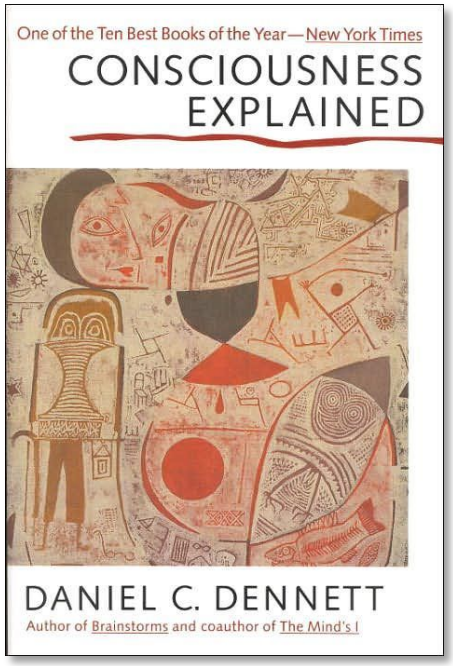
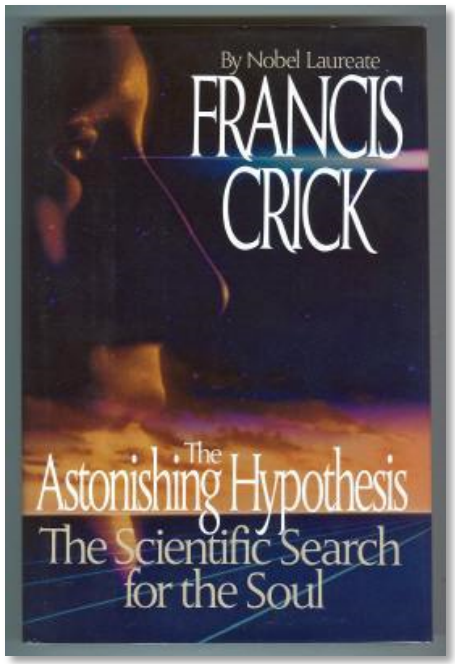
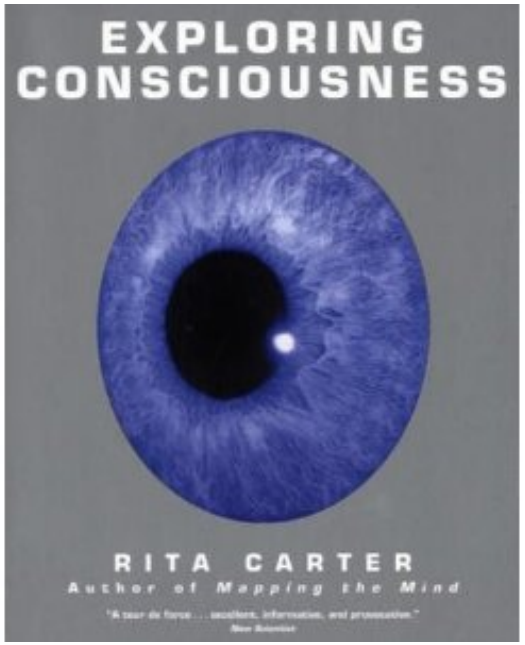
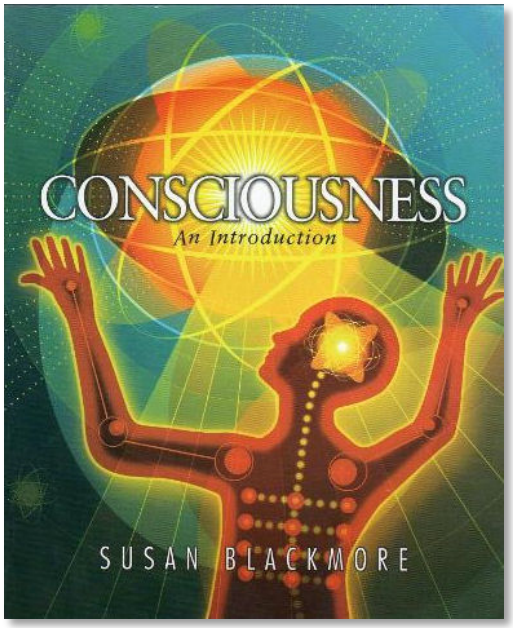
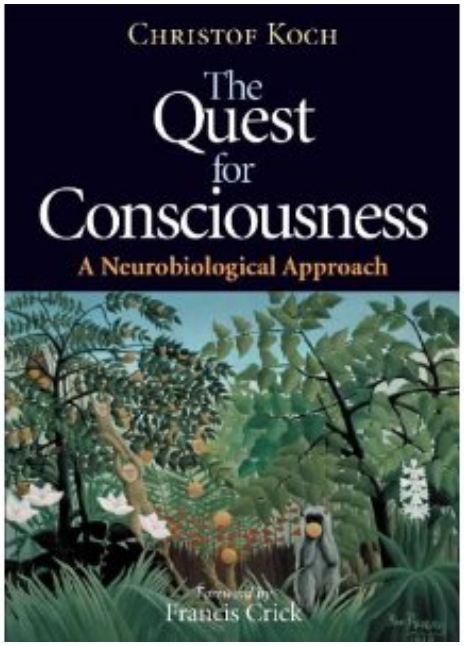
The Neural Correlate of Consciousness

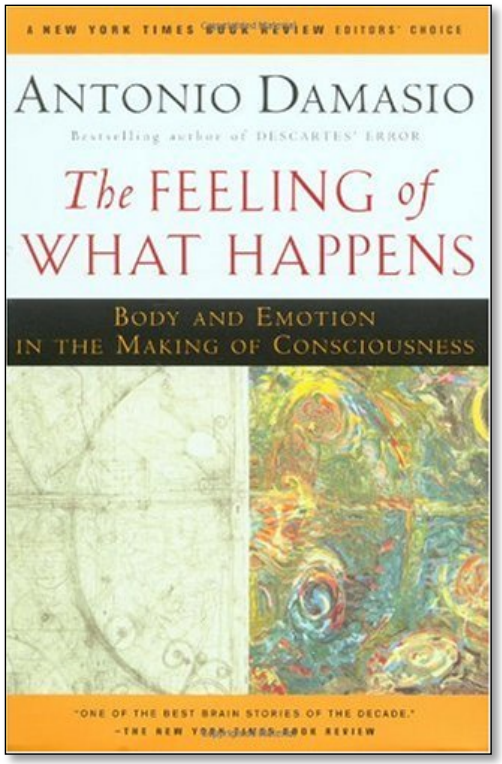




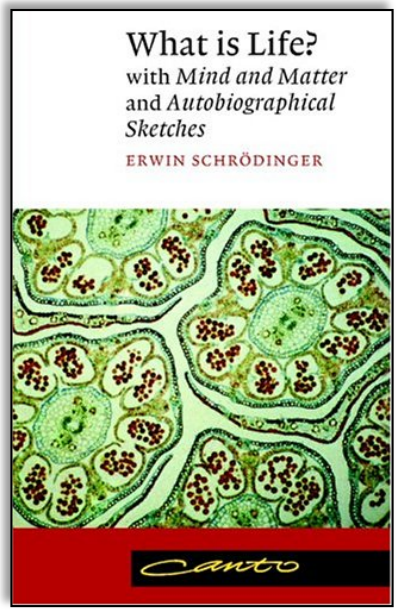








The Neural Correlate of Consciousness



What is Life? (Erwin Schrodinger, 1944)

"..living matter, while not eluding the 'laws of physics' as established up to date, is likely to involve 'other laws of physics' hitherto unknown, which however, once they have been revealed, will form just as integral a part of science as the former.

The functions of consciousness…

Summarizing all information that pertains to the current state of the organism and its environment and ensuring this compact summary is accessible to the planning areas of the brain.

Christof Koch (2007) Trends in Cognitive Sciences 11:16-22.

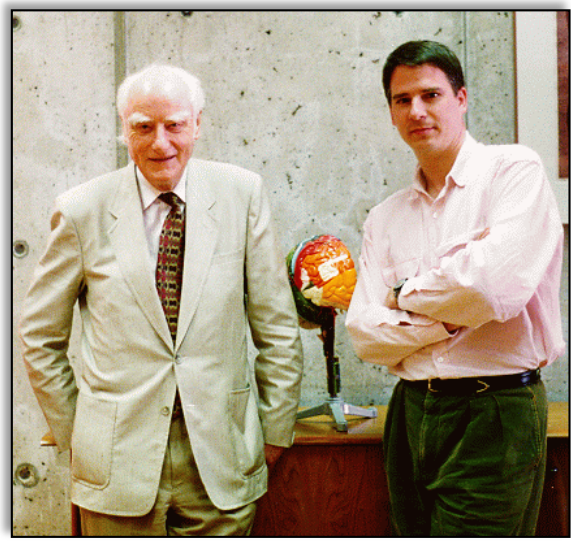
Psychophysics - what is the behavioural correlate of physical stimuli?
or, conversely - what is the physical correlate of the behavioural report?

NCC - what is the neural correlate of the behavioral report?

The neural correlate of consciousness...

The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.

Crick & Koch (1998) Cerebral Cortex. 8: 97-107

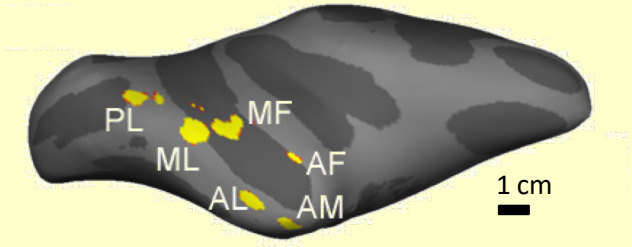


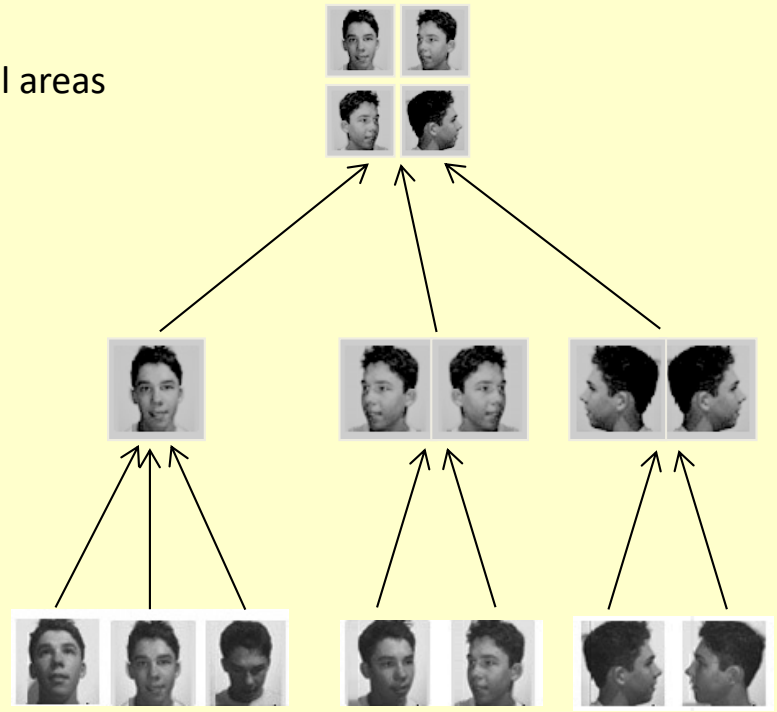
Crick & Koch

Proposition: *'perceiving' is being aware of the information encoded by each area's feature detectors.*

e.g. Face perception:
-an amalgam of information from separate cortical areas

Friewald & Tsao (2010) Hierarchical model





AM patch:
View invariant
Subject specific
latency = 124 msec

AL patch:
View symmetrical
latency = 104 msec

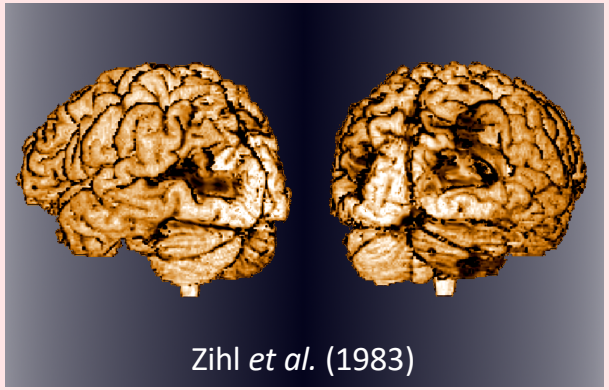
ML/MF patches:
View selective
Subject inspecific
latency = 88 msec

(NB. Invariance to translation & invariance to size)

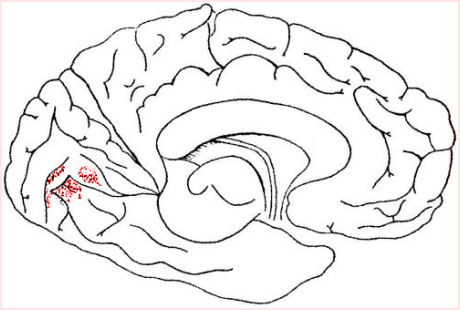
e.g. Akinetopsia, achromatopsia:

Area V5 direction-selective cells encode motion;

Area V4 hue-selective cells encode colour .

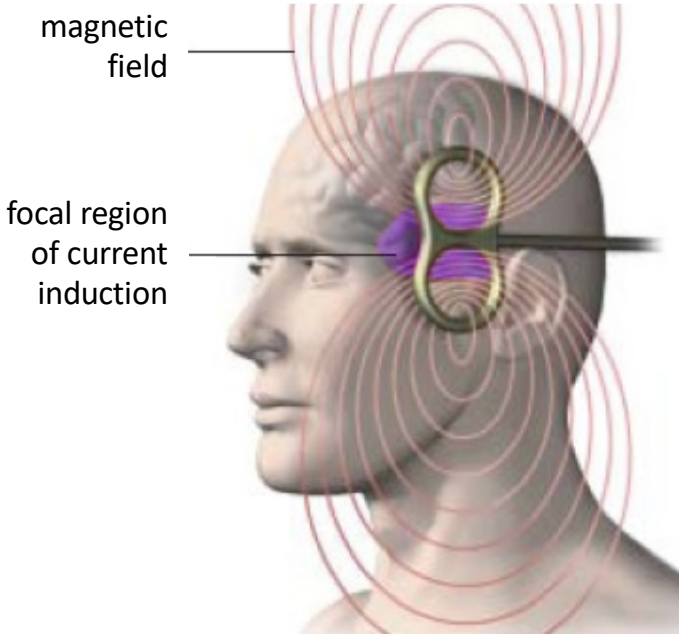


Zihl *et al.* (1983)

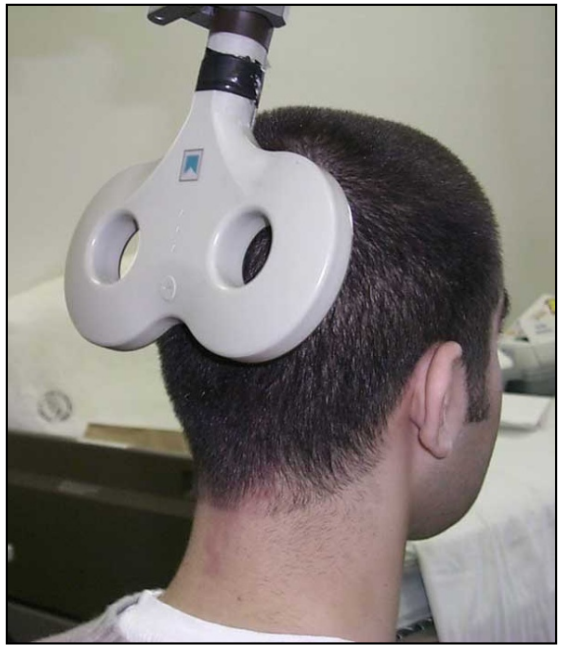


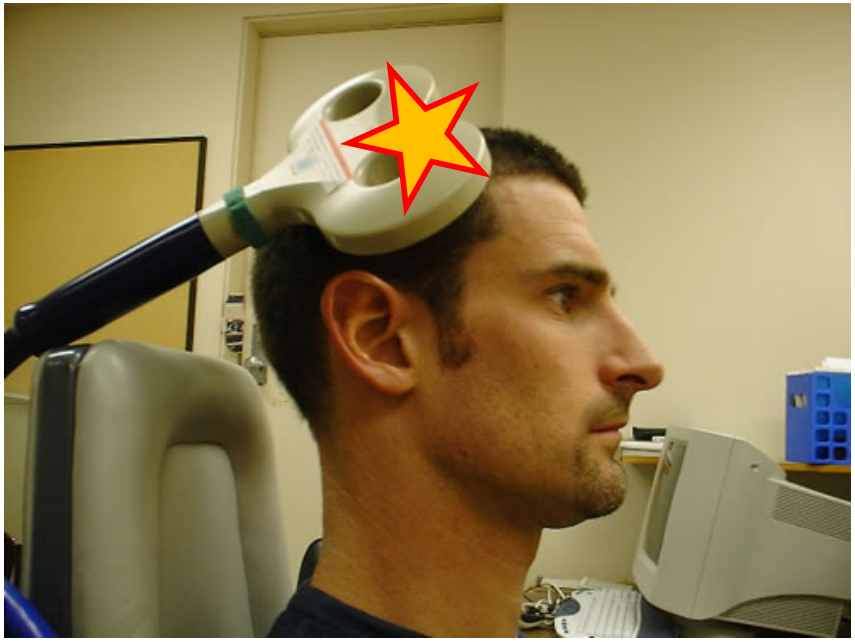
Louis Verrey (1888)

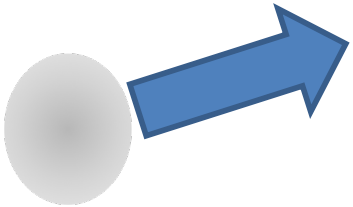
Transcranial Magnetic Stimulation (TMS)



TMS over visual cortex produces a 'phosphene'
(Grk. 'light-show')





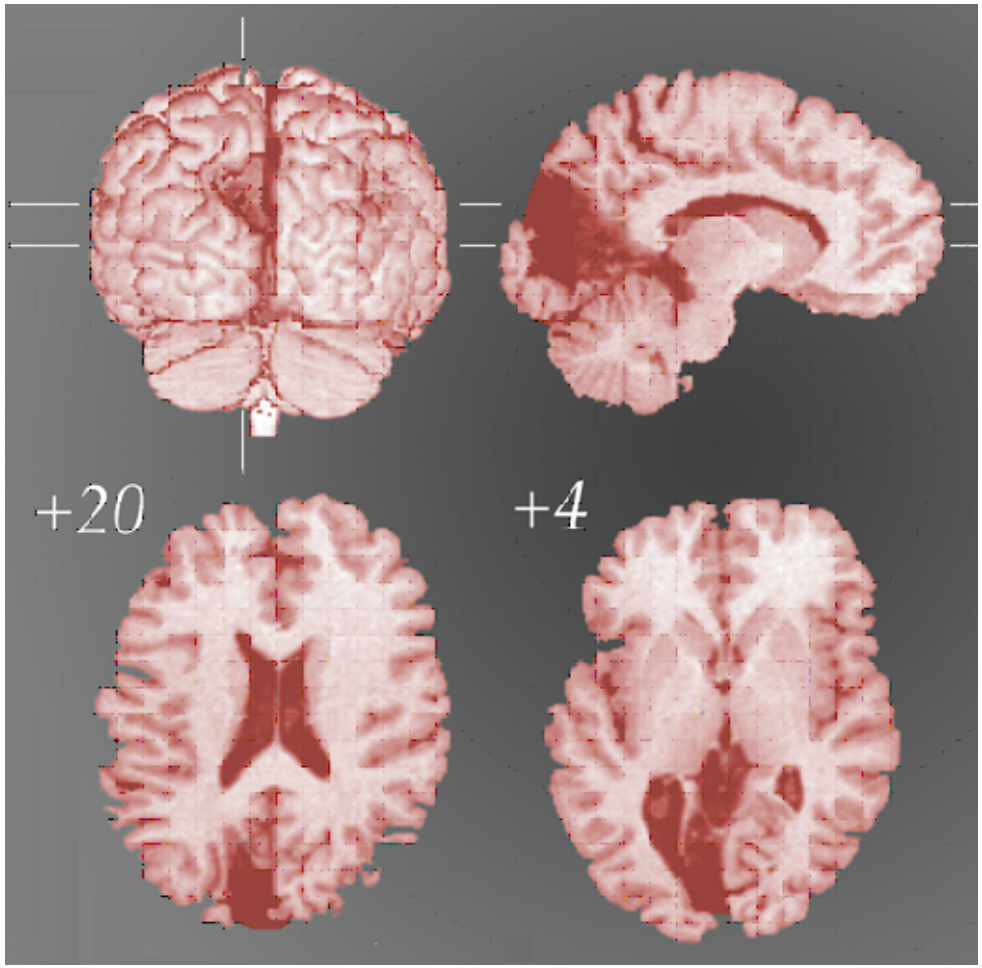


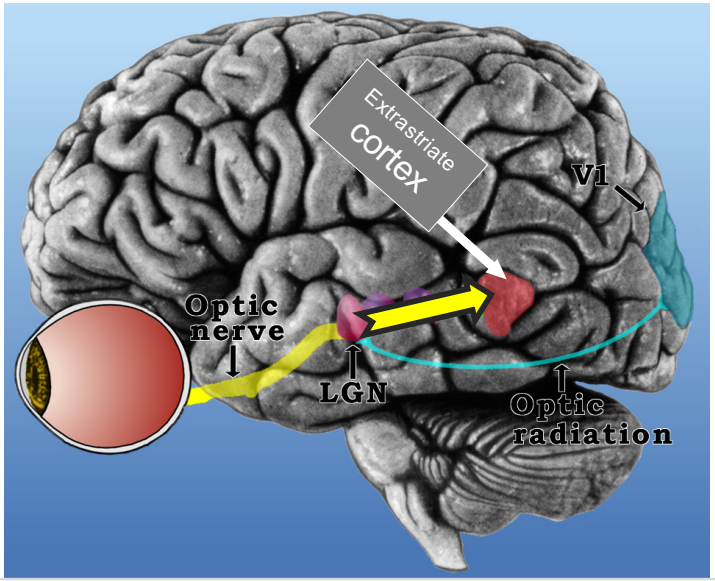
TMS over area V5 produces a moving phosphene

Proposition: ‘perceiving’ is being aware of the information encoded by each area’s feature detectors.

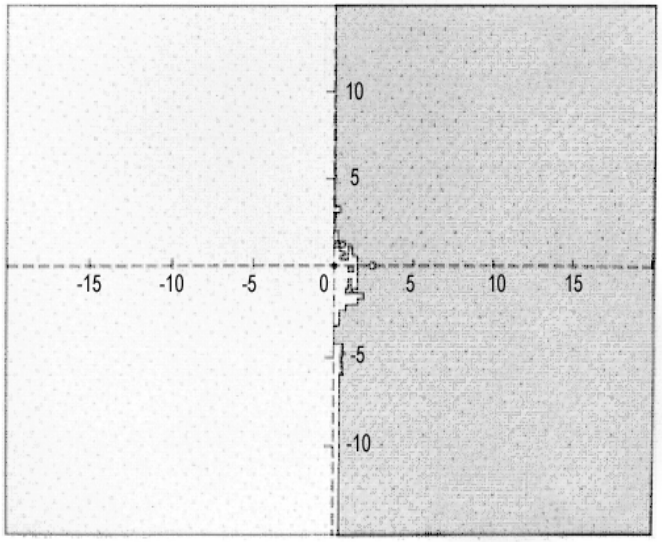
BUT – can information be encoded, yet not reach awareness ?

1. e.g. ‘blindsight’ (subject GY)





Schmid *et al.* (2010) [ref 1]
Blindsight depends on the lateral geniculate nucleus



Hemifield with normal sight

Hemifield with ‘blindsight’

Proposition: ‘perceiving’ is being aware of the information encoded by each area’s feature detectors.

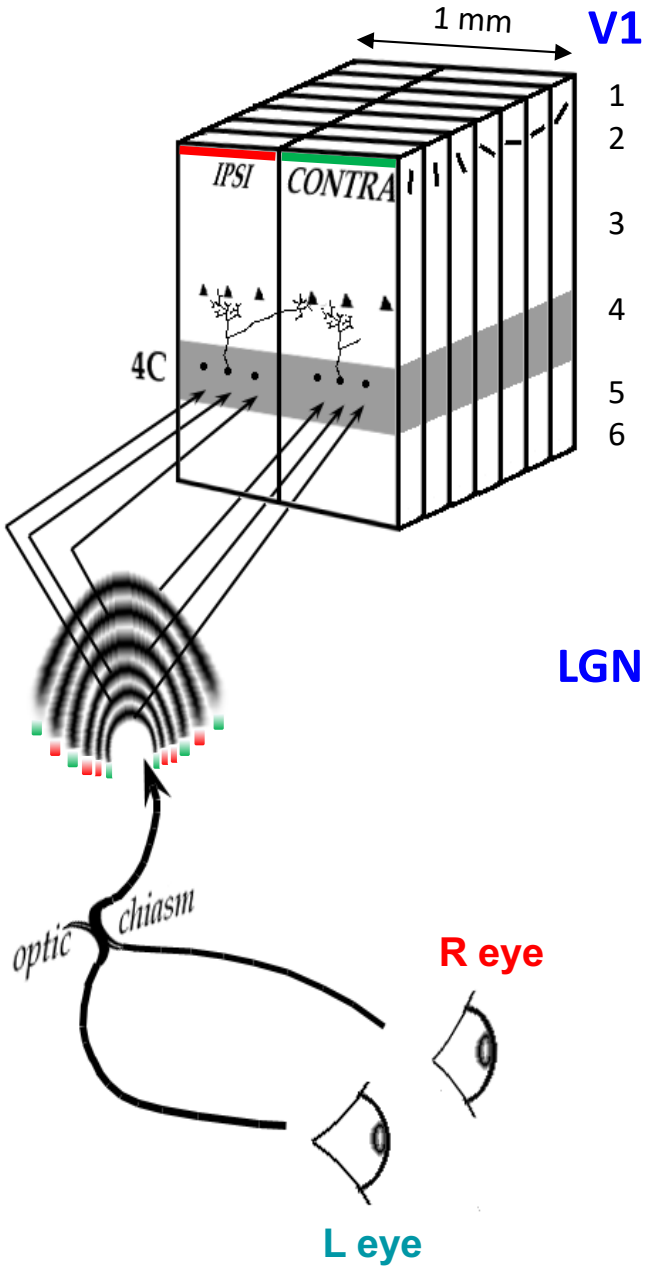
BUT – can information be encoded, yet not reach awareness ?

1. e.g. ‘blindsight’ (subject GY).

2(a) e.g. monocular neurons in layer 4C of V1;

2(b) e.g. V1 neurons whose spatial frequency sensitivity exceeding perceptual acuity; [ref 2]

3. ‘**Psychophysical magic: rendering the visible invisible**’ (Kim & Blake, 2005).
e.g. ‘visual masking’
e.g. ‘motion-induced blindness’ [ref 3]
e.g. ‘change blindness’
e.g. ‘inattentional blindness’



Change Blindness: Demonstration

Casual viewing provides the ‘gist’ of a scene;
- attention is required to appreciate details.

The neural correlate of consciousness...

The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.

Crick & Koch (1998) Cerebral Cortex. 8: 97-107

The previous examples demonstrate that some forms of neural activity may occur, but not lead to awareness. They may be *necessary* for visual awareness, but are not *sufficient* for visual awareness.

'Anatomical' interpretation of NCC

- Which parts of the brain..?
- Which circuits within/between which areas?

'Physiological' interpretation of NCC

- What pattern of activity..?
- What timing of activity..?

