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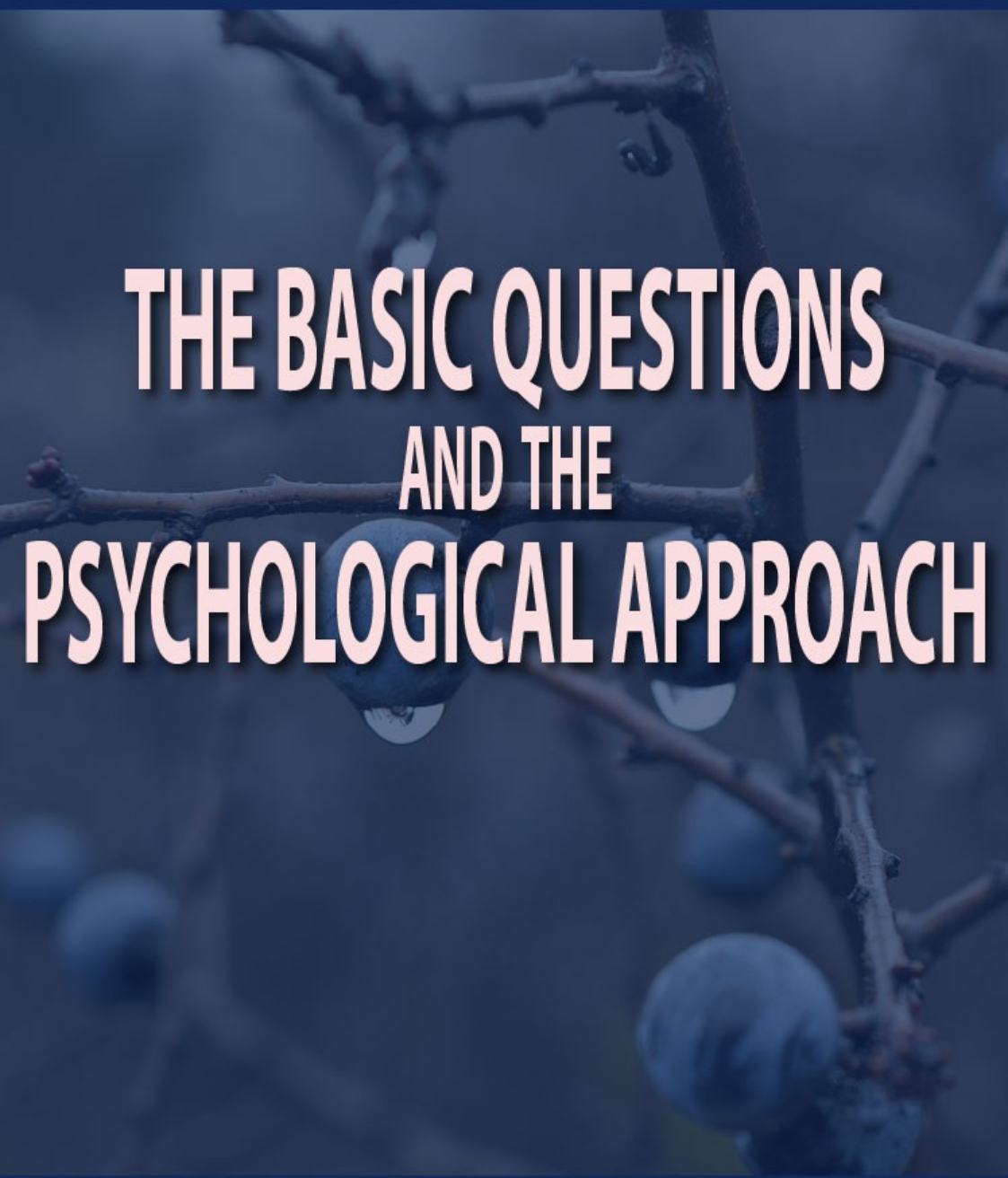

THE BASIC QUESTIONS AND THE PSYCHOLOGICAL APPROACH

Severe and Mild Depression

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Common is the sorrow that visits the human being when an adverse event hits his precarious existence or when the discrepancy between the way life is and the way it possibly could be becomes the center of his fervid reflection. In some people this sorrow comes and goes repeatedly, and in some others, only from time to time. It is painful, delays actions, and generally heals, often but not always after deepening its host's understanding and hastening his maturation.

