# The Classification of Endogenous Psychoses

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*5th Edition*

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# FOREWORD

The concepts of bipolar and unipolar disease as applied to manic-depressive disease originated with Karl Leonhard. He first referred to the distinction in the first edition of this book in 1957[1] and elaborated it in an article published in 1962.[2] This fifth edition of his *Aufteilung der Endogenen Psychosen* is being presented in an English translation as a fifth edition, to keep the edition numbers the same in Germany and in the English speaking world, under the English title, *The Classification of Endogenous Psychoses*.

The leading current American approaches to clinical psychiatry are either Kraepelinian, Meyerian, or Freudian. Leonhard has been working systematically over the past 30 years to present another approach to clinical psychiatry, one which does not slavishly follow any of those approaches and is very different from most American perspectives and also from such perspective as that of Slater and Roth in Great Britain.

Perhaps his most important contributions are his profound insight into the variations in clinical manifestations and his correlation of these with diagnosis and with family studies. His book will provide the English-speaking world with a new and, I believe, fascinating way of approaching clinical psychiatry.

The aspects I should like to emphasize are inherent in Professor Leonhard's division of the psychoses into 38 different clinical illnesses, each of which is classified primarily as a Phasic Psychosis, Cycloid Psychosis, Unsystematic Schizophrenia, Systematic Schizophrenia, and Combined-Systematic Schizophrenia. Subsidiary divisions of these

[1]Leonhard, K, *Aufteilung der Endogenen Psychosen* (First ed.), Berlin, Akademie-Verlag, 1957.

[2]Leonhard, K, Korff, I, Schulz, H, "Die temperament in den familien der monopolaren und bipolaren phasischen psychosen," *Psychiatry Neurol.*, 143: 416-434, 1962.

five major classifications make up the 38 illnesses. Additionally, there are extensive chapters on:

1. Age of Onset, Sex Distribution, and Number of Phases in the Phasic Psychoses (including the cycloids).
2. Incidence of Psychoses in the Families of the Phasic Psychoses (including the cycloids).
3. Age of Onset, Sex Distribution, and Course in the Schizophrenias.
4. Number of Psychoses in the Families of Schizophrenias.
5. Genetic and Psychosocial Influences on the Origin of Schizophrenias and Cycloid Psychoses.

Professor Leonhard's book will, I believe, make a significant difference in the way in which both American psychiatrists and psychiatrists from other countries look at psychotic disorders whether or not Leonhard is wholly correct and whether or not there are some errors that will come to light only when his work has been fully exposed to investigators here and abroad. What is certain is that he has a number of novel observations and conceptions that have not been exploited or even described in the American or English psychiatric literature.

ELI ROBINS, M.D.

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ELI ROBINS, M.D.
RUSSELL BERMAN

# PREFACE TO THE ENGLISH LANGUAGE EDITION

(From the Current 5th German Edition)

While this English language edition was being prepared, my colleagues and I were completing a large series of tests in Germany. With the help of Frau v. Trostorff, 634 chronic schizophrenics were investigated and 500 of them were statistically evaluated. The evaluation brought many new results to light which are included in this new edition. Cataphasia and periodic catatonia can now be described more precisely in terms of the symptoms and their range. There are new results for the systematic schizophrenias, particularly in the combined cases and since each combination is rare, every observation is important. Thus, it became necessary to rewrite the section on combined-systematic schizophrenias. In addition, a different test series produced new results regarding the origin of schizophrenia. An examination of the differential diagnosis in twins pointed to psychosocial causes of systematic schizophrenia. Although, we had found a relatively small number of psychoses in these families earlier and had come to the conclusion that heredity apparently does play an important role, we had been unable to comment on other causes which are now discussed in the final chapter. Thus there are some essential improvements, but no fundamental changes. My opinion has been confirmed, that independent forms of endogenous psychoses must be differentiated and described.

KARL LEONHARD

# PREFACE TO THE FOURTH EDITION:

After I had improved the clinical aspect of the third edition by means of the newly won knowledge, the necessity to go over the statistical description and to relate it to the newer results developed. This was possible through investigations which some of my associates carried out. My earlier statistical results had been gained from a set of patients without a fully stable differential nosology, as described in the preface to the third edition. After the clinical improvements, the statistics for cases with secure diagnoses had to be redone as well. Thus the statistical section, like the clinical, became more self-contained. Finally, the genetically different nature of the different groups and forms becomes considerably clearer now than it was before.

Karl Leonhard
Berlin, June 1968

# PREFACE TO THE THIRD EDITION:

In the first and second editions of *Division of Endogenous Psychoses*, there was still some confusion about the boundary between curable and incurable forms. Phasic psychoses, on the one hand, and systematic schizophrenias, on the other, certainly stood in very clear opposition to each other, but the line between cycloid psychoses and unsystematic schizophrenia still appeared partially unclear because of the many misdiagnoses. So as not to hide any confusion in the first and second editions, I reported all the cases where misdiagnoses became apparent due to the catamnesis or the family picture of the immediate and extended family. Furthermore, I listed all relevant cases in which no differential-diagnostic questions appeared in family members. It was my duty to decide whether or not the same diagnosis in the proband was the correct one. In this third edition, I continue to describe cases when I can draw on the family picture, but many unclear cases, in which the proband's diagnosis was uncertain or did not agree with that in the family members previously described, are not included. My justification for this is the confirmation of my opinions that have appeared since the last edition. In particular, a larger study I undertook with Frau v. Trostorff* showed that we could diagnose the cycloid psychoses (which are of decisive importance in dividing the phasic from the progressive cases) with much greater certainty since nearly all of them proved catamnestically to be correct. In this edition, I have also included cases described by others as schizophrenia, but by myself as cycloid psychosis. Generally, the cases corresponding to my expectation were cured. I was able to show that no less than half the cases, described with the term schizophrenia in its usual broad meaning, recover.

*Leonhard, K. and S. v. Trostorff., *Pronostische Diagnose der endogenen Psychosen*, Fischer Jena, 1964.

To improve the nosology, I worked, on the one hand, through all the cases referred to in the first and second editions; that way I had learned to observe things better. Furthermore, I was able to try my concepts out on all the cases that I was seeing in the clinic. My ideas changed to the greatest extent in regard to the clinical picture of schizophrenia, i. e., cataphasia, as will be seen in its description in this third edition.

Although many cases which had been unclear could now be left out because of the strengthened nosology, a large number of histories still had to be discussed. This was necessary to the presentation of a clear picture of what I wanted to show. With the multifaceted "polymorphic" and "unsystematic" forms, the only way to show the symptomatology is through a series of cases and family pictures and patterns. With the pure and systematic forms, a few cases would have been sufficient in each form, but I had to continue to list all related cases, since the pure, i. e., systematic character, if it really exists, should recur in secondary cases in the family.

The third edition is, in general, in comparison with the two first editions, more self-contained, which will, I hope, be a benefit.

Karl Leonhard
December 1965

# INTRODUCTION

For decades psychiatry has been struggling for a new orientation but can obviously not find it, the diversity of opinions in psychiatry being greater today than ever. Kraepelin's teachings have been rejected, but whenever nosological questions are raised, his dichotomy of the endogenous psychoses reappears. One has moved away from the overemphasis on heredity, replacing it with the causality of psychosocial factors, without, however, knowing what they look like. Although the attempt was made to explain the genesis with psychoanalytic methods and to delineate the difference from neuroses, the empirical evidence was insufficient. Some claim that diagnoses should be scuttled altogether and thus re-embrace, albeit based on new theses, the theory of the "unitary psychosis," which was current more than a 100 years ago and has reappeared in new clothing. It is claimed that schizophrenics could be cured with psychotherapy, but they are everywhere treated with neuroleptic medicines and sometimes even with leucotomy.

I will not enter this altercation but rather will speak as an empirical clinician. In this stance, I repeatedly observe differences among the endogenous psychoses, which are at times great, and which hardly appear to be bridgeable. What could a melancholy have in common with a hebephrenia? Or solely within the context of the schizophrenias, how could one unite a fantastic paraphrenia with a negativistic catatonia. Kraepelin's classification into only two forms has been damaging. He himself attempted many finer distinctions with great enthusiasm and continued open-mindedness, but his followers ignored this; they only saw the coarse division into schizophrenia, i. e., dementia praecox, and the manic-depressive disease. The theory of the psychic diseases, for which there seemed to be no external cause, was thus simplified in a frightening manner. While the related neurological discipline recognized hundreds of endogenous diseases and continues to describe more,

psychiatry perceives only two. While neurologists attempt to describe the heritability of each one of the genetic forms of endogenous diseases and in fact at times describe several heritability patterns for forms which are clinically identical, psychiatrists are still arguing over the heritability of schizophrenia (in the singular!)—as if so varying and at times extremely different pictures would correspond not only to a single disease but also to a single heritability pattern! Neural muscular atrophy, which in fact appears to be a clinical unit, displays four different heritability patterns.

