Beta Brain Waves as an Index of Alertness

Abstract. Reaction times of human subjects are reliably shorter when the signal to respond is given during spontaneous low voltage, fast (beta) brain waves than they are when the signal is given during spontaneous alpha waves. The mean difference of 12 milliseconds is, however, trivial in comparison to the advantage to be expected from forewarning.

The alpha rhythms of Berger are a reliable, although in some persons and under some conditions not infallible, index of relaxed waking; the beta rhythms are typical of alertness and active response (*1*). Further, the alpha cycle, at least, reflects an excitability cycle which has been demonstrated by thresholds of cortical responsiveness (*2*), reaction times (*3*), and accuracy of perception (*4*)—all three showing that peak excitability occurs with the depolarization or surface-negative phase of cortical activity. A similar excitability cycle for the high-frequency beta waves has been hypothesized as a possible explanation of the associated alertness. The speculation is that peak excitability periods recur so rapidly as to be effectively continuous (*5*).

Reaction time, too, is an index of alertness, and has been known for a long time to be a function of the duration of the foreperiod—the interval between "ready" and "go" (*6*). The optimum foreperiod has been shown to be 1 to 2 seconds. Perhaps beta waves are invariably present during this optimum foreperiod, and not always present during longer or shorter foreperiods, but this conjecture has not been studied adequately. Lindsley, however, has reported that in a study of reaction time in which one of the test conditions was omission of the "ready" signal—essentially a condition of a long and variable foreperiod—he found no difference between the trials in which his subjects, when signaled, were showing alpha activity and the trials in which they were showing beta activity (*5*). If this finding can be verified, then one may question whether alpha and beta waves per se are of differential importance to brain functioning, although they could still be useful superficial indices.

In the present study the reaction times of 20 young men and women, who were asked to press a button in response to a light flash, were measured. The flash was presented without forewarning and during times when the subjects were exhibiting spontaneously either alpha or beta waves. The experiment was conducted in a darkened, quiet room. During the 30 minutes in which the subjects were becoming accustomed to the dark, they were fitted with electrodes placed bilaterally over the visual, motor, and frontal cortical areas, and with a muscle electrode, for recording the onset of contraction, placed over the digitorum profundus of the dominant arm. Brain activity of the right visual cortex was led off from the amplifier of the electroencephalograph and fed simultaneously into a monitoring oscilloscope and a trigger mechanism (*4*), which provided an electronic feedback system. The system enabled the experimenter to use the subject's brain rhythms to trigger stimuli during any preselected phase of any preselected frequency of the brain rhythm. Such frequencies used for both alpha and beta waves were the dominant ones of each subject, individually. The preselected phase settings were aimed at ensuring that the neural activity from the retina arrived at the occipital cortex coincidentally with peak cortical excitability. The light was set to emit a 10-μsec flash 40 msec prior to peak cortical excitability; this relation can be seen in the stimulus artifact in the upper right-visual-to-vertex trace of Fig. 1.

We recorded activity of the six cortical areas, muscle activity of the digitorum profundus, signal flash, and closure of the subject's response switch (Fig. 1). Reaction times were scored for those trials in which the signal flash was unequivocally within an interval of alpha or beta activity, as called for on a predetermined random schedule. Forty scorable responses were obtained for each subject for each brain wave pattern.

The mean reaction time, from signal flash to switch closure, during beta activity was 221 msec, and during alpha, 233 msec. The 12-msec difference is reliable ($F = 35.8$; $df = 1, 19$; $p < .001$). For 18 of the 20 subjects, mean reaction times were faster during beta activity than during alpha. For 14 of the 20 subjects individually, the mean reaction times were reliably different (p's $< .05$); all of these favored beta.

