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Research Recognition Award Paper

Applied Psychophysiology and Biofeedback of Event-Related Potentials (Brain Waves): Historical Perspective, Review, Future Directions[1]

J. Peter Rosenfeld[2]
Northwestern University

This paper reviews the efforts of workers in the 1960s-1980s to demonstrate voluntary control of exogenously evoked (event-related) potentials in visual, somatic sensory, and auditory systems in rats, cats, and humans. The first part of the paper reviews the conceptual foundation and development of the work—it actually arose from traditional sensory coding and neural correlates of behavior studies. The second part summarizes recent applications of the method in the area of pain control. In reviewing these matters, the major effort is directed at revealing how the ideas unfolded in very human, day-to-day, anecdotal terms. There is not much of an attempt to formally review the literature, which is cited for consultation elsewhere. In the same spirit, many possible future experiments are suggested by way of elucidating the key remaining questions in the area.

Descriptor Key Words: event-related potential; evoked response; P300; operant-controlled brain activity.

[1]Some of the research reported here was supported by NIH grants DE05204 and GM23696. I have tried to make this material of interest to the general readership of this journal by emphasizing how, on a day-to-day basis, we thought about and planned our studies. Thus, I've tried to tell a true story of where the ideas came from and how some of them got executed, rather than write a formal review (several of which can be found in the references for this paper). I would really appreciate your comments. If nothing else, a postcard from you will give me an idea about how many people are reading this paper, and I'd like to know.
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ORIGINS AND THEORETICAL OVERVIEW

The various biofeedback modalities now in use have had varying and often unusual histories. To be sure, some of the early psychophysiologists in the late 1950s and early 1960s had the notion that if emotions have physiological expressions, it might be possible to modify the psyche via operant modification of its physiological output. Biofeedback-assisted psychotherapy and relaxation can both trace at least one root back to these ideas from which they logically follow. On the other hand, as most of us are aware, much biofeedback arose from an issue in learning theory, as contemplated by one of the most eminent alumni of the Hull seminar in learning at Yale, Neal Miller. The issue concerned whether there was one, versus two (or several), fundamental learning mechanisms. The two-process theorists noted that there were two learning paradigms—classical conditioning and instrumental conditioning—which had evolved separately and had various differences. For example, while operant or instrumental conditioning seemed well suited to skeletal muscular systems (used, e.g., in bar pressing, key pecking, maze learning) and had a poor history of success with the ANS, classical or Pavlovian conditioning was first demonstrated on an ANS response (salivation) and continued to succeed as a means of modifying the ANS. Miller observed that unbiased, systematic efforts to obtain voluntary (instrumental) control of ANS responses had never really been attempted. The demonstrations from his laboratory in the 1960s that operant control of the ANS was quite achievable—despite attendant controversies as partly summarized by his 1974 chapter (Miller & Dworkin, 1974)—provided solid support for one-process theorists. Of course, these demonstrations also gave impetus to much clinically oriented biofeedback research.

Those of us involved in the operant control of event-related potentials (ERPs) started from an even more distant place: the classic neurophysiological fields of sensory coding and neural correlates of behavior. Steve Fox was the first to attempt ERP conditioning (Fox & Rudell, 1968). As he informally related to me while I was his graduate student, Fox was interested in how the brain represented or coded information. Having studied for a time at the Brain Research Institute at UCLA, Fox was schooled (and schooled *his* students) in the early work of Hubel and Weisel, Mountcastle, Bulloch, Granit, and others who, even a quarter century ago, were making great strides in developing our current knowledge of how the brain represents incoming sensory information, as well as outgoing motor commands. But presumably since he was a psychology Ph.D., from the University of Michigan, it was the psychological-behavioral event in whose neural representation Fox was interested. When he moved to the University of Iowa about 1964 (I arrived at nearly the same time), psychology was under the influence

of Iowa's longtime department chairman (and another eminent graduate of the Hull seminar), Kenneth Spence. Spence exemplified the midcentury behaviorist tradition of theory development in psychology, in which experimenters spent their time developing laws of behavior relating operationally defined behavioral variables. In this era, like-minded *physiological* psychologists were spending their time attempting to demonstrate physiological (especially neural) correlates of the laws being developed at the more molar-behavioral level by their experimentalist colleagues. Fox appreciated intuitively—for while a brilliant innovator and charismatic mentor, he was never much for disciplined, logical, or formal thinking—that there was some kind of inherent mismatch between behavioral and neurophysiological phenomena as formulated by most of his contemporary researchers seeking neural correlates of behavior. This inherent mismatch, he felt, doomed contemporary approaches of physiological psychologists to reliable neural correlates of behavior. For example, behavioral phenomena of interest (learning) took several trials to become measurable in dichotomous events (such as bar presses, conditioned response occurrences), whereas the putative neurophysiological correlates of these events (such as unit discharges and evoked potentials) had epochs measurable in milliseconds. It would be ultimately impossible, Fox argued, to meaningfully correlate such events that existed on "different time bases." This was Fox's somewhat raw appreciation of the problem of "*reduction*" in science, which was formally considered by Gustav Bergmann (1966). (Bergman was a former secretary to Albert Einstein and a member of the Vienna Circle of Operationalist Philosophy; he was imported to Iowa by Kenneth Spence to become the latter's logical overseer of his work in behavior theory.)

