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21 March 1960

I have read Firsoff's criticism of my earlier note and I find that no changes are necessary either in my results given there or in my comments relating to an earlier note by Firsoff.

Costain, Elsmore, and Whitfield have published the details of their method of estimating the maximum density of the lunar atmosphere from the radio observations of the lunar occultation of the Crab nebula (*1*). Firsoff is further in error when he states that Rishbeth and Little have observed a response from the radio source associated with Kepler's nova when the source was 3′ inside the limb of the moon. It is explicitly stated in the Rishbeth and Little paper that it was the visible remnant of Kepler's nova that lay 3′ inside the limb of the moon. It is further explained by Rishbeth and Little that the best available position would put the radio source much nearer the limb than the optical remnant.

It seems clear that the kinetic temperature of particles rebounding from the surface of the moon will depend very little on the radiation temperature of the moon, as assumed by Firsoff. Hence, the lunar atmosphere will undoubtly not be at the surface temperature of the moon but will approximate the temperature of the interplanetary gas. The issue is somewhat obscured by Firsoff's manipulation of rather well established astronomical constants such as the mass and radius of the moon, and because he arbitrarily cuts off the lunar atmosphere 1000 km above the lunar surface. Thus it happens that the particle density of $10^7/cm^3$ does not follow from Firsoff's assumptions in a straightforward manner. Firsoff's assumptions given in his original report (which include the claim that the interplanetary density of heavy particles with molecular weight 25 is about $10^3/cm^3$) lead to an atmospheric density at the lunar surface of about $6 \times 10^{17}/cm^3$, a value which is too high by orders of magnitude.

The concept of a critical level or base of an exosphere has been in the literature for years (*2*). A simple definition of the critical level may be given as the region in an atmosphere where a characteristic mean free path is equal to the local scale height. Firsoff's "important factors" (i) and (iii) follow immediately from this definition, and (iii) has also been discussed by Öpik, who considers how the escape rate decreases when the thickness of an atmosphere becomes less than one mean free path. Firsoff's point (ii) is still incorrect, and he is further guilty of quoting van de Hulst out of context. The sentence quoted by Firsoff together with the very next sentence read as follows: "In an isothermal gas the light electrons have a tendency to segregate from the heavier protons. Long ago Pannekoek and Rosseland showed that a minute separation of charge suffices to create an electric field **E** that compensates this tendency by drawing the protons up and the electrons down."

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28 March 1960

Electrographic Evidence of Impaired Brain Function in Chronically Anxious Patients

Abstract. In a study of cerebral function by electroencephalographic techniques the following observations have been made. (i) In intact subjects, repeated stimulation with bright light causes a predictable change (alpha blocking) in the electroencephalogram, whereas repeated auditory stimulation does not. (ii) If, however, an auditory stimulus is presented repeatedly just before the visual stimulus, the sound temporarily but predictably acquires the property of the light to suppress the alpha activity. (iii) This linkage between sound and light occurs much less frequently in human subjects with known amounts of structural brain damage. (iv) A similar electrophysiological defect, implying impairment of brain function, occurs in patients showing severe anxiety during prolonged periods of difficulty in over-all adaptation.

While it is obvious that prolonged periods of poor life adjustment linked with anxiety impair the ability of an individual to function at his most effective level, the demonstration of a defect in brain function by electrophysical means has been difficult to obtain. Studies of the microscopic structure of the nervous system have revealed no significant changes in the brains of persons suffering from the common neuroses and psychoses; studies of the function of the brain as recorded in the resting electroencephalogram have shown only slight, if any, deviation from the normal; studies of the highest integrative functions as evidenced by behavior, attitudes, and thought are limited by the wide variability of motivation and cooperation of such patients.

