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THE NEURAL PROCESSES GENERATING VISUAL PHENOMENAL CONSCIOUSNESS: ERP AND NEURONAVIGATED BRAIN STIMULATION STUDIES

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ABSTRACT

One of the greatest conundrums to the contemporary science is the relation between consciousness and brain activity, and one of the specific questions is how neural activity can generate vivid subjective experiences. Studies focusing on visual consciousness have become essential in solving the empirical questions of consciousness. The main aim of this thesis is to clarify the relation between visual consciousness and the neural and electrophysiological processes of the brain. By applying electroencephalography and functional magnetic resonance image-guided transcranial magnetic stimulation (TMS), we investigated the links between conscious perception and attention, the temporal evolution of visual consciousness during stimulus processing, the causal roles of primary visual cortex (V1), visual area 2 (V2) and lateral occipital cortex (LO) in the generation of visual consciousness and also the methodological issues concerning the accuracy of targeting TMS to V1.

The results showed that the first effects of visual consciousness on electrophysiological responses (about 140 ms after the stimulus-onset) appeared earlier than the effects of selective attention, and also in the unattended condition, suggesting that visual consciousness and selective attention are two independent phenomena which have distinct underlying neural mechanisms. In addition, while it is well known that V1 is necessary for visual awareness, the results of the present thesis suggest that also the abutting visual area V2 is a prerequisite for conscious perception. In our studies, the activation in V2 was necessary for the conscious perception of change in contrast for a shorter period of time than in the case of more detailed conscious perception. We also found that TMS in LO suppressed the conscious perception of object shape when TMS was delivered in two distinct time windows, the latter corresponding with the timing of the ERPs related to the conscious perception of coherent object shape. The result supports the view that LO is crucial in conscious perception of object coherency and is likely to be directly involved in the generation of visual consciousness.

Furthermore, we found that visual sensations, or phosphenes, elicited by the TMS of V1 were brighter than identically induced phosphenes arising from V2. These findings demonstrate that V1 contributes more to the generation of the sensation of brightness than does V2. The results also suggest that top-down activation from V2 to V1 is probably associated with phosphene generation.

The results of the methodological study imply that when a commonly used landmark (2 cm above the inion) is used in targeting TMS to V1, the TMS-induced electric field is likely to be highest in dorsal V2. When V1 was targeted according to the individual retinotopic data, the electric field was highest in V1 only in half of the participants. This result suggests that if the objective is to study the role of V1 with TMS methodology, at least functional maps of V1 and V2 should be applied with computational model of the TMS-induced electric field in V1 and V2.

Finally, the results of this thesis imply that different features of attention contribute differently to visual consciousness, and thus, the theoretical model which is built up of the relationship between visual consciousness and attention should acknowledge these differences. Future studies should also explore the possibility that visual consciousness consists of several processing stages, each of which have their distinct underlying neural mechanisms.

TIIVISTELMÄ

Tajunnallisuus ja sen suhde aivojen neuraalisiin tapahtumiin on yksi tieteen suurimmista ratkaisemattomista kysymyksistä. Tyypillisesti tajunnallisuudella viitataan fenomenaaliseen tajuntaan eli yksilön elämykselliseen ja välittömään kokemukseen tietystä sisällöstä. Tajunnallinen näkeminen eli visuaalinen tajunta on noussut keskiöön tajunnan neuraalisten korrelaattien tutkimuksessa. Tarkastelen tässä tutkimuksessa aivokuoren aktivaation ja visuaalisen tajunnan välistä korrelaatio- ja kausaalisuhdetta elektroenkefalografian (EEG), toiminnallisten magneettikuvien avulla ohjatun transkraniaalisen magneettistimulaation (TMS) sekä TMS:n indusoiman sähkökentän mallinnuksen avulla. Erityisesti tavoitteena on tarkentaa näönvaraisen tajunnan ja tarkkaavaisuuden välistä suhdetta, tajunnan ajallista kehittymistä, ensimmäisen näköaivokuoren alueen (alue V1), alueen V2 ja lateraalisen näköaivokuoren (LO-alue) roolia visuaalisessa tajunnassa. Väitöskirja koostuu viidestä osatutkimuksesta.

Tulokset osoittivat, että varhaisimmat visuaalisen tajunnan vaikutukset tapahtumasidonnaisiin herätevasteisiin (ERP) tulivat esiin noin 140 ms ärsykkeen esittämisen jälkeen ja selvästi ennen valikoivan tarkkaavaisuuden vaikutusta sekä riippumatta valikoivan tarkkaavaisuuden vaikutuksesta. Tulos viittaa siihen, että visuaalisen tajunnan ja valikoivan tarkkaavaisuuden taustalla on erilliset neuraaliset prosessit.

Alueen V1 tiedetään olevan välttämätön normaalille näönvaraiselle tajunnalliselle kokemukselle, mutta kolmannen osatutkimuksen tulokset tukevat oletusta, että myös viereinen alue V2 on välttämätön normaalille visuaaliselle tajunnalle. Lisäksi havaittiin, että aktivaatio alueella V2 oli välttämätöntä visuaalisen ärsykkeen yksityiskohtien prosessoinnille pidempään kuin tajunnallisuudelle ärsykkeen läsnäolosta. LO-alueen stimulointi TMS:lla taas ehkäisi tajunnallisen kokemuksen tutusta objektista kahdessa erillisessä aikaikkunassa, joista jälkimmäisen ajoitus korreloi tajuntaan liittyvän tyypillisen ERP-vasteen ajoituksen kanssa. Tutkimustulos tuo tukea näkemykselle jonka mukaan LO-alueen aktivaatio liittyy suoraan niihin prosesseihin, jotka generoivat tajunnallisen havainnon objektista.

Okkipitaalilohkon TMS- ja sähköstimuloinnin tiedetään aiheuttavan subjektiivisia valoaistimuksia eli fosfeeneja. Tutkimuksessa havaittiin, että alueen V1 ja alueen V2 stimuloinnin avulla tuotetut fosfeenit ovat keskenään hyvin samankaltaisia muodon, värin sekä koon osalta, mutta alueen V1 stimuloinnissa tuotetut fosfeenit olivat kaikilla tutkittavilla kirkkaampia kuin alueen V2 stimuloinnilla tuotetut fosfeenit.

Menetelmällisessä tutkimuksessa havaittiin, että vaikka TMS-pulssi oli suunnattu alueelle V1 toiminnallisten magneettikuvien tai kallon muodon mukaan, oli todennäköisempää, että indusoitu sähkökenttä oli ollut voimakkaampi alueen V2 päällä. Toisaalta toisen osatutkimuksen tulokset osoittivat, että joillekin tutkittaville alueen V1 TMS-stimulaatio oli mahdollista, kun erityistä huomiota kiinnitettiin retinotooppisten edustusalueiden valitsemiseen ja hyödynnettiin sähkökentänmallinnusmenetelmää.

Kokonaisuudessaan tämän tutkimuksen tulokset viittaavat siihen, että eri tarkkaavaisuuden muodot vaikuttavat eri tavoin näönvaraiseen tajuntaan, ja näin ollen, teoreettisen mallin visuaalisen tajunnan ja tarkkaavaisuuden välisestä suhteesta tulisi ottaa huomioon nämä erot. Tulevissa tutkimuksissa tulisi myös selvittää mahdollisuutta, jonka mukaan näönvarainen tajunta koostuu useista prosessointi tasosta, joista jokaisella on erilliset hermostolliset perustansa.

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DEFINITIONS AND ABBREVIATIONS

ANOVA	analysis of variance
BOLD	blood oxygenation level dependent
CMVC	constitutive mechanisms of visual consciousness
EEG	electroencephalogram
E-field	electric field
ERP	event-related response
FEF	frontal eye fields
fMRI	functional magnetic resonance imaging
IT	inferior temporal
LGN	lateral geniculate nucleus
LO	lateral occipital cortex
LO1	lateral occipital area 1
LO2	lateral occipital area 2
LP	late positivity
MEG	magnetoencephalography
mffMRI	multifocal functional magnetic resonance imaging
MRI	magnetic resonance imaging
MT	middle temporal area
NBS	Navigated Brain Stimulation
NCC	neural correlates of consciousness
SN	selection negativity
SOA	stimulus onset asynchrony
TMS	transcranial magnetic stimulation
V1	visual area 1, primary visual cortex
V2	visual area 2
V2d	dorsal visual area 2
V3	visual area 3
V3a	visual area 3a
V3d	dorsal visual area 3
hV4	human visual area 4
V5	visual area 5
V7	visual area 7
V8	visual area 8
VAN	visual awareness negativity

LIST OF ORIGINAL PUBLICATIONS

This thesis is based on the following articles:

Study I: Koivisto, M., Revonsuo, A. & Salminen, N. (2005). Independence of visual awareness from attention at early processing stages. *Neuroreport, 16*, 815-822. [a]

Study II: Salminen-Vaparanta, N., Noreika, V., Revonsuo, A., Koivisto, M. & Vanni, S. (2012). Is selective primary visual cortex stimulation achievable with TMS? *Human Brain Mapping, 33,* 652-665.[b]

Study III: Salminen-Vaparanta, N., Koivisto, M., Noreika, V., Vanni, S. & Revonsuo, A. (2012). Neuronavigated transcranial magnetic stimulation suggests that area V2 is necessary for visual awareness. *Neuropsychologia, 50,* 1621-1627.[c]

Study IV: Salminen-Vaparanta, N., Vanni, S., Noreika, V., Valiulis, V., Móró, L. & Revonsuo, A. (2014). Subjective characteristics of TMS-induced phosphenes originating in human V1 or V2. *Cerebral Cortex, 24*, 2751-2760.[d]

Study V: Salminen-Vaparanta, N., Koivisto, M., Henriksson, L., Noreika, V., Quaß, G., & Revonsuo, A. (2014). *Conscious perception of object coherency depends on the contribution of LO during two separate time intervals: evidence from transcranial magnetic stimulation.* Manuscript submitted for publication.

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1. BACKGROUND OF THE THESIS

1.1. Introduction

How is it possible that material tissue, such as the brain, can generate vivid subjective experiences, a phenomenon called consciousness? The relationship between mind and brain is an age-old conundrum that has intrigued both scientists and laypersons for centuries. The question has been approached by using empirical methods at least for the past 100 years. The first methods included observations of patients with neuropsychological disorders, electrical brain stimulation and behavioural experiments. Although there is still debate among scientists about the exact definition of consciousness, the number of studies aiming to identify a particular neural correlate of consciousness has increased considerably since the early 1990's. Nevertheless, despite more than two decades of research (Crick & Koch, 1990), it still remains unsolved which specific neural processes generate the conscious perception, and thus, for current neuroscience, one of the major tasks is to find the neural mechanisms necessary and sufficient for consciousness (e.g., Dehaene & Changeux, 2011; Koch, 2004, 2012; Metzinger, 2000; Revonsuo, 2006).

In this thesis, my main aim is to concentrate on the empirical problem of consciousness: *Which brain regions and mechanisms are necessary and sufficient to generate consciousness?* This question has been called "an easy problem" (Chalmers, 1995). Easy problems are thought to be easy, as they can be answered only by referring to objectively measureable cognitive and neural mechanisms and to the causal interaction between these two mechanisms (Chalmers, 1995), although recent years of consciousness studies have demonstrated that the easy problems are actually not so easy to solve. I will approach this problem by studying the effects of transcranial magnetic stimulation (TMS) in visual cortex on consciousness and by tracking the temporal dimension of consciousness by using electroencephalography (EEG). The more philosophical questions related to consciousness are how any physical entity or process can have or produce any subjective experiences, why do we have phenomenal experiences in this material world and why specific, empirically found, neural mechanisms are associated with consciousness. These questions are called "hard problem" (Chalmers, 1995), because the answers cannot be described by purely referring to cognitive and neural mechanisms. Hence, I will not be dealing with them in this thesis.

1.2. Definitions of consciousness

One of the most famous definitions of consciousness is by Thomas Nagel (1974, p.436) who said that "an organism has conscious mental states if and only if there is something that it is like to be that organism – something it is like for the organism". This definition outlines the most important feature of consciousness: *subjective experience*. Consciousness can refer to the *level of consciousness as a background state* or to *the contents of the consciousness* (Chalmers, 2000; Frith, Perry, & Lumer, 1999). Consciousness as a background state refers to an overall state of consciousness (Chalmers, 2000). The states of consciousness can vary from a nonconscious state to a fully conscious one where one is able to have conscious experiences (e.g., from brain death, dreamless sleep or coma to vivid subjective experiences). Whereas the contents of consciousness, which are the focus of this thesis, refer to the specific subjective experiences in consciousness.

Phenomenal consciousness (or primary consciousness; Edelman, 1992, 2003; Farthing, 1991) can be defined as phenomenal subjective experience of a specific content (Block, 1995, 1996, 2001; Revonsuo, 2006). The content in phenomenal consciousness can be, for example, an unpleasant pain that we feel when we hit our toe on the corner of the wall. These specific subjective experiences are called *qualia* in the philosophical literature (Lewis, 1929). When we are awake, our everyday life consists of a stream of qualia in the phenomenal consciousness[1]. Even during deep sleep, when we have dreams, we may experience vivid subjective experiences, that is, phenomenal consciousness. The most important features of particular contents of phenomenal consciousness are presence, location, duration and intensity (Revonsuo, 2006). According to Revonsuo, presence means that the contents of phenomenal consciousness are directly and immediately available here and now to the experiencing subject, and the contents of phenomenal consciousness are experienced from the perspective of a "world for me". Particular contents of consciousness are spatially related to each other and therefore have a location within the world for me. The particular contents of phenomenal consciousness can also occur with various durations and intensities, so, for example, a toothache can be something that you hardly even notice or it can be so painful that you are not able to think about anything else, except the pain.

Phenomenal consciousness in the visual modality is called *visual consciousness.* Crick and Koch (1990) proposed that empirical consciousness studies should first focus on visual consciousness, as the neurophysiology of vision is well-known, and visual perception is relatively easy to manipulate experimentally. In this thesis, I concentrate only on visual consciousness, not on the other modalities of phenomenal consciousness (auditory, taste, smell, etc.). In the literature, the words "consciousness" and "awareness" are often, but not always, used as synonyms. ”Awareness of” usually refers to the veridical conscious perception of real physical stimuli whereas "phenomenal consciousness" as such does not necessarily require external stimuli (as in dreams, hallucinations, etc.). So, according to this distinction, ”visual consciousness” is a more comprehensive concept than ”visual awareness” is, but both refer to the same phenomenon: subjective experience. In other

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[1] The situations where a discontinuity of consciousness occurs are exceptions. For example, in an epileptic absence seizure, shock or exquisite panic attack the person is awake but acts automatically without having any conscious experiences.

words, when subjective experience is triggered by the physical stimulation of an external object, it can be termed as "aware of" or "conscious perception of" or "consciousness of", but for internally arisen experiences the only appropriate word is "consciousness".

The concept of reflective consciousness (Farthing, 1991; Revonsuo, 2006) is more controversial than the concept of phenomenal consciousness. It has been proposed that the contents of the phenomenal consciousness that are in the focus of attention would transfer into the reflective consciousness. For example, when we are in the hairdresser's having a new haircut and evaluate whether we are satisfied with the new hairstyle or not, our hair, which is first in the content of phenomenal consciousness is after the focusing of attention in the reflective consciousness. When the content emerges in the reflective consciousness, it is available for the processes of evaluation and verbal description. Thus, phenomenal consciousness would be a necessary state for reflective consciousness (Lamme, 2004; Revonsuo, 2006). Working memory, on the other hand, refers to a short-term memory storage system of the brain (Atkinson & Shiffrin, 1968; Baddeley, 1992). Like reflective consciousness, it is limited in its capacity but refers solely to cognitive functions. The concept of reflective consciousness shares also a lot of similarities with the well-known concept of "access consciousness", introduced by Ned Block (1995, 2001, 2005). This concept refers to the state where the contents of phenomenal consciousness are available for the access of, for example, reasoning, memory or reports. Not all scientists agree with these definitions, but for the purposes of the present thesis, *I accept the concepts of phenomenal and reflective consciousness as a starting point*.[2] Importantly, when phenomenal consciousness is studied by using subjective reports as an indication of phenomenal consciousness, the content is also processed in reflective consciousness. Thus, when studied in this manner, phenomenal consciousness can be accessed only indirectly (via reflective consciousness).

1.3. Definitions of attention

Cognitive and neural systems of attention and visual awareness overlap in several manners (for review see Rees & Lavie, 2001), and there is still disagreement of the links between the two phenomena (e.g., de Brigard & Prinz, 2010; Tallon-Baudry, 2012). Visual attention is defined as a selection process where attention selects certain locations and objects from the visual input (Pashler, 1998). The selected information is processed faster and deeper than the input outside of the focus of attention, enabling the content in the focus of attention to access cognitive processes and memory (Egeth & Yantis, 1997; Posner, 1994).

In addition, there are two processes of visual attention that also have distinct neural processes (Zani, Avella, Lilli, & Proverbio, 1999). When a specific location in the visual

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[2] Since the concepts of "access" and "reflective consciousness" are closely linked, I could also select the concept of access consciousness instead of reflective consciousness to refer to a processing stage where the content of phenomenal consciousness is available for the processes of evaluation and verbal report.

field is in the focus of attention, it is called *spatial attention.* When a certain object or visual stimulus feature is in the focus of attention, it is called object attention, or *selective (object) attention* (see Yantis, 2000). In practice, there is constant interplay between these two processes. Here, I will systematically refer to object attention with the term selective attention. Attention can be controlled by the salience of the visual stimulus in a stimulus-driven manner (i.e. *bottom-up attention*) or it can be controlled by a specific goal as a result of the motivations of the observer, so called *top-down attention* (see Behrmann, Geng, & Shomstein, 2004; Yantis, 2000).

