Cell motility was measured by the degree of the fluctuation of the light intensity which pass through optical microscope (× 150) observing systems. The photodiode was mounted on the eye-piece lense and its output was fed into the operational amplifier and finally connected to memory oscilloscope. The degree of cell motility was determined by the average amplitude of the intensity fluctuation caused by sperm moving.

In Fig. 1, the membrane potential ($\Delta\varphi$) of the sperm was plotted against the various concentration of potassium. Under the normal condition of incubation solution, the membrane potential of the sperm was about -20 mV, and it was decreased linearly with potassium concentration. Finally, it became nearly 0 when potassium concentration reached 200 mM.

Under the same range of potassium concentrations, the intracellular pH was measured by using ^{31}P-NMR. The results are shown in Fig. 2. From 10 mM to 40 mM of potassium concentration, the pH_{int} was constant (=6.8) and then decreased linearly with K^+ concentration. The same kind of decrease in pH_{int} was also obtained when the pH of the incubation solution was changed to acidic (Fig. 3). In both cases, high K^+ concentration (=200 mM) or low pH (=5.0) of external solution, pH_{int} was decreased about 0.2 unit and surprisingly, cell motility was inhibited absolutely.

From these results, we can conclude that external high potassium concentration led to the acidification of pH_{int} and reached to the immobilization of sperm. The mechanism why high potassium resulted in low pH_{int} and why pH_{int} stops flagellum motion is not clear at present but it might be very convenient to assume the existence of H^+-K^+ exchange mechanism.

Considering the simillarity in the structure of sperm flagellum and hair cell cillia,[6] these results suggest that high potassium content in endolymph of cochlea might be necessary to inhibit self moving of the hair cell cilia and increase the sensitivity to the mechanical stimulation on it..

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Inhibition on cochlear nerve fibers by the sound-activated olivocochlear bundle

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The olivocochlear bundle (OCB) described by Rasmussen descends from the ascending auditory pathway, the superior olivary complex, to the receptor site in the cochlea. In addition to 500 myelinated OCB fibers estimated by Rasmussen, numerous unmyelinated

efferent fibers were histochemically found and the total number of OCB fibers were estimated at 1700-1800. An abundant distribution of the olivocochlear efferent synapses in the cochlea suggests the central control of transduction. The functional significance of the OCB, however, is not yet well understood in spite of the extensive studies and several proposals concerning its possible functions.

Considering that the afferent activity in the cochlea is inhibited by the electrically activated crossed OCB (COCB) and the OCB neurons are activated by a sound, it is an attractive hypothesis that the transduction is modified by the descending OCB which is activated by the input sound, *i.e.* the OCB contributes to a feedback loop. Pfalz denied the assumption from his observation that an intense sound stimulation to an ear, by which the OCB might be bilaterally activated, did not inhibit the cochlear action potentials in response to clicks presented to the other ear.

In the present study interactions in the cochlea between two sounds presented to the both ears were analysed to examine whether or not the activity of a single cochlear nerve fiber can be modified by the sound-activated OCB.

The neuronal activity was isolated under Nembutal anaesthesia with Flaxedil paralization from the cochlear nerve of cats whose another cochlear nerve was completely transsected. A pure tone burst to the ear contralateral to the recording site (c-tone) activated a cochlear nerve fiber, when the c-tone was enough intense and its frequency was not far from the neuron's best frequency. The difference in threshold between the c-tone and an ipsilaterally applied another tone burst (i-tone) at the same frequency exceeded 40 dB.

Changing the phase difference between a simultaneously presented c- and i-tone from the same oscillator, the discharge rate of neuron was changed according to the phase difference. Difference between the most favourable phase angle, and the least favourable, to activate the neuron was found to be π. If the frequency difference between the c- and i-tone was slight and their phases were locked at particular angles at their respective starts in every stimulus trial, the pst-histogram of responses to the binaural stimuli showed ripples which synchronized with the beat between the two sounds. Holding the i-tone at the neuron's best frequency and just above its threshold, the frequency and intensity of c-tone were widely changed. The i-tone response was suppressed by the c-tone whose frequency range was higher and/or lower than the best frequency. The latency and time course of the suppression were exactly same as those of the *two tone suppression* caused by the monaurally applied two sound.