Less common—but frequent enough to constitute a major psychiatric concern—is the sorrow that does not abate with the passage of time, that seems exaggerated in relation to the supposed precipitating event, or inappropriate, or unrelated to any discernible cause, or replacing a more congruous emotion. This sorrow slows down, interrupts, or disrupts one's actions; it spreads a sense of anguish which may become difficult to contain; at times it tends to

expand relentlessly into a psyche which seems endless in its capacity to experience mental pain; often it recurs even after appearing to be healed. This emotional state is generally called depression.

Is it just a feeling? Is it a syndrome? Is it a disease? It is a feeling but, contrary to common sorrow and sadness, it is also a syndrome insofar as it brings about severe alterations of psychological and some somatic functions. Whether or not it is a disease depends on the definition given to the term disease. If "disease" means a condition that causes a dysfunction of the organism, irrespective of evidence of cellular pathology and irrespective of the nature of the cause which determined it, we can certainly call depression a disease. Again, we must carefully differentiate depression from sadness and normal sorrow, which is a topic we shall take into consideration in chapter 5. Depression thus implies deviation from the normal way of experiencing some emotions, including sadness and sorrow.

One of the basic and most frequently asked questions is whether this deviation from the normal is *dependent upon external circumstances.* Many authors have emphatically stressed that depression is not dependent upon external circumstances; at times a

dependency seems to exist, but it is illusory. An unpleasant event which would elicit only temporary sadness or sorrow in the normal person, brings about in the patient a psychiatric illness that soon reveals an autonomous process. This psychiatric illness is at first difficult to distinguish from normal sadness, but eventually it is accompanied by agitation and restlessness or by severe retardation, inhibition, reduction in responsiveness to external stimuli, and recurrent sequences of gloomy and pessimistic thoughts. In some cases —those labeled manic-depressive—this psychiatric picture alternates with periods of euphoria and motor excitement.

If this condition is an illness or a medical entity, and if it is not found to be connected with external circumstances and has an autonomous development, then the conclusion drawn by many is that it is an endogenous condition; that is, originating within the organism itself.

When such a position is taken, research along certain lines of inquiry receives momentum. Inasmuch as neuropathological research proved to be completely useless since the time of Kraepelin (1921), the major fields investigated have been the genetic and the

biochemical. It is not within the scope of this book to cover these two vast fields. We shall mention only that several authors have found evidence that at least in biphasic manic-depressive psychosis (a disorder characterized by both depressive and manic attacks) there is a genetic factor. (See Mendels, 1974; Cadoret and Tanna, 1977.) Perris (1966) also reached the conclusion that patients who suffer from both manic and depressive episodes are to be genetically distinguished from those who suffer only from depressive attacks. In a subsequent article, Perris (1976) reached the moderate conclusion that "the combined results of clinical and biological studies of affective disorders support the hypothesis that genetic factors are of some importance for the occurrence of at least some groups of these disorders" (especially those presenting both the manic and the depressive phases).

Contrasting with quite a large number of authors who have concluded that a genetic factor may be involved in the typical biphasic manic-depressive disorder is the work of Odegard (1963), which showed no genetic difference between psychotic and neurotic forms of depression. In summary, a genetic factor seems to have been almost convincingly demonstrated only in typical cases of manic-depressive

psychosis. In them, too, however, it seems to be in the nature of a predisposition, and not a factor sufficient in itself to bring about the clinical condition.

If studies on heredity are far from conclusive, biochemical studies are even more so. The most promising line of research has followed the catecholamine hypothesis, according to which depression may be connected with decreased activity of some amine synaptic neurotransmitters, probably norepinephrine and dopamine. Conversely, in manic states there would be increased activity of these amines. This hypothesis and others have not gone beyond the hypothetical stage. (See Mendels, Stern, and Frazer, 1976.)

Another issue which to my knowledge has not been investigated is that the biological predisposition may consist of a greater facility to activate specific neuronal spatio-temporal patterns in the brain. These spatio-temporal patterns ultimately would engage more intensively than usual those parts of the limbic system or other parts of the brain that mediate the phenomenon of depression.

