Sight and blindness in the same person: Gating in the visual system[1]

Hans Strasburger[a] & Bruno Waldvogel[b]

[a]Institute of Medical Psychology, Ludwig Maximilian University, Munich, Germany,
[b]Practice for Psychotherapy, Munich, Germany

Abstract

We present the case of a patient having dissociative identity disorder (DID) who – after 15 years of misdiagnosed cortical blindness – step by step regained sight during psychotherapeutic treatment. At first only a few personality states regained vision whereas others remained blind. This could be confirmed by electrophysiological measurement, in which visual evoked potentials (VEPs) were absent in the blind personality states but were normal and stable in the seeing states. A switch between these states could happen within seconds. We assume a top-down modulation of activity in the primary visual pathway as a neural basis of such psychogenic blindness, possibly at the level of the thalamus. VEPs therefore do not allow separating psychogenic blindness from organic disruption of the visual pathway. In summary, psychogenic blindness seems to suppress visual information at an early neural stage.

Keywords
Dissociative identity disorder, conversion disorders, psychogenic blindness, visual evoked potentials (VEP, VECP), visual pathway, thalamus, LGN

Introduction

Some years ago, BT, a then 33-year old blind patient who was diagnosed as showing dissociative identity disorder (DID; ICD 10: F44.81) was referred from a psychiatric clinic to one of the authors (BW) for psychotherapy. The patient appeared with her guide dog and, with respect to her blindness, reported having suffered an accident thirteen years earlier. This, she reported, first caused severe visual impairment and then, gradually, total blindness. Further information was obtained from a university ophthalmic hospital's expert report, commissioned by the social-assistance authorities after the onset of full blindness.

The expert concluded cortical blindness from craniocerebral trauma. Ocular defects were excluded by a standard exam including slit lamp examination of the anterior ocular segments, ocular fundus inspection (three-mirror contact lens), intraocular tension measurement (applanation tonometer), and Humphrey refractometry (showing 2 dpt myopia). The ocular motor apparatus seemed intact with the eyes moving in parallel and with intact voluntary but no pursuit eye movements present. Acuity as measured by a nystagmus drum (Kotowski ophthalmoscope) was markedly decreased, to 0.1 (OS and OD). All subjective measures, however, showed almost complete blindness: These included laser interference acuity, Goldmann perimetry, and Worth-dot and Bagolini binocular-vision tests. Notable was the total absence of adverse-effects reflexes like watering, winking, or startle response to glare. A residual subjective function was the ability to detect a glaring light close

[1] This paper is a translation from Waldvogel et al. (2007), expanded with current new ideas as to its interpretation.

to the eye, with colored after image, yet without being able to tell the direction of the light source.

The findings from pattern visual evoked potentials (VEPs) were decisive for the diagnosis, however. Despite cooperation (monitored by infra-red fixation control), neither foveal-size (3 deg visual angle), nor perifoveal-size (13 deg) or large-size (30 deg) contrast-reversing checkerboard stimuli evoked detectable potentials. Flash VEPs, in contrast, showed normal amplitude and latency of the P100. This was taken as evidence for lacking visual cortex activity with patterned stimuli but remaining capability of light detection.

Diagnostics

The referral diagnosis of a dissociative identity disorder (then referred to as "multiple personality disorder") soon manifested itself in the psychotherapeutic treatment by spontaneous changes of "identity". By and by, more than ten personalities presented themselves that differed, respectively, by name, reported age, gender, attitudes, personal inclinations, aptitudes, temperament, and other character traits. Voice, gesture, and facial expressions were markedly different. In certain states the patient could communicate in English only, in others in German only, and in still others spoke both languages. In her childhood she had spent some years in an English speaking country and had there spoken English only. These observations concurred with those in the psychiatric clinic where she had been treated prior to the psychotherapy and also with third-party anamnestic reports (patient's friends). All criteria required by the DSM-IV (the applicable version during the treatment) for diagnosing DID were thereby fulfilled.

The term *DID,* compared to the older (and in the ICD-10 still used) term *multiple personality disorder,* is the more precise one to describe the phenomenon and has prevailed in the pertinent literature for many years (Gast, 2004; see Şar, 2014, and Dorahy et al., 2014, for recent reviews). For an operationalised diagnostic, the *Structured Clinical Interview for DSM-IV Dissociative Disorders* (SCID-D) has proved a particularly reliable and valid choice among several screening instruments and interview strategies (Steinberg, 1994; Steinberg, Hall, Craig, & Cichitti, 2004; German translation: Gast, Oswald, & Zündorf, 2000). The operationalised DSM-IV criteria by the SCID-D also led to the diagnosis of DID for our patient.

