A CONCEPTUAL HISTORY OF ANXIETY AND DEPRESSION

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1. INTRODUCTION

For more than 2500 years, physicians have distinguished the clinical conditions which we call affective or anxiety disorder from such everyday feelings as fear, restlessness, and despondency, feelings which overwhelm each of us, at one time or another.
Contrary to what might be expected, case descriptions from the past bear often remarkable resemblances to patients encountered in modern day clinical practice. Whoever one consults, whether it is Aristotle, Galen, Burton or the 19th century alienists, images of a suggestive reality are evoked, images in which we can easily recognize the depressive, anxious and melancholic individual of our own era. There are similarities in symptomatology and course, as well as in the distinction between normality and pathology.
On the other hand, there are also considerable disparities in language and frame of reference, in conceptualization and in interpretation. From the time of Hippocrates up until well into the 17th century, the description and interpretation of anxiety and depression were dominated by the doctrine of bodily fluids (*humores*). And, until quite recently, all manner of ideas involving neural energy overshadowed discussions of phenomena such as neurasthenia, inhibition, and motor agitation.
These disparities have, traditionally, been given particular emphasis. The undeniably impressive growth of our knowledge is seen as evidence of the superiority of contemporary explanatory models. Conversely, ideas which were current from Antiquity up until the 19th century, are considered to be of no more than historical interest - simply a fanciful mythology for enthusiasts. The history of medicine has become a somewhat quaint activity, pursued by a handful of specialists. This may or may not be considered regrettable. However, these disparities once again become relevant at a deeper level of discussion, for clinicians as well as for scientific investigators. I refer here to the level of medicine's basic concepts and to the cultural and ideological strata from which these concepts derive their meaning.
A consideration of the foundations of medicine helps, for example, to put into perspective the already overly strict separation of symptoms and explanatory models. Symptoms are not natural phenomena, in that they are not the invariable expressions of an unchanging biological substratum. Whatever one's concept of disease, what the patient says is always based upon interpretation, at least to a certain extent. That interpretation can be colored by whatever is considered to be normal or abnormal in a particular culture. Explanatory models, on the other hand, are not simply conjured up out of the blue. They are neither arbitrary nor coincidental, but are instead closely linked to whatever paradigms are currently fashionable in the various branches of science. Moreover, they are always interpretations of a reality which is already interpreted by the afflicted person and those around him.
Accordingly, we cannot pretend that depression and anxiety are natural phenomena which have consistently been expressed and experienced in the same way down through the centuries. The view according to which only the explanatory models have changed and not the phenomena themselves, should be rejected. Concentrating purely on differences at the level of the explanatory models, can easily turn the history of medicine into a study of scientific folklore; as if, with the passage of time, only the explanatory models have undergone change and not the signs and symptoms of the disorders. Notwithstanding the above mentioned similarities in clinical picture and course, the symptoms of anxiety and depression also have changed, i.e. their relevance to what counts as disease and their meaning as an expression of disease.
Seen from this point of view, the study of the history of medicine suddenly becomes extremely relevant to a clear understanding of all sorts of current explanatory models. The medical history of anxiety and depression is, therefore, not simply concerned with internal scientific development. It also involves the interplay of cultural changes and psychopathological phenomena, including the

scientific interpretations of such phenomena.
In this chapter several leading concepts in the history of anxiety and depression will be summarized. Instead of delving into historical detail, the emphasis will be on concepts and, particularly, on the paradigm shifts associated with the changes in conceptual content.
Anyone interested in the detail is referred to the existing literature on the history of medicine, particularly to the outstanding studies of Jackson (1986) and Klibansky, Panofsky, and Saxl (1964). Also of interest are the studies by Ackerknecht (1968), Beek (1969), Berrios (1988), Flashar (1966), Foucault (1965), Gardiner, Metcalf, and Beebe-Center (1937), King (1978), Leibbrand and Wettley (1961), Lewis (1934a), Roccatagliata (1986), Rosen (1969), Starobinski (1960), Zilboorg (1941).