I am not in a position to say whether these altered patterns

ultimately could be subsumed in the category of altered biochemical events. But it seems to me that we should not bypass in a cavalier fashion a level of investigation which has been followed in the studies of other functions— for instance, language. Obviously we cannot believe that there is a center of sadness and depression which is similar to language centers or even to the pleasure center that the experiments by Olds and Milner (1954) suggest. If areas which mediate sadness and depression do exist, they probably consist of many cerebral areas, not contiguous, but working together through neuronal associations and constituting what Luria (1966, 1.973) called a functional system.

In contrast with my hesitation in taking any position about the previous matter is the security with which I can make the following affirmation: in the several decades spent in psychiatric practice and research, I have never treated for a considerable length of time a case of depression about which I could say that there was no psychological factor involved. I have never seen a patient about whom I could say that his depression was unrelated to a prior anguish, or about whom I could say that his depression came from nowhere and its origin had to be sought exclusively in a metabolic disorder. And yet I want to stress

that my experience deals predominantly with cases of severe depression that many authors call endogenous. Bemporad (who is not the author of this chapter), joins me in asserting that in the mild cases of depression which he has treated there was always some psychological factor involved.

Does this mean that the position taken in this book is that every case of depression is a *reactive* depression? Not at all, at least in the sense in which the word reaction is used by many psychiatrists. The concept of psychiatric illness as a reaction is attributed by some to Karl Bonhoeffer, who described what he called "exogenous reactions" (1910). However, Bonhoeffer referred to diseases of the brain caused by external toxic agents. He interpreted mental illness as a reaction to a physical alteration.

In the United States the concept of depression as a reaction derives from the works and teachings of Adolph Meyer. Although Meyer was much more interested in schizophrenia, he also enunciated for depression some principles that followed his general psychiatric orientation. He spoke of patients who "are apt to *react* with a peculiar depressive reaction where others get along with fair balance" (1908a).

(Italics mine.) In the same article Meyer stated, . . the etiology thus involves (1) constitutional make-up and (2) a precipitating factor; and in our eagerness we cut out the latter and only speak of the heredity or constitutional make-up. It is my contention that we must use *both* facts and that of the two, for *prevention* and for special characterization of the make-up, the precipitating factor is of the greater importance because it alone gives us an idea of the actual defect and a suggestion as to how to strengthen the person that he may become resistive." (Italics in the original.)

Adolph Meyer's words, written in 1908, retain their poignancy today. Meyer stressed that both factors have to be considered. He italicized "both" but not "reaction." Although Meyer referred to the precipitating factor as being important "because it alone gives us an idea of the actual defect," American psychiatry subsequently gave the emphasis to the concept of reaction. But what was the nature of the defect to which Meyer referred? We know from his other works and especially from his studies on schizophrenia that he felt both the "actual defect" and the reaction were the result of the total life experience of the patient as well as of his biological endowment (the psychobiologic set). However, if Meyer was not able to penetrate

sufficiently into the depth psychology of the schizophrenic, even though he studied him longitudinally, he was even less able to do so with the depressed patient. Although Meyer very correctly stressed that "the reaction" would not occur in psychiatric patients if they had not been prepared for it, he investigated this preparation in a vague and unsatisfactory way; namely as "substitutive activity" (Meyer, 1908*b).* As a result of Meyer's influence, American psychiatrists could be divided into two groups: the group who gave importance only to the reaction and saw the patient as a passive entity dominated by the external event; and the group who retained the old conception that the patient was suffering from an endogenous disease and therefore was to be viewed as an organism at the mercy of its chromosomic or metabolic destiny.

It is not the aim of this book to demonstrate or deny the existence of constitutional or biochemical factors. Whether such factors exist is not of major relevance to our theme, according to which other factors — psychological factors—must exist or at least coexist. Inasmuch as every psychological event requires neurophysiological and biochemical mechanism for its occurrence, in this book the existence of such mechanisms is reaffirmed but not studied.

Although the authors of this book give major importance to psychological factors, they do not subscribe to the usual concept of reaction. When the authors use the word “reaction” to describe some forms of depression in which the precipitating event seems of major importance (for instance, in the so-called reactive depression), they follow a commonly adopted terminology in order to prevent possible confusion.