Therapy and Course of Treatment

In her fourth year of psychotherapy[2] – to which she always appeared with her guide dog – the patient all of a sudden recognized a few individual words on the title page of a magazine in one of her adolescent male identity states, right after one of the therapy sessions. Interestingly, recognition at that stage was limited to whole words and did not involve or allow recognizing the letters that constituted those words. It was further limited to that one personality state. In subsequent sessions, recognition generalized to particularly brightly lit objects and then, quite rapidly, to everything visible. By employing hypnotherapeutic techniques, visual capability could further be generalized to other personality states. For a few months, now, increasingly more sighted personality states coexisted with increasingly fewer states that were totally blind. Sighted and blind states could alternate within seconds.

[2] For psychotherapy of DID in German see Eckhardt-Henn & Hoffmann, 2004, and Reddemann, Hofmann, & Gast, 2004.

Visual Evoked Potentials in a Sighted and Blind State

In that phase when sighted and blind states coexisted, pattern VEPs were acquired to objectify the patient's reports and inquire in how far these reports were accompanied by electrophysiological correlates.

Methods

EEG data were acquired with a standard VEP recording system (Neuropack-2 by Nihon Kohden; electrode impedance < 5 kOhm; bandpass 0.5–100 Hz; 50-Hz notch filter; active electrode at Oz; reference electrode at Fpz). Measurements were performed with binocularly viewed checkerboard stimuli with small and large checks (check size: 0.15° or 1.2° visual angle, corresponding to 2.4 and 0.3 cpd fundamental, respectively; display size: 6.7°×9.3° on a 12" b/w CRT monitor [JVC TM-122] in a darkened room; viewing distance 150 cm; dioptric correction) and time-synchronous averaging of 32 cycles, for both low (1 Hz, transient VEP) and high frequency (10 Hz, steady-state VEP) square-wave modulation. After a recording of two minutes duration there was a five-minute break. During this break, the switch in visual faculty was evoked by her therapist calling on the desired name (in a balanced sequence: sighted, blind, and sighted). Fixation (within about 2°) and blinking, as well as absence of squint, were monitored by an additional observer. Measurements were done at around 6 pm; the patient was awake, motivated, and relaxed. The psychotherapist was present at all times.

Results

Results are shown in Figure 1 and 2. The first series of measurements were in a sighted personality state. VEPs were reliably repeatable, with amplitudes over 10 µV and normal transient latencies of 104–106 ms, for both transient (1 Hz) and steady-state (10 Hz) stimulation (Figure 1a). In the subsequent blind state, in contrast, reliable VEPs were recorded neither with transient stimulation (1 Hz, Fig. 2a) at low spatial frequency, nor with steady-state stimulation (10 Hz, Fig. 2b) at high spatial frequency.

Discussion

Psychogenic loss of conscious visual perception, i.e., a loss without obvious organic defect, was described in the 19th century by Charcot (1872), Janet (1893), and by Freud (1910) (see Stone et al., 2005, for an overview). Since we first reported on BT (Waldvogel et al., 2007), one other similar case has been reported (Bhuvaneswar & Spiegel, 2013). Schoenfeld et al. (2011) studied neural mechanisms of hysterical partial blindness (in two visual-field quadrants) before and after psychotherapy. The present case is remarkable because psychogenic blindness was restricted to certain dissociated personality states and could be abolished immediately by a switch to an alternate personality. Equally remarkable, however, is – after many years of total blindness with all signs of an organic origin – the sudden onset of partial vision, and subsequently the rapid alternation between apparent total blindness and almost normal vision.

This capability of a rapid alternation excludes any explanation based on neural restitution of a central nervous lesion of the visual pathway. Even though plasticity of visual brain function long after the time of lesion is no longer called into question on principle, both spontaneous recovery and success of a neuro-rehabilitative training are tedious, tenacious processes with ever so small improvements over time (e.g. Kasten, Wüst, Behrens-Baumann, & Sabel, 1998; Poggel, Treutwein, Sabel, & Strasburger, 2015). Spontaneous recovery, moreover, typically happens right after the time of lesion (training success, in contrast, seems to be independent

of time-after-lesion; Poggel, Mueller, Kasten, & Sabel, 2008). A reorganization of synaptic connections after craniocerebral injury as a basis for the regained eyesight is thus highly unlikely. Rather we assume that the craniocerebral injury and a thereby caused temporary impairment of vision likely acted as a prime for a psychogenic blindness.

