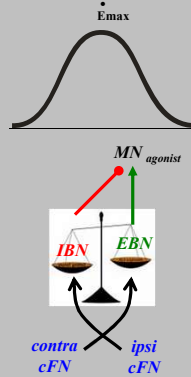
« bilateral » hypothesis



Goffart, Chen & Sparks *Journal of Neurophysiology* 2004

How does cFN inactivation alter saccade amplitude ?

« bilateral » hypothesis

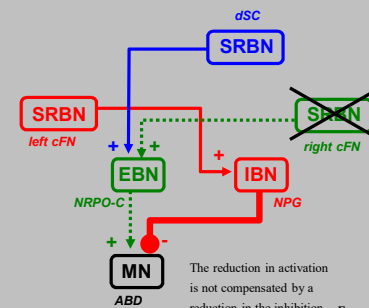


For every saccade, the bilateral cFN activity regulates the balance of activity between EBNs and IBNs

(Goffart, Chen & Sparks JNP 2004).

Bilateral cFN activity controls the balance EBNs/IBNs

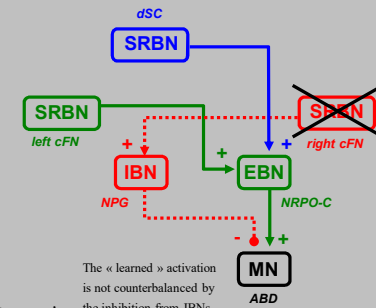
Contralesional hypometria



The reduction in activation is not compensated by a reduction in the inhibition which is exerted by IBNs and which results from the unaffected cFN

From the motoneurons' « point of view »

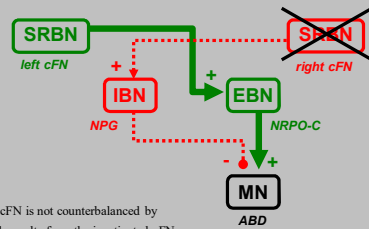
Ipsilesional hypermetria



The « learned » activation is not counterbalanced by the inhibition from IBNs which results from the inactivated cFN

Bilateral cFN activity controls the balance EBNs/IBNs

Ipsipulsion of vertical saccades



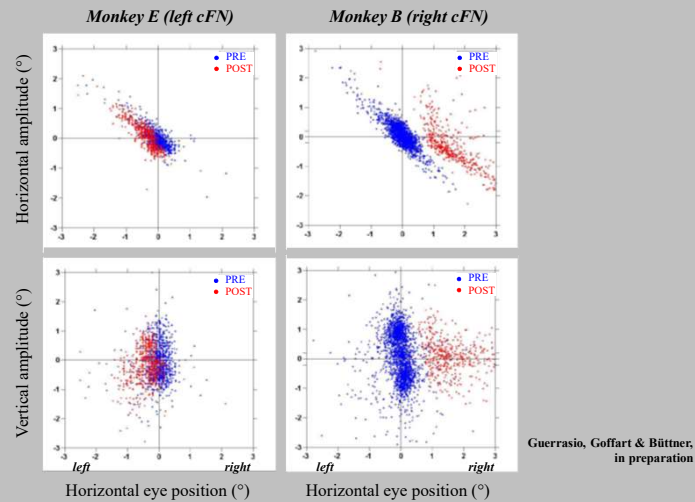
The burst from the unaffected cFN is not counterbalanced by the inhibition from IBNs which results from the inactivated cFN.

The ipsipulsion increases with saccade size because in normal conditions, the number of spikes that IBN generate during vertical saccades, increases with saccade size (Scudder, Fuchs & Langer JNP 1988).

Fixation offset and **dysmetria** :

- > Two different deficits ?
- > One same deficit ?
(dysmetria of fixational saccades) ?

