Neural Correlates of Conscious Perception

The Role of Primary Visual Cortex in Visual Awareness

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List of abbreviations

2-AFC	2-alternative forced choice task
ARAS	Ascending reticular activation system
BEM	Boundary element model
BOLD	Blood oxygen level dependent
CRF	Contrast response function (physiological)
CRT	Cathode ray tube
CT	Computer tomography
CTF	Contrast transducer function (psychophysical)
ECD	Equivalent current dipole
EEG	Electroencephalography
EPSP	Excitatory postsynaptic potential
ERP	Event-related potential
FFA	Fusiform face area
FMRI	Functional magnetic resonance imaging
INUS	Insufficent but necessary part of an unnecessary but sufficient condition
IPSP	Inhibitory postsynaptic potential
ISI	Inter-stimulus interval
ICA	Independent component analysis
IT	Inferior temporal lobe
JND	Just noticeable difference
KO	Kinetic occipital area
LFP	Local field potential
LGN	Lateral geniculate nucleus
LO	Lateral occipital area
LOC	Lateral occipital complex
MEG	Magnetoencephalography
MRI	Magnetic resonance imaging
MUA	Multi unit activity
OFA	Occipital face area

PCA	Principal component analysis
PET	Positron emission tomography
PFs	Posterior fusiform gyrus
POS	Parieto-occipital sulcus
PPA	Parahippocampal place area
PVS	Persistent vegetative state
RF	Receptive field
SEM	Standard error of mean
SQUID	Superconducting quantum interference device
TMS	Transcranial magnetic stimulation
TvC	Threshold versus contrast function
VEP	Visual evoked potential
VFD	Visual field deficit

Preface

The study presented here is the attempt to shed light on an aspect of conscious perception that has often been conflated with other issues in the literature: *representation* of low-level dimensions of conscious perception. The question is not, what background conditions enable us to become aware of stimuli. Nor is the focus on processes that enable or prevent access to complex representations in visual cortex. Rather, the question is, where certain dimensions of conscious perception are represented in the brain. This question is of special importance, because it attempts to directly answer the age-old question, which brain areas encode which dimensions of our perceptual space. As such it can only be a starting point in a long series of experiments, and should be thought of more as an exemplification of a specific methodological and conceptual approach. The reader who is not interested in the general framework can confine himself to reading the last 3 chapters which present a study on perceived contrast that can stand alone as a contribution to the field of contrast perception. I chose to present the entire framework here – perhaps unusual for an experimental thesis – because it makes the models and assumptions underlying my research more transparent and summarizes my proposal how to scientifically attempt to bridge the gap between conscious perception and brain processes.

Several formal notes should be made in advance. First, the spelling of citations was used *exactly* as it appears in MEDLINE. This means, that German names may be occasionally spelt in an unusual way (e.g. ”Struber” instead of “Strüber” but “Goebel” instead of “Göbel”). This was done to increase the clarity for readers who wish to search for the articles in online databases. To maintain a common style, also the names of other German authors were “anglicised” according to common rules (“Koehler” instead of “Köhler”). Second, a considerable number of pilot experiments were performed in order to increase the signal to noise ratio of the evoked neuromagnetic signal, to optimise the trade-off between psychophysics and physiology and to calibrate the nonlinearities of the visual stimulation system. I decided not to present these pilot experiments here, simply because they in themselves do not directly contribute to the question posed in this study. On the other

hand some preliminary data from other studies I have performed with Prof. Jochen Braun and Prof. Hans-Jochen Heinze will be presented in the section on shapes and objects. This is “work in progress” and thus cannot be presented in full detail.

Before starting I would like to thank several people for their support. The academic climate at the universities of Bremen and Magdeburg has brought me in contact with numerous people who have greatly influenced this work. Prof. Gerhard Roth and Prof. Michael Stadler, the two reviewers, have strongly supported my academic development and have also presented me with an environment that has strongly shaped my ideas. Prof. Hans-Jochen Heinze has strongly supported my studies and has also provided me with an infrastructure without which the present study would never have been possible. Furthermore I wish to thank (without regard of role and title and in the order of appearance): Margarete Haynes, David Haynes, Daniel Strüber, Peter Kruse, Canan Basar-Eroglu, Günter Vetter, Manfred Stöckler, Sven Schütt, Harald Schmidt, Thomas Metzinger, Achim Stephan, Klaus Pawelzik, Uwe Opolka, Tillman Hagner, Claus Tempelmann, Max Hopf, Stefan Knape, Udo Ernst, Jochen Braun, Manfred Herrmann, Nina Nönnig, Claudia Grubich, Geraint Rees and Elliot Freeman. Some people may be surprised to find themselves on this list, but I believe this to be a good place to let them know that interaction with them has influenced me.

Abstract

This study investigates which neural populations represent low-level dimensions of conscious perception. First, a general framework is presented that will allow the separation of different aspects of the study of visual awareness. A set of six criteria is developed that allows one to assess whether a neural population could in principle represent a dimension of conscious perception. These criteria are then applied to previous studies on the neurophysiology and neuropsychology of conscious perception. It is demonstrated that the conscious perception of the dimensions of colour, motion and object identity is represented in extrastriate visual cortex in a modular fashion. Then it is demonstrated that currently available data indicate that brightness and perceived contrast are likely to be represented in primary visual cortex. In the following empirical section a study on the relationship between perceived contrast and activity in primary visual cortex is performed using a combination of EEG, MEG and psychophysics. The perceived contrast of flashed stimuli was measured and compared to synchronously recorded neuromagnetic responses. When a target grating is flashed into a larger, surrounding grating, its contrast was perceived to be lower when both gratings are oriented collinearly rather than orthogonally. This effect can be used to dissociate the physical and the perceived contrast of the target grating. Transient potentials and magnetic fields evoked by the flashed target gratings were recorded and compared to psychophysical judgements of perceived contrast. Both early (100 ms) and late (150 ms) transients were reduced in amplitude when targets were flashed into a collinear rather than orthogonal surround, mimicking the situation for perceived but not physical contrast. At all investigated contrast levels, the amplitudes of electrophysiological transients correlated better with perceived than with physical target contrast. This holds especially for the late transient. Source localisation indicated that the transients in question are likely to originate in primary visual cortex. The study presented here is the first ever to study perceptual constancy by recording psychophysics and physiological responses synchronously. The results identify the activity of primary visual cortex as the most likely neural basis of perceived contrast.

Chapter 1

An empirical framework for studying visual awareness

Introduction

Consider the picture in Fig. 1A. Simply observing its different features can demonstrate the complexity every study of visual awareness is confronted with. When focussing at a small spatial scale of analysis one can see local variations of brightness, hue and saturation. At an intermediate level of analysis one can see local elements grouped to surfaces such as the uniform texture of the hair. At a shape based level of processing different objects are segmented such as the eyes or the nose, each enclosed by contours. And at an object based level of processing one recognizes the entire spatial configuration shown as a female face. Although it is possible to attend preferentially to one level of analysis (say the object category) aspects of all other levels jointly contribute to the perceptual experience. The different levels can be emphasised by transforming the picture using a set of Gabor wavelets (Fig. 1B,C). This shows how this left image may be represented in simple cells in V1. Although the image is composed of isolated elements of varying orientation and contrast these are still spatially integrated into contours and the overall face shape is still recognisable. Again it is possible to focus on different levels of integration – the elements, the contours or the entire object – but our perception is jointly determined by these multiple levels of analysis.

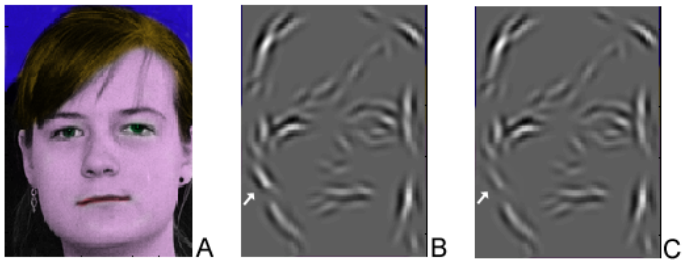

Fig. 1: (A) A chromatically distorted image of a female face. Figure B shows a transformation of this image that is obtained by only showing a Gabor wavelet with best matching orientation and contrast. Figure C is obtained from B by decreasing the contrast of one element (see arrows).

Many studies of visual awareness have focussed on the high-level aspect of this multilevel percept (Farah, 1994, 1995; Kanwisher, 2001; Logothetis, 1998; Milner &

Goodale, 1995; Rees & Lavie, 2001). It has been shown that the representation of different high-level features such as colour, motion or shape occurs in a modular fashion in different areas of the visual cortex. Perceptual deficits after brain lesions can be highly selective and affect specifically one subclass of high-level category, depending on where the lesions occur (Damasio, Yamada, Damasio, Corbett, & McKee, 1980; Farah, 1996; Farah, McMullen, & Meyer, 1991; Heywood, Wilson, & Cowey, 1987; Zihl, von Cramon, & Mai, 1983). Also brain imaging studies have revealed that similar areas are selectively activated when different feature classes are processed (Bartels & Zeki, 2000; Culham, He, Dukelow, & Verstraten, 2001; Grill-Spector, Kourtzi, & Kanwisher, 2001; Hadjikhani, Liu, Dale, Cavanagh, & Tootell, 1998). Some studies have demonstrated that this activity nicely correlates with perception (Grill-Spector, Kushnir, Hendler, & Malach, 2000; He, Cohen, & Hu, 1998). However little has been said about the conscious representation of the most elementary, local[1] features of such an image, such as brightness and contrast. Consider the difference between Fig. 1B and 1C: The contrast of one of the oriented elements composing the left cheek has been reduced. This difference can be readily perceived but it does not seem to have an effect on the perceived continuity of the contours or on recognition of the object as a face. Perception can change along the high-level dimensions and the low-level dimensions independently.

The key question in this study is which areas in the brain directly encode our conscious perception of these most elementary spatial features. Specifically the question will be if low-level visual dimensions of conscious perception such as brightness, contrast or even spatial patterns and contours are represented in primary visual cortex. This question was chosen for several reasons. First, there are controversial positions as to whether primary visual cortex could represent any dimension of conscious experience at all (Block, 1996; Crick & Koch, 1995; Koch & Braun, 1996a, 1996b; Lennie, 1998; Pollen, 1995; Rees, Kreiman, & Koch, 2002; Stoerig, 2001). However several recent results point towards a close correlation between V1 activity and conscious perception (Kosslyn et al., 1999; Macknik & Haglund, 1999; Macknik & Livingstone, 1998; Pascual-Leone & Walsh, 2001; Polonsky, Blake, Braun, & Heeger, 2000; Rossi, Rittenhouse, & Paradiso, 1996; Super, Spekreijse, & Lamme, 2001). Concentrating on feature dimensions that are more likely to be represented in extrastriate visual areas may have misled previous

authors to doubt that any feature dimension could be represented in V1. Second, of all visual areas primary visual cortex is that about which most is known. This will allow an analysis to build upon a large set of empirical data. Third, there is a strong advantage in studying dimensions of perceptual magnitude such as brightness and contrast. In contrast to dimensions such as colour hue or direction of motion perceived magnitude is believed to be represented by the mean activity of cortical cells. If this is the case then differences in perceived magnitude will result in absolute differences in spike rates in a population, whereas differences in say motion direction are more likely to be encoded by changes to population vectors without changing average activity (Treue, Hol, & Rauber, 2000). Because all current methods for extracranial recording of neural activity in humans (electroencephalography, magnetoencephalography, positron emission tomography, functional magnetic resonance imaging and near infrared spectroscopy) average across large populations of neurons and measure their mean level of activity, perceived magnitude is very suitable for correlation studies.

How do we proceed empirically in order to find out which brain areas *represent* certain feature dimensions of conscious perception[2] (such as brightness, contrast or hue)? A large number of paradigms have been considered to be relevant to visual awareness, but only a limited number can specifically contribute to this problem of representation. The implications of discrimination studies for visual awareness for example are not straightforward, because it is known that subjects can perform above chance on discrimination tasks without subjectively perceiving differences between stimuli (Kolb & Braun, 1995; Stoerig & Cowey, 1997). A first strategy could be to attempt to correlate our perceptual experience with neural processes. If we were interested in perceived brightness we could present a bright and a dark stimulus, and correlate the different perceptual states with the physiological state of the brain. One could assume that perceived brightness must be represented exactly where the brain responds differently to the two stimuli. However there is a severe problem with this approach: The resulting set of correlates is far too large. Even the luminance distribution on the CRT monitor correlates nicely with our perception, as does the graded receptor potential of retinal cones, the spike rate of cells in lateral geniculate nucleus, and also (if we let the subject press one of two buttons in response to either the dark or bright stimulus) the subpopulation of neurons in motor cortex executing

our behavioural response. Many of these correlates are not relevant. The correlating luminance distribution of the CRT monitor certainly does not represent perceived brightness. Not only because we believe that our brain states are sufficient to account for perceptual experiences, but also because the CRT monitor is not necessary for our perception of brightness. Brightness sensations can also be caused by exerting mechanical force on the eye. We can also exclude the retina from representing any visual feature because it is possible to evoke sensations by directly stimulating visual cortex and thus bypassing the retina as will be shown later in more detail (Brindley & Lewin, 1968). Thus, the CRT monitor and the retina cannot be necessary conditions of visual experiences. So simply searching for empirical correlation does not help. We are interested in the subset of correlates that are necessary for a perceptual experience to occur, rather than those that happen to be merely accidental.

A different strategy could be to search for necessary conditions of visual experiences. However the entire set of necessary conditions is again far too large because it includes all unspecific background conditions. It includes conditions that are necessary for any type of experience such as activity of the brainstem reticular formation producing *wakefulness* (Moruzzi & Magoun, 1949; Parvizi & Damasio, 2001). One of the meanings of the word "consciousness" relates to this necessary condition: being awake, aware of the environment and responsive, as opposed to sleeping or being in coma. However the requirement of wakefulness is far too general because it applies to experiences of every modality. A second necessary condition is *representation*. This refers to some property of a neural population (such as mean spike rate or phase coherency) that encodes a specific dimension of visual experience. This can be compared to a "dataset" that is able to encode every perceptually different state along a feature dimension by adopting a different state. A third necessary condition refers to our ability to *access* this representation. In clinical patients with neglect syndrome (Driver & Vuilleumier, 2001) or in certain experimental paradigms, such as attentional blink (Raymond, Shapiro, & Arnell, 1992), visual brain areas can have a highly complex representation of a stimulus without the subjects being aware of it (Rees et al., 2000; Vogel, Luck, & Shapiro, 1998). The rest of this chapter will present a brief review of the two main background conditions: wakefulness and access. The following chapters will be concerned only with the issue of representation. First formal and empirical criteria will be presented that will help

decide if a neural population can be said to represent a certain dimension of conscious experience. These criteria will be applied to high-level and low-level visual features separately. Then a study will be presented that aims at answering the question whether *perceived contrast* is encoded in primary visual cortex.

Background conditions: Wakefulness and access

Wakefulness

"Consciousness" in one of its meanings refers to a very unspecific state where the individual is awake, perceives, responds to the environment and shows at least rudimentary signs of cognitive processing. This requires wakefulness, a form of general arousal that affects all modalities alike. Originally wakefulness was believed to reflect activation by a unitary "ascending reticular activation system" (ARAS), projecting from the reticular formation of the brainstem to the thalamus and from there diffusely to cortex (Jasper, 1949; Moruzzi & Magoun, 1949)[3]. Subsequent research has revealed that the ARAS is not a unitary system but consists of a complex network originating from various functionally distinct brainstem nuclei with different neurotransmitters and projecting to both intralaminar and reticular nuclei of the thalamus, as well as the basal forebrain and directly to cortex (Parvizi & Damasio, 2001; Steriade, 1996; Steriade, McCormick, & Sejnowski, 1993).

Besides the brainstem reticular formation the thalamus also plays a major role in arousal. During sleep for example signal transfer through the thalamic lateral geniculate nucleus is reduced by about 50 % (Coenen & Vendrik, 1972) and cells switch from "transmission mode" to "burst mode" (McCormick & Bal, 1997). This greatly attenuates afferent signals reaching visual cortex via the LGN. Functional brain imaging studies show that a transition from conscious to unconscious in humans (such as during sleep, anaesthesia or clinically during the persistent vegetative state) is accompanied by global decreases of cerebral activity. The strongest decreases are observed in the brain stem and in the thalamus, which is in accord with the special role of reticular formation and thalamus (especially its reticular and intralaminar nuclei) in the control of arousal (Alkire, Haier, & Fallon, 2000; Bonhomme et al., 2001; Braun et al., 1997; Fiset et al., 1999; Hofle et al., 1997; Maquet, 1997, 2000)[4].

An important distinction has to be made between wakefulness and awareness. Wakefulness does not imply that subjects are aware of anything. Epilepsy patients for example can show all criteria of wakefulness despite being completely unconscious. During petit mal or "absence" epileptic seizures patients can be capable of complex automatic behaviour, which involves representing and responding to external events, in absence of any awareness (Young & Wijdicks, 1998)[5]. A similar distinction between wakefulness and awareness can be found in patients in the so-called persistent vegetative state (PVS). These have a normal sleep-wake cycle and open their eyes spontaneously during the wake phases (Jennett & Plum, 1972; Kinney, Korein, Panigrahy, Dikkes, & Goode, 1994; Kinney & Samuels, 1994; Laureys, Lemaire, Maquet, Phillips, & Franck, 1999; Zeman, 1997). However they show no sign of awareness of the environment, no sign of cognitive activity and no reproducible voluntary responses to external events. This syndrome has been termed "wakefulness without awareness" (Andrews et al. 1996). In PVS the brainstem typically remains intact, which explains why vegetative functions and sleep-wake cycles still function. However cortical processing is severely reduced, which points towards the importance of neocortical processing for awareness, as has been previously pointed out (Roth, 1994)[6].

In many clinical cases it is difficult to distinguish between unconscious but awake patients and patients who are fully conscious but unable to respond due to motor paralysis. These latter cases of "locked-in" syndrome occur most frequently after brainstem lesions at the level of the pons (Patterson & Grabois, 1986). Similar to PVS-patients locked-in patients exhibit wakefulness without responses to external stimuli, but they are consciously aware of their environment (Bauer, Gerstenbrand, & Rumpl, 1979; Boyce, 2000; Patterson & Grabois, 1986; Plum & Posner, 1980). These patients are unable to communicate with the external world due to a lack of motor efferents. Often the only method for communication is the use of vertical eye-movements and the upper eyelids[7], which can be used to communicate via Morse code and often reveal a near-to-normal cognitive status (Feldman, 1971). In rare cases even the voluntary control over eyelids and eye-movements is lost, a syndrome called "total locked-in" (Bauer et al., 1979). Because it is difficult to assess whether awake but unresponsive patients lack awareness of external events or lack the ability to

communicate (Bernat, 2002; Giacino et al., 2002; Menon et al., 1998; Schiff & Plum, 1999) many patients suffering from locked-in syndrome have been incorrectly diagnosed as having the vegetative state syndrome, despite consciously perceiving and processing their environment for many years (Andrews, Murphy, Munday, & Littlewood, 1996; Childs, Mercer, & Childs, 1993). This was only discovered after offering more adequate forms of communication that do not rely on eye movements (Andrews et al., 1996; Childs et al., 1993; Kmietowicz, 2000).

It was originally believed that the higher neocortical blood flow level of locked-in as compared to PVS patients could differentiate between the two groups (Levy et al., 1987). However several studies have provided evidence for activity in neocortical areas even in clear cases of persistent vegetative state (Laureys et al., 1999; Menon et al., 1998; Schiff et al., 2002). Most strikingly cortical areas typically related to high-level processing have shown to be activated in PVS patients as indicated by mismatch negativity and P300 evoked responses (Kotchoubey, Lang, Bostanov, & Birbaumer, 2002). Even high-level visual processing has been demonstrated in one PVS patient. Despite the complete lack of any sign of consciousness her EEG showed an auditory evoked P300 to oddball stimuli and when presented with face stimuli (Menon et al., 1998) she showed increased blood-flow in the right fusiform gyrus, the cortical locus of high-level face-processing (Haxby et al., 1994; Kanwisher, McDermott, & Chun, 1997; Puce, Allison, Gore, & McCarthy, 1995). This leaves one with a dilemma: Either one trusts the physiological data and believes that the patients are minimally aware because ventral visual areas show a high depth of visual processing. Or one trusts the lack of behavioural responses and believes that the rudimentary cortical activity in high-level visual areas is not sufficient for visual awareness. The decision as to whether a patient is believed to be aware but unresponsive or completely unaware has immense consequences. Life supporting systems can be withdrawn from a patient who is unaware, whereas this is not the case for conscious patients. This has lead to methodological and ethical debates[8], which demonstrates that a clear physiological index of awareness is of greatest clinical relevance.

Access

Some patients who are clearly awake, responsive and have no sensory deficits can nonetheless fail to become aware of certain external stimuli. This failure of access mostly occurs in patients with unilateral brain damages to the inferior parietal lobe (especially right-hemispheric lesions) and has been termed the neglect syndrome (reviewed in Driver & Vuilleumier, 2001). Neglect patients fail to notice stimuli spontaneously if they are presented in the visual field contralateral to the side of the lesion. However they can perceive them when explicitly cued to their position[9].

Neglect is a prime example of a dissociation between the perceptual representation of a stimulus and its accessibility. It is not due to an impairment in the depth of stimulus processing and representation but to a postperceptual lack of access. This is demonstrated by a number of findings. Whether a stimulus is perceived or not does not depend only on its retinotopic coordinates. The neglected region of visual space can be modulated by direction of gaze, so that a retinotopic position from which information was previously inaccessible becomes accessible (Nadeau & Heilman, 1991). Turning of the trunk without change in the position of the stimulus in retinotopic space can also counteract neglect (Nadeau & Heilman, 1991). Also, the impairment does not abruptly stop at the vertical meridian (as would be expected if it were a failure of selection from the damaged hemisphere), but shows a gradual, continuous decrease (Pouget & Driver, 2000).

Further evidence that neglect is a failure of selection comes from a phenomenon called “extinction”. In some patients stimuli presented in isolation to the contralesional visual field are readily perceived and only fail to reach awareness when presented together with a stimulus on the normal side. In these cases a “conflict” occurs and only the stimulus on the unaffected side is perceived, whereas the other stimulus appears to be extinguished. The failure to reach awareness also depends on high-level attributes of the stimulus such as object category. A fear-related stimulus is less likely to be extinguished than a neutral stimulus (Vuilleumier & Schwartz, 2001), suggesting that the extinguished stimulus is processed up to the level of object recognition. Evidence for this also comes from brain imaging studies showing that extinguished object stimuli nonetheless specifically activate object-selective modules

in inferior temporal cortex (Driver, Vuilleumier, Eimer, & Rees, 2001; Rees et al., 2000). Further evidence for extensive sensory processing in neglect is that extinction can be counteracted by perceptual grouping processes (Gilchrist, Humphreys, & Riddoch, 1996; Ward, Goodrich, & Driver, 2001), as demonstrated by the fact that extinction of bisected lines depended strongly on whether the two lines could be perceptually grouped (Mattingley, Davis, & Driver, 1997). Failures of accessibility have also been demonstrated in other cases. Patient D.F. of Milner and coworkers was unable to consciously access shape information after lesions to the ventral visual stream (Milner et al., 1991). However she could use shape information to guide actions of her hand. This again demonstrates a dissociation between representation and availability for awareness.

Deficits of accessibility can also be studied in normal subjects using experimental paradigms such as "inattentional blindness" (Mack & Rock, 1998; Simons, 2000), "change blindness" (Rensink, 2002; Simons & Levin, 1997) and "attentional blink" (Raymond et al., 1992). In inattentional blindness a subject does not become aware of a stimulus when it is presented unexpectedly, spatial attention is deployed elsewhere in the visual field and the event does not lead to attentional capture. In these cases the target is often not perceived (Mack & Rock, 1998; Simons, 2000). This has been studied using low-level visual stimuli (Mack & Rock, 1998) as well as natural image sequences (Fig. 2)(Simons & Chabris, 1999) and has been taken as a prime example that attention is the gatekeeper to visual awareness.

In change blindness a subject does not become aware of a small change between two different presentations of a visual image, despite explicit expectation and knowledge that something does change (Rensink, 2002; Simons & Levin, 1997). Interestingly an fMRI study has shown that even an undetected change can nonetheless be unconsciously registered by ventral stream visual areas, suggesting that object processing is available up to the degree of change detection (Beck, Rees, Frith, & Lavie, 2001).

In the attentional blink paradigm subjects are presented with rapid sequences of stimuli, some of which contain potential targets and have to be responded to (Raymond et al., 1992). If a target is followed within short time (400-600 ms) by a

second target the probability that the latter is detected is decreased, a phenomenon termed “attentional blink”. This can clearly not be due to sensory degradation by forward or backward masking effects, because the first target is easily perceived. Evidence from visual evoked potentials shows that the locus of selection is likely to be “postperceptual”. Components indicating both early sensory processing (P1, N1) and semantic analysis (N400) are not attenuated, whereas the P300 component is completely suppressed (Luck, Vogel, & Shapiro, 1996; Vogel et al., 1998).

Fig. 2: The famous “monkey-event” used to study inattentional blindness: Subjects view a video in which two teams play basketball. They are instructed to count the number of passes. After about 45 seconds a person wearing a gorilla suit walks through the scene. 73 % of subjects engaged in the counting task fail to notice the “gorilla event” (Simons, 2000; Simons & Chabris, 1999).

It is important to notice that accessibility is a more general requirement than selective attention. If subjects perform a complex foveal feature discrimination task leading to a complete engagement of selective attention at the point of fixation they can nonetheless perceive (detect) target stimuli outside the focus of attention if they are expected to occur, despite partly losing information on their spatial properties (Lee, Koch, & Braun, 1997). Similarly it has been demonstrated that the perceived brightness of stimuli does not change under conditions of inattention (Prinzmetal, Nwachuku, Bodanski, Blumenfeld, & Shimizu, 1997). On the one hand this questions whether the crucial variable in inattentional blindness is really attention, or possibly rather expectation (Braun, 2001). But it also suggests that selective attention is not a necessary condition for awareness, simply because we are often aware of stimuli that fall outside our focus of attention.

Chapter 2

Formal and empirical criteria

Empirical criteria for conscious representation

If one is interested in the question of representation it is important to ensure that one is really studying representation and not access. As shown in the previous chapter given that a subject is awake his failure to become aware of a stimulus can be due to problems of representation or of access. The brain can have very complex representations of a stimulus despite one's being unaware of it. In order to empirically assess whether a brain area is involved in representing a dimension of conscious perception rather than in accessing it, it is necessary to show that this area responds to small perceivable differences along that dimension with different states. Otherwise the area cannot be said to have a "representation", simply because we cannot explain differences in perception by different states of this area. For example changes in the physical contrast of stimuli lead to changes in their perceived contrast. Activity in parietal and frontal visual areas does not correlate with changes in physical contrast, but in several early visual areas it does (Boynton, Demb, Glover, & Heeger, 1999). This means that parietal and frontal areas cannot represent the dimension of perceived contrast. Representation requires that certain mapping requirements on the co-occurrence of perceptual and neural states be fulfilled. Furthermore it is important to separate necessary from accidental correlations between perceptual and neural states. In what follows a simple heuristics is followed. If a property of a neural population N is to represent a dimension of conscious perception Q then we have to assess both, that it fulfils certain mapping requirements so that every different qualitative state can actually be represented and that it is a necessary condition for a percept of the class Q (Fig. 3).

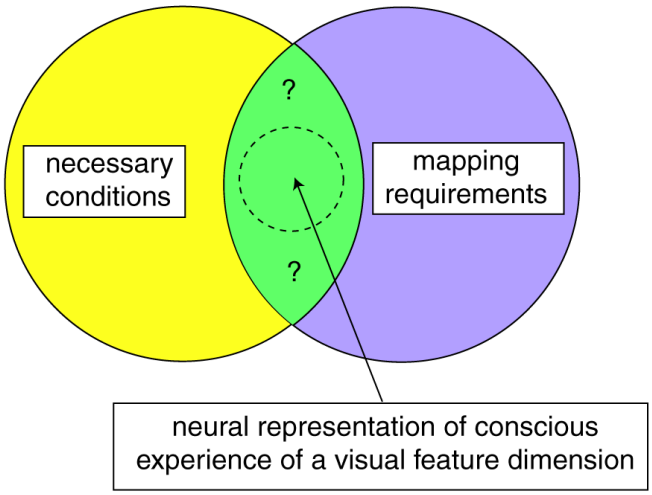

Fig. 3: At least two criteria will have to be fulfilled in order to support a claim that a property of a neural population represents a dimension of conscious perception. On the one hand activity in that neural population has to be a necessary condition for percepts of this type. On the other hand certain mapping requirements will have to be fulfilled (see below), so that the neural population can be said to "represent" a dimension of conscious perception. The union of the neural processes that fulfil both criteria certainly includes the neural representation of a dimension of conscious perception. It could possibly also include other neural processes (indicated by the question-marks), which also fulfil both criteria, but it will provide a first framework to discard a large number of neural processes that certainly do not fulfil these requirements.

Mapping requirements for representation

In most previous studies the mapping criterion employed was simple "correlation", which was used rather intuitively. The neural population representing a dimension of conscious experience was called a "neural correlate of consciousness" (Block, 1996; Chalmers, 1996; Crick & Koch, 1995, 1998). However correlation as an empirical criterion is too weak. In the statistical sense correlation suggests simply that some part of the variance of the neural state can be explained by the variance in the qualitative state (and vice versa because correlation is a symmetric measure). On the other hand requiring a perfect correlation as criterion is too strong because it pre-supposes a linear relationship between the neural and qualitative dimensions. If we want to speak of a "representation" we will have different requirements. Ideally one should be able to infer the exact qualitative state from the neural representation. On the one hand we want the same qualitative state to always lead to the same neural representation. At

the same time we do not want two qualitative states to share the same representation. This can be cast in more formal terms that mathematically define an "injective function". Let Q be the set of all perceivably different percepts (qualia) with respect to a certain feature dimension. Let N be the set of possible states of a certain neural population[10].

The function f: Q → N mapping qualitative states onto neural states can only be a representation of Q in N if the following three requirements are fulfilled:

1. Totality: $\forall q \in Q : \exists n \in N : f(q) = n$

2. Single-valuedness $\forall q_1, q_2 \in Q : q_1 = q_2 \Rightarrow f(q_1) = f(q_2)$

3. Injectivity: $\forall q_1, q_2 \in Q : q_1 \neq q_2 \Rightarrow f(q_1) \neq f(q_2)$

The first two conditions ensure that we can actually speak of a "function", which is defined as a subclass of all relations $R \subseteq Q \times N$ that fulfil these two criteria. The first ensures that for every perceptual state there is a neural state assigned to it. The second condition means that only one neural state is assigned to every perceptual state. When conditions 1 and 2 are met then exactly one neural state will be assigned to each perceptual state. Condition 3 is necessary to ensure that the same neural state does not "represent" two different perceptual states (Fig. 4). However we can allow for some neural states not to represent any perceptual state, which means that we do not have to require our function to be surjective[11]. Note that these three conditions are necessary but not sufficient for the neural population N to represent dimension Q. They formulate a minimal requirement for the mapping.

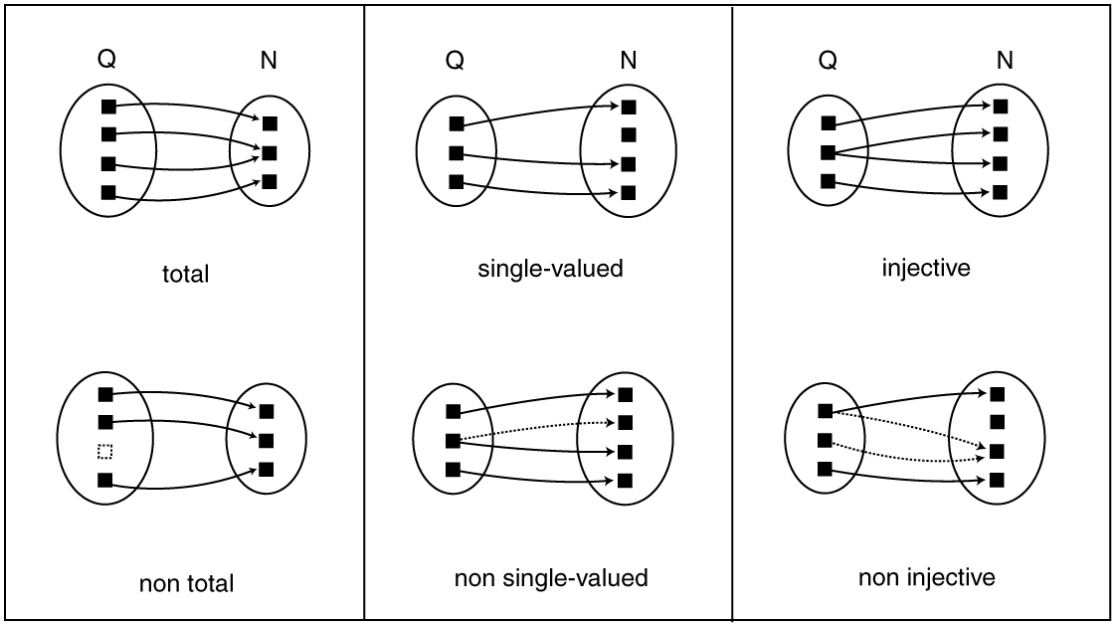

Fig. 4: Top row: Formal mapping criteria for the co-occurrence of qualitative states and neural states that have to be fulfilled if the state of a neural population N is to represent a qualitative state Q. Totality (left) refers to the fact that a neural state is assigned to every qualitative state. Single-valuedness (middle) refers to the fact that the same qualitative state cannot be assigned to two different neural states. Injectivity (right) means that the same neural state cannot represent two different qualitative states. Bottom row: Violations of the formal mapping criteria (dotted lines) can occur as failures of totality, for example when one qualitative state does not have a neural state assigned to it (left), of single-valuedness, if a qualitative state is assigned to more than one neural state, and of injectivity, if two qualitative states are assigned to the same neural state.

These three formal mapping criteria are rather abstract, so the next step is to ask for empirical ways to test these criteria. The first criterion (totality) is easy to meet, because it only means that the state of the neural system has to be defined whenever a conscious perception occurs. This is independent of our knowledge of this state and thus does not require that we have actually measured it[12]. The criteria of single-valuedness and injectivity however will require more extensive discussion.

Single-valuedness

The requirement of single-valuedness can be empirically tested by examining the state of the neural population for repeated occurrences of the same qualitative state. Testing for single-valuedness can be done in different ways. For some features this can be studied by matching paradigms where a feature is modulated by its context. For

example perceived colour hue of a region in the visual field is influenced by its chromatic context (Judd, 1940; Land, 1959a, 1959b). This is a highly useful feature, which enables us to discard for the spectral composition of the illuminating light by taking into account the spectral statistics of the context. Under different illumination conditions stimuli with very different spectra can be perceived to have the same colour hue. This can be exploited to test for single-valuedness. If the state of a neural system is to represent the perceived colour hue then it will have to show the same response to these stimuli that are physically different, but perceived to be the same. This is a very powerful paradigm to test whether a representation is based on physical or perceptual properties because it allows dissociating the two. The same logic will be applied to develop the empirical paradigm for contrast perception used in this study.

For high-level features single-valuedness can be studied using invariance paradigms. High-level features such as motion or object identity can be defined by different sets of low-level features (Fig. 5). The same shapes and motion patterns can be defined by contrasts of luminance, colour, contrast, texture or even direction of motion. Single-valuedness is fulfilled if a neural population responds invariantly of the different low-level realisations of the complex feature (Fig. 6).

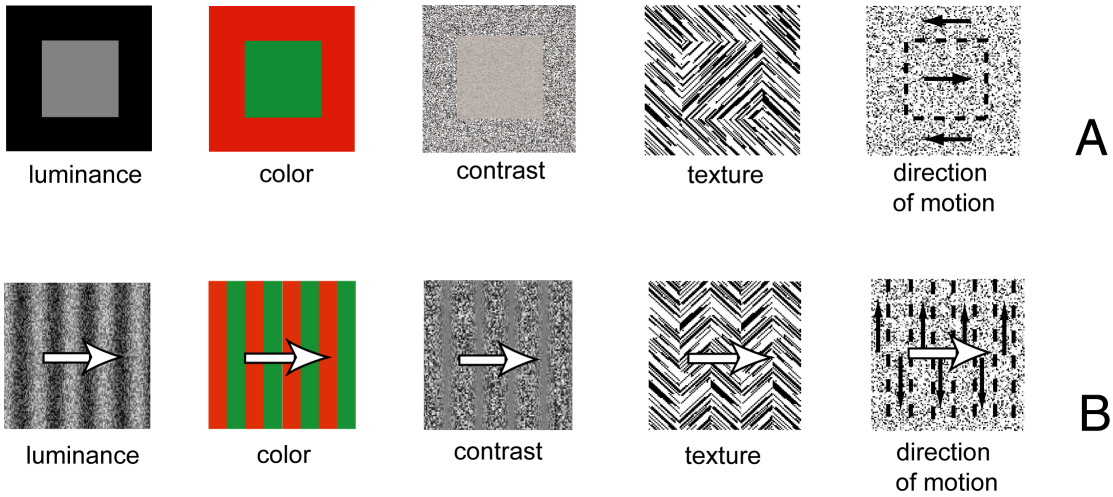

Fig. 5: Cue invariance of shape (A) and motion processing (B). Borders between areas of different luminance, colour, contrast, texture and direction of motion can be used to evoke highly similar shape or motion percepts. The stimuli in the bottom row are generated by movement of the local borders.

Empirical criterion of single-valuedness

The mapping of a perceptual dimension to states of a neural population can justifiably be assumed to be single-valued (according to the best currently available scientific knowledge) if in every case studied so far identical perceptual states were mapped to identical neural states.

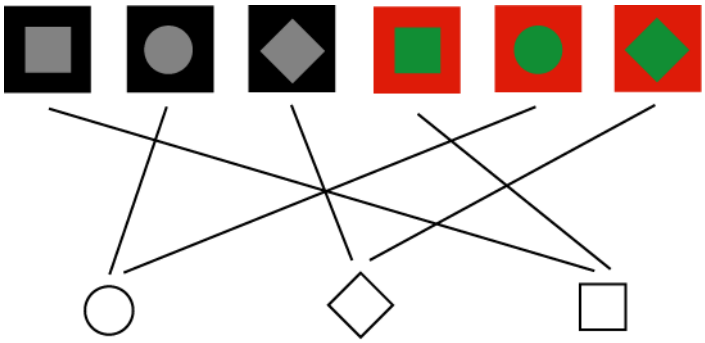

Fig. 6: In order to fulfil the criterion of single-valuedness, repeated occurrences of the same perceived object shape defined by different low-level cues (here luminance and colour contrast) should lead to the same neural activation vector in a population of neurons representing shape (indicated by the shapes in the bottom row). The representation should be invariant with respect to the low-level visual features.

Injectivity

Injectivity requires that every perceptual state be mapped to a different neural state. This is hard to assess for the entire set of perceptual states, but certain empirical approaches allow one to at least falsify this by testing for two consequences of injectivity. First, if injectivity holds, the state of the neural system will change for every change in the perceptual state. This will be termed “covariance”. Second, if injectivity holds, the neural system will have the resolution or grain to provide a different neural state for each perceptual state. This will be termed “grain”. Note that these two criteria are necessary but not sufficient for injectivity. This means that we can only falsify injectivity by testing for these criteria. If covariance, grain or both conditions fail then the mapping is not injective and thus a candidate neural population cannot represent that perceptual dimension.

Empirical criterion of injectivity

The mapping of a certain perceptual dimension to states of a neural population can justifiably be assumed to be injective (according to the best currently available scientific knowledge) if in every case studied so far (1) the state of the neural population changes when the perceptual state changes ("covariance") and (2) the resolution of responses in the neural population is sufficient to account for all different perceptual states ("grain").

Covariance

Intuitively this condition ensures that the neural population is not an unspecific background condition of any type of perception (such as wakefulness), but actually changes with our specific feature. In most studies this criterion is the only one studied at all. If a percept is only varied along a single feature dimension necessary background conditions such as wakefulness or attentional selection will remain constant. A large number of paradigms, each with specific strengths and weaknesses allow one to assess covariance.

Covariation with stimulus features

The easiest way to study perceptual covariance is to selectively stimulate the visual system with stimuli changing along only one feature dimension. As in the example above, one could present stimuli with varying luminance or contrast and record which parts of the visual system also change their response properties. Although this will yield a rather large set of correlates, including early sensory processing stages that are not directly related to conscious representation, it can nonetheless be used to rule out certain candidates, namely those that fail to correlate with the specific feature dimension. For example neurons in inferior temporal cortex respond strongly to different shapes of stimuli, but are largely invariant as to the local features by which these are defined (Sary, Vogels, Kovacs, & Orban, 1995; Sary, Vogels, & Orban, 1993; Vogels & Orban, 1996). These inferior temporal neurons cannot represent low-level visual features, because they do not covary with them. Selective stimulation is very suitable for studies of high-level visual representation. The strategy here is to change only the high-level properties of the stimuli and at the same time keep the low-level feature statistics constant[13], as is done in studies of representation of objects (Grill-Spector et al., 2001).

Perceptual changes without changes in stimulation

Covariance can also be studied in situations where perception changes but stimulation does not. One paradigm that follows this strategy is *multistable perception*, which has historically been considered one of the most important paradigms for visual awareness (Logothetis, 1998). Reversible figures (reviewed in Kruse & Stadler, 1995) and binocular rivalry stimuli (Leopold & Logothetis, 1999; Levelt, 1965; Wheatstone, 1838) have the property of leading to changes between different perceptual interpretations despite constant stimulation. These stimuli have the advantage that all exogenous, early sensory processes remain in a steady state due to the constant stimulation. If perception changes, activity in early areas that do not participate in representation will stay constant whereas areas encoding the percept will change their activity. Multistable stimuli can help to narrow down the candidates for perceptual representation by giving a hint at the earliest point at which a visual process correlates with a perceptual change.

However one has to be careful when interpreting perceptual alternations during multistable perception. Reversible figures, such as the Necker cube (Necker, 1832) have the problem that the perceptual change occurs for a high-level feature (such as 3D perspective) but the elementary, local spatial pattern does not change (Fig. 7, left). So perceptual reversals in reversible figures can only shed light on perceptual representation of high-level visual features. With binocular rivalry the situation is even more difficult. The perceptual change occurs at many feature levels in parallel, such as the object category and the local spatial pattern (Fig. 7, right). Theoretically this can be overcome when the two stimuli differ along a simple visual dimension (brightness or contrast) rather than object category (Polonsky et al., 2000). Multistable stimuli also have the caveat that it is difficult to assess whether a perceptual change was due to a change in a perceptual representation, or due to a change in the *access* of a perceptual representation[14].