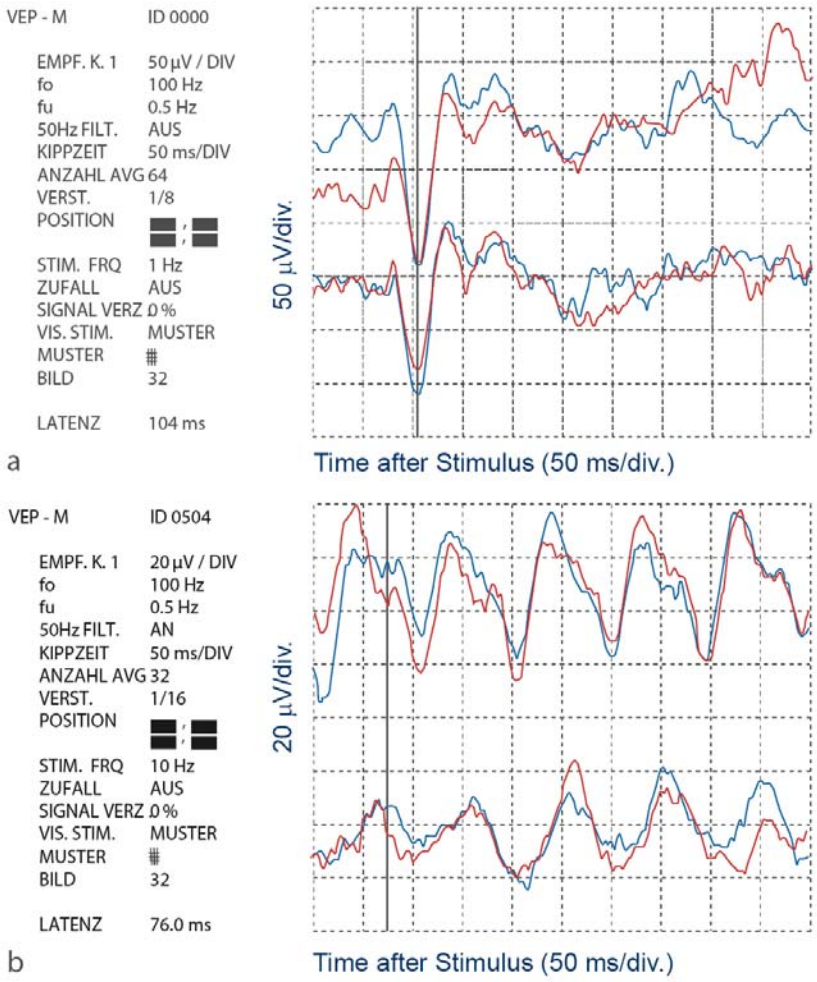

Figure 1. Pattern-evoked potentials in a sighted personality state, with (a) transient, 1-Hz stimulation, and (b) steady-state, 10-Hz stimulation. Traces are color coded for better visibility.

The phenomenon of a temporary suppression of the afferent neural signal in the healthy human that is reversible at any time is more common than it would first appear. Images of the two eyes that contradict each other temporarily, e.g. when squinting or with dichoptic stimulation, lead to a transient, partial or full, suppression of one eye's image. A mild suppression occurs already in the case of a pronounced ocular dominance. Visual information is also modulated – enhanced or suppressed – by spatial selective attention. So brain mechanisms *are* present that allow modulation of the incoming information, acting on either the entire visual field or some parts thereof.