While still at the University of Michigan, Fox had the opportunity to observe some early studies of James Olds in which Olds trained rats to produce particular patterns of single neuronal discharge. Olds, so Fox told us, soon abandoned this work. He dropped the operant neural conditioning studies because he had been searching for learning centers in brain, which he felt he could claim to have found had he been able to demonstrate operantly conditioned neural events that occurred *without* a correlated peripheral expression—i.e., a concomitantly occurring motor event. As Fox related it to us, when Olds noted tail and body movements accompanying the conditioned neural events, he feared he was observing "mere" motor commands, and lost interest. (He might also have been distracted by exponentially growing fame associated with his putative reward system discovery as embodied in the self-stimulation phenomenon.) Fox, however, saw in Olds's discarded, first neural biofeedback demonstration, the possibility of a whole new approach to the study of neural coding of behavior. When he moved shortly thereafter to Iowa, Fox put his "operant controlled neural event" (OCNE)

program into effect. Having seen that it was possible to voluntarily control a neural event, Fox had an original series of insightful ideas about how a neural conditioning methodology might be utilized in a systematic attempt to decode the neural representation of behavior.

One subset of his ideas that appeared intuitively compelling on first thought did not hold up to later analytic scrutiny, although an *empirically* productive direction did ultimately emerge—Fox's controversial notion that since the first chore in a neural decoding program was *identification* of the neural parameters that could serve as neural coding "characters," the OCNE approach should be first used to identify these characters. This could be accomplished upon each successive demonstration of a new conditionable neural parameter. Fox argued that if a neural parameter (e.g., evoked potential amplitude) was conditionable, it had to be a potential psychological code, for why else would it respond to the psychological manipulation (e.g., of the neural reinforcement contingency)?

Remembering Olds's original cynicism would have served Fox well here. For although a conditionable neural parameter *could* be a psychological code, it could also be a simple motor code. For example, it is obvious that as one uncontroversially conditions a bar-press response, the motor neurons generating it (as well as the somatosensory feedback neurons encoding it) become conditioned also. The rat learns what he must do for the reward, and his motor system executes the commands, its neural activity changing of necessity along with his operantly changed behavior. These neural events do not necessarily encode a true psychological event (e.g., *learning*); they encode the motoric substrate of bar-press performance. Conditioning these same neurons directly, then, is not of great interest.[3]

The productive line of research that emerged from Fox's early neural coding formulations began with my doctoral dissertation, the basic impetus for which came from Steve. He was, as always, interested in the neural coding of behavioral events. He was aware, from the early work of Mountcastle, Poggio, and Werner (1962) at Johns Hopkins, that as an animal moves during the normal course of behavior, information from the moving limbs about their motion is relayed to the central nervous system. In fact, in the joints of the rotating limbs, pressure receptors emit an action potential (nerve impulse) discharge whose rate is proportional to the pressure in the joint (which, of course, varies with the *angle of joint angle rotation*). This rate code is integrated and relayed up to somatosensory cortical levels.

Fox was interested in decoding behavioral representation by EEG-derived population macropotential amplitude, not by single neuronal discharge, since in 1965 it was virtually impossible to reliably record single neuronal discharge over the course of a week in a *freely-moving awake* animal (the usual subect of an animal learning experiment). In an extremely influential paper (Fox & O'Brien, 1965), Fox had shown that the amplitude, at every point in a sensory cortical evoked (macropotential) response, was tightly correlated to the cotemporaneous neuronal impulse probability in a single neuronal member of the cell population generating the macropotential. This is not the place to explain why this single-cell-impulse/population-amplitude relationship exists.[4] Suffice it here to note that since the population evoked macropotential (EP) amplitude is related to the neuronal discharge, if the cortical neuronal discharge encodes the movement of a limb (Mountcastle et al., 1962), so should the amplitude of the macropotential EP evoked by the behaving limb encode the movement. Fox thus persuaded me (as a 3rd-year graduate student) that the OCNE method offered a whole new approach to the study of neural coding of behaviors.

We trained cats to execute stereotyped reaching movements from the floor to the wall of their 2.5-foot sided chambers. These movements evoked somatosensory cortical evoked potentials in the contralateral postcentral gyrus (primary somatosensory cortex). Whereas the followers of the neural-correlates-of-behavior approach trained behavior and looked back correlatively to see what the correlated effects in brain activity would be, we would reverse this process and operantly condition the movement-evoked potential (MEP) attributes and then see what aspects of behavior would also change. Somewhere in this persuasive argument of Fox, the (erroneous, but widely circulated) notion of treating the *brain-wave as the independent* (i.e., *directly manipulated*) *variable* was born.

Although I sensed that there were mistakes in Steve's pitch, I also sensed (as opposed to contemplated; Steve by example taught me that intuition was not always bad) that there was a potentially productive research program in it somewhere, so we proceeded. Indeed, we did show (Rosenfeld & Fox, 1971, 1972a, 1972b) that cats could alter MEPs under operant control and that predicted changes in the evoking movements followed. But what was the significance of this demonstration? It took a somewhat searching oral defense and a grueling NIH site visit in 1971, after I arrived at Northwestern, to force me to think carefully through the loosely conceptualized, but inspired and original, ideas of my mentor in order to find and appreciate their true value.

[3]We much later appreciated (Rosenfeld & Hetzler, 1979) that a nontrivial example of neural conditioning—for very different reasons, as will be considered later—required the *removal* of correlated motor activity.

