intuitive, since our most basic experience informs us that our choice of perspective regarding how we apprehend our mental states makes a real difference in how we respond to them. For those who insist that only material causes matter such intuitions must be denied as illusory. Fortunately, as we have attempted to demonstrate in the body of this chapter, physics as it is currently understood and practiced, i.e., quantum physics, offers a precise scientifically grounded solution to this apparent conflict between the observed reality of our emotional experience and the requirements of rigorous data-based explanation.

A word about the meaning of the term data may begin to point us toward a more pragmatic perspective on these issues. For the materialist *all* possible data *must* in principle be provided, at least potentially, by the five senses (perhaps aided by technical advances in data acquisition) and their contact with the external world. But the way in which the experiments on the neuropsychology of emotional self-regulation are actually designed and performed belies this perspective: the use of language and other modes of communication that refer to phenomena that are intrinsically experiential and non-reducible to materialist terminology (e.g., Actively reappraise this scene; Be mindful now, etc.) are a critical and irreplaceable part of the experimental instructions, training and set-up. From the materialist's point of view the only aspects of the entire experimental set-up that count as real are the brain data itself, any observable bodily phenomena, and the actual physical sounds of the verbal reports the subjects make to the investigators, as recorded by marks on rating scale sheets, etc. The concept that the subjects *did something* with their minds *per se* that actually influenced their emotional responses and the related brain data collected is strictly prohibited by the materialist perspective. The idea that the subjects actually *experienced* any internal change in feeling state on account of their mental acts is considered entirely out-of-bounds. Only what is externally observable counts as real in any ultimate sense. One is reminded of the old anecdote about two strict behaviorists who meet at a conference. After imbibing some alcoholic beverage they proceed to a hotel room and engage in intensive copulatory activity. Pulses soar, blood surges, engorges, and subsides. At the conclusion of the physiological excitation process, one turns to the other and sighs, "Well, I know it was great for you. How was it for me?"

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CHAPTER 8

EEG biofeedback ("Neurofeedback") and affective disorders

J. Peter Rosenfeld and Elsa Baehr

Introduction

Organization of this chapter

After a definition of the term *Neurofeedback,* and a brief discussion of its underlying principles, this chapter will review the evidence that at least some examples of operant EEG conditioning – such as the voluntary control of brain waves - are not trivially mediated by familiar phenomena. Then, a presentation of examples of the functional significance of neurofeedback will be given; one experimental and one clinical illustration will finally be followed by an example of the limits of this new clinical protocol.

Definition with underlying principles

The term *Neurofeedback* is a recently coined shorthand term for operant conditioning of neural activity. Nowadays, it is frequently used in clinical contexts. In general, the neural activity can be any form of individual or population measures of the activities of neurons, and indeed there have been reports of operant conditioning of action potentials from single cortical neurons (Fetz & Finnochio 1971) as well as more frequent reports of conditioning of spontaneous EEG (Kamiya 1969) and its time-locked derivatives, i.e., event-related potentials (ERPs) from animal brains (Fox & Rudell 1968) or human scalps (Rosenfeld, Rudell, & Fox 1969). Operant conditioning of any neural event means that a subject is rewarded for (voluntarily) changing the future probability of the specific event. Obviously, this is the prevailing operant model of neurofeedback. This model is close to the original formulation of operant conditioning as articulated by Skinner in the 1930s: training a rat to press a

