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Visual emotion

From input to behavioral output

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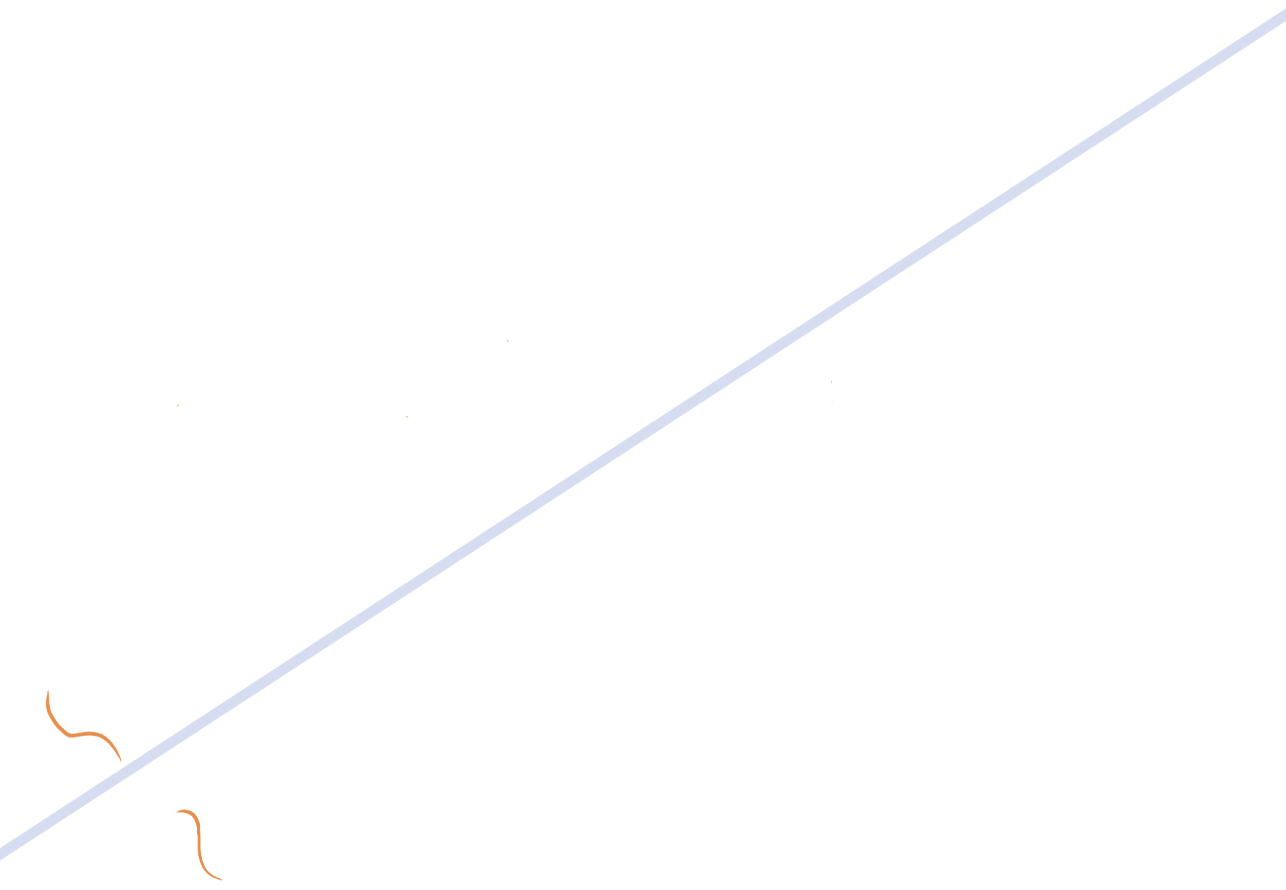
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General introduction



General introduction

Patients with brain damage may encounter social, emotional and (neuro)-psychological problems in daily life. Examples of neuropsychological consequences after brain damage include language or memory problems.¹ These language or memory problems are relatively distinct and readily recognized by patients and their families. It is obvious that a person who is suddenly, after an incident, unable to express him or herself verbally is suffering from a serious deficit. In addition, patients may have deficits in other neuropsychological domains, which may not so easily be noticed. Examples of these ‘invisible’ consequences include deficits in visual functions (such as the perception of shape or motion) or deficits in the ability to perceive facial emotional expressions. Despite their apparent ‘invisibility’ for the outside world, these deficits can have adverse behavioral consequences^{2,3} and they are related to a reduced quality of life.³ The current dissertation was set out to investigate these various visual functions and their interrelationships in brain-damaged patients. The studies in this dissertation are part of a large multi-center cohort study, aimed at assessing the Functional Architecture of the Brain for Vision (FAB4V).