The development would certainly have been different if Wernicke, Kraepelin's main opponent, had not died so early. He, who like no one else could pick out and describe the characteristic single-pictures from the plentitude of endogenous psychoses, would certainly have remained very one-sided without Kraepelin's etiological method of consideration, but his classical attempts would have at least resulted in the interest in clinical psychiatry's individual pictures remaining alive. Even today, there are still very few psychiatrists who have read Wernicke's textbook. Whoever does so, receives an impression of how much more the precise clinical description of endogenous psychoses meant to him than today's authors, who only ask whether it's schizophrenia or manic-depression, or perhaps not even that anymore. One should not devote much time to his theoretical writings which always end up with a circular theory, but should read the description of the diseases. Wernicke, who had so many students in brain pathology, did not have them in the field of psychiatry. Only Kleist picked up his teachings and followed the same path. His spirit and the spirit of Wernicke are in my book.

If not two but rather many endogenous psychoses are to be differentiated, then psychiatry becomes of course a difficult science. However, this differentiation cannot be avoided, if we are to get out of the dead end of the present theory of endogenous psychoses. For the phasic psychoses, the investigations by Angst, Perris, Winokur, and others seem to have finally demonstrated that unipolar and bipolar endogenous depressions must be separated genetically and that many further differentiations must yet be made. In the realm of schizophrenias, a peculiar contradiction has been apparent for years. Most psychiatrists agree with Eugen Bleuler, who once spoke of a "group of schizophrenias" and not of one schizophrenia, but most scientific publications sound as if there were only one schizophrenia. This contradictory posture leads us no closer to the solution of the many problems related to schizophrenia. If each separate form cannot be isolated, then one should at least distinguish the various subgroups, each of which constitutes a relative unit. There are five distinguishable subgroups:

1. unipolar phasic psychoses
2. manic-depressive disease

3. cycloid psychoses
4. unsystematic schizophrenias
5. systematic schizophrenias.

The last mentioned forms deserve particular attention, since they correspond to the largest group of patients, mainly chronic, but with a particularly low rate of psychoses among relatives. An alarming result, which we have emphasized in our investigations recently, underscores the urgency of work on these most severe forms of schizophrenia. In an investigative series of 72 pairs of twins, we found systematic schizophrenias only in fraternal twins, but never in a case of identical twins. Thus psychosocial factors, which were previously associated mainly with milder forms of schizophrenia, appear to predominate in the origin of these schizophrenias.

If, due to the uncovering of many individual forms of endogenous psychoses, psychiatry becomes a difficult medical discipline, I believe it can only be advantageous. One will be forced to delve into the very different individual pictures. The rewards of this effort will be not only scientific, but also practical, since a differentiated nosology will lead to a differentiated prognosis. Kraepelin once considered his dichotomy in prognostic terms, but his prognostic goals were not upheld in total success. On the one hand there remained of course the benign manic-depressive disease, but in the cases considered to be schizophrenic, there are nearly as many cases which recover as those which end in a defect. Thus today the diagnosis of schizophrenia implies nothing about the prognosis. However with a differentiated nosology, one can tell the patients and their family that the motility psychosis, which one has distinguished from the picture of a catatonia, that the happiness psychosis, which has been separated from a paranoid schizophrenia, that the anxiety psychosis, or the confusion psychosis, that all these psychoses end with a total recovery. Similarly, therapy would take very different directions depending on whether the psychosis is of a kind which leads to spontaneous recovery, or if it could lead to a defect if left untreated. Unfortunately I often see cycloid psychotics, who are kept in a toxic-diseased state by continuous medication, although they would be absolutely healthy without this medication. If the continuous medication could prevent further phases, then it would be justified, but this is unfortunately not the case. Thus patients, who might intermittently be healthy for longer periods or perhaps even forever, are kept in chronic, toxic states, which are frequently accompanied by extrapyramidal disorders. Whenever I could, I liberated patients with endogenously cured cycloid psychoses from their toxic disease. I will not take a position here on the question of prophylaxis with lithium.

I want to add, that every psychiatrist must plunge into his or her discipline when the diagnosis becomes difficult. Precisely the ease, with which a psychiatrist can produce a diagnosis today—he can hardly miss,

if he simply chooses between the two possibilities, manic-depressive disease and schizophrenia—has led to the inability of many even to see and describe a psychiatric picture correctly. Instead of the description, one finds hackneyed psychiatric terms with which the disease—usually schizophrenia—is described and, allegedly, correctly evaluated.

If one wants to prove that an endogenously appearing psychiatric picture corresponds to a separate disease form, then it must be shown that it repeatedly occurs in a similar form. It is particularly important if the same history repeats in one family, for, from that, one could deduce that the genetically same disease does in fact produce the same clinical picture. If, on the other hand, the psychoses in one family display significant differences, then one can deduce a broad range of symptoms for the same disease, as long as several diseases are not present in the same family. I have explored the possibilities that develop due to the presence of several cases in one family. The result was that some psychic diseases always recurred in the same form, while others seemed to have a broad range of symptoms. In order, thus, to prevent decisive evidence, I have particularly listed cases below where other cases existed in the same family, so that they may all be compared with each other.

Since I also wanted to reveal the cases I refer to from a statistical point of view, I could not choose them at will, but rather I had to restrict myself to certain ones. In reference to the phasic diseases, I took the cases which were admitted to the Frankfurt Nerve clinic in the years 1938 to 1942. I worked on these cases with Neele and later added the statistics. Neele[1] has already presented some of the insights that developed from that work. I had to proceed otherwise with the schizophrenias, since I wanted cases which started at least ten years ago, so that their course could be well observed. I obtained these cases through Kleist's[2] carrying out many catamnestic investigations and frequently consulting with me on them. I included all cases that I saw myself. There were also 55 patients who were in the clinic in the years 1937–1940 and for whom the disease onset lay at least 10 years in the past. Through the kindness of Bruno Schulz, I could examine a large number of older schizophrenics in Bavarian institutions. In total there were 526 phasic psychoses, including both circular and cycloid forms, and 325 schizophrenics.

In Berlin I was able to increase the number of the latter greatly. In the past years I examined 634 schizophrenics in psychiatric hospitals with predominately chronic patients and, with the help of Frau v. Trostorff, was able to carry out a statistical evaluation of 500 cases (250 men and 250 women). Thus the number of schizophrenics on which I base my

[1]Neele, *Phasic Psychoses*, Leipzig, 1949.

[2]Kleist, *Nervenarzt*, 1943, p. 1, and 1947, p. 481. My own comments are in the *Allg. Z. psychiatr.*, 1942 to 1944.

clinical observations rises to 971 and the number of statistically evaluated cases to 837. The number of phasic psychosis increased through the additions of the cases researched by people who worked with me to the number of 1163. I hardly need to mention that I confronted the many patients with endogenous psychoses whom I saw daily, not only with routine interest but with scientific curiosity.

# The Classification of Endogenous Psychoses

# PART 1

# The Phasic Psychoses

# CHAPTER 1.

# MANIC-DEPRESSIVE DISEASE

Until very recently nearly all psychiatrists were united in the opinion that the manic and the depressive disease pictures were all part of the manic-depressive disease. It was the work of Angst[1] and Perris[2] that helped spread my theory that unipolar and bipolar diseases were separable. I carried out my first investigations together with Neele, and Kleist[3], with whom we were working, shared our opinion. Previously, Kleist had claimed that there was no independent manic-depressive disease, but rather only a melancholy and a mania with a certain reciprocal affinity. Thus he had already claimed the independence of the unipolar forms, but had gone too far by totally denying the independent existence of the manic-depressive disease. The genetic difference between the unipolar and bipolar forms was seen in that the manic-depressive had a significantly higher rate of psychoses among relatives than did unipolar forms. Winokur[4] and others followed in this line of thinking.

