ingly difficult and, as yet, but imperfectly understood, namely, radiation, both solar and terrestrial, and absorption, including their various effects on the atmosphere. Here the author is dealing with one of his favorite subjects and the result is correspondingly excellent, even though his widely distributed castigations may seem a trifle severe. Then the subject changes to a discussion of the motions, both horizontal and vertical, of a compressible fluid over a rotating sphere and the interactions of passing discrete currents of this fluid upon each other. These problems give pause to the beginner, but they have the advantage of being more fully solved than almost any others in the whole field of meteorology.

Following this are 61 pages on atmospheric turbulence and associated problems, pages which fortunately, and despite their importance, not many practical meteorologists have to master before entering upon their work. Sixteen pages of mathematical reasoning are given to the transformations of energy in the atmosphere. After this the problems of the genesis and maintenance of the cyclone and anticyclone are discussed. This leads quickly to a consideration of discrete air masses and their fronts, in which all that has been done in "air mass analysis" is given a friendly but unstampeded judgment.

The last chapter discusses with equal frankness the general circulation of the atmosphere, about which, physical geographies and elementary works on meteorology to the contrary notwithstanding, astonishingly little is really known.

A few brief but useful tables make up an appendix, and there is a good index. But what of the errors? There aren't any, worth mentioning.

Many a meteorologist will find this book hard reading, but it is so valuable that he should keep it, like Shakespeare and the Bible, close at hand for appearance's sake, at least.

W. J. HUMPHREYS

U. S. WEATHER BUREAU

SPECIAL ARTICLES

FURTHER OBSERVATIONS ON THE POTENTIAL RHYTHMS OF THE CEREBRAL CORTEX DURING SLEEP

IN a previous communication[1] we have reported certain observations made on human subjects whose brain rhythms have been recorded as they appeared between two electrodes placed on the forehead and crown of head. The record of each subject was usually of seven hours' duration, starting as the subject retired. Customarily the subject's pulse rate, respiration rate and any movements made during the night were also recorded. The outstanding discovery was that trains of regular waves of a particular character could be produced when certain sounds were made while the subject was asleep. The same sounds produced no waves when the subject was awake.

Since the previous communication the installation has been extended so that the rhythms from two parts of the head could be recorded simultaneously by means of two matched amplifiers and recorders. These were checked, prior to each run, against sinusoidal potentials of known frequency and voltage. The three electrodes were placed on the midline: one on the high forehead, the second on the crown and the third on the occiput. The region between the first and second electrodes will be referred to as the "front area" and that between the second and third electrodes as the "back area." Eleven persons ranging in age from 5 to 48 years have been investigated either during an all-night sleep or an after-lunch nap.

[1] SCIENCE, 81: 597, 1935.

We have distinguished at least four characteristic types of waves which have appeared in both areas. Fig. 1 shows typical examples, which we have named for convenience of description (E) spindles, (D) trains, (C) saw-tooth, (B) random.

When an adult subject first retired for the night, "trains" appeared in both areas in great number. These continued after the subject had fallen asleep for some time, gradually becoming less numerous, shorter and of lower amplitude, finally changing to the "random" type. This change is illustrated in Fig. 1-A. If the subject awakened during the night the "trains" usually appeared at once and then changed gradually to the random type as he sank into deep sleep again. The impression was gained that a change in the level of consciousness was connected with this change in type of wave. Children and young persons in very deep sleep showed the random type predominantly with only occasional trains or spindles. A sudden change from the random type to trains could be caused by speaking to a drowsing subject. In every case observed both areas have changed in the general type of wave simultaneously.

On the other hand, when trains and spindles were appearing in both areas, the individual trains and spindles usually showed no correspondence in time of appearance. They might appear in the front area with none in the back, and *vice versa,* or they might appear simultaneously in both but last for a shorter time in one, or their frequency might be different. All possible degrees of correspondence as regards

