

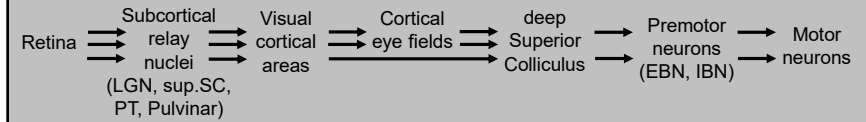
European Conference on Eye Movements
August 2019
Alicante, Spain

Cerebellar control of saccades by the size of the active population in the caudal fastigial nucleus

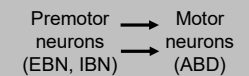
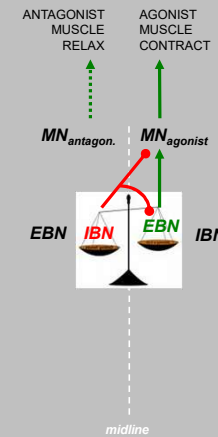
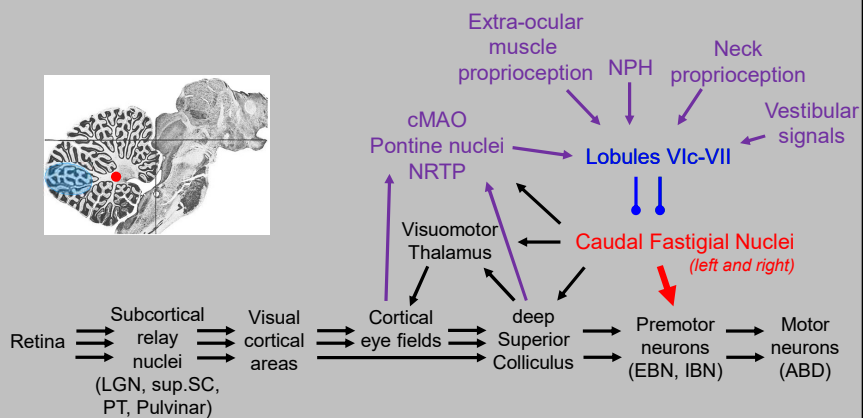
Laurent Goffart, Clara Burrelly & Julie Quinet

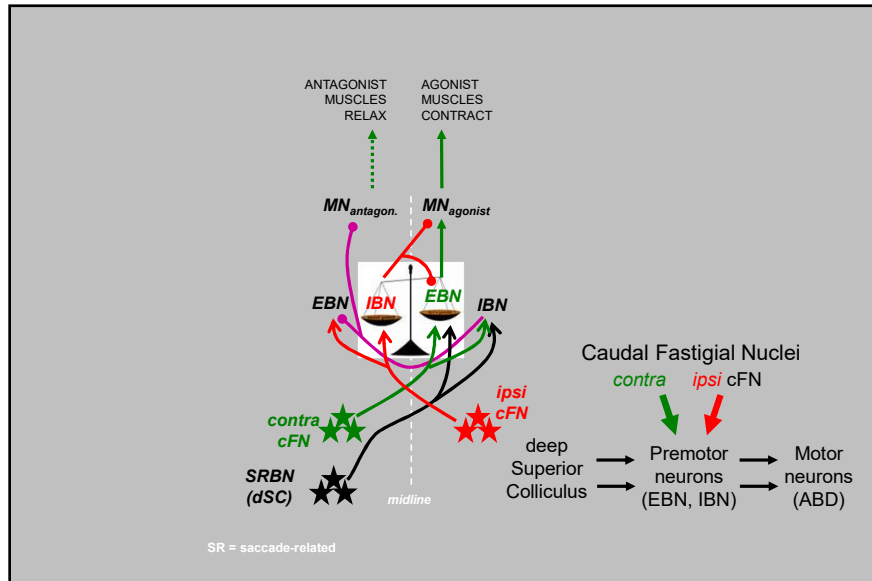
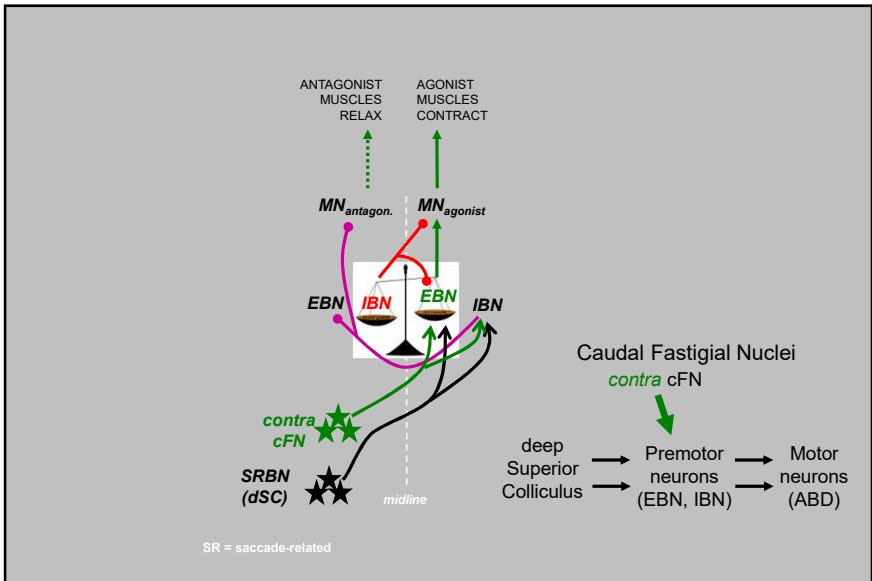
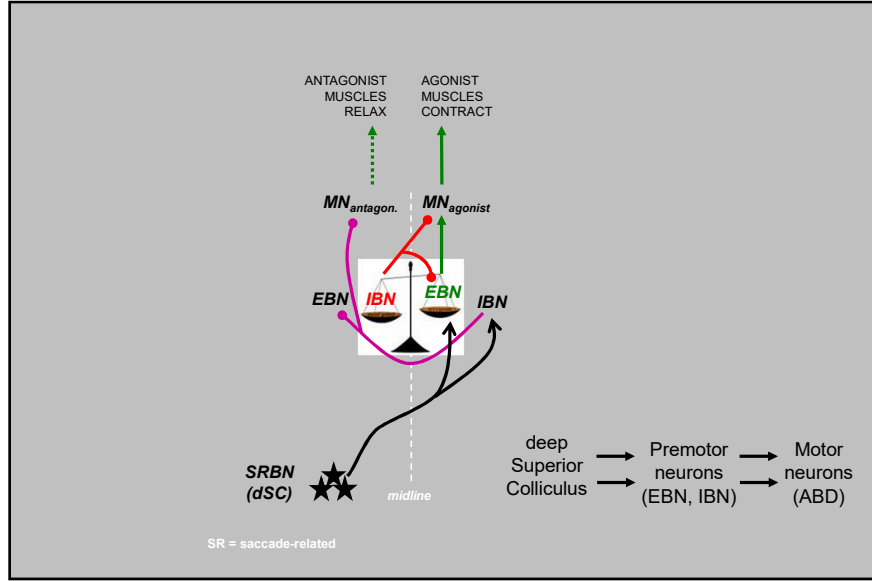
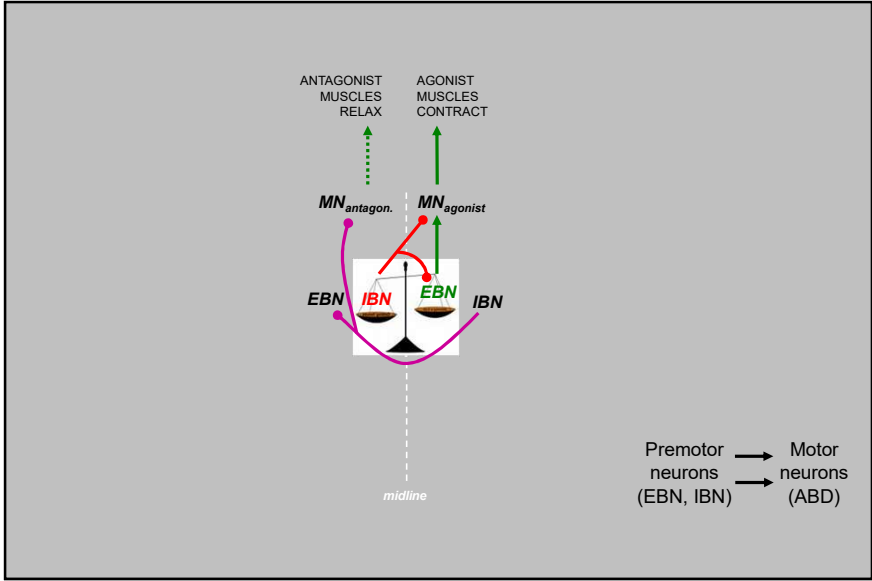
CNRS
INT, Marseille, France

