

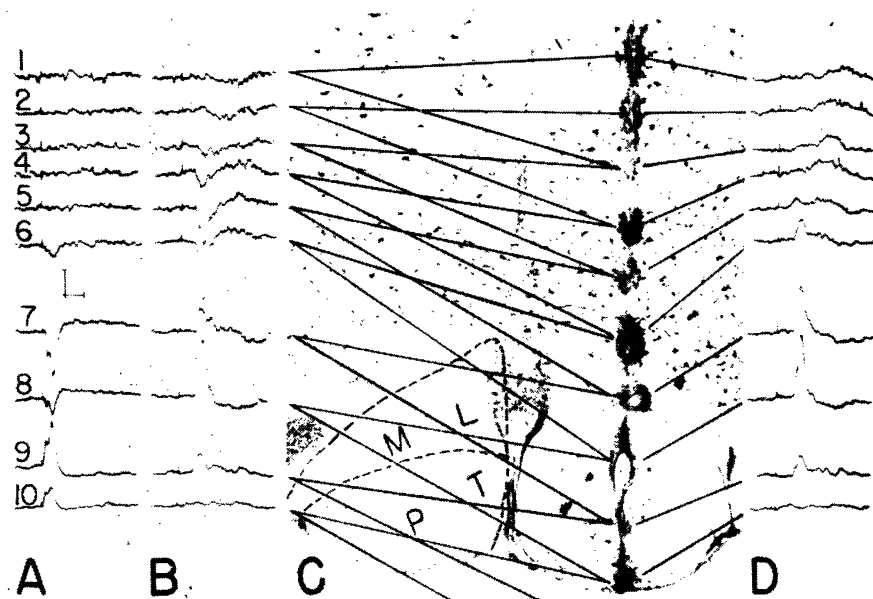


Fig. 1. (A) Bipolar recording of response to motor cortex stimulation. (B) Bipolar recording of response to nerve stimulation. (C) Lines showing recording sites in histological section; the approximate anatomical location of medial lemniscus and pyramidal tract is marked. (D) Monopolar recording of "sensory" responses as in B; upward deflection indicates relative positivity at upper electrode. Calibration 100 μ v and 5 msec.

basilar artery to the pons. Because of the toughness of the medullary pia, the electrode was usually inserted to maximal depth at the start; subsequent records from more superficial points were made at 0.5-mm intervals as it was withdrawn. All electrode tracts were identified histologically using Marshall's technique (3).

The active lead in the medulla always gave a positive response, presumably the consequence of leading from the injured regions of active axons. In every case, the response to cortical stimulus lay superficial to the area responsive to nerve stimulus (Fig. 1). Histological check revealed

that the former identifies the pyramidal tract; the latter, the medial lemniscus. Bipolar differential recording of the potential gradients gave distinctly separate reversals of the cortical and nerve-activated potentials at depths about 1 mm apart (Fig. 1A,8,9 and B,6,7). Both dorsal and ventral to the maximal lemniscus response, similar but rapidly diminishing smaller potentials could be recorded.

The small "sensory" potential in the dorsal portion of the pyramid (Fig. 1B,9) was often entirely absent at the trapezoid body where auditory fibers intervene between the diverging medial lemniscus and corticospinal tract. Nerve and cortical stimuli were given together and at various intervals when the electrode was properly situated to record both responses. The potentials always added algebraically without interaction. Reduction of shock strength at both stimulus sites resulted in a uniform fall in response at all recording positions.

In the cerebral peduncle where the medial lemniscus has moved away from the pyramidal fibers, a cortical stimulus elicited a large potential, while a nerve shock that fired somatosensory cortex produced no response (Fig. 2).

The latency of the nerve response in the medulla was about 4 to 5 msec. With monopolar recording (sometimes also with the bipolar leads), there was a subsequent slow wave at 8 to 10 msec (Fig. 1D). This could be recorded through most of the medulla and at times even in the overlying pool. It was often irregular in form, and in the dorsal reticular substance there were some related spike potentials. The late potential may be

assigned to a diffuse response of the medullary gray. Occasional irregularities in shape when this wave was recorded from the pyramidal tract suggested that a descending cortical reflex response to the sensory volley might also be present. Two attempts to test this hypothesis by severing the tract at the pons were not entirely satisfactory; the response pattern did not change significantly.

From the data presented, it is reasonable to assign the potentials recorded from the pyramidal tract after sensory stimulation to current spread from the subjacent medial lemniscus. The misinterpretation of Brodal and Kaada may be explained by their exclusive reliance on monopolar recording, and by their lack of precise anatomical control of electrode placement. The results obtained do not negate the histological demonstration of ascending axons in the pyramid, although the morphological data have been questioned elsewhere (4). At this time there is no reason to qualify Sherrington's functional definition of the pyramidal tract as a descending internuncial pathway (5, 6).

