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Excerpt
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Part 1

**Clinical, diagnostic, and therapeutic
aspects of bipolar disorders**

Introduction

Bipolar disorders, classically known as “manic–depressive psychosis,” is a serious, chronic, and relapsing mental disorder. Despite the growing efficacy of available pharmacological tools, bipolar-affective disorders have continued to be a significant source of morbidity and mortality, doing serious harm to the quality of life of sufferers. They are the sixth cause of disability worldwide (López and Murray, 1998) and, serious and chronic as they are, represent a heavy financial and social burden – both direct (hospitalizations and consumption of medical resources) and indirect (constant days missed from work and loss of productivity) (Wyatt and Henter, 1995; Goetzl et al., 2003).

The incidence of bipolar disorders amounts to approximately 4% of the adult population (Hirschfeld et al., 2003), but may reach 6.5% of the population at large if minor and atypical forms are included (Angst, 1995). The consequences of the disorder and its subsequent relapses for the individual and for family members, combined with the high risk of mortality by suicide (Vieta et al., 1992, 1997a, b, c; Tsai et al., 2002) suggest that a multiple therapeutic effort must be made, going beyond while at the same time supportive of drug therapy.

Bipolar disorders through history

The first references to mania and melancholia go back to Araeteus of Capadocia in the second century BC. For centuries, the term “mania” was used to refer to agitation syndromes, whatever their origin. The first attempts to find a solid biological substrate for mania arose with the liberalization of cadaver dissection and the advent of the anatomical–clinical methods. However, it was not until the nineteenth century that the concepts of mania and depression were linked conceptually and clinically through the first detailed descriptions of the *folie circulaire* by Falret and the *folie à double forme* by Baillarger – both the conditions characterized by states of excitation, sadness, and lucid intervals of variable duration. The introduction of recurrence and the cyclicity to descriptions of the disorder is one of the breakthrough moments in the history of psychiatry, and even discussion of the authorship of the concept makes quite an interesting story (Pichot, 1995). Falret and Baillarger simultaneously, in 1854, described the cyclicity of the disorder. These two doctors worked in the same city (Paris), were of similar age, and were the disciples of a single master, Esquirol, who had actually taught almost all French psychiatrists of the era. They kept up a brisk polemic about who was first to define the concept, each claiming to have described it first, albeit as part of lectures given to young medical students (in the case of Falret) or in a clinical session at the Academy of Medicine (Baillarger). What is certain is that this polemic took over their lives and poisoned their personal relationship. The gulf could not be bridged even when the two met face to face at a conference on February 14, 1854. As stubborn as they were brilliant, each persisted in the conviction that he was first, and the other was the upstart. The story had a happy, somewhat bitter–sweet, ending when Falret’s son, also a psychiatrist and the successor of Baillarger, settled the dispute after the deaths of the two savants. With the wisdom of a Solomon but somewhat gratuitously, he attributed the genesis of the idea to both, and in a fine gesture started a fund-raising campaign to erect a monument

to Baillarger. On July 7, 1894, the French, masters of the subtle art of diplomacy, concluded what they rather high-mindedly called “a battle between giants” in a ceremony presided over by the busts of the two geniuses, who observed the evolution of mental disorders from the gardens of the most prestigious psychiatrist in Paris. The contest ended in a draw, and was a major step forward for psychiatry.

Much earlier, in the eighteenth century, although far more anecdotally, the Spanish doctor Piquer Arrufat described the disease of King Ferdinand VI as a mania–melancholia. Arrufat’s writings, fortunately revived in a fine new edition prepared by Vieta and Barcia (2000), are seasoned with moments of great clinical wisdom and inspiration (almost surprising for his time), and with curiosities, particularly with regard to the treatments essayed.

But the one who truly defined the outlines of the disorder by introducing the longitudinal study as an essential diagnostic tool was Emil Kraepelin whose work *Manic–Depressive Insanity and Paranoia* was a watershed in setting out the nosological aspects of bipolar disorders. He drew the boundaries of manic–depressive psychosis with schizophrenia, described the episodic course of the disorder, formulated its inheritability, and characterized its main clinical forms. Kraepelin’s work was followed and extensively developed by psychiatrists in Europe, although less so in North America which was influenced more by the ideas of Adolf Meyer and the psychoanalysts who fled Europe with the advent of Nazism.