[4]It is intuitively reasonable that if, as Fox hypothesized, the EEG or derived evoked potential (EP) amplitude is the complex integral of postsynaptic potentials (PSPs) from the recorded population, and if PSPs reflect proximity of neurons to their impulse/discharge thresholds or excitabilities, then impulse probability, which is an obvious expression of excitability, should correlate with EEG and EP amplitude.

First, let's clear away the misconceptions: Putting a reinforcement contingency on a brain wave rather than on a behavior does not alter the status of either as a *dependent* variable. The only thing we directly manipulate here is the reinforcement contingency, the *real independent variable.* We don't know in advance if the animal's brain waves or behavior will change (separately or together) in consequence of our manipulations. If we did, there would be no point in doing the experiment! Second, we were not really decoding behaviors as psychological phenomena of interest to our predecessors and contemporaries, who were more interested in neural coding of cognitive processes such as learning phenomena. We were dealing with simple motor processes (limb movements)—"behaviors" to be sure, but simple ones (microbehaviors) that were of more interest to physiologists than to psychologists. Finally, nothing we reported in our 1971–1972 papers established any coding relationship, based on *causal necessity,* linking even our microbehaviors and neural activity. We simply showed that when you condition the MEP, the evoking movement changes. This wasn't much of an improvement over the old neural correlators that Fox repeatedly took to task (e.g., Fox & Rudell, 1968; Fox, 1970) for merely reporting correlations from which causal lawful interpretations do not automatically follow. In fact, *our* findings were correlative also.

But here is what finally occurred to me as representing possible real value in this work: We could train cats to change discrete MEP components evoked by *natural* movements. We could also have trained them to change discrete components of their movements. We didn't actually do this, but common sense suggests (and literature shows) that patterns of movement can be developed with training (isn't this what atheletic coaches do?). What could be attempted *next* is a study in which cats are rewarded for generating the same changes in MEPs already reported in 1972, but with the added reward contingency that the movements expected (from the 1972 correlations) to accompany the MEP changes be *prohibited.* (Since in these studies a computer monitors and controls all aspects of MEPs and movements instantaneously, and then determines whether or not a reward is due, this hypothetical, complex reinforcement contingency is quite feasible.) If we were to find that cats prevented from executing previously seen movement correlates of conditioned MEP changes are out-performed by cats who are not so restricted (i.e., in movement), then it would be reasonable to argue that the particular movement effect is necessary (and thus is encoded in) the conditioned neural event.[5]

It is worth acknowledging that not much was done with the OCNE/movement paradigm after 1972, nor did we demonstrate anything beyond

[5]This hypothetical study has several subtle complexities, which were discussed recently in detail elsewhere (Rosenfeld, in press).

the feasibility of the approach in the first place. It is also worth emphasizing that this proposed combined application of reinforcement contingency to movement and neural activity is the *only* way to decode *voluntary,* naturally occurring (i.e., physiological) movement. The traditional approaches (e.g., Mountcastle et al., 1962) involve passive limb rotation (by experimenters) of heavily restrained and sedated animals. The neural representation of such movement probably differs significantly from the representation of normal movement, not only because of the emotional consequences of restraint and neuropharmacological effects of the sedative, but also because passive movement differs fundamentally from voluntary movement. The latter involves central feedback from the motor cortical command (e.g., via corticopontocerebellar pathways), which is absent in voluntary movement. To get at neural coding (by whatever neural activities; i.e., not just MEPs) of voluntary movement, *neural biofeedback may be an indispensable tool.*

Fox's other idea about the utility of his OCNE program was that it could be used to determine the intrinsic rules of any putative neural coding language. For example, suppose a foreign espionage agent was eavesdropping on encrypted broadcasts from our airbases to aircraft. His aim, like Fox's, is translation of a code. For simplicity's sake, suppose further that our coding scheme consisted of a letter replacement arrangement such that a given letter would be represented by a different letter, the one that ordinarily follows the former by two letters, with wraparound. The alphabet would thus be represented as shown below; *a* becomes *y, b* becomes *z,* etc. My name would be spelled *PMQCLDCJB.*

a b c d e f g h i j k l m n o p q r s t u v w x y z
↑ ↑
y z a b c d e f g h i j k l m n o p q r s t u v w x

Fox would have argued that in order to decode the scheme, the rules of the language plus a knowledge of our alphabet would first be necessary to obtain. For example, suppose the agent came to see that in the coded broadcasts, the character *g* always came before *c* except after *a,* and that a *o* was always followed by an *s.* If he then saw the coded word *OSGCR* and had hypothesized that *o* and *s* were encoded *q* and *u,* respectively, and that *g* and *c* were *i* and *e,* he would then know that *n* was a coded *t* and that *OSGCR* = *quiet.* Having knowledge of these five letters might soon lead to observation that each was a two-shifted encryptation. Cracking the code entirely would shortly follow.