Anatomical Conception:

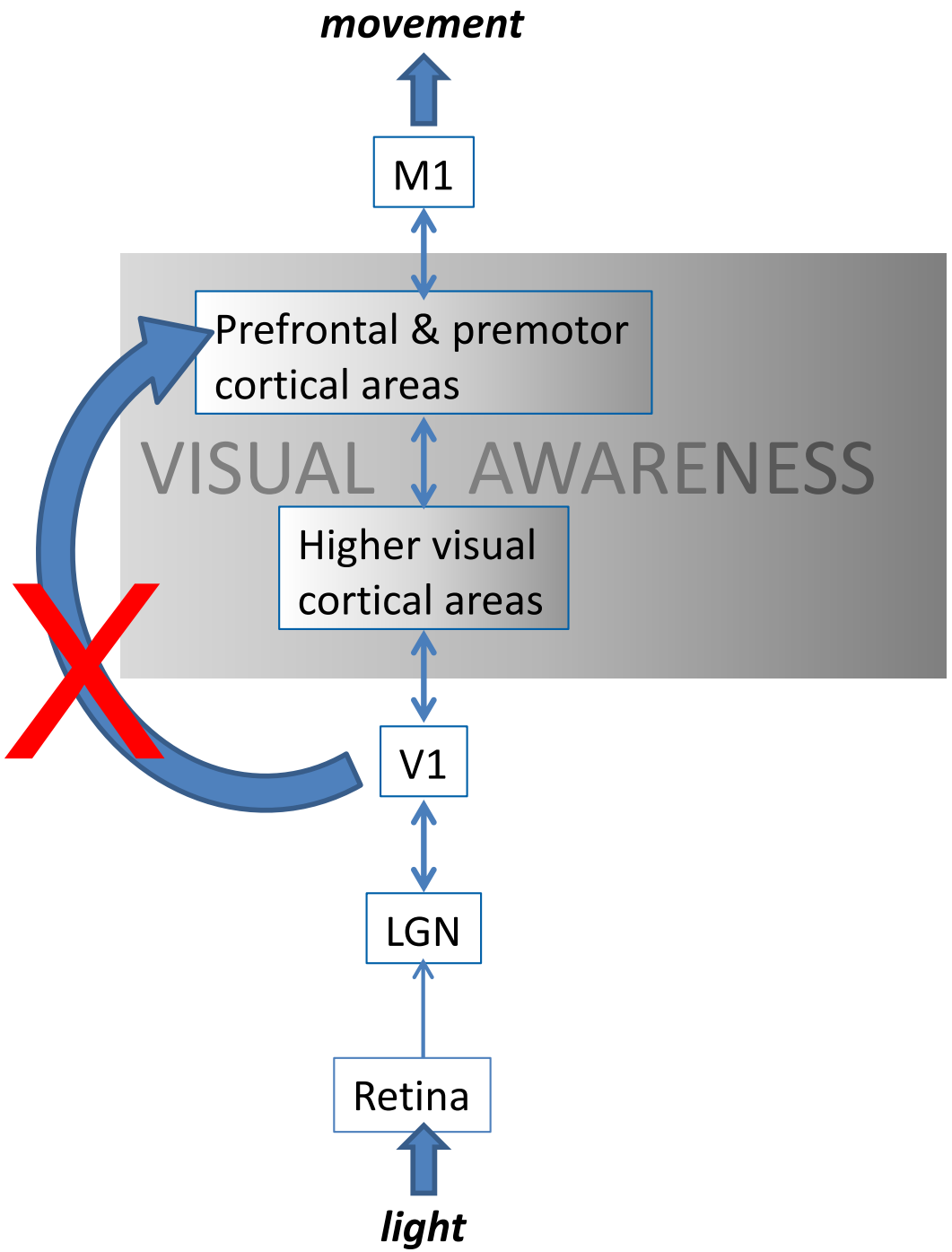
- is the *retina* a component of the NCC ?

Claim	Evidence	Verdict
YES	Loss of awareness in retinal blindness	INVALID (ignores de-afferentation of higher areas)
NO	TMS stimulation over V1 produces phosphenes in the retinally blind	VALID
NO	Hallucinatory visual perception within the blind field of retinally blind subjects (Charles Bonnet syndrome, 1760)	VALID

Crick & Koch (1995) [ref 5] Are we aware of neural activity in primary visual cortex ?

FUNCTION OF CONSCIOUSNESS:

Summarizing all information that pertains to the current state of the organism and its environment and ensuring this compact summary is accessible to the planning areas of the brain.

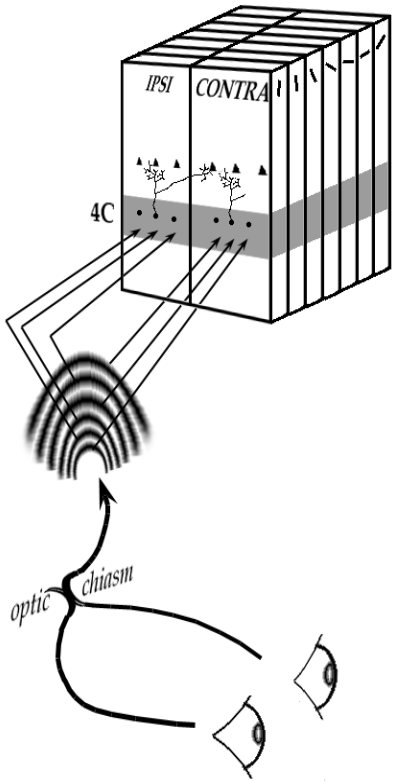


These connections do not exist

"All we are hypothesizing is that the activity in V1 does not directly enter awareness."

Is area **V1** a component of the NCC ?

Claim	Evidence	Verdict
NO	No direct communication between V1 and 'decisional /command' centres of cortex	?
NO	Monocular cells in V1 do not register a conscious percept	INCONCLUSIVE (may not generalise to all V1 neurons)



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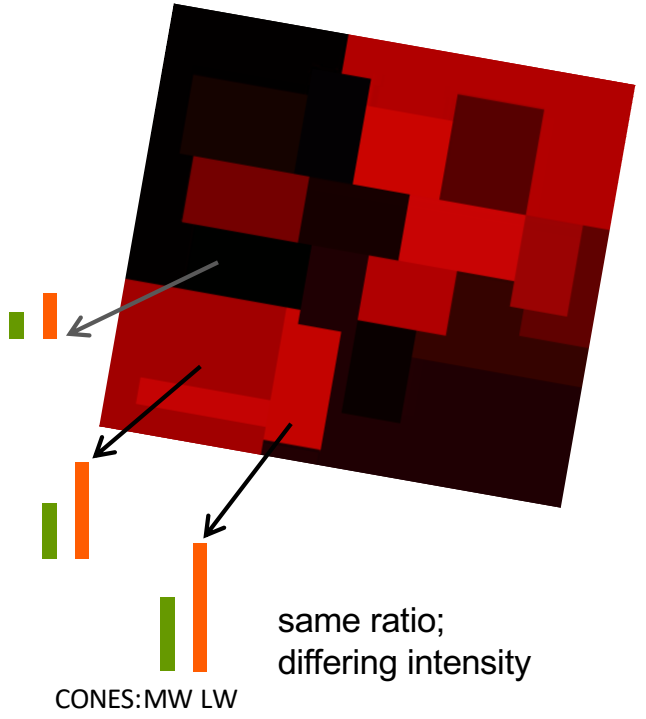

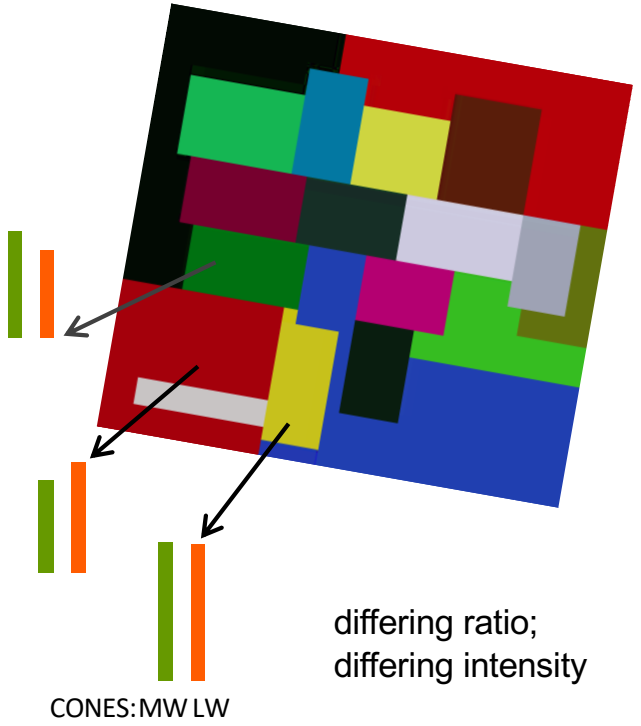

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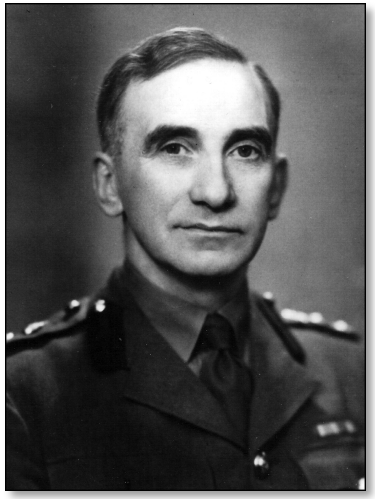
NO	Colour cells in V1 do not exhibit the Land Mondrian (colour constancy) effect , unlike cells in V4	INCONCLUSIVE (V1 cells may be necessary, but not sufficient for colour percept)
NO	Awareness of motion with V1 lesion: 'type 2' 'blindsight' or 'Riddoch Syndrome' [ref 6]	VALID... but trivial: *infer V1 is not NCC for 'residual' motion perception.*



Riddoch, 1917: Dissociation of visual perceptions due to occipital injuries, with especial reference to appreciation of movement.

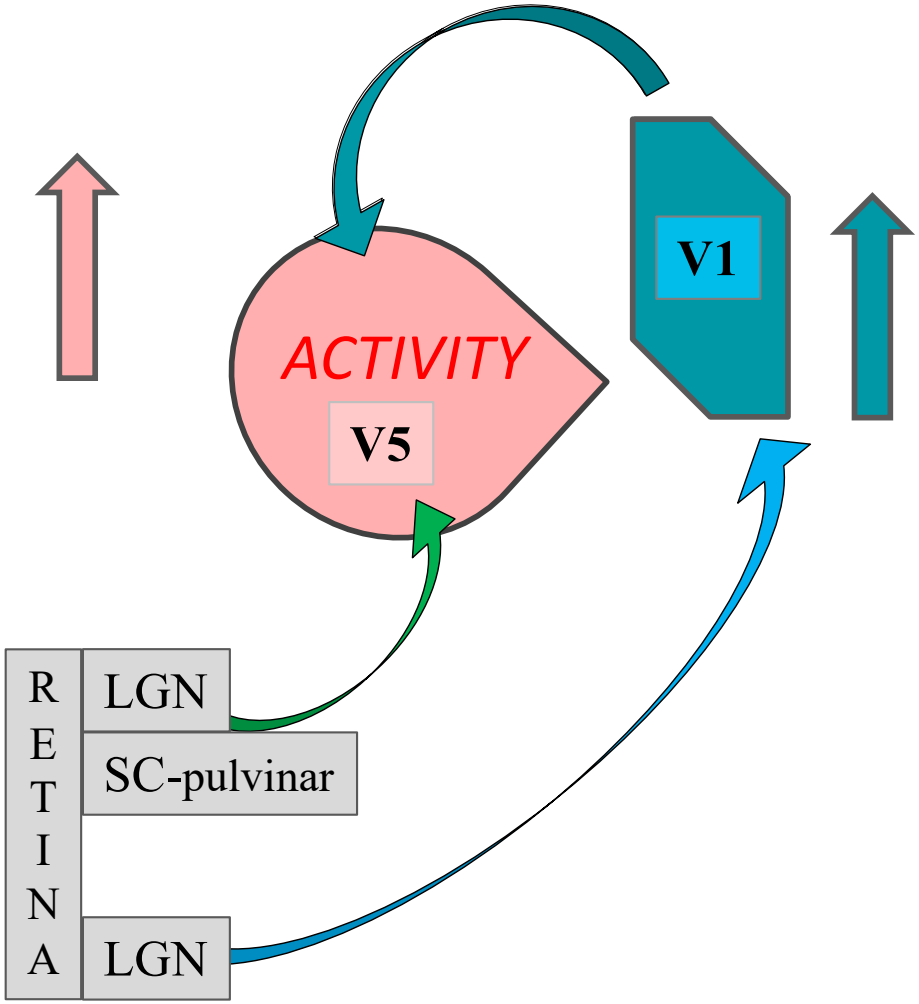
"A shadowy, foggy sensation of motion to which blind subjects could attribute neither colour nor form"

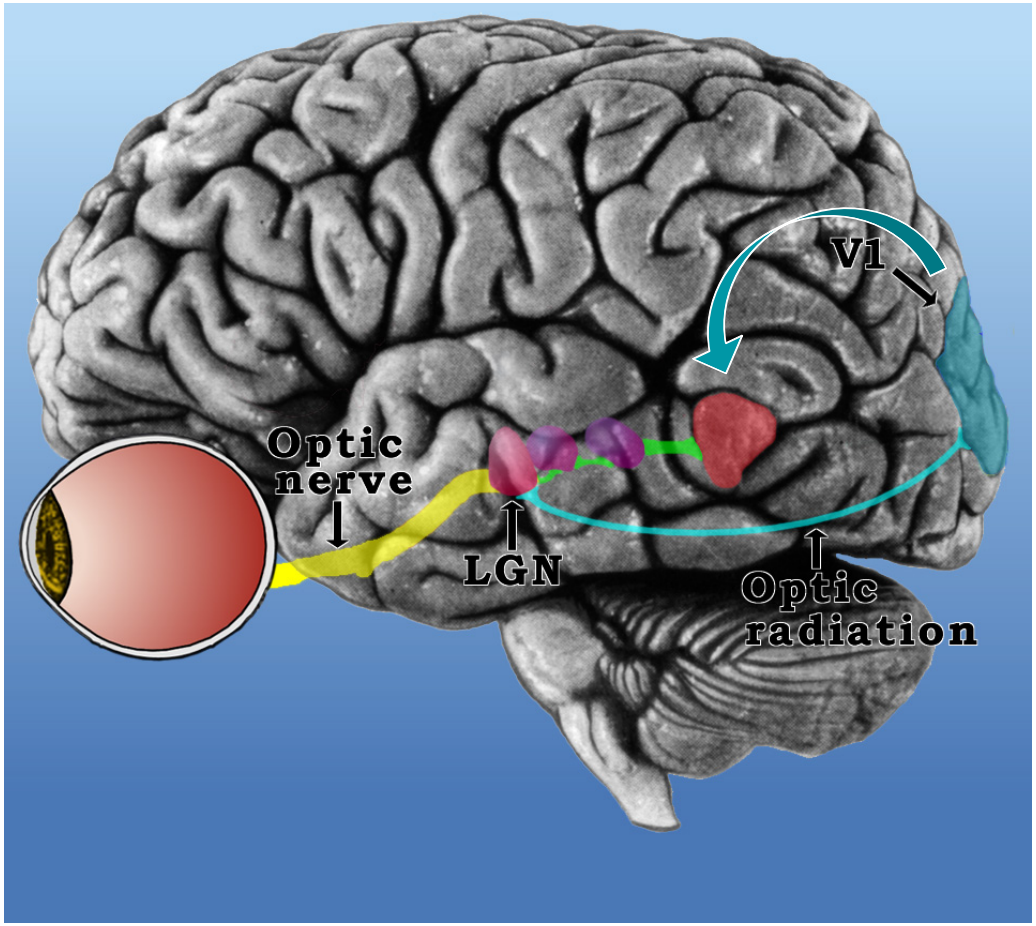
NCC = *The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.*

Model of residual motion vision with NCC excluding V1

'NO' CAMP

NCC



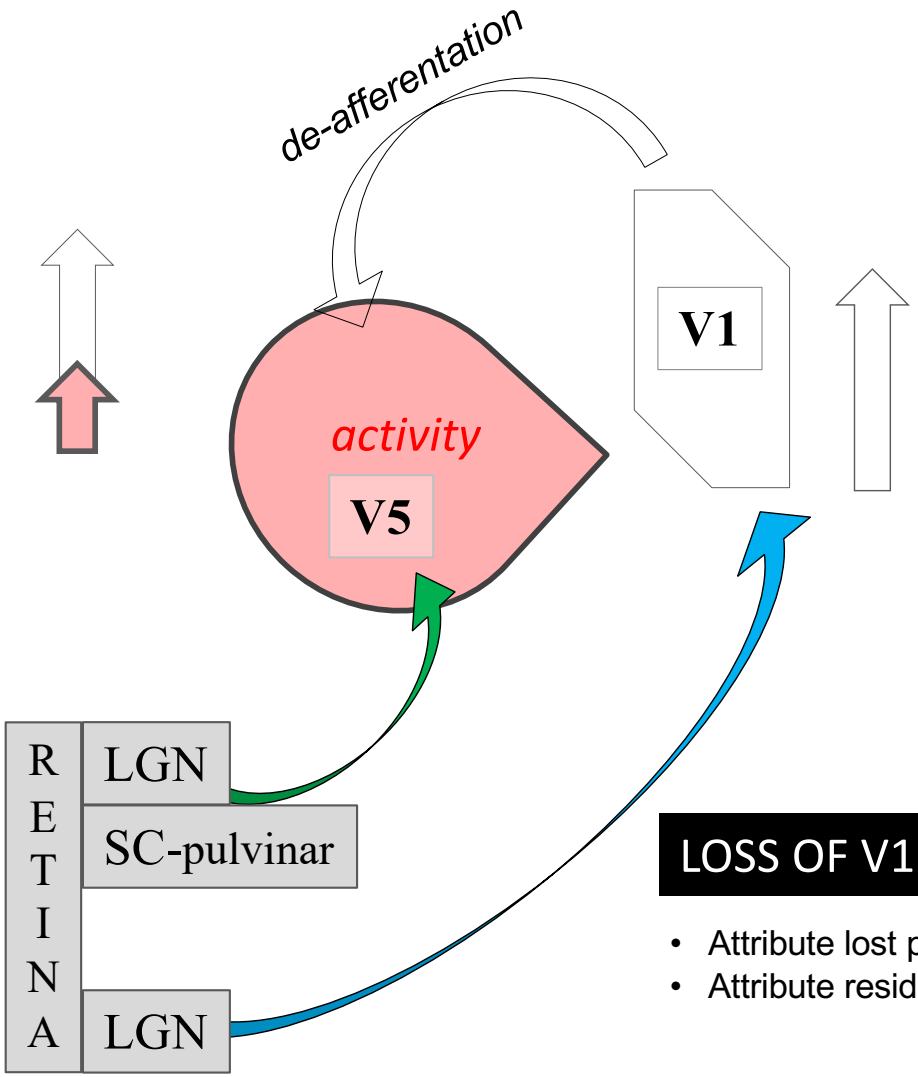


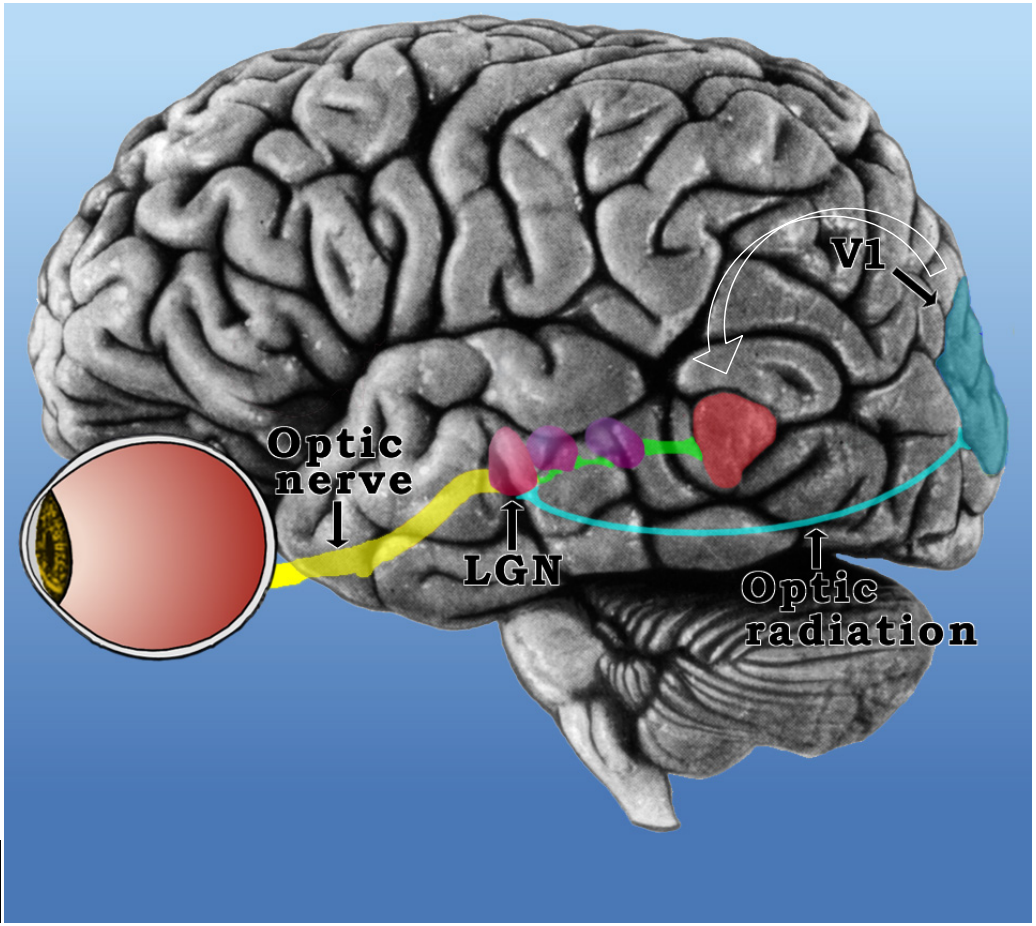
INTACT BRAIN – NORMAL FUNCTIONING

Model of residual motion vision with NCC excluding V1

'NO' CAMP

NCC





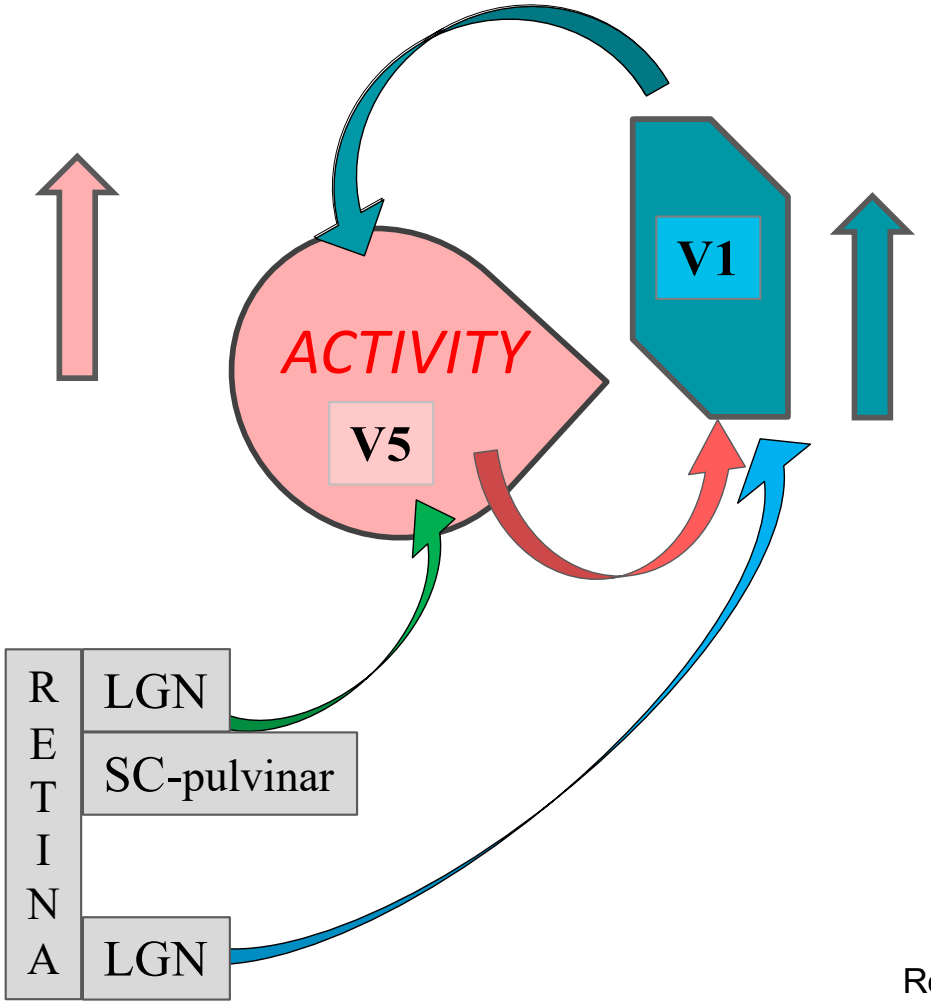
LOSS OF V1

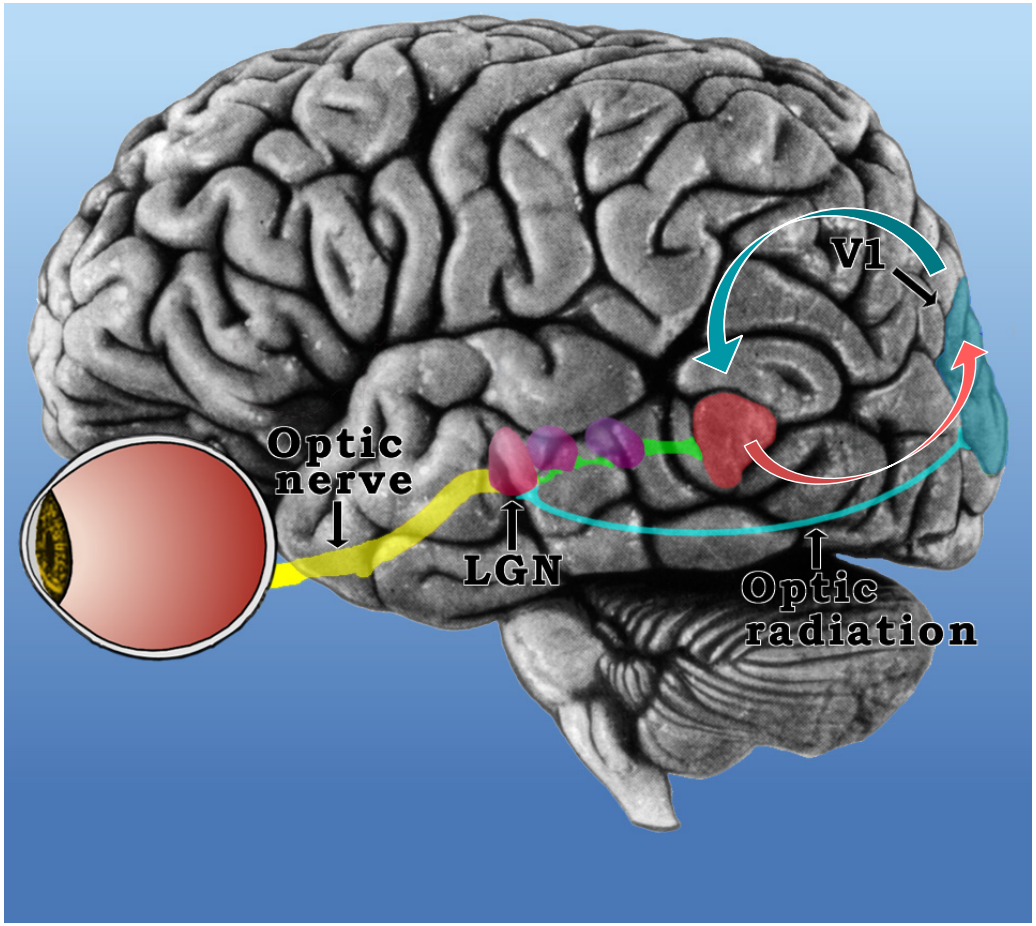
- Attribute lost perceptual capability to de-afferentation of V5;
- Attribute residual perceptual capability to remaining, reduced V5 activation.