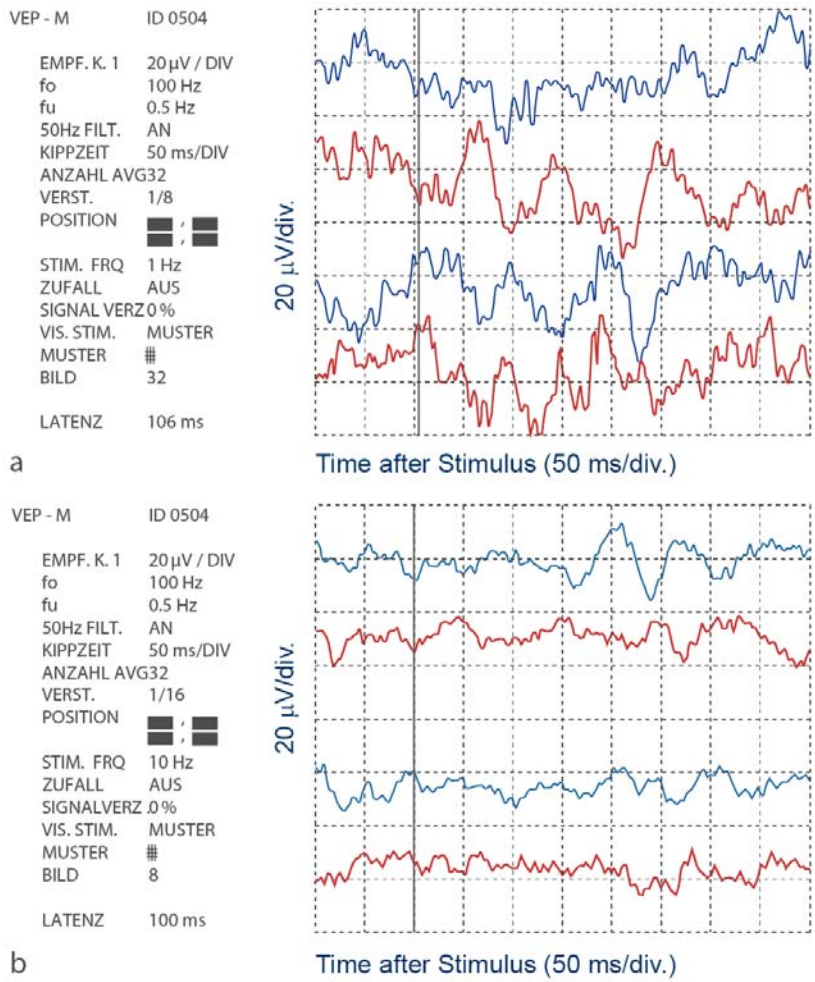

Figure 2. Pattern-evoked potentials in a "blind" personality state, with (a) transient, 1-Hz stimulation, and (b) steady-state, 10-Hz stimulation. Terms as in Figure 1.

Where in the brain that is happening is the subject of current research. Likely sites would be the lateral geniculate body of the thalamus (LGN; often referred to as a "gateway to the cortex") or cortical areas. Indeed, Wunderlich et al. (2005) and Haynes et al. (2005) independently showed eye-specific modulation of neural activity in the human LGN during binocular rivalry by fMRI. Both author groups concluded that the LGN plays a central role in eye-specific dominance and suppression. Wunderlich et al. (2005) suggested that the LGN may act as an early gatekeeper of visual awareness.

A further example for a modulation of the incoming visual information is selective spatial attention (see Gazzaniga, 1999; Chalupa & Werner, 2004, Carrasco, 2011, for reviews). The concept dates back to the 19th century; Johannes Müller in 1825 explained that fixation and spatial attention can be decoupled and Hermann Helmholtz (1871) showed it experimentally (cf. Strasburger, Rentschler, & Jüttner, 2011). Attentional modulation has been shown to happen in cortical areas (Moran & Desimone, 1985; Hillyard, Vogel, & Luck, 1998; Luck & Hillyard, 1999), and was long-time assumed to be restricted to the cortical level (Mehta, Ulbert, & Schroeder, 2000). More recent work, however, has shown modulation could occur earlier in the pathway. O'Connor et al. (2002) showed by fMRI that LGN activity was increased by attended, and decreased by neglected stimuli. Schneider & Kastner (2009) corroborated and extended these findings. This led Kastner & Pinsk (2004) and Saalmann &

Kastner (2009) to suggest that, while neural mechanisms of selective attention operate at multiple stages in the visual system, the earliest of these is the LGN, the role of which is that of an early gatekeeper in controlling neural gain. The modulatory input to the LGN may come from the pulvinar and the thalamic reticular nucleus (TRN) (Saalmann & Kastner, 2009; see also Scolari, Seidl-Rathkopf, & Kastner, 2015), a notion originally proposed by Crick (1984).

