

Coupling between the Horizontal and Vertical Saccade Generators after Inactivation of the Caudal Fastigial Nucleus in the Monkey

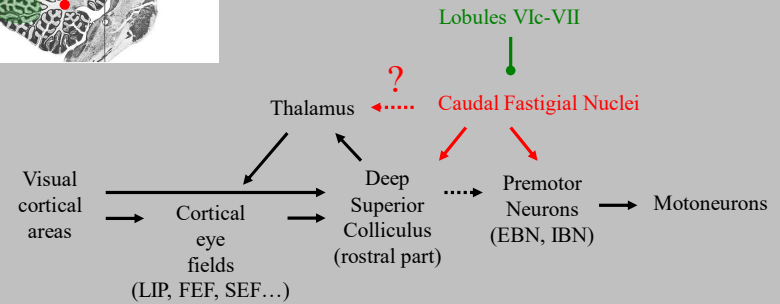
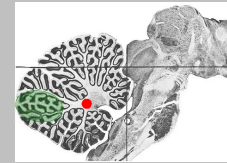
Laurent Goffart & Julie Quinet

CNRS

DYVA

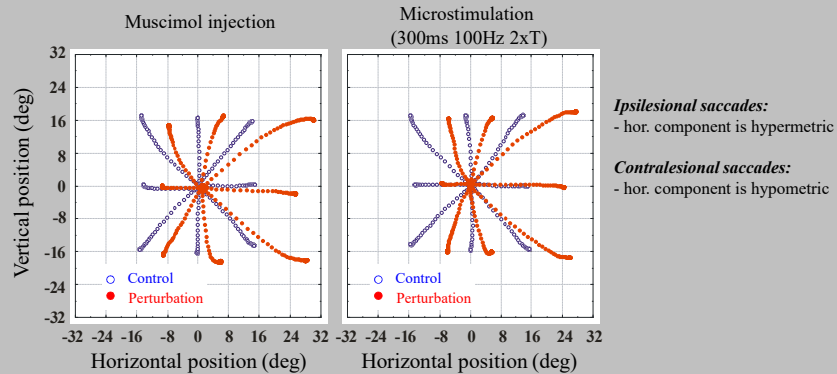
Institut de Neurosciences Cognitives de la Méditerranée
Marseille, France

The medio-posterior cerebellum



cFN inactivation leads to horizontal dysmetria

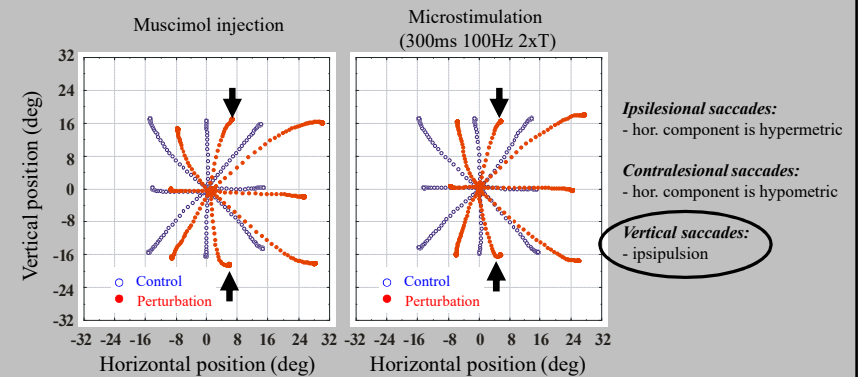
right caudal Fastigial Nucleus



Goffart & Quinet unpubl. obs.

cFN inactivation leads to horizontal dysmetria

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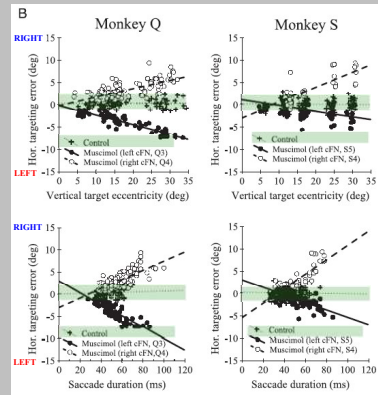


Goffart & Quinet unpubl. obs.

