

TOWARD A PSYCHOBIOLOGICAL THEORY OF BORDERLINE PERSONALITY DISORDER:

Is Irritability the Red Thread that
Runs Through Borderline Conditions?

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ABSTRACT

This paper proposes a psychobiological model of the borderline conditions that explores the role of a hyperirritability that may either antedate parent-child interactions or stand apart from traditional developmental stages. It suggests that one pathway toward this hyperirritability is the traumatic effect of abuse, which may alter the neuroregulatory response system in ways that cannot be accounted for in purely developmental models. The therapeutic implications of this model are reviewed.

*... The brain is placed between two orders of stimulation, those which proceed from the nerves of the external senses, and those which it receives from the nerves of the internal viscera.... (once) Excitants... having acted with too great energy, and during too long a time, the brain... assumes a state of irritation; innervation becomes excessive, which appears by an augmentation of sensation and motion. F.J.V. Broussais, *On Irritation & Insanity*, tr.: T. Cooper, M.D., 1831, p. 233.*

So then, this exuberant activity of memory, its bizarre combinations of the imagination, reduce themselves, physiologically speaking, to an excessively lively and tenacious action—to an irritation of the intracranial nervous substance subserving the operations of the intellect.... memory cannot be explained other than as a cerebral excitation, which renews itself even in the absence of the cause which had long ago provoked it. F.J.V. Broussais, 1828, pp. 447-8. (Tr. by the author).

May not these melancholy departures from ordinary and healthy modes of thought, impulse and action constitute evidence ... of undetected, unperceived, unrecognized mental disease, in all probability arising from cerebral irritation...? F. Winslow, 1861 (p.160).

INTRODUCTORY REMARKS

The current definitions of borderline personality disorder (BPD) derive mainly from earlier criteria promulgated by Kernberg (1967) and Gunderson and Singer (1975). Spitzer et al (1979) selected key elements from these formulations in establishing what was then called the *unstable* variant of borderline personality. A year later these eight items were incorporated into the criteria for borderline personality disorder in DSM-III.

Any theory about the etiology of borderline conditions must take into account the typical clinical picture evoked by this term. Subtle shifts in usage over the years make it important to keep in mind the evolution of the borderline label, so that the various theories may be evaluated in accordance with the nosological species they were attempting to explain. Elsewhere I have commented in some detail upon this evolution (Stone, 1977, 1980, 1985). Borderline at first described a group of heterogeneous and loosely defined patient-types situated between the turn-of-the-century concepts of psychosis and neurosis. Included in this nebulous region were psychopaths (Prichard, 1838; Lombroso, 1878), *formes frustes* of manic-depressive psychosis (Kraepelin, 1909), dilute forms of schizophrenia (Bleuler, 1911; Oberndorf, 1930; Moore, 1921) along with a number of other nondelusional emotional or mildly organic disorders (Rosse, 1890). No coherent theory of causation emerged, so long as the term remained so nonspecific. In the absence of common clinical features, the search for common etiological factors would in any case have been futile.

This situation changed fifty years ago with the publication of Stern's paper on borderline conditions (1938). With this article, borderline found a home within the psychoanalytic community, where it was to remain, until the psychogeneticists provided it with more specific descriptors in 1968 (Kety, Rosenthal, Wender, & Shulsinger). These investigators had been zeroing in on the then prevalent notion that borderline signified proximity to schizophrenia. Many prominent psychoanalysts at midcentury wrote of borderline in this fashion—often as an aside—since they were interested not so much in etiology as in psychotherapy (Deutsch, 1942; Bychowksi, 1953; Knight, 1953). The schizophrenia of which the borderline patient was either a close or a distant cousin was the broad Bleulerian schizophrenia, not the more narrowly defined schizophrenia of contemporary research criteria. Thus the analysts at midcentury, if they focused on distal etiology at all, assumed that whatever abnormalities underlay schizophrenia were, by extension, also present, albeit to a lesser degree, in the borderline cases. For a long time the psychoanalytic community ignored or underestimated the biological underpinnings of schizophrenia, likewise of borderline schizophrenia (Rosen, 1949; Rosenfeld, 1956; Arlow & Brenner, 1964; Jackson, 1960).

In the mid 1960s Kernberg fashioned a more coherent theory of borderline conditions, selecting a few attributes mentioned by his psychoanalytic predecessors (Deutsch, Klein, Polatin, and Frosch) as central to his conception of borderline personality organization. He described his main inclusion criteria—diffusion and adequate reality testing capacity—in psychological terms, though acknowledging a contributory role from the biological side in the form of (excess) innate aggress-

sion. The latter, Kernberg postulated, was the *primum mobile* behind the often dramatic displays of rage and aggressivity one confronts, either in the anamnesis or during one's actual clinical work with borderline patients. Jean Bergeret's concept of *la violence fondamentale* (1984) is closely related to this hypothesized innate aggression.

Though the theoretical formulations of Kernberg and Bergeret are more systematic in their approach to borderline states than were the earlier psychoanalytic interpretations, they are still predominantly psychological, oriented toward clarifying therapeutic rather than etiological questions and as a result still spread out, in the case material to which they relate, over a wide territory, when viewed from the perspective of distal etiology. Around 11 percent of the population would satisfy the Kernberg criteria for borderline personality organization (Stone, 1988). This domain includes DSM schizotypals, BPD patients, sociopaths, agoraphobes, substance abusers, most dysthymic and cyclothymic personalities, and still other groups. The dysthymic and cyclothymic patients may be viewed as borderline with respect to manic depression in the same way most schizotypals could be viewed as within the penumbra of classic schizophrenia. Clearly, there are no common biological roots governing this heterogeneous domain. A proportion (varying in percentage with the sample) of BPD patients manifest serious affective disorders, often of the dysthymic or of the bipolar-II type. The question arises in those cases whether one is dealing with comorbidity (two conditions coincidentally occurring together), primacy of the affective disorder (where the BPD would be a latter-day secondary personality deformation), or conjoint etiology (both springing from a common biological source).

Leaving this thorny issue aside for the moment, suffice it to say that until now psychoanalytic theories of the borderline have remained either purely or predominantly psychological, if for no other reason than that many of their spokesmen were much more acquainted with information stemming from psychological/psychodynamic sources than from neurophysiologic sources (viz., Masterson, 1982; Kohut, 1971, 1977; Muslin & Val, 1987). Until recently, this distantiation—of mind from the black box within which it is contained—was even more pronounced than in Freud himself—who was said to have abandoned biology once he put down his pen from his 1895 Project, but who, in fact, remained more congenial to the notion of constitutional/organic underpinnings than were many of his students and successors.

THE CURRENT DEFINITION OF BORDERLINE PERSONALITY DISORDER

One outgrowth of the decades' long controversy about borderline conditions has been the creation of a more objectifiable and homogeneous definition. Actually we now have two partly overlapping, partly disjunctive definitions: namely, that of Gunderson and the one sanctioned by DSM-III-R. Both are polythetic, in that not every diagnostic item need be fulfilled before the diagnosis can be established. The Gunderson definition is tighter in that four out of five inclusion criteria must be satisfied (1. work impairment, 2. impulsivity—especially manipulative suicide gestures, 3. brief psychotic episodes, 4. good socialization, but 5. severe disturbances in intimate relationships). Any two Gunderson borderlines must have at least three items in common (viz., #1,2,3,4; 2,3,4,5). The DSM-III-R sys-

tem, requiring only five of eight items, allows 93 combinations; any two cases need only overlap on two items (viz., #1,2,3,4 & 5 vs. #4,5,6,7 & 8). A borderline patient with identity disturbance, labile affect, impulsivity, rage and self-damaging acts might have little in common (etiologically, dynamically, or prognostically) with another exhibiting emptiness, rejection sensitivity, disturbed relationships, identity disturbance, and labile affect.

The newer, official definition (of BPD) tends to capture within it a more affectively-tinged patient population than was the case with the earlier definitions. Akiskal (1981) and I (1981) have commented upon this semantic shift, in essence a manifestation of what might be called *biometric drift*, and have presented pedigree-data that would support the contention that a significant proportion of BPD cases could also be considered borderline with respect to manic-depressive disorder. The DSM-III-R definition, in fact, bears an eerie resemblance to the 19th century (and earlier) descriptions of the *sanguineocholeric* and *melancholicocholeric* temperaments (Griesinger, 1871). We would translate these as *hypomanic/irritable* or *depressive/irritable*, which imply the very same combination of *angry* and *moody* qualities that dominate the current conception of the borderline personality. *Plus ça change, plus c'est la meme chose!*

One may chafe at the notion that the past hundred years in this realm of psychiatry, instead of aligning themselves into a straight march of progress, have secretly curved back into quaint Galenic ideas about temperament and the Four Elements. What if we were to approach the problem a different way, seeking out the common attributes amongst all the popular definitions of borderline, present and past, in hopes of developing a picture of the quintessential borderline patient, about whose diagnosis all clinicians and investigators, whatever their age and educational background, could agree? If a uniform and nosologically more homogeneous picture emerged, perhaps this might point the way to common etiological factors—the hitherto concealed and elusive red thread of borderline personality. Several investigators have already turned their attention to this task.

