mune to infection by both 3C and 1D phages, but it continues to be sensitive to infection by phage NA1. Conversely, strain HS37 infected with phage NA1 is immune to infection by the homologous phage, but remains sensitive to phages 3C and 1D.

Electron micrographs were prepared of the bacteriophages according to procedures of Eklund and Poysky (*8*); photomicrographs of phages 3C and 1D were presented in a previous paper (*8*). Phage NA1, which is responsible for inducing nontoxigenic *C. botulinum* type C to produce the alpha toxin of *C. novyi*, exhibits a polyhedral head 65 nm in diameter and a tail 160 nm long and 5 nm in diameter. The sheath surrounding the tail is 68 nm long and 20 nm wide. Phage NA1 of *C. novyi* is similar in morphology to the phages that induce toxicity of *C. botulinum* types C and D (*8*).

These studies show that *C. botulinum* type C strain 162 ceases to produce toxin when cured of its prophage. This nontoxigenic derivative can then be converted to another toxigenic species, *C. novyi* type A, by infection with bacteriophage NA1 or converted to *C. botulinum* type C by bacteriophage 3C or to *C. botulinum* type D by bacteriophage ID. The toxigenicity and the type of toxin produced by this common bacterial strain depends upon the continued participation of specific bacteriophages. Further studies are needed to determine whether *C. novyi* type A can be cured of its prophage and then converted to *C. botulinum* type C or type D by bacteriophages.

The finding that *C. botulinum* type C can be converted to *C. novyi* type A opens new areas of research in the formation of these different toxins and possible control of botulism and gas gangrene in man and animals. Our results suggest that in nature a common bacterial strain could be responsible for both diseases. The specific bacteriophage that infects this common bacterial strain would therefore govern not only the toxigenicity of the bacterium but also the resulting disease.

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Aversive Situational Effects on Alpha Feedback Training

Abstract. *Anticipation of electric shock did not depress alpha activity in a feedback situation though it was associated with reported anxiety and heightened arousal indexed by greatly increased heart rate and number of spontaneous skin conductance responses. Contrary to previous reports, a reduction in alpha activity is not a necessary consequence of apprehension or heightened arousal.*

Considerable public and professional interest has been aroused in the use of alpha feedback training. The many beneficial results that have been claimed for the technique appear to be based on observations that anxiety-arousing stimuli and situations that are associated with sudden increases in levels of arousal produce sharp, dramatic, transient drops in electroencephalographic (EEG) alpha density. As early as 1929, Berger noted that worry and apprehension result in depression of the alpha rhythm (*1*). After reviewing the literature, Williams (*2*) considered the depression of alpha activity with apprehension a well-established phenomenon. It has generally been assumed, therefore, that as individuals are trained to augment alpha density they concomitantly learn to regulate their level of arousal and also gain control over the degree of experienced anxiety.

We have previously noted that alpha feedback training does not allow individuals to exceed their own optimal baseline, obtained from a nondrowsy subject under relaxed conditions with eyes closed in a totally darkened room (*3*). Under conditions when alpha density had initially been depressed by ambient light, however, subjects could learn to produce alpha activity with their eyes open, a circumstance which had previously interfered with alpha production, by learning to ignore their surroundings and avoiding use of their oculomotor system. It seemed likely that if anxiety suppresses alpha activity, individuals learning to augment this rhythm with feedback training would simultaneously be learning to prevent the increased physiological arousal and the subjective components of the anxiety experience.

The present study sought to test this rationale empirically, and focused on the relationship between both baseline alpha density and alpha density in a feedback situation under conditions of experimentally varied anxiety and arousal levels. Male college students were solicited for a study in EEG conditioning, and were told that the study would involve one session of physiological recording. During this screening session, considerable care was taken to establish rapport with each subject as occipital (O^2) to right mastoid EEG, electrooculographic, electrocardiographic, and skin conductance electrodes were attached. Records were obtained in both darkness and in dim ambient light, including eyes-open and eyes-closed 3-minute baselines and three 5-minute trials under each light condition, using alpha identification and auditory feedback procedures described previously (*3, 4*). Only after the completion of this first session were those individuals who had at least 25 percent alpha density during the initial eyes-closed baselines asked whether they would be interested in returning for a further experiment involving harmless but quite painful electric shock. It was our intent to create an ambiguous situation where subjects would be moderately anxious for the succeeding session.