Visual perception

The investigation of the visual brain originated with electrophysiological and tracing examinations of animals^{4,5} and was refined with the emergence of functional neuroimaging techniques, such as functional Magnetic Resonance Imaging (fMRI). These neuroimaging techniques allowed the examination of visual functions in healthy human subjects.⁶ The available evidence has shown that roughly a quarter of the brain is dedicated to the perception and understanding of the visual world. In addition, the physiological studies have shown the presence of over forty retinotopic maps in the posterior brain.⁶⁻⁸ The neurons in these maps are tuned to various aspects of the visual world, such as shape, orientation and motion.⁵ However, despite the large number of studies on the visual brain, there is still debate about the functional organization of the different visual primitives. Overall, there are two conflicting views.

The first view argues that the different retinotopic maps are hierarchically organized into a limited number of pathways. An example is the two-visual pathway model, which claims that there are two major pathways: a ventral pathway and a dorsal pathway, both starting in the primary visual cortex.⁹ The ventral pathway runs

towards the mesial temporal lobe and is used for the recognition of, for instance, text or faces. The dorsal pathway runs towards the frontal lobe and is related to the spatial aspects of the outside world and is used for action planning. This model furthermore assumes that the complexity of visual information within each pathway increases in a systematic manner. Therefore, according to this model, lower-order visual functions (e.g. brightness perception) or mid-range visual functions (e.g. shape or texture perception) are expected to precede higher-order visual cognitive functions, such as object recognition and facial emotion recognition.

The second view assumes a more parallel organization of the different visual functions with many interconnections between the different routes.^{10,11} This view has been summarized in the 'patchwork model', proposed by De Haan and Cowey (2011).¹² This patchwork model of the visual brain assumes the existence of many overlapping networks with different nodes for the different visual features. The investigation whether the visual system is better described in terms of the two-pathway model or the patchwork model is a central question in the FAB4V-study.

Still, it is clear that a large amount of the brain is dedicated to visual perceptual functions. Therefore, it is not surprising that deficits in visual perception have been found in up to sixty percent of the patients who suffered brain damage.^{13,14} Examples of these visual deficits include visual field problems, blurred vision, color perception problems and visuo-constructional and visuospatial problems.^{13,14} Deficits in these visual perceptual functions can negatively influence driving behavior and have been found to be predictive of the chronic functional outcome after brain damage.¹⁵

Visual emotion perception

The perception of emotional facial expressions can be seen as a higher-order cognitive function comprising advanced processing of visual information. Emotion perception is a key component of social cognition, which involves the functions that enable us to behave adequately in social situations.¹⁶ Other aspects of social cognition include the understanding of behavior and intentions of others and empathic behavior.¹⁷ With regard to the perception of emotions, six basic emotions can be discerned: anger, disgust, fear, happiness, surprise and sadness.¹⁸

With respect to the neural underpinnings of facial emotion recognition, the fronto-temporal brain networks are known to play an important role.¹⁹ However, with regard to the specific neural correlates of emotion recognition, previous studies have presented conflicting information. For example, it is still unclear whether

there is a lateralization effect. Some studies indicated that particularly the right hemisphere is important in guiding emotion recognition,²⁰⁻²² while others did not show evidence for emotion recognition to be lateralized.²³ In addition, there is still debate about whether there are separable networks responsible for the recognition of the six basic emotions (anger, disgust, happiness, fear, sadness and surprise) or whether there is a general 'emotion perception network'. The existence of a common emotion perception network was suggested by a meta-analysis,²⁴ while in contrast, two other meta-analyses argued that there are distinct networks for the six basic emotions.^{19,25}