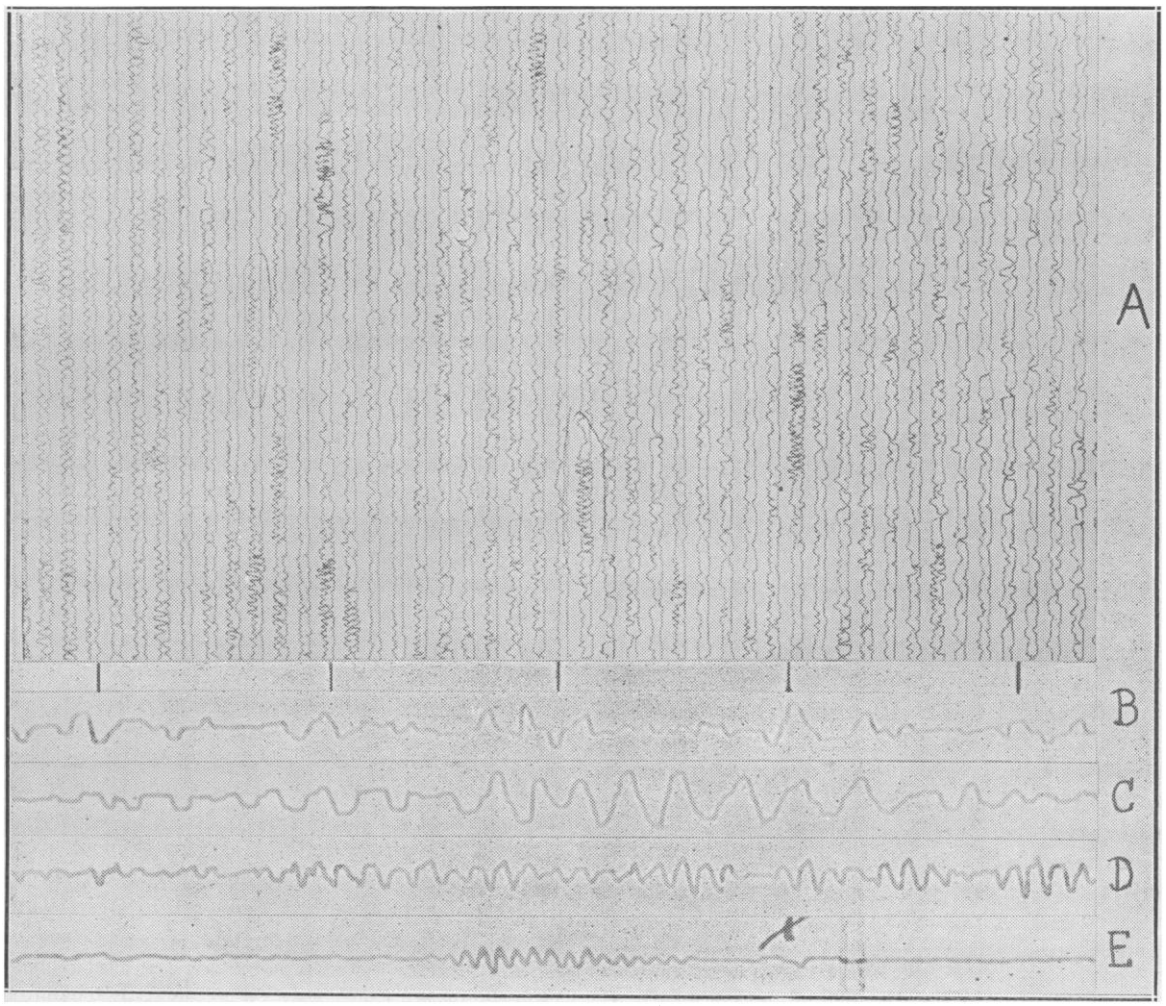

FIG. 1. A shows brain potential rhythms from two areas of the cortex. The photograph is a small section from a large continuous record taken on an eight-foot drum, 45 inches in circumference, revolving once a minute. The section shows only eight seconds out of each minute (time, in seconds, at left). Read from top to bottom and from left to right. The left line in each pair gives the potentials from the front area of the cortex, the right line the back area. B, C, D, E. Enlarged photographs of four types of waves which we have called (B) "random," (C) "saw-tooth," (D) "trains," (E) "spindles." Time in seconds at top of B (read left to right). Note in A the change in character of the waves from "trains" and "spindles" to the "random" type over the forty-five minute period. Note also how often the "trains" appear in one area and not in the other.

amplitude, frequency and time of appearance were observed between the rhythms from the front and back areas. This is also illustrated in Fig. 1-A. These observations seem to indicate that the individual rhythms arise in different regions of the cortex, but that the gradual change of type from "trains" to "random" is dependent on a change connected with the cortex as a whole.

We found also that the trains appearing as the result of sound stimuli sent to a sleeping subject were much more marked in the front area than in the back. This was at first unexpected, as the temporal cortical end station for auditory stimuli is as near or nearer the back area as the front, but would be readily understandable if trains were due to a change in level of consciousness in the front area, brought about by the sound disturbances.

While the foregoing observations apply to the great majority of the subjects so far studied, we have found a number whose brain rhythms were of quite special types. So far we have observed the "saw-tooth" type of wave only in children and only when they have just fallen asleep. A peculiar type of wave of large amplitude lasting from two to four minutes has appeared in subjects during deep sleep without any movement or change in pulse or respiratory rate. In animals (dogs, rats) types of waves have appeared quite different from any observed on human subjects. Deep anesthesia stopped the waves in a rat completely. They began again when the

anesthesia became less deep. We will reserve the discussion of these matters for a later paper.

ALFRED L. LOOMIS
E. NEWTON HARVEY
GARRET HOBART

LOOMIS LABORATORY
TUXEDO PARK, N. Y.