Ipsipulsion of vertical saccades increases with :

- target eccentricity

- saccade duration

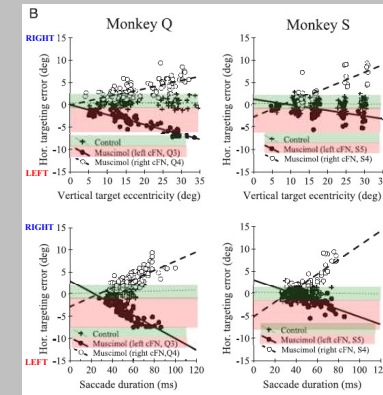


modified from Goffart, Chen & Sparks JNP 2004

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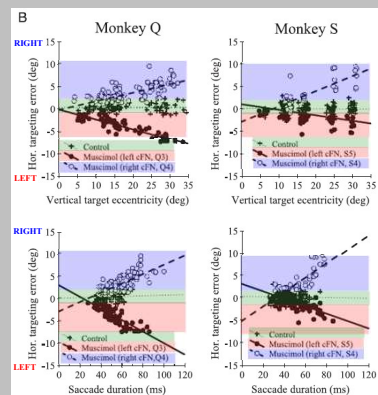


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Ipsipulsion of vertical saccades increases with :

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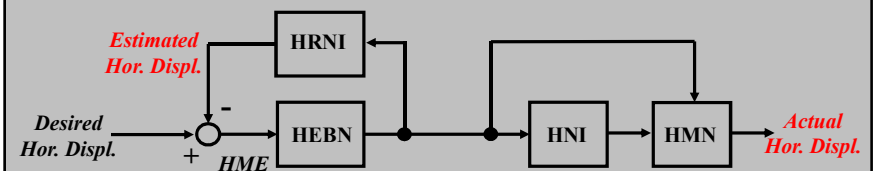
- saccade duration



modified from Goffart, Chen & Sparks JNP 2004

Dysmetria and the feedback control of saccades

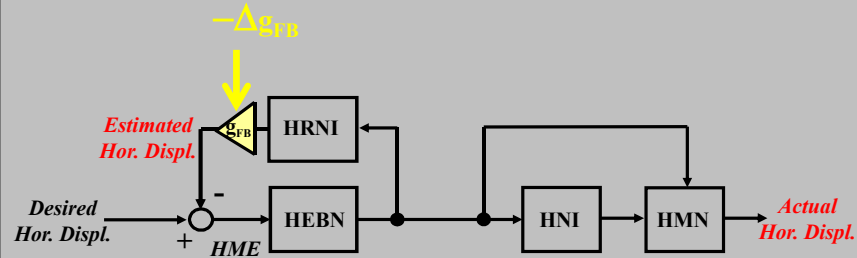
Mismatch between - the actual displacement (AHD) and
- its representation by the feedback signals (EHD)



HME = Hor. motor error

Hypermetria

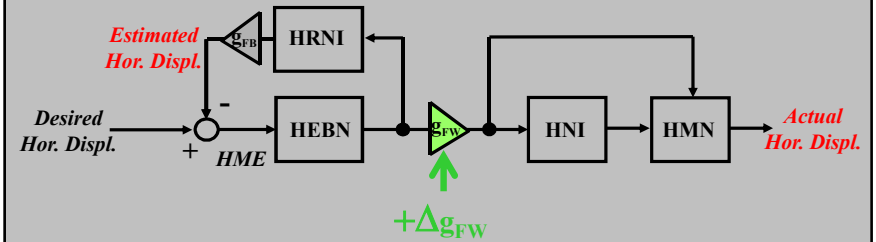
if actual displacement is underestimated (EHD < AHD)
because of a gain reduction ($-\Delta g_{FB}$)



HME = Hor. motor error

Hypermetria

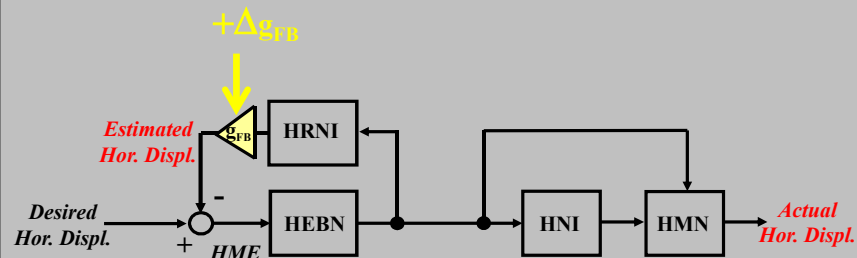
if $-\Delta g_{FB}$ or if $+\Delta g_{FW}$