1.4. Definitions of neural correlates of consciousness

In empirical consciousness studies, as well as in this thesis, one of the goals is to find the *neural correlates of consciousness (NCC).* Literature includes several definitions for this concept. According to Chalmers (2000, p. 20), "a content NCC is a neural representational system N such that the content of N directly correlates with the content of consciousness". Importantly, this definition includes merely the correlations between consciousness and brain activation. However, also the causal relationship between consciousness and brain processes should be identified. According to Koch (2004, p.16), NCC is "a *minimal* set of neuronal events and mechanisms jointly sufficient for a specific conscious percept" and according to the definition of Ned Block (2005), phenomenal NCC can be defined as the minimal neural basis of the phenomenal content of an experience. Given that this kind of definition goes further than pure correlation, it has been proposed that maybe the term "neural correlates of consciousness" should be replaced by the term "constitutive mechanisms of consciousness" (Revonsuo, 2006). The central question is what kind of neural processes generate *visual consciousness.* Therefore, one of the main objectives of this thesis is to explore *the minimal neural basis sufficient for a specific content of consciousness,* that is, *the constitutive mechanisms of visual consciousness (CMVC).*

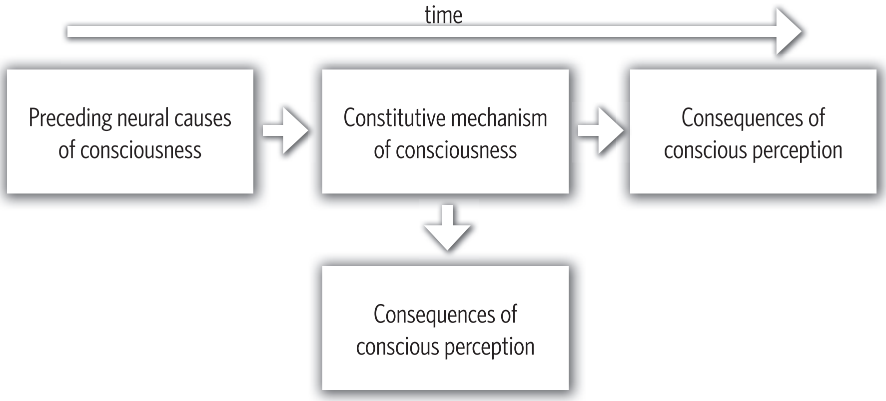

Figure 1. Diagram of the neural processes which are related to consciousness. The diagram is modified from the views of Aru et al. (2012) and Revonsuo (2006).

It is also important to distinguish the processes which are not directly related to consciousness (Miller, 2001). In addition to defining the NCC, Revonsuo (2006) has also identified the preceding causes of NCC, calling these etiological explanations (Fig. 1). These are the processes that precede visual consciousness and are necessary for it. Aru, Bachmann, Singer, and Melloni (2012) also dissociate the processes ensuing from consciousness, calling these processes the consequences of conscious perception. These processes can also take place simultaneously with the conscious experience.

2. REVIEW OF THE LITERATURE

2.1. Visual signal processing

To comprehend the neural basis of visual consciousness, it is crucial to understand how visual perception in general is generated in the visual system. In the human eye, photoreceptors of the retina receive the visual input. The centre of the visual field is coded by the highest density of photoreceptors, which are located in the fovea of the eye. From the eye, the visual input ascends to lateral geniculate nucleus (LGN) via the optic nerve and the optic tract. LGN includes magno-, parvo- and koniocellular layers. The majority of the output from LGN to the cortex goes to the primary visual cortex (or V1, Broadman area 17, striate cortex). Three major pathways leaving from LGN terminate to V1 (for review see Lennie & Movshon, 2005). The pathway from the magnocellular LGN carries information on coarse achromatic contrast and motion. The pathway from the koniocellular LGN (blob pathway) is specialized in carrying information on blue-yellow contrast and the parvocellular (parvo-interblob) pathway is specialized in carrying information on red-green contrast as well as fine details. Some of the visual input does not ascend to visual cortex at all. About 10 % of the axons of the ganglion cells in the retina do not have synapses in the LGN but instead in other nuclei (e.g., superior colliculus). Some of these nuclei have connections to visual cortex. For example, the superior colliculus has direct connections especially to the dorsal stream areas V3 and V5 (Lyon, Nassi, & Callaway, 2010).

In macaque monkeys, over 30 cortical areas have been identified that process visual input (Felleman & Van Essen, 1991). The exact number of visual areas in humans is still unclear. Here, I will cover only the most important areas from the perspective of this thesis. Cortical areas can be set to a hierarchical order according to anatomical connections. At the bottom in the anatomical hierarchy is V1, most of which is located in the interhemispheric fissure and in the calcarine sulcus. In the left hemisphere, V1 receives visual volley from the contralateral visual field, whereas in the right hemisphere it receives visual input from the left visual field. The small field of central or foveal vision is represented in V1 by a large area of neural tissue, allowing high-acuity central vision. Thus, our sight has higher resolution for the contents at the centre of the visual field than for the contents at the periphery. V1 consists of six layers. Axons from the LGN terminate to different layers in V1, most of them to layer IVc. The neurons in V1 are tuned up for specific features of visual input, such as position, ocular dominance, orientation, direction of motion, binocular disparity, spatial frequency or wavelength.

V1 is called striate cortex because the cortical tissue appears striped due to a band of myelinated axons when neural tissue preparations are microscopically observed.

It is surrounded by extrastriate areas (including V2, V3, V4 and V5) which do not histologically have a striped appearance. V2 is the adjacent area of V1, and in primates, V1 and V2 are linked with strong bidirectional connections. The sizes of the receptive fields (regions of visual space where the visual stimulus can elicit neural responses) are larger in the higher visual areas than in V1 (Smith, Singh, Williams, & Greenlee, 2001). Also the complexity of the receptive fields increases with the visual hierarchy. Regarding the role of V2 in visual perception, single-unit recordings in monkeys demonstrate that neurons in V2 are tuned up for the low-level features of stimuli such as size, orientation and colour (e.g., Gegenfurtner, Kiper, & Fenstemaker, 1996; Hubel & Livingstone, 1987), to some basic shapes such as cross and angle (Hegdé & Van Essen, 2000, 2003, 2007), and illusory contours (for review see Seghier & Vuilleumier, 2006; von der Heydt, Peterhans, & Baumgartner, 1984).

Beyond V1 and V2, two different visual processing streams have traditionally been distinguished in humans: the ventral and the dorsal stream (Goodale & Milner, 1992). Roughly speaking, the ventral stream includes the pathway from V1 to V2, then, to hV4 (or V8, Hadjikhani, Liu, Dale, Cavanagh, & Tootell, 1998) and thence to occipitotemporal and inferior temporal (IT) cortex. The dorsal pathway consists of connections from V1 to V5 (or V1-V2-V5) and thence to the parietal cortex. In the dorsal stream, the features of the neurons are relatively similar to those of the magnocellular ones in V1, whereas in the ventral stream, both parvo- and magnocellular neurons are involved. Studies done with non-human primates suggest that most of the cells in V5 are direction selective (Dubner & Zeki, 1971). In the parietal cortex, activation in the specialized regions is associated with abstract representation of space, spatial location of objects and feature binding (Andersen, Essick, & Siegel, 1985; Andersen, Snyder, Bradley, & Xing, 1997; Friedman-Hill, Robertson, & Treisman, 1995; Galletti, Battaglini, & Fattori, 1995; Holmes, 1918; for review see Pollen, 1999).

In the ventral stream, many of the cells in V4 are colour, shape and orientation selective (Hegdé & Van Essen, 2007; Zeki, 1973). Yet, it should be noted that human hV4 is not necessarily homologous to monkeys' V4 (Hadjikhani et al., 1998; see also Brewer, Liu, Wade, & Wandell, 2005; Lueck et al., 1989; Wade, Brewer, Rieger, & Wandell, 2002). Functional imaging studies show that unconscious and conscious object perception is associated with activity in several areas of the ventral visual pathway from V1 to IT cortex (e.g., Chao, Haxby, & Martin, 1999; Henriksson, Karvonen, Salminen-Vaparanta, Railo, & Vanni, 2012; Martin, Wiggs, Ungerleider, & Haxby, 1996; for a review see Grill-Spector, 2003). These areas comprise for example the lateral occipital (LO) area (which can be divided into LO1 and LO2; Larsson & Heeger, 2006), V7, the occipitotemporal sulcus, the inferior temporal gyrus and the anterior and middle fusiform gyrus. The specific features of visual input and the cognitive processes involved in the processing of objects determine exactly which cortical regions are active (e.g., Grill-Spector, Kushnir, Hendler, & Malach, 2000; Martin et al., 1996; Moore & Price, 1999). In particular, the activation in LO increases when visual stimuli contain any coherent object shape (whether familiar or novel), but not when non-objects lack a coherent shape (e.g., Grill-Spector et al., 1998; Grill-Spector, et al., 2000; Kourtzi & Kanwisher, 2000, 2001; Malach et al., 1995; Vanni, Revonsuo, Saarinen, & Hari, 1996).

The information concerning functional connectivity between cortical areas is primarily based on studies carried out with non-human primates. These studies have shown that cortical afferent processing consists of feedforward, feedback and horizontal processing streams. Corticocortical feedforward projection starts typically from supragranual layers and ascends to granual layer, whereas feedback projection projects primarily from infragranual and supragranual layers and terminates in the supragranual layers (e.g. Maunsell & Van Essen, 1983; Felleman & Van Essen, 1991; Stone, Dreher, & Leventhal, 1979). In the feedforward processing, the visual volley from V1 converges to higher cortical areas via neurons that code visual input from the retinotopically equivalent region relative to that of V1 (Bullier et al., 1996; Salin & Bullier, 1995). The majority of the connections from V1 terminate in other extrastriate areas via V2, but some of the connections from V1 reach higher visual areas (e.g., V3, V4 and V5) without V2 as a mediator (e.g., Burkhalter, Felleman, Newsome, & Van Essen, 1986; Felleman, Burkhalter, & Van Essen, 1997; Felleman & Van Essen, 1991; Maunsell & Van Essen, 1983; Van Essen, Newsome, Maunsell, & Bixby, 1986). In the feedback processing, visual input from the higher cortical regions is projected to the visual areas which are located lower in the anatomical hierarchy. Direct descending connections to V1 include projections from V3, V4 (Rockland & van Hoesen, 1994), V5 (Shipp & Zeki, 1989; Ungerleider & Desimone, 1986) and IT cortex (Felleman & Van Essen, 1991; Kennedy & Bullier, 1985; Rockland & Drash, 1996; Rockland & van Hoesen, 1994). Based on the studies done with non-human primates, it has been suggested that the role of the feedback projection is modulatory rather than driving in the modulation of the responses in the visual areas that are lower in the anatomical hierarchy (Macknik & Martinez-Conde, 2009). Both feedforward and feedback connections between the visual areas can be extremely fast (less than 5 ms) at least in non-human primates (Girard, Hupé, & Bullier, 2001). Horizontal connections within each visual area are slow and, in V1, the region corresponding to these connections can reach only up to 0.6 ° in the visual field (for review see Bullier, 2001).

Regarding the timing of visual processing in humans, the first visual afferent ascends to V1 approximately 40–60 ms after the stimulus-onset (e.g., Clark, Fan, & Hillyard, 1995; Vanni et al., 2004; Wilson, Babb, Halgren, & Crandall, 1983). Studies with non-human primates show that activation is spread to V2 only a few milliseconds later (Nowak, Munk, Girard, & Bullier, 1995; Raiguel, Lagae, Gulyàs, & Orban, 1989) [3]. Some of the cells in V1 are actually activated even later than the cells in V2 (Nowak et al., 1995); the

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[3] Due to the bigger size of the human brain compared to that of monkeys, the latencies in humans are slightly longer than in monkeys.

latency difference between the ascending magnocellular and parvocellular pathway is longer than the latency difference between V1 and V2 (Bullier & Nowak, 1995) denoting that signals of the magnocellular pathway reach extrastriate areas and return to V1 before the volley from the parvocellular pathway ascends to V1 (Bullier, Hupé, James, & Girard, 1996). Similarly, also several other regions are activated within a few milliseconds after the activation in V1 (e.g., V5 and frontal eye fields [FEF], Hupé, James, Girard, Lomber, et al., 2001; for review see Bullier, 2001). For example, the feedback process from MT to V1 is already activated with the earliest component of the V1 response (Hupé, James, Girard, Lomber, et al., 2001). Thus, the visual signal processing in the cortex is parallel rather than serial (Bullier & Nowak, 1995), which means that visual input is processed simultaneously in various extrastriate areas and in V1 (see also Raiguel et al., 1989). In humans, event-related EEG responses to coherent images start to diverge from scrambled ones only 100 ms post-stimulus (Schendan & Lucia, 2010), and neural responses to discrimination between animal and non-animal pictures can be dissociated from each other around 150 ms after the stimulus-onset (Thorpe, Fize, & Marlot, 1996). These results imply that our brain can discriminate visual objects very rapidly – within less than 200 ms.

2.2. The neural correlates of visual consciousness

The basic idea behind the brain imaging experiments searching for the CMVC is that a participant carries out a behavioural task where visual stimuli and experimental procedure are kept otherwise exactly the same, but the only thing that changes between the two conditions is consciousness of the visual stimulus (Frith et al., 1999). While in imaging studies, the objective is to compare the brain activity between these two conditions, in brain stimulation studies, neural activation is interfered with while a participant is carrying out a visual detection or discrimination task.

In a typical experimental setup, subjective ratings are used to determine whether a participant is conscious of the stimulus or not. In addition, behavioural forced-choice responses have been used as an indicator for conscious perception. Nevertheless, forced-choice responses do not invariably covary with conscious percepts as a forced-choice response has to be given despite the fact that the participant might be completely uncertain about her/his subjective perception or did not see the visual stimulus at all. Furthermore, the stimulus may remain below the subjective perceptual threshold, although it is above the objectively measured threshold, as demonstrated, for example, with unconscious priming procedure (e.g., Naccache & Dehaene, 2001; for review see Henson, 2003). In the following literature review, I concentrate on the most relevant findings which were made before the present studies were conducted. Recent findings will be covered in Discussion.

2.2.1. Localizing neural mechanisms of consciousness: evidence from single cell recordings and functional imaging studies

In a study by Sheinberg & Logothetis (1997), monkeys were given a binocular rivalry task while the neural responses were recorded in several visual areas and correlated with the change of dominance between the two rivalrous stimuli. In binocular rivalry, one visual stimulus is presented to one eye and a different visual stimulus to the other eye at the same time. Sheinberg and Logothetis found that the activity levels of most cells in the inferotemporal cortex are correlated with the changes of visual awareness. In addition, Logothetis (1998) demonstrated that binocular rivalry in monkeys resulted in firing of cells in the ventral stream whose activation correlated with a state of visual consciousness in such a manner that the activation level was changed as a function of state of consciousness in 15% of the measured cells in V1 and V2 and in 90% of the measured cells in inferotemporal cortex. Quiroga, Mukamel, Isham, Malach, and Fried (2008) found consciousness-related single-neuron activity in human middle temporal area (MT). Thus, the results from single cell recordings suggest that cortical areas along the ventral stream travelling from V1 to temporal lobe play an important role in generating specific conscious contents.

In humans, studies using magnetoencephalography (MEG) have demonstrated that activation in the occipitotemporal regions, particularly in the lateral occipital complex (Vanni et al., 1996) and the right posterior infero-temporal region (Liu, Paradis, Yahia-Cherif, & Tallon-Baudry, 2012), is increased for consciously seen stimuli. Also functional magnetic resonance imaging (fMRI) response in LO is increased for the recognized objects (Grill-Spector et al., 2000) and letters (Kleinschmidt, Büchel, Zeki, & Frackowiak, 1998). Further, activation in an area 1 cm anterior to the fusiform face area shows a strong correlation to the object recognition (Bar et al., 2001). Tong, Nakayama, Vaughn and Kanwisher (1998) applied the binocular rivalry paradigm by presenting faces to one visual field and houses to another, and they showed that the average fMRI response increased in the cortical region whose preferred stimulus emerged in visual consciousness. Along similar lines, patients with schizophrenia showed activation in the modality-specific cortical areas while they were having hallucinations (Silbersweig et al., 1995). Patients suffering from Charles Bonnet syndrome are mentally healthy but have vivid hallucinations typically due to deafferentation. Similar to the patients with schizophrenia, these patients' extrastriate areas are activated during the visual hallucinations (Ffytche et al., 1998) in a manner that the contents of visual consciousness reflect the specialization of the specific visual area. Altogether, these results demonstrate that the magnitude of the neural response in the cortical regions that are specialized in certain stimulus features is correlated with the content entering consciousness. In contrast to the findings of activation in the specialized areas, some fMRI studies show that also V1, and specific regions in the parietal and prefrontal cortex respond to conscious perceptions (Dehaene et al., 2001; Haynes, Driver, & Rees, 2005; for review see Dehaene & Changeux, 2011). Unmasked (consciously perceived) words have been shown to induce activation in the frontal and parietal areas compared to responses to masked words (Dehaene et al., 2001). Polonsky, Blake, Braun, and Heeger (2000) applied the binocular rivalry paradigm by presenting low and high contrast stimuli and found that blood oxygenation levels (BOLD signal) of both V1 and other extrastriate areas fluctuated according to the perception in a way that activity in V1 was increased when participants reported seeing a high contrast pattern whereas it decreased when participants reported seeing a low contrast pattern. Also changes in surface luminance are associated with changes in fMRI responses in V1 and V2/V3, and reflected also as changes in perceived brightness (Haynes, Lotto, & Rees, 2004).

An evident problem in interpreting fMRI studies is the limited temporal resolution of fMRI. EEG studies have indicated that the generation of visual consciousness probably includes several fast and locally separated neural processes that occur within the first 450 ms from stimulus-onset (e.g., Wilenius-Emet, Revonsuo, & Ojanen, 2004; for review see Koivisto & Revonsuo, 2010; Railo, Koivisto, & Revonsuo, 2011). In fMRI studies, the temporal resolution is at best one or two seconds which is clearly not sufficient to get detailed information about the temporal evolution of how brain activity dynamically develops when visual stimuli are rapidly processed in the visual cortex and consciously perceived.

2.2.2. Timing of awareness as studied with EEG and single cell recordings

The timing of visual awareness has been studied with various methods by investigating the timing of visual consciousness related brain responses. The most commonly used method is EEG, and other methods used include MEG, intracranial recordings and single cell recordings. By measuring changes in the BOLD signal, fMRI provides excellent spatial resolution for tracking consciousness related brain activity, whereas in EEG and MEG the electrical activity is measured more directly, and thus, these methods provide a temporal resolution of only one millisecond. MEG, single cell recordings and EEG allow (similar to fMRI) measurements that reflect the *correlation* between neural activity and visual awareness.