From the retinal input to the oculomotor output



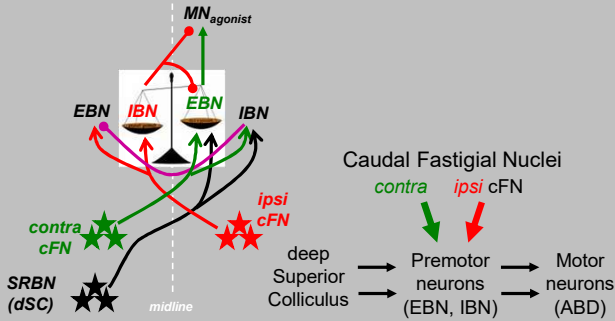
From the retinal input to the oculomotor output





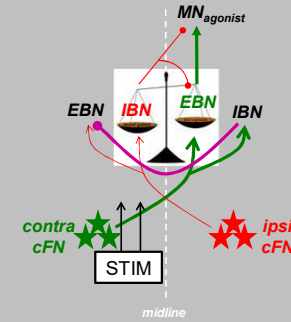
Bilateral hypothesis

For every saccade (horizontal, oblique or vertical), the left and right cFN regulate the balance of activity between the excitatory and inhibitory input (from EBNs and IBNs) to the motor and Internuclear neurons in the abducens nucleus (Journal of Neurophysiology 2004).



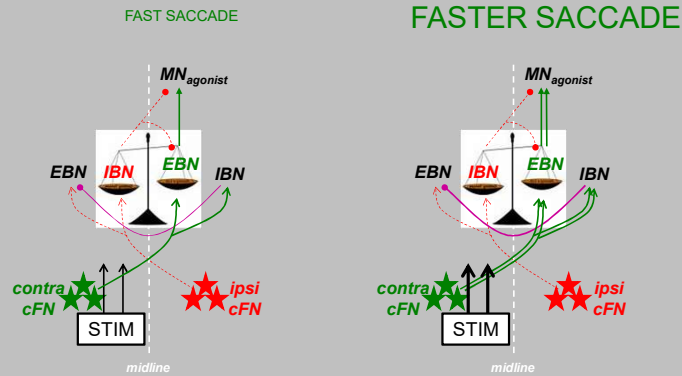
cFN stimulation evokes a contralateral saccade

Noda, Murakami, Yamada, Tamada, Tamaki & Aso T Journal of Neurophysiology 1988 (head fixed monkey)
Quinet & Goffart Journal of Neurophysiology 2009 (head free monkey)



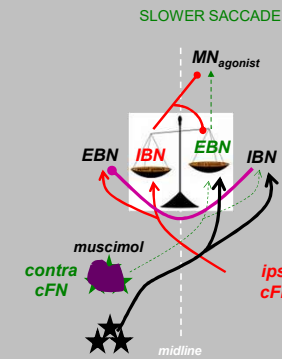
Peak velocity of saccades increases with current

Quinet & Goffart Journal of Neurophysiology 2015 (head fixed monkey)



cFN inactivation reduces the velocity of contralateral saccades without changing their duration → hypometria

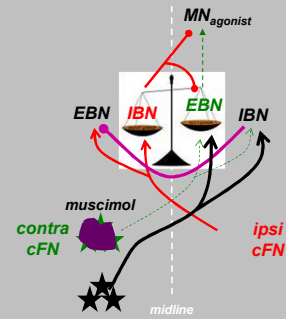
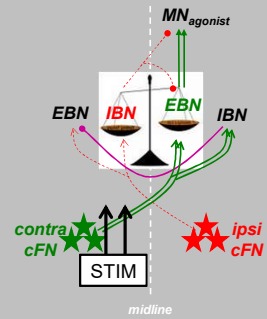
Goffart, Chen & Sparks Journal of Neurophysiology 2004 (head fixed monkey)
Quinet & Goffart Journal of Neurophysiology 2005, 2007 (head free monkey)



HYPOTHESIS : the number of active cFN neurons influences the velocity of contralateral saccades