HME = Hor. motor error

Hypometria

if actual displacement is overestimated (EHD > AHD)
because of a gain increase ($+\Delta g_{FB}$)

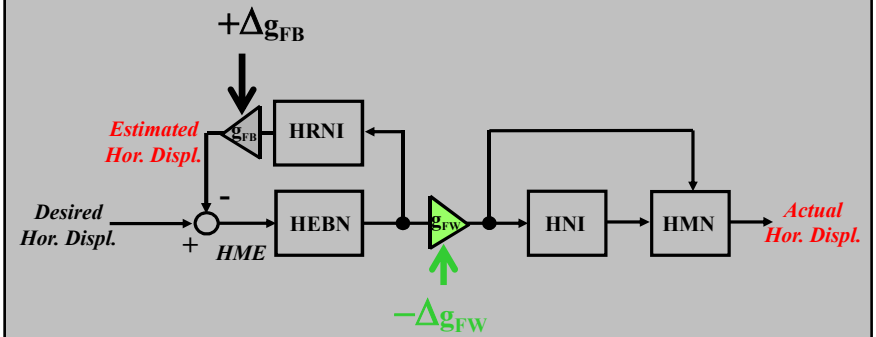


DHD = Desired. hor. displ.

HME = Hor. motor error

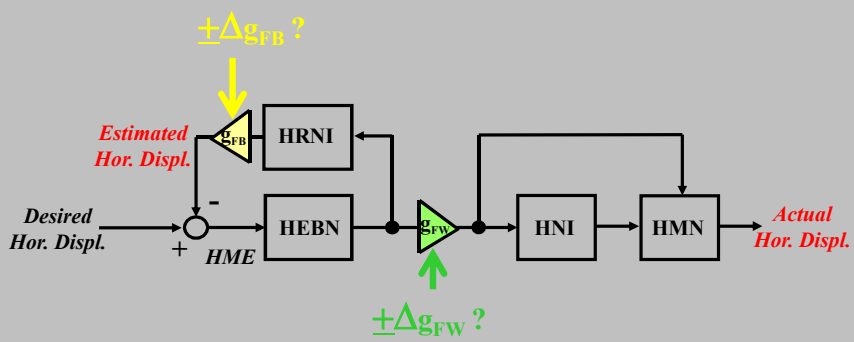
Hypometria

if $+\Delta g_{FB}$ or if $-\Delta g_{FW}$



HME = Hor. motor error

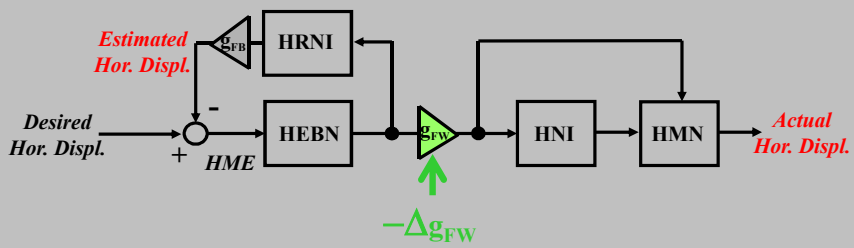
Functional localization of horizontal dysmetria after cFN inactivation



