also possible that early crystallization of plagioclase grains causes them to be isolated from each other by later forming minerals such as quartz and potassium feldspar. Quartz, however, does not seem to affect the development of neighboring grains, and it also appears that no mineral affects the development of other mineral species (*1*).

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Note

1. This research was financed by the Harry Carothers Wiess fund of the department of geology of Rice Institute. We thank John M. Whitfield for helping to assemble the collection studied.

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On the Presence of 3-Hydroxytyramine in Brain

The compound 3-hydroxytyramine has attracted interest as a probable intermediate in the biosynthesis of noradrenaline and adrenaline and also as a possible neurohumoral agent. It has been shown to occur in the urine (*1*), in the adrenals (*2, 3*), and in the heart (*2*) of sheep and in the splenic nerve of the ox (*4*). The study of this compound has been hampered by lack of sensitive and specific assay methods. Apart from bioassay techniques, only the fluorimetric ethylenediamine condensation method of Weil-Malherbe and Bone (*5*) appears to be sufficiently sensitive for biological purposes. However, with this method the fluorescence spectra obtained from 3-hydroxytyramine and adrenaline are almost identical (*6*). In the fluorimetric method of Euler and Floding (*7*), the fluorescence obtained from 3-hydroxytyramine is very weak and amounts to only a few percent of that obtained from noradrenaline or adrenaline.

Recently we observed, however, that if the *p*H of samples prepared essentially according to this method was adjusted to about 5 by means of acetic acid, a fairly strong fluorescence developed. Furthermore, the activation and fluorescence peaks (345 and 410 mμ, respectively, as read in an Aminco-Bowman spectrophotofluorimeter) were at much shorter wavelengths than those obtained from noradrenaline and adrenaline, so that these compounds did not interfere, even if they were present in comparably large amounts.

Using this technique in combination with ion-exchange chromatography (Dowex 50), we have started to investigate the 3-hydroxytyramine content of various tissues. We have thus found that 3-hydroxytyramine is present in rabbit brain in an amount of about 0.4 μg/g, which is roughly equal to the amount of noradrenaline in this tissue. This may indicate that the function of 3-hydroxytyramine is not merely that of a precursor. The following criteria argue for the identity of the apparent 3-hydroxytyramine in brain with authentic 3-hydroxytyramine: (i) identical activation and fluorescence peaks, (ii) similar behavior on an ion-exchange column, and (iii) identical R_f values on paper chromatography.

Like noradrenaline (*8*), 3-hydroxytyramine is made to disappear almost completely from brain by intravenous injection of reserpine (5 mg/kg). On the other hand, the injection of the precursor 3,4-dihydroxyphenylalanine (150 mg of the DL form per kilogram, intravenously) caused a very marked increase in the 3-hydroxytyramine content of the brain (to about 2 μg/g in less than 1 hour). This was accompanied by central excitation (*9*). Both these phenomena were markedly enhanced by pretreatment with iproniazid (Marsilid). Simultaneous changes in the noradrenaline level of the brain were much less pronounced if present at all (*10*).

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References and Notes

1. P. Holtz, K. Credner, G. Kroneberg, *Arch. exptl. Pathol. Pharmakol. Naunyn-Schmiedeberg's* **204**, 228 (1947); U. S. von Euler, U. Hamberg, S. Hellner, *Biochem. J.* **49**, 655 (1951).
2. M. Goodall, *Acta Physiol. Scand. Suppl.* 24, 42 (1951).
3. D. M. Shepherd and G. B. West, *J. Physiol. (London)* 120, 15 (1953).
4. H. J. Schümann, *Arch. exptl. Pathol. Pharmakol. Naunyn-Schmiedeberg's* 227, 566 (1956).
5. H. Weil-Malherbe and A. Bone, *Biochem. J.* 51, 311 (1952).
6. J. Kägi, M. Burger, K. Giger, *Arch. exptl. Pathol. Pharmakol. Naunyn-Schmiedeberg's* 230, 470 (1957).
7. U. S. von Euler and I. Floding, *Acta Physiol. Scand. Suppl.* 33, 45 (1955).
8. A. Carlsson *et al.*, in S. Garattini and V. Ghetti, *Psychotropic Drugs* (Amsterdam, 1957).
9. A. Carlsson, M. Lindqvist, T. Magnusson, *Nature* 180, 1200 (1957).
10. A detailed discussion of these results is in preparation.

4 November 1957

Upper Atmosphere Densities from Minitrack Observations on Sputnik I

The analysis of Minitrack (*1*) data on the first U.S.S.R. satellite, 1957 Alpha 2 (*2*) provides information on the density of the atmosphere (*3*) above the perigee altitude of 232 km. We find that the observed rate of change of period for Alpha 2 may be explained by a model atmosphere which is in agreement with recently obtained data on air density and temperature at altitudes (*4, 5*) up to ~200 km and constitutes a reasonable extrapolation of these measurements to higher altitudes. With allowance for the estimated probable errors in the density at 200 km and for the uncertainty in the orbit elements and ballistic drag parameter of Alpha 2, the data still yield a relatively unambiguous determination of density up to 400 km.