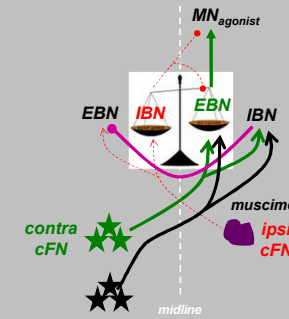
ELECTRICAL MICROSTIMULATION

PHARMACOLOGICAL INACTIVATION



cFN inactivation makes ipsilesional saccades hypermetric

Goffart, Chen & Sparks *Journal of Neurophysiology* 2004
Quinet & Goffart *Journal of Neurophysiology* 2005, 2007 (*head free monkey*)



RESEARCH ARTICLE Modeling Inter-trial Variability of Saccade Trajectories: Effects of Lesions of the Oculomotor Part of the Fastigial Nucleus

Thomas Eggert^{1*}, Farrel R. Robinson², Andreas Straube²

¹ Department of Neurology, Ludwig-Maximilians-University, Munich, Germany, ² Department of Biological Structure, University of Washington, Seattle, Washington, United States of America

PLOS Computational Biology | DOI:10.1371/journal.pcbi.1004866 June 28, 2016

This study investigates the inter-trial variability of saccade trajectories observed in five rhesus macaques (*Macaca mulatta*). For each time point during a saccade, the inter-trial variance of eye position and its covariance with eye end position were evaluated. Data were modeled by a superposition of three noise components due to 1) planning noise, 2) signal-dependent motor noise, and 3) signal-dependent premotor noise entering within an internal feedback loop. Both planning noise and signal-dependent motor noise (together called accumulating noise) predict a simple S-shaped variance increase during saccades, which was not sufficient to explain the data. Adding noise within an internal feedback loop enabled the model to mimic variance/covariance structure in each monkey, and to estimate the noise amplitudes and the feedback gain. Feedback noise had little effect on end point noise, which was dominated by accumulating noise. This analysis was further extended to saccades executed during inactivation of the oculomotor part of the fastigial nucleus (cFN) on one side of the cerebellum. Saccades ipsiversive to an inactivated cFN showed more end point variance than did normal saccades. During cFN inactivation, eye position statistically more strongly coupled to eye position at saccade onset could fit the variance/covariance structure of ipsiversive and contralateral effects on saccade noise are explained by a decrease of the

Saccades ipsiversive to an inactivated cFN showed more end point variance than did normal saccades.

increase of planning and/or signal-dependent motor noise. The decrease of the fitted feedback gain is consistent with previous studies suggesting a role for the cerebellum in an internal feedback mechanism. Increased end point variance did not result from impaired feedback but from the increase of accumulating noise. The effects of cFN inactivation on saccade noise indicate that the effects of cFN inactivation cannot be explained entirely with the cFN's direct connections to the saccade-related premotor centers in the brainstem.

RESEARCH ARTICLE Modeling Inter-trial Variability of Saccade Trajectories: Effects of Lesions of the Oculomotor Part of the Fastigial Nucleus

Thomas Eggert^{1*}, Farrel R. Robinson², Andreas Straube²

¹ Department of Neurology, Ludwig-Maximilians-University, Munich, Germany, ² Department of Biological Structure, University of Washington, Seattle, Washington, United States of America

PLOS Computational Biology | DOI:10.1371/journal.pcbi.1004866 June 28, 2016

This study investigates the inter-trial variability of saccade trajectories observed in five rhesus macaques (*Macaca mulatta*). For each time point during a saccade, the inter-trial variance of eye position and its covariance with eye end position were evaluated. Data were modeled by a superposition of three noise components due to 1) planning noise, 2) signal-dependent motor noise, and 3) signal-dependent premotor noise entering within an internal feedback loop. Both planning noise and signal-dependent motor noise (together called accumulating noise) predict a simple S-shaped variance increase during saccades, which was not sufficient to explain the data. Adding noise within an internal feedback loop enabled the model to mimic variance/covariance structure in each monkey, and to estimate the noise amplitudes and the feedback gain. Feedback noise had little effect on end point noise, which was dominated by accumulating noise. This analysis was further extended to saccades executed during inactivation of the oculomotor part of the fastigial nucleus (cFN) on one side of the cerebellum. Saccades ipsiversive to an inactivated cFN showed more end point variance than did normal saccades. During cFN inactivation, eye position statistically more strongly coupled to eye position at saccade onset could fit the variance/covariance structure of ipsiversive and contralateral effects on saccade noise are explained by a decrease of the

Saccades ipsiversive to an inactivated cFN showed more end point variance than did normal saccades.

increase of planning and/or signal-dependent motor noise. The decrease of the fitted feedback gain is consistent with previous studies suggesting a role for the cerebellum in an internal feedback mechanism. Increased end point variance did not result from impaired feedback but from the increase of accumulating noise. The effects of cFN inactivation on saccade noise indicate that the effects of cFN inactivation cannot be explained entirely with the cFN's direct connections to the saccade-related premotor centers in the brainstem.

"accumulating noise" =
planning noise + signal-dependent noise

About the increased endpoint variance for ipsilesional saccades

RESEARCH ARTICLE
 Modeling Inter-trial Variability of Saccade Trajectories: Effects of Lesions of the Oculomotor Part of the Fastigial Nucleus

Thomas Eggert^{1*}, Farrel R. Robinson², Andreas Straube¹

¹ Department of Neurology, Ludwig-Maximilians-University, Munich, Germany, ² Department of Biological Structure, University of Washington, Seattle, Washington, United States of America

PLOS Computational Biology | DOI:10.1371/journal.pcbi.1004866 June 28, 2016

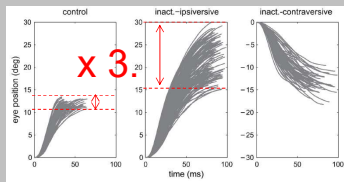


Figure 1

About the increased endpoint variance for ipsilesional saccades

RESEARCH ARTICLE
 Modeling Inter-trial Variability of Saccade Trajectories: Effects of Lesions of the Oculomotor Part of the Fastigial Nucleus

Thomas Eggert^{1*}, Farrel R. Robinson², Andreas Straube¹

¹ Department of Neurology, Ludwig-Maximilians-University, Munich, Germany, ² Department of Biological Structure, University of Washington, Seattle, Washington, United States of America

PLOS Computational Biology | DOI:10.1371/journal.pcbi.1004866 June 28, 2016

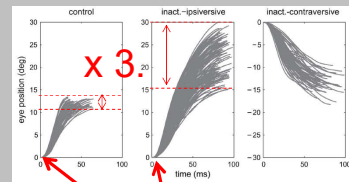


Figure 1

NOT EXACT

About the increased endpoint variance for ipsilesional saccades

RESEARCH ARTICLE
 Modeling Inter-trial Variability of Saccade Trajectories: Effects of Lesions of the Oculomotor Part of the Fastigial Nucleus