The effect of visual consciousness on the event-related potentials (ERP) has been studied by presenting visual stimuli close to the subjective threshold, and by comparing the ERPs to the consciously seen stimuli with those to the unseen stimuli. The most common finding from these studies is a negative amplitude shift which peaks around 260 ms after the stimulus-onset (Wilenius-Emet et al., 2004; for reviews, see Koivisto & Revonsuo, 2010; Railo et al., 2011), that is, *visual awareness negativity* (*VAN*, Ojanen, Revonsuo, & Sams, 2003). VAN to conscious perception of high contrast stimuli peaks around 260 ms after stimulus-onset and around 400 ms to low contrast stimuli (Wilenius & Revonsuo, 2007), and it is observed regardless of whether familiar object images or meaningless scrambled images are presented (Wilenius-Emet et al., 2004). However, some other studies have shown that visual awareness-related activity would be observed only as enhanced positivity in the P3 time window (Salti, Bar-Haim, & Lamy, 2012; for a review see Dehaene & Changeux, 2011). Interestingly, the brain activity that predicts whether a consequent stimulus will reach consciousness or not, can be observed in the oscillations in MEG already before the onset of the visual stimulus presentation. The phase and power of alpha (Mathewson, Gratton, Fabiani, Beck, & Ro, 2009; Romei, Brodbeck et al., 2008; Romei, Rihs, Brodbeck, & Thut, 2008) or gamma activity (Wyart & Tallon-Baudry, 2009) can predict whether a visual stimulus or a phosphene is seen or not. Increased phase and power of alpha is related to decreases in detection (Mathewson et al., 2009; Wyart & Tallon-Baudry, 2009; see also Hanslmayr et al., 2007), whereas increase in prestimulus gamma-band activity is related to increased detectability of the stimulus (Aru, & Bachmann, 2009; Wyart & Tallon-Baudry, 2009, see also Hanslmayr et al., 2007). Melloni et al. (2007) reported that the difference between stimulus visibility and invisibility can be observed within 100 ms after the stimulus-onset as increased mean phase synchrony at 50–57 Hz for visible stimuli. Further, gamma-band activity (in addition to the visual awareness related prestimulus phase activity) was shown to be related to the detection of a stimulus and the activation was localized to LO (Wyart & Tallon-Baudry, 2009). Quiroga et al. (2008) studied specifically the responses of neurons in MT and reported consciousness-related activation in a large time window from 300 ms onwards. The challenge of interpreting the results from EEG, MEG and single cell recordings is that visual consciousness is preceded and followed by processes that may alter depending on the conscious state (see Fig.1) and, thus, it is difficult to show which of the effects actually are part of the CMVC.

In addition to electrophysiological studies, the timing of visual awareness has also been studied with purely behavioural methods, for example by using the visual masking paradigm (e.g., Enns & Di Lollo, 2000). In several studies, it has been demonstrated that when two visual stimuli are presented one after another with brief durations, if the latency between the two stimuli is short, the perception of the first stimulus is impaired. The masking effect disappears when the latency between the two stimuli is lengthened up to 100 ms. These results give clear support to the idea that we do not become aware of the visual stimulus until about 100 ms after the stimulus-onset.

2.2.3. Deficits in consciousness: evidence from studies of patients with brain injuries

Intact primary visual cortex is required for normal conscious visual perception: removal of V1 results in blindness in the whole visual field in humans, and injury to a part of the V1 causes scotomas, that is, loss of vision in a part of the visual field (Holmes, 1918; Tong, 2003)[4]. Although V1 and the pathway to it are necessary for externally induced conscious visual percepts, there is evidence that they are not necessary for visual consciousness itself: Patients who have a lesion in V1 are still able to have dreams with vivid visual

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[4] In addition, a lesion in any of the regions in the visual afferent pathway from the eye to the V1 will cause perceptual blindness in humans.

contents (Solms, 1997). Furthermore, patients who have damaged optic radiation can still have visual hallucinations, and thus, visual phenomenology (Anderson & Rizzo, 1994), suggesting that visual consciousness is possible without direct projections from the retina to the LGN. Similarly, sensations of flash of light, or phosphenes, can be induced in patients whose neural connections from LGN to V1 are destroyed (Brindley & Lewin, 1968; Dobelle & Miladejovsky, 1974).

There are only few reports of patients suffering from injuries that have specifically covered V2, V3, or V3A. Lesions roughly of V2/V3 have been reported to result in visual field defects (Horton & Hoyt, 1991; Slotnick & Moo, 2003; see also McFadzean & Hadley, 1997)[5], but isolated lesions within V3 have not been reported to result in scotomas (see Pollen, 1999). Lesions of both hV4 areas can induce complete inability to see colours, a phenomenon called achromatopsia (Damasio, Yamada, Damasio, Corbett, & McKee, 1980; Meadows, 1974; Sacks, 1995; Zeki, 1990). These patients are unable to see colours in their dreams or imagine how colours would look like. If the lesion is only in one hemisphere, then the colours have disappeared only from the contralateral hemifield (Zeki, 1992). Importantly, conscious achromatic contrast discrimination remains intact after the damaged V4 areas (Heywood & Cowey, 1987). Patients with lesions in V5 may lose their ability to see visual motion, a phenomenon known as akinetopsia (Zihl, von Cramon, & Mai, 1983). It is noteworthy that although the number of reported lesions in the occipitotemporal cortex is high, the aforementioned deficits in consciousness are relatively infrequently reported. Yet, the results are in agreement with a view that hV4 is necessary for the visual consciousness of colours and V5 is necessary for the consciousness of motion.

Bilateral lesions in the mesial occipitotemporal region may result in an inability to recognize faces, known as prosopagnosia (Damasio, Damasio, & Van Hoesen, 1982), and injury in the LO may cause inability to consciously see and identify objects, a deficit called visual form agnosia or apperceptive agnosia (e.g., Damasio et al., 1982; Goodale, Milner, Jakobson, & Carey, 1991; Heider, 2000; James, Culham, Humphrey, Milner, & Goodale, 2003; Konen, Behrmann, Nishimura, & Kastner, 2011; McIntosh, Dijkerman, Mon-Williams, & Milner, 2004; Rubens & Benson, 1971). Although these patients are unable to consciously discriminate specific shapes or the orientation of an object, they can consciously see variations in contrast, colours and motion. Taken together, these results demonstrate that the activation in the areas that are specialized in specific visual features is necessary for the corresponding visual features to appear in the phenomenal consciousness.

In addition to the areas in the ventral stream, also injury to the parietal lobe (or the temporo parietal junction, Damasio, 1999; Friedrich, Egly, Rafal, & Beck, 1998) has been

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[5] Merigan, Nealey and Maunsell (1993) demonstrated with monkeys that complex orientation discrimination is impaired due to injury in V2, but contrast discrimination in simpler tasks remains intact after lesion in V2.

shown to influence visual consciousness. Patients suffering from a unilateral visuospatial neglect (or visuospatial agnosia [Heilman, Watson, & Valenstein, 2012]) syndrome have difficulties to consciously see objects in one hemifield due to an injury in the parietal cortex. Typically a lesion in the right parietal cortex impairs visuo-spatial attention to the left perceptual field, and the patients may seem to be completely unaware of the stimuli in their left perceptual field (Heilman et al., 2012; Kerkhoff, 2001; Vallar, 1998). However, lesions in the left parietal cortex induce typically milder symptoms than lesions in the right hemisphere and also higher inter-individual variations: neglect can be in either ipsi- or contralateral hemifield (Weintraub, Daffner, Ahern, Price, & Mesulam, 1996). It is still unclear which cortical region(s) exactly is (/are) necessary and sufficient to produce neglect (for reviews see e.g., Behrman, et al., 2004; Pollen, 2011). In Balint's syndrome patients are able to observe only one object at a time, a phenomenon that is called simultanagnosia (Bálint, 1995; Friedman-Hill, Robertson, Desimone, & Ungerleider, 2003; Jackson, Swainson, Mort, Husain, & Jackson, 2009). These patients have injuries in the parietal lobes of both hemispheres. Patients with extinction have difficulties to perceive stimuli in the contralesional hemifield when presented simultaneously with a stimulus in the ipsilesional hemifield. Rees et al. (2000) demonstrated that patients with the extinction of the left visual field still have activation in the ventral stream when a stimulus is presented to the left visual field although they are unconscious of this stimulus. Vuilleumier et al. (2001) showed with neglect and extinction patients that the N1 potential is evoked for face stimuli presented in the neglected visual field. The stimuli in the neglected visual field induce also activation in the contralateral V1 and IT cortex. Injury to the (right) parietal cortex may also result in the disappearance of dreams in all sensory modalities (Solms, 1997). It has been proposed that visuospatial neglect could be explained by an attentional deficit, as these patients have intact primary sensory pathways and intact ventral visual streams (Cohen, Romero, Servan-Schreiber, & Farah, 1994; Heilman et al., 2012). Another possibility is that the (inferior) parietal lobe is involved in spatiality, a process considered to be necessary for phenomenal consciousness (Clark, 2000; Revonsuo, 2006), or that neglect is due both to the competition of attention between the two hemifields and to spatiality.

There are a few reports that damage in prefrontal and frontal cortical areas may also cause unilateral visuospatial neglect (Damasio, Damasio, & Chui, 1980; Heilman & Valenstein, 1972; Maeshima, Funahashi, Ogura, Itakura, & Komai, 1994), and that patients with damage to the anterior or bilateral posterior cingulate cortex may have disturbances in consciousness (Damasio, 1999). Yet, compared to the reports of parietal lesion induced neglect, these reports are rare[6]. All in all, data from neuropsychological patients suggests that V1 is the only cortical area whose removal clearly induces perceptual blindness.

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[6] There are also several other brain regions whose removal has been shown to induce unilateral neglect, e.g., mesencephalic reticular formation (Watson, Heilman, Miller, & King, 1974). However, in this thesis, my main goal is to focus on the cortical basis of visual consciousness, and thus, the role of subcortical regions is not discussed further.

The roles of V2, V3, parietal cortex and prefrontal cortex in visual consciousness are less clear, but there is some evidence that also these areas are necessary for perceptual visual consciousness. In addition, specific regions in the ventral and dorsal stream are necessary for the specific contents or features of visual consciousness. Nevertheless, in the cortical lesions of neurological patients, the damaged brain area typically covers also the neural tissue of the adjacent areas, axonal tracts to other areas or does not cover the whole area. Thus, in most cases it is probable that the lesion area is not only limited to the region of interest preventing further conclusions about the role of particular areas in conscious perception.

2.2.4. Evidence from brain stimulation studies

While functional imaging studies or electromagnetic sensing of brain activity cannot reveal causal relationships between brain activation and consciousness, studies where the normal neuronal processing is in some way experimentally modulated or disturbed can indicate whether a specific area or connections from that area to other areas is/are necessary for visual consciousness or for a given cognitive process. By using TMS, the effects of such interference on behavioural responses and cognitive functions can be investigated. Thus, it allows one to investigate the *causal relationship* between neural activity and consciousness. In TMS, the TMS coil containing copper wires is held against the head. A rapidly changing magnetic field is induced under the coil, which in turn induces electrical current in the brain, thus causing a depolarisation of neurons. It is likely that TMS influences directly cortical columns instead of horizontal axons (Fox et al., 2004). TMS is nowadays a widely used method in studies on the neural mechanisms underlying visual perception and consciousness (e.g., Amassian et al., 1989; Corthout, Uttl, Ziemann, Cowey, & Hallett, 1999; Corthout, Uttl, Walsh, Hallett, & Cowey, 1999; Epstein & Zangaladze, 1996; Kastner, Demmer, & Ziemann, 1998; Pascual-Leone & Walsh, 2001; Paulus, Korinth, Wischer, & Tergau, 1999; for reviews see Cowey, 2005; Kammer, 2007).

Phosphene studies

TMS stimulation of visual areas located early in the anatomical hierarchy, V5 or parietal cortex can induce fleeting subjective visual sensations or phosphenes (e.g., Bestmann, Ruff, Blakemore, Driver, & Thilo, 2007; Deblieck, Thompson, Iacoboni, & Wu, 2008; Fernandez et al., 2002; Kammer, Puls, Erb, & Grodd, 2005; Marg & Rudiak, 1994; Meyer, Diehl, Steinmetz, Britton, & Benecke, 1991; Ray, Meador, Epstein, Loring, & Day, 1998; Stewart, Walsh, & Rothwell, 2001). Also stimulation via intracortical electrodes located in the V1 (Brindley & Lewin, 1968; Dobelle & Mladejovsky, 1974; see also Schmidt et al. 1996) and other regions in the occipital and temporal lobes of epilepsy patients induces phosphenes (Lee, Hong, Seo, Tae, & Hong, 2000; Murphey, Maunsell, Beauchamp, & Yoshor, 2009). Phosphene thresholds are lower when spatial attention is directed towards the visual field location whose cortical representation is stimulated (Bestmann et al., 2007). Stimulation thresholds to produce phosphenes vary between different individuals, as well as the subjective characteristics of phosphenes, but the size of the phosphenes correlates positively with stimulation intensity (e.g., Kammer, Puls, Erb, et al., 2005). Typically, white or grey phosphenes are reported, but they can also contain colours and have various shapes (Marg & Rudiak, 1994).

By inducing sensations, one can study the role of the specific area, or connections from that specific area to other brain areas in the emergence of consciousness. The results concerning differences in the characteristics of phosphenes induced by striate or extrastriate cortex stimulation are contradictory[7]. Murphey et al. (2009) mapped V1, V2, V3 and several other regions in the visual cortex with fMRI to target the intracranial electrical stimulation to these areas. Participants' task was to report the size, location, colour, and complexity of the phosphenes. Murphey et al. reported that the characteristics of the phosphenes did not vary between the different cortical areas. Kammer, Puls, Erb, et al. (2005) guided TMS stimulation with the help of individually defined maps of V1, V2 and V3 and compared the elicited phosphenes between these areas. Participants were instructed to draw a contour of phosphene on the screen. However, the TMS-induced electric field (E-field) was not modelled, so it was not entirely clear in which of the areas the induced E-field was the highest. In line with the results of Murphey et al., Kammer, Puls, Erb, et al. reported that the phosphenes between the areas were similar. An opposite finding was made by Lee and colleagues (2000), who demonstrated that the phosphenes which were elicited by cortical electrodes placed nearby the calcarine sulcus were described more bloblike than the phosphenes which were induced further away from the calcarine sulcus. The latter ones were described as more complex. However, Lee and colleagues did not use functional imaging to map the areas in the visual cortex, so it remained open which of the areas exactly elicited more complex phosphenes. Consequently, the subjective characteristics of phosphenes between the areas located early in the anatomical hierarchy have never been compared within neurologically intact humans by using a method that would reliably target V1 and V2.

Suppression studies

Magnetic stimulation at stronger stimulator output intensities than what is required to induce phosphenes impairs visual detection and may even produce a momentary scotoma (e.g., Abrahamyan, Clifford, Arabzadeh, & Harris, 2011; Amassian et al., 1989; Corthout, Hallett, & Cowey, 2002, 2003; Corthout, Uttl, Juan, Hallett, & Cowey, 2000; Corthout, Uttl, Walsh, et al., 1999; Corthout, Uttl, Ziemann et al., 1999; Epstein & Zangaladze, 1996; Kammer, 1999; Kammer, Puls, Erb, et al. 2005; Kammer, Puls, Strasburger, Hill, & Wichmann, 2005; Kastner et al., 1998; Koivisto, Railo, Revonsuo, Vanni, & Salminen-

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[7] The exception is the motion area V5/MT, the stimulation of which typically produces large, moving phosphenes (Pascual-Leone & Walsh, 2001; Stewart, Battelli, Walsh, & Cowey, 1999).

Vaparanta, 2011; Miller, Fendrich, Eliassen, Demirel, & Gazzaniga, 1996; Paulus et al., 1999; Ro, Breitmeyer, Burton, Singhal, & Lane, 2003; Thielscher, Reichenbach, Uğurbil, & Uludağ, 2010). When two or more pulses are delivered in quick succession, lower stimulation intensity is required than when only a single pulse is induced. By interrupting the neural processes with TMS and varying the delay between the visual stimulus and the TMS pulse, one can track the exact point in time (if there is one) when a specific area is required in the process of visual consciousness. Typically, stimulation of visual areas which are located early in the anatomical hierarchy induces impairment in visual perception when the TMS pulse is delivered 80-120 ms after the onset of the visual stimulus (Amassian et al., 1989).

By varying the intensity of magnetic stimulation one can affect the level of performance in stimulus detection tasks. Kammer, Puls, Strasburger, et al. (2005) demonstrated that by increasing the TMS intensity the onset of TMS-induced impairment in visual perception became faster. Abrahamyan et al. (2011) showed that the E-field strength under the phosphene threshold facilitated visual detection whereas the E-field strength at the phosphene threshold had no effect on performance. In addition, Thielscher et al. (2010) demonstrated that when the TMS stimulator output intensity is cut down by about 15% from the level required to induce visual suppression, the proportion of correct responses rises from the chance level to 100% correct. These results show that visual suppression diminishes fast when the stimulator output intensity is reduced, and that TMS at the phosphene threshold intensity is not sufficient to induce impairment in visual detection. Therefore, if the aim is to produce impairment in visual detection by stimulating a specific visual area, the stimulation intensity should be at the phosphene threshold intensity or below in the adjacent non-targeted areas.

The neural processes and the visual areas underlying the generation of TMS-induced visual field defects are not well-understood. V1, extrastriate cortex and subcortical structures have all been proposed to be involved in TMS-induced effects on visual perception (Kammer, Puls, Erb, et al., 2005). Kastner and colleagues (1998) presented that V1 and extrastriate areas contribute differently to the generation of visual scotomas, in a manner that the visual field deficits within 1–3° are induced by magnetic stimulation of V1, whereas visual field deficits slightly further in the periphery (within 4–9°) are due to V2/V3-stimulation. Some researchers have suggested that scotomas are produced purely by V2/V3 stimulation (e.g., Potts et al., 1998; Thielscher et al., 2010). Thielscher and colleagues (2010) modelled the TMS-induced E-field with a spherical model in the occipital lobe combined with functional images of V1, dorsal V2 (V2d) and dorsal V3 (V3d) while participants were performing a detection task and found that the TMS-induced E-field was highest in V2d or V3d. However, some earlier studies suggested that phosphenes and scotomas originate in V1 (Juan & Walsh, 2003; Meyer et al., 1991; Pascual-Leone & Walsh, 2001).

At the time when the studies of this thesis were conducted, published studies concerning the effect of TMS on visual consciousness (although not on visual perception as such) were rare. Among others, studies using forced-choice responses as an indicator of consciousness have shown that when TMS is targeted to V2, discrimination of orientation is impaired (Thielscher et al., 2010) and that TMS in V1/V2 disrupts the recognition of animals in two distinct time windows: at 100 ms and at 220 ms after the stimulus-onset (Camprodon, Zohary, Brodbeck, & Pascual-Leone, 2010). The latter result suggests that V1/V2 contributes to visual perception also at the later latencies – at the latencies whose timing roughly overlaps with the electrophysiological responses for visual awareness (Koivisto & Revonsuo, 2010; Railo et al., 2011). The studies using subjective ratings as an indicator of visual consciousness have shown, for example, that when a moving phosphene is produced with TMS to V5 (Pascual-Leone & Walsh, 2001) or when a real motion is viewed (Koivisto, Mäntylä, & Silvanto, 2010; Silvanto, Lavie, & Walsh, 2005), the subjective experience of the movement can be suppressed by delivering TMS to V1 shortly after delivering TMS to V5. In our earlier study, TMS in LO impaired conscious perception of natural scenes at 150 ms after visual stimulus-onset and TMS in V1/V2 interfered with conscious perception at 180 ms after visual stimulus-onset (Koivisto, Railo, Revonsuo, et al., 2011). However, we did not investigate the effect of TMS at later latencies (> 200 ms), which might correspond more closely with the peak latency of the typical awareness-related activity in electrophysiological and MEG studies (e.g., Koivisto, Revonsuo, & Lehtonen, 2006; Liu et al., 2012; Quiroga et al., 2008; Vanni et al., 1996; Wilenius & Revonsuo, 2007; Wilenius-Emet et al., 2004).

