correspond, temporally, to the extracellular negativity. Furthermore, when recording with an electrode with a fortunate tip size, extracellular unitary discharges could be seen riding upon the slow negative wave. Such negativity, then, seems to represent an envelope of spike activity and associated EPSPs.

An analysis of extracellular unitary responses was undertaken; 165 units were examined. The conduction velocity of fibres from the deep temporal lobe was determined from the analysis of the difference in latencies of extracellular units, with stimulating electrodes in both the anterior amygdaloid area and the diagonal band of Broca. The modal conduction velocity from the amygdala to the nDBB, of 11 estimates, was 0.6 M/sec; with a range of 0.5 M/sec to 0.9 M/sec. GLOOR[3] has reported a conduction velocity of fibres from basolateral amygdala stimulation to the anterior hypothalamus of 1 M/sec.

The relative stability of latency for unitary discharges with changes in stimulus intensity is shown in D. With submaximal stimulation, beginning at 1.4 times threshold (1.4 T), the latency was 18 msec. The latency remained stable until threshold (1.0 T) is reached where the latency increased to 22 msec. This relative constancy in discharge latency is taken to represent the monosynaptic excitation of cells. The latency shift at threshold is understandable in terms of slow rise times and temporal dispersion of the EPSPs. In addition to those units which showed stable latencies, units were encounted which showed marked instability, sometimes even at a constant stimulus intensity. These, undoubtably, represent polysynaptic activation.

With the stria terminalis severed, stable, as well as unstable, extracellular units could still be recorded to amygdala and prepyriform cortex stimulation. Consideration of the conduction velocity and conduction distance made the observed latencies of the stable units reasonable for a monosynaptic connection. These results suggest a combination mono- and polysynaptic input from the amygdala and/or prepyriform cortex to the nDBB.

Extracellular units which displayed stability in latency showed durations of test response suppression with paired-stimulus testing from 100 msec to 1000 msec. The modal time to recovery was 400 msec. An example is shown in E. The upper trace is the test control. The conditioning responses are not shown. As seen in this instance, the unitary discharge is absent in a 200 msec interval, but re-appears in a 400 msec interval. This characteristic recovery time was also seen for the test field response. No differences were found in the behavior of the test responses between amygdala and prepyriform cortex stimulation.

Extracellular units in the nDBB responding to deep temporal lobe stimulation could be antidromically activated by IFim stimulation; 45 of 133 units tested were antidromically by IFim stimulation. F shows an example where amygdala stimulation evokes two unitary discharges and where IFim stimulation produces a short latency discharge. The latency in this instance was 1.5 msec. Paired-stimulus testing of the unit with an IFim-IFim combination found it to recover in a 2.5 msec interval, suggesting its antidromic nature. The modal conduction velocity of the antidromically activated units was 4.0 M/sec, with value ranging from 2.8 M/sec to 4.8 M/sec.

There is a problem concerning the origin of the projections to the nDBB. RAISMAN[12] and DEOLMOS[13] indicate that amygdala projections are restricted to the tract of the diagonal band. Moreover, the prepyriform cortex has been shown to project through the amygdala[5,9]. Hence, in this study, amygdala stimulation should also activate fibres of passage from the prepyriform cortex. What contribution the amygdala makes to the projection to the nDBB could not be determined with the present techniques. This difficulty led us to examine inputs from both areas and include these results in the term 'deep temporal lobe projections'.

These results suggest that the deep temporal lobe projections to the nDBB are monosynaptically excitatory and that some of the target neurons project to the hippocampal formation. The nDBB, therefore, provides a synaptic station for prepyriform cortex and possible amygdala input for the influence of hippocampal activity[14].

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Adrenaline and the Electrogenic Sodium Pump in *Rana catesbeiana* Sympathetic Ganglion Cells

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Summary. The effect of adrenaline on the Na^+-pump in bullfrog (*Rana catesbeiana*) sympathetic ganglion cells was studied by use of electrophysiological methods. The rate of removal of excess Na^+ injected into a ganglion cell was increased by adrenaline. The K^+-activated hyperpolarization of cell membrane, which might be produced by an electrogenic Na^+-pump, was also increased by adrenaline. These results suggested that adrenaline was able to accelerate the Na^+-pump, possibly the electrogenic Na^+-pump.