The VEP, both in its clinically customary paradigm with checkerboard stimuli or in the paradigm that is more prevailing in basic research with sine wave stimuli of defined spatial frequency (Strasburger, Murray, & Remky, 1993) has long been considered a reliable indicator of the intactness of the primary, retino-cortical pathway. VEPs are abnormal in the presence of any lesion along the anterior visual pathway (Halliday, 1982; Behbehani, 2007) and are used forensically to identify malingering. Area V1, specifically its part within the calcarine fissure, is widely considered to be the origin of the VEPs early N75 and the prominent P100 component (Di Russo et al., 2005). Classical VEP studies in cases of psychogenic blindness have hitherto shown normal pattern VEPs (Halliday, 1982; Altenmüller, Diener, & Dichgans, 1989). So, alternative reasons for the absent or very low VEP in our case need to be considered. The most common behavioral conditions that can typically attenuate electrophysiological visually evoked responses, notably inadequate compliance and vigilance and eccentric fixation, were not apparent with BT as assessed by an outside observer. An also likely source, insufficient fixation, is not an overly critical factor since large stimuli were used (note that larger stimuli do not necessarily lead to a larger P100, probably due to signal cancellation. Furthermore, the preceding expert investigation that was reported in the case history used infrared fixation control so that imprecise fixation, though certainly present, appears unlikely to be the cause of the absent VEP. Defocusing (inaccurate accommodation), however, is a further potential factor that is not reliably visible to an outside observer. In BT's everyday life, her pupils seemed somewhat dilated in her blind state, probably due to anxiety with increased sympathetic activity, which would slightly increase the effect of bad accommodation. The normal flash VEP in the very first exam would conform to heavy defocus. Such defocusing, however, would need to exceed an estimated 10 dpt for the large check size, however, or would have differential effect on high vs. low spatial frequencies, which was not the case. So, except for uncertainties with respect to the role of accommodation, the finding of an absent VEP in the blind state is strong evidence that the incoming neural information is blocked in some way in the primary pathway, i.e. in LGN or V1. In particular the spontaneous switching between states – within seconds – supports such an interpretation. It might be mentioned that the VEP does not require conscious perception or sustained attention since a VEP can be also recorded in an anesthetized state (Pang & Bonds, 1991). Also, the modulation being an effect of hypnosis is very unlikely. In a study by Spiegel et al. (1985) P100 amplitude was reduced but not abolished under hypnosis (Spiegel, Cutcomb, Ren, & Pribram, 1985).

The time course of BT's recovery of vision in her sighted personality states (prior to our examinations) is of interest in the light of our findings. Recovery seems in some respect to have progressed from higher to lower levels of visual function. BT recognized complete words before she recognized the constituent letters, and as her vision in her seeing states improved there was a progression from reading large high-contrast text, to recognizing the colors of words, low contrast print, and inverted contrast patterns. Depth perception and vision/motor coordination were still by no means normal in the seeing states – the patient often bumped into objects or missed an object when reaching for it. The recovery pattern

for reading is commensurate with research showing that the letter recognition is neither necessary nor sufficient for word recognition (Kennedy, Radach, Heller, & Pynte, 2000). Recognizing single letters is mostly a task of pattern recognition (cf. Strasburger et al., 2011) whereas word recognition involves language skills (cf. Nazir & Huckauf, 2008).

In addition to its implications for the brain's ability to control the inflow of visual information, the present case bears on discussions of the ontology of dissociative identity disorders. In the literature on consciousness and the nature of the self, dissociative identity disorder has been taken as important evidence for the formulation of a scientific theory of the self (Humphrey & Dennett, 1989; Velleman, 2005). However, questions regarding the validity of the phenomenon have complicated the picture. From the onset, the nosological description of dissociative identity disorders has been accompanied by an ongoing controversy about whether this disorder might be a cultural and therapeutic artifact (Ross, 2006). Recent psychobiological evidence has shown that different personality states are correlated with differing cortical activation patterns (e.g. Hopper et al., 2002; Reinders et al., 2003; Reinders et al., 2006, Schlumpf et al., 2013) and has demonstrated neural correlates of switching between personality states (Tsai, Condie, Wu, & Chang, 1999; Savoy, Frederick, Keuroghlian, & Wolk, 2012, Wolk, Savoy, & Frederick, 2012; see Şar, 2014 and Dorahy et al., 2014, for review). Yet that would still be compatible with a skeptical view that personality states are just metaphors reflecting differences in higher-level cognitive processing (Merckelbach, Devilly, & Rassin, 2002), or viewpoints that personality states result from therapeutic suggestions or elaborate forms of role playing (Deeley, 2003; Piper & Merskey, 2004). The case of BT contributes to this controversy by demonstrating that differences between personality states are not limited to higher level processing but can differ with respect to the fundamental processing of early sensory information and corresponding perceptual change. It therefore provides compelling evidence for the existence of the dissociated identities in a more biological sense.

Acknowledgements

A. Ullrich kindly performed the VEP acquisition. We thank Nick Wade for language corrections and Uwe Mattler, Dorothe Poggel, Peter Dechent, Richard Fendrich, and Torsten Wüstenberg for comments on, and contributions to, a related manuscript (in preparation) on fMRI results for the same patient. Michael Bach pointed out the possible involvement of visual accommodation.