In the last decades, an increasing amount of research has investigated deficits in emotion recognition and its behavioral consequences in patients with various neurological diseases. For instance, Yuvaraj et al. (2013) found that emotion recognition deficits in stroke patients were related to deficits in interpersonal behavior.²² Furthermore, in patients with a subarachnoid hemorrhage, deficits in emotion recognition were found to be related to apathy and to an impaired self-awareness.²⁶ In addition, in patients with TBI, it was found that a worse ability to recognize emotions was related to a lower amount of independent activities²⁷ and to increased risk-taking behavior.²⁸ Lastly, in patients with neurodegenerative diseases, deficits in emotion recognition were found to be related to apathy²⁹ and to impaired decision-making behavior.³⁰ In sum, studies in various neurological patient groups have indicated that deficits in emotion recognition occur frequently and that these deficits can underlie disturbances on a behavioral level.

Visual risk-assessment

Another social cognitive function is decision making in potentially risky situations, which is known to be guided by emotional processes.³¹ According to the somatic marker hypothesis, decisions are guided by emotion-related bodily sensations ('markers').³¹ These markers are based on previous experiences, where a particular event becomes associated with a particular emotion. Encountering this event a next time will unconsciously provoke this emotion, which will in turn influence the decision to be made.

Patients with damage in the frontal lobe frequently show problems in daily-life decision-making behavior, leading to risky situations.³² Although many studies have indicated a specific role of the ventromedial prefrontal cortex in decision-making behavior in hazardous situations,³³ other studies have shown that other brain structures can be involved in this type of decision-making as well. These

structures include other frontal areas such as the dorsolateral prefrontal cortex, but also temporal³⁴ and cerebellar areas.³⁵ The presence of increased risk-taking behavior can have severe consequences for a safe daily-life functioning, especially in traffic situations. For instance, it has been found that impaired decision-making behavior in patients can be predictive of a decreased driving performance and that they are accident prone.³⁶

Stroke

Stroke is a common neurological condition. It is one of the major causes of acquired chronic disability and the third largest cause of death in the Netherlands.³⁷⁻³⁹ There are two types of strokes: hemorrhagic stroke and ischemic stroke. A hemorrhagic stroke occurs when a blood vessel ruptures. This puts pressure on the brain, which subsequently leads to a loss of blood to the adjacent brain areas. The majority of strokes (87%), however, involves ischemic strokes.^{40,41} During an ischemic stroke, the blood supply to a part of the brain is blocked by a thrombus or blood clot. This results in a lack of oxygen in the affected brain region of blood supply. This causes symptoms such as a paralyzed arm or confused speech, depending on the particular brain region. Brain regions supplied by the middle cerebral artery are the most commonly affected areas by a stroke.⁴² The most common risk factors for stroke include smoking, hypertension and diabetes.^{37,39} The recommended treatment options for patients with an acute ischemic stroke involve intravenous thrombolysis with alteplase or mechanical thrombectomy with the aim of breaking down or removing the clot that is blocking the blood flow.⁴³⁻⁴⁵ The application of these treatment options has significantly reduced the mortality rate after ischemic stroke.^{45,46} Nevertheless, because of the higher survival rate, there is also a larger number of patients with chronic, functional complaints. In particular, in addition to the physical consequences of a stroke, patients may face a number of social and (neuro)psychological problems in everyday life, including impairments in visual functions and emotion perception, as described earlier. These neuropsychological deficits can in turn have a great impact on the quality of life in brain damaged patients.

