WHEN TO DO WHAT WITH CRIMINAL OFFENDERS

Douglas A. Quirk, Ontario Correctional Institute and
George von Hilsheimer, Maitland, Florida

Introduction

In the usual case, biofeedback is used to train voluntary self-regulation of a so-called involuntary physiological response which is associated with a specified kind of anomalous behaviour. In principle, it is presumed that some cuing stimulus is required to remind the client when to reinstate the learned voluntary self-regulation. However, the nature of the stimulus cue used to signal the need for self-regulation is not always clear.

In some instances, time is the main cuing stimulus. After a pre-established interval of time, the learned self-regulatory acts may be initiated. Meditation, once learned, tends to be done on a time schedule.

In some instances, the setting in which the self-regulation is to take place is implicit during training, and the setting may then become the cuing stimulus. Since work or school performance deficiencies are often the reason behind biofeedback training for hyperactivity or attention deficit, the client is likely to be thinking about the work or school setting while attending for the training. Thus, entering the setting may cue self-regulation.

In some instances, the client is able to detect a specific and noteworthy prodrome which signals the onset of the condition for which the self-regulation training was undertaken. An aura occurs just prior to the onset of many cortical-focus epileptic seizures and some attacks of migraine. When they occur, these prodromes can be used by the client to cue the need for learned self-regulation. Indeed, there is some suspicion that it is those seizures and migraines which follow a recognizable aura that can be controlled best by means of self-regulation biofeedback training.

However, it is not yet clear whether a cuing stimulus, such as time, setting or aura, is required to evoke self-regulation in order to ensure that biofeedback learning is maintained in self-regulation of symptomatic behaviour. Of course, there are those who consider the question of a cuing stimulus irrelevant. These practitioners tend to hold that biofeedback training provides the client with means by which to self-regulate that must be practised daily in order to be maintained. There are at least two problems with this point of view.

First, under this view, responsibility for failure to maintain the effects of biofeedback training can be transferred to the client. While convenient, if the therapist does not assume the responsibility, there is little motivation to develop means by which to increase future successes. The therapist is surely the

one paid to be the expert to ensure effectiveness of the training.

Second, even if the therapist clearly and repeatedly states that, to be effective, the learned self-regulation must be maintained by constant practice, many clients just will not hear the instruction. Most clients view biofeedback training through the expectation, fostered in health practice, that they will be 'cured' of their presenting problems. Indeed, most clients will believe they are paying for a cure for their symptoms. Any statements to the contrary will tend not be heard or will lead the client to find help elsewhere. When the practitioner's and the client's agendas differ, it is as though each speaks a language the other does not understand.

The present report adopts the assumption that biofeedback training is concerned with modifying presenting problems or symptoms. It adopts the assumption that biofeedback training effects are the responsibility of the therapist. And it addresses the question of whether it is possible to arrange for effective and lasting consequences of biofeedback in conditions where there is no recognizable cuing stimulus or prodrome, and thus by means that may not involve voluntary self-regulation.

Syndrome Prodromes (Selection of Problems)

Some conditions or syndromes display a recognizable prodrome which signals the imminent occurrence of the symptoms. Many cortically-focused epilepsies are associated with a pre-seizure aura, the experience involved being sufficiently unexplainable as to be noteworthy. The same is true in some cases of migraine. PMS often has a recognizable prodrome. In addition to the regularity of menstruation which provides a time cue, weight increases due to fluid retention and/or an unexpected increase in irritability may serve as anticipatory stimuli. In these cases, it is possible during training to use the prodromes as implicit cues to which evocation of the learned skills can become attached or learned.

Most conditions, however, are associated with no recognizable prodrome which might be used as a cue to evoke learned skills. In schizophrenia, although the person may be terribly upset and uncomfortable, the connection between self-awareness and personal control over subjective states tends to be lost. Even if the person is aware of the need for corrective measures, personal role in modification of the symptoms seems remote and inconceivable.

Among offenders, it might seem that the person would know when he or she is about to perform a criminal act. However, in many cases, either the need for the offensive act is too great or the person is not well attuned to the risks involved. In either case, the voluntary evocation of learned corrective techniques may be difficult or impeded.

The point is that in conditions such as these there is either

no cuing prodrome or the prodrome may be too weak to cue corrective measures. The importance of an absent or weak prodrome lies in the likelihood that voluntary self-regulation, however well learned, will not be re-activated at the times when it is needed. This fact means that the conventional methods of biofeedback self-regulation training are apt to fail in such conditions.

Instead, it may be necessary in conditions such as these to employ learning procedures that do not depend upon self-regulation. That is, it may sometimes be necessary to establish a stable and continuous automatic habit to behave differently at all times, or at least at times cued by specific types of internal or external stimuli. The present report is concerned with the establishment of continuous automatic habits in conditions that are essentially prodromeless.

Subjects' Symptoms' Symptoms (Selection of Subjects and Responses)

But what kinds of responses and automatic habits could be trained which might modify conditions such as schizophrenia or criminality? An answer to this question might be found by looking at ancient history -- with apologies for belabouring the obvious.

Joe Kamiya's demonstration that recorded EEG activity could be altered by feedback of that activity, not only launched biofeedback as a field, it also suggested that the electrical activity of the brain was just a kind of behaviour that could be trained like any other. In turn, this led to the idea that any pathological (or desirable) state, related to recognizable features of the EEG, could be modified by getting the person to alter his or her EEG. That idea was the seed from which grew Forrester's habituation training for triggerable epileptics and Sterman's conditioning of SMR to inhibit epileptic seizures. It also made it possible to normalize the learning performances of people exhibiting attention deficiencies and hyperactivity. But how widely might this idea be applied?

(1) Some epilepsies, whose focus lies in the deep recesses of the old brain, are variously referred to as partial and complex non-convulsive seizures, paroxysmal events, sub-ictal states or seizure equivalents. These deep-brain epilepsies have features which make them difficult to recognize. First, they are seldom associated with convulsions, losses of consciousness or even periodic unusual events which might suggest the need for a specific investigation. Second, their epileptic equivalents often seem to be accountable as behavioural derivatives of the social history. Third, they are not readily or reliably diagnosed by means of the EEG, partly because they are not as easily triggered as cortical epilepsies by conventional means such as photic stimulation or hyper-ventilation. In these deep-brain epilepsies there is rarely a prodrome, and the person can rarely tell when a seizure occurs.

If the epileptic discharge in these seizures involves Olds and Milner's 'drive centre', the person may exhibit uncontrollable paroxysms of rage, sexual drive, hunger, satiety, alcohol use or other excitant automatisms such as fire-setting. Given that, it is obvious that a correctional population is most likely to contain relatively large numbers of people exhibiting behaviours such as those evoked by this kind of epileptic discharge. Indeed, in a study of the serious offenses among one year's admissions to a correctional treatment centre, we found that 40% of the arsonists, 30% of the assaultists and 25% of the rapists were subject to deep-brain seizures. By way of contrast, under 2% of the 'garden varieties' of less 'dangerous' offenders exhibited this deep-brain epileptic syndrome.

If Sterman's procedure for training SMR in the EEG is effective in treating the epilepsies, we thought it would be interesting to discover whether it could be used in epileptics of this kind to modify their future criminal conduct. If this could be achieved, the generality of Sterman's method would be widened to a degree. There is no recognizable prodrome or characteristic behaviour which might provide a cuing stimulus for the client to re-activate any learned corrective strategy. If SMR training reduces criminal recidivism in offenders with deep-brain seizures, then the SMR training may be said to have become a stable new habit in these people which requires no maintenance exercises. That is, if deep-brain epileptic events, that are not visible in the EEG, could be inhibited by means of SMR training, it might be argued that trained increase in the amount of SMR activity has a 'curative', rather than merely an ameliorative, effect on the epilepsies.

In our first study, then, SMR activity in the EEG was used as the operant response to be increased by training. The subjects were 77 incarcerated criminal offenders, all males, mostly displaying the more dangerous types of offenses, who gave evidence, on test investigation, of deep-brain epileptic events -- presumably underlying the excitement-seeking actions, 'blind' rages or sex drive excesses involved in their criminal conduct. Since the EEG rarely reveals pathological behaviour in deep-brain epilepsies, the therapist is not explicitly or implicitly cued in the training activities. And since the client is unlikely to notice subjective indications of these epileptic events, he or she is unable to re-evoke the learned behaviour on cue during or after training.

(2) The group of conditions called the schizophrenias offers another setting in which it might be expected that any prodromes would not usefully be recognizable by the client or the therapist. Schizophrenia is also a condition in which biofeedback seems an unlikely treatment choice. The relevance to the present topic of

this group of conditions is based on two observations.

First, as Mednick and others have shown, one main feature of the schizophrenias is stimulus over-generalization. Different stimuli are responded to as if they were equivalent. That is, the person becomes undiscriminating with respect to internal and external stimulus events. One consequence of this phenomenon is that the person becomes vague and unresponsive to subtle cuing events. Thus, even if there is a prodrome which the client might use to activate learned corrective measures, he or she is unlikely to be able to recognize or use the prodrome as a cuing stimulus. Moreover, if there is a prodrome that the therapist might use as a cue during training, it is unlikely to be recognizable by the therapist. This last statement is justified by the fact that, for over a hundred years, the schizophrenias have proved to be utterly mystifying to a host of psychologically trained workers.

Second, as Mednick has shown, another main feature of the schizophrenias is the maintenance of an under-responsive or muted autonomic nervous system as a defence against catastrophic hyper-arousability under stress. This feature was demonstrated by Mednick while monitoring the galvanic skin resistance (GSR) or the electrodermal response (EDR). There is some reason to suppose that these catastrophic reactions are central to the schizophrenic condition. If so, it is possible that trained modification of the GSR might offer a biofeedback means to treat the schizophrenias.

Of course, there are several difficulties which seem to stand in the way of using this approach to treatment for schizophrenia. First, the GSR is known to provide a highly variable (unstable) measure of responses. This, however, is true mainly when it is used to measure responses to discrete test stimuli. Variability of this kind is much less of an issue in extended monitoring of the response. Second, the GSR tends to reflect anxiety arousal of which the client must surely be aware as a kind of cuing prodrome. However, as a group, schizophrenics are said to exhibit 'flat' affect, which means that they tend to be under-responsive to changes in their internal or external stimulus environments. Moreover, their inability to cope with catastrophic anxiety arousal is a main characteristic which defines the condition. In turn, this might mean that learned coping might offer some hope as a treatment strategy. Third, it is generally understood by psychiatrists that the high levels of anxiety arousal are symptoms or consequences of schizophrenic pathology. If true, the most that might be hoped from trained modification of anxiety is that some symptoms might be ameliorated slightly -- the purpose claimed for the use of psychotropic medications. Still, the untested validity of the psychiatric understanding might usefully be challenged. For example, what if anxiety could be shown to be the cause of the

pathology instead of its effect? In order to test that question it would be necessary to demonstrate 'cures' of schizophrenia by treatment modification of anxiety or arousal.

In our second study, then, GSR or EDR is used as the operant response to be trained towards 'comfort'. The subjects were 40 psychiatric in-patients, all female, all of whom carried the 'diagnosis' of schizophrenia as at least part of the label assigned by psychiatrists to their conditions. It seems unlikely that the clients would have been able to recognize or use any cuing prodromes in order to modify their symptoms. This last statement seems justified by the fact that these people had been in-patients bearing the same diagnoses for an average of nine years prior to the present treatment contact. Since the symptoms manifested by these patients were highly variable, there was also no specific cue which might have been used by the therapist as an anchor in the biofeedback training. It is true that there are ranges of basal skin resistances (BSR) which tend to represent degrees of approach to 'mental health', and which, in addition to their use to help in deciding when treatment was completed, might unintentionally have cued therapist conduct during training.

Caveat

The two studies reported here are flawed. Both employ old data, are subject to the limitations imposed on data collection and analysis prior to the advent of modern computer technology, and both employed antique equipment. Moreover, both studies contain confounds which limit the confidence with which statements can be made concerning the factors contributing to therapeutic effects. For these reasons, the results are expressed in rather more general terms than is usual in a research report. While this caveat needs to be borne in mind, it should also be observed that this paper is intended as a discussion of prodromeless syndromes. Its purpose is to suggest some wider applications of biofeedback for the future. As such, the studies reported can best be understood as pilot work.

Pre-scribed Prescriptions (Selection of Methods)

(1) In our first study, the operant response selected for biofeedback conditioning was SMR in the EEG. Bipolar contact EEG electrodes were installed over the C3 and C4 sites, with the ground placed in the middle of the forehead. The EEG signal was fed into an Autogen 120a. The instrument digitizes the incoming signal, and responds with audible feedback only while the recorded EEG is within a pre-set frame defined by frequency (set at 12-14 Hz) and amplitude (set at 10-30 Mv). That is, the frequency modulated (or white noise, if preferred) feedback options were discontinuously present and were contingent upon the presence of the defined SMR activity from the C3-C4 sites. Subjects were told that the audio feedback, when it occurred, was to be understood as the trainer's

way of saying 'fantastic', or as a reward for desirable behaviour. The subjects commonly complained that they had no idea at all what they were doing or why the (usually) whistling sounds occurred.

Once it had discharged itself, the instrument's percentage of time meter was set to cumulate time-in-feedback at 1,000 seconds. This was done to permit two fairly stable records of SMR learning which might be observed during sessions -- the one record made just after the middle and the other at the end of the half-hour training sessions. Sessions were held once or twice a week, starting when trainer time was available, and terminating when the incarcerated subject was released from his sentence.

From the perspective of an evaluation of the effects of SMR training on the subjects, this study was flawed. Some pilot work had been done using this application of SMR training only. While the effects of the training on follow-up behaviour seemed to have held up in most cases, the trained increases were 'lost' in a few cases. Inquiry led to the conclusion that the 'loss' of trained effects in these cases followed intense anxiety/panic attacks or a trauma. In order to reduce the risk of such 'losses' from anxiety attacks in the present study's subjects, all the subjects reported here were concomitantly trained with the same GSR training method to be described in the second study (below). Thus, although we believe that the effects achieved with the subjects from the first study were due to the SMR component of the biofeedback treatment, we are unable to prove our contention. Moreover, since all the subjects were treated with both the EEG and GSR biofeedback methods at all their training sessions, we are unable to partial out the effects of one or the other from the results.

(2) In our second study, the operant response selected for biofeedback conditioning was the GSR or EDR. Contact electrodes were installed on the right hand, with one electrode in the centre of the palm, and the other in the middle of the back of the hand. The skin resistance was recorded using a SCARS2 programming unit. It displays the GSR level in thousands (or hundreds) of ohms, to a maximum level of 1.5 megohms. The unit performs a compare function between the GSR meter value and the last value stored in the unit. When the meter value exceeds the stored value by one thousand (1K) ohms, the new value is stored and the unit pulses a change in the slide being viewed by the subject. That is, video slide change is effected discontinuously and serves as a 'reward' contingent upon successive increases in skin resistance (GSR). The 'reward' value of slide change has been demonstrated elsewhere by Quirk. Subjects were told that each new slide change was the trainer's way of saying 'good' as a reward for desirable behaviour. Some subjects complained that they did not know what they were doing, or why the slides changed.

The therapeutic record included the starting, ending, highest and lowest GSR values, along with the category of slides used and the number of slides shown/changed during each half-hour training session. The slides depicted ordinary events from daily life, but were selected and organized into sets (trays) to represent various stimulus situations in which clients commonly experience anxiety. Slide trays had been assembled to represent such anxiety stimuli as A-Ambiguity, B-Body boundary permeability, C-Condensation of unrelated stimuli, D-Distorted images, E-Exhibition of the body, F-Authorities, G-Guilt, H-Heights, I-Isolation, J-Minor authority, K-Claustrophobic settings, and the like. Partly, the slides were shown in order to provide a context other than the lab in which training took place, and to which contexts it was hoped that the training would generalize. Partly, they were shown to have some stimulus ongoing which, being changed, could afford a discontinuous and contingent (reinforcing) feedback for progressive changes in the physiological response being monitored.

This study might be considered by some to have been flawed because, in addition to the above GSR biofeedback training, all of the patients received anxiety-relief conditioning for five minutes twice each day. This procedure, described by Wolpe, involves conditioning, to a repeated verbal cue, a relief response brought about by terminating anticipation of mild discomfort from tickle shock to the fingers. This psychological 'tranquillizing' agent was used with all these patients as a way of replacing for them their expected tranquillizing medications. These medications were withdrawn well before patients began the biofeedback treatment. The reason for delaying start-up with the biofeedback treatment is that it had been observed in other cases that a small percentage of the patients recovered following withdrawal of their tranquillizing medications. The addition of the anxiety-relief conditioning to the treatment regimen, however, did not affect any symptoms other than the intensity of anxiety in these patients, or in others who were treated for prolonged periods with anxiety-relief conditioning alone. That is, in spite of what may appear to be a confound, it is likely that the additional use of anxiety-relief conditioning is not a significant confound in the present study.

Results

(1) On average, one and a half years after the offenders who were accepted into this treatment had been released from their sentences, their justice system records were reviewed. Of those 17 who received 0 to 4 half-hour training sessions, 65% had been re-convicted of criminal offenses. Of the 10 who had received 34 or more half-hour training sessions, 20% had been re-convicted of criminal offenses. These findings are consistent with those of an earlier, smaller study of which the present study is a replication.

Intermediate amounts of training were found to be associated with intermediate recidivism rates. Among these subjects, follow-up interval and age were not related to the rates of recidivism, nor were they related to the observed relationship between amount of training and recidivism rates.

(2) Following the treatment regimen described above, most of the patients were deemed by psychiatric conferences to be recovered and were discharged from the hospital. All those who had recovered and were discharged were seen in occasional follow-up contacts for at least two years after discharge from the hospital. By the end of this follow-up period, 85% of the forty women trained with the GSR biofeedback procedure were still functioning as normal people, were self-supporting and had required no psychotropic medications or psychotherapy during the two years post-discharge. One of the 'failure' cases, who was deemed to have recovered following the present treatment, obtained out-patient psychotherapy during the follow-up period. The other five 'failure' cases had not recovered from this treatment, and they were later treated with various forms of behaviour therapy.

Discussion

The caveat stated earlier should be recalled when considering the sketchy results reported. The results are not intended to offer definitive proof of any contention that dangerous criminals and schizophrenics can be treated effectively with biofeedback methods. Instead, the results are intended to offer preliminary indications that wider than might be expected therapeutic effects may be possible by means of biofeedback procedures devised to take account of some client characteristics.

In particular, the results are intended to suggest that even some prodromeless conditions may be susceptible to biofeedback interventions using appropriate applications. If the training is structured to foster the establishment of a new and stable habit to respond in healthy ranges of appropriately selected physiologic responses, lasting, even self-strengthening, resistance to symptoms may be possible. In these studies, the physiological responses monitored were considered to be operant responses and, as in learning studies with animals, the responses were subjected to reinforcements to shape new habits independent of voluntary control or understanding. It is assumed that the habits thus developed would be self-strengthening since, if properly selected, they should eventuate in self-reinforcement by enhancing the efficiency of the person's functioning.

The other issue addressed in this report bears upon selection of the physiological responses to be monitored and trained. In many conditions, there is a recordable physiological response which varies concomitantly with the symptoms. It is probably relevant

only to the preferences of the particular investigator whether the physiology or the symptom drives, controls or causes the other. Regardless of the 'causal' functions involved, if a recordable physiological response can be found which does vary with important phases of symptom production, trained modification of that related physiological response may serve in some cases to modify the symptoms. Certainly, this hypothesis merits further investigation.

In the first study referenced here, a deep-brain epilepsy was detected in a subset of relatively dangerous offenders. Under the hypothesis that Sterman's method for SMR training offers a general treatment for epilepsy, it was used with these offenders apparently with stable effects in reducing their subsequent criminal behaviour as indicated by justice system records.

In the second study referenced here, a group of chronic schizophrenic patients were treated with a procedure described elsewhere by Quirk. GSR modulation training was used with these patients under the general hypothesis that Mednick's observations of catastrophic autonomic nervous system reactions, which can be recorded on the GSR, are centrally associated with the maintenance of schizophrenic symptomatology. The results obtained over a two-year follow-up justify the tentative conclusion that schizophrenia may be susceptible to treatment using suitably designed means for GSR biofeedback conditioning.