bar is simply a matter of setting up a reinforcement contingency such that any time the bar is pressed, the rat is positively reinforced or rewarded with, say, a food pellet. If the rat is motivated (e.g., hungry), he will learn the association between response and reward and will eagerly press the bar. Operant or voluntary control in humans is often also by trial and error, perhaps aided with a bit of coaching. When we learn a new word processor program, it is not always obvious how to achieve an effect, such as bolding. We try a few things, perhaps coached by icons, and we learn whatever works, such as the association between the "B" icon press and the bolding effect. Unfortunately, the neural mechanism of this simple trial-and-error learning process remains as unknown as in Skinner's time, even for the most simple of operant responses. In the neurofeedback situation, we assume that a certain desired brain wave pattern is like a bar press, and we reward the subject following emission of these target neural events. As in operant behavioral conditioning, the target behaviors – neural or ordinary – have to be in the animal's ordinary behavioral repertory prior to training. Indeed in our first studies of evoked potential amplitude training described below, we would observe the subject for one or more days of baseline with no rewards just to collect the amplitude distribution. Then we would reward animals for increasing the probability of one tail of this *normal* distribution of pre-training amplitudes. That is, the amplitude had to be within the normal repertory of amplitudes. How the animals learned to shift their distributions for reward remains a mystery. What was critical to us was to eliminate some obvious trivial mechanisms (this is discussed in the next section). In some human clinical situations, matters become slightly more complicated, as operant neural training may be preceded and/or accompanied by relaxation training, and/or various other therapies. These other components of a neurofeedback training package probably have non-specific effects, and may be unnecessary.

There may be one additional complication regarding the mechanism of operant neural conditioning vs. operant conditioning of familiar behaviors (bar presses), and that complication merits noting. This is the issue of what fundamental event gets conditioned, specifically. Do subjects learning to change the amplitude of an EEG wave have direct, non-trivial control over that wave? Or do the subjects change something else which influences or is reflected in that EEG event? If, for example, a visual evoked potential is changed by the subject's learning to close his eyes or stare into the light source, this is mediated conditioning. Indeed, it is trivially mediated, as discussed in the next section. On the other hand, the conditioning could be mediated, but in a novel, non-trivial fashion. Or it could be unmediated and direct. With ordinary behavioral

conditioning, one can readily identify the target behavior (e.g., the bar press) and assume that the subject can experience the behavior also that results in the reward. If not the behavior, then the somatic sensory feedback from the behaving limb, its efferent control and/or collateral feedback discharge. With neural events, there is no known physiological feedback from the conditioned brain part which sends information to awareness systems. The learning mechanism becomes an even deeper conondrum.

The trivial mediation issue

S. S. Fox and Rudell's use of operant controlled neural events (*OCNE*) was introduced as a new methodology for clarifying neural coding of behavior. However, their initial 1968 paper in *Science* caused interest in the neuroscience community not so much because it was a new approach to neural coding, but because prior to this report, it was widely believed that the pattern of components in the photically evoked ERPs in the cat's cortex was not subject to self-regulation, but represented the hard wired, sequential summations of postsynaptic potentials in the visual cortex in response to a consistent high intensity stroboscopic light source. The cats of Fox and Rudell dramatically changed their brain responses following a regimen of training in which they received a small amount of milk (pumped directly into their mouths) following emission of responses of low, pre-training probability. Thus, reactions to this report included some nay-saying. For example, it was noted that the conditioned cats were free to move about in their conditioning chambers and thus able to voluntarily alter their photic ERPs by (uninterestingly) changing their receptor orientation to the light source. Or they could execute either phasic or tonic movements whose somtosensory feedback could, via reticular formation and its widespread reticulocortical connections, alter the photic ERPs (also uninteresting and not novel). A series of papers followed (summarized by Rosenfeld 1990) which ultimately ruled out all possible sources of such trivial mediation of operant controlled ERPs. In particular, Rosenfeld, Hetzler, Birkel, Antoinetti, and Kowatch (1976) demonstrated that in total darkness, rats could be trained (via rewarding brain stimulation in the lateral hypothalamus) to systematically alter the amplitude of early and late components of the visual ERP elicited by mild shock to the optic chiasm, each shock presented at random intervals. It had been thus clearly shown that organisms could voluntarily control internal neural activity utilizing some novel mechanism. This was a novel operant response that could be used to test the generality of putative laws of learn-

ing derived from experiments on other (usually voluntary motor) systems (see Rosenfeld et al. 1976). (Incidentally, even in our earliest studies, we informally visually observed both cats and people during the operant ERP conditioning and never saw any sign of trivial mediation.)