Model of residual motion vision with NCC including V1

'YES' CAMP

NCC





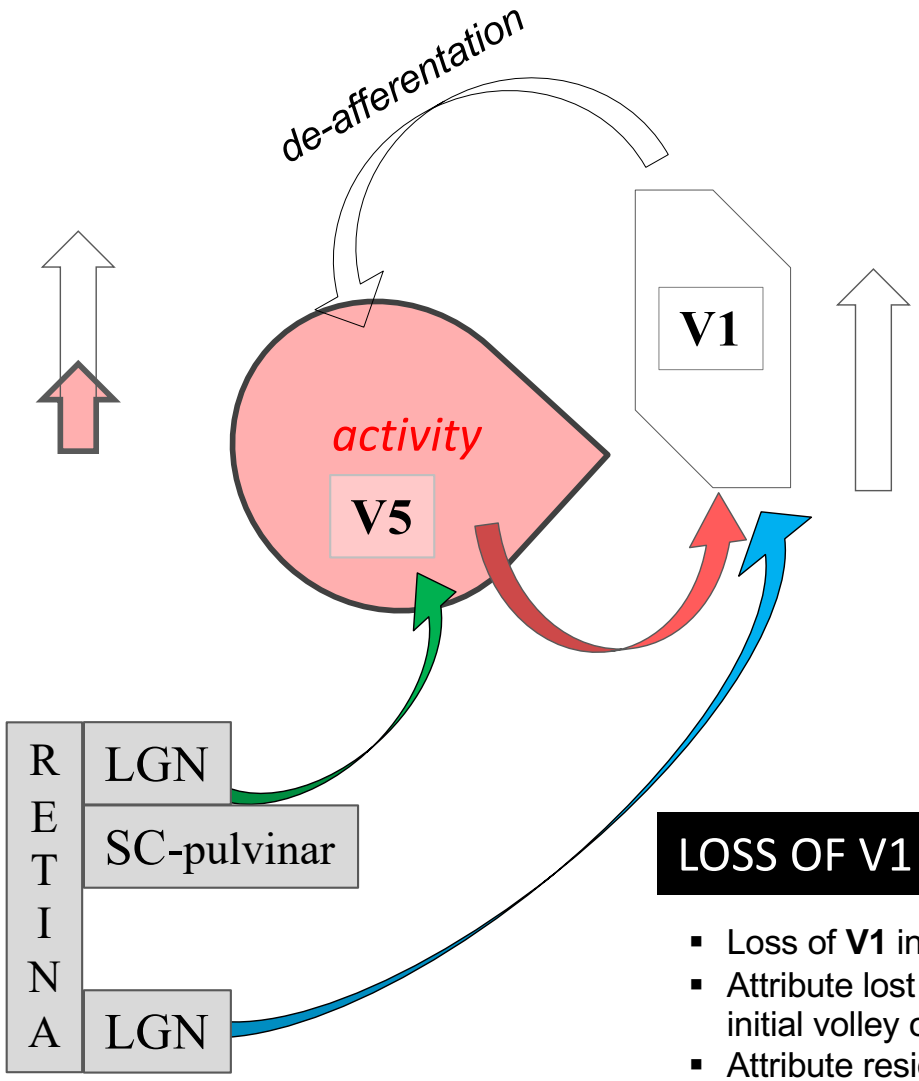
INTACT BRAIN – NORMAL FUNCTIONING

Reciprocal connection from **V5** to **V1** creates a positive feedback loop:

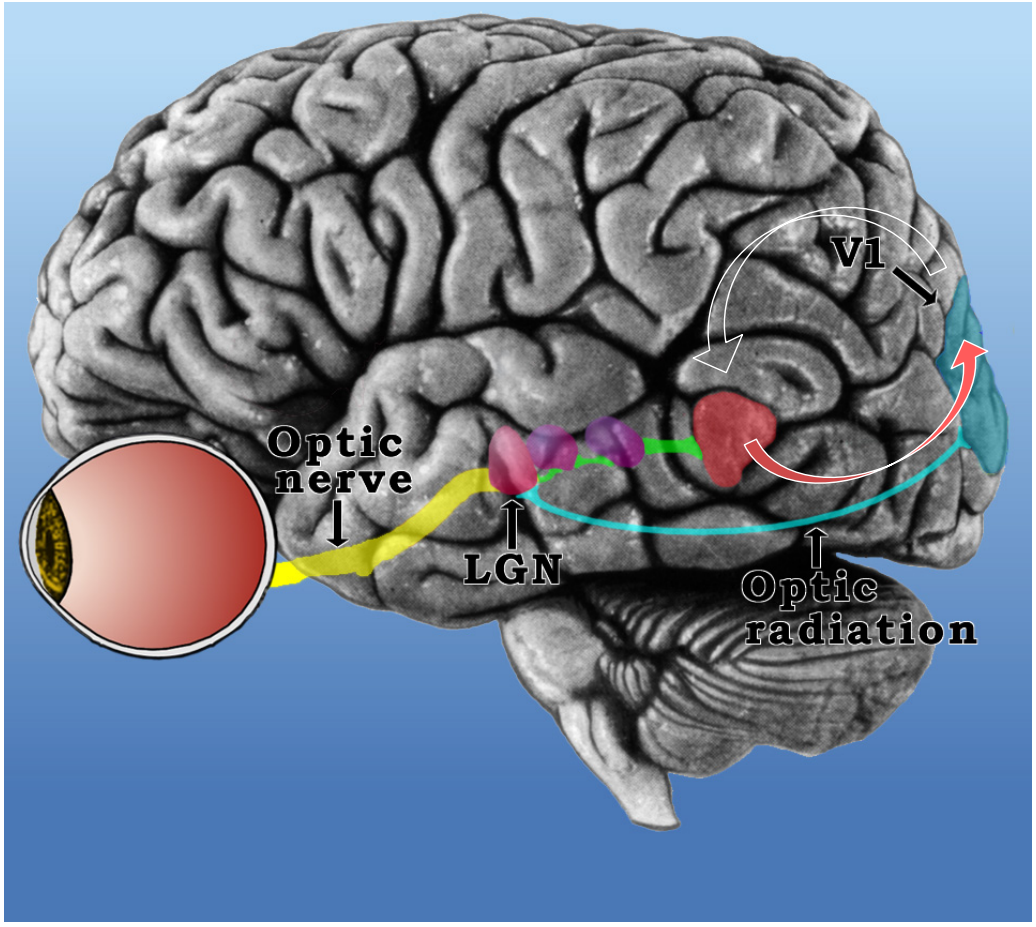
Model of residual motion vision with NCC including V1

'YES' CAMP

NCC



LOSS OF V1



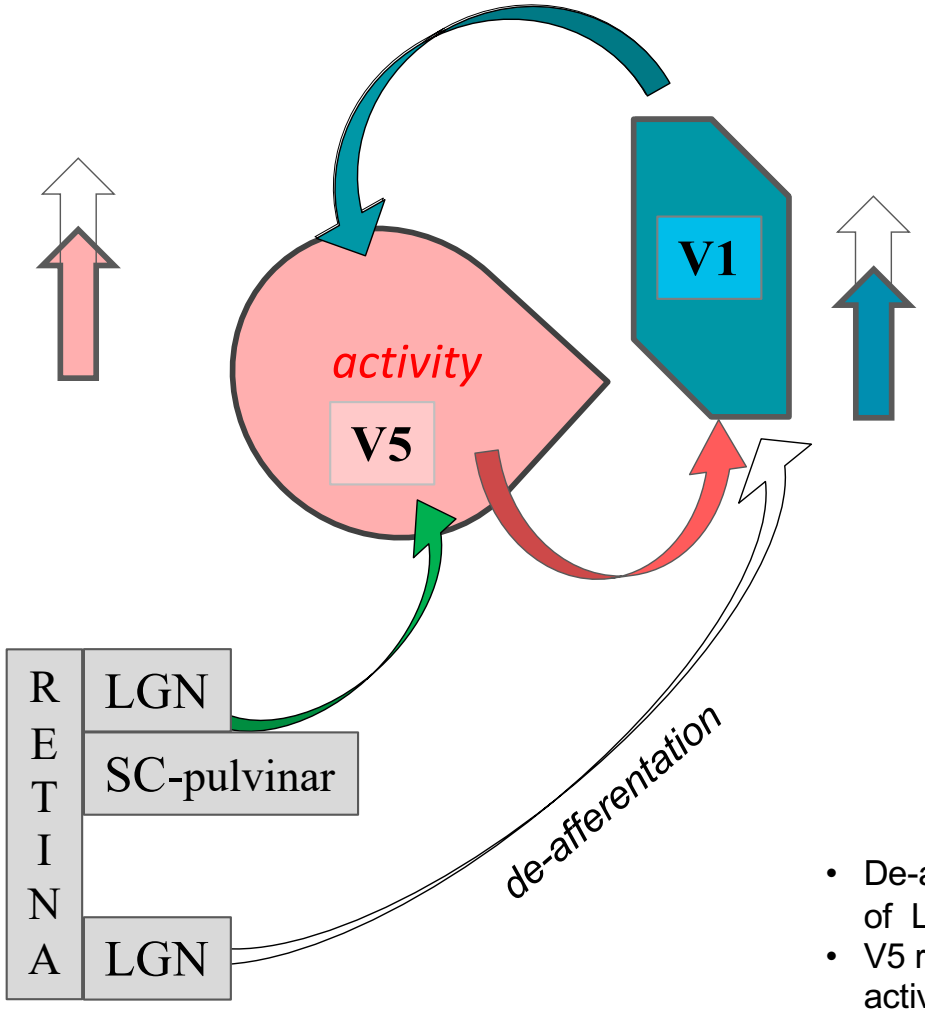
- Loss of **V1** inactivates the feedback loop.
- Attribute lost perceptual capability to de-afferentation of V5, removing the initial volley of activity and the sustained 'loop' activity;
- Attribute residual perceptual capability to remaining, reduced V5 activation.

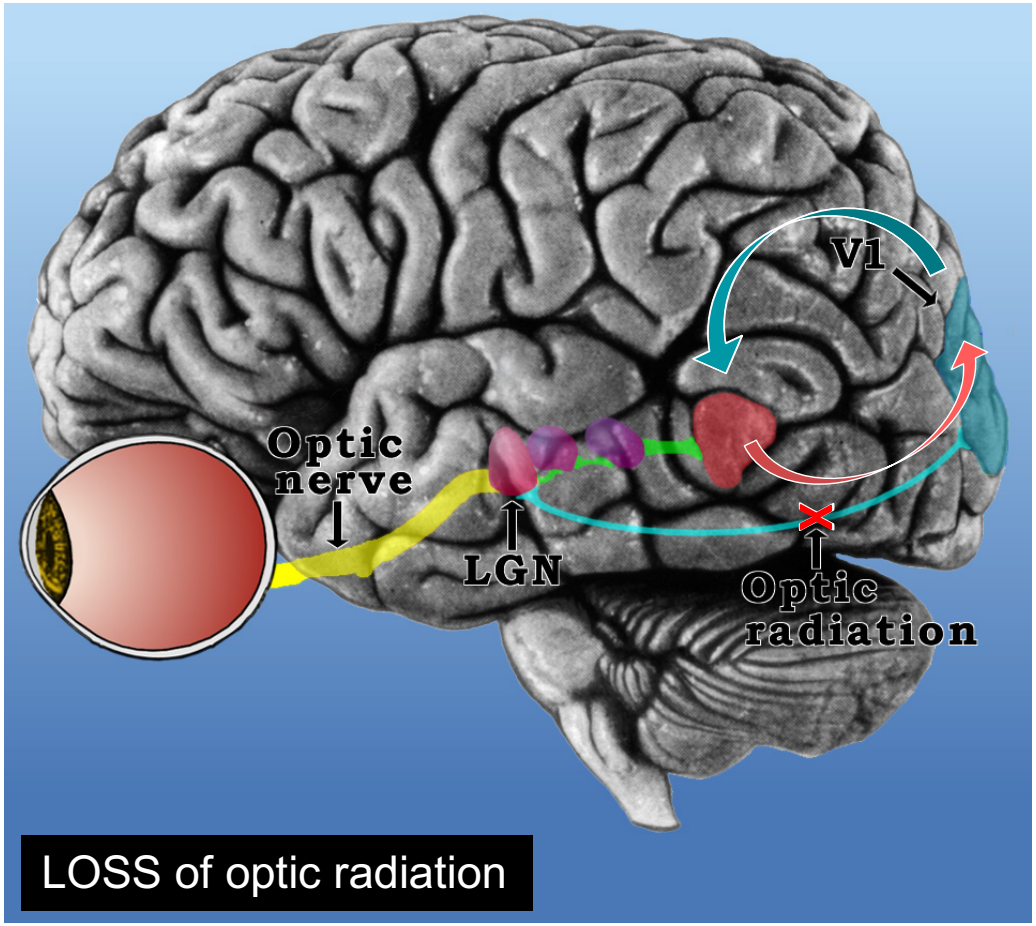
Model of residual motion vision with NCC including V1

'YES' CAMP

'THOUGHT EXPERIMENT'

NCC





- De-afferentation of V1 and V5 is here attained by selective destruction of LGN input to V1;
- V5 remains activated by direct input from LGN or SC/pulvinar; this activity may be sustained by the feedback loop with V1;
- Attribute residual perceptual capability to remaining, but less reduced activation of V5 ***and*** V1 => *BETTER RESIDUAL VISION ?*

Is area **V1** a component of the NCC ?

Claim	Evidence	Verdict
NO	No direct communication between V1 and 'decisional /command' centres of cortex	?
NO	Monocular cells in V1 do not register a conscious percept	INCONCLUSIVE (may not generalise to all V1 neurons)
NO	Colour cells in V1 do not exhibit the Land Mondrian (colour constancy) effect , unlike cells in V4	INCONCLUSIVE (V1 cells may be necessary, but not sufficient for colour percept)
NO	Awareness of motion with V1 lesion/ 'blindsight' ('Riddoch Syndrome') [ref 6]	VALID... but trivial: *infer V1 is not NCC for 'residual' motion perception.*
NO	99% loss of normal motion vision with V1 lesion is ascribed to deafferentation of higher visual areas	INVALID (ignores V1 participation in recurrent circuitry)

NCC = *The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.*

Is area V1 a component of the NCC ?

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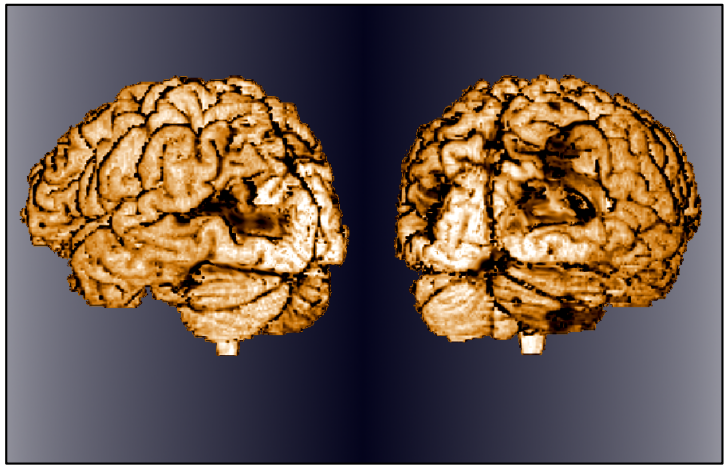
NO	Colour cells in V1 do not exhibit the Land Mondrian (colour constancy) effect , unlike cells in V4	INCONCLUSIVE (V1 cells may be necessary, but not sufficient for colour percept)
NO	Awareness of motion in blindsight ('Riddoch phenomenon')	VALID… but trivial: *infer V1 is not NCC for 'residual' motion perception.*
NO	99% loss of normal motion vision with V1 lesion is ascribed to deafferentation of higher visual areas	INVALID (Ignores V1 participation in recurrent circuitry)
NO	Akinetopsia in case LM (bilateral lesion of V5), with V1 intact (i.e. including direction-selective neurons in layer 4B of V1 that provide direct output to V5).	*… infer V1 is not NCC for motion…?* *No: perhaps V1 is necessary, but not sufficient?*

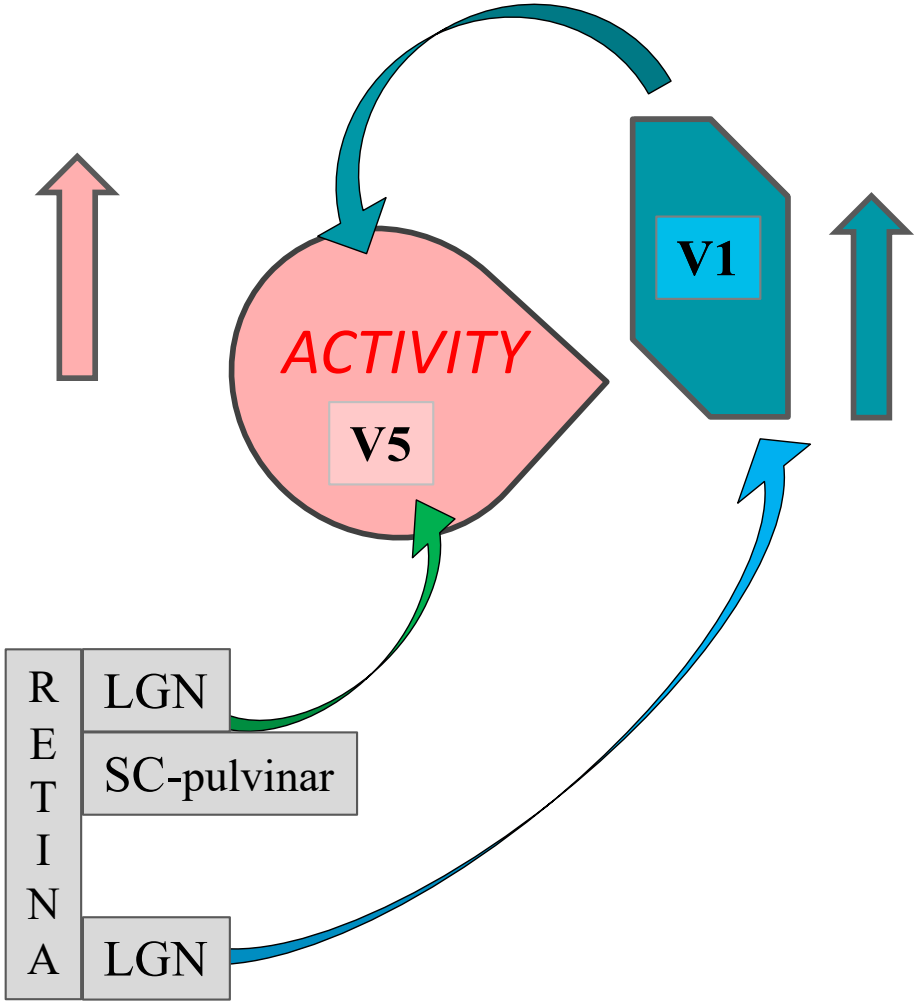


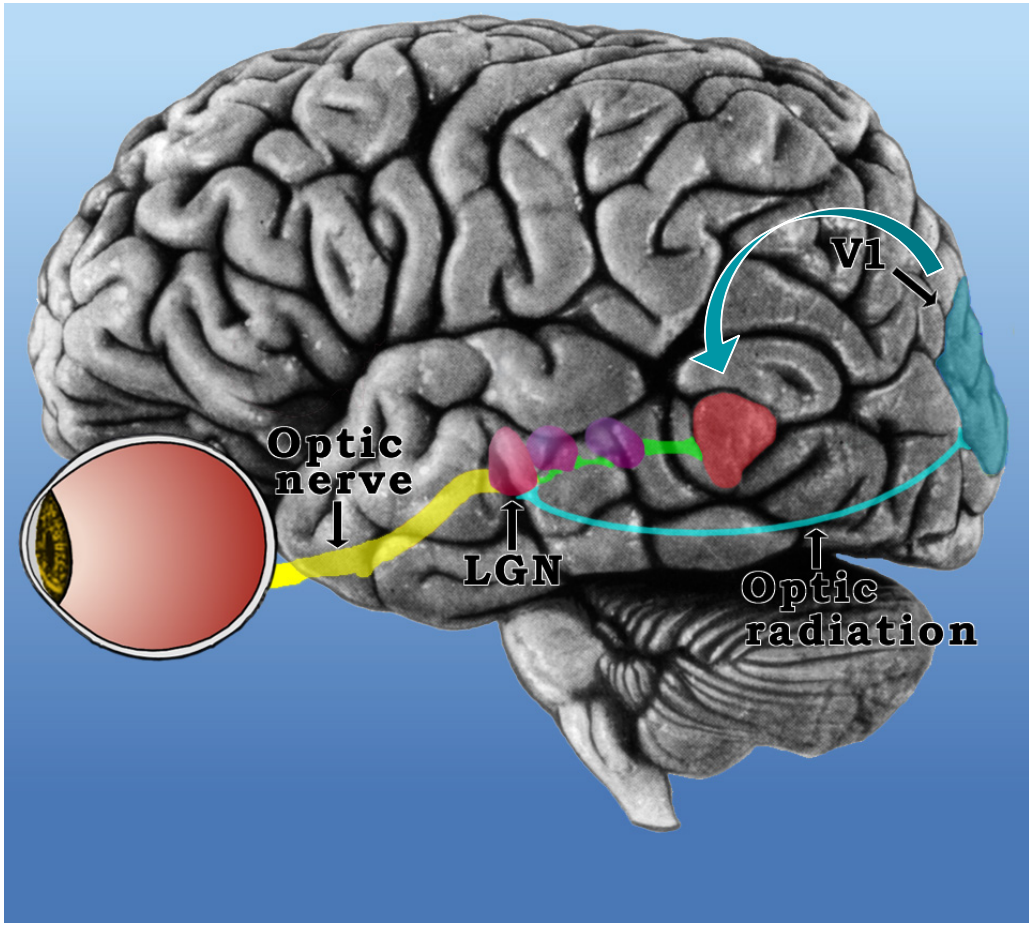
NCC = *The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.*