When adrenaline (Ad) is directly applied to bullfrog's sympathetic ganglia, the ganglion cells produce depolarizing (Ad-depolarization) and also hyperpolarizing (Ad-hyperpolarization) responses[1]. The nature of Ad-hyperpolarization is essentially similar to that of the slow inhibitory postsynaptic potential (slow IPSP)[1], which seems to be produced by an electrogenic sodium pump[2]. It was, therefore, suggested that Ad might be able to accelerate the electrogenic sodium pump and thus to produce the Ad-hyperpolarization[1]. The present experiment was designed to demonstrate the experimental

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evidence by which this hypothetical concept could be supported.

Isolated paravertebral sympathetic ganglia of bullfrogs (*Rana catesbeiana*) were used. Experimental arrangements for intracellular recordings of action potentials of single ganglion cells, and also for the sucrose-gap method to record membrane potential changes of ganglion cells, were described elsewhere[2,3]. Intracellular injections of sodium ions were made through a single intracellular microelectrode connected to the input bridge circuit and also to a pulse generator[4]. A glass capillarly microelectrode filled with 2 m*M* NaCl was used for both sodium injection and potential recording; the resistance of these electrodes was ranged between 20–30 megohm. Action potentials of a cell were produced by applying antidromic stimulations to postganglionic nerve fibres. Ionic compositions (in m*M*/1000 cm^3 H_2O) of Ringer's solution were as follows: 112 m*M* NaCl, 2 m*M* KCl, 1.8 m*M* $CaCl_2$ and 2 m*M* $NaHCO_3$. 2 m*M* KCl was simply omitted from the Ringer's solution for preparing the K-free Ringer's solution. A preparation was continuously perfused with a solution flowing through a channel ($50 \times 5 \times 4$ mm) at the rate of 0.2 cm^3/sec; a perfusate in the channel could be replaced completely by another perfusate within approximately 5 sec. Drugs added to the solutions were adrenaline bitartrate (SIGMA) and ouabain (Merck). All experiments were carried out at 20–24 °C.

The effect of intracellular iontophoretic injection of sodium ions on the action potentials was studied with cat's spinal motoneurones, in order to analyze the rate of extrusion of injected excess sodium ions[5]. It was shown in this experiment that characteristic changes in action potentials occurred after a sodium injection, and the recovery time course of these changes was approximately exponential[5]. Similar results were obtained from present preparations. Since a single intracellular electrode was used for both recording and injection in the present experiment, the value of resting membrane potential was ignored after sodium injections. Thus, the recovery time course of action potentials, of which configurations were changed by intracellular sodium injections (5×10^{-9} A for 30 sec), was analyzed by measuring the amplitude of after-hyperpolarization of these action potentials[5].

Immediately after a sodium injection, the amplitude of spike potentials showed either an increase or no changes, while the amplitude of after-hyperpolarization (see Figure 1) was consistently and markedly decreased. The decrease in the amplitude of after-hyperpolarization was gradually restored and its recovery time course was approximately exponential (Figure 1). The recovery time constant obtained from 12 cells was 75 ± 9 (mean ±SD) sec. Effects of 0.005 m*M* ouabain and 0.3 m*M* Ad on the recovery time constant of these cells were studied. Both resting and action potentials of these cells showed no significant changes between 10 and 30 min after an ad-