In retrospect, such self-regulation of brain activity in a non-trivial manner could have been anticipated. In Rosenfeld et al. (1969), humans instructed to sit still and alter acoustic ERPs explained their successful ability to generate such alterations by referring to use of their imaginations. We knew nothing about how imagining activity was encoded in EEG and how it might influence externally evoked activity, and so we tended to write off these responses, (imagining activity was also not a popular element of any hypothesis at a place such as Kenneth Spence's highly behaviorally oriented department of Psychology where our early work was done). Nevertheless it can be simply stated today that the key assumption of any model of operant neural conditioning is that there must be an input to the conditioned neuronal organization from another neuronal organization over which a subject has direct control. We take as given the fact that individuals can control the contents of their imaginations to a large extent from moment to moment. The fact that individuals can alter ERPs in a non-trivial manner and report using imagination to do so means that the neuronal organizations mediating imagination activity must be connected to the altered neuronal populations. This need not be the only mechanism of operant neural control or neurofeedback.

Functional significance: Experimental studies

The fact that individuals could alter visual ERPs in a non-trivial manner begged the question of functional significance. After all, the sizeable changes in the amplitudes of visual ERPs that were being produced by operant conditioning meant that the patterns of post-synaptic potential integration giving rise to EEG and ERP phenomena must themselves be changing. The neurons generating such post-synaptic potentials were likely to be functioning neurons in the visual cortex. Thus one could wonder to what extent vision was being influenced by operant control of visual ERPs. We next addressed ourselves to this question of functional significance, however for various reasons (see Rosenfeld 1990), we moved from the visual modality to the pain modality. Therefore, instead of recording from visual cortex, we recorded from the somatic sensory cortex (a terminus of major pain systems) and instead of evoking potentials by stimulating the optic tracts, we delivered *non-painful* shocks to the trigeminal

tract, the neural pathway which transmits orofacial and dental pain information to the central nervous system. Clearly, high-level shocks to this tract would produce pain. This path was our ERP-eliciting locus, and had to be stimulated several hundred times, every few seconds during a daily training run. The level of stimulation used had to be kept at non-painful levels and so was adjusted to be just high enough to evoke to-be-conditioned potentials in the somatic sensory cortex. To measure the effects of operantly conditioning these trigeminally evoked, cortical ERPs on pain perception, we slowly increased the heat in a heater attached to the rats' faces. We assumed that when the rat started rubbing at the heaters, the heat was approaching painful levels and we immediately shut off the heat, recording the time from heat onset to first face rub as the pain tolerance index. The results (Dowman & Rosenfeld 1983, 1985), to our amazement, were that all rats with histologically confirmed correct electrode placements were readily able to increase as well as decrease (depending on the reinforcement contingency we set) the amplitude of the selected ERP component, *and the consequence of this neural training was a systematic change in the pain tolerance index. Also, the degree of this change was "clinically significant"* since it corresponded to the change produced by a moderate dose of morphine.

These results did stimulate us to explore whether they would generalize to human clinical situations, and we did initiate such work (Rosenfeld, Silvia, Weitkunat, & Dowman 1985; Weitkunat & Rosenfeld 1986), however we did not carry it forward for various reasons. One reason was that we had demonstrated effects in animals *with experimentally induced pain.* Human pain in clinical situations is very different. Clinical pain tends to be tonic in nature, unlike our rapidly increasing heat bursts in rats which tended to peak in 5 seconds. Also, chronic clinical pain is also often accompanied by psychological stress and/or anxiety and/or depression. Our results in rats did serve the purpose of demonstrating functional significance, if not a novel clinical modality, and so we decided to leave it to others to explore the clinical pain situation. There was a theoretical issue also. The rationale for the somatic sensory evoked potential training effect on pain was our knowledge that there existed neurons, called "wide dynamic range neurons," in somatic sensory cortex, which were activated both by innocuous somatic sensation, such as our non-noxious shocks to the trigeminal tract, as well as by pain such as that produced by our facial heaters. We reasoned that if the operant conditioning procedure could change the states of these neurons during the training session, their states would still be altered when the actually painful heat burst was applied immediately after the last daily training trial, leading to altered pain tolerance. This worked, as we have seen, but we had no idea how long this altered state would last beyond