HME = Hor. motor error

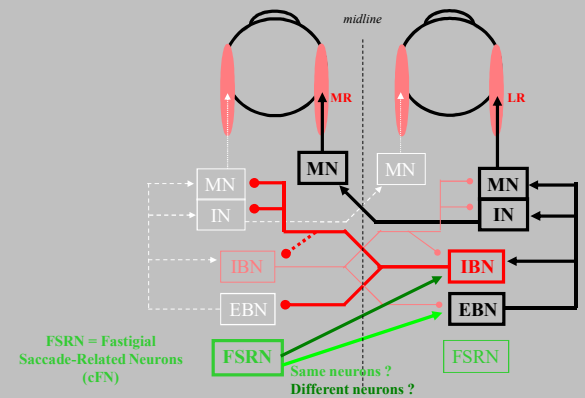
Contralesional hypometria

1. Lack of compensation for unexpected changes in eye position (Noda, Murakami & Warabi, 1991)



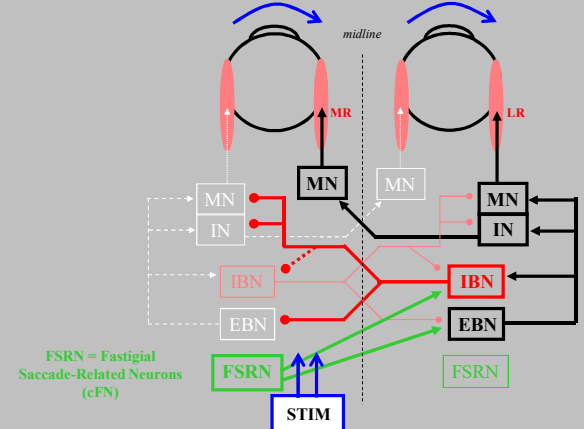
Background

- a. cFN neurons project toward the contralateral PMRF (Noda, Sugita & Ikeda JCN 1990)

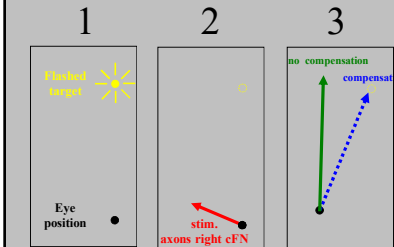
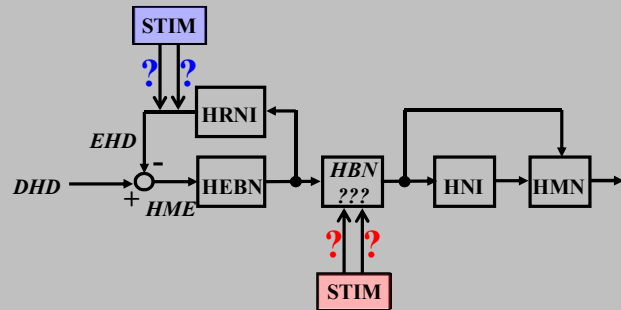


Background

- a. cFN neurons project toward the contralateral PMRF (Noda, Sugita & Ikeda JCN 1990)
- b. Stimulation of axons evokes a contralateral saccade (Noda, Murakami, Yamada, Tamaki & Aso JNP 1988)



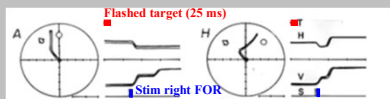
Does the fastigial stimulation influence neural elements located **inside** or **downstream** from the feedback loop ?



Experimental protocol (Mays & Sparks' paradigm) :

1. Flashed target for generating a saccade
2. Stimulation during the reaction time to **change eye position**
3. Test saccade accuracy after perturbation
(accuracy = **compensation** = Δ eye position is fed back
inaccuracy = **no compensation** = stimulation is downstream from feedback)

No compensation for a change in eye position induced by fastigial stimulation

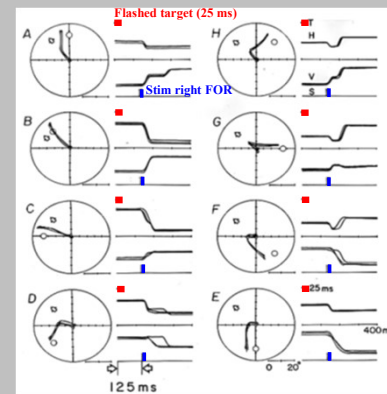


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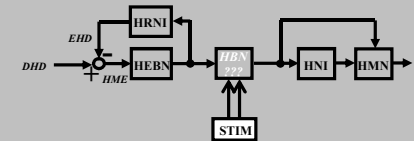
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Modified from Noda, Murakami & Warabi Neurosc. Res. 1991