Early study of electroencephalograms of human subjects showed that repeated stimulation by a bright light predictably provoked disappearance of the alpha activity whereas repeated presentation of an auditory stimulus soon ceased to have any effect on the electroencephalogram. In the 1930's it was noted (*1*) that after a subject's brain waves failed to show a response to an auditory stimulus, if the sound were then paired with a bright light and made to routinely precede the light by a fixed time interval, the sound itself might suppress the alpha activity just as the light had. Such a phenomenon has been known as a temporary cerebral connection or a conditioned cerebral response (it being understood that the phenomenon does not fulfill the criteria for Pavlovian conditioning). These conditioned cerebral responses, for reasons not yet established, are poorly sustained in man.

Since the development of conditioned cerebral responses is a measurable manifestation of brain function which demands minimal cooperation of the subject, and since such responses were found to occur much less frequently in human subjects with impaired function due to brain damage resulting from loss of known amounts of the cerebral hemispheres (*2*), it seemed appropriate to study this phenomenon in patients exhibiting sustained and severe anxiety. Studies were carried out on 23 "control" subjects without evidence of central nervous system dysfunction and on 15 patients who exhibited the consequences of long unresolved adaptive difficulties. They expressed severe anxiety most of the time and conspicuously showed many signs of it. They complained of thinking difficulties, low frustration tolerance, and fatiguability and were to some degree depressed and hostile. Adaptive and compensatory devices were few, poorly developed, and poorly maintained. They were free of the effects of drugs, of infectious, degenerative, neoplastic, or traumatic disease, and of other evidence of gross structural defects of the cerebral hemispheres. Their mean age was 35 as compared with a mean age of 30 for the control group.

The subject was seated in a quiet and semidarkened room and the test procedure was described to him in general terms in order to allay apprehension. Light stimulation was provided by a 150-watt frosted bulb, with a white reflector placed approximately 12 in. from the subject's eyes. Auditory stimu-

lation was produced by a Beltone audiometer, which delivered a sound to one ear at 500 cy/sec and approximately 40 db above the level of audibility. The brain waves were recorded with a Grass model 3 electroencephalograph, frontal, central, and occipital or frontal, temporal, and occipital electrode placements being used. All subjects showed an alpha rhythm present at rest for at least 50 percent of the test period.

The subject was first presented with the light stimulus of 3-sec duration several times to ascertain that light provoked the usual suppression of alpha activity over the occipital regions. This was followed after a brief interval by a sound stimulus of 4-sec duration, repeatedly presented until at least five successive tone presentations failed to suppress alpha activity. The subject was then exposed to paired sound and light stimuli 50 times at irregular intervals, the sound appearing 0.8 to 1.0 sec before the light, with both continuing simultaneously for 3 sec. The interval between the sound and light was automatically timed and remained constant in each individual.

The resting electroencephalogram was evaluated, and the number of conditioned cerebral responses appearing in 50 presentations was ascertained by one of us (C. E. W.) without knowledge of whether the record was obtained from a control subject or from a patient. A conditioned cerebral response was considered to have occurred when the alpha rhythm was obliterated or strikingly depressed following the presentation of the tone and before the appearance of the light.

The resting electroencephalograms of the two groups were indistinguishable. In 23 control subjects the number of conditioned cerebral responses appearing in 50 paired sound-light stimulations ranged from 5 to 16, with a mean of 10.9, a median of 11, and a standard deviation of ± 3.25. In the 15 patients the number of conditioned cerebral responses ranged from 2 to 14, with a mean of 4.8, a median of 4, and a standard deviation of ± 3.28. The difference between these two means is statistically significant, the likelihood of their occurring by chance being less than 1 percent.

These observations demonstrate that the ability to develop conditioned cerebral responses is significantly impaired in a group of patients showing prolonged difficulties in adaptation and severe anxiety when compared with the ability to develop such responses in a group of subjects without obvious impairment of nervous system function. These data are of interest from several standpoints. First, they demonstrate, in subjects suffering from a so-called "functional" nervous system disease without evidence of gross structural abnormalities, failure of the brain, as indicated by its electrical activity, to respond in a normal fashion to external stimuli.