The mean reaction time, from signal flash to the first detectable response of the digitorum profundus, during beta

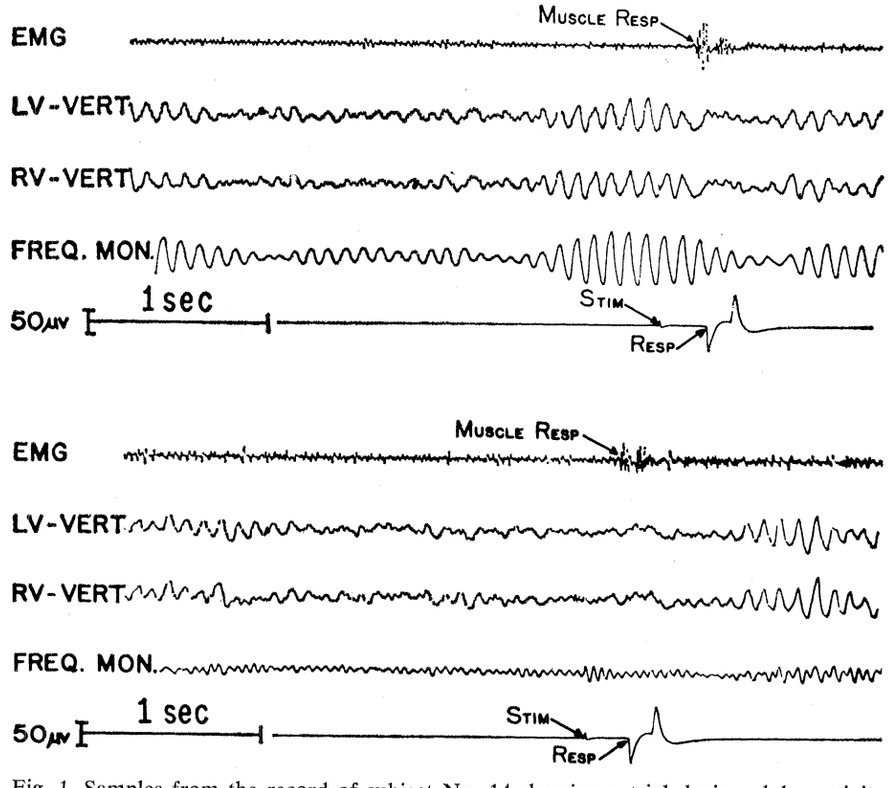

Fig. 1. Samples from the record of subject No. 14 showing a trial during alpha activity (top) and a trial during beta activity (bottom). The electroencephalographic tracings from the frontal and motor cortex electrodes have been trimmed off. EMG, electromyogram; LV-VERT, left visual to vertex; RV-VERT, right visual to vertex.

activity was 160 msec, and during alpha, 171 msec. The difference, 11 msec, is so nearly the same as that for the means for switch closure as to force the conclusion that all of the time advantage of the beta condition was prior to the muscle activity.

A 12-msec difference in reaction latency is not a long time. In conditions similar to ours, Lindsley (*5*) reported a saving of 74 msec by use of a foreperiod of 0.3 to 1 sec compared with the unspecified foreperiod produced by omitting the "ready" signal. Telford (*6*) found that foreperiods of 1 and 2 sec produced reaction times of 90 msec less than foreperiods of 0.5 sec. Woodrow (*6*) reported 60-msec savings when he compared foreperiods of 2 sec with foreperiods of 24 sec. Clearly, the alertness indexed by beta waves, if alertness it is, is not the magnitude of alertness caused by optimal forewarning.

But in spite of being small, the difference of 12 msec is real, and we propose two alternative explanations. The first is that there is no fundamental difference between the alpha and beta reaction times, but that we were more successful in hitting the exact peak of excitability in beta than in alpha. Lindsley's failure to find a difference between spontaneous alpha and beta reaction times is irrelevant here because he did not control for the phase of the excitability cycle. But against this possibility of no fundamental difference is the fact that intraindividual variability of beta frequencies is greater than the variability of alpha frequencies. Our experimental error should thus have reduced, rather than produced, the beta advantage.