Perry and Klerman (1978), for example, compared several systems including those of Grinker, Werble, and Drye (1968), Kernberg (1967), and Gunderson (1975). Grinker (1968) delineated four subtypes, of which the core borderline syndrome was characterized by vacillating involvement with others, anger acted out, depression, and inconsistent self-identity. Perry and Klerman (1979) noted that, of the many different traits and symptoms mentioned by the authors they surveyed, the one universal item was impulsivity, of which Grinker's *anger acted out* may be understood as an equivalent. Gunderson (1975) mentions this trait by name; Kernberg (1967) does also, though as one of his three *nonspecific features*. Other contributors also allude to this clinical feature, in describing syndromes quite similar to BPD, though using different terminology. Frosch (1977), in this connection, limned the features of *character impulse disorders*, to which he appended a synonymous term, *impulse-ridden character*. The latter derives from Wilhelm Reich's 1925 monograph of the same name (*der treibhafter Charakter*). Frosch (1977) distinguished between *symptom impulse disorders*, of which kleptomania, pyromania, exhibitionism, and temporal lobe episodic dyscontrol were some examples (p. 298) and the *impulse-ridden character*, where impulsivity permeates character structure as a pathognomonic feature and is not limited to any one type of impulsive act. Frosch expressed the belief that "real and traumatic occurrences... at crucial stages in

psychic development play a significant role in determining the jump from fantasy into action" (p.306). He felt Freud came to underestimate the importance of these factors (as when he cast doubt on the occurrence of actual incest, in contrast to mere fantasies, in hysterics). Frosch also gave considerable weight to constitutional factors, alongside environmental factors, as of causative significance, stressing *individual variations in the intensity of drives and needs* and possible differences in infant motility patterns (a point stressed by Fries and Woolf, 1953). Alluding to the biological amalgam of inheritance, intrauterine life, and perinatal factors, Frosch cautions that the innate strength of the drive(s) may not be as important as the rapidity of increase in their intensity, possibly because of a disturbance in the *built-in delay apparatus* (p.306). We shall have occasion to return to this point later in the discussion.

Though Frosch's impulse-ridden characters resemble certain types of patients now subsumed under the heading of BPD, he advocated the term *psychotic character* (1964, 1970) as a replacement for what was then being called *borderline*. Shortly after Frosch's 1977 paper, Liebowitz and Klein (1979) offered a new set of criteria for the hysteroid dysphoric patient, revised from Klein's original description (1969). The hysteroid dysphoric label has not gained widespread popularity; its syndromal validity has been questioned by Spitzer & Williams (1982). The clinical description, nevertheless, answers closely to that of the quintessential borderline patient. Liebowitz and Klein do not mention impulsivity in their outline, but several varieties of impulsive behavior (substance abuse, self destructive acts, chaotic sexuality) are written into their description. While males are by no means free of irascible or impulsive behavior, the overall picture of hysteroid dysphoria derives from the clinical encounter with certain impulsive female patients who happen to be particularly chaotic, moody, unreasonable, tempestuous, and irritable. This syndrome (Spitzer's caveat aside) represents an extreme of the histrionic patient, and resembles both the *hysteroid* patients described by Easser and Lesser (1965) and the *infantile personality* variant of borderline sketched by Kernberg (1967). In the psychobiographies of criminals, one occasionally comes upon an extreme version of the hysteroid dysphoric, where chaotic impulsivity, wildly oscillating extremes of love and hate, and extreme promiscuity constitute a kind of behavioral psychosis. The case of Cindy Ray Campbell (Olsen, 1987) who conspired to kill her parents, having first filled her boyfriend's ears with lurid tales of sexual molestation by her father (not proven in this case), is a striking example. Extreme cases of BPD (where all eight items are positive; where the frantic behavior is especially dramatic) often present a history of incest, marked impulsivity, and episodic dissociative phenomena.

IRRITABILITY: THE NEUROPHYSIOLOGICAL RED THREAD?

Psychoanalytic theoreticians have sought to explain borderline conditions as the outgrowth of certain unfavorable dynamic constellations (chiefly involving mother and child) occurring over stretches of time considered critical to the later development of borderline personality. Kernberg selected the Eriksonian concept (1956) of identity diffusion as an essential feature. Impulsivity, along with heightened vulnerability to stressors (*low anxiety tolerance*) were accorded nonspecific status. The current generation in psychiatry, however, is inclined to

lend great weight to biological factors, if and when they can be detected. In the light of this attitudinal shift, even *preoedipal* factors (such as problems in separation/individuation and what Balint (1969) described as the *basic fault*) seem like players who have entered late in the game. What now intrigues us are various *preoedipal* factors, relevant to the birth period, to intrauterine life or to the basic genome.

If certain biological—specifically, neurophysiological—abnormalities can be picked apart from the alphabet soup of causative factors that are truly characteristic of and etiologically significant for borderline personality, then some of the preoedipal and other dynamic factors hitherto relied on as explanatory may have to be placed somewhat to the side as epiphenomena or latter-day contributors to the psychobiological liability underlying BPD. A similar shift in our thinking has already taken place vis-a-vis schizophrenia. Bleulerian concepts held sway in the United States for 60 years; delusions and hallucinations were regarded as secondary attributes. But in the current rebiologization (if you can pardon the neologism) of psychiatry, what was secondary has become primary and vice versa. So it may be with impulsivity and identity diffusion.

Impulsivity, the red thread running through most definitions of the borderline case, may be understood as the behavioral manifestation of some underlying predisposition to action-oriented (as opposed to modulated, reason-oriented) responses. Borderlines *behave* impulsively; often they give evidence of inordinate irritability of the nervous system. This irritability appears in many instances as the neurophysiological precursor of impulsivity. In earlier papers (Stone 1977, 1980), I stressed the importance of predisposition to manic depressive disorder as the wellspring of the irritability in borderline patients, destined later to surface as tantrums in childhood; and as moodiness and impulsivity in subsequent life-stages. Here, I was postulating that *innate aggression* might be the expression of risk genes for manic depressive illness. In certain cases other innate factors, such as temporal lobe epilepsy, have etiological significance. Neurophysiological abnormalities have been invoked to explain cases of *episodic dyscontrol* (Andrulonis, 1981) in certain violence prone borderline patients. The irritability associated with the severer forms of premenstrual syndrome would also appear to stem from biological origins, whose nature remains obscure. Many borderline women exhibit this late luteal phase irritability in the form of impulsive/destructive behavior that may be quite uncharacteristic of them during other phases of the cycle (Stone, 1982). Irritability, and the impulsivity associated with it, is a common feature of bipolar I and II affective disorders. Its presence is often accompanied by the kind of reckless and provocative behavior that inspires in diagnosticians the notion of *BPD comorbidity*. Indeed, the *quintessential* borderline besides exhibiting the hysteroid dysphoric features mentioned earlier, frequently shows hypomania, after the manner of the bipolar II or cyclothymic persons which they either are or closely resemble.

The inordinate/inappropriate anger that figures as a diagnostic item for BPD may present itself as *borderline rage*—an unofficial designation for the momentary, extremely intense and irrational, often murderous anger demonstrated at various times by borderline patients. The comment is actually circular: patients often become defined as borderline in large part because of their propensity to this excess of rage. When the provocation seems partly justified (husband makes a contemptuous remark

about the way dinner was prepared) we speak of an overreaction. In many instances, the provocation exists only in the eyes of the borderline person and would be totally unpredictable to any reasonable person (husband presents wife with a dozen roses for their anniversary; unbeknownst to him the florist inadvertently included only eleven; wife notices and upbraids husband mercilessly as a cheap uncaring bastard who thought he could get away with saving two dollars...). These paranoid reactions so misconstrue expectable reality as to constitute brief psychotic episodes (as stressed by Gunderson and Singer) [1975]. The wife may hurl the flowers at her dumbfounded spouse, or else storm out of the house—in either event one has witnessed the transformation of irritability into impulsivity.

As an outgrowth of my recent work in environmental antecedents of BPD (Stone, Stone & Hurt, 1987; Stone, 1988a), I would now add certain early traumata to the list of factors capable of deranging neuroregulatory mechanisms in the manner we encounter in borderline patients. Among these, physical abuse and sexual molestation figure most prominently. Their impact will be affected by the timing, the chronicity, and the intensity of the abuse. Multiple sources of abuse (viz., physical and sexual) may be more pathogenic than single sources; sexual molestation by relatives (especially, older relatives), more pathogenic than nonincestuous molestation (Brown, et al., 1973; Cross & Hirschfeld, 1986; Grant et al., 1982; Paykel et al., 1975; and Browne & Finkelhor, 1987). The situation with abusive family environments is comparable to other traumatic environments (e.g., combat during wartime), that induce post-traumatic neuroses or other post-traumatic syndromes resembling the endogenous psychoses. Kolb (1987) has recently written about these syndromes, commenting upon the reverberating central nervous system circuits that trauma appears to set in motion. It is this chronic hyperexcitability or irritability that leads to the behavioral over-reactivity, nightmares, etc. by which we recognize the post-traumatic disorder.

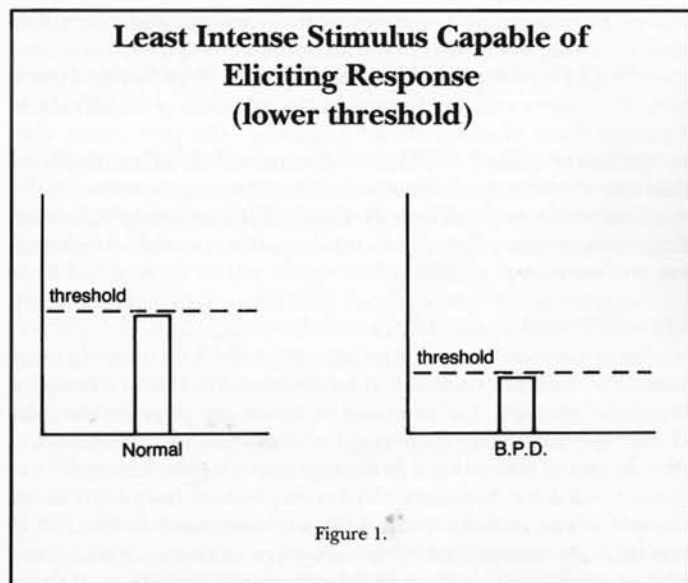
We need to ask at this point whether we have the means available to pass beyond clinical description and speculative formulations toward a neurophysiological model that might give us a better grasp, pictorially at least, if not mathematically, of the phenomena peculiar to borderline and related disorders of the sort outlined above. In the next section, I offer several diagrams and comments that may help achieve this better conceptual grasp.