Thomas Eggert^{1*}, Farrel R. Robinson², Andreas Straube¹

¹ Department of Neurology, Ludwig-Maximilians-University, Munich, Germany, ² Department of Biological Structure, University of Washington, Seattle, Washington, United States of America

PLOS Computational Biology | DOI:10.1371/journal.pcbi.1004866 June 28, 2016

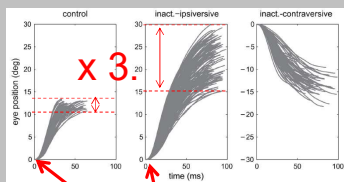


Figure 1

NOT EXACT

Deficits in Saccades and Fixation During Muscimol Inactivation of the Caudal Fastigial Nucleus in the Rhesus Monkey

Laurent Goffart^{1,2,3}, Longtang L. Chen² and David L. Sparks^{1,2,3}
¹ Department of Psychology, University of Pennsylvania, Philadelphia, Pennsylvania 19104; ² Department of Neuroscience, Baylor College of Medicine, Houston, Texas 77030; and ³ Virginia Dromaeur de la Vision et de l'Action, Institut de Neurosciences Cognitives et de Médiémoire, Institut Minc de Recherche CNRS Centre National de la Recherche Scientifique—Université de la Méditerranée, 13402 Marseille, France

TABLE 1. Comparison of the horizontal and vertical position of the eye prior to the saccade toward the target LED before and after muscimol injection in the cFN.

Experiment	Injection	Session	n	Horizontal Eye Position				Vertical Eye Position			
				Mean	Range	Difference	Mean	Range	Difference		
Q1	Left cFN (0.4 µl)	Control	378	-0.5 ± 0.6	-1.5, 1.3	-1.2*	0.0 ± 0.6	-1.5, 1.0	0.4*		
		Muscimol	614	-1.7 ± 1.0	-6.5, 2.0	-2.4*	0.4 ± 0.6	-1.6, 3.0	-0.1 NS		
Q2	Left cFN (0.4 µl)	Control	605	0.6 ± 0.6	-1.1, 2.4	-2.4*	0.2 ± 0.7	-2.5, 2.3	-0.1 NS		
		Muscimol	502	-1.8 ± 1.6	-6.6, 4.0	-0.1	0.1 ± 1.6	-6.0, 6.1	-0.3*		
Q3	Left cFN (0.8 µl)	Control	787	0.2 ± 0.2	-1.2, 2.3	-1.2*	0.2 ± 0.2	-1.5, 2.4	-0.1 NS		
		Muscimol	1011	-1.2 ± 1.1	-7.0, 2.9	-0.2	0.2 ± 0.6	-3.6, 2.6	-0.2*		
Q4	Right cFN (0.6 µl)	Control	680	-0.2 ± 1.0	-7.7, 7.0	2.0*	0.3 ± 0.9	-3.0, 3.1	-0.2*		
		Muscimol	962	1.6 ± 1.3	-3.5, 6.0	-1.4*	0.1 ± 0.8	-3.1, 3.2	0.1 NS		
J1	Left cFN (1.0 µl)	Control	335	0.0 ± 0.6	-1.3, 2.3	-1.4*	-0.7 ± 0.3	-1.7, 1.2	0.1 NS		
		Muscimol	341	-1.6 ± 1.1	-6.4, 3.0	-0.6	-0.6 ± 0.6	-2.2, 2.0	-0.4*		
J2	Left cFN (0.6 µl)	Control	199	0.1 ± 0.7	-1.7, 1.4	-1.3*	-0.5 ± 0.4	-1.7, 0.9	-0.4*		
		Muscimol	378	-1.2 ± 0.8	-3.4, 3.0	-0.4*	-0.9 ± 0.4	-2.4, 0.9	-0.2*		
J3	Right cFN (0.6 µl)	Control	488	0.0 ± 0.8	-2.3, 2.0	-0.4*	0.0 ± 0.5	-1.2, 1.9	-0.2*		
		Muscimol	470	-0.4 ± 0.7	-2.8, 3.0	-0.2	-0.2 ± 0.4	-1.4, 1.0	-0.1 NS		
J4	Right cFN (0.8 µl)	Control	796	0.1 ± 0.6	-2.4, 1.7	0.8*	0.0 ± 0.4	-1.2, 1.7	-1.0*		
		Muscimol	422	0.9 ± 0.7	-2.5, 3.2	-0.3*	-0.2 ± 0.4	-1.4, 1.0	-0.1 NS		
J5	Left cFN (0.8 µl)	Control	471	0.0 ± 0.8	-2.3, 2.2	-0.3*	0.0 ± 0.5	-1.6, 2.1	-0.1*		
		Muscimol	469	-0.3 ± 0.8	-3.2, 2.7	-0.1	-0.1 ± 0.5	-1.6, 1.4	-0.1 NS		
J7	Right cFN (1.3 µl)	Control	38	0.3 ± 0.8	-2.0, 2.1	0.3*	-0.1 ± 0.4	-1.2, 1.2	0.2*		
		Muscimol	154	0.6 ± 1.0	-1.6, 3.9	0.1	0.1 ± 0.6	-1.4, 2.3	0.1 NS		

Difference was tested with a Student's t-test for independent samples; NS: no statistically significant difference; *P < 0.05; LED, light-emitting diode; cFN, caudal fastigial nucleus.

Increase in the variability of starting positions

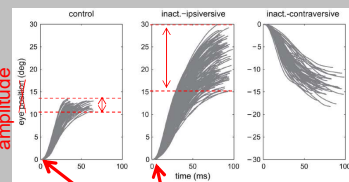
About the increased endpoint variance for ipsilesional saccades

RESEARCH ARTICLE
 Modeling Inter-trial Variability of Saccade Trajectories: Effects of Lesions of the Oculomotor Part of the Fastigial Nucleus

Thomas Eggert^{1*}, Farrel R. Robinson², Andreas Straube¹

¹ Department of Neurology, Ludwig-Maximilians-University, Munich, Germany, ² Department of Biological Structure, University of Washington, Seattle, Washington, United States of America

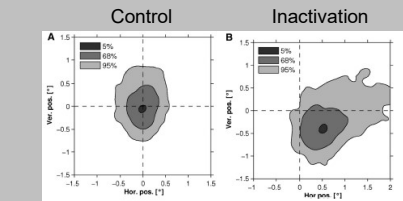
PLOS Computational Biology | DOI:10.1371/journal.pcbi.1004866 June 28, 2016



