each experiment, is particularly noticeable. This means that *G. petersii* prefers to respond to an exteroceptive electric stimulus, perceived through its electroreceptors, by an electric organ discharge within about 11 msec.

As pointed out by electrophysiological work, 7–8 msec are required to conduct the command signal from the medulla oblongata to the electric organ in mormyrids[4]. Hence less than 4 msec remain for the transmission of the coded receptor signal to the medulla oblongata, via lateral nerve, lobus lateralis, and mesencephalon. Among the different electrosensory receptor types (Mormyromasts, tuberous ('Knollen'-), and ampullary organs), and their connections to the brain, found in mormyrids, only the tuberous receptors and their 'fast' junctions, and cerebral connections, are capable of such a rapid signal transmission[5–7]. This pathway comprises neurons with axons of large diametre, and probably electrical synapses between them[8].

Although a latency of about 11 msec seems to be the minimal reaction time of *G. petersii* to an external electric stimulus, the fish is free to respond after a longer time as well, as seen in Figure 1c. Shorter latencies than the minimal time were also observed, beginning at zero latency. In this case, it must be assumed that the pulse-making decision had already taken place before the *M. rume* pulse occurred, and thus could not have been altered by sensory input.

The observation reported in this paper demonstrates a hitherto unknown interaction between the discharges of 2 electric fish at the interval level, indicating the existence of an extremely rapid reflex arc. This reflex arc seems to be specifically involved in the perception of, and in responding to, the electric signals of congeners because of 2 reasons: 1. The threshold to short electric stimuli is low in tuberous receptors, compared with the other electroreceptor types[5], enabling the fish to detect a conspecific, when the distance between them is ca. 30 cm or less[9]; 2. When responding to the fish's proper discharge, tuberous receptor evoked activity in ganglion cells of the lobus lateralis was found to be inhibited[10]. This excludes the possibility of a significance of tuberous receptors, and their cerebral connections, in electrolocation, and suggests their importance in 'communication'.

Zusammenfassung. Während des agonistischen Verhaltens beantwortet *G. petersii* elektrische Organentladungen eines *M. rume* statistisch bevorzugt nach einer Latenzzeit von nur ca. 11 msec. Dies deutet auf die Existenz einer besonders raschen Reflexbahn hin, deren spezifische Aufgabe offenbar die Wahrnehmung und Beantwortung elektrischer Fremdsignale ist.

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The Effects of Reticular Stimulation on Digastric Reflex Activity During Sleep and Wakefulness

The spontaneous variations in digastric reflex (jaw-opening) activity during the course of sleep and wakefulness have been extensively investigated[1]. The orbital cortical control of this reflex during sleep and wakefulness in freely moving cats has also been described[2,3]. In immobilized cats, which presumably are awake, electrical stimulation of the reticular formation can modify digastric reflex activity[4,5]. We were therefore interested in determining whether stimulation of the reticular formation, in freely moving unanesthetized cats, would influence this reflex not only during wakefulness but also during sleep. In this paper we present data which indicate that electrical stimulation of the reticular formation exerts a consistent pattern of digastric reflex inhibition throughout quiet sleep and active sleep as well as during wakefulness.

The details of the experimental preparation for stimulation and recording with permanently placed electrodes in the freely moving cat have been previously described[1]. 6 adult cats were used in this experiment. Bipolar stimulating electrodes were placed in the ponto-mesencephalic reticular tegmentum. The digastric reflex was induced by electrical stimulation of the inferior dental nerve with a pair of stainless steel screws embedded in the mandibular canal. The reflex was monitored electromyographically by a pair of bipolar electrodes placed around the anterior belly of the digastric muscle. Other bipolar electrodes were used to record the frontal cortical EEG, eye movements (EOG) and neck EMG. The digastric reflex was monitored oscilloscopically and was recorded on a polygraph by utilizing a peak-amplitude, time-expanding electronic circuit.

Experimental sessions, which were started at least week after electrode implantation, were conducted in an environmental chamber where the animal was able to move about freely. The behavior of the animal was observed through a one-way window. Each experimental session lasted approximately 3 h, during which time continuous recordings of consecutive cycles of sleep and wakefulness were obtained. The digastric reflex was induced continuously throughout each session at rates of 0.5 to 1 per sec. The amplitude of the digastric reflex which followed short pulse train reticular stimulation (1–4 pulses; conditioning-test latency 0–20 msec) was compared with the amplitude of immediately preceding control reflexes during the states of wakefulness, quiet sleep and active sleep. The intensity of the reticular