TOWARD A NEUROPHYSIOLOGICAL MODEL OF BORDERLINE HYPERIRRITABILITY

The following comments and their pictorial accompaniments necessarily represent gross oversimplifications of how the brain may malfunction in the disorders whose essence I strive to understand. With its 15 billion neurones, countless interconnections and upwards of 200 neurotransmitters, the brain is a machine of surpassing complexity. Nonetheless, like the rudimentary devices we fashion and regulate, the brain receives its input (stimuli from the external world, from the body that houses it, and from itself), does some work with this input and produces an output, consisting of signals that are transformed into action, thought, and emotion. This output, and presumably the machinery behind it, is disordered in borderline patients in a number of characteristic ways.

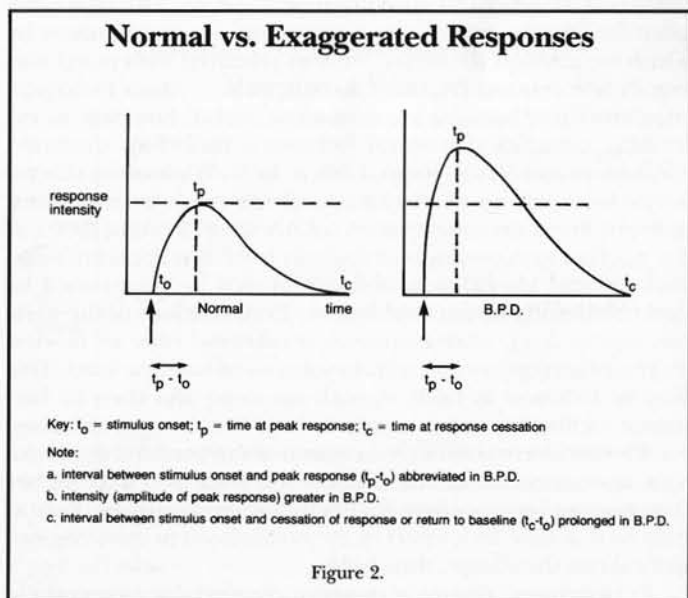
LOWERED THRESHOLD

Borderline persons often react, especially in situations of interpersonal stress, to stimuli that would be too weak to elicit a response from normal persons. One could illustrate this phenomenon as a function of threshold differences, as in Figure 1.



EXAGGERATED RESPONSE

Whether in reaction to stimuli ordinarily capable of eliciting a response or to weak stimuli, the response in borderline persons may be excessively rapid or excessively intense or both. Under certain circumstances the response, once switched on, may remain on longer than would be in keeping with optimal adaptation; i.e., the duration of response may be unduly prolonged. This difference is portrayed schematically in Figure 2.



The heightened amplitude of response may be the expression of augmentation—the designation for supranormal response in nerve units(s) following photic, auditory, or other stimuli. Augmentation in evoked potentials is characteristic of bipolar patients (cf. Shagass et al., 1977). Faulty functioning of a neural inhibitory mechanism might also lead to overshooting, too sudden firing, or inadequate damping of nerve impulse.

Similar enhancement of neural response has been demonstrated in relation to a number of monoamine and other substances acting upon synaptic transmission. Augmentation and speeding up of the peak response, followed by prolonged duration of response can be effected by the addition of dopamine to a preparation of sympathetic ganglion cells pretreated with acetylcholine (Libet, 1984). In the same article, Libet mentions that the monoamines appear to have important roles in the motivational aspects of behavior and that norepinephrine and dopamine systems figure in cerebral pathways mediating learning and memory (p.427).

CHAOTIC OSCILLATIONS

An important facet of normal responsivity in a nervous system is the ability to contain a noxious stimulus within a range of response-intensity that does not threaten the physical integrity of the system. Body proteins, for example, are destroyed at temperatures beyond the boiling point of water, hence a (heat-) stimulus becomes dangerous where heat cannot be dissipated fast enough to maintain temperature below 212 F. Presumably, an optimal nervous system would operate with a built-in danger-zone, such that heat stimuli well short of those that could kill tissue already, lead to avoidant or else to more effective heat-dissipating activity. An ideal ("normal") nervous system would operate smoothly at a baseline activity level well below the danger threshold, such that ordinary stimuli would not push the response-system into the danger-zone. Analogously, additional calories sufficient to raise temperature 2 degrees will do very little to water at 150 but will convert water at 211 into steam.

Physiological control was, as Rapp points out (1986), formerly viewed as subserving the function of restoring transiently disturbed systems to an ideal homeostatic state. This view is implicit in Freud's hypotheses concerning the mechanisms by which we attempt to reduce tension (equated with pain) and regain homeostasis (equated with pleasure). Many biological regulatory mechanisms are now understood, however, as exhibiting complex dynamical behaviors, including sustained oscillations and chaos (Rapp, 1986, p. 179). While at the macroscopic level persons may behave, under normal circumstances, as nearly linear dynamic systems (cf. Abraham & Shaw, 1980), at the microscopic (neurophysiological) level, this apparent near-linearity and smoothness of function may be maintained by systems showing distinct oscillation. Perturbations of the nervous system lead, under optimal conditions, only to modest increases in response intensity beyond some baseline level. This may be followed by fairly smooth damping and then by harmonic oscillations within a narrow (i.e., *gentle*) range of values.

The hyperirritable nervous system of the borderline, in contrast, operates as though the baseline level of activity were higher than normal (analogous to higher initial temperature). Perturbations that have little effect in the normal system drive responsivity above the danger threshold.

Furthermore, damping down to comfortable homeostatic

levels either is slow or tends not to occur at all, unless the system is driven, via additional stimulus, near to a point of destruction. Once this level is reached, the responsivity quickly descends to the baseline level.

A related characteristic of the *overheated* system is its tendency to oscillate widely and wildly (rather than narrowly and harmonically). At successive time intervals in the operation of the dynamic system, intensity values at each next interval tend to be markedly different from the preceding value. The fluctuations become nonperiodic: each new value is (and remains infinitely) different from all preceding values. Nonlinear oscillations of this sort remain, nevertheless, within certain upper and lower bounds and within definable ranges. Such systems are said to exhibit chaos (Gleick, 1987), as opposed to randomness (where, ultimately, all possible values would eventually be visited by the system). This phenomenon is discernible in the weather, as Lorenz (1963) demonstrated: temperature over time behaves nonlinearly and nonperiodically and therefore remains unpredictable except over very short time-spans.

By way of illustrating these different types of systemic behavior, one may have recourse to certain equations that have applicability to related phenomena (Gleick, 1987). Population growths in ecological systems, for example, behave as though next year's population (of deer, shark, etc.) is dependent upon this year's population (= "x"), upon the growth rate, "r" (comparable to the amount of heating or some other nonlinear quantity) and to another factor, "1-x," that tends to keep the system within bounds (as x increases, [1-x] decreases). The resulting equation, $X_{n+1} = rx(1-x)$, suggests that increasing the growth parameter r would eventually yield a higher stable population; decreasing r, a lower population. Interestingly, for values of r within a certain range, successive values of x_{n+1} ("next year's population") oscillate narrowly about a presumed equilibrium value. At increased values of r, it becomes clear, however, that there is no equilibrium; that the values of x_{n+1} never converge to a single value. Either they begin to oscillate widely (with varying period-lengths) or, at a still higher value of the r-parameter, they exhibit chaotic fluctuation, never visiting the same value twice, no matter how many iterations of the equation. Figure 3 shows the resulting trajectories for a variety of r-values. The abscissa is the "time" axis (here: succeeding iterations of the equation); the y-axis shows successive values of x_{n+1} ("next year's" population). I use the values chosen by Gleick: initial $x=0.02$; $1-x=0.98$. With growth-parameter $r=2.9$, x rapidly increases (after 4 "years") to a value between 0.6 and 0.7. After 25 iterations, the trajectory settles down to a gentle harmonic oscillation between 0.657 and 0.653, though never touching the "average" value of 0.655.

Modest increments in the parameter (viz., $r=3.1$; 3.3 ; 3.5) produce much wider, though still periodic, oscillations that never settle anywhere near their hypothetical average value. Viewed another way, the line representing the successive x-values suddenly appears to bifurcate into low and high values (which appear in alternate cycles). Further small increments produce still wilder oscillations that not only never touch an apparent average but appear chaotic through many iterations. With $r=3.75$, for example, x bounces about, sometimes seeming to show a periodicity of 5, then becoming chaotic, before finally settling down to a period-5, oscillation through values 0.85, 0.478, 0.936, 0.225, 0.653, 0.85....