2. NOMENCLATURE

Before commencing with our historical review, a few comments about terminology.
First of all, it should be realized that the generally accepted distinction between anxiety and depression is of comparatively recent vintage. The first non-phobic form of anxiety to take its place in the description of disease did so as recently as the middle of the 19th century. Flemming's *Über Praecordialangst*, which dates from 1848, was cited by Schmidt-Degenhard (1986) as the first medico-psychiatric text exclusively devoted to a non-phobic form of anxiety.
Of course, this does not mean that subtle variations in the spectrum of anxiety and depressive disorders had not been observed and described prior to this. Evidently, however, there was no recognition of the need for a systematic distinction between anxiety and depression. For a long time, both were encompassed by the broad concept of melancholia. Since the *Corpus Hippocraticum* (5th. century B.C.), fear and despondency have been referred to as the prominent characteristics of melancholia.
The terms *melancholia* (Greek: *melaina cholè*, black bile) and *hypochondria* (Greek: *hypochondrios*, under the breastbone) are therefore of ancient vintage. The same applies to the concept of *mania*. The word *depression* (Latin: *deprimere*, to press down) gradually came into use during the 18th century (Jackson 1986, pp. 145-146).
Unlike the term *phobia* (Greek: *phobos*, fear), the term anxiety has neither Greek nor Latin origins. The word *anxiety* (German: *Angst*, worry) probably derives from the Indo-Germanic root *Angh*, which means to narrow, to constrict, or to strangulate (von Baeyer & von Baeyer-Katte 1971; Häfner 1971; Lewis 1967). This root reappears in the Greek word *anchein* which means to strangle, to suffocate or to press shut. The root *Angh* has also survived in Latin, for example in *angor* (suffocation; feeling of entrapment) and *anxietas* (shrink back fearfully; being overly concerned). Fear derives from the German stem *freisa* or *frasa*. The term panic on the other hand has a Greek background, namely *Pan* or *Panikos*, the Greek god of the forest and of shepherds, who was thought to have caused panic amongst the Persians at Marathon.
The boundaries of the different terms are rather vague. This is particularly true of the term *melancholia*, which covers practically all forms of non-organically determined psychopathology. In summary, however, it can be stated that despondency is a central element in numerous terms for depression, whereas in terms referring to anxiety the emphasis is often on sensations of tightness and constriction in the region of the chest and throat.

3. A HISTORY OF THE CONCEPTS OF ANXIETY AND DEPRESSION BASED UPON THE CONCEPT OF MELANCHOLIA

3.1 Ancient Greece and Rome

Western psychiatry, just like somatic medicine, has its roots in Greek natural philosophy. In this philosophy, the traditional explanations of mental illness, based upon the supernatural, gradually diminish in significance. Clinical observation and reasoning become established practice. Natural philosophers attempt to elucidate the universal principle behind observed phenomena. They observe heaven and earth, the orbits of heavenly bodies and the course of the seasons, as well as the cycle of ascension, splendor, and decline in the living and the non-living worlds. They are dissatisfied with demonological explanations of mental illness, such as those found in the works of Homer, for example.
This does not mean that moments of speculation become a thing of the past, as can be seen from the *Corpus Hippocraticum*, for example. This work consists of a series of 70 medical texts dating from the 5th century B.C., which are attributed to Hippocrates and his pupils. The *Corpus* contains the earliest formulation of the theory of the four humors, or bodily fluids. This humoral theory was a modified version of the view, first encountered in the works of Empedocles, that the universe is made up of a mixture of four elements: earth, fire, air and water. Empedocles himself was probably influenced by the Pythagorean school's doctrine of the 'harmony of the spheres', which placed strong emphasis on notions such as tuning and equilibrium. According to the humoral theory, disease results from a disturbance in the natural balance (*dyscrasia*) of the elements.
Blood, yellow bile, black bile and phlegm are the four bodily fluids or humors distinguished in the Hippocratic texts. These fluids were considered to be influenced by the seasons. Accordingly, blood would increase in the spring, yellow bile in the summer, black bile in the fall, and phlegm in the winter. In addition, each of the humors was associated with a pair of primary qualities. Thus blood was associated with heat and wetness, yellow bile with heat and dryness, black bile with dryness and cold, and phlegm with cold and wetness (see fig 1).