However the two disease forms also have different clinical pictures. The bipolar form displays a considerably more colorful appearance; it

[1]Angst, I., *Zur Aetiologie und Nosologie endogener depressiver Psychosen,* Berlin-Heidelberg-New York, Springer, 1966.

[2]Perris, C., "Study of Bipolar (Manic-depressive) and Unipolar Recurrent Depressive Psychoses," *Act. Psychiat. Scand., Suppl. 194,* Munksgaard, Copenhagen 1966.

[3]Kleist, K., *Nervenarzt,* 16: 1 1943; and 1947, 18: 481 p. 481. My own comments in *Allg. Z. Paychiatr,* 122, 39: 1943.

[4]Winokur, G., Clayton, P., Reich, Th., *Manic-depresseive Illness,* St. Louis, Mosby, 1969.

varies not only between the two poles, but in each phase offers different pictures. The unipolar forms, of which there are several (Angst, Perris, Winokur do not treat this) return, in a periodic course, with the same symptomatology. Every individual form is characterized by a syndrome associated with no other form and not even related transitionally to any other forms. On the other hand, in bipolar cases no clear syndromes can be described since there are many transitions between various formations and the picture may even be distorted during one phase. Thus, one can generally recognize a bipolar form during the first phase. In the same sense, one is also in the position to recognize as bipolar those forms which only accidentally swing toward one pole but which contain the potential toward the other pole. Consequently the differentiation is better made between polymorphic (bipolar) and pure (unipolar) forms. Both possibilities of differentiation will be explored in the statistical section so that nothing will be assumed that should first be proven. In the clinical section however, the line can be drawn correctly and immediately between the polymorphic and the pure forms. The number of polymorphic disease forms with no tendency at all to the opposite phase is very small, insofar as one considers mild indications (of the other phase) important. The opposite phase will merely hint at itself not infrequently though it cannot fail to be recognized. Manic patients can have a depressive mood for hours, depressive patients can display a noticeable excitation with gaiety and busyness while their depression disappears; anxious patients are not infrequently suddenly ecstatic; on the other hand, ecstatic patients may have short anxious oscillations; hyperkinesis will often be interrupted by short periods of lack-of-motion while akinesis can have short agitations. The quick course of all these variations may not be valued as the expression of separate phases, but it does show the disease-potential toward the other pole. Furthermore, depressive patients can often be excited by encouragement and can be led out of their depression. They become lively, talkative, and hardly show a depressed mood. It can, in fact, occur that a patient, during such an encouragement-discussion, becomes so lively that one is finally forced to describe him as hypomanic. Afterwards he or she returns directly to the depressive mood. With the essentially unipolar forms, there are no signs of lability toward the other poles; a unipolar melancholic never shows a manic trait—no matter how long it lasts or how often it returns—and a unipolar mania never has a trace of depression. One should expect reactive depressions only when a manic patient realizes after the end of the psychosis what he or she has done during the disease. But such depression can generally be differentiated from depressive oscillations of a manic-depressive disease by the presence of normal motivation.

The clinical picture of manic-depressive insanity described by Kraepelin is well known. *But since one has continually differentiated it only from schizophrenia and has disregarded the other phasic psy-*

*choses, i.e., the pure forms and the cycloid psychoses, the basic understanding of the range of symptoms, which in any case is broad, must be reviewed.*

In one group of the cases, the symptomatic picture is restricted to the manic and the melancholic basic syndrome. The first is composed of euphoria, that can easily develop into annoyance, elevated self-consciousness, flight of ideas, pressure of speech, and busyness. While the second includes a depressive mood with ennui, feelings of insufficiency, thought blocks, psychomotor retardation, difficulty in making decisions and depressive idea construction. We will also find these syndromes in the pure mania and the pure melancholy—there they are in fact necessary forms, while in the manic-depressive disease these syndromes only rarely appear in their pure form. If that is the case, then only the bipolarity makes the correct diagnosis possible.

More frequent in manic-depressive disease are those cases in whom the basic syndrome of the mania as of the melancholia, or of both, has changed. Essential single symptoms can be absent. A mania can be merely a euphoric mood without flight of ideas and busyness—that is the origin of an "unproductive mania." If the busyness is absent, then the patients can develop flight of ideas and pressure of speech, but show no other restless motions. Conversely, the euphoric mood can be absent, while flight of ideas and busyness are present. Similarly in melancholia, the depressive mood and the consequent inferiority ideas will alone be present, while thought inhibitions and psychomotor retardation are absent. Then the depression takes on a slightly agitated character, without a further atypicality being suggested. Every depressive feeling tries to find release since it is painful, and even if an inhibition or retardation is absent, the depressive affect displays a slightly anxious character. If the anxiety appears more purely, then the affect takes on a form atypical for the manic-depressive disease. (I will return to that point.) If, on the other hand, the depressed mood is absent, so that only the thought and psychomotor inhibitions are present—or perhaps only one of them—then simple inhibited states arise. Of course, the depressive affect is generally only temporarily absent; on the other hand confusion with motiveless states of other genesis is possible.

The incompleteness of manias and melancholies—I want to speak of manic and melancholic "partial states"—can go further. With the pure euphorias and depressions we will see that there are different forms of affect and thus different forms of affective disorders. Harried depressions, hypochondriacal depressions, enthusiastic euphorias, confabulatory euphoria, and so on, apparently arise when only one type of feeling is affected by the disease. Thus there is a dissociation within the sphere of feelings, and such dissociations will be at times agitated by the manic-depressive disease, so that its partial states will resemble a harried depression, an unproductive euphoria, and so on. One seldom finds this phenomenon markedly developed, but one often observes that

in one phase of the manic-depressive disease one or the other type of feeling will be more affected, so that there will at least be hints of one of the pure euphorias or depressions.

More seldom than the partial states are the mixed states in which in the place of the missing symptom the symptom of the opposite pole appears. Kraepelin described this extensively. Euphoria can be tied to a psychomotor inhibition. The latter can in fact be extreme and a manic stupor arises. Unproductive manias, already mentioned as partial states, can also be mixed states. When a thought inhibition joins a euphoric mood and a busyness, then an unproductivity of thought and speech replaces the manic flight of ideas. Conversely, the manic thought disorder can join a depression thus creating a depression with flight of ideas accompanied by press of speech. No other excitations must be present; the excitement in thought alone can lead to the pressure of speech. If, on the other hand, a psychomotor excitation joins the depression, then the decision is difficult whether the affect is only looking for release and is led to anxious excitation, or whether there is a manic component in the excitation. The latter (the manic component) is certain if a busyness without relation to the depressive mood is recognizable.

Other variations of the manic-depressive disease arise when the disease-events go beyond the normal margins and create symptoms otherwise associated with the cycloids (i. e., also a bipolar disease form). Best known of the manic-depression variations is the confused mania in which the thought disorder has a character otherwise associated with the confusion psychosis. There is not necessarily a consciousness disorder. In the hyperkinetic phenomena there are traces of the motility psychosis and, in the ecstatic phenomena, there are traces of the happiness psychosis and in the otherwise manic picture. In the stuporous depression, the inhibition takes on the importance of mutism, in the confusion psychosis and in the motility psychosis of akinesis. Severe anxiety corresponds to the affective disorder of the anxiety psychosis. It is questionable if the consciousness is involved in the manic-depressive disease, as it often is in the cycloid psychoses. One could suspect it in extremely excited or confused manias, but judgment is difficult since these patients receive sleeping pills regularly.

The total picture of the manic-depressive disease will appear when I describe single cases. The most informative cases are those in which other cases of the disease in the same family have been clinically described, since the range of symptoms in the same family can be observed. First I list only those patients who themselves and their relatives show only the typical symptoms of manic-depressive disease. In one group of these cases, atypical traits were apparently absent because the phases appeared in particularly mild forms. As we shall see, this relates to the fact that a greater intensity of the disease process is

especially able to create atypical traits in the sense of the cycloid psychoses.

The described cases come from my Frankfurt investigations and are found in the first edition of the book. That it is thus a question of older cases is actually advantageous, since no neuroleptic treatment was carried out then.