Targeting V1 with TMS methodology

V1 is probably the most widely targeted visual area with TMS. In targeting V1 with TMS, at least three different stimulation procedures have been applied. There has been a general assumption in TMS studies that when the stimulation site is approximately from 1 to 3 cm above inion (and sometimes also 1–2 cm lateral from the midline), the E-field is the strongest in V1, although extrastriate areas might also be affected. Thus, the conventional method is to target the magnetic pulse few centimetres above the inion or according to the international 10/20 system (e.g., electrode site Oz [Jasper, 1958]). This method, however, assumes that the location of the underlying brain structures corresponds with the shape of the scull similarly with different individuals despite the fact that there is inter-individual variation between the location of the calcarine fissure and the shape of the posterior part of the scull (Steinmetz, Fürst, & Meyer, 1989). Also the size of V1 on the surface of the brain varies notably across individuals (Amunts, Malikovic, Mohlberg, Schormann, & Zilles, 2000). In addition to the anatomical landmark method, also the mapping (or hunting) method has been used to target V1. In the mapping method, pulses are delivered to various sites in the posterior part of the head (above the inion) to find an optimal location for producing phosphenes or visual field deficit (e.g., Boroojerdi, Prager, Muellbacher, & Cohen, 2000; Rauschecker, Bestmann, Walsh, & Thilo, 2004; Ro et al., 2003). The selected site for TMS is either the site which produces phosphenes in the optimal visual field location or the one which produces the most vivid phosphenes. This method ignores the fact that stimulation of several other areas in the visual cortex besides V1 can elicit phosphenes (Murphey et al., 2009). A relatively rarely used method is to allocate the coil over the target location according to the individual cortical anatomy by applying MRI images and a neuronavigated system. However, given that only a small portion of V1 is located on the outermost surface of the cortex (Amunts et al., 2000; Stensaas, Eddington, & Dobelle, 1974), the MRI-guided method is also insufficient for determining whether the E-field actually is the strongest in V1, or not.

As mentioned above, Thielscher and colleagues (2010) searched for the occipital site with the strongest visual field deficit by using a hunting method, and the results suggested that the E-field was actually strongest in V2d instead of V1. Changing the coil orientation did not shift the site where stimulation induced maximal drop in correct responses (Thielscher et al., 2010). In their study, the peak of the visual suppression occurred later when the E-field strength was highest in V3d than when the E-field strength was highest in V2d. Thus, their results demonstrate that a sufficient accuracy can be achieved in targeting the TMS pulse to the V2d or V3d at least in a visual detection task when a visual stimulus is presented in a specific location in the visual field. Furthermore, by comparing different locations of the figure-of-eight coil on the scalp with the visual suppression in the specific visual field location, Thielscher et al. showed that visual performance can increase from chance level to maximum performance by changing the coil position only with 7 mm. Nevertheless, they also reported that they did not find a coil position that would have induced a higher E-field in V1 than in the V2.[8]

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[8] In addition to considering the accuracy of stimulation in interpreting the results of TMS studies, one should also take into account the possible remote effects of TMS. Concurrent TMS-fMRI studies (for a review see Bestmann, Ruff, Driver, & Blankenburg, 2008) have demonstrated that delivering TMS pulses to a specific area may result in changes in the regional blood flow of another brain region. For example, TMS in FEF results in BOLD response changes in the visual cortex (V1-V4; Ruff et al., 2006). Another study showed that TMS in the angular gyrus induces activity in extrastriate areas (Heinen et al., 2011). It is worth noting that these results do not show that the behavioural effect was due to the remote effect. What these results demonstrate instead is that there was a specific behavioural effect due to the TMS and that after the TMS stimulation there were some changes in the regional blood flow in a specific remote brain region. The behavioural effect may be related to the local effects within the area under the TMS coil or, alternatively, to the effects within the remote area, or to the interaction between these two areas. Nevertheless, these results demonstrate that the behavioural effects induced by TMS of a specific brain area might actually be due to the influence of TMS on some other area instead. Thus, this should always be taken into consideration when interpreting the results of the TMS studies. Importantly, the role of the remote effects can be tested by applying single pulse TMS to both areas (to the TMS-targeted area and to the area where fMRI is showing the remote effects). In addition, it is worth noting that the effects of TMS are not pathway- or channel-specific (Breitmeyer, Ro, & Ogmen, 2004).

2.3. The neural correlates of attention

As aforementioned, cognitive and neural systems of attention and visual consciousness share several similarities (for review see Rees & Lavie, 2001), and the links between these two phenomena are still unclear (Tallon-Baudry, 2012). Thus, one of the purposes of this thesis is to elucidate this relationship. To that end, the previous studies concerning the neural basis of visual attention are briefly reviewed.

2.3.1. Localizing neural mechanisms of attention: evidence from functional imaging studies and single cell recordings

Studies with neuropsychological patients and functional imaging studies have revealed crucial information about the neural basis of attention. Activation of dorsolateral prefrontal cortex, posterior parietal cortex, anterior cingulate gyrus and the pulvinar nucleus of thalamus have been shown to be associated with visual attention (e.g., Kastner & Ungerleider, 2000; Mesulam, 1990; Nobre et al., 1997; Rossi, Pessoa, Desimone, & Ungerleider, 2009; Yeung, Nystrom, Aronson, & Cohen, 2006). Furthermore, directing attention to a specific feature or location, facilitates activity in the cortical regions that are specialized in processing that feature or location (Brefczynski & DeYoe, 1999; Corbetta, Miezin, Dobmeyer, Shulman, & Petersen, 1991). The effect of spatial attention on activity in V1, V2 and V4 was studied with single cell recordings in monkeys, and the results suggest that cells in V2 and V4 are modulated by spatial attention whereas the cells in V1 are not (Luck, Chelazzi, Hillyard, & Desimone, 1997). Also other studies combining PET and ERP have demonstrated that directing attention to a specific location increases regional blood flow in extrastriate areas (Heinze et al., 1994; Mangun et al., 2001; Mangun, Hopfinger, Kussmaul, Fletcher, & Heinze, 1997).

2.3.2. Timing of attention: ERP studies

Most of the published studies suggest that visual attention does not influence the earliest evoked potential, C1 (Clark & Hillyard, 1996; Heinze et al., 1994; Martínez et al., 1999), but there are also contradictory findings (Kelly, Gomez-Ramirez, & Foxe, 2008; Zani & Proverbio, 2009). Focusing spatial attention on the specific location in the visual field enhances the amplitudes of P1 (80–130 ms) and N1 (140–200 ms) (Hillyard & Anllo-Vento, 1998; Mangun & Hillyard, 1991). In contrast to spatial attention, the electrophysiological response to selective object attention has been shown to emerge later than at P1 or N1 time window. The typical finding for selective attention is a broad negative amplitude shift (*selection negativity, SN*), which starts 140–180 ms after the onset of the visual stimulus and continues for about 200 ms (Harter & Aine, 1984; Harter & Guido, 1980). In the typical experimental set-up, the task of the participant is to discriminate the target stimulus from the nontarget stimuli. SN is best observed as a difference wave, where the responses to the nontargets are subtracted from the targets (Hillyard & Anllo-Vento, 1998). The onset of the SN is determined by the properties of the visual stimulus (Hillyard

& Anllo-Vento, 1998). In the studies where SN has been observed, selective attention has been focused on the orientation (Previc & Harter, 1982), shape (Smid, Mulder, Mulder, & Brands, 1992), colour (Anllo-Vento, Luck, & Hillyard, 1998), spatial frequency (Harter & Previc, 1978) or the direction of a movement (Anllo-Vento & Hillyard, 1996). SN is often followed by *late positivity* (LP) in the P3 time window (e.g., Zani & Proverbio, 1995). In addition to SN and LP, also frontal selection positivity has been reported as a response to selective attention (Baas, Keneman, & Magnun, 2002; Smid, Jakob, & Heinze, 1999).

2.4. Models of consciousness

Several models have been presented on the conceptual structure and the neural basis of consciousness, and there is still disagreement on the relation between visual consciousness and attention (e.g., de Brigard & Prinz, 2010; Tallon-Baudry, 2012). In contemporary cognitive neuroscience, two main models of the conceptual structure of visual awareness can be dissociated. According to a traditional view, subjective visual experience requires attention, and there are no subjective contents if attention has not been focused (Baars, 1997, de Brigard & Prinz, 2010; Dehaene & Naccache, 2001; Mack & Rock, 1998). In this model, attention can be described as a gateway to consciousness. It is worth noting that this view does not dissociate between phenomenal and reflective consciousness, but instead regards reportable perception as consciousness (i.e., reflective consciousness according to Revonsuo [2006] and Farthing [1991] or access consciousness according to Block [1995, 2001, 2005]). In addition, Dehaene, Changeux, Naccache, Sackur, and Sergent (2006) distinguish a *preconscious* state which refers to a perceptual processing stage where, amongst other things, the energy and duration of the visual stimulus are sufficient for the stimulus to be detected consciously if attention is directed towards it. According to Dehaene et al. (2006) and Dehaene and Changeux (2011), the stimulus is subliminal if it is not consciously detected although attention is directed towards it. In an alternative model, contents of visual awareness emerge independently of and prior to selective attention (Lamme, 2003, 2004). Thence, attention can be focused on some of the contents of visual awareness for more elaborate processing in the reflective consciousness.

Concerning the neural basis of visual consciousness, the views vary even more than with the conceptual structure of consciousness. Several models of the neural basis of visual perception suggest that re-entrant processing from the higher cortical areas back to the visual areas which are located early in the anatomical hierarchy is related to various visual processes, such as formation of fine-grained or medium-grained visual representations (Pollen, 1999), visual awareness (Hochstein & Ahissar, 2002; Lamme, 2003; Lamme & Roelfsema, 2000), or figure-ground segmentation (Bullier, 2001). The most well-known of these models is probably Lamme's model (e.g., Lamme, 2003; Lamme & Roelfsema, 2000) which states that visual consciousness is generated via recurrent (both feedback and horizontal) cortical processing in the ventral stream areas[9]. Lamme (2000, 2010) poses that this recurrent processing is a direct correlate of visual awareness, that is, the timing of recurrent processing correlates with the timing of visual awareness. Thus, according to this model, the emergence of visual consciousness does not require, for example, neural processing in the prefrontal cortex, and V1 is seen as a subcomponent in a larger recurrent network underlying visual consciousness (Lamme, 2004). Furthermore, according to the models of Hochstein and Ahissar (2002) and Campana and Tallon-Baudry (2013), (conscious) visual perception arises through two stage processes. In the first stage, a coarse visual perception (e.g., awareness of stimulus presence or "vision at a glance", Hochstein & Ahissar, 2002) is generated by a rapid feedforward sweep and in the second stage, a more detailed visual perception (e.g., awareness of stimulus features or "vision with scrutiny") is descended through cortical backward processing. Furthermore, Campana and Tallon-Baudry (2013) define coarse but *vivid* and detailed but *vivid* consciousness. According to this segregation, when we first perceive something, we have a coarse visual consciousness and over the time it evolves into a more detailed visual consciousness.

In contrast to the models that emphasize feedback projections from the higher cortical areas to the V1 or V2 in generation of visual awareness, there are views that emphasize the roles of the parietal cortex and of the regions in the prefrontal cortex in the generation of visual awareness. According to the global workspace theory (Baars, 1988, 1997, 2002), the contents of working memory correlate with consciousness and the neural representations of these contents are distributed broadly in the brain. In particular, Dehaene and Naccache (2001) have proposed that the neural architecture of global workspace consists of long distance neuronal connections which link specialized brain areas, and consciousness emerges from this phenomenon. Thus, the view by Dehaene and Naccache does not specify the exact brain regions whose activation would constitute CMVC[10]. Instead, the widespread neural connections would be necessary for consciousness. Importantly, the visual information processing purely in the visual cortex is not sufficient for visual awareness according to their view. [11]

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[9] The phenomenon of blindsight can be explained by residual neural activity in the extra striate areas and in the other higher cortical areas (e.g. Lamme, 2001); In blindsight, the feedforward sweep to these areas and the recurrent processing within the extra striate areas and between the higher cortical areas and the extra-striate areas have remained. It has been proposed that since the feedforward sweep is still present, although degraded, recurrent processing between higher areas and V1 and horizontal processing within V1 are essential to visual consciousness (Lamme, 2001).

[10] Nevertheless, in the later article the roles of prefrontal and parietal regions have been emphasized (Dehaene & Changeux, 2011).

[11] There are also several other influental models of the neural correlates of consciousness, such as Microconsciousness theory (Zeki, 2003; Zeki & Bartels, 1999), the Dynamic core theory (Tononi & Edelman, 1998), the Information integration theory (Tononi, 2004, 2012), the Thalamocortical binding theory (Llinás, 2001; Llinás, Ribary, Contreras, Pedroarena, 1998) and "the consciousness as the feeling of what happens" (Damasio, 1999). These theories, although influential, are not discussed further, because they were not directly tested in the studies of the present thesis.

3. THE AIMS OF THE PRESENT THESIS

The question of the relation between visual awareness and attention (the question of *Study I*) is central in the current studies of consciousness and cognition (Posner, 1994). There is even a possibility that attention and consciousness are so closely related that they share similar neural circuits (for a recent review see Tallon-Baudry, 2012). This view implies that the concepts of attention and consciousness refer to the same phenomenon. Naturally, the opposite view is that attention and consciousness are two different and (at least partly) independent phenomena. In *Study I*, we tested these two opposing theories concerning the relationship between attention and consciousness, Lamme's model and the model suggested by De Brigard and Prinz (2010), Dehaene and Naccache (2001) and Mack and Rock (1998). The former view predicts that the electrophysiological correlate of visual consciousness can be generated earlier than and independent of the effect of attention whereas the latter view predicts that the electrophysiological correlates of visual awareness depend on the attentional selection and should not occur for stimuli remaining outside the focus of attention. We aimed to find out whether visual consciousness and selective visual attention are different phenomena with neural processes that can be dissociated from each other and, if they were different, which one precedes the other.

The role of V1 in visual perception and consciousness is particularly interesting given that there is no clear agreement whether V1 is part of the CMVC or not. V1 may only be a mediator of neural afferents to higher cortical areas which are actually part of the CMVC (e.g., Bar et al., 2001; Crick & Koch, 1995), or alternatively, V1 itself might be part of the CMVC (e.g., Lamme, 2003, 2004). The role of V1 in visual perception has been investigated with TMS for about two decades in a considerable number of studies (e.g., Boyer, Harrison, & Ro, 2005; Heinen, Jolij, & Lamme, 2005; Juan & Walsh, 2003; Kosslyn et al., 1999; Pascual-Leone & Walsh, 2001; Sack, van der Mark, Schuhmann, Schwarzbach, & Goebel, 2009; Silvanto et al., 2005; for review see Kammer, 2007). The previous studies which have targeted V1 according to the anatomical landmarks in the head have not defined in which visual area the E-field strength actually was the strongest. In *Study II*, one of our aims was to observe to which extent V1 and V2 are affected when V1 is targeted according to the traditional paradigm relying on the external anatomical landmark. We directed TMS pulses 2 cm above the inion and modelled the TMS-induced E-field distribution of the individually defined V1 and V2 with navigation software (eXimia, Nexstim Ltd., Helsinki, Finland). We speculated that this result would help to draw inferences of the other TMS studies investigating visual awareness, where the aim has been to target V1 with more coarse TMS methods. Second, we investigated how accurately V1 can be stimulated if magnetic stimulation is directed according to the individually defined maps of V1 and V2 combined with brain navigation and TMS-induced E-field modelling. This experiment was particularly important for clarifying the prospects of studying V1 and V2 in our own future studies.

One of the particular research interests behind the present thesis was to study the *causal* role of specific cortical areas in the generation of visual consciousness, and for that purpose, TMS was employed. Specifically in *Studies III–V,* we investigated the roles of V1, V2 and LO in visual awareness by targeting TMS to these areas and observing the effects of stimulation on visual awareness. The motivation behind these studies was twofold: our aim was to study the processes that occur before visual awareness is generated and the neural processes that are directly related to visual awareness. While there are a considerable number of studies concerning the role of V1 in visual consciousness, the role of V2 in conscious perception is not well understood. Given that V1 and V2 are so densely connected with each other, it is plausible that V2 might also be a prerequisite for the generation of visual awareness. The aim of *Study III* was to explore the role of V2 in visual awareness in humans. In this study, we applied the experimental procedure that was introduced in *Study II*. With this procedure, specific early visual areas can be targeted with TMS, particularly V1 and V2.

CMVC can also be explored by studying the subjective characteristics of phosphenes. The roles of V1 and V2 in TMS-induced visual sensations were studied by stimulating these areas with TMS in *Study IV*. Our objective was to investigate whether or not the phosphenes induced from V1 stimulation are equivalent with those elicited from V2 stimulation in their subjective characteristics. We reasoned that this experiment might disclose new information concerning the roles of V1 and V2 as part of the CMVC, because specific features of visual sensations might correspond with the expected neural ensemble of the area (Lee et al., 2000).

Compared to the study by Kammer, Puls, Erb, and colleagues (2005), the advantage in our study was that we targeted the V1 and V2 with the help of TMS-induced E-field modelling in V1 and V2 and used a phosphene questionnaire to statistically analyse the participants' reports on the subjective characteristics of the phosphenes.

In *Study V*, our objective was to investigate the causal role of LO in the conscious perception of coherent images. We reasoned that if the activity in LO which has been shown to correlate with visual consciousness (Liu et al., 2012; Vanni et al., 1996) were a prerequisite for the conscious perception of object coherency, then TMS in LO at the time window ranging from about 200 ms to 340 ms should interfere with visual awareness. Alternatively, if the activation in LO at the late latencies were not necessary for visual consciousness, then TMS in LO should have no effect on conscious visual perception at the late latencies. Consequently, with this experimental set-up we were able to infer whether activation in LO is part of the actual CMVC or the neural processes preceding it.