WILLIAM M. LANDAU

Division of Neurology and
Electroencephalography, Washington
University School of Medicine,
St. Louis, Missouri

References and Notes

1. A. Brodal and B. R. Kaada, *J. Neurophysiol.* 16, 565 (1953).
2. A. Brodal and F. Walberg, *Arch. Neurol. Psychiat.* 98, 755 (1952).
3. W. H. Marshall, *Stain Technol.* 15, 133 (1940).
4. P. Glees and W. J. H. Nauta, *Monatsschr. Psychiat. Neurol.* 129, 74 (1955); F. Morin, *Am. J. Physiol.* 183, 245 (1955).
5. This work was supported in part by U.S. Public Health Service grant B-882.
6. While this paper was in press an abstract with similar conclusions became available: H. D. Patton and V. E. Amassian, *Am. J. Physiol.* 183, 650 (1955).

31 October 1955

Pluto Not a Planet?

The recent announcement of G. P. Kuiper—with the usual fanfare of a sensational magazine, radio, and TV accompaniment that we have come to expect of him—that Pluto might not be an original planet, strikes most astronomers somewhat humorously, coming, as it does, nearly 20 years after the original suggestion to this effect by R. A. Lyttleton.

In the December number of the *Monthly Notices of the Royal Astronomical Society* for the year 1936, Lyttleton published the ingenious suggestion that Pluto might have been a satellite of Neptune and that it and the present big satellite Triton might have gone through a very close approach to each other, with the result that Pluto was removed from Neptune's control and Triton turned around to become the outstanding

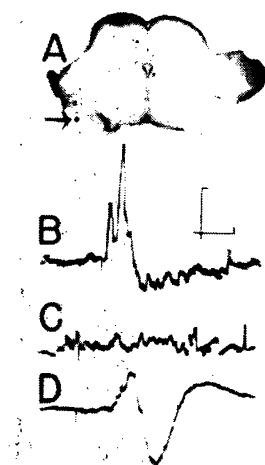


Fig. 2. (A) Recording site in cerebral peduncle. (B) Response to cortical stimulus. (C) Absent response to nerve stimulus. (D) Posterior sigmoid cortex response simultaneous with C. Calibration 100 μ v and 5 msec.

"wrong way Corrigan" of the solar system. But, while Lyttleton even showed how the whole thing could have happened, and also that perhaps it happened not so very long ago, astronomically speaking, it is one of those things we may never be able to prove or disprove.

It is an interesting speculation, and, in fact, at the University of Minnesota, I have regularly discussed this in class since 1937. One might even speculate further, and, as a sort of joke, suggest that if Pluto were once a moon of Neptune it might well have come through Neptune's atmosphere, which contains a lot of methane—and got covered with soot. This is not only appropriate for the god of the underworld but might explain why Pluto gives so little light. From its mass—which is not too certainly determined—we guess that Pluto should be nearly as large as the earth but, from its feeble light, it seems only a little larger than the moon. Of course, there are still many difficulties, the most outstanding perhaps being the large mass of Pluto. Most satellites are something like 10,000 times less massive than their primary (at most). Our moon is only 81.5 times smaller in mass than the earth, but if Pluto has been a satellite of Neptune, its mass is only 20 times smaller than that of its primary.

But all these are wild speculations and I am sure that they must have occurred to hundreds of others. Only scientists, in general, do not rush into print, dusting off old theories and presenting them as new.

WILLEM J. LUYTEN

Department of Astronomy,
University of Minnesota, Minneapolis

22 March 1956

Recombination in Bacteria

I should like to add my comments to those of J. Lederberg [*Science* **122**, 920 (1955)] on the recent news report [*Science* **122**, 278 (1955)] concerning recombination in the colon bacillus. In microorganisms, recombination of hereditary characters involves precisely that and nothing more. Until the mechanism of meiosis has been adequately defined, either by cytological procedures or by chromosome maps, and the existence of a standard meiotic apparatus has been established, it is not possible to determine how recombination has been achieved.

Recombination of hereditary characteristics in microorganisms may be the result of at least six different mechanisms: (i) crossing-over at meiosis, (ii) gene conversion, (iii) transduction, (iv) transformation, (v) misassortment of autonomous extrachromosomal heredi-

tary particles, and (vi) mutation induced by substrate. The last named is particularly important when the recombinants are isolated from a selective medium, as in genetical analysis of the colon bacillus. A seventh possible mechanism of recombination is mitotic crossing-over, but its demonstration depends on assumptions concerning genic stability that have recently undergone drastic revision.