*Case 1:* Elisabeth Kol, born in 1908, became depressive in September, 1937, made self-accusations, was afraid her father would die, and nearly stopped speaking. She appeared normal from January to March 1938, then an excitation began with pressure of speech and activity. Thus she entered the clinic, had flight of ideas, was partially happy, partially irritable. After she had calmed down, she was released. In 1921, the *mother's sister* had spent ten days in Institution T due to a depressive mood. She had to ponder over religious questions, made self-accusations, and complained about a lack of feeling toward her children. She did not become ill again.

*Case 2:* Ernst Hoh, born in 1886, has gone through nine phases of depression with retardation, indecisiveness, feelings of insufficiency, and of manias, with elevated self-consciousness, busyness, pressure of speech, flight of ideas, and a somewhat querulous behavior. In the clinic he was typically manic with a certain irritability. The *father* suffered from depressed moods going back to his youth—which were not described more closely—and died of arteriosclerosis in sanatorium R.

*Case 3:* Illse May, born in 1912, had a depressive phase triggered reactively with anxious fears, ennui and inhibition, and a gay mania which led to spending much money. The *mother* (also a patient) had had repeated depressions with hysterical overtones. In the foreground stood complaints about lack of feeling and her own insufficiency, and also traces of fear.

*Case 4:* Sybille May, born in 1883, had earlier had repeated depressions, which are not better described. In 1939 she had a mania in the clinic that had only one querulous trait. Her *sister* had a sick mood in the menopause and made many self-accusations.

The cases listed up to here show nothing really atypical. If both poles had not been obvious in the clinical picture, then pure mania or pure melancholy could have been suspected. However, that is not very common and the cases which follow are different. In rare cases it could happen that a manic-depressive disease would be imitated by the accidental confluence in one person of a pure mania and a pure melancholy in a purely external connection. The family histories in two of my cases point to such an interpretation.

*Case 5:* Stefan Sud, born in 1900, who had only entered the clinic for observation, went through repeated mild manias with heightened self-consciousness, wealth of ideas, industriousness, and mild inhibitions. There were no psychoses in the family, but we found out the following about the parents: The *father* was always very industrious, co-founder of many societies, holder of many honorary positions, and ten times an honorable member. The *mother* is quiet, sensitive, and religious. After the marriage she went through a depression that went away after hydrotherapy.

*Case 6:* Anna Peis, born in 1904, went through several depressions accompanied by a tendency to suicide, self-accusations, indecisiveness, and a feeling of bodily heaviness, as well as manias of lighter natures, but which still contained all the symptoms. There is no inherited psychosis, but again a definite family history of temperaments in the family. A *brother* is mildly depressed, another *brother* is hypomanic. The mother is grave, conscientious, industrious. The father, on the other hand, is vivacious, humorous, and always sang a lot.

In these two cases, one could think that the father passed on the tendency toward pure mania and that the mother passed on the tendency to pure melancholy. On the other hand, it is also possible that the peculiarities of temperament derive from the manic-depressive disease which does not always emphasize both poles. In most of the cases of manic-depressive psychosis, the atypical symptomatology alone shows that it is not a mixture of pure mania and pure melancholy. This will become clearer through further case studies.

*Case 7:* Maria Ma, born in 1901, has experienced many depressions since the age twenty-five. In 1942 she was despairing for two days, then exhibited a pressure of speech in a mood at times depressed and at times gay. This state continued for a few days in the clinic, than a severe inhibition followed with nearly complete mutism. After a few days, a challenging jocularity was observed. Irritable, complaining, excited and stuporous days followed. Then a recognizable mixed-state began in which the patient lay in bed the whole day without moving, but displayed flight of ideas or incoherence in her broken, linguistic expressions. After further changes, the phase ended four months after it had started.

*Case 8:* Elise Jun, born in 1893, was anxious, perplexed, and retarded during her first illness in 1930. She also ran randomly around the room. In 1940 she became anxious again, developed simultaneously a pressure of motion, spoke, screamed, and ripped things up. The anxiety continued in the clinic, but was not accompanied by a confused retardation. A *brother* had had a stupor during the war in 1917 which was diagnosed as psychogenic. After admission to institution W., a mania followed, during which only a somewhat excited affect was observable.

In the last mentioned patient, apparently a depressive mood was mixed with a manic pressure to motion at home. In case 7, a continual change and mixing of the symptoms is easily noticeable.

In the following cases, the atypicality is particularly noticeable in the affect. The emotions are a multilevel construction, and can therefore be disturbed in varied ways. In the manic and melancholic basic syndromes, the emotions are pushed fully toward one or the other side; in atypical forms, one or the other type of feeling can be more strongly affected. Then, the previously mentioned clinical pictures begin to arise which are reminiscent of the pure forms.

*Case 9:* Dorothy Drus, born in 1890, experienced a "nervous exhaustion" in 1926–27. In 1938 she was in the nerve clinic G. for 4 weeks for a neurasthenic-depressive state. In 1940 she offered a similar picture in the Frankfurt clinic; with her mood being more labile than depressive. Suicide attempts had a demonstrative character. Then a strong fear replaced the lability, and it became even stronger in the institute G., to which she was moved. The patient cried, complained, begged, was full of despair and tried to pull her jacket closed tight around her neck. After an ebbing of the fear, she asked, uniformly and urgingly, for her release. In May she recovered suddenly and was released in a happy mood. In 1918 the *father* had had a mania that was often interrupted by a crying mood but which had no other atypical characteristics.

If one had only observed this patient during her excitement, then one would have had to judge her illness to be a harried depression, due to her uniform expressions of fear. Due to the very different picture in the rest of the course, this diagnosis must be rejected. The very varied picture reveals the correction to manic-depessive disease; this is also supported by the father's mania.

*Case 10:* Anna Hell, born in 1914, in 1931 experienced anxious excitement, hallucinated ("the walls say I am a thief"), and was difficult to diagnose. Then she became increasingly stuporous and was finally fully motionless. There was no longer depressive affect, and at times the patient would even smile. After a quarter of a year, she recovered. In 1937 she returned to the clinic and was now hypomanic, somewhat forward, without distance. In 1939 she was again depressive and complained in the clinic particularly of an alienation of feeling: she had no more heart and no more feelings, the blossoming of nature meant nothing more to her, everything was so odorless. Her child and her husband seemed strange to her. She felt inwardly changed, that she is another. There was also a feeling of inability to accomplish anything. After six weeks the depression disappeared, and she was then hypomanic again. The *daughter of a sister of the mother* went through several hyperkinetic and stuporous phases, but later became insipid (dull, dumb, flat).

The hereditary connection of the relative's psychosis with the psychosis of the subject is uncertain; the relationship is also not close. The subject, after short hallucinatory fear, went through an akinetic state, no longer recognizable as depressive. Later I will speak of similar states. Here I am interested in the second phase which was shaped fully differently and which reveals the multifacetedness of the manic-depressive disease. In this phase the alienation phenomena were in the foreground. In the basic melancholic system too, the complaint about a subjectively felt cooling of the emotions is often heard. With the subject, however, the complaint that she had lost her normal emotions was the only depressive symptom. There was also a feeling of insufficiency. According to the picture in the "impoverished-second phase," one would assume a disease of "non-participatory-participation depression," as will be better seen from the presentation of the pure forms. The first phase and the hypomanic interphases, show, however, that the "alienation depression" was only one episode within a manic-depressive disease.

*Case 11:* Otto Wil, born in 1907, developed feelings of anxiety in 1938 and was brought unconscious to the clinic after a suicide attempt with sleeping pills. Here he was very inhibited, indecisive, made self-accusations, complained of a lack of interest and claimed he would never get well. There were also hypochrondriacal complaints: his body was out of line, everything hung to the right, hands and feet would fall asleep, the legs jerked, there was something wrong in the lower abdomen, the intestine has ceased functioning. After a period of six months, the above phenomena disappeared without the general picture being essentially changed. In 1926 a *sister* made a reactive suicide attempt, but became depressive in 1934 for no reason and was brought to institute M. She displayed certain hypochondriacal traits and was partially inhibited and partially anxiously excited. After one year she became manic with a pressure of speech and flight of ideas, and during the decline of the mania she displayed a temporary muteness with forced motion. Then she became depressive again for a short period before finally recovering. A *second sister* arrived in 1943 in the Frankfurt clinic with a depression, which at first appeared with inhibition, dislike of work, hopelessness, anxious fears, and indeterminate bodily complaints. After a few weeks it turned into a severe excitation, during which the patient cried inarticulately, struck out aimlessly with her fists, and spoke of poisoning. The mood was fearful but also with annoyed components. She died in the excitation during the appearance of a bronchopneumonia. The *father* of the three patients entered institution M at the age of 53 due to a depression. With mild oscillations, he remained stuporous for 2 years, moaned, but despite the severe inhibition, he rubbed his hands together in a lacing motion and bemoaned himself. Finally the psychosis healed. A quiet, shy manner remained. He died in 1933 without any further psychic illness.