Obviously, a real encryptation, let alone a neural code, would have more complex rules, but the approach to decoding would still begin with *working out the internal rules of the code.* The OCNE approach could help accom-

plish this with putative neural codes. The neural event of major interest to Fox was the sensory evoked potential (EP). It was possible that each of the obvious attributes of EPs—the slope and amplitude of each of the six or more components usually seen in an EP—were potentially independent, information-carrying characters of a neural code. If this hypothesis is correct and if each component slope and each component amplitude can exist in two states (e.g., high and low), then 24 characters are available to form words (6 components times 2 attributes—slope and amplitude—times 2 states—high and low). A 24-independent-character code compares well to our 26-character alphabet code, not all of whose characters are independent since *u* always follows *q* and *i* comes before *e* except after *c*, etc. But how do we see if we really do have these characters available, and what their mutual interdependencies are? Apply a reinforcement contingency, Fox would answer.

The earlier (*exogenous,* see Donchin, Ritter, & McCallum, 1978) components (up to 100 msec) of an event-related potential (ERP) really comprise the sensory-evoked responses (EPs) of the cortical (or scalp) recorded ERP. The later (*endogenous*) components (100–1,000 msec) are thought to be related to psychological events (and, as in the case of the P300 wave at 300–800 msec, can be recorded in the *absence* of an expected physical stimulus). Thus the earliest OCNE studies focused on 100- to 200-msec components that were relatively variable exogenous waves and mediated by reticulocortical rather than specific sensory thalamocortical pathways (Fox & Rudell, 1968; Rosenfeld, Rudell, & Fox, 1969). But even though the 100- to 200-msec EPs were more variable than 10- to 50-msec components, they too were exogenously evoked sensory responses. Indeed, because the neurophysiological community in the 1960s regarded the 100- to 200-msec components as "hard-wired" responses to sensory input, the initial report of their conditioning (Fox & Rudell, 1968) provoked much excitement. Fox was not amazed since he predicted that they would condition. He had already noted their spontaneous trial-to-trial variability in the face of relatively constant stimulus parameters, and argued that changing psychological states affecting their substrate pathways were the likely sources of the variance. Thus, it may be inaccurate to regard them as purely exogenous (as many people still do) since the cat's ability to voluntarily control them indicates their partial determination by endogenous influences.

Are the *earliest* components (< 20 msec) purely exogeneous? Most of us, even in Iowa City, the birthplace of operantly controlled ERPs, must have felt so since we mostly avoided the early components. Alan Rudell—probably the most hard-nosed of us at the time—was not so certain. He knew that lateral geniculate (visual pathway) responses recorded simultaneously with conditioned cortical responses did not correlate (Rudell, 1970). He also

had gone further than anyone at the time to control stimulus parameters by locking the cats in place, thereby fixing receptor orientation (Rudell, 1970), while successfully training them to alter flash-evoked ERPs. Maybe visual phenomena and operant-controlled visual evoked components were completely independent, strange as that sounds. This hypothesis, of course, bears directly on how many characters (independently conditionable) are available for coding, and returns us to the point raised two paragraphs ago.

Very briefly, Rudell reported (Rudell & Fox, 1972) that the earliest "primary" flash-evoked components readily conditioned, to the surprise of the neurophysiology community, as well to some of his Iowa City colleagues. Moreover, not too long after that, he demonstrated operant control of cortical components evoked one or two synapses below the cortex via an electric shock to the optic radiations of the internal capsule (Rosenfeld & Rudell, 1976). We had to begin thinking that *purely exogeneous components of any latency did not exist!* Quite apart from Fox's concerns about working out neural coding laws (which Rudell's work really didn't directly address; he asked only about *available* coding characters), Alan's findings about the conditionability of early evoked components made me (at least) appreciate something important about cortical neuronal function: Neurons in sensory cortex have *functional* inputs from nonsensory connections.

Everyone knew about the input anatomically; everything in cortex is connected to everything else in cortex. But the conditionability, independent of sensory pathways, suggested that these nonsensory anatomical connections mediated the mental state the subject generated, which modulated the sensory response delivered over the sensory pathways. It became clear that the sensory stimulation was a mere technicality: We were just using it to reliably locate the time locus in ERPs at which subjects could cause cortical changes via *other* mechanisms. What are these?

This is the question I raised about operant EP control: How do they do it? (Rosenfeld, 1974). We don't know the answer to this date, but chances are that cognitive processes—imagined events—are involved, as we learned very early on by asking human subjects about their OCNE strategies (Rosenfeld et al., 1969). This, incidentally, suggests that *ERP biofeedback could be used to confirm guided imagery* in much the same way that EMG biofeedback is used to confirm relaxation training! This has never been done (probably because it seems a bit technically daunting, but it really wouldn't be that hard to do).

Nor has there been a really systematic attempt to follow through on Fox's suggestion of using the OCNE approach to work out internal rules of a neural language in ERPs. Rudell just broached the issue by beginning a catalog of conditionable components. No one has yet systematically addressed

the issue of which attributes of which components are independently conditionable, although we have often observed some evidence of independence of amplitude effects in neighboring components, even without explicitly requiring them in the reinforcement contingency (Rosenfeld, 1977). That is, we don't know intrinsic rules such as "*i* before *e* except after *c*." In ERP terms, this might be a rule that states, "When you condition an increase in component X amplitude, its slope and the amplitude of another component, Y, must decrease." The only way to validate such hypothetical rules is by seeing if they can or can't be disrupted under operant neural control.