NOT EXACT

Fastigial Oculomotor Region and the Control of Foveation During Fixation

Laurence Guersanis¹, Julie Quidant², Ulrich Blittner¹, and Laurent Goffart³
¹ Department of Neurology, Klinikum Großhadern, LMU, München, Germany; ² Unité 534, Institut National de la Santé et de la Recherche Médicale/Université Claude Bernard—Lyon 1, IFR 19 Institut Fédératif des Neurosciences de Lyon, Rive, France; and ³ Institut de Neurosciences Cognitives et de la Médiémoire, Unité Minc Recherche 6191, Centre National de la Recherche Scientifique, Aix-Marseille Université, Marseille, France



Increase in the variability of starting positions

RESEARCH ARTICLE
Modeling Inter-trial Variability of Saccade Trajectories: Effects of Lesions of the Oculomotor Part of the Fastigial Nucleus
 Thomas Eggert^{1*}, Farel R. Robinson², Andreas Straube¹
¹ Department of Neurology, Ludwig-Maximilians-University, Munich, Germany, ² Department of Biological Structure, University of Washington, Seattle, Washington, United States of America

J Neurophysiol 92: 3355–3367, 2004. First published June 30, 2004; doi:10.1152/jn.01199.2003.

Deficits in Saccades and Fixation During Muscimol Inactivation of the Caudal Fastigial Nucleus in the Rhesus Monkey

Laurent Goffart,^{1,2,3} Longtang L. Chen,² and David L. Sparks,^{1,2}
¹Department of Psychology, University of Pennsylvania, Philadelphia, Pennsylvania 19104, ²Department of Neuroscience, Baylor College of Medicine, Houston, Texas 77030, and ³Equipe Dynamique de la Vision et de l'Action, Institut de Neurosciences Cognitives de la Méditerranée, Unité Mixte de Recherche 693 Centre National de la Recherche Scientifique—Université de la Méditerranée, 13402 Marseille, France

Abstract
 This study investigates the inter-trial variability of saccade trajectories observed in five rhesus macaques (*Macaca mulatta*). For each time point during a saccade, the inter-trial variance of eye position and its covariance with eye end position were evaluated. Data were modeled by a superposition of three noise components due to 1) planning noise, 2) signal-dependent motor noise, and 3) signal-dependent premotor noise entering within an internal feedback loop. Both planning noise and signal-dependent motor noise (together called accumulating noise) predict a simple S-shaped variance increase during saccades, which was not sufficient to explain the data. Adding noise within an internal feedback loop enabled the model to mimic variance/covariance structure in each monkey, and to estimate the noise amplitudes and the feedback gain. Feedback noise had little effect on end-point noise, which was dominated by accumulating noise. This analysis was further extended to saccades executed during inactivation of the caudal fastigial nucleus (cFN) on one side of the cerebellum. Saccades ipsiversive to an inactivated cFN showed more end-point variance than did normal saccades. During cFN inactivation, eye position during saccades was statistically more strongly coupled to eye position at saccade end. The proposed model could fit the variance/covariance structure of ipsiversive and contraversive saccades. Inactivation effects on saccade noise are explained by a decrease of the feedback gain and an increase of planning and/or signal-dependent motor noise. The decrease of the fitted feedback gain is consistent with previous studies suggesting a role for the cerebellum in an internal feedback mechanism. Increased end-point variance did not result from impaired feedback but from the increase of accumulating noise. The effects of cFN inactivation on saccade noise indicate that the effects of cFN inactivation cannot be explained entirely with the cFN's direct connections to the saccade-related premotor centers in the brainstem.

Introduction
 Saccades are rapid eye movements that are essential for visual perception. They are initiated by a burst of activity in the saccade-related premotor centers in the brainstem. The saccade trajectory is determined by the interaction of these centers and the oculomotor system. The cerebellum is known to play a role in the control of saccades, particularly in the timing and accuracy of the movements. Inactivation of the caudal fastigial nucleus (cFN) of the cerebellum has been shown to affect saccade performance in rhesus monkeys. This study aims to investigate the effects of cFN inactivation on saccade noise and endpoint variance.

Methods
 Five rhesus macaques were used in this study. Saccades were recorded during control conditions and during inactivation of the cFN. The inter-trial variability of saccade trajectories was analyzed using a model that incorporates planning noise, signal-dependent motor noise, and signal-dependent premotor noise. The model was fitted to the experimental data to estimate the noise amplitudes and the feedback gain.

Results
 The results show that saccades ipsiversive to an inactivated cFN exhibit increased endpoint variance compared to control saccades. This increase in variance is not due to impaired feedback but rather to an increase in accumulating noise. The feedback gain is also decreased during cFN inactivation.

Conclusions
 The effects of cFN inactivation on saccade noise and endpoint variance are explained by a decrease in feedback gain and an increase in accumulating noise. This study provides insight into the role of the cerebellum in saccade control.

Keywords
 saccades, noise, cerebellum, fastigial nucleus, endpoint variance, feedback gain, accumulating noise.

Figure 1: Overall saccade amplitude

Figure 2: Maximum velocity

Figure 3: No particular increase in variance

RESEARCH ARTICLE
Modeling Inter-trial Variability of Saccade Trajectories: Effects of Lesions of the Oculomotor Part of the Fastigial Nucleus
 Thomas Eggert^{1*}, Farel R. Robinson², Andreas Straube¹
¹ Department of Neurology, Ludwig-Maximilians-University, Munich, Germany, ² Department of Biological Structure, University of Washington, Seattle, Washington, United States of America

J Neurophysiol 92: 3355–3367, 2004. First published June 30, 2004; doi:10.1152/jn.01199.2003.

