EEG measures the difference in voltage, or potential, between two electrodes. In contrast, MEG measures the absolute magnitude of the magnetic field and does not require a reference. Both EEG and MEG measure brain activity from the surface; from the scalp and outside the skull, respectively. To localize brain activity from the recorded electric and magnetic fields, the so-called inverse problem must be solved. That is, to make a reasonable guess about current sources and their locations based on measurements from the surface. Unfortunately, theory shows that there is no unique solution to this problem. In additon, EEG is affected by the conductivities of the skull and scalp much more than MEG. Therefore, interpretation of EEG signals, in particular for source localization, will require more precise knowledge of the thickness and conductivities of the tissues in the head. Consequently, MEG has a better spatial resolution (Fig. 1). Under favorable conditions the source location can be determined with a precision of a few millimeters (Hari, 1988).

MEG is completely noninvasive and can record physiological signals in the order of milliseconds. Thus, it is possible to follow the rapid neuronal changes in the brain. MEG also provides direct measure of neuronal activity, whereas other functional brain imaging techniques, i.e. single-photon-emission computed tomography (SPECT), positron-emission tomography (PET), and functional magnetic resonance imaging (fMRI), measure hemodynamic response, and so, provide an indirect measure of neuronal activity. The time resolution, therefore, is much better in MEG than in those methods (Fig. 1).

The first measurement of magnetic field in human brain was carried out at the Massachusetts Institute of Technology by David Cohen (1972). He measured the spontaneous activity of a healthy subject and the abnormal brain

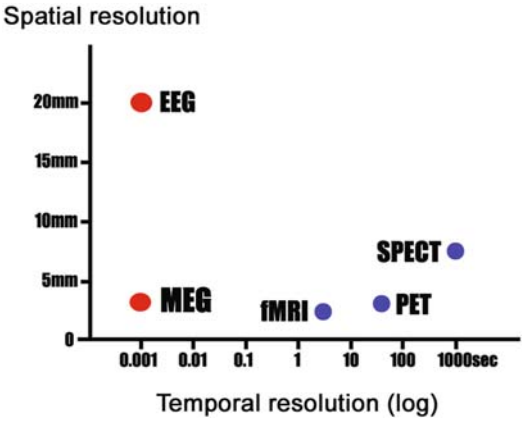

Fig. 1 Temporal and spatial resolution of brain imaging techniques
EEG Electroencephalography; MEG Magnetoencephalography; SPECT Single-photon-emission computed tomography; PET Positron-emission tomography; fMRI Functional magnetic resonance imaging