From the 22 subjects in the first session who were asked to participate

in the shock experiment, 10 subjects agreed to continue. These subjects did not differ from the subjects who did not participate further in terms of baseline alpha densities ($t = .01$, $P > .20$). For the second session the same electrode placements and equipment were used, and, in addition, two shock electrodes were identified as such and conspicuously attached to the subject's lower leg. Care was taken not to explain when the shocks would occur, in the hope of increasing general feelings of apprehension. The remainder of the session was run in total darkness to avoid the confounding effects of ambient light.

After initial eyes-closed and eyes-open baseline periods, there followed four 5-minute feedback trials identical to those which subjects had received during the first session. Only then were subjects instructed about the shock part of the experiment. They were told that their task was still to keep alpha activity on, but at times a third tone would be present in addition to the usual alpha and no-alpha tones. They were told that whenever the third tone was on they were in danger of being shocked, but that this tone only occurred along with the no-alpha tone. Thus the jeopardy tone sounded only in the absence of alpha activity, and therefore the more alpha activity they produced, the less likely they were to be shocked.

Each subject was then given five 5-minute feedback trials. Each 5-minute trial was divided into ten contiguous half-minute segments. Five of these were labeled jeopardy segments during which the third (or shock) tone was always presented simultaneously with the no-alpha tone. The remaining five half-minutes were nonjeopardy segments. The two types of segments were ordered in a pseudorandom sequence with the restriction that the first segment always be nonjeopardy. During each of the 5-minute trial periods at such time as the jeopardy tones were on, subjects were given one or a maximum of two shocks to the leg electrodes. These shocks were delivered from a constant-current, battery-powered source, and were 60 hertz, varying randomly in 0.5-ma steps from 0.5 to 3.5 ma.

At the completion of final baseline measures, the subjects were interviewed about their experiences. All ten subjects returned for a third and identical session where much of the novelty of

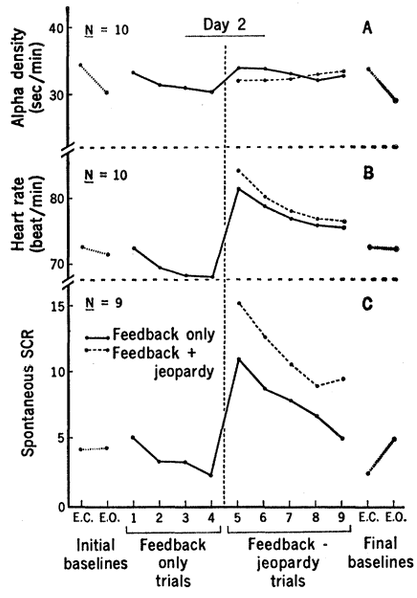

Fig. 1. Alpha density (in seconds of activity per minute), heart rate, and spontaneous skin conductance response (SCR) data for the first day with shock (day 2). E.C., eyes closed; E.O., eyes open.

the second (first shock) session was absent.

It was expected that the apprehension associated with returning for an ambiguous shock experiment in the second session would decrease alpha density during the initial baseline measures. From such a depressed baseline, a gradual increase in alpha density was

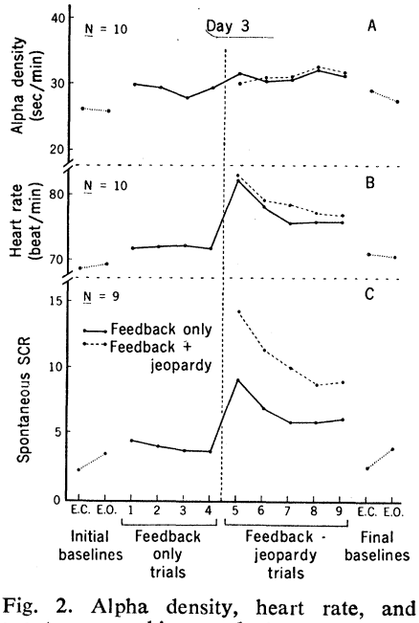

Fig. 2. Alpha density, heart rate, and spontaneous skin conductance response data for the second day with shock (day 3).

then anticipated over the four 5-minute initial trials, much as had been observed previously in the presence of ambient light (*3*). A comparison between eyes-closed baseline alpha densities on day 1 and day 2 showed that, although there was a slight drop from 59.1 to 57.9 percent, the difference was not significant ($t = .22$, $P > .20$). Since anticipation of shock failed to decrease alpha baseline density, it is hardly surprising that no significant increases were observed during the feedback trials in total darkness ($t = .80$, $P > .20$).