Almost as important as the work of Kraepelin were the studies by Leonhard, who postulated a separation between bipolar and unipolar forms of affective disorders from clinical, evolutionary, and familial differences. This division was independently validated by Angst and Perris in 1966. Their studies, together with those of the North American group led by George Winokur, became the scientific and clinical basis for the early classifications of affective disorders based on the use of standardized criteria.

In parallel to the evolution in nosological concepts, the history of bipolar disorders is marked by the discovery of lithium salts. Lithium was tested on human beings for the first time in 1949 when the Australian scientist, John Cade, described its “tranquilizing” properties. Next, Scandinavian psychiatrists, especially the Mogens Schou group, conducted the first clinical trials and demonstrated its antimanic activity. Due to its molecular simplicity and therapeutic specificity, lithium continues to be a fascinating drug and its prophylactic effect has not been surpassed even today.

Diagnosis and classification

The diagnosis of bipolar disorders and its episodes is based on purely clinical criteria which are hence subject to controversy and interpretation. Nonetheless, bipolar disorders (especially type I) has in its favor a validity of construct and long-term stability that are greater than those of other mental disorders. Unlike anxiety, depression, or psychosis, mania is one of the most specific concepts in psychiatric nosology. The *Diagnostic and Statistical Manual for Mental Disorders 4th edition* (DSM-IV) diagnostic criteria for schizophrenia require exclusion of a manic picture, but the reverse is not the case. Even so, manifold symptoms are observed in other pathologies.

In its time, DSM-IV had some novel features over the 3rd revised edition, DSM-III-R: incorporation of type II bipolar disorders as a category of its own, including cases previously classified as unspecified bipolar disorders; inclusion in the affective disorder section of substance-induced mood disorders or organic illness; and incorporation of a series of specifications with prognostic value. Some of these specifications are:

- “with catatonic characteristics,” a specification added because many catatonic presentations are associated with mood disorders rather than with schizophrenia;
- “with atypical characteristics,” meaning a depressive phase characterized by mood reactivity, reverse vegetative symptoms and hypersensitivity to rejection, and were probably incorporated with a view to their therapeutic implications; and
- “postpartum-initiated,” a specification indicating a better prognosis but with particular vulnerability to recurring with each childbirth.

Longitudinal-course specifications were also incorporated to give information on the degree of interepisodic recovery. Rapid cycling was also recognized,

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in view of its poorer prognosis, poor response to lithium, and risks of antidepressant treatment in these patients. The seasonality and melancholic symptom criteria were slightly modified. Other changes had to do with the duration of the mania and hypomania symptoms, and the classification of pharmacogenic manias and hypomanias as substance-induced syndromes, although this is without doubt one aspect that needs to improve: in view of the non-specificity of some symptoms, future psychiatric taxonomists will have to include longer temporal criteria or withdraw these temporal criteria in order not to under-diagnose and hence leave untreated many of those affected by the less classical forms of disorder entities. In this sense, the future criteria for hypomania should look at the possibility of including episodes lasting less than 4 days – a time limit that certainly has no empirical support. A number of studies support this argument and argue for broadening the bipolar spectrum (Akiskal et al., 2000; Benazzi, 2001). On the other hand, more attention needs to be paid to the role of ethnocultural variables in the presentation of manic symptomatology, which often mask it and make diagnosis difficult, especially if we are talking about hypomania (Kirmayer and Groleau, 2001). All these may lead us to consider hypomania as a far more frequent syndrome than what has been established thus far, with a prevalence of up to 6.5% (Angst, 1998), and to bear in mind the possibility of it presenting in a mild form in which cognition plays a key role (Colom et al., 2002).

The WHO classification, the International Classification of Disease, 10th edition (ICD-10), is fairly similar but does not include cyclothymia in the definition of bipolar disorders; also, cases of unipolar mania and type II bipolar disorders are classified in a residual category (“other bipolar disorders”). It is also certain, however, that some preliminary criteria were introduced for incorporating type II bipolar disorders, likely to be in a specific category in the 11th edition of the ICD.