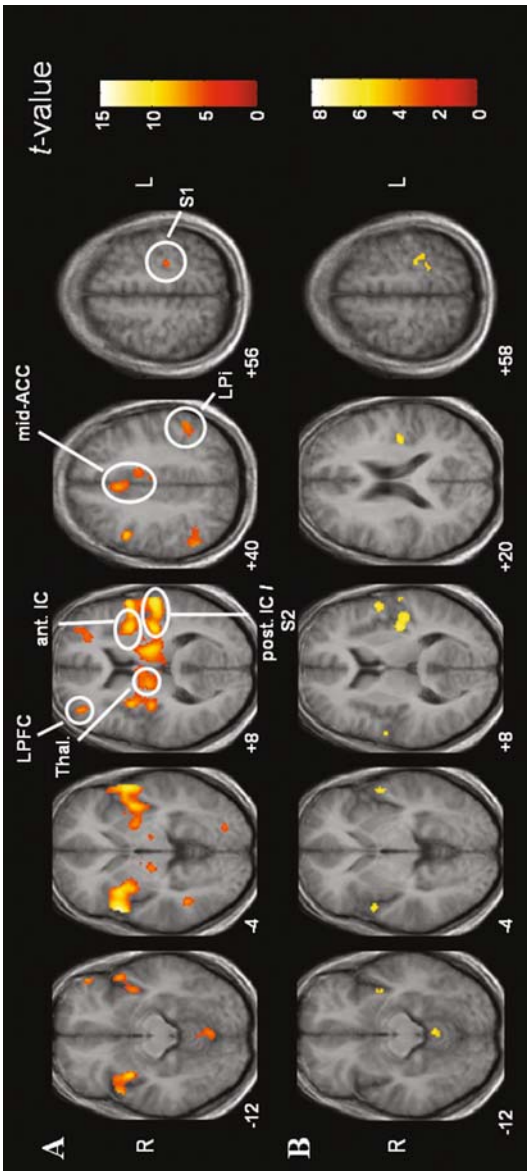

Fig. 2 Effects of distraction on cerebral pain processing. (**A**) Without distraction heat stimulation activates the sensory-discriminative pain coding system (e.g.: S1, S2, lateral thalamus, posterior insular cortex) and affective-motivational system (medial thalamus, ACC, anterior insular cortex). (**B**) With distraction, induced by the Stroop-task, the same noxious stimulation is no longer able to activate the former pain network. Image right is brain left. (From Valet et al., 2004; reprinted with permission. Pain 109, 399–408)

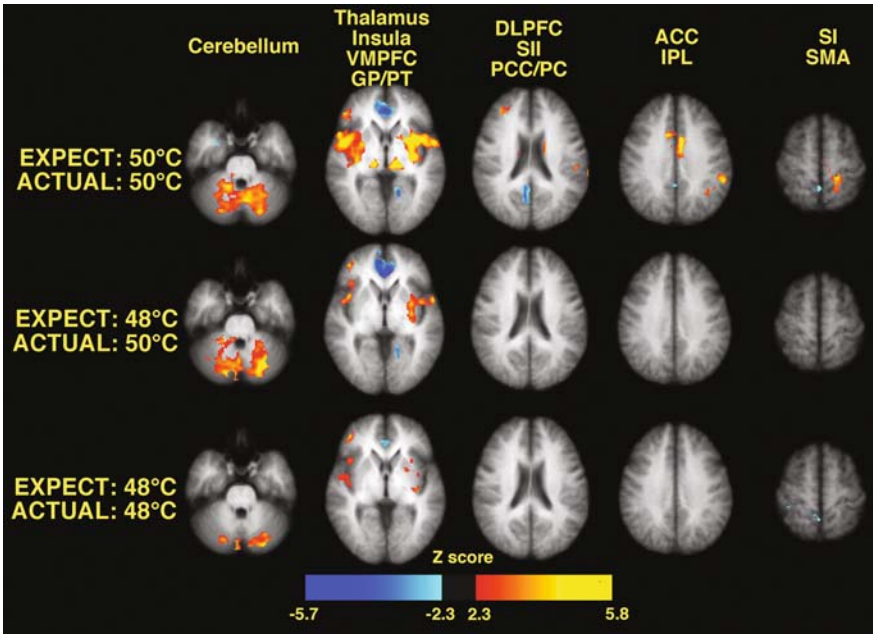

Fig. 3 Expectations for decreased pain significantly reduce pain-related brain activation during 50°C stimulation. Image right is brain left. (From Koyama et al., 2005; Copyright (2005) National Academy of Sciences, U.S.A)

area. Rostral ACC portions tended to have greater expectation-related activation, and caudal portions tended to have greater pain-intensity-related activation. When subjects expected a 48°C stimulus, but received a 50°C stimulus, no detectable activation was found in ACC and S1 (Fig. 3, middle row), when compared to a correctly signaled 50°C stimulus (Fig. 3, upper row). The degree of activation in the insula, dorsolateral prefrontal cortex, S2 and other areas were also significantly reduced. In fact, after decreased expectations of pain the pain-intensity-related activation closely resembled that evoked by correctly signaled 48°C stimuli (Fig. 3, bottom row).

Taken together, ACC, S1 and S2 likely represent critical pathways for the integration of expectation-related information with afferent sensory information (Koyama et al., 2005). Connections exist between ACC/insula and S1/S2, thus, all of these cortical areas receiving afferent nociceptive information can be modulated by expectation-induced information. There is emerging, although still incomplete evidence that at least part of the expectancy effect is mediated by descending pain modulatory circuits (Matre et al., 2006; Wager et al., 2006).