Neurodegenerative diseases

A neurodegenerative disease (NDD) is the condition in which neurons in the central nervous system are progressively degenerating.⁴⁷ NDD's are an increasingly common cause of mortality and morbidity.⁴⁸ Frequently encountered NDD's include, amongst

others, Alzheimer's Disease, Frontotemporal Dementia and Huntington's Disease. Many NDD's share several clinical features, such as disturbances in memory or executive functioning. In addition to cognitive decline, patients with an NDD may show behavioral or personality changes.⁴⁹ In recent years, accumulating evidence has shown that deficits in social cognitive functions, such as deficits in facial emotion recognition, may underlie these behavioral changes in these patients.⁵⁰

General aim and outline of the thesis

The main objective of this dissertation is to investigate the prerequisites for and consequences of deficits in visual emotion recognition in brain damaged patients. The ability to adequately perceive facial emotional expressions could be seen as an intermediate stage in the entire process between the visual 'input' and the behavioral 'output'. Problems at the 'input' phase, such as deficits in mid-range visual functions (e.g. shape, motion or texture), could possibly influence the perception of emotional expressions. An impaired emotion perception could, in turn, interfere with adequate higher-order social cognitive behavior, such as risk-taking behavior, of which it is known to be guided by emotional processes.³¹

In **Chapter 1**, a general introduction to the subject is given. **Chapter 2** presents the results of a study in which a new diagnostic set-up is used to investigate the prevalence and co-occurrence of specific 'mid-range' visual functions, in a large cohort of stroke patients. **Chapter 3** describes the possible knock-on effects of the different 'mid-range' visual functions on higher-order visual cognitive functions, including visual emotion perception, visuo-construction and visual memory in a group of stroke patients. In **Chapter 4**, an intriguing case of a patient with a visual-perceptual disorder is described. This patient perceived the outside world at about seventy percent of the actual size, after a right occipito-parietal stroke. **Chapter 5** zooms in on emotion recognition in stroke patients and its neural underpinnings. **Chapter 6** presents the results of a study on the consequences of cerebellar stroke for emotion recognition and risk-taking behavior, including the relationship between these constructs. In **Chapter 7**, the relationship between emotion recognition and risk-taking behavior is further investigated, in a patient group with neurodegenerative damage to frontal-subcortical circuits, which are known to be involved in emotion recognition. **Chapter 8** provides a general discussion of the preceding chapters with overall conclusions and implications of the findings.

References

- 1 Nys GM., van Zandvoort MJ., van der Worp H., *et al.* Early cognitive impairment predicts long-term depressive symptoms and quality of life after stroke. *J Neurol Sci* 2006; 247: 149–56.
- 2 Lai SM, Studenski S, Duncan PW, Perera S. Persisting consequences of stroke measured by the stroke impact scale. *Stroke* 2002; 33: 1840–4.
- 3 Cooper CL, Phillips LH, Johnston M, Radlak B, Hamilton S, McLeod MJ. Links between emotion perception and social participation restriction following stroke. *Brain Inj* 2014; 28: 122–6.
- 4 Hubel DH, Wiesel TN. Receptive fields of single neurones in the cat's striate cortex. *J Physiol* 1959; 148: 574–91.
- 5 Livingstone M, Hubel D. Segregation of form, color, movement, and depth: anatomy, physiology, and perception. *Science* 1988; 240: 740–9.
- 6 Larsson J, Heeger DJ. Two retinotopic visual areas in human lateral occipital cortex. *J Neurosci* 2006; 26: 13128–42.
- 7 Dougherty RF, Koch VM, Brewer AA, Fischer B, Modersitzki J, Wandell BA. Visual field representations and locations of visual areas V1/2/3 in human visual cortex. *J Vis* 2003; 3: 1.
- 8 Van Essen DC. Functional organization of primate visual cortex. *Cereb Cortex* 1985; 259–329.
- 9 Goodale MA, Milner AD. Separate visual pathways for perception and action. *Trends Neurosci* 1992; 15: 20–5.
- 10 Gomez J, Pestilli F, Witthoft N, *et al.* Functionally Defined White Matter Reveals Segregated Pathways in Human Ventral Temporal Cortex Associated with Category-Specific Processing. *Neuron* 2015; 85: 216–27.
- 11 Kravitz DJ, Saleem KS, Baker IB, Mishkin M. A new neural framework for visuospatial processing. *Nat Rev Neurosci* 2011; 12: 217–30.
- 12 de Haan EHF, Cowey A. On the usefulness of 'what' and 'where' pathways in vision. *Trends Cogn Sci* 2011; 15: 460–6.
- 13 Rowe FJ. Visual perceptual consequences of stroke. *Strabismus* 2009; 17: 24–8.
- 14 Neumann G, Schaadt A-K, Reinhart S, Kerkhoff G. Clinical and Psychometric Evaluations of the Cerebral Vision Screening Questionnaire in 461 Nonaphasic Individuals Poststroke. *Neurorehabil Neural Repair* 2016; 30: 187–98.

