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"Neural correlates of (visual) consciousness on different spatial and temporal scales; Approaches to evaluate the causal contribution of brain regions and coding principles for conscious perception"

(Report by Hiroaki Hamada & Jelle Bruineberg)

Finding the Neural Correlates of Consciousness by Professor Melanie Wilke

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Introduction

What is the relationship between consciousness and the brain? One way of understanding this relationship has been by looking at neural correlates of conscious experience in human and animals. Prof. Wilke gave an overview of experimental studies covering enabling factors, actual substrates, and relevant impairments of visual consciousness, and she also discussed some intrinsic difficulties of studies in the neural correlates of consciousness (NCC).

Section 1: Enabling factors (Prerequisites)

First, Professor Wilke talked about those factors that enable conscious perception but are not themselves involved in conscious processing. These include some basic conditions such as general alertness, being presented with some sensory input (i.e. not being in a completely dark room), and an intact transmission of sensory information to early visual areas. For further enabling factors, it is first necessary to take a closer look at consciousness itself. First, one can distinguish between different levels of consciousness, based on ocular, verbal and motor (re)actions. Second, behavioral observation suggests that there are two dimensions to consciousness: arousal and awareness. Dissociations between these two can be found in for example vegatative state and minimally conscious (MC) state patients. Further neural enabling factors can be found through the analysis of lesions of patients in a vegetative state, with locked-in syndrome or in a MC state.

Particularly, an important sysmte here might be the Ascending Reticular Activating System, a structure in the brainstem. Lesions in this region interfere with the level of consciousness. For example, locked-in syndrome patients typically have a lesion between the reticular formation and the pons (the pontine basis). Lesions of the upper brainstem may lead to vegetative and MC states. One cortical region that seems to correlate with the level of consciousness is the precuneus and the adjacent posterior cingulate cortex, these regions show most activity in conscious activity and virtually no activity in patients in a vegetative state (Laureys et al., 2004).

In practice, it can be very difficult to distinguish between a patient in a vegetative state and a patient in a MC state, since both show no motor output. One particularly fascinating methodology for assessing minimal consciousness is presented by Owen et al. (2008). They asked a patient to think about two different activities (for instance, playing tennis and opening a door) when wanting to respond “yes” and “no” respectively to a question. Using fMRI, they found differentiated brain activity for both answers showing that the patient understood and could respond to asked questions, and hence indicating it being conscious. Further research with Deep Brain Stimulation suggests that, at least in some cases, the level of consciousness can be improved in MC-state patients by stimulation of the thalamus (Schiff et al. 2007).

Summarizing, besides some more basic conditions, neural enabling factors for consciousness seem to be active nuclei in the reticular formation and brain stem and cholinergic release in the cortico-thalamic complex.

Section 2: Actual Substrates (Content)

Although the abovementioned processes are important, they are generally not considered to be parts of the NCC in a narrow sense. The NCC is supposed to pin down the minimal neural mechanisms that are sufficient for any one conscious percept under constant background conditions (Koch, 2004) and are therefore more related to the *content* of consciousness. One inherent difficult in the study of the NCC is the unreliability of the relations between 1.) the awareness (or phenomenal) state and a behavioral indication and 2.) the brain state and the measure of the brain activity. No-report paradigms (see Nao Tsuchiya's lecture) and improved neuroscientific methods might overcome these difficulties.

The general logic of the study of the NCC is to have a paradigm in which subjects report whether they saw a given stimulus or not (either a masked stimulus, a visual illusion etc.). The difference in brain activity between consciously perceived and not perceived stimuli could then be an indication of the NCC. Presumed actual correlates can be both on the scale of neural firing and local neural oscillations, as well as more global processes of coherency and synchronicity as suggested by Global Workspace Theory (Dehaene & Changeux, 2011)

Section 3: Consequences (Cognitive/Motor Output)

Third, the intrinsic functions of visual awareness were discussed with disorders of visual perception, such as V1-lesion and neglect syndrome. V1 lesioned people, who are blind but have the ability to report visual stimuli, have been discovered. Such ability is called as blindsight. Humphrey et al. reported that a V1 lesioned monkey, called Helen, is able to avoid obstacles during free moving without visual perception (Humprey et al., 1970). In line with this, blindsight subjects are found to have dissociation between vision and action, and the LGN-pulvinar pathway might enable the patients to implement actions without visual perception (Cowey, 2010; Schmid et al., 2012). Furthermore, as another example of disorders of conscious vision, Prof. Wilke raised neglect syndrome. Neglect symptoms are characterized by biased posture and loss of ipsilesional exploration. Etiology of neglect symptoms has been studied. Based on several studies, the hemispheric imbalance model is proposed as a neglect model (Corbetta et al., 2005). The model suggests that ipsilesional exploration bias is caused by over-activation of the contralateral hemisphere due to damages of the ipsilateral hemisphere. Consistent with the model, inactivation of the lateral intraparietal area (LIP) caused a loss of saccades toward cotralesional space, while inactivation of the medial intraparietal sulcus (MIP) induced a reduction of amplitude of reaching but no effect on saccades (Wilke et al., 2012; Hwang et al., 2012). A function of the dorsal pulvinar nucleus is also studied, and both reaching and saccades are found to be biased toward ipsilesional space (Wilke et al., 2010). However, whether these inactivations cause perceptual deficits is still unclear.

Student discussion and Conclusion

In the student discussions, we discussed central problems in studies of the NCC. One concern was that the concept of an NCC already makes rather neuro-centric assumptions without explicitly defending them. Another group discussed the correct spatial/temporal scale to look for the NCC is discussed. This group concluded that the

appropriate temporal scale might depend differ with different sensory systems, while the spatial scale is dependent on the size of an animals' nervous system. A lot of the groups were struggling with and discussing the rather abstract presentation of IIT the day before and this more concrete example of the neurobiology of consciousness. Questions were raised such as: how would phi change with particular lesions in the brain that experimentally reduce the level of consciousness?

Overall, Prof. Wilke gave us a great and thought-provoking summary of the neuroscientific study of consciousness. We are looking forward to progress in studies of the NCC and integrity with theory of consciousness such as IIT.

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