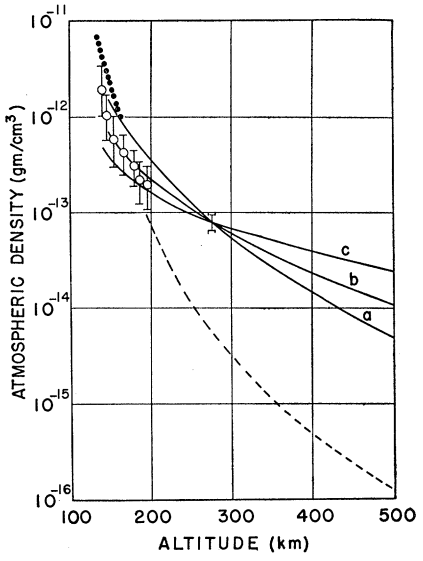

Fig. 1. Curves *a, b,* and *c* represent density distributions adjusted for simultaneous agreement with the rocket measurements and the α2 data. The dashed curve is the ARDC model atmosphere.

The determination of the density from the rate of change of the orbital period depends on the values of the ballistic drag parameter and the orbit constants of Alpha 2. The present calculations are based on a ballistic drag parameter of 89 ± 11 kg/m², derived from U.S.S.R. announcements of mass and area (*6*). The relevant orbit elements were deduced from Minitrack observations between 14 and 25 October, and their average values for that interval are as follows: perigee altitude = 232 ± 5 km; eccentricity = 0.047 ± 0.004; latitude of perigee = $39° \pm 6°$; equatorial inclination = $64.5° \pm 0.3°$; rate of change of period = 0.045 ± 0.003 min/day.

Our results are shown in Fig. 1. The solid lines represent three model atmospheres (*a, b,* and *c*) which agree with the rate of change of period of Alpha 2 and also fall within the limits of probable error in the rocket measurements of density up to 185 km. The data of Horowitz and LaGow (*4*) are indicated by circles, and the data of Byram, Chubb, and Friedman (*5*) by a dotted line. The dashed curve is the atmosphere proposed by Mizner and Ripley (*7*). The spread in the solid curves above 275 km indi-

cates the uncertainty in the density at high altitudes corresponding to the probable errors in the rocket data for altitudes below 200 km. The separation between the brackets at 275 km represents the additional uncertainty in the density when allowance is made for the quoted probable errors in the ballistic drag parameter and perigee altitude.

It is interesting to note that at 400 km the densities indicated by the present analysis are larger than those of the ARDC atmosphere (*6*) by a factor of approximately 40, and estimates of satellite lifetime are therefore reduced by the same factor for orbits with this perigee altitude. For example, a satellite with a mass of 10 kg and diameter of 50 cm, moving in an orbit with initial perigee and apogee altitudes of 400 km and 1000 km, respectively, has a lifetime of ~4 years in the ARDC atmosphere, versus 38 days in the present model (*b*).

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References and Notes

1. J. Mengel, *Elec. Eng.* **76**, 666 (1957).
2. We follow the notation adopted in the announcements of the Harvard College Observatory, according to which a satellite will be identified by the year of launching, by a Greek letter indicating the order of launching, and by an Arabic numeral denoting visual brightness, the last added only when several objects appear during one launching. Thus the brightest object appearing in the first launching of 1957 is labelled 1957 α1, the next brightest, α2, and so forth.
3. We are indebted to Mr. H. E. LaGow and Drs. H. Friedman, H. E. Newell, J. A. O'Keefe, and F. C. Johnson for valuable discussions on the properties of the upper atmosphere. We are also grateful to Mr. W. F. Cahill of the National Bureau of Standards for the preparation of the machine program for integration of the equations of motion, and to Mr. J. H. Wegstein for his collaboration in writing the earlier forms of that program.
4. R. Horowitz and H. E. LaGow, *J. Geophys. Research* **62**, 57 (1957).
5. E. T. Byram, T. A. Chubb, H. Friedman, "Dissociation of oxygen at high altitudes," in *The Threshold of Space*, M. Zelikoff, Ed. (Pergamon, New York, 1957).
6. Mass = 83.4 kg; diameter = 58 cm; total antenna length = 812 cm; mean antenna radius = 1.0 ± 0.3 cm (estimated). Extreme assumptions regarding the mechanism of reflection at the satellite surface lead to values for the drag coefficient, C_d, which vary from 2.0 to 2.7, hence we take $C_d = 2.3 \pm 0.3$. The ballistic drag parameter, Mass/$C_d \times$ Area, is then 89 ± 11 kg/m^2.
7. R. A. Mizner and W. S. Ripley, Air Research and Development Command (ARDC) Atmosphere, *ASTIA Document 110233* (1956).