References

Altenmüller, E., Diener, H. C., & Dichgans, J. (1989). Visuell evozierte Potentiale [Visual evoked potentials]. In M. Stöhr, J. Dichgans, H. C. Diener & U. W. Büttner (Eds.), *Evozierte Potentiale. 2nd edition* (pp. 279-382). Berlin, Heidelberg, New York: Springer.

Behbehani, R. (2007). Clinical approach to optic neuropathies. *Clinical Ophthalmology (Auckland, N.Z.), 1*(3), 233–246.

Bhuvaneswar, C., & Spiegel, D. (2013). An Eye for an I: A 35-year-old woman with fluctuating oculomotor deficits and dissociative identity disorder. *International Journal of Clinical and Experimental Hypnosis, 61:3*, 351-370.

Carrasco, M. (2011). Visual attention: the past 25 years. *Vision Research, 51*(13), 1484-1525.

Chalupa, L. M., & Werner, J. S. (Eds.). (2004). *The visual neurosciences* (Vol. 2). Cambridge, Mass., London, England: MIT Press.

Charcot, J. M. (1872). *Leçons sur les maladies du système nerveux faites à la sal-pêtrière [Lessons on diseases of the nervous system made at the Salpêtrière]*. Paris: A. Delahaye.

Crick, F. (1984). Function of the thalamic reticular complex: the searchlight hypothesis. *Proceedings of the National Academy of Sciences, USA, 81*(14), 4586-4590.

Deeley, P. Q. (2003). Social, cognitive, and neural constraints on subjectivity and agency: implications for dissociative identity disorder. *Philosophy, Psychiatry and Psychology, 10*(2), 161-167.

Di Russo, F., Pitzalis, S., Spitoni, G., Aprile, T., Patri, F., Spinelli, D., et al. (2005). Identification of the neural sources of the pattern-reversal VEP. *NeuroImage, 24*(3), 874– 886.

Dorahy, M. J., Brand, B. L., Şar, V., Krüger, C., Stavropoulos, P., Martínez-Taboas, A., et al. (2014). Dissociative identity disorder: An empirical overview. *Australian & New Zealand Journal of Psychiatry, 48*(5), 402–417.

Eckhardt-Henn, A., & Hoffmann, S. O. (Eds.). (2004). *Dissoziative Bewußtseinsstörungen [Dissociative disorders of consciousness]*. Stuttgart, New York: Schattauer.

Freud, S. (1910). Die psychogene Sehstörung in psychoanalytischer Auffassung [The psycho-analytic view of psychogenic disturbance of vision]. In A. Freud, M. Bonaparte, E. Bibring, W. Hoffer, K. E & O. Osakower (Eds.), *Gesammelte Werke VIII [Collected Works VII]* (pp. 94-102). Frankfurt: Fischer.

Gast, U. (2004). Dissoziative Identitätsstörung – valides und dennoch reformbedürftiges Konzept [Dissociative Identity Disorder - Valid concept, yet in need of reform]. In L. Reddemann, A. Hofmann & U. Gast (Eds.), *Psychotherapie der dissoziativen Störungen [Psychotherapy of dissociative disorders]* (pp. 26-36). Stuttgart, New York: Georg Thieme Verlag.

Gast, U., Oswald, T., & Zündorf, F. (Eds.). (2000). *Das Strukturierte Klinische Interview für DSM-IV Dissoziative Störungen (SKID-D). Deutsche Fassung [The Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D)]*. Göttingen: Hogrefe.

Gazzaniga, M. S. (1999). The New Cognitive Neurosciences, Chpt. V: Attention. In M. S. Gazzaniga (Ed.), (2nd ed., pp. 619-724). Cambridge, MA: Bradford Book, MIT Press.

Halliday, A. M. (Ed.). (1982). *Evoked potentials in clinical testing, p. 575*. Edinburgh: Churchill Livingstone.

Haynes, J., Deichmann, R., & Rees, G. (2005). Eye-specific effects of binocular rivalry in the human lateral geniculate nucleus. *Nature, 438*(7067), 496-499.

Helmholtz, H. (1871). Über die Zeit, welche nöthig ist, damit ein Gesichtseindruck zum Bewusstsein kommt [On the time required for a visual impression to come into consciousness]. *Monatsberichte der Königlichen Preußischen Akademie der Wissenschaften zu Berlin [Monthly reports of the Royal Prussian Academy of Sciences, Berlin], Juni*, 333-337.