Fixational saccades become inaccurate

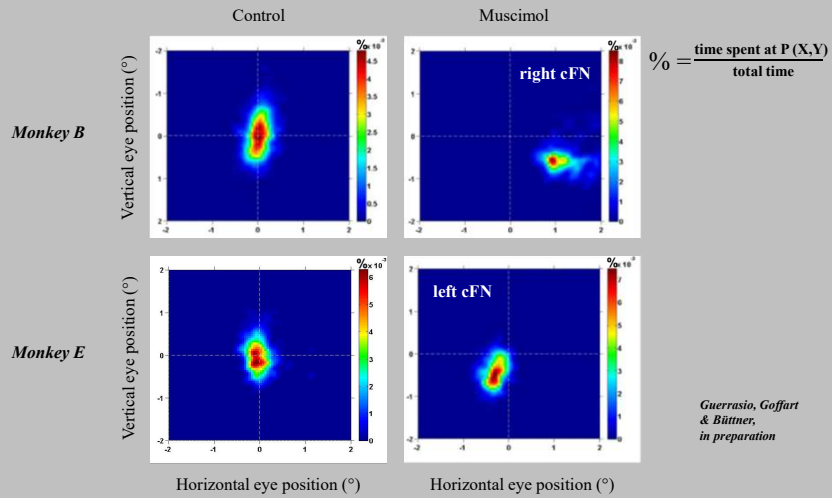


Fixational saccades become inaccurate

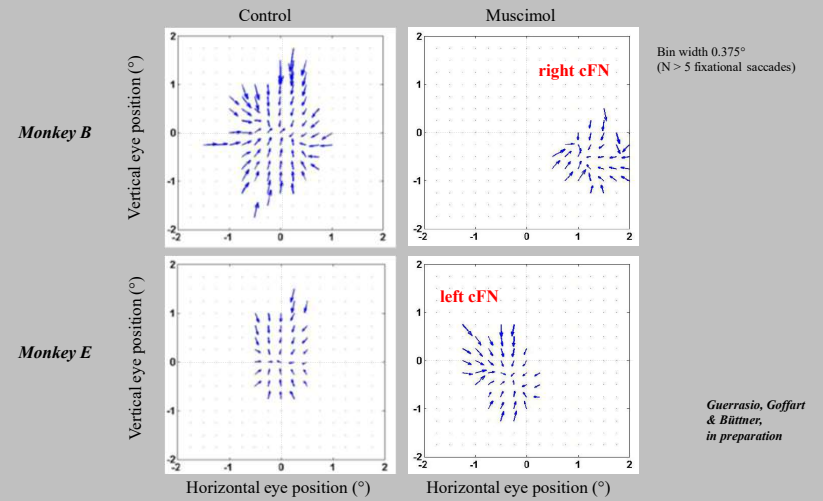
BUT

there are additional observations

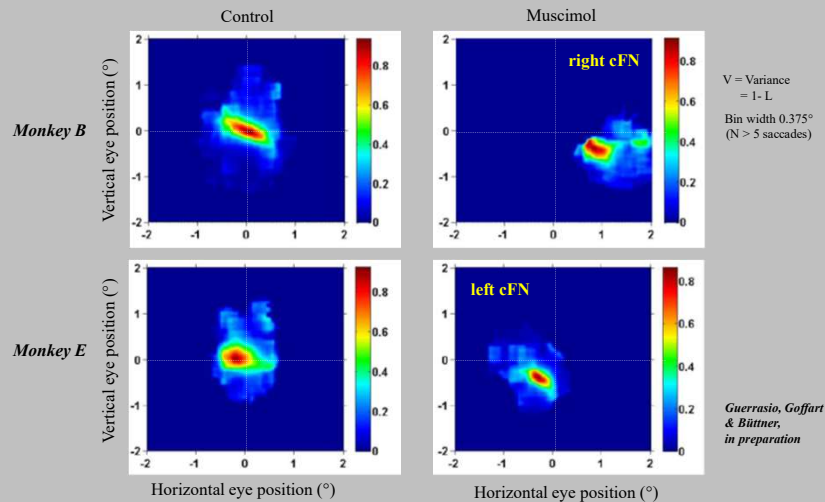
Fixation offset : spatio-temporal map



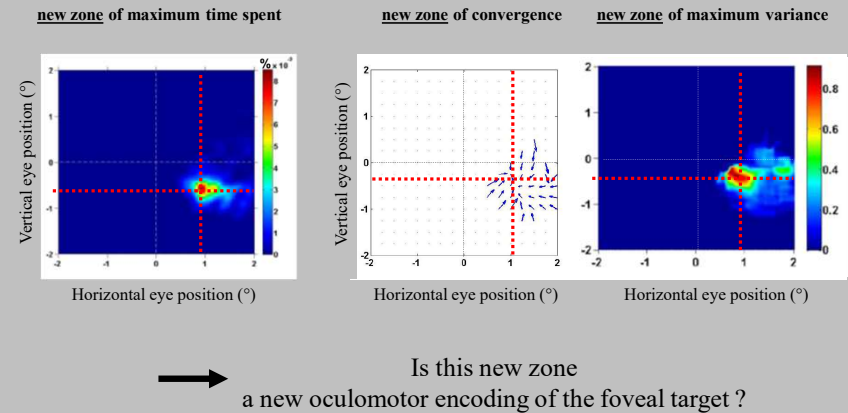
Vector field : convergence zone for fixational saccades



Vector field : zone of maximum variance



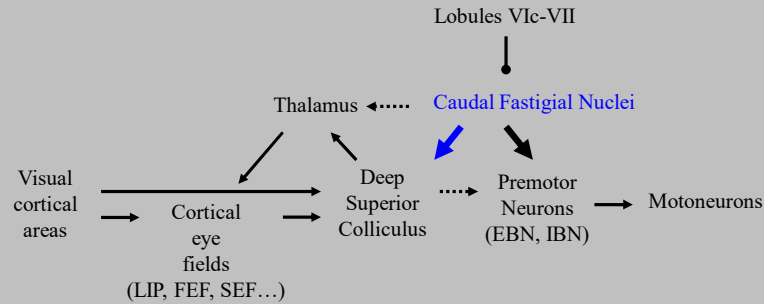
Fixational saccades become inaccurate BUT



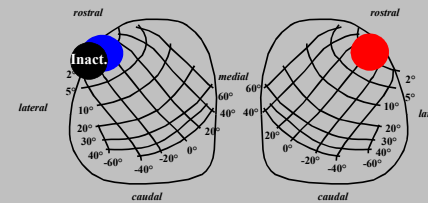
Fastigio-tectal projections could participate in the oculomotor encoding of foveal targets

May, Hartwich-Young, Nelson, Sparks & Porter Neuroscience 1990

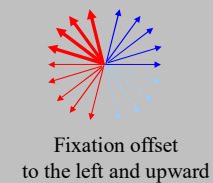
The fastigiotelectal pathway is derived from cells in the caudal fastigial nucleus and projects bilaterally to the rostral end of the intermediate gray layer.



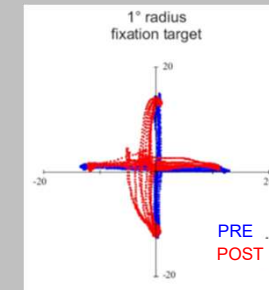
HYPOTHESIS



PREDICTION



RESULT



Goffart, Hafed & Krauzlis SFN 2006

Conclusions

The Fastigial Oculomotor Region (FOR) ensures the spatial congruence between :
 - the location of a stationary visual target (peripheral or foveal locus of activity) and
 - the endpoint of a saccade toward this location (oculomotor commands).

The FOR is mostly involved in the control of the amplitude of the horizontal component
 (more information during the symposium #10 "Vertical eye movements" at the Barany meeting).

The control is exerted during fixation and during saccades by the balance of activity between the left and right FOR.

During saccadic gaze shifts, the control is restricted to the oculomotor system (or « small » gaze shifts). Additional mechanisms must be recruited for coupling the eye saccades with head movements and/or body movements during locomotion.

The two FOR influence the accuracy of saccades by their projections toward the EBN/IBN region. They participate to encoding the location of foveal targets by their projections toward the rostral SC.

Perturbation of the rostral SC does not affect the accuracy of saccades to peripheral targets but leads to a fixation offset which is similar to that observed after cFN inactivation.

Acknowledgments

Work on the caudal Fastigial Nucleus:

Julie Quinet, Eye-head coordination lab, Rochester, USA
 David L. Sparks, Division of Neuroscience, Houston, USA
 Lorenzo Guerrasio, Dept of Neurology, München, Germany
 Ulrich Büttner, Dept of Neurology, München, Germany
 Sandrine Hugues, INCM, Marseille, France

Work on the rostral Superior Colliculus:

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

