at posterior cerebral locations which are not grossly pathological (Jasper, 1949; Penfield & Jasper, 1954). The alpha rhythm under these conditions is of larger amplitude than the alpha rhythm of EEG obtained through scalp leads. This difference in amplitude reflects shunting by these surroundings of the skull. All electrocortical activity suffers shunting by the intact CSF, dura, and brain to the same extent. Whether the brain pulsates mechanically or not makes no difference in the waveform of the alpha rhythm. The possible artifact in electroencephalography which Kennedy’s model implies therefore seems non-existent.

REFERENCES


(Received June 17, 1960)

**ON THE ORIGIN OF THE EEG ALPHA RHYTHM**

IAN OSWALD

*University of Edinburgh*

In reporting some ingenious experiments Kennedy (1959) suggests that oscillating fluctuations of electrical potential recorded in the EEG may be artifacts resulting from the mechanical (arterial) pulsation of a charged gel in a rigid case. Kennedy confines his attention to the alpha rhythm and to a rhythm which he calls the anterior temporal rhythm, originally claimed to be associated with thinking (Kennedy, Gottsdanker, Armington, & Gray, 1948), although, to the best of my belief, independent confirmation is still lacking.

Kennedy proposes that variations in the amount of human alpha rhythm result from variations in local blood flow in the brain, as a result, he implies, of the activity of the autonomic nervous system. Brain waves were shown to vary in association with changes of cerebral blood flow by Ingvar (1955) and Ingvar and Söderburg (1956), following reticular formation stimulation, but the blood flow changes always occurred several seconds after the electrical changes, just as the peripheral vasoconstriction which follows a startling stimulus has a latency of 1-5 seconds, compared with 0.3 seconds for alpha blocking in man. It is possible, by repetitively varying what may be called “attention,” to cause fluctuations between the alpha rhythm picture of wakefulness and the nonalpha picture of light sleep, or alternatively, the nonalpha picture of high level wakefulness, in an exact and rhythmic fashion, with rhythms having periods of 1-2 seconds (Oswald, 1959b, 1960). Vaso-motor responses are too slow to account...
for such accurate, rapid, and rhythmic alterations.

The alpha rhythm can be replaced by high voltage slow waves, not only during the cerebral vasoconstriction of hyperventilation, but also during the cortical hyperaemia and congestion of encephalitis. If a patient with slow waves owing to encephalitis hyperventilates, causing cerebral vasoconstriction, the slow waves become more marked; they do not change to alpha rhythm during the time of some "optimum" vasomotor state.

The alpha rhythm disappears at the onset of sleep, and Kennedy quotes Shepard's work during the early years of this century to support the view that increase of cerebral blood flow causes the change of the EEG. Other workers, before and since, obtained contrary results but, using modern techniques, Mangold, Sokoloff, Conner, Kleinerman, Therman, and Kety (1955) did find evidence of a small but significant increase of cerebral blood flow. Central nervous system responsiveness is lowered in sleep, including the responsiveness of the respiratory centre to carbon dioxide, the arterial concentration of which rises during sleep (Bellville, Howland, Seed, & Houde, 1959; Magnussen, 1944). Carbon dioxide is a very potent vasodilator and it is believed that this is responsible for the increased cerebral blood flow in sleep (Robin, Whaley, Crump, & Travis, 1958). The disappearance of the alpha rhythm at this time is preceded by a slowing of the rhythm. Yet when carbon dioxide is inhaled during wakefulness, causing a big increase of cerebral blood flow (Kety & Schmidt, 1948), the alpha rhythm does not slow but becomes faster (Gibbs, Williams, & Gibbs, 1940).

Inhalation of a low oxygen-high carbon dioxide mixture sufficient to produce an enormous increase in cerebral blood flow (Kety & Schmidt, 1948) can be without effect on the EEG frequency distribution (Holmberg, 1953).

The Figure 2 of Kennedy's paper apparently shows the artificial "alpha rhythm" fluctuating in amplitude at the rate of the "pulse." There are theoretical reasons for believing that this might occur in man for quite different reasons, and I observed a case in which awareness fluctuated with the arterial pulse (Oswald, 1959a). Subsequent attempts of mine, using superimposition photography, to demonstrate fluctuation of alpha "envelope" amplitude with the pulse met with no success.

Kennedy claims that a hole in the skull will greatly modify or abolish the alpha rhythm. Although he quotes the writings of Jasper, he does not mention the recording of alpha rhythm from the exposed brain of the conscious human, shown by Penfield and Jasper (1954, p. 187). Kennedy's crucial experiment remains unconvincing, for he shows us but two selected parts of the EEG record from his subject, in each of which only a couple of seconds of eyes-closed record are shown. Had he presented hundreds of such examples, completely unselected, to an independent observer denied all knowledge of the presence or absence of the "damping mechanism," that observer could then have made judgments as to the degree of alpha rhythm present, and we might have been in a better position to judge the reliability of the phenomenon in question.

According to Kennedy the alpha rhythm should be extremely sensitive to changes of cerebrospinal fluid pressure. A simpler crucial experiment lies in the examination of the effects of variations of this pressure in normal people. Over a number of years, and for a variety of reasons, I have studied alpha rhythms from the same individuals in both the upright and the prone positions and have never noticed any difference in the alpha rhythms, despite the fact that the cerebrospinal fluid pressure within the skull varies considerably with posture. A further simple, deliberate means of testing Kennedy's hypothesis was provided by jugular vein compression (Queckenstedt's maneuver) and also forced expiration against a closed glottis. These procedures both cause a sudden, large rise of cerebrospinal fluid pressure within the skull. These maneuvers, when first attempted with two normal subjects, caused alpha blocking, but when they were re-
peated half a dozen times, so that the subjects became used to them, no change of alpha rhythm was to be seen.

It would be of interest to learn Kennedy's views on the "following" of the human occipital EEG rhythms at the frequencies of a flickering photic stimulator. In some persons these rhythms may follow faithfully the frequency of the flicker from 2 to 20 cycles per second.

If I follow Kennedy correctly he does not imply that all EEG waves could be attributed to the phenomenon he has demonstrated. Indeed I suspect he would be hard put to it to explain the vast quantity of observations made in recent years on the brains of cats lacking major portions of their calvaria, or that most striking feature of the human EEG during medium depth sleep, namely the K complex, with its short latency and composite pattern of slow and fast waves following a sensory stimulus. It would then be necessary to claim that the human alpha rhythm is a special case.

As I fail to find Kennedy's arguments convincing, I shall continue to believe that the alpha rhythm comes and goes in relation to increased alertness (Oswald, 1957) and especially visual alertness (Oswald, 1959c) on the one hand, and light sleep on the other, by reason of mechanisms which embrace EEG phenomena as a coherent whole.

REFERENCES


OSWALD, I. The EEG visual imagery and attention. *Quart. J. exp. Psychol.*, 1957, 9, 113-118.

OSWALD, I. A case of fluctuation of awareness with the pulse. *Quart. J. exp. Psychol.*, 1959, 11, 45-48. (a)


OSWALD, I. The human alpha rhythm and visual alertness. *EEG clin. Neurophysiol.*, 1959, 11, 601. (c)


(Received August 16, 1960)