Functional neural mechanisms of psychogenic paralysis: “hysteria” and hypnosis

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Dissociation of body and mind: from neurology to psychiatry

Clinical lesson on hysteria at the Salpêtrière , Paris, 1887

Hysteria = conversion

DSM-IV (APA 2000)

“SOMATOFORM DISORDER”

- **loss or distortion of neurological function, e.g. paralysis, anaesthesia, blindness**
- **not explained by organic lesion or another medical disease**
- **related to psychological stress or conflict**
- **not consciously produced or intentionally feigned**

Hysteria = conversion = functional disorder

~~DSM-IV (APA 2000)~~ → **DSM-V** (APA 2013)

"SOMATOFORM DISORDER"

- **loss or distortion of neurological function, e.g. paralysis, anaesthesia, blindness**
- **not explained by organic lesion or another medical disease**
- ~~related to psychological stress or conflict~~
- ~~not consciously produced or intentionally feigned~~
- **clinical evidence of inconsistency or incongruity**
- **significant distress or impairment in social functioning**

ICD-10 (WHO 1993)

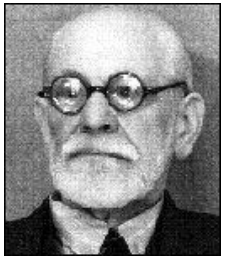
"DISSOCIATIVE DISORDER"

- **motor / sensory / cognitive / personality change**
- **no physical disorder explaining the symptoms**
- **association in time with stressful events or needs**

Body and mind in conversion: from clinical observations to theories

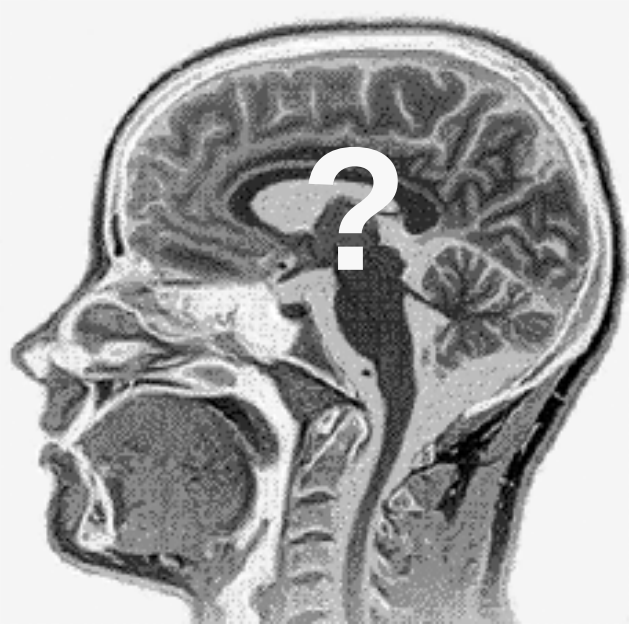
- **Charcot 1887-1892:** Invisible alteration in nervous system (neurosis), anomaly in motor will; caused by imagination, suggestions, altered psychological states (cf. hypnosis)

- **Janet 1889-1894:** Dissociation between conscious and unconscious control of behaviour or thoughts; reflecting lack of integration (due to hypnosis, emotions, fixed ideas, etc.)

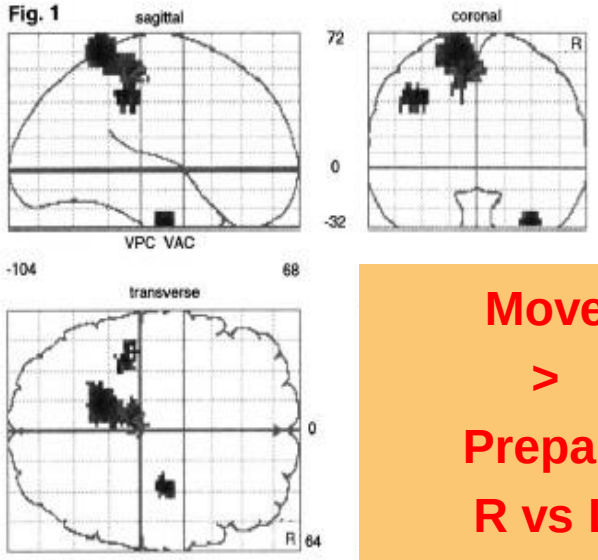


- **Freud & Breuer 1895-1909:** Affective motives and conflicts, unconsciously repressed and transformed into symbolic complaints (“conversion”)

How emotion and stress are « converted » into neurological symptoms? (e.g. paralysis)



PET imaging of conversion paralysis

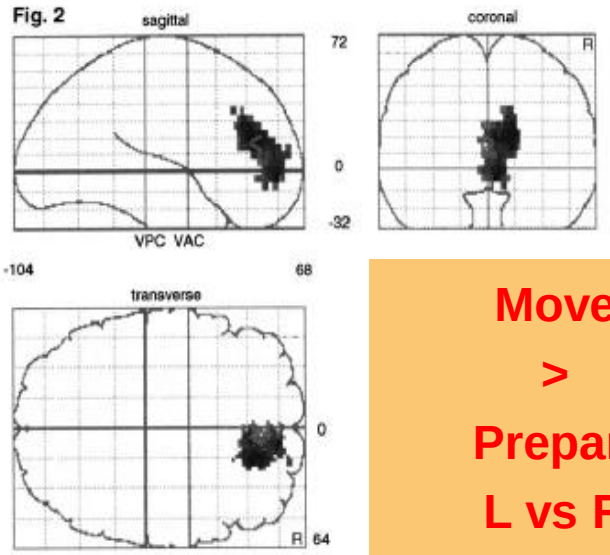


Move > Prepare
R vs L

- 45 year-old right handed woman
- **left leg** paralysis for 2 and 1/2 years

Inhibition?

or conflict?
error monitoring?

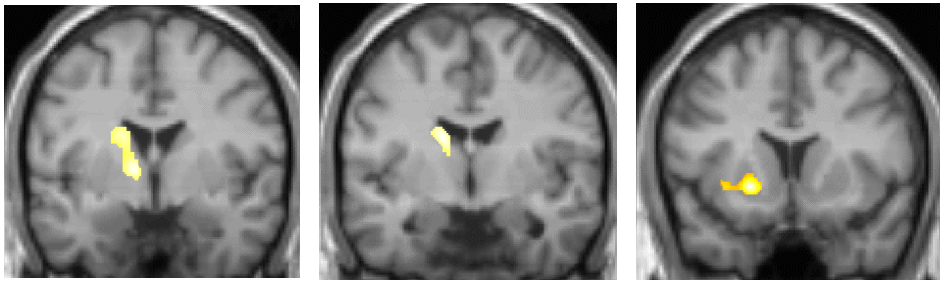


Move > Prepare
L vs R

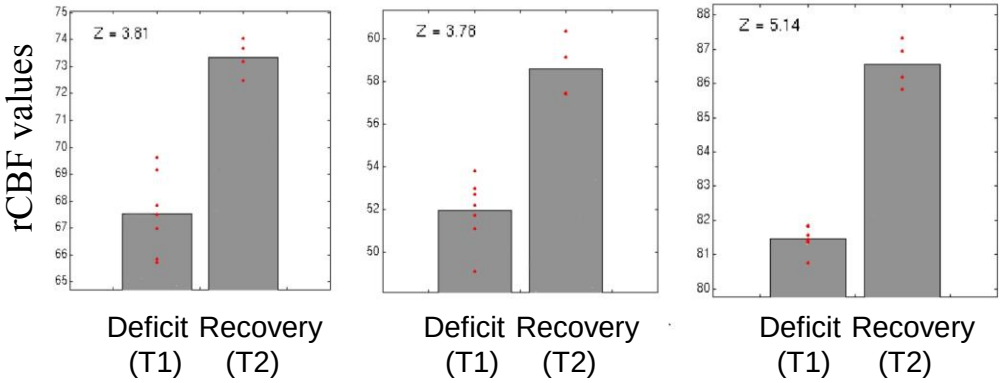
Marshall et al., Cognition 1997

SPECT of conversion paralysis and recovery

stimulation T2 (recovery) > T1 (deficit)



Thalamus *Caudate* *Putamen/Pallidum*



Tc99 SPECT scan + SPM96

- *rest + bilat vibro-tactile stimulation*
- *during deficit (T1) vs after recovery (T2)*

Unilateral motor (± sensory) loss

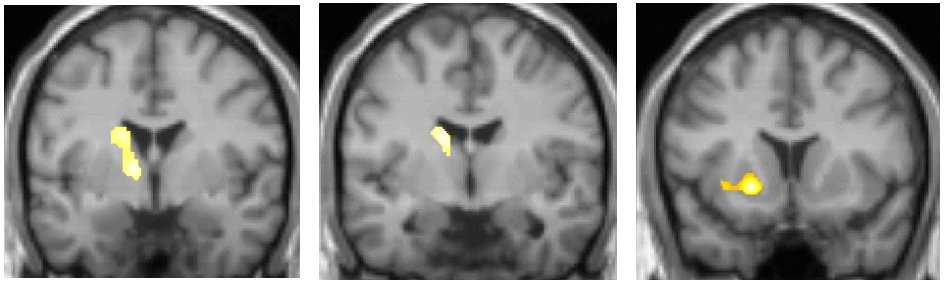
- n = 7 patients (16-54 year-old)
- onset < 2 months
- acute or chronic stress factors all cases
- follow-up until recovery (8-18 weeks)