The main objectives of the present thesis are summarized as follows:

1. to investigate whether or not visual consciousness and selective attention are separate phenomena which have separate electrophysiological responses (*Study I*)
2. to find out by analysing the TMS-induced E-field distribution which visual areas are the most affected when V1 is targeted with TMS by relying on the traditional paradigm, the external anatomical landmark method (*Study II*)
3. to explore how accurately, if at all, V1 can be stimulated with TMS by applying individual functional retinotopic maps with simultaneous brain navigation and TMS-induced E-field modelling (*Study II*)
4. to investigate whether or not processing in V2 is a prerequisite for the generation of visual consciousness *(Studies III and V)*
5. to explore the subjective characteristics of the TMS-induced phosphenes produced by V1 or V2 stimulation *(Study IV)*
6. to investigate whether LO is a subcomponent of CMVC of object coherency *(Study V)*

4. MATERIALS AND METHODS

4.1. Participants and ethical issues

In each study, the recruited participants were neurologically healthy. All of them had either normal or corrected-to-normal vision. In *Study I*, we report the data from 12 participants (age 18–28 years), *in Study II* from nine participants (age 20–28 years), *in Study III* from seven participants (21–27 years), *in Study IV* from four participants (age 22–28 years) and *in Study V* from seven participants (age 22–28 years).

Each study was conducted with the written informed consent of each participant. For the fMRI, the experiments were approved by the local ethics committee of the Hospital District of Helsinki and Uusimaa. For the TMS in *Studies II* and *III*, the ethics committee of The Hospital District of Southwest Finland approved the protocol. For the TMS in *Study V* and the EEG, the local ethics committee of the University of Turku gave the formal ethical permission.

4.2. EEG

EEG was used in *Study I*, and it was recorded with the NeuroScan software and equipment by using 20 tin electrodes (Electro-Cap International Inc., USA), arranged according to the international 10/20 system. EEG was amplified with SynAmps with the digitization rate of 500 Hz and cut-off frequencies at 0.05 and 100 Hz. The average activity between 100 and 0 ms preceding the visual stimulus was used as a baseline level. Trials with artefacts (> 70 µV) were rejected after the recording. For ERP analysis, the data was averaged time-locked to stimulus-onset.

4.3. Functional magnetic resonance imaging

FMRI was used in *Studies II–V*, and the imaging data was obtained by using a 3T MRI scanner (Signa™ Excite, General Electric Inc., WI, USA), combined with a phased-array 8-channel head coil. The values for the single shot gradient-echo echo-planar imaging sequence were: repetition time 1800 ms, echo time 30 ms, matrix 64 × 64, flip angle 60˚. Field of view was 16 cm and slice thickness 2.5 mm in *Studies II–IV,* whereas they were 20 cm and 3.0 mm respectively for *Study V*. High-resolution MRI images of the head were obtained with spoiled gradient echo sequence. The fMRI data were processed with the SPM2 software package (Wellcome Department of Imaging Neuroscience, London, UK) of Matlab™ for *Studies II* to *IV* and with SPM8 for *Study V*.

In *Studies II–V*, 24 retinotopic representations in the V1 and V2 were mapped for each participant with multifocal fMRI (mffMRI) (for details, see Henriksson et al., 2012; Vanni, Henriksson, & James, 2005). The multifocal stimulus extended from 1° to 12° eccentricity in the visual field. The visual stimuli were presented with a data projector (Christie X3™, Christie Digital Systems Ltd., Monchengladbach, Germany) and the timing was controlled with Presentation™ software (Neurobehavioral Systems Inc., Albany, California, USA). The boundaries between the V1, V2 and V3 were determined from SPMt maps which were superimposed on an anatomical 3D image, and equivalent coordinates were determined for each region's centre of representation (i.e. subareas). For the participants taking part in *Study V*, we also mapped the LO. In the object mapping, grayscale images of objects (obtained from www.freeimages.co.uk and www.morguefile.com) were presented (3 eccentricities, 8 polar angles) in a block design. The visual stimuli were presented at the fovea, meridians (mean eccentricity: 5°) and at the oblique polar angles (mean eccentricity: 2.1°). In order to facilitate the determination of the area LO in *Study V*, we also defined the motion selective region (V5). The LO was determined as the cortical area which activates for objects and is located between V3 and V5.

For *Studies II* (mffMRI-guided approach)*, III and IV,* we aimed to choose the optimal subarea for the TMS stimulation for each individual on the basis of functional anatomy of V1 and V2. In these studies, the selected subarea was always either the representation of region 8 or of region 6 (Fig. 2). For the participants in *Study V*, to stimulate V2d with TMS, we used the cortical representation of region 6. For LO stimulation, we used the cortical representation of the object stimuli presented in the lower left visual field quadrant at a mean eccentricity of 2.1°.

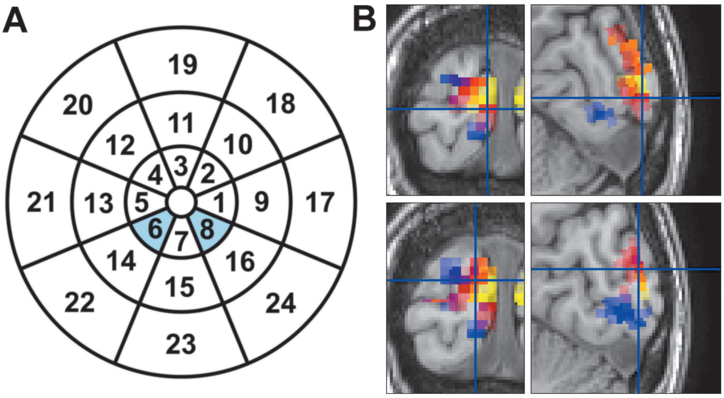

Figure 2. A. The regions in the visual field whose cortical representations (subareas) were mapped with mffMRI. The regions whose cortical representations were targeted with TMS are indicated with light blue. **B**. Examples of multifocal subareas in one representative participant in coronal and sagittal plane. In the upper figures, the crosshair points the location of V1 whereas in the lower figures the V2 is similarly indicated. Red shows the cortical representation of region 8, yellow of region 7 and blue of region 1. The border between V1 and V2 is in the subarea 7 (yellow region).

4.4. Transcranial magnetic stimulation

TMS was used in *Studies II–V*, and eXimia TMS magnetic stimulator was applied for magnetic stimulation. The figure-of-eight Nexstim bipulse coil was used for *Studies II, III* and *V*. In *Study IV*, figure-of-eight monopulse coil was used. In *Studies II*, *III* and *V*, the E-field of the second wave of the biphasic pulse was directed from lateral to medial (Corthout, Barker, & Cowey, 2001), whereas in *Study IV* the E-field was directed perpendicular to the sulcal bank of the target site.

The spatial relation between the brain and the TMS coil was monitored with an MRI-guided Navigated Brain Stimulation (NBS) system (eXimia 2.1.1 for *Studies II–IV* and eXimia 2.2.1. for *Study V*). The TMS-induced intracranial E-field distribution was modelled and visualized on the participant's structural images by the NBS system during each TMS pulse. Spherical conductor model was used to model the E-field (Heller & van Hulsteyn, 1992; Ilmoniemi, Ruohonen, & Karhu, 1999; Sarvas, 1987). The focal area of the stimulation hotspot of the TMS-induced E-field refers to 98% of the maximum stimulating E-field which is calculated 20 mm below the coil in the spherical conductor model representing the human head (see Ruohonen & Karhu, 2010). The size of the hotspot is approximately 0.68 cm^2, and the accuracy comprising all error sources of NBS is 5.7 mm (see Ruohonen & Karhu, 2010).

In *Study III*, where V2 was the target for stimulation, the TMS-induced E-field strength was higher in V2 than in V1 for all the participants. The same applied for V3 at least for the five tested participants. Likewise, in *Study IV*, where both V1 and V2 were targeted separately, the E-field strength was above the phosphene threshold only in the targeted visual area. In *Study V*, constant stimulation intensity was used for all participants, and the impact of the stimulation on the adjacent area of the target area was not controlled.

4.5. Stimuli and experimental set up

4.5.1. Study I

During the experiment, the visual stimuli (three black letters) and a black pattern mask were presented at the centre of the computer screen on a white background. The duration of the stimulus was 16.5 ms and it was followed by a blank screen for either 16.5 or 116 ms, and then by the mask (stimulus onset asynchrony, SOA = 33 or 133 ms). After the mask, a blank screen appeared after which the next trial begun. Each letter was a target in one of the three blocks and a nontarget in the other two blocks. The participants were instructed to press a button when they consciously perceived a target letter. In the control experiment (N=8), a constant stimulus–mask SOA was used. In the control experiment, the procedure was the same as explained above.

4.5.2. Studies II and III

Detection experiment

In the detection task, three grey letters (diameter 0.23°) served as visual stimuli. They were presented on a grey background by using Presentation™ software. Each trial started with an appearance of the fixation cross followed by a visual stimulus for 16.5 ms and then again by the fixation cross. For participants whose subarea 6 was stimulated with TMS, the visual stimulus was presented either to region 6 in the lower left field or to region 2 in the upper right field. Alternatively, for participants whose subarea 8 was stimulated, the visual stimulus was presented either to region 8 or to region 4. The participants were instructed to identify the letter. After the forced choice response, participants rated their subjective visual experience of the stimulus with the scale: *(1) I saw the stimulus clearly, that is, I saw at least a feature of the letter from which I could recognize it, (2) I did not see the stimulus clearly, but I saw a trace on the screen, or (3) I did not see anything at all, only the fixation point.* TMS pulses were targeted to V1 in *Study II* and to V2 in *Study III*. Pulses were delivered randomly at 9 different visual SOAs, ranging from 24 to 184 ms in steps of 20 ms.

Stimulation intensity and phosphene thresholds

To help to evaluate the impact of TMS on each visual area, we calculated the phosphene thresholds for each participant. The idea was to find the lowest TMS intensity in relation to the phosphene threshold which induces visual suppression. The phosphene threshold was defined as the pulse intensity that produces phosphenes in 50% of the pulses (e.g., Deblieck et al., 2008; Kammer, Beck, Erb, & Grodd, 2001), and it was determined with the maximum likelihood threshold hunting procedure (Awiszus, 2003) for 4 participants. For 5 participants, in *Study II* and for 3 participants in *Study III*, the phosphene threshold was estimated by delivering the pulses in steps of one percentage unit with the intensity ranging from 30% to 55% of the stimulator output. The pulses were delivered in a randomized order.

Pilot studies with three participants suggested that the optimal stimulator output intensity for the visual suppression was 120% of the individual phosphene threshold. The experimental set-up in the pilot study was similar to that in the main experiment.

Localization of V1: The external anatomical landmark and mffMRI-guided approaches (Study II)

When TMS pulses were targeted on the basis of the external anatomical landmark, 2 cm above the inion, we modelled the E-field distribution in V1 and V2 (N=8). TMS pulses were delivered at the intensity of 120% of the participant's own phosphene threshold. Specifically, E-field strength was modelled in the centre of each of the subareas of the V1 and V2 for each participant.

When the magnetic stimulation was directed according to the individual functional anatomy of V1 and V2 (mffMRI-guided approach) during the visual detection task (n= 9), we modelled the E-field distribution in V1 and V2. In particular, we compared the E-field strength in the targeted V1 subarea with the E-field strength of the retinotopically corresponding V2d subarea.

4.5.3. Study IV

The method of constant stimuli (see Kammer et al., 2001) was used to determine the phosphene thresholds for V1 and V2 stimulation. After the determination of the phosphene thresholds, 100 phosphenes were induced for each participant by magnetically stimulating V1 and another 100 phosphenes were elicited by stimulating V2d. TMS intensity was 110% of the individual phosphene threshold of the stimulated area (V1/V2). Participants' task was to draw a picture of the phosphene on the screen and to fill in the Phosphene Questionnaire form which was presented after the drawing. The phosphene drawings revealed the exact location and extent of each phosphene in the visual field.

4.5.4. Study V

Line drawings of familiar objects and non-objects ("scrambled images") served as visual stimuli. In each trial, a fixation cross was followed by a visual stimulus for 13.3 ms in the upper right or lower left visual field region and then again by the fixation cross and a blank period. The participants were instructed to discriminate whether a scrambled image or a familiar object was presented, and to assess their subjective experience *(1 = I saw a shape of the familiar object, 2 = I saw an incoherent figure, 3 = I saw a brief change in contrast but I am not sure what it was, 4 = I did not see anything at all, just a blank screen).* The TMS pulses were delivered to V2d and LO randomly at 17 different SOAs, ranging from 20 to 340 ms. Each TMS block included trials with no TMS pulses.

4.6. Data analyses

In Study I, the statistical analyses focused on the N1 (130–200 ms), N2 (200–260 ms) and P3 (290–700 ms) time windows. The analyses of variance (ANOVAs, factors: Awareness [2: aware, unaware], Attention [2: target, nontarget], Lobe [3] and Hemisphere [2]) were used in the analyses of mean amplitudes from occipital, posterior temporal and parietal electrodes.

In Study II when the external anatomical landmark approach was applied, first, the five subareas with the highest E-field strength were chosen from each participant to estimate the E-field distribution between occipital areas (V1 vs. V2 vs. V1/V2 border), the hemispheres, visual fields (the upper vs. the lower) and the eccentricity (1–3.2° vs. 3.2–6.7°). Second, only the subareas of V1 and V2 with the maximal E-field strength were compared. Third, the V1 subarea with the maximal E-field strength was compared with the E-field strength of the retinotopically equivalent V2 subarea. Respectively, the V2 subarea where the E-field strength was the strongest was compared with the retinotopically corresponding V1 subarea. When the pulse was directed to V1 by using mffMRI-guided approach, we compared the E-field strength in targeted V1 subarea with the E-field strength in the retinotopically corresponding V2d. *In Study II and III*, to analyse how different SOAs (9: 24–184 ms) and visual fields (2: upper, lower) affected the proportion of correct responses in letter discrimination (and ratings of subjective awareness in *Study III*), we carried out repeated measures ANOVA.

In Study IV, we compiled 27 research questions in total concerning the subjective characteristics of phosphenes based on the Phosphene Questionnaire. For each participant, the Mann-Whitney test was used to analyse the quantity of phosphenes in a single stimulation, the number of phosphenes with just one colour or shade in a single stimulation, the number of different basic shapes in a single stimulation, the number of achromatic phosphenes, the total number of unique hues versus other hues in a single stimulation and the brightness of phosphenes. To analyse categorical phosphene features (e.g., exact colours, shapes, area [empty/filled inside], location of phosphene), the Chi-square test was used individually for each participant. Fisher's exact test was used to analyse the values when the number of observations was small.

In Study V, for subjective ratings, only the data of the familiar objects as visual stimuli were analysed. For the analyses of subjective ratings, we had two categories: conscious perception of the coherent object vs. conscious perception of the change in contrast. To analyse the data in *Study V*, repeated measures ANOVA (cortical area [LO/ V2] and SOA [20–340 ms]) was used.

5. RESULTS

5.1. Electrophysiological correlates of visual awareness and selective attention (Study I)

Study I addressed the relationship between object attention and awareness. Another aim was to investigate the critical time window where conscious visual information processing dissociates from unconscious processing. The participants had conscious perception of 95% of the target letters at the long SOA whereas only 5% of them were perceived at the short SOA. Thus, at the SOA of 133 ms the stimulus was evidently above the subjective threshold and at the 33 ms SOA it was below. By computing difference waves between targets and nontargets (SN), we aimed to coarsely map the time window where the effect of attention was reflected in ERPs. The SN was observed 160–300 ms after the stimulus, simultaneously with the N1 and N2 potentials. Similarly, VAN (i.e. the difference between consciously seen and unseen stimuli) concurred with the N1 and N2 potentials (Fig. 3). LP was observed after both SN and VAN, and it overlapped with the P3 potential (290–700 ms).

In the N1 time window (130–200 ms), deflections to consciously seen stimuli were more negative than deflections to stimuli that remained in the unconscious level. ERPs to targets were more negative than those to nontargets. Awareness and Attention as factors did not have any interaction effects in the N1 time window, which is in agreement with the view that they are independent processes at least in this time window. In the N2 time window (200–260 ms), amplitudes to consciously seen stimuli were more negative than amplitudes to unconscious stimuli. In addition, targets showed more negativity than nontargets. Contrary to the N1 time window, Awareness and Attention interacted at the electrode sites above the left hemisphere and the temporal lobe. Further analyses revealed that deflections to consciously seen targets were more negative than deflections to consciously seen nontargets and deflections to consciously seen nontargets were more negative than to unconscious nontargets. Thus, despite the interaction effect in the time window from 200 to 260 ms, a clear VAN was also elicited for nontargets, revealing support for a view that it emerges independently of selective attention.

For LPs in the 290–700 ms (P3) time window, targets showed more positive ERPs than nontargets, and ERPs to consciously seen stimuli were more positive than ERPs to those that remained at the unconscious level. The difference was greatest at the parietal sites. The interaction between Awareness and Attention as factors and Awareness, Attention and Lobe as factors showed that in parietal electrodes, responses to consciously seen nontargets were more positive than those to nontargets that remained at the unconscious level.

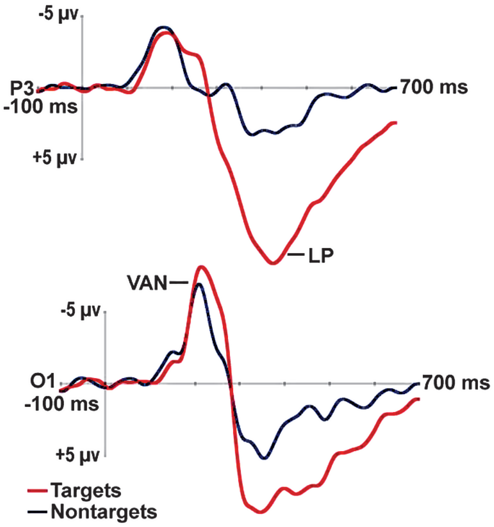

Figure 3. The difference waves between consciously seen and not consciously seen stimuli for targets and nontargets in *Study I* (N=12).

In addition, we compared amplitudes between consciously seen and not seen stimuli and amplitudes between target and nontarget stimuli in the left occipital (O1) and in the left temporal (T5) electrodes at 120, 140, 160, 180 and 200 ms after the stimulus-onset. The purpose of this analysis was to determine the onset of the effects of consciousness and attention. The results showed that the effect of consciousness was statistically significant 140 ms after the stimulus-onset. In contrast, the effect of attention became significant 180 ms after the stimulus. Thus, the effects of awareness emerged 40 ms before those of attention.