The last mentioned patient offered at age 53 the picture I have described as characteristic for the "involutional depression," which often develops into the "congealing regressive depression," that is, an anxious-stuporous behavior, in which, despite the sever inhibition, there is an unrest in the hands, often accompanied by moans.

In my sample of patients with phasic psychoses, I found my position upheld that this anxious-stuporous behavior form of depression almost only occurs in persons of climacteric age. However, the development into a congealing regressive depression need not follow. In the few cases out of other age groups, it was a question of complications through arteriosclerosis or fever. The lacinglike motion here could be a delirious sign. I cannot decide if the climacterium can have a similar effect in the sense of an exogenous noxa. With the patient described above, the psychoses of his three children speak for his also being a manic-depressive disease, and not an involuntial depression.

The mixed-state of the first sister of the subject is of interest. In this state she offered a mute restlessness, most likely an excitation with thought inhibition. Immediately beforehand, she had experienced excitation, flight of ideas, and further tendency toward depression followed. The second sister, too, seemed to have a mixed-state during her excitation, but with the sudden switch from a previously inhibited constitution, it is more probable that the excitement was of a manic nature, while the affect still remained depressive. Both sisters had only mild hypochondriacal traits. Because they were very marked in the subject, I have described the relations here.

These peculiar complaints that have been presented here will occupy us later with the hypochondriacal depressions. If there were not the simultaneous presence of melancholic symptoms in the form of inhibition, indecisiveness, self-accusations and hopelessness, then one would have to suspect a hypochondriacal depression. There were no fears about bodily well-being—that is also called hypochondriacal—but actual bodily misperceptions, which are directly reminiscent of the hypochondriacal hallucinations of schizophrenics. The total picture of the depression also speaks for its really being a manic-depressive illness. This confirmation is also found in the relatives who display manic traits. Depression had, therefore, once again attacked a certain type of feeling and thus created the similarity with a pure form.

*Case 12:* Marta Emm was born in 1907 and years before her first severe diseases she had depressions in which she spoke of death. In 1936 she entered the clinic, was somewhat fearful, complained, and expressed self-accusations that she was a bad mother. Further, peculiar bodily complaints appeared—she had the feeling that her head was too large and the hands too long; the stomach had changed; the intestines no longer worked. But not only her body, the whole environment seemed changed, everything seemed larger. She also had the feeling that people

were laughing at her. After four weeks she was released, recovered. In 1940 she became ill again. This time feelings of insufficiency appeared, a feeling of incapability of dealing with anything. There was a hopelessness that she would never get well. She only lacked the courage for suicide. She lacked motivation, was insipid, and inhibited. There were also the misperceptions and alienations; she had stomach cramps as if she had to vomit; a feeling in her head that it was growing larger. Everything external seemed strange to her, she felt as if she no longer belonged to humanity. She could no longer grasp ideas correctly. After four months she was improved and released. A *sister* of hers was in a clinic six times. She offered generally a melancholy picture with inhibition, occasionally she would have anxious excitations, and alienation phenomena were sometimes recognizable. She had short manic excitations twice, one a manic stupor during which she spoke no word but laughed silently and tried to put on jewelry. The *father* had a labile mood and was in this clinic for a short period in 1930 due to a reactive depression.

In Marta Emm we find a connection between phenomena of hypochondria and alienation. The latter are different, however, than those in case 10. There we found a subjectively perceived cooling of feeling while here there is a feeling of strangeness toward the patient's own body and external events. We will see in the pure forms that this form of alienation is a frequent accompanying phenomenon of hypochondriacal depression. This connection can also be initiated by manic-depressive insanity. The melancholic symptoms, the inhibition, and the lack of energy with feelings of inadequacy all also point to its not being a true hypochondriacal depression, which is associated characteristically with agitation. The illness of the sister also confirms the presence of a manic-depressive disease. Her mixed state of a manic stupor is worthy of interest.

*Case 13:* Hedwig Schwab, born in 1905, became melancholic in 1932, and believed she was being spied on, that the newspapers were writing about her, that people were playing games with her. She entered the clinic after a suicide attempt. Here she was inhibited, laconic, spoke in a monotonous voice about her suicide, "I was so dumb, no one dies at all; it just never happens that people die." She confirmed her self-references but she complained more about hypochondriacal problems. She felt as if worms were crawling around in her body, she must have a child in her stomach, she no longer has a real body, her stomach is missing. She was released seven months later in a happy but equable mood. In 1936 she became ill again, was more complaining than deeply depressive and often had a smiling expression. In the intelligence test, she easily fell into flight of ideas. This time no hypochondriacal ideas were present, but rather many ideas of self-reference. People were maligning her and, although she had killed no one, someone wanted to kill her and put

poison in her food. After four months she was again happy and equalized and could be released. In 1938 she entered the clinic for a third time. She was very labile, "cried and laughed simultaneously," expressed her ideas of reference, such as, the people singing in the street were singing about her, and so on. Her whole environment seemed to her to be changed. After three months this phase ended completely. The *mother* had gone through many depressions, and, in institution I, exhibited a manic state with paranoid-querulous traits.

In the first depression, which went deepest, hypochondriacal ideas were present, which were again connected with alienation phenomena. Schwab also believed her stomach was missing and that neither she nor other people could die. In the first depression and more markedly in the following two, ideas of reference became apparent and dominate the picture to a large extent. We will return to the relationship between anxiety and the ideas of reference in relation to the anxiety psychoses. When the anxiety is minor and is not particularly recognizable externally, then one thinks of the suspicious depression which we will examine under the pure forms. Thus again, a pure form is initiated by the manic-depressive illness. The similarity is, of course, only approximate. In the first phase, the nihilistic hypochondriacal coloration goes in a fully different direction, and in both phases, the varying affect cannot be overseen. Once there was a definite mixed state with flight of ideas and a depressive basic mood. The bipolarity of the psychosis was even more clear in the mother.

*Case 14:* Luise Zieg, born in 1906, went through mild depressions in 1925, 1928 and 1934, and entered the clinic in 1938 due to a new depression. In between, particularly in 1929, she had mild manias. In depression she was somewhat inhibited, indecisive, expressed self-accusations and feelings of inadequacy. In each depression there were ruminations. She tortured herself with all kinds of ideas, which did bear relations to her depressive ideas, but whose baselessness she recognized. Thus she always had the forced throught that she would do something to herself or would do something bad. With the disappearance of the depression, the forced ideas also disappeared. There is no inherited psychoses, that is, in her family there were no psychotics, only psychopaths, hypomanic-hypomelancholic personalities, and the abnormality of one *sister*. She was very conscientious and suffered chronically from compulsive ideas. She always had to think about the dangers for the lives of her children and imagine that she had missed something.

The subject Zieg is listed for the anancastic coloration of her depression. There are often hints of compulsive phenomena in depressions, particularly in inhibited forms with indecisiveness, but they rarely come so far forward as to dominate the picture. This case shows how the anancastic coloration should be explained, since the sister is a clearly

compulsive neurotic personality. A markedly anancastic coloration of depression probably only appears when the manic-depressive tendency is accompanied by a compulsive neurotic tendency. It is then strengthened by the depression and the indecisiveness. It is, however, something altogether different when compulsive neurotics become depressed due to a reaction to their disease phenomena which they try to escape in vain. In this form of "compulsive depression," one recognizes—as with reactive depressions—that the depression is fully directed at its cause. Melancholies of compulsive neurotic coloration, on the other hand, show their depression fully independently of the compulsive thoughts.

*Case 15:* Wilhelmire Trit, born in 1924, was in a clinic in 1939 and 1940, and in between was in the Institution M. She changed often, was partially anxious-depressive with ideas of reference, partially manic with euphoria, excessive speech and flight of ideas. During both clinical stays she displayed states of somewhat imaginative superciliousness without excitement. Thus she claimed, among other things, she had police work to do especially assigned to her by Hitler, to whom she had sworn loyalty. She only need to click her tongue and a police officer would come and help her. She had a secret mission. Her mother had been in Institution G in 1917–1918, after having spent an excited period at home in 1914. In the institute she sang, danced, and screamed. Then she offered a playful restlessness without linguistic expressions, let herself fall out of bed, climbed on the window and smiled to everyone. Then this restlessness disappeared while the mood remained gay and had a so-called "markedly dull-witted, silly trait." Then the patient became depressive again, then manic, and finally fully equalized, so that she could be released.

