Discussion, Part IV, and Overview

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An analysis of the contents of this volume indicates that the most important concepts discussed in the interface between stress and mental disorder are: life events, vulnerability, crises, and episodes. While life events occupy the center of attention in this volume, it is to be hoped that in the not-too-distant future, a deeper understanding of vulnerability itself will have reached the point where it can serve as the focus of a symposium.

Regarding the chapters in the foregoing section, both Dr. Rose and Dr. Jenkins deal with the stressors provided by the hazardous occupation of air traffic controllers (ATC), an occupation most ingeniously suited to elicit stressful responses in its practitioners since threatening events are continuously in the offing. Dr. Rose deals with the physiological responses, while Dr. Jenkins deals mostly with the psychological and social. Although the data collected on this project are not yet fully analyzed, they already provide opportunities for analyzing the relationship between stressors and psychological dysfunction. These data may cast light on the perennial question of the specific pathway by which a psychologically threatening situation is transduced into a physiological response. It is easy enough to conceive how the injection of a suitable drug will bring about a physiological response, but how does an environmental threat get encoded into a bodily response?

Among the attempts to answer this question are the theoretical frameworks provided first by the James-Lange theory and later by Cannon for fear and anxiety in the “fight or flight” paradigm presumably mediated by the neuroendocrine system. Another attempt is the “conservation and withdrawal” hypothesis of Engle and Schmale (5) for depression mediated by lowered metabolism and increased parasympathetic activity. Selye’s hypothesis regarding the sensitivity of adrenocortical hormones to stressors in the environment has not met with universal acceptance (10,11). More recently, however, as Rose points out, other hormones, including growth hormone, prolactin, and testosterone, have been shown to be involved in the responses of the organism to stressors. Whether or not these can serve as general paradigms for the transformation of environmentally originating threats into physiological responses remains to be demonstrated. Perhaps the data on the ATC will provide new leads to solving this problem.
Another type of information which this study is eminently suited to provide is the selection of indicators for the beginnings of crises and their termination. By monitoring the physiological and psychological characteristics of the ATC in situations in which the effects of the stressors become evident, we may be able to discover which of the physiological and psychological changes can serve as markers for the beginnings and ends of crises, miniepisodes, or maxiepisodes.

The partial analysis of the data has thus far proved disappointing to the investigators. To their surprise, they found no relationship between rises in their three physiological indicators (blood pressure, cortisol, and growth hormone) measured during the apparently stressful on-the-job situations and increases in psychological and psychiatric problems. Although in the general literature, and also in this study, increases in the above-mentioned indicators are reported to be predictive of various physical illnesses, they do not seem to be predictive for the psychological area in the ATC. One wonders whether the stresses experienced by the ATC are sufficiently intense to warrant the expectation of a relationship, especially since no actual assessment of the stress has been reported. As Rose points out, the low cortisol levels observed and the absence of growth hormone indicators suggest that most controllers were not under acute stress when tested, at least not as acute as that caused by parachute jumping and upcoming examinations. Why the stress that the ATC undergo is sufficient to trigger physical but not mental distress and/or disorders is a most intriguing question. Is this merely an example of specificity, as Rose argues, or are there any protective mechanisms that operate in the case of mental distress and/or disorders which do not operate in physical disorders? It is to this question that Dr. Jenkins devotes himself; he tries to provide an answer by introducing the concept of moderator variables.