NB: stimulation vs rest, scan T1: symmetric effects in sensory-motor cx

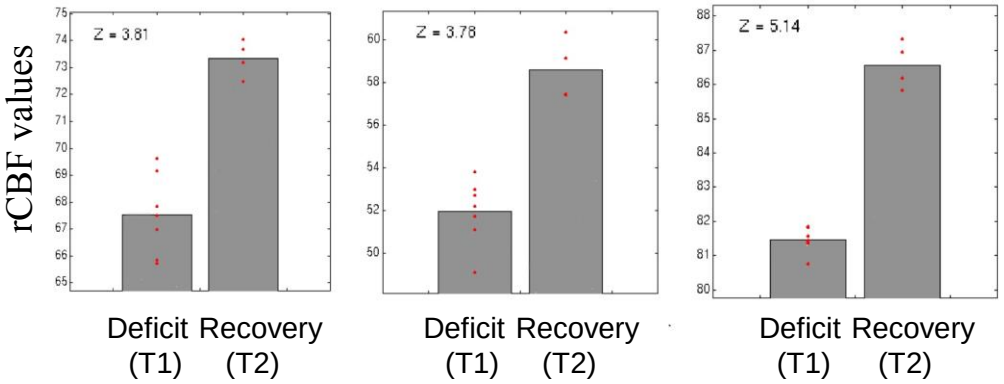
Vuilleumier et al., Brain 2001

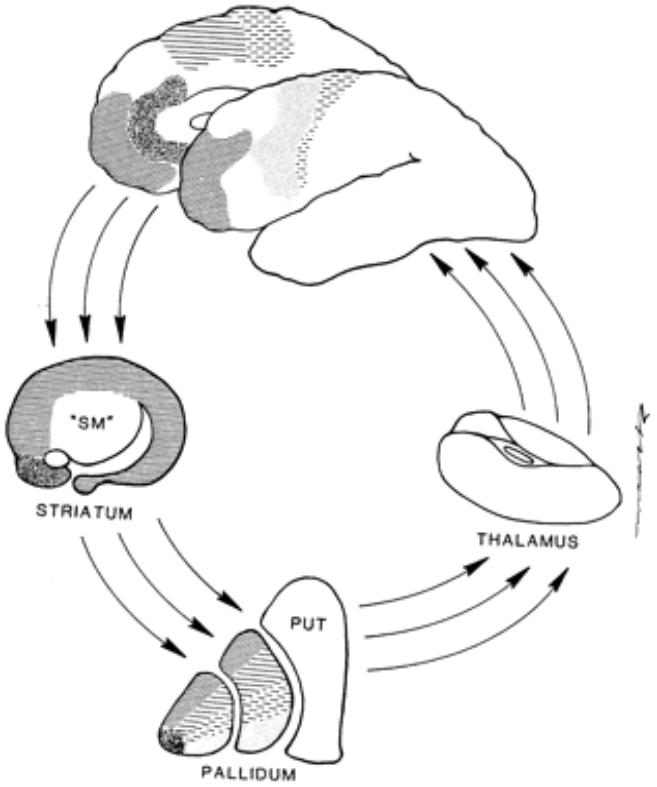
SPECT of conversion paralysis and recovery

stimulation T2 (recovery) > T1 (deficit)



Thalamus ***Caudate*** ***Putamen/Pallidum***

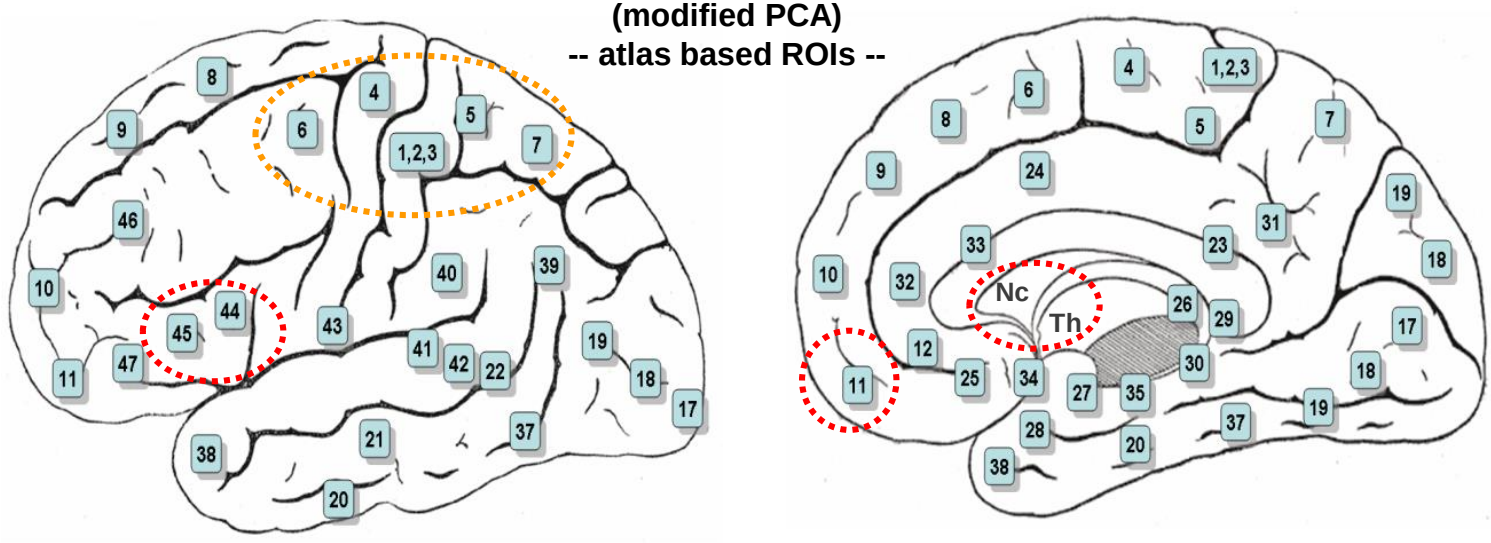




Cortical-basal ganglia loops:
convergence of motor and motivational signals

Functional network analysis

Scaled Subprofile Model (SSM) analysis
(modified PCA)
-- atlas based ROIs --

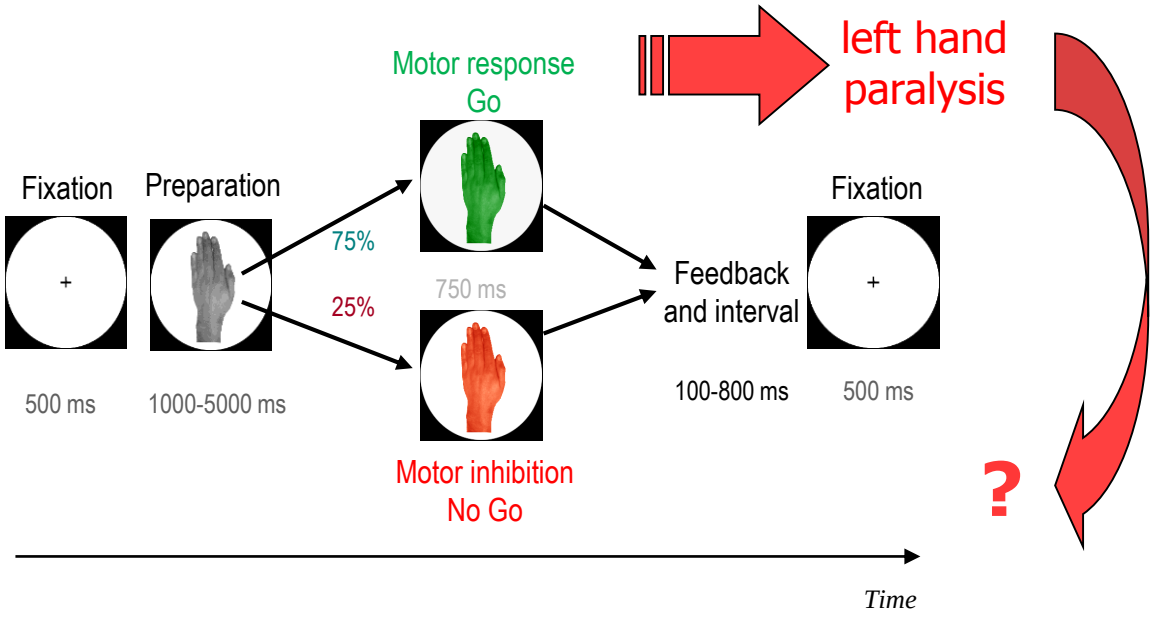


• ***Sensori-motor network:*** *primary motor and somatosensory areas (BA4-6, 1-2-3, 5-7), associated with* ***stimulation*** *(T1 and T2 scan, bilateral).*

• ***Fronto-limbic network:*** *<u>prefrontal</u> cx (BA11,44-45) & <u>subcortical</u> regions (caudate-thalamus), associated with* ***conversion deficit*** *(T1 scan, ↑ contra hemisphere).*

Vuilleumier et al., Brain 2001

Motor inhibition vs impaired intention?