I didn't follow up the initial OCNE thrusts for a variety of reasons. First of all, I graduated in 1971 and went to Northwestern. To continue my doctoral studies of movement coding I needed my own research grant. I did not get a grant for this work with my first application, and since I did manage to get funding for other projects, and since new assistant professors are under pressures to produce, I proceeded with the other projects. The other projects I refer to pertained to "how do they do it?" questions. That was *my* interest from the beginning. How can cats, rats, and people change sensory evoked components? Fox didn't seem to care that much; I did. My master's research, the first study I did in the OCNE lab, was a "how do they do it?" study. Rudell and Fox had worked with cats; one can't *ask* a cat how it changes visual evoked responses. Nor were any hints forthcoming via direct observation of a cat during a successful conditioning run. My idea was to repeat the Fox and Rudell (1968) cat study in humans, whom one *could* ask about mediation. As noted, we did this, and the human subjects reported *imagining* various things as their mediation strategy (Rosenfeld et al., 1969), which was probably true and intuitively reasonable. After this, Fox got me interested in the movement studies, but when I went off on my own to Northwestern, I got back to my own original question: How do they do it?

In fact, I was much more concerned with ruling *out* possible *trivial* ways they could have been doing it; this is the mediation issue I worked on with Bruce Hetzler at Northwestern from 1970 to 1975. I didn't want to devote my research career to pursuing trivial phenomena, so I felt impelled to satisfy myself that voluntarily controlled ERPs were not trivially mediated.

Before getting into this, a distinction should be made between the situations with a subject's own movement-evoked versus "ordinary" sensory-evoked potentials.

There could be some confusion here. In view of the voluntary movement studies, one could legitimately wonder why there would be concern about mediation—i.e., how the subjects voluntarily control brain waves. After all, we had already shown that when dealing with a wave evoked by somatosensory feedback from the joints subserving a voluntary movement, subjects change the waves by voluntarily changing the evoking movements.

Nothing out of the ordinary here. In fact, the application of the OCNE procedure to movement decoding is a special, *exceptional* case of neural biofeedback. It is the case of our having (and taking advantage of) good *a priori* knowledge of the major source of input to a sensory-evoked response: the sensory input. We were asking the cats to change the movement-evoked wave and, in effect, inviting them to do so by changing the movement evoking this sensory input. This was the case since, apart from a maximum allowed duration of movement, we had no restrictions on the movement whatsoever. The cats could execute any movement they liked; i.e., we made no attempt to keep the sensory input—the movement feedback—constant. On the contrary, we wanted and expected the cats to vary movements from trial to trial so that they could readily learn which type of movements would produce waves that met the reward contingency. Our interest was in the relationship (the encoding) of sensory feedback from voluntary movement and neural activity.

In contrast with this situation, all the other studies of operantly controlled ERPs, as noted above, included at least an implicit attempt to keep the sensory source of the ERPs *constant*. Until the studies we reported in 1976 (Rosenfeld et al., 1976), this was usually accomplished by having a photic stimulator set at constant intensity as the animal was contained and run within a box with walls, floor, and ceiling painted a highly reflective white. In Rosenfeld et al. (1969), *earphones* were used to deliver tones at constant intensity, pitch, duration, rise-time, etc. As noted earlier, the sensory stimuli were virtually incidental to our interests in these studies. They served to mark the origin and origin-referred time loci in the recording epoch. The psychological influences on the recorded (conditioned) waves were our major interests, and the sensory evoking stimuli were at best analogous to carrier frequencies in broadcasting. When we listen to AM or FM, we are listening not to carriers of the signal but to the sound-encoded amplitude modulations or frequency modulations that ride on the carriers. When we look for operantly conditioned changes in a sensory-evoked component, we are interested in the operant *changes* in these components, not their pretraining values, which are direct functions of stimulus parameters. What is of novel scientific interest in the movement-encoding studies is knowledge of how the brain represents sensory feedback from voluntary movement. The OCNE approach is a tool to that end. What was and remains of interest in the other neural operant studies is how (over what pathways, using what strategies, etc.) subjects can cause psychological events to influence components whose basic presence is determined by constant sensory (extrinsic) input.

Indeed, it became important to me in the 1970s to make absolutely certain that when subjects voluntarily change sensory-evoked brain waves, they *cannot* do so by altering sensory input or by generating movements produc-