Hillyard, S. A., Vogel, E. K., & Luck, S. J. (1998). Sensory gain control (amplification) as a mechanism of selective attention: electrophysiological and neuroimaging evidence. *Philosophical Transactions of the Royal Society of London B, Biological Sciences, 353*(353), 1257-1270.

Hopper, A., Ciorciari, J., Johnson, G., Spensley, J., Sergejew, A., & Stough, C. (2002). EEG coherence and dissociative identity disorder. *J Trauma Dissociation, 3*(1), 75-88.

Humphrey, N., & Dennett, D. C. (1989). Speaking for ourselves. *Raritan, 9*(1), 68-98.

Janet, P. M. F. (1893). *L'État mental des hystériques [The mental state of the hysterical]* (Vol. vol. I. Les stigmates mentaux [Vol. I. Mental stigmata]). Paris: Rueff et Cie.

Kasten, E., Wüst, S., Behrens-Baumann, W., & Sabel, B. A. (1998). Computer-based training for the treatment of partial blindness. *Nature Medicine, 4*(9), 1083–1087.

Kastner, S., & Pinsk, M. A. (2004). Visual attention as a multilevel selection process. *Cognitive, Affective, & Behavioral Neuroscience, 4*(4), 483-500.

Kennedy, A., Radach, R., Heller, D., & Pynte, J. (Eds.). (2000). *Reading as a perceptual process*: North Holland.

Luck, S. J., & Hillyard, S. A. (1999). The operation of selective attention at multiple stages of processing: Evidence from human and monkey electrophysiology. In M. S. Gazzaniga (Ed.), *The New Cognitive Neurosciences* (2nd ed., pp. 687-700). Cambridge, MA: Bradford Book, MIT Press.

Mehta, A. D., Ulbert, I., & Schroeder, C. E. (2000). Intermodal selective attention in monkeys. I: distribution and timing of effects across visual areas. *Cereb Cortex, 10*(4), 343–358.

Merckelbach, H., Devilly, G. J., & Rassin, E. (2002). Alters in dissociative identity disorder: Metaphors or genuine entities? *Current Psychiatry Report, 22*(4), 481-497.

Moran, J., & Desimone, R. (1985). Selective attention gates visual processing in the extrastriate cortex. *Science, 229*(4715), 782-784.

Nazir, T. A., & Huckauf, A. (2008). The visual skill "reading". In E. L. Grigorenko & A. J. Naples (Eds.), *Single word reading : Cognitive, Behavioral and Biological Perspectives*. New York: Lawrence Erlbaum (LEA Books).

O'Connor, D. H., Fukui, M. M., Pinsk, M. A., & Kastner, S. (2002). Attention modulates responses in the human lateral geniculate nucleus. *Nature Neuroscience, 5*(11), 1203-1209.

Pang, X. D., & Bonds, A. B. (1991). Visual evoked potential responses of the anesthetized cat to contrast modulation of grating patterns. *Vision Research, 31*(9), 1509-1516.

Piper, A., & Merskey, H. (2004). The persistence of folly: critical examination of dissociative identity disorder. Part II. The defence and decline of multiple personality or dissociative identity disorder. *Canadian Journal of Psychiatry, 49*(10), 678--683.

Poggel, D. A., Mueller, I., Kasten, E., & Sabel, B. A. (2008). Multifactorial predictors and outcome variables of vision restoration training in patients with post-geniculate visual field loss. *Restorative Neurology and Neuroscience, 26*(4,5), 321–339.

Poggel, D. A., Treutwein, B., Sabel, B. A., & Strasburger, H. (2015). A matter of time: Improvement of visual temporal processing during training-induced restoration of light detection performance. *Frontiers in Psychology, section Perception Science*, 6:22.

Reddemann, L., Hofmann, A., & Gast, U. (Eds.). (2004). *Psychotherapie der dissoziativen Störungen [Psychotherapy of dissociative disorders]*. Stuttgart, New York: Georg Thieme Verlag.

Reinders, A. A. T. S., Nijenhuis, E. R. S., Paans, A. M. J., Korf, J., Willemsen, A. T. M., & den Boer, J. A. (2003). One brain, two selves. *NeuroImage, 20*(4), 2119-2125.

Reinders, A. A. T. S., Nijenhuis, E. R. S., Quak, J., Korf, J., Haaksma, J., Paans, A. M. J., et al. (2006). Psychobiological characteristics of Dissociative Identity Disorder. A symptom provocation study. *Biological Psychiatry, 60*, 730-740.