Dr. Jenkins points out that one of the reasons why, in previous investigations, the impact of life event stressors did not invariably lead to reports of discomfort and/or mental disorder was the simplicity of the two-variable design, which is usually employed. He proposes a much broader design along the lines of system-analysis theory and formulates a table in which the various levels of functioning, from the biological through the psychological, the interpersonal and the sociocultural, constitute the ordinate axis and the levels of response suggested by Selye constitute the abscissa axis, giving rise to a $4 \times 5$ table or 20 rubrics. Unfortunately, Selye's assumptions of the alarm reaction, general adaptation syndrome followed by the "exhaustion" syndrome, have never been generally accepted by biologists (10,11). Whether they will prove suitable for the levels above the biological remains to be seen. However, the test of a model is not whether it is "true" but whether or not it provides hypotheses for testing, which the model does provide. One hypothesis that Dr. Jenkins examines is whether the expected relationship between life event stressors and psychopathology can be modified by moderator variables. Thus he finds that educational mobility, anomie (as measured by the Srole scale), church attendance, and frequency of growth hormone responsiveness modify the expected relationship
between life event stressors (as measured by Paykel et al.) and psychopathology, as measured by the Psychiatric Status Schedule (PSS). The educationally less mobile (those who do not excel their fathers) show less psychopathology (impulse dyscontrol) in response to similar life event stressors than do the educationally mobile (those who do excel their fathers). This also holds true of the nonanomic, steady churchgoers, and those who are low in growth hormone responsiveness (those who gave no measurable responses in their serum growth assay).¹

On the other hand, excelling one's father educationally, scoring high on the anomic scale, irregular or infrequent attendance at church, and high responses in growth hormones tend to make people more vulnerable to life event stressors. Furthermore, the intensity of the stress induced by the life event stressors does not relate to the amount of psychopathology in the vulnerable, whereas those who are not vulnerable to these factors show the expected relationship between intensity of stress and psychopathology. Apparently, the vulnerable are more sensitive and responsive to life event stressors, regardless of the amount of stress involved.

Surprisingly, the degree of utilization of social coping resources (e.g., counseling) does not generally protect against life event stressors. However, the nonutilizers of social coping resources show the expected relationship between degree of life event stressors and psychopathology, while the utilizers do not. Here again the utilizers seem to be more responsive to life event stressors, regardless of the amount of stress induced.

The psychological dysfunction measures used in this study are based on the PSS, which obtains information on the five variables used in this study—sources on subjective stress, impulse control, alcohol abuse, work role, and mate role. Perhaps it would have been more directly informative if data on some of these variables could have been obtained more directly from the work history or daily record of the ATC.

It is interesting that one well-known mediating variable—the type of personality known as Type A (15)—was not considered by the researchers in the ATC studies. It seems a likely candidate for serving as a moderator variable.

If we adopt the vulnerability model for explaining why similar levels of stress do not produce similar dysfunction, we could conclude that mobility in educational level, anomic, and growth hormone responsiveness could be regarded as vulnerability markers for the development of psychological crises in response to life event stressors. Thus those who show upward mobility in education, high level of anomic, poor church attendance, and sensitive growth hormone responsiveness are at greater risk of showing psychopathological behavior, such as impulse control dysfunction, regardless of the level of stress induced by the impinging life event stressors. Whether or not they can also lead to episodes of disorder is not indicated in the reported data.

Dr. Wender's chapter raises several issues that bear directly on the interrela-

¹ The life event stress was based on self-ratings of distress due to life change.
tionships between stressors and mental disorder. The question he raises regarding the relative effects of continued stressful niches versus changes in level of stressors on the production of psychopathology has never been adequately answered, but his negative conclusions regarding the role of early rearing practices and socioeconomic status on mental disorders remain debatable. These issues are discussed in the next section.

Interaction Among Life Events, Vulnerability, and Episodes of Illness

The question behind most discussions of life events is: why doesn't the same event produce the same results in everyone? Why do some individuals, when faced with a life trauma, succumb to an episode, while others contain the traumatic effect homeostatically? To analyze the possible answers to this question, we must take a somewhat broader view of the entire problem of the detection and description of psychological dysfunction and mental disorders and their etiology.