Model of akinetopsia with NCC excluding V1

'NO' CAMP

NCC



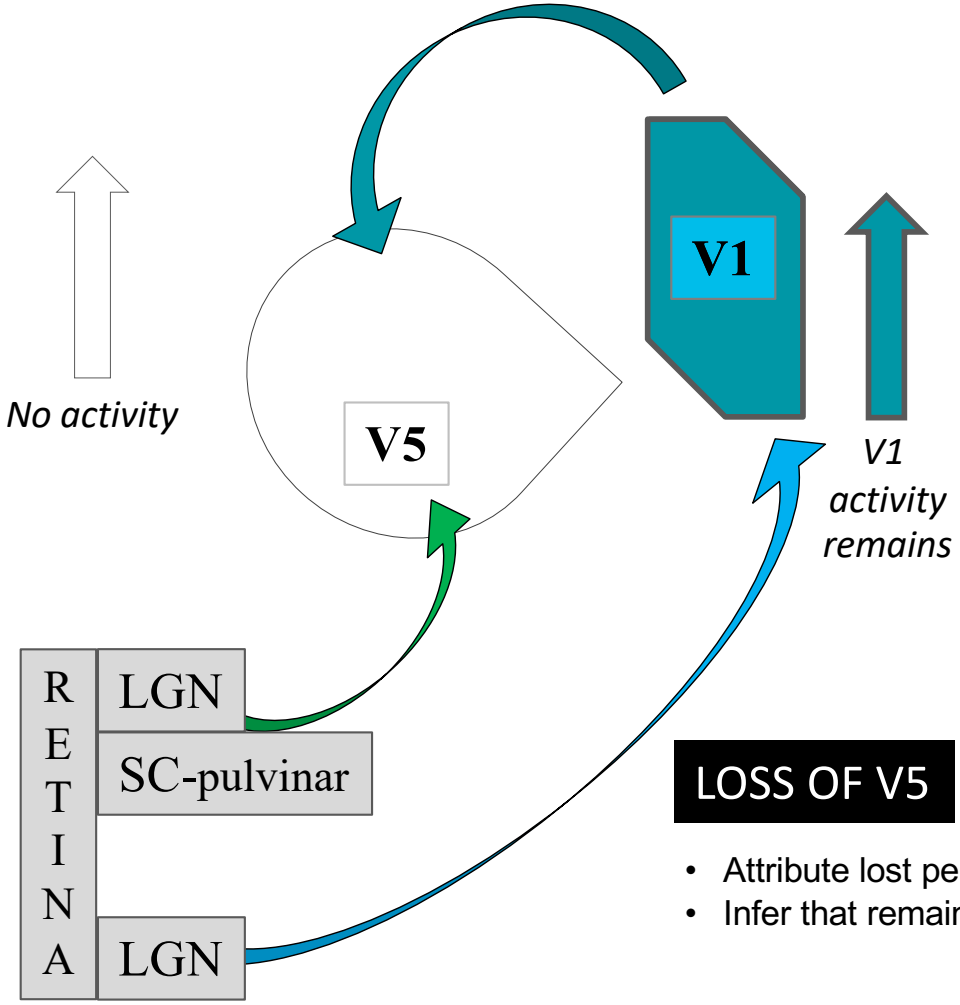


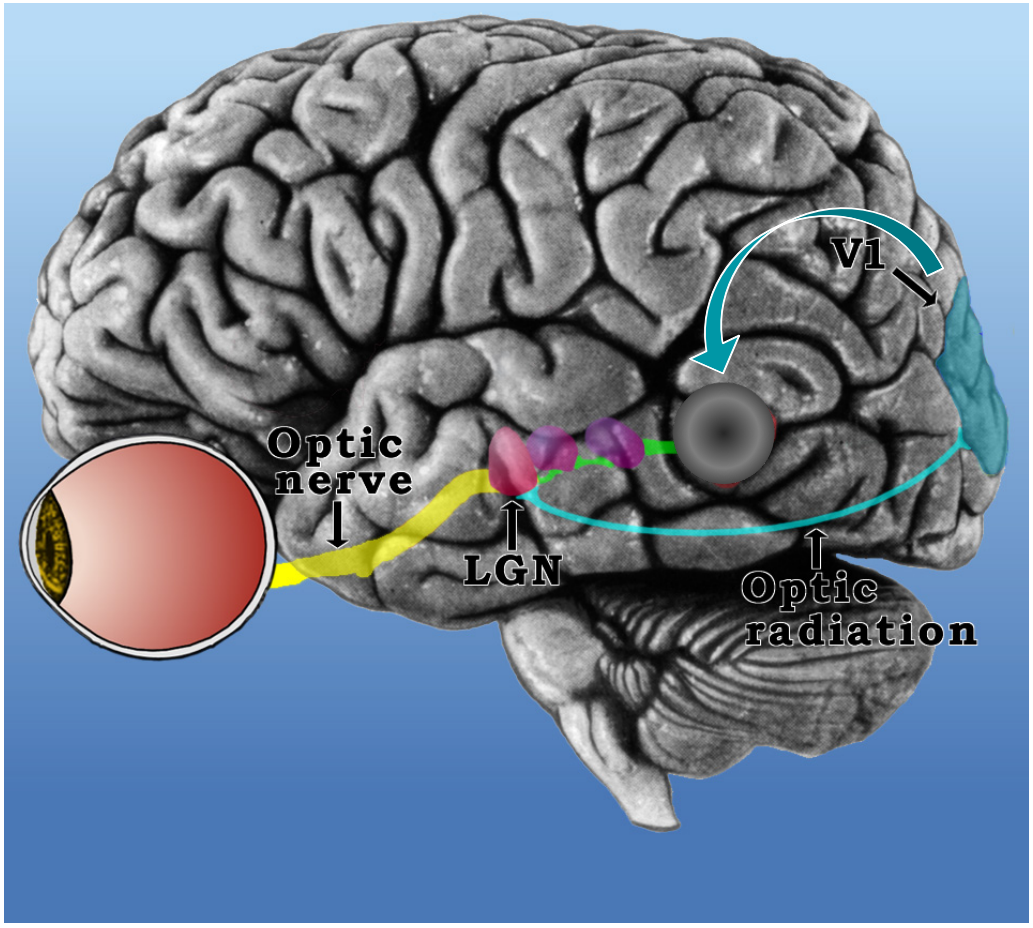
INTACT BRAIN – NORMAL FUNCTIONING

Model of akinetopsia with NCC excluding V1

'NO' CAMP

NCC





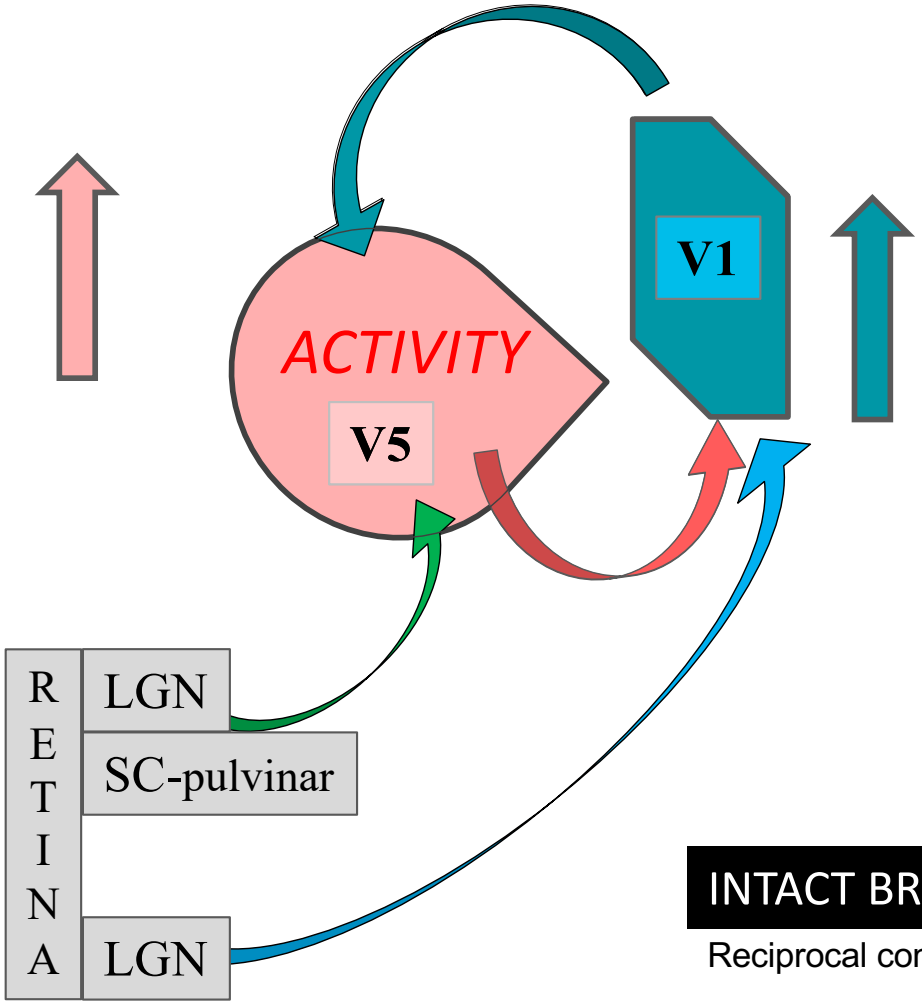
LOSS OF V5

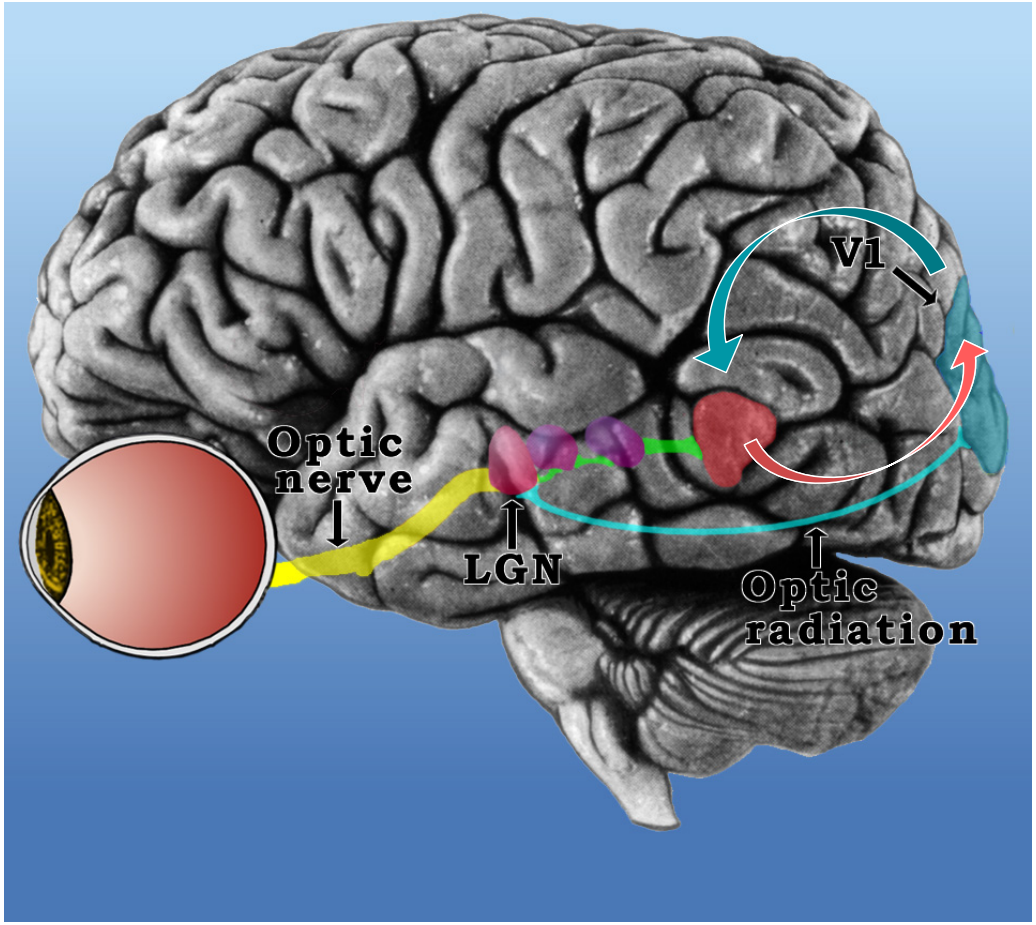
- Attribute lost perceptual capability to destruction of V5;
- Infer that remaining activation of V1 is not part of NCC.

Model of akinetopsia with NCC including V1

'YES' CAMP

NCC





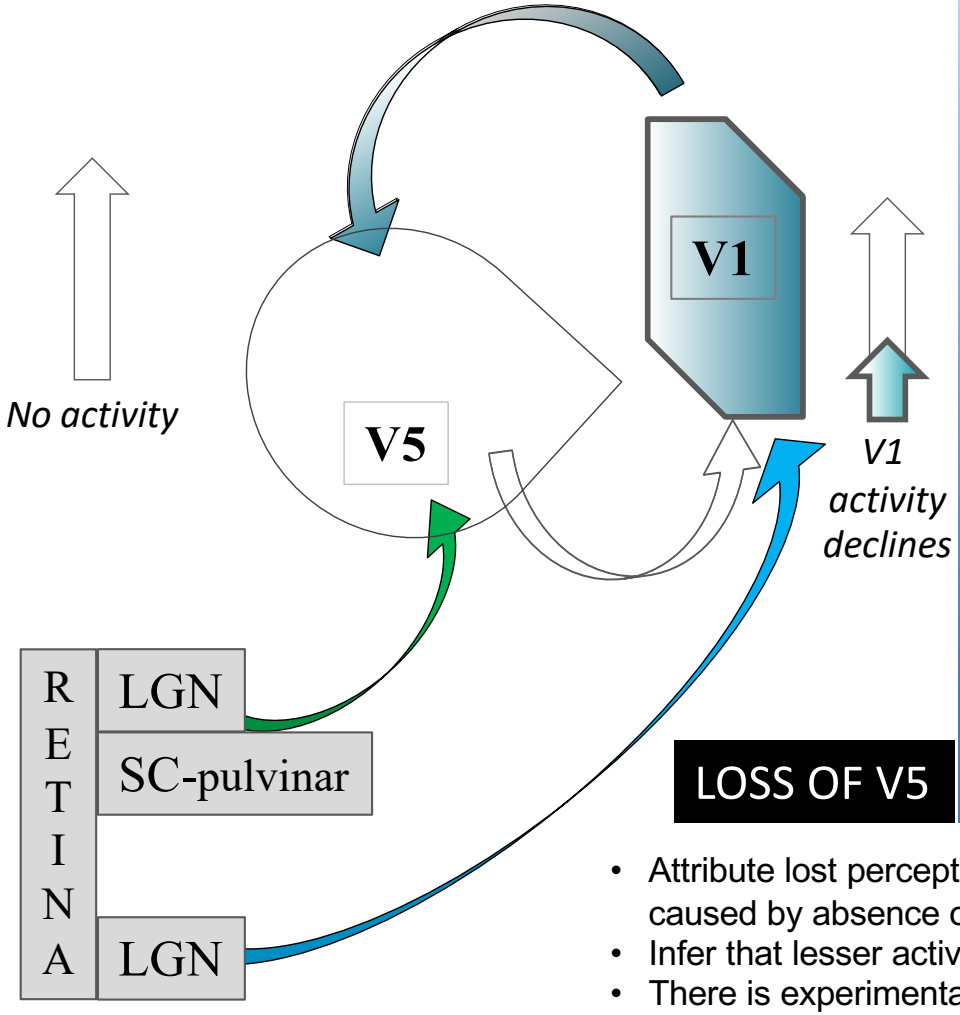
INTACT BRAIN – NORMAL FUNCTIONING

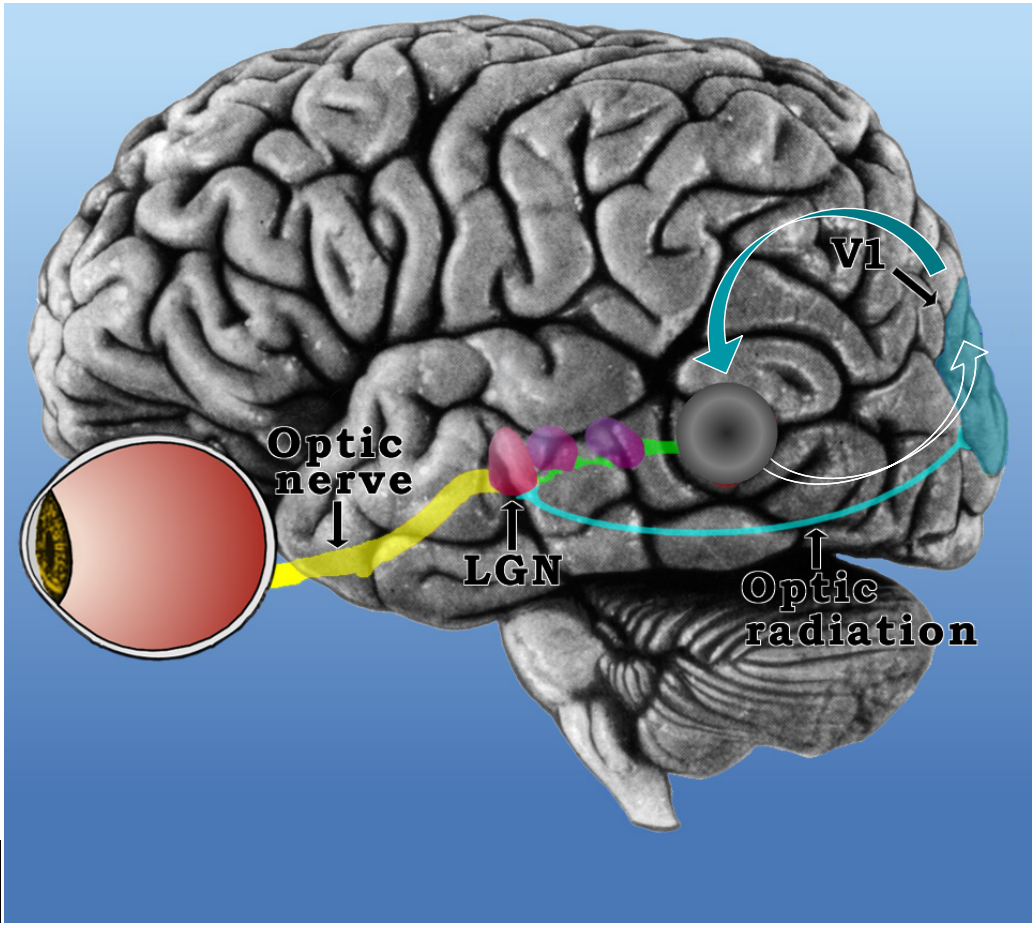
Reciprocal connection from **V5** to **V1** creates a positive feedback loop:

Model of akinetopsia with NCC including V1

'YES' CAMP

NCC



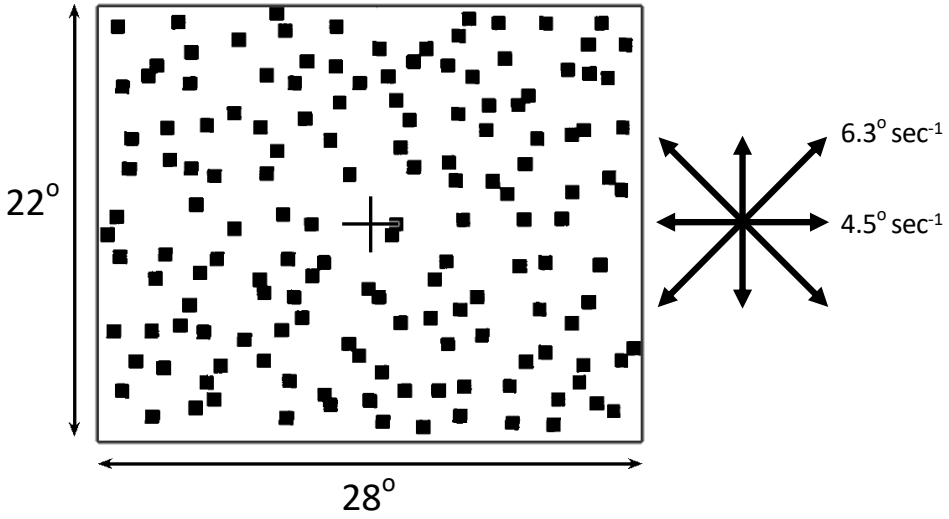


LOSS OF V5

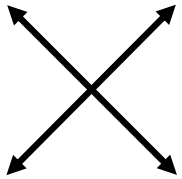
- Attribute lost perceptual capability to destruction of V5, AND reduced activation of V1, caused by absence of feedback from V5;
- Infer that lesser activation of V1 may be NCC, and contribute to residual motion vision;
- There is experimental evidence to corroborate this model (next slide).

The brain activity related to residual motion vision in a patient with bilateral lesions of V5.

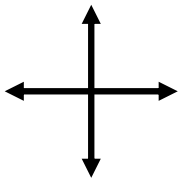
Shipp *et al.* (1994) [ref 7]



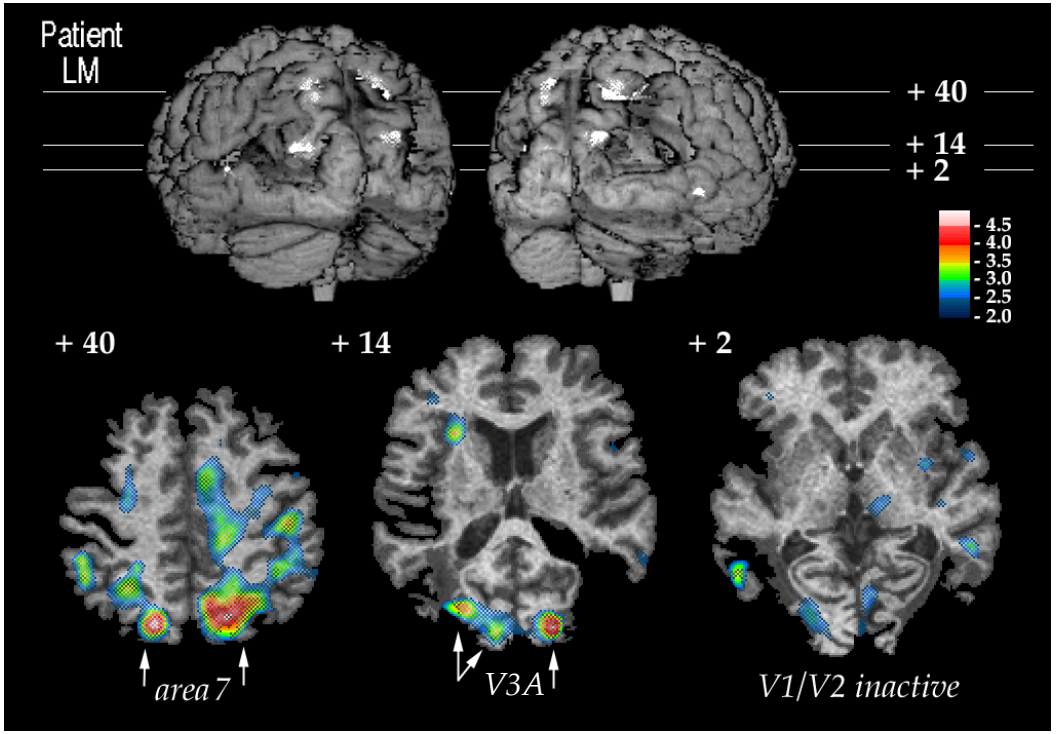
DIRECTION DISCRIMINATION TASK

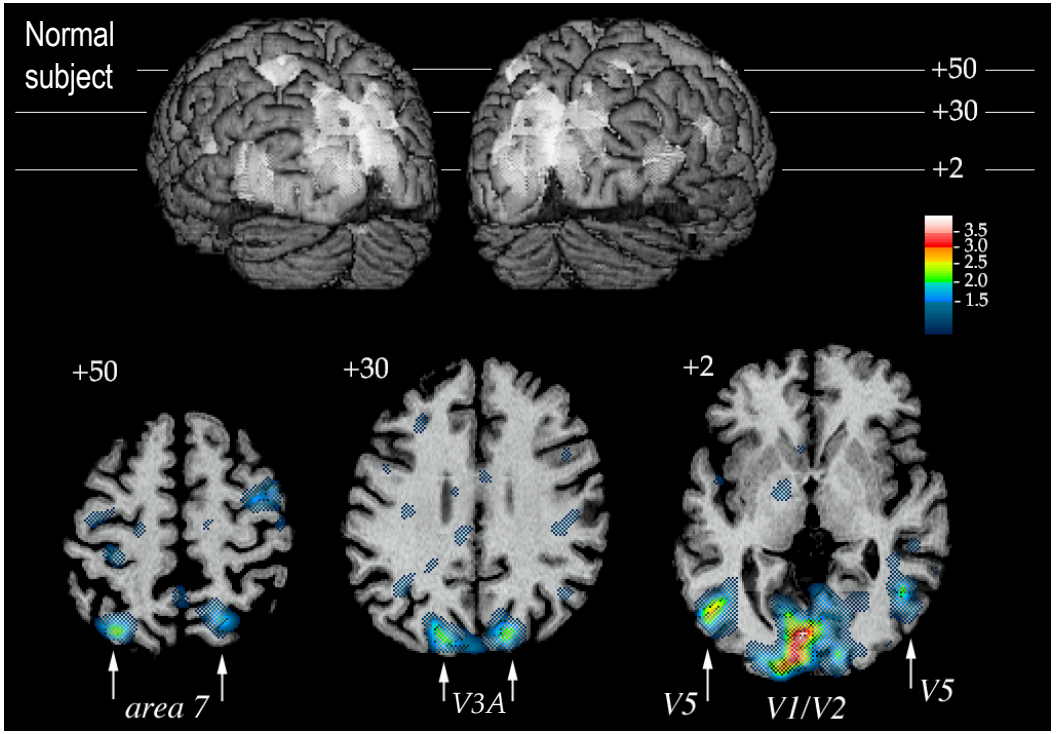


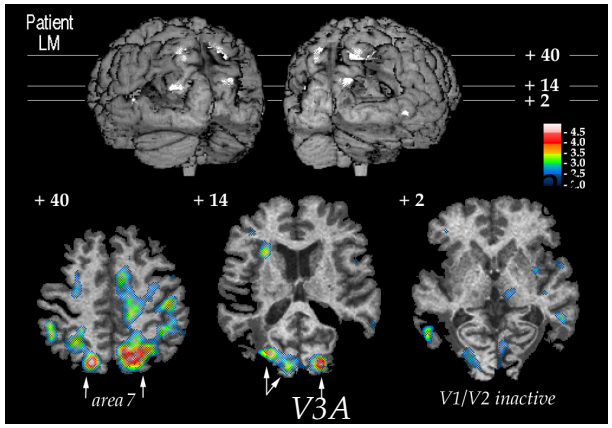
Direction: 0% correct
Axis: 0% correct

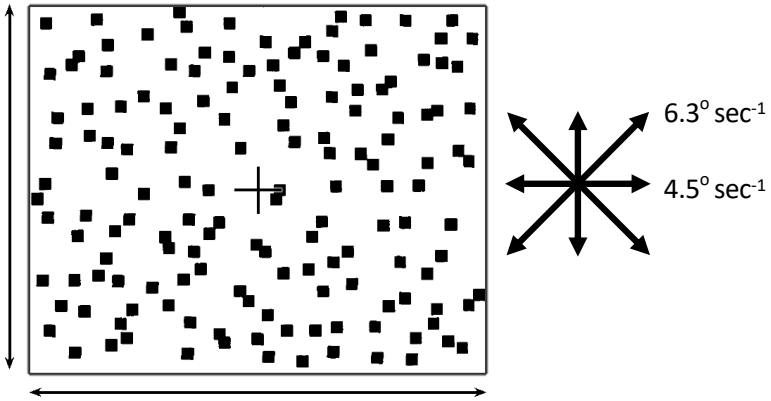


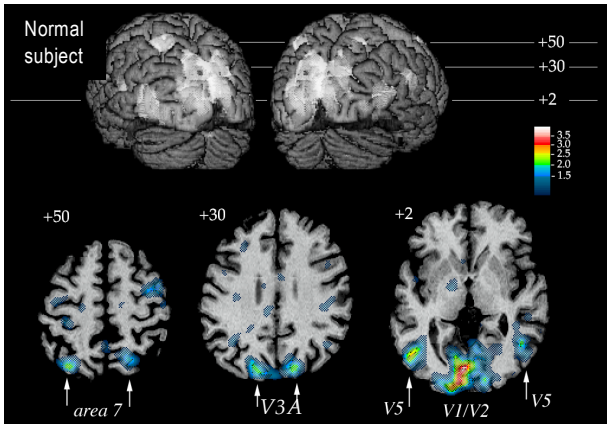
Direction: 82% correct
Axis: 100% correct











The previous slide shows a PET scan of the brain of akinetopsic subject LM, when viewing a display of coherently drifting rectangles; the image shows the differential activity caused by motion v static displays.

The speed of drift motion was slow; at this speed LM does have some residual motion awareness.

The lower image (or above right) shows equivalent activity elicited in a normal subject.

Note that LM shows activation in areas that appear to correspond to V3A and area 7. However there is no activation in V5 (destroyed bilaterally by the lesions), and no activation in V1 (which is intact). The lack of activation of V1 in LM implies that the relative activation of V1 in a normal subject for motion v static requires feedback from V5.

- Hence V1 should not be thought to be functioning normally in LM.
- Hence it would be irrational to rule out V1 from the NCC on the basis of this evidence.

ALSO: LM is not accurate at discriminating motion direction.

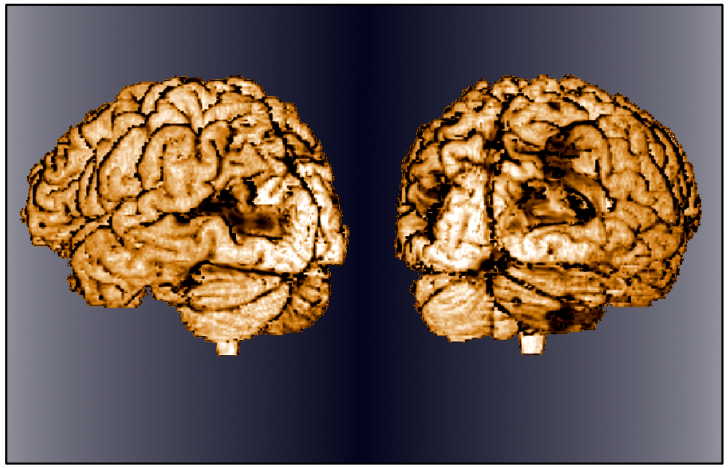
(a) Performance at identifying motion in the oblique directions was 0% - she persistently reported seeing motion in one of the cardinal directions;

(b) Performance at identifying motion in the cardinal directions was 82% - all errors were reports of direction 180 deg opposite;

(c) Residual performance in LM is indicated to arise from activation of areas V3A and area 7. Perhaps these areas are capable of encoding motion perpendicular to the edges of the rectangular elements of the display, but poor at encoding direction proper.

Is area V1 a component of the NCC ?