Current explanations of recombination in the colon bacillus assume (i) a normal standard meiotic mechanism and (ii) a single mechanism of recombination, namely, meiotic crossing-over; deviations from the anticipated results are explained in terms of abnormalities of the assumed mechanism. In the absence of tetrad analysis, it seems necessary to withhold judgment in view of the possibility that other recombinatorial mechanisms may be involved.

CARL C. LINDEGREN

Biological Research Laboratory,
Southern Illinois University, Carbondale

19 March 1956

Urea Complexes of Lithium Chloride

The pharmaceutical importance of the urea addition complexes of calcium chloride and calcium iodide was first investigated by Greenbaum (1). A systematic study of the urea addition complexes of the alkaline earth halides has recently been extended by Pande and Bhatnagar (2). By use of the monovariation method (3), complexes of the general formula,



where X is chloride, bromide, or iodide and n is equal to $\frac{1}{2}$, 1, 2, and 4 were found. Because of the similarity between the reactions of lithium salts and the corresponding alkaline earth compounds, the complexes with urea should also be of a similar nature. This was indeed found to be the case.

The complexes between urea and lithium chloride in solution were determined using the monovariation method applied to two physicochemical properties of the system, the relative viscosity and the index of refraction. A total of 23 solutions were prepared, each containing 5.10 ml of 1.967*M* LiCl solution and 0 to 21.00 ml of a 2.00*M* urea solution. Each mixture was then diluted to a total of 100 ml.

The relative viscosity of each solution was measured using an Ostwald viscometer in a water bath at $25 \pm 0.02^\circ\text{C}$. The index of refraction of each solution was measured with an Abbe refractometer at the same temperature.

The results of the viscosity measurements of each solution are shown in Fig. 1. The index of refraction curve is not shown but was of a similar appearance. From the curve, a maximum point of viscosity indicates a complex between urea and the lithium chloride in solution. It can be seen that there are three maxima in the curve, corresponding to complexes having the following compositions $\text{LiCl} \cdot \text{CO}(\text{NH}_2)_2$, $\text{LiCl} \cdot 2\text{CO}(\text{NH}_2)_2$, and $\text{LiCl} \cdot 3\text{CO}(\text{NH}_2)_2$. A complex having the composition $2\text{LiCl} \cdot \text{CO}(\text{NH}_2)_2$ was not found, as was the case with the barium halides.

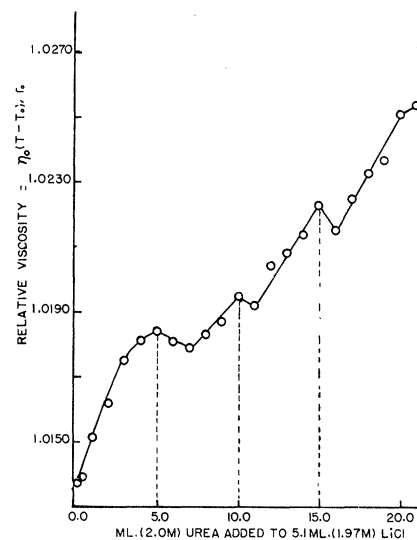


Fig. 1. Viscosity of the urea-lithium chloride system.

The nature of the bonding in these complexes is not known but is presumably an ion-dipole attraction between the metal ion and the nitrogen atoms of the urea molecule. As a consequence, the complexes are relatively unstable. In the cases where the calcium halide complexes have been isolated in the crystalline state (1, 4), it was found that they were hygroscopic and easily soluble in water but insoluble in organic solvents.

WILLIAM G. MCGAVOCK

Trinity University, San Antonio, Texas

JOHN M. BRYANT*

WESLEY W. WENDLANDT

Department of Chemistry and
Chemical Engineering, Texas
Technological College, Lubbock

References and Notes

1. F. R. Greenbaum, *Am. J. Pharm.* **100**, 600 (1928).
 2. C. S. Pande and M. P. Bhatnagar, *J. Indian Chem. Soc.* **31**, 402, 405 (1954); *Z. physik. Chem.* **203**, 369, 375 (1954); **204**, 183 (1955).
 3. M. R. Nayar and C. S. Pande, *Proc. Indian Acad. Sci.* **A27**, 286 (1948).
 4. Y. Okuda and K. Fujikawa, *J. Tokyo Chem. Soc.* **40**, 404 (1919).
- * Work performed at Trinity University as partial fulfillment of the requirements for the M.S. degree, 1955.

7 November 1955