Perhaps the most striking progress made in the field of psychopathology has been the emergence of scientifically constructed instruments for the description of psychopathology. Instead of depending on the free-floating interview of the past, we now have, at least for research purposes, systematic structured or semistructured interviews which yield reliable descriptions of a person's psychopathology. We can go even further and provide operational, or at least objective, criteria for selecting the best diagnostic category for a given individual. Once we ask about the validity and significance of the diagnostic category or of the dimensions elicited from the interview, however, we realize that we still lack good answers to these questions. Before we can answer them, we must know the etiology of the disorders. While predictive validity based on outcome, or treatment, can be of help, outcome criteria are so dependent on the ecological niche from which the patient emerges and the one to which he returns that such results are usually not generalizable. Depending for validity on treatment outcome, i.e., whether or not a given treatment produces a specific improvement in a given diagnosis, suffers from the same kinds of problems as outcome in general and is subject to the continual changing armamentarium of treatment methods.

After considering prediction and treatment outcome as validity criteria and realizing that concurrent validity is nothing more than a form of consensual reliability, we have left content and construct validity. We can exclude content validity from further consideration since the currently used interviewing methods do cover the entire waterfront of psychopathology. As for construct validity, we must postulate the constructs involved in the etiology of mental crises and episodes of disorder; this may be the only open road to validity that we now have.

Our ignorance of etiology is truly abysmal. How does one proceed to study etiology under such conditions? The only avenue open is to invent "as if"
etologies in the form of scientific models, develop hypotheses based on these models, and test them for their tenability by direct observations in experimental or naturally occurring situations.

Elsewhere (17) I have proposed six such models ranging from the molecular genetic model to the ecological field model. Each is so wide in scope that only by specifying assumptions and delimiting the scope can psychopathologists make progress.

Such an attempt has been made by providing more specific assumptions for the ecological, developmental, learning, genetic, internal environment, and neuropathological models (18). However, because each of these models has been standing alone, very rarely interacting with the others, it has become necessary to provide a second order model to integrate them. For this purpose, the vulnerability model has been proposed. According to this model, each of the scientific models of etiology should provide markers in its own domain which differentiate the vulnerable from the rest of the population. Since no one of these models is in itself a sufficient and necessary cause of psychopathology, the vulnerable person is characterized by the interaction among all of these models in the form of the pattern of markers which he possesses across all the models; but this pattern alone is not sufficient to initiate an episode. It must be triggered by some life event stressor, which produces a crisis, which in turn may develop into an episode in the vulnerable (19). What are the implications of this vulnerability model for the future direction of stress research?

The role that stress plays in this vulnerability model is threefold. First, it can serve as a triggering mechanism for eliciting a brief crisis or a longer enduring episode in a vulnerable individual. Second, it can produce an environmentally as opposed to a genetically based vulnerability as a result of an early noxious event often going back as far as the intrauterine existence or childhood, adolescent, or later developmental stress. Third, a persistent continuing stress may also generate a degree of vulnerability sufficient to engender an episode when a triggering event occurs.

Barrett has classified life events according to the first two categories but added an additional category of life events which serves to maintain the disorder once the episode is begun. Here is where behavioral analysis following the learning theory model can be of help, since behavior modification workers establish the contingencies that maintain deviant behavior as the targets for therapeutic intervention.

One of the basic questions implicit in the various discussions and which Barrett raises is whether the triggering events are specific for inducing various types of crises or episodes or whether they are merely happenstances for which no systematic classification or dimensions can be found. He concludes that particular life events and the degree of stress they produce can distinguish between the five subtypes of neurotic disorders he studied.

Thus far we have been content to accept life events at face value without requiring a rigorous definition. But are there any criteria that can be applied to differentiate events that serve as triggers of crises from those that do not?
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In searching for criteria to distinguish between life events that serve as triggers and those that do not, the following have been suggested: those that (a) produce losses (exists) or gains (entrances), (b) are undesirable, (c) are novel, (d) are unexpected, (e) are un anticipated, (f) are uncontrollable, and (g) require considerable readjustment of daily routine. Another aspect of the impact of life events, according to Bruce Dohrenwend, is the individual's own subjective rating of the potential stressfulness of an event. While the subjective rating may be a reflection rather than a cause of psychopathology, this rating may indicate the degree to which the individual would find the event subjectively threatening. It would be well to find a common denominator for the criteria by which life events can be defined. Perhaps the impact of the life event on the social network support system which surrounds the individual may serve as a carrier wave on which the criteria listed above can impinge. This may be the common denominator whose constriction or disruption, or even expansion, brings about the crises and/or subsequent episode. One could argue that the other criteria bring about their stressor effect only insofar as they overwhelm the defenses provided by the social network supporting system. This is why, for example, the presence of a confidant protects against depression and why certain losses can be withstood as long as the social network remains intact.