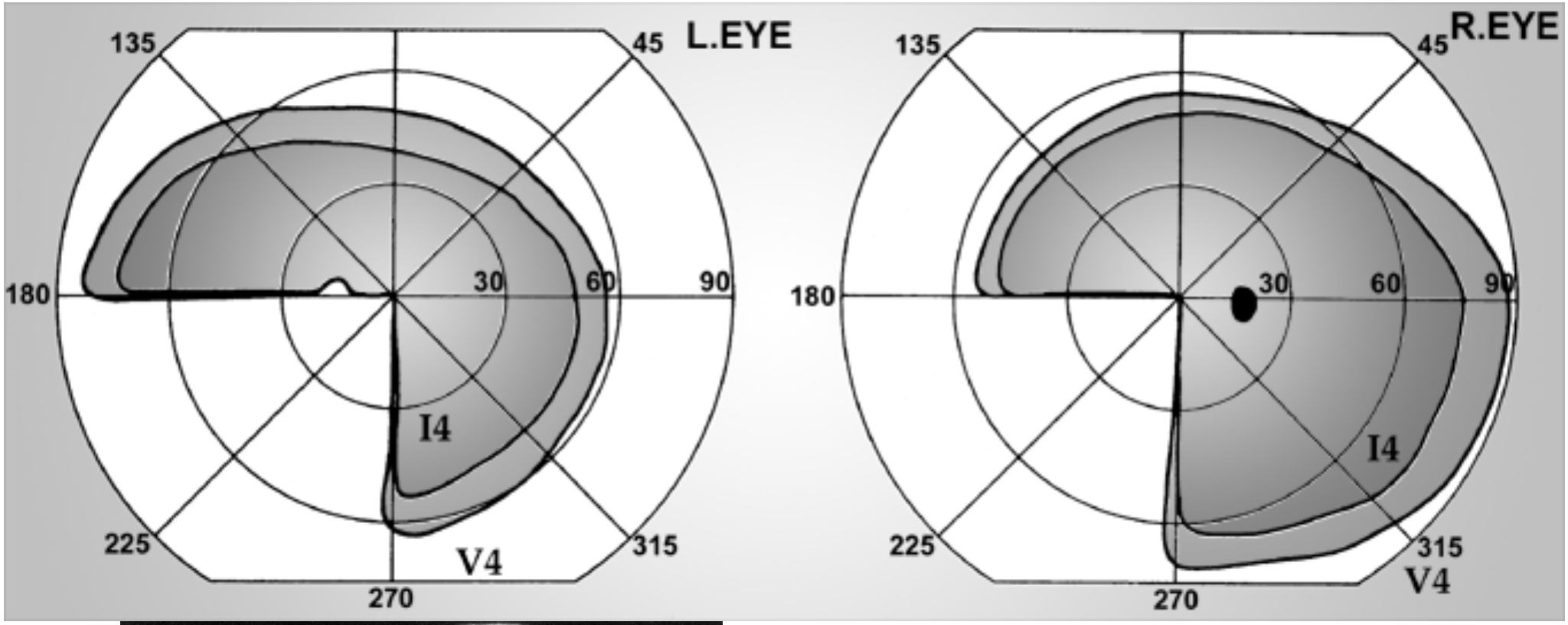
Claim	Evidence	Verdict
NO	No direct communication between V1 and 'decisional /command ' centres of cortex	?
NO	Monocular cells in V1 do not register a conscious percept	INCONCLUSIVE (may not generalise to all V1 neurons)
NO	Colour cells in V1 do not exhibit the Land Mondrian (colour constancy) effect , unlike cells in V4	INCONCLUSIVE (V1 cells may be necessary, but not sufficient for colour percept)
NO	Awareness of motion in blindsight ('Riddoch phenomenon')	VALID… but trivial: *infer V1 is not NCC for 'residual' motion perception.*
NO	99% loss of normal motion vision with V1 lesion is ascribed to deafferentation of higher visual areas	INVALID (Ignores V1 participation in recurrent circuitry)
NO	Akinetopsia in case LM (bilateral lesion of V5), with V1 intact (i.e. including direction-selective neurons in layer 4B of V1 that provide direct output to V5).	INVALID (ignores V1 participation in recurrent circuitry)

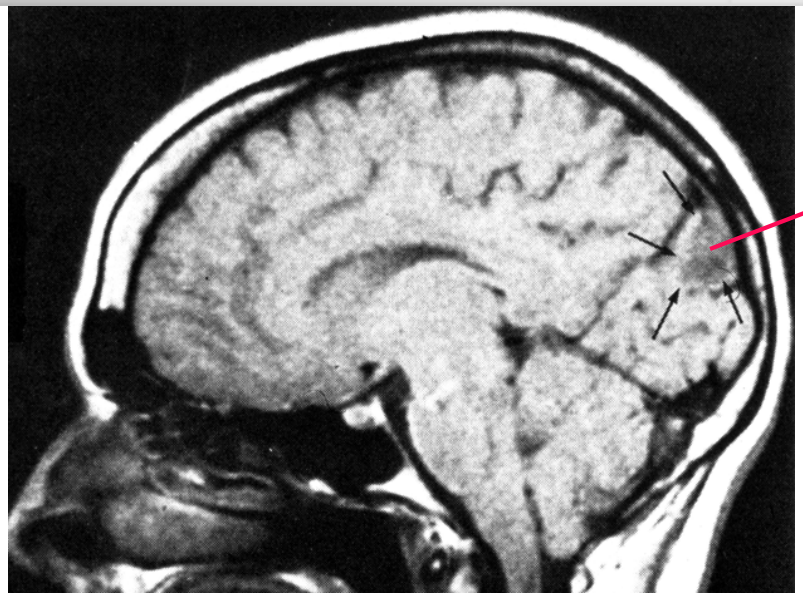


NCC = *The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.*

Horton & Hoyt (1991)

Cortical Representation





Quadrantanopia from V2 lesion

Excised tumour:

Destruction of superior contralateral quadrant representation in V2/V3

Is area V1 a component of the NCC ?

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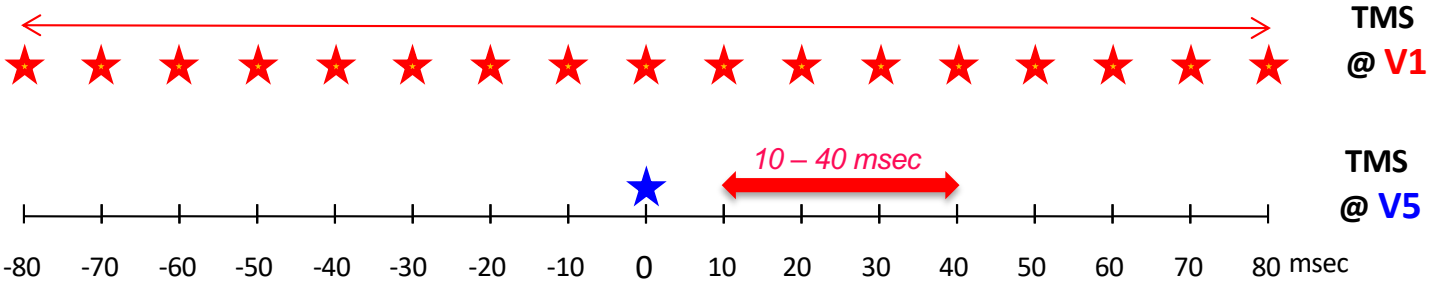
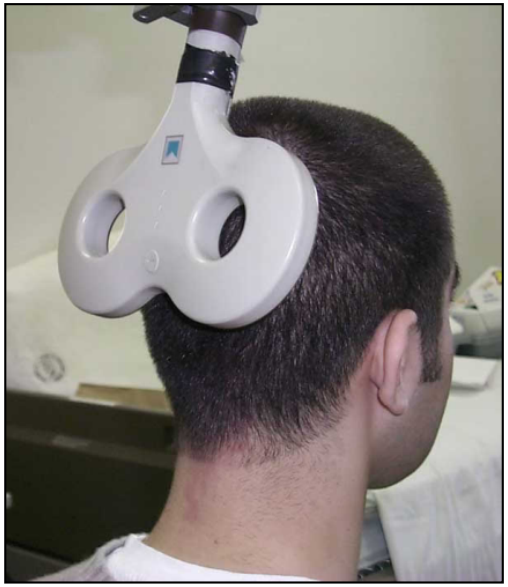
NO	Quadrantanopia resulting from unilateral, dorsal V2/V3 lesion, with V1 intact	INVALID (ignores V1 participation in recurrent circuitry)

NCC = *The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.*

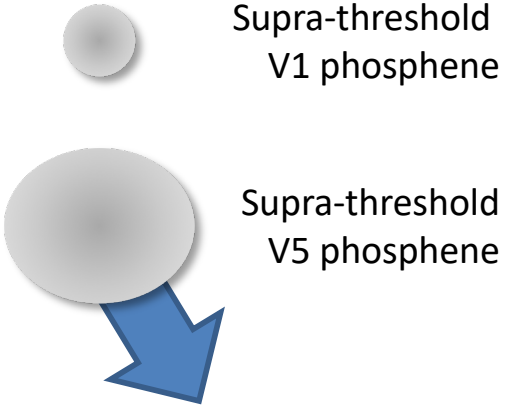
Is area V1 a component of the NCC ?

Silvanto *et al.* (2005) [ref 8] *'V1 activity gates awareness of motion'*
(TMS over V1 modulates visual feedback from V5, and affects motion percepts of phosphenes).





TMS @ **V5**	TMS @ **V1** (+10 to +40 msec)	phosphene percept
Supra-threshold	Sub-threshold	Nil (implying suppression of V5 phosphene)
Sub-threshold	Supra-threshold	Moving & larger than V1 phosphene ("*a mixture of V1 & V5 phosphenes*")
Sub-threshold	Sub-threshold	Nil



Conclusion: processing of re-entrant signals in V1 can affect percepts (negatively or positively)

Is area V1 a component of the NCC ?

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NO	Quadrantanopia resulting from unilateral, dorsal V2/V3 lesion, with V1 intact	INVALID (ignores V1 participation in recurrent circuitry)
YES	TMS to V1, disrupting feedback from V5, impairs motion perception	VALID ? (if rudimentary)

NCC = *The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.*

The neural correlate of consciousness...

The minimal neuronal mechanisms jointly sufficient for any one specific conscious percept.

Crick & Koch (1998) Cerebral Cortex. 8: 97-107

'Anatomical' interpretation of NCC

- Which parts of the brain..?
- Which circuits within/between which areas?

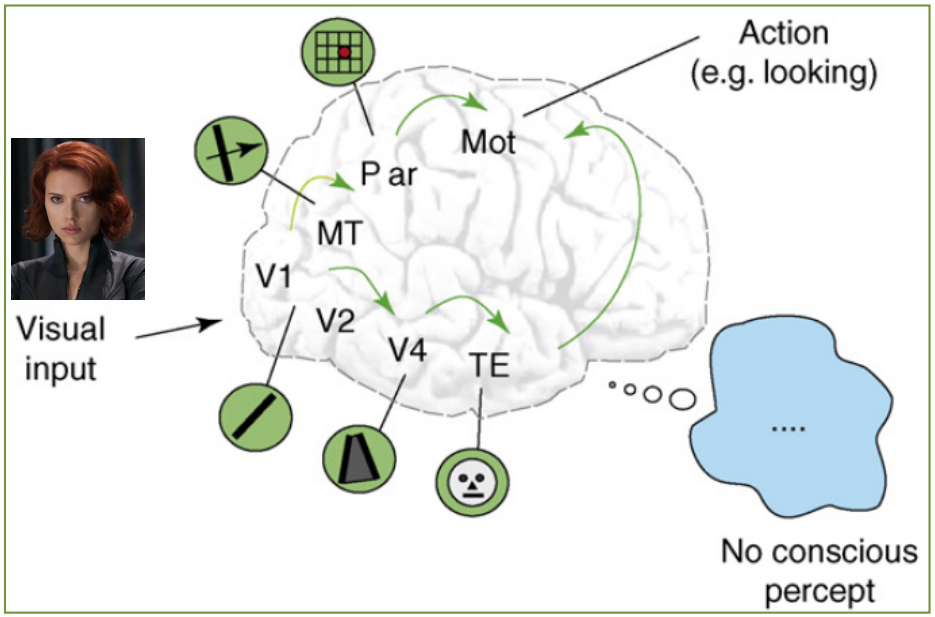
Dfficulties in resolving the apparently simple question of whether area V1 is, or is not NCC suggest that an 'anatomical' conception of NCC is not helpful. Does a 'physiological' conception of NCC prove to be more insightful ?

'Physiological' interpretation of NCC

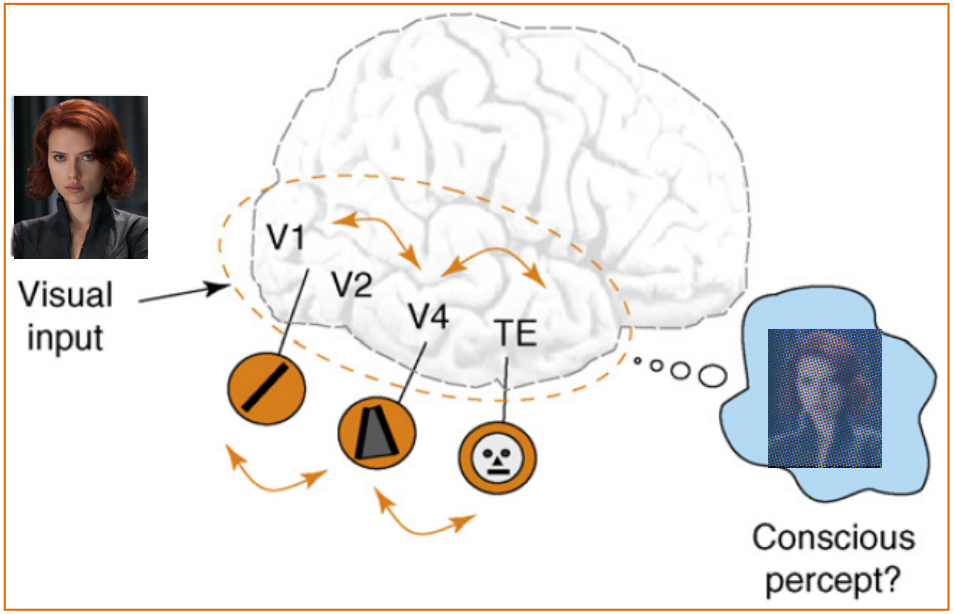
- What pattern of activity..?
- What timing of activity..?

Lamme (2006) Theory that recurrent (or re-entrant) processing is the key component of NCC

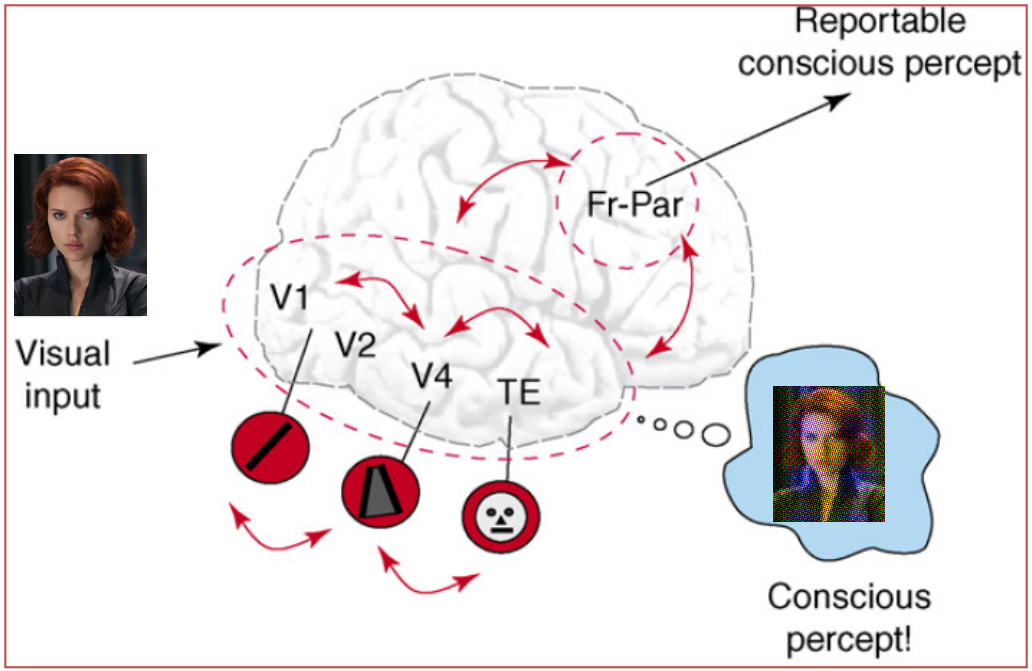
The feedforward sweep



Localised recurrent processing

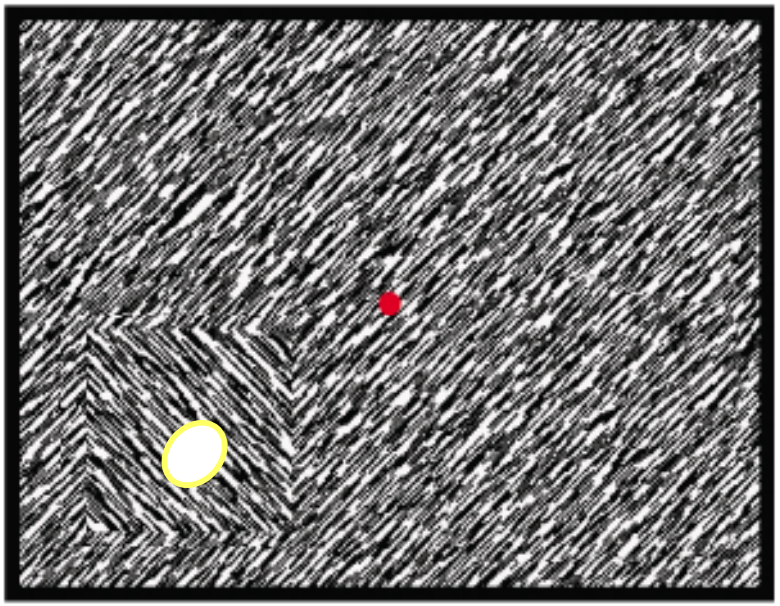


Widespread recurrent processing

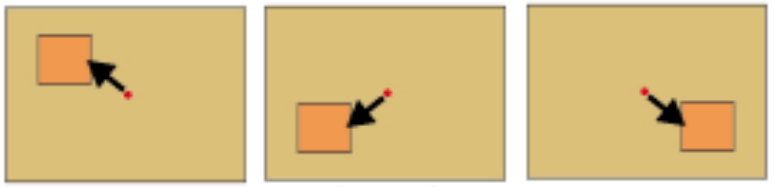


'Phenomenal' Consciousness
v.
'Access' Consciousness

Super *et al.* (2001) V1 (multiunit) activity correlating with target detection (or visibility) [ref 10]



80% target trials
(3 possible locations)



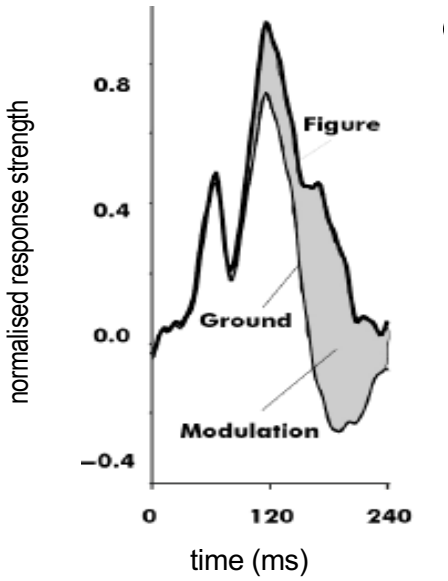
20% 'catch' trials

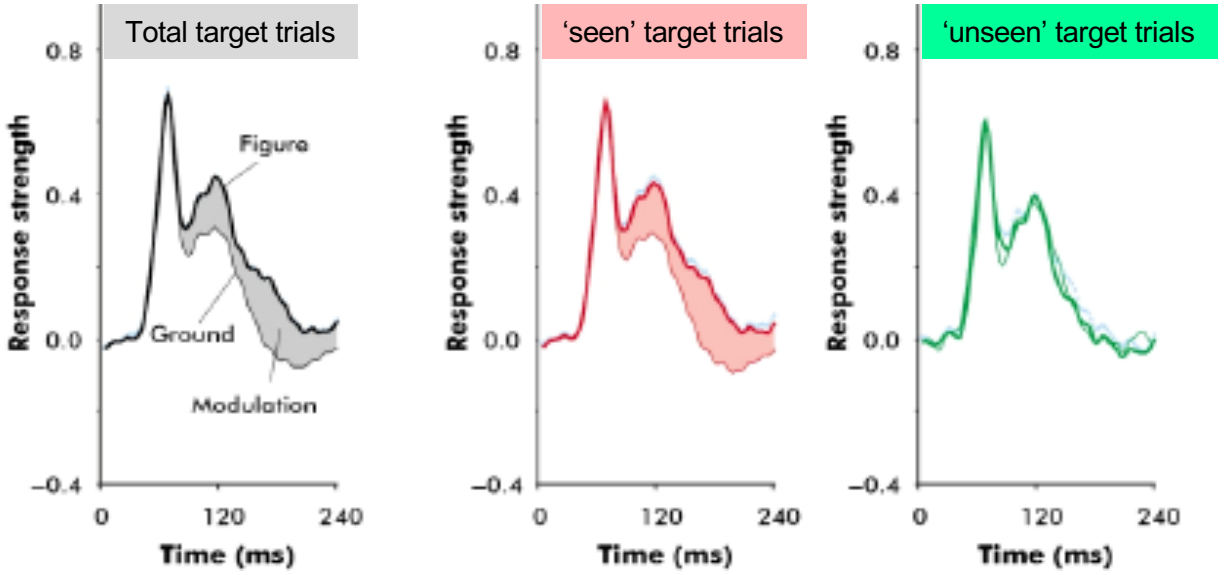
Task:

saccade to figure;

<u>or</u>

maintain fixation.





Super *et al.* (2001) V1 (multiunit) activity correlating with target detection (or visibility) [ref 10]

Note on this study:

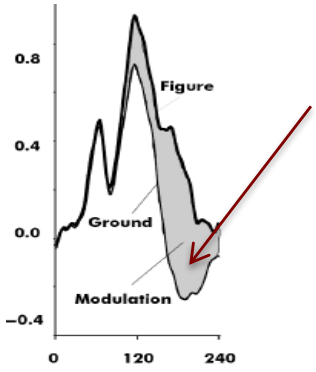
Stimulus display & figure/ground detection task

A 'figure' is defined by the orientation of its texture, orthogonal to that of the background; the two orientations used are switched from trial to trial, so the figure is not associated with any particular orientation. Monkeys are trained to detect the onset of the figure stimulus, and to make a saccade (rapid eye movement) to the centre of the stimulus as soon as they see it. They start each trial by fixating on the red spot at screen centre. The figure target may appear at one of three possible locations or, in so-called 'catch' trials, no target is displayed and the monkey is rewarded for continuing to fixate centrally.

Because the figure is not easy to see (especially when fixating centrally) there is a certain proportion of target trials in which the figure is displayed but is missed by the monkey. These are called 'unseen' target trials.

Data collection and analysis

The activity of cells in area V1 is recorded when their receptive field (RF) is stimulated by the figure, or by the ground. The figure is substantially larger than the RF, and all its boundaries lie outside the RF. Hence the cell cannot detect the presence or absence of the figure by analysing the orientation presented within its RF. However, at about 100 msec after target onset (well before the monkey makes a saccade) there is a small increase in cell firing rate when the RF coincides with the figure rather than the ground stimulus (shown by the filled-in, grey area of the response histogram).



When this response data is averaged across lots of trials, but split into two groups – 'seen' versus 'unseen' target trials – the difference between figure and ground responses is only present in the seen target trials; it is absent from the 'unseen' target trials.

Interpretation

The increased activity associated with the figure stimulus is thought to be produced by backward connections from higher areas to V1, modulating the activity of the recorded cells. In other words, higher areas are thought to be the first to detect the figure, and to signal this information back to V1. But, this only happens if the animal is aware of the figure; in 'unseen' target trials, the monkey behaviourally has failed to see the target, and the modulation of V1 cells associated with target detection is not observed. Hence the modulation of activity can be regarded as a neural correlate of figure detection, and potentially a candidate for an NCC mechanism.

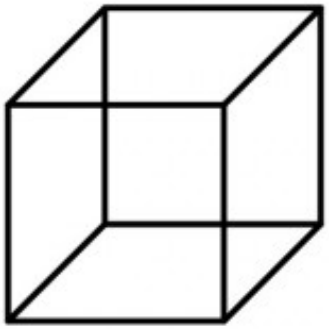
Bistable (or multistable) percepts

Experimental strategy to study NCC

Bistable percepts:

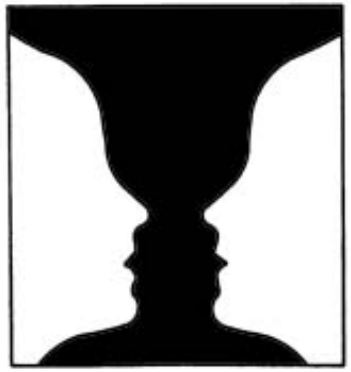
- The 2 (or more) interpretations are mutually intolerant: only one is seen at a time.
- This implies a competitive or suppressive interaction between the neural networks that sustain each percept

- Non-fluctuating activity concerns basic processing of the fixed retinal image;
- Activity fluctuating with rivalling percepts may be a component of N.C.C.



Necker Cube

Swiss Crystallographer
Louis Necker (1832)



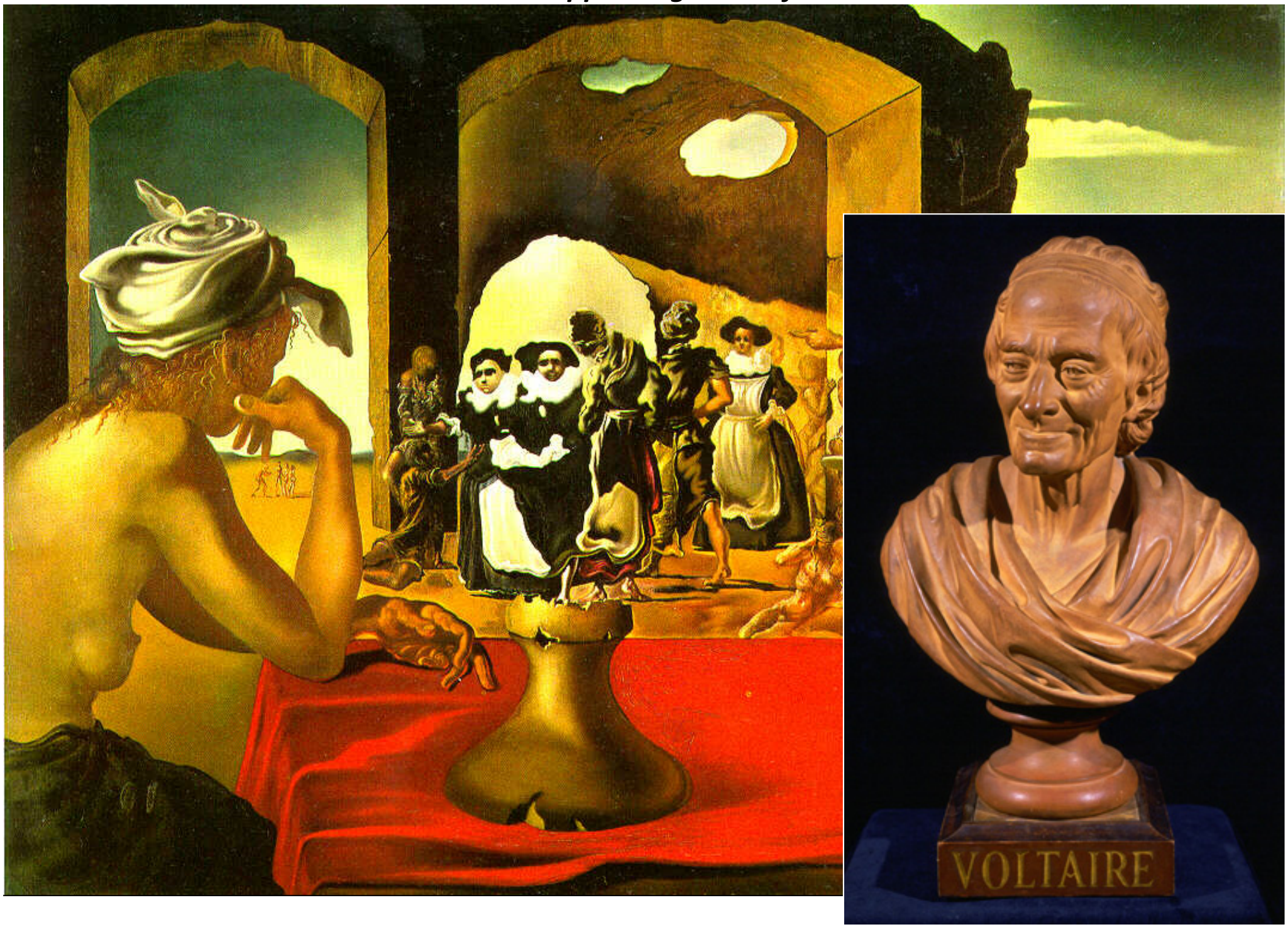
Ruben's Vase

Danish psychologist
Edgar Rubin (1915)

Elusive Arch

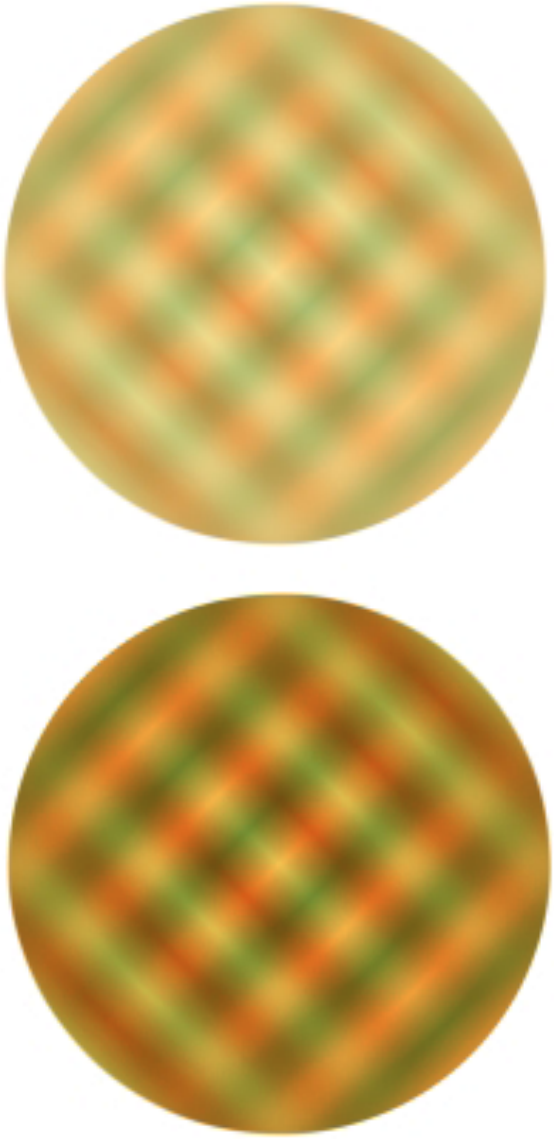
Serbian Psychologist
Dejan Todorović (2005)

Salvador Dali – ***'Slave Market with Disappearing Bust of Voltaire'***



Experimental strategy to study NCC

The natural phenomenon of ‘Monocular Rivalry’



Experimental strategy to study NCC

The natural phenomenon of 'Binocular Rivalry' (a.k.a. 'Retinal Rivalry')

Advantages of binocular rivalry as a bistable percept for experimental study:

1. No restriction of image content – can select stimuli to suit the response properties of the area of visual cortex being studied.