ing correlated neural events capable of satisfying the neural reinforcement contingency. Nontrivial discovery in science means novel discovery, not roundabout rediscovery of existing knowledge. We already knew (from Rosenfeld & Fox, 1972b) that subjects could alter neural activity by altering movement. It was also known that the size of a photic-evoked ERP is determined by brightness of the evoking flash. Thus, it would have been disappointing indeed to have learned that the original operant conditioning of cats' photic-evoked responses (Fox & Rudell, 1968) was mediated by the cats' looking into or away from the evoking flash stimulus, thereby indirectly regulating stimulus intensity. I have written extensively about this "trivial mediation" issue elsewhere (Rosenfeld & Hetzler, 1979; Rosenfeld, Hetzler, Birkel, Antonetti, & Kowatch, 1976; Rosenfeld et al., 1983) and will here state only that prior to the Rosenfeld et al. (1976) report (including Bruce Hetzler's Ph.D. studies, which, along with Al Rudell's pioneering work and Bob Dowman's 1980s Ph.D. studies to be described below, I consider to be among the most significant OCNE studies ever reported), there were no completely controlled, ironclad demonstrations that operant ERP conditioning was a nontrivial phenomenon; Rudell's work in Fox's lab was very suggestive, but Hetzler did the absolutely convincing study. He at once ruled out mediating movement-related activity along with indirect stimulus parameter control, yet the rats still learned, and learned readily and convincingly, to alter visually evoked ERP amplitude both upward and downward. The control for movement-related activity was threefold and partly indirect; the interested reader is referred to Rosenfeld et al. (1976) for details. It can be noted briefly here that the stimuli were presented at random intervals, so that the rats could not timelock a movement to the intertrial interval such that its phasic somatosensory feedback or efferent command could generate a criterion-satisfying amplitude. Also, the component conditioned had a relatively early latency of 70 msec; i.e., the stimulus itself could not be a cue for a phasic, mediating movement since the 70-msec component occurred prior to the time that the subject could command a movement. Hetzler's method of removing the animal's ability to indirectly control stimulus parameters by altering receptor orientation (looking into or away from the light) was truly heroic: The stimulus was a constant mild electric shock to the optic chiasm via a chronically implanted electrode (to which the rats could, obviously, not control their orientation). Moreover, prior to being run in total darkness, the rat was dark-adapted for several hours in the home cage (to stabilize the visual system) and transferred to the running chamber in total darkness. (Only Hetzler, a skilled magician in his spare time, had the hand skills to do this feat.) Indeed, Hetzler had such extraordinary total controls planned that I was somewhat dubious and anxious about the project's success. If it failed, I would

face the possibility of having spent 7 years of my life chasing a trivial phenomenon. No wonder I remained in Europe that summer for a few weeks after the end of the 1976 NATO-supported (Munich) biofeedback meetings—Bruce was in the middle of his studies and I was probably afraid to return home to see a possible failure!

Happily, it worked—but when I realized this, I became even more bewildered, and excited about this phenomenon's real significance than I had back in 1967 when Al Rudell and I stood outside his running chambers, peering through spyholes at one of his cats who was generating hit after hit after hit while giving no hint in his overt behavior as to how he was doing it! We still don't know. Indeed in 1984–1985, Bob Dowman and I spent 30 hours repeatedly viewing videotapes of his rats as their sensory ERPs were being nontrivially conditioned in a very different kind of experimental situation (to be reviewed shortly; Dowman & Rosenfeld, 1985a). We could not point to a mediator then, either; we could not even cautiously suggest one.

Actually, it is good that there is no simple (let alone trivial) mediation. Apart from reasons already suggested, it is worth pointing out that (as I have written elsewhere: Rosenfeld & Hetzler, 1979; Rosenfeld et al., 1976) one way to conceive of nontrivially mediated, operantly conditioned neural events is as representing conditioning in a *novel response system.* These new systems are necessary for testing the generality of suspected laws of learning. For example, the ideal CS-US interval for classically conditioned eyelid responses may be about 500 msec, but if one utilizes a different system (such as the taste aversion conditioning system), one can observe an ideal interval of several hours.

This argument may sound impressive (and it has fit needs of significance sections of grant applications), but candidly, I never did have any plans to test the generality of putative operant laws.The nontrivial, operantly controlled neural event is a mystery whose mediation will probably remain elusive for a while, even though the phenomenon is and will remain robust. This is what, for me, makes it so perpetually engaging.

On the other hand, of course, perhaps we have a pretty good idea of what subjects are doing after all: They are using their imaginations and manipulating cognitive contents. That's what they told us in 1969 (Rosenfeld et al., 1969), when my bewilderment at watching Rudell's cats prompted me to persuade Steve Fox to let me transpose the Rudell experiment to humans, who could be *asked* about their strategies of mediation. These verbal reports are still problematic, however, because we don't yet have means of independently confirming through direct observation that people are using imagination when they report doing so. We *can,* for example, confirm reported muscle tensing via EMG recording, but there is no "EIG" ("electroimagina-

gram") yet available. So in operant ERP control we have a phenomenon not trivially mediated by simple observable behavioral correlates and probably aided by unobservable cognitive, self-manipulative strategies.

I'm not suggesting that there is no way to ever get at the putative cognitive mediation. I think it will take some really innovative thinking, novel methodologies, and dedication. It must surely be done in humans, if cognitive mediation mechanisms are to be investigated. This should be technically easier, of course, even if the conceptualization is not. It is striking to me that so few people have pursued the mediation of *any* biofeedback phenomena, but remain content to utilize the phenomena clinically. Only Rudell, Hetzler, and I have done systematic mediation studies with neural biofeedback, and even these were designed to *eliminate* certain trivial mediators, rather than reveal the actual mediation mechanisms. Moreover, I must admit that the application possibilities have attracted me also in recent years.

APPLICATIONS

When I returned from Europe in the summer of 1976 and Bruce Hetzler showed me the remarkable set of data he had collected for his dissertation—the data set leading to the report that operant EP conditioning in awake, unrestrained rats is not trivially mediated (Rosenfeld et al., 1976)—I felt a sense of release (and relief). There could not longer be any doubt that there was a real phenomenon here. We still didn't know how the subjects did it, but now we knew how they did *not* do it (i.e., trivially), and so felt free to pursue functional significance.