Insert *Figure 1*: The Four Humors

To the Greek physician, disease was caused by a disturbance in the natural balance of the bodily fluids. This balance was influenced by all sorts of factors, such as seasonal changes, climate, geographical conditions, age, mental effort, as well as eating and drinking habits. The Greeks were well aware, for example, of the link between depressive phenomena and the fall. In addition to these factors, certain people were temperamentally predisposed to melancholia. The term temperament refers to a person's humoral constitution. Due to an excess of black bile, or to an increased susceptibility of the black bile to heat or cold, some people could have a natural tendency towards melancholia.
This suggests that the balance between the humors reflects a much broader biopsychological and ecological equilibrium. This is indeed the case. The ancient concept of disease must be seen against the background of the then popular idea of a fundamental likeliness of macroscosm and microcosm. Universe at large is a well-orderdered macrocosm. Its changes are reflected at the level of microcosm, the individual body for instance. This theme was to dominate the concept of disease for at least two millennia. It left no room for the principle of linear (unidirectional) causality, which began to dominate medicine in the middle of the 18th century. Nor can it be equated to the late 19th century concept of homeostasis, since this concept presupposes the idea of internal feedback, a notion which was quite foreign to the ancient Greeks. In Antiquity, disease was seen as a disorder reflected on all levels of existence, rather than as the consequence of an internal disorder. The excess of black bile in melancholia was the analogue of changes in the seasons, in dietary habits,

and in psychological constitution (cf. Temkin 1973; 1977, pp. 422-5). The origins and conclusion of disease were not confined to the relative isolation of the body. Instead, disease reflected changes on all sorts of levels within the macrocosm.
The 20th century reader may suspect that there are conceptual problems here, however none seemed to exist for the Greek physician-philosopher. He seemed quite uninterested in the question of how all these different processes interacted with one other, choosing instead to ignore the problem. Some have suggested, for example, that the Greek outlook could not accommodate a psychogenic cause of mental illness. This is factually incorrect, since the literature of that time includes many examples of scholars becoming depressed through excessive study, and of melancholics consumed by feelings of guilt, hatred, or grief over a lost love. In addition, there is also a conceptual misunderstanding here. This occurs whenever modern ways of thinking begin to dominate the interpretation of humoral pathology. The humors are then reduced to purely biological phenomena (comparable to neurotransmitters) and the lovelorn state, or that of being overworked, to mere matters of psychology. Greek physicians undeniably thought of the *melainè cholè* as a substance which was both visible and tangible, even though they had never actually seen it. Nevertheless, they persisted in associating this unseen substance with all kinds of effects at the psychological and behavioral levels. From a 20th century point of view, this association could be seen as a metaphor. To the Greek physician, however, the notion of atrabiliousness (black bitterness) was a condensation of all sorts of very real experiences and perceptions. In short, even though the emphasis lay on what is now referred to as the biological component, the psychological connotation still was implied by the terms for the bodily fluids[1]. According to Aretaeus, bile means anger and black, much or furious:

> "... in certain of these cases, there is neither flatulence nor black bile, but mere anger and grief, and sad dejection of mind; and these were called melancholics, because the terms bile and anger are synonymous in import, and likewise black with much and furious"
>
> (Aretaeus; via Jackson 1986, p. 40).

For all that, black bile was the last substance to be ranked amongst the true bodily fluids. Initially interpreted as a breakdown product of yellow bile, black bile was first described as a natural constituent of the body in the *Corpus Hippocraticum*. Its change in status can probably be attributed to the dark-colored urine and feces observed in malaria sufferers and in patients with hepatic disease or gastric bleeding.
However, rather more than five centuries were to pass between this reference to black bile in the Hippocratic texts and the first summarized description of its effects. This summary, which can be found in the medical works of *Galen* (131-201 AD), was to serve as a model for medical thinking for centuries to come.
Galen owed a great deal to the work of *Rufus of Ephesus* (circa 100 AD), who me must thank for a description of various melancholic delusions, amongst other things. One such delusion was that of being an earthen pot, another was that of lacking a head. Rufus also influenced Arabic medicine and, through it, the medicine of the Middle Ages. It was Rufus from whom the great *Ishaq ibn Imran*, of 10th century Baghdad, reputedly derived his ideas about melancholia. The latter's work was to become the direct source for *De Melancholia* by *Constantinus Africanus* (11th century), a text which enjoyed great authority during the Middle Ages and the Renaissance (Klibansky, Panofsky, and Saxl, 1964, pp. 82 et seq).
The distinction between the three forms of melancholia, which he may have derived from Rufus, was considered by Galen to be of particular significance. He distinguished the following forms:

1. a *generalized* form of melancholia, with the blood being full of black bile;
2. a *cerebral* form of melancholia, which only affected the brain;
3. an *hypochondriacal* form of melancholia involving the organs of the upper abdomen (cf. Galenus, pp. 89-94; Jackson 1986, p. 37; Leibbrand and Wettley 1961, pp. 122-125).

The first form, unlike the second, could be accompanied by other phenomena e.g. discoloration of the skin, cirrhosis of the liver and the accumulation of fluid. The mental manifestations of both the generalized and the cerebral forms were due to obstructed blood vessels in the brain, as a result of a thickening of the blood. Galen suspected that this obstruction led to a blockage of the channels through which the so-called *pneuma animalis* flowed. This *pneuma* was an ether-like substance, made up of small, lightweight, highly animated particles. From ancient times until well into the 17th century, it has been associated with all kinds of mental functions, including perception and imagination. In other cases, according to Galen, black bile caused cerebral tissue damage, leading to the impairment of intellectual functions in particular. In the third form of melancholia, disease symptoms were not interpreted as resulting from black bile, as such. Instead they were caused by a vapor emanating from this fluid, as a result of local warming in the hypochondrium. This smoky vapor, according to Galen, rose up into the brain, obscuring thought. It was this mental obscuration which explained the anxiety seen in melancholics. Galen compared it to the darkness of night, which induces a causeless fear in many people:

> "As external darkness renders almost all persons fearful, with the exception of a few naturally audacious ones or those who were specially trained, thus the color of the black bile induces fear when its darkness throws a shadow over the area of thought [in the brain]".
>
> (Galenus, p. 93).

Apart from generating this vapour, such local warming also converted one of the other bodily fluids to black bile, thereby producing an excess. Under circumstances such as this, melancholia would be characterized by heat rather than coldness. According to some later writers of the Galenic school, this explained motor restlessness and behavioral disorders, an interpretation with which Burton concurred in 1621.
In terms of treatment, it was the distinction between the three forms of melancholia, which became of primary importance. Phlebotomy, the pre-eminent therapy for the generalized form, was ineffective in the treatment of the other two forms of melancholia. These required alternative measures to be taken. Mention is made of changes in eating and drinking habits, the use of emetics and laxatives, and attaining a correct balance between rest and physical exercise.
Galen was aware that, whilst the manifestations of anxiety and depression are tremendously varied, the heart of melancholia consists of despondency and anxiety, especially the fear of death:

> "Although each melancholic patient acts quite differently than the others, all of them exhibit fear or despondency. They find fault with life and people; but not all want to die. For some the fear of death is of principle concern during melancholy. Others again will appear to you quite bizarre because they dread death and desire to die at the same time.
>
> Therefore, it seems correct that Hippocrates classified all their symptoms into two groups: fear and despondency. Because of this despondency patients hate everyone whom they see, are constantly sullen and appear terrified, like children or uneducated adults in deepest darkness."