Deficits in Saccades and Fixation During Muscimol Inactivation of the Caudal Fastigial Nucleus in the Rhesus Monkey

Laurent Goffart,^{1,2,3} Longtang L. Chen,² and David L. Sparks,^{1,2}
¹Department of Psychology, University of Pennsylvania, Philadelphia, Pennsylvania 19104, ²Department of Neuroscience, Baylor College of Medicine, Houston, Texas 77030, and ³Equipe Dynamique de la Vision et de l'Action, Institut de Neurosciences Cognitives de la Méditerranée, Unité Mixte de Recherche 693 Centre National de la Recherche Scientifique—Université de la Méditerranée, 13402 Marseille, France

Abstract
 This study investigates the inter-trial variability of saccade trajectories observed in five rhesus macaques (*Macaca mulatta*). For each time point during a saccade, the inter-trial variance of eye position and its covariance with eye end position were evaluated. Data were modeled by a superposition of three noise components due to 1) planning noise, 2) signal-dependent motor noise, and 3) signal-dependent premotor noise entering within an internal feedback loop. Both planning noise and signal-dependent motor noise (together called accumulating noise) predict a simple S-shaped variance increase during saccades, which was not sufficient to explain the data. Adding noise within an internal feedback loop enabled the model to mimic variance/covariance structure in each monkey, and to estimate the noise amplitudes and the feedback gain. Feedback noise had little effect on end-point noise, which was dominated by accumulating noise. This analysis was further extended to saccades executed during inactivation of the caudal fastigial nucleus (cFN) on one side of the cerebellum. Saccades ipsiversive to an inactivated cFN showed more end-point variance than did normal saccades. During cFN inactivation, eye position during saccades was statistically more strongly coupled to eye position at saccade end. The proposed model could fit the variance/covariance structure of ipsiversive and contraversive saccades. Inactivation effects on saccade noise are explained by a decrease of the feedback gain and an increase of planning and/or signal-dependent motor noise. The decrease of the fitted feedback gain is consistent with previous studies suggesting a role for the cerebellum in an internal feedback mechanism. Increased end-point variance did not result from impaired feedback but from the increase of accumulating noise. The effects of cFN inactivation on saccade noise indicate that the effects of cFN inactivation cannot be explained entirely with the cFN's direct connections to the saccade-related premotor centers in the brainstem.

Introduction
 Saccades are rapid eye movements that are essential for visual perception. They are initiated by a burst of activity in the saccade-related premotor centers in the brainstem. The saccade trajectory is determined by the interaction of these centers and the oculomotor system. The cerebellum is known to play a role in the control of saccades, particularly in the timing and accuracy of the movements. Inactivation of the caudal fastigial nucleus (cFN) of the cerebellum has been shown to affect saccade performance in rhesus monkeys. This study aims to investigate the effects of cFN inactivation on saccade noise and endpoint variance.

Methods
 Five rhesus macaques were used in this study. Saccades were recorded during control conditions and during inactivation of the cFN. The inter-trial variability of saccade trajectories was analyzed using a model that incorporates planning noise, signal-dependent motor noise, and signal-dependent premotor noise. The model was fitted to the experimental data to estimate the noise amplitudes and the feedback gain.

Results
 The results show that saccades ipsiversive to an inactivated cFN exhibit increased endpoint variance compared to control saccades. This increase in variance is not due to impaired feedback but rather to an increase in accumulating noise. The feedback gain is also decreased during cFN inactivation.

Conclusions
 The effects of cFN inactivation on saccade noise and endpoint variance are explained by a decrease in feedback gain and an increase in accumulating noise. This study provides insight into the role of the cerebellum in saccade control.

Keywords
 saccades, noise, cerebellum, fastigial nucleus, endpoint variance, feedback gain, accumulating noise.

Figure 1: Overall saccade amplitude

Figure 2: Maximum velocity

Figure 3: No particular increase in variance

Example of NO increased endpoint variance for ipsilesional saccades

Figure 1: Vertical eye position vs Horizontal eye position

Figure 2: Horizontal final eye position vs Trial #

Figure 3: muscimol injection in the left cFN

○ ● 16UP
 ○ ● HOR
 ○ ● 16DOWN

Other tested target locations (+/-8,0); (0,+/-8); (+/-8,+/-8)

Example of NO increased endpoint variance for ipsilesional saccades

Figure 1: Vertical eye position vs Horizontal eye position

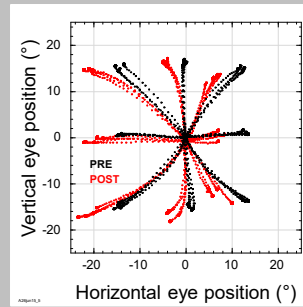
Figure 2: Horizontal final eye position vs Trial #

Figure 3: muscimol injection in the left cFN

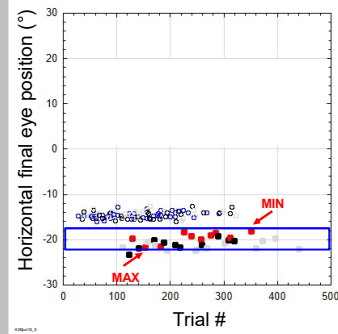
○ ● 16UP
 ○ ● HOR
 ○ ● 16DOWN

Other tested target locations (+/-8,0); (0,+/-8); (+/-8,+/-8)

Example of NO increased endpoint variance for ipsilesional saccades

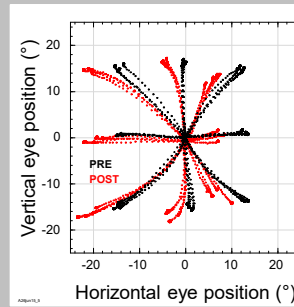


muscimol injection in the left cFN

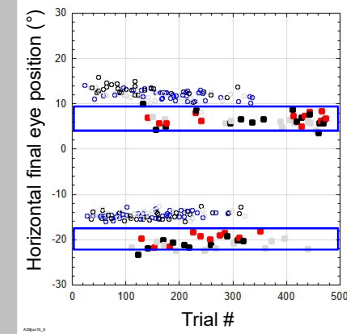


○ ● 16UP
 ○ ● HOR
 ○ ● 16DOWN
 Other tested target locations (+/-8,0); (0,+/-8); (+/-8,+/-8)

Example of NO increased endpoint variance for ipsilesional saccades



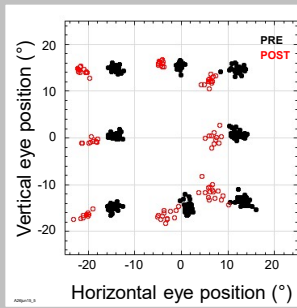
muscimol injection in the left cFN



○ ● 16UP
 ○ ● HOR
 ○ ● 16DOWN
 Other tested target locations (+/-8,0); (0,+/-8); (+/-8,+/-8)