After having seen several pictures of manic-depressive illness that resembled pure depressions, here we have euphoric phases that are reminiscent of pure euphorias. The somewhat imaginative-expansive states of the subject resemble the imaginative euphorias, as will be seen later. The basically ecstatic mood is not masked, but still recognizable, particularly in the way ideas appear. The festiveness with which the ideas are expressed divides the megalomania of the subject from the playful megalomania of the manias and points toward a single type of feelings being emphasized.

The *mother* of the subject possibly had another kind of feeling attached for a period. Her simple friendliness and its contentlessness, which gives it a silly appearance, is reminiscent of the "unproductive euphoria," which we will examine with the pure forms. In any case, some states of simple happiness of the manic-depressive illness resemble the unproductive euphorias.

*Case 16:* Valentin Wak, born in 1914, went through a depression in 1937 with inhibition, feelings of inadequacy, and sick fears. One half year

after his recovery he became ill again and was temporarily anxious with ideas of reference. Then he became euphoric and entered the clinic. Here he demanded 100,000 men in order to occupy Offenbach, claimed to be the richest man from O., to have taken over control of innumerable firms and to have written 25,000 business letters. He claims to have been in the Foreign Legion, to have been a pilot in the Spanish War, and to have crashed three times. The excitation increased and soon developed into a confused manic state with considerable hyperkinesis. After entering Institution W, he varied between manic periods and shorter depressive periods. In depression he was at times stuporous, in the mania there were expansive and hyperkinetic traits. About a year-and-a-half after the start of his second phase, he calmed down. He returned later to the clinic and was clearly hypomanic.

I have presented this subject, because in one, albeit short, episode of his manic-depressive disease, he presented an expansive-confabulatory picture, as we will meet again with the pure forms.

*Case 17:* Wilhelm Goy, born in 1865, became ill in 1917 for the first time. He became excited, accused other people of embezzlement, gave statements to the police, wanted to establish justice everywhere, and wrote long letters. He messed up his apartment and claimed thieves had been there. He also yelled at his wife and hit her. In the clinic he accused his wife of adultery and insulted the doctors. He heard a nurse saying he was crazy. Generally he was annoyed, but also periodically gay and generally very talkative. After two months he calmed down and could be released. He remained healthy until 1941, and then he became excited again; he was for justice and the "Führer." He developed an excessive speech and lively gestures, and would often shift from the gay mood into tears. There were no probable signs of senile dementia or arteriosclerosis. He entered Institution H and remained hypomanic until his death in 1943. His *daughter* was in Institution K in 1918. She was at first manic, then depressive, then she displayed a mixed state with anxious excitation and flight of ideas. She became manic again with expansive phenomena: she was the lord over war and the German Reich. She eventually calmed down and was released three-quarters of a year after her admission. In 1926 she spent four weeks in the Frankfurt Clinic and complained of many problems which, in connection with a lachrymose whining were interpreted to be symptoms of hysteria. An imbecility was proven. In 1936 she returned to the clinic and was confused-excited. After three weeks she was moved to Institution H where she varied between normal and manic periods. She died in the institution in 1937.

This patient is of interest because of her paranoid traits. It is known that people suffering manias are often querulous and frequently have a markedly paranoid coloration. Generally it can then be shown that the

mood is not purely elevated, but can easily change into irritation. The mood is often also found in many manias that are not at all paranoid or querulous. Thus it alone cannot be responsible for the paranoid trait. If there are fixed delusions which oppose the manic flexibility, then there must be some other cause. Often it is a paranoid nature which had always been present, but which breaks out due to the manic irritability, and at times it is a mixed state. We have seen that there are distrusting depressions in which ideas of reference are markedly apparent. If a depression of this form combines with manic activity, then a paranoid state can undoubtedly develop. The ill humor does not have to be any deeper, since the distrustful depressions are generally not very deep and are even more insipid in the content of manic flexibility. However, one can go further theoretically. If the different types of feelings do not have to be similarly distorted, then it is also conceivable that they can be distorted in different directions; that there are mixed states within the affect itself. Thus a distrustful-depressive mood could be fully covered by a euphoric mood of the other types of feelings. There then would be distrustful reinterpretations despite the otherwise euphoric mood. Specht is thinking of something similar when speaking of a mixed affect in paranoia.

*Case 18:* Lina Krapp has, since 1910, experienced four depressive and many manic phases. The latter had mild degrees of paranoid aspects, at times leading to severe confusion. A *sister* had been mentally ill twice in an institution and recovered both times. There was no medical history.

*Case 19:* Bernhard Tab, born in 1889, was manic in 1934 and in 1939, after anxious introductory phases. His was a mania with an intermittently apparent lability toward the depressive side. A *sister* had a periodic mania that began with severe confusion and finally became chronic. There were also hyperkinetic traits in the excitement and transient depressive states in between. The mother had also had periodic confused manias with the misrecognition of individuals and once she was temporarily depressed.

*Case 20:* Therse Fick, born in 1912, went through a confused mania, beginning with the misrecognition of people and mood lability, and a melancholy with strong inhibitions, particularly thought inhibitions. The *mother's mother* had several melancholies with inhibition, indecisiveness and a confused mania. Two *sisters of this diseased grandmother* were also temporarily mentally ill. There was no medical history available for one, the other had a mania with severe confusion. Of the latter, two daughters were temporarily mentally ill: one had an inhibited depression with some anxiety; the other had two manias with a fully incoherent thought process and the misrecognition of people.

The confused manias appear in marked form almost only in women. The subject Tab (Case 19) was simply manic, while his female relatives went through confused manias. The confusion is determined through the incoherence of the thought process, which can be understood as the highest degree of flight of ideas. We will find it to be otherwise in the confusion psychosis. Generally, there is simultaneously a high degree of force to speech, since the excitement is much more related to a thought disorder than other excitements. The mood in confused manias is often no longer purely euphoric, but rather labile with a tendency to temporarily slip into depression. Perhaps this mood lability is not only an expression of manic-depressive bipolarity, but is rather partially comparable to the lability of symptomatic neurasthenias and psychoses; hence a sign that the manic-depressive psychosis has reached an endotoxic level. At times, as the following case shows, sensuous misperceptions accompany the confusion. There, too, one could see an endotoxic effect of the gradually heightened disease process, particularly because the consciousness is somewhat unclear in the height of the disease.

*Case 21:* Paula Schul, born in 1910, became ill in 1927 with a depression involving religious scruples and thoughts of sin. She believed she was lost and destined for hell. Soon an excitement developed with flight of ideas and excessive speech, while the mood remained depressive and ideas of sin continued. Then the mood too became manic, the excitement increased. The thought process became incoherent. She heard, as she said and as she confirmed after her recovery, many voices; she perceived a smell of death and had visual experiences. Thus she saw a golden beam on the wall, she saw her dead cousin dressed in white, and her father confessor sitting on her bed. Recovery occurred after oscillations, so that Schul was released after a half year. One year later she returned for an examination. She had no more psychic abnormalities.

For the patient Schul, who had visual hallucinations, we must consider the possibility of an unclear consciousness, even if only of a low degree.

In the listed cases of confused manias, the entire picture, including the familial evidence, leaves no doubt about their being manic-depressive diseases. But the more severe the confusion becomes, and the more misrecognition of persons and hallucinations occur, the more one has to ask the question of whether the diagnosis should be confused mania or if it should be one of confusion psychosis, which we will deal with extensively later.

The differential diagnosis is often more difficult in stuporous states. The perplexed stupor is characteristic for the inhibited confusion; in the manic-depressive disease, on the other hand, a particularly strong inhibition can lead to a depressive stupor.

*Case 22:* Emilie Rauch, born 1890, was in the clinic eight times due to depression. In between there were short manic states with excitement and annoyance. In depression she had some ideas of reference, but otherwise little content. The inhibition was very marked, and became stronger in all phases, approaching stupor. The patient seemed somewhat confused, but her facial expression revealed her depressive mood. The *mother's sister* was institutionalized six times, four times due to a mania with no essential atypicalities, and twice due to melancholy which was accompanied by moderate inhibition and few contents.