(Galenus, p. 93)

The link between melancholia and mania had already been established by Aretaeus of Cappadocia, who lived around 150 AD (cf. Leibbrand & Wettley 1961, pp. 111-116). However, in the work of Galen, this link is conspicuous by its very absence. In the Hippocratic texts, the term *mania* was frequently used when referring to mental illness in general, even though the link with the action of black bile had already been established. In the centuries which followed, mania and melancholia gradually became delineated as disorders having a certain periodicity, but with contrasting outward expressions. Nevertheless we must exercise caution, and not be over hasty in identifying these ailments with the present-day, bipolar disorder. The term *melancholia* still has very wide connotations, incorporating many different forms of psychosis and all kinds of neurotic symptoms. *Mania*, on the other hand, specifically refers to the various forms of emotional restlessness and motor excitation.
In fact, mania and melancholia together encompass virtually the entire field of prolonged psychopathology, that is, chronic diseases not associated with fever. The third form of mental illness, *phrenitis*, is both acute and associated with fever. The obvious comparison here is with delirious visions and acute psychoses. Epilepsy had a place all of its own. Viewed by the Greco-Roman world as a "sacred disease", it includes forms which are transitional between classical epilepsy and melancholia.

3.2 The Middle Ages

During the Middle Ages, ideas about anxiety and depression actually changed very little. Scholars continued to build upon the foundations created by the Hippocratic-Galenic school. For many centuries, Alexandria, with its enormous library, was the center of research and contemplation. One area of Byzantine medicine, as it is known, is particularly worthy of mention in connection with the subject under discussion. This concerns the work of compilers such as *Oribasius of Pergamon* (325-403), *Alexander of Tralles* (525-605) and *Paul of Aegina* (625-690). These scholars classified existing ideas from many different sources, without adding any significant contributions of their own.
At about the same time, the work of the Greek authors began to be translated into the Semitic languages. This task was carried out by Christians who had fled the Byzantine Empire, as well as by Arab authors. This was how Arabic medicine came to assimilate its Byzantine inheritance, in addition to influences from India and even China. By the end of the first millennium, writers from the Eastern Caliphate (Baghdad), such as *Rhazes* (865-923) and *Avicenna* (980-1037), had produced medical treatises of their own. Avicenna's *Canon of Medicine*, in particular, was to dominate medical ideas for centuries to come. From Persia came another significant figure, *Ishaq ibn Imran* (early 10th century), who has been referred to previously. It was his work on melancholia which inspired the great and influential treatises on the subject by the encyclopedist *Constantinus Africanus* (1020?-1087). Originally from North Africa, Constantinus subsequently lived in Salerno and later moved to Monte Cassano. His work reflects that of Ishaq, in that he also devoted an extraordinary amount of consideration to psychogenic causes of melancholia. Later on, famous scholars from the Western Caliphate (Spain), such as *Averroes* (1126-1198) and *Maimonides* (1135-1204), were also to exert an influence on medicine. In the late Middle Ages, however, authors dealing with melancholia mainly referred to the works of Avicenna and Constantinus (Jackson 1986, p. 63).
In the late Middle Ages, medical knowledge was mainly concentrated in monasteries and in cathedral schools. Moreover, various university medical schools were in the ascendent, the best known of these being Montpellier, Bologna and Padua. In addition to continued classification, some scholars now began to apply morality to humoral pathology (cf. Klibansky, Panofsky, and Saxl, 1964,

pp. 105-110). This led to melancholics being described by some as degenerate, along with phlegmatics and cholerics. Sanguinics, on the other hand, were considered to represent man, as God had intended he should be, at the Creation. Melancholia was also associated with *acedia*, a sort of listlessness and restless boredom, accompanied by a longing for change of environment. As long ago as the fourth century, Cassianus described this condition in the monks of desert monasteries, not far from Alexandria. The afternoon demon would appear around the sixth hour. It bred in the monks a loathing for their own cells, a disdain of the other brothers and a slothful unwillingness to take part in the routine activities of monastic life. Acedia, in the guise of Sloth, was to become one of the Seven Deadly Sins of the Middle Ages (cf. Jackson 1986, pp. 65-77; Klibansky, Panofsky, and Saxl, 1964, pp. 300 et seq).