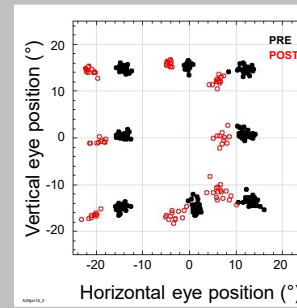
Saccade endpoints

muscimol injection in the left cFN

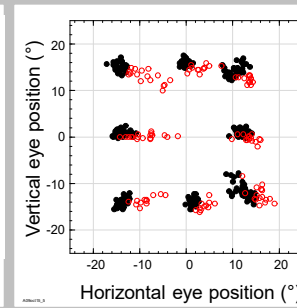


Saccade endpoints

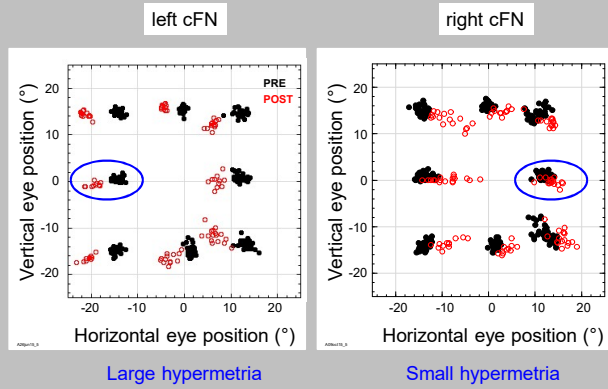
left cFN



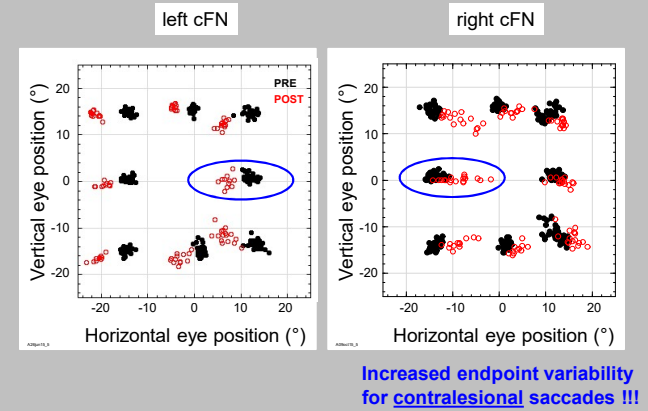
right cFN



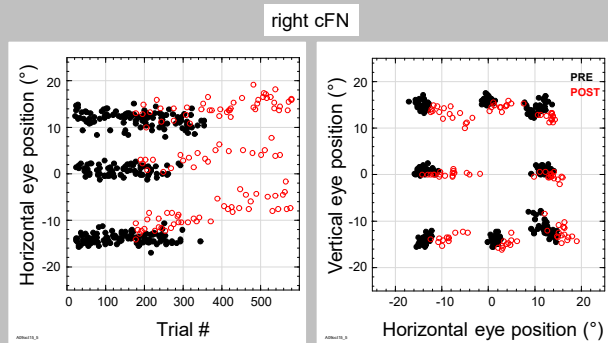
Searching for symmetry



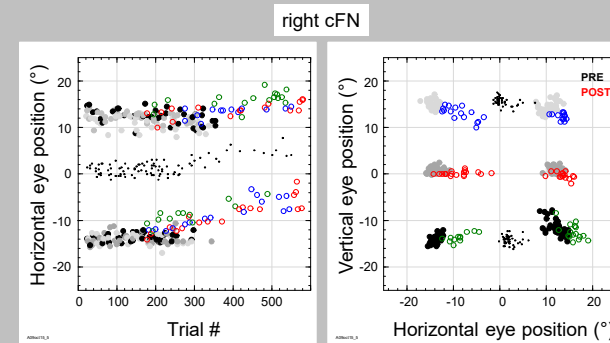
Searching for symmetry



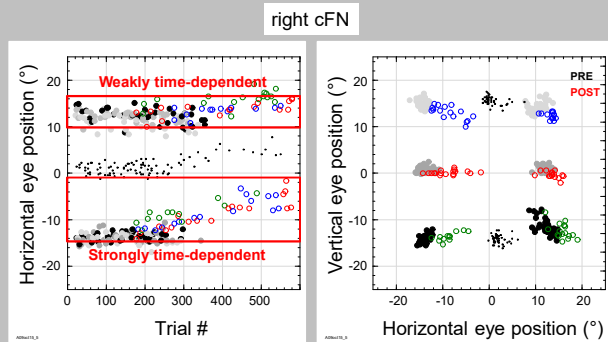
Time-dependent endpoint variability



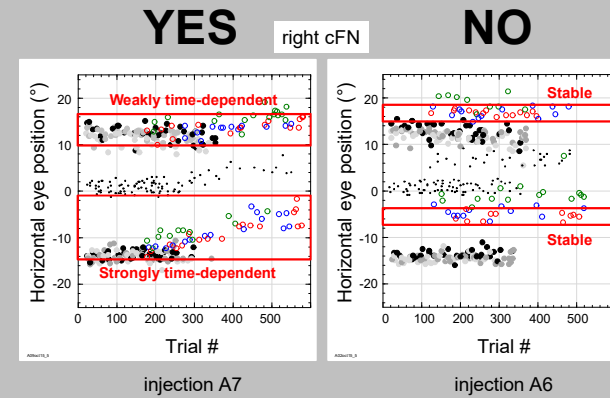
Time-dependent endpoint variability



Time-dependent endpoint variability for contralesional saccades



Time-dependent endpoint variability for contralesional saccades

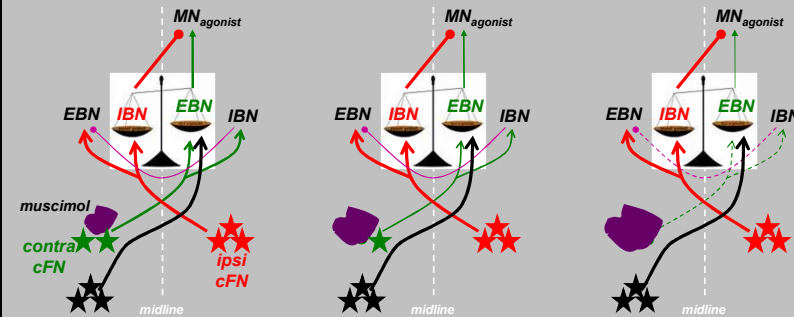


For some injection, the magnitude of dysmetria increases with the trial number.

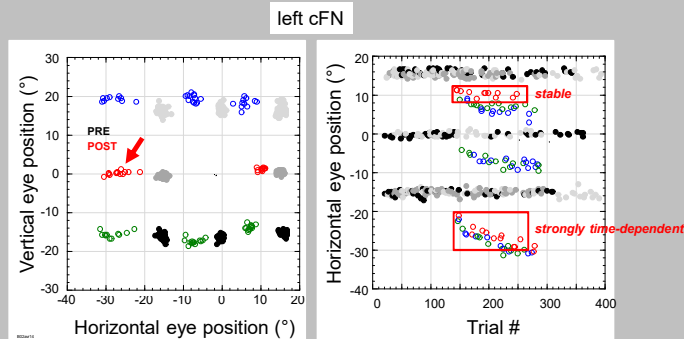
For some injection, the magnitude of dysmetria increases with the trial number.

