O-033 Synchronized firing of event-related neurons in fronto-opeculo-insular cortex in monkey performing a GO/NOGO taste discrimination task

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In the cortical taste area of anesthetized rats, neighboring taste neurons have similar taste responses and fire spikes in a highly synchronized fashion (Nakamura & Ogawa 1997). In the present study we recorded multiple unitary activities in the fronto-operculo-insular cortex of a monkey while she was performing a GO/NOGO taste discrimination task, and we attempted to study whether pairs of neurons discharge synchronously and share the same response features. Spike wave forms were sampled at rate of 33 k Hz, digitized and stored in the memory of an IBM-compatible computer. A few spike wave forms of neural activities were separated by the cluster cutting method off-line, and a few distinct spike trains were identified accordingly. Neuron pairs (n=339) were recorded in which one of the partner neurons discharged in relation to one of the event in the task, e.g., Cues, LED, Lever, or Reward events. They showed a high tendency to respond to other event when they responded to a given event. Cross correlation calculation revealed that 24 % of the pairs discharged synchronously. Cross-correlated neuron pairs tended to show responsiveness to the same set of task events. It is highly probable that cross-correlated neuron pairs locate in the same functional column, if present.

O-038 The influence of prediction on voluntary saccades reaction times

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Animals predict changes in the environment based on experience. They adjust the prediction according to sensory signal and take action correctly. Carpenter and Williams(1995) showed reaction time of human subjects in saccade task decreased as probability of stimulus presentation increased. This suggests humans predict stimulus presentation. This study presents a psychophysical experiment investigating how the prediction in saccade task may be affected by sensory signal. Furthermore, reaction time in the saccade task is predicted by LATER model (Carpenter, 1981).
METHOD: Subjedts are asked to make saccade to one of two light spots in two situations. One is that a target spot is indicated in a certain probability and the other is that a light spot already indicates the target.
RESULTS: Preliminary data show reaction time decreases with the probability in the former situation and the latter.
CONCLUSION: This suggests the prediciton can not be eliminated by sensory stimulus.

O-034 Activated brain regions by olfactory and gustatory stimulation in unanesthetized behaving monkey: A PET study

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To reveal activated brain regions by olfactory or gustatory stimulation,regional cerebral blood flow (rCBF) was measured by use of positron emissiontomography (PET) and behaving rhesus monkeys. We used acetic acid and applejuice for olfactory and gustatory stimuli. The monkeys were trained to sitin a special apparatus with a head fixation and to receive passively thestimuli during PET scans. A significant increase in rCBF was observed inthe prepyriform area, amygdala and substantia innominata by the olfactorystimulation. These regions were also activated by the gustatorystimulation, probably due to their flavors. The gustatory stimulationselectively increased rCBF in the insula/frontal operculum, orbitofrontalcortex, inferior frontal gyrus and cerebellum. These results indicate thatthe chemical sensing areas could be activated in monkey brain, including theprimary olfactory and gustatory cortices by passive stimulation and PET.Thus, animal PET might be used in the research of chemical sense tointegrate the results from cognitive studies in humans and those frominvasive studies in monkeys.

O-039 Analysis of inter-spike interval statistics of cortical neurons

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Cortical neurons generate irregular spike sequences. But many sequences differ from a entirely random sequence (Poisson process) in their inter-spike interval statistics. We estimate inter-spike interval statistics: the coefficient of variation (CV), the skewness coefficient (SK), and the correlation coefficient of consecutive intervals (COR), from unit spiking data recorded in monkey prefrontal cortex and MT area. The prefrontal data have a bias to exhibit either large SK/CV or large COR, while the MT data have a bias to exhibit small SK but large CV and small or negative COR. Here we attempt to understand the difference by projecting (CV, SK, COR) on the simple spiking model: Markov switching at every spike between two Poisson processes (one is active state, and the other inactive). We can see the difference between prefrontal and MT clearly in staying time scales of active and inactive states. In the prefrontal case, the staying time scales are apt to be large relatively to the mean inter-spike interval. SK is large with the staying time balance biased to active, and otherwise, COR is large. In the MT case, the staying time scales are apt to be not so large and the balance is biased to inactive. The staying time scale can be considered as time scale of temporal correlation in the incoming synaputic inputs. This implys that the difference in the spiking statistics is caused by the difference in the time scales of synaptic inputs correlations.

O-041 Oxytocin release from the pituitary after noxious stimuli and CCK injections under a hyperosmotic condition

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Noxious stimuli activate noradrenergic neurones in the medulla oblongata, facilitate noradrenaline release in the hypothalamus and, as a result, induce oxytocin release from the pituitary. The magnitude of activation of oxytocin-secreting neurosecretory cells after noxious stimuli has been shown to be reduced under a hyperosmotic condition. Hyperosmotic stimuli activate NMDA receptors in the hypothalamus and NMDA receptor activation reduces noradrenaline release in the hypothalamus. It is thus possible that reducing effects of osmotic stimuli upon oxytocin release after noxious stimuli is due to activation of NMDA receptors in the hypothalamus. In the present study, we examined effects of osmotic stimuli on oxytocin release after peripheral injection of cholecystokinin octapeptide (CCK). We also examined effects of noradrenaline release in the supraoptic nucleus (SON). Oxytocin release after CCK was not attenuated under a hyperosmotic condition. Noradrenaline release after noxious stimuli was augmented by local perfusion of an NMAD receptor antagonist into the SON. Oxytocin release after noxious stimuli was augmented by an NMDA receptor antagonist. All these data are consistent with a view that osmotic stimuli reduce oxytocin release after noxious stimuli by inhibiting noradrenaline release via activation of NMDA receptors in the hypothalamus.

O-042 Effect of vasopressin upon noradrenaline release in the supraoptic nucleus.

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Vasopressin is synthesized in the magnocellular neurons of the supraoptic and paraventricular nucleus in the hypothalamus. Vasopressin is released from the dendrites in addition to neurohypophysial axon terminals of the magnocellular neurons. Dendritically released vasopressin has been claimed to facilitate noradrenaline release in the supraoptic nucleus. In the present study, we examined effects of locally applied vasopressin or a vasopressin V1a receptor antagonist (OPC21268) on noradrenaline release in the supraoptic nucleus after high K^+solution in urethane-anaesthetized rats. A microdialysis probe was inserted into the supraoptic nucleus and the noradrenaline concentrations in the dialysate were measured by HPLC in conjunction with electrochemical detection. Vasopressin applied via the microdialysis probe did not significantly change the basal level of noradrenaline release in the supraoptic nucleus. Infusion of high K^+ solution into the supraoptic nucleus increased the noradrenaline release. Vasopressin application increased the noradrenaline release after high K^+ solution, while oxytocin did not significantly change the noradrenaline release after high K^+ solution. An vasopressin V1a receptor antagonist decreased the noradrenaline release after high K^+ solution in a dose-related manner. These data support the view that dendritically released vasopressin facilitates noradrenaline release in the supraoptic nucleus via vasopressin V1a receptors.