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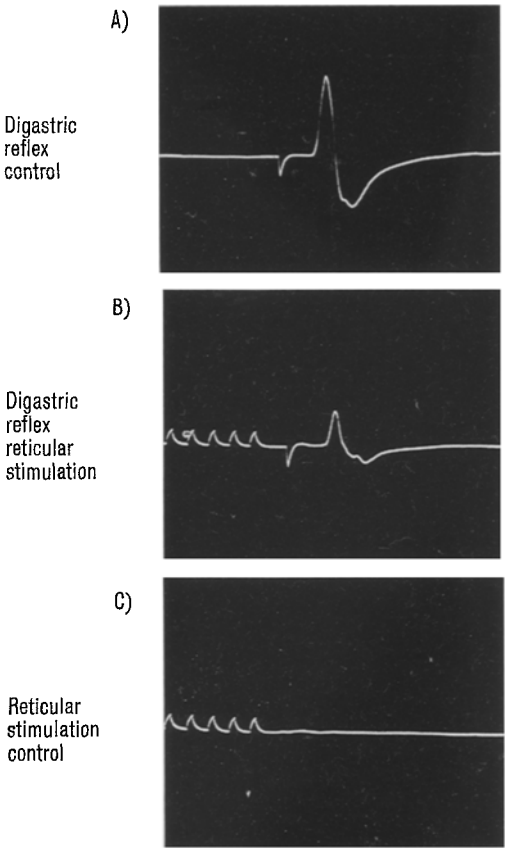

Fig. 1. Oscilloscopic recordings of digastric reflex inhibition following reticular stimulation. Note that the reticular stimulus alone did not induce digastric activity. All recording was obtained when the cat was awake. Inferior Dental Nerve: 0.4 V, 0.4 msec. Reticular stimulation: 1.5 V, 1 msec, 5 pulses. Calibration: 50 μV, 5 msec.

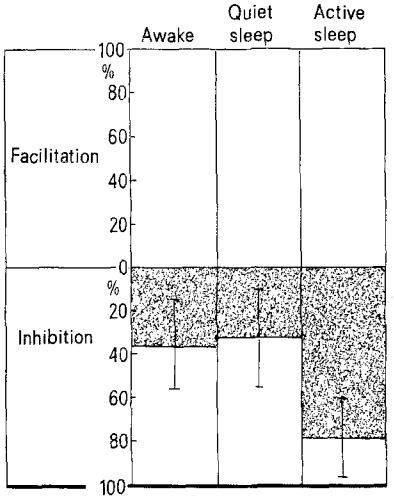

Fig. 2. Graphical representation of digastric reflex inhibition induced by reticular stimulation. Each bar indicates % inhibition. The amplitude of the digastric reflex immediately preceding reticular stimulation was used as the baseline control amplitude. The standard deviation is indicated by the vertical line in the middle of each bar. The data for each state were based upon the amplitudes of 40 control reflexes and 40 reflexes preceded by reticular stimulation.

stimulation was adjusted so that no overt behavioral response was induced. During active sleep, the intensity of dental nerve stimulation occasionally had to be increased due to the spontaneous depression of the reflex which is prominent during this state[1].

An example of the digastric reflex in the control condition and preceded by reticular stimulation is shown in Figure 1. The effect of reticular stimulation on the induced digastric reflex was clearly inhibitory (Figure 1, A and B). Inhibition was observed at all conditioning-test latencies between 0 and 20 msec. Note that no response was evoked in the digastric muscle in conjunction with independent excitation of the reticular formation (Figure 1C).

Figure 2 represents a graphical and statistical description of the inhibitory effect of reticular stimulation on the digastric reflex during sleep and wakefulness. Inhibition was observed in all animals in every session – during arousal accompanying wakefulness, during the spindle bursts of quiet sleep, and during the rapid eye movements of active sleep. Thus, there were no qualitative differences in the inhibitory effect during intrastate variations in activity.

Almost without exception the inhibitory function of the reticular stimulus (as expressed by the % decrease from baseline level) was more pronounced during active sleep than during wakefulness or quiet sleep. As the duration, voltage or number of reticular pulses were increased, the degree of inhibition was enhanced.

CHASE and MCGINTY[2] observed that orbital cortical stimulation led to digastric reflex facilitation and an evoked digastric discharge. The effects of orbital stimulation were examined during sleep and wakefulness under conditions similar to those described in this paper[3]. In the previous study, digastric activity was facilitated by orbital stimulation during all states of sleep and wakefulness, whereas in the present investigation reticular stimulation induced reflex inhibition throughout these same states. The contrasting effect may be attributed to the difference in the function of these brain sites. However, during the stage of active sleep, orbital facilitation was clearly reduced, whereas reticular inhibition was perhaps most potent during this state. It would be tempting to consider the possibility that this reflex is primarily controlled during active sleep by the reticular system and during wakefulness by orbital cortical discharge. The facilitation of this reflex by orbital excitation[2], the fact that during wakefulness the orbital effect is strongest[3] and the suggestion of an increase in the effectiveness of reticular inhibition during active sleep support this hypothesis[6].

Résumé. La stimulation électrique de la formation réticulaire mésencéphalique fut appliquée 0–20 msec avant l'induction du réflexe digastrique chez des chats libres. Le reflexe digastrique fut induit par stimulation électrique du nerf dentaire inférieur. La stimulation réticulaire eut pour résultat l'inhibition de ce réflexe pendant la veille, le sommeil tranquille et le sommeil actif.

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