*Case 23:* Lina Wof, born in 1886, went through a mania in 1939, with some querulous traits. Eleven years earlier she had been in the clinic due to a depression—then she was anxious, at first somewhat excited, was afraid of being killed, moaned uniformly, but then became inhibited, finally stuporous, with an anxious-confused facial expression. The *sister* experienced her first depression at 17, at 30 she went through a confused mania in the nerve clinic H, and at 34 she went through a depression, at first with anxious excitement, then with severe inhibition, nearly a stupor. At 40 and at 44 she had mixed states in which pressure of speech and flight of ideas combined with a labile mood oscillating between gaiety and anxiety.

The last mentioned patient shows the approach of melancholic inhibition to stupor. Since the depression considerably outweighed the confusion, the correct diagnosis could be made. The other course of the cases and the evidence from the relatives confirm the presence of a manic-depressive disease, on the other hand, a particularly strong both manias and depressions, Wof's sister had manias, depressions, and mixed states. In one depression her inhibition nearly became stuporous, otherwise this peculiarity does not appear among the diseased relatives.

The confused stupor, as we will later see, is characterized by ideas of reference and meaning. These ideas cannot be determined during the stupor, but can only be asked about later. With less severe inhibitions, they can appear in the psychosis. Confusion dominated the psychosis then as well. The manic-depressive disease may also offer differential diagnostic difficulties in relation to the confusion psychosis, as the following case shows.

*Case 24:* Ruth Geit, born in 1913, went through short, depressive illnesses in 1937 and 1941 and became ill again in 1942. She was very confused, and asked, "What is happening with me?" She wanted to leave her apartment in her pajamas to see where she really was, everything seemed changed to her. She also felt observed, referred conversations to herself, heard accusations that she did not care enough for her children. She also had other self-accusations. When the doctor came, she claimed that he could not be a doctor. In the clinic she hardly expressed these ideas anymore she was so inhibited, but they seemed to

continue to exist and to be seen in her confused facial expression. She was now fully stuporous, gave no answers. Then she became increasingly anxious, ran restlessly around the ward and heard voices with anxious contents, "you're going to jail, you must die immediately," Four weeks after admission and about four months after the onset of the phase, the mood disappeared and changed into gaiety, accompanied by excessive speech, and flight of ideas with jokingly aggressive remarks. Then recovery set in. The *father,* Friedrich Geit (also a subject), was depressive almost annually since 1918, infrequently annoyed and aroused. He was in the Frankfurt Clinic in 1919, 1941 and 1946. Each time he was somewhat anxious, easily agitated, suffered from fears, had ideas of reference and had to periodically compulsively use ugly expressions. The depressions always disappeared fairly soon. His *mother* had a depression in 1890 and in 1891 entered Institution I. She offered the picture of an agitated depression with anxious restlessness and self-accusations. She died of Addison's disease one half year after admission. The clinical picture imitated a "harried depression," as we have already seen in case 9.

The ideas of reference and meaning present in the subject are often considered typical for schizophrenia. I will return to that in reference to the anxiety psychosis and the confusion psychosis. This case should not only show that that which we find in the confused stupor is also present in the manic-depressive disease. The assumption of a manic-depressive psychosis—and not of a confusion—was based in the severely depressive mood recognizable behind all ideas and the depressive states without the ideas. There was also a short manic state, as well. The family history confirms the diagnosis of manic-depressive disease.

*Case 25:* Rosa Ost, born in 1879, was in the Frankfurt Clinic six times and also went through milder phases at home. She was partially typically manic or typically melancholic but often atypical traits appeared, particularly, during mania she would often become confused with the misrecognition of persons, and in depression she became stuporous. Often the two states followed each other quickly. A *brother* went through a depression in 1921, which was characterized by inhibition and indecisiveness. Later he frequently had milder depressions. In 1919 a sister was anxiously excited, in 1923 stuporous-depressive, in 1930 severely inhibited and depressive, and in 1939 partially anxious and partially irritable and arrogant, in the sense of a manic-depressive mixed-state.

In this subject, both the manic and melancholic phases of the picture of the state show transitions to the confusion psychosis, since during the former she often becomes confused, and during the latter, stuporous. A propensity to severe inhibition appears to be present in the siblings as well. I interpret confusion and stupor in the manic-depressive disease as an expression for the psychosis being very deep. Similarly as with the

picture of the confusion psychosis, the manic-depressive disease can resemble the picture of the motility psychosis, as these cases should show.

*Case 26:* Maria Bat, born in 1892, was in the Frankfurt Clinic eight times for manias, which were often followed by inhibited-depressive swings. In her excitement she was generally confused and displayed hyperkinetic traits. For example, she danced, threw herself on the bed, stood up again, ran through the room, shook her head, so that her hair flew about, beat rhythmically on the wall, spread out her arms and made many other expressive movements. A *brother* experienced manic and depressive phases at home and in 1915, in the Frankfurt Clinic, offered the picture of a mania with expansive-confabulatory traits.

*Case 27:* Kath Hof, born in 1877, went through melancholies with inhibitions and ideas of sin in 1928 and 1935, and in 1937 a mixed state in which a depressive mood with self-accusations was accompanied by a confused excitement. In 1933 and 1940, she had manias, accompanied by confusion and, above all, hyperkinetic phenomena, which, as is stated once, produced "the picture of a motility psychosis." She jumped around, gesticulated, made gymnastic movements, and swung her arms. A *sister* went through a confused mania in 1932 in Institution L, with many hyperkinetic traits and frequent, intermittent mild depressive-inhibited states. A *second sister* had, in 1927 in the Frankfurt Clinic, a confused mania with hints of suggestion ideas and then remained chronically hypomanic with irritability and self-references.

The two patients last mentioned went through excitations greater than manic activity, which took on the form of hyperkinesis as we will see with the motility psychosis. The rest of the clinical picture of the subjects and their relatives show that there is no reason to doubt the diagnosis of manic-depressive disease.

*Case 28:* Hildegard Mor, born in 1895, had been in Institution I ten times since 1922 and in the Frankfurt Clinic once in 1940. She went through manias that were often somewhat confused; mixed states, in which a depressive mood combined with confused excitation, or gaiety and flight of ideas combined with psychomotor inhibition; and depressions with stuporous behavior. Then she was fully rigid, resistant, had to be fed with a tube and stayed in the same position for hours. In 1928, the *mother* went through a mania of fourteen days, with no more complete description. In 1933 she had a depression, which in Institution I changed quickly through an irritable state into a mania and was associated with confusion and ideas of grandeur. A *sister of the mother* had a manic excitation in 1927 in Institution W, which is not described, and in 1928 in Institution M, a melancholy with self-accusations, ideas of impoverishment, and mild anxious excitements.

In this subject, the stupor accompanying the depression is not comparable to the perplexed stupor of the confusion psychosis but rather, to the akinesis of the motility psychosis. This is seen in the severity of the impoverishment of movement, which made tube feeding temporarily necessary, and the stiffening of expressive movements and the maintenance of positions. According to these symptoms, the loss of motion is not due to thought inhibitions and growing indecision, as in the perplexed stupor, but rather to a disorder in the psychomotor mechanism. The rest of the clinical pictures shows again that it is the manic-depression disease, and this is confirmed by the picture of the relatives. Thus the manic-depressive disease can extend over into the picture of the akinetic motility psychosis.

A case (#13) was presented above, in which an anxious, basic mood combined with ideas of reference, so that the syndrome of "suspicious depression" arose. As was mentioned there, one is reminded of the "paranoid anxiety psychosis," i. e., one pole of the anxiety-happiness psychosis, when the anxiety is particularly salient. Such forms also appear in the manic-depressive disease.