The second possibility is that the beta advantage comes from an accumulated effect of interneural facilitation produced by heightened activity in the reticular activating system. There is considerable evidence that the reticular system is responsible for beta waves (*7*) and that it effects interneural facilitation (*5, 8*). We know of no contradictory evidence, but clarification must await an improvement of technique which will insure hitting the peak excitability of both alpha and beta rhythms.

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5 March 1962

Action of Acetylcholine on Bivalve Hearts

Abstract. The isolated hearts of a comprehensive selection of bivalves were tested to determine the distribution of the excitor and depressor effects of acetylcholine. No broad relationships are obvious although some intrafamily uniformity of response exists. The hearts of most species were both excited and depressed, probably by separate actions of acetylcholine. From these data we can conclude that bivalve heart muscle conforms closely to other molluscan muscle in its pharmacological reactions to acetylcholine.

The depressor effect of acetylcholine on most bivalve hearts and its excitor effect on hearts of *Mytilus* species are well known (*1*). Pilgrim has also found that some bivalve hearts are depressed at low concentrations and excited at high concentrations of acetylcholine (*2*). A similar "combination response" has been recorded from hearts of *Spisula solidissima* (*3*) and *Anodonta cygnea* (*4*).

We observed the effects of acetylcholine on the isolated ventricles of 39 species from 20 families, in order to determine the distribution of responses within the class Bivalvia. Most hearts were isolated in water-jacketed baths with tension applied between the auricles (*5*), but the atypical construction of ostreid and anomiid hearts does not allow such an arrangement. In these animals the heart was stretched in the direction of the normal beat. Temperature was controlled throughout each experiment. The perfusion fluid for marine species was either natural or artificial (*6*) sea water. For freshwater animals a 5-percent artificial sea water solution made with $10^{-3}M$ Na_2HPO_4 (*p*H 7.6) was used. The isolated hearts of many species were quiescent and were induced to beat with 5-hydroxytryptamine (10^{-7} to $10^{-5}M$). Heart beat was recorded by means of an isotonic lever on a smoked drum; tension on the lever was varied between 200 and 1000 mg, depending on the size of the heart.

The responses do not appear to be distributed phylogenetically (Table 1). Thus, depressor, excitor, and combination effects occur both in the Pteriomorphia (*7*) and Lamellibranchia and in both primitive forms (*Glycymeris, Cardita*) and highly evolved forms (*Chlamys, Panope*). No depression was observed in the Protobranchiata. Also, no correlation exists between effect and such morphological features as relative size of adductors, symmetry of body and mantle/shell (*8*), or condition of the gills. Finally, there is no relationship between response and physiological or ecological factors such as activity, burrowing and boring, attachment to substrate, mode of feeding, or exposure during low tides.

Although there are no broad systematic relationships correlated with the acetylcholine responses, there is some uniformity within families. The venerids, which have been widely studied, all have low thresholds (10^{-12} to 10^{-8} *M*) and for the most part show no excitation. Pilgrim (*2*) obtained similar results from hearts of *Dosinia anus, Protothaca crassicosta*, and *Chione stutchburyi*. The Mactridae show only the combination response to acetylcholine. This is also true for the family Amphidesmatidae (Mactracea) (*2*). Characteristically, hearts from the family Mytilidae have high thresholds (5×10^{-8} to to 2×10^{-6} *M*) and excitation predominates. Ostreid hearts have equally high thresholds but no typical excitatory response ever occurs. Frequency always decreases but amplitude changes are variable. Similar effects have been noted *for Ostrea hefferdi* (*2*), *Ostrea laperousei* (*9*), and *Crassostrea angulata* (*10*). The striking uniqueness of the responses of the oysters and mytilids relative to the responses of other Pteriomorphia would tend to support Cox's raising of these two groups to the ordinal level (*7*).

Two general statements can be made regarding the distribution of these acetylcholine responses. First, there are very few species in which concentrations of acetylcholine lower than those producing excitation did not result in depression. Even in *Mytilus edulis* and *Modiolus modiolus* some depression was observed in most of the hearts tested.