The drug diffuses with the trial number.

As the drug diffuses, the number of inactivated neurons increases = the number of active neurons diminishes.
 Reduced number of active neurons → reduced peak velocity unchanged duration → enhanced hypometria.



Time-dependent endpoint variability also for ipsilesional saccades

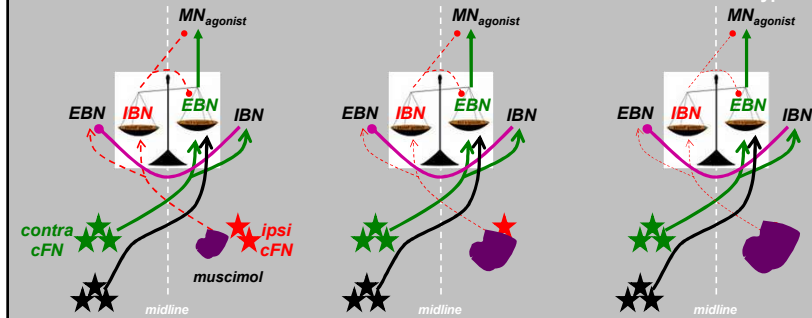


For some injection, the magnitude of dysmetria increases with the trial number.

The drug diffuses with the trial number.

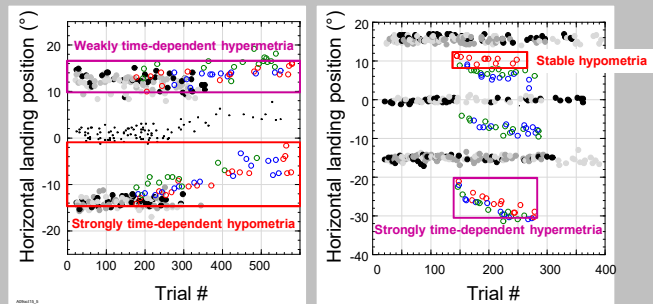
As the drug diffuses, the number of inactivated neurons increases = the number of active neurons diminishes.

Reduced number of active neurons → unchanged/enhanced peak velocity → enhanced duration → enhanced hypermetria



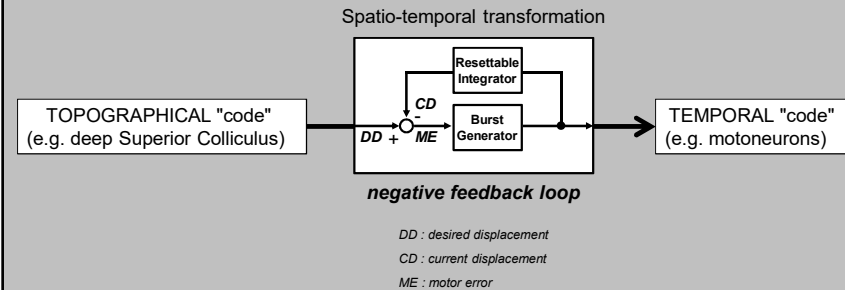
CONCLUSION

1. The **NUMBER** of active neurons contribute to the saccade dynamics
2. Different evolutions of ipsi/contra-lesional dysmetria indicate **TWO UNRELATED DEFICITS**



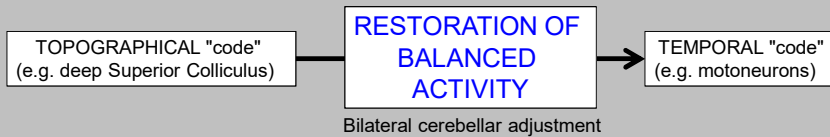
CONCLUSION

1. The **NUMBER** of active neurons contribute to the saccade dynamics
2. Different evolutions of ipsi/contra-lesional dysmetria indicate **TWO UNRELATED DEFICITS**
3. Between the topographical and temporal "codes", a negative feedback loop



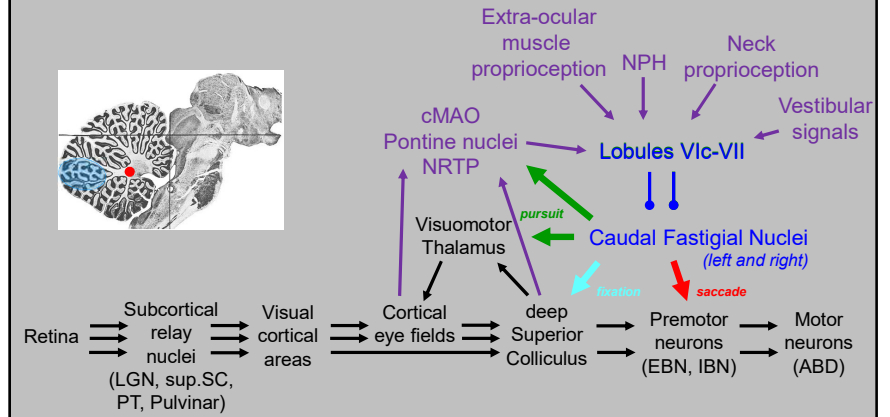
CONCLUSION

1. The **NUMBER** of active neurons contribute to the saccade dynamics
2. Different evolutions of ipsi/contra-lesional dysmetria indicate **TWO UNRELATED DEFICITS**
3. Between the topographical and temporal "codes", the **RESTORATION OF SYMMETRY**

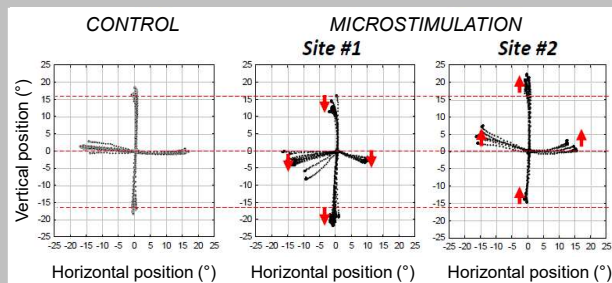


The orienting movement is not saccadic (abrupt and very rapid) for any teleological purpose. It is saccadic merely because the flow of activity involves the activity of neurons whose pattern of firing rate is bursting.

Working hypothesis : Cerebellar adjustment of bilateral activity



DYSMETRIA OF VERTICAL COMPONENT



Acknowledgments