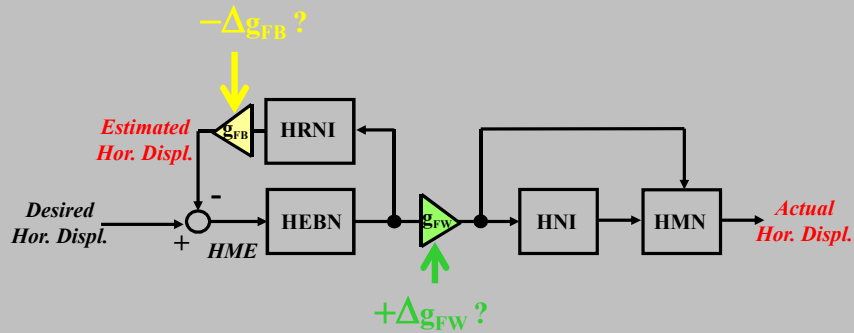
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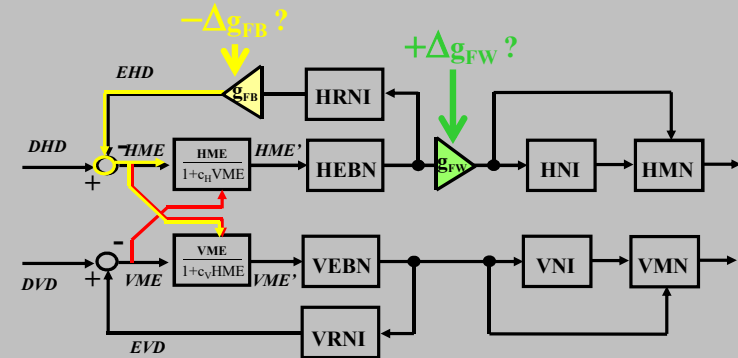
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Ipsilesional hypermetria

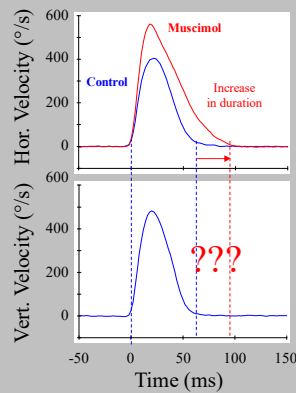


Testing oblique saccades to determine where Δg takes place



Modified from Becker & Jürgens VR 1990

Testing oblique saccades to determine where Δg takes place

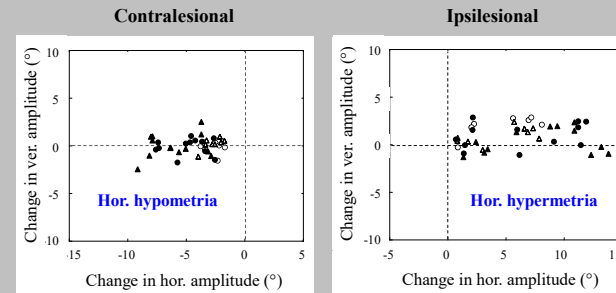


Note the increase in hor. peak velocity
(= a change in acceleration of ipsilesional saccades can happen)

Is the vertical saccade generator informed of the increased duration in the horizontal component?

Dysmetria

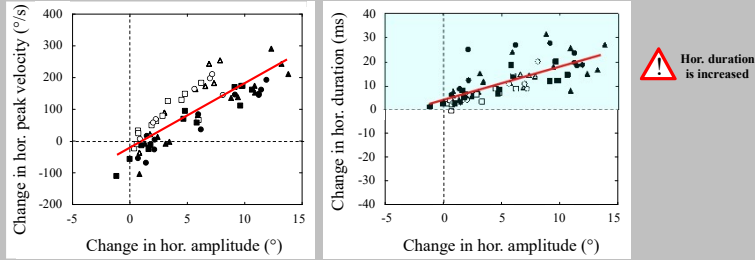
Oblique saccades



\triangle \blacktriangle 8°, 16° oblique upward
 \circ \bullet 8°, 16° oblique downward
 N=13 injections in 2 monkeys

Ipsilesional saccades (hypermetric)

Horizontal component (oblique and cardinal saccades)

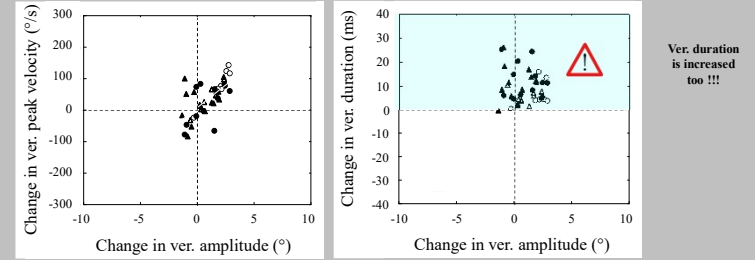


- △ ▲ 8°, 16° oblique upward
- ■ 8°, 16° horizontal
- ● 8°, 16° oblique downward

N=13 injections in 2 monkeys

Ipsilesional saccades (hypermetric)