It requires at this highly unstable state, only a tiny increment in the parameter (equivalent to a slight increase in heat) to

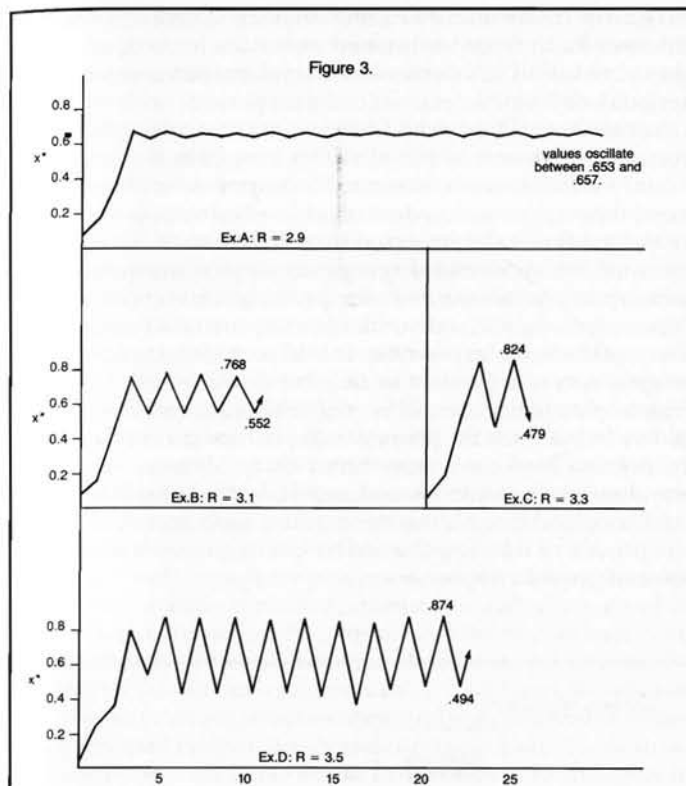
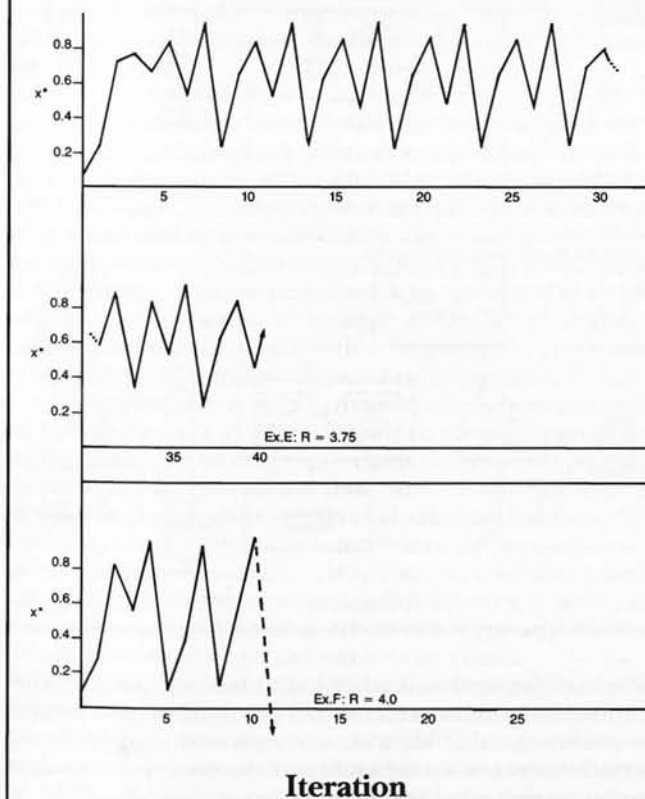


Figure 3. cont'd



drive the system into chaos. At $r=4.0$, the oscillations become so wide as to surpass the maximum x -value (1.0) at the 27th iteration, whereafter the value drops to zero and the system collapses. If the system were an ecological one, the 28th year would see the extinction of the population. If the system were heated water, this iteration would correlate with boiling away. With respect to a nervous system, irritability would have reached a level incompatible with the survival of the system; one would witness its breakdown or destruction.

PROVOCATION AND CRASH: THE BORDERLINE TANTRUM

Shifting our focus from the microsystem of nerve or nerve-tract to the macrosystem of the individual, we note comparable behavior in the irritable/borderline patient: wide and *unpredictable* oscillations of mood, oscillations that are actually predictable only as to the range within which they vary, but not predictable as to their timing. As an example, the range might include just two regions: adoration and jealous hatred, the *high* and *low* points of a bifurcated response-system. But precisely when the mood of adoration will be in force, and when jealous hatred will take over is unpredictable. The unpredictability of mood shift appears to be an expression of the same phenomenon that underlies the unpredictability of climatic shifts; namely, sensitive dependence upon initial conditions (cf. Gleick, 1987, p.23). By this is meant the inherent instability of the system, such that tiny and in essence unpredictable perturbations set in motion huge shifts in the system's state. This effect is characteristic of borderline behavior. The response to the gift of eleven roses (in section 3) is exemplary.

In the tantrum or provocation-and-crash behavior, also characteristic of the borderline, the response to certain stimuli is not only exaggerated but seems incapable of dying out back to the baseline or relaxed level unless spiked still further to the peak or maximum tolerable level for the system. This situation is analogous to sex and other consummatory behaviors. Once stimulated to near-orgasm, for example, one tends to remain

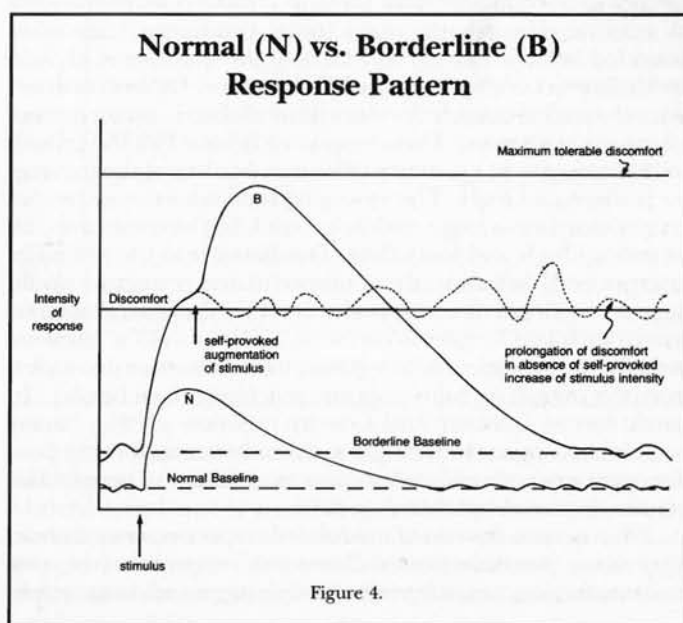


Figure 4.

in an uncomfortable state, unable to relax, until pushed further to the peak/orgastic level. Borderline persons, caught in situations that arouse in them moderately intense anger often provoke the person engendering this emotion until a state of maximal fury (*borderline rage*) is reached. There is usually an outbreak of impulsive behavior at this moment ("flash point"), in the form of hitting, hurling, screaming, etc., followed by rapid relaxation and abrupt recovery of more rational thinking. One may represent this phenomenon graphically, contrasting it with normal dynamics, as in Figure 4.

A FURTHER COMMENT ON SENSITIVE DEPENDENCE STIMULUS BOUNDEDNESS; DISTURBANCES OF MEMORY

We drew attention in the preceding section to sensitive dependence upon initial conditions. By initial is meant not the distal or original—but the immediately preceding conditions. The rage outburst after the flower gift follows immediately upon the chance discovery there were only eleven roses in the box. The factor determining the response here is not the long history of the relationship between the two participants but rather the last stimulus in what might be a many year's long sequence of stimuli. Let us assume for didactic purposes that in the above example ten years of living together had accumulated in the memories of the two participants, and that 93 percent of the discretely measurable interactions were favorable, when viewed by persons of conventional sensibilities. Only 7 percent of the time was the husband forgetful, neglectful, disappointing, hypercritical, etc. Given this history, a normal spouse would reflect momentarily upon this favorable history, average in the new stimulus into the large memory-bank of preceding experiences and conclude that her husband was (a) unaware of the oversight and (b) meant no ill in any case. Her response might be graphed as in Figure 4, the *normal* curve, to show the stimulus, the mild (surprise-) reaction and the rapid return to a relaxed state.

The borderline spouse, in contrast, behaves in a manner that Allan Frances, in a personal communication, characterized as *stimulus-bound*. This term is the appropriate psychological equivalent to the physicist's/meteorologist's concept of sensitive dependence upon initial conditions. Stimulus-bounded behavior would also take on the qualities of chaotic and unpredictable variation as defined above. Other characteristics of stimulus-boundedness are unmodulated, instantaneous, *all-or-none* responses. These responses behave like the growth (or heated-system) equations of section 4-C, where the parameter has been set high. The emerging state-values cease to converge toward or to hug a middle-ground, but bifurcate into (alternating) high and low values. Translated into the system of interpersonal behavior, these unmodulated responses would appear to reflect a disconnectedness within the central nervous system such that long-term memory, mechanisms for assessing statistical probabilities, and integrating mechanisms are decoupled from the overall stimulus-response machinery (see below). In particular, as Gardner and Cowdry mention (1985), "many borderlines appear to have great difficulty remembering positive experiences or maintaining a positive image of people who genuinely care about them" (p.390).

What occurs, instead of modulated responses, are extreme, all-or-none, survival-oriented "knee-jerk" responses (viz., passionate clinging, striking, verbally vilifying, murdering...); i.e.,

positive or (more often) negative violence. These reactions are the ones Rado (1956) subsumed under the heading of "emergency" reactions, encountered in very disturbed persons under ordinary circumstances or in ordinary persons under extreme circumstances. The model I have been constructing here is of course reminiscent of Freud's 1895 Project in the sense that Freud's psi neurones constituted a memory system and at the same time a feedback system capable of inhibiting the (all-or-none) tendencies of the unmodulated neurones. The set of all these inhibitory/modulating neurones approximates to, though is not quite coextensive with, the psychoanalytic concept of *ego*. Neurophysiological and nonlinear mathematical research over the past few decades permit us to add such elements to Freud's original system as (in the case of borderline/irritable behavior) chaotic fluctuation, response augmentation, etc. Figure 5 depicts schematically the propositions put forward in this section. In portion B of the Figure Short-Term Memory is (at least transitorily) decoupled from Long-Term Memory; Probability-Analyzing and Integrating mechanisms no longer dominate in the process of selecting the final response from the total repertoire of possible responses.