the end of the training session. This was another loose end that we felt needed tying up prior to a foray into the clinical pain problem. However, our major reservation was that our approach had been indirect in that the ERPs evoked by non-noxious stimulation were themselves not a pain index, yet their alteration was (not unreasonably) expected to lead to pain change. We decided that before a clinical application of operant neural control was attempted, we would wait until someone found a robust indicator of some human pathology.

Functional significance: Clinical application for depression

We did not have a long wait. Indeed Richard J. Davidson and colleagues had commenced a program of study of the neural mechanisms of emotion at about the time we had been training ERP changes in the rat somatic sensory cortex. Davidson's highly influential program of research has dealt with many aspects of emotion, theoretical and applied (see Davidson 1995, and other chapters in this volume by Fox & Lane), but for us, the key papers were those by Henriques and Davidson (1990, 1991). These papers showed that, as predicted by the Davidson group's other work and theorization, depression was characterized by a characteristic frontal cortical asymmetry in comparison to the EEG from a non-depressed control group. Normal frontal cortical asymmetry tended to produce roughly equal amounts of right and left cortical activation, or more left than right activation. Depression correlated with a reduced activation on the left side. This was expected and predicted since many other studies from this laboratory had demonstrated that manipulations tending to produce negative affect also led to a reduction in left frontal activation and an increase in right frontal activation. The EEG results, in turn, were predicted from Davidson's model in which (to oversimplify) there is a positive, approach emotion system in the left frontal cortex, but a negative, withdrawal emotional system in the right frontal cortex. Now having a direct neural correlate of affect, we decided to see whether or not the EEG asymmetry could be operantly conditioned, and if so, to determine the emotional consequence, if any, in a clinical situation.

In Davidson's studies and in ours to be described, activation asymmetry was indexed (inversely) by EEG Alpha (8–12 Hz) power. Thus an active cortex would have a paucity of alpha, and an idling cortex would have excess alpha (the reasons for the inverse indexing are in Rosenfeld 2000). There are two formulae which have been used to index moment to moment alpha asymmetry: $A[1] = \log R - \log L$ where R and L are right and left alpha power or magnitude;

$A[2] = (R - L)/(R + L)$. As these *Asymmetry scores* correlate at .98 (Rosenfeld 2000) even though they are not mathematically equivalent, both formulae may be used interchangeably for moment to moment assessment of alpha asymmetry, where the higher the score, the greater the relative ratio of right to left alpha, the lower the ratio of right to left activation, and presumably, the greater the positive affect. In summarizing, for patients, asymmetry results for a session, as well as for research, we found that the mean A[1] or mean A[2] scores were not as good as a percentage (PCT) score we developed: this was simply the percent of time that A[2] was greater than zero. In one session, we had found that the PCT scores discriminated depressed and normal persons better than the mean A[2] score, as might be expected, since a mean A[1] or A[2] index would be much more influenced by extreme values (Baehr, Rosenfeld, Baehr, & Earnest 1998) than would the PCT index. Our neurofeedback training method in these early studies was lifted from our work with human ERPs (Rosenfeld et al. 1969): we would observe the alpha power in one or more baseline sessions, and then reward subjects with a pleasant tone sound for producing A[1] scores 0.7 standard deviations greater than the baseline mean. Our methods with clinical cases are similar and detailed in Rosenfeld (2000).