In the control experiment, participants responded to 52% of the targets and to 2% of the nontargets. ERPs to consciously seen and not seen targets began to diverge after 100 ms, with deflections to consciously seen targets revealing stronger negativity. ANOVA with Awareness, Lobe and Hemisphere as factors showed that this difference was significant in 130–200 ms and 200–260 ms time windows. The physical differences between conscious and unconscious conditions can have a considerable effect on ERP waveforms (Bachmann, 2009), but the results of the control experiment give support for the results of the main experiment and, thus, for the view that VAN does not result from the physical differences between conscious (long SOA) and unconscious (short SOA) conditions.

5.2. Accuracy of V1 stimulation with TMS (Study II)

5.2.1. Localization of V1 by using the external anatomical landmark method

The E-field distribution within the five most affected subareas of each participant showed that on average V2 had the strongest E-field. Further, 74% of these subareas represented the visual field region between 1° and 3.2 ° and 26% the visual field area between 3.2 ° and 6.7 °. Only 5% of the visual field regions corresponding to the most affected subareas represented the upper visual field. In addition, none of the selected subareas in V1 represented the upper visual field. When only the subareas with the maximal E-field strength were compared, the difference in the E-field strengths between V1 and V2 was less than 10% for five of the participants, but for three participants the E-field was 20% stronger in the V2 than in the V1 subareas. Out of all V1 subareas, the E-field was strongest in subarea 6 for seven out of eight participants.

The calculation of the angle between the direction of the electric current and the direction of the underlying gyrus/sulcus at the approximate centre of the subareas showed similarly broad variability in the orientation of the underlying gyrus/sulcus for V1, V2 and V1/V2 border. Thus, the cortical anisotropy was not likely to affect the main results.

5.2.2. Localization of V1 by using the mffMRI-guided stimulation approach

When TMS pulses were directed to V1 during the visual detection task (n= 9) according to the individual functional anatomy of V1 and V2, the TMS-induced E-field strength was modelled in the targeted V1 subarea and retinotopically equivalent V2d subarea. The results showed that when V1 was targeted, the E-field strength was higher in V1 than in V2d only in four out of the nine participants (mean difference 20.1%). For the rest of the participants, the E-field was stronger in V2d than in V1. For two of them, the difference in E-field between V2d and V1 was 32%, whereas for the remaining participants the corresponding difference was less than 10%. As mentioned above, the stimulator output intensity which induced suppression was 120% of the phosphene threshold intensity. In other words, at the phosphene threshold intensity, the E-field strength was 17% lower than the E-field strength that induced visual suppression. Thus, logical conclusion is that if the E-field strengths between the V1 and V2d varied more than 17%, the subarea with the weaker E-field strength did not induce impairment in responses but TMS influenced processing in the subarea with the higher E-field. This interpretation assumes that cortical excitability does not vary between V1 and V2 which has been demonstrated at least with non-human primates (Murphey & Maunsell, 2007). Because of the size of V1 and V2 subareas, the focal area of the TMS-induced E-field hotspot and the specified system accuracy of the eXimia NBS system we could not reliably evaluate the anisotropy of the underlying sulcus or gyrus.

The psychophysical data revealed that response accuracy impaired for the lower visual field stimuli when TMS pulses were delivered at stimulus-TMS SOAs of 64 ms, 84 ms and 184 ms (Fig. 4). The performance for the upper visual field stimuli was not affected by TMS stimulation.

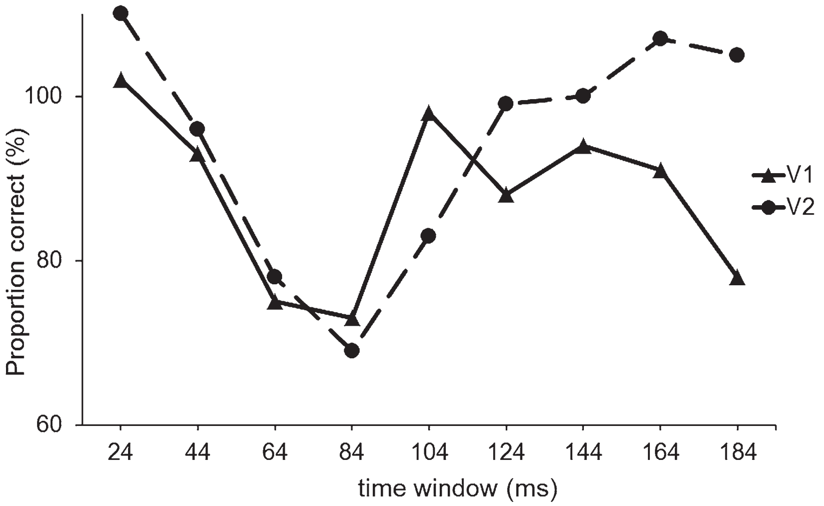

Figure 4. The proportion of the correct responses for the lower visual field stimuli for the participants to whom the E-field strength was the highest in V1 (dashed line) and for the participants to whom the E-field strength was the highest in V2 (solid line) in *Study II*. The results are scaled in relation to the responses to the upper visual field stimuli, in a way that 100% correct represents the response to the upper visual stimuli.

5.3. The role of V2 in visual consciousness (Study III)

To analyse the conscious perception of contrast change, we combined the subjective ratings of trials where the participants reported either having seen the stimulus clearly or having seen just a trace of the stimulus. Thus, in the remaining of the trials the participants reported subjectively not being conscious of the stimulus. The conscious perception of contrast change diminished for the lower visual field stimuli when the pulses were delivered at 64 or 84 ms SOA. TMS did not have an effect on conscious perception of stimuli which were presented to the upper visual field (the cortical representation of which was not stimulated), suggesting that transient scotoma was not caused by nonspecific effects of TMS.

For the conscious perception of stimulus features, the dependent measure was the percentage of trials where participants reported that they saw clearly at least some feature of the letter stimulus. TMS impaired the conscious perception of stimulus features for the lower visual field stimuli at SOAs from 44 to 104 ms. The conscious perception of stimulus features was not affected for the upper visual field stimuli. We also analysed the effect of TMS on letter discrimination accuracy. Stimulation of V2d did not have an effect on discrimination of the upper visual field stimuli, but the proportion of the correct responses was decreased for the lower visual field stimuli when the pulses were delivered at SOAs from 44 to 104 ms.

5.4. The roles of V1 and V2 in TMS-induced subjective sensations (Study IV)

In *Study IV*, we investigated the subjective characteristics of phosphenes induced by V1 or V2d stimulation. There was no systematic difference between the phosphene thresholds induced from V1 and V2d stimulation. The phosphenes elicited by V1 stimulation were statistically significantly brighter than the phosphenes which were evoked by V2d stimulation. The result was consistent for each participant. The other features of subjective characteristics of phosphenes evoked from V1 and V2d stimulation were not systematically different between the areas but seemed to depend more on the individual.

The analyses of the phosphene locations in the visual field revealed that phosphenes tended to appear to the contralateral visual field relative to the stimulation site. For three participants out of four in more than 50% of the trials, the phosphenes were located in the peripheral visual field areas (12–39° from the fixation), although the electric current was directed to the cortical representation of around 1–3° in the visual field. Furthermore, there even were visual field regions where phosphenes were seen in more than 50% of the trials, although the E-field strength in the corresponding subarea was below 50% of the individual phosphene threshold. Thus, it is improbable that the neurons of the cortical representations of such peripheral visual field regions would be directly depolarized with TMS due to their deep location in the brain.

5.5. The roles of LO and V2/V1 in visual awareness (Study V)

TMS of LO at the SOAs of 120 ms and from 300 ms to 320 ms impaired the conscious perception of coherent objects. TMS to V2/V1 had an effect on the conscious perception of coherent objects when the pulse was delivered between 60 and 120 ms after the onset of the visual stimulus. TMS affected the conscious perception of change in contrast differently during LO and V2/V1 stimulation, in a way that TMS in V2/V1 interfered with the conscious perception of change in contrast. However, given that in V2/V1 stimulation none of the SOAs revealed statistically significant effects, the source of this result remains open. In the control condition, we delivered TMS to the vertex while participants (N=7) carried out the same behavioural task as in the main experiment. TMS pulses did not have an effect on the conscious perception of coherent objects, supporting further the idea that the nonspecific effects of TMS cannot explain the TMS-induced visual suppression in the main experiment.

In addition, we analysed the scrambled image versus coherent object discrimination accuracy. TMS to V2d impaired the discrimination accuracy at the SOAs of 60 and 80 ms. TMS to LO did not have an effect on discrimination accuracy.

6. DISCUSSION

In this thesis, my main goal was to shed light on the neural basis of visual consciousness by empirically exploring the following main questions: Are visual consciousness and selective attention two distinct phenomena? What are the roles of the functionally early visual areas and LO in the most fundamental forms of visual consciousness: the conscious perception of contrast change, and the consciousness of brightness? What is the role of these areas in the conscious perception of stimulus features and coherent objects? Which theoretical model of visual consciousness do our results support? In addition to the main questions, my additional objective was to observe which areas are affected by TMS targeted to V1 and, moreover, if it is possible to target specifically V1 without also significantly stimulating neighbouring regions.

The main results showed that the initial effects of visual consciousness (about 140 ms after the stimulus-onset) on electrophysiological responses appeared earlier than the effects of attention and also in the presence or lack of attention. The initial effects of attention emerged independently of consciousness. The results imply that *visual consciousness and selective attention are independent phenomena*, and given that visual consciousness is a phenomenon that is separate from selective attention, it should also be studied as such. The results of the present thesis also imply that reliance on purely external anatomical landmarks is not sufficient in TMS studies if the aim is to determine the functional role of a particular visual area. *If the TMS pulse is directed 2 cm above the inion, the E-field strength is likely to be strongest in V2d.* The results also implicate that if the objective is to study the role of V1 with TMS methodology, navigated brain stimulation with coil positions based on detailed maps of individual functional anatomy and computational modelling of the TMS-induced E-field distribution in V1 and V2 are required. Regarding the neural processes preceding CMVC, as shown earlier for V1, the results implied that also *V2 is necessary for conscious visual perception*. The results are also in accordance with the view that the contribution of V2 is necessary for a shorter time window for conscious perception of contrast change than for conscious perception of more complex stimuli. In addition, we found that *the phosphenes elicited by the TMS of V1 were brighter than identically induced phosphenes arising from the adjacent area V2*. These findings suggest stronger V1 contribution to the generation of subjective light sensation in the human brain. The results of this thesis also suggest that LO is necessary for the conscious perception of coherent object shape in two different time windows. The timing of the later dip might correlate with the timing of the ERPs related to the visual awareness of coherent object shape. Thus, these results give support for a view that *activation in LO is directly involved in the generation of visual awareness.*

6.1. The relation between visual awareness and attention as studied with ERPs

In *Study I*, we compared Lamme's model (2003, 2004) and the model presented by Mack and Rock (1998). One of our objectives was to find out whether the negativity that is related to awareness (i.e. VAN) and the negativity that is related to selective attention (i.e. SN) represent the same phenomenon or two distinct phenomena. If they represent different phenomena, our aim was to observe which one precedes the other. Our results showed distinct electrophysiological correlates for awareness and selective attention. The initial part of VAN (up to 200 ms) was independent of selective attention, but attention had an effect on the later part of it (200–260 ms). This result is in agreement with the view that when attention is focused on some content in the consciousness, this content might consciously be seen differently than the unselected contents. SN emerged for target stimuli independently of visual consciousness: it appeared for both targets that the participants consciously perceived and targets that they were unconscious of. This result is in accordance with the view that selective attention can affect visual input although the visual stimulus is not consciously seen, thus converging with several other studies (e.g., Faivre & Kouider, 2011; Naccache, Blandin, & Dehaene, 2002).

Attention had almost no effect on P3 for the stimuli which were not consciously detected, implying that the nonconscious visual stimuli were not processed further in the late positivity time window. Given that P3 was strongly affected by the manipulation of attention for the conscious stimuli, the late positivity seems also to partly reflect selective attention. The results converge clearly with the earlier studies showing that LP is related to the neural processes underlying cognitive processes, such as working memory (e.g., Polich & Kok, 1995). Taken together, earlier results and the results of this thesis suggest that LP reflects neural processes related to other phenomena than pure visual consciousness.

An alternative interpretation of this result states that visual awareness actually involves two separate processes (de Brigard & Prinz, 2010). The first one would be the so called "state of availability/detectability" and the second "detection". According to this interpretation, only the later process (detection) would correlate with visual awareness, and thus the early part of the VAN (or the VAN as a whole) would not represent visual awareness but some preceding processes instead (detectability). Another possible interpretation of the results is that LP actually reflects the consequences of visual consciousness, VAN visual consciousness and P1 the preceding prerequisite processes of consciousness. To dissociate the awareness related processes from each other, these alternative models should be tested in future studies, for example, by studying the effects of TMS in particular brain areas on subjective awareness (Aru et al., 2012).

There are some limitations in *Study I* that should be noticed. First, the participants gave a response immediately after they had seen the target stimulus. Thus, the neural processes related to preparing and giving the response were not controlled for and could have reflected in the electrophysiological responses as a difference between conscious and unconscious target stimuli. However, if the responses would purely explain the enhanced negativity for conscious stimuli, the negativity should not have emerged for conscious nontarget stimuli, because the nontarget stimuli were never responded to. Importantly, the early effects on electrophysiological responses between conscious and unconscious stimuli were observable for both nontargets and targets in the same time window. Therefore, it is highly unlikely that the VAN would only reflect the motor responses. Second, it is noteworthy that in this study, we used letters as visual stimuli. It is not entirely clear whether the present results concerning the timing of selective attention would be replicated if some other visual stimuli embodying some other visual characteristics would be used. For example, the effects of selective attention on colour (Anllo-Vento et al., 1998) or motion (Valdes-Sosa, Bobes, Rodriguez, & Pinilla, 1998) are reflected in ERPs already in P1 time window. On the other hand, Koivisto and Revonsuo (2008b) demonstrated that the effects of attention on the spatial frequency or orientation are tracked in ERPs later than the effects of visual consciousness.

In *Study I*, we manipulated selective (feature/object based) attention, not spatial attention. Thus, in our experiment, the visual stimuli were always in the focus of spatial attention. In contrast to selective attention, electrophysiological studies suggest that spatial attention is required in order to have a conscious perception of an object. Wyart and Tallon-Baudry (2008) demonstrated with MEG that high-frequency gamma-band oscillations are increased for spatially attended stimuli (either seen or unseen), whereas elevation of low-frequency gamma-band oscillations are related to consciously seen stimuli which are either attended or not-attended. This finding fits well with the view that spatial attention and visual awareness have distinct neural correlates. Nevertheless, by manipulating both visual awareness and spatial attention in the same experimental set-up, Koivisto, Kainulainen and Revonsuo (2009) showed that VAN emerges only for the stimuli on which spatial attention is focused. They found only a slight effect of consciousness in the LP time window. Thus, if we accept the view that VAN is the direct correlate of visual awareness, the results suggest that spatial attention is required for normal visual awareness converging with other studies (Chica, Paz-Alonso, Valero-Cabré, & Bartolomeo, 2013; for reviews see Deouell, 2002; Driver & Vuilleumier, 2001). The result of Koivisto et al. (2009) concurs also well with the finding that in a passive viewing condition the early part of VAN is reduced (Koivisto & Revonsuo, 2008b), and with the findings from neuropsychological patients suffering from a unilateral hemispatial neglect syndrome (Heilman et al., 2012; Kerkhoff, 2001; Vallar, 1998). Thus, it is possible that without spatial attention no features of the stimuli are perceived consciously, although the visual input is processed in the ventral stream (see also Koivisto et al., 2009; Revonsuo, 2006).

Concerning the theoretical models of the relationship between attention and consciousness, as mentioned above, our results from *Study I* provide evidence that selective, feature based attention is not necessary for visual awareness. This result is in line with Lamme's model and consistent with later published studies (Koivisto & Revonsuo, 2008b; Koivisto et al., 2006). Nevertheless, the studies by Koivisto et al. (2009), Lamy, Salti, and Bar-Haim (2009) and Salti et al. (2012) support more closely the model presented by Dehaene and Naccache (2001) and Mack and Rock (1998). These results strongly suggest that visual awareness is dependent on attention, although awareness and spatial attention are not the same phenomenon (Wyart & Tallon-Baudry, 2008). Consequently, the current dominant models of the relationship between consciousness and attention are likely to overly simplify the explanation of the complex nature of consciousness. Recently, Tallon-Baudry (2012) introduced a new model of the relationship between visual consciousness and attention, which she calls cumulative influence model. According to this view, distinct neural mechanisms underlie attention and consciousness, and both influence the final stage of decision, although with differing force. Importantly, this model suggests that consciousness and attention influence each other at the level of decision, not before. This model seems to explain the current data from electrophysiological recordings. According to an alternative explanation, different forms of attention influence consciousness in different manners. For example, spatial attention might actually be a gateway to visual consciousness but pure feature based selective attention would not. In other words, the neural processes related to spatial attention would influence the neural processes that are required to generate visual consciousness: in most of the circumstances, spatial attention would be required to boost the generation of visual consciousness. The neural processes related to selective attention would instead only facilitate the neural processes related to visual consciousness but the absence of selective attention would not solely be able to prevent a specific content from emerging into consciousness. This interpretation would also receive support from the current study and from earlier findings (e.g., Karns & Knight, 2009; Kelly et al., 2008; Koivisto et al., 2006; Koivisto et al., 2009; Lamy et al., 2009; Salti et al., 2012).

6.2. The causal relationship between visual consciousness and neural activation in V1, V2 and the ventral stream

6.2.1. Targeting primary visual cortex with TMS in visual perception studies

We modelled the E-field distribution when the traditional anatomical landmark method was used for targeting the TMS. Specifically, we aimed at answering whether V1 receives stronger impact than V2 from magnetic stimulation when the centre of the TMS coil is placed 2 cm above the inion to target V1. We reasoned that this could open new doors for the interpretation of earlier studies and literature. The modelling of the TMS-induced E-field distribution showed that the traditional stimulation site of 2 cm above the inion results in a stronger E-field in V2 than in V1. Nevertheless, there was also notable variation between individuals in the areas that received the strongest impact. For three of the eight participants, the E-field strength in V2 was notably stronger than in V1, whereas for the rest of the participants the strengths of the induced E-fields between V1 and V2 were relatively similar. These results are in agreement with earlier findings which indicate that the standard occipital landmarks of the international 10–20 system are not consistently related to specific underlying cortical brain areas across different individuals due to considerable variation in individual brain anatomy and its spatial relation to the external anatomical landmarks on the skull (Okamoto et al., 2004; Steinmetz et al., 1989; Towle et al., 1993). In addition, we found that 90% of the most affected subareas in V1 extended on the visual field 1–3.2^{o} from fixation, while in V2, the percentage of subareas at this eccentricity was considerably lower (65%). These results converge well with Kastner et al. (1998) who suggested that the visual field deficits at 1–3° might be caused by the stimulation of V1, V2 and V3, whereas visual field deficits at eccentricities 4-7° are caused only by the stimulation of V2 and V3.