Hammer et al. (8) and Mueller (14) have provided a review of the studies in which the clout of life events can be explained on the basis of their impact on the personal social network. It is interesting to note that of the 43 items in the life event schedule provided by the Social Readjustment Rating Scale of Holmes and Rahe (9), fully 37 (86%) involve reduction in the social interconnectedness of the individual (death of spouse, divorce). Thus the social network serves two functions in our vulnerability model: (a) an etiological function, as one vulnerability marker of the ecotype insofar as a skewed or a constricted social network contributed to by early traumas or by an accumulation of past or present continuing stressful conditions may characterize the individual, and (b) as an agent for absorbing or failing to absorb the impact of immediate life event stressors.

Regarding the question of the valence (either good or bad) attached to the triggering life event, there seems to be a consensus that only the noxious life events have sufficient impact to trigger stress leading to a crisis or episode. Thus the type and quality of the social network which a person develops may serve as a bulwark against the life exigencies which are likely to provoke crises.

It is likely that the social network may serve as a psychosocial index of vulnerability in the same way that consanguinity with a mentally ill person serves as a genetic index of vulnerability. In other words, just as consanguinity with a mental patient identifies the risk of developing an episode on genetic grounds, the social network support in which an individual is imbedded identifies the risk of developing a crisis or episode on nongenetic or environmental grounds.

Dohrenwend (3,4) recently found that self-reporting instruments as screening devices for mental health do not discriminate clinically observed psychopathol-
ogy from nonclinical cases. It is likely that scores on these techniques measure not psychopathology but demoralization (7) and hence may lead to crises in everyone but to the development of psychopathology only in the vulnerable. If we had measures of vulnerability, we could separate out those for whom the scores on these instruments relate to psychopathology (the vulnerable) from those for whom no relationship to clinically recognizable psychopathology exists (the nonvulnerable).

It is clear in this volume that in addition to the vulnerability to crises and episodes which are based on genetic grounds, there are a considerable number of sources leading to crises, episodes, or deviant behavior in general which are not based on genetics but on life experience. We can classify these experiences as single traumas (such as death of a parent) or as continuing stresses (living in a deprived ecological niche). This nongenetic vulnerability can be related to the various etiological models proposed earlier. In fact, we can postulate etiological bases or etiotypes for each of these models even as we postulate a genotype for the genetic model. Thus we can postulate an ecotype for the ecological model, an auxanotype2 for the developmental model, a mathetotype3 for the learning theory model, a chemotype for the nongenetic aspects of the internal environment model, and a neurophysiotype for the neurophysiological model. Certain characteristics, indicators, or markers that indicate vulnerability can be classified under these various etiotypes. It is sometimes difficult to categorize the various markers in accordance with the procrustean schema we have laid down, so that the classification is still highly tentative. Thus for the ecotype we have as examples the following potential markers of vulnerability to psychological disturbance: (a) deviant social networks, (b) anomic, (c) socioeconomic status, (d) overcrowding, and (e) unemployment outside of the home.

One of the most potent arguments for vulnerability markers arising from the ecological model is Brenner's chapter. He claims that the economic cycles are etiological factors in the development of mental disorders (2).

How to integrate the research methodology initiated by Brenner's economic paradigm with the future endeavors in stress research presents a problem. Although the results of Brenner's work are quite impressive, they suffer from the well-known ecological fallacy. Furthermore, the conclusion that rate of unemployment is associated with rate of mental hospitalization is not supported by the data of Kasl. Perhaps the fault lies with the unrepresentativeness of first admission rates, since it has long been recognized that they do not tell the entire story regarding the incidence of mental disorders. On the other hand, Kasl's findings may suffer from the clinical fallacy of basing a generalization on a single case study, reminiscent of the claims that Buerger's disease was a Jewish disease, found only in Jews, until the disorder developed in the King of England.