The reason that does not permit us to embrace the concept of reaction, even in cases where the precipitating event exists and is likely to be of the greatest importance, is the conclusion drawn from our studies that such an event would not have become a precipitating factor unless it had a special meaning for the patient, and consequently an assumed pathogenetic power. Thus the environment and the patient both contribute to the transformation of the event into a cause: the environment, by offering the contingency of the event; the patient, by attributing either consciously or unconsciously a special meaning to the event. Furthermore, the event would not be given such a meaning and would not be endowed with such power if the patient had not integrated his past life experiences and personality in specific ways.

For many years I have indicated (and Bemporad, through clinical work, has also confirmed) that a preceding ideology prepares the ground for depression. The ideology is responsible for the special meaning given to the precipitating event and for the way the patient deals with that event. When there is no recognizable precipitating event, there is nevertheless a special preexisting ideology which to some extent consciously, and to a much larger extent unconsciously, has prepared the ground for the depressive outcome. The ideology, which had an integrative function in the life of the patient and was used as a defense, now makes him experience a state of partial or total loss, helplessness, or hopelessness which is accompanied by depression. Whereas the depression generally remains as a subjective and conscious phenomenon, the cognitive substratum may become partially or totally unconscious.

In this book we shall illustrate how this ideology came to be. In many cases its origin can be traced to childhood events. However, it will be apparent to the reader that although we consider childhood experiences very important, in agreement with classic Freudian tenets, we do not consider them the exclusive determinants of a psychopathological course of events as it unfolds in the life history of

the patient.

In the terminology of general systems theory, the psyche is not a closed system but a system open to continuous influences from factors occurring outside the system (Bertalanffy, 1956). Psychopathological structures are also open systems. They are states of various degrees of improbability that are maintained by negative psychological entropy coming from outside the original system. An open system such as the psyche follows the principle of equifinality; the final state is not unequivocally determined by the initial condition. Each stage of life is under the influence of the previous stages, but not in rigid or ineluctable ways. Other factors intervene. Early experiences participate in causing depression only when, together with other factors, they facilitate the formation of an ideology which will lead to unfavorable patterns of living.

Several issues emerge from the foregoing. Even though we speak of patterns of living and of specific behavior throughout this book, we do not focus on the external behavior, but on the ideology and subsequent mechanisms that lead to the formation of these patterns. It is for this reason that our approach is called cognitive. Although I have

done psychiatric research on cognition since 1946, my first writings on cognition in reference to depression appeared in 1959 and 1962. Since 1963 Aaron Beck has also stressed the cognitive approach to depression. The approach that I have proposed and to which Bemporad has added a number of original contributions differs from Beck's in that our approach is longitudinal and dynamic. It does not stress the point that the patient is depressed because he has depressive thoughts, but it puts in evidence a cognitive history whose existence was to a considerable extent unconscious. Many people experience reluctance to accept a cognitive approach lest the affective life and especially motivation, conscious and unconscious, be disregarded or not recognized in its full role. This apprehension particularly is felt in connection with the study and treatment of affective disorders, in which the major deviation from the normal involves affects.

A prevailing cultural anti-intellectualism has caused misapprehensions and distortions even in the fields of psychiatry and psychoanalysis. A cognitive approach stresses a fact which is very seldom acknowledged in psychiatry and psychoanalysis: at a human level most emotions do not exist without a cognitive substratum. The

expansion of the neocortex and consequently of our cognitive functions also has permitted an expansion of our affective life. In a classic paper published in 1937, Papez demonstrated that several parts of the rhinencephalon and archipallium (included now after MacLean, 1959, in the limbic system) are not used for olfactory functions in the human being, but for experiencing emotion. In spite of the diminished importance of olfaction, these areas have expanded rather than decreased in man, and have become associated with vast neocortical areas. Elsewhere (Arieti, 1967) I have shown that in the human being elementary emotions, which can exist without or with relatively little cognitive counterpart—such as tension, fear, and rage —are changed into higher emotions (anxiety, anger, depression, hate, and so forth) through the intervention of complicated cognitive mechanisms. It is because of these connections with a potentially infinite realm of symbolic cognition that the emotional life of the human being also becomes immense and potentially infinite. In chapter 5 I shall illustrate how cognitive elements can give rise to sadness and depression.