Our first study in the operant conditioning (neurofeedback) of frontal alpha asymmetry was reported by Rosenfeld, Cha, Blair, and Gotlib (1995). In this study in which we were working with normal subjects from the introductory psychology participant pool and whose training time was, therefore, limited, we subjected the subjects to only three days of training (most neurofeedback regimens in current use call for upwards of 30–40 sessions). Nevertheless, 9 of 13 subjects showed significant (doubling) of their baseline A[1] scores. This encouraged our plan to transpose the paradigm into the clinical situation. In the meantime, an independent lab (however with our software) extended our work by demonstrating that the asymmetry changes could be generated, and that these changes could be made bidirectionally, and that there were emotional consequences: subjects trained to increase A[1] showed greater positive affect to happy and neutral films than did subjects trained to decrease A[1], as would be predicted from Davidson's model (Allen & Cavendar 1996).

These results were certainly encouraging with respect to our clinical ambitions, however since no patients with real affective disorders were involved, the studies were merely preliminary. Nevertheless, these studies were very relevant to a theoretical issue with which Davidson's group and many other workers have long been grappling: the state-trait issue. Henriques and Davidson (1990, 1991) had shown, as we have seen, that in one study, never depressed persons had A[1] scores much higher than currently depressed persons; in another

study they showed that never depressed persons had A[1] scores also much greater than previously depressed but *currently remitted, i.e., non-depressed persons*. They saw these data as supporting the notion that alpha asymmetry was a trait marker rather than a state marker; i.e., persons with the depressed asymmetry pattern had an innate "affective style" which made them vulnerable to depressive episodes in response to the stresses of life. We actually extended these findings by comparing all three groups within one study and observed that currently depressed and previously depressed but *currently remitted*, i.e., *non-depressed* persons both had lower A[1] scores than never depressed persons (Gotlib, Ranganath, & Rosenfeld 1998). This was consistent with the Henriques and Davidson studies, and the trait model of activation asymmetry. However, the neurofeedback studies immediately dislodged us from the pure trait position, because we reasoned that if the asymmetry pattern were fixed and innate, it would seem impossible to change it appreciably with any intervention. Indeed, the Henriques and Davidson studies, as well as the Gotlib et al. study, could be explained without a trait model: it could be the case that the normal asymmetry pattern is present in all individuals prior to a first depressive episode. Once this episode occurs, the pattern is changed and tends to linger. In this formulation, the asymmetry pattern is a result, not an antecedent of depression. However, it could be countered that the *range* of asymmetry values in an individual is fixed, and neurofeedback can produce changes only within the narrower individual range. The range is what may vary among individuals and the range may show properties of a trait. Our clinical studies with real patients, as described below, however, will indicate that neurofeedback can produce EEG and emotional changes which shift depressed patients' asymmetry scores well out of the depressive range as affect is improved. There is much other evidence that alpha asymmetry is a state marker in that it varies with the nasal cycle (Quinn 1998), and with simple passage of time over days, and that its value on a given day predicts affective response in psychotherapy (Rosenfeld, Baehr, Gotlib, & Ranganath 1996). All these results are summarized in detail in Rosenfeld (2000).

In order to deal with issues involving magnitude of change in patients or controls, it became necessary to develop our PCT index (see above) and to compare its values in patients vs. normals. Previous studies of this type had utilized either A[1] or A[2] indices. Baehr et al. (1998) undertook precisely this novel study involving 13 currently depressed out-patients and 11 individuals free of depressive affect. It was the first time PCT as a dependent variable was studied in this way. All diagnoses were based on DSM-IV interviews and on scores on the Beck Depression Inventory (BDI – a score of 10 was the

Table 1.

PCT at end of therapy	PCT follow-up	(years later)	BDI at end	BDI follow-up
(1) 84	86	(1 year)	3	3
(2) 86	66	(3 years)	4	4
(3) 69	69	(5 years)	2	3

cut-off for depression). We found that PCT scores <56 always characterized depressed persons, and that scores >59 characterized non-depressed affect. Between values of 56 and 59, were PCT scores from 4 of the 24 cases (17%), 2 depressed subjects and 2 non-depressed subjects. Of course there were significant *group* differences in A[2], PCT, and BDI, which supported the Henriques and Davidson (1990, 1991) and Gotlib et al. (1998) studies, however only the PCT score (and not the A[2] score) nicely discriminated *individual* members of the two groups.