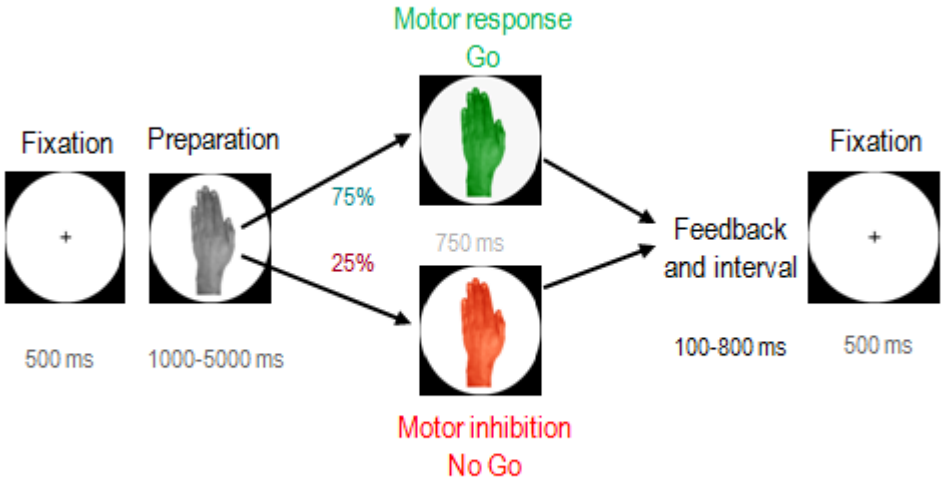
Modified go-gono paradigm, event-related fMRI

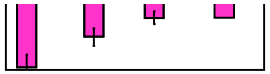
Similar mechanisms for conversion paralysis (CP) and hypnotic paralysis (HP)?

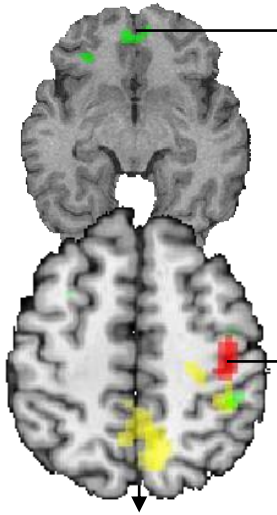
How different from voluntary simulation?

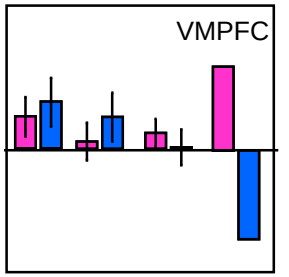
Neuron 2009; 62(6):862-75
Neuroimage 2009; 47(3):1026-37
Ann Medico-Psychol 2010; 168(4):306-10

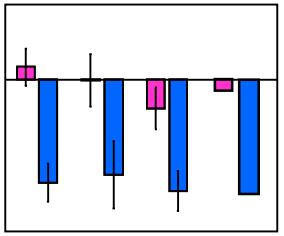
Results (1) - Motor preparation

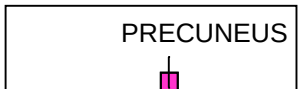


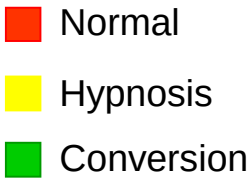




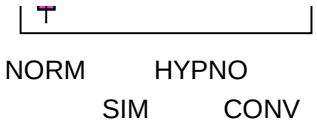






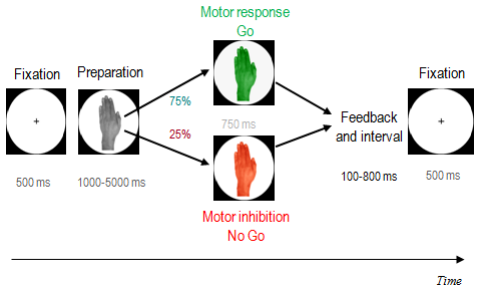


→ **Intact voluntary activation of contra M1 in both CP and HP**

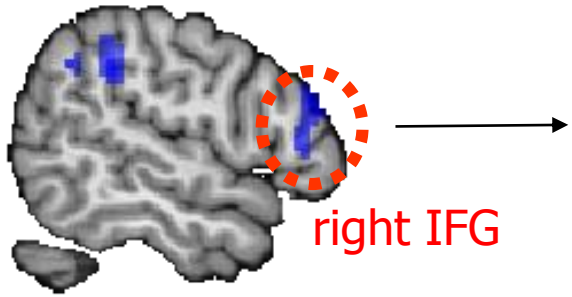


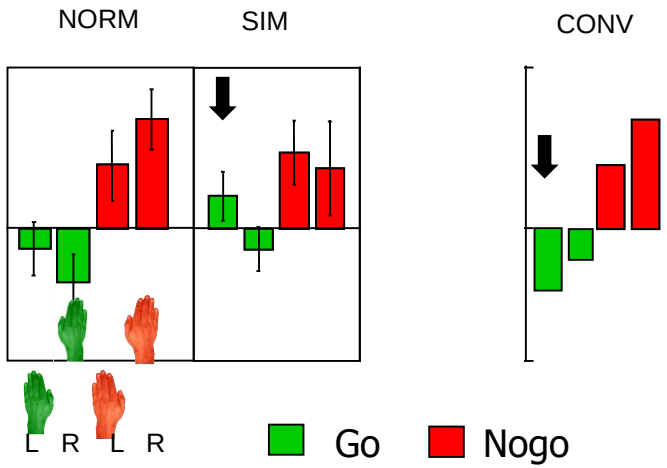
Neuron 2009; 62(6):862-75

Results (2) - Motor inhibition



Nogo > Go
in normal condition

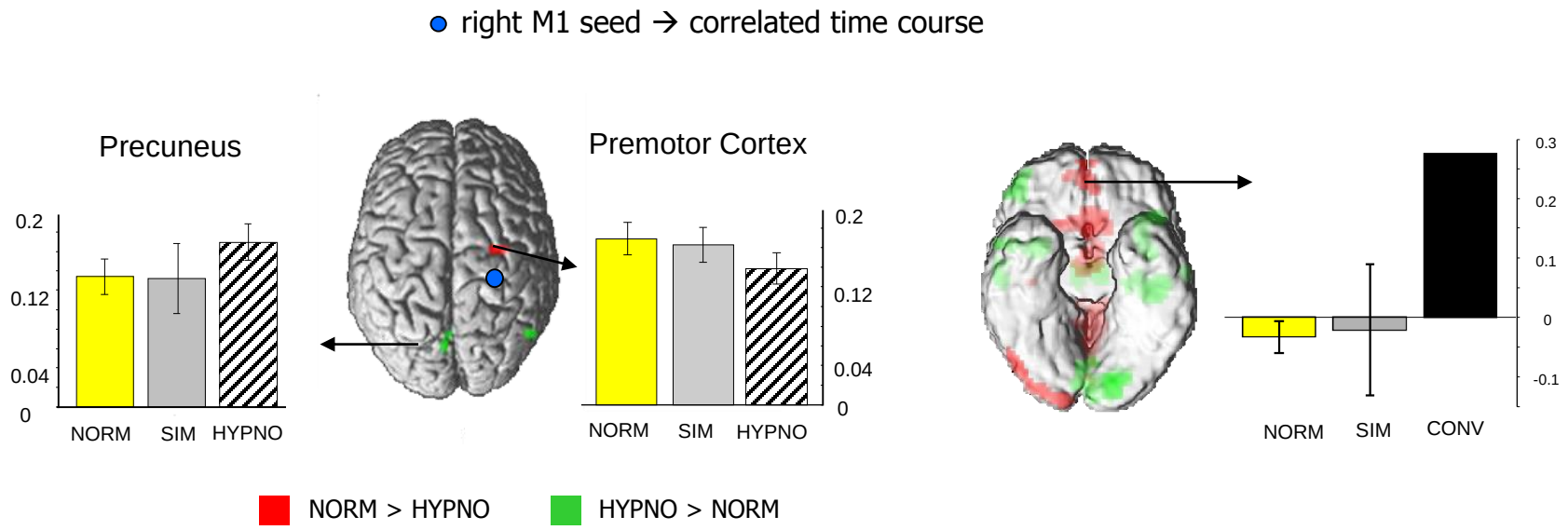




→ **voluntary inhibitory control mediated by rIFG**
(cf. Aron et al. 2007, 2015; Hampshire et al. 2010; etc.)

→ **recruited by NoGo trials and simulators (not "paralysed Go")**

Results (3) - functional connectivity of M1



1. Less connected with **premotor cortex** (vs normal state)

2. More connected to **precuneus** *under hypnosis*

3. More connected to **vmPFC** *in conversion paralysis*

Conclusion:

- Motor intentions still activate **M1** despite paralysis, in both conversion and hypnosis
- Inhibitory (executive) control mechanisms mediated by **rIFG** are not differentially recruited during paralysis
- Conversion and hypnotic paralysis are associated with distinctive activity patterns: **VMPFC** in conversion vs **Precuneus** in hypnosis (*motor preparation and connectivity*), **rIFG** in hypnosis (*across all conditions*)

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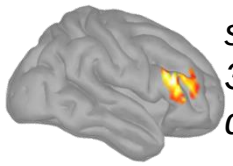
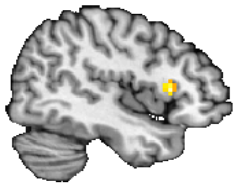
- Conversion and hypnotic paralysis are associated with distinctive activity patterns: **VMPFC** in conversion vs **Precuneus** in hypnosis (*motor preparation and connectivity*), **right IFG** in hypnosis (*across all conditions*)
- right IFG also :



selective activation in EEG sources 350-400 ms post "go" cue during hypnotic paralysis

Cojan et al., Cortex 2013, 49:423-36

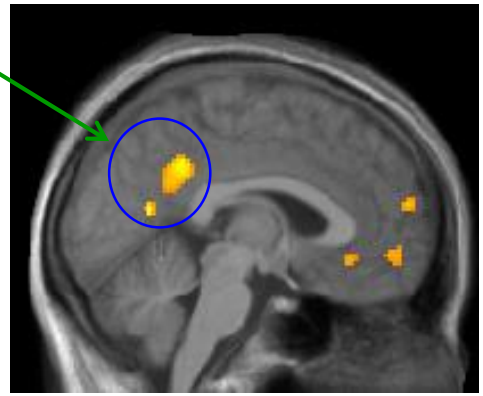


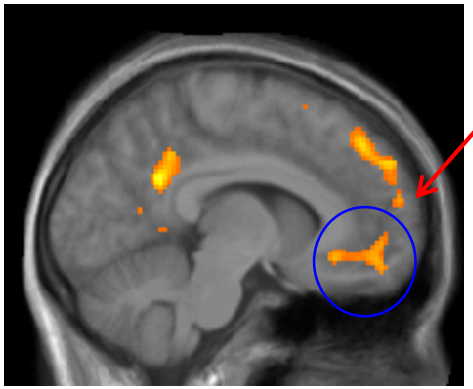
activation in attention (flanker) task predicting higher susceptibility to hypnosis

Cojan et al., NIMG 2015, 117:367–74

Precuneus and ventro-medial PFC: access to self-related representations

more activated in hypnosis





more activated in conversion

e.g., mental imagery with self-centered components (Cavanna 2006), self-related episodic memory (Lou 2004), self-reflective processing (Baars 2003).

e.g., rating personal traits or affective preferences (Mitchell 2006), first-person perspective for past or future events (D'Argembeau 2007), evaluation and regulation of emotions (Oschner 2004).