- 15 Nys GMS, Van Zandvoort MJE, De Kort PLM, *et al.* The prognostic value of domain-specific cognitive abilities in acute first-ever stroke. *Neurology* 2005; 64: 821–7.
- 16 Adolphs R. Neural systems for recognizing emotion. *Curr Opin Neurobiol* 2002; 12: 169–77.
- 17 Borod JC. *The Neuropsychology of Emotion*. New York: Oxford University Press, 2000.
- 18 Ekman P. An Argument for Basic Emotions. *Cogn Emot* 1992; 6: 169–200.
- 19 Fusar-Poli P, Placentino A, Carletti F, *et al.* Functional atlas of emotional faces processing: a voxel-based meta-analysis of 105 functional magnetic resonance imaging studies. *J Psychiatry Neurosci* 2009; 34: 418–32.
- 20 Adolphs R, Damasio H, Tranel D, Cooper G, Damasio AR. A Role for Somatosensory Cortices in the Visual Recognition of Emotion as Revealed by Three-Dimensional Lesion Mapping. *J Neurosci* 2000; 20: 2683–90.
- 21 Philippi CL, Mehta S, Grabowski T, Adolphs R, Rudrauf D. Damage to association fiber tracts impairs recognition of the facial expression of emotion. *J Neurosci* 2009; 29: 15089–99.
- 22 Yuvaraj R, Murugappan M, Norlinah MI, Sundaraj K, Khairiyah M. Review of emotion recognition in stroke patients. *Dement Geriatr Cogn Disord* 2013; 36: 179–96.
- 23 Campanella F, Shallice T, Ius T, Fabbro F, Skrap M. Impact of brain tumour location on emotion and personality: a voxel-based lesion–symptom mapping study on mentalization processes. *Brain* 2014; 137: 2532–45.
- 24 Lindquist KA, Wager TD, Kober H, Bliss-Moreau E, Barrett LF. The brain basis of emotion: A meta-analytic review. *Behav Brain Sci* 2012; 35: 121–202.
- 25 Vytal K, Hamann S. Neuroimaging support for discrete neural correlates of basic emotions: A voxel-based meta-analysis. *J Cogn Neurosci* 2010; 22: 2864–85.
- 26 Buunk AM, Spikman JM, Veenstra WS, *et al.* Social cognition impairments after aneurysmal subarachnoid haemorrhage: Associations with deficits in interpersonal behaviour, apathy, and impaired self-awareness. *Neuropsychologia* 2017; 103: 131–9.
- 27 May M, Milders M, Downey B, *et al.* Social Behavior and Impairments in Social Cognition Following Traumatic Brain Injury. *J Int Neuropsychol Soc* 2017; 23: 400–11.