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2 Suggested by E. I. Burdock.
3 Suggested by Kurt Selzinger.
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Only by utilizing the broad economic studies as pathfinders for clinical investigations and in turn using clinical results as a basis for verifying the results of economic surveys will we be able to profit from the interaction between these two fields.

For the auxanotype (development) we have as examples the following potential markers: (a) persistent nutritional deprivation in childhood, (b) maternal toxemias during gestation, (c) failure to develop intimacy in friendships during adolescence, (d) lack of confidant in adulthood, (e) loss of mother before age 11 and father before age 17, and (f) parental school truancy or dropout, which Robbins reports as being transmitted to the next generation.

For the learning theory model, the indicators or markers of vulnerability to psychopathology which Jenkins traced out of his data on ATC are: (a) infrequent or no church attendance, and (b) utilization of social coping resources, which are presumably learned behaviors. There is a likelihood that being too realistic about one's social competence may serve as a marker for dysphoria and depression. Mischel (13) reports that the self-ratings of depressives for social competence were more similar to the ratings of observers than was the case with normals and with nondepressed psychiatric patients. This greater similarity persisted even in remission. Thus a certain amount of unrealistic euphoria regarding social competence seems to be necessary for normal adjustment.

For the genetic model itself, the most likely marker is consanguinity with an individual who has had or is now having an episode of mental disorder. The internal environment model provides such markers as monoamine oxidase levels in the blood platelets and growth hormone assays. The neurophysiological model has many potential markers: pupillographic responses, evoked potentials, and reaction time. These and the previously mentioned markers have been discussed elsewhere (20).

The fact that none of these markers is by itself pathognomonic of psychopathology necessitates that we consider the patterning of the markers (or their interaction) as the basis for determining vulnerability. As is well known, however, being vulnerable does not necessarily lead to an episode of psychopathology. A triggering life event is needed for eliciting this vulnerability by inducing sufficient stress to produce a crisis. This crisis, which occurs in everyone subjected to stressors, may be contained homeostatically or, if it surpasses the individual's tolerance threshold, develop into an episode. This is where stress and vulnerability meet. The contributions of this volume and their significance for the future inheres in the attempts at classifying and dimensionalizing the life event stressors capable of inducing crises and episodes.

One of the more trenchant contributions in this direction was made in the chapter by Hurst. He examined empirically and to some extent theoretically the content of life event schedules, their scoring, and the clustering of the items of the schedule into sections. After analyzing the present state of the art along these three dimensions, he concludes that it is nearly impossible to develop a universal schedule applicable to all populations and all purposes and that by
subdividing the schedule into specific content categories, more focused research becomes possible. His comparison of the different schedules and their scoring with the special schedule developed for the ATC illustrates the importance of content, scoring, and clustering on the outcome of the research.

Dohrenwend (3,4) classifies life events into a triad of pathogenic elements consisting of (a) fateful loss events (bereavement), (b) physical illness and injury, and (c) disruption of social network. He indicates that if these factors overwhelm the internal and external mediating or protective factors, an episode of mental illness will ensue. He raises an interesting problem regarding the effect of extreme traumatic or fateful situations. While most would agree that at least transient psychopathology occurs in everyone under such traumas, opinions differ as to whether severe psychopathology develops in all of those exposed to such traumas. Thus the experience of concentration camp survivors seems to indicate, according to Dohrenwend, that this stress-induced psychopathology persists and even makes them more prone to physical illness and early death. On the other hand, Slater and Slater (16) found that in the survivors of a group of individuals buried by a blockbuster during the London Blitz, although all suffered transient traumas, only those who had a history of previous mental disorder in themselves or their families developed a severe, longer lasting episode.