Ross, C. A. (2006). Dissociative Identity Disorder. *Current Psychosis & Therapeutics Reports, 4*, 112-116.

Saalmann, Y. B., & Kastner, S. (2009). Gain control in the visual thalamus during perception and cognition. *Current Opinion in Neurobiology, 19*(4), 1-7.

Şar, V. (2014). The Many Faces of Dissociation: Opportunities for Innovative Research in Psychiatry. Review. *Clinical Psychopharmacology and Neuroscience, 12*(3), 171-179.

Savoy, R. L., Frederick, B. B., Keuroghlian, A. S., & Wolk, P. C. (2012). Voluntary switching between identities in dissociative identity disorder: A functional MRI case study. *Cogn. Neurosci., 3*(2), 112-119.

Schlumpf, Y. R., Nijenhuis, E. R. S., Chalavi, S., Weder, E. V., Zimmermann, E., Luechinger, R., et al. (2013). Dissociative partdependent biopsychosocial reactions to backward masked

angry and neutral faces: An fMRI study of dissociative identity disorder. *Neuroimage Clin*(3), 54-64.

Schneider, K. A., & Kastner, S. (2009). Effects of sustained spatial attention in the human lateral geniculate nucleus and superior colliculus. *J. Neurosci., 29*(6), 1784–1795.

Schoenfeld, M. A., Hassa, T., Hopf, J.-M., Eulitz, C., & Schmidt, R. (2011). Neural Correlates of Hysterical Blindness. *Cerebral Cortex, 21*(October), 2394--2398.

Scolari, M., Seidl-Rathkopf, K. N., & Kastner, S. (2015). Functions of the human frontoparietal attention network: Evidence from neuroimaging. *Current Opinion in Behavioral Sciences, 1*, 32–39.

Spiegel, D., Cutcomb, S., Ren, C., & Pribram, K. (1985). Hypnotic hallucination alters evoked potentials. *Journal of Abnormal Psychology, 94*(3), 249-255.

Steinberg, M. (1994). *The structured clinical interview for DSM-IV-dissociative disorders – revised (SCID-D)*. Washington, DC: American Psychiatric Press.

Steinberg, M., Hall, P., Craig, L., & Cichitti, D. (2004). Diagnostik valider und vorgetäuschter Dissoziation mit dem strukturellen klinischen Interview für dissoziative Störungen SCID-D-R – Richtlinien für klinische und forensische Untersuchungen [Diagnosis of valid and fake dissociation with the Structural Clinical Interview for Dissociative Disorders SCID-DR – guidelines for clinical and forensic investigations]. In L. Reddemann, A. Hofmann & U. Gast (Eds.), *Psychotherapie der dissoziativen Störungen [Psychotherapy of dissociative disorders]* (pp. 151–167). Stuttgart, New York: Georg Thieme Verlag.

Stone, J., Smyth, R., Carson, A., Lewis, S., Prescott, R., Warlow, C., et al. (2005). Systematic review of misdiagnosis of conversion symptoms and ‘‘hysteria’’. *BMJ*, 331:989.

Strasburger, H., Murray, I., & Remky, A. (1993). Sustained and transient mechanisms in the steady-state visual evoked potential. Onset presentation compared to pattern reversal. *Clin. Vision Sci., 8*(3), 211-234.

Strasburger, H., Rentschler, I., & Jüttner, M. (2011). Peripheral vision and pattern recognition: a review. *Journal of Vision, 11*(5), 1-82.

Tsai, G. E., Condie, D., Wu, M. T., & Chang, I. W. (1999). Functional magnetic resonance imaging of personality switches in a woman with dissociative identity disorder. *Harv Rev Psychiatry, 7*(2), 119-122.

Velleman, J. D. (2005). The self as narrator. In *Autonomy and the Challenges to Liberalism: New Essays* (pp. 56-76). Cambridge: Cambridge University Press.

Waldvogel, B., Ullrich, A., & Strasburger, H. (2007). Blind und sehend in einer Person. Schlußfolgerungen zur Psychoneurobiologie des Sehens [Sighted and blind in one person. A case report and conclusions on the psychoneurobiology of vision]. *Der Nervenarzt, 78*(11), 1303-1309.

Wolk, P. C., Savoy, R. L., & Frederick, B. B. (2012). The neural correlates of vertical splitting in a single case study. *Neuropsychoanalysis, 14*(2), 157-163.

Wunderlich, K., Schneider, K. A., & Kastner, S. (2005). Neural correlates of binocular rivalry in the human lateral geniculate nucleus. *Nature Neuroscience, 8*(11), 1595–1602.