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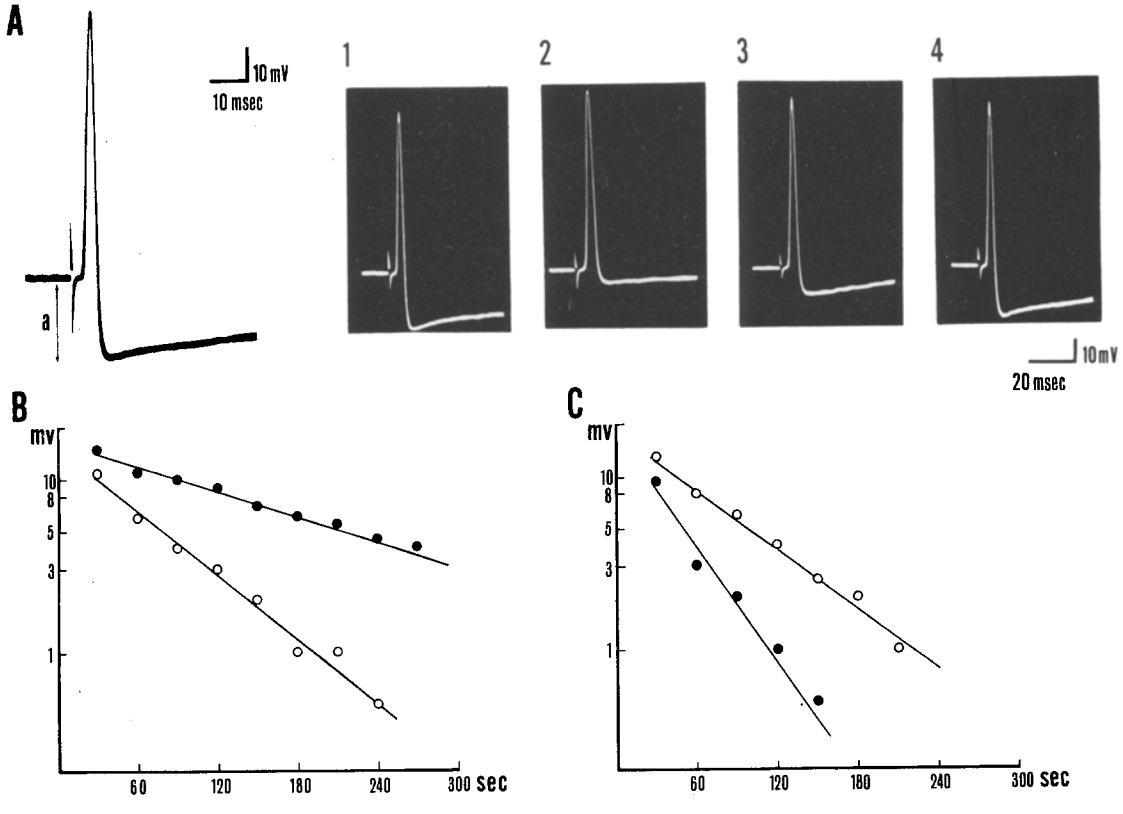

Fig. 1. Recovery time courses of the after-hyperpolarization of action potentials changed by intracellular sodium injections. Sodium ions were injected into ganglion cells by applying a constant direct cathodal current (5×10^{-9} A) for 30 sec through an intracellular sodium electrode. The amplitude (shown by *a* in A) of after-hyperpolarizations is transiently depressed after a sodium injection, as seen in A where records 1, 2, 3 and 4 were taken before, 30 sec, 90 sec and 300 sec after an injection, respectively. The recovery time course of amplitude of after-hyperpolarization is shown in B and C, which were obtained from two different cells. In B and C, differences between the value of *a* before and after a sodium injection are plotted in mV against the time after an injection, with (closed circles) and without (open circles) ouabain (B) or Ad (C).

dition of each drug. The recovery time constant of the amplitude of after-hyperpolarization after sodium injections, however, was reversibly increased during this period in the presence of ouabain (Figure 1); the recovery time constants measured from 6 cells were 188 ± 20 sec. On the other hand, it was reversibly decreased during this period in the presence of 0.3 m*M* Ad (Figure 1); the recovery time constants measured from 6 cells were 40 ± 10 sec.

The potassium-activated hyperpolarization was demonstrated with rabbit non-myelinated nerve fibres[6]. A similar potassium-activated hyperpolarization was recorded from present preparations by use of the sucrose-gap method[7]. In the present experiment, isolated ganglia were initially perfused with the K-free Ringer's solutionfor 60 min. When the perfusate was changed to the Ringer's solution (containing 2 m*M* KCl), the potassium-activated hyperpolarization of ganglion cells, which reached a maximum value (2–4 mV) within approximately 3 min which was sustained thereafter, could be consistently recorded (Figure 2). The maximum amplitude of the potassium-activated hyperpolarization of a preparation was fairly constant between 60 and 180 min in the K-free Ringer's solution, provided each application of the Ringer's solution for approximately 3 min was repeated at an interval of 15 min. These potassium-activated hyperpolarizations were completely and reversibly abolished in the presence of 0.005 m*M* ouabain[7].