- 28 Visser-Keizer AC, Westerhof-Evers HJ, Gerritsen MJJ, Van der Naalt J, Spikman JM. To fear is to gain? The role of fear recognition in risky decision making in TBI patients and healthy controls. *PLoS One* 2016; 11: 1–16.
- 29 Robert G, Le Jeune F, Dondaine T, *et al.* Apathy and impaired emotional facial recognition networks overlap in Parkinson's disease: A PET study with conjunction analyses. *J Neurol Neurosurg Psychiatry* 2014; 85: 1153–8.
- 30 Ibarretxe-Bilbao N, Junque C, Tolosa E, *et al.* Neuroanatomical correlates of impaired decision-making and facial emotion recognition in early Parkinson's disease. *Eur J Neurosci* 2009; 30: 1162–71.
- 31 Damasio AR, Tranel D, Damasio HC. Somatic markers and the guidance of behavior: Theory and preliminary testing. In: *Frontal Lobe Function and Dysfunction*. New York: Oxford University Press, 1991: 217–29.
- 32 MacPherson SE, Phillips LH, Della Sala S, Cantagallo A. Iowa gambling task impairment is not specific to ventromedial prefrontal lesions. *Clin Neuropsychol* 2009; 23: 510–22.
- 33 Clark L, Bechara A, Damasio H, Aitken MRF, Sahakian BJ, Robbins TW. Differential effects of insular and ventromedial prefrontal cortex lesions on risky decision-making. *Brain* 2008; 131: 1311–22.
- 34 Von Siebenthal Z, Boucher O, Rouleau I, Lassonde M, Lepore F, Nguyen DK. Decision-making impairments following insular and medial temporal lobe resection for drug-resistant epilepsy. *Soc Cogn Affect Neurosci* 2017; 12: 128–37.
- 35 Schmahmann JD. From movement to thought: Anatomic substrates of the cerebellar contribution to cognitive processing. *Hum Brain Mapp* 1996; 4: 174–98.
- 36 Motta K, Lee H, Falkmer T. Post-stroke driving: Examining the effect of executive dysfunction. *J Safety Res* 2014; 49: 33–8.
- 37 Tsendsuren S, Li CS, Liu CC. Incidence and Risk Factors for Stroke among 14 European Countries. *Int J Aging Hum Dev* 2016; 84: 66–87.
- 38 Thrift AG, Dewey HM, Macdonell RAL, McNeil JJ, Donnan GA. Stroke incidence on the east coast of Australia: The North East Melbourne Stroke Incidence Study (NEMESIS). *Stroke* 2000; 31: 2087–92.
- 39 Struijs JN, Van Genugten MLL, Evers SMAA, Ament AJHA, Baan CA, Van Den Bos GAM. Modeling the future burden of stroke in the Netherlands: Impact of aging, smoking, and hypertension. *Stroke* 2005; 36: 1648–55.
- 40 Unibaso-Markaida I, Iraurgi I, Ortiz-Marqués N, Martínez-Rodríguez

- S. Degree of functionality and perception of health-related quality of life in people with moderate stroke: Differences between ischemic and hemorrhagic typology. *Behav Neurol* 2019; 2019.
- 41 Benjamin EJ, Blaha MJ, Chiuve SE, *et al.* Heart Disease and Stroke Statistics—2017 Update: A Report From the American Heart Association. *Circulation* 2017; 135: e146–603.
- 42 Rorden C, Karnath HO. Using human brain lesions to infer function: A relic from a past era in the fMRI age? *Nat Rev Neurosci* 2004; 5: 812–9.
- 43 Hacke W, Kaste M, Bluhmki E, *et al.* Thrombolysis with alteplase 3 to 4.5 hours after acute ischemic stroke. *N Engl J Med* 2008; 359: 1317–29.
- 44 Luan D, Zhang Y, Yang Q, *et al.* Efficacy and safety of intravenous thrombolysis in patients with unknown onset stroke: A meta-analysis. *Behav Neurol* 2019; 2019.
- 45 Goyal M, Menon BK, Van Zwam WH, *et al.* Endovascular thrombectomy after large-vessel ischaemic stroke: A meta-analysis of individual patient data from five randomised trials. *Lancet* 2016; 387: 1723–31.
- 46 Muret W, Rudd A, Wolfe CDA, Douiri A. Long-Term Survival After Intravenous Thrombolysis for Ischemic Stroke. *Stroke* 2018; 49: 607–13.
- 47 Amor S, Puentes F, Baker D, Van Der Valk P. Inflammation in neurodegenerative diseases. *Immunology*. 2010; 129: 154–69.
- 48 Erkinen MG, Kim M, Geschwind MD. Clinical Neurology and Epidemiology of the Major Neurodegenerative Diseases. *Cold Spring Harb Perspect Biol* 2018; 10.
- 49 Desmarais P, Lanctôt KL, Masellis M, Black SE, Herrmann N. Social inappropriateness in neurodegenerative disorders. *Int Psychogeriatrics* 2018; 30: 197–207.
- 50 Kumfor F, Irish M, Leyton C, *et al.* Tracking the progression of social cognition in neurodegenerative disorders. *J Neurol Neurosurg Psychiatry* 2014; 85: 1076–83.