Fig. 7: The left shows perceptual changes when viewing the Necker cube. A change occurs in 3D perspective but the perceived local brightness pattern remains identical. The right shows perceptual changes during binocular rivalry. Here both the high-level features (monkey versus sun) and the local spatial patterns change. Simply finding a neuron that follows the perceptual changes in binocular rivalry will not say if this neuron represents a high or a low-level visual feature.

Visual masking

One method to study a graded change in a perceptual dimension without changing the stimulus with respect to that dimension is visual masking. Masking of a target occurs when it is presented in close spatial or temporal vicinity of a second stimulus. In *backwards pattern masking* a target is followed after a short time by a mask that is presented to the same position in the visual field and is typically some form of noise pattern. Variation of the time between the two stimuli can change perceivability of the target from complete invisibility to complete visibility (Fig. 8). Using this method it has been demonstrated that activity in human object processing areas exactly follows the perceptual threshold. In *metacontrast masking* (Breitmeyer & Ogmen, 2000), where the target and mask only share a contour, the target remains visible but its perceptual representation is degraded with respect to certain perceptual dimensions. The perceived brightness can be reduced (Bridgeman & Leff, 1979) or the spatial extent of the stimulus can be misjudged (Macknik & Livingstone, 1998).

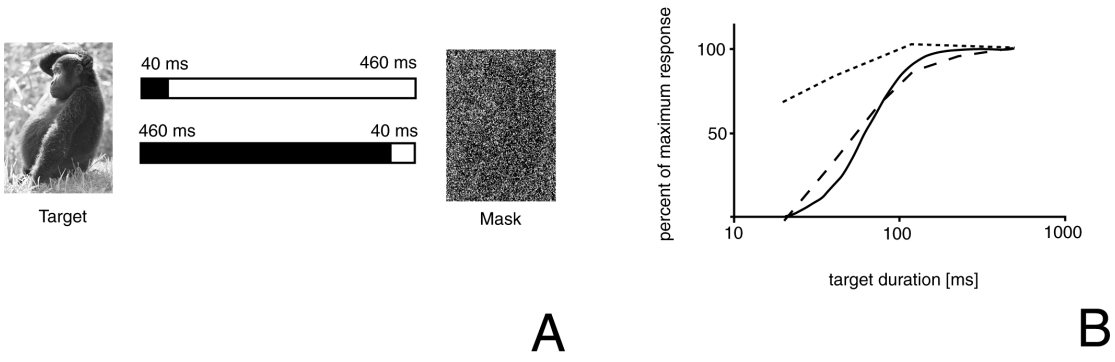

Fig. 8: (A) Stimulus design used for backwards masking of object recognition (Grill-Spector et al., 2000). The target is presented for a variable duration between 20 and 500 ms and followed by a mask. Recognition performance increases with target duration in a way shown by the solid line in (B). The dashed and dotted lines in B show the responses of visual areas lateral occipital (LO) and V1 respectively. LO which is the major cortical site of human object processing (Grill-Spector et al., 2001), correlates closely with the perceptual threshold.

Direct cortical stimulation

Direct cortical stimulation can also be used to provide evidence for covariance. If we activate a neural population by directly stimulating it, we can set the starting point of a causal chain directly and thus observe the effects of a change in neural state on the perceptual dimension of interest. It has been known since the earliest studies (Foerster, 1929; Krause, 1924; Penfield & Rasmussen, 1950) that electrical stimulation of occipital visual areas during surgical operations can elicit visual hallucinations ranging from simple colourless points of light, so called “phosphenes”, to complex hallucinations of motion, colour hues and objects (coloured balls, butterflies, human figures), depending on the site of stimulation. However these reports were restricted by the limited time available during neurosurgery. Brindley and Lewin (1968) performed the first extensive study on a 52-year-old patient who was blind after loss of major parts of both retinae. They implanted a prosthesis consisting of 80 platinum electrodes that spanned the medial wall covering the parts of the cuneus and lingual gyrus directly surrounding the calcarine sulcus (Fig. 9).

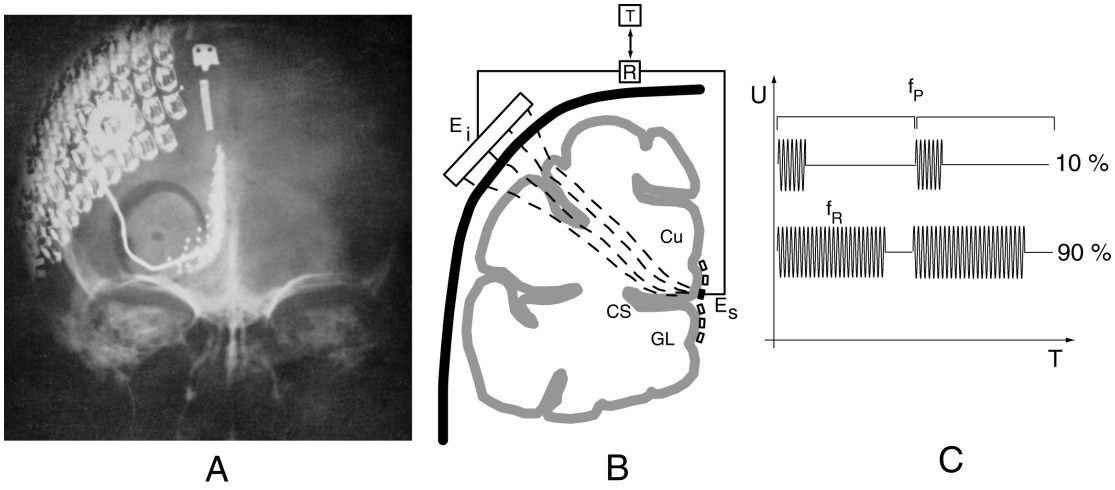

Fig. 9: (A) Coronal X-ray picture of Brindley and Lewin's (1968) device for independent stimulation of different locations of the medial wall representation of V1 in a blind subject. (B) Schematic drawing of A (T=radio transmitter; R=radio receiver; E_s=stimulation electrode; E_i=indifference electrode; Cu=cuneus; GL=gyrus lingualis; CS=calcarine sulcus). The electrodes were controlled by radio receivers implanted outside the skull beneath the skin, which in turn were stimulated by radio transmitters above the skin. Stimulation could thus be applied by each electrode separately and consisted of high frequency alternating current (6.0 or 9.5 MHz) pulsed in packages of variable frequency. (C) Demonstration of stimulation timing and 10 % and 90 % duty cycles (U=voltage; T=time; f_P=pulse frequency; f_R=radio frequency). Perceived brightness correlates with the duty cycle, being stronger for the 10 % than for the 90 % stimulus.

Upon stimulation the patient perceived very small spots of light "like a star in the sky" or like "the size of a grain of sago at arm's length" (ibid. p. 483). Recently a study by Lee and coworkers combined a large sample size of epilepsy patients with precise localisation of electrodes to perform the most comprehensive study on electrocortical stimulation of subjects with largely normal vision (Lee, Hong, Seo, Tae, & Hong, 2000). They studied 23 patients using a magnetic resonance imaging (MRI) and computer tomography (CT) coregistration technique for precise individual localisation. They classified the sensations as (1) Simple forms (uncoloured small flashing light points); (2) Intermediate form (uncoloured or coloured geometric shapes such as triangles, diamonds and stars); (3) Complex forms (animals, people, landscapes and sequences from autobiographic memory). They also localised sites leading to colour and motion percepts, temporary scotoma, visual illusions and visual experiences accompanied by sounds. While stimulation of the striate cortex and occipital pole resulted in only simple form sensations, stimulation of cuneus and

lingual gyrus (corresponding to visual areas V2 and V3) results in mainly intermediate form sensations. Complex form sensations and colour and motion sensation are evoked by stimulation of various extrastriate regions, according to what would be expected from functional specialisation of visual areas.

Grain

This condition supplements the condition of covariance to ensure that for every perceivably different state the neural population adopts a different state. Grain means that the resolution of the neural population has the capacity to represent the grain of perception. It implies a lower bound for the resolution of the neural representation, but not necessarily an upper bound. If we are able to perceive 150 different colour hues between 430 and 650 nm (Halsey & Chapanis, 1951) then our neural representation of colour hue will have to be different for each one in order to allow a representational mapping. The grain of perceptual resolution is very difficult to measure. One may assume that this question can be answered by studying the discriminability of visual features. But it has long been known that discrimination has a finer grain than perception, because subjects can make many discriminations without subjective confidence that they are performing above chance (Kolb & Braun, 1995; Stoerig & Cowey, 1997). However we can use discrimination as a first estimate of perceptual grain because if anything it will overestimate the required resolution. If a neural population can be shown to be able to account for discrimination it will have sufficient grain to account for conscious perception[15].

Isomorphism and perceived magnitude

The abovementioned criteria are a minimum set of requirements on the co-occurrence of perceptual and neural states. It is only possible to explain a perceptual state by the occurrence of a neural state if the same perceptual state is mapped to the same neural state on repeated occurrences and if different perceptual states are mapped to different neural states. These basic requirements allow one to treat different classes of perceptual states by a general methodological approach. It allows one to treat perceived object identity in a similar way to perceived magnitude of brightness or contrast. The latter two are real perceptual "dimensions" in the sense that for example states of perceived brightness are ordered according to their perceived magnitude. Perceived object identity however is more complex, because different perceptual

states are not ordered along a single dimension but are represented in a high-dimensional feature space.

A minimal requirement for a dimension of perceived magnitude[16] is that the perceptual states are ordered in a transitive fashion, which means that if q_2 is (say) brighter than q_1 and q_3 is brighter than q_2 then q_3 is also brighter than q_1. Ideally we would want this transitive relation also to be preserved by the neural representation. If brightness were represented by mean response amplitude of a neural population and one stimulus is perceived as brighter than another then we also want its neural response amplitude to be higher:

Representation of perceived magnitude: $\forall q_1, q_2 \in Q : A(q_1, q_2) \Rightarrow B(f(q_1), f(q_2))$

where A is a relation defined over the perceptual dimension (such as for example "is brighter than") and B is a relation defined over the neural population (e.g. "has a higher response amplitude than"). This requires the preservation of a relational property of a perceptual dimension by the neural dimension and goes beyond the mapping requirements formulated above. It postulates an *isomorphism* to hold between a dimension of perception and a property of the neural dimension representing it. The idea that the neural population representing a perceptual dimension has to preserve its order has a long history (Fechner, 1860; Mueller, 1896) and has been especially propagated by Gestalt theory (Koehler, 1920; Metzger, 1963; Stadler & Kruse, 1994).

Representation and strong necessity

After having assessed that a neural population N can fulfil the mapping requirements the second criterion will need to be tested: Is activity in N necessary for a certain dimension of conscious perception? Testing this is not as easy as it may seem. As stated above an intact retina can be thought of as "necessary" for visual experiences, because destroying both retinae results in peripheral blindness. But visual experiences can be caused even in blind people without retinae by directly stimulating visual cortex, as shown above (Brindley & Lewin, 1968). So intact retinae are necessary only for a specific way of causing visual percepts, namely via distal stimuli. This

"necessity" may be thought of as an insufficient but necessary part of an unnecessary but sufficient condition (Mackie, 1965). This is known as an INUS-condition[17]. The unnecessary but sufficient condition here is simply one way to cause visual percepts, namely by visual stimulation through the retina. In this causal chain the retina is necessary, but not in others. When we are interested in perceptual representation, however, we want activity in that neural population to be a necessary part of every sufficient condition. Necessary in the sense used here means absolutely or strongly necessary. We require that there is no sufficient condition that does not have the necessary condition as part of it.

> *Definition: Weak necessity*
> Activity in a neural population is weakly necessary for a certain class of visual percepts if activity in that neural population is a necessary but insufficient part of at least one unnecessary but sufficient condition under which such percepts can be produced.

> *Definition: Strong necessity*
> Activity in a neural population is strongly necessary for a certain class of visual percepts if activity in that neural population is a necessary but insufficient part of all sufficient conditions under which such percepts can be produced.

Strong necessity involves weak necessity, namely weak necessity in the entire set of sufficient conditions. The former (weak) necessity is easy to prove. All one has to do is for example destroy the retinae and observe if visual perception is disrupted. But how do we assess strong necessity? This is very difficult because it is a statement about the set of all possible sufficient conditions, which may be infinite and thus does not lend itself to empirical testing. All we can do is assume that a certain neural population is strongly necessary if under all conditions so far studied it has been weakly necessary, i.e. there has never been a case where a visual percept occurred without activity in that neural population. This can be tested by two different research strategies. On the one hand we can test if this particular visual percept could occur despite disrupting activity in the neural population, either by lesions or by temporal inactivation, say using transcranial magnetic stimulation. On the other hand we can

test if it is possible to evoke a certain percept by stimulating a different area when we can be sure that the neural population under investigation is not affected. In either case we could falsify the claim of strong necessity. So we can formulate an empirical criterion of strong necessity that splits up into two empirical sub-conditions:

> *Empirical criterion of strong necessity*
> Activity in a neural population can be justifiably assumed to be strongly necessary for a certain class of visual percepts (according to the best currently available scientific knowledge) if (1) in every case studied so far the disruption of activity in that population has lead to a loss of percepts of that class ("selective disruption") and (2) no case has been shown where it was possible to evoke this class of percepts without activating that neural population ("selective stimulation").

Selective disruption

To test the first condition that selective disruption of the neural population does not spare perceptual dimension Q one would in theory have to examine every case where a candidate area was lesioned and show that visual percepts of a specific class were always lost. This will lead to a set of areas of varying specificity. On the one hand we will find areas that are highly selective because they are strongly necessary only for certain subclasses of visual percepts. In all cases so far studied MT lesions have always lead to deficits in motion perception (akinetopsia), certain fusiform lesions have always lead to deficits in face perception (prosopagnosia) and lesions of V1 and V2 have always lead to visual field deficits such as scotoma and hemianopias (Heywood & Cowey, 1998; Heywood & Cowey, 1987; Heywood, Gadotti, & Cowey, 1992; Holmes & Lister, 1916; Horton & Hoyt, 1991a, 1991b; Kitajima et al., 1998; McFadzean & Hadley, 1997; Spalding, 1952; Zeki, 1991; Zihl et al., 1983)[18]. On the other hand we will find conditions that are strongly necessary for more general classes of percepts, such as activity in the brainstem reticular formation. The background conditions wakefulness and access have been discussed in chapter 1. Although these are strongly necessary they are ruled out because they do not fulfil the mapping requirements stated above.

A different, less dramatic method to study selective disruption is transcranial magnetic stimulation (TMS)(Fig. 10). TMS was first introduced by Barker in 1985 and is a non-invasive and painless method to induce cortical currents through the intact skull (Barker, Jalinous, & Freeston, 1985). In contrast to electrocortical stimulation TMS has the advantage of being available for normal subjects[19]. TMS can be used to disrupt visual processing with great temporal and acceptable spatial precision. Fig. 11 shows an experiment where TMS pulses are applied above the occipital pole at varying intervals after a target stimulus was presented. At a time delay of around 100 ms target processing is strongly disrupted, suggesting that processing at this time in the region of V1/V2 is necessary for perception.

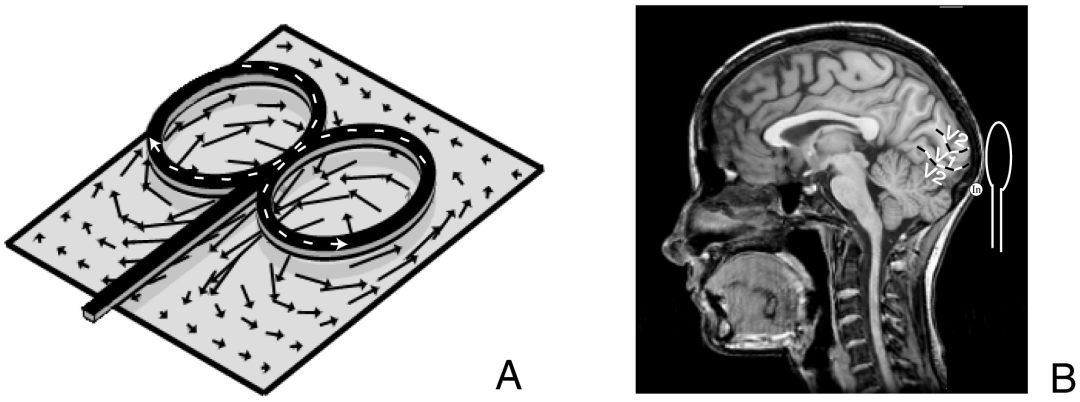

Fig. 10: In TMS short pulses of electrical current of up to 8000 A are induced in a stimulation coil (A) for less than 1 ms. This induces a magnetic field surrounding the coil which in turn induces an electric field which induces an intracortical current (Walsh & Rushworth, 1999). Here a double coil is shown with its coil current (dashed white arrows) and tissue current (black arrows)[20]. (B) Relative positions of coil and primary and secondary visual cortices are shown here on a sagittal T1-weighted anatomical MR image. The position of the foveal representation of V1 is on the posterior surface of the brain and can be reasonably well estimated as 2 cm above the Inion (In)[21].

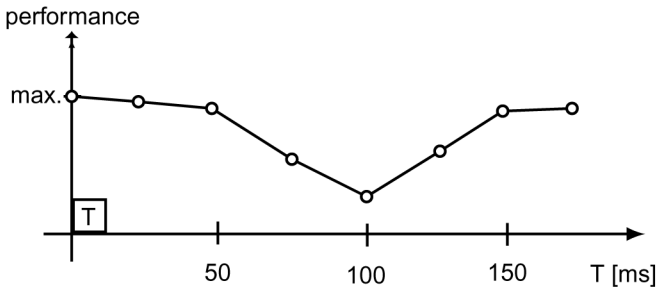

Fig. 11: Disruptive effects of a TMS pulse applied over the occipital pole on perception of a target (T) as a function of time between target and magnetic pulse (reproduced after Amassian et al., 1993). The exact cause of the disruptive effect is not clear to date but it is typically considered to be caused by inhibitory post synaptic potentials due to the induced current, which is supported by the finding of decreases in regional blood flow with increased magnetic stimulation (Paus & Wolforth, 1998).

Selective stimulation

The second condition (that it is not possible to evoke the class of visual experiences by stimulation of a different neural population without at the same time stimulating the area of interest) can be addressed by direct cortical stimulation. As mentioned above with this method it is possible to set the starting point of a neural cascade of events more precisely than by distal stimulation[22]. In this way it is possible to activate certain cortical areas directly, bypassing the afferent pathways. It can be used to assess whether an early visual area (cortical or subcortical) is a necessary condition by stimulating successively higher visual areas and checking whether a sensation along the visual dimension of interest can still be evoked. If it is possible to evoke a certain class of visual experiences by stimulating area V_n in the visual hierarchy then it could be argued that areas V_1 to V_{n-1} are not necessary, as long as signal spread backwards to these areas can be excluded[23]. The main strength is that it can be used to falsify a hypothesis that an area is strongly necessary for a specific class of visual percepts. For example the retina is not strongly necessary because direct electrical stimulation of the visual cortex also leads to visual sensations. Data on direct (intracranial) cortical stimulation is rare because it is only available from clinical studies[24]. An alternative for studies in normal subjects is TMS, which can also be used to elicit phosphenes when applied over visual areas (Cowey & Walsh, 2001).

Summary

Two *formal* criteria have been developed to assess whether basic mapping requirements between perceptual and neural states are fulfilled. The first requires that the same perceptual state must co-occur with the same neural state under repeated occurrences, even if the perceptual state is produced by very different stimuli. The second requires that different perceptual states always co-occur with different neural states. An additional formal criterion is relevant for dimensions of perceptual magnitude. It assesses whether isomorphism holds between relations defined on perceptual states and relations defined on the neural states representing them. Furthermore the criterion of strong necessity has to be met.

This has lead to 6 *empirical* criteria that are summarized in Tab. 1. These criteria can be used to test and falsify the hypothesis that a neural population represents a certain dimension of conscious perception. In the following two chapters these criteria will be applied to re-examine data from previous studies on representation of several feature dimensions. Chapter 3 will present evidence that the feature dimensions perceived colour, perceived motion and perceived object identity are likely to be represented in extrastriate visual cortex. Chapter 4 will demonstrate that primary visual cortex is the most promising candidate for the representation of the low-level visual feature dimensions perceived brightness and perceived contrast. In chapter 5 a study on perceived contrast will be presented. This study will demonstrate that for perceived contrast all criteria formulated here are fulfilled by neural processes in primary visual cortex.

Formal criterion	Empirical falsification criteria
Single-valuedness	Repeated occurrences of the same percept (e.g. in a constancy paradigm) lead to different activation patterns in neural population N
Injectivity	Failure of covariance of neural population N when a percept changes along a dimension of interest
	Lack of grain or resolution of neural population N to represent differences between percepts
Isomorphism	Lack of preservation of a relational property between two perceptual states in their neural representation
Necessity	Any percept including dimension Q after lesioning or disrupting processing in neural population N
	Any percept including dimension Q by stimulation of a different population than N when it can be excluded that neural population N was activated

Tab. 1: Summary of empirical falsification criteria. The postulate that neural population N represents the conscious feature dimension Q would be falsified if any of the empirical criteria (right) were answered with "yes".

Chapter 3

Representation of high-level features in extrastriate visual cortex

Colour

Human brain imaging studies have shown that many cortical visual areas (V1, V2, V3/VP, V4, V4α/V8) respond strongly to pure colour stimuli, i.e. their activity covaries with stimuli changing only in colour contrast where luminance contrast is kept zero or constant (Beauchamp, Haxby, Jennings, & DeYoe, 1999; Chao & Martin, 1999; Engel, Zhang, & Wandell, 1997; Hadjikhani et al., 1998; Kleinschmidt, Lee, Requardt, & Frahm, 1996; Lueck et al., 1989; McKeefry & Zeki, 1997). Even the MT complex can be driven by isoluminant colour stimuli (Seidemann, Poirson, Wandell, & Newsome, 1999; Wandell et al., 1999). Monkey single-cell studies originally suggested that V4 is the main cortical colour processing module because it was shown to have a large number of colour selective cells (Zeki, 1973). In humans an area on the border of the inferior occipital and temporal lobes also responded stronger to colour-defined than to luminance-defined Mondrian stimuli in early PET studies and was subsequently believed to be the human homologue of monkey area V4 (Bartels & Zeki, 2000; Lueck et al., 1989; Zeki et al., 1991).

However as research progressed the colour sensitivity of monkey V4 was questioned because subsequent studies often failed to show an abundance of colour sensitive cells in this area (Dean, 1979; Heywood et al., 1992; Schein, Marrocco, & de Monasterio, 1982). Also V4 was shown to be strongly involved in other visual processes such as form perception and spatial attention (Allison et al., 1993; Gallant, Braun, & Van Essen, 1993; Gallant, Connor, Rakshit, Lewis, & Van Essen, 1996; Moran & Desimone, 1985; Schiller, 1993; Walsh, Butler, Carden, & Kulikowski, 1992). In subsequent research regions further anterior on the monkey temporal lobe were demonstrated to have a high population of colour selective cells (Cowey & Heywood, 1995; Heywood, Gaffan, & Cowey, 1995; Komatsu, Ideura, Kaji, & Yamane, 1992). Furthermore several functional imaging studies demonstrated that a region anterior to the purported human V4 was strongly activated by colour stimuli (Bartels & Zeki, 2000; Beauchamp et al., 1999; Hadjikhani et al., 1998; Lueck et al., 1989). There was great debate as to the precise location and nomenclature of this region (V4α or V8), especially as it was defined by different authors either on anatomical considerations or

by retinotopic mapping (Fig. 12). Hadjikhani and coworkers (1998) for example found no colour sensitivity in V4 but only in the more anterior retinotopically defined V8[25].

V8 activity is more closely correlated to conscious colour perception than V4, because it is the only area that has been demonstrated to respond to perceived colour-aftereffects (Hadjikhani et al., 1998). Direct cortical stimulation studies show that colour sensations are mostly evoked by sites at the fusiform and lingual gyri, again matching both V4 and V8. Stimulation at other sites (such as V1, V2 or MT) does not lead to colour sensations (Brindley, Donaldson, Falconer, & Rushton, 1972; Brindley & Lewin, 1968; Dobelle, Mladejovsky, & Girvin, 1974; Lee et al., 2000)[26].

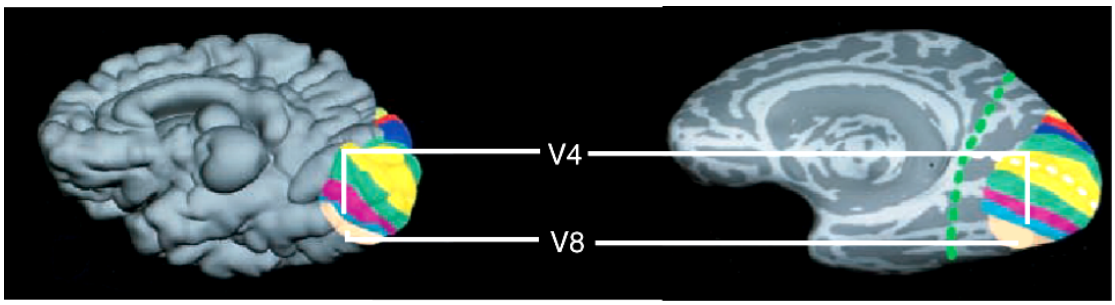

Fig. 12: The two proposed colour areas (overlaid on a retinotopic map taken from Tootell et al. 2000): V4 is the first colour area of Zeki and V8 refers to the second colour area termed by other authors V4α (Bartels & Zeki, 2000; Hadjikhani et al., 1998).

The phenomenon of colour constancy can help assess the criterion of single-valuedness by examining whether physically different stimuli that are perceived to have the same colour hue are neurally encoded in the same way. It is known that the perceived colour hue of an area in the visual field depends on the spectral statistics of the surrounding areas (Judd, 1940; Land, 1959a, 1959b)[27]. This reflects the ability of the visual system to discard for the effect of the spectral composition of the illumination. Several studies have indicated that a subpopulation of cells in V4 exhibit colour constancy whereas neurons in V1 and V2 do not (Kusunoki, Moutoussis, & Zeki, 2001; Moutoussis & Zeki, 2002; Zeki, 1983)[28].

It has long been shown that intact cortex around the border of fusiform and lingual gyri is necessary for colour perception, because lesions of this region in human

subjects always lead to impaired colour perception up to the complete loss of colour vision (Damasio et al., 1980; Zeki, 1990)[29]. Also, lesions to this region that spare V1 typically lead to a loss of colour perception (Le et al., 2002). Due to the extent of most lesions it is not possible to say whether this "achromatopsia" is due to a loss of area V4 or V8. The fact that direct cortical stimulation in the region of V4/V8 leads to colour perception whereas stimulation of other areas typically does not, could mean that early visual areas V1 and V2 are not strongly necessary for colour percepts, as long as backflow of signals from V4/V8 can be excluded. Furthermore lesions in area MT+ of the dorsal visual stream typically have no effect on colour perception (Vaina, 1994).

Summary

Areas V8 and V4 show a tight covariation with purely chromatic stimulus changes. Some evidence also points towards the fact that V4 responds to perceived colour hue independent of illumination conditions, indicating that the single-valuedness criterion may be met in V4. V8 but not V4 responds to perceived colour after-images. Little is known about whether V8 shows colour-constancy and whether responses in either V4 or V8 can account for the grain of colour space. Lesion studies and direct cortical stimulation studies reveal that the area around the human fusiform and lingual gyri is most likely to be the only colour sensitive area that is strongly necessary for conscious colour perception. Thus, V4 and V8 are the only regions in which perceived colour hue could be directly represented, but between these two the matter is still open. A study of colour constancy in V8 could help resolve this issue.

Motion

Functional brain imaging studies in humans have shown that a number of visual areas (V1, V3A, V4, MT+, KO, LOC) respond strongly when stimulated with moving as compared to stationary or flickering stimuli (Fig. 13)(Albright, 1992; Culham et al., 2001; Dupont et al., 1997; Dupont, Orban, De Bruyn, Verbruggen, & Mortelmans, 1994; Greenlee, 2000; Smith, Greenlee, Singh, Kraemer, & Hennig, 1998; Tootell et al., 1997; Tootell, Reppas, Dale et al., 1995; Zeki et al., 1991)[30]. In monkeys the most specialised areas for visual motion processing are MT and MST, where most cells are

direction selective (Lagae, Maes, Raiguel, Xiao, & Orban, 1994)[31]. In most human brain imaging studies MT and MST have not been separated and are combined to the so-called motion complex MT+, which is located in the region between inferior temporal sulcus and lateral occipital sulcus (Dumoulin et al., 2000)[32].

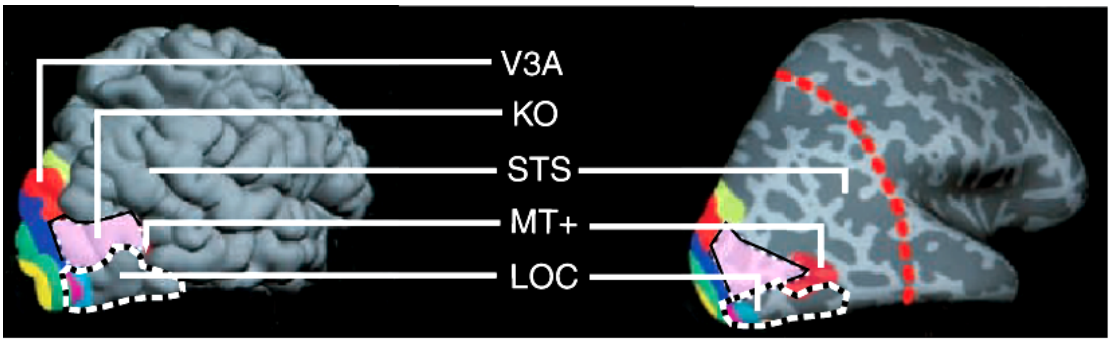

Fig. 13: Major cortical areas involved in motion processing (overlaid on a retinotopic map taken from Tootell et al. 2000): MT+ is buried in the inferior temporal sulcus and is only visible after inflating the surface.

Monkey MT/MST and human MT+ show the closest level of covariance with conscious motion perception. During binocular rivalry MT responds to purely perceptual changes of motion direction (Logothetis & Schall, 1989) and in humans it responds to purely perceptual changes of motion direction in multistable stimuli (Sterzer, Russ, Preibisch, & Kleinschmidt, 2002). MT/MT+ also responds to apparent motion stimuli, precisely within the displacement limits for which humans perceive apparent motion (Goebel, Khorram-Sefat, Muckli, Hacker, & Singer, 1998; Kaneoke, Bundou, Koyama, Suzuki, & Kakigi, 1997; Mikami, Newsome, & Wurtz, 1986). When two gratings drifting in different directions are superimposed, human observers perceive motion in the direction of the compound pattern rather in the direction of the component gratings (Fig. 14). In accord with human perception a percentage of cells in MT/MT+ is known to respond to the direction of pattern rather than component motion (Adelson & Movshon, 1982; Huk & Heeger, 2002; Stoner & Albright, 1992b). MT+ even responds in cases where motion is perceived without a moving stimulus. It responds during perception of illusory motion figures (Zeki, Watson, & Frackowiak, 1993), during imagined motion (Goebel et al., 1998), during perceptual motion aftereffects (Culham et al., 1999; He et al., 1998; Huk, Ress, & Heeger, 2001;

Tootell, Reppas, Dale et al., 1995) and even during perception of stimuli with implied motion (Kourtzi & Kanwisher, 2000).

Cortical microstimulation of MT can also induce a perceptual bias in motion direction judgement tasks in monkeys (Celebrini & Newsome, 1995; Salzman, Britten, & Newsome, 1990; Salzman, Murasugi, Britten, & Newsome, 1992). Transcranial as well as direct cortical stimulation of MT+ in epilepsy patients can induce motion hallucinations (Lee et al., 2000; Pascual-Leone & Walsh, 2001; Penfield & Rasmussen, 1950). However movement sensations can also be induced by direct cortical stimulation of V1 (Lee et al., 2000), which could be due to signal flow from V1 to MT+, which have strong mutual projections (Felleman & Van Essen, 1991). Stimulation of other areas rarely leads to motion perception (Lee et al., 2000) indicating that motion perception fails to correlate with activity in these areas.

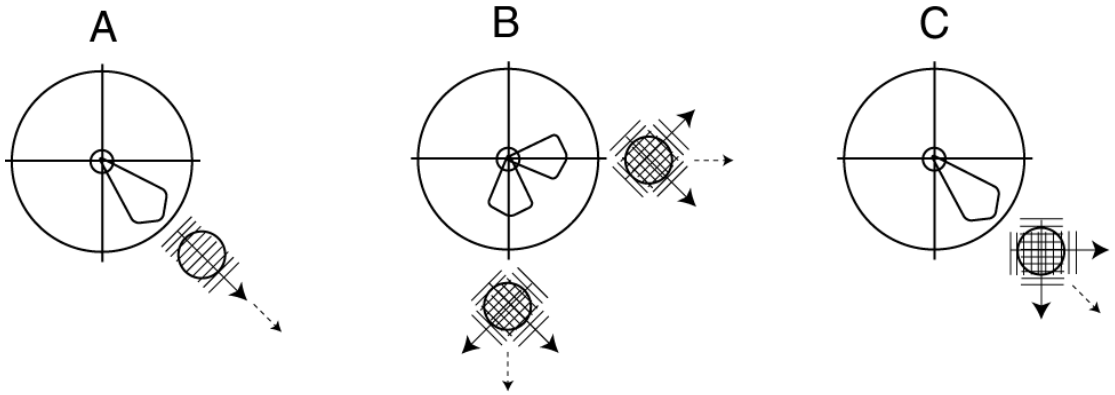

Fig. 14: Component versus pattern motion. The plots show the responses of direction selective neurons to motion as a function of angle. The distance from the centre indicates the response amplitude. The small circle outside the large circle symbolises to the receptive field. When a human observer views the patterns through the circular aperture he will perceive the direction indicated by the broken arrow. (A) Response profile of a direction selective neuron shows preferred direction of approx. 315°. (B) A component motion cell with a direction preference of 315° when measured with single gratings will respond to a superposition of two gratings that are drifting in orthogonal directions maximally when either of the components drifts in the preferred direction. (C) A pattern motion cell responds maximally when the pattern (to be precise its crossings) drifts in the preferred direction. Neuron B cannot be said to represent perceived direction of pattern motion, whereas neuron C can. (Movshon, Adelson, Gizzi, & Newsome, 1986).

In monkeys individual and pooled single unit responses in MT and MST have been shown to have a sensitivity comparable to psychophysical performance (Britten,

Shadlen, Newsome, & Movshon, 1992; Celebrini & Newsome, 1994; Mikami et al., 1986; Newsome, Mikami, & Wurtz, 1986; Shadlen, Britten, Newsome, & Movshon, 1996), suggesting that they can account for the grain of perceived motion. A fact that may question whether the grain is available is that motion trajectories are clearly localisable in visual space whereas MT+ is only coarsely retinopic (Gattass & Gross, 1981).

Other cortical areas have been demonstrated to play a major role in motion processing, but it is not clear how they relate to conscious motion perception. Among these are V3A, which is involved in direction discrimination (Cornette et al., 1998), motion imagery (Goebel et al., 1998), and perception of second order motion (Smith et al., 1998), 3D shape from motion (Paradis et al., 2000) and presumably also flow fields (de Jong, Shipp, Skidmore, Frackowiak, & Zeki, 1994)[33]. A further area was identified by different authors as kinetic occipital (KO) (Dupont et al., 1997; Orban et al., 1995; Van Oostende, Sunaert, Van Hecke, Marchal, & Orban, 1997) or V3B (Smith et al., 1998). It lies very close but slightly posterior to MT+[34] and is sensitive to borders where coherent motion fields change direction[35]. This could be used to segment borders of objects (Dupont et al., 1997; Orban et al., 1995; Paradis et al., 2000; Van Oostende et al., 1997). Surprisingly areas in the ventral visual stream that are specialised for shape and object processing (Milner & Goodale, 1995; Ungerleider & Mishkin, 1982) also contain motion sensitive neurons. Among these areas are V4, which contains cells that are selective to direction of motion (Desimone & Schein, 1987; Tolias, Smirnakis, Augath, Trinath, & Logothetis, 2001) and the so-called lateral occipital complex (LOC) that responds strongly to objects defined by motion cues (Grill-Spector, Kushnir, Edelman, Itzchak, & Malach, 1998) and to motion coherence (Braddick, O'Brien, Wattam-Bell, Atkinson, & Turner, 2000). Although these other motion areas covary with certain aspects of motion perception their role in representation of perceived motion is not clear.

Perceived motion can be evoked by movement of borders defined by various low-level cues (see Fig. 5, chapter 2). Area MT+ responds to motion defined by most cues such as luminance (Smith et al., 1998; Tootell, Reppas, Kwong et al., 1995), colour (Saito, Tanaka, Isono, Yasuda, & Mikami, 1989; Seidemann et al., 1999; Tootell, Reppas, Kwong et al., 1995; Wandell et al., 1999), contrast (Albright, 1992; Smith et

al., 1998) and even illusory contours (Seghier et al., 2000). Even responses to coherent motion are form-cue invariant in MT (Stoner & Albright, 1992a). This may be a first hint towards single-valuedness. However to fully support this hypothesis it would be necessary to demonstrate that the population response in MT+ is the same when the same perceived direction of motion is evoked by different cues. A result pointing in this direction is that population responses in monkey MT can be used to account for the perceived direction of movement of "motion metamers". Observers cannot distinguish more that two sets of superimposed random dot patterns moving in independent directions and perceive only two sets. This means that physically very different stimuli can lead to the same motion percept. This can be explained by the specific mechanisms with which MT recovers motion directions from population activity (Treue et al., 2000; Williams, Tweten, & Sekuler, 1991).

Using electrocortical stimulation in epilepsy patients and TMS in normal subjects it has been shown that disruption of processing in MT+ leads to a reduction or even complete loss of motion perception (Beckers & Homberg, 1992; Beckers & Zeki, 1995; Blanke, Landis, Safran, & Seeck, 2002; Hotson, Braun, Herzberg, & Boman, 1994), whereas stimulation over V1 only has weak effects (Beckers & Zeki, 1995). Using direct cortical stimulation over human MT+ it is possible to disrupt motion perception as selectively as single motion directions (Blanke et al., 2002). Patients with lesions in the region of the occipito-parieto-temporal junction, corresponding to the location of MT+, often have cortical motion blindness or "akinetopsia" (McLeod, Heywood, Driver, & Zihl, 1989; Plant, Laxer, Barbaro, Schiffman, & Nakayama, 1993; Rizzo, Nawrot, & Zihl, 1995; Zeki, 1991; Zihl et al., 1983; Zihl, von Cramon, Mai, & Schmid, 1991). However a closer look at these cases reveals that even for the prototypical and clearest case of akinetopsia demonstrated so far, patient L.Y. (Baker, Hess, & Zihl, 1991; Hess, Baker, & Zihl, 1989; McLeod et al., 1989; Rizzo et al., 1995; Zihl et al., 1983; Zihl et al., 1991), the loss of motion perception is far from absolute and is better characterised as partial and highly selective ("semi-akinetopsia"). Although she can be assumed to have bilateral lesions in MT+ (Zihl et al., 1983; Zihl et al., 1991) she still has considerable residual motion vision, especially at lower velocities, below approx. 15 deg/s (Rizzo et al., 1995; Zihl et al., 1991). No patient has been reported so far to have a complete loss of motion perception with an intact visual field for detection and acuity[36]. There is also evidence for spared

perception of biological motion after MT+ lesions (Vaina, Lemay, Bienfang, Choi, & Nakayama, 1990).

It is clear that several visual areas are not necessary for motion perception. Patients with severe deficits in perception of colours, shapes and objects after lesions to areas in the occipito-temporal areas show normal performance on motion tasks (Gallant, Shoup, & Mazer, 2000; Vaina, 1994; Vaina, Cowey, Eskew, LeMay, & Kemper, 2001), providing evidence that the ventral stream is not strongly necessary for motion perception. Evidence that primary visual cortex is not necessary for motion perception comes from patients with Riddoch syndrome. This refers to patients who are blind due to lesions in V1 (and possibly parts of V2) and show no residual object discrimination but nonetheless report to see pure objectless motion qualia, perhaps similar to Wertheimer's "pure phi" motion (Wertheimer, 1912)[37]. Riddoch syndrome was first described in the early 20th century (Riddoch, 1917; Zeki & Ffytche, 1998). Residual motion perception in Riddoch patients is different from blindsight because subjects report to *consciously* perceive motion rather than just perform above chance with the subjective impression of guessing. The Riddoch syndrome shows that visual awareness of motion is possible without V1[38].

Summary

There is strong support for a direct link between neural processes in MT+ and visual awareness of motion. Lesions to MT+ as well as disruption of MT+ lead to disruption of motion perception. Lesions to most other visual areas (even V1) can leave motion perception selectively spared, meaning that they are not strongly necessary for motion percepts. Processing in MT+ is not a necessary condition for *every* type of motion perception, simply because lesions to MT+ can spare perception of slow motion and of biological motion. This is in contrast to colour perception that can be completely disrupted by very local lesions in the fusiform gyrus. However MT+ is strongly necessary for perception of fast movement. Furthermore MT+ is the region that fulfils the mapping criteria best. It produces motion sensations that most closely match perception and can even account for the grain of motion perception. MT+ shows the strongest invariance as to the low-level cues by which motion is defined and can even account for the perception of "motion metamers", meaning that it may also fulfil the

criterion of single-valuedness. It seems that no single visual area can account for all motion qualia, but MT+ can account well for perception of fast motion[39].

Shape and objects

The main cortical sites of object processing reside in temporal cortex between the occipito-temporal junction and the inferior temporal lobe (Fig. 15). The main areas are V4, TEO and TE in monkeys (TEO and TE are often jointly named IT) and V4, lateral occipital (LO) and posterior fusiform gyrus (pFs) in humans[40]. These areas are typically labelled the "what" or "ventral" stream of visual processing (Livingstone & Hubel, 1988; Milner & Goodale, 1995; Ungerleider & Mishkin, 1982). Outside the ventral visual stream object selective activity can also be found in the kinetic occipital area (KO), which is also located around the occipito-temporal junction (Dupont et al., 1997; Orban et al., 1995; Van Oostende et al., 1997) and processes shapes defined by motion borders; furthermore in MT+ (Kourtzi, Bulthoff, Erb, & Grodd, 2002), the parietal lobe around area V7 (Grill-Spector et al., 2001), and in hippocampus and amygdala[41]. However, very little is known about the role of these other visual areas in object perception.