INTRANASAL OR GASTROINTESTINAL PORTAL OF ENTRY IN POLIOMYELITIS[1]

IN a recent issue of SCIENCE, Faber[2] reported data on 57 monkeys in which poliomyelitis virus had been introduced intranasally. His facts are admitted. He concluded that, since the virus passed through the brain stem and spinal cord from above downward, and because the virus was found present in all levels of the cord just before paralysis appeared, no explanation of the usually earlier and greater paralysis of the lumbar area was possible other than that offered by Fairbrother and Hurst,[3] *i.e.*, that the virus usually involves the anterior horn cells of the lumbar area because of greater susceptibility of these cells. Shall we deduce, then, that in cases with only bulbar poliomyelitis or only cervical horn cell involvement or only seventh nerve palsy the lumbar motor areas of these patients are not susceptible, or that in these particular patients the motor cells of those areas involved are more susceptible? How does one explain why the cervical enlargement is often hit later, *i.e.*, after the lumbar area has already become involved? With the theory of susceptibility, one would have to think that there are degrees of motor cell susceptibility depending upon location and involvement. If the cells of the lumbar area have such a high degree of susceptibility they should respond promptly to local injection of virus into the lumbar area of the experimental animal. But they do not thus respond, for even when the virus is injected directly into this enlargement, there is a delay before the disease is produced.[4]

I feel that in the monkey the disease results when two factors combine and the combination material destroys motor cells. One of these factors is monkey cord virus and the other is the toxic material produced in the animal's gut. In the human being the causative agent usually enters the digestive system "ready made," *i.e.*, it is already combined and capable of producing cell destruction immediately. All the vagaries of the disease both in the *M. rhesus* monkey and the human being, even the reason why the lumbar area seems usually to be first involved, are easily understood in the light of this theory. Much experimental and clinical work has been done that points to a relationship between the gastrointestinal tract, the sympathetic nervous system and the production of the experimental disease.[5] But ignoring this evidence, what else can be said? When virus is given either intracerebrally or intranasally, the experimental animal usually gets quadriplegia and dies. Poliomyelitis is thus produced, but even though this is poliomyelitis, *it is not the kind of poliomyelitis that is seen in the human being.* Our objective should be not only to produce the disease, but to produce it as it appears clinically. If we can produce poliomyelitis in the experimental animal in the same way as it is seen in the human being, it is plausible to suppose that the route taken to produce it in the monkey might be the same as that taken by the virus in the human being.[6,7]

Faber further states that the theory of gastrointestinal invasion has few remaining advocates since the very convincing study of Clark, Roberts and Preston.[8] The facts shown by these workers do not rule out the gastrointestinal tract as a probable portal of entry. Clark, *et al.*, put virus into the intestines of monkeys that are normally not susceptible to poliomyelitis, and they did not produce the disease. When one realizes that poliomyelitis virus acts almost like an enzymic catalyst with an obligate affinity for gray fibers and that the natural disease can be produced only when the axis cylinders of the gray fibers contact the virus and absorb it, it is easy to understand why these workers did not produce the disease. When I caused the virus to come in contact with these fibers, the disease was easily produced.[6]

The fact that intranasal inoculation is unsuccessful if the connection between the gray-fibered olfactory nerve and central nervous system is severed is interesting. However, transection of the olfactory nerve would be of importance only if such section made it impossible for us to produce the disease by the gastrointestinal tract.

If our theory as to the production of paralysis in the monkey is right then it is obvious that passive immune serum of great value can not be obtained by injecting monkeys with the virus alone. A high-titered antiserum was rapidly produced by injecting

[1] From the Department of Pediatrics, Western Reserve University, and the Division of Contagious Diseases, City Hospital, Cleveland, Ohio.

[2] H. K. Faber, SCIENCE, 82: 42, 1935.

[3] R. W. Fairbrother and E. W. Hurst, *Jour. Path. and Bact.*, 33: 17, 1930.

[4] J. A. Toomey, *Proc. Soc. Exp. Biol. and Med.*, 32: 1185, 1935.

[5] *Idem, Annals Int. Med.*, 8: 854, 1935. *Jour. Prev. Med.*, 6: 379 and 397, 1932. *Proc. Soc. Exp. Biol. and Med.:* 30: 1082, 1933; 31: 502, 680, 702, 1015, 1934; 32: 423, 869, 1935. *Am. Jour. Dis. Child.:* 45: 1211, 1933; 46: 730, 1933; 47: 573, 1934; 48: 30, 1934. *Jour. Inf. Dis.*, 54: 74, 1934.

[6] J. A. Toomey, *Proc. Soc. Exp. Biol. and Med.*, 31: 680, 1934.

[7] *Idem, Proc. Soc. Exp. Biol. and Med.*, 32: 628, 1935.

[8] P. F. Clark, D. J. Roberts and W. S. Preston, Jr., *Jour. Prev. Med.*, 6: 47, 1932.