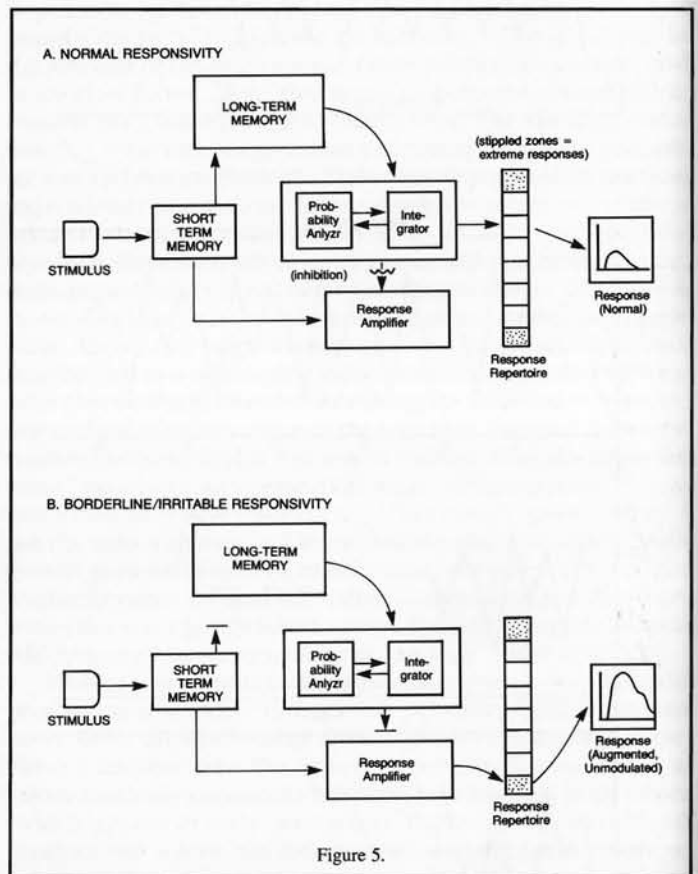


Figure 5.

The *amplifier*, pertinent to borderline behavior, may become operative, as alluded to earlier, either as a result of hereditary or constitutional influences or of early-environmental traumatic influences (or a combination of the two), in the form of altered activation states that tend to persist throughout life.

Figure 5 also presents, in abstract form, a schema which has its counterpart in what is becoming increasingly understood as the neuroanatomy of memory (Iverson, 1983; Mishkin et al., 1984; Kesner, 1984; Murray & Mishkin, 1986; Zola-Morgan, Squire, & Mishkin, 1982; Squire & Butters, 1984). Other contributions to the schema stem from the neuromolecular investigations of Kandel et al. (1987) and Hoffman (1987).

The *long-term memory* component may be analogized to areas of the neocortex, including the frontal lobes, among whose functions are the monitoring and organization of input via thought and planning (which exert an inhibiting/modulating effect upon various response-tendencies). The *short-term memory* component may correspond to portions of the limbic system that subserve (a) the translation of tactile- into visual impressions (the amygdala) (Mishkin, 1988) and (b) visual recognition (the hippocampus). Recognition-memory enhanced by cholinergic agents, relates to a cognitive and potentially conscious memory-system. The long-term retention of remembered experiences can apparently be enhanced by epinephrine (viz., during states of emotional arousal: [McGaugh et al., 1984]). Simultaneously, stimuli are processed, and their enregistrements stored, in a behavioral memory-system (also called *procedural- or habit memory* [Kesner, 1984]) involved in the development and reinforcement of habits, even (as Mishkin points out) in the absence of recallable representations. The latter, non-cognitive, memory is not dependent upon limbic structures but rather upon the basal ganglia, the caudate and putamen. These structures have recently been implicated in obsessive thoughts in obsessive-compulsive patients, studied via positron emission tomography (PET scan) by several groups of investigators (Schmeck, 1988).

Both the hippocampus and amygdala are important in the process by which objects gain associative meaning, helping to bring to mind (conscious recall) something or someone not currently present.

The *Analyzer-Integrator* component may involve the neocortex (including the frontal lobes) in communication with the limbic and basal ganglia structures, permitting on-the-spot comparison of new stimuli with impressions already stored in long-term memory. This process mediates decisions as to whether what is now before one is desirable, safe or dangerous. Existing memory, as Kesner (1984) hypothesizes may normally be in an inactive state. Critical neural regions can then be rendered active in the presence of an appropriate set of attributes—external or internal stimuli—that heighten the probability of enduring changes in the organization of memory.

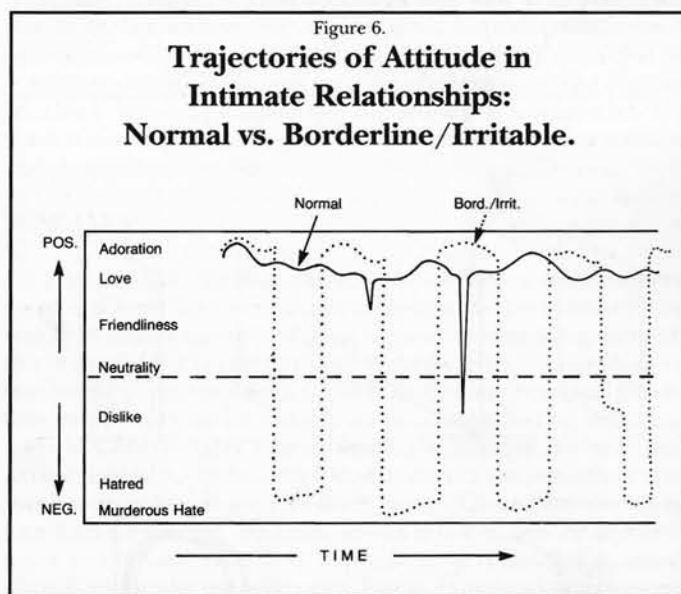
These processes may be partly conscious, or at times wholly automatic—especially in situations that are perceived as threatening welfare or survival (and which provoke an epinephrine response). Programs in the noncognitive, basal ganglia memory system permit speedy, reflex-like survival decisions. Severe traumata in early life can apparently inscribe an overactive and overly inclusive “program” (based on an *overlearned memory*), bringing it about that new stimuli that are even faintly reminiscent of the original noxious events can trigger the same catastrophic response engendered by those events.

Hoffman (1987) refers to these abnormalities in the memory system as *parasitic foci*. According to Hoffman’s hypothesis, these foci foster false assumptions, and then trigger stereotyped maladaptive responses, based on a partial or even meaningless *similarity*. The individual hurriedly concludes there is an equiva-

lence between some new, non-threatening stimulus and certain old memories of truly dangerous situations. Examples might be a woman, victimized incestuously as a child, who now cowers in the presence of a trustworthy and harmless man; or a man, beaten repeatedly as a child, who breaks out in a sweat, without knowing why, upon seeing a jar of whipped cream. The *Response Amplifier* component might include such structures as the thalamus, hypothalamus and corpus striatum. The latter, via dopaminergic transmission, facilitates integration of information from cortical and limbic sites (Iverson, 1983). The hypothalamus is involved in various emotional response-patterns; viz., rage (which may be mobilized by stimulation of the lateral portions) or aggressivity/excitability (which may be provoked by lesions of the medial hypothalamus) (Kupferman, 1985). Similarly, the posterior hypothalamus seems necessary to the phenomenon of *sham rage* as described in decorticate animals (Buck, 1976). The limbic structures also appear to be involved in emotional tone, as suggested by Kluver and Bucy (1937)—who noted that removal of the amygdala, anterior hippocampus and temporal neocortex rendered aggressive animals placid.

WILD OSCILLATIONS IN ATTITUDE

What is striking about borderline/irritable persons is not only their tendency to swing from one attitudinal extreme to the other at unpredictable intervals in the course of close relationships, but to hug the extremes for such a large proportion of time as to spend almost none in the middle ground, much as might a sailboat that lacked a keel. The history of the relationship may be represented by a trajectory in a state-space, as in Figure 6.



In the diagram the attitude of the borderline/irritable person is seen to oscillate over maximal amplitudes, spending only a brief interval in a nonextreme state. By way of comparison, the normal person’s attitude-history may be plotted as a nearly straight line (punctuated by a few brief descents, as during a *spat*, toward the negative pole). Here the attitude history has a

clear-cut average, whereas the borderline's attitude-history admits of no meaningful average. Typical of chaotic systems (such as stock-market crashes), not all intermediate values are visited as the attitude swings from high to low or *vice versa*; i.e., may drop precipitously from adoration to hate without stopping off at friendliness, tolerance, etc.

STORMY RELATIONSHIPS

In a previous communication (Stone, 1988), I commented upon the irresolvable conflictual states characteristic of the intimate relationships of borderline patients. Many (especially those who were traumatized physically or sexually as children) enter a love relationship craving intimacy almost immediately (partly as an antidote to intolerable loneliness), while barely suppressing an intense mistrust (especially pronounced in incest-victims). If one plots the evolving two-dimensional system over time, where both love and mistrust can vary from minimal to maximal values, a point in a plane may be used to represent the two values at any given moment. Usually the relationship progresses in such a way that the borderline person is conscious of minimal mistrust initially, with love increasing toward a maximum. As the latter position is approached, apprehension and mistrust (what if he rejects me?, what if he mistreats me?) suddenly escalate, often ushering in an angry scene, accusations, etc., whose net effect is to alienate the lover, creating a rapid swing toward minimal love. The relationship may be severed at this point. But such rupture brings about an intolerable state of loneliness again, predisposing the borderline person to (a) propose a reconciliation, amidst a (temporary) abrogation of consciously felt mistrust, and to (b) a rapid shift, once again, toward the *ideal* position of maximal love/minimal mistrust. Such a series of state spaces is portrayed in Figure 7, part A.