Local contours

Processing of shapes and objects involves a succession of stages, each with an increasing level of abstraction. For this reason successive areas covary with increasingly abstract properties of shapes and objects as one proceeds down the ventral processing stream. First of all the visual field has to be segmented into areas separated by contours. These contours can be defined by texture or motion borders and even by remote inducers as in the Kanizsa triangle. Single cells respond to most of these contours (even to illusory contours) as early as V1 (Grosof, Shapley, & Hawken, 1993; Lamme, Rodriguez-Rodriguez, & Spekreijse, 1999; Lee & Nguyen, 2001; Reppas et al., 1997; Sheth, Sharma, Rao, & Sur, 1996) and V2 (Reppas et al., 1997; Sheth et al., 1996; von der Heydt, Peterhans, & Baumgartner, 1984).

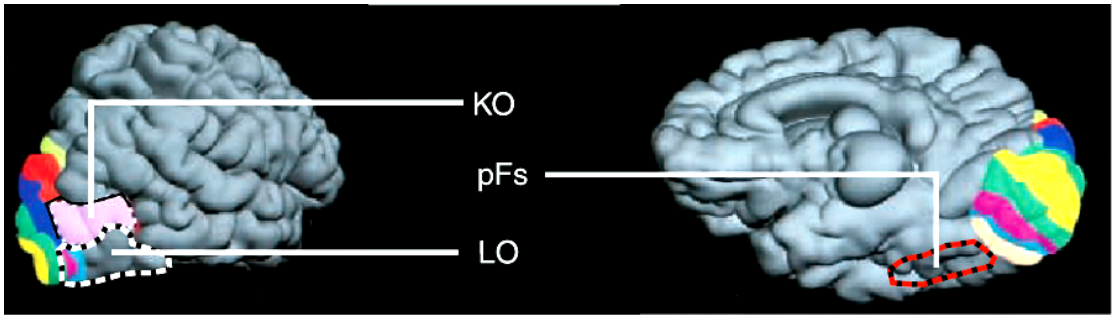

Fig. 15: The major cortical areas involved in processing objects (overlaid on a retinotopic map by Tootell et al. 2000). The lateral occipital complex LOC is mapped differently by several groups. Grill-Spector and coworkers subdivide it into lateral occipital (LO, black and white dashed line) and posterior fusiform (pFs, black and red dashed line). Sometimes LOa (lateral occipital anterior) is used instead of pFs but that is slightly misleading because the pFs is located on the temporal and not on the occipital lobe (Grill-Spector et al., 2001). PFs is believed to contain further subdivisions, e.g. regions processing mainly faces (fusiform face area, FFA) or letters. It is to date not clear how the *functionally* defined LOC relates to the *retinotopically* defined areas such as V4 and V8. The kinetic occipital area (KO, also mapped by other authors as V4d on retinotopic grounds) is strongly involved in processing motion boundaries (Dupont et al., 1997; Orban et al., 1995; Van Oostende et al., 1997).

With Jochen Braun from Plymouth University I have recently conducted a study that allowed to study contour processing without changing any local feature statistics (Braun, Haynes, & Heinze, 2002). These stimuli were introduced by David Field and consist of fields of randomly oriented Gabor patches within which the perception of contours is evoked purely by changing the orientation of the elements (Fig. 16A)(Field, Hayes, & Hess, 1993). Long snake-like figures can be generated as long as the angle between the axes of successive elements does not exceed 60°. This type of contour integration can be explained by the contextual modulation of receptive field properties of cells in primary visual cortex. A receptive field (RF) is defined as the region in the visual field that – when stimulated with a "suitable" stimulus - can directly lead to a change in a cell's spike rate. It was found however, that stimuli placed beyond this "classical" receptive field could modulate spike rates when the classical RF was already stimulated by an optimal stimulus, although they could not drive the classical RF directly (Allman, Miezin, & McGuinness, 1985; Fitzpatrick, 2000; Kapadia, Westheimer, & Gilbert, 2000; Maffei & Fiorentini, 1976; Nelson & Frost, 1978, 1985; Sengpiel, Sen, & Blakemore, 1997). In most studies these influences "from beyond the classical receptive field" are found to be inhibitory for

surround stimuli iso-oriented to the classical RF's preferred orientation and neutral to excitatory at orthogonal orientations (Blakemore & Tobin, 1972; Levitt & Lund, 1997; Nelson & Frost, 1978; Sengpiel et al., 1997; Walker, Ohzawa, & Freeman, 1999), although there are occasional deviations from this pattern (Kapadia, Ito, Gilbert, & Westheimer, 1995; Maffei & Fiorentini, 1976) [42]. Several authors (Kapadia et al., 1995; Kapadia et al., 2000; Nelson & Frost, 1985) have demonstrated that the effect for iso-oriented stimuli is not isotropic but instead is excitatory if the surround stimulus in on the main axis of the receptive field and inhibitory at the sides (Fig. 16B). This is a natural candidate to explain contour integration for the snake-stimuli, because contours are perceived precisely when successive contours fall within each other's enhancement fields.

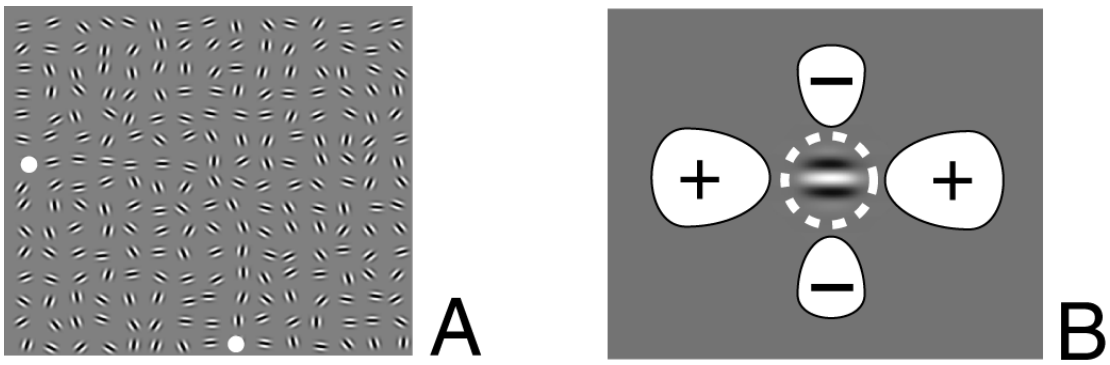

Fig. 16: (A) "Snake" contour stimuli: Array of randomly oriented Gabor-patches into which a curved contour was inserted (connecting the two white dots) by constraining the orientation difference of successive elements to a maximum of 60°. (B) Fields of surround suppression and enhancement for a simple cell in primary visual cortex (schematically reproduced from Kapadia et al. 2000). The greyscale plot in the centre shows the linear response profile of the classical receptive field, in this case with a horizontal orientation tuning. The dashed white line shows the border of the classical RF. The fields in the surround show regions where a surround stimulus that is iso-oriented to the classical RF and does not change the cell's spike rate when presented alone nonetheless exerts an excitatory (+) or suppressive (-) influence upon the response when the classical RF is optimally stimulated.

We used functional magnetic resonance imaging (fMRI) to assess the involvement of extrastriate and striate cortex in contour integration. Stimuli were Gabor stimuli matched to the V1 receptive field properties (Daugman, 1985; Marcelja, 1980) (Fig. 17, left). Stimuli were presented in two ways: (A) In a mini-block design as alternating sequences of 9 random and 9 contour stimuli with an inter-stimulus interval (ISI) of 1 second; (B) As a randomised sequence spaced by randomised

(exponentially distributed) inter-stimulus intervals of 1 to 5 seconds (Hinrichs et al., 2000). In both experiments contour stimuli evoked stronger activation as random stimuli in large areas of cortex previously related to contour and object perception (LO, LOa, V4v and V4d/KO), shown in Fig. 17 (right) for the mini-block condition. However we found only weak enhancement in primary visual cortex.

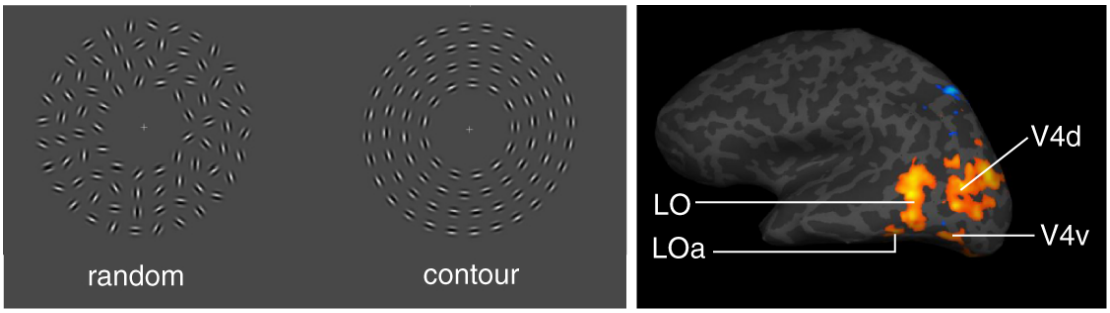

Fig. 17: Visual responses to contour integration: Lateral occipital areas. The two figures on the left are Gabor stimuli designed to match the receptive field properties of primary visual cortex. In the "random" stimulus orientations were chosen randomly, whereas in the "contour" stimulus they were chosen to yield integrated contours. The figure on the right shows how several visual areas typically involved in shape perception (LO, V4 and weaker LOa) respond stronger to the uniform stimulus than the random stimulus.

A closer inspection of the activation time-courses of single striate voxels revealed a strong saturation effect, most likely due to saturation of the BOLD-signal rather than neural adaptation effects. So we chose to change the timing parameters to a spaced event-related design with an ISI of 16 seconds. We also ran "localiser-scans" using high-contrast checkerboard annuli and mapped the horizontal and vertical meridians in order to be able to restrict our analysis to primary visual cortex. This revealed a clearly stronger activation of primary visual cortex by contour stimuli (Fig. 18A,B). We also recorded EEG and MEG evoked responses, which revealed that the contour-integration response builds up in V1 only at a later stage of processing (Fig. 18C). At an early stage of processing the random stimuli evoked stronger responses. This could be due to either feedback from extrastriate cortex or propagation of neural signals along horizontal connections linking iso-oriented orientation columns (Gilbert, 1992; Gilbert & Wiesel, 1979; Malach, Amir, Harel, & Grinvald, 1993; Martin & Whitteridge, 1984; Mitchison & Crick, 1982; Rockland, Lund, & Humphrey, 1982; Schmidt, Kim, Singer, Bonhoeffer, & Lowel, 1997).

This study presents evidence that contour integration without any corresponding changes in low-level features (such as luminance) is present as early as primary visual cortex. Thus "pure contours" could be represented in V1, although at this point it cannot be said if the V1 representation is an epiphenomenal feedback effect from higher visual areas.

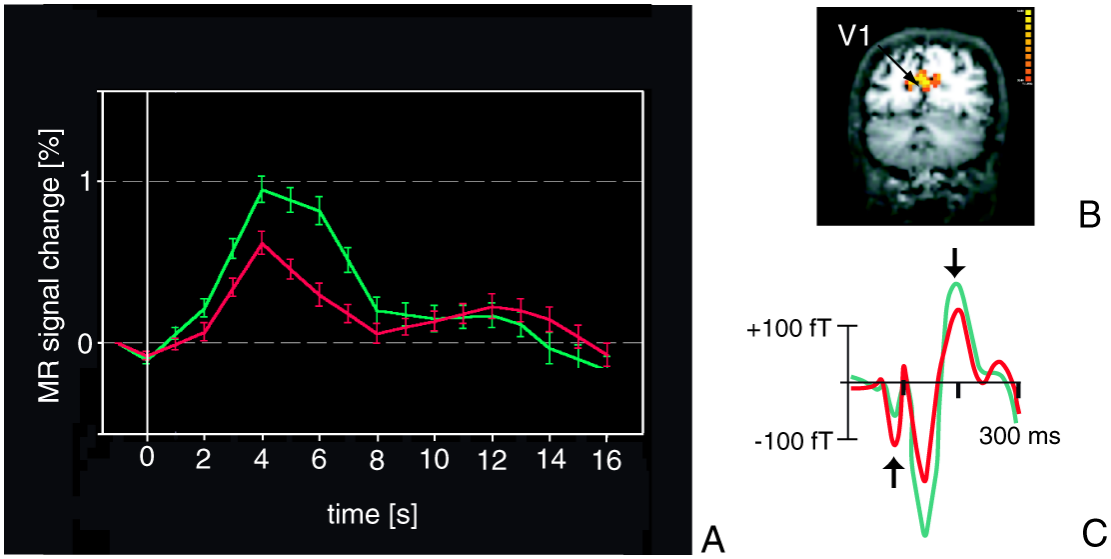

Fig. 18: Visual responses to contour integration in primary visual cortex (averaged across 6 subjects). (A) Event-related time-courses of BOLD-fMRI signal change in the region of primary visual cortex shown in (B). Red and green are the responses to random and uniform stimuli respectively (see Fig. 17, left). The stimulus containing the integrated contour clearly evokes a stronger response in V1. (Error bars = +/- 1 SEM). (C) Event-related MEG response to a left occipital sensor. The arrows point to the early feed-forward and late feedback responses presumably generated in V1. As can be seen during the first processing stage the random stimulus evokes stronger activity in V1, whereas at the feedback stage (which shows an inverted topology to the early response) the response to the collinear stimulus is stronger.

Intermediate shapes

Following the major projections of the monkey ventral stream the next major area is V4, which is generally considered a site of intermediate shape processing (Desimone & Schein, 1987; Gallant et al., 1993; Gallant et al., 1996; Pasupathy & Connor, 1999). On the one hand V4 neurons respond well to typical V1 stimuli such as gratings (Desimone & Schein, 1987). On the other hand they also show a selectivity to stimuli

defined in other than Cartesian basis systems such as polar or hyperbolic stimuli (Gallant et al., 1993; Gallant et al., 1996; Wilkinson et al., 2000)(Fig. 19).

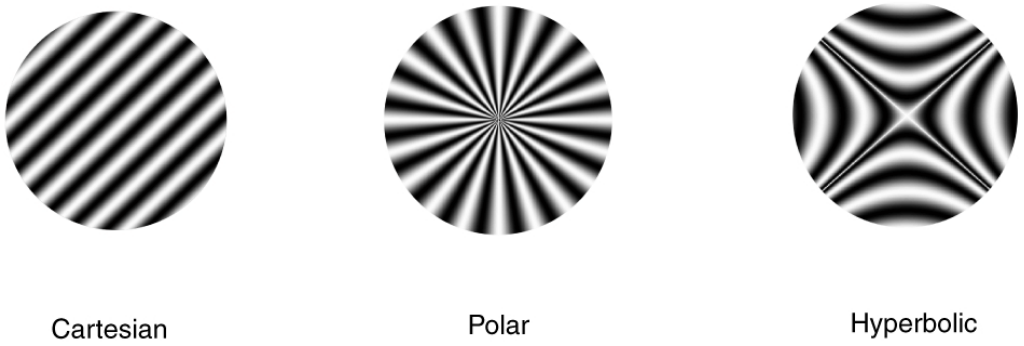

Fig. 19: Complex shape processing in V4: Cartesian, polar and hyperbolic gratings similar to those used by Gallant and coworkers (1993).

Complex shapes and objects

In monkeys, representation of complex shapes begins with inferior temporal areas TEO and TE. These areas are still ordered into columns where neighbouring cells show similar response profiles for complex geometrical shapes (Fujita, Tanaka, Ito, & Cheng, 1992; Tanaka, 1996)[43]. In human PET and fMRI studies a large cortical area covering the lateral occipital cortex and fusiform gyrus can be selectively activated during complex shape and object perception which is referred to as the lateral occipital complex (LOC)(Grill-Spector et al., 2001; Haxby et al., 1991; Malach et al., 1995).

LOC is identified functionally (rather than retinotopically) by comparing responses to objects versus scrambled objects, a method designed to keep low-level features identical and only manipulate the presence or absence of a complex shape (Lerner, Hendler, Ben-Bashat, Harel, & Malach, 2001). LOC can be further subdivided into the two main areas LO (lateral occipital) and pFs (posterior fusiform) or lateral occipital anterior (LOa), which roughly correspond to monkey TEO and TE. Because LO and pFs are defined functionally rather than retinotopically it is not clear if in some studies LO also includes areas V4v and V8 (see for example Lerner, Hendler, & Malach, 2002).

Besides the subdivision into LO and pFs the lateral occipital complex also shows subregions that are selective to specific object categories. The most prominent example of these submodules is the so-called fusiform face area FFA, which is selectively activated by faces versus other objects (Gauthier, Tarr et al., 2000; Haxby et al., 1994; Kanwisher et al., 1997; Puce et al., 1995). An additional occipital face area (OFA) was also found (OFA, Gauthier, Skudlarski, Gore, & Anderson, 2000) and other temporal areas have been found that are selective to different categories such as letters (Puce, Allison, Asgari, Gore, & McCarthy, 1996), chairs (Ishai, Ungerleider, Martin, Schouten, & Haxby, 1999) or views of houses (Aguirre, Zarahn, & D'Esposito, 1998; Epstein & Kanwisher, 1998). More recent functional imaging studies have questioned this strong object modularity by demonstrating that the fusiform face area is also strongly activated by overlearned novel categories (Gauthier, Skudlarski et al., 2000; Gauthier & Tarr, 1997, 2002; Gauthier, Tarr, Anderson, Skudlarski, & Gore, 1999; Rossion, Gauthier, Goffaux, Tarr, & Crommelinck, 2002; Tarr & Gauthier, 2000)[44]. Also different categories of objects do not activate mutually exclusive regions of cortex, but rather differ in their specific pattern of activation in a number of temporal areas (Ishai et al., 2000; Ishai et al., 1999). This provides evidence that inferior temporal cortex may be rather organized in a "featurotopic" fashion holding a "dictionary of complex shapes" (Riesenhuber & Poggio, 2000; Tanaka, 1996).

Activity in monkey IT and human LOC correlates not only closely with high-level features of objects but also with object perception. When subjects view thresholded black and white images as in Fig. 20 (left) they will perceive a complex spatial pattern but are often not able to recognize the depicted object. However after exposition to a non degraded version of the stimulus (learning phase, Fig. 20, middle) they will recognize even a degraded object without problem. This is a prototypical rapid perceptual learning process and allows comparison of processing of the same stimulus with and without recognition. Dolan compared stimuli before and after recognition learning in a PET study (Dolan et al., 1997). The same stimuli evoked stronger activity in inferior temporal cortex after recognition learning. A possible interpretation of this result is that neurons in inferior temporal cortex respond not only to the complex shapes but also to the objects these shapes resemble. Unrecognised

stimuli are processed up to the level of complex shape processing, but because they are unfamiliar they cannot be assigned to an object category.

However the interpretation is not so straightforward. It can be demonstrated behaviourally and physiologically that even unrecognised stimuli are processed up to a categorical level. Behaviourally there is evidence for repetition priming for unrecognised objects (Bar & Biederman, 1999; James, Humphrey, Gati, Menon, & Goodale, 2000).

Rather than using an all-or-nothing rapid learning design the border between unrecognised and recognised objects categories can be crossed more gradually by employing backward masking designs (Fig. 8, chapter 2). Backwards masking of objects has been studied at the level of single cells in monkeys IT (Kovacs, Vogels, & Orban, 1995; Rolls, Tovee, & Panzeri, 1999). Kovács and coworkers (1995) recorded from shape-selective neurons in macaque area IT with pattern masking stimuli. Even for stimuli with strongly reduced discriminability due to masking they found clear shape-selective responses. But these responses were temporally brief and were interrupted by presentation of the mask. Grill-Spector and coworkers (2000) however have shown that in humans the activity in LOC (but not in V1 and in the dorsal stream) covaries closely with the perceptual threshold of object awareness. Following an interpretation by Kovács et al. (1995) one could assume that awareness requires the activity to be integrated over a certain temporal duration in able to be accessed. Thus, pattern backwards masking may not be a problem of representation but a problem of access.

Further evidence for a close correlation between activity in inferior temporal cortex and conscious object perception comes from studies of binocular rivalry. When conflicting (non-fusible) stimuli are presented to both eyes (e.g. using prisms, shutter glasses or chromatic filters), one of the two inputs will be temporarily suppressed and the observer will perceive an alternation between the images presented to the individual eyes. In monkeys it has been demonstrated that inferotemporal areas show the strongest correlation with the current percept (Leopold & Logothetis, 1999).

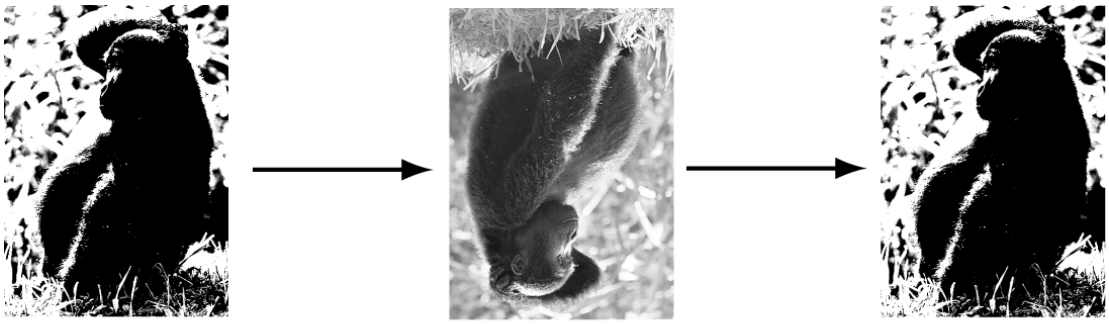

Fig. 20: Degraded (thresholded) image presented during pre-learning phase that is not recognized at the first exposure by most subjects (left). Then in a learning session subjects view a non-degraded version of the stimulus (the reader has to turn the page upside down to view the stimulus). Afterwards the degraded stimulus is easily recognized.

In humans it has been demonstrated that this perceptual alternation occurs in a highly category specific fashion. As mentioned above there is a certain degree of categorical specialisation in inferior temporal cortex, which is most pronounced for faces and houses. If conflicting face and house stimuli are presented to both eyes the dominance phases of face and house percepts are strongly correlated with increased activity in the corresponding specialised areas (Fig. 21)(Tong, Nakayama, Vaughan, & Kanwisher, 1998). Further evidence for a close correlation comes from direct cortical stimulation of the region of the LOC. In humans this leads to hallucinations of complex objects such as animals, faces, body parts and landscapes (Lee et al., 2000; Penfield & Rasmussen, 1950). Direct cortical stimulation of regions other than LOC does not result in complex object percepts (Lee et al., 2000).

For shape processing there is an increasing tendency towards invariance over local cues as one processes down the ventral stream in both monkeys and in humans. It has been demonstrated that object representations in monkey inferior temporal cortex are largely invariant of cue type (Sary et al., 1993). Human LO and pFs also respond well to objects regardless of whether they are defined by texture, colour or motion borders, lines or illusory contours (Grill-Spector et al., 1998; Kastner, De Weerd, & Ungerleider, 2000; Mendola, Dale, Fischl, Liu, & Tootell, 1999).

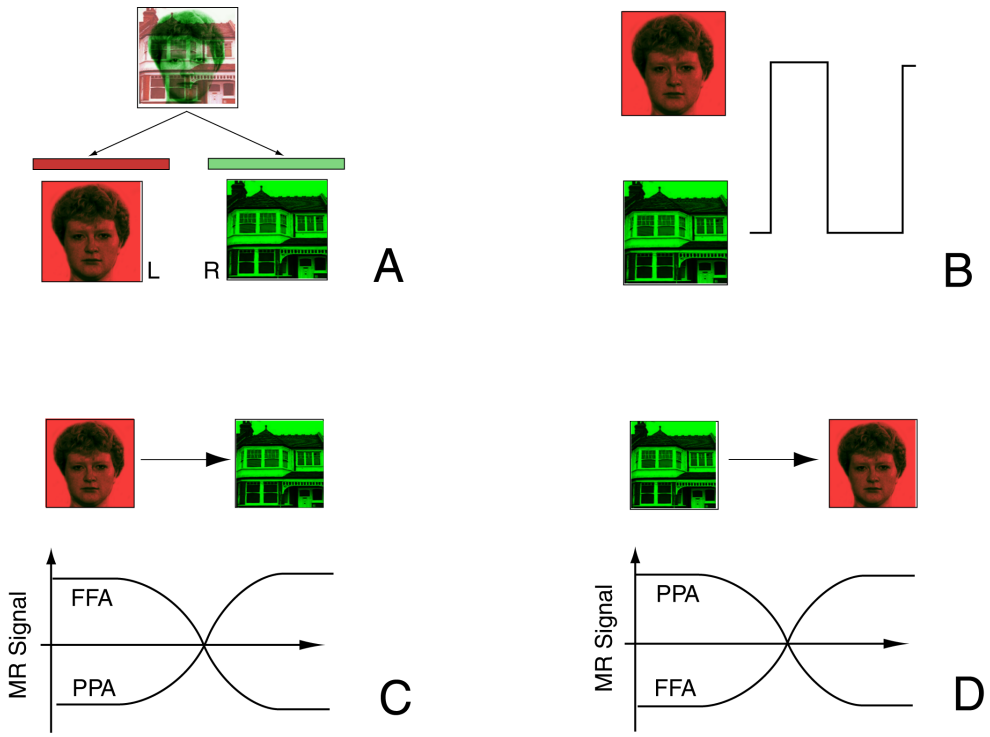

Fig. 21: (A) Tong et al.'s (1998) binocular rivalry scenario: A green filtered face picture and a red filtered house picture are presented superimposed. When left and right eyes view the picture through red and green filters respectively only one object can be seen in either eye. After a (mostly short) phase of fusion an observer's perception will select only one of the objects and stochastically alternate between the two (B). (C) Transition from a dominant face percept to a house percept leads to a decrease in activation in face processing areas (FFA) and an increase in house / scene processing areas (PPA)(schematically reproduced from Tong 1998). (D) As in C but for a transition from house to face.

For shape and object perception even complex types of *spatial* invariances can be found (Fig. 22A). As single-cell recording is practically unavailable in humans the generalisation characteristics of single neurons in temporal cortex have to be estimated indirectly using an fMRI selective adaptation paradigm. The logic employed is that if a neuron generalises across multiple views of an object then repeated stimulation with different views of the object should lead to a reduced response due to neural adaptation or due to priming (Buckner et al., 1998; for a more elaborate interpretation see James et al., 2000; Schacter & Buckner, 1998). Thus the degree of response reduction can be taken as a measure of generalisation. Using such a repetition paradigm it has been shown that cells in human LOC are largely invariant to changes in size and position of objects (Grill-Spector et al., 2001; Grill-Spector et

al., 1999)[45]. Invariance to more complex manipulations was nicely demonstrated in monkey inferior temporal cortex (Baylis & Driver, 2001) (Fig. 22B). Baylis and Driver examined invariance of IT neurons to contrast polarity, mirror inversion and figure-ground reversal. They found a strong correlation between the responses of IT neurons to the original stimulus and the contrast inverted and mirror stimuli, but not between response to original stimulus and figure-ground inverted stimulus. Invariance has also been demonstrated for lighting conditions in monkeys (Hietanen, Perrett, Oram, Benson, & Dittrich, 1992), but not in humans (Grill-Spector et al., 1999). While there is a strong tendency towards most types of invariance in the inferior temporal lobes there is only weak (if any) generalisation over different viewpoints, especially over rotations in depth. This has been shown for monkeys (Logothetis & Sheinberg, 1996; Perrett, Hietanen, Oram, & Benson, 1992; Tanaka, 1996) and humans (Grill-Spector et al., 1999)[46]. This finding is paralleled by behavioural demonstrations of high costs of object rotation (Bulthoff & Edelman, 1992; Bulthoff, Edelman, & Tarr, 1995; Logothetis et al., 1994). Thus, there is hardly any evidence of object-centred representations in the visual system.

Lesions to the temporal lobes constantly lead to deficits in object processing (Farah, 1995; Humphreys, 1999). Large lesions typically lead to a general loss of object perception whereas small lesions can highly selectively affect single object categories, the best studied of which is face perception (Damasio et al., 1980; McNeil & Warrington, 1993; Sergent, Ohta, & MacDonald, 1992; Sergent & Signoret, 1992). Other selective object recognition deficits have also been found. In one extreme case a farmer was reported to be unable to recognize his cows despite intact recognition of human faces, a deficit coined “zooagnosia” (Assal, Favre, & Anderes, 1984). Further evidence that activity in LOC is necessary for conscious perception of shapes and objects comes from transcranial magnetic stimulation (TMS) and direct cortical stimulation of the temporal lobe. TMS pulses applied over Brodman’s area 37 can deteriorate performance in picture naming tasks (Stewart, Ellison, Walsh, & Cowey, 2001). Direct cortical stimulation of the temporal lobes has been shown to interfere with object processing in monkeys (Goldrich & Stamm, 1971) and humans (Fried, Mateer, Ojemann, Wohns, & Fedio, 1982). Lesions in the region of MT+ do not lead to deterioration of shape discrimination and object recognition (Vaina, 1994).

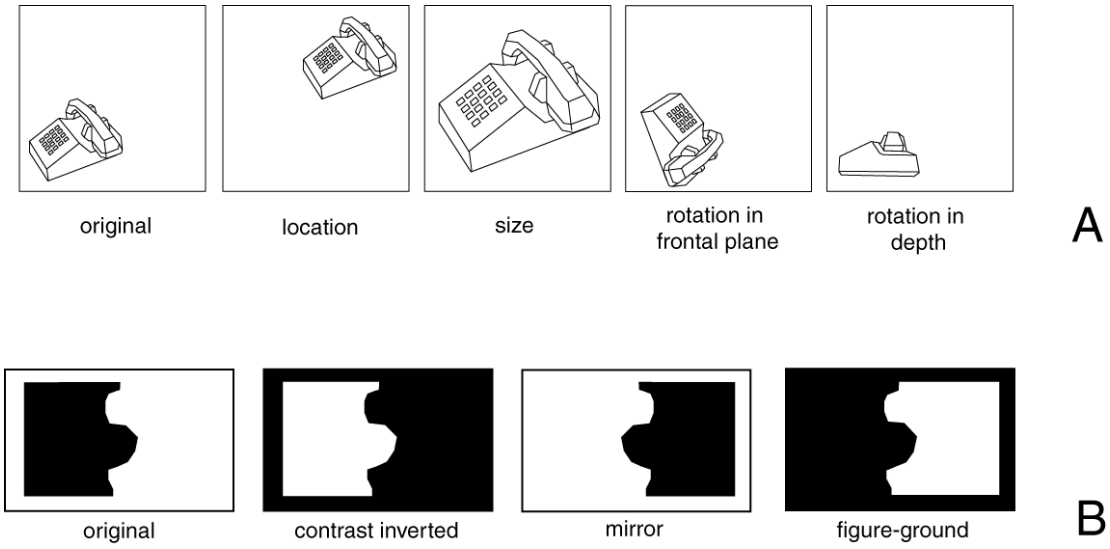

Fig. 22: Principles of invariance in shape and object perception. (A) Spatial invariances of location, size and rotation: Invariance of location refers to representations that do not depend upon where in the visual field an object is presented. Invariance to size means that the mapping of visual features is independent of the scaling of the object. Viewpoint invariance is the trickiest of all and refers to the fact that the representation of an object is invariant to rotation in the frontal plane or in depth. Invariance over rotation in the plane is easier to achieve than rotation in depth. One instance of an object can be matched to an in-plane rotated exemplar by pure 2D rotation of the projection. However rotation in depth can reveal completely different parts of object that were previously occluded requiring either exemplars from multiple viewpoints or a 3D model for matching (Riesenhuber & Poggio, 2000). (B) More complex types are invariances to contrast polarity, mirror image and figure-ground used by Baylis and Driver (2001).

Summary

To summarize, the human visual area LOC covaries with conscious perception of abstract shape and object properties strongest of all visual areas. In many cases it is the only area to covary with object perception. Furthermore there is strong evidence for an independence of inferior temporal responses from low-level features, position, scale, contrast polarity and mirror-inflection. This is a first hint towards single-valuedness because it implies that the abstract properties of shapes defined by different cues are represented by the same neural population activity. Lesions and disruptions in the region of LOC lead to selective deficits of object perception. Little can be currently said on the grain of representation, because no discrimination studies are available. Together these results strongly support the claim that conscious

representation of complex shapes and objects occurs in the human lateral occipital complex.

Summary: High-level features and extrastriate visual cortex

In this chapter the use of the framework presented in chapter 2 has been demonstrated by using it to re-analyse results from previous studies on perception of high-level visual features. Is has been demonstrated that V1 is not necessary for perception of colour hue, motion and complex shapes and objects. Although under normal circumstances V1 is an important stage in processing of these stimulus categories it is possibly to by-pass V1 and evoke high-level percepts by stimulating the according areas directly (Lee et al., 2000). The different high-level features are represented in a distributed fashion and conscious perception can break down selectively for each of these feature categories after lesions in the regions representing them. Interestingly, the strong invariance of areas specialised in motion and shape processing with respect to low-level visual features means that these areas cannot represent the low-level visual features by which the complex features are defined. The involvement of V1 in processing local contour information has already been demonstrated. The next chapters will investigate whether V1 can possibly encode our conscious perception of the low-level visual features of brightness and perceived contrast.

Chapter 4

Representation of low-level features in primary visual cortex

Perceived brightness

Due to the predominance of research on luminance contrast the responses of visual cortex to homogenous illumination have only rarely been directly studied[47]. In the few studies available it was demonstrated that a substantial proportion of cells in cat and monkey striate cortex can be characterized as "luxotonic" in showing a modulation of their responses by homogenous changes in illumination (Bartlett & Doty, 1974; Kayama, Riso, Bartlett, & Doty, 1979; Kinoshita & Komatsu, 2001; Komatsu, Murakami, & Kinoshita, 1996; MacEvoy, Kim, & Paradiso, 1998; Maguire & Baizer, 1982; Rossi & Paradiso, 1999; Rossi et al., 1996). Luxotonic cells also give a sustained measure of luminance level rather than a transient measure of luminance change, which matches the fact that perceived brightness[48] remains elevated after sustained increments in luminance[49] (Bartlett & Doty, 1974).

Photergic and scotergic cells in V1

Kinoshita and Komatsu (2001) performed the most extensive study so far on luminance representation in monkey V1. They investigated the luminance dependency of responses to uniform stimuli of varying luminance on a uniform grey background. They found the modulation to be strongest for later, sustained phases of processing. Three different profiles were observed: 67 % of luxotonic cells increased their response rate monotonously with luminance (photergic or "bright type"); 25 % of cells monotonously increased their spike rate with *decreasing* luminance (scotergic or "dark type")[50]. If primary visual cortex were to represent perceived brightness one might expect that the spike rate increases monotonously with luminance, which would only fit to the "bright type" cells of Kinoshita and Komatsu (2001). An alternative explanation would be that the visual system employs a double strategy and explicitly signals darkness and brightness separately. It has long been known that retinal ganglion cells can be subdivided into two subclasses, the on-centre and off-centre type (Kuffler, 1953). This provides evidence for a bipolar rather than unipolar processing of luminance. Also, there is evidence that grey rather than black may be the zero-point of brightness perception. In the absence of visual stimulation in the

state of dark adaptation observers typically perceive the visual field in a homogenous grey ("Eigengrau") rather than black (Aubert, 1865).

Phosphenes

Brindley and Lewin (1968) used the electrocortical stimulation device shown in Fig. 9 to stimulate the region of primary visual cortex. Their subject perceived small, bright, isolated, point-like flashes (phosphenes) that were reproducibly localised in retinotopic space (Brindley & Lewin, 1968)(Fig. 22). The phosphenes were mainly elicited around the vertical meridian (12 o'clock and 6 o'clock positions) as would be expected from stimulating the cortex just outside the calcarine sulcus according to retinotopic mapping of visual field onto cortex. The size of phosphenes increases with eccentricity as would be expected due to the cortical magnification factor (Cowey & Rolls, 1974). Phosphenes were restricted to the area immediately surrounding the calcarine sulcus and stimulation from more distant electrodes (most parts of V2) did not evoke phosphenes. Others have reported that stimulation of extrastriate visual areas leads to complex phosphenes, such as intermediate and complex shapes, colours and motion, but not to localised points (Lee et al., 2000).

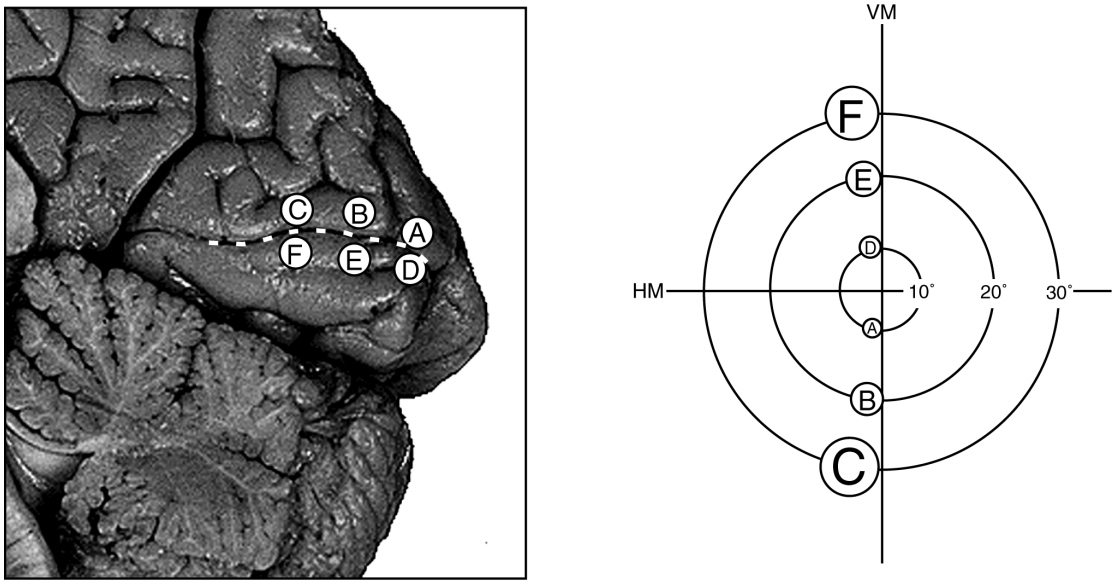

Fig. 22: Schematic drawing of the relationship between stimulation site along the sulcus calcarinus of the right hemisphere (dashed line) and perceived visual field location and size of phosphenes elicited by Brindley and Lewin (1968) (HM=horizontal meridian; VM=vertical meridian). The size of phosphenes scales with cortical magnification factor (Cowey & Rolls, 1974).

Brindley and Lewin (1968) were able to evoke well-defined spatial dot-patterns by stimulating multiple sites in V1 simultaneously. Their aim was to transmit pixelated letter patterns in order to develop artificial visual prostheses for retinally blind patients. V1 is the only cortical site from which high-resolution stationary phosphenes can be reliably evoked. This has led this field of research to focus on V1 as the most promising site for an implant grid providing a "scoreboard-like" display matrix for transmission of information (Dobelle & Mladejovsky, 1974; Dobelle, Mladejovsky, Evans, Roberts, & Girvin, 1976; Dobelle et al., 1974; Girvin, 1988; Stensaas, Eddington, & Dobelle, 1974). If the sensations produced by V1 stimulation were merely epiphenomenal and due to passing on of activation to extrastriate areas such as V2 and V3 one would expect to be able to produce the same small localisable sensations by stimulation of V2 or V3 directly, which is not the case[51].

Metacontrast masking

One method that allows varying the perceived brightness of a target independent of its physical luminance is metacontrast masking. This occurs when a target stimulus (typically a disk of uniform luminance) is presented upon a dark background and is followed by a mask stimulus, that shares a contour with the target (typically an annulus or ring). For a certain time delay between offset of the target and onset of the mask (typically around approx. 50-100 ms) the perceived brightness of the target stimulus is strongly reduced (e.g. Bridgeman & Leff, 1979), but detection is normally not affected (Kahnemann, 1968). Thus the subject is aware of the presence of the stimulus but it appears to be darker (Bridgeman & Leff, 1979). The detailed mechanisms of metacontrast masking are not understood to the present day[52]. Psychophysical data suggest a locus in early retinotopic visual cortex. Metacontrast masking is possible even when target and mask are presented dichoptically (Kolers & Rosner, 1960; Schiller, 1968) suggesting that it does not occur before binocular processing in visual cortex. The fact that metacontrast masking scales with eccentricity similar to cortical magnification factor (Bridgeman & Leff, 1979) and that it falls rapidly with the distance between target and mask contours (Alpern, 1953; Kolers & Rosner, 1960; Werner, 1940), suggests that it occurs in early retinotopic cortex, presumably in an area with small receptive fields.

Several single-cell studies point towards V1 as the major site of metacontrast masking[53]. In V1 effects of metacontrast masking have been found on the transient onset responses (Macknik & Haglund, 1999; Macknik, Martinez-Conde, & Haglund, 2000), on secondary (possibly feedback) discharges (Bridgeman, 1975, 1980) and on off-responses (Macknik & Livingstone, 1998). The modulation of late striate responses fits to the fact that luminance-dependency of luxotonic cells is strongest at the late response phases (Kinoshita & Komatsu, 2001). It also presents a straightforward account of the paradox that reaction times for detection are not influenced by metacontrast masking. Detection may be mediated by a low criterion and could be based on the earliest striate responses, whereas judgement of perceptual magnitude may depend on the later stages of processing. A nice demonstration of the effects of metacontrast masking in V1 can be seen in Fig. 23. Optical imaging responses to target stimuli are strongly suppressed under masking conditions in a highly retinotopic fashion. Although these results provide a possible correlate of perceived brightness reduction in primary visual cortex a direct link has not yet been demonstrated.

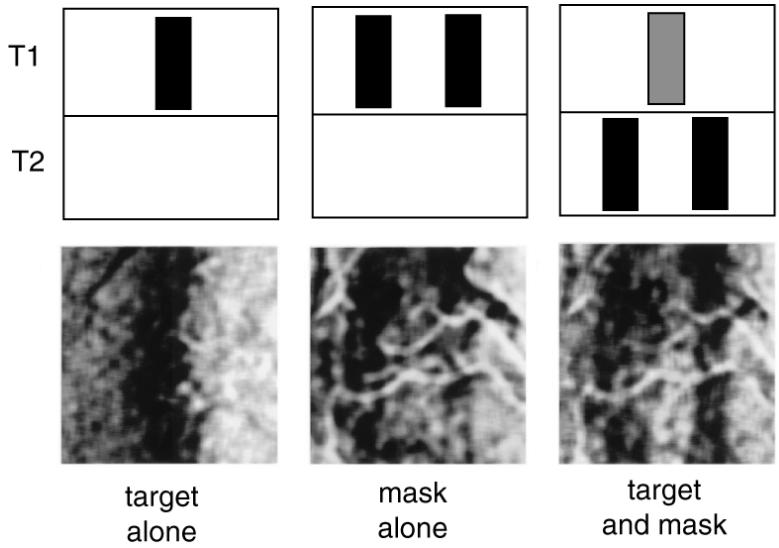

Fig. 23: The effect of metacontrast masking on the retinotopic representation of a target bar in primary visual cortex as demonstrated using optical imaging (Macknik & Haglund, 1999). Presentation of the target alone (left) or mask alone (middle) alternating with empty frames leads to retinotopic representations of these stimuli in area V1. If target and masks are alternated with timing parameters for which the target is invisible in human observers (right) the representation of the target in V1 is strongly suppressed.