One particular development in the continuing systematizing of humoral pathology, was an accentuation of the difference between two forms of melancholia. In addition to melancholia as a result of an excess of natural black bile, a second form was discerned, being caused by an excess of unnatural black bile. Unnatural black bile was thought to be produced by the combustion, or degeneration, of one of the four bodily fluids. In cases of an excess of natural black bile the characteristics of the melancholic temperament became more prominent. In such patients, meditation gave way to brooding. Their previously sincere and caring attitude to life, plunged into one of anxiety and gloom. They would stare at a single point, be incommunicative and avoid all contact. Beek, whose *Waanzin in de Middeleeuwen* (Madness in the Middle Ages) is, unfortunately, not available in an English translation, summarizes the writings of many authors as follows:

> "An excess of melancholic humor, which is thick and sediment-like, imparts a red color to the head. The patient also experiences a feeling of heavy-headedness. He tastes a bitter-sweet taste, the sediment of the humor. The pulse is weak and the veins full. The urine is thick and red-colored."
>
> (Beek 1969, p. 98, my translation; cf. also Klibansky, Panofsky, and Saxl 1964, pp. 86-90)

Although the combustion or degeneration of black bile presents a broadly similar picture, there is the added element of a pre-occupation with death:

> "They become agitated about funerals. Believing that they are about to die, they lie on graves and collect the bones of the dead. The pulse is hard and tense, the urine is lead-colored and thin."
>
> (Beek 1969, p. 98)

The picture which developed as a result of burned yellow bile was one of mania. Patients ranted, raved and screamed all day long. Referring, once again, to Beek:

> "They lie awake. They exhibit excessive movement, jumping and running around. They are reckless and quarrelsome, wanting to beat those around them, preferably with an iron bar. In the grip of the mania, they throw themselves through windows. The choleric temperament is characterized by a lack of inhibition, wild behavior, recklessness, constant motion and rage. Such patients have a lemon-colored complexion, their urine is thin and yellowish, the pulse hard and rapid. It is thought that sufferers do not feel the cold because the combustion of the bile keeps them warm."
>
> (Beek 1969, p. 98)

Conversely, the combustion of blood produces feelings of happiness rather than of sadness:

> "They talk and laugh the whole time, wanting to dance and make merry all whole day long. Their temperament type is one of happiness, laughter and loquacity. Their urine is thick and reddish brown."
>
> (Beek 1969, p. 98)

Degeneration of phlegm, on the other hand:

> "induces apathy, inertia and absent-mindedness. Feeling heavy-headed, these patients neither move nor laugh, nor do they feel joy. In people with this type of temperament, inertia, drowsiness and forgetfulness come to the fore. Characterized by a moist mouth and nostrils, and a pale white complexion, they are referred to as lymphatics. Believing themselves to be fish, they ask for water all day long and pine for a river, or the sea. Their pulse is small and weak, their urine pale, whitish and of medium thickness."
>
> (Beek 1969, p.98)

In tracing the origins of melancholia, factors other than the humors (combusted or otherwise) must be taken into consideration. These were the complexio (the temperament and primary qualities) of the brain and the condition of the rest of the body. A brain which is overly dry, or excessively cold, has an increased susceptibility to disease. A weak heart lowers the threshold against developing melancholia. Particular and frequent mention is made of the relationship between heart and head. Heart palpitations, for example, are the expression of an affection of the heart. Obviously, there is a relationship between the malfunctioning of this organ and a person's state of mind. After all, did not the *spiritus vitalis* ascend from the heart to the brain, where it influenced the *spiritus animalis*?
In medieval medicine, the nature of these influences was connected to the localization of functions in the different ventricles. The anterior (or lateral) ventricles were associated with imagination (*imaginatio*), the median (or third) ventricle with reasoning (*ratio*) and the posterior (or fourth) ventricle with memory (*memoria*). Melancholia was usually linked with a disorder of the middle ventricle. Conditions which involve hallucinations and delusions, such as mania and psychotic depression, were based upon disturbances to the equilibrium of the anterior ventricles.
Classification was extended to include therapeutic procedures. Polypharmacy had been popular even in ancient times, with leading roles being played by black hellebore and extracts of mandragora. In addition, regulation of the so-called *non-naturalia* (external or environmental factors) remained an important part of the therapeutic arsenal. Traditionally, six such factors were identified. These were air, physical exercise and rest, waking and sleeping, food and drink, retention and excretion of body products and the passions of the soul.
As regards *Air*: The patient should be kept in a warm, moist environment, the door of his house should preferably face east.
Rest and Exercise: The aim should be to reduce sensory excitation and to achieve relaxation. The patient should preferably be nursed in a dark room. The walls should be bare of pictures, since these might over-stimulate his imagination.
A not overly arduous walk, when the time is ripe, is beneficial for the maintenance of body temperature, as are massages and hot baths. An excess of body heat, however, dries out the body and causes melancholia.
Waking and Sleeping: The patient should sleep neither too little nor too much.
Food and Drink: This sickness can result both from overeating and from excessive fasting (as seen in