These binaural phenomena observed in the deafferented animals can be explained from their physical characteristics as direct mechanical interactions in the ipsilateral cochlea between an i-tone and a crosstalk of c-tone from the contralateral ear.

In normal cats with the intact right and left cochlear nerve the same mechanical interactions between binaurally applied two sounds were observed on most cochlear nerve fibers as those in the deafferented animals. Among these neurons we observed quite a few neurons which showed a binaural interaction other than the mechanical. The minority of neurons had no inhibitory area which might be observed on secondary neurons for an i-tone stimulation. Unlike the majority of neurons, they were not activated by a c-tone at the neuron's characteristic frequency (c-CF-tone) even at its available maximum intensity, but their spontaneous activity was clearly suppressed by an extremely intense c-CF-tone. During presentation of an i-tone at the neuron's best frequency (i-CF-tone) and just above its threshold, the sound-evoked discharges were suppressed by the c-CF-tone similar to the

spontaneous activity. The pst-histogram of responses to the simultaneously presented two tones at the frequencies slightly different from each other had no ripples corresponding the beat between the two sounds regardless whether or not their phases were locked at their starts, but the histogram showed a slowly progressing monophasic suppression. The suppression by the c-tone on the spontaneous activity and on the sound-evoked activity grew up slowly to its maximum and maintained the plateau with slightly progressive decrease till cessation of the suppressing sound. The discharge rate then recovered gradually to its control level with or without rebound.

The latency, the peak latency and the recovery time of the suppression after cessation of a c-tone ranged 10-28 msec, 20-84 msec and 60-280 msec respectively. These values were not inconsistent with those for the inhibition caused by the electrical stimulation of the COCB. Increasing the intensity of one of the two sounds by more than 15 dB, the mechanical interaction observed on the majority of cochlear nerve fibers became no more recognizable, whereas the suppression on the minority of neurons became more dominant and the extent of suppression increased with elevation of the suppressing c-tone level.

The c-tone frequency was just the neuron's best frequency and the c-tone leakage from the contralateral ear was strong enough at the cochlea under observation to activate the neuron. The c-tone did not activate the neuron, but suppressed it. The suppression can not be considered to be due to a direct action of the leaked c-tone on the opposite cochlea but it may result from the interaural neural inhibition. As any neural connections are not morphologically found between afferent dendrites and the efferent synapses in the cochlea are solely originated from the OCB, the inhibition might come from via the OCB.

An effective c-CF-tone level for the inhibition was so high that it exceeded 60 dB SPL and was around 100 dB SPL in some neurons. The population of cochlear nerve fibers which were inhibited by a c-CF-tone was small; less than 10 % of the fibers examined precisely in the present study. It was reported that the electrical stimulation of the crossed OCB beneath the fourth ventlicle inhibited most of cochlear nerve fibers. These facts imply that the OCB might not be expected to be an automatic gain control system, but, besides an input sound, synaptic convergence of any other additional gating signals to the OCB neurons in the superior olivary complex, probably from the higher level of the CNS, might be necessary to activate the OCB extensively enough to inhibit the transduction in the cochlea.

Structural bases of crustacean mechano-sensory hair function

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Crustacea and insect are the main member of arthropod, whose external surface is protected with sclerotized chitinous cuticle. Those animals therefore must have cuticular structure on the exoskeleton as sensory interface with the environment. Hair structure is employed as the crustacean cuticular mechanoreceptor. Antennular basal segment of crayfish carries several groups of mechano-sensory hairs including those of the statocyst