V1 signals brightness and not luminance

A different way to study changes in perceived brightness without changing physical luminance is to exploit surround effects. It is known that human brightness ratings are strongly influenced by the luminance distribution of contextual stimuli (Adelson, 1993, 1999). In the simplest case this occurs over very small angular distances and leads to border enhancement (Mach-bands), illusory dots (in the Hermann grid illusion) or illusory lines (Fig. 24A). There are also contextual modulations across large angular distances as in the "simultaneous contrast effect". A uniform surface with fixed luminance on a uniformly illuminated background is perceived to have lower brightness the higher the luminance of the background is as would be expected if the visual system took into account the mean background luminance to discount for the illuminant (Fig. 24B). There are also more complex variants of such long range contextual modulations such as the border-induced Craik-O'Brien-Cornsweet illusion (Cornsweet, 1970), or the corrugated plaid illusion (Adelson, 1999) the latter even revealing the involvement of 3D geometry and lighting interpretation (Fig. 24C).

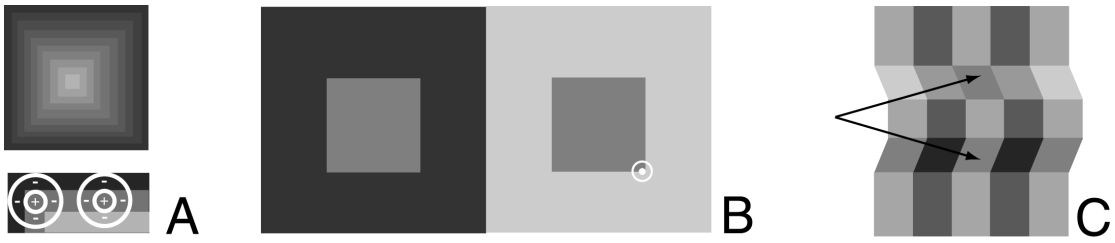

Fig. 24: Contextual influences on perceived brightness. (A) Top: A 2-dimensional variation on the Hermann grid illusion (Adelson, 1999). Bright lines are seen on the diagonals that have no physical correlate in the stimulus and can be explained by the surround-surround organisation of cells in optic nerve (Kuffler, 1953), LGN (De Valois & Pease, 1971) or of concentric cells in striate cortex (Spillmann, Ransom-Hogg, & Oehler, 1987). The inhibitory surrounds of cells centred on the corners receive less luminance than those centred on the sides (bottom). (B) Simultaneous contrast: The left central square is perceived as brighter than that on the right although both have the same luminance. This demonstrates that contextual effects occur over much larger distances than the receptive field sizes of retinal and LGN cells (circle). (C) Corrugated plaid effect (Adelson, 1993, 1999): Interpretation of reflectance characteristics, geometry and lighting condition leads to a perceived difference in brightness between the two surfaces indicated by the arrows although both are isoluminant.

Whereas contextual modulation of perceived brightness over small spatial scales (as in Fig. 24A) can be accounted for by the receptive field properties of the optic nerve, of LGN and striate cortex of cats (Spillmann et al., 1987; Syrkin, Yinon, & Gur, 1994) and LGN of monkeys (De Valois & Pease, 1971), simultaneous contrast, integrating over larger areas of the visual field, was not found to operate at that early level, possibly due to the small size of receptive fields (De Valois & Pease, 1971; Rossi & Paradiso, 1999). Several studies have been performed on the neurophysiology of surround influences on striate responses to luminance. Rossi and coworkers (1996) demonstrated that surround modulation of cat striate cortical responses to luminance shows a complex profile similar to human perception. They used a dynamic variant of the simultaneous contrast stimuli in Fig. 24B. Luminance of either surround or surround was sinusoidally modulated (Rossi & Paradiso, 1999; Rossi et al., 1996). When the luminance of the surround is sinusoidally modulated both perception and luxotonic cells correlate in-phase with this modulation (Fig. 25A). If the surround luminance is modulated and the surround luminance remains at a medium grey level, both perceived brightness of the centre and luxotonic cells respond in counter-phase this modulation (Fig. 25B). This is due to the fact that a grey stimulus on a dark surround is perceived to be brighter than on a bright surround. If the centre is dark surround modulation neither leads to a change of perceived brightness of the centre nor to a modulation of responses of luxotonic cells (Fig. 25C)[54].

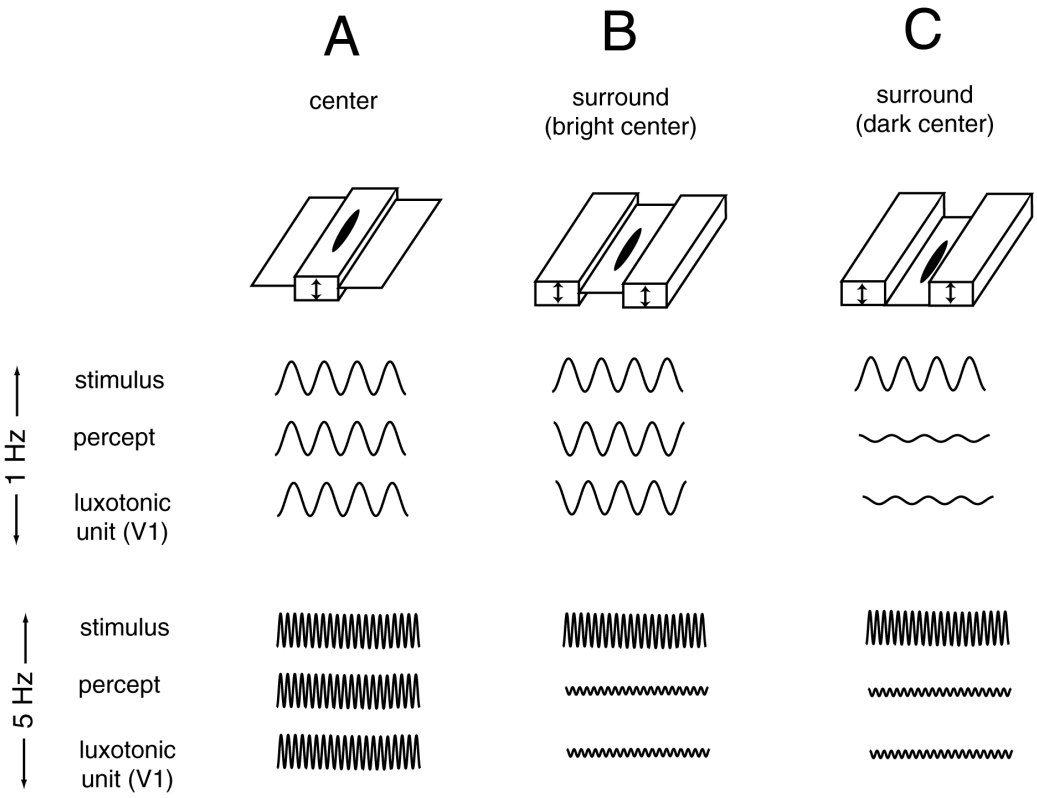

Fig. 25: Close physiological correlation between contextual modulation of perceived brightness and responses of luxotonic units in cat primary visual cortex (Rossi & Paradiso, 1999; Rossi et al., 1996). The top row shows the stimulation applied. (A) Central stimulation of an area covering the receptive field (dark ellipse) and its immediate surround. The luminance in that area is modulated sinusoidally at 1 Hz and 5 Hz as shown in the rows labelled "stimulus". The surrounds are kept at a constant medium luminance level ("grey"). Human observers perceive a sinusoidal modulation of the centre at both frequencies and in-phase with the stimulation. Luxotonic units in cat V1 closely parallel human perception. (B) Sinusoidal stimulation of the surround only where the centre remains at a constant medium luminance. At 1 Hz human observers perceive the centre to change brightness in counter-phase with the surround modulation. The same holds for luxotonic cells in V1 (note that both show the same phase-shift). At 5 Hz both the perceptual as well as the luxotonic responses are absent due to the low-pass characteristics of the effect (Rossi & Paradiso, 1996, 1999; Rossi et al., 1996). (C) If the centre is kept at a very low constant luminance level ("black") the perceptual and physiological responses are absent at both frequencies.

In a follow-up study MacEvoy and Paradiso (2001) could even demonstrate lightness constancy for luxotonic units using achromatic Mondrian stimuli composed of multiple rectangles with different luminances, similar to those used to study colour constancy (Fig. 26). If luminance values in the whole display (including the square covering the cell's receptive field) were changed multiplicatively in accordance with an overall increase in illumination the cell's response remained practically constant,

as does the perceived brightness of the square. If however only the luminance in the square covering the receptive field was changed the cells clearly responded with a change in spike rate. This provides evidence for single-valuedness, because physically very different luminances elicit identical V1 responses when they are perceived to be equally bright.

There are several other studies showing a close relationship between brightness perception and striate cortical activity. Some authors have demonstrated a close relationship between monkey psychophysics and striate physiology for brightness filling-in of dark fields covering the blind spot (Komatsu et al., 1996)[55]. There is also a close relationship between the perception of luminance flicker and striate activity, which is more controversial and will thus be discussed in a separate chapter[56].

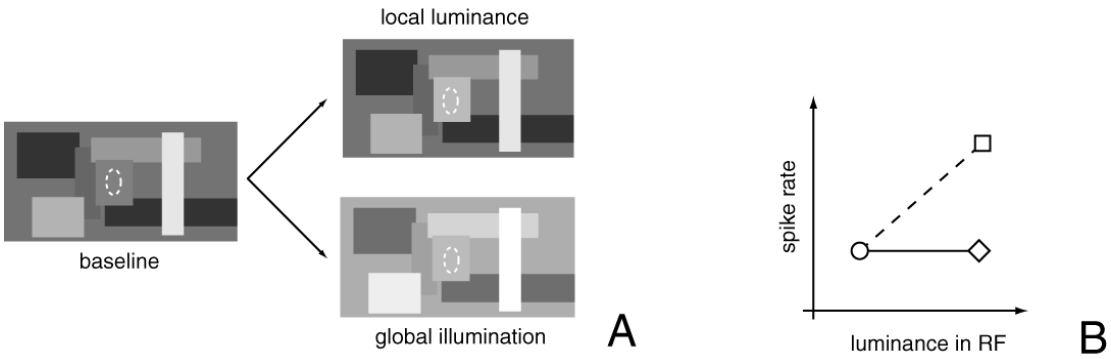

Fig. 26: (A) Achromatic Mondrian stimulus. The local luminance change stimulus was obtained from the baseline stimulus by increasing the luminance of the central rectangle only. The global illumination stimulus was obtained by multiplying all luminance values with a constant factor as would occur in case of a change in global illumination. (B) Schematic responses of a luxotonic cell with a receptive field centred on the central rectangle (dashed ellipse in A). Circle: Response to the baseline stimulus. Square: The increase of local luminance alone leads to an increase in spike rate. Human observers perceive the central rectangle to become lighter. Diamond: The same increase in local luminance that does not lead to an increase in perceived lightness (because it is perceived to be the consequence of a global change in illumination) does not lead to an increase in spike rate (MacEvoy & Paradiso, 2001).

Visual field deficits

Lesions to striate cortex lead to visual field deficits that show a close retinotopic correlation to the region destroyed (Holmes & Lister, 1916; Horton & Hoyt, 1991b;

Kitajima et al., 1998; McFadzean, Brosnahan, Hadley, & Mutlukan, 1994; Spalding, 1952). These visual field deficits range from small localised scotoma to the complete loss of left or right visual hemifield (hemianopia). In these parts of the visual field any conscious perception of brightness, contrast and form is lost[57] and cannot even be artificially induced by TMS stimulation anywhere over the visual cortex (Cowey & Walsh, 2000). Many early studies on visual field deficits lack a detailed neuroanatomical localisation (e.g. Spalding, 1952) and many recent studies report lesions that are not confined to a single cortical lesion but also involve extrastriate visual areas (Barbur, Watson, Frackowiak, & Zeki, 1993; McFadzean et al., 1994). Using high-resolution anatomical imaging methods such as CT and MRI it has recently been possible to demonstrate that highly isolated lesions confined to V1 cause localised scotoma (Horton & Hoyt, 1991b; Kitajima et al., 1998; Spector, Glaser, David, & Vining, 1981) (Fig. 27). Patients who have lesions to V4, MT or even the complete temporal lobes[58] lose specialised high-level visual perception but not their capacity for perceiving low-level visual features (Gallant et al., 2000; Huxlin & Merigan, 1998). Spared perception of low-level features after lesions to these areas has also been demonstrated in monkeys (Merigan, 1996; Schiller, 1993). Lesions to V2 and V3 can also lead to visual field deficits, which often affect exactly one quadrant of the visual field (Horton & Hoyt, 1991a; Jones, Waggoner, & Hoyt, 1999; McFadzean & Hadley, 1997). In a study by Merigan and coworkers lesions of V2 in monkeys did not lead to a blind region. Injections of ibotenic acid in V2 did not lead to significant changes of visual acuity and contrast sensitivity. Lesions to V1 on the other hand severely disrupted visual acuity in the position of the visual field represented in the damaged cortex (Merigan, Nealey, & Maunsell, 1993).

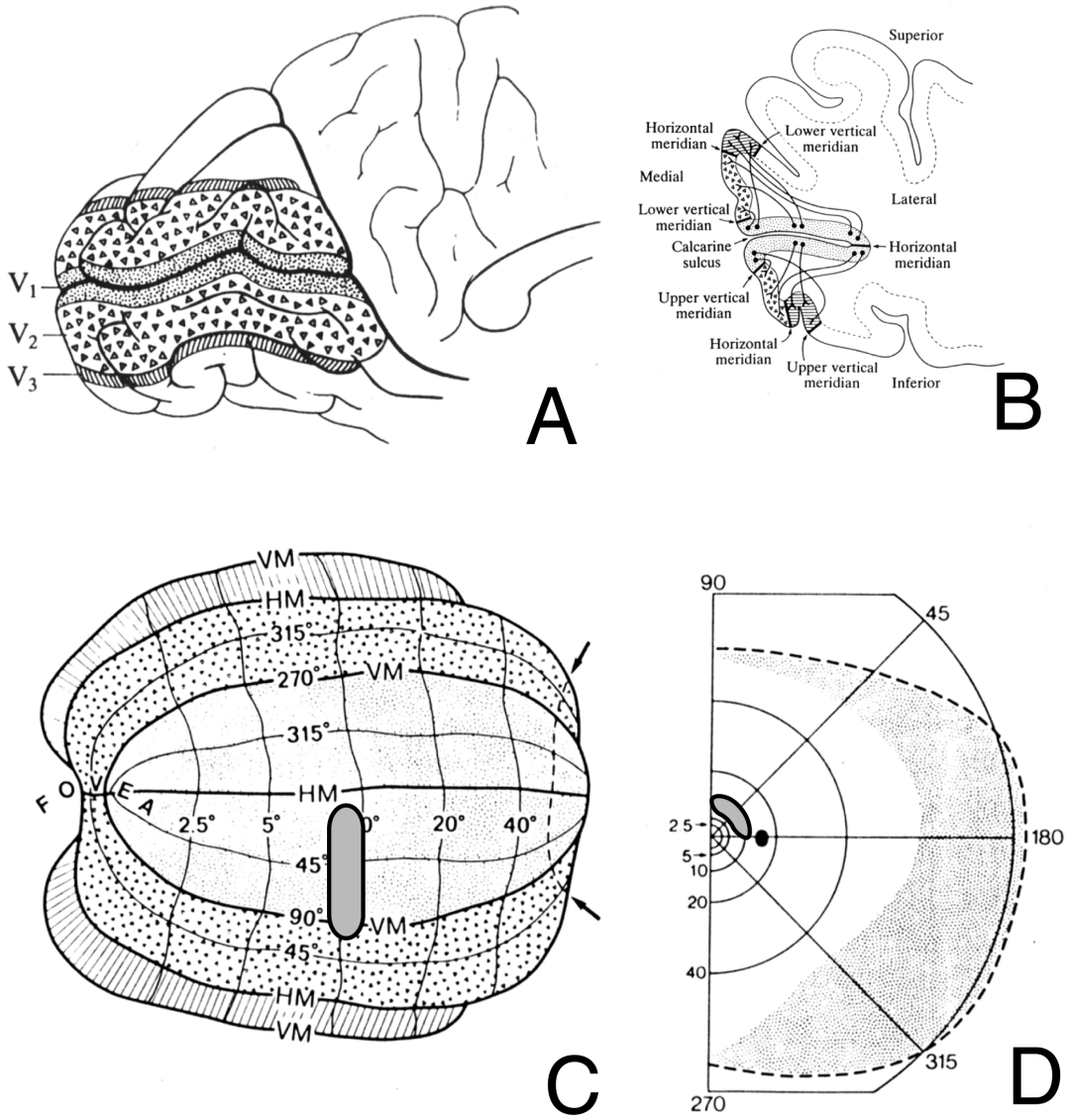

Fig. 27: Retinotopic map of a right visual hemifield in left striate cortex. (A) Location of V1 (stippled), V2 (triangles) and V3 (hatched) along the calcarine sulcus, cuneus and lingual gyrus. V1 is only partly visible because its main extent lies within the depth of the calcarine sulcus, but it becomes visible in a coronal section (B). (C) Shows a detailed map of the visual hemifield onto the polar-coordinate representation in a flattened striate cortex. Eccentricity is mapped as the depth in the interhemispheric fissure. The fovea is at the occipital pole and the periphery is at the crossing between calcarine sulcus and parieto-occipital sulcus. Angle is mapped as shown in B and C: The horizontal meridian is in the base of the calcarine sulcus. The lower vertical meridian is on the lower cuneal surface and marks the border between V1 and the lower right quadrant of V2. The upper vertical meridian is mapped to the upper lingual gyrus and marks the border between V1 and the upper right quadrant of V2. The foveal region occupies a far larger region of cortex than the periphery: The central 10° are mapped to approx. 50% of the striate cortex. The arrows indicate the border between representations of the binocular and monocular (peripheral) field. The grey inset in (D) shows a scotoma after a selective lesion to the region highlighted in (C). The lesion was localised using anatomical T1-weighted MRI scans (modified from Horton & Hoyt, 1991a, 1991b).

TMS over V1 disrupts brightness perception

Brightness processing in V1 can also be disrupted by TMS (Amassian et al., 1989). In the typical experiment a foveal target is presented and after a variable delay a TMS pulse is given. If the pulse occurs within a window of around 60 to 140 ms after stimulus onset (Fig. 28A) the perception of the target is severely degraded ("blurred") or it is even completely invisible. Interestingly the disruptive effect can even be used to make stimuli *visible*: In a backwards-masking setting where a target is followed by a mask after 100 ms the target is rendered invisible. If however a TMS pulse is applied in a time windows estimated to disrupt the V1 processing of the *mask* (approx. 120 ms after the mask and 220 ms after the target) the target is again visible (Fig. 28B). The time window in which performance is deteriorated is typically considered to reflect disruption of the striate stage of feed-forward visual processing of the target or mask. However recent experiments have revealed a further, earlier time window around 30 ms during which also disrupts visual processing (Corthout, Uttl, Walsh, Hallett, & Cowey, 1999; Corthout, Uttl, Ziemann, Cowey, & Hallett, 1999)(Fig. 28C). This is interpreted as interfering with the earliest afferent volley reaching V1 through the geniculostriate pathway and is in accord with earliest onset latencies in primary visual cortex (Bullier, 2001). The second disruptive time window around 60 to 140 ms with a much stronger detrimental effect could be operating at a *feedback stage* of visual processing (Lamme et al., 1998), suggesting that feedback from extrastriate activity is necessary for the subject to become aware of the target stimulus. These late effects also match the properties of luxotonic cells (Kinoshita & Komatsu, 2001) and of metacontrast masking (Bridgeman, 1975, 1980; Macknik & Livingstone, 1998).

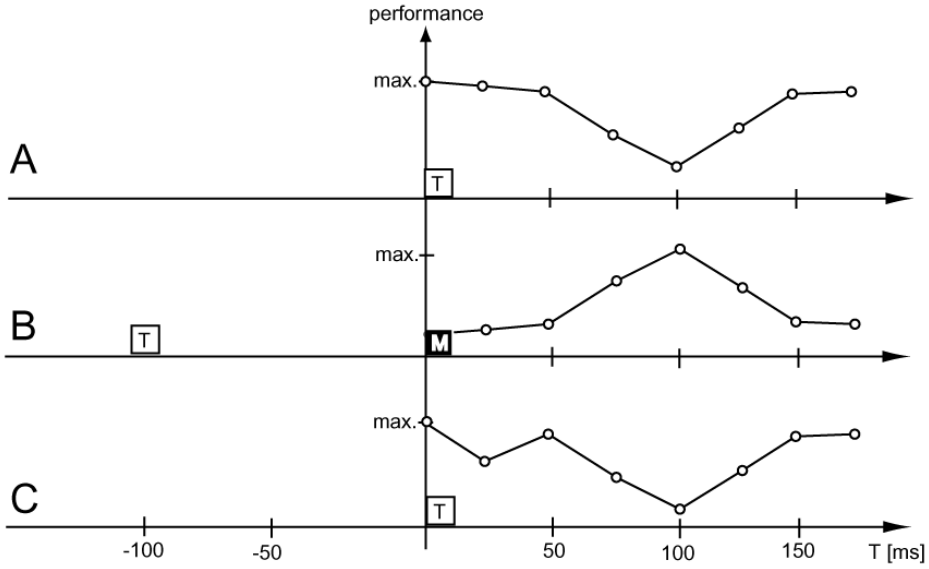

Fig. 28: Disruptive effects of a TMS pulse applied over the occipital pole on target perception as a function of time between target and magnetic pulse (reproduced after Amassian et al., 1993). (A) A target ("T", for example a set of characters or a light flash) is presented at T = 0 ms. The performance rate (e.g. rate of correct detection) remains at maximum for pulses with onset simultaneously to the target up to approx. 80 ms. If the pulse is given between 80 ms and 140 ms after target onset (depending on stimulus intensity and type of stimulus used) the performance drops due to cortical disruption of target processing. If the pulse occurs beyond approx. 140 ms after target onset the performance is not altered. (B) Unmasking of backwards masking: Recognition of a target presented at –100 ms is disrupted by a backwards pattern mask ("M") occurring at 0 ms and performance is low. If a magnetic pulse is given around 80 to 140 ms after *mask* onset the cortical processing of the *mask* is disrupted and the target is perceived - i.e. the performance increases ("unmasking"). (C) Some authors (Corthout, Uttl, Walsh et al., 1999; Corthout, Uttl, Ziemann et al., 1999) have observed an early and a late disruption time window, possibly revealing two stages of processing in primary visual cortex (Lamme et al., 1998).

The effect of the pulse has been shown to have a *graded* effect on perceived brightness and the disruption can be compensated by increasing stimulus intensity (Fig. 29A). There is strong evidence that the same mechanisms underlie disruptive effects and phosphenes induced by TMS and that both occur at a very early retinotopic level of processing. Typically phosphenes and transient visual field deficits co-occur. If the position of the coil is kept fixed and subjects are first asked to indicate the location of the phosphene in the visual field and then undergo perimetric threshold measurements it can be demonstrated that VFD and phosphenes share the

same locations in the visual field (Fig. 29B) (Kammer, 1999; Kastner, Demmer, & Ziemann, 1998).

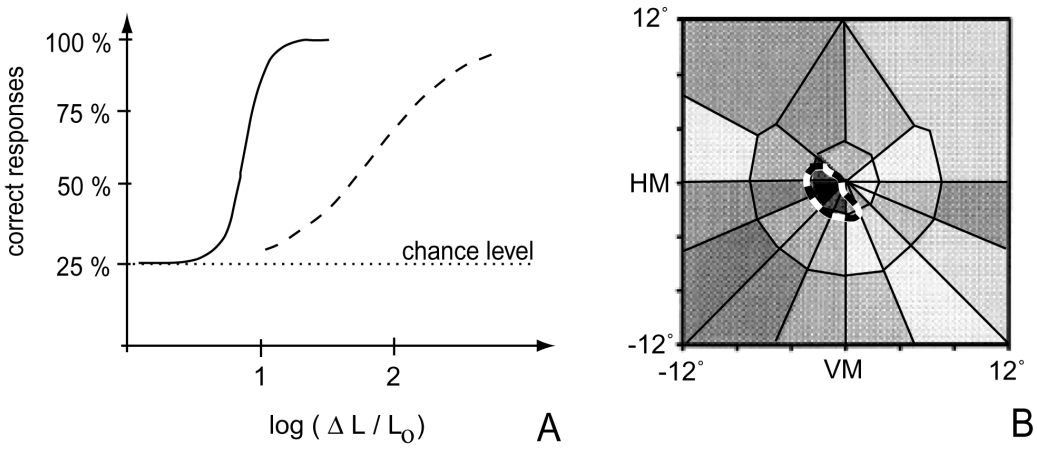

Fig. 29: Psychophysical data redrawn from two studies by Thomas Kammer (Kammer, 1999; Kammer & Nusseck, 1998). (A) The proportion of trials in which the orientation of a U-shaped target was correctly identified as a function of contrast. The identification threshold was clearly shifted upwards for targets followed after 120 ms by a magnetic pulse (dashed line) versus targets without pulse (solid line). This result shows that the TMS pulse has a graded effect on the neural representation rather than completely disrupting processing. (B) The co-location of perceived phosphenes (black and white dashed line) and threshold enhancement ("graded" scotoma) for a fixed position of the coil. The shading of the segments indicates the threshold increase by a TMS pulse for each position in the visual field. The dark shaded location with the highest increase in threshold is at the same position as the phosphene (HM = horizontal meridian; VM = vertical meridian).

Summary

Primary visual cortex has a subpopulation of cells that closely covaries with luminance changes and with a number of perceptual phenomena known to influence perceived brightness. This covariance is weak for early, transient phases of V1 single-cell responses but strong for later, sustained phases. Responses of luxotonic cells also exhibit isomorphism with perceived brightness in the sense that stimuli that are perceived to be brighter also lead to higher response amplitudes in "bright" cells and reduced responses in "dark" cells. Furthermore V1 signals are the same when the same brightness perception is achieved with very different luminances, thus the mapping between brightness and responses in V1 luxotonic cells also exhibits single-

valuedness. Little is known about the grain of brightness representation. V1 is strongly necessary for brightness perception, as shown by the visual field deficits following lesions and TMS pulses. The role of V2 in perceived brightness is not yet clear, because too few studies have attempted to record V2 responses to homogenous luminance changes. However the fact that electrocortical stimulation of V2 typically leads to more complex sensations implies that it does not represent the fine spatial grain of perceived brightness, whereas this is known to be the case for V1.

Contrast perception

Contrast is a low-level perceptual dimension of intensity that has been extensively studied. Contrast can be defined as the difference in luminance between the brightest and darkest regions in a visual stimulus divided by the mean luminance. This means that contrast defines the modulation depth as a proportion of background luminance (Fig. 30). A considerable body of data is available, both on the neural processing of contrast and on psychophysical aspects of contrast perception. Due to the receptive field organisation of cortical cells luminance contrast has received more attention than luminance. Especially cells in primary visual cortex are often modelled as indifferent to luminance and only responsive to luminance contrast[59]. The abundance of data available makes contrast a very suitable dimension for studying the representation of *perceived* magnitude.

Physiological responses to luminance contrast

Most visual areas respond to increases in contrast with monotonous increases in activity (Boynton et al., 1999), however with quite distinct profiles. Whereas magnocellular cells in LGN and cells in V3 and MT have a high contrast gain and saturate at low contrasts cells in parvocellular layers of LGN and in striate cortex have a low contrast gain and give incremental response changes right up to the maximum contrast of 1 (Fig. 31).

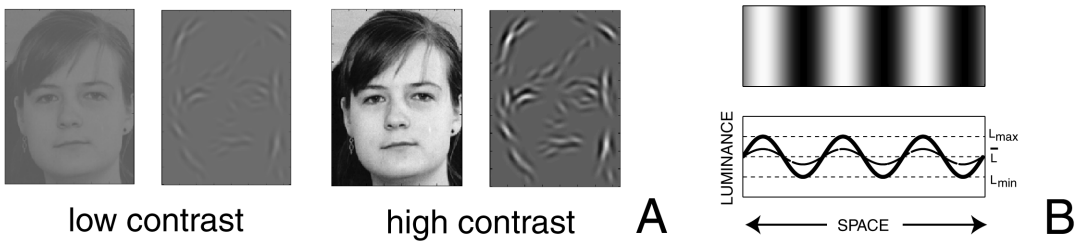

Fig. 30: Definition of luminance contrast: (A) Two pictures of a face with low contrast and high contrast. Note how only the luminance modulation depth but not the mean luminance changes. The two stripy pictures are obtained by plotting for each position the Gabor patch with an orientation that correlates strongest with the stimulus. (B) Luminance profile of a sine-wave grating. The top picture shows a 2-D image and the bottom picture shows the luminance profile of a horizontal line through it. The bold line shows a high contrast and the thin line a low contrast stimulus. Michelson contrast is defined as the difference between maximum and minimum contrast in the picture divided by the sum of the two.

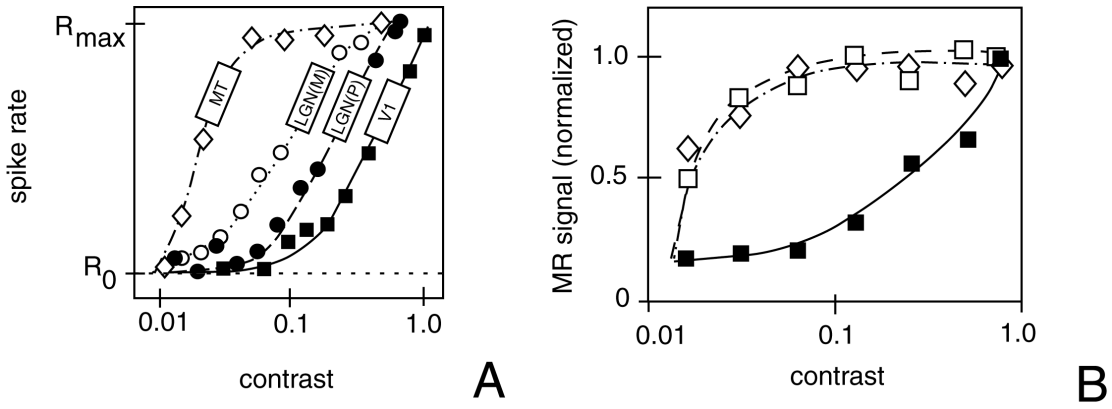

Fig. 31: (A) Responses of various macaque visual areas to stimulus contrast. Open circles, dotted line: LGN magnocellular layers; closed circles, dashed line: LGN parvocellular layers; filled squares, solid line: V1 simple cell; diamonds, dash-dotted line: MT (adapted from Sclar, Maunsell, & Lennie, 1990). R_0 refers to baseline spike rate and R_{max} to maximally saturated spike rate. (B) Responses of 3 human visual areas to stimuli of varying contrast. Filled squares, solid line: V1; diamonds, dash-dotted line: MT; open squares, dashed line: area V3 (redrawn fromTootell et al., 1998).

Striate cortex shows a biphasic dependency on contrast, which is best visible when plotting the contrast responses on a linear axis (Fig. 32A and Fig. 32B, dashed line).

For low contrast levels (just above absolute threshold) the response increases very fast with changes in contrast. In this region the response can be described by a power function rising with an exponent above 1 (accelerating branch). In the high contrast range this gives way to a second phase where contrast responses increase to an exponent below 1 (compressive branch).

Various model functions have been fitted to the contrast-response functions (Fig. 32A), but the model that captures the two branches best is typically achieved using a variant of the so-called hyperbolic ratio function (Albrecht & Hamilton, 1982; Boynton et al., 1999; Boynton, Engel, Glover, & Heeger, 1996; Geisler & Albrecht, 1997; Li & Creutzfeldt, 1984):

$$R(C) = R_{\max} \frac{C^{p+q}}{C^q + \sigma^q} \qquad (1)$$

where R(C) denotes response at contrast C, R_{max} is the maximum response and p, q are two parameters defining the slopes in the two branches. If $C << \sigma$ the denominator is dominated by the constant σ^q and the function rises approximately to the power p + q. If $C << \sigma$ the denominator is dominated by C^q and the function rises approximately to the power p. The constant σ^q is called the "semisaturation constant".

Fig. 32B compares the "average" striate single cell contrast response to a measure of a population response. The dashed line shows the hyperbolic ratio function obtained from monkey V1 by averaging the parameters of hyperbolic ratio function fits across 98 cells (Albrecht & Hamilton, 1982). This standard V1 single cell contrast response function saturates around 50% above which it would not be possible to give graded responses to contrast. The dotted line on the other hand shows the response of one human subject's V1 as measured using BOLD-fMRI. Clearly this response also shows compression in the high-contrast range but it still retains a graded response to contrast. This discrepancy between average single-cell and fMRI data can be explained by the considerable variability of single-cell contrast response functions. Fitted semisaturation constants vary between 1 and 40 and the exponent q varies between 0 and 8 (Albrecht & Hamilton, 1982). Thus, each cell has its dynamic range

(the steep part of the contrast response function) at a different contrast and can thus contribute with a graded response to different contrast ranges of the population response. Interestingly there is no difference between the contrast response functions of simple and complex cells (Albrecht & Hamilton, 1982).

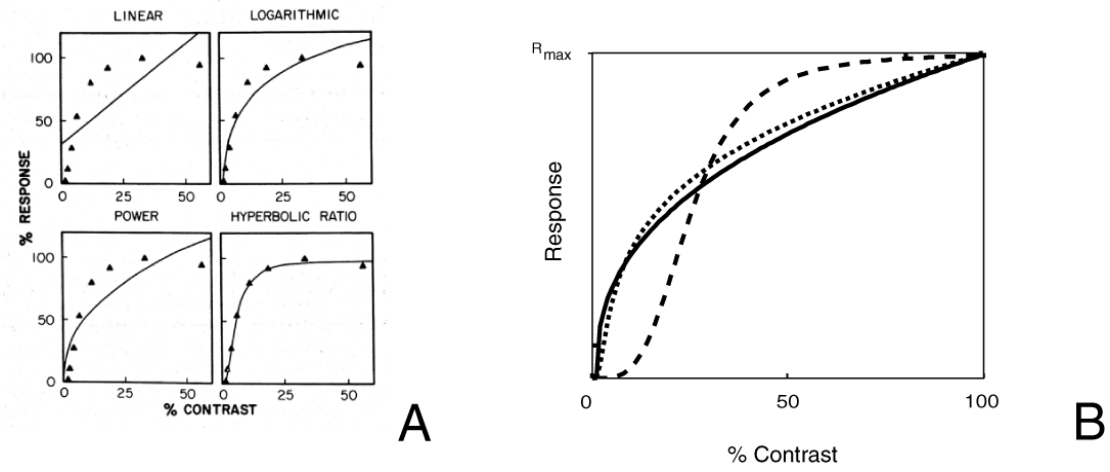

Fig. 32: (A) Fit of various model functions to the contrast response function of a striate simple cell (taken from Albrecht & Hamilton, 1982). The hyperbolic ratio function gave the best fit with the data. Note that the apparent difference between the responses of striate cells in Fig. 31A is due to the logarithmic versus linear scaling of contrast. (B) Contrast representation in V1 calculated as the hyperbolic ratio function with parameters averaged across 98 monkey cells (dashed line, data taken from table 5 in Albrecht & Hamilton, 1982), the fMRI response of one human subject (dotted line, taken from table 1 in Boynton et al., 1999) and the hypothetical contrast transducer function obtained from behavioural data which is used to explain human contrast discrimination performance (solid line, taken from Legge & Foley, 1980).

V1 can account for contrast discrimination

It has long been noted that there is a close relationship between contrast discrimination and responses in V1. In the prototypical study an observer is confronted with two stimuli, one with a baseline or pedestal contrast and one with a slightly higher contrast. He has to judge, which of the two stimuli has the higher contrast (Fig. 33, top left). The contrast difference that is necessary for the observer to reach a criterion proportion of correct responses is called the increment threshold.

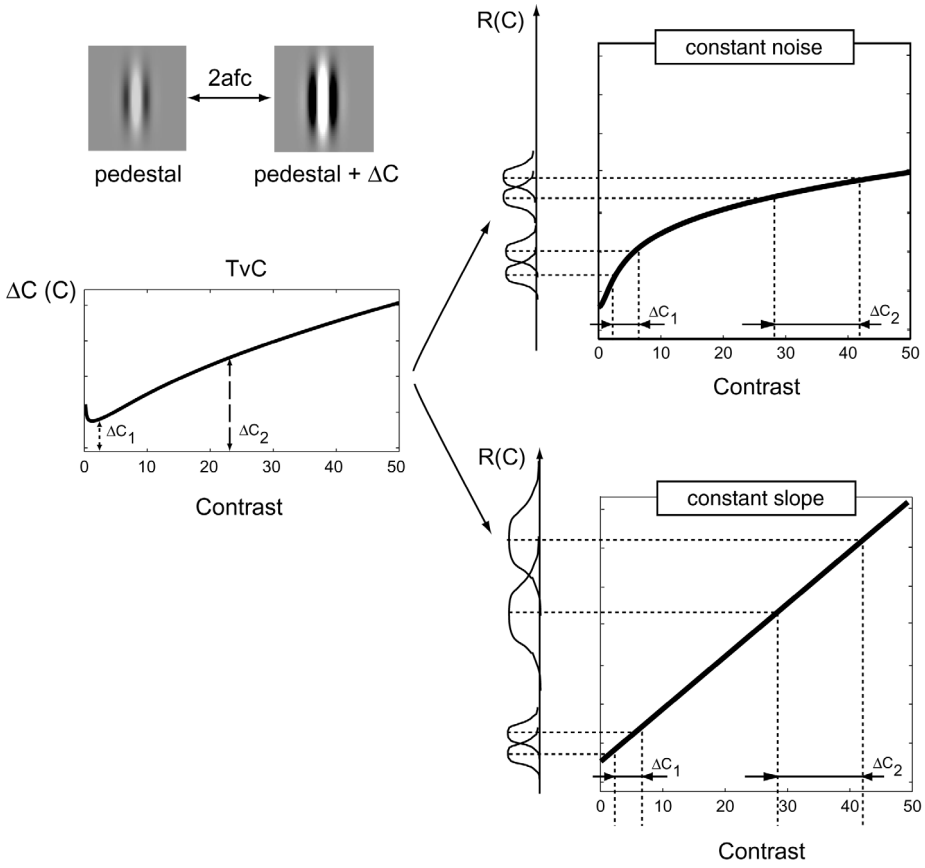

Fig. 33: Schematic relationship between neural response amplitude and discrimination performance. Two extreme models are shown: Top right: the increase in increment threshold (left) with contrast is accounted for by changing the slope of the nonlinear contrast transducer function but keeping noise constant (top right). Bottom right: The change in increment threshold is accounted for by a change in noise where the transducer has a constant slope. Most psychophysical models assume the constant noise model (Boynton et al., 1999; Foley, 1994; Legge & Foley, 1980; Zenger-Landolt & Koch, 2001).

Weber was one the first to study increment thresholds and he postulated a constant ratio between increment threshold and baseline magnitude (Weber, 1834). The Weber-fraction

$$\frac{\Delta C}{C} = k \qquad (2)$$

was thought to be a constant (C denotes physical stimulus intensity). This is clearly not the case for contrast data. The increment threshold ΔC does not rise linearly with baseline contrast (as would be expected from transforming the equation to ΔC = kC), but slower and also has a "dip" in the range of low contrasts (Fig. 33, middle left).

Contrast discrimination performance can be explained by a very simple model. It is assumed that each stimulus (C and C+ΔC) results in a physiological response and that a correct discrimination can be made if the two physiological responses differ by a criterion amount. The imperfection of not being able to discriminate infinitesimally small differences can be explained by assuming that the physiological response is obscured by additive noise resulting in two random response distributions with different mean (due to the contrast difference) but same dispersion. A criterion performance in discrimination is reached when there is a specific separation of the two distributions (Fig. 33, top right). The subject will make a certain number of errors due to the fact that in a fixed proportion of trials the weaker stimulus will evoke a stronger response because of the additive noise. By measuring discrimination thresholds at different contrast levels it is possible to construct a hypothetical nonlinear *contrast transfer function* (CTF) whose slope decreases with contrast in order to account for the increase in increment threshold with contrast. It is assumed that the luminance distribution of the stimulus is first *linearly* multiplied with the filter profile of each cell or psychophysical "channel". Then the output of this linear stage is passed through this nonlinear CTF (reviewed in Olzak and Thomas 1999). If the slope of the CTF becomes shallower more contrast increment is required to achieve the same response increment. Thus the threshold rises with contrast because discrimination performance is proportional to one over the slope of the CTF. Under the assumption of constant noise it is thus possible to calculate the profile of the CTF from the discrimination data.

If however the assumption of constant noise is dropped the problem becomes ambiguous. In the extreme case an increase in threshold can be interpreted as due to either a decrease in slope at constant noise or due to an increase in noise at constant slope (bottom right). All that can be known is that the response distributions have fixed overlap, which can be achieved by an infinite number of different combinations of mean and distribution. At the level of single cells response variance is roughly proportional to mean response (Geisler & Albrecht, 1997; Tolhurst, Movshon, & Dean, 1983) and not constant. Little is known about response variance at the population level. Despite this fact most psychophysical models assume that noise is

constant and additive (Boynton et al., 1999; Foley, 1994; Legge & Foley, 1980; Zenger-Landolt & Koch, 2001), which is partly supported by recent psychophysical evidence (Gorea & Sagi, 2001).

The constant noise CTFs have striking similarities to contrast response functions recorded from monkey and human primary visual cortex that were presented above. The transducer is also modelled as a hyperbolic ratio function (equation 1). The parameters thus estimated show a very close match to striate contrast responses. Geisler and Albrecht (1997) investigated this issue in great detail and showed that behavioural performance could not well be explained by single cells but was best predicted by the entire population, either by the envelope of the most sensitive cells or by optimally pooled single cell responses. This comparison between psychophysical performance and single-cell data is partly limited because behavioural and physiological data were obtained in separate studies. The parameters p, q and σ depend on various stimulus features, such as spatial frequency, that are not matched between these studies. Contrast response functions measured with human fMRI also show great similarity to these transducers. Boynton and coworkers (1999) demonstrated that human contrast discrimination can be modelled by assuming that it is based on the fMRI contrast response of V1. Fig. 32B shows the close correspondence between the human fMRI contrast response function and a psychophysical contrast transducer function. Other studies have demonstrated that contrast discrimination performance can be accounted for by statistically pooling the responses of the entire population of single-cell responses in primary visual cortex (Geisler & Albrecht, 1997; Itti, Koch, & Braun, 2000).