*Case 29:* Hedwig Schleub, born in 1906, became ill in 1942, believed she was being followed by the Gestapo and claimed her boarder was working with them. She believed the people on the street were observing her, and that a baby carriage in the steet was only a observation place from which to spy on her. She reinterpreted every noise in the apartment and looked for the Gestapo's "bug." She also heard voices and believed to have heard her sister. In the clinic she confirmed these ideas, but also produced self-accusations that she had falsely signed bills and that she was not worthy of her girl friend. At first she was rather motionless and perplexed, then she became anxiously excited, begged for help, and clung to people. After a few weeks, the anxiety decreased and was replaced by a hypomanic state. After a relapse into anxiety, again with ideas of reference, a hypomanic nature remained established. The patient was lively, talkative, laughed a lot and became querulous when she was not immediately released. A *mother's sister* had been in the Frankfurt Clinic in 1917 where she was depressive, lachrymose, involved with hypochondriacal ideas that a nerve was strained, that her emotional nerves were gone, that her head felt as if it were bleeding inside, and that the heart was burning. She was periodically anxiously excited. She became healthy and did not become ill again.

In Schleub, the ideas of reference with an anxious basic mood played a large role. There were, however, also melancholic symptoms. The clearly hypomanic state probably was the reason for the diagnosis of manic-depressive disease. The diagnosis can be considered confirmed by the depression of the mother's sister who had no ideas of reference.

*Case 30:* Josef Gopf, born in 1871, was in the Frankfurt Clinic five times between 1923 and 1940. He had only mild depressive moods, but marked manias, which were partially typical, and partially accompanied by ideas of happiness (blessing). Thus, in 1923 he wanted to go through the world as an evangelist; he claimed he was a spirit and needed nothing to eat. A *sister* spent 1937 in Institution L in a stuporous depression and later committed suicide.

*Case 31:* Minna Hen, born in 1894, was in the Frankfurt Clinic in 1938, 1940, and 1942. She had depressions, accompanied by self-accusations and, occasionally, by severe anxiety. She also had manias which were generally typical but which occasionally had ecstatic traits. She would then declaim religious sayings in a pathetic manner. In 1934 in the Frankfurt Clinic, a *sister* went through a mild melancholy of neurasthenic coloration.

In these cases, the depressions have different colorations, but remain within the margins of manic-depressive disease. In the mania, on the other hand, characteristics appear associated with the happiness psychosis. In further cases we will see that both poles of the manic-depressive disease can be reminiscent of the anxiety-happiness psychosis.

*Case 32:* Elise Ew, born in 1907, presented a very variable picture in the Frankfurt Clinic in 1939. She was partially typically manic, partially ecstatic, and partially anxious with ideas of reference and poisoning, as well as olfactory hallucinations. The *mother* went through a mild melancholy with self-accusations and mild anxiety in 1918 in Institution M. A *sister of the father* had depressions in 1927 and 1928, accompanied by severe inhibition, indecision, ideas of impoverishment, and fears.

In these last cases we find paranoid traits, ideas of self-reference and perceptual illusions in the depression, and ideas of happiness in the mania, but in no case so much as to doubt the diagnosis of manic-depressive disease.

In a few cases, which were counted as manic-depressive disease, the post examination history of the subject or the familial picture showed that the assumption of a curable, phasic illness had been possibly incorrect. Such diagnostic difficulties between the manic-depressive disease and the schizophrenias are very rare, if the concepts are strictly interpreted.

## SUMMARY

Thus I have prescaled the symptomatology of the manic-depressive disease and the range of the symptoms. It can be essentially grasped if one begins with the manic and the melancholic basic syndrome, and understands atypical forms to be mixed states, partial states, which

occasionally mimic pure euphorias and depressions, or an extension of the symptomatology into that of the other bipolar forms. Certain similarities to the cycloid psychosis can be understood as an increase in degree of severity—thus confused manic and stuporous depressive pictures. Mixed states, severity increases, and traits of the anxiety-happiness psychosis, confusion psychosis, and motility psychosis thus make up the atypicalities. The picture will be filled out by the statistical results.

Certain points of view, however, which could be of clinical importance, we neither discussed here nor there, since I have nothing new to add to them. I have no new results on the length of the separate phases which, for manias, is considered to be one quarter to one half a year, and, for melancholies, one half to three quarters of a year. My results also confirmed the fact that phases can exceptionally last only hours or that they can continues for years and, in fact, become chronic. I have ignored the normal temperaments associated with the manic-depressive disease, not because I saw no possibility of adding to what is known, but because special investigations were planned.[5,6,7] It is clear that hypomanic, hypomelancholic, and cyclothymic temperaments are present in the families of the patients as kinds of dilutions of mania, melancholy, and the manic-depressive mixed state. Beyond this, it is of great interest how many similarities and differences exist to the temperaments associated with the other bipolar psychoses.

[5]Leonhard, K., Korff, I., Schulz, H., "Die temperament in den familien der monopolaren und bipolaren phasischen," *Psychosen psychiat. Neurol.* 143: 416–434, 1962.

[6]Leonhard, K., "Die prapsychetische temperamente bei den monopolaren und bipolaren phasischen," *Psychosen Psychiat. Neurol.* 146: 105–115, 1963.

[7]Leonard, K., et. al., *Normale und abnorme Personlichkeiten,* Berlin, Verlag Volk Psychoses," *Psych. Neur. Med. Psychol.* 15: 203–206, 1963.

# CHAPTER 2.

# PURE MELANCHOLY AND PURE MANIA

The full import of the polymorphousness of the manic-depressive disease only becomes clear when the pure forms of the disease with their stable symptomatic pictures are discussed, particularly the pure melancholy and the pure mania. Some points have already been touched upon, since the variable syndromes of the manic-depressive disease occasionally mimic the pure forms.

The "basic syndromes," with which the discussion of the manic-depressive disease began, also represent the pictures of pure melancholy and pure mania. Thus the functional area that is affected by those forms is also the center of activity for the manic-depressive disease.

## PURE MELANCHOLY

The first and most cardinal symptom of melancholy is the depressed mood. There can be an implicit anxiety, but more often apathy gives the depression its characteristic form. The indifferent behavior could make the patients seem "sad," but this concept, reflecting a higher mental state of mind, does not correctly describe this melancholic depression; it appears to be more a physical decline of the feelings and does not correspond to mental sadness. When K. Schneider speaks of "vital" depression, one can think particularly of pure melancholy. Depression is not to be described with terms of normal psychology; only the somewhat general term of "depressedness" can be understood in the sense of this

motiveless (i. e. incomprehensible by the normal mentality) mood. In mild cases, the patients appear almost more apathetic than actually depressive; in severe cases, however, deep suffering develops. We will later find anxiety to be a special component of the emotional disorders of the pure depressions. It is certainly also included in melancholic depression but is generally covered up by other emotional components and by inhibition. It is often subjectively indicated. High degrees of anxiety, however, prohibit the diagnosis of pure melancholy, particularly, if an anxiety leading to severe reactions does not occur here.

The second cardinal symptom is psychomotor inhibition which accompanies the depression of melancholy, and can be of higher or lower degrees, generally running parallel to the depth of the mood. In pure melancholy, the inhibition never reaches to stupor. A slow speech pattern, a monotonous and soft voice, a somewhat slow carrying out of all motions, a poverty of expressive movements, all these characterize melancholic inhibition. This does not restrict the ability to carry out daily functions; the patient can feed himself, as long as an absence of hunger does not occur, he can go to the bathroom himself, and the ability to speak is not lost. Stuporous depressions are common in the margins of manic-depressive disease; they also appear in other bipolar psychoses, but not in the margins of pure forms.

Thought inhibitions, the third cardinal symptom of pure melancholy, become recognizable when the patient is asked questions which require a certain, albeit small, amount of thought. The reaction times are markedly longer than what could be explained by a psychomotor inhibition alone. While the simplest questions, about personal data for example, can still be answered quickly, it takes a disproportionately long time to receive answers about data not immediately available or about somewhat complicated relationships from the past. Often one finds that difficult questions are not even understood the first time. Simple questions in intelligence tests, which normally require hardly any thought, can be answered quickly. But depending on intelligence and educational level and the degree of severity, the thought inhibition becomes apparent and leads to noticeably long reaction times. Thought inhibition, too, does not assume high degrees in pure melancholy; here we find no mutism developing from thought inhibition.

Other symptoms of the pure melancholy can be deduced from the three cardinal symptoms. Thought inhibition and the psychomotor inhibition make decisiveness more difficult. Thus, indecision is an important symptom of pure melancholy. In milder cases it is often not easy to demonstrate the presence of an inhibition, but at least the subjectively perceived increase in the difficulty of making decisions points out the inhibition. While the patient may be able to operate somewhat freely in an already moving process, he or she has a hard time beginning the process itself. Even when it is not very significant, these individuals do not come to a decision, that is, the last act of the