Cross-sectional diagnosis

Manic phase

As we know, the basic symptomatology of the manic phase of bipolar disorders is defined from a limited period of time in which there is a mood change featuring not only euphoria but also expansiveness and irritability, with accompanying symptoms including excessive self-esteem or grandiosity

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(which can be delusional), reduced sleep, logorrhea, racing thoughts, distractibility, increased involvement in pleasurable or high-risk activities while disdaining them, and psychomotor anxiety or agitation. In distinguishing it from hypomania, it is felt that in mania the change must be serious enough to bring about a sharp deterioration in sufferers' social/job activity, or to require them to be hospitalized so that they are protected from hurting themselves or others. Other associated symptoms may be emotional lability, anxiety, and dysphoria. When there are hallucinations or delusional ideas, the content is usually, but not always, related to mood. In fact, there is mounting evidence that symptoms traditionally considered exclusive to schizophrenia are present in bipolar patients during a severe manic episode (McElroy et al., 1996). Among these symptoms, which can be even more frequent than the so-called "mood-congruent" symptoms (Tohen et al., 1992) are Kurt Schneider's first-rank symptoms. In ICD-10, these patients are classified as schizoaffective. This is one of the most fundamental sources of diagnostic confusion between schizophrenia and manic–depressive psychosis – a confusion that may affect about one-fourth of bipolar patients (Vieta et al., 1994).

Hypomanic phase

The characteristic picture of hypomanic episodes consists of a predominantly elevated, expansive, or irritable mood, and actual manic symptoms for a given period of time, but not to a degree such that there is a marked deterioration in social or job performance, or such as to require hospitalization. In general, all the symptoms tend to be milder than in mania, and there is no psychotic symptomatology. Hypomania is a difficult syndrome to detect, especially with hindsight, and the difficulties in its diagnosis are the main source of errors in identifying type II bipolar disorder, which is confused with unipolar disorder and personality disorders. The borderline between hypomania and nonpathological elevated emotions is difficult to pin down, especially in highly educated individuals. Certain patterns of socially positive behavior (extreme sociability, good organizational skills, unstoppable decision-making and drive) are combined with the pathological expression of altered mood. The problem with such apparently virtuous hypomania is that the patient says she or he is making up for lost time, which is the strongest predictor of the next depressive episode.

From a more psychological standpoint, the cognitive–behavioral model has proven useful thus far in explaining cognitive function in depression

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(Beck, 1976) and, more recently, the implications of cognitive features such as self-esteem (Winters and Neale, 1985), attributional style, coping skills (Lam and Wong, 1997), and the decision-making process (Murphy et al., 2001) in bipolar disorders. The cognitive model fits perfectly into the medical model of the disorder, without discussing it and complementing it, partly because its great research tradition makes it easily testable empirically. Here it is very interesting to single out studies that find cognitive vulnerability in bipolar patients that is qualitatively and quantitatively similar to that of unipolar depressive patients (Scott et al., 2000b).

The cognitive model of mania (Colom et al., 2002) fully assumes that it is organically based and, taking a somewhat more than descriptive approach, points to the existence of distorted thinking, as occurs with depression. Mania, however, is characterized by a “positive” cognitive triad: over-optimistic view of oneself, the world, and the future. Manic-automatic thoughts are positive cognitions and interpretations that do not square with reality. As with depression, these are rigid, inflexible, and unrealistic thoughts (Colom et al., 2002). Beyond these suppositions, we feel that cognitions play a key role in hypomania, both as a triggering or aggravating factor and as a therapeutic tool designed to improve three aspects in particular: (a) organization of behavior, that is reduction of stimuli, postponing decisions, increasing the sleep pattern; (b) analysis of cognitions; and (c) improvement in therapeutic adherence, traditionally poor in bipolar patients. The superiority of this approach to dynamic and psychogenic models is due to the fact that it is less reductionist to a psychological model, so that it can be adjusted and combined with the medical/biological approach. At the present time, there is definite evidence that certain behavioral interventions can bring about biological changes; for example, it has been shown that sleep deprivation can bring on hypomania (Wehr et al., 1982). This phenomenon has been described, albeit anecdotally, for cognitive therapy (Kingdon et al., 1986).