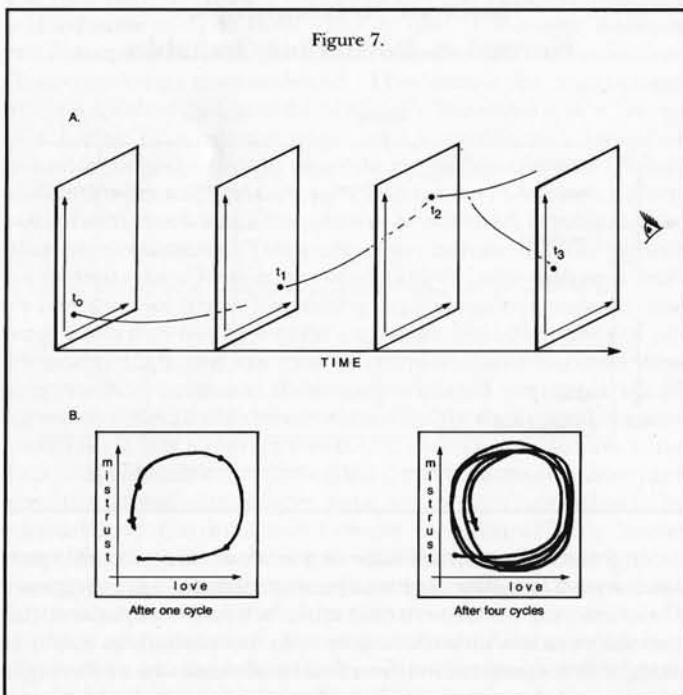
Were one to view these vertically aligned state-spaces from some distance, from the position of the eye in Figure 7, one would catch a glimpse of the system's trajectory. The trajectory is quasi-circular, as shown in Figure 7, Part B. Figure 7, Part B shows the history of the relationship after four such cycles. The system is nonlinear and only approximately periodic: each successive cycle resembles its predecessors, but does not touch precisely the same points. If the relationship were to last through many such cycles, the donut-shaped space in 7-C would mostly be filled in; no points would lie inside or outside this space (i.e., the system is chaotic but not random). This situation would be characteristic of a rapidly oscillating, *stormy* relationship. The basic trajectory would tend not to change without successful therapy, though the cycles would cease if the love partner declined reconciliation.

DISCUSSION

Many of the clinical features of borderline personality disorder, both in its fully developed adult form and in its childhood and adolescent antecedents, may be understood as the expressions of excessive CNS irritability.

This excess irritability might stem from a variety of factors, including decreased inhibition (*viz.*, failure of frontal lobe dopaminergic tracts to exert their customary modulating influence in response to certain stresses) or increased excitability (*viz.*, from activation-states set abnormally high within memory-modules oversensitized by various traumatic experiences in the past).

Affective disorder is common in BPD, but affective disorders without concomitant irritability are customarily accorded only an "affective" label (major depressive disorder, unipolar depression, atypical depression...). Once melded with rage, manipulative suicide gestures, or other manifestations of irritability/hostility, the diagnosis shifts toward BPD (or BPD with affective comorbidity). During the childhood of future borderlines restlessness, tantrums, irascibility, impatience and demandingness are prominent traits. These qualities either persist throughout adolescence or take on the attributes of moodiness, poor self-discipline, a tendency to create *scenes* and impulsivity in such forms as substance abuse, promiscuity, truancy, petty thievery, or even more violent antisocial behavior. Self injury and suicide gestures often have their onset during the adolescence of borderline persons. As the syndrome crystallizes in its adult form, chaotic oscillations in mood and attitude become apparent—especially in close relationships. Serious identity disturbance becomes apparent, accompanied by *splitting* and a strikingly unintegrated view of the self. The latter may stem from several sources. Persons with quasiperiodic shifts between calmness and extreme irascibility (as in those with concomitant late luteal-phase irritability), behave literally as two different people. Hyperirritability may also interfere so grossly with application to studies, hobbies, and vocational pursuits as to make mastery of any one task or interest impossible. One may see extreme *flightiness*, or desultoriness where a hobby or other interest is taken up with enthusiasm one week and dropped the next. Delay or permanent disruption of identity-formation in the vocational area is a frequent result. Identity disturbance may also arise from sources unrelated to irritability. Akiskal's example (1981) of the albino young woman born to black parents is one of many such instances. The point here, however,



is that identity disturbance in BPD is frequently an epiphenomenon of the underlying irritability. When this is so, the latter deserves pride of place in the array of causative factors.

As a kind of extreme case of identity disturbance, the syndrome of multiple personality disorder often overlaps phenomenologically with BPD. Kluft (1985), Bliss (1986), have shown that sexual or physical abuse is a near universal antecedent in multiple personality disorder. Usually the abuse had been severe and chronic; often the perpetrator had been a parent. The extreme dependence of a young child upon its parents fosters a love and a loyalty that are unextinguishable even in the face of outrageous mistreatment. Incestuous or harsh corporal abuse produce a hatred that may exceed the child's powers of forming an integrated picture either of the offending parent or of himself. Chaotic and wide oscillations of attitude may ensue (analogous to the equations relating to chaotic oscillations with the parameter set high, discussed earlier), the extremes becoming encapsulated, via dissociative phenomena, into alternating or multiple personalities. The patient with multiple personality disorder appears vulnerable to an abrupt functional tuning-out of entire blocks of memory, under the impact of specific emotion-laden events. Many borderline and multiple personality disorder patients also show state-dependent memory, in the areas of past traumata: material encountered in a particular context is contextually bound (cf. McClelland & Rumelhart, 1986, p.206) and can be retrieved later only during events that closely mirror the original context. Example: a borderline incest-victim suppressed the memory of her father's molestation (anally, when she was six) except at such times when her husband attempted this form of contact, which then elicited terror and flight.

Braun and Sachs (1985) also allude to state-dependent memory in connection with multiple personality disorder. These authors postulate the development, in this syndrome, of alternate memory systems containing information the main system was "...unwilling or unable to integrate" (p.49). In their example, the walled-off information centered around anger toward authority figures; the alternate personality (during whose dominance the anger is permitted to surface) may then "pop up whenever the host personality feels he... is pushed around..." (p.49) by some controlling figure.

Traumata, as we have noted, can lead to permanent changes in the activation states within the memory modules that encode them. Kandel and his colleagues (1987) have begun to elucidate the mechanisms by which trauma might activate (within the neurone ensembles that compose these modules) previously nonexpressed genes (via activation of the 5-hydroxytryptophan/cyclic AMP system) such that an environmental condition can create lasting neurophysiological alterations (including the synthesis of different proteins) similar to those found in inherited mental disorders. What remains unclear is why the trauma-induced hyperirritability manifests itself in one person as post-traumatic stress disorder (without personality "alternates" or impulsivity), in another, as BPD (with impulsivity but no *alternates*), and in a third, as multiple personality disorder. I do not think this variation can be wholly explained on environmental/psychodynamic grounds. The traumata may be similar across cases of all three. Certain brains are probably "wired" at birth in such a way as to predispose, given subsequent trauma, to one or another of these clinical syndromes. Dissociative states less extreme than multiple personality disorder occur with some

regularity in BPD in the form of depersonalization or derealization. These often follow in the wake of severe stress; viz., romantic rejection, quarrel with a parent, etc. These states are comparable, in the model I have presented here, to the situation in the section on Provocation and Crash: a hyperirritable nervous system is pushed toward a state of near-maximal discomfort, where relaxation is no longer possible unless the irritable state is pushed further to the extreme. In borderline persons suffering dissociative experiences of this sort their solution is often a violent act: suicide gesture, self-mutilation, smashing property, hurting someone (cf. Gardner & Cowdry, p. 397). These acts constitute the orgasmic tremor that ushers in the longed-for reduction of irritability. Within the context of a tempestuous love relationship it should be added, a self-injurious act committed in the presence of a temporarily irate partner will routinely have the effect of a circuit switch: the partner's fury gives way at once to loving/protective feelings. This restoration of positive attitude may itself bring about a sudden reduction of irritability. But in other stressful situations, borderline persons may induce this *switch* reaction by violent means, even in the absence of other people.

Borderline as well as bipolar disorders are both often associated with *sensation seeking*. Prolonged states of low stimulation lead to an intolerable boredom, for which their antidote is to jolt the nervous system with an intense, exciting experience. The latter may have either valence (a vicious argument, passionate lovemaking, reckless driving, seeing a horror-show...). Possibly this sensation seeking represents a less extreme example of the provocation-and-crash, orgasmic-like phenomena such persons induce under conditions of still greater irritability. Sensation seeking may also take the forms of abusing substances, courting danger via shoplifting, fare-beating, picking fights in bars and a host of other impulsive acts. That the impulsive BPD/borderline may be the most important subtype is substantiated by Hurt (1988), who has shown that, of the 93 combinations of DSM items capable of establishing the diagnosis, those involving impulsivity are the most numerous. His study is based on a pooling of 465 BPD patients from several in- and outpatient samples.

SUMMARY

The psychobiological model of borderline development proposed here places emphasis on heightened central nervous system irritability as the key factor, the *red thread* running through the bulk of what is currently subsumed under the heading of borderline personality disorder (BPD). Pre- and postnatal factors may individually or in concert set in motion lasting deformations of CNS circuitry that substantially increase the reactivity (irritability) of the system, such that it remains perpetually nearer some neurophysiological boiling point. These factors constitute a chaos engine, bringing about quicker, less modulated, more wildly oscillating and unpredictable responses to many stimuli, especially noxious ones. Figure 8 highlights in systems terms these interrelationships aligned on their corresponding levels: the neurophysiological (hyperirritability), the symptomatic (primary BPD traits) and the interpersonal (stormy relationships). Certain Dissociative phenomena, strictly (multiple personality, fugue states) and loosely (splitting, identity disturbance) defined, occupy an intermediate position in the hierarchy of systems, since they reflect a commingling of cogni-

tive and affective abnormalities often have their origin in sharply polarized attitudes toward caretakers and significant others, provoked sometimes by real abuse, sometimes by pre-existing CNS hyperirritability in the absence of environmental abuse. Either way one is left with split and unintegratable images of self and other.

