fMRI: From Mapping to Mechanisms

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In the late 19th century the early investigations of brain function were dominated by the concept of functional segregation. This approach was driven largely by the data available to scientists of that era. Patients with circumscribed lesions were found who were impaired in one particular ability while other abilities remained largely intact. Functional brain imaging avoids many of the problems of lesion studies, but, here too, the field has been dominated by the doctrine of functional segregation. Nevertheless, it is implicit in the subtraction method that brain regions communicate with each other. Only in the mid nineties, the technique has shown its usefulness in revealing mechanisms of brain function. The latter is achieved by suitable experimental designs and novel multivariate analysis techniques that allow the estimation of functional interactions between brain areas. With respect to experimental design, many studies have used fMRI to probe the response characteristics of a certain cortical or subcortical area, rather than to try and identify areas which are activated by a certain cognitive component. In short, the field has witnessed a transition from asking the question 'where' to asking the question 'how'.

In a series of behavioral, fMRI and electroencephalography (EEG) experiments we examined the influence of cognitive load on the processing of irrelevant visual background objects. In particular, we addressed two questions: (i) is this modulation dependent on regional activity (i.e. phasic)? (ii) At what processing stage does this modulation take place? Cognitive load was manipulated by a working memory (WM) task and concurrently the processing of irrelevant visual objects was assessed with fMRI and EEG. To examine the dependency of this modulation on intrinsic activity, we varied the activity level of visual areas by presenting objects with different levels of degradation. Activity in the lateral occipital complex (LOC) increased with object visibility and was phasically modulated by WM load. Event related potentials revealed that this phasic modulation occurred ~170 ms after stimulus onset, indicative of an early selection under high load. In summary, this experiment revealed the mechanism behind attentional modulation of visual processing by working memory, namely a phasic modulation of activity in the ventral visual pathway (LOC). In addition combining fMRI with EEG allowed us to precisely estimate the time point at which this modulation occurs.

Classically, neuroscience is divided into different topics like "attention", "learning" or "emotional processing". Sometimes it appears as if this distinction is carved in stone and different topics are studied independent of each other. But this is not necessarily the case. In the second part of the talk I will show examples in which we deliberately studied the combination of two different neuroscientific topics namely "attention" and "pain", using fMRI. In the first part of the study, we were interested how concomitantly applied painful stimuli can modulate visual perception by using a similar experimental design as described above, when we investigated the modulatory effect of working memory on visual perception. We found a similar pattern, namely a modulation of LOC responses by increasing pain intensity. In addition, we also investigated the reverse namely the effect cognition has on pain processing. This was studied by investigating the effect of placebo analgesia, i.e. volunteers were stimulated with a laser pain but beforehand either treated with a local analgesic or a control cream. In reality, both the control and the analgesic creams were identical and did not contain any analgesic compound, yet still we observed a marked decrease in perceived pain intensity when subjects were told that their skin was treated with the "analgesic cream". FMRI revealed a prominent role of the rostral anterior cingulate cortex (rACC) in mediating this effect, more precisely, the coupling between the rACC and the amygdala and between rACC and the PAG were modulated by the placebo effect, showing (i) that cognition can have a strong influence on pain perception and (ii) how this mechanism is implemented in the human brain.