Perceived contrast

In what follows the main question will be how a different perceptual measure, perceived magnitude of contrast, is related to these contrast transducer and contrast response functions. Whereas contrast discrimination is a purely local measure of perceived difference, perceived contrast is an absolute measure of perceived magnitude. Historically Fechner proposed a way to link increment thresholds to perceived magnitude (Fechner, 1860). He assumed that the difference in perceived magnitude between two stimuli differing by a single discrimination threshold or “just

noticeable difference" (JND) is equal, regardless of the baseline level at which it is studied. This can be stated as

$$\Delta\Psi = \Psi(C + \Delta C) - \Psi(C) \qquad (3)$$

where C denotes physical stimulus intensity, Ψ perceived magnitude and ΔC the increment threshold. Fechner assumed $\Delta\Psi$ to be constant.

This provides a linking hypothesis between contrast discrimination and perceived contrast. Sensation is proportional to the number of JNDs above absolute threshold. However, it is known that discrimination performance is not always a good indicator of a subjectively perceived difference between two stimuli (Kolb & Braun, 1995; Stoerig & Cowey, 1997). For example under certain conditions subjects can perform well on discrimination without any subjective confidence in the correctness of their judgements (Kolb & Braun, 1995). This indicates that just noticeable differences do not necessarily correspond to single "quanta" of perceived magnitude, but possibly to less[60].

A different linking hypothesis could be provided by the contrast transducer function. In this view the perceived contrast would be proportional to the amplitude of the CTF (Xing & Heeger, 2001). If one assumes constant noise then the contrast transducer function and Fechner's perceived magnitude have equal predictions. However, as shown above, the shape of the contrast transducer function strongly depends on the noise assumption. It is possibly to account for the increase in discrimination threshold with increased pedestal contrast either by assuming that the transducer function becomes shallower, or by assuming that noise increases. In the extreme case one could assume a linear contrast transducer function.

Given that there is no obvious linking hypothesis it is necessary to study perceived contrast directly. There are several direct approaches to the relationship between physical contrast and perceptual magnitude (Fig. 34). These measures give either a direct measure of contrast (luminance matching) or a measure of the contrast ratio between two stimuli (contrast halving, direct scaling). There are considerable

differences between perceived magnitude functions even when estimated using similar methods (reviewed in Georgeson, 1991). The relationship between perceived contrast has been reported to be a linear (Cannon, 1979; Kulikowski, 1976), logarithmic (Fiorentini & Maffei, 1973) or a power function (Cannon, 1985; Franzen & Berkley, 1975; Gottesman, Rubin, & Legge, 1981; Hamerly, Quick, & Reichert, 1977) with exponents varying as far as between 0.3 and 1.7. As Cannon has showed for direct scaling studies this may partly be due to range effects (Cannon, 1984). His more recent models of perceived contrast employ transducers which are also very similar to the contrast transducer functions and striate contrast responses mentioned above (Cannon & Fullenkamp, 1991b; Cannon, 1985). All have similar exponents in the high contrast range. A tempting view would be that perceived contrast depends on the mean and discrimination depends on the noise and slope of the same CTF.

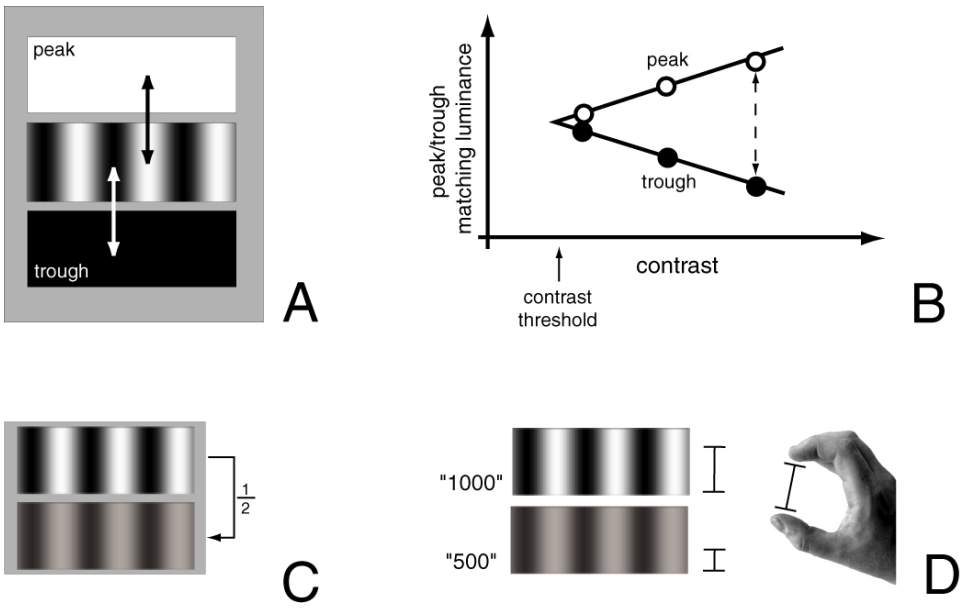

Fig. 34: Various methods used for direct estimation of perceived contrast. (A) Matching of perceived luminance of peaks and troughs with that of a standard stimulus with homogonous luminance (Bryngdahl, 1966; Fiorentini & Maffei, 1973). The peak and trough judgements are then plotted for different contrasts as shown in (B). For constant mean luminance the difference is a measure of perceived contrast (dashed line). (C) Contrast halving: The observer is required to set the adjustable lower grating to half the contrast of the top grating (Kulikowski, 1976). (D) Direct scaling using magnitude estimation (Stevens 1960): The observer is required to rate perceived contrast either by assigning a number (left) or by adjusting the finger span to the according value (right). The ratio between the judgements is believed to reflect the ratio between the perceived magnitude (Franzen & Berkley, 1975).

A few studies have directly assessed the relationship between neural response amplitude and perceived contrast. In a recent study Polonsky and coworkers (2000) studied binocular rivalry stimuli with gratings that differ in orientation and contrast between the two eyes. The subjects' perception alternates between high-contrast gratings of one orientation and low-contrast gratings of an orientation orthogonal to it. Responses in primary visual cortex closely correlated with these changes in perceived contrast, rising when perception switched to the high-contrast grating and falling when perception switched to the low-contrast grating.

Fiorentini and Maffei (1973) compared perceived contrast functions obtained using luminance matching (Bryngdahl 1966, see Fig. 34A,B) to steady-state VEPs taken from the literature (Campbell & Kulikowski, 1972). They found that both perceived contrast and steady state VEP amplitude showed a logarithmic dependency on physical contrast. Franzen and Berkley (1975) extended this using a direct scaling method. They fitted perceived contrast using power functions and found that the exponents of the power functions for perceived contrast and Campbell and Maffei's (1972) steady-state VEPs closely matched. However promising these two studies are they both bring up serious questions. First they are limited by the fact that psychophysical and electrophysiological data were gathered from different subjects. Other studies have shown a considerable difference between exponents of perceived magnitude functions for different subjects (e.g. Cannon 1985, table 1). Second it is difficult to determine the cortical generators of Campbell and Maffei's (1972) steady-state VEPs, especially as the authors only used a single pair of electrodes. Thus it remains unclear if their contrast-response functions reflect activity of primary visual cortex. The third and weakest point is that Fiorentini and Maffei's (1973) and Franzen and Berkley's (1975) perceived magnitude functions are very different compared to more recent models of perceived magnitude that take range effects into account (Cannon 1984, 1985). Thus it is unclear if the psychophysical side of their correlation holds up to modern views of perceived contrast.

To summarize, activity in V1 can explain contrast discrimination performance and there is some evidence that it might represent perceived contrast. Stimuli that are perceived to have a higher contrast also lead to higher response amplitudes in V1,

pointing towards an isomorphism between perceived contrast and V1 response amplitude. Furthermore V1 is strongly necessary for perceived contrast: The lesions to primary visual cortex discussed above that lead to complete blindness also lead to a loss of contrast perception. This has also been demonstrated for monkeys (Merigan et al. 1993; Cowey & Stoerig 1995). It has also been demonstrated above that TMS pulses over the occipital pole disrupt both luminance and contrast perception[61]. Other visual areas however do not seem to be strongly necessary for contrast perception. Extensive lesions to either ventral or dorsal stream can leave contrast sensitivity largely unaffected (Plant et al., 1993; Vaina, 1994). However to date there has been no demonstration that repeated occurrences of the same perceived magnitude of contrast lead to the same responses in V1, as has been shown for perceived brightness by the constancy studies of MacEvoy and Paradiso (2001).

Chapter 5

An empirical study of perceived contrast

Lateral masking and perceived contrast

Taken together there are several indications that V1 activity may represent perceived contrast, but the direct demonstration has not yet been made. Luckily there is also a direct route to assess whether striate activity correlates with perceived contrast that does not rely on the debated shape of perceived contrast functions. If one were able to dissociate physical contrast and perceived contrast one could test whether striate cortical responses are identical in situations where physically different stimuli appear to have the same contrast. Such an approach was followed by Goodyear and coworkers (2000). They studied one amblyopic patient with a monocular decrease in contrast perception[62]. The contrast of a stimulus presented to the pathological eye has to be increased for it to match the contrast of a stimulus presented to the normal eye. Using BOLD-fMRI they demonstrated that stimuli that were physically different but perceived to have the same contrast evoked the same responses in a region of interest spanning V1 and V2 (Goodyear, Nicolle, Humphrey, & Menon, 2000). Thus the mapping of perceived contrast to V1/V2 response amplitudes may exhibit single-valuedness, at least in one amblyopic patient.

One way to change perceived contrast independent of physical contrast in normal subjects is to use a phenomenon called “lateral masking” (Fig. 36). If a target grating is presented surrounded by a larger area of grating its perceived contrast is reduced (Cannon & Fullenkamp, 1991a, 1993; Chubb, Sperling, & Solomon, 1989; Ejima & Takahashi, 1985; Snowden & Hammett, 1998; Solomon, Sperling, & Chubb, 1993). This effect is known as “lateral masking” and it suggests that some mechanism for spatial pooling has to be incorporated to account for contrast perception.

Most studies on contrast discrimination and perceived contrast reported above have studied simple grating or Gabor stimuli that were spatially confined and were presented in a background field of homogenous grey. In the classical psychophysical view these stimuli should trigger a single visual channel, defined as a filter with a specific tuning to orientation, spatial frequency, phase and position. The increment threshold and perceived contrast data are interpreted as shown in Fig. 35A. The

spatial input pattern is multiplied with a filter profile to yield the response at the linear filter stage. This linear output is passed into a nonlinear contrast transducer function for each channel separately and then gaussian or poissonian noise is added (not shown).

At an early stage these channels are believed to be independent, but it has long been known that at further processing stages interactions between these channels occur. When stimuli consist of a superposition of components that optimally drive different filters (i.e. two superimposed Gabor patches with orthogonal orientations) the filters interact leading to changes in detection and discrimination thresholds (Itti et al., 2000; Olzak & Thomas, 1991; Tolhurst & Barfield, 1978). The observer's response can then not be explained by assuming that he has direct access to the independent low-level channels. Interactions occurring between superimposed stimuli mean that filters tuned to the same position in the visual field interact. This interaction is typically modelled as a broadband divisive inhibition, where each unit's response is divided by a normalisation term that depends on a weighted sum of the responses of differently tuned filters (Fig. 35B)(Foley, 1994; Itti et al., 2000; Olzak & Thomas, 1999).

Interactions are not restricted to filters at the same location in the visual field but also occur between filters that have no overlap but respond to nearby positions in the visual field. Detection and discrimination of Gabor patches can be modulated by placing other stimuli in their surround (Polat & Sagi, 1993, 1994; Snowden & Hammett, 1998; Zenger, Braun, & Koch, 2000; Zenger & Sagi, 1996; Zenger-Landolt & Koch, 2001). This effect can be modelled by including divisive inhibition from the immediate spatial surround into the model shown in Fig. 35A (Yu & Levi, 2000). It has also been shown that surround modulation may involve more than divisive inhibition and may also include subtractive inhibition (Zenger-Landolt & Koch, 2001).

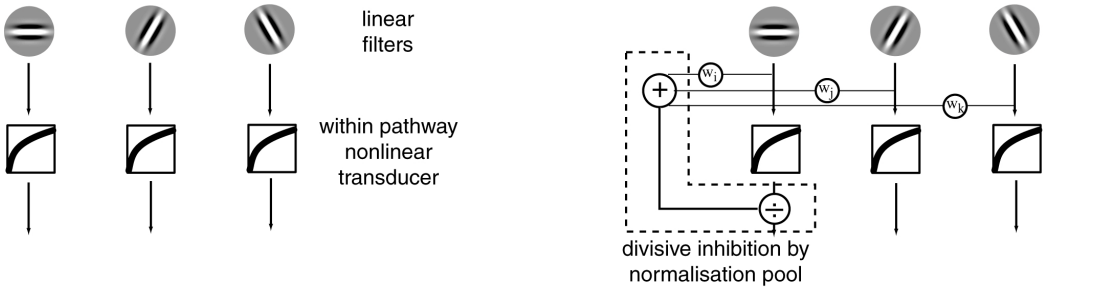

Fig. 35: (A) The "multiple channels, direct access model" (Olzak & Thomas, 1999). The input at a certain position in the visual field is multiplied with a set of independent linear filters tuned to different orientations, spatial frequencies and phases. The output is then independently transformed by a within pathway nonlinearity as shown in the previous chapter to account for differences in contrast discrimination at different pedestal contrasts. It was assumed that the observer's response can be directly based on the outcome of these independent channels. (B) A simple model incorporating interactions between channels. To account for interactions between overlapping spatial filters the output of one channel (shown here for the channel on the left which has a horizontal orientation preference) is divided by a weighted sum (w_i … w_k) of the outputs of filters tuned to other orientations, spatial frequencies and phases. To account for surround interactions the inhibition pool has to be extended to include input from spatial filters tuned to surrounding positions in the visual field.

Not just contrast discrimination but also perceived contrast is modulated by surrounding stimuli suggesting that it is also subject to surround normalisation (Cannon & Fullenkamp, 1996; Xing & Heeger, 2001)(Fig. 36). Numerous studies have revealed the key properties of this surround modulation of perceived contrast. The most important are: (1) Modulation depends on the contrast ratio between center and surround and not on absolute contrast although minor departures from this rule have been demonstrated (Cannon & Fullenkamp, 1993, 1996; Ejima & Takahashi, 1985; Snowden & Hammett, 1998; Xing & Heeger, 2001). Suppression is strongest for high-contrast surrounds and low-contrast targets. If the contrast ratio is inverted (i.e. the surround has a lower contrast than the target) the effect can occasionally be inverted, resulting in enhancement rather than suppression (Cannon & Fullenkamp, 1993; Ejima & Takahashi, 1985; Xing & Heeger, 2001). (2) Suppression is composed of a basic untuned effect and an effect tuned to a specific bandwidth of spatial frequencies, orientations and speeds (Cannon & Fullenkamp, 1991a; Solomon et al., 1993; Takeuchi & De Valois, 2000). The perceived contrast of the grating that is iso-oriented to the surround is lower than for that within an orthogonal surround. Thus by

varying relative orientation one has the possibility to modulate perceived contrast without locally varying stimulus contrast. (3) There is no binocular transfer when the target and surround are presented to different eyes (Chubb et al., 1989). The fact that the effect is on the one hand monocular and on the other hand orientation-selective seems to strongly suggest a role of layer IV in primary visual cortex (Solomon et al., 1993). On the one hand V1 is the last processing stage with substantial populations of cells with monocular dominance in visual cortex (LeVay, Hubel, & Wiesel, 1975). On the other hand it is the first stage of orientation-selective processing (at least in the feed-forward sweep) which is necessary to account for the orientation tuning of lateral masking.

Lateral masking allows one to perform a study similar to Goodyear et al. (2000) on normal patients and also introduce an additional constraint. It is possible to dissociate perceived contrast and physical contrast in a similar way as was done using the amblyopic patient. By choosing a target stimulus in a parallel surround and with fixed contrast as a "standard" stimulus, one can determine psychophysically how much contrast a stimulus in an orthogonal surround will require to match psychophysically (Fig. 36). The orthogonal matching stimulus will have a lower physical contrast because the reduction in perceived contrast is stronger for the parallel stimulus. This makes it possible to compare stimuli that either physically have the same contrast or are perceived to have the same contrast and assess whether V1 is a processing stage that correlates with physical or with perceived contrast.

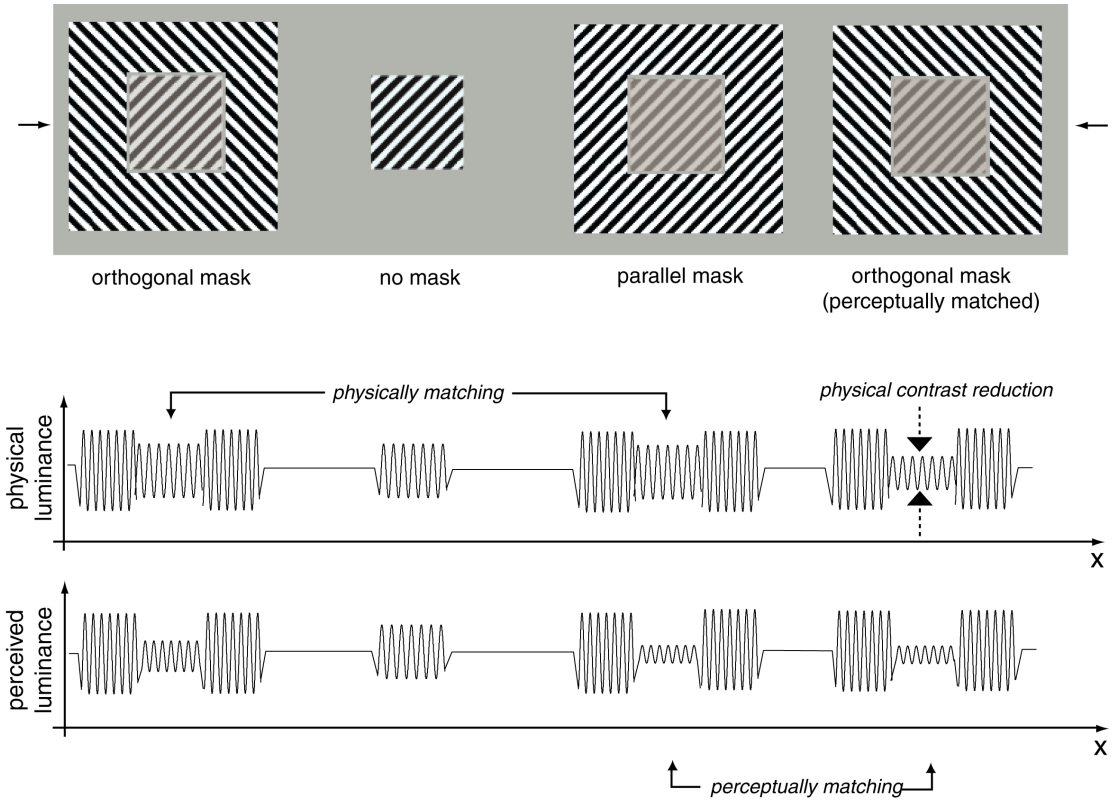

Fig. 36: (Top) Demonstration of the effect of lateral masking on perceived contrast. The stimulus in the middle left ("no mask") is a standard rect-wave grating with medium contrast. If the stimulus is surrounded by a high-contrast masking grating the contrast is perceived to be suppressed (see the 2 adjacent stimuli). The suppression is stronger for iso-oriented ("parallel mask") than for orthogonal surrounds ("orthogonal mask"). (The effect had to be artificially enhanced here due to the fact that the luminance transfer characteristics of the print are uncontrolled. This has several disrupting effects. First, the print does not provide a grey-scale resolution that is sufficient. Second, the mean luminance cannot be kept constant for the different areas, which introduces additional perceived *baseline luminance* changes due to the effect of simultaneous contrast. If the observer squints the eyes and views the stimulus at a certain distance the whole surface should appear in a homogenous grey, which it does not. The stimuli in the current study were presented with an LCD video projector and its luminance transfer characteristics were precisely controlled using a spot-photometer to calibrate the gamma correction). (Middle) This schematically shows the physical distribution of luminance in a section between the two arrows shown in the top stimulus. The physical contrasts (luminance modulation amplitudes) of the left three targets are equal. The rightmost stimulus shows an orthogonal target whose *physical* contrast has been *reduced* so that it *perceptually matches* the parallel target. (Bottom) Despite being physically identical the perceived contrasts are highest for the unflanked central stimulus, lower for the orthogonally flanked stimulus and lowest for the target with parallel surround.

Here a combined EEG and MEG study will be performed. This will allow comparing the individual components of the evoked responses for physically matching and for

perceptually matching stimuli. The responses from an area representing perceived contrast should be the same for perceptually matching stimuli. When comparing responses to the physically matching stimuli, the orthogonal targets should evoke larger responses because they appear to have higher contrast. On the other hand, if an area encodes physical contrast, the response to the parallel and orthogonal stimuli with equal physical contrast should be the same, and the response to the perceptually matching (but physically reduced) orthogonal stimulus should be reduced.

Several reasons have lead to the choice of EEG and MEG rather than functional magnetic resonance imaging (fMRI) for physiological recording[63]. First EEG and MEG are direct measures of neural activity. Both measure electrical and magnetic properties of currents that follow excitatory and inhibitory postsynaptic potentials (see below). Second, EEG and MEG allow to separate individual stages of processing in the various areas. In the review of perceived brightness it has been shown, that there could be two striate response phases, where the later phase correlates better with perception. To test this requires a temporal resolution that is not available in functional magnetic resonance imaging[64].

Brief review of EEG and MEG technology

Electroencephalography (EEG) and magnetoencephalography (MEG) both measure different electromagnetic consequences of excitatory and inhibitory postsynaptic potentials. Release of an excitatory neurotransmitter by the presynaptic cell into the synaptic cleft will lead to a local inflow of cations into the postsynaptic cell. This locally depolarises the postsynaptic cell from its resting potential of approx. –70 mV (relative to the extracellular space) and creates an excitatory postsynaptic potential (EPSP). Fig. 37A shows schematically the current flow after an EPSP in a spine on the apical dendrite of a typical cortical pyramidal cell. Within the immediate vicinity of the synapse there is an intracellular current source and an extracellular current sink. Because the cell membrane dividing source and sink has a high resistance and the intracellular fluid has a low resistance the current follows the path of lowest resistance and the EPSP spreads along the length of the dendrite down into the soma. There the intracellular space has a large cross-section and the membrane has a large surface

both contributing to an overall low resistance. Depending on the characteristic length of the cell[65] and its geometry (especially the distance between soma and synapse) a large part of the current takes the longer low-resistance path and flows back to the synapse via the soma. In the extracellular space the current flows in a more extended fashion and spreads over a larger volume. The circuit in Fig. 37A can be modelled in a simplified fashion as an electrical current dipole with a positive pole at the soma and a negative pole at the synapse (Fig. 37B, top)(de Munck, van Dijk, & Spekreijse, 1988; Sarvas, 1987).

The surface recorded EEG measures the electric potential between a small area on the scalp and a reference that should be chosen to be electrically "neutral"[66]. If one were able to measure the electric scalp potential generated by a single EPSP it would be strongly spatially blurred by the high resistance of the skull. This means that even if the surface of the electrode was reduced to a single point it would still record the activity averaged over of a large number of neurons in the underlying cortex. The typical scalp electrode measures the space-averaged activity of more than 10^7 neurons (Nunez, 1981). If the synaptic events in these neurons occurred randomly in time and with random polarity the surface recorded potential would be zero due to this spatial averaging. Thus some degree of synchronicity of the occurrence of EPSPs or IPSPs is necessary for a "population potential" to build up and exceed the noise level created by background synaptic events.

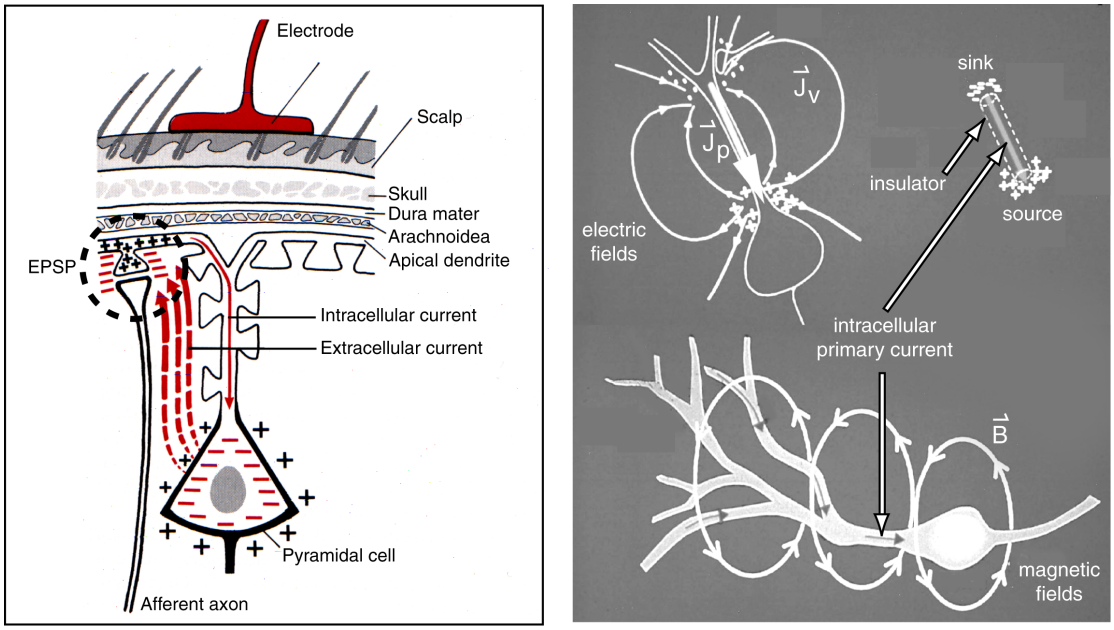

Fig. 37: Idealised model of cortical generators of surface EEG and MEG signals. (A) The surface recorded EEG and MEG is believed to be largely generated by EPSPs or IPSPs occurring synchronously in large populations of cortical pyramidal cells. Pyramidal cells are oriented orthogonal to the surface of the cortex, have long apical dendrites with few branches and thus dendritic EPSPs and IPSPs can lead to an optimum separation of current source and current sink. (B) Top left: Schematic view of A with the intracellular "primary" current density J_p and the extracellular "volume" current density J_v. Extracellularly the soma acts as a current source and the synaptic region acts as a current sink. Bottom: The intracellular primary current generates a concentric magnetic field B. The local magnetic fields generated by extracellular currents are largely cancelled out. Top right: The circuit can be modelled by an equivalent current dipole (de Munck et al., 1988; Hamalainen, Hari, Ilmoniemi, Knuutila, & Lounasmaa, 1993; Sarvas, 1987) which gives a good approximation of the main electric and magnetic properties of the real circuit and can also be used to model small cortical areas with adjacent, co-aligned neural dipoles (Nunez, 1981). If the electric and magnetic properties of the individual brain compartments are known and also the position, orientation and strength of the dipole, the surface recorded electric and magnetic fields can be uniquely calculated. The current dipole model consists of an intracellular current that is shielded from extracellular space by electrical insulation (infinite resistance). It has a current source on one end and a current sink on the other end.

MEG measures a different biophysical property of the circuit shown in Fig. 37A. Each current flow is accompanied by a magnetic field the direction of which is tangential to the current and can be estimated by the right hand thumb rule (Fig. 37B, bottom). The sensory evoked magnetic fields recorded at the scalp measure around 5 to 500 femtotesla (10^{-15} T) and are thus very weak. The earth's static magnetic field is

stronger by a factor of around 10^9 and the dynamic environmental noise is stronger by a factor of around 10^6 (Hamalainen et al., 1993; Hari, 1993; Malmivuo & Plonsey, 1995). Thus, the measurement of neuromagnetism is confronted with two severe problems: The low signal strength and the low signal to noise ratio.

Extremely weak magnetic fields can be recorded by superconducting quantum interference device sensors (SQUIDs) which transform magnetic flux into voltage and have a very high sensitivity (Brenner, Williamson, & Kaufman, 1975). They are kept below their critical superconducting temperature by liquid helium at –269°C. The cerebral magnetic field is not directly picked up by the SQUIDs but by a set of superconducting induction coils (flux transformers) whose axes are roughly orthogonal to the surface of the head and each of which have a second coil coupled to a SQUID (Brenner et al., 1975; Hamalainen et al., 1993). In the BTI MEG system used for the current study 148 such induction coils ("sensors") are fitted into a "helmet" into which the subject's head is inserted (Fig. 38A).

The severe background noise problem is encountered by using magnetically shielded recording chambers (Fig. 38A, MS). It is also possible to use reference sensors placed at a distance from the receiving coil in the recording chamber that can be used to subtract magnetic fields generated by distant sources. Magnetic field strength falls off as the square of distance. Distant magnetic fields induce virtually identical currents in the recording and reference coils giving no net output when both are subtracted. Magnetic fields due to sources near to the recording coil will induce a considerably larger current in the recording than reference coil leading to a net output. This procedure reduces the noise from distant irrelevant sources[67].

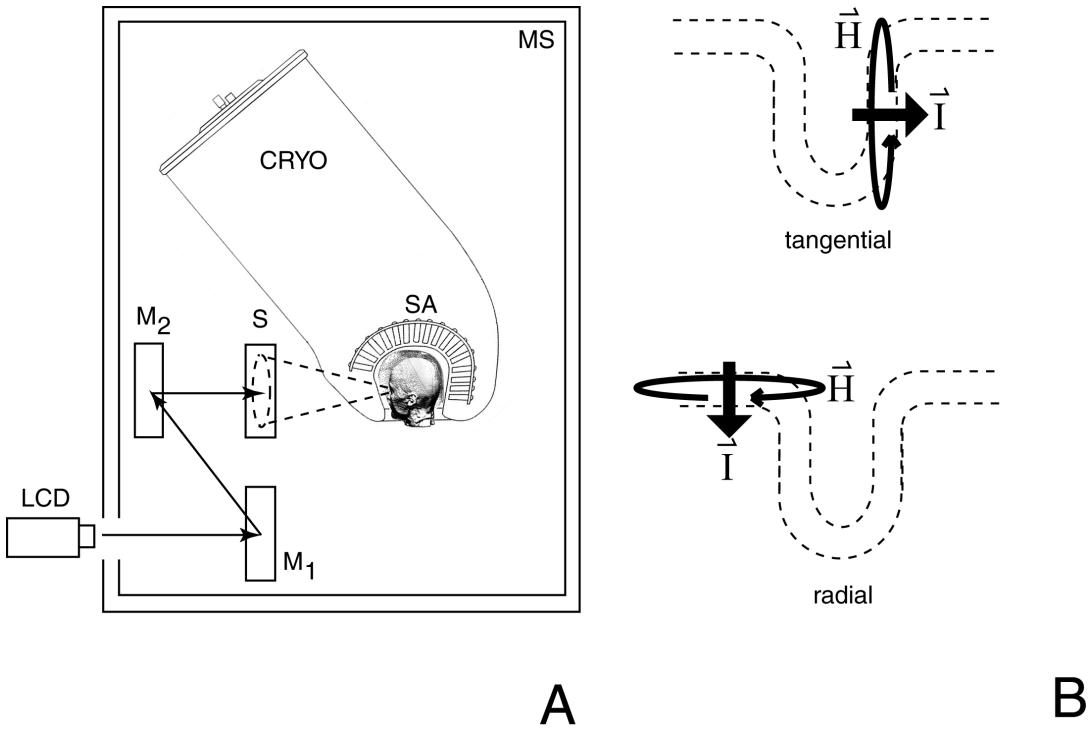

Fig. 38: (A) Recording set-up of the BTI Magnes 148 channel MEG system in Magdeburg. The subject's head is positioned within an array of sensors (SA) oriented roughly orthogonal to the surface of the skull. The sensors are fitted into a large cryostat (CRYO) where they are cooled to –269°C by liquid helium. Subject and cryo are placed within a magnetically shielded recording chamber (MS) to reduce artefacts due to external noise sources. Stimuli are projected from an LCD projector (LCD) through a small hole in the chamber and reflected by two mirrors (M_1, M_2) onto the back of a rear-projection screen (S), which can be viewed by the subject. (B) Only fields generated by current sources tangential to the surface of the head generate magnetic fields outside the head. Thus in most cases only sources in the sulci of the neocortex can be recorded. Radial sources on the surface of the brain are invisible to the MEG and have to be detected by concurrent recording of EEG (I=current; H=magnetic field strength).

Magnetic fields have the advantage over electrical fields that the magnetic permeability of the different compartments of the head is close to that of vacuum which means that they are not distorted as electric fields are by the high resistance of the skull (Lounasmaa, Hamalainen, Hari, & Salmelin, 1996). Also, the magnetic field recorded at the scalp is mainly generated by the highly localised *intracellular* current in the dendritic tree (primary current) whereas the surface-recorded electric potential is a consequence of the spread of electric current over a large volume. However, in a spherical symmetrical volume conductor (which can be used as a model of the head) only a dipole oriented tangentially to the surface can generate an external magnetic field. Thus, all dipoles oriented radially to the surface will not be recordable. This

means that MEG can only record signals from generators buried in the sulci, whereas surface dipoles are invisible to magnetic recording (Malmivuo & Plonsey, 1995)(Fig. 38B).

Even after noise reduction the amplitudes of evoked electric and magnetic fields generated by neural processing of single visual stimuli are typically too weak to be detected. This is not only due to the uncontrolled external noise but also to neural background noise generated by the majority of neurons that are not involved in processing of the stimulus. There is a solution that allows separation of signal and noise, the so-called method of "selective averaging". This exploits the fact that the background noise is not correlated with the evoked signal and will thus cancel out if repeated samples are averaged. The evoked signal on the other hand will not be cancelled as long as its individual deflections occur at a constant latency after each stimulus (Fig. 39). The result is an averaged evoked electric and magnetic signal for each electrode and sensor, which can be used to interpolate the scalp topography of the electric and magnetic fields generated by the neural process.

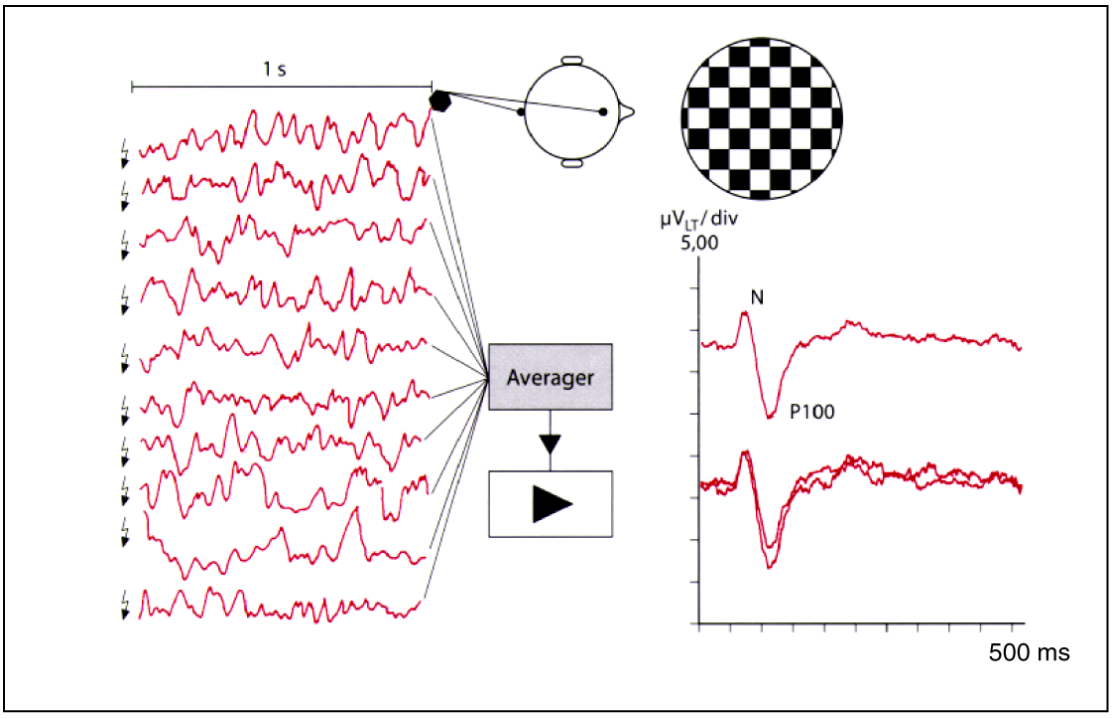

Fig. 39: Selective averaging procedure: The response evoked by a contrast reversal of a checkerboard stimulus is not detectable in the raw EEG. If repeated samples of the EEG time-locked to the reversal are averaged the background EEG is averaged out and the evoked signal becomes visible, here with a biphasic negative-positive deflection.

This scalp topography can be used to infer the underlying generators. If the position, orientation and amplitude of a neural generator is known and also the electromagnetic properties of the different compartments of the head then the electric and magnetic fields recorded at the scalp can be be uniquely calculated (Hamalainen & Sarvas, 1989). The simplest way to model the electromagnetic properties of the head is as a sphere that demarks the boundary between scalp and air. This is adequate for magnetic fields, but for electric fields the model will also have to take into account the different conductivity of brain, skull and skin. This is typically achieved by modelling four compartments based on their mutual boundary surfaces (boundary element model, BEM)(Fuchs, Wagner, & Kastner, 2001; Hamalainen & Sarvas, 1989)[68]. These compartments are (1) brain and cerebrospinal fluid, (2) skull, (3) skin and (4) air. Boundary-element models are reconstructed for individual subjects on the basis of anatomical T1 MR images.

Although the *forward* solution from a neural generator to a scalp recorded field is uniquely solvable there is no unique solution to the "inverse problem" of inferring the generator from a given scalp-recorded field (Hamalainen et al., 1993). The same recorded field could have in theory been generated by an infinite number of intracerebral dipole constellations[69]. Luckily the solution space can be highly constrained by allowing only physiologically and functionally plausible dipole solutions (Hari, Levanen, & Raij, 2000; Ilmoniemi, 1993; Scherg & Berg, 1991). First, the solution space can be restricted to the grey matter, owing to the fact that macroscopically recordable fields are believed to be generated mainly by cortical pyramidal cells. A second restriction of the solution space can be made by functional considerations. For example the main evoked signals by visual stimuli should be located in the visual rather than in the auditory cortex.

Two main factors may be taken as evidence for a higher spatial resolution of MEG source analysis: The small distortion of magnetic fields by the skull and the fact that the signal is based upon the intracellular primary current rather than the volume current. Recent results however show that depending on dipole position and orientation, number of recording channels and accuracy of the volume conductor model either EEG or MEG can give the better estimate when recorded alone (Barth,

Sutherling, Broffman, & Beatty, 1986; Cohen & Cuffin, 1991; Cohen et al., 1990; Cuffin, 2001; Cuffin, Schomer, Ives, & Blume, 2001; Fuchs et al., 1998; Huizenga, van Zuijen, Heslenfeld, & Molenaar, 2001; Leahy, Mosher, Spencer, Huang, & Lewine, 1998; Liu, Dale, & Belliveau, 2002; Lopes da Silva, Wieringa, & Peters, 1991; Mosher, Spencer, Leahy, & Lewis, 1993; Rose, Sato, Ducla-Soares, & Kufta, 1991). For this reason the method used in the current study employs a co-registration of EEG and MEG, which jointly contribute to improve the quality of source estimation which under optimal conditions can have an error of less than 5 mm.

Methods

Subjects

8 right-handed subjects (2 male, 6 female, age range 21 to 27) with normal vision (uncorrected) participated in the experiment. All subjects had prior experience with EEG/MEG recordings. The experimental procedures were in conformity with the Declaration of Helsinki.

Visual stimuli

Masking background stimuli were two large (9.8° x 9.8°) square areas of high contrast grating (0.79 Michelson luminance contrast) oriented at either 45° or 135° (Fig. 40, bottom left) into which 16 small squares of grey were simultaneously inserted (Fig. 40, top left). Target stimuli were 16 square patches of rectangular-wave grating (6 cpd, 1.1° x 1.1°) oriented at 45° or 135° with variable Michelson luminance contrasts of 0.13, 0.20, 0.32 and 0.50. The 16 targets were presented simultaneously within the masking background. Mean luminance was 194 cd/m^2 over the entire display. In accordance with most previous studies of lateral masking the parallel targets were chosed to be in-phase with the surround. In order to decrease border effects an isoluminant band of 0.1° was inserted between targets and masks. The border was not smoothed following the results of Cannon and Fullenkamp showing that the "sharpness" of the edge had no influence on masking (Cannon & Fullenkamp, 1991a). All displays contained a central fixation spot. The high spatial frequency of 6 cpd was

chosen after extensive pilot experiments. It was found to be the best trade-off between a high response amplitude and a maximisation of the striate contribution to the evoked responses.

Stimuli were generated using an XG-SV1E LCD projector (SHARP Electronics Europe, Hamburg, Germany) that projected via two mirrors onto a rear projection screen in the shielded recording chamber. This was necessary to prevent electrical interference in the electromagnetic recordings. A hardware gamma correction was calibrated using an LS 110 spot photometer (Minolta Europe, Langenhagen, Germany) to ensure linear luminance transfer characteristics.

Procedure

The subject triggered each trial with a keypress (Fig. 40, right). A trial began with the presentation of the mask alone for 2000 ms. The mask remained present throughout the entire duration of a trial. A sequence of 8 identical stimuli, either parallel or orthogonal, was flashed into the holes in the background, each for 250 ms with a (uniformly distributed) random inter-stimulus interval of 650-850 ms. After a gap of 2000 ms a second sequence was presented with the same timing but using the other orientation. Two thousand ms later the mask disappeared and the subject was required to give a response indicating which of the two stimulus trains had had the higher contrast. In this modified 2-alternative forced choice paradigm the parallel stimuli were “standard” stimuli against which orthogonal stimuli of different contrasts were tested, allowing one to estimate the orthogonal stimulus which was a subjective “match” to a given parallel stimulus. Our design departs from previous studies on lateral masking in two ways. Normally targets and masks are presented synchronously, which was changed in our case in order to reduce the interference between transient mask and target responses. Second, the stimuli were presented in groups of 8, a change which was made in order to increase the number of stimuli recorded for each condition. The design was optimised in pilot experiments to yield an optimum trade-off between number of events available for physiology and for psychophysics.