2. The entire percept switches – not just the interpretation; hence may be able to observe fluctuating activity at earlier stages of analysis.

Dichoptic stimulus

LEFT EYE RIGHT EYE

percept

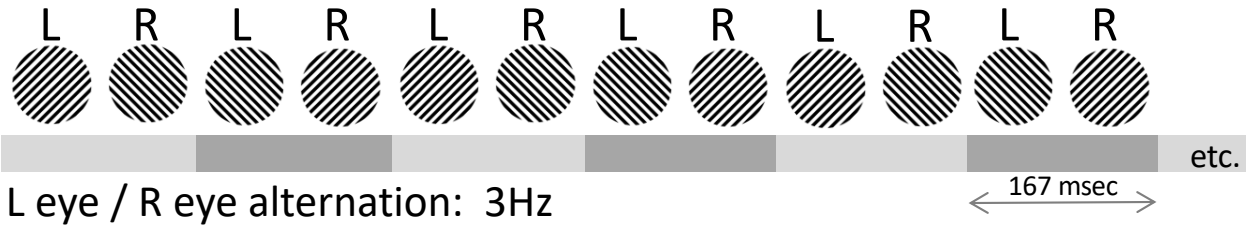
time →

'piecemeal rivalry'

Logothetis, Leopold & Sheinberg (1996) What is rivalling during retinal rivalry ? [ref. 11]

CONVENTIONAL:
The rival stimuli are continuously presented to each eye
(static dichoptic stimulation)

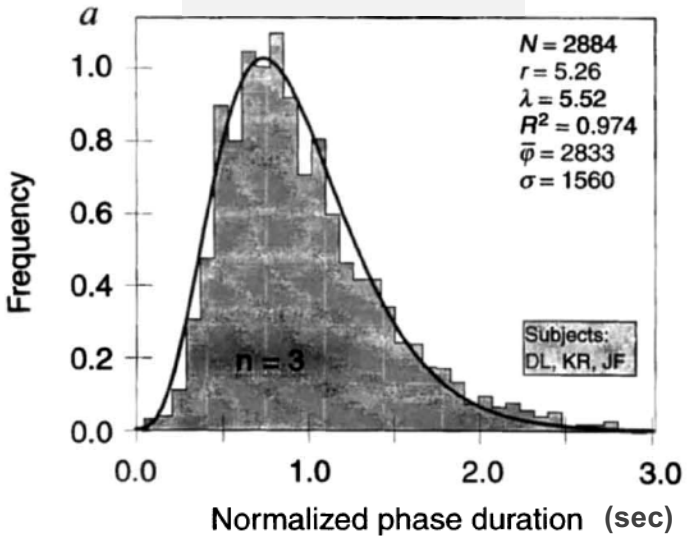
ALTERNATING:
The rival stimuli are swapped between the eyes at 3Hz
(dynamic dichoptic stimulation)



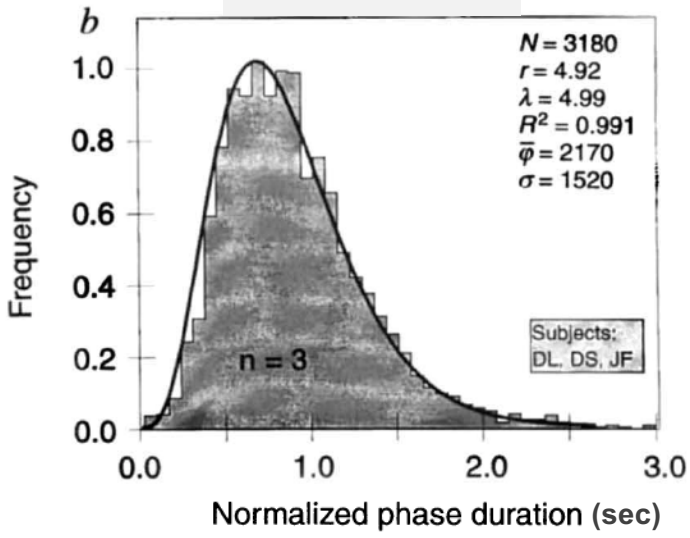
L eye / R eye alternation: 3Hz

Result:

CONVENTIONAL



ALTERNATING

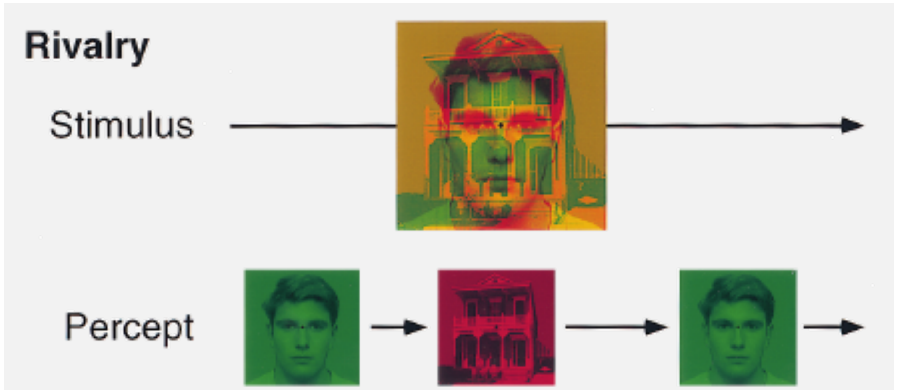


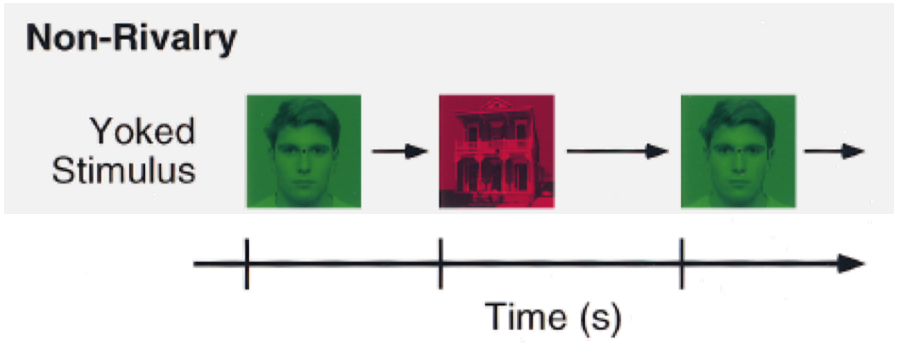
Conclusion:

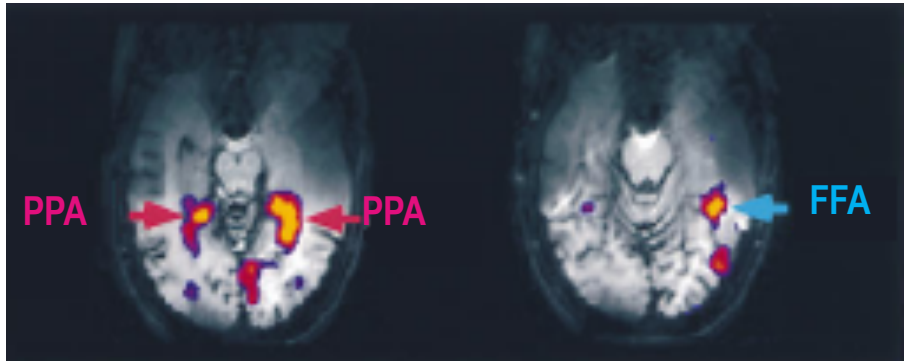
"The rivalry experienced when monocular stimuli are continually swapped between the eyes is indistinguishable from conventional binocular rivalry."

Inference: *that rivalry reflects competition, not only between neurons with R or L eye ocular dominance, but also between higher level neurons selective for objects/features presented to the R or L eye.*

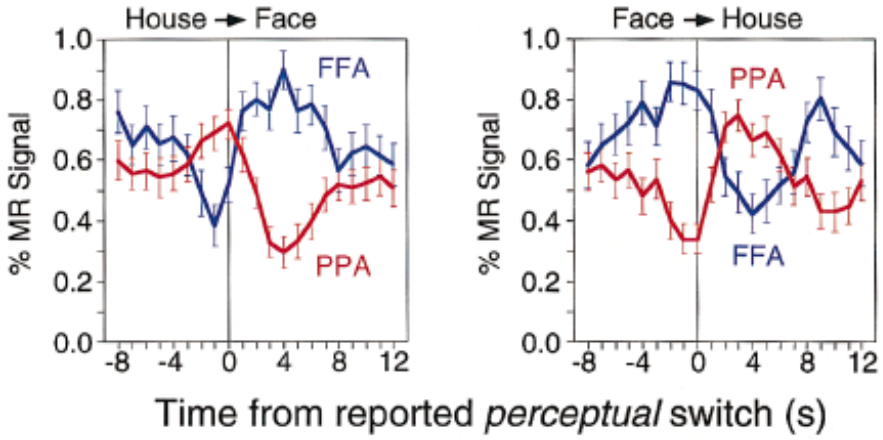
Tong *et al.* (1998) *(fMRI)* activity correlating with rivalrous percepts in *human* 'face' & 'place' areas (FFA & PPA) [ref. 12]

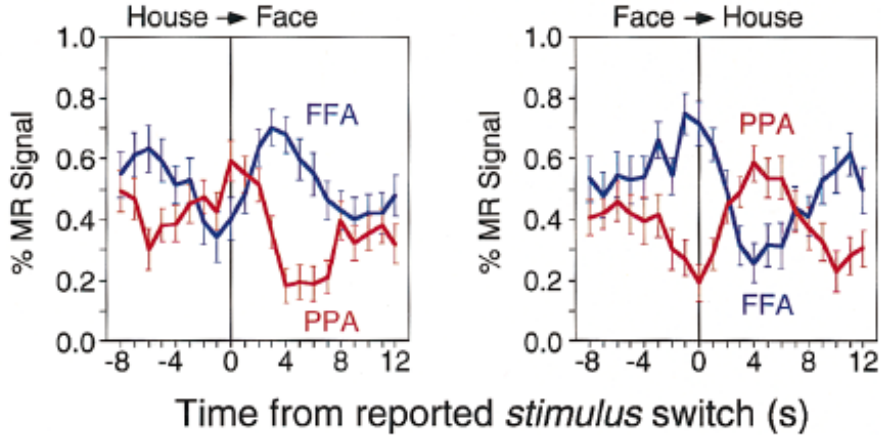






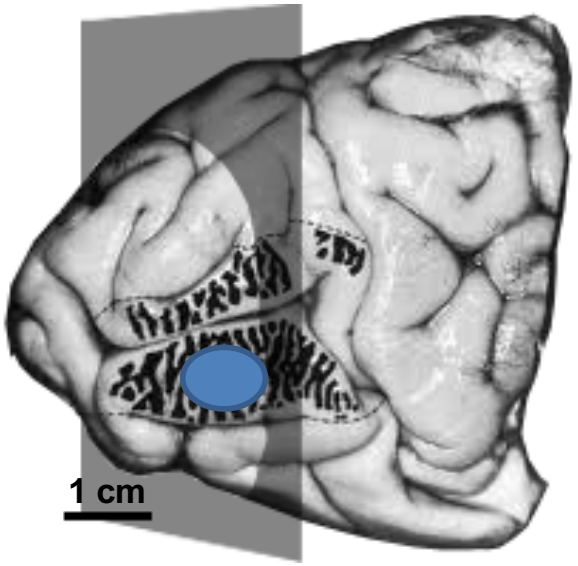
Parahippocampal Place Area Fusiform Face Area

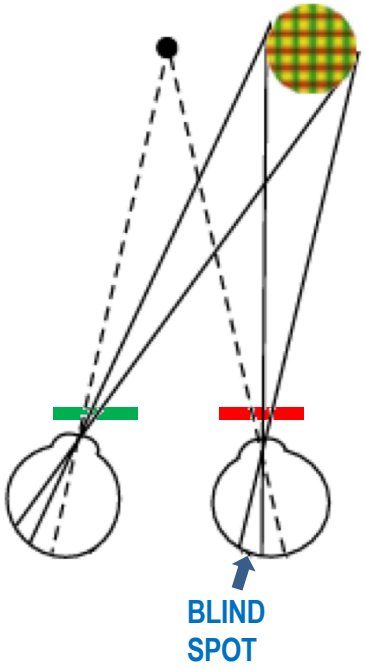


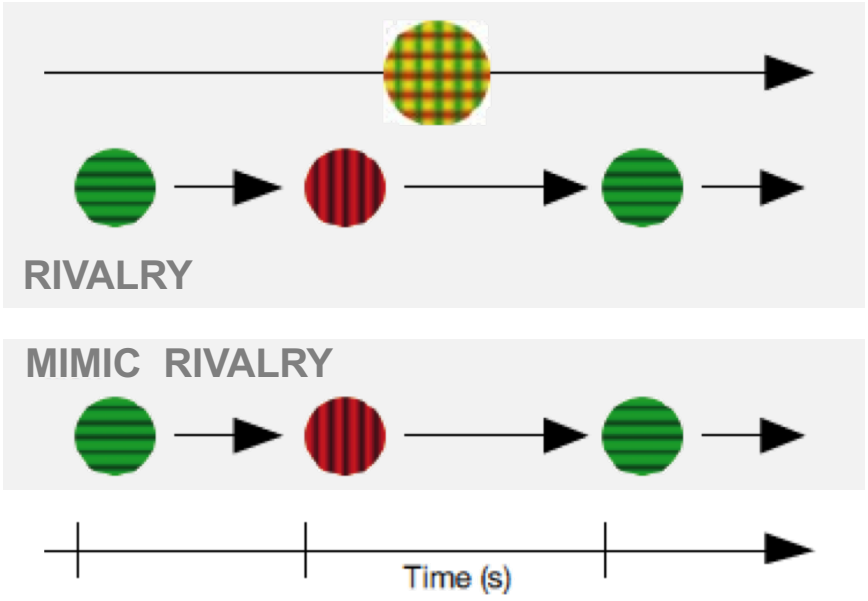


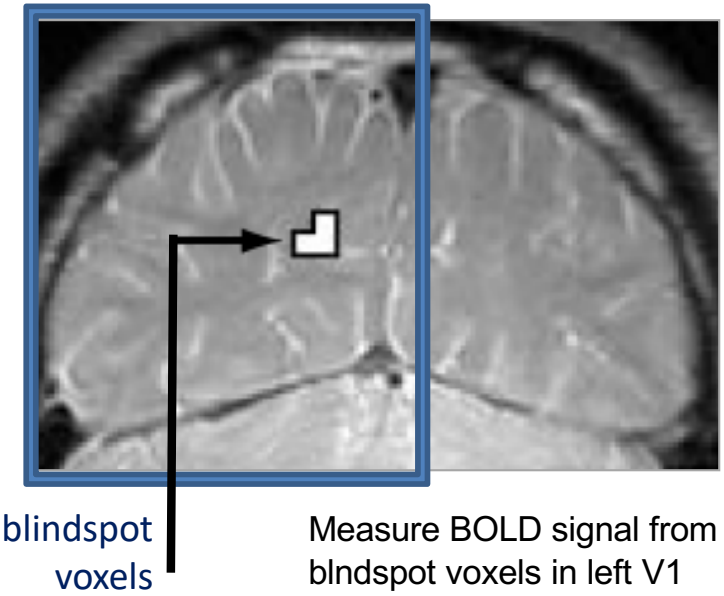
Averaged data from a single subject

Tong & Engel (2001) [ref13] *(fMRI)* activity correlating with rivalrous percepts in *human V1*

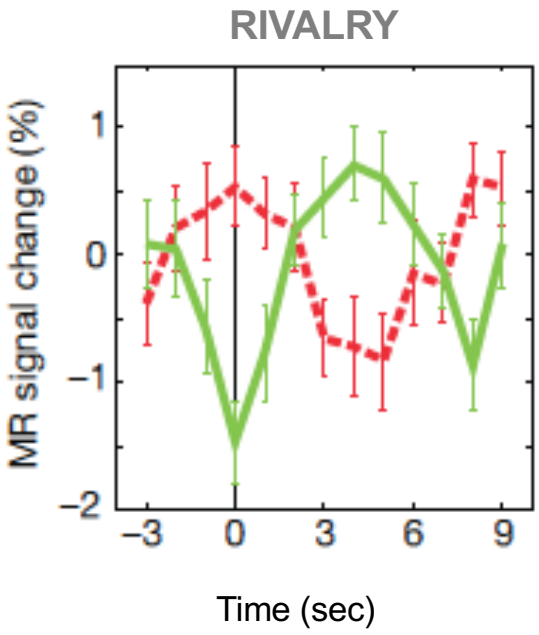


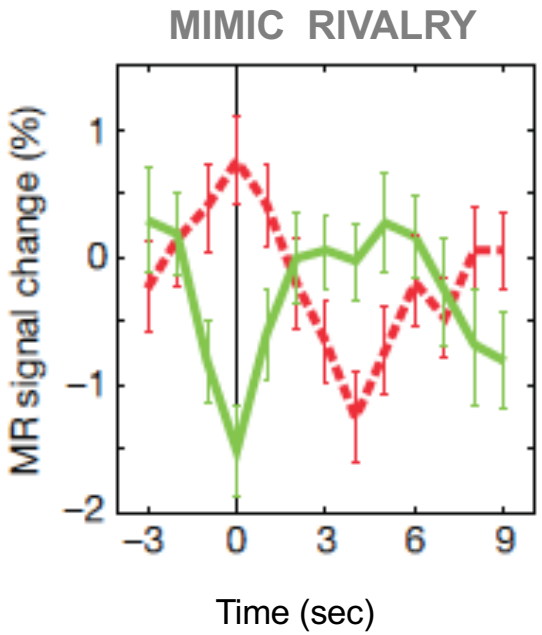






Measure BOLD signal from
blndspot voxels in left V1



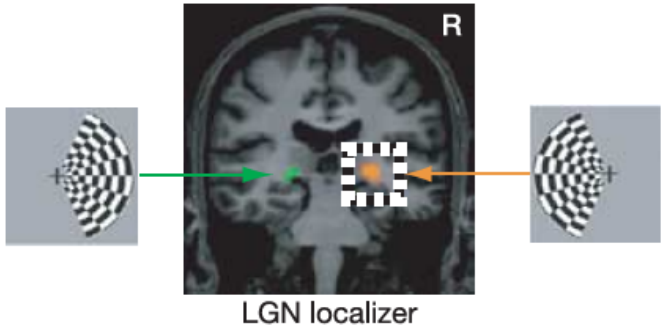


Notes on Tong & Engel (2001) [ref 13]

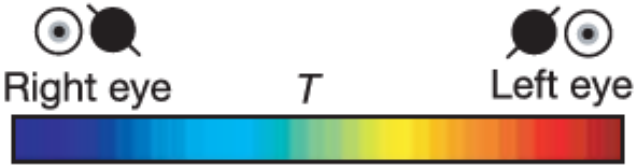
Interocular rivalry revealed in the human cortical blind-spot representation

- The image is placed so as to fall on the blind spot of the right eye. The blind spot representation in contralateral V1 is around 10 x 5 mm in size; this is considerably wider than a human ocular dominance column in V1, and the only brain location where MRI voxels (using MR technology as of 2001) can sample the signals from a single eye.
- The image is a grid formed of red vertical and green horizontal stripes; R & L eyes are equipped with red and green filters to give rivalrous images of a vertical or horizontal grating in either eye.
- The size of the grid on the retina is about twice the size of the blind spot; the image falling on the blind spot is perceptually filled in (by cortical mechanisms not studied here); hence the subject is not aware that it is placed over the blind spot, and sees the stimulus equally well through either eye.
- In the 'mimic rivalry' condition, the subject is shown a sequence of non-rivalrous vertical and horizontal gratings, presented monocularly using the same sequence of alternations as reported in a previous rivalry scan.
- Analyse BOLD signal recorded from voxels in left V1 coinciding with representation of blindspot in R eye;
- MR signal change plots: time zero in the green trace marks reports of a switch in percept from (red)/vertical blind spot grating (seen by contralateral R eye) to (green)/horizontal grating (seen by ipsilateral L eye at a non-blindspot location). Time zero in the red trace marks report of the opposite switch in percept.
- Conclusion: BOLD activity in the representation of the left eye blind spot fluctuates with the perception, or not, of the stimulus shown to that eye. Note the delay of a few seconds between report of perceptual change and subsequent peak (or trough) of BOLD signal, known as 'haemodynamic lag'.

Haynes *et al.* (2005) *(fMRI)* activity correlating with rivalrous percepts in human LGN & V1 [ref 14]



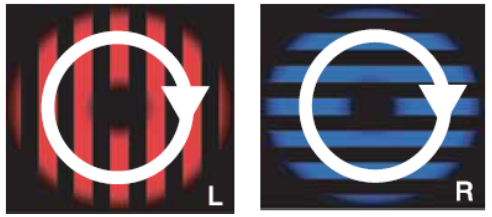
LGN localizer



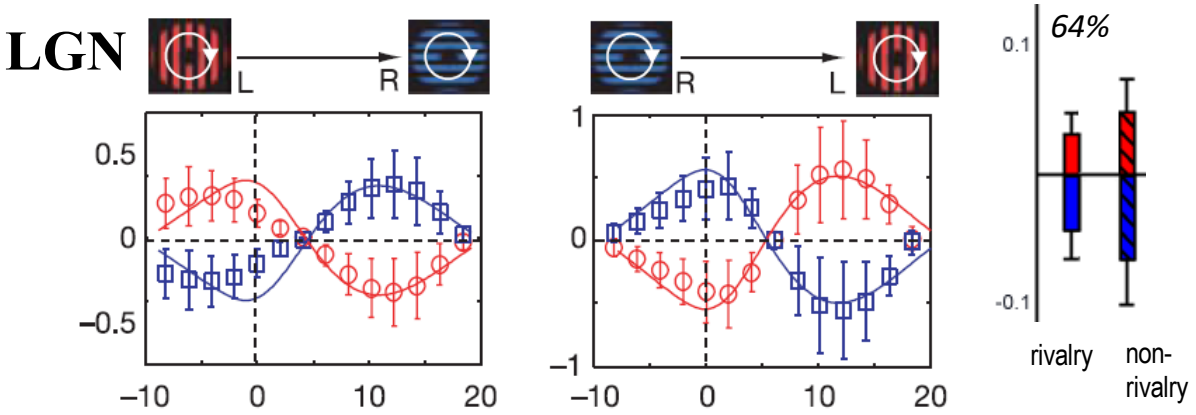

LGN

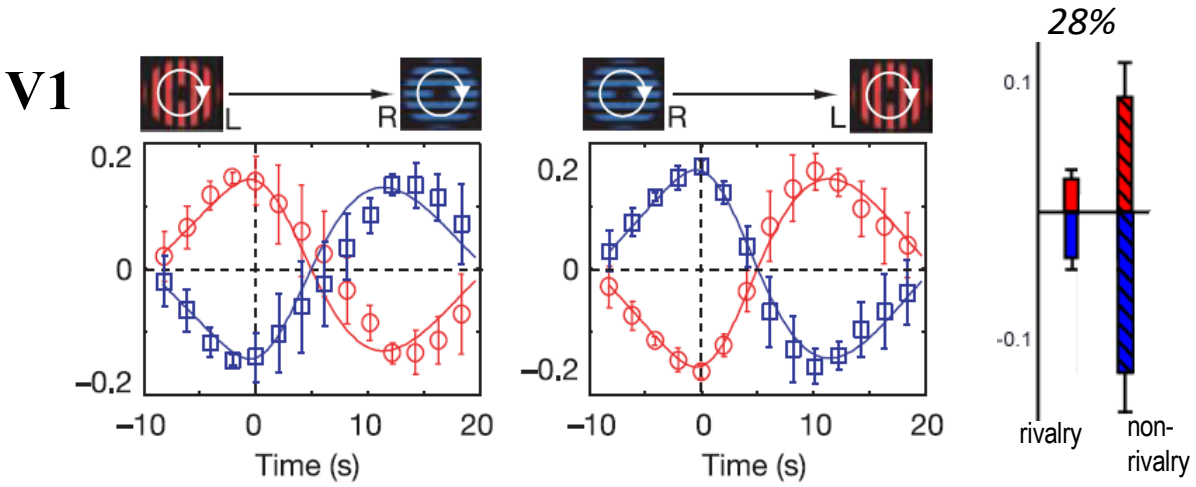
High resolution imaging:
– 1.5 mm cubic voxels.

50% LGN voxels have a significant R or L eye bias.