To study more specifically the role of V1 and V2 in visual consciousness, we explored whether TMS could be used to stimulate V1 and V2 separately. In particular, we asked whether it is possible to selectively stimulate V1 when the area is targeted by using functional images of V1 and V2, combined with the modelling of the TMS-induced E-field distribution in V1 and V2. When TMS pulses were directed to V1 by using mffMRI, V1 was the most affected area for about half of the participants. Thus, retinotopic maps increased the probability of hitting V1, but it was not possible in each and every participant, or at the general level. Our result is at odds with the earlier finding which suggests that there is no position of coil or visual stimulus that would yield stronger E-fields in V1 than in V2 (Thielscher et al., 2010). Our results, instead, imply that selective stimulation of V1 depends on the individual functional anatomy of V1 and V2 and that it can be achieved in some human participants. There are some differences in the experimental procedures between our and Thielscher et al.'s study which may explain the differing results. In our study, more functional subareas were investigated for each participant than in Thielscher et al.'s study, because we mapped 24 retinotopic subareas in two hemispheres from V1 and V2 while Thielscher et al. (2010) mapped dorsal V1, V2 and V3 in one hemisphere. In addition, in our study more participants (N=9) were examined than in Thielscher et al.'s study (N=4). Furthermore, in *Study II* we compared only the equivalent retinotopic regions in V1 and V2d whereas Thielscher et al. compared the whole mapped visual area V1 and V2d with each other. However, in *Study IV* we also compared only the specific subarea (in V1 or V2d) with all the nontargeted subareas of the adjacent visual area (V2 or V1). So, the comparison method in *Study II* is not likely to explain the differences between our study and the study by Thielscher et al.

It should, however, be noted that in this study we used the spherical model, but it is likely that using more realistic models (which would take into account the conductivities of the tissues and the direction of the E-field in respect with the pyramidal cells, see e.g., Fox et al., 2004; Opitz, Windhoff, Heidemann, Turner, & Thielscher, 2011; Thielscher, Opitz, & Windhoff, 2011) could have made the locating of the E-field distribution more accurate. Importantly, the spherical model has been commonly evaluated as sufficiently accurate for the modelling of the occipital lobe (Davey, 2008; Hämäläinen & Sarvas, 1989; Ruohonen & Karhu, 2010; Tarkiainen, Liljeström, Seppä, & Salmelin, 2003; Thielscher et al., 2010). Nonetheless, it is not entirely clear whether phosphene and suppression thresholds are lower if the current is directed perpendicular to the underlying sulcal bank (Fox et al., 2004; Kammer, Vorwerg, & Herrnberger, 2007). In our studies, we used perpendicular current direction with respect to the underlying sulcus when we delivered the pulses with a coil that delivers monophasic pulses, whereas with the coil that delivers biphasic pulses (inducing bidirectional current) the current was not directed according to the underlying sulcus. Thus, it is possible that the accuracy of the stimulation in *Studies II* (the mffMRI-guided approach), *III* and *V* might have been improved if the current would have been directed perpendicular to the average direction of the sulcus. However, we also noticed that due to the complex surface geometry in the occipital lobe in relation to the TMS hot spot, it is complicated to direct the electric current definitely perpendicular to the sulcal bank. It is more likely that neurons in various orientations are affected.

In summary, our results suggest that if the stimulation area is not carefully controlled, it is not possible to conclude anything about the role of any particular visual area which is located early in the anatomical hierarchy. Thus, our results together with those of Thielscher et al. (2010) should be acknowledged when inferences are drawn from the earlier studies where the aim has been to stimulate V1 and the stimulation site has not been controlled: Differences in the individual functional anatomy of V1 and V2 and even V3 might, to some extent, influence the variable results in TMS studies investigating visual perception. The results imply that in the previous studies in which external anatomical landmarks or MRI images have been used to target V1, the most probable stimulation site has actually been V2. *Thus, the results suggest that instead of referring to the stimulation site in such studies as "V1 with possible contamination of V2", it might be more accurate to say "V2 with possible contamination of V1"*. In addition, it should be acknowledged that in all TMS studies, converging evidence from TMS, neuroimaging and lesion studies is critical.

6.2.2. The neural basis of consciousness of contrast change

What TMS-induced suppression can tell us about conscious perception of contrast change?

One of my main objectives in this thesis was to study the role of V2 in the conscious perception of contrast change – a fundamental form of visual awareness. We found that TMS targeted to V2 impaired the conscious perception of change in contrast, supporting the view that intact functioning of V2 is necessary for visual awareness. Conversely, the direct neural connections from V1 to the extrastriate areas (e.g., V3, V3a, V4, and V5) are probably not sufficient for conscious detection of change in contrast. Alternatively, TMS pulse to V2 may disturb normal processing in the whole network.

In *Study III*, magnetic stimulation of V2d at 84 ms after the stimulus-onset suppressed the conscious perception of change in contrast. Based on the measurements of electrophysiological responses in humans, activation in V1 is estimated to begin 40–60 ms after the stimulus-onset (e.g., Clark et al., 1995; Vanni et al., 2004; Wilson et al., 1983) and studies with non-human primates show that activation spreads to V2 only after a few milliseconds (Nowak et al., 1995; Raiguel et al., 1989). Thus, when the TMS pulse was delivered 84 ms after the stimulus-onset, it is probable that the visual input had already spread to higher cortical areas. This suggests that the initial neural afferent from V1 through V2 to the other brain areas (within approximately 80 ms from the stimulus-onset) might not be sufficient to arouse conscious perception. Thus, it is possible that V2 is necessary for conscious perception because of local visual volley processing in V2, or alternatively, the top-down projection from V2 to V1 is a prerequisite for conscious perception via modulating the activation in V1. While there are contradictory findings on the influence of V2 inactivation on responses in V1 (Hupé, James, Girard, & Bullier, 2001; Sandell & Schiller, 1982; see also, Salin & Bullier, 1995), the signals in non-human primate's V1 are affected by the feedback from V2 at least slightly (Bullier et al., 1996), and the majority of the cortical feedback activation of V1 descend from V2 (Barone, Batardiere, Knoblauch, & Kennedy, 2000). The third possibility is that V2 is important purely as a mediator of neural activation between V1 and higher cortical areas.

In contrast to the results of *Study III*, in *Study V*, TMS in V2/V1 suppressed only slightly the conscious perception of contrast change and it was not entirely clear in which time window the suppression occurred. It should, however, be noted that in *Study V*, our main objective was to examine the influence of magnetic stimulation on the conscious perception of object coherency, not the conscious perception of change in contrast. Therefore, in *Study V* the visual stimuli were wider (the diameter of the stimulus was 1.3°) than in *Study III* (.2°). Consequently, it was to be anticipated that magnetic stimulation would not totally wipe out the awareness of the visual stimulus in *Study V*.

In conclusion, the results of this thesis suggest that in addition to V1, also V2 seems to be another cortical area necessary for visual awareness. Perhaps, generation of visual consciousness requires bidirectional processing between V1 and V2. Future studies should explore whether other extrastriate areas (e.g., V3, V3a, V4) are also necessary for the conscious perception of contrast change. Furthermore, as discussed in the Introduction, the role of parietal cortex in the awareness of contrast change is not entirely clear. The results from the patients suffering a lesion in the parietal cortex and having hemispatial neglect give support for the view that activation in the parietal cortex is necessary for visual awareness. However, the parietal cortex consists of several subareas and there are no published studies to date that would have investigated causal relation between the conscious perception of contrast change and the activations of specific regions in the parietal cortex with healthy humans. Although studies suggest that V1 and V2 are necessary for conscious perception which has been generated via visual input through the geniculate-striate pathway, given that the removal of V1 (and maybe also V2) does not result in the cessation of dreams, these two areas are probably necessary only for conscious perception which has been generated through the geniculate-striate pathway but not for visual consciousness which is generated internally without stimulus input (i.e. dreaming). Nevertheless, although lesions in V1 (and V2) (Solms, 1997) do not result in the cessation of dreaming, it does not prove that these areas are not part of the neural correlates of conscious visual perception. It is still possible that V1 and V2 are part of the CMVC which is generated through geniculate-striate pathway but not the CMVC which is elicited in dreaming. In contrast to removal of V1 and V2, the removal of PPC results in the cessation of dreams (Solms, 1997), giving support for the view that the PPC is also likely necessary for internally generated visual consciousness. These important issues should be explored in future studies with neurologically healthy participants.

TMS-induced visual sensations

We compared the subjective characteristics of phosphenes which were produced either by magnetic stimulation of V1 or V2d in *Study IV*. We reasoned that this might disclose new information concerning the roles of V1 and V2 as part of the CMVC, because specific features of visual sensations might correspond with the expected neural ensemble of the area (Lee et al., 2000). We found that the phosphene thresholds in V1 and V2d did not systematically differ from each other, which concurs with earlier findings (Kammer et al., 2001, Kammer, Puls, Erb, et al., 2005; Lee et al., 2000; Murphey et al., 2009), and suggests that both the stimulation of V1 and the stimulation of V2 are similarly capable of producing subjective sensations. The results based on subjective reports also showed that the characteristics of V1 and V2d phosphenes were very much alike. The most consistent and prominent difference between the phosphenes was observed in their brightness (i.e. perceived luminance). The phosphenes which were produced by stimulating V1 were reported as being brighter than the phosphenes which were induced by stimulating V2d, suggesting that local processing in V1 or projections from V1 to other brain regions arouses more intense sensations of brightness than processing within V2 or via connections from V2 to the other areas. The more intense brightness induced by TMS in V1 cannot be explained by higher TMS intensity in this area, because the phosphene thresholds between V1 and V2d did not differ, and thus, both areas seemed to be equally responsive to TMS.

The neural correlates of brightness in humans are not well understood. Cornelissen, Wade, Vladusich, Dougherty, and Wandell (2006) studied the neural correlates of brightness with human participants by using fMRI and reported activation in the higher cortical areas than V1 or V2 to be associated with brightness perception. On the other hand, single cell recordings in cats (MacEvoy, Kim, & Paradiso, 1998; Rossi & Paradiso, 1999; Rossi, Rittenhouse, & Paradiso, 1996) and non-human primates (Kinoshita & Komatsu, 2001) demonstrate that a specific ensemble of V1 neurons responds to brightness. Also thin stripes in V2 are known to contribute to brightness perception (Lu & Roe, 2007). In addition, in a recent fMRI study Salmela and Vanni (2013) demonstrated that V1, V2 and V3 respond to brightness (see also van de Ven, Jans, Goebel, & De Weerd, 2012). Thus, the majority of the studies suggest that brightness perception is associated with the activation of V1 and V2.

Kammer, Puls, Erb, et al. (2005) and Murphey et al. (2009) did not report systematic differences in the quality of phosphenes between V1 and V2 stimulation. Unfortunately, in these studies, the participants were not asked to evaluate or report the subjective perceived brightness of the phosphenes they experienced. In addition, in their studies the descriptions of phosphenes were based on drawings and either free recall (Kammer, Puls, Erb, et al., 2005) or an interview (Murphey et al., 2009). In contrast, the strength of our study was the systematic, structured method for collecting data concerning the subjective visual features of the perceived phosphenes. This was accomplished by using the Phosphene Questionnaire (the form is available in *Study IV*, Supplementary data) and carrying out statistical analyses. Overall, our results imply that systematic analyses of subjective ratings might unveil some information of the phosphene features that could be missed when subjective reports about the perceived phosphenes are collected by using a less systematic method and after a temporal delay. In addition, Kammer, Puls, Erb, et al. (2005) used information of functional anatomy to guide TMS to V1 and V2, but no TMS-induced E-field modelling was applied which has been shown to be crucial in targeting V1 selectively (*Study II*; Thielscher et al., 2010). Thus, it is conceivable that their method was not sufficiently accurate to target V1 and V2.

It could be argued that if both areas, V1 and V2, were equally sensitive to TMS, they should also produce equally bright phosphenes. However, this interpretation presumes that phosphenes are produced in a continuum from very low contrast to bright. In other words, according to this view when phosphenes are produced precisely at the phosphene threshold intensity or at the lowest E-field strength which produces phosphenes, the brightness of TMS-induced phosphenes is always equal between the visual areas. Nevertheless, it is not clear, whether conscious perception is an all-or-none phenomenon or whether it is a gradually generated phenomenon (see Bachmann, 2013). Our results suggest that TMS-induced E-field triggers a depolarization of neurons which produces visual sensations whose brightness varies between the areas. Thus, this result gives support for the view that visual sensation, at least at the phosphene threshold, is closer to all-or-none phenomenon than to a gradually generated one. This interpretation is also in agreement with the recent study by Sekar, Findley, Poeppel and Llinás (2013). In their MEG study, the response amplitudes of the event-related fields were similar for the conditions where stimulus was perceived either with high or low certainty, and the response amplitude which was observed for perceived stimuli was not observed in the unconscious condition.

Murphey et al. (2009) demonstrated that the stimulation of several visual areas (e.g., V3, V4/V8, PPA) induces phosphenes, but the higher visual areas were less sensitive to induce phosphenes than the areas closer to the calcarine sulcus (Murphey et al., 2009). In other words, by using the same stimulation intensity, the probability of an electrode to produce phosphene sensation decreased in proportion to the distance from the calcarine sulcus. Taken together, our results and those of Murphey et al. suggest that early visual areas (V1, V2) are equally sensitive to induce phosphenes, although the phosphenes generated from V2 stimulation are less bright than the phosphenes induced from V1 stimulation. Importantly, to have a sensation of phosphenes, luminance (or brightness) sensitive neurons need to be activated. As aforementioned, it is not entirely clear which brain areas contain neurons that are sensitive to brightness. The most probable cortical sites include V1 and V2. An interesting question is whether higher extrastriate and temporal areas (e.g., V4/V8, LO, V5, PPA) also contain brightness sensitive neurons or whether top-down activation from higher temporal and visual areas back to V1–V3 are required for the generation of phosphenes. Indeed, some results of this thesis were consistent with the view that feedback activation is related to the generation of conscious perceptions, which would fit to the models presented by Lamme and Roelfsema (2000) and Pollen (1999). We found that the locations of phosphenes in the visual field did not correspond with the predicted locations which were based on the E-field distribution in the cortex. Given that the phosphenes typically appeared at a larger eccentricity than the target region of the E-field, it is likely that TMS activated a rapid feedforward-feedback-track, which has been shown to be related to the surround modulation of classical receptive field responses in V1 (Angelucci & Bressloff, 2006; Schwabe, Ichida, Shushruth, Mangapathy, & Angelucci, 2010; Schwabe, Obermayer, Angelucci, & Bressloff, 2006), comprising far surround (Ichida, Schwabe, Bressloff, & Angelucci, 2007). Given that the corresponding region of the horizontal connections can reach only up to 0.6° in the visual field (for review see Bullier, 2001), horizontal connections cannot explain the large spreading of activation as presented here. The view that connections from the TMS-targeted site to other brain areas underlie the generation of phosphenes is in agreement with some earlier studies in humans (Cowey & Walsh, 2000; Pascual-Leone & Walsh, 2001) and in monkeys (Tolias et al., 2005).

TMS targeted at the FEF modulates activity in V1 and extrastriate areas (Ruff et al., 2006). Some results suggest that TMS of FEF impairs the conscious perception of contrast change (O'Shea, Muggleton, Cowey, & Walsh, 2004) whereas other studies suggest the opposite (Grosbras & Paus, 2003; Silvanto, Lavie, & Walsh, 2006). Chica, Valero-Cabré, Paz-Alonso, and Bartolomeo (2014) demonstrated that rTMS to left FEF improved the detection of ipsilateral stimuli and decreased the detection of contralateral stimuli. All in all, magnetic stimulation of FEF seems to have an effect on the conscious perception of contrast change. However, it is not entirely clear whether it is actually the remote activity from FEF to visual areas that has an effect on consciousness or whether the activity of FEF in itself has an effect on consciousness. Yet, it is likely that FEF, or connections from FEF to visual areas, contribute to the conscious perception of contrast change.

In summary, *Study IV* provides strong causal evidence on that the stimulation of V1 and V2 are equally capable of generating subjective light sensations but that the activation of V1 makes a stronger contribution to the intensity of the basic experience of light. Altogether, the results from *Studies III* and *IV* support the view that both V1 and V2 or connections from these areas to the other areas are important (and even necessary) to perceive brightness and changes in it.

6.2.3. The neural basis of consciousness of stimulus features

In addition to investigating the neural basis of conscious perception of brightness, my objective was to study the necessary time windows of processing in V1, V2 and LO in the *conscious perception of stimulus features*. The effects of TMS on conscious perception of stimulus features were explored in two separate studies (*Studies III* and *V*) by investigating in which time windows TMS disturbs the conscious perception of the features of letters or objects. In particular, we were interested to find out whether TMS to V2 would prevent visual awareness in two separate time windows or in one broad time scale as it would be predicted from Lamme's model (2003, 2004, 2006). We also aimed at discovering whether the conscious perception of contrast change is impaired by TMS to V2 in a different time window than the conscious perception of stimulus features. The models of Hochstein and Ahissar (2002) and Campana and Tallon-Baudry (2013) predict longer processing periods in V1 and V2 for the conscious perception of stimulus features than for the conscious perception of contrast change.

In *Study III*, the activation in V2d was found to be a prerequisite for the conscious perception of change in contrast for a shorter duration (up to 84 ms) than for the conscious perception of stimulus features and letter discrimination (up to 104 ms) which involves more elaborate visual processing. In *Study V*, we investigated the time intervals during which LO and V2/V1 are necessary for the conscious perception of a coherent object. Concerning the results of differences between coarse and complex perception, a similar pattern of results was found in *Study V* as in *Study III*. In *Study V*, TMS of V2/V1 impaired the accuracy of the discrimination between scrambled and coherent objects up to 80 ms whereas the conscious perception of a coherent object was disrupted up to 120 ms. These patterns of results converge well with those of Koivisto, Railo, and Salminen-Vaparanta (2011) whose results implied that the activation in V2/V1 is a prerequisite for a longer duration for the conscious perception of a bar or an arrow and discriminating an arrow shape than for discriminating the orientation of a bar. These findings suggest that the classical TMS-induced dip occurring around 100 ms post-stimulus due to the stimulation of V1/V2/V3 (Amassian et al., 1989) may be related to different processing stages: the initial part of the decline might be caused by impairment in the (conscious) perception of contrast change and simple orientation discrimination, while the later part might be associated with the discrimination and conscious perception of more complex stimulus features. Thus, regarding the models of neural basis of visual consciousness, the results of this thesis and the results from earlier studies (de Graaf, Goebel, & Sack, 2012; Koivisto, Railo, & Salminen-Vaparanta, 2011; Koivisto & Silvanto, 2012) support the view that coarse conscious perception requires processing in V2 (and maybe in V1) for a shorter period of time than the generation of more detailed visual perception, which is clearly in agreement with the view by Campana and Tallon-Baudry (2013) and Hochstein and Ahissar (2002). Nevertheless, although their model explains part of the neural basis of generation of different contents of visual consciousness, it leaves open the question of specific CMVC.