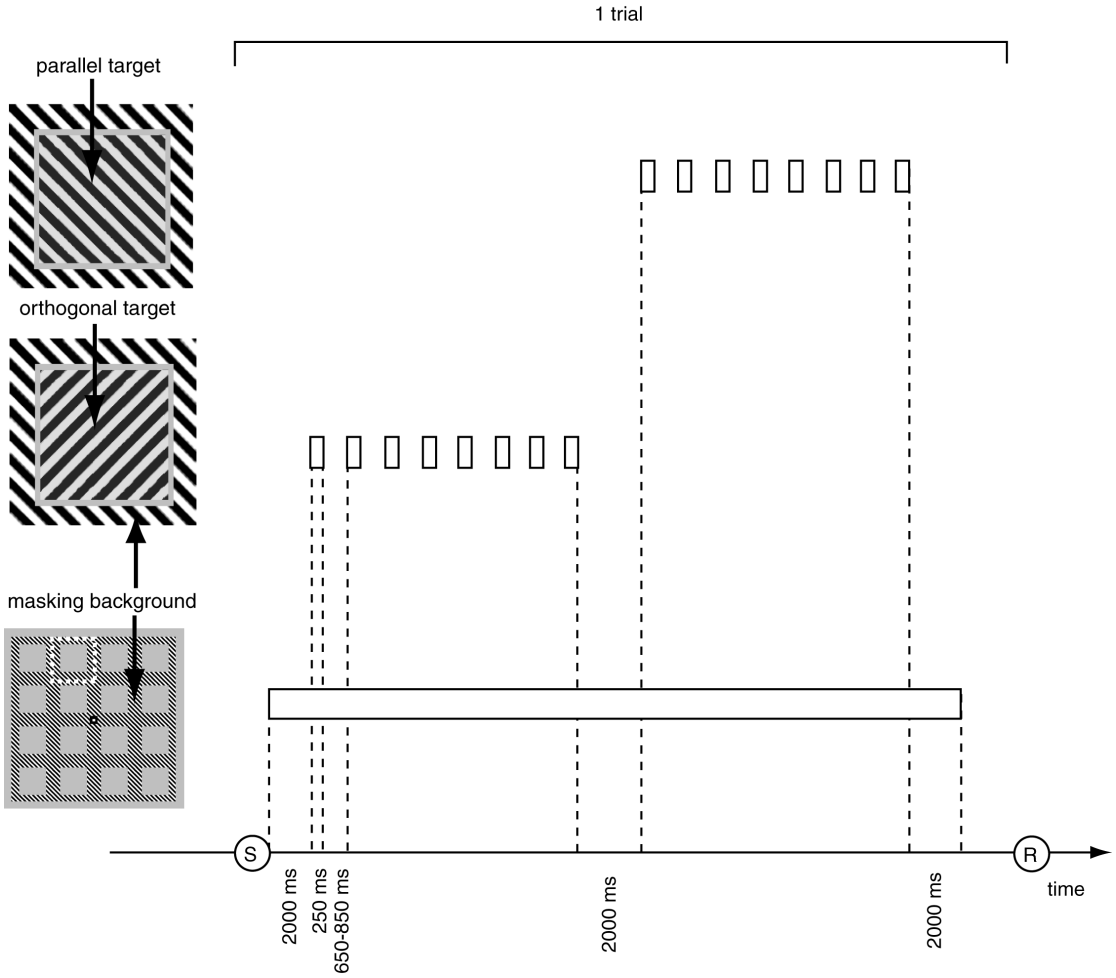

Fig 40: Bottom left: masking background. Middle and top left: a blown-up view of the area indicated by the dashed white square with superimposed orthogonal and parallel targets. Right: timing of an individual trial. S indicates the keypress starting the trial and R the subject's response.

Following preliminary studies the high contrast parallel stimulus (0.50) was paired with (compared to) orthogonal stimuli with contrasts 0.20, 0.32 or 0.50 (high contrast condition). The low contrast parallel stimulus (0.32) was paired with orthogonal stimuli with contrasts of 0.13, 0.20 or 0.32 (low contrast condition). Due to this design the total number of presentations was different for each stimulus category (600 for the two parallel stimuli, 400 for the middle two contrast levels of the orthogonal stimuli and 200 for the lowest and highest orthogonal stimuli). Conditions, contrast levels and orientations were randomly intermixed. Subjects were instructed to maintain fixation throughout each trial. Fixation was monitored during the sessions using an infrared camera.

EEG/MEG data acquisition

148 channel MEG and 32 channel EEG were simultaneously recorded at a sampling rate of 254 Hz filtered with a bandpass of 0.1-100 Hz. MEG was acquired with a BTI Magnes 2500 whole-head MEG system with 148 magnetometers (Biomagnetic Technologies, San Diego, USA). EEG was recorded using a 32 channel Synamps amplifier (NeuroScan, Herndon, USA) with an electrode cap (Electrocap International, Eaton, OH) covering the channels Fz, Cz, Pz, Oz, Iz, Fp1, Fp2, F3, F4, F7, F8, T7, T8, C3, C4, P3, P4, O9, O10, P7, P8, FC1, FC2, CP1, CP2, PO3, PO4, PO7, PO8 plus extra electrodes for right horizontal EOG, right vertical EOG and left mastoid. EEG channels were referenced to right mastoid and re-referenced offline to the average of right and left mastoid. MEG was subjected to an online noise reduction process that removed a weighted sum of environmentally induced magnetic noise (first order spatial gradients of the field) recorded by eight remote reference channels that do not pick up brain activity. Locations of EEG electrodes and MEG sensors were registered using a 3Space Fastrak system (Polhemus, Colchester, USA) with a common reference system defined relative to three anatomical landmarks (nasion, left/right preauricular points). These were coregistered with the individuals' anatomical T1 magnetic resonance scans. In order to further enhance the individual peaks and remove contribution of low-frequency noise the data were digitally highpass-filtered at 3 Hz which does not significantly alter amplitudes of the early visual evoked components (Skuse & Burke, 1990). Then data were sorted into stimulus-locked epochs of 600 ms length with 100 ms pretrigger and subjected to artefact rejection which removed epochs with peak-to-peak amplitudes exceeding a criterion of 3.0×10^{-12} T for MEG or 100 μV for EEG data.

Recording of transient rather than steady-state visual evoked responses (VERs) was chosen for several reasons: (1) Transient VERs have the advantage of minimizing the contribution of motion processing, which is typically observed for counter-phase reversing gratings used in steady-state designs (Murray & Kulikowski, 1983). (2) They allow segregation of time-courses into separable components corresponding to different processing stages. (3) They allow more straightforward equivalent current dipole modelling.

Results

Behavioural data

The orthogonal contrast which matches the parallel standard was estimated by interpolating the data with a Weibull cumulative distribution function (Weibull, 1951) for each condition and subject. The matching contrast can be found where the Weibull function takes a value of p = 0.5, which is where it is equally likely that the subject will judge either stimulus to be stronger.

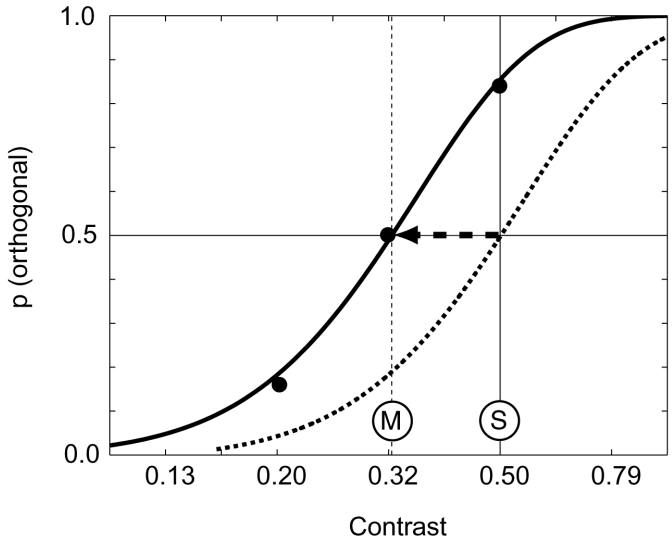

Fig. 41: Psychophysical equality threshold estimation for high contrast condition: A high contrast standard parallel target S is paired with 3 different levels of orthogonal stimuli. The filled circles indicate the proportion of trials in which the subject (ka81) chose the orthogonal stimulus to have higher contrast than the parallel standard stimulus as a function of the orthogonal contrast level tested. The point M where the probability that either of two stimuli is chosen is equal is interpolated by fitting a Weibull function to the data. If lateral masking were not orientation-tuned S would be equal to M and the judgements would be predicted by the hypothetical dashed line.

Although our stimulus design departed from the literature a strong masking effect was measured. Fig. 41 shows psychophysical results for one subject in the high-contrast condition. The proportion of trials in which an orthogonal stimulus train of a certain contrast level (either 0.20, 0.32 or 0.50) is judged as stronger than the parallel stimulus train of 0.50 is plotted on the ordinate. The data show that for physically equal stimuli the orthogonal stimulus is judged as higher than the parallel stimulus in 80 % of the trials. An orthogonal stimulus of about 0.33 is judged as equal to the

parallel standard in this subject. This occurs at $p_{orthogonal}$=0.5 where both stimuli are chosen equally often and neither of the two stimuli appears to have a higher contrast. The difference between parallel standard and matching contrast indicates a reduction in perceived contrast of the parallel stimulus due to the orientation tuning of lateral masking. This reduction is present for both contrast levels and all subjects. Across all subjects matching orthogonal grating contrasts are 0.18 for the 0.32 standard stimulus (one sample t-test: $t_{[7]} = -7.5$, $p < 0.001$) and 0.33 for the 0.50 standard stimulus (one sample t-test: $t_{[7]} = -9.1$, $p < 0.001$)(Fig. 42). Thus in both high and low contrast conditions an orthogonal stimulus requires only approx. 60 % of the contrast of a parallel stimulus to match.

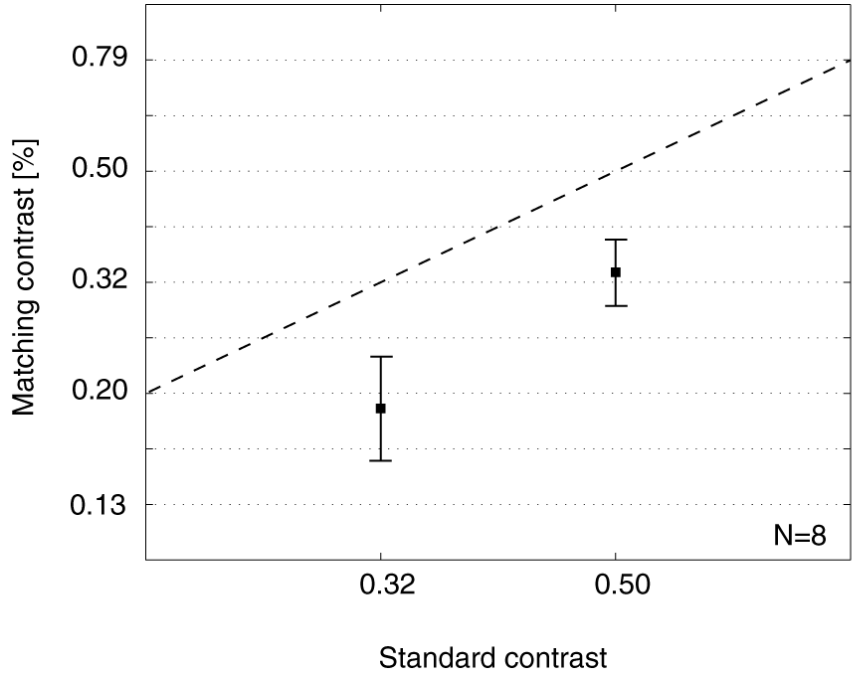

Fig. 42: Threshold reduction averaged across all 8 subjects. The dashed line shows the orthogonal matching contrasts that would be expected if there were no effect of orientation on perceived contrast. Error bars show +/- 1 SEM.

Waveforms and topographies

Electrical and magnetic responses followed a similar 3 phase waveform (Fig. 43A, left): The posterior EEG channels showed a sequence of positive-negative-positive deflections: (I) A positive component with onset latency around 80 ms and peak latency 100-150 ms (P100); (II) a negative component with onset latency 120-180ms

and peak latency 150-200 ms (N150); (III) a positive component with onset latency 180-220 ms and peak latency 210-280 ms (P250). The MEG channels showed highly similar temporal profiles, but with opposite polarities for left and right hemisphere channels resulting in bipolar fields. The MEG components will be labelled in analogy to their electric counterparts with "M" (M100, M150, M250). All 3 major deflections show a reduction of amplitude for the parallel compared to the orthogonal stimulus with the same physical contrast.

Fig. 43A also shows the electric and magnetic field topographies interpolated from the raw evoked signals for the time point 110 ms after stimulus onset, which is the peak of the early deflection. The electric field over the scalp shows a strong positivity centred on the occipital pole. The magnetic field shows a bipolar field distribution that is orthogonal to the electric field as would be expected if both fields were generated by the same source. The topographies are shown for both orthogonal and parallel stimuli of the same contrast (0.50). The scale has been individually set to demonstrate that both topographies are strikingly similar, except for a linear scaling factor. This topographical similarity is further demonstrated by the results of a cluster analysis (Everitt, 1993). Cluster analysis has been used by other groups in order to provide a segmentation of event-related brain topographies (Michel et al., 2001; Pascual-Marqui, Michel, & Lehmann, 1995). Here it will be used to assess the similarity of topographies for the different stimulus conditions.

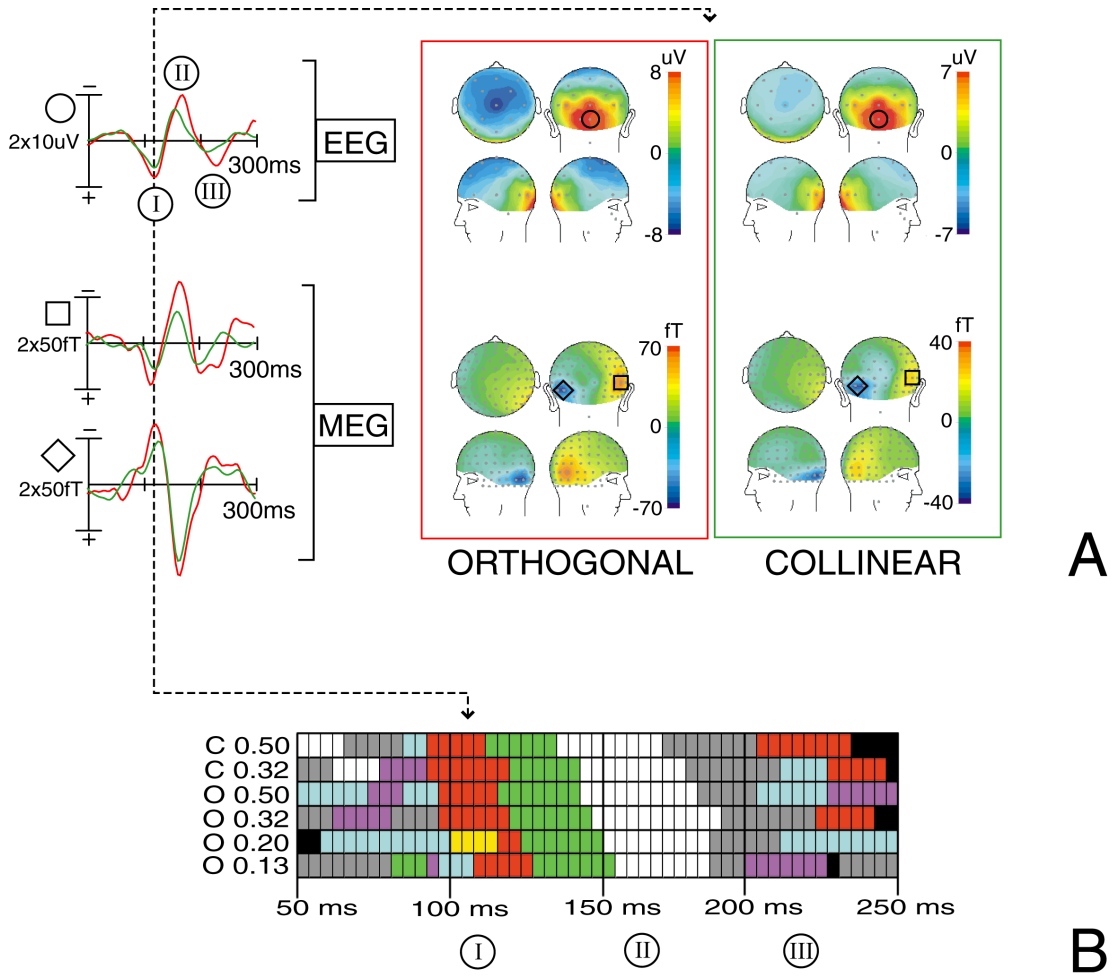

Fig. 43: (A) Left: Evoked electric and magnetic brain responses to orthogonal (red) and parallel stimuli (green) of same contrast (0.50) over occipital pole (subject ka81). EEG and MEG response are highly similar and left occipital and right occipital magnetic responses are mutually inverted as would be expected for a single dominant generator. Parallel responses are clearly reduced for all 3 major components (P100/M100 (I), N150/M150 (II), P250/M250 (III)). Stimulus onset was at 0 ms. Right: response topographies for the time indicated by the dashed vertical line (110 ms). The top shows EEG and the bottom MEG data. The figures are individually scaled to demonstrate that the topography is similar for both conditions. The circle, box and diamond symbols indicate the scalp location of the channels shown on the left. (B) Results of cluster analysis for the same subject shown between 50 and 250 ms. Each cluster is coded with a colour, each row is a stimulus category c and each column is a sample point t. This demonstrates that the topographies of the evoked responses of the 6 stimulus types (P 0.50, P 0.32, O 0.50, O 0.32, O 0.20 and O 0.13) are very similar at similar latencies. This means that the topographies evoked by the different stimuli are all similar except for a scaling factor.

A 177-dimensional topography vector was calculated for each time-point sampled between 0 and 300 ms. This vector consisted of the amplitudes of the 148 magnetic plus 29 electric channels (excluding the 2 eye-channels and the left mastoid) scaled to a unit length of 1 (Fig. 44).

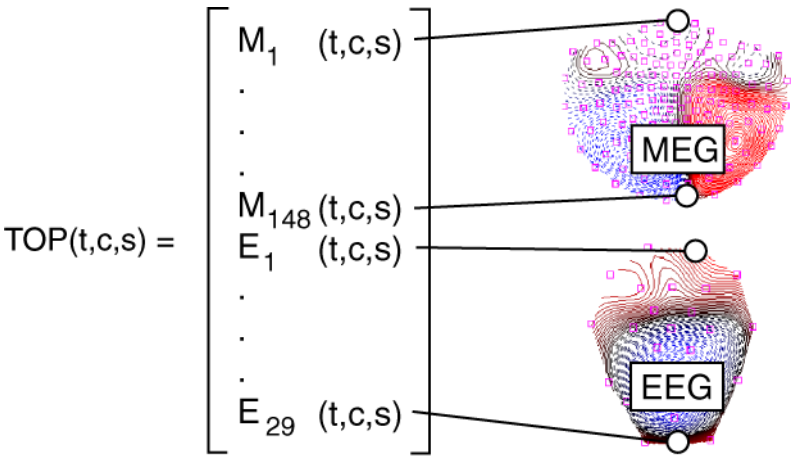

Fig. 44: Construction of a 177-dimensional topography vector for each time point (t), condition (c) and subject (s).

The 450 topography vectors from all 75 time points and 6 conditions of one subject are then fed into a single cluster analysis. In a first step the distance between all possible pairs of the 300 vectors is computed using a Euclidian distance metric:

$$d_{i,j} = \sqrt{\sum_{k=1}^{177} (x_i(k) - x_j(k))^2} \quad . \tag{4}$$

where $d_{i,j}$ denotes the Euclidian distance between topography vectors x_i and x_j and k runs through the 177 channels. Based upon the Euclidian distance the 450 vectors were segmented into 10 clusters based upon the average distance D(r,s) between all n_r vectors x_{ri} in cluster r and all n_s vectors x_{sj} in cluster s:

$$D_{r,s} = \frac{1}{n_r n_s} \sum_{i=1}^{n_r} \sum_{j=1}^{n_s} d_{ri,sj} \quad . \tag{5}$$

Analyses were performed using the MATLAB statistics toolbox (Mathworks, Natick, USA). For further information on the clustering algorithms see Everitt (1993). The cluster analysis procedure is very different compared to analysing topography with principal component analysis (PCA)(Maier, Dagnelie, Spekreijse, & van Dijk, 1987; Skrandies, 1989) or independent component analysis (ICA)(Makeig, Jung, Bell, Ghahremani, & Sejnowski, 1997), because the topography for each time point is assigned to exactly one cluster. PCA and ICA on the other hand reconstruct the topography for each time point by a superposition of several weighted "basis topographies", which are either orthogonal (PCA) or statistically independent (ICA). Cluster analysis was preferred to PCA and ICA because it requires only a minimal set of assumptions. PCA assumes that different basis vectors (topographies) are orthogonal, a requirement that is problematic for evoked electric and magnetic brain responses, because different sources can produce non-orthogonal fields. ICA requires a number of observations that is larger than the number of dimensions, which is not given for our data, where the topography vector is 177-dimensional and the number of samples during the main deflections of the evoked response is 75.

The results for one subject are shown in Fig. 43B beneath the original waveforms and topographies. Before the first major deflection (I) the clusters are distributed randomly. During the first two major components (I,II) the responses for all conditions are assigned to the same topography cluster at a fixed latency. This means that the topographies are highly similar at a given latency, except for a possible linear scaling factor. The minor latency differences are presumably an effect of stimulus contrast, which is known to affect latency (Vassilev & Manahilov, 1986). Beginning with the last major deflection (III) the clusters are again distributed randomly. The results for all subjects are shown in Fig. 45 and confirm this pattern.

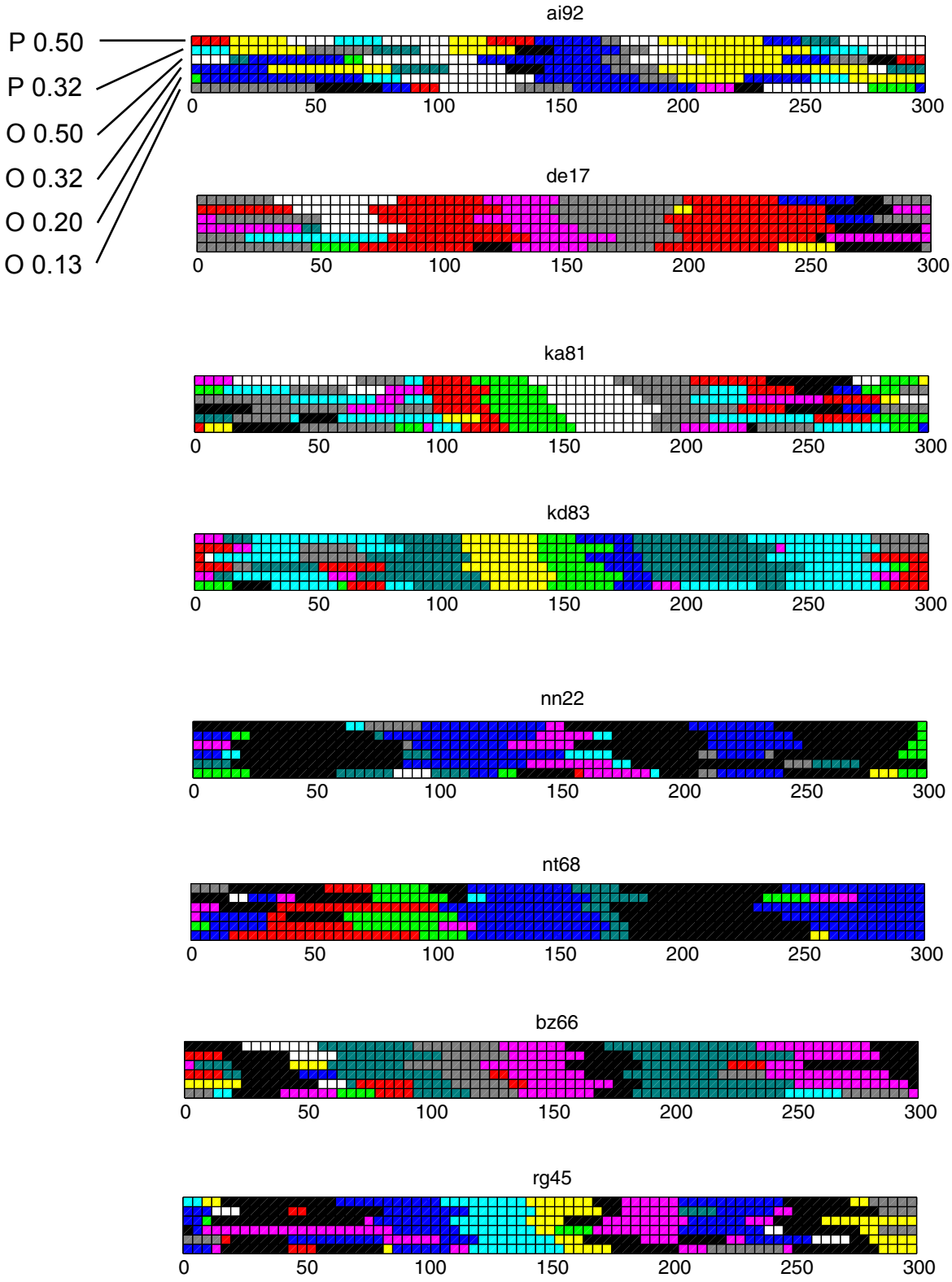

Fig. 45: Results of cluster analysis for all subjects. Each of the 10 clusters is coded with a colour, each row is a stimulus category c and each column is a sample point t. It can be seen that in most cases the topographies evoked by different stimuli are highly similar at the same time points, as is indicated by the fact that colour (cluster) does not change in a vertical section across each diagram. All subjects show cross-stimulus topographical similarity around the time of main deflections whereas before 100 ms and after 250 ms topographies are randomly distributed and the cross-stimulus similarity only holds for a few subjects (kd83, nn22).

Source localisation

There is considerable disagreement in the literature on the striate and extrastriate contributions to the early deflections of evoked electric and magnetic fields (Aine, Supek, & George, 1995; Aine et al., 1996; Foxe & Simpson, 2002; Ikeda et al., 1998; Jeffreys & Axford, 1972a, 1972b; Maier et al., 1987; Portin, Salenius, Salmelin, & Hari, 1998; Schroeder, Mehta, & Givre, 1998; Schroeder, Tenke, Givre, Arezzo, & Vaughan, 1991; Seki et al., 1996). This can be mainly attributed to differences in spatial and temporal stimulus parameters (quadrant, eccentricity, spatial frequency, onset versus reversal versus offset responses) to which early components respond sensitively. Studies of action potentials, local field potentials and current source densities in multiple visual areas and cortical layers reveal that synaptic activity is temporally extended, occurs synchronously in multiple areas, shows signs of feedback and polarity inversion at different processing stages and depends critically on spatial and temporal stimulus properties (Bullier, 2001; Creutzfeldt & Kuhnt, 1973; Lamme & Roelfsema, 2000; Lamme et al., 1998; Maunsell & Gibson, 1992; Schroeder et al., 1998; Schroeder et al., 1991). Thus when using novel stimuli it is impossible to infer the generators by simply referring to the literature. Instead it is necessary to localise the dominant generators by using equivalent dipole modelling. According to its receptive-field tuning V1 should contribute strongly to early deflections for stimuli with high spatial frequencies as in our case.

As the anatomical representation of a stimulus in V1 can be well estimated using retinotopic considerations (Aine et al., 1996; Horton & Hoyt, 1991b) two dipole seed points were placed to the lateral side of the calcarine sulcus of either hemisphere at a depth of 2 cm from the occipital pole (Fig. 46). Then two equivalent current dipoles (ECD) with fixed orientation and fixed location were fitted using a Nelder-Mead simplex minimisation algorithm (Nelder & Mead, 1965). Dipoles were constrained within a radius of 5 mm of the seeds and fitted using software package CURRY (Neuroscan, El Paso, USA) to the whole time period between 0 ms and 300 ms. A 3-sphere boundary element model (BEM) was used and dipoles were fitted to EEG and MEG data concurrently. In order to reduce noise due to contributions of non-visual areas the MEG channels were restricted to a circular set of 72 sensors centred on Pz.

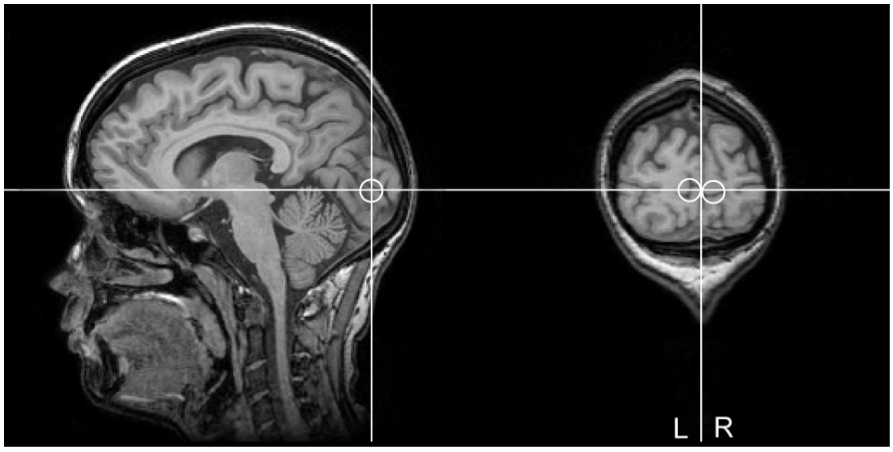

Fig. 46: Bounding spheres of the two-dipole fit in the calcarine sulcus shown on top of an anatomical T1-weighted MR-image.

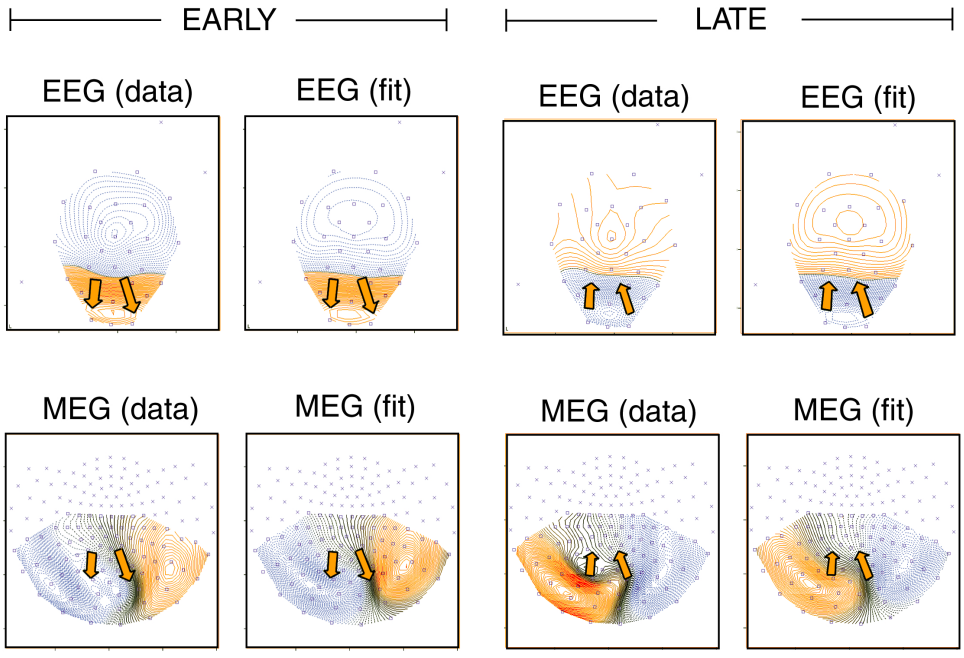

Fig. 47: Measured ("data") and fitted ("fit") response topographies for the peaks of the two striate components (subject ka81). The left shows P100/M100 ("early"), the right N150/M150 data ("late"). The quality of fits is striking, especially regarding that dipoles (orange arrows) were placed on a-priori retinotopical assumptions. The N150/M150 is an inversion of the P100/M100 response. Data are shown as 2-D projections with posterior right at the bottom right.

The variance accounted for by two striate dipoles is 87.7 % (6.39 std) for the P100/M100 peak, 88.1 % (4.21 std) for the N150/M150 peak and 70.1 % (15.74 std) for the P250/M250 peak. Thus the early two components are dominated by striate responses, whereas the later component seems to have significant influences from other areas. The good fit between data and our model-guided forward solution for the striate dipoles for the early two components is shown for one subject in Fig. 47.

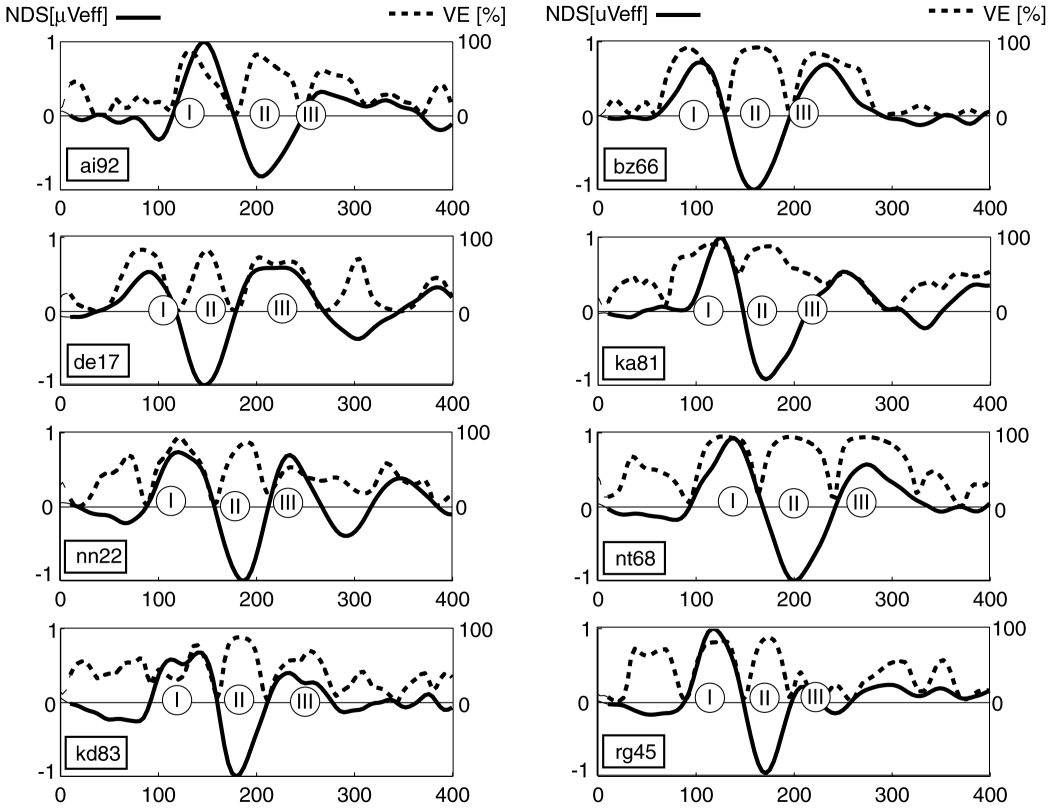

Fig. 48: Normalized dipole strengths (NDS) and variance explained (VE) by the two-striate dipole forward solution for all eight subjects studied. The theoretical dipoles give a good explanation of the two early peaks. The Roman numbers indicate the locations of the 3 major deflections in the raw data. The peak V1 dipole strength coincides closely with these peaks.

Fig. 48 shows the time courses of normalized dipole strengths and percentages of variance explained for each subject. The quality of the fit is surprising given the complex stimulus shown and the fact that dipole locations were chosen on a-priori anatomical assumptions. It is also surprising that the N150/M150 component was so clearly an inversion of the P100/M100 component (Fig. 47)[70]. Similar results have

been reported by other authors and may point to re-entrant processing effects (Aine et al. 1995). The third component P250/M250 also had significant striate contributions, but the decrease in quality of fit suggests strong contributions from extrastriate visual areas. Besides the main effects the data also point towards the existence of an earlier striate effect (around 30-80ms), which is rather weak (as can be seen from the dipole strength) but nonetheless explains a considerable amount of variance for four of the subjects (nn22, nt68, kd83 and rg45). This effect did not show any difference between parallel and orthogonal stimuli.

Contrast response functions

For quantitative analysis of contrast responses peak amplitudes of the major evoked electric and magnetic responses were measured for each contrast level, stimulus type and subject. For MEG components left and right hemisphere deflections were averaged across the peak negative and peak positive channels. For EEG components the peak left and right hemisphere channels were averaged. In order to combine data across subjects the response amplitudes were normalised for each subject and component individually by dividing the data by their mean. These data were then used to plot contrast response functions (CRFs) for parallel and orthogonal stimuli (Fig. 49). The data show the typical profile increasing monotonously with contrast (Boynton et al., 1999; Campbell & Kulikowski, 1972; Gopfert, Muller, Breuer, & Greenlee, 1998; Murray & Kulikowski, 1983; Tootell et al., 1998)[71]. For all electric and magnetic deflections the responses to collinear stimuli were reduced as compared to orthogonal stimuli. For two subjects (kd83, nn22) the electric and magnetic P250 were too weak to be measured so for this component the results will be based on the data of only 6 subjects.

CRFs were also computed for the peaks in the time-courses of estimated dipole strengths for the orthogonal stimuli in order to obtain a more pure estimate of striate cortical activity. These data were fit using the hyperbolic ratio-function, which is a standard model of contrast-dependent responses in primary visual cortex as mentioned in the previous chapter (Albrecht & Hamilton, 1982; Boynton et al., 1999; Geisler & Albrecht, 1997; Li & Creutzfeldt, 1984; Sclar et al., 1990).

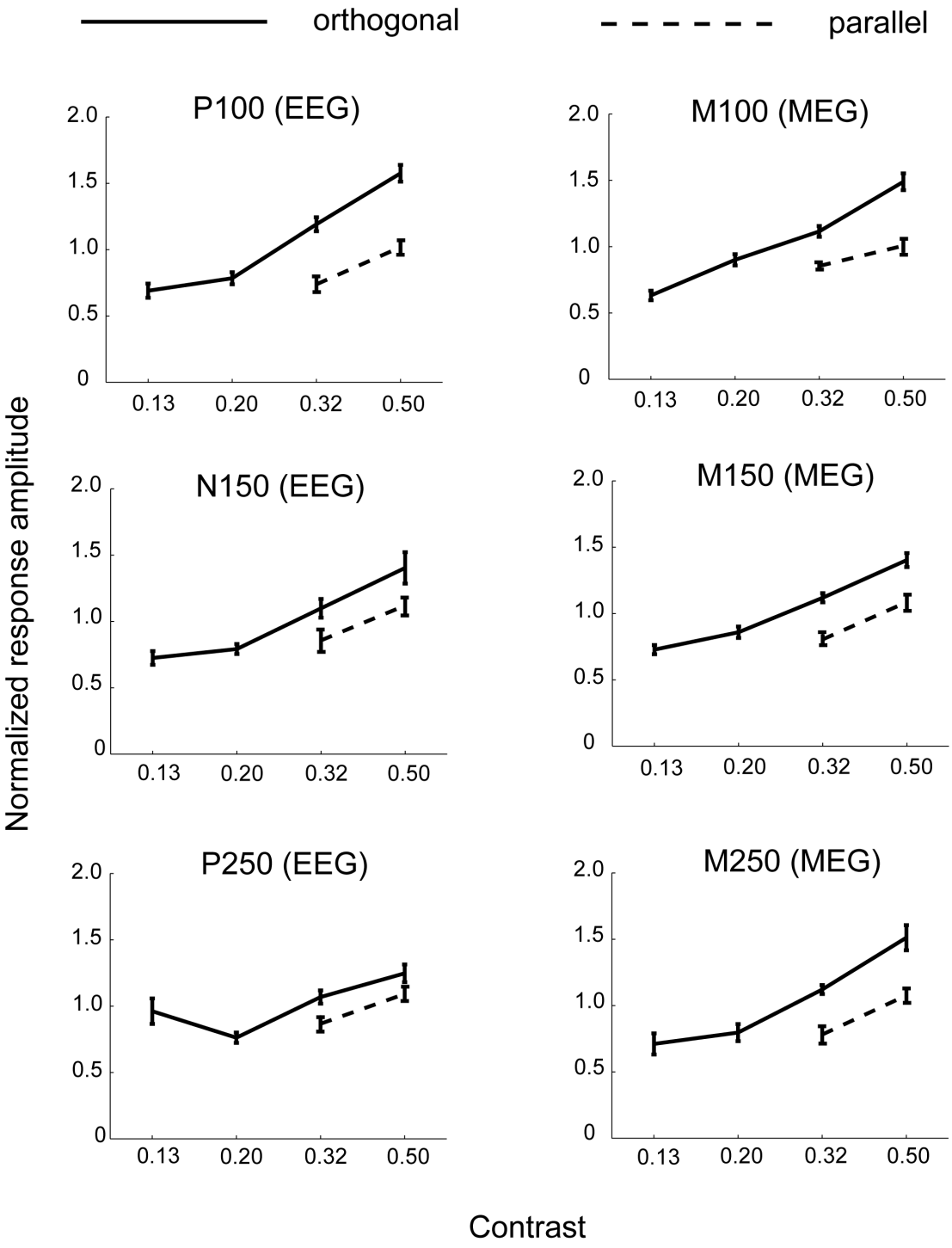

Fig. 49: Contrast response functions for all components normalized and averaged across subjects. Error bars = +/- 1 SEM.

Prediction of psychophysics by physiology

In order to assess the degree to which striate activity predicts perceived magnitude of contrast it was necessary to compare the striate responses for the parallel standard stimuli with the according orthogonal matching stimuli. The responses to the orthogonal matching stimuli were interpolated using the hyperbolic ratio model fitted for each subject. In order to assess how well perceived contrast is predicted by activity in primary visual cortex the analysis was restricted to the striate dipole time-courses of the first two deflections that had a dominant origin in V1. Fig. 50 shows the CRFs of peak dipole amplitudes for the early striate component for parallel and orthogonal stimuli for one subject. The filled circle and filled square show the respective responses for two stimuli S (parallel) and M (orthogonal) matching in perceived contrast.