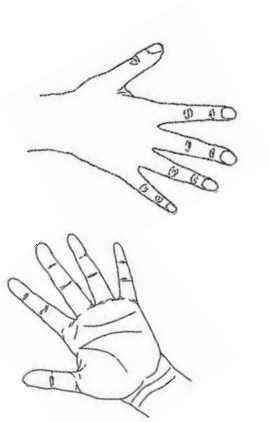
"imaginary self"

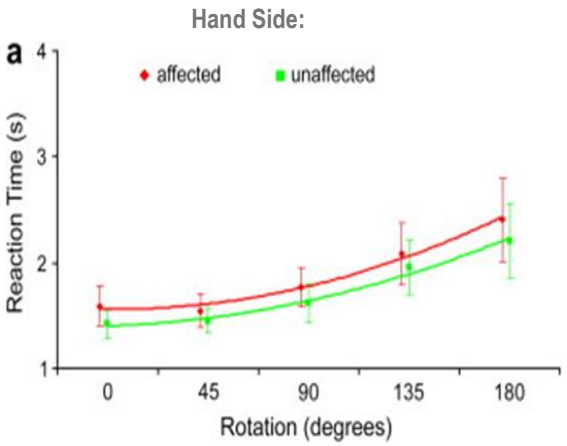
"affective self"

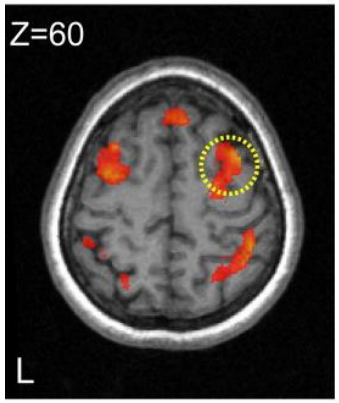
VMPFC activated by motor imagery in conversion: increased self-monitoring?

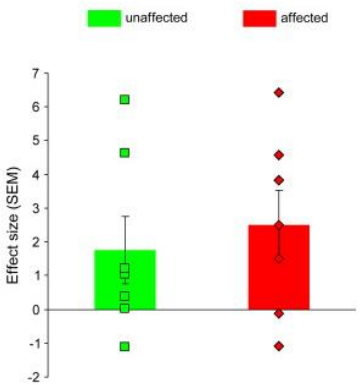
DeLange, Toni & Roelofs (2008):

- implicit and explicit motor imagery tasks (hand rotation)
- 7 patients with conversion paralysis

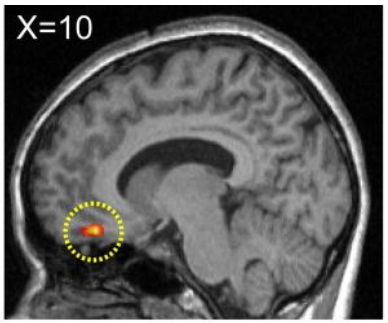


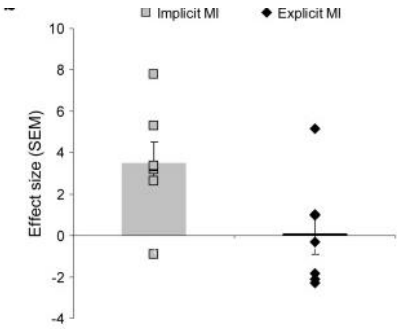






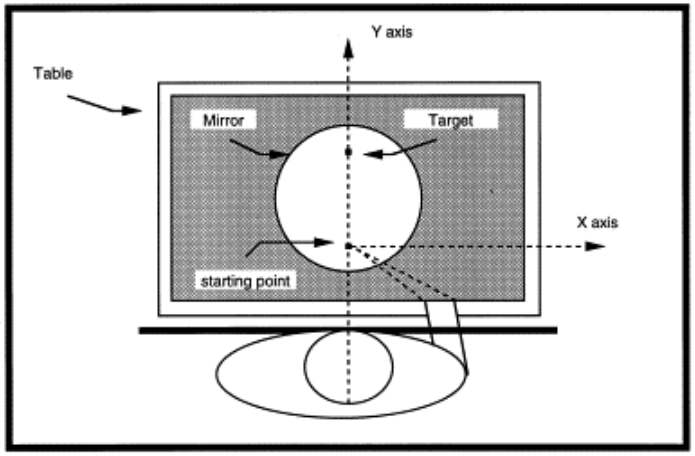
→ Normal activation in motor cx for both hands (during implicit and explicit task)

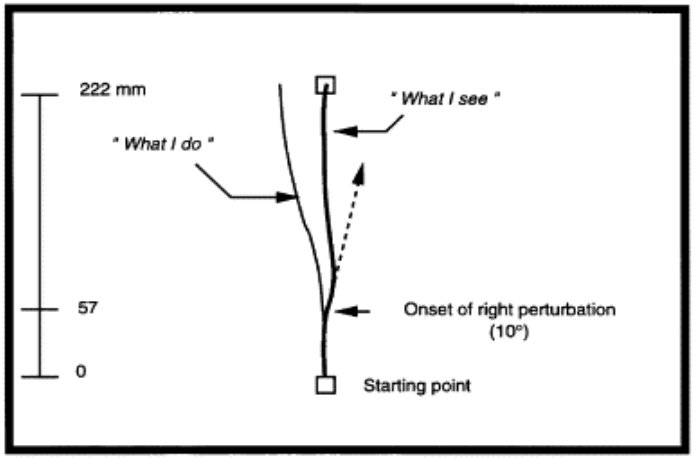




→ Selective increase in VMPFC for affected hand (during implicit task)

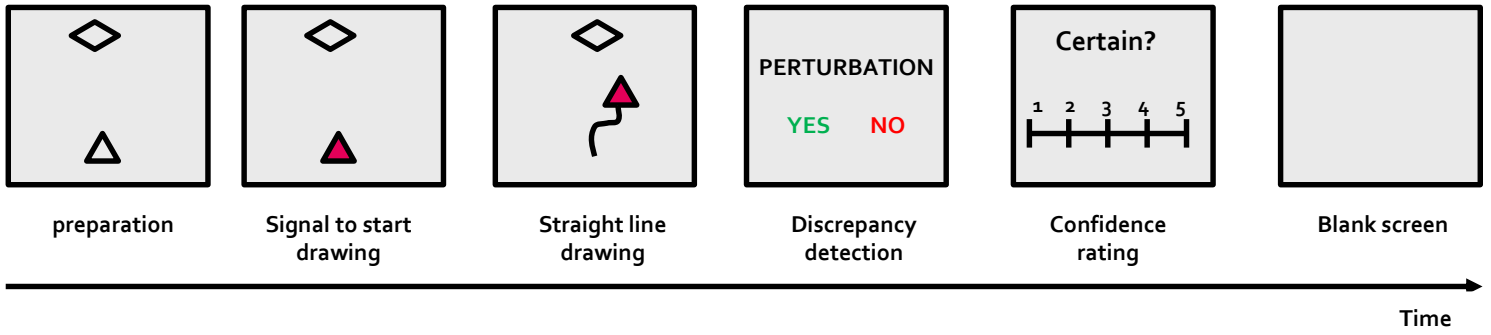
Self-monitoring and motor awareness



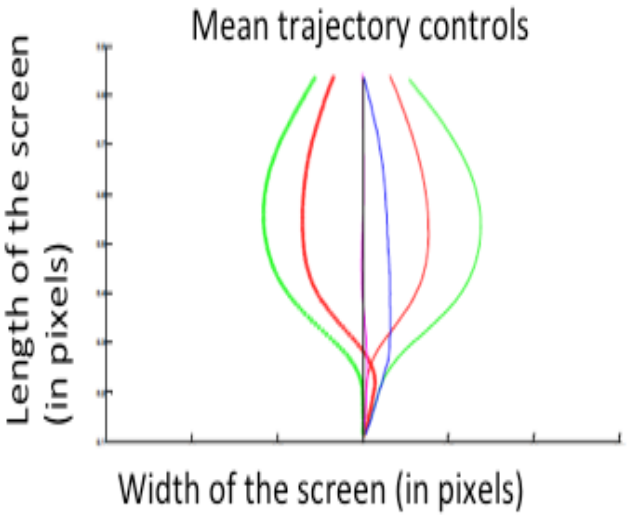


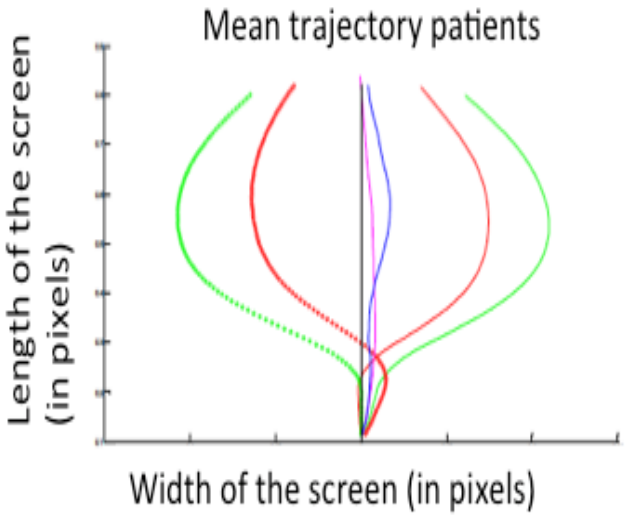
Fourneret & Jeannerod, 1998

1) Training
2) Threshold procedure
3) Test sessions (2x 104 trials)