The results of the present thesis did not provide an unambiguous answer to the question on whether activation in V1 or V2 is necessary for conscious perception also after the classical TMS-induced impairment taking place around 100 ms after stimulus-onset. Importantly, there are studies suggesting that activation in V2/V1 is necessary for visual perception in the later time scale than the classical TMS-induced dip (Camprodon et al., 2010; Juan & Walsh, 2003; Koivisto & Silvanto, 2012; Wokke, Sligte, Scholte, & Lamme, 2012; Wokke, Vandenbroucke, Scholte, & Lamme, 2013). For example, Wokke and colleagues showed that TMS in V2/V1 interfered with the subjective perception of illusory contours around 160–180 ms after stimulus-onset (Wokke et al., 2013) and figure-ground segregation around 240–260 ms after the onset of a stimulus (Wokke et al., 2012). Koivisto and Silvanto (2012), on the other hand, demonstrated that TMS on V2/V1 impaired orientation discrimination in a task that required feature binding in a relatively long time window, starting from around 90 ms after stimulus-onset and continuing until 240 ms after stimulus-onset. In our earlier study (Koivisto, Railo, Revonsuo, et al., 2011), we demonstrated that TMS in V1/V2 disrupted the awareness of natural scenes up to 180 ms post-stimulus. Notably, only Koivisto and Silvanto (2012) and Koivisto, Railo, Revonsuo, et al. (2011) used an experimental set-up which actually required subjective ratings of visual awareness. Other studies used accuracy to evaluate perception, which may also be influenced by unconscious processes.

Activation in V3, V4, parietal cortex and several occipito-temporal regions is associated with the visual perception of various shapes or objects (Grill-Spector, 2003; Hedgé & Van Essen, 2000, 2003, 2007). Whether these areas are necessary for the conscious perception of features or shapes is not clear at all, and there are only few studies which have investigated this issue. Koivisto, Lähteenmäki, Kaasinen, Parkkola, and Railo (2014) applied TMS to intraparietal sulcus and demonstrated that TMS in IPS 90 ms post-stimulus impaired the conscious perception of stimulus shape. The results of this thesis showed that TMS in LO suppressed the conscious perception of an object coherency during two separate time windows (at around 120 ms and around 310 ms after the stimulus-onset). This finding is in accordance with several earlier reports which indicate that LO responds more strongly to coherent shapes than to scrambled images (e.g., Grill-Spector et al., 1998, 2000; Malach et al., 1995; Kourtzi & Kanwisher, 2000, 2001; Vanni et al., 1996) and also with reports of patients suffering from visual agnosia due to injury in LO (e.g., Goodale et al., 1991; Heider, 2000; James et al., 2003; Konen et al., 2011; McIntosh et al., 2004). Unfortunately, the roles of LO and other areas in the ventral stream, and especially their role in late latencies, has not been previously studied in detail with brain stimulation methods. In Koivisto, Railo, Revonsuo, et al. (2011), LO was found to be necessary for the conscious perception of natural scenes up to 150 ms, and in Wokke et al. (2013) the LO was necessary to illusory contour up to 100 ms. By observing subjective ratings of feature binding, Koivisto and Silvanto (2012) found that TMS in the angular gyrus impaired conscious feature binding when TMS was applied 180 ms after the stimulus-onset. The result suggests that the angular gyrus is necessary for the visual awareness of feature conjunctions. Future studies should apply brain stimulation to aforementioned areas in the ventral and dorsal stream to study the necessary activation periods of these areas. Optimally, by using the same visual stimulus and applying single pulse TMS to several different cortical areas it might be possible to identify the participation of these areas to the CMVC. However, in the typical current TMS experiment set-ups, TMS is delivered only to one or two region(s) of interest.

In addition, to study the consciousness of visual features by aiming to suppress visual awareness, we observed the qualitative differences of phosphenes generated by the stimulation of V1 or V2. Our objective was to find out whether the specific features of visual sensations correspond with the expected neuronal ensemble of the area. We found that the shapes and colours of phosphenes were highly alike regardless of whether they were induced by V1 or V2 stimulation. The results of Murphey et al. (2009) are in accordance with our finding, as they did not find systematic differences in phosphene features between the stimulation of functionally mapped V1 and V2 or between any other functionally defined area (V3, lateral occipital cortex, V4, V8, MT, parahippocampal place area and fusiform face area). Together our results and the results by Murphey et al. suggest that the neural mechanisms related to phosphene perception could be related to the activation of a widely distributed neural network. Interestingly, Murphey et al. did not find (or did not report) moving phosphenes from the stimulation of V5, which is surprising given that it has been shown in several earlier studies (e.g., Pascual-Leone & Walsh, 2001; Stewart et al., 1999). In contrast to our and Murphey and colleagues' results, Lee et al. (2000) reported that the more complex geometric shapes of phosphenes (e.g., triangles, diamonds) are produced via electrodes placed on the extrastriate cortex and the simpler ones by striate cortex stimulation. Compared to the study by Murphey et al., Lee and colleagues used larger number of electrodes, which might to some extend explain the differences in results. In addition, Lee and colleagues did not map the functional anatomy of the visual cortex, leaving open which visual areas were actually stimulated. All in all, the contradictory findings concerning the relationship between phosphene complexity and different visual areas remains to be solved in future studies.

6.3. Timing of visual awareness as studied with EEG, MEG and single neuron recordings

To study the timing of visual consciousness, we aimed in *Study I* to find out in which time windows the electrophysiological responses to conscious and unconscious stimuli start to diverge from each other. We found that the effects of consciousness emerged as early as 140 ms after the presentation of the visual stimulus. The prominent negativity was observable for about 160 ms. The result converges with several ERP studies also using a masking (Koivisto & Revonsuo, 2007, 2008a; Koivisto et al., 2006; Wilenius-Emet et al., 2004), repetition blindness (Koivisto & Revonsuo, 2008b) or reduced contrast stimuli paradigm (Koivisto et al., 2008; Pins & ffytche, 2003), although the exact onset of negativity varies between the studies depending on the experimental paradigm used (see also Bachmann, 2009). In addition, the MEG studies also support the view that the difference between consciously seen stimuli as compared to the stimuli that remained in the unconscious level is associated with posterior activity between 200–300 ms after the onset of the visual stimulus (Liu et al., 2012; see also Sekar et al., 2013; Vanni et al., 1996).

In contrast to the studies which have reported VAN as the earliest neural correlate of consciousness, there are studies which report the first consciousness related changes in electrophysiological responses as increased positivity in the P1 time window (peaking around 100–130 ms) for subjectively seen stimuli (Pins & Ffytche, 2003). In a later study, Koivisto et al. (2008) aimed to replicate the study by Pins and Ffytche (2003), but they failed to find the consciousness related enhancement for P1. In addition, Wilenius and Revonsuo (2007) also reported enhancement in P1 for consciously seen stimuli. P1 is strengthened by spatial attention and it has been argued that the effect in the studies by Pins and ffytche and Wilenius and Revonsuo is actually related to the fluctuation of spatial attention instead of visual awareness (Koivisto & Revonsuo, 2010; Railo et al., 2011; Wilenius & Revonsuo, 2007). The fact that the majority of studies have not reported any enhancement of P1 for stimuli entering consciousness is in agreement with the view that P1 is not directly related to visual awareness. Given that most of the studies favour the time window in which VAN appears (rather than the earlier time window in which P1 appears) as the time window where the CMVC take place (for review see Koivisto & Revonsuo, 2010), there are good reasons to believe and a lot of converging evidence to support the belief that the electrophysiological wave called P1 does not directly correlate with visual awareness. This would suggest that the P1 would reflect some preceding prerequisite processes of visual consciousness.

Although the majority of published studies on VAN report that VAN and LP occur in tandem, LP following VAN, there are also some studies which report correlation only between LP and visual consciousness, without an observable VAN (Aru & Bachmann, 2009; Babiloni, Vecchio, Miriello, Romani, & Rossini, 2006; Lamy et al., 2009; Salti et al., 2012; van Aalderen-Smeets, Oostenveld, & Schwarzbach, 2006). In accordance with the interpretation that visual consciousness correlates with neural activity in the later time window where LP occurs, Quiroga et al. (2008) found consciousness-related activity in late time windows between 300 and 1000 ms post-stimulus in a study using single-neuron recordings in the human visual area MT. Babiloni et al. (2006) and Lamy et al. (2009) used a masking paradigm and van Aalderen-Smeets et al. (2006) used metacontrast masking. What is common for the studies by Babiloni et al., Lamy et al. and Salti et al. is that the visual stimulus was presented in an unpredictable location and not in the central visual field. Thus, the subjective perception of the visual stimuli was probably considerably coarser in these studies than in the studies where VAN has been observed, and it is possible that the conscious visual perception of the stimulus was simply too weak to produce VAN. It has even been argued that it is not entirely clear whether in the studies by Babiloni et al. and Lamy et al. the participants actually saw the target stimulus or whether they just based their perception on the transient signal (Breitmeyer & Öğmen, 2006; Koivisto & Revonsuo, 2010). Furthermore, Koivisto and Revonsuo (2010) argue that given that in van Aalderen-Smeets et al. (2006) the MEG components purely to the masks were not reported, the results of the study are not reliable. To prove that VAN is *not* the neural correlate of consciousness but instead a byproduct of some other cognitive phenomenon or the result of some features in the experimental set-up, one should conduct a study showing that the difference in ERPs for conscious versus unconscious stimuli would be seen in two different conditions in LP time window whereas VAN would be seen only in one of these two conditions (Koivisto & Revonsuo, 2010). An experimental procedure such as this would demonstrate the cause of VAN if it is not due to consciousness but some other perceptual or cognitive process instead. To the best of my knowledge, so far, such studies are yet to be published. There are, however, studies showing VAN in two conditions but LP (or only a weak LP) in only one condition (Koivisto & Revonsuo, 2007, 2008b; Koivisto et al., 2006; *Study I*). Obviously, the relation between VAN/LP and visual awareness should still be studied further. One possibility is that actually both VAN and LP reflect visual awareness depending on its quality. According to this view, visual stimulus in the central vision or strong/complex stimulus in the peripheral vision would be associated with VAN, whereas coarser stimulus in the peripheral vision would elicit only LP. The differences in the strength of VAN could represent a difference in the intensity of visual awareness. Support for this interpretation comes from the study by Koivisto & Revonsuo (2008b) who demonstrated that VAN is attenuated when the stimuli are presented in the central vision but are not actively attended to (so called passive viewing condition). This hypothesis could be tested in future studies. One should, however, consider the possibility that the earliest neural processes directly related to visual awareness cannot be detected with ERPs but with some other electrophysiological analysis method instead (e.g., oscillations in the EEG). Nevertheless, given that the consciousness related changes in the oscillations in the EEG are observed already in the prestimulus phase (Mathewson et al., 2009; Romei, Brodbeck, et al., 2008; Romei, Rihs, et al., 2008; Wyart & Tallon-Baudry, 2009), it is difficult (without taking advantage of other methods, e.g., TMS) to decide which of these phases would be a direct neural correlate of consciousness.

Consequently, the overwhelming majority of the ERP studies are consistent with the view that VAN is the earliest ERP correlate of visual awareness, suggesting that visual awareness occurs around 200–300 ms after stimulus-onset for high contrast stimuli. While VAN has been linked to phenomenal consciousness, LP has been suggested to be associated with the reflective consciousness (Koivisto & Revonsuo, 2008a; Koivisto et al., 2006). This interpretation is in harmony with the results of *Study I*. However, the finding concerning the timing of visual awareness should be approached with caution as there is still inconsistency in the results of the electrophysiological correlates of visual awareness. Second, as mentioned above, even if there is a tight correlation between visual awareness and specific electrophysiological responses, this does not necessarily imply that those responses are a part of the direct CMVC (Bachmann, 2009).

6.4. Are V1, V2 and LO direct neural correlates of consciousness?

So far, I have discussed the cortical areas necessary for conscious visual perception, but the question remains whether these areas are part of the CMVC or just a part of the preceding neural processes which enable visual awareness at later latencies. As discussed above, electromagnetic sensing of brain activity in humans implies that the emergence of visual consciousness can be observed at latencies around 200–300 ms after stimulus-onset or at later latencies. Concerning the question of whether recurrent processing in V1 or V2 would be part of the CMVC, the results of this thesis were controversial. *Study II* showed that the accuracy of the responses was impaired when the TMS pulse was targeted to V1 180 ms after the onset of the stimulus. In our *Studies III* and *V*, we demonstrated that TMS of V2 wiped out the conscious perception of stimulus presence when the pulse was delivered within about 80 ms after the stimulus-onset, the conscious perception of letter stimulus features was impaired up to 100 ms post-stimulus and the conscious perception of coherent objects up to about 120 ms post-stimulus. Thus, *the TMS-induced suppression occurs in our studies earlier than the awareness related ERP or MEG deflections do*. Consequently, the majority of the studies in this thesis imply that top-down activation to these areas (or the operation of horizontal connections) is not part of the CMVC. Instead, our results support the view that the activation in V2 is a prerequisite for conscious perception at the initial processing stages which precede CMVC but is not alone sufficient for consciousness to emerge.

However, the issue is not as simple as it seems. To the best of my knowledge, there are so far no published studies that would have targeted specifically either V1 or V2 to observe the effects of TMS on visual awareness in the late time windows, but as mentioned above, there are a few studies which have not specifically targeted V1 or V2, suggesting that V2/V1 are necessary for visual perception in the later time window than when the classical 100 ms dip takes place (Camprodon et al., 2010; Juan & Walsh, 2003; Koivisto & Silvanto, 2012; Wokke et al., 2012, 2013). In these studies, the TMS pulse disrupted the subjective perception when the pulse was delivered around 180 ms (Koivisto, Railo, Revonsuo, et al., 2011a) or 240 ms (Koivisto & Silvanto, 2012) after the stimulus-onset. It is not entirely clear whether the 180 ms delay is sufficiently long for the generation of visual consciousness. The study showing visual suppression in the time scale that would more probably correlate with CMVC has used a task which requires feature binding (Koivisto & Silvanto, 2012). Thus, it is conceivable that the necessity of recurrent processing for visual awareness depends on the content of visual awareness. For instance, feature binding is a cognitively more demanding perceptual process than the detection of stimulus presence. Thus, it is possible that when more complex visual stimuli are used, also late top-down processing to V1 or V2 is required. This issue should be clarified in future studies by investigating the roles of V1 and V2 in the late time windows. One problem is that because different stimuli are used in different studies, the studies are seldom directly comparable and only the replication of several earlier studies by, optimally, applying neuronavigated TMS or intracranial recordings with human participants would help to solve the issue.

Unfortunately, as mentioned above there are so far only few published studies that have concentrated on the necessary role of LO, other areas in the ventral stream and prefrontal or parietal cortex in visual awareness at the late time windows. In this thesis, given that interference with the visual input processing in LO at around 310 ms after the onset of the visual stimulus suppressed the conscious perception of coherent object shape, it seems that LO is not only a part of the underlying neural circuits that lead to the phenomenal experience of objects *at the later time intervals*, but in addition *it is a part of the CMVC of the coherency of the objects*. However, it is highly likely that LO is only one part of the wider neural network required for the generation and maintenance of the conscious perception of objects.

7. CONCLUSION

As a summary, the most important findings of the present thesis imply that:

1. visual consciousness and selective attention are initially independent phenomena, and they should be studied as distinct from each other.
2. reliance on purely external anatomical landmarks or MRI is not sufficient in TMS studies if the aim is to determine the functional role of V1 or V2.
3. V1 can be selectively stimulated in a subset of participants, but successful targeting of TMS to V1 requires detailed retinotopic maps of individual functional anatomy combined with computational modelling of the TMS-induced E-field distribution in the visual cortex.
4. as shown earlier for the V1, also the activation in the adjacent area V2 is necessary for conscious visual experience.
5. activation in V2 is necessary for a coarse conscious visual perception for a shorter period of time than for the more detailed visual perception.
6. phosphenes elicited by the TMS of V1 are brighter than identically induced phosphenes arising from the adjacent area V2.
7. the earliest effects of visual consciousness in ERPs can be observed at 140 ms from the stimulus-onset.
8. LO is directly involved in the generation of conscious perception of coherent objects and is thus part of the CMVC.

In this thesis, I aimed to study the phenomenon of visual consciousness and the neural basis of it with several research questions and by using divergent methods, ERPs, fMRI-guided TMS and behavioural reports. I found support for the view that different features of attention contribute differently to visual consciousness, and the theoretical model which is built up of the relationship between visual consciousness and attention should acknowledge these differences. It is likely that the model which has purely two factors, visual consciousness and visual attention, are overly simplified. One possibility that should be acknowledged in future studies is that consciousness might consist of several processing stages which each have their distinct underlying neural mechanisms. The experimental procedures used in this thesis and other empirical research can provide help to dissociate these processing stages from each other.

We also found that the commonly used methodologies in TMS studies in targeting V1 (anatomical landmark method, hunting method, MRI images) that do not use TMS-induced E-field modelling are most probably insufficient to accurately target that area, and if those methods are used it is important to acknowledge that actually V2 is probably investigated in these studies. On the other hand, concerning the neural basis of stimulus presence the results of this thesis suggest that V1 and V2 might be equally important in the generation of visual awareness and that higher cortical areas cannot generate conscious visual perception without the stimulus first being processed in V1 and V2. It is likely that V1 and V2 operate in intensive interaction when generating visual consciousness. Support for this view comes from our finding that top-down activation from V2 to V1 is probably associated with the generation of TMS-induced visual sensation. We also found that V1 and V2 were equally likely to produce phosphenes. According to functional anatomy of V3, it is likely that also V3 is part of the processes generating visual conscious perception, but future studies are required to solve the role of V3 in visual awareness.

Concerning the easy problem of consciousness, although the present results suggest that activation in the ventral stream area LO might be part of the CMVC and thus directly involved in the generation of visual consciousness, these results do not rule out the possibility that activation in the frontal and parietal regions might also be a prerequisite for the generation of conscious perceptions (Dehaene & Changeux, 2011). Neither does this result rule out the possibility that information integration in the thalamocortical system (Tononi, 2004, 2012; see also Edelman, 2003) or visual input processing in a widely distributed neural network (global workspace, Baars, 1988, 1997, 2002; Dehaene & Naccache, 2001) constitutes the basis of visual conscious perception. These questions remain to be addressed in future studies.

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