At the beginning of this century, Bleuler and Jung introduced into psychiatry the concept of "complex." According to Laplanche and

Pontalis (1973) a complex is an "organized group of ideas and memories of great affective force which are either partly or totally unconscious. Complexes are constituted on the basis of the interpersonal relationships of childhood history; they may serve to structure all levels of the psyche: emotions, attitudes, adapted behavior."

The concept of complex was embraced immediately by classic psychoanalysts, but Freud soon found it to be an unsatisfactory theoretical notion (Jones, 1955). Since then it has lost popularity in psychoanalytic and psychiatric circles, although gaining great popularity in common parlance. A complex is referred to in this book as a cognitive construct or as a system of constructs. The old term "complex" has lost value in professional circles because not enough importance was given to its cognitive components. It is understood that when we refer to a construct we do not connote something static, but something potentially capable of changing throughout the life of the individual; something which is altered by life events and at the same time is a promoter of other life events.

Although it is important to study the transformation from the

cognitive to the affective components of the psyche, it is equally important to study why in our psychiatric cases it is so difficult for the patient to free himself from the intense feeling of depression. Often he cannot escape from the feeling of having lost what was most valuable to him, either something specific or something very vague, undefinable, or impossible to express with words. In serious cases the patient mourns the most profound loss, the loss of life's meaning—an experience which reflects in magnified form an original loss or a series of factual or symbolic losses. In these very serious cases the patient feels or acts as if he had reached an inevitable conclusion that his life is meaningless and worthless. The intense depression that accompanies this apparent conclusion actually betrays the patient's attachment to and love for life, and his inherent premise that life is meaningful and should be worthwhile. There is thus something psychologically positive even in this deep depression. In his inner self the patient is not one of those people who consider the events of the cosmos to be due to random collisions of atoms, transformed by chance into organized unities, and completely independent from the needs of the human heart.

Outside the realm of pathology, is there anything similar to this

deep depression which implies the inevitability of what is dreaded most? Yes: the *tragic* situation. The relation between depression and tragedy will be studied in chapter 16.

Before proceeding to discuss further the cognitive approach, I must mention that the other psychological interpretations of depression are reviewed by Bemporad in chapter 2. In chapter 16 the relation between depression and some ideologies to which the patient is exposed in his sociocultural environment also are described and discussed.

The major themes of this book will be, however, the psychopathology and psychotherapy of the individual patient—child, adolescent, and adult. When we go beyond the study of manifest symptomatology and make our first acquaintance with the history of the depressed patient, we feel as if an invisible force, running throughout his life, has brought him to his present predicament. One of the main aims of this book is to make visible that invisible force, by showing how it came into existence and sustained itself on many facts, internal and external, individual and sociocultural; most of them unconscious and others conscious, but with unconscious ramifications.

Another purpose of this book is to illustrate in detail our psychotherapeutic approach. The psychotherapist intends to affect the psychological etiological factors and mechanisms to such an extent that even when biological mechanisms enter into the picture, by themselves they will not be able to maintain the disorder, at least with the same degree of intensity. A successful psychotherapy also will make much less likely the recurrence of the disorder. We hope that our case reports will support our optimism. We have come to the conclusion that most cases of depression, ranging from mild to severe, benefit from psychotherapy. We do not object to the use of other types of treatment in some cases, but we feel that for many patients the psychotherapeutic approach will prove very rewarding, even when other types of therapy have failed. Combined forms of treatment also can be used in a few selected cases.

Although the patient is by far our major concern, we should not omit considering that the psychotherapeutic approach also is rewarding for the therapist, who learns in greater depth some dimensions of human existence. As I wrote elsewhere (1976), when we successfully treat a patient who has been severely depressed we experience a burst of joy because we have helped a person who is

happy to have known us. But we also feel a secret joy because we have come to know the patient, and in knowing him we know more of ourselves. There is always a resonance in our heart for the anguish of the depressed which does not seem to us completely unfounded, but similar to ours, and containing a partial truth based on the human predicament.

This study of the depressed person will show how we ourselves can contribute to our own sorrow with the strange ways in which we mix and give meaning to our ideas and feelings. We shall learn that the study of life circumstances is important, but that even more important is the study of our ideas about these circumstances, our ideals and what we do with them, and how we use them to create feelings. This study will explore, and we hope to some extent enlighten, not just our pathology but our so-called normality; not just our despair but our confident expectation; not just our loneliness but also our ways of helping each other and reinforcing the human bond.

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