Vertical component

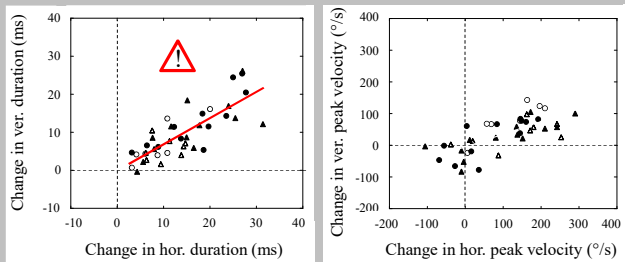


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N=13 injections in 2 monkeys

Ipsilesional saccades (hypermetric)

Coupling H-V components

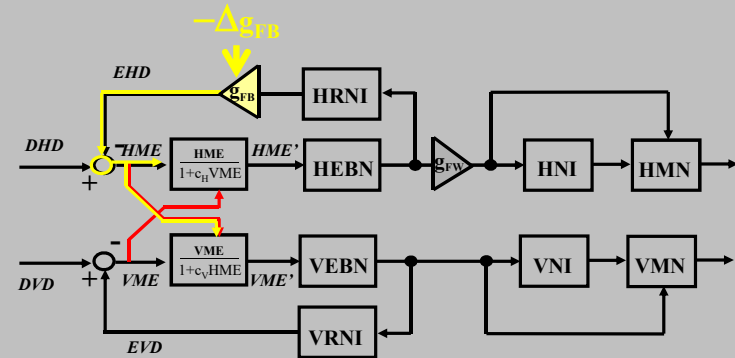


- △ ▲ 8°, 16° oblique upward
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N=13 injections in 2 monkeys

Ipsilesional saccades : conclusion

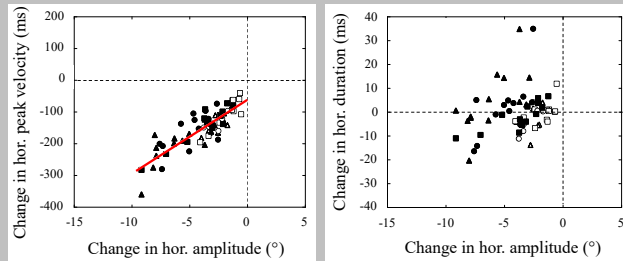
Correlated increases in horizontal and vertical duration



Modified from Becker & Jürgens VR 1990

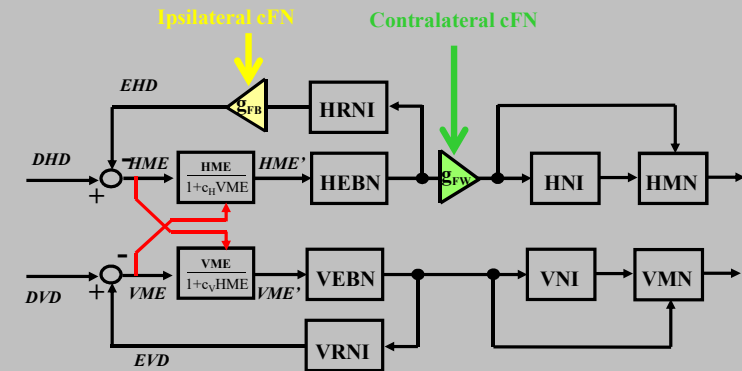
Contralesional saccades (hypometric)

Horizontal component (oblique and cardinal saccades)



▲ 8°, 16° oblique upward
 ■ 8°, 16° horizontal
 ● 8°, 16° oblique downward
 N=13 injections in 2 monkeys

Bilateral control of saccade accuracy



Modified from Becker & Jürgens VR 1990

Conclusions and summary

Ipsilesional oblique saccades

The amplitude and duration of the horizontal component are increased. Depending on the injections, the amplitude of the vertical component is increased, decreased or unchanged. But its duration is consistently increased. These increases in horizontal and vertical durations are correlated.