Second, such observations may perhaps help to explain why electroencephalographic studies have been largely disappointing in showing impairment of the highest integrative functions of man. Numerous investigations have revealed only minor differences between the electroencephalograms of normally functioning subjects and those of patients with severe neuroses and psychoses. Perhaps more sensitive methods of measuring responsiveness in the electroencephalogram may demonstrate other evidences of impairment in the "functional" disorders of the brain.

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9 February 1960

Chromosome Number of the Chimpanzee, Pan troglodytes

Abstract. The chromosome numbers of nine chimpanzees (*Pan troglodytes*) have been determined by the bone marrow technique. The diploid number in this species is 48, with a probable XX-XY sex chromosome constitution.

In 1940, Yeager, Painter, and Yerkes (*1*), from an examination of spermatogonial diakineses, reported the diploid chromosome number of a chimpanzee to be 48. No further reports of chromosome numbers in the anthropoid apes have been presented, nor has there been confirmation of the original report since the development of improved cytogenetic techniques. The latter point is of particular interest because of the recent revision of the human chromosome number from $2n = 48$ to $2n = 46$ (*2*).

We have been able to ascertain the chromosome numbers of nine individuals of *Pan troglodytes* (seven males and two females), which were sacrificed because of infection with tuberculosis (*3*). Anesthesia was induced with ether and maintained for 3 to 5 hours with Nembutal. This period afforded sufficient time for the action of the mitotic poisons used: either colchicine, 0.25 mg/kg injected intraperitoneally, or Colcemid (*4*), 6 mg per animal injected intravenously.

Bone marrow was obtained from the proximal third of the humerus (in one case from the radius); it was suspended in 1.12 percent sodium citrate at 37°C for 20 to 30 minutes, centrifuged, and either fixed in cold alcohol–acetic acid and prepared by the Feulgen squash method (*5*) or fixed in 50 percent acetic acid and stained with lactic-acetic orcein and then squashed (*6*). Counts of suitable metaphase plates were made directly from the preparations.

Table 1. Distribution of chromosome numbers.

Specimen		Number of cells scored for various chromosome numbers				
		45	46	47	48	48+
P53	♀	1	1	3	10	1
P60	♀	1	1	4	18	0
P66	♂	1	2	3	8	0
P74	♂	0	0	1	8	1
P79	♂	0	1	4	10	0
P102	♂	0	0	0	5	1
P108	♂	0	0	1	7	1
P118	♂	0	0	1	4	0
N613	♂	0	5	11	58	4
Total		3	10	28	128	8

Table 1 presents the distribution of chromosome numbers obtained in the nine individuals and confirms the observation of Yeager, Painter, and Yerkes (*1*) that the diploid chromosome number in this anthropoid ape is 48.

Figure 1 is a metaphase plate from a female individual (P53) prepared by the Feulgen method.

Comparison of the chromosomes of the sexes suggests an XX-XY sex chromosome constitution, the X being a moderately large metacentric and the Y probably a very small metacentric chromosome. No evolutionary significance can as yet be attributed merely to the difference in chromosome number between man ($2n = 46$) and the

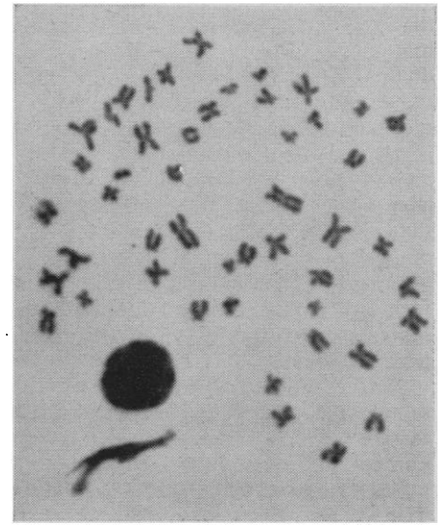

Fig. 1. Metaphase plate from a *Pan troglodytes* female, prepared by the Feulgen method: $2n = 48$.