23 January 1958

Localization of Stretch Reflexes by Recurrent Inhibition

Feedback discrimination between active cells, and hence between their functional fields, appears to be a general property of nervous systems. When a cell, or group of cells, is activated, neighboring cells are frequently inhibited, and contrast between active and inactive zones is thereby enhanced. Such interaction has been described in the eye of *Limulus* (*1*), the cat's cochlea (*2*) and retina (*3*), the cat's trapezoid body (*4*), and the afferent path to the cat's somatosensory cortex (*5*). It is the purpose of this report to show that the antidromic inhibition which results from spinal motoneuron activity (*6*) represents a similar discriminatory mechanism; it creates a negative feedback that is instrumental to the localization of stretch reflexes and thus aids in the control of fine movement.

Antidromic activation of motor nuclei inhibits monosynaptic reflexes of neighboring nuclei (*6*). During the period of inhibition a repetitive internuncial discharge occurs (*7*), but lack of anatomical evidence dissuaded Renshaw (*7*) from linking the two phenomena causally. As the time course of motoneuron aftercurrents is very similar to that of the inhibition, current between neighboring cells might be responsible for the inhibition (*8*).

Recent work in which microelectrodes were utilized has provided strong evidence that the inhibition is mediated through the interneurons described by Renshaw, as a result of their having been activated cholinergically by recurrent motor axon collaterals (*9*). To strengthen the chain of evidence, a comparison has been made between the actions of an anticholinergic drug on (i) the inhibition of neighboring motoneurons, (ii) the electrical signs of activity in Renshaw cells, and (iii) the antidromic electrotonus of motoneurons reflecting current between them, as recorded on ventral roots.

All preparations were immobilized by intravenous injections of Flaxedil. Figure 1 shows that an intravenous injection of 0.5 mg of dihydro-β-erythroidine (DHE) (*10*) per kilogram reduces in magnitude the inhibition exerted by an antidromic volley in one fraction of the first sacral ventral root upon a monosynaptic reflex elicted by stimulation of the corresponding dorsal root and recorded in the remainder of the ventral root. Coincidentally (Fig. 2*B*), DHE shortens the duration of Renshaw cell discharge, as has been previously described (*9*). The small change in ventral root electrotonus (Fig. 2*A*) does not exceed changes seen between controls recorded at various times during the experiment. It is evident that DHE differentiates between antidromic inhibition and Renshaw cell discharge, on the one hand, and ventral root electrotonus on the other.

Since the inhibitory system is acti-

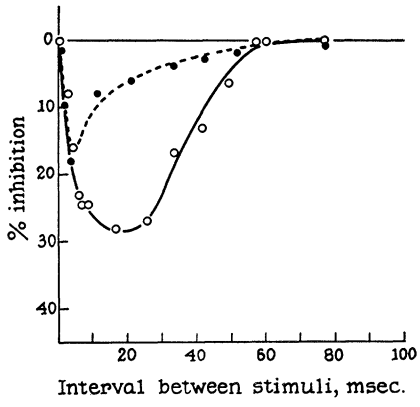

Fig. 1. Antidromic inhibitory curve obtained in a decerebrate preparation. For procedure, see text. Circles show inhibition before, dots after, intravenous injection of DHE. Each point was determined from 20 superimposed reflex responses.

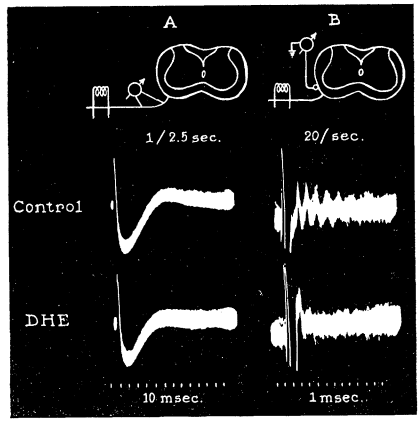

Fig. 2. Same experiment as illustrated in Fig. 1; (A) ventral root electrotonus, (B) Renshaw cell discharge, before and after injection of DHE. Diagrams show experimental arrangements.

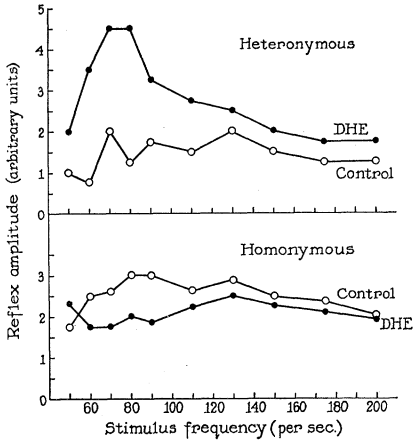

Fig. 3. Decerebrate preparation. For procedure see text. Graph shows, in arbitrary units, the effect of an intravenous injection of DHE on homonymous and heteronymous responses. Each point represents the average size of reflexes superimposed during a 1-second exposure.