Emotion. Emotion may be defined as a physiological state in which an intense affective experience is accompanied by physiological reactions to the inciting event (eg: laughter, crying, preparations to attack or flee, etc.). Emotions such as fear and anxiety have divergent effects on human pain thresholds;

component of pain) there is a correlation in ACC activity (area 24) accompanied by reduced pain unpleasantness ratings while pain intensity ratings is unchanged (Rainville et al., 1997). This study was the first to provide direct evidence of a specific encoding for pain unpleasantness in the ACC. In another study by the same group, suggestions were given to reduce pain intensity (Hofbauer et al., 2001). This time pain intensity ratings were accompanied by changes in S1 activity without changing ACC activity.

Faymonville and colleagues, using hypnosis and PET, confirmed the role of ACC (area 24) in encoding pain affect and showed that it also plays a role in encoding pain intensity (Faymonville et al., 2000). This has later been confirmed (Büchel et al., 2002). A subsequent study, by the same authors, used a functional connectivity approach to demonstrate that area 24 modulates a large cortical and subcortical network. Compared to normal alertness (rest and mental imagery), the hypnotic state enhanced the functional modulation between midcingulate cortex (area 24) and bilateral insula, pregenual anterior cingulate cortex, pre-supplementary motor area, right prefrontal cortex and striatum, thalamus and brainstem (Faymonville et al., 2003), see Fig. 4. Two recent connectivity analyses also confirms that the rostral ACC is capable of activating caudal regions involved in nociceptive modulation, such as PAG and nucleus cuneiformis (Bingel et al., 2006; Wager, Scott, & Zubieta, 2007).

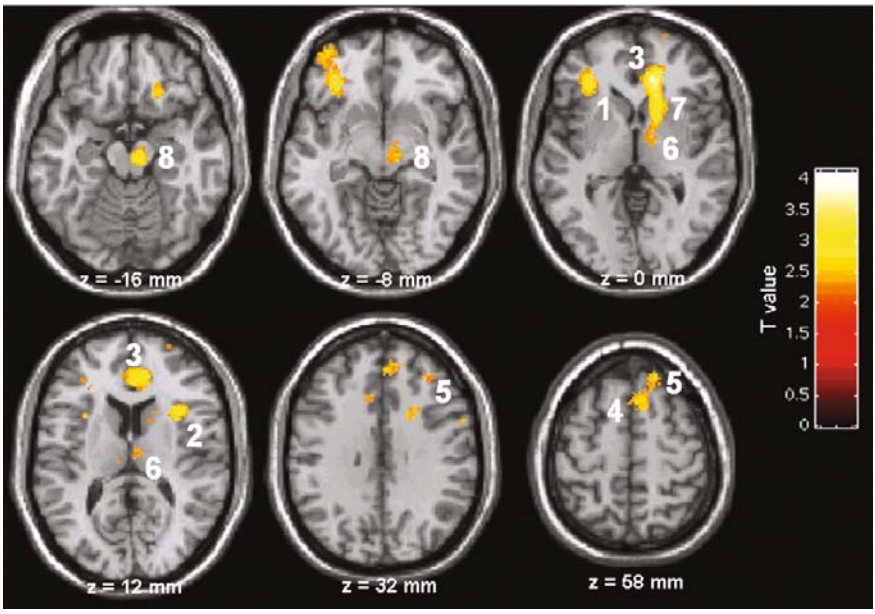

Fig. 4 Regions that showed an increased functional connectivity with midcingulate cortex in hypnosis relative to normal alertness rest and mental imagery. 1,2: Insula, 3: pregenual cortex, 4: Pre-supplementary motor area, 5: superior frontal gyrus, 6: Thalamus, 7: Caudate nucleus, 8: Midbrain/brainstem. (From Faymonville et al., 2003; reprinted with permission)

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