The potassium-activated hyperpolarizations initiated before, during and after an application of 0.3 m*M* Ad were recorded and compared with each other, in order to examine the effect of Ad and its reversibility. The potassium-activated hyperpolarization produced in the presence of Ad was markedly augmented compared with that recorded in the absence of Ad (Figure 2). Such an augmentation of the potassium-activated hyperpolarization was observed consistently after Ad was applied for more than 10–20 min. The increases in the maximum amplitude observed from 8 preparations were 46 ± 13%.

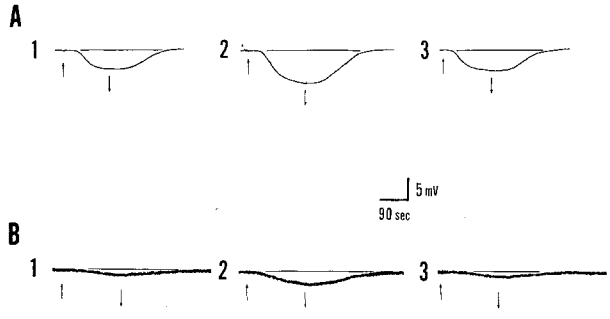

Fig. 2. Potassium-activated hyperpolarizations of bullfrog's sympathetic ganglion cells, being recorded by the sucrose-gap method (A) and an intracellular microelectrode (B). Records were taken before (1), 30 min after an addition of 0.3 m*M* Ad to the perfusate and 60 min after withdrawal of Ad (3). Note reversible increase in the amplitude of potassium-activated hyperpolarizations under the effect of Ad.

The potassium-activated hyperpolarization of a single ganglion cell, which reached a maximum value (2–3 mV) within approximately 3 min, was also recorded by an intracellular microelectrode. Its maximum amplitude was fairly constant between 60 and 180 min in the K-free Ringer's solution, provided each application of the Ringer's solution for approximately 3 min was repeated at an interval of 15 min. When 0.3 m*M* Ad was added to the K-free Ringer's solution, the resting membrane potential of a cell showed no, or small, depolarization (a few milivolts) and the membrane resistance (input resistance) showed no detectable changes. The potassium-activated hyperpolarizations was augmented in the presence of Ad (Figure 2); the increases in the maximum amplitude observed from 8 preparations were 32 ± 8%.

The recovery time constant of positive after-hyperpolarization of action potentials was assumed to characterize the rate of sodium removal, namely the rate of sodium pump from a cell[5]. This assumption was supported by the present result that the recovery time constant was markedly increased by the action of ouabain in a small concentration. On the other hand, the present experiment demonstrated that the recovery time constant was markedly decreased in the presence of Ad. Thus, the rate of sodium pump appeared to be increased in the presence of Ad. The present experiment also demonstrated that the potassium-activated hyperpolarization was augmented in the presence of Ad. If one assumes that the potassium-activated hyperpolarization is generated by an electrogenic sodium pump[6], the electrogenic sodium pump appeared to be accelerated in the presence of Ad. In conclusion, the present experiment results altogether suggested that Ad was able to accelerate the sodium pump, possibly the electrogenic sodium pump. This supports our hypothetical concept that the neurotransmitter, such as Ad, is able to regulate the membrane potential by controlling the active ionic transport.

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The Renal Concentrating Ability of Newly Born Brattleboro Rats (Hereditary Diabetes Insipidus)

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Summary. In Brattleboro rats, there was no difference in urine osmolality between animals with and without diabetes insipidus after water deprivation up to age 14 days, and it appeared at age 18 days due to increase of osmolality in non-diabetic individuals.

Vasopressin (VP) regulates body water content by acting on water reabsorption in the distal segment of the nephron. In young rats, this activity is less pronounced and newly born rats excrete a urine of low osmolality even during dehydration[1–3]. The content of VP in the neurohypophysis of the newly born is lower than in adult animals, but is still sufficient to play a role in osmoregulation[4–6]. The response of the kidneys depends

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