The findings of Slater and Slater (16) would demand the prior existence of a vulnerability; according to Dohrenwend, no prior vulnerability seems necessary, everyone being at risk of developing a psychopathological episode if the stress is sufficiently severe.

Although Dohrenwend places disruption of social networks on the same level as the other two members of the triad, it could be hypothesized that fateful loss events as well as physical illness and injury bring about their effects at least in part through constriction or alteration of the social network, since bereavement of a loved one certainly is a disruption of the network and physical illness threatens to disrupt or constrain it. This hypothesis can be tested by noting how much of the variance of the stressful life events can be attributed to alteration in the social network.

In addition to the rational logical approaches (one might even call them clinical), there are certain statistical methodological aids that could have been used. First, even the logical categories for clustering life events could be subjected to statistical analysis to determine whether the items in a given cluster are homogeneous and whether any of the items within a given cluster show higher correlations with another cluster. Hurst's objection to the use of covariance between items because they assume or presume a causal connection is somewhat puzzling in this context.

Furthermore, once a set or pattern of items is established by clustering, it might be possible to cluster people according to the established patterns of items. This might produce homogeneous groups of individuals in whom the relationship between life event stress and psychopathology could become clear. Perhaps Lazarsfeld's latent structure analysis or some of the other typological
techniques now so popular could be applied to this end (1,6,12). One of the outcomes of such analyses might be an attempt to obtain the factor structure of stress underlying the various dimensions in the clusters of items. This could be based on the actual results of the application of the schedules to a variety of populations as well as on the ratings of experts for the importance or intensity of the items. Perhaps such a factor analysis could answer the question of what the criteria are for designating a given occurrence as a life event. The suggestions listed earlier as criteria for distinguishing an event from a nonevent could be weighed against the factor structure.

An examination of the siblings of the ATC with the same instruments used in the study would cast considerable light on the relationship of vulnerability to the occurrences of life event stressors. Since siblings share not only half their gene pool but a considerable portion of their life experience and ecological influences as well, the role of vulnerability in the life of probands and siblings could be evaluated.

SUMMARY

The role of life event stressors in psychopathology is a relatively new field of investigation. The earlier views tended to regard life event stressors as precipitants of psychopathology; i.e., they tended to precipitate an episode which would have occurred without the life event. Today we tend to classify life event stressors as either triggering events, which initiate an episode in the vulnerable, or as causes in themselves. The primary question facing the investigator is why the same life event stressor leaves some individuals untouched while in others it leads to either a crisis or an episode of mental disorder. This discussion provides a theoretical answer in the form of the vulnerability hypothesis, which assumes that the occurrence of a crisis requires the interaction of a life event stressor and a vulnerable individual, such vulnerability being based on either a hereditary transmitted liability or an environmentally produced or transmitted nonhereditary liability to a crisis, which may develop into an episode in the vulnerable. This vulnerability hypothesis can serve as an integrator for the various types of approaches to the question of the role of stressors in mental disorder, since it encompasses the entire range of liabilities based on ecological, developmental, learning theory, genetic, internal environment, and neurophysiological models of etiology.

To test the tenability of the vulnerability hypothesis, we must discover the markers that differentiate the vulnerable from the nonvulnerable. The work under the various scientific models listed above is providing such markers. Since no single marker is pathognomonic, the search is for patterns of markers that cut across the various models. Thus each of the models and the discipline from which it springs can offer its disciplinary contribution to the interdisciplinary goal of patterns of markers that identify the vulnerable. Once these markers are known, the contingent life event triggers necessary to transform the latent
vulnerability into an episode can be determined and therapeutic intervention instituted for preventing either the initial episode or any subsequent episode. After the triggering events are found, families of patients as well as patients themselves can be taught to avoid the occurrence of the noxious events or desensitize the patient to their occurrence if they are inevitable. Advances in mental health are likely to come in the future as in the past not from therapeutic interventions after an episode of psychopathology has occurred but from modification of conditions which lead to psychopathology.

REFERENCES