Stormy relationships appear as the interpersonal consequence of the foregoing abnormalities. In place of stability and reasoned responses, one sees sensitive dependence upon the interpersonal events of the immediate past, and wild oscillations in attitude.

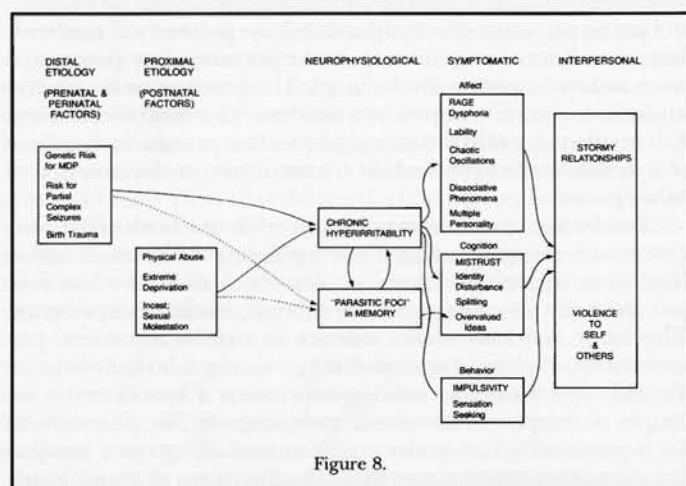


Figure 8.

THERAPEUTIC IMPLICATIONS

The psychobiological model proposed here highlights a hyperirritability whose origins either antedate parent-child interaction or else stand to one side of the traditional developmental stages. The traumatic effects of abuse, that is, alter the neuroregulatory response system in ways that cannot be accounted for by maladaptive patterns in the separation/individuation or any other stage of early development. The irritability of borderline patients tends to resist the ameliorative effects of otherwise correct interpretations concerning early maladaptive interactional patterns.

We may view this neurophysiologically as an example of (a) the *noncognitive* memory system, activated long ago by traumatic experience, overwhelming the modulating capability of the neocortex, or, in the former instance, (b) the noncognitive memory system gaining ascendancy, even in the absence of trauma, because of constitutional/genetic defects in those parts of the cortex (esp. the frontal lobes) that normally enregister emotional memories and modulate response to recent stimuli. Frosch alluded to mechanisms that might "overflow the cortical control apparatus" in an earlier paper (1977, p. 306).

Reduction of irritability will often take precedence over other therapeutic goals, especially at the outset, in one's work with borderline patients.

The more intense the irritability and the more dramatic its symptomatic expression, the more appropriate will be the use

of medications such as carbamazepine or lithium. This is especially true in instances of repetitive self-injurious acts or of paramenstrual intensification of irritability.

In other instances, cognitive- or behavior-modification approaches will be necessary, as mechanisms helping the patient gain mastery over psychodynamically obvious but nevertheless still-uncontrollable impulses. Overeaters Anonymous, Alcoholics Anonymous and similar organizations have proven their value as better first lines of defense (in bulimia, alcoholism, etc.) than interpretive technics alone.

Psychotherapy, much of which will have to be of a highly supportive, eclectic and flexible sort, will usually be necessary for long periods of time. A steady long-term working alliance with the same therapist has in itself a soothing quality—a point made earlier by Giovacchini (1982)—which helps reduce the irritability of borderline patients. This will be true particularly for patients who have experienced severe parental deprivation. Most borderline patients need education about the average expectable interpersonal environment, since they have usually been programmed to deal with atypical, often abusive, behavior on the part of parents or other intimates.

Another aspect of this cognitive/supportive approach is problem-solving: helping the patient expand his repertoire of possible responses in the stressful interpersonal situations to which he is most vulnerable. This introduces modulation and measured reaction where explosive, catastrophic responses had held sway beforehand.

Further reduction of irritability may be achieved in selected cases via a shift toward a more exploratory therapy. Distortions in the patient's perception of others may be corrected in this fashion through focus on transference. Borderline patients exhibiting the temperaments (Stone, 1979) associated with manic-depressive illness are often irritable, impatient and demanding from birth, even when reared by fairly calm and reasonable parents. Because of their imperious drives, strong cravings and demanding natures, they experience even an adequately nurturing parent as depriving or withholding. Such patients develop a hypercritical attitude and see their irritability as completely justified, (cf. Spungen, 1983).

In general, borderline hyperirritability born (largely) of constitutional factors in the absence of poor or traumatic nurturing presents fewer therapeutic problems than similar clinical configurations engendered by abuse or severe neglect. In the former instance, once the irritable symptoms are brought under control, the patient will ordinarily be able to see the central figures of his life in more human and more realistic ways. Those who have been truly injured, however, retain next-to-ineradicable traces of the traumatic experiences in their *habit* (or *memory-without-record*, as described by Squire, 1987, p. 169) memory-system. These traces easily override the evidence of one's senses (about the innocuousness of someone in the here-and-now who just happens to resemble a harmful figure from the past). This distinction would help to account, I believe, for the relative ease with which the Oedipus Complex of a never-molested woman can be resolved by psychoanalysis and why the same interpretive interventions usually prove ineffective with the incest-victim, whether the latter manifest a primarily cognitive abnormality (e.g., pathological jealousy, dissociative states), a primarily impulsive disorder (e.g., BPD with chaotic sexuality and rage outbursts) or both.

REFERENCES

- Abraham, R.H. & Shaw, C.D. (1981). *Dynamics: The Geometry of Behavior, I: Periodic Behavior*. Santa Cruz, Calif: Aerial Press, Inc.
- Akiskal, H. (1981). Subaffective disorders: Dysthymic, cyclothymic and bipolar II disorders in the "borderline" realm. *Psychiatric Clinics of North America*, 4, 25-46.
- American Psychiatric Association. *Diagnostic and Statistical Manual, 3rd Edition*. Washington, DC: Author.
- American Psychiatric Association. *Diagnostic and Statistical Manual, 3rd Edition, Revised*. Washington, DC: Author.
- Andrulonis, P.A., Glueck, B.C., Stroebel, C.F., Vogel, N.C, Shapiro, A.L., & Aldridge, D.M. (1981). Organic brain dysfunction and the borderline syndrome. In M. Stone (Ed.), *Symposium on Borderline Disorders*. *Psychiatric Clinics of North America*, 4 (1), 47-66.
- Arlow, J., & Brenner, C. (1964). *Psychoanalytic Concepts and the Structural Theory*. New York: International Universities Press.
- Balint, M. (1969). *The Basic Fault*. New York: Brunner/Mazel.
- Bergeret, J. (1984). *La Violence Fondamentale*. Paris: Dunod.
- Bleuler, E. (1911). *Dementia Praecox, oder die Gruppe der Schizophrenien*. Leipzig: Deuticke.
- Bliss, E. (1986). *Multiple Personality, Allied Disorders and Hypnosis*. New York: Oxford University Press.
- Braun, B.G., & Sachs, R.G. (1985). The development of multiple personality disorder: Predisposing, precipitating & perpetuating factors. In R.P. Kluff (Ed.): *Childhood Antecedents of Multiple Personality*. Washington, DC: American Psychiatric Press, pp. 37-64.
- Broussais, F.J.V. (1828). *De L'Irritation et de la Folie*. Paris: Delaunay.
- Broussais, F.J.V. (1831). *On Irritation and Insanity*. Tr.: T. Cooper. Columbia, S.C.: S.J. Morris.
- Brown, G.W., Sklair, F., Harris, T.O., & Birley, J.L.T. (1973). Life events and psychiatric disorders. *Psychological Medicine*, 3, 74-87.
- Browne, A., & Finkelhor, D. (1986). Impact of child sexual abuse. *Psychological Bulletin*, 99, 66-77.
- Buck, R. (1976). *Human Motivation and Emotion*. New York: John Wiley & Sons.
- Bychowski, G. (1953). The problem of latent psychosis. *Journal of the American Psychoanalytic Association*, 4, 484-503.
- Cross, C.K., & Hirschfeld, R.M.A. (1986). Psychosocial factors and suicidal behavior: Life events, early loss and personality. In J. Mann, and M. Stanley (Eds.), *Psychobiology of Suicidal Behavior*. New York: New York Academy of Science Press, pp. 77-89.
- Deutsch, H. (1942). Some forms of emotional disturbances and their relationships to schizophrenia. *Psychoanalytic Quarterly*, 11, 301-321.
- Easser, R.R., & Lesser, S. (1965). Hysterical personality: A reevaluation. *Psychoanalytic Quarterly*, 34, 390-402.
- Erikson, E.H. (1956). The problem of ego identity. *Journal of American Psychoanalytic Association*, 4, 56-121.
- Freud, S. (1966). Project for a scientific psychology. In J. Strachey (Ed. and Trans.) *The Standard Edition of the complete psychological works of Sigmund Freud* (Vol. 1, pp 283-413). Longon: Hogarth Press. (Original work published in 1895).
- Fries, M. & Wolf, P.J. (1953). Some hypotheses on the role of congenital activity type in personality development. *Psychoanalytic Study of the Child*, 8, 48-62.
- Frosch, J. (1964). The psychotic character: Clinical psychiatric considerations. *Psychoanalytic Quarterly*, 38, 81-96.
- Frosch, J. (1970). Psychoanalytic considerations of the psychotic character. *Journal of the American Psychoanalytic Association*, 18, 24-50.
- Gardner, D.L., & Cowdry, R.W. (1985). Suicidal and parasuicidal behavior in borderline personality disorder. *Psychiatric Clinics of North America*, 8, 389-403.
- Giovacchini, P.L. (1982). *Technical Factors in the Treatment of the Severely Disturbed Patient*. New York: J. Aronson, Inc.
- Gleick, J. (1987). *Chaos: Making a New Science*. New York: Viking.
- Grant, I., Yager, J., Sweetwood, H.L. & Olshen, R. (1982). Life events and symptoms. *Archives of General Psychiatry*, 39, 598-605.
- Griesinger, W. (1871). *Die Pathologie und Therapie der psychischen Krankheiten*. Second Edition. Braunschweig: Verlag von Friederich Wreden.
- Grinker, R.R., Sr., Werble, B., & Drye, R.C. (1968). *The Borderline Syndrome*. New York: Basic Books.
- Gunderson, J.G., & Singer, M.T. (1975). Defining borderline patients: An overview. *American Journal of Psychiatry*, 132, 1-10.
- Hoffman, R. (1987). Computer simulations of neural information processing and the schizophrenia-mania dichotomy. *Archives of General Psychiatry*, 44, 178-188.
- Hurt, S. (1988). *Cluster Analysis of Borderline Subtypes*. Paper presented at the First International Congress of Personality Disorders, Copenhagen,

Denmark. August 4.