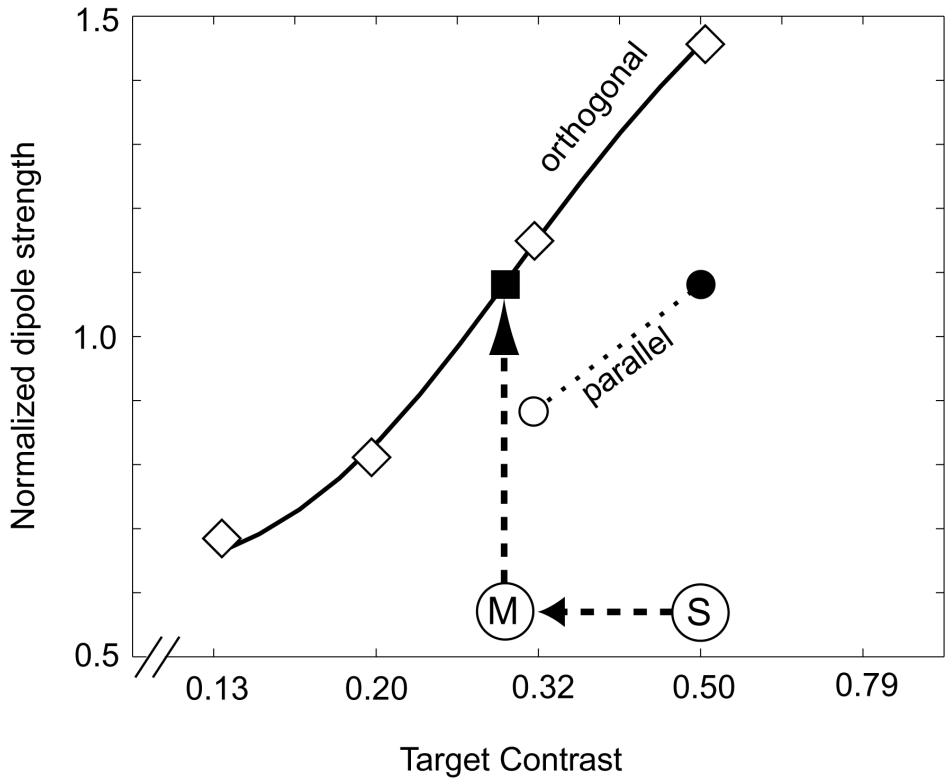

Fig. 50: Dipole contrast response functions (subject ka81): Peak dipole amplitudes for the early striate component (P100/M100) as a function of contrast shown for parallel (open and filled circles) and orthogonal stimuli (open diamonds). The neural response to the perceptually matching orthogonal stimulus (filled square) of a high contrast parallel standard is interpolated from the contrast response data of the orthogonal stimuli for each subject by fitting a hyperbolic ratio function (equation 1). In the case of a perfect match between response amplitude and perceived contrast the normalized dipole strengths for the standard and matching stimuli should be identical (filled circle and the filled square).

If striate activity correlates with perceived contrast the responses to these two perceptually matching stimuli should be identical. For early and late striate components and both contrast levels the response amplitudes to the two physically identical stimuli are different and the responses to perceptually matching stimuli are similar (Fig. 51). Thus stimuli that are perceived to have the same contrast generate similar V1 responses. This holds especially for the late striate component, whereas the early striate component shows a slight deviation at high contrasts. Further analysis shows that there is a strong correlation for individual subjects between the reduction of perceived contrast and the reduction of the response to the parallel standard stimulus (P100/M100, low contrast: Pearson's r = 0.87; P100/M100, high contrast: Pearson's r = 0.77; N150/M150, low contrast: Pearson's r = 0.87; N150/M150, high contrast: Pearson's r = 0.62).

Tab. 2 shows the parameters estimated for equation (1) collapsed across subjects which can be used for a further comparison with previously published psychophysical data (Boynton et al., 1999; Cannon, 1985; Legge, 1981; Legge & Foley, 1980). The exponent p that governs the response behaviour for mid to high-contrast stimuli ($C>>\sigma$) is similar for both response components (about 0.3) and comparable to those found in previous studies on contrast discrimination (Legge & Foley, 1980) and magnitude scaling (Cannon, 1985). It is also similar to the exponents fit to single cell responses in cat and monkey striate cortex (Geisler & Albrecht, 1997), as well as to fMRI contrast-response functions in human striate cortex (Boynton et al., 1999). In psychophysical models the inflection point in the contrast representation function is used to explain the dip of the threshold versus contrast (TvC) function which occurs at low contrasts around 0.01 (Fig. 33, middle left)(Legge, 1981; Legge & Foley, 1980). Also the inflection point max (R'(C)) of our contrast-response functions was computed. For the late striate component (0.03 for N150/M150) the estimate is in a similar range as human psychophysics, but not for the early striate component (0.13 for P100/M100).

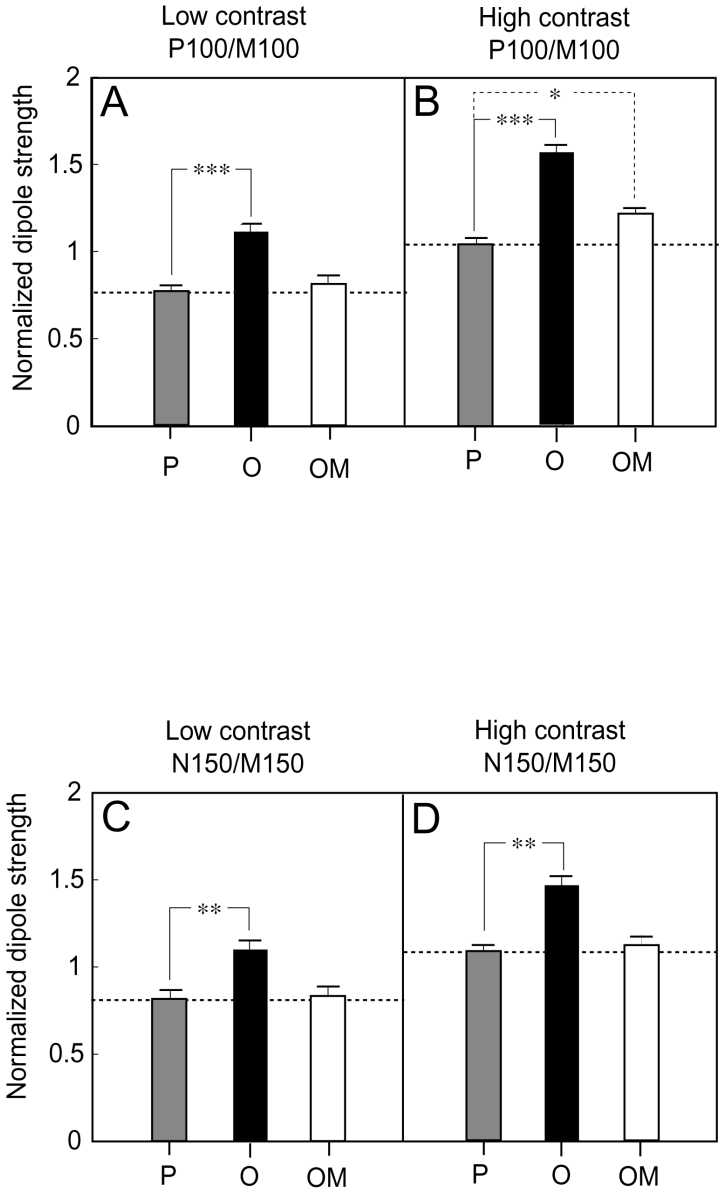

Fig. 51: Mean response amplitudes averaged across subjects of striate components to parallel standard stimuli (P), orthogonal stimuli with same physical contrast (O) and same perceived contrast (OM). Response amplitudes are different for physically identical stimuli (P, O) and similar for perceptually matching stimuli (P, OM). Especially the N150/M150 component closely parallels perception. The dotted line shows the location of the mean for the parallel stimulus. Statistics were computed to test for an overall effect of stimulus category and single tests were performed for the pairs (P - O) and (P - OM): (A) ANOVA $F_{[1;7]} = 33.6$, $p < 0.001$; paired-samples t-test for comparison O-P $t_{[7]} = -5.8$, $p < 0.001$; comparison O-OM $t_{[7]} = -0.8$, $p = 0.454$; (B) $F_{[1;7]} = 88.1$, $p < 0.001$, comparison O-P $t_{[7]} = -9.4$, $p < 0.001$, comparison O-OM $t_{[7]} = 2.8$, $p = 0.028$; (C) $F_{[1;7]} = 13.8$, $p = 0.007$, comparison O-P $t_{[7]} = -3.7$, $p = 0.008$; comparison O-OM $t_{[7]} = -0.743$, $p = 0.482$; (D) $F_{[1;7]} = 12.7$, $p = 0.009$; comparison O-P $t_{[7]} = -3.5$, $p = 0.010$, comparison O-OM $t_{[7]} = -1.2$, $p = 0.288$. Error bars display +/- 1 SEM.

	p	Q	σ	max (R')	RMS (fit)
P100/M100	0.36	1.36	0.23	0.13	0.06
N150/M150	0.29	1.06	0.13	0.03	0.03

Tab. 2: Parameters of the fitted hyperbolic ratio functions collapsed across 8 subjects.

Discussion

Using a lateral masking paradigm that allowed the dissociation of physical and perceived contrast, it has been demonstrated that activity of primary visual cortex correlates closely with perceived contrast but not with physical contrast. Target stimuli that were perceived to have the same contrast but whose physical contrasts differed as much as 40 % lead to the same response amplitude in V1. The data also demonstrate a temporal development in the rescaling process. The first striate deflection already predicts perceived better than physical contrast. The second deflection fits the perceptual data even better, and also extrapolates well beyond the range of contrasts measured to predict the dip in the contrast discrimination data for low contrasts shown in previous studies (Boynton et al., 1999; Legge, 1981; Legge & Foley, 1980). Both components also allow good prediction of individual differences in perceived contrast reduction.

Previous results showing a lack of binocular transfer in lateral masking (Chubb et al., 1989) despite its orientation selectivity have been used to argue for a strong role of primary visual cortex in the representation of perceived contrast. On the one hand V1 is the last processing stage with substantial populations of cells with monocular dominance in visual cortex (LeVay et al., 1975). On the other hand it is the first stage of orientation-selective processing and can thus account for the orientation tuning of lateral masking. Subcortical visual neurons in the lateral geniculate nucleus (LGN) are not orientation selective, so targets with the same physical contrast should be masked in a similar manner by parallel and orthogonal flanks, at least in the feed-forward sweep of processing. Likewise, a lower contrast orthogonal stimulus matched in perceived contrast to the parallel stimulus should evoke less LGN activation. If the

stimulus representation in V1 were similarly based on physical contrast responses, evoked V1 responses should also follow this pattern. Our results, however, show similar responses for the contrast metamers and different responses for physically identical stimuli.

Numerous studies at the level of single cells and populations have demonstrated that primary visual cortex exhibits surround-effects that can account for the current data (Blakemore & Tobin, 1972; Grinvald, Lieke, Frostig, & Hildesheim, 1994; Kapadia et al., 2000; Levitt & Lund, 1997; Nelson & Frost, 1978; Polat, Mizobe, Pettet, Kasamatsu, & Norcia, 1998; Sengpiel et al., 1997; Walker et al., 1999). Specifically, several studies have directly shown the influence of surround effects on contrast transfer functions (Polat et al., 1998), and the dependency of surround effects on the relative contrast between centre and surround (Levitt & Lund, 1997; Polat et al., 1998; Somers et al., 1998; Toth, Rao, Kim, Somers, & Sur, 1996). It is possible that the anatomical substrate of this surround modulation is feedback from higher visual areas. Mutual feed-forward and feedback connections are known to exist between V1 and many extrastriate visual areas (Bullier, 2001; Lamme & Roelfsema, 2000; Lamme et al., 1998; Salin & Bullier, 1995). However the most detailed study so far showed that inactivation of V2 has no effect on the surround modulation of responses in V1 (Hupe, James, Girard, & Bullier, 2001). A second candidate is the rich plexus of horizontal connections in primary visual cortex. These connections have a range of up to 8 mm and tend to preferentially link iso-oriented orientation columns (Gilbert, 1992; Gilbert & Wiesel, 1979; Malach et al., 1993; Martin & Whitteridge, 1984; Mitchison & Crick, 1982; Rockland et al., 1982; Schmidt et al., 1997). This orientation anisotropy is of special interest because it may be able to account for the orientation tuning of lateral masking. The temporal dynamics observed in our study may provide a further clue as to the mechanisms. Horizontal connections are slow (ca. 0.1-0.3 m/s, cf. Bringuier, Chavane, Glaeser, & Fregnac, 1999; Girard, Hupe, & Bullier, 2001; Grinvald et al., 1994) whereas feed-forward and feedback connections are fast (ca. 3.5 m/s, cf. Girard et al., 2001). Based on estimates by Bullier (2001) a feed-forward-feedback cycle between V1 and V2 could be completed within 4 ms. Horizontal propagation across a distance of half the size of our targets (0.55°) should take approx. 55 ms. The fact that our second component predicts perception better thus fits in with a slow horizontal integration process.

It should be noted that some other masking paradigms can produce an opposite effect of cross-orientation inhibition (Burr & Morrone, 1987; Morrone, Burr, & Maffei, 1982; Morrone, Burr, & Speed, 1987) when using superimposed targets and masks. These effects can be explained by models of local divisive inhibition (Carandini, Heeger, & Movshon, 1997). Our results also differ from those of Polat and Norcia, who observed facilitation for collinear and suppression for orthogonal target-flank combinations (Polat & Norcia, 1996, 1998). However these authors used steady-state visual evoked potentials, rendering it difficult to judge whether they recorded predominantly striate activity. They also found their collinear facilitation effects at far lower contrasts than used here. A subsequent single-cell study of the same authors revealed a biphasic dependency of surround interactions on contrast, with the interaction being facilitatory for low and inhibitory for high target contrasts (Polat et al., 1998). In their earlier studies Polat and Norcia presumably recorded from the low-contrast end, while here recordings were taken from the high-contrast end of this biphasic function.

Chapter 6

General discussion

Perceived contrast and primary visual cortex

The present study has for the first time demonstrated a close relationship between perceived contrast and response amplitudes in primary visual cortex in a population of normal subjects. Several visual areas respond to monotonous increases in contrast with monotonous increases in their response amplitude (Boynton et al., 1999) and could thus be counted as possible candidates to represent perceived contrast. The few previous studies directly investigating the relationship have relied on controversial assumptions about the shape of the perceived contrast function, which limits their ability to link neural responses to perception (Fiorentini & Maffei, 1973; Franzen & Berkley, 1975). They also relied on steady-state VEPs that bear no simple relationship to underlying visual areas. The current study has demonstrated that even stimuli whose physical contrast differs by as much as 40% but that are perceived to have the same contrast evoke responses of the same amplitude at a late stage of processing in V1. Furthermore, the study has for the first time concurrently recorded psychophysics and physiology in such a constancy paradigm. The studies on *brightness* constancy in V1 on the other hand have relied on general assumptions on the similarity between perception in humans and cats (MacEvoy & Paradiso, 2001)[72].

The experiment can now be interpreted within the framework formulated in chapter 2. It presents evidence that two important criteria are fulfilled. First, when two stimuli are physically identical but one is perceived to have a higher contrast then the response amplitude for that stimulus is higher. This presents evidence for the isomorphism requirement:

$$\forall q_1, q_2 \in Q : A(q_1, q_2) \Rightarrow B(f(q_1), f(q_2)).$$

This means that the perceptual relation A ("q_1 is perceived to have higher contrast than q_2") is preserved at a late stage of processing in V1 by the relation B ("the late striate response to q_1 has a higher amplitude than the response to q_2"). Furthermore the study provides evidence for single-valuedness since the same perceived magnitude

corresponds to the same response amplitude even when using very different physical contrasts:

$$\forall q_1, q_2 \in Q : q_1 = q_2 \Rightarrow f(q_1) = f(q_2).$$

The study has also revealed that the close correspondence between perceived contrast and V1 response amplitude holds best for later stages of processing (the "late" striate component). This temporal unfolding of the response might even give a further explanation as to why several studies failed to find a correlation between V1 responses and perceptual experience. It suggests that in some cases this correlation may only be present for the late temporal stages of V1 processing. Supporting this idea, Kinoshita and Komatsu (2001) showed that representation of the luminance of large homogenous fields is present at late (sustained) but not at early (transient) phases of striate processing. Likewise, Super and coworkers (2001) demonstrated that conscious perception of texture-defined figures critically depends on late rather than early striate responses. They believe this is a consequence of feedback processes from extrastriate visual areas. Several other authors have stressed the role of re-entrant processing for visual awareness (Bridgeman, 1975, 1980, 2001; Di Lollo, Enns, & Rensink, 2000; Lamme & Roelfsema, 2000; Lamme, Super, Landman, Roelfsema, & Spekreijse, 2000; Stoerig, 2001).

A considerable body of further evidence has been presented that adds to these results and shows that V1 also fulfils other criteria formulated in chapter 2. On the one hand striate cortex is strongly necessary for contrast perception, which does not hold for other visual areas except V2 (and possibly V3). The clearest points of evidence for this being that lesions to V1 always lead to blindness, and it is not possible to elicit phosphenes by stimulating extrastriate cortex in these patients (Cowey & Walsh, 2000). V1 is also the cortical site at which the injectivity criterion is most closely fulfilled:

$$\forall q_1, q_2 \in Q : q_1 \neq q_2 \Rightarrow f(q_1) \neq f(q_2).$$

To recapitulate, this states that different perceived contrasts will always have to be mapped to different response amplitudes. It was proposed in chapter 2 to assess injectivity by testing for its consequences.

The first consequence is covariance, which has been shown to hold above for the relationship between perceived contrast and V1 responses. V1 correlates with changes of perceived contrast in binocular rivalry (Polonsky et al., 2000) and shows a similar scaling with contrast as single-cell and population responses in V1 (Boynton et al., 1999; Cannon, 1985; Geisler & Albrecht, 1997; Georgeson, 1991; Sclar et al., 1990).

The second consequence is that the V1 would have to have the resolutional grain to represent differences in perceived contrast. It can be assumed that V1 has this grain, simply because V1 can account for contrast discrimination (Boynton et al., 1999; Geisler & Albrecht, 1997) and that requires at least the same resolution as perceived differences, if not an even higher resolution. In this respect perceived contrast is one of the only dimensions for which the grain requirement has been demonstrated at all.

Future studies may help further elucidate the role of extrastriate visual areas in contrast perception. This would need to be studied using single-cell recording in animals or using fMRI, which can give a detailed resolution of the different extrastriate visual areas (DeYoe, Bandettini, Neitz, Miller, & Winans, 1994; DeYoe et al., 1996; Engel, Glover, & Wandell, 1997; Engel et al., 1994), however with a lack of temporal resolution. Preliminary fMRI studies performed by the author in Magdeburg have confirmed the possibility of recording contrast response functions in fMRI, as has been shown by other authors (Boynton et al., 1999; Tootell et al., 1998). However the logic of the current study was not applicable because it proved impossible to separate target and mask responses. First, the *spatial* resolution of fMRI was not high enough to enable one to construct stimuli, where target and mask would activate different volume elements (voxels). Stimuli that were large enough to do so were constructed according to the known human cortical magnification factor (Horton & Hoyt, 1991b), but in these stimuli the masking effect was strongly reduced. A different strategy would be to separate target and mask transients in time, as in the present study. However, due to the temporal extension of the BOLD response the targets and masks would have to be separated so far in time, that eye movements

could not be prevented during individual trials. However, a different route is available and will be approached in a future study. Perceived contrast can also be modulated by the adaptation state of the visual system. Thus, orientation selective adaptation can be used to reduce the perceived contrast of targets in a similar manner as in the present study (Georgeson, 1991). This will again enable the separation of physical and perceived contrast and will allow the comparison of responses evoked by various visual areas to stimuli that are perceived to have the same contrast. A further route will be to compare the shape of the perceived contrast function using direct magnitude estimation (Cannon, 1985) to the shape of contrast response functions in various visual areas. Together with the present study this should provide a very detailed picture of the representation of perceived contrast in the visual system. Furthermore, it will be attempted to compare the degree of brain state coherency between the first and the second striate components. It could be hypothesized, that the degree of coherency may be higher during the late striate process, reflecting an integrated stage of processing (Rodriguez et al., 1999; Varela, Lachaux, Rodriguez, & Martinerie, 2001; von der Malsburg, 1997). A set of spectral and non-spectral measured to this ends has recently been developed and is currently being applied to neuromagnetic data (Ernst, Haynes, Heinze, & Pawelzik, 2002).

Refutation of some counter-arguments

The current study has delineated the watershed between exogenous representation of physical stimulus properties and the perceptual level of representation. The results demonstrate that the watershed might be as early as primary visual cortex, thus putting into question claims that V1 cannot directly encode a subdimension of conscious perception (Crick & Koch, 1995, 1998; Koch & Braun, 1996a, 1996b; Rees et al., 2002). The counter-examples that have been forward to support this claim will now be discussed. Most of these were aimed to show a failure of correlation between V1 response properties and certain properties of our conscious experience.

V1 does not correlate with perception

On the one hand are arguments based on data that V1 does not represent certain features such as perceived depth or perceived colour. However important these data

are in identifying the cortical representation of these specific features, they do not imply that other features such as perceived brightness and contrast could not be represented in V1. The above discussion of high-level features has shown that they are most likely to be represented in a distributed fashion in extrastriate visual cortex. Crick and Koch (1998) for example argue that the fact that responses of colour-selective cells in V1 do not exhibit colour constancy means that we cannot be aware of activity in these striate cells. However, as pointed out above, there are only 2 areas that could fulfil the necessity and mapping requirements that would be needed for an area to represent perceived colour, namely V4 and V8. V1 certainly does not represent perceived colour, simply because stimulation of V1 rarely leads to *coloured* phosphenes, whereas stimulation of the fusiform gyrus does.

Depth perception

Crick and Koch (1998) have also argued that primary visual cortex does not correlate with depth perception. Neurons in V1 respond to stereoscopic depth cues that do not lead to depth perception. Early studies suggested an important role of primary visual cortex for stereoscopic depth perception (Barlow, Blakemore, & Pettigrew, 1967; Pettigrew, Nikara, & Bishop, 1968) and some single neurons in V1 are known to respond to disparity as precisely as psychophysical depth judgements (Prince, Pointon, Cumming, & Parker, 2000). However, V1 responds equally to correlated (contrast matched) as to anti-correlated (contrast reversed) stereoscopically displaced dots, only the former of which lead to a percept of depth. Thus V1 processing is most likely a stage prior to the solution of the correspondence problem (Cumming & Parker, 1997, 2000). V1 also responds to absolute (or retinal) disparity but not to relative disparity, which refers to the difference in absolute disparities between two points in different depths (Cumming & Parker, 2000). Perceptual measures however have been shown to be highly sensitive to relative disparity (Westheimer, 1979). Thus V1 is unlikely to represent perceived depth.

There are however plenty more candidates: It has been shown that also various other visual areas (V2, V3, V3A, V4, IT, MT, MST) respond more or less vigorously to disparity (extensively reviewed in Gonzalez & Perez, 1998). Two of these areas (V3A and MT+) have been demonstrated to be closely related to psychophysical measures[73].

Binocular rivalry

There was a large and still ongoing debate on the locus of perceptual alternation in binocular rivalry. At first sight the lateral geniculate nucleus (LGN) and monocular cells in layer 4 of primary visual cortex would seem highly suitable candidates to account for binocular rivalry. Inter-ocular suppression is known to exist in LGN but it occurs also for stimuli that do not lead to rivalry[74]. The role of V1 in binocular rivalry is somewhat inconclusive: some authors find evidence for rivalry[75] and others do not. In a number of experiments recording from single cells in monkey visual cortex Logothetis and co-workers found that V1 only slightly correlated with the dominant percept in rivalry, but that specialised extrastriate areas showed strong correlation depending on the features involved (reviewed in Blake & Logothetis, 2002; Leopold & Logothetis, 1999; Logothetis, 1998). This was supported by several human fMRI studies involving playback (Lumer, Friston, & Rees, 1998), autocorrelation (Lumer & Rees, 1999) and category tagging paradigms (Tong et al., 1998).

However two recent experiments specifically designed to assess the participation of V1 in rivalry have lead to a different picture. In most earlier studies one of the two rivalling inputs is a complex shape or object, stimuli for which inferior temporal lobe structures are known to be highly selective. Thus there is not only a local change in spatial pattern but also in the mapping to an object category (see chapter 2). The study by Polonsky and coworkers (2000) used conflicting orientation stimuli "tagged" with the low level feature contrast (Fig. 52). This rivalry was purely based on low level features and did not permit complex semantic interpretation. The authors could demonstrate that the V1 signal was high during dominance of the orientation that was tagged with the high contrast and low during dominance of the orientation tagged with the low contrast. A study by Tong further investigated the role of inter-ocular switching by comparing BOLD fMRI responses from the monocular region in V1 representing the blind spot (Tong & Engel, 2001). For the blind spot representation BOLD fMRI activity is higher during phases of ipsilateral eye dominance than contralateral eye dominance. Using event related potentials it was recently demonstrated that bottom-up processing of visual stimuli presented to the suppressed eye are significantly attenuated (as compared to the dominant eye) as early as striate

cortex (de Labra & Valle-Inclan, 2001; Valle-Inclan, Hackley, de Labra, & Alvarez, 1999).

How can this lack of correlation in single unit recordings from V1 be reconciled with the findings of Polonsky et al. (2000) and Tong et al (2001)? One possible explanation could be found in the data of Fries. They recorded from the primary visual cortex of awake strabismic cats during inter-ocular rivalry. Using optokinetic nystagmus as an indicator of perceptual dominance they could show that neurons coding a currently dominant percept increased their synchrony as measured by cross correlation whereas non dominant percepts decreased their synchrony (Fries, Roelfsema, Engel, Konig, & Singer, 1997). This is in accord with a model by Lumer. Perceptual rivalry between input from both eyes may be due to a lack of synchronisability between monocular inputs (Lumer, 1998). If the relative timing of spikes is what leads to perceptual dominance then perceptual alternation could possibly be achieved with only small changes in spike rates.

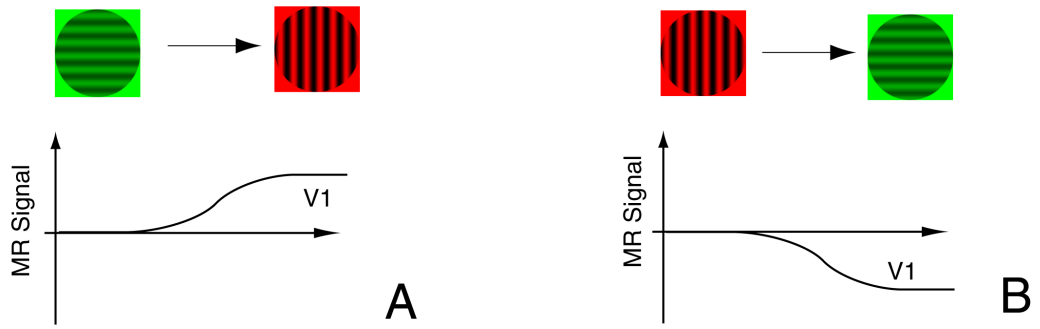

Fig. 52: Binocular rivalry between gratings of orthogonal orientations that bear no complex object interpretation. The two orientations are tagged with a "contrast marker": When the low contrast stimulus (green) gives way to the high contrast stimulus (red) there is an increase in activity in primary visual cortex and vice versa (Polonsky et al. 2000). The contrast difference alone does not lead to rivalry.

No perceptual effects of changes in single-cell responses

If there were cases where perception changes along a certain low-level feature dimension but not the response in V1 this would mean a violation of the covariance

criterion and would mean V1 cannot account for this perceptual difference by adopting a different state. As discussed just now, the cases that have been brought forward either relate to visual features that are far more likely to be represented in other visual areas, or to cases where closer scrutiny does actually show a covariance of V1 if the appropriate stimuli are chosen and if the right response property of the neural population is chosen (e.g. spike synchronisation). A different type of argument against representation of a feature dimension in V1 would be the demonstration that perception does not always follow response changes in V1. If the neural population that hypothetically encodes a feature dimension changes its state then one would expect that the perceptual state also changes[76]. This has been supported by two empirical paradigms, orientation selective adaptation without awareness and flicker fusion.

Selective adaptation without visual awareness

In a series of behavioural experiments Sheng He and others (He, Cavanagh, & Intriligator, 1996; He & MacLeod, 2001) demonstrated that orientation-specific adaptation is possible with grating stimuli even when observers cannot perceive their orientation. To achieve this the adapting grating had to be either (a) presented at great eccentricities and surrounded by masks (He et al., 1996) or (b) have such a high spatial frequency that it was beyond the resolvability threshold, which was achieved using laser interferometer patterns (He & MacLeod, 2001). The unperceived orientations of the gratings nonetheless lead to adaptation, which was measured indirectly as a reduced contrast sensitivity and by tilt aftereffects on gratings presented after the adaptation. As orientation selective adaptation can not occur before V1 this ingenious study suggests that it is possible to activate cells in primary visual cortex selectively (i.e. differentially) without awareness of the stimulus, thus questioning if V1 responses really correlate with awareness of low-level visual features (here spatial pattern).

One way to respond to this argument would of course be to drop the assumption that V1 represents any dimension of visual awareness, because our perception does not correlate with these V1 responses leading to orientation selective adaptation. This would mean sacrificing the established body of evidence presented above (showing that necessity and mapping requirements are fulfilled in V1), and switching to a

hypothetical different neural population. However other neural populations fail to correlate with low-level perception in far more serious ways, such as not being necessary at all for low-level visual perception. Maybe a revisit of the mapping requirements could point towards a different solution. Possibly the problem is one of choosing the right subset of cells in V1. Not every neuron in V1 must necessarily correlate with a given perceptual dimension. V1 has a vast number of functionally distinct cells, so that it may have subsets of cells for different feature dimensions (Fig. 53). The simple cell model used in many studies that has a tuning varying in orientation and spatial frequency and that can be used to account for elementary contrast and spatial pattern perception is certainly a strong simplification. One further functional distinction may exist between cells representing perceived contrast and perceived brightness.

Orientation selectivity can be found as early as the first recipient layers 4Cα and 4Cβ in primary visual cortex (Hawken & Parker, 1984). Thus, it could be hypothesized, that the high-resolution adapting stimuli of He and MacLeod (2001) selectively adapt the input layer, but do not get passed on to the output stages of primary visual cortex. Also, it has been shown in the present study that lateral masking occurs as early as the first responses in primary visual cortex. Thus, the fact that He and Cavanagh's lateral masking stimulus is invisible could be due to an early bottom-up filtering process, preventing the stimulus from participating in the late stages of representation, which correlate with conscious perception.

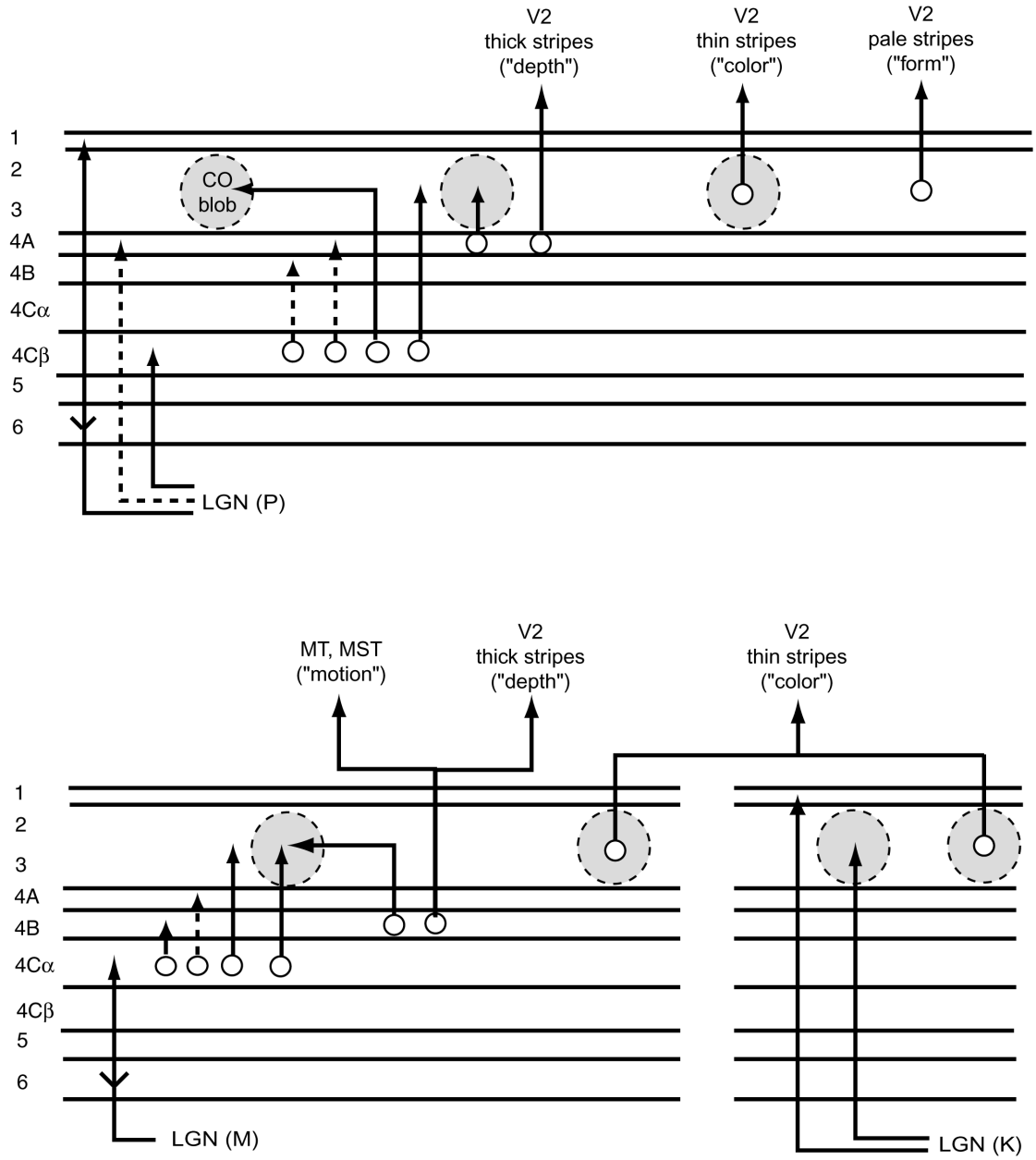

Fig. 53: Major feed-forward routes through primary visual cortex taken by projections from parvocellular (P), magnocellular (M) and koniocellular (K) cells in lateral geniculate nucleus. The connections are ordered by source layer (Callaway, 1998; Casagrande & Kaas, 1994; Kuljis, 1994; Lund, Yoshioka, & Levitt, 1994; Merigan & Maunsell, 1993; Peters, 1994; Preuss, Qi, & Kaas, 1999). The separated retinogeniculate streams project to separate layers of primary visual cortex[77]. The magnocellular layers project to layer 4Cα[78], and from there the main projections go via 4B to MT and the thick stripes of V2. The parvocellular layers of LGN project in their majority to layer 4Cβ and from there the main projections pass blob and interblob regions of layer 2/3 and project to different subdivisions of V2. The koniocellular layers project in their majority to the cytochrome-oxydase blobs in layer 2/3. Furthermore at least two within-area feedback loops have been identified within V1 (Callaway, 1998). The complexity of layers and connectivities is paralleled by a multitude of response profiles with different selectivity profiles (DeValois & DeValois, 1990; Lennie, 1998).

V1 responds to flicker beyond flicker fusion frequency

The most discussed piece of evidence within this category is the fact that activity in primary visual cortex can follow stimulus changes that occur at far higher frequencies than we are able to perceive (Crick & Koch, 1998; Gur & Snodderly, 1997; Rees et al., 2002). If the luminance of a uniform field is modulated at a mid range frequency (say 5 Hz) the EEG potential recorded over the occipital pole will show a strong oscillatory contribution at precisely the frequency of the stimulus along with weaker responses at the subharmonics and at higher harmonics (Herrmann, 2001; Lyskov, Ponomarev, Sandstrom, Mild, & Medvedev, 1998; Maier et al., 1987; Regan, 1968; Spekreijse, Estevez, & Reis, 1977; Sternheim & Cavonius, 1972; Van der Tweel & Lunel, 1965). Interestingly the frequency of the fundamental can be recorded even beyond typical frequency values for flicker fusion (Herrmann, 2001; Lyskov et al., 1998; Spekreijse et al., 1977; Van der Tweel & Lunel, 1965), where the perception of flicker gives way to the perception of a uniform grey surface. This can be interpreted to indicate that primary visual cortex has *information* about stimulus features that we do not perceive.

As with most visual thresholds the flicker fusion border is not fixed but strongly dependent on stimulus properties such as size or modulation depth (Spekreijse et al., 1977; Van der Tweel & Lunel, 1965; Watson, 1986). Modulation depth refers to the luminance difference between the bright and dark phases of the flickering stimulus. If modulation depth is increased, flicker is perceived at higher frequencies. This can be seen in Fig. 54A. In order to perceive a flicker at 10 Hz one has to choose a modulation depth of more than approx. 5 % (dashed line). In order to perceive a flicker of 20 Hz one has to choose a modulation of over approx. 50 % (dotted line). In this case a very small surface was modulated. Using different stimulus parameters values of above 50 Hz can be obtained.

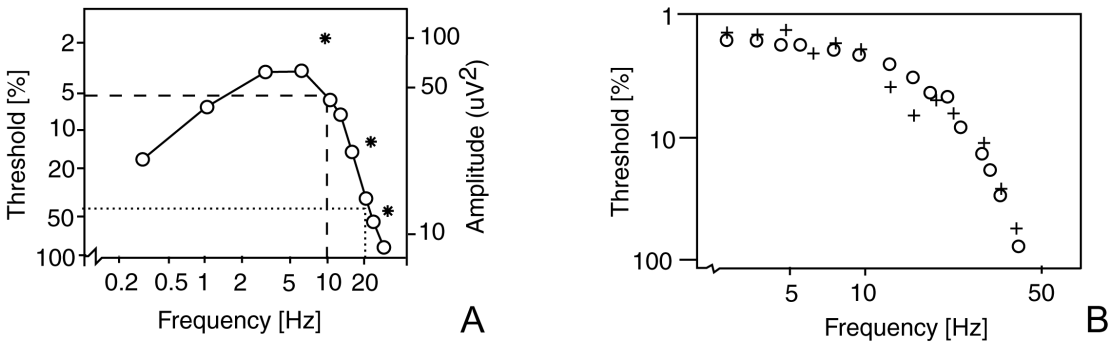

Fig. 54: (A) Left axis, circles: Flicker fusion thresholds modified from Spekreijse et al. (1977). The critical modulation depth is plotted as a function of flicker frequency for a stimulus of 22' (dashed and dotted lines: threshold modulation depths for 10 Hz and 20 Hz respectively). Right axis, stars: Amplitude of the steady state fundamental in the frequency range where the perceptual transition from flicker to a uniform surface occurs (data estimated visually from Herrmann 2001 figures 2 and 3). (B) Concurrent measurement of flicker fusion threshold (o) and steady state VEP thresholds (+) using small grating stimuli (Sternheim & Cavonius 1972).

Recordings in cat and monkey primary and secondary visual cortex (Gur & Snodderly, 1997; Rager & Singer, 1998) have confirmed that both these areas show steady state responses up to approx. 50-60 Hz (as shown using single cell recordings, multi-unit recordings and local field potentials)[79]. The fact that steady state VEPs, single cells and local field potentials follow stimulus frequencies beyond flicker fusion should not obscure the fact that the amplitude of the response in most cases does show a strong decrease in the range of flicker fusion (Fig. 54A, right axis). Due to the dependency of thresholds on other stimulus parameters it seems necessary to record the critical fusion frequency and the steady state VEP amplitude concurrently. This was done in a study by Sternheim & Cavonius (1972). They used phase reversing gratings that have an additional advantage of keeping overall retinal illumination constant throughout a cycle. After measuring the response amplitudes for various modulation depths they extrapolated the data to obtain the physiological threshold modulation depth. Using this procedure the perceptual and physiological thresholds closely match (Fig. 54B). An interesting result regarding the temporal resolution of perception and responses in V1 comes from Brindley and Lewin's (1968) studies using direct cortical stimulation of the primary visual cortex. Surprisingly there was no flicker fusion at high frequencies. The flicker rate was directly related to the frequency of the pulsed stimulation (see Fig. 9C) even far above

100 Hz[80]. This points to a close correlation between V1 response modulation and perceived brightness.

A recent study by Rager and Singer (1998) may help towards understanding the fact that high frequency responses can often be recorded from V1 despite our inability to perceive flicker. They stimulated with frequencies between 2 and 50 Hz and recorded multi-unit activity and local field potentials from primary and secondary visual cortex in anaesthetised cats (Fig. 55). An inspection of their data reveals that multi-unit activity in V2 is actually stronger than in V1 at higher frequencies (compare their figures 4C, 5C) bringing into question whether the high frequency steady state EEG response is actually mainly generated in V1. Also multi-unit activity and local field potentials show a strong decrease in the range of critical fusion frequency, especially when stimulated with stimuli confined to their receptive fields. The authors were also able to demonstrate that the responses to high-frequency flicker are not monotonous functions of frequency but show prominent strong peaks existence of resonance frequencies at multiples of certain low fundamentals. Herrmann (2001) showed the same for steady-state EEG. These are interpreted to reflect *resonance phenomena* related to endogenous rhythms around 40 Hz which have been brought in connection with feature binding and visual awareness (Basar-Eroglu, Struber, Schurmann, Stadler, & Basar, 1996; Crick & Koch, 1990; Eckhorn et al., 1988; Engel, Fries, Konig, Brecht, & Singer, 1999; Engel & Singer, 2001; Gray & Singer, 1989; Metzinger, 1995; Treisman, 1996; von der Malsburg, 1997).