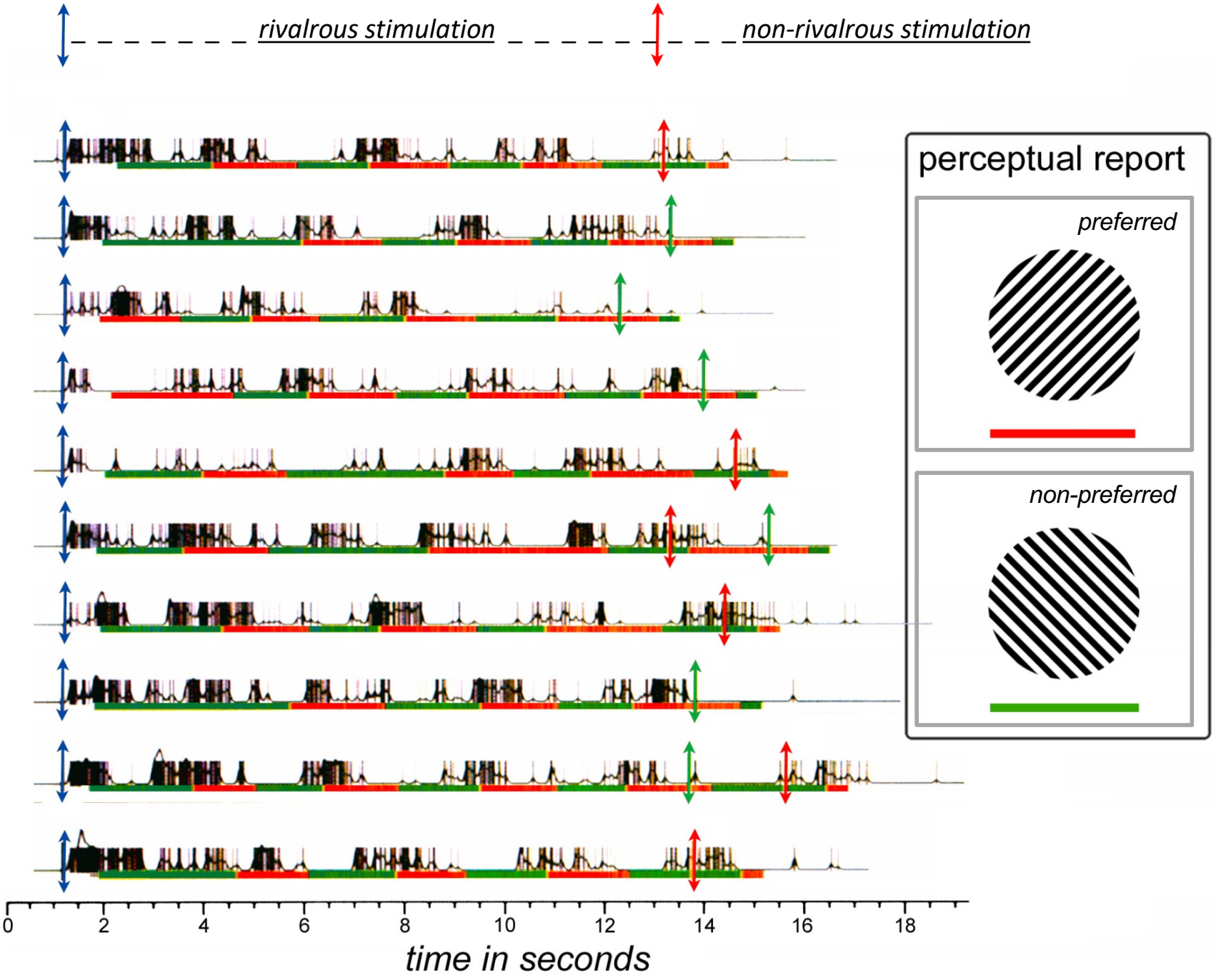
dichoptic stimulus



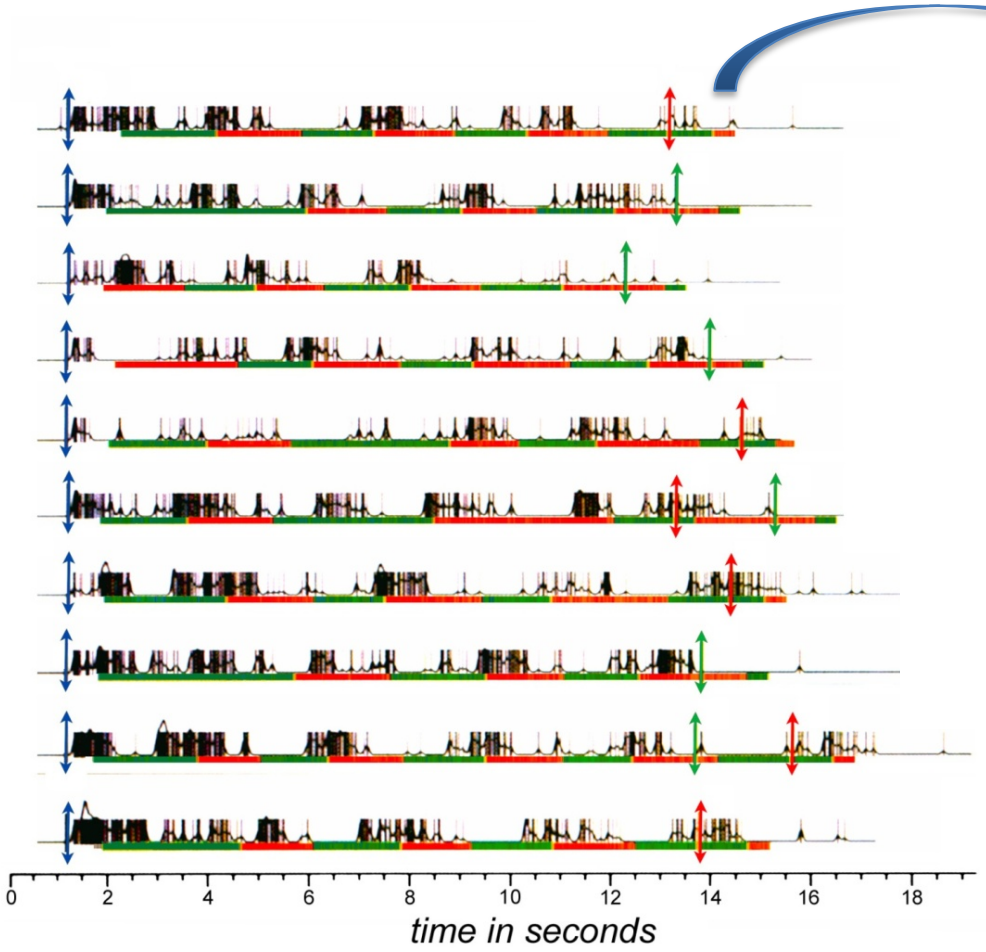


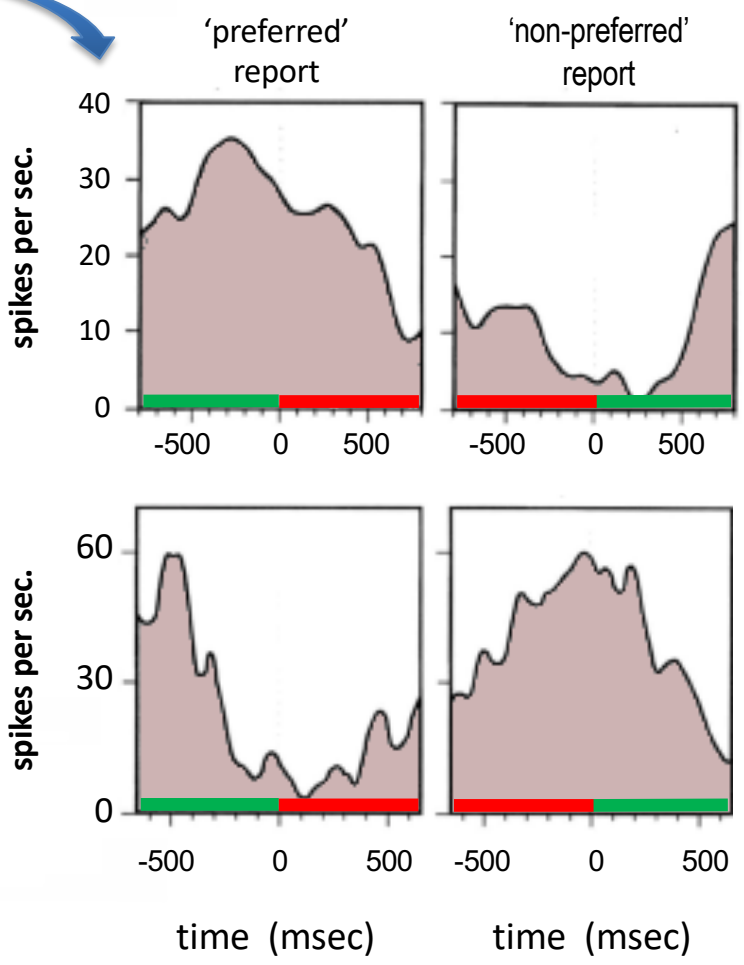
Interpretation: **LGN** & **V1** are both components of NCC !

Leopold & Logothetis (1996) activity correlating with rivalrous percepts in area V4 [15]



Leopold & Logothetis (1996) activity correlating with rivalrous percepts in area V4 [15]





18%

9%

Area V4:

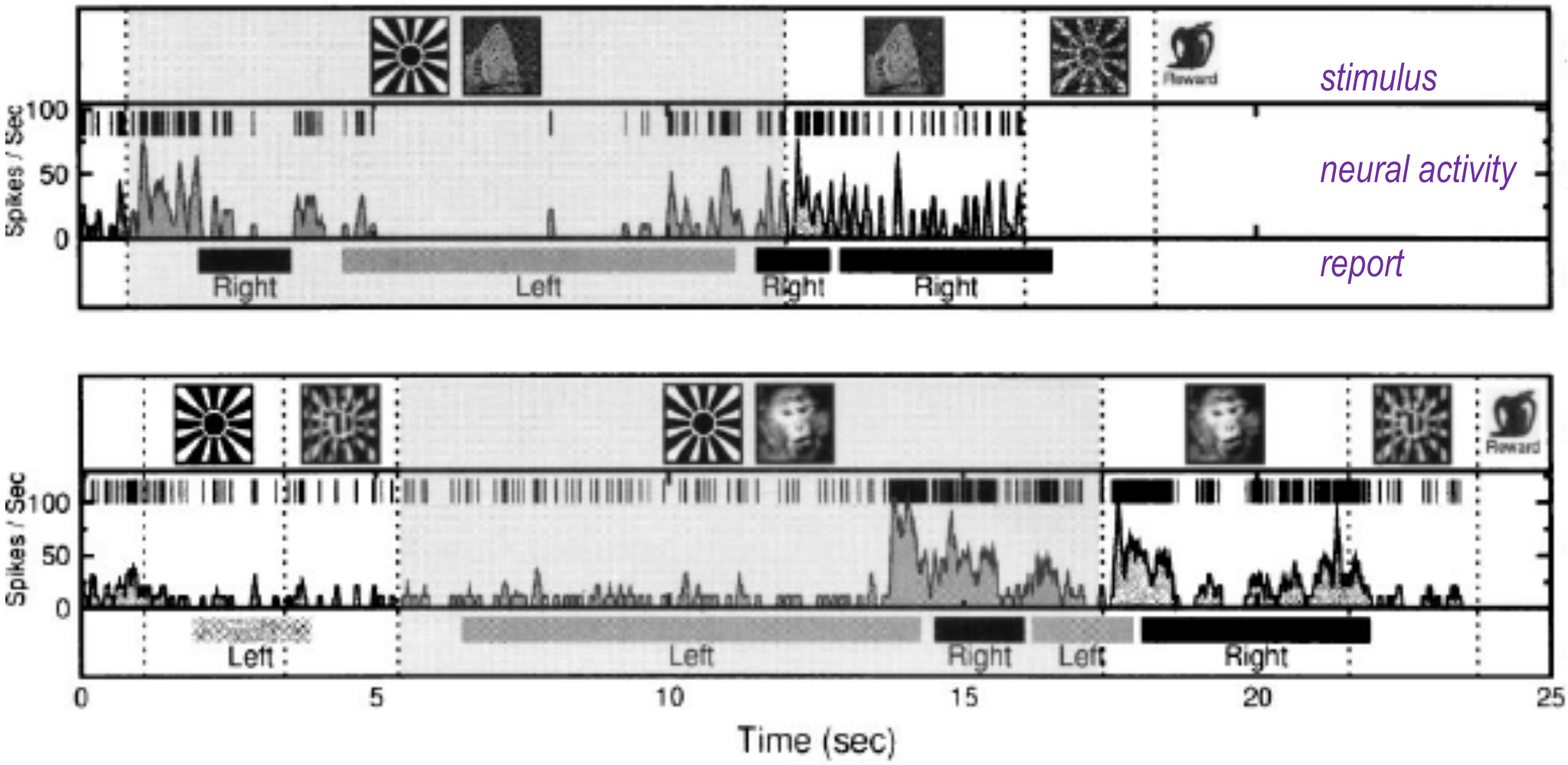
- **18%** of neurons are more active whilst the monkey reports seeing their preferred orientation = 'preferred report'.
- **9%** of neurons are more active whilst the monkey reports seeing their non-preferred orientation = 'non-preferred report'.
- **12%** of neurons lacking a preferred orientation also had activity modulating with the reported percept.
- **61%** of neurons did not have activity modulating with the reported percept.

non-oriented 12%

Fraction of modulating neurons, V4 39%

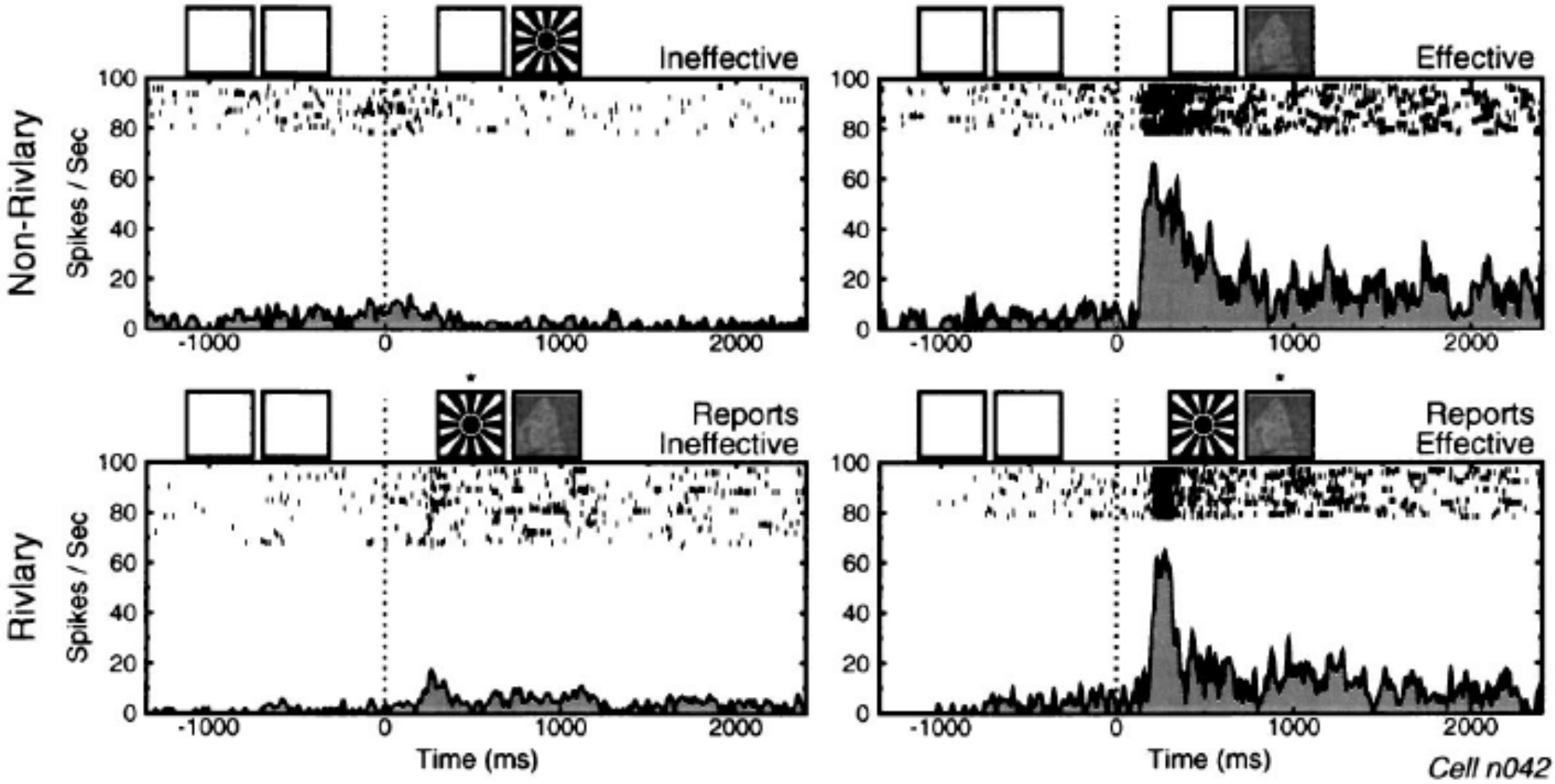
Fraction of modulating neurons, V1/V2 18%

Sheinberg & Logothetis (1997) activity correlating with rivalrous percepts in inferotemporal cortex [ref. 16]



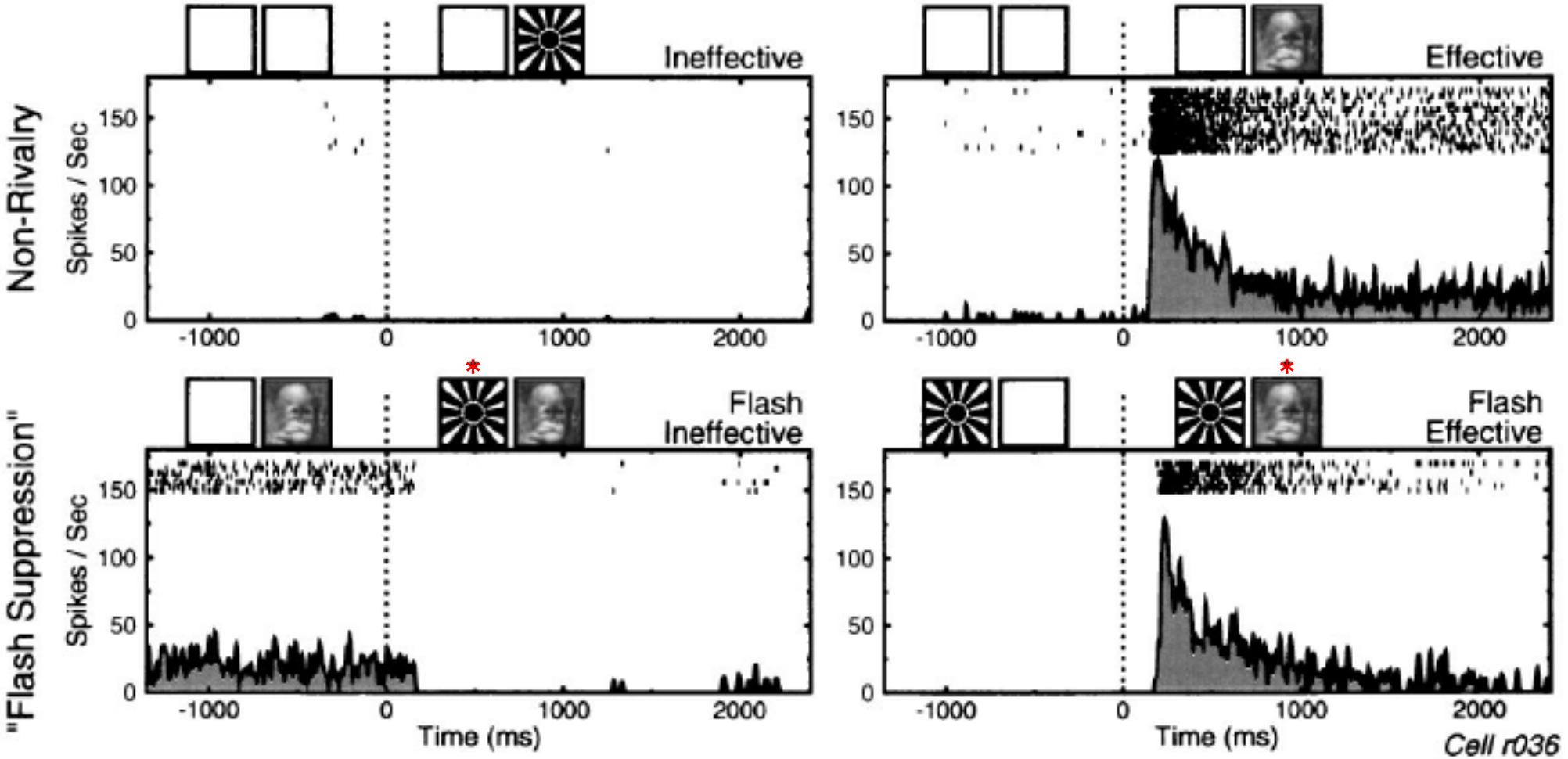
Fraction of modulating neurons, IT cortex 90%

Sheinberg & Logothetis (1997) activity correlating with rivalrous percepts in inferotemporal cortex [ref. 16]



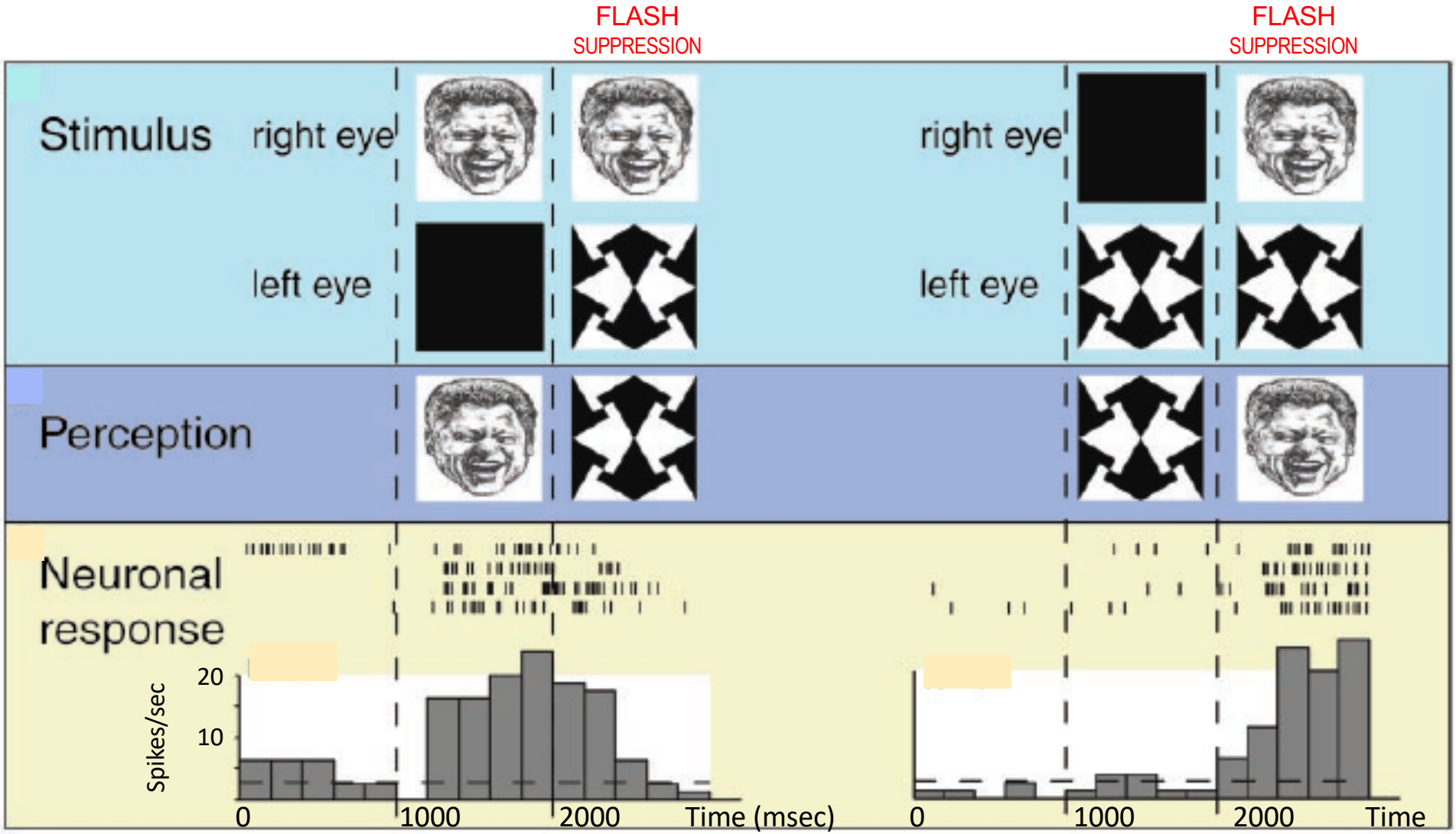
Fraction of modulating neurons, IT cortex 90%

Sheinberg & Logothetis (1997) activity correlating with rivalrous percepts in inferotemporal cortex [ref. 16]



Fraction of modulating neurons, IT cortex 90%

Kreiman, Koch *et al.* (2002) activity correlating with rivalrous percepts in *human* medial temporal lobe (hippocampus, amygdala, entorhinal cortex) [ref. 17]



Fraction of modulating neurons, human MTL cortex 70%

Summary of rivalry outcomes:-

% cell spiking activity, or fMRI BOLD signal, correlating with perceptual report

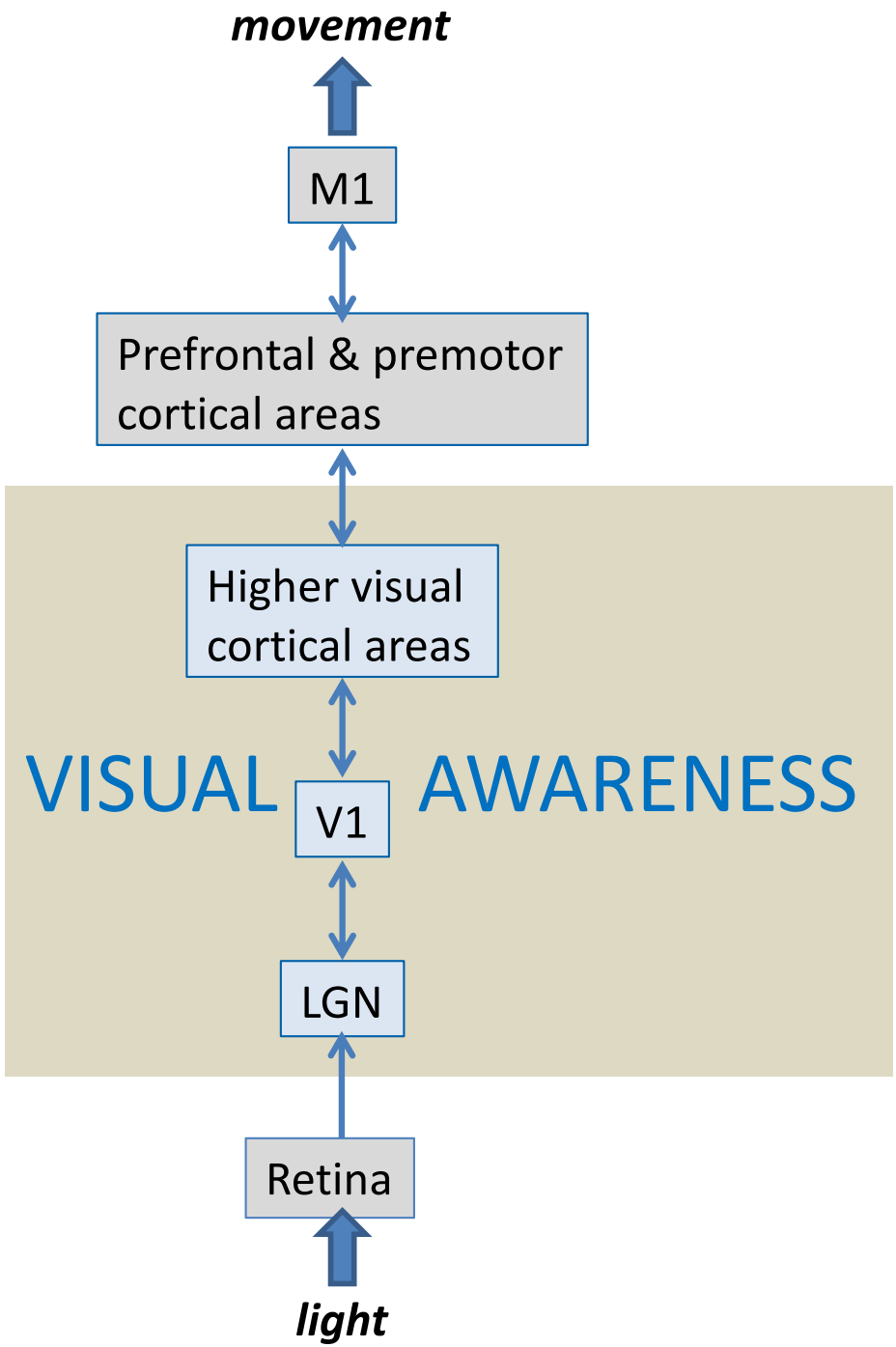
	BRAIN REGION	species	SINGLE UNIT (spiking activity)	fMRI (**BOLD** signal)
	LGN	M.m. & H.s.	0%	YES
	V1	M.m. & H.s.	18%	YES
	V2	M.m. & H.s.	18%	YES
	V4	M.m. & H.s.	39%	YES
Inferotemporal cortex	IT	M.m.	90%	
Medial Temporal Lobe	MTL	H.s.	70%	
Fusiform Face Area	FFA	H.s.		YES
Parahippocampal Place Area	PPA	H.s.		YES
	Trend ?		*Clear Hierarchical Trend*	*No Trend*

M.m. Macaca mulatta, rhesus monkey
H.s. Homo sapiens, human

Anatomical conception of NCC: summary

fMRI:
Binocular rivalry experiments imply that visual NCC extends from LGN to higher visual centres – i.e. includes all of visual system linked by reciprocal connectivity.