The next step was, finally, to attempt the neurofeedback therapy involving operant conditioning of the A[2] score in clinically depressed out-patients (from the private clinical practice of the present second author, E.B.). These patients were all on medications with doses stabilized months before the beginning of the neurofeedback treatment (see Rosenfeld 2000, where details of the training protocol and patient demographics may also be found along with the results for four individual cases.) Psychotherapy sessions alternated with pure neurofeedback training sessions. Figure 1 below shows summary data. The figure shows that the neurofeedback protocol led to changes in the PCT scores from about 50, sd = 8.7 (i.e., well within the depressed range) to about 71, sd = 7.7 (well into to range of normal affect). The corresponding BDI scores fell from 28 (sd = 13.4) to 4.7 (sd = 6.2). The DSM-IV diagnoses after neurofeedback also indicated clear recovery from depression.

Since this initial study, two additional patients from the practice of Elsa Baehr were put successfully through the asymmetry protocol (making a total of six), and an independent investigator also reported success, for the first time, with an adolescent patient (Earnest 1999). The next question to investigate, which had not yet been posed, concerned the issue of long term results. That is, how long after the last therapy session do the positive effects – both EEG and clinical – endure? Baehr, Rosenfeld, and Baehr (2002) were able to examine three of the original six patients tested at one, three, and five years, respectively, after the last neurofeedback training session. The results for the three cases are as shown in Table 1.

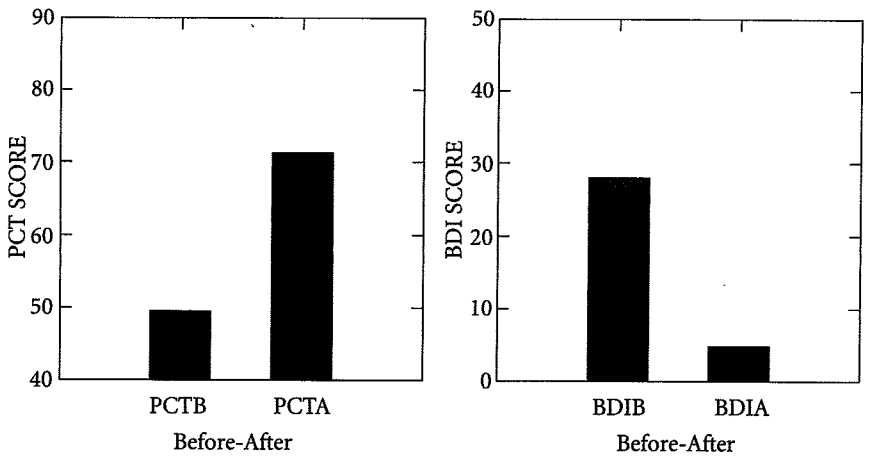

Figure 1. Mean PCT and BDI scores in a group of 5 previously depressed patients, before and after training with the alpha asymmetry protocol.

It is noted that the mean PCT score for these cases before therapy was 51.3 and the mean BDI score was 35.5. Clearly, the neurofeedback protocol appears to result in PCT and BDI scores well out of the depressive ranges of >58 and <10, respectively, and these values hold up after 1–5 years (Table 1).