the ascetics). Food must be easily digestible. Vegetables such as lentils and beans give rise to flatulence and therefore cause melancholia. Peppery spices, garlic, leeks and onions must be avoided since they can burn the humors. The same applies to both mature and salted meat, the meat of forest animals, mature cheese, vinegar and fish. A heavy wine, rich in sediment, causes sickness, whereas a light, young wine can actually raise the spirits.
Retention and Excretion: Melancholia can result from the accumulation of those bodily fluids which are normally discharged, such as menstrual blood, sperm, or hemorrhoidal blood. The same is true of feces. Evacuation sometimes requires mechanical assistance, for example in constipation, the non-appearance of menstruation, or the non-bleeding of hemorrhoids. Coitus is generally to be recommended, although men with low potency should avoid over-indulgence. Bathing, whether in a herbal bath or just with plain water, is an important therapeutic agent, which can also facilitate evacuation. However, excessively hot baths can induce madness.
Finally, regarding *Passions of the Soul*: Excessive fear, hatred and grief should be avoided. The same applies to excessive study and to intense preoccupation with a particular task. Nevertheless, anxious and inhibited melancholics can actually be cured by either a fierce rage or a sudden shock. Generally speaking, however, these passions should be kept under control. Discussion and philosophical reading can both be useful in calming the patient.[2]

3.3 Intermezzo: Melancholia as a Characteristic of Genius

We have placed great emphasis on the continuity of the medical debates regarding melancholia. This continuous dialogue spans the centuries, from ancient times up to the Middle Ages and, as we shall see, even beyond. There are, in all, three closely related concepts:

1. natural black bile;
2. the disorder of melancholia, based on either
   a) an excess of natural black bile, or
   b) the combustion of one of the four body humors
   (*melancholia adusta*);
3. a chronic predominance of natural black bile in people with a melancholic-type temperament.

In spite of the allegorizing and moralizing interpretations, the concept of melancholia retained its link with humoral pathology. This doctrine, however, was not entirely undisputed in ancient times. The empirical school, for example, considered the theory of bodily fluids to be too speculative. There was also the methodist school, which sought refuge in a much simpler classification of disease (involving the *status strictus* versus *status laxus* or, in other words, the respective contraction and relaxation of the so-called internal pores). We also found that, with regard to the temperaments, there was some doubt about the normalcy of the character variations (cf. note 1).
There is another line which, in terms of the theme of this review, is even more important. This proceeds from Plato and Aristotle, via the Florentine School of the Middle Ages (Marsilio Ficino) and Robert Burton (1621) in The Renaissance, to William James (1902) in the present century. It is a line of thought which regards the melancholic as exhibiting certain traits of genius. There is the famous opening line from the thirtieth book of Aristotle's *Problemata*:

> "Why is it that all those who have become eminent in philosophy or politics or poetry or the arts are so clearly of an atrabilious temperament, and some of them to such an extent as to be affected by diseases caused by black bile, as is said to have happened to Heracles among the heroes?"

(Aristotle, Problemata, 953a 10)

Aristotle seeks a natural explanation for this "madness which comes from the gods". This is in contrast with Plato's *Phaedrus* (244b-250), which offers a mythological description. Aristotle suggests that the eminence of the poets, politicians and philosophers in question could be ascribed to an optimum warmth of the black bile. Black bile, by its very nature, was thought to be sensitive to changes of temperature. When cooled, it brought about "apoplexy or torpor or despondency or fear". Heating induced "cheerfulness accompanied by song and