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The adaptive brain: A neurophysiological perspective

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ABSTRACT

When an individual is learning a new skill, recovering from a brain damage, or participating in an intervention program, plastic changes take place in the brain. However, brain plasticity, intensively studied in animals, is not readily accessible in humans to whom invasive research methods cannot be applied without valid clinical or therapeutic reasons. Animal models, in turn, do not provide information about higher mental functions like language or music. Evoked neural responses have shed new light to the mechanisms underlying learning and recovery, however. Of particular interest are those higher order neural responses that can be recorded even with absence of attention, such as the mismatch negativity (MMN) and N1. They enable one to determine plastic neural changes even in patients who are unable to communicate and in infants learning a language.

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1. Introduction

Intensive research during the last decades has changed our view of the brain's capacity to adapt to new or special circumstances. By now, it has been proven that in its most extreme forms, brain plasticity may even extend from one sensory

Abbreviations: CI, cochlear implant; EEG, electroencephalography; EMF, event-related magnetic field; ERP, event-related brain potential; MEG, magnetoencephalography; MMN, mismatch negativity; MMNm, magnetic mismatch negativity.

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system to another (for reviews, see Rauschecker, 2002; Kujala et al., 2000; Bavelier and Neville, 2002). In this case, the cortical areas deprived of input due to a complete damage to sensory receptors (e.g., peripheral blindness or deafness) leads to the responsiveness of these areas to the input originally received by the other sensory receptors. Reorganization taking place within the different modalities has been even more intensively investigated, however. For example, the amputation of a finger does not leave the corresponding brain representation area "silent". Instead, this area will process input from the neighbouring fingers, which is reflected as an increased responsiveness of the neurons in this area to the stimulation of the adjacent fingers (Merzenich et al., 1984). Analogous phenomena were also demonstrated in the visual (Kaas et al., 1990) and auditory modalities (Robertson and Irvine, 1989). Furthermore, intensive sensory training, too, causes plastic cortical changes. For example, sound-frequency discrimination training increases the cortical representation area of the corresponding frequencies (Recanzone et al., 1993). In this case, a larger number of neurons become responsive to the trained frequencies.

Our understanding of the cortical-map plasticity within the sensory systems is largely based on animal models (for a review, see Kaas, 2001) even though some of these phenomena, such as training-induced enlargement of a representation area or the spreading of adjacent representations to an area deprived of input, were demonstrated in humans, too (Pascual-Leone and Torres, 1993; Rossini et al., 1994; Jäncke et al., 2001; Weiss et al., 2004). Research on animal neurophysiology has given the basis for understanding physiological changes underlying learning. At the cellular lever, a corner stone of learning is long-term potentiation (LTP). LTP is an enhancement of synaptic transmission, which may last from hours to lifetime (Kaas, 2001). LTP is suggested to have a role in experience-dependent plasticity because of the strong correlation between critical periods for LTP induction and naturally occurring plasticity. Furthermore, procedures disrupting LTP also disrupt experience-dependent plasticity. LTP appears to behave according to the principles suggested by Hebb (1949): synapse is strengthened if the pre- and postsynaptic cells fire in synchrony. Learning affects the receptive-field size of neurons (Jenkins et al., 1990; Recanzone et al., 1993) as well as the width of cortical columns (Recanzone et al., 1992) and representational areas (Recanzone et al., 1992), that is, the number of neurons driven by the stimuli.

However, animal models are not fully informative on plasticity associated with higher order cognitive functions, like neural reorganization caused by the acquisition of language or musical skills, which are inherently human phenomena. Although some authors see strong parallels between human and animal languages (Rauschecker and Scott, 2009) and the underlying neuronal substrates do indeed share some of the relevant structures, it is clear that the degree of complexity reached by the human language (numerous phonemes, ten thousands of words, flexible syntactic rules) goes beyond the capacities of non-human primates, and it is noteworthy that this difference may have a correlate in the underlying neuroanatomical substrate (Rilling et al., 2008).