It occurred to me that however they managed to do so, these rats of ours were altering the state of neurons in the sensory (visual) cortex that normally play a crucial role in visual perception. Was the rats' visual perception then changing in consequence of the operant changes in sensory neurons?[6]

To ask this question experimentally in rats required a complex study indeed. One cannot do a visual perception study in rats as easily as one can in humans, who can be asked about what they see. In animals, one would ordinarily set up a visual discrimination paradigm (e.g., a brightness discrimination), and once the rats were trained to discriminate, say, black from white, one could progressively decrease the discriminability of the stimuli until the discrimination could no longer be made. The seriously daunting aspect of this approach involves the necessary combination of discrimination and

[6]It was around this time (1976) that I realized I should start becoming more active in what we then called BSA (Biofeedback Society of America), since this was a question about a new biofeedback application.

OCNE training required by the research question. If we knew that operantly controlled changes in visual cortical function were long-enduring (i.e., they lasted for several days), then we could simply do the brightness discrimination test the day after the last OCNE training day. Of course, we didn't know whether there would be *any* functional effect—even a short-lasting one. After all, this is the question the experiment is designed to ask. Therefore the study would require an interweaving of brain-wave and brightness-discrimination training so that discrimination test trials could be given immediately following a successful series of brain-wave hit trials. The rats would thus, in effect, be learning two tasks at one time. Maybe rats can manage such a feat, but upon consideration of the difficulties involved, I decided to get out of the visual system and into the pain business, which, as will be soon clear, allowed for a much easier test of functional significance.

There were actually a number of inducements for me to transpose the question of functional significance of operantly controlled EPs into the somatosensory (pain) system. The first, of course, was the one I alluded to in the previous paragraph, the issue of feasibility. In contrast to vision or other senses, the pain sensation leads to innate defense reactions in animals (and people)—i.e., a reaction that they do not need to be trained to execute. Thus, there already existed a palette of pain tests for use in rats. Most of these involved nociceptive responses to noxious stimulation of the lower body. For example, in the tail-flick test, a beam of focused light is applied to the rat's tail, which flicks when the heat mounts to a painful level. My own laboratory, which since 1972 had been as involved with traditional studies of pain and analgesia mechanisms (e.g., Rosenfeld & Stocco, 1980, 1981; Broton & Rosenfeld, 1982; 1985) as with neuronal biofeedback, had developed a method of testing pain in orofacial areas (Rosenfeld, Broton, & Clavier, 1978). It was the device we would ultimately decide to use for our functional significance studies of operant EP control, since the EPs we would be conditioning were to be recorded in orofacial representation areas of brainstem and cortex. This device involved a simple heater attached to the rat's face, overlying the cheekbone. Heat was applied until the rat began vigorous rubbing at the device so as to dislodge this noxious heat source. We would of course terminate the heat upon first nociceptive response, and the time from heat onset to offset was recorded as an index of inverse heat sensitivity (inverse because the longer the rat could tolerate the heat, the less sensitive he was). This system was one among many available in which the rat's response (the face rub) was innate and thus required no training to conceivably confuse the rat in the midst of his EP training.

Besides satisfying the issue of feasibility, the pain sensation was ideal for making the case that operant EP control was functionally significant in the sense of being useful; pain is the most common symptom of many pathol-

ogies. If neural biofeedback could be rigorously shown to have a direct, specific analgesic effect, all other things notwithstanding, I would have the gratification of knowing that my scientific career had some tangible benefit to society.

One final consideration in choosing to move, after many years, from the visual to the somatosensory (pain) cortex: The role of pain in disease—its "relevance"—greatly increased the likelihood of obtaining research grants to support the work. These three considerations—feasibility, personal gratification, and greater availability of grant funds—made for an utterly compelling reason to shift from visual to pain-related event-related potentials.

The next decisions were practical: What stimulus should be used to evoke the potentials? From where should they be recorded? How should functional effectiveness (analgesia) be measured?

EP conditioning in unrestrained, awake rats can occur in 1 session or 10 sessions, depending on the particular rat's intelligence and personality. One session cannot be counted on; therefore one must expect a multisession training period. If the somatosensory evoking stimulus is to remain constant (which of course it must), it probably is not a good idea to plan on constant *peripheral* stimulus delivery, as one can in a human subject. One can, in a human, reattach stimulating devices (e.g., for electric shock) in more or less the same place several days running. This can't be easily done in an unrestrained rat, who would probably spend most of the training session trying to dislodge the electrodes (or mechanical stimulators or whatever). Moreover, it is by no means certain that peripheral tissue would stay unchanged after several days of any kind of stimulation. If the tissue changes, so will the stimuli.

It quickly became clear that we would have to again apply stimuli centrally as electric shocks to the pain pathways. Obviously, animals cannot and do not attempt to alter indwelling, chronically implanted electrodes, of whose presence they are probably unaware. Moreover, since we had to implant *recording* electrodes for long-term recording, why not implant *stimulating* electrodes also? The thing to be careful about here, however, is to be sure that the stimuli applied through the electrodes were themselves not painful. It takes 500 trials per session for several days (1 session/day) to train EPs. To plan on delivering this many hurtful stimuli to an animal in training would not only be cruel, it would also be self-defeating, since traumatized creatures are unlikely to learn anything except helplessness. So we had to plan to deliver *nonpainful electric stimuli to pain pathways.* Was this an impossibly paradoxical plan? Not really, because (as detailed in Rosenfeld, Dowman, Heinricher, & Silvia, 1984) there are some somatosensory cortical neurons (wide dynamic range neurons) that respond to nonpainful as well as to painful stimuli. Our thinking was that we might succeed in nonpainfully activating them repeatedly for conditioning purposes, and thus, via conditioning, put them in an altered state so that when their response to rare painful *probe stimuli* was tested later, the analgesia would be detectable. One final advantage to using central stimulation: The receptor orientation factor would be controlled with respect to the trivial mediation issue (as Hetzler and I did earlier in the visual system).