This correlation suggests that the changes in the horizontal component are fed back to the mechanisms coupling the horizontal and vertical components of vertical saccades. If the feedback control of saccade amplitude relies upon the same feedback as the cross-coupling mechanisms, our results indicate that the ipsilesional dysmetria is due to an impaired estimate of the current horizontal eye displacement.

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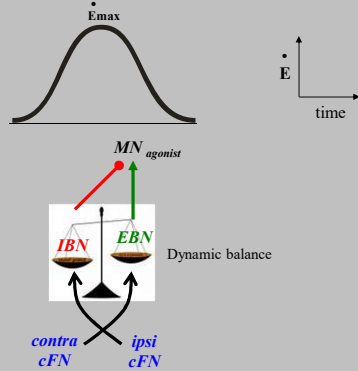
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Contralesional oblique saccades

The reduction in horizontal amplitude is associated with a correlated change in horizontal peak velocity without consistent changes in the vertical component. Consistent with the results of Noda et al. (1992), our results suggest that during these saccades, the cFN influences neural elements which are located downstream from the local feedback loop. The existence of such elements is supported by results from Sparks et al (1988) after pontine stimulation.

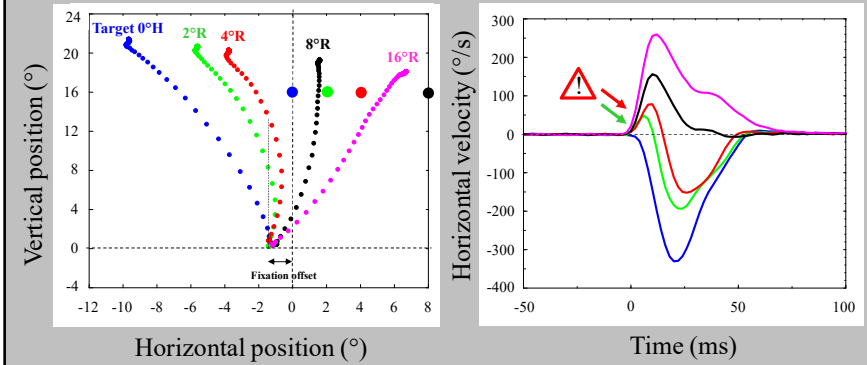
« Bilateral » hypothesis

For every saccade, horizontal, oblique or vertical,
the bilateral cFN activity regulates the balance of activity
between EBNs and IBNs (see Goffart, Chen & Sparks JNP 2004 page 3366).

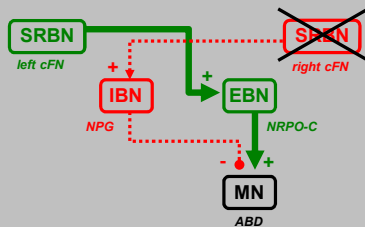


Bilateral control of the on-going trajectory (from saccade onset to saccade end)

Muscimol injection in the left caudal Fastigial Nucleus



Ipsipulsion of vertical saccades

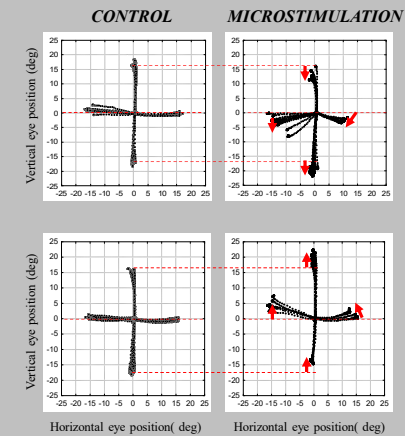


The burst from the unaffected cFN and its effects on EBN are not counterbalanced by the inhibition from IBNs which results from the inactivated cFN. The ipsipulsion increases with saccade size because in normal conditions, the number of spikes that IBN generate during vertical saccades, increases with saccade size (Scudder, Fuchs & Langer JNP 1988).

During vertical saccades, the cFN helps the IBN to “filter” the appropriate amount of EBN activity for the agonist muscles. The bilateral activity controls the on-going trajectory. Slight changes in this balance affects the trajectory of saccades, during ANY saccades (i.e. adapted or not) and during its complete course (i.e. from saccade onset to saccade end).

Dysmetria of the vertical component

Still undefined region



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Sandrine Hugues, INCM, Marseille, France

Ousmane Dieng, INCM, Marseille, France