Iverson, D.S. (1983). Brain lesions and memory in animals: A reappraisal. In J.A. Deutsch (Ed.), *The Psychological Basis of Memory*. New York: Academic Press, pp. 139-198.

Jackson, D. (1960). *The Etiology of Schizophrenia*. New York: Basic Books, Inc.

Kandel, E.R., Castellucci, V.F., Goelet, P., & Schacher, S. (1987). Cell-biological interrelationships between short-term and long-term memory. In E.R. Kandel (Ed.), *Molecular Neurobiology and Psychiatry*. New York: Raven Press, pp. 111-132.

Kernberg, O.F. (1967). Borderline Personality Organization. *Journal of the American Psychoanalytic Association*, 15, 641-685.

Kesner, R. (1984). The neurobiology of memory: Implicit and explicit assumptions. In E. Lynch, J. McGaugh, & N.M. Weinberger (Eds.), *Neurobiology of Learning and Memory*. New York: The Guilford Press, pp. 111-134.

Kety, S.S., Rosenthal, D., Wender, P.H., & Shulsinger, F. (1968). Mental illness in the biological and adoptive families of adopted schizophrenics. In Rosenthal, & S.S. Kety (Eds.), *Transmission of Schizophrenia*. Oxford: Pergamon Press, pp. 345-362.

Kluft, R.P. (Ed.) (1985). *Childhood Antecedents of Multiple Personality*. Washington, DC: American Psychiatric Press, Inc.

Kluver, H. & Bucy, P.C. (1937). Psychic blindness and other symptoms following bilateral temporal lobectomy in Rhesus monkeys. *American Journal of Psychology*, 119, 352-353.

Knight, R.P. (1953). Borderline states. *Bulletin of the Menninger Clinic*, 17, 1-12.

Kohut, H. (1971). *Analysis of the Self*. New York: International Universities Press.

Kohut, H. (1977). *Restoration of the Self*. New York: International Universities Press.

Kolb, L.C. (1987). A neuropsychological hypothesis explaining posttraumatic stress disorder. *American Journal of Psychiatry*, 144, 989-995.

Kraepelin, E. (1909). *Psychiatrie*. Leipzig: Verlag von J.A. Barth.

Kupferman, I. (1985). Hypothalamus & limbic system: I: Peptidergic neurons, homeostasis, and emotional behavior, II: Motivation. In E.R. Kandel, & J.H. Schwartz (Eds.), *Principles of Neural Science, 2nd Edition*. New York: Elsevier, pp. 611-635.

Libet, B. (1984). Heterosynaptic interaction at a sympathetic neuron as a model for induction and storage of a postsynaptic memory trace. In G. Lynch, J.L. McGaugh, & N.M. Weinberger, (Eds.): *Neurobiology of Learning and Memory*. New York: Guilford Press, pp. 405-430.

Liebowitz, M.R., & Klein, D.F. (1979). Hysteroid dysphoria. *Psychiatric Clinics of North America*, 2, 555-575.

Lombroso, C. (1878). *L'uomo Delinquente*. Firenze: Fratelli, Bocca.

Lorenz, E.N. (1963). Deterministic nonperiodic flow. *Journal of Atmospheric Science*, 20, 130-141.

Masterson, J.F. (1981). *The Narcissistic & Borderline Disorders*. New York: Brunner/Mazel.

McGaugh, J., Liang, K.C., Bennett, C. & Sternberg, D.B. (1984). Adrenergic influences on memory storage: Interaction of peripheral and central systems. In G. Lynch, J. McGaugh, & N.M. Weinberger (Eds.), *Neurobiology of Learning and Memory*. New York: The Guilford Press, pp. 313-332.

McClelland, J.L. & Rumelhart, D.E. (1986). A distributed model of human learning and memory. In *Parallel Distributed Processing*. Vol. II. Cambridge, Mass.: The MIT Press, pp. 170-215.

Mishkin, M. (1988). *Two types of memory*. Unpublished manuscript.

Mishkin, M., Malamut, B., & Bachevalier, J. (1984). Memories and habits. Two neural systems. In G. Lynch, J. McGaugh, & N.M. Weinberger (Eds.), *Neurobiology of Learning & Memory*. New York: Guilford, pp. 65-77.

Moore, T.V. (1921). The parataxes: A study and analysis of certain borderline mental states. *Psychoanalytic Review*, 8, 252-283.

Murray, A., & Mishkin, M. (1986). Visual recognition in monkeys following rhinal cortical ablations combined with either amygdalotomy or hippocampectomy. *Journal of the Neurosciences*, 6, 1991-2003.

Muslin, H.L., & Val., E.R. (1987). *The Psychotherapy of the Self*. New York: Brunner/Mazel.

Oberndorf, C.F. (1930). The psychoanalysis of borderline cases. *New York State Journal of Medicine*, 30, 648-651.

Olsen, J. (1987) *Cold Kill*. New York: Atheneum.

Paykel, E.S., Prusoff, B.A., & Myers, J.K. (1975). Suicide attempts and recent life events: A controlled comparison. *Archives of General Psychiatry*, 32, 327-333.

Perry, J.C., & Klerman, G.L. (1978). The borderline patient. *Archives of General Psychiatry*, 35, 141-150.

Prichard, J.C. (1835). *A Treatise on Insanity*. London: Sherwood, Gilbert & Piper.

- Rado, S. (1956). *Psychoanalysis of Behavior: Collected Papers*. New York: Grune & Stratton.
- Rapp, P.E. (1986). Oscillations and chaos in cellular metabolism and psychological systems. In A.V. Holden (Ed.), *Chaos*. Princeton, N.J.: Princeton University Press, pp. 179-208.
- Reich, W. (1925). *Der triebhafte Charakter*. Leipzig: Psychoanalytischer Verlag.
- Rosen, J. (1947). The treatment of schizophrenic psychosis by direct analytic therapy. *Psychiatric Quarterly*, 21, 3-37.
- Rosenfeld, H.A. (1965). *Psychotic States: Psychoanalytic Approach*. New York: International Universities Press.
- Rosse, I.C. (1980). Clinical evidences of borderline insanity. *Journal of Nervous and Mental Disease*, 17, 669-683.
- Schmeck, H.M., Jr. (1988). Region in brain is linked to obsessive disorder. *New York Times*, March 8, C-1.
- Shagass, C., Roemer, R.A., Straumanis, J.J. & Amadeo, M. (1977). Evoked potential correlates of psychoses. Presented at the 32nd Annual Meeting of the Society for Biological Psychiatry, Toronto.
- Spitzer, R.L. & Williams, J.B.W. (1982). Hysteroid dysphoria: An unsuccessful attempt to demonstrate syndromal validity. *American Journal of Psychiatry*, 139, 1286-1291.
- Spungen, D. (1983). *And I don't Want to Live this Life*. New York: Villard Books.
- Squire, L.R. (1987). *Memory & Brain*. New York: Oxford University Press.
- Squire, L. & Butters, N. (1984). *Neuropsychology of Memory*. New York: Guilford Press.
- Stern, A. (1938). Psychoanalytic investigation and therapy in the borderline group of neuroses. *Psychoanalytic Quarterly*, 7, 467-489.
- Stone, M.H. (1977). The borderline syndrome: Evolution of the term, genetic aspects and prognosis. *American Journal of Psychotherapy*, 31, 345-365.
- Stone, M.H. (1979). A psychoanalytic approach to abnormalities of temperament. *American Journal of Psychotherapy*, 33, 263-280.
- Stone, M.H. (1980). *The Borderline Syndrome*. New York: McGraw Hill.
- Stone, M.H. (1981). Borderline syndromes: A consideration of subtypes and an overview, directions for research. *Psychiatric Clinics of North America*, 4, 3-24.
- Stone, M.H. (1982). Borderline conditions and the menstrual cycle. In R. Freidman, (Ed.), *Behavior and the Menstrual Cycle*. New York: M. Dekker & Co., pp. 317-344.
- Stone, M.H. (1985). Borderline syndromes. In J. Cavenar, & R. Michels, (Eds.), *Psychiatry* Vol. 1, Ch. 17. Philadelphia: J.B. Lippincott, Co., pp. 1-15.
- Stone, M.H., Stone, D. & Hurt, S. (1987). Natural history of borderline patients treated by intensive hospitalization. *Psychiatric Clinics of North America*, 10, 185-206.
- Stone, M.H. (1988). The borderline domain: the "inner script" and other common psychodynamics. In J. Howells (Ed.): *Modern Perspectives in Psychiatry* (in press).
- Stone, M.H. (In press). Incest in Borderline Patients. American Psychiatric Association Press. In R. Kluff (Ed.), *Incest-Related Syndromes of Adult Psychopathology*. Washington, DC: American Psychiatric Association Press.
- Winslow, F. (1861). *On Obscure Diseases of the Brain and Disorders of the Mind*. 2nd Edition. London: John W. Davies.
- Zola-Morgan, S., Squire, L.R., & Mishkin, M. (1982). The neuroanatomy of amnesia: Amygdala-hippocampus vs. temporal stem, *Science*, 218, 1337-1339.