1.1. Involuntarily elicited neurophysiological responses

A feasible approach to the dynamics of plastic brain changes in humans is to record stimulus- or event-locked synchronous activity of neural populations giving rise to neural evoked responses (or event-related brain potentials, ERPs; and event-related magnetic fields, EMFs; Hari et al., 2000), which permit one to address stimulus processing with a millisecond's temporal accuracy. With these responses, stimulus reception and discrimination as well as stimulus recognition can be studied (Näätänen, 1990, 1992). Some of these responses can be elicited even involuntarily, irrespective of the individual's primary task or direction of attention. Such responses are of particular significance when one is interested in brain functions of individuals who cannot adequately communicate, like severely aphasic patients, sleeping individuals, or infants.

Neural stimulus reception can be addressed by recording stimulus-elicited P1, N1, and P2 responses of which the N1 response has been most extensively studied. The N1 peaks at about 100 ms after stimulus onsets, offsets, or changes in stimulus energy. Therefore, the N1 can be used to monitor plastic changes in the central afferent system induced by stimulation. It has been suggested that N1 reflects stimulus representation area in the cortex (Näätänen, 1992). Stimulus repetition diminishes the N1 amplitude, which appears to reflect the refractoriness of the neural populations stimulated by the input (for a review, see Näätänen and Picton, 1987). Additionally, the N1 is modulated by latent inhibition mechanisms (Sable et al., 2004). The N1 has a negative polarity and reaches its amplitude maximum over the fronto-central scalp areas for sounds (Näätänen and Picton, 1987). Its generators for auditory stimuli are located in the supratemporal plane and lateral areas of the auditory cortices and in frontal areas (Giard et al., 1994; Näätänen and Picton, 1987).

The neural basis and plasticity of stimulus discrimination, in turn, can be best investigated with the mismatch negativity (MMN) response (Näätänen et al., 1978), which is elicited by any discriminable auditory change (Sams et al., 1985; Tiitinen et al., 1994; Kujala et al., 2001a; for reviews, see Näätänen et al., 2005, 2007). When a deviant stimulus is presented after a string of similar sounds, then an MMN is elicited at 150–250 ms after change onset (Näätänen et al., 2007). The main MMN generators are located in the supratemporal auditory cortices and frontal areas (for a review, see Alho, 1995), but also parietal-cortex sources have been found (Lavikainen et al., 1994). Acoustic changes elicit stronger MMNs in the right than left temporal lobe, whereas MMNs for speech sound changes are left-hemisphere preponderant (Kujala et al., 2007).

The MMN is based on a memory representation of the auditory past up to about 4–15 s (Mäntysalo and Näätänen, 1987; Cowan et al., 1993; Ulanovsky et al., 2004), reflecting an auditory cortex response to any violation of regularities in the auditory scene (Winkler et al., 2009). That is, the MMN is even elicited in the absence of a physical sound change if the incoming stimulus violates some aspect of the regularity present in the past auditory input. Thus, the MMN reflects higher order memory processes than the N1, which is elicited by a change in the energy or physical properties of a stimulus (Näätänen and Picton, 1987). Furthermore, the MMN and N1 generation seem to have at least partly distinct neurochemical mechanisms, since the blockage of NMDA receptors abolishes the MMN but does not affect the generation of obligatory sound-elicited responses in the auditory cortex (Javitt et al., 1996). At the neuronal level, both N1 and MMN have been associated with stimulus-specific adaptation (SSA), which is a stronger reduction of a neuron's response to a repetitive stimulus than to a rare stimulus (Ulanovsky et al., 2004). It may underlie the maintenance and update of auditory representations, and its high sensitivity to small deviations and fast time course suggests that it encodes relationships between sounds and detects deviations (Winkler et al., 2009). Subcortical and cortical SSA recorded in animals occurs earlier than the N1 or MMN (Nelken and Ulanovsky, 2007), and, therefore, presumably does not reflect identical processes but rather their precursors.

A strong relationship has been found between the MMN parameters and the discrimination ability in behavioural tests (Fig. 1; Näätänen et al., 1993; Kujala et al., 2001a; Novitski et al., 2004; for reviews, see Kujala et al., 2007; Näätänen et al., 2007). MMN is large for large and easily-discriminable sound differences,