The inner sense of time: how the body and brain create our experience of duration.

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On reviewing the diversity of time perception models, it becomes clear that no consensus exists on how and where in the brain temporal information is processed, too many different and competing models exist. However, over the last decade a set of functional models of time perception has been developed, accompanied by empirical evidence from animal research on neural ensemble activity as well as from neuroimaging studies in humans, that point to a specific neural mechanism: the experience of duration might emerge from climbing neural activity, the monotonic increase of neural firing rate across time. I will present my own fMRI and psychophysiological evidence of neurophysiological activity in circumscribed areas of the human brain involved in the encoding of time. Time-activity curves of neural activation during an fMRI duration reproduction task show that activity within bilateral posterior insula represents duration of multiple seconds. Given the close connection between posterior insula and ascending body signals, I suggest that the accumulation of physiological changes in body states constitutes our experience of time. In a further study, a positive relationship was detected between duration reproduction accuracy and the slope of cardiac slowing, which showed a monotonic increase over time during the encoding of temporal intervals. These findings are discussed in a novel framework where physiological changes of the body, the basis of our feeling states, form an internal signal to encode the duration of external events.

Developmental psychology of timing and time perception: Implications for developmental disabilities.

By Melissa J. Allman, Johns Hopkins University School of Medicine, and Kennedy Krieger Institute, Baltimore, MD.

The development of timing and time perception is a critical component in successful adaptation, learning, social and non-social behavior and cognitive development-temporal processing is one of the basic mechanisms of cerebral function. Electrophysiological and behavioral findings reveal young, typically developing infants reveal sensitivity to the temporal quality of events, and this ability continues to develop through childhood. Atypical development of timing and time perception represents a schema for understanding the basis of autism and its associated deficits; in communication, sensory processing and social reciprocity, and a proclivity for restricted and repetitive behaviors and interests. The temporal deficit hypothesis of autism will be reviewed and evaluated, with a particular emphasis on fMRI and behavioral findings that support the idea that children with autism subjectively experience time differently.

Temporal event-coding: What can we learn from patients with schizophrenia?

By Anne Giersch, CR1 INSERM, Dept. of Psychiatry, University Hospital of Strasbourg, France

Results observed in patients with schizophrenia question the mechanisms underlying temporal event-coding. At a clinical level these patients display a disturbed sense of continuity. Experimentally, they are impaired in duration judgments, but also in discriminating between simultaneous and asynchronous events. However, recent studies showed that 'simultaneous' responses are not associated with a fusion of events in time. During the tasks, subjects decided whether two squares were displayed simultaneously or asynchronously, and gave their response by hitting a left or right response key. We repeatedly showed that when stimuli are asynchronous and displayed on opposite sides, manual responses are biased to the side of either the first or second stimulus. Such a bias allows exploring the implicit processing of asynchronies when those are not explicitly detected by subjects. Results have been replicated in four studies and show that patients distinguish events in time at an implicit level even when explicitly judging such events to be synchronous. In addition, their implicit responses differ qualitatively from those observed in controls, for asynchronies as short as 8-17 ms. There is a clear dissociation between results at short and large asynchronies, that question the mechanisms underlying the implicit coding of events in time, not only in patients, but also in controls. It is as if controls would code events relative to one another at an implicit level, even when asynchronies are shorter than 20 ms. We will show results in healthy volunteers that test this hypothesis directly.

Corticostriatal Mechanisms of Timing Deficits in Parkinson's Disease

By D.L. Harrington, Research Service, Veterans Affairs San Diego Healthcare System & Department of Radiology, University of California San Diego, San Diego, CA, USA

Timing is a ubiquitous component of many behaviors. Though the striatum is thought to be essential for interval timing, the experience of time emerges from interactions within integrated corticostriatal networks. This likely explains temporal processing impairments in many clinical disorders, including ones that are not associated with basal ganglia dysfunction. Key components of timing networks include regions of the motor and the executive circuits, but other centers are also involved depending on the behavioral context. In this presentation, I will discuss Parkinson's disease (PD) where an understanding of the neural mechanisms of timing deficits and their response to dopamine therapy has been hampered by a paucity of functional imaging investigations.

We examined brain functioning in PD and healthy adult volunteers as they underwent functional magnetic resonance imaging (fMRI) while performing a

time perception task, in which they judged whether a comparison interval (CI) was longer or shorter than a standard interval (SI). To better understand sources of time perception deficits, brain activity during two phases of a trial was separated. One phase was associated with encoding the SI, which emphasizes timing and working memory, and the other phase with encoding the CI and making a response, which emphasizes timing and decision making. The results showed that timing was impaired in PD. Deficits were partly related to hypoactivation of the striatum during both phases of a trial. In addition, different patterns of cortical dysfunction were found during the two phases in the motor and the executive circuits, sensory integration centers, and a memory hub. Dopamine therapy did not alleviate timing deficits, and had only circumscribed effects on regional activation. However, striatal connectivity with the cortex was modulated by dopamine therapy. I will discuss the implications of these results for understanding corticostriatal mechanisms of temporal processing deficits in PD and the role of dopamine therapy. Future directions will be suggested that may advance an understanding of the neural control of timing in PD and other neurological disorders.