The presence of cognitive changes during hypomania and depression phases is indubitable, and forms part of its diagnostic criteria. However, just as nihilistic, fatalistic thinking and underevaluation of one’s own capabilities should be understood not as a causal factor of depression but as a symptom of depression, the positive thinking characteristic of hypomania must also be considered a symptom, not a cause. The presence of changes in the method of processing information during a hypomanic episode has an etiologic value

per se, so that it must be recognized as playing an important role when “nourishing” the severity of the symptoms, as cognitive changes generally involve behavioral changes and the latter do aggravate an episode, beginning an actual hypomanic spiral that feeds on itself. This is why detection of cognitive changes has a great therapeutic value favoring early intervention in hypomanic phases when the spiral is less powerful. The states of emotional exaltation triggered by substances or physical diseases would be a good model, or at any rate the least bad model, for interpreting the cognitive changes that occur during a hypomanic episode (Vieta and Cirera, 1997). The effects of stimulants, for example, may mimic some of the phenomena observable in hypomanic patients, although the short duration of the elating effect prevents the cognitive changes from settling in and progressing.

To describe such cognitive changes in isolation is no easy task, as they appear to be an interwoven whole and do not usually exist independently. Some of them would be quantitative (i.e. consist of accentuation of a feature that was also present in asymptomatic periods) and others would be qualitative (i.e. would correspond to “new” modes of thinking for the patient). Thus, it is easier to describe a cognitive style qualitatively characteristic of the hypomanic phases, which we decided to call “anastrophic thinking” by contrast to the catastrophic thinking of depressive phases. This particular way of processing information would basically include overevaluation of the ego, a positive interpretation of reality, and unwarranted, disproportionate, and uncritical optimism.

The intrusive behaviors of hypomanic periods are strongly connected to the above-mentioned cognitive changes and in their turn contribute to worsening the patient’s condition. Just as Beck (1979) defines some “depressogenic” suppositions which by themselves and by modifying the subject’s behavior aggravate depressive symptoms, we would venture to propose, based on the Beck model itself, the existence of an “elatogenic” hypothesis (from the Greek *elata*, which Kahlbaum connects to thymic exaltation as distinct from *melana*, the root of the term “melancholia”). The existence of these concepts would be linked to the presence of an exalted mood that would be part of a psychobiological process whose presence would give rise to a series of anastrophic and anastrogenic cognitions.

Trying to establish a causal order between emotional, cognitive, and behavioral states beyond the contingency itself is virtually impossible without

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falling back somewhat on theoretical speculation. What is certain is that there appears to exist a feedback process between emotion and cognition, a process that affects behavior which eventually closes the cycle, modifying certain biological aspects through, for example, sleep deprivation. This cycle starts up again with the severity of the syndrome increasing each time with a snowball effect, increasing in size and speed to a veritable avalanche. A clear example of this phenomenon is the absence of fatigue in the manic patient; the greater the number of stimuli and activity, the more she or he becomes involved in them and the less time she or he spends asleep, this creating a vicious cycle with a crescendo effect on the symptomatology.

Depressive phase

The depressive phase of bipolar disorders has some features that distinguish it from unipolar endogenous depression on the one hand and reactive or situational depression on the other hand. The depressive phase of manic–depressive disorder is often accompanied by apathy predominating over sadness, psychomotor inhibition over anxiety, and hypersomnia over insomnia. Another difference described in the literature on bipolar and unipolar depressions is that, in the former, there is less anorexia and weight loss while emotional lability and the probability of developing psychotic symptoms in severe cases are greater. Epidemiologically, the age at which the disorder begins, in bipolar depression sufferers is younger and the incidence of postpartum episodes is greater. Other differences characteristic of bipolar forms are a family history of mania and completed suicide, and good response to lithium (Dunner, 1980). In young patients, the presence of catatonic stupor is frequent, and in the elderly, pseudodementia is likewise frequent.

Mixed phase

Mixed states are characterized by the simultaneous appearance of manic and depressive symptoms in different combinations, depending on mood swings, and cognitive and behavioral changes. The most common form, called depressive mania, is a picture characterized by hyperactivity and psychomotor anxiety, global insomnia, tachypsychia, and logorrhea, combined with depressive thinking, weeping and emotional lability, and often-delusional guilt feelings, all of which can be in various combinations. With the present criteria of DSM-IV, the appearance of a mixed state necessarily carries the