Neurophysiology:
Single neuron recordings under binocular rivalry imply an ascending gradient of significance for NCC activity.



‘Pattern of activity’ conception of NCC: re-entrant signalling & synchronisation of neural firing

fMRI:

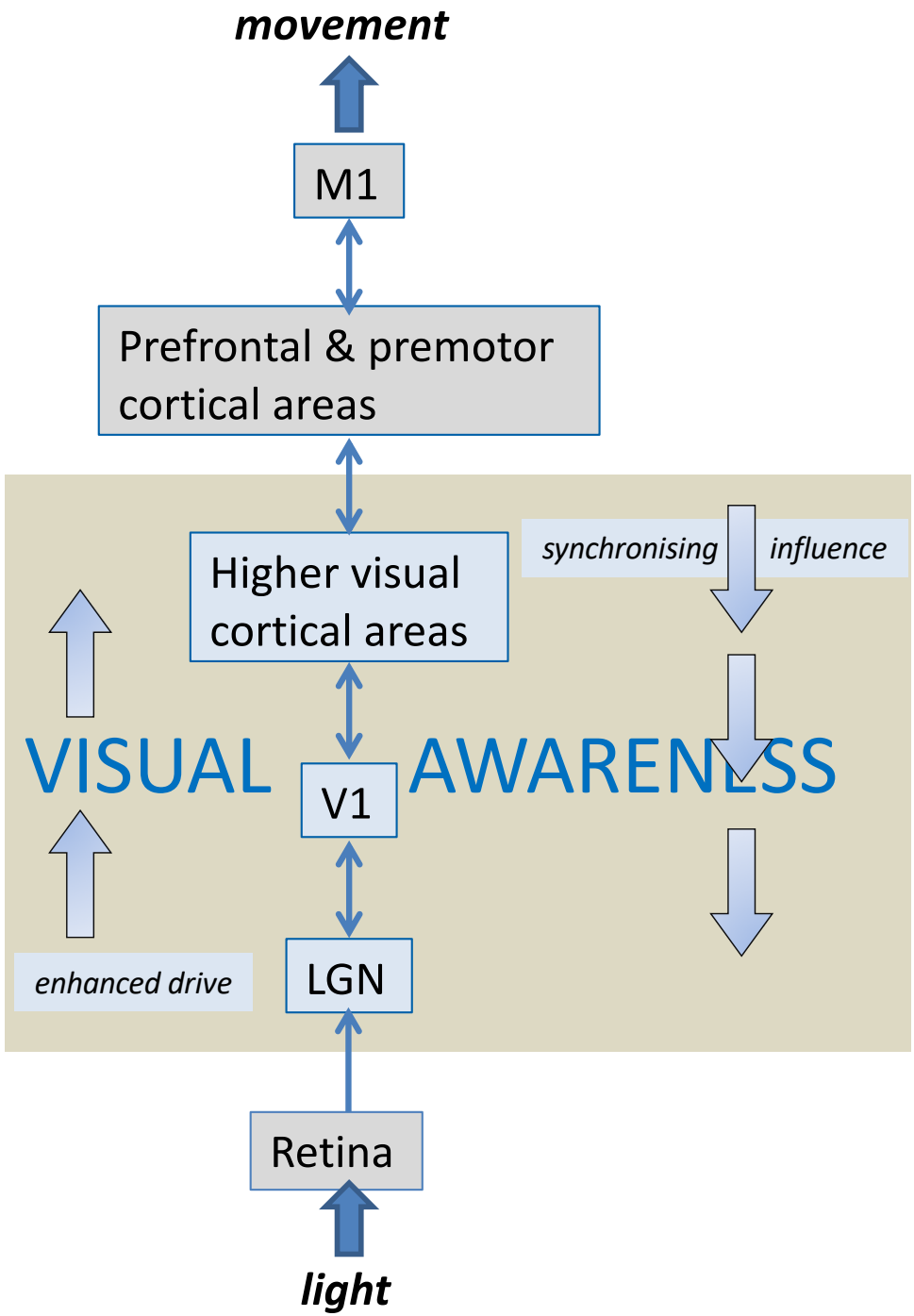
BOLD signal is influenced by all components of neural computation, not just spiking activity - i.e. includes subthreshold somatodendritic membrane potential changes, including inhibitory post-synaptic potentials.

Neurophysiology:

Such modulatory components of activation by descending pathways may act to synchronise neural firing, without changing rate of neural firing;

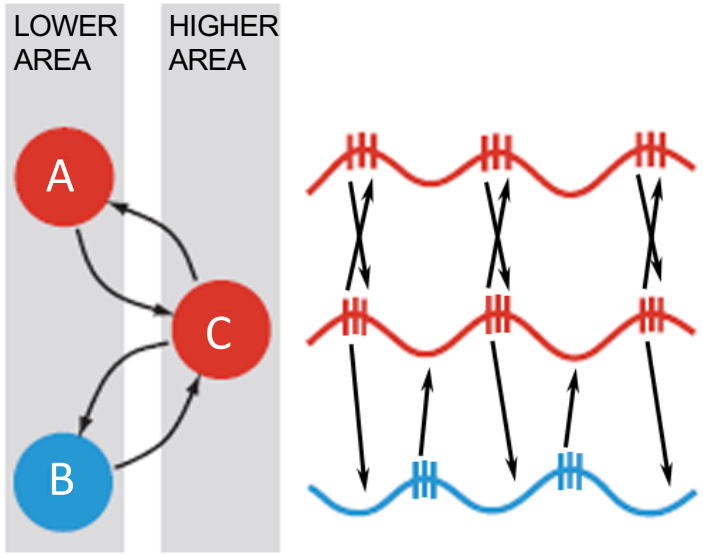
Synchronised neural activity in one group of neurons is more effective at driving other neurons, and propagating along neural pathways.

NB. *Predictive coding theory* envisages that backward pathways transmit both ‘predictions’, and ‘precision’. Precision is related to attention, and controls the gain of forward (error) signals. Hence precision is the component of backward signals that is more likely to exert a ‘synchronising influence’.



Gamma rhythm and synchronisation of activity: *'communication through coherence'* (Fries 2009)

Human EEG oscillatory rhythms:	
delta	0 - 4 Hz
theta	4 - 7 Hz
alpha	8 - 13 Hz
beta	14 - 30 Hz
gamma	30 - 60 Hz

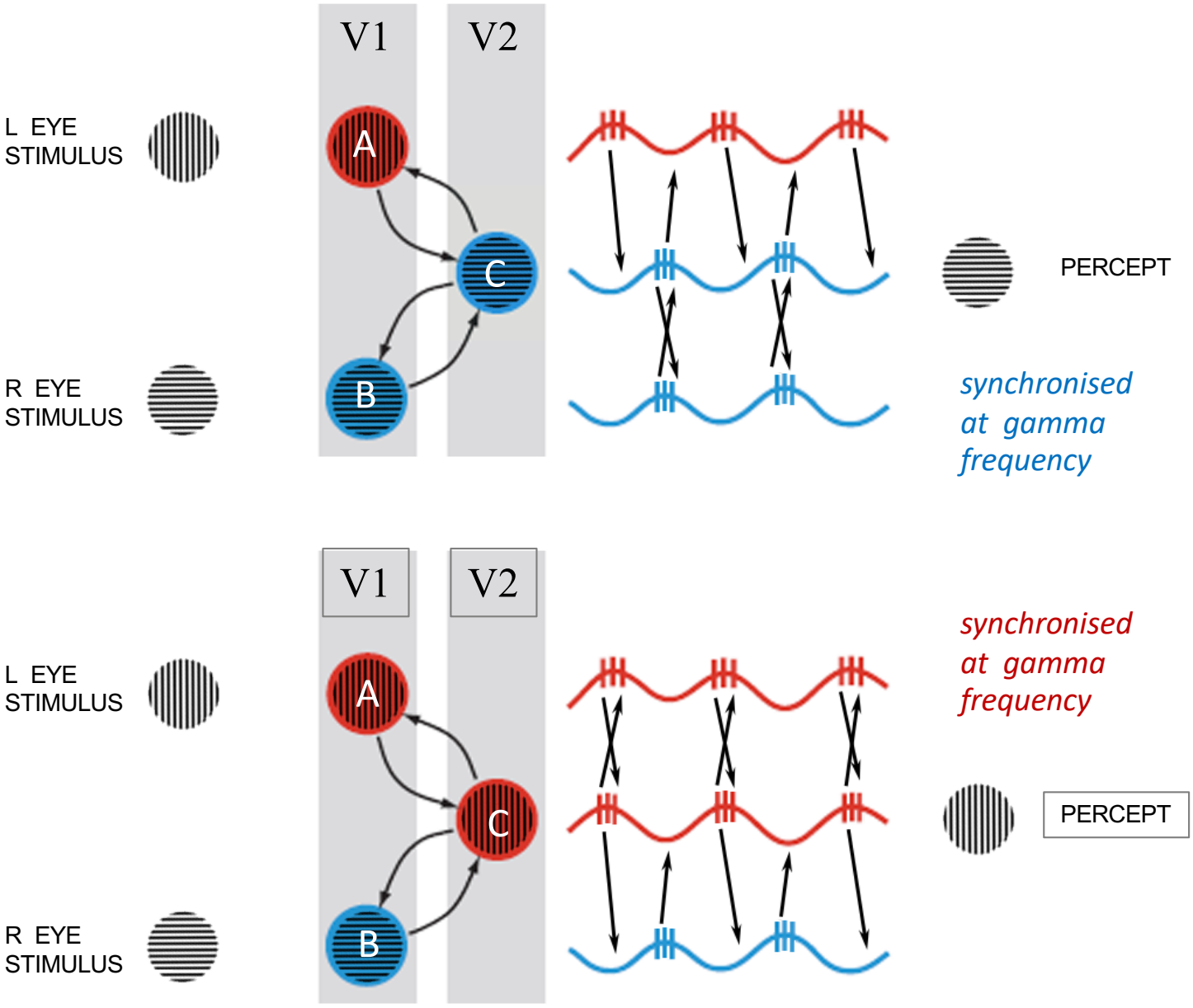


synchronised at gamma frequency

NB: in cerebral cortex, all long distance (inter-areal) connections are excitatory, and made by pyramidal cells; inhibitory connections are short range, and formed by a diverse variety of local inter-neurons.

Local cell networks (composed of excitatory & inhibitory neurons) exhibit an intrinsic gamma rhythm of excitability (known as the gamma cycle); transmission of signals from network A to network C is facilitated if their gamma cycles are in phase (hence spikes from A arrive at C when C is maximally excitable). Conversely, transmission from B to C is attenuated if B and C are out of phase.

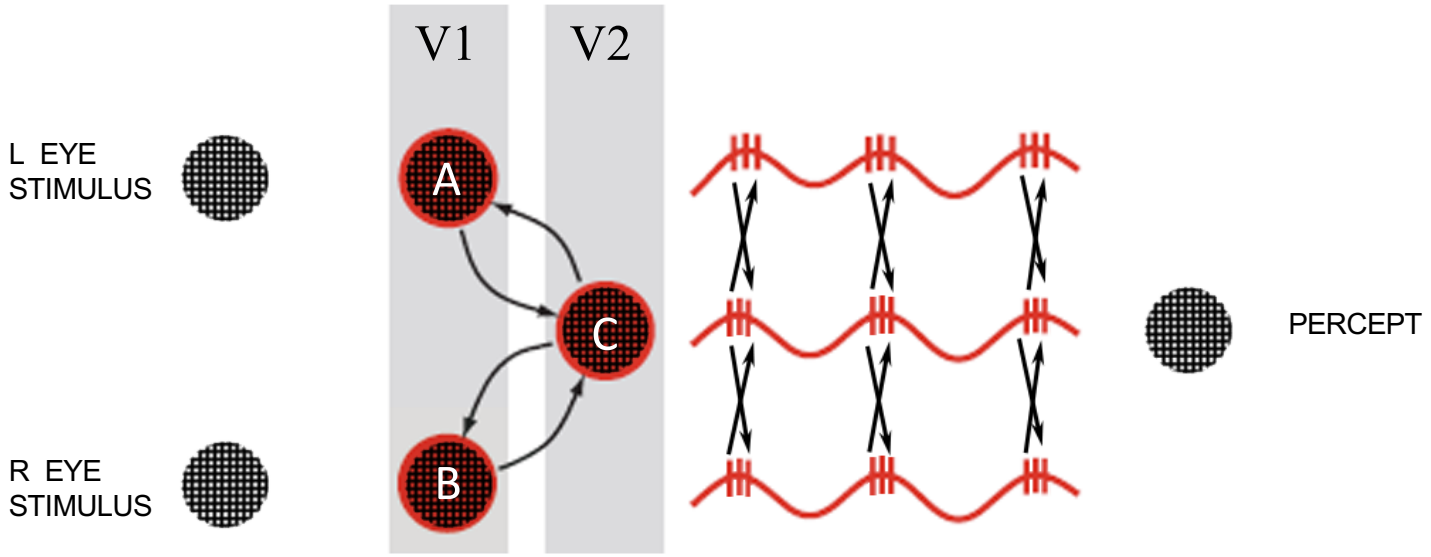
Gamma rhythm and synchronisation of activity (Fries 2009)



Fluctuating synchronicity of C with A, or C with B, could be one way of accounting for changes of perceptual dominance in binocular rivalry.

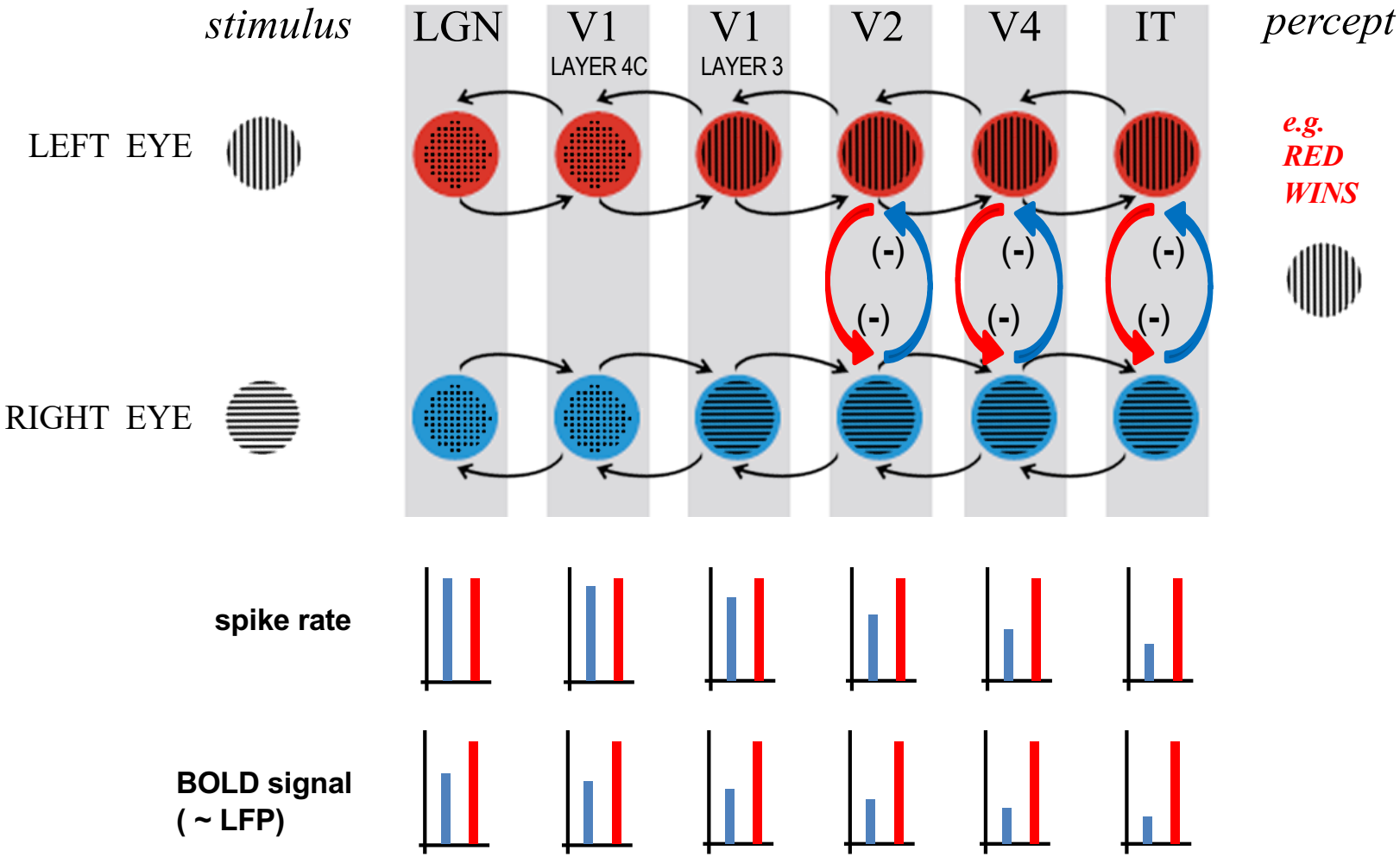
[NB. in this, highly schematic example, network C would comprise both vertical and horizontally tuned cells]

Gamma rhythm and synchronisation of activity (Fries 2009)



NB. Broader synchronicity between networks is the norm when both eyes are shown the same stimulus; dichoptic stimulation may act to induce desynchronisation between networks driven by (or selective for) dissimilar stimuli in either eye.

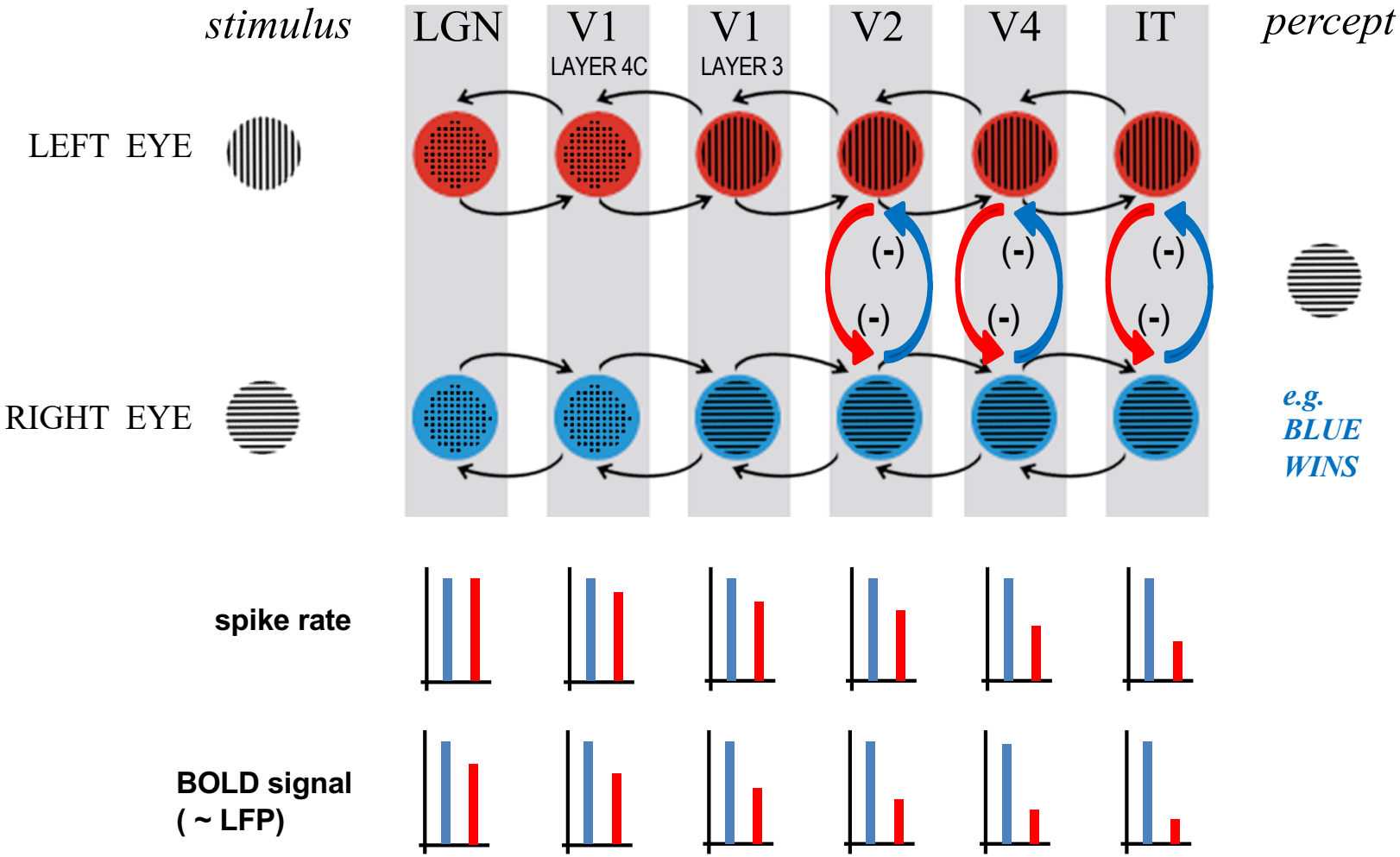
Models of trans-hierarchical synchronisation & rivalry



In this theoretical schematic example, dichoptic stimulation gives rise to two hierarchical chains, gamma synchronised at different phases to each other. The absence of synchronisation between the chains may minimise excitatory (mutually supportive) interactions between them, whilst enhancing inhibitory (mutually suppressive) interaction.
Here 'red' activity (representing the vertical grating) is dominant over 'blue' activity at levels hosting an inhibitory interaction (e.g. V2, V4 & IT). The balance of spiking activity is more equal in V1, and LGN, where absence (or lower incidence) of orientation selective cells implies an absence of selective inhibition between oppositely tuned neurons.

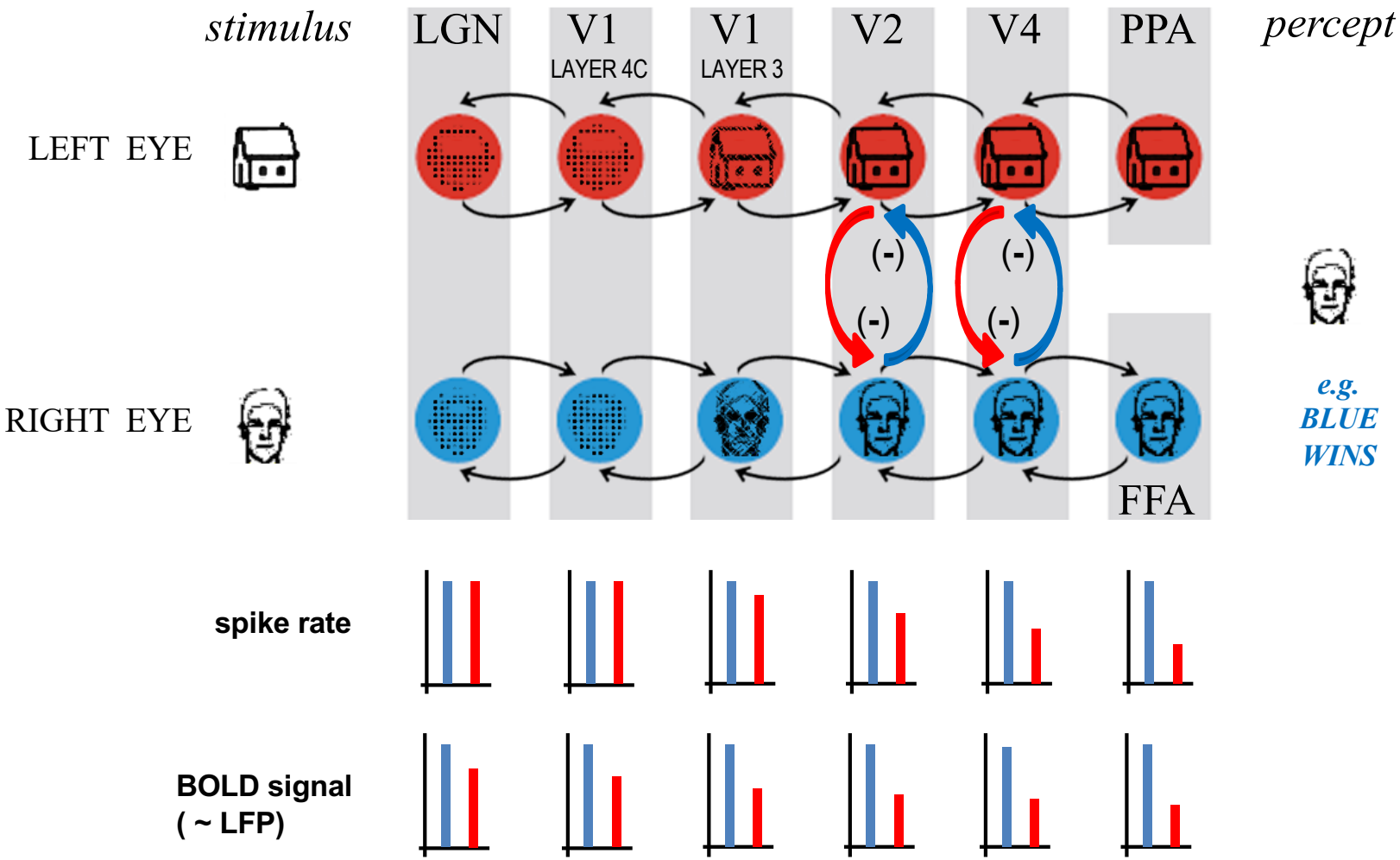
Explanation continues on next slide…

Models of trans-hierarchical synchronisation & rivalry



Here 'blue' activity (representing the horizontal grating) is dominant over 'red' activity at levels hosting an inhibitory interaction (e.g. V2, V4 & IT). The balance of spiking activity is more equal in V1, and LGN, where an absence of orientation selective cells implies an absence of selective inhibition between oppositely tuned neurons.
BOLD signal, by contrast, is representative of total synaptic activity, as opposed to spiking rate of active cells. This synaptic activity includes synapses driven by backward connections. Activity in the backward pathways should diminish while the chain is not perceptually dominant - hence BOLD signal may diminish at early stages of the non-dominant pathway, e.g. as as demonstrated for LGN and V1 (Haynes et al 2005).

Models of trans-hierarchical synchronisation & rivalry



This conjectural example uses face/house rivalry to address the situation where the two rival percepts involve different, separate areas toward the end of each chain - here FFA (fusiform face area) and PPA (parahippocampal place area).
It is unlikely that specific, mutually inhibitory, inter-areal connections exist between all possible pairs of 'end' areas that might be engaged by any given pair of stimuli chosen by an experimenter for dichoptic stimulation.
Hence the inhibitory interaction between the two chains is more likely to be instantiated by intrinsic connections within intermediate areas (e.g. V2, V4). This may have two consequences in the non-dominant chain: (a) reduced induction of BOLD signal over the backward pathway (as explained previously); (b) lesser drive of spiking activity, and BOLD signal (in FFA or PPA) over the forward pathway.

Summary

The NCC is/are “the minimal neuronal mechanisms jointly sufficient for any one specific conscious percept”.

‘Minimal mechanism’ may include:

- *which neurons* – specified by area, layer, & neural type (morphology, transmitter etc);
- *pattern of activity* – synchronisation and phase of oscillatory activity;
- *timing of activity* – forward going activity versus modulation contingent on recurrent/re-entrant feedback.

These ideas are unlikely to be mutually inconsistent: feedback circuits are probably essential to the mechanisms by which synchronised activity is established and propagated; neurons of different types (excitatory pyramidal relay neurons v. inhibitory interneurons), in different layers, play different key roles in forward and backward circuitry.

It is not a simple task to dissect the visual system into anatomical components and/or functional mechanisms that are, or are not part of the NCC !