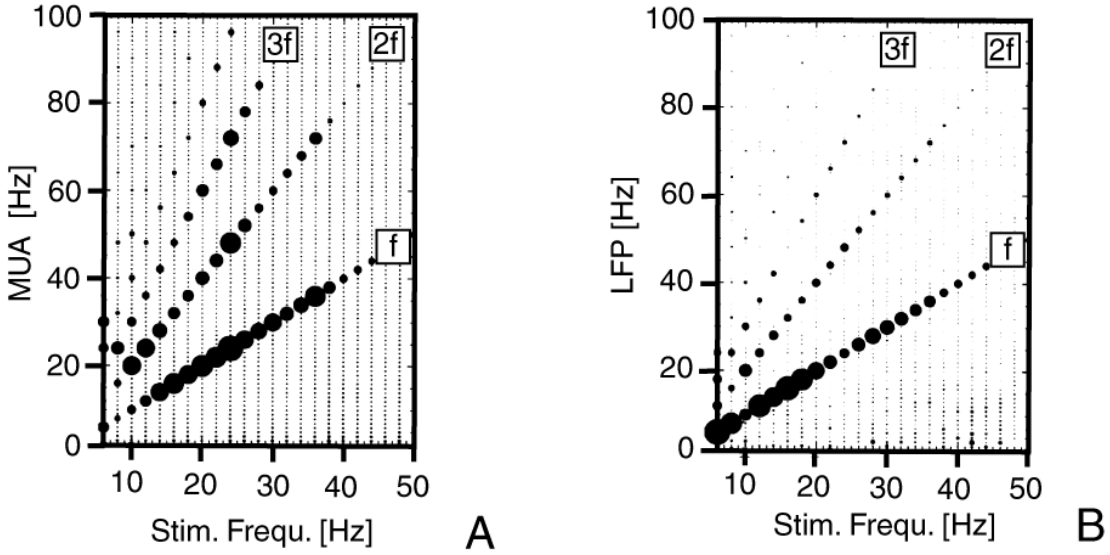

Fig. 55: High frequency responses as resonance phenomena. The power spectrum is shown as a function of stimulation frequency for (A) multi-unit activity (MUA) and (B) local field potential (LFP). Power is indicated as the diameter of the filled circle for each position on the stimulation frequency / response frequency grid. A power spectrum for a single stimulation frequency can be viewed by selecting a vertical line in the plot. The f, 2f and 3f symbols indicate responses at the fundamental frequency and the first two harmonics. It is clear that the response amplitude strongly declines for both MUA and LFP between 20 and 50 Hz. Multi unit activity was obtained by bandpass filtering the electrode signal from 1-100 Hz. Local field potential was obtained by bandpass filtering the signal from 1-3 kHz. Modified plots from Rager and Singer (1998), figures 11 C, E.

Lack of connectivity

One popular defining criterion of visual awareness is that to be aware of representations means to have them under executive control for arbitrary action patterns, especially for verbal report (Crick & Koch 1995, 1998; Chalmers 1996). Crick and Koch (1995, 1998) argue that this means that the neural population has to have a direct projection to "the planning stages of the brain", i.e. to prefrontal cortex. They support this by the fact that complete ablation of the frontal lobes in monkeys leads to complete blindness, despite response properties in primary visual cortex remaining largely normal (Nakamura & Mishkin, 1986). This position is then used to argue that V1 could not represent any feature dimension, because it lacks a direct projection to the prefrontal cortex and to the frontal lobes in general, and thus processes in V1 could not be accessible in the same way as our conscious perception.

Other authors have criticised this notion that neurons in decision space must necessarily have a *direct* connection to those in perceptual space (Block, 1996; Pollen, 1995). A different substrate that could be used for executive access could be the extensive feedback projections that most visual areas have onto V1 (Fig. 56). Bullier (2001) for example has estimated that a feedback projection from V2 to V1 could be completed within 2 ms. V2 in turn has mutual projections to several areas in the frontal lobes (Felleman & Van Essen, 1991). The feedback projections from V2 to V1 are slightly more branched than the feed-forward projections, but nonetheless largely preserve retinotopy (Rockland, 1994, 1997). This means that there is a suitable neural candidate that could account for executive access to V1, namely via fast V2 to V1 connections.

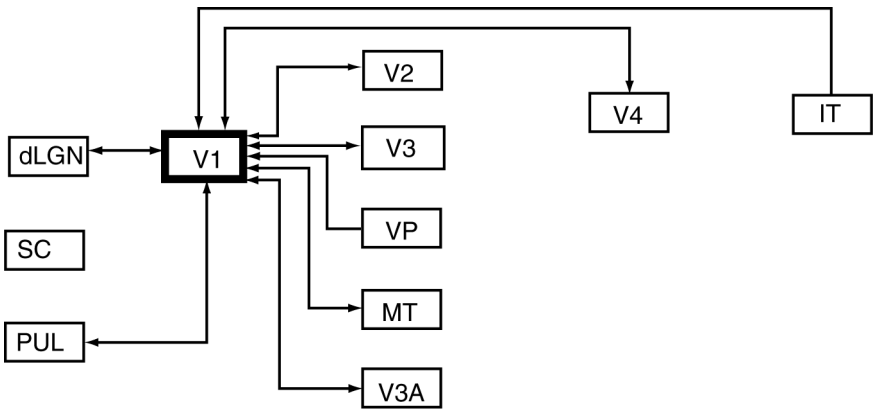

Fig. 56: Feed-forward and feedback connections to and from visual area V1 (based on data by Bullier et al., 1994; Casagrande & Kaas, 1994; Felleman & Van Essen, 1991; Kuljis, 1994; Lamme & Roelfsema, 2000; Merigan & Maunsell, 1993; Rockland, 1994, 1997)(PUL=pulvinar; SC=superior colliculus).

Modularity and binding

In the preceding chapters it has been demonstrated that some dimensions of conscious perception are most likely to be represented in primary visual cortex, whereas others are most likely to be represented in a number of other visual areas. There is obviously a clear "multiple dissociation" between the sites representing different features. As has been shown above perceived high-velocity motion is most likely to be represented in the human MT+ complex, whereas perceived colour hues are represented in the fusiform gyrus and representation of high-level object properties occurs in lateral

occipital cortex and in the fusiform gyrus. This goes beyond the previously postulated dissociation between areas that are specialised for processing different visual features and demonstrates that our conscious perception of these features is also modularly represented. This naturally brings up the question of why perceptual experience is that of a coherent "whole", rather than of a fragmented set of different feature dimensions.

The search for a mechanism of integration is also known as the "binding problem". This refers to the question of how the brain achieves the integration of representations that are processed by different neural populations into a coherent percept. These neural populations can be separated *within* visual areas, such as in cases where objects in different parts of the visual field are grouped according to Gestalt laws such as proximity, similarity, common motion or common fate (Wertheimer, 1923). It has been demonstrated that the brain may use time as an additional dimension to separate neural representations belonging to different objects (Engel, Roelfsema, Fries, Brecht, & Singer, 1997). Spike cross-correlation between neurons processing spatially separated stimuli that are perceived as belonging to the same object is enhanced and shows marked peaks pointing towards a phase-coupling of their responses (Engel, Konig, Kreiter, & Singer, 1991; Freiwald, Kreiter, & Singer, 1995). This within-area integration is the simplest case of the binding problem. If binding is to be achieved between different feature dimensions a mechanism has to be devised that can provide functional integration between the neural populations representing these different features. As some different feature dimensions are represented in different visual areas (such as luminance contrast and colour hue), the synchronisation account would predict spike synchronisation between different visual areas, which has been demonstrated (Engel, Kreiter, Konig, & Singer, 1991).

Other authors have proposed similar temporal correlation mechanisms based on re-entrant processing, which have been shown to be able to account for Gestalt grouping (Sporns, Tononi, & Edelman, 1991). If re-entrant processing is not only necessary for feature binding but also for visual awareness, as proposed by several authors (Bridgeman, 1971, 2001; Damasio, 1989; Lamme & Roelfsema, 2000; Super et al., 2001; Tononi & Edelman, 1998), this would give a natural account of why the closest match between conscious perception and evoked responses occurs only at later stages of processing. This has been demonstrated for contrast in the current study, where the

second striate response matches perceived contrast better than the first. As reported above, the better match between perception and late responses has also been shown for perceived brightness (Kinoshita & Komatsu, 2001), metacontrast masking (Bridgeman, 1975, 1980; Macknik & Livingstone, 1998) and texture segmentation (Super et al., 2001). Furthermore, disruption of feedback can render stimuli invisible (Pascual-Leone & Walsh, 2001). All of these studies have demonstrated a close correlation between perception and late processing stages in primary visual cortex[81].

Final remarks

An eliminativist position states that the "temperature of a gas is nothing else than the mean kinetic energy of its molecules". If one were an eliminativist one may interpret the results of the present study to mean that "perceived contrast is nothing else than the mean response amplitude of a subpopulation of cells in striate cortex". On the other hand, if one were a property dualist one may now argue that "perceived contrast is a phenomenal property of the response amplitude of a subpopulation of cells in striate cortex". The results of the current study do not allow deciding between the various metaphysical positions that have been proposed for the relationship between conscious experience and brain states. However, it does present a first step in the reduction of a dimension of conscious perception onto a physiological property of a population of cells in primary visual cortex.

Our everyday models of perceptual processes are specified at a number of different levels of resolution. A psychophysicist who is not interested in the neural basis of perception may interpret an experiment on perceived contrast as follows:

> *The subject is presented with visual stimuli varying along the dimension of luminance contrast. He perceives each as a spatial pattern with a certain contrast determined by a non-linear contrast transducer function, recalls which numbers he has assigned to other perceived contrasts, maps the current sensation on this scale and responds with the according number.*

A different view may now be offered, where subjective perceptual and cognitive concepts have been replaced by the neural processes taking the same causal roles:

> *The subject's retina is presented with visual stimuli varying along the dimension of luminance contrast. These lead to a cascade of events in the visual system, one of which is a response of varying amplitude in a subset of cells in primary visual cortex at a late stage of processing. A set of distributed representations is reactivated that associates response amplitudes of this population with an activation vector in the parietal representation of the "number line" (Dehaene & Cohen, 1995; Pinel, Dehaene, Riviere, & LeBihan, 2001), which is the basis for the subject's differential response.*

In a neural theory of contrast perception every occurrence of a percept with a particular magnitude of contrast will be replaced by a certain response amplitude in this neural population, to which the perceived magnitude is mapped. In every case when a percept changes along this dimension, the neural response will adopt a different state, and the percept can be mapped to this neural state, which further on inherits its causal role.

Of course, by following this research program, the "explanatory gap" (Levine, 1983) — or "hard problem" (Chalmers, 1996) — remains unsolved, in the sense that even if we had established a perfect neural substitute for perceived contrast, we would still fail to know *why it is* that these brain-processes are accompanied by just these qualitative experiential states. This problem holds for the same reason that we do not have an answer to the question of *why* the world is such that gravitation decreases as the square of the distance between two masses increases. "That's the way it is" is all a scientist can answer, and several ontologies may be constructed upon the same set of empirical data (Smart, 1959). It is however possible to follow a *nomological* research strategy (investigating which laws govern the behaviour of qualitative states) even if the *ontological* problem (of what qualitative states "are") is principally unsolvable. The search for the neural representation of dimensions of conscious perception can thus be viewed as the search for bridge laws (Nagel 1961) that map phenomenal onto

physiological concepts. If such bridge laws were known, one would be able to predict a subject's conscious perception by a measurement performed on his brain. In the current study it has been shown that this is possible, for the low-level dimension of perceived contrast.

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Footnotes

[1] Even if perceived brightness and contrast at a position in the visual field depend on spatial context (Adelson, 1999; Ejima & Takahashi, 1985) the effect of this surround modulation is nonetheless a change in perceived brightness or contrast at a specific local position in the visual field, thus justifying calling perceived brightness and contrast "local" aspects of conscious perception.

[2] The term "perception" rather than "experience" was chosen because of its greater familiarity, although experience is the more adequate term. Perception implies an external object that is being perceived. In the case of visual imagery, hallucinations, direct cortical stimulation, etc. there is no external object being perceived, but the observer has an experience of certain visual features, often giving rise to his belief that they were evoked by an external object. The term "visual features" was used to allow for the modular representation of the various dimensions of conscious perception.

[3] The importance of the brainstem reticular formation for control of arousal was demonstrated by showing that its stimulation in lightly anaesthetised animals had a desynchronising effect on the EEG, which is typically observed when animals and humans wake up (Moruzzi & Magoun, 1949). Also brain transsections at the level of the midbrain, that separate the reticular formation from thalamus and cortex, lead to irreversible sleep whereas transsections below the reticular formation at the lower brainstem leave the animal awake but largely immobile with spared ability to respond to auditory and visual stimuli by eye and face movements (Bremer, 1935). In humans lesions of the brainstem at the level of the upper reticular formation can lead to coma, whereas this is not necessarily the case for lower lesions at the level of the medulla (Plum & Posner, 1980).

[4] These structures are also involved in arousal changes in waking subjects (Kinomura, Larsson, Gulyas, & Roland, 1996; Portas et al., 1998).

[5] Anecdotal reports mention opening doors and taking streetcar rides (Young & Wijdicks, 1998).

[6] In PVS patients cortical cerebral metabolism as assessed with fluorodeoxyglucose-PET is below 50% of normals and matches that encountered during deep barbiturate anaesthesia (Plum, Schiff, Ribary, & Llinas, 1998; Schiff et al., 2002). Similarly post-mortem studies have found extensive neocortical and thalamic degeneration (Kinney et al., 1994; Kinney & Samuels, 1994; Zeman, 1997).

[7] Vertical eye-movements and movement of upper eyelids are controlled by cranial nerve III (oculomotor nerve), which originates from the oculomotor nucleus in the midbrain at the level of the superior colliculus. Horizontal eye-movements however are controlled by cranial nerve VI (abducens nerve) originating from the nucleus abducens in the caudal pons. This explains why vertical but not horizontal eye-movements are often spared in locked-in syndrome, which occurs mostly after brain stem lesions at the level of the pons.

[8] See the discussion in the New England Journal of Medicine, Vol. 331 (20), pp. 1378.

[9] Under certain circumstances hemineglect can be falsely classified as hemianopia (Kooistra & Heilman, 1989), but visual neglect can occur even when there is no blind region in the visual field (Halligan, Marshall, & Wade, 1990).

[10] When the term "state" is used it is not meant to imply a single physiological state of the system, defined down to the microphysical level, but to a state defined as a *function* over a neural population, such as "mean activity" or "coherency".

[11] Some possible states of the neural system may simply be irrelevant for conscious perception.

[12] Cases of lesions are the only cases where the neural state is not defined. A perceptual dimension can trivially not be represented by a neural population if it can still occur when the neural population is lesioned.

[13] Mostly the low-level feature statistics are not kept constant in a satisfactory way. This is due to the method used, where a grid is applied to the image and then the positions of individual cells are randomised. This of course changes the spectral composition of the image, leading to the alternative interpretation that visual areas found in these studies do not differ in their selectivity to high-level features but in their spatial-frequency selectivity.

[14] Visual imagery is a different paradigm that can be used to study changes in perception without changes in stimulation. A subject is asked to imagine a spatial pattern without any exogenous stimulus (reviewed in Kosslyn, Ganis, & Thompson, 2001). Visual imagery is interesting because it allows one to correlate a perceptual change that occurs purely intentionally, to a change in brain state. However the imagined spatial pattern is largely uncontrollable and thus the perceptual state is only weakly defined. Perceptual illusions also fall into this category. If a stationary stimulus is perceived to move then motion perception can be studied independent of motion stimulation (Zeki, Watson, & Frackowiak, 1993). This can also occur in the form of aftereffects. If a moving stimulus is viewed for a certain time visual motion detectors are adapted. A subsequently presented stationary stimulus is perceived to move. This can also be used to study motion perception without motion stimulation (Huk, Ress, & Heeger, 2001).

[15] However this can only be positive evidence. If a neural population is unable to account for discrimination, it cannot be ruled out that it might have sufficient grain for perceptual representation.

[16] This does not hold for example for perceived orientation.

[17] An example may make this clear. Consider a forest fire "caused" by a short circuit. Was the short circuit a necessary condition for the fire to occur? No, because the fire could have been caused in other ways, for example by a burning cigarette. The short circuit is also not sufficient for the fire to occur because the same fire would not have been caused if the forest had not been dry. But the short circuit can be interpreted as a "cause", because it is a necessary part of one sufficient set of conditions. However this sufficient set of conditions is not necessary, because other sets of sufficient conditions can exist which do not make the cause "short circuit" necessary (e.g. a burning cigarette plus a dry forest). So the short circuit is an insufficient but necessary part of a condition that is itself unnecessary but sufficient for the result (hence INUS).

[18] A note of caution should be added here when interpreting results from lesion studies. After lesions of primary visual cortex for example more is lost than simply activity in V1. As V1 is the main route of signal flow into visual cortex, many visual areas show strongly reduced activity after V1 lesions. Early extrastriate visual areas V2 and V3/VP, as well as areas in the temporal pathway (V4, IT) are largely

silenced after V1 lesions, whereas V3A and MT still show activity (Bullier, Girard, & Salin, 1994; Milner & Goodale, 1995; Rocha-Miranda, Bender, Gross, & Mishkin, 1975). A lesion in V1 will leave responses in the superior colliculus and pulvinar largely unchanged but will lead to retrograde destruction of parts of LGN and even retina (Cowey & Stoerig, 1989; Cowey, Stoerig, & Perry, 1989).

[19] Transcranial electrical stimulation on the other hand has the disadvantage of being strongly obscured and blurred by the high resistance of the skull (Merton & Morton, 1980; Nathan, Sinha, Gordon, Lesser, & Thakor, 1993).

[20] These "figure-of-eight" coils induce the most focal intracerebral currents.

[21] Studying V1 with TMS has problems. Electromagnetic modelling using three layer brain models of brain, skull and scalp predicts that current drops to 75 % within the first 10 mm of tissue (Roth, Cohen, & Hallett, 1991) and thus only reaches cortex on the surface of the brain. The parts of V1 representing peripheral vision are buried in the depth of the calcarine sulcus and are separated from the scalp by V2. Thus V1 cannot be disrupted without at the same time disrupting activity in V2 and the representation of the most peripheral visual field can possibly not be studied at all.

[22] Direct cortical stimulation can be interpreted to allow statements about the direction of "causality" between neural and mental states. However direct cortical stimulation is no different in this respect to a distal stimulus, which can also be thought of as a cause of a sensation. The real advantage of cortical stimulation is that it allows setting the starting point of a chain of neural events with much greater precision.

[23] Due to the immense reciprocal feed-forward and feedback connectivity in visual cortex stimulation of one visual area cannot be expected to be restricted to the site of stimulation (Callaway, 1998; Casagrande & Kaas, 1994; Felleman & Van Essen, 1991; Lamme & Roelfsema, 2000; Lamme, Super, & Spekreijse, 1998; Lennie, 1998; Merigan & Maunsell, 1993; Rockland, 1994). Effects will also certainly depend on the layers stimulated. So the conclusion that areas V_1 to V_{n-1} can be ruled out in this way is slightly optimistic. Co-recordings of EEG and TMS have shown that activity does spread significantly from the site of stimulation (Ilmoniemi et al., 1997).

[24] Intracranial stimulation is available from three main patient populations: (1) Patients in neurosurgical operations for removal of tumours and epileptogenic foci (Penfield & Rasmussen, 1950). (2) Blind patients participating in studies on artificial implants for visual prostheses (Brindley & Lewin, 1968; Dobelle, Mladejovsky, Evans, Roberts, & Girvin, 1976). (3) Patients with temporarily implanted electrodes for diagnostics of epileptogenic foci over the lateral and medial surfaces of the occipital and temporal lobes (Lee et al., 2000).

[25] They confirmed that the second colour area V8 contains a representation of the entire contralateral visual hemifield. There are divergent results and interpretations as to whether "ventral V4" contains a complete representation of the visual field (Bartels & Zeki, 2000) or only an upper quadrant (Hadjikhani et al., 1998), with the corresponding lower quadrant in dorsal occipital cortex (Tootell & Hadjikhani, 2001).

[26] Penfield and Rasmussen claim to have produced colour sensations by stimulation of primary visual cortex. They even claim that the "calcarine image was more often coloured while images produced

from the secondary visual zone more often consisted of colourless light" (p. 142 in Penfield & Rasmussen, 1950). However Penfield and Rasmussen never stimulated the calcarine region inside the interhemispheric fissure, where the major part of V1 is located. It cannot be clearly reconstructed from their data whether stimulation leading to colour sensations really did occur in V1, or possibly rather in extrastriate visual cortex. Furthermore, their data contradict most results of direct cortical stimulation studies performed using chronically implanted electrode grids directly covering a large extent of V1 (Brindley et al., 1972; Brindley & Lewin, 1968; Dobelle et al., 1974; Lee et al., 2000).

[27] The spectral composition of light that is reflected by an object is a joint function of the invariant reflectance properties of its surface and the spectrum of the illuminating light. Colour constancy refers to the fact that an object's perceived colour is not determined by the reflected spectrum, but by the invariant reflectance properties of the object, which is achieved by discarding for the illuminant (Judd, 1940; Land, 1959a, 1959b). To achieve this our visual system is able to take into account complex properties such as local and global context (Kraft & Brainard, 1999), overall scene statistics (Brown & MacLeod, 1997; Golz & MacLeod, 2002) and even physical knowledge of mutual illumination (Bloj, Kersten, & Hurlbert, 1999).

[28] Some human lesion studies suggest that colour constancy in V4 may only be computed by interaction with other areas. Patients with selective colour constancy deficits that were still able to discriminate and name colours typically had lesions around the superior and medial temporal gyrus, but not in fusiform gyrus (Ruttiger et al., 1999).

[29] In monkeys a complete ablation of area V4, which some authors believe to be the monkey colour area, does not severely affect colour discrimination (Heywood et al., 1992). However this only questions whether V4 is really the major monkey colour area, especially as there are several failures to find an abundance of colour-selective cells in V4. Recently some authors have reported that in a rare case of a blind patient with spared colour vision colour stimuli only activated area V1 but not V4, whereas in a normal subject both V1 and V4 were activated (Zeki, Aglioti, McKeefry, & Berlucchi, 1999). The patient lacked colour constancy and his colour perception was purely wavelength-based. Zeki et al. interpret this as evidence that V1 alone can provide for conscious colour perception, albeit only in its native wavelength-based format of representation. However careful analysis of this study reveals that if the threshold criteria for control subject and patient are matched then also the patient shows activation of V4.

[30] Although in some animals motion selectivity can be found as early as in the retina (Barlow, Hill, & Levick, 1964) motion processing in primates is a cortical phenomenon (Culham et al., 2001).

[31] MST differs from MT mainly in its response to complex flow patterns such as rotation, contrast and expansion (Graziano, Andersen, & Snowden, 1994; Tanaka, Fukada, & Saito, 1989).

[32] Similar to monkey MT human MT+ contains a complete representation of the contralateral visual field and even extends up to 20° into the ipsilateral visual field (Tootell, Mendola, Hadjikhani, Liu, & Dale, 1998; Tootell, Reppas, Kwong et al., 1995). There is some evidence that human MT+ may also be retinotopically organised (Kansaku et al., 2001). However, it is not clear if these authors provide evidence for retinotopic organisation or for the functional subdivision between MT and MST. MST is

believed to be specialised for more complex motion patterns such as rotation or expansion (Graziano et al., 1994).

[33] The activity assigned to dorsal visual area V3 by the authors of this study could possibly reflect V3A. This fine-grained distinction is impossible to make based on PET data, for which no retinotopic mapping is available. V3A is also strongly involved in processing of stereoscopic depth (Gonzalez & Perez, 1998)

[34] Due to the proximity of MT+ and KO it is actually possible that many supposedly selective neurological or experimental MT lesions in fact involve both areas (Beckers & Homberg, 1992; Beckers & Zeki, 1995; Plant, Laxer, Barbaro, Schiffman, & Nakayama, 1993; Zihl et al., 1983).

[35] Sensitivity to motion-defined texture and shape was also demonstrated as early as V1 by other authors (Reppas, Niyogi, Dale, Sereno, & Tootell, 1997).

[36] This is paralleled by the fact that macaque monkeys do not completely lose motion vision after MT/MST ablations (Rudolph & Pasternak, 1999).

[37] One patient reports to see motion as "a black shadow moving on a black background" (Zeki & Ffytche 1998, p. 30).

[38] Zeki and Ffytche (1998) could even show for their Riddoch patient G.Y. that MT+ was activated by motion. Awareness versus unawareness of motion in Riddoch patients was correlated with increased activity in MT+. Other authors even found stronger activation of superior colliculi in the same patient G.Y. in the unaware mode suggesting a shift from cortical to subcortical processing (Sahraie et al., 1997). Surprisingly this residual motion vision can even occur for isoluminant chromatic stimuli (Guo, Benson, & Blakemore, 1998). However, it was not possible to elicit motion phosphenes in G.Y. by application of TMS anywhere over visual cortex of his lesioned hemisphere (Cowey & Walsh, 2000).

[39] Possibly it is necessary to subdivide motion perception into a number of smaller feature classes. The massive redundancy and parallel processing of motion in the visual system (Culham et al., 2001; Greenlee, 2000; Sunaert, Van Hecke, Marchal, & Orban, 1999) may not be surprising given that motion is closely tied to the general problem of temporal integration. First there seems to be a subdivision dependent on speed and cue-type. Some authors (Gegenfurtner & Hawken, 1995, 1996; Rieger, Gegenfurtner, Tempelmann, & Heinze, 2002) have provided evidence for a dissociation between slow motion of chromatic stimuli processed by the parvo-stream and in V3A, V7 and V4 and of fast motion of luminance-defined stimuli processed by the magno-stream and in MT+. This fits to the fact stated above that MT+ lesions selectively impair processing of fast motion stimuli. KO and LOC may not represent so much motion itself but rather shapes and objects defined by motion borders. There is also evidence for a double dissociation between first and second order (contrast-defined) motion perception (Vaina & Cowey, 1996; Vaina, Cowey, & Kennedy, 1999; Vaina, Makris, Kennedy, & Cowey, 1998).

[40] It is not yet clear how the functional subdivisions of monkey temporal cortex map onto human cortex.

[41] The latter two have been studied in a rare case of single neuron recording from awake human brain (Kreiman, Koch, & Fried, 2000) and seem to be involved in memory retrieval and emotional analysis respectively.

[42] Contrast plays a strong role in surround effects. Sometimes complete reversions of the direction of effects are found at different contrast levels (Levitt & Lund, 1997; Polat, Mizobe, Pettet, Kasamatsu, & Norcia, 1998).

[43] It is not clear if single cells in these areas represent even more complex objects such as specific categories of animals. One has to be careful in interpreting the fact that a neuron can be driven strongly by a complex object such as a "tiger" to mean that the neuron is a "cardinal cell" (Barlow, 1995) for this specific object. Often the cell can just as well be driven by simplified versions of the stimulus, such as a striped area plus two circles (Tanaka, 1996). An alternative model is that categories are represented as population vectors over many cells encoding different complex shape primitives. In the latter sense monkey IT could provide a "dictionary of complex shapes" (Riesenhuber & Poggio, 2000; Tanaka, 1996).

[44] There has been a strong debate about possible confounds in studies demonstrating specialised object category modules (Gauthier, Behrmann, & Tarr, 1999; Humphreys & Forde, 2001; Ishai, Ungerleider, Martin, & Haxby, 2000). On the one hand prosopagnosia is mostly correlated with deficits in other object classes, such as animals, foods or plants (Farah, 1995) putting into question its pure face-selectivity. An alternative interpretation could be a deficit in a specialised cortical area for living (as compared to non-living) objects. But this is difficult to reconcile with the fact that recognition of automobile models is also often disrupted together with face recognition (Farah, 1995). Possibly other incidental differences between the object categories play an important role: the degree of similarity or confusability between members in a subcategory (or the degree of crowding in the local surround of an objects' activation vector in some high-dimensional feature space), structural complexity and degree of familiarity or overlearning. Furthermore certain objects typically call for certain types of processing. While we are often in a situation of having to make and remember fine discriminations between faces this is not the case for birds, cows or automobiles. Given all these possible confounds the hypothetical categorical selectivity of certain regions could be a mere artefact due to uncontrolled covariates.

[45] Position and size invariance can be largely attributed to the large size of receptive fields in inferior temporal cortex (Logothetis & Sheinberg, 1996; Logothetis, Pauls, Bulthoff, & Poggio, 1994; Tovee, Rolls, & Azzopardi, 1994).

[46] The minor response generalisations observed can mostly be attributed to invariance to mirror inflection rather than to viewpoint (Fig. 4 in Logothetis & Sheinberg, 1996).

[47] Little is known about the processing of brightness in the visual system. After the initial demonstrations that receptive fields of cat retinal ganglion cells were organised into concentric excitatory and inhibitory rings (Kuffler, 1953) it was believed that luminance contrast rather than absolute luminance was the basic achromatic representational unit in the visual system. Subsequently it was shown that also cells in lateral geniculate nucleus and visual cortex are composed of excitatory and inhibitory zones and respond only weakly to homogenous illumination across their entire receptive

fields (Hubel & Wiesel, 1959). This preference of contrast (along with neural adaptation) is important to account for the ability of our visual system to discard the influence of overall illumination level, which can vary by a factor of up to 10^8 between different viewing conditions such as weak starlight and strong sunlight (Hood & Finkelstein, 1986).

[48] Luminance is an objective measure of the power within the visible spectrum projected per steradian in a given direction per unit area of a surface. The subjective sensation evoked by this luminance varying between "dark" and "bright" is termed "brightness". The relationship between luminance and brightness is complex, because it depends on dynamic factors such as the adaptation state of the photoreceptors, and (as shown below) on the luminance of the spatial context. Because we are interested in subjective perception we will be looking for the neural representation of (perceived) brightness, not luminance.

[49] The authors recorded peri-stimulus time histograms (PSTH) during extended phases after changes in luminance. Many luxotonic cells responded in such a sustained fashion showing only minor effects of light adaptation. A subpopulation of cells maintained their enhanced discharge rate to a light increment even up to the longest time measured (one hour).

[50] The remaining 8 % of cells showed a "V"-shaped response profile with the minimum response obtained when the luminance was the same as that of the background ("V-shaped type").

[51] Brindley and Lewin (1968) also found that perceived brightness rose monotonously with the duty cycle of electrocortical stimulation pulses demonstrating a close relationship between perceptual magnitude and total current flow (see Fig. 9 C).

[52] Several controversial theories still co-exist: Inhibitory influences of transient on sustained visual channels (Breitmeyer & Ganz 1976; Breitmeyer & Ogmen 2000), retinocortical feedback loops (Purushothaman et al. 2000), co-occurrence of specific geniculostriate projections and unspecific thalamocortical arousal (Bachmann 1997), filling-in from edges (Macknik et al. 2000) and models combining feedback and lateral inhibition (Bridgeman 1971, 1977; Francis 1997). The diversity of positions is due to the fact that each theory postulates a complex network mechanism that goes far beyond currently available single-cell data.

[53] At a level of single cells the effects of metacontrast masking have been studied in various subcortical and cortical areas of both cats and monkeys. Retinal ganglion cells and cells in the lateral geniculate nucleus (Bridgeman, 1975; Gruesser, Petersen, & Sasowski, 1965; Schiller, 1968) exhibit surround masking, but with different temporal profiles than those for which metacontrast masking occurs. Macknik et al. (2000) demonstrated that in conditions where stimulus and mask are cyclically repeated single cell responses in LGN do exhibit suppression, which is strongest for small target-mask distances and decreases with increasing distance, as in metacontrast masking. This may point towards a partial account of metacontrast masking by LGN mechanisms. However the fact that dichoptic stimuli, where target and mask are presented to different eyes, also lead to strong masking effects points towards a predominantly cortical locus of suppression. When one records from later visual area V4, surround masking can still be observed, but again with different temporal parameters (Kondo & Komatsu, 2000). Early physiological studies on the neural basis of metacontrast masking used visual evoked potentials.

These have led to somewhat diverging results, some authors finding no differences between target responses for masked and unmasked stimuli (Schiller & Chorover, 1966), some authors finding differences in mid-latency components around 200 ms (Vaughan & Silverstein, 1968). Besides physical differences such as luminance and contrast level and physiological differences such as dark versus light adaptation these diverging results may be attributed to the difficulty of isolating the evoked responses to the target mask. Because they occur in close temporal succession the evoked potentials show substantial overlap.

[54] Similar results were obtained in macaque V1 by Kinoshita and Komatsu (2001) who found three classes of surround modulation: Type I (response independent of surround luminance), type II (decreasing response with increasing surround luminance) and type III (increasing response with increasing surround luminance). The type II cells are in accord with the surround normalisation of perceived brightness.

[55] Texture filling-in on the other hand may have its earliest correlates in areas V2 and V3 (De Weerd, Gattass, Desimone, & Ungerleider, 1995).

[56] The role of V2 in brightness representation is currently not clear. Using optical imaging and single unit recording it has been demonstrated that single cell activity not only in V1 but also V2 follows border-induced brightness modulation in the Craik-O'Brien-Cornweet illusion (Hung, Ramsden, Chen, & Roe, 2001). In the same study it was also demonstrated for the first time that V2 responds strongly to homogenous changes of luminance across large parts of the visual field.

[57] Visual field defects can be classified as absolute and relative, the latter referring to cases where perception is not completely disrupted but severely changed with reduced sensitivity and acuity. There is controversy as to the mechanisms of this residual vision which some authors believe to be purely extrastriate (Kleiser, Wittsack, Niedeggen, Goebel, & Stoerig, 2001) and others believe to rely on residual islands of intact cortex in V1 (Fendrich, Wessinger, & Gazzaniga, 1992). Even in absolutely blind parts of the visual field some capacity for discrimination may remain despite lack of any awareness of the stimuli. This phenomenon is known as "blindsight" and has also been demonstrated in monkeys where lesions can be set with much greater precision (Cowey & Stoerig, 1995; Stoerig & Cowey, 1997).

[58] A temporal lobectomy will not lead to visual field deficits only as long as the optic radiation is spared. The optic radiation cannot take a direct route from LGN to calcarine sulcus but has to loop around the lateral ventricle meaning it has to pass through the white matter of the temporal lobe.

[59] This is of course a doubtful practice in the light of the results on luminance processing in V1 reported above.

[60] It would possibly be more appropriate to speak of "just discriminable differences".

[61] The stimuli studied in TMS are a mixture of luminance and contrast stimuli, because on the one hand they increase overall luminance and on the other hand they increase contrast by introducing a luminance border.

[62] The authors also studied contrast perception in normal subjects, but the dissociation was only available for the amblyopic patient.

[63] Positron-emission tomography (PET) was not considered because it employs substances marked by positron-emitting radioactive isotopes, such as 2-deoxyglucose to measure regional metabolism or $H_2{}^{15}O$ to measure regional blood flow. This procedure exposes the subject to radioactivity and should only be used in clinically justified circumstances and when no other method is available.

[64] FMRI signals are only indirectly related to neural activity. Blood oxygenation level dependent contrast (BOLD) fMRI signals measure local inhomogeneity of magnetic fields in small volumes of tissue via the dephasing rate of proton spins. The inhomogeneity changes with the level of blood oxygenation because the magnetic susceptibility of oxygenated haemoglobin is low and that of deoxygenated haemoglobin is high. An increase in deoxygenated haemoglobin leads to an increased inhomogeneity of the local magnetic field, which leads to a faster dephasing of proton spins. The blood oxygenation level first shows a very small decrease after neural activity due to increased metabolic demand, followed by a strong increase, presumably as a consequence of pre-emptive vascular auto-regulation. The initial decrease in blood oxygenation is too small to be measured in most studies, but the temporally extended increase in blood oxygenation has a high signal to noise ratio and can be measured with MRI scanners.

[65] The characteristic length of a dendrite is the distance along the membrane after which a local EPSP or IPSP has fallen to 1/e of its value. The characteristic length is a measure of electrotonic spread in the dendritic tree and depends on several parameters such as the membrane resistance and the axial intracellular resistance, which in turn depends on the cross-sectional area of the dendrite (Shepherd, 1999).

[66] "Neutral" refers to the fact that the reference electrode should pick up as little as possible of the evoked responses and should not pick up any other signals systematically related to the stimulus.

[67] In the current study the magnetic field strength is measured with single coils that measure magnetic flux caused by both local and distant sources (magnetometer). A different technology uses double-coils where one coil is equivalent to the magnetometer and the other coil is wound inversely and thus effectively subtracts the distant field that is constant for both coils (gradiometer).

[68] The boundaries are modelled using triangular mesh surfaces, i.e. surfaces approximated by (typically several thousand) triangles.

[69] To put it differently: The function mapping dipole space onto topography space is single-valued but non-injective.

[70] This was not evident in the cluster analysis, which does not exploit symmetry in the data, but interprets the vectors A and –A as belonging to two different clusters.

[71] The typical biphasic shape of CRFs is not visible here because the data are presumably only recorded from the region above the inflection point in the CRF.

[72] No behavioural data were recorded, simply because the cats were anaesthetised.

[73] When applying electrical stimulation to disparity selective cells in MT it is possible to apply a bias to depth judgements of awake and behaving rhesus monkeys suggesting a role for MT+ in perceived depth (DeAngelis, Cumming, & Newsome, 1998, 2000). However lesions of MT+ (and V4) did not lead to any deficit in stereopsis in rhesus monkeys (Schiller, 1993). In a human fMRI study area V3A

(however not MT) matched psychophysical performance very closely (Backus, Fleet, Parker, & Heeger, 2001). Area V3A is distinct from V3, is located on the superior occipital cortex and contains a compete representation of the visual field (Tootell et al., 1997). Transcranial magnetic stimulation over V3A has been shown to disrupt stereopsis in humans (Takayama & Sugishita, 1994). Monkey V3A response properties have also been shown to be dependent on direction of gaze, thus V3A is possibly on the border between retinotopic and spatiotopic processing (Galletti & Battaglini, 1989). This is supported by the vicinity of V3A to parietal cortex which is the main cortical site of spatiotopic representation in egocentric and possibly also allocentric coordinates (Duhamel, Bremmer, BenHamed, & Graf, 1997; Landis, 2000; Rizzolatti, Fogassi, & Gallese, 1997). The discrepancy on the role of MT in depth processing can be explained by specialisation of V3A and MT to fine and coarse stereopsis respectively (DeAngelis et al., 2000).

[74] In LGN representations for each eye are separated but lay in adjacent laminae. Thus corticofugal feedback onto LGN - which is known to derive from various visual areas (Lin & Kaas, 1977) - could selectively gate or suppress input from one eye by suppressing input passing through individual laminae. Inhibitory inter-ocular interactions are known to exist in LGN (Schroeder, Tenke, Arezzo, & Vaughan, 1990; Sengpiel, Blakemore, & Harrad, 1995). The spike rate of an LGN cell that is stimulated by a grating presented to its dominant eye is modulated by a grating presented to the non-dominant eye. But in contrast to stimuli that lead to perceptual rivalry this inhibition is not specific to gratings of orthogonal orientations (as one would actually expect at a stage prior to orientation selectivity). So far no evidence for rivalry in LGN was found - neither in anaesthetised cats (Sengpiel et al., 1995) nor awake monkeys (Lehky & Maunsell, 1996).

[75] In recording from layer 4 of cat area 17 Sengpiel et al. (1995) found no evidence for binocular rivalry in monocularly driven cells, but strong rivalry in binocularly driven cells suggesting a locus of rivalry at or beyond striate cortex. This was observed when a grating of preferred orientation was presented to one eye and an orthogonal (non preferred) grating was presented to the other eye a few seconds later. The tuning properties of the suppression effect in cat area 17 show great similarity to human psychophysical data on binocular rivalry (Sengpiel, 1997).

[76] To be precise, this does not necessarily violate the mapping criteria formulated in chapter 2. It is highly unlikely that every cell in V1 participates in encoding every low-level feature. Luxotonic cells for example only make up a subset of V1 cells, but they nonetheless can account well for perceived brightness. Thus, this issue may be resolved by splitting V1 into subpopulations that represent different feature dimensions, such as perceived contrast or brightness.

[77] Even the most comprehensive reviews of primary visual cortex, which is the visual area for which the greatest body of data exists, admit that the true complexity of axonal and dendritic arborisation of real visual neurons can only be approximated in current models (Callaway, 1998; Casagrande & Kaas, 1994; Kuljis, 1994; Lund et al., 1994; Peters, 1994). This is further complicated by the fact that most connectivity data is only available for monkeys and not fur humans. Although for example macaque and human visual cortex show a high degree of similarity there are also some clear differences (e.g. in layer 4A of V1, Preuss et al., 1999) the functional implications of which are currently unknown.

[78] This nomenclature refers to the classical numbering of striate cortical layers by Lund and Boothe which is derived from Brodman (Brodman, 1909; Lund, Lund, Hendrickson, Bunt, & Fuchs, 1975). Although revised labelling systems have been proposed recently (Boyd, Mavity-Hudson, & Casagrande, 2000; Kaas & Collins, 2001; Kuljis, 1994) the Lund and Boothe system is still dominant in the literature.

[79] Gur and Snodderly (1997) also showed in monkeys that V1 colour opponent cells followed chromatic flicker of isoluminant stimuli up to 30 Hz which is beyond the typical chromatic flicker fusion threshold. However as discussed above the hypothetical representation of perceived colour is not in V1 but in the fusiform gyrus. Also it can be questioned whether the authors' use of a photometer with human V_λ calibration can reliably indicate isoluminance for macaque monkeys, which are known to have a slightly different wavelength sensitivity (Dobkins, Thiele, & Albright, 2000; Jacobs & Deegan, 1997).

[80] The authors took great care to ensure that the patient really experienced flicker at these high frequencies and not slower fundamentals as could have arisen from technical artefacts or a misunderstanding of the term "flicker".

[81] Synchronisation is not only a possible way to integrate disparate neural representations belonging to the same perceptual object. It has also been proposed as a mechanism of perceptual selection in multistable perception and binocular rivalry (Basar-Eroglu et al., 1996; Engel & Singer, 2001; Fries et al., 1997; Lumer, 1998). For humans this has been most impressively studied with the so-called "frequency tagging" paradigm (Srinivasan, Russell, Edelman, & Tononi, 1999; Tononi, Srinivasan, Russell, & Edelman, 1998). Two stimuli that lead to binocular rivalry when presented dichoptically can each be tagged by a different fundamental flicker frequency. This enables one to compare how widely activity evoked by each stimulus is disseminated in the brain, by comparing the power in the according stimulus' fundamental frequency in the steady state EEG/MEG under conditions of perceptual dominance and suppression. With this paradigm Tononi and co-workers have shown that perceptual selection of one of two percepts leads to wide dissemination of activity deriving from that area across many brain areas, including many non-visual areas of the parietal and frontal lobes. Also, the interhemispheric phase coherency at the selected stimulus frequency is largely increased. This points towards synchronisation and coherency as two fundamental mechanisms of functional integration and selection. Information from selected percepts may be made globally available to other areas by exploiting the neurocomputational advantages of synchronous activation. Global availability has been suggested by many authors as a fundamental property of conscious representations (reviewed in Baars, 2002). However it is not clear, whether this "global availability" of "information" means that the activation pattern of a selected representation is re-represented in other visual areas. If an observer is in two different perceptual states that only differ in a slight change of perceived contrast, a "global distribution of information" would mean that this difference is reproduced in every brain area that has this information. Alternatively, the information could be represented in only one cortical area and other areas would be able to access it. One is again confronted with the functional mapping problem and one has to account for the "information" available to these areas in terms of different states. Thus all areas

in which the information is reproduced have to fulfil the mapping requirements outlined in chapter 2. Current evidence says, that this information is not globally distributed. A simple counter-example is that a change in contrast does not differentially activate non-visual areas (Boynton et al., 1999). Thus, even in a global workspace individual dimensions of conscious perception are still locally represented.