Where, then, to electrically stimulate in the somatosensory pain system? Rudell had shown cortical conditioning of EPs recorded to shocks to the optic radiations just one synapse removed from cortex (Rosenfeld & Rudell, 1976), and Hetzler conditioned cortical potentials in response to sensory pathways immediately upon their entrance to brain in the optic chiasm (Rosenfeld et al., 1976), so we had a wide range of choices. Actually, the first question to answer concerned where to *record* (i.e., condition) the EPs: at the bottom or the top of the neuraxis? Wide dynamic range neurons occur at the secondary sensory (dorsal horn) level as well as in cortex. We had no way of knowing *a priori* where in the neuraxis conditioning would have the largest functional effect (if conditioning anywhere would have any effect). I had two excellent graduate students at the time, Mary Heinricher and Bob Dowman. I sent Mary to the "bottom" (the primary afferent terminal area) and Bob to the "top" (somatosensory cortex) of the neuraxis to record the to-be-conditoned EPs. If these first attempts failed, Mary would move up a synapse (to, say, ventral basal complex of thalamus) and Bob down (to reticular nucleus cuneatus) until we met with success somewhere in between.

This decision made the other (where to put the *stimulating* electrodes) automatic: If we were going to record EPs from the primary afferent terminal region, we would have to evoke them at an even lower level. There is only one level lower than the primary terminal area in the CNS—the incoming primary afferent axons—so this became our target area. It was accessible in two places, one being the spinal cord dorsal horn. To chronically implant this area in a tiny rat spinal cord was all but impossible (likewise the related primary terminal area for *recording*). Happily, the other possible stimulating target, the primary descending trigeminal tract (PDTT) in medulla, was accessible stereotactically, and the derived electrode leads could be led to the standard skull socket for chronic recording. Moreover, we had experience in this system from our pure trigeminal system studies (Broton & Rosenfeld, 1982, 1985, 1986). Thus, the stimulating target was to be PDTT. The primary afferent termination area (for recording and conditioning by Heinricher) would be the trigeminal nuclear complex, and the cortical conditioning area (for recording by Dowman) would be the orofacial somatosensory cortex. Since the trigeminal system represents orofacial pain, the orofacial heater described above was used to assay functional significance.

As detailed in Dowman, Rosenfeld, and Heinricher (1983), Heinricher, Rosenfeld, and Dowman (1983), Dowman and Rosenfeld (1985a, 1985b), Rosenfeld et al. (1984), and Rosenfeld, Silvia, Weitkunat, and Dowman (1985), the results were that (a) trigeminal nuclear potentials were conditioned (though with difficulty), but there was *no* evidence of accompanying analgesia: (b) trigeminal evoked *cortical* potentials are readily conditioned, and there is a robust accompanying alteration in pain sensitivity; (c) the cortical effects are specific to the conditioned cortex (other areas' responses are unchanged); (d) the analgesia is specific to the side of the face represented in the conditioned side of cortex; (e) the pain sense, but not other somatic sensations, show changed sensitivities with cortical EP conditioning; and finally, (f) the analgesic effects are reversed by the opiate antagonist naloxone. These results were most exciting not only because we established functional significance (analgesia) but because the functional effect was too *specific* to be of a trivial or placebo nature; i.e., there was "somatotopic, submodality, and opiate-receptor-specific analgesia" (as I have been heard to say). I might also add that the *degree* of analgesia was significant; it was comparable to the effect of a moderate morphine injection.

These results were extended to normal humans in 1985 (Rosenfeld et al., 1985, resulting in a story in the popular press; Travis, 1984) and now await testing on real-pain patients. In the meantime, my colleagues in Europe (Miltner, Larbig, & Braun, 1988) reported similar effects using a vastly different type of stimulation (this was most gratifying!). I hope others with access to real-pain patient populations and interest in developing a new kind of biofeedback-produced pain relief will contact me to collaborate.[7]

EPILOGUE

I do hope that others will get involved in this research. Fox himself warned me two decades ago that OCNE research, which sometimes seems of borderline scientific seriousness to some traditional researchers, would not be popular (read: easy to fund) in hard times for scientific research. He was right. It has not been easy to keep this all going, and I have been starting to do some other things lately (applied psychophysiology of the P3 event-related potential in various kinds of personality, pain, and honesty assessment; e.g., Rosenfeld, Angell, Johnson, & Ivanov, 1989). Neither do I wish

[7]For a few years (1985–1988), it was difficult for me to access such a population locally (where "access" means "rigorously experiment upon").

to imply that those named here are the only ones who have worked in the area. Michel Roger (1984) in France and AAPB's own Bill Finley (1984) have published laudable efforts in both the theoretical (mostly Roger) and applied (mostly Finley) areas involving operant EP control. There were others[8] also involved without whose help the work would never have come even this far.

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[8]E.g., Dave Walker, Bob Owen, Bill Kosnik, Dianne Antonetti, and especially Paul Birkel, Bob Silvia, and Rolf Weitkunat (Weitkunat & Rosenfeld, 1986).

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