Totally unexpected, however, were the consequences of the shock instructions and the subsequent differences between the jeopardy and no-jeopardy segments. The top part of Fig. 1 shows the alpha density for the ten subjects during baselines and both feedback and jeopardy trials for the second session. The vertical dotted line shows the period during which shock instructions were presented to the subjects. In contrast to what was anticipated in terms of past published research, the shock instructions failed to lead to any significant diminution in alpha density. Indeed, there was a slight, nonsignificant increase in alpha density during the second session ($t = 1.01$, $P > .20$). Equally surprising was the transient nature of the drop in alpha density during the jeopardy segments of the trial. Although there was a significant difference during the first 5-minute period between the jeopardy and no-jeopardy segments ($t = 2.43$, $P < .05$), this difference disappeared by the third trial.

One of the most plausible explanations for these results would be that the attempt to induce feelings of apprehension or fear in the self-selected subsample of subjects who volunteered for the shock experiment was unsuccessful. During postexperimental interviews, however, subjects characteristically expressed their vague qualms about being shocked in the early part of the session, and most described acute discomfort and apprehension during the jeopardy trials. Although these reports suggested that the manipulation was successful, objective evidence regarding other (and often uncorrelated) measures of arousal levels was sought in the examination of heart rate data and in the frequency of nonspecific skin conductance responses (SCR's).

The average heart rate for the ten subjects on session 2 is displayed in

Fig. 1B. During the session, heart rate increased significantly in response to the shock instructions, a change of more than 10 beats per minute from an average of about 73 beats per minute in the earlier part of the session ($t = 2.98$, $P < .01$). Heart rate was distinctly elevated during the jeopardy segments of the shock trials ($t = 2.05$, $P < .05$), and when shock trials were over, heart rate returned to baseline levels.

The frequency of nonspecific SCR's is shown in Fig. 1C. As with heart rate, there is a dramatic increase in SCR's following shock instructions ($t = 7.62$, $P < .01$). Although the frequency of SCR's remains high during both jeopardy and no-jeopardy segments of the shock trials, the jeopardy segments are significantly higher ($t = 7.52$, $P < .01$). Very similar patterns of response for the three measures are seen during the third session, displayed in Fig. 2.

Although these data show clear evidence of heart rate and electrodermal responses to the threat of shock, consistent with the subjects' postexperimental reports, there are no concomitant decreases in alpha density. Even the differences in alpha density between the jeopardy periods, when shocks were actually administered, and the no-jeopardy periods are remarkably fleeting. It appears that neither the apprehension about the shock session reflected in the second session baseline, nor the more intense concern following the shock instructions, nor even the acute fear of being shocked, resulted in the anticipated sharp drops in alpha density.

In the present context, the lack of appropriate control groups and the special nature of the volunteer sample make it impossible to determine the effects that alpha feedback training might have had on the subjects' remarkable stability of alpha density throughout this experiment or the effects feedback could have on other populations. One cannot discount the unlikely possibility that within the course of 50 minutes of feedback training received prior to the shock instructions subjects learned sufficient control of their alpha densities to permit them to maintain these densities in the face of shock threat, as they were instructed and motivated to do, in spite of both large autonomic reactions to the shock instructions and the lack of manifest learning in total darkness during the screening session.

Whatever effect alpha feedback training had upon the alpha density shown by the subjects in this experiment, there clearly is a lack of the expected relationship between alpha density and the apprehension, anxiety, fear, or arousal level of the subjects. The discrepancy between these observations and previous reports suggesting a link between alpha density on the one hand and subjective state and arousal on the other requires further attention. Possibly the data suggesting a link between these two phenomena reflect a fortuitous combination of situation-specific factors and mediating influences which are not yet understood. It is worth noting in this regard that in the present studies alpha density was recorded for relatively long periods in total darkness. Care was taken to thoroughly adapt the visual orienting response and to control the novelty of the procedure. On the other hand, those studies which have reported the alpha blocking effects of anxiety and arousal were essentially novel situations, and there was little specific concern about the visual activity which Mulholland (*5*) has shown to be the major factor in alpha variation within any given single nondrowsy subject. It is plausible that the drop in alpha density in previous studies ascribed to arousal was actually a response to novelty or attempted visual activity provoked by the same stimuli responsible for emotional arousal, or both.

Regardless of how the discrepancies between the present and previously reported findings are ultimately resolved, the data presented here demonstrate that it is possible for the subjects to report the experience of apprehension or fear as well as manifesting the autonomic concomitants of such experiences without associated changes in alpha density. These data not only raise theoretical questions about the meaning of alpha density but also challenge the widely accepted rationale for using alpha feedback training as a means of teaching individuals control over their own levels of anxiety. Further basic research seems indicated prior to considering alpha feedback training for widespread clinical application.

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