Behavioral performance





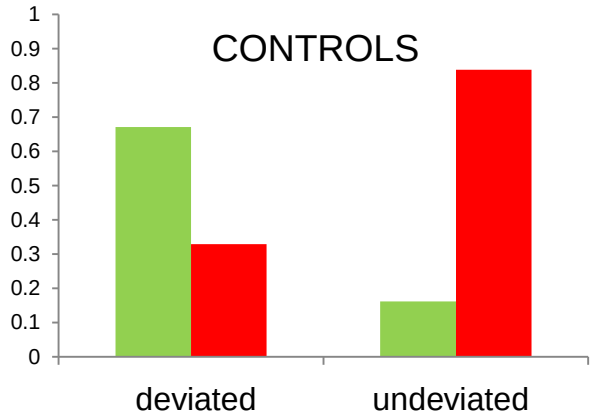
Trial type:

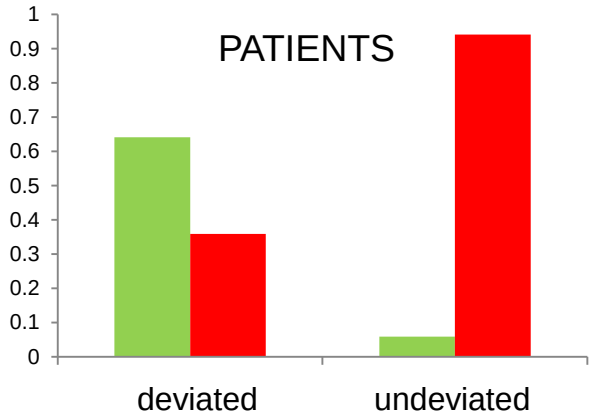
- Undeviated
- Deviated + «No» (leftward)
- Deviated + «Yes» (leftward)

Deviation thresholds:

Controls: mean 13.3° ±8.3

Patients: mean 21.4° ±8.4



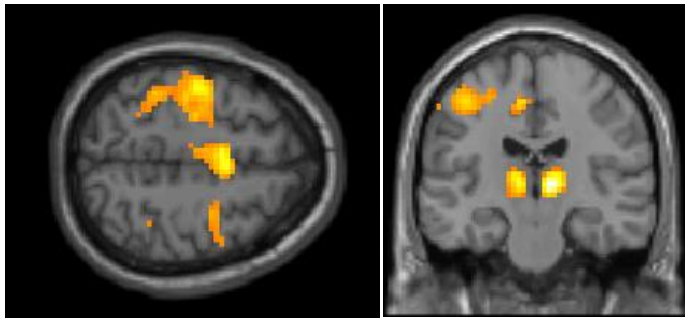


% of responses:

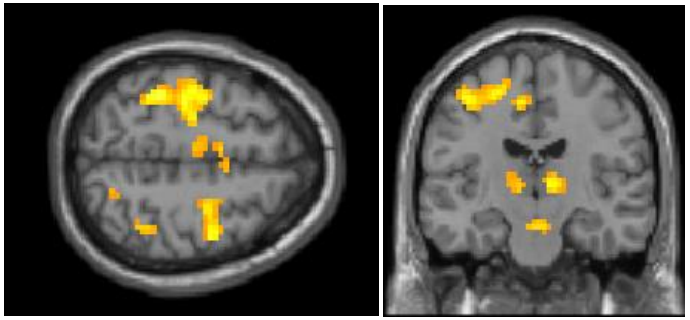
- yes
- no

fMRI results (1): movement period

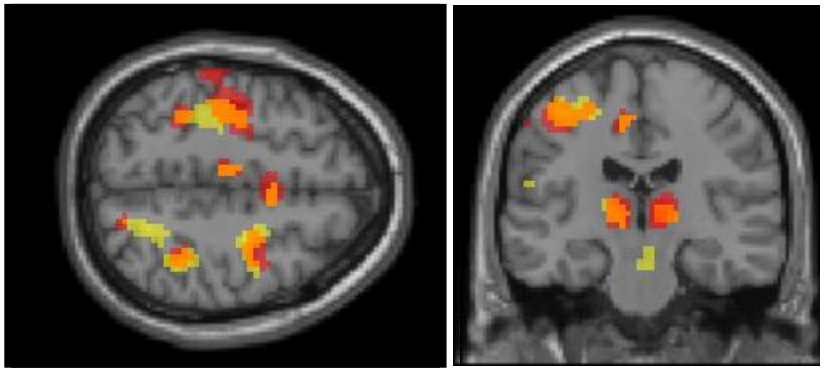
Healthy Controls



Conversion Patients



HC

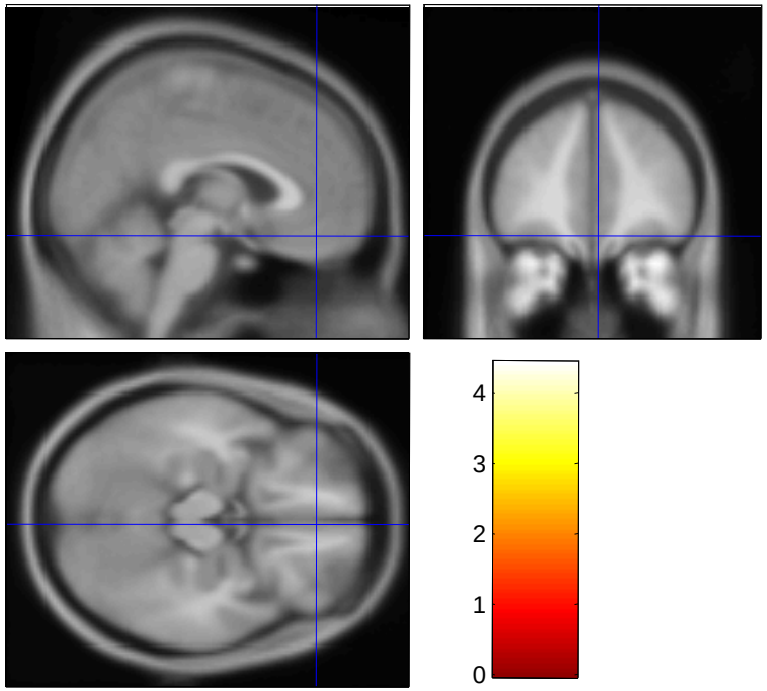


CD

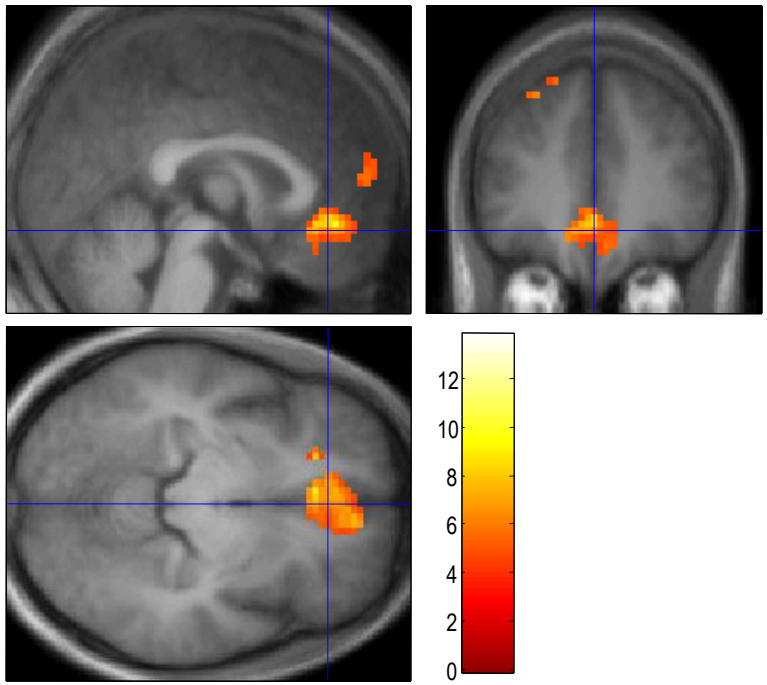
→ **similar motor network**

fMRI results (2): movement preparation

Healthy Controls



Conversion Patients



z= 4.04, p< 0.001
K=65 (fwe)

→ **activation to anticipation of movement, rather than monitoring its execution**

Summary

1) Increasing insights on neural substrates of "psychogenic" paralysis:

- cf. Charcot: functional changes without lesion in motor pathways
- but: no loss in "motor intention" or imagery (M1, both conversion and hypnosis)
- distinct from simulation or voluntary inhibition (e.g. no-go)

2) Commonalities, but also differences between conversion and hypnosis:

- cf. Charcot / Janet: connectivity of motor system with internal representations
- but: increases in vmPFC (affective representations) for *conversion*
  vs. increases in precuneus (imaginal/mnesic representations) and rIFG (cognitive control) for *hypnosis*

3) Changes in cortico-basal ganglia loops during conversion paralysis:

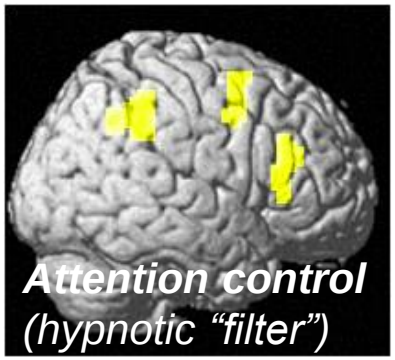
- pathways by which emotional states can modulate/block motor execution
- cf. Freud: may arise unconsciously, be experienced as non-volitional, and modulated by past history / learning

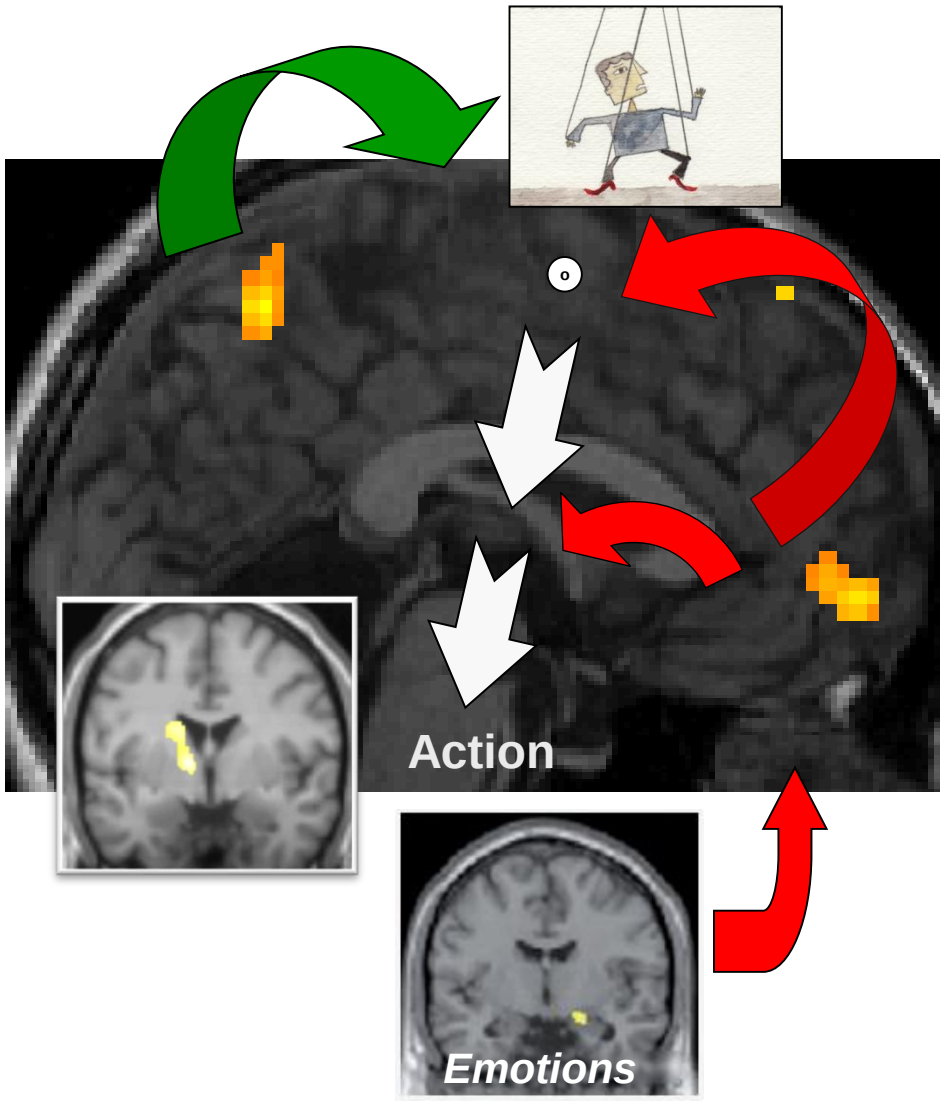
4) Distinctive role of right IFG in hypnosis:

- related to hypnotic suggestion and individual susceptibility
- role in cognitive control / attentional focus and filtering

Action control by self relevant representations

Hypnotic suggestion:
sensory imagery and memories
(imaginary self)





Conversion:
self-relevant affective representations and memories
(affective self)

THANK YOU FOR YOUR ATTENTION

Thanks to:

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"Poor hysterics. First they were treated as victims of sexual trouble... then of moral perversity and mediocrity... then of imagination. Among the various rehabilitation which our age has seen, none are more deserving or humane. It is a real disease, but a mental disease."

W. James (1896)

Neurophysiology of conversion disorders

- **ERPs** (SEP, VEP, BAEP) & **MEG**: standard tests are usually normal …

- but Ø near threshold ? **(e.g. Levy & Behrman 1970, n= 7)**
- but Ø habituation? (SEP: **Moldofsky & England 1975, n= 5;** SCRs: **Horvath et al 1980, n= 11)**
- but Ø P300 ? **(Lorenz et al. 1998, n= 1);** CNV **(Drake, 1990);** MMN **(James et al. 1989, n= 10)**
- or ↓ attentional N1 in VEP ? **(e.g. Schoenfeld et al., 2011, n= 1)**

- EEG spectrum: ↓ratio high/low frequency L/R frontal **(Drake et al. 1980, n= 10)**

- **TMS:** normal + symmetric MEPs **(Meyer et al. 1992, n= 15),**

  decreased excitability in contra RH, but no change with recovery **(Foong et al. 1997, n= 2) ;**
  or decreased only during movement imagination **(Liepert et al 2008, n= 8)**

- **SPECT, PET, fMRI**

Neuroimaging of conversion disorders

Motor disorders:

- **HMPAO-SPECT**: decreased R parietal, increased R frontal **(Tiihonen et al. 95, n= 1);** decreased L temporal or L parietal **(Yazici et al. 98, n= 5);** decreased R frontobasal and temporal, increased L insula **(Saladini et al. 2006, n= 1)**
- **ECD-SPECT:** decreased activity in basal ganglia & thalamus **(Vuilleumier et al. 2000, n= 7)**
- **PET**: increased orbitofrontal + cingulate **(Marshall et al. 97, n= 1);** decreased L frontal **(Spence et al. 2000, n= 3)**
- **fMRI**: decreases in motor cx **(Burgmer et al. 06, n= 4)** and medial prefrontal **(DeLange et al. 07, n= 8)**

Somatosensory disorders:

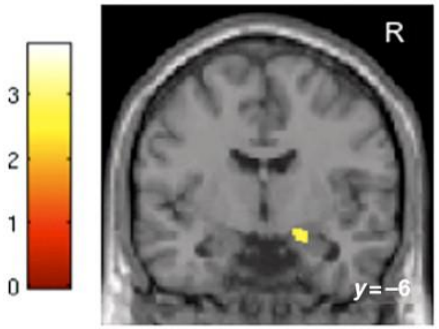
- **fMRI:** decreased SI, SII, thalamus, insula, ACC post/SMA + increases in rostral ACC -- but heterogenous **(Mailis-Gagnon et al. 03, n= 4)**
- **fMRI:** no activation of SI during unilateral stimulation -- but during bilateral **(Ghaffar et al 06, n=3).**

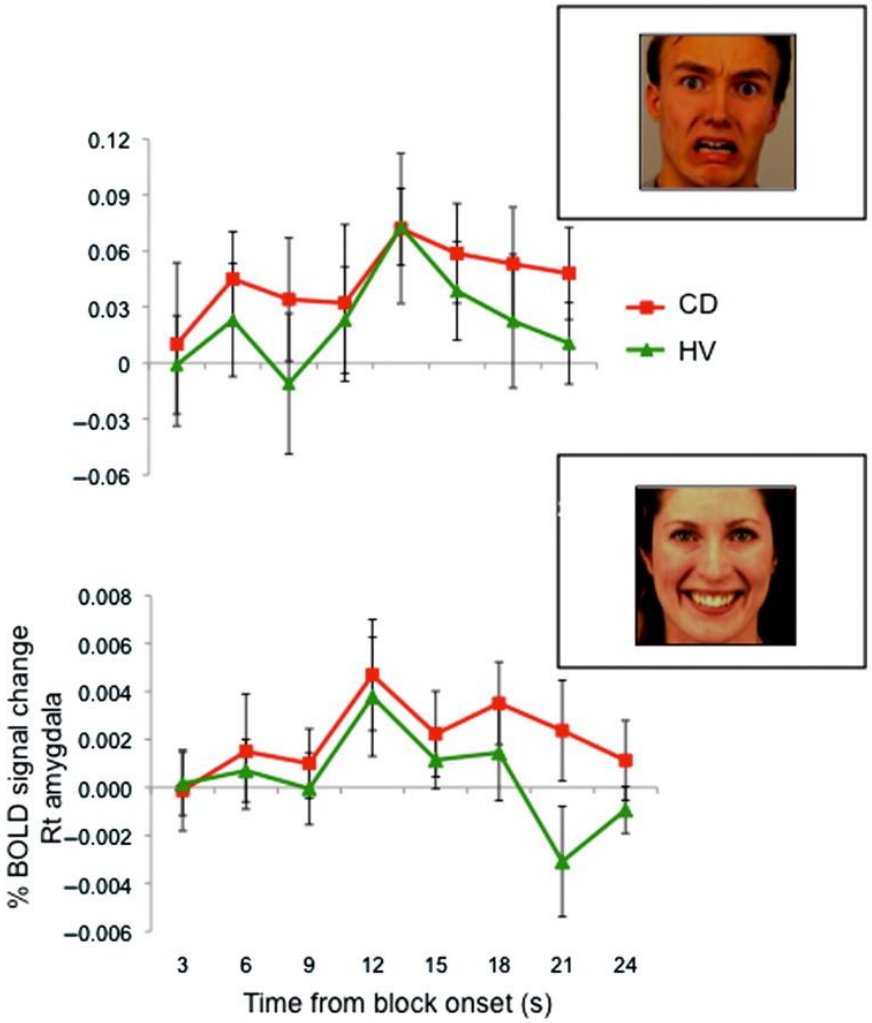
Visual disorders:

- **fMRI:** decreases in occipital cx + increases in thalamus, striatum, inferior frontal cx **(Werring et al. 04, n= 5)**

Emotion reactivity in conversion patients

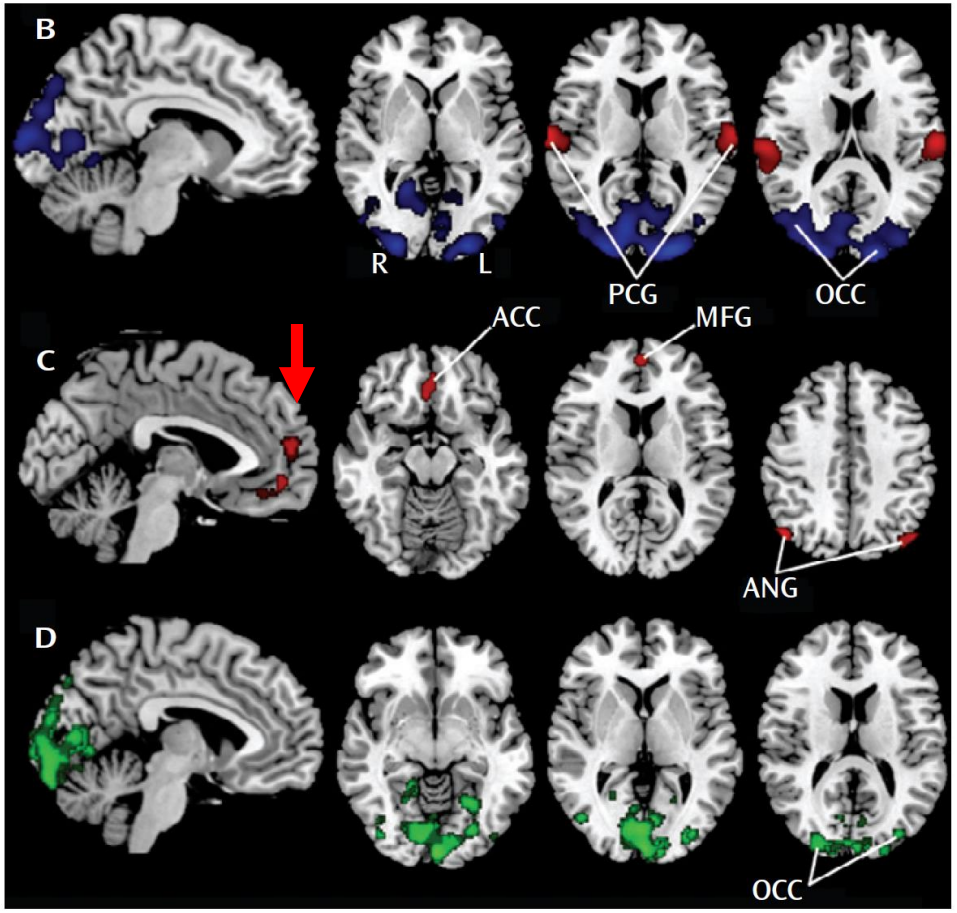
- N = 16 patients with CD
- Positive movement disorders (tremor, dystonia or gait)



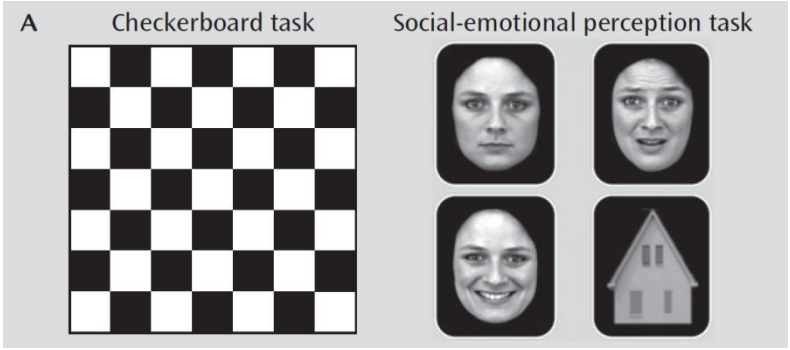


Voon et al. 2010

fMRI of conversion blindness



Decreased activity during visual loss *(faces, not checkerboard)*
Increased activity during visual loss *(emotion > neutral)*
Increased connectivity with ACC, MFG, and ANG during visual loss



- 25 year-old man
- recurrent episodes of blindness, preceded by visual hallucinations
- triggered by death of close friend

Becker et al., Am J Psy 2013

Network analysis using ICA during motor task

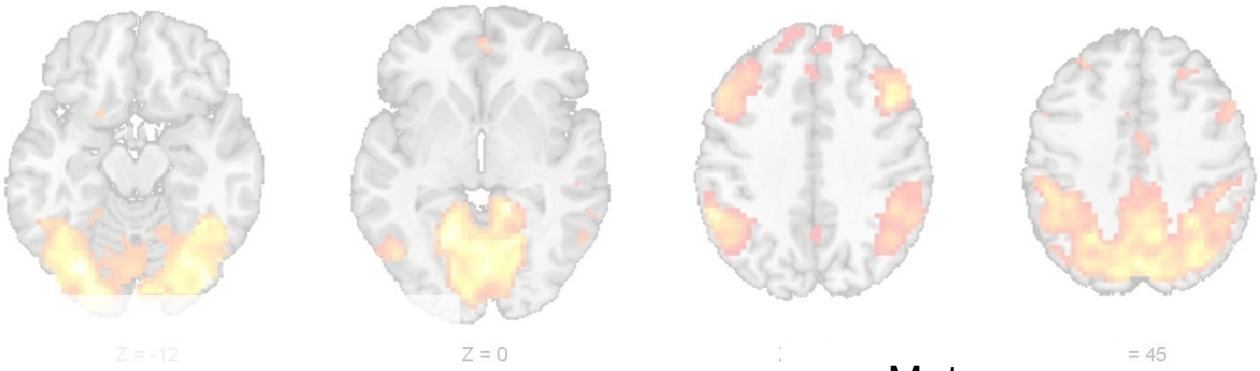
→ 7 distinct coherent networks:

Visual – extrastriate

Visual – striate

Parieto-frontal

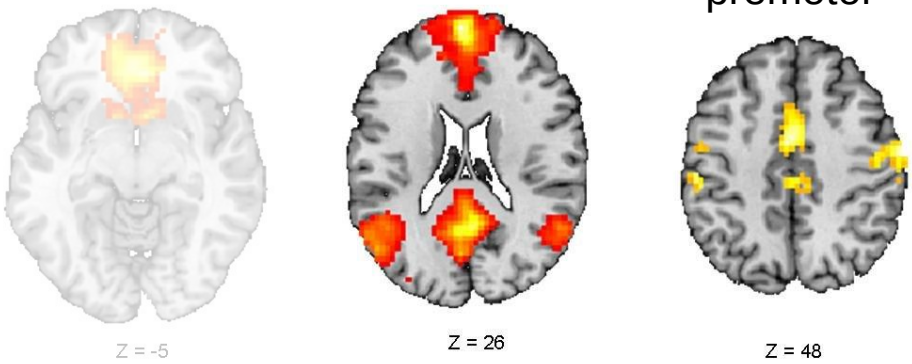
Parietal - posteromedial



VMPFC

Default mode

Motor - premotor

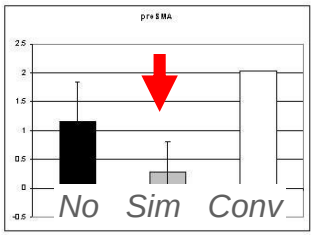


→Difference in patient with L arm paresis vs controls

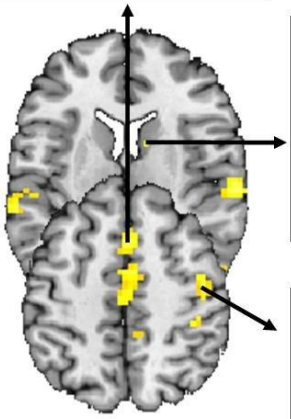
Cojan & Vuilleumier, 2011

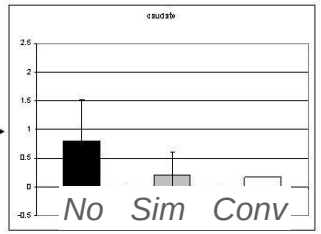
Network analysis using ICA during motor task

Motor network

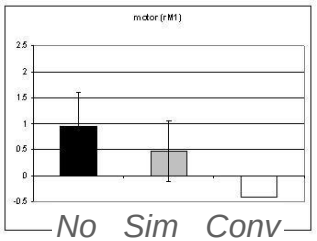


preSMA / cACC





Caudate R



Motor cx (M1)

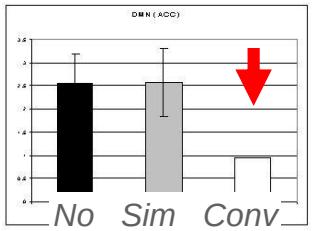
Cojan & Vuilleumier, 2011

Network analysis using ICA during motor task

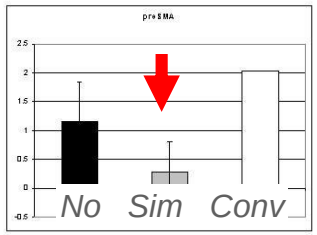
Default mode network

Motor network

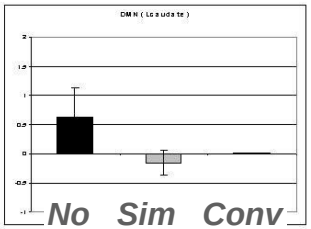
VMPFC



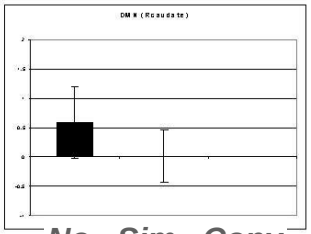
preSMA / SMA

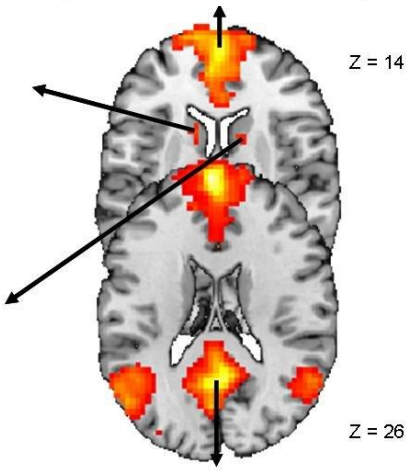


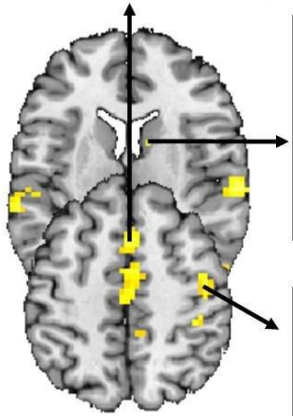
Caudate L

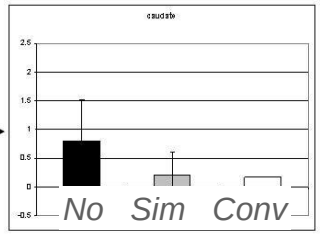


Caudate R

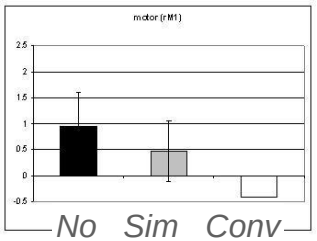






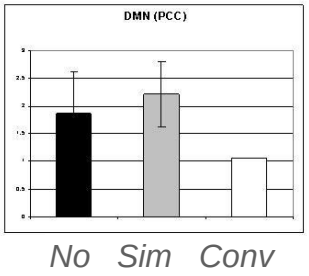


Caudate R



Motor cx (M1)

PCC



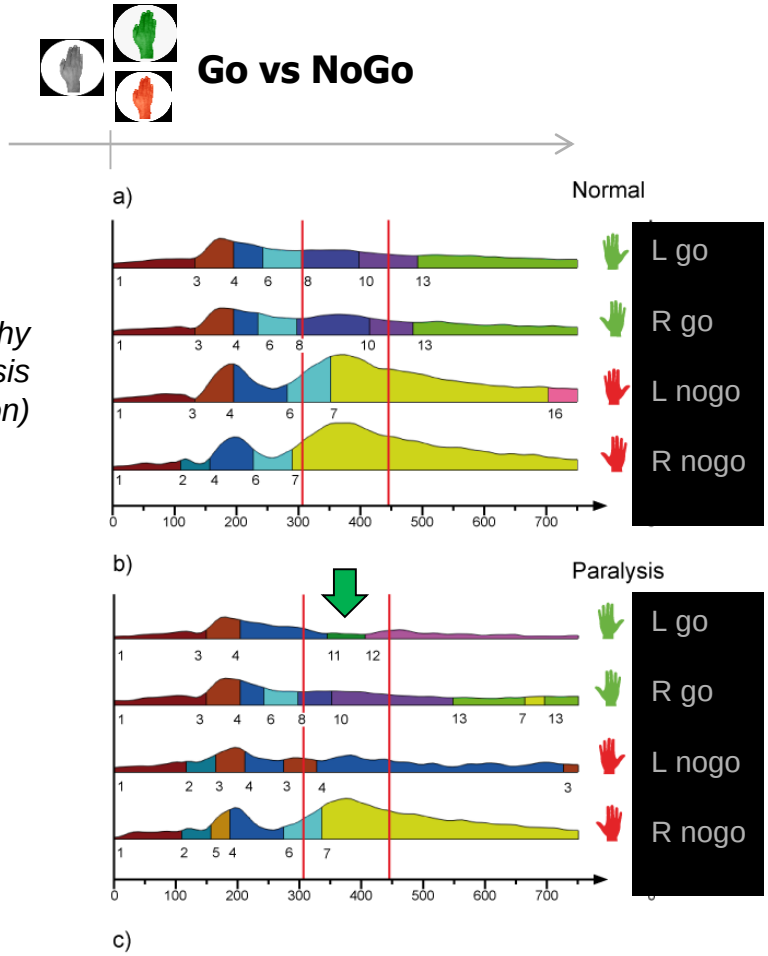
→ **Reduced connectivity with DMN for VMPFC and caudate, only for conversion**

→ **Reduced connectivity with motor network for R caudate, in both conditions**

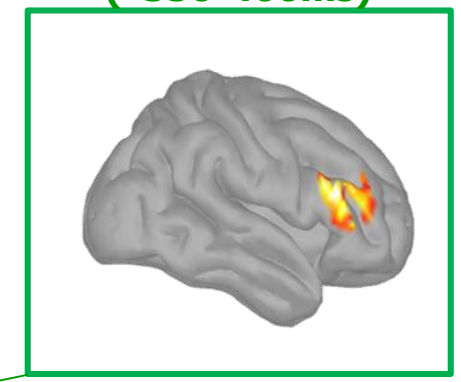
Cojan & Vuilleumier, 2011

EEG time-course of impaired motor execution under hypnotic suggestion

EEG topography
micro-state analysis
(clustering-based segmentation)



Topographical map11: "left paralysis" (~350-400ms)



Right IFG

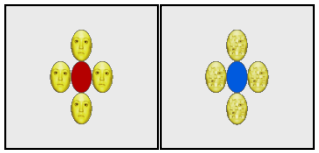
Cojan et al., Cortex 2013, 49:423-36

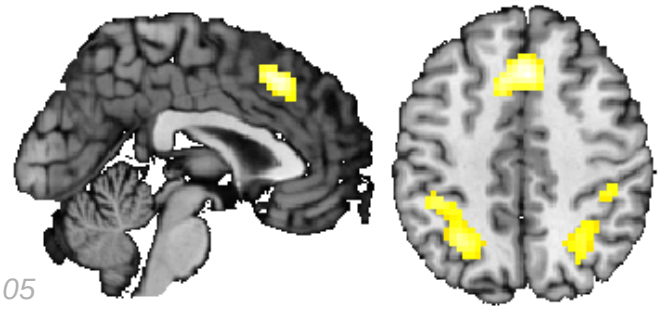
Individual susceptibility to hypnosis and selective attention

Main effect of distractors:

INCONG > CONG FLANKERS

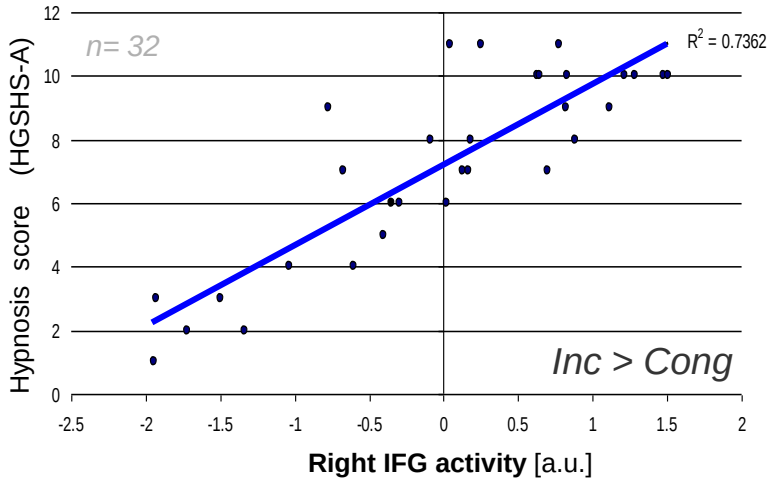
Flanker task (central face color)



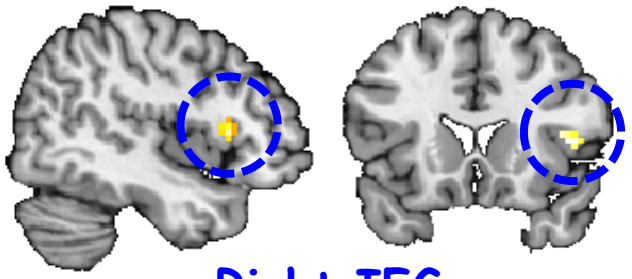


all p<.001, FDR p<.05

ACC R & L IPS



Whole brain correlation x hypnosis scores (+)



Right IFG

Cojan et al., NIMG 2015; 117:367–374