These results allow us to suggest that it is possible, with a specific neurofeedback paradigm, to alter neural correlates of depression in a human patient, which, in turn, results in long-enduring emotional changes. Only the lack of a solid control group tempers this suggestion so that it cannot yet be a conclusion. We want to be able to say that it is the *learning of a specific change in the EEG which produces the psychological change.* However, given only the data so far collected, we cannot say this. This is because it could be objected that it is the patient contact and/or the psychotherapy component of the treatment package and/or the charisma of the therapist which produces the lifting of the depressive affect. This could happen in various possible ways: the psychotheraputic component of the experience could be a corrective emotional experience which results directly in positive affect which is then reflected, as expected, in the EEG; or, alternatively, the entire therapy experience could directly alter the A[2] scores, resulting in improved affect. In either case, it could be that *any* neurofeedback paradigm would have resulted in the same positive outcome. An ideal control would be a group just like the one treated with the asymmetry protocol, however given some other neurofeedback reinforcement contingency operating on a neural activity which should not affect the relative

left-right frontal cortical activation ratio. We have not run such a control group at this point, although it is noted that some of the original depressed patients had been in other therapy treatments (showing no affective improvement) for years prior to use of the asymmetry training protocol. Indeed one such patient had been in various forms of pharmacotherapy and psychotherapy – including one other ineffective neurofeedback therapy – for 12 years with one of us (E.B.) prior to showing any effects with the asymmetry training protocol.

An important finding in these clinical studies, even without a control group, is the fact that large changes in both PCT score and clinical function (as measured by BDI score and psychiatric interview) could be generated under experimental control, because these findings bear directly on the state-trait issue raised above. These findings showed that functionally effective changes in the PCT score – not just small changes within an innate range – could be generated by imposing the protocol. Even if it were the case that individuals are each born with an innate and characteristic asymmetry range, the fact that this range can be dramatically changed on a long-term basis with use of a clinically effective treatment protocol (including various components plus neurofeedback) suggests that the asymmetry score is not a rigid trait. However, we are *not* attempting to argue that once the neurofeedback protocol has been successfully applied and the PCT significantly changed, it cannot regress again, as illustrated in the next section.

Premenstrual Dysphoric Disorder (PMDD)

We have recently had the opportunity to study five cases of depressed women who were also suffering from *Premenstrual Dysphoric Disorder* (PMDD). This syndrome was first officially listed in DSM-IV (Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition 1994). A woman who has PMDD experiences *severe depression*, irritability and/or mood swings, which interfere with functioning. The symptoms begin 7–10 days prior to menstruation during what is called the luteal phase of the menses. Sometimes PMDD has been conceptualized as an extreme form of PMS (premenstrual syndrome), but it is known that the symptoms of PMDD are much more severe, and that PMDD has specific metabolic correlates (Endicott 2001). One of these women had been dose-stabilized on Zoloft, another on Wellbutrin, and a third on Prozac. Two others were untreated medically. All were run through the Alpha asymmetry training protocol. All these women initially presented with PCT scores well into the depressed range (<50), but as they were treated with neurofeed-

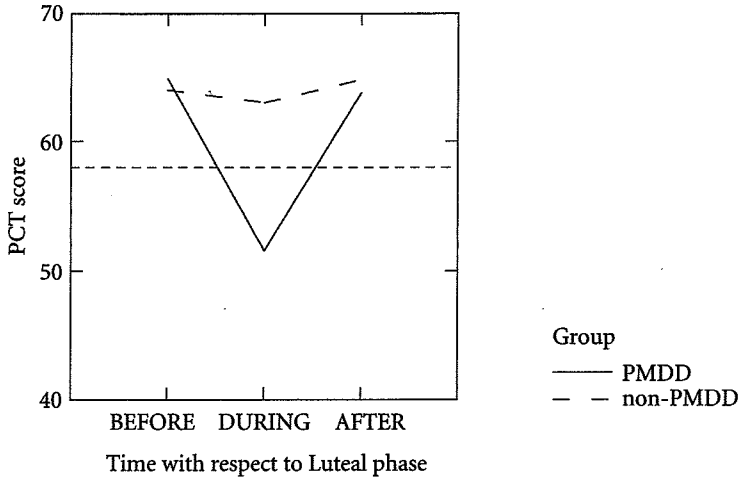

Figure 2. PCT scores of PMDD patient group and non-PMDD patient control groups before, during, and after the Luteal phases of their menstrual cycles.

back, they made significant gains, well out of the depressed range. They mostly remained in the non-depressed range and showed correspondingly normal affect, however during the luteal phase of their cycles, they each showed profound regressions as seen in Figure 2, in which a group of five demographically matched control cases (one on Zoloft, one on Hypericin, three unmedicated) are superimposed. These control cases also presented with depression, but were not diagnosed with PMDD. Only one cycle's worth of data is shown here, however data from second cycles are virtually identical. These data indicate two important points: (1) Although the alpha asymmetry training is effective at dramatically altering PCT scores and affect, these conditioned changes are not invulnerable to life's vicissitudes, such as severe PMDD luteal symptoms. (2) Aside from these relatively formal observations with PMDD cases, we have noted informally that previously depressed, non-PMDD patients in final successful phases of asymmetry training will show occasional regressions (in PCT and affect) always closely traceable to negative life events.

One concludes that the asymmetry protocol is a promising treatment modality for affective disorders, and that the measure itself (PCT) may be a useful diagnostic index for affect – it certainly discriminates PMDD cases and other women. The first conclusion must be tempered by our lack of systematic control data without which we do not know whether or not the therapeutic effects are specific to the asymmetry protocol. The second conclusion (about

diagnostics) must be tempered by our presentation of only five PMDD cases; given the rareness of PMDD it will take some time to gather more data. One additional conclusion which seems pretty safe to put forth is that our use of the asymmetry protocol has indicated that frontal cortical activation asymmetry is not simply a hard-wired, immutable trait, but is a reasonable state index and may be altered under operant control.

Summary

In this chapter, the term *Neurofeedback* was defined as a voluntary change in brain activity resulting from rewarding of target neural events, as in operant conditioning. It was shown with one example, the conditioning of evoked potential amplitude, that the conditioning is not trivially mediated. Then, via a literature review, it was shown that operant control of evoked potential amplitude in somatic sensory cortex produced changes in experimental pain threshold in animals. A clinical application of neurofeedback was then presented: the treatment of depression via the operant conditioning of left-right frontal cortical activation. Finally, it was noted that some sources of depression, such as the extremely negative affect and its EEG correlate experienced by women with premenstrual dysphoric disorder during the luteal phase of their menses, cannot be controlled either with neurofeedback and/or medication.

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CHAPTER 9

Consciousness, emotional self-regulation, and the psychosomatic network

Relevance to oral biology and medicine

Francesco Chiappelli, Paolo Prolo, Elaina Cajulis, Scott Harper, Elaine Sunga, and Edna Concepcion

Introduction: Consciousness and volitional control

From the viewpoint of philosophy, whence we should begin since it may be considered the master of all sciences (*scientia ancilla philosophiae*), the interpretation of consciousness has ranged from the "sense-certainty" of Heraclitus, to Plato's myth of the Cave. Here, the "soul" was said to progress a self-edifying journey from an initial state similar to that of a prisoner trapped at the lowest level of knowing and being in a world of hellish visions of sheer illusion. By an act of volition, the "soul-prisoner" became free, and commenced the long and arduous ascent out of the cave (equivalent perhaps to today's "emotional self-regulation" – *vide infra*). The "soul" eventually emerged into the clarity of the heavenly intelligible world - consciousness. In modern philosophy, Hegel described consciousness as rising through a series of historical levels. It preceded the view proposed by Kant, who revolutionized Modern philosophical thought by defending that it is the representation that makes the object possible, rather than the reverse. Kant argued that it is the consciousness of the object, rather than the object itself, that makes the representation of the object possible: that is, the human mind is the active originator of experience, through consciousness, not merely the passive recipient of perception. In the kantian view, perceptual inputs are processed, recognized, and integrated by the conscious mind, lest they be categorized as noise. Hence the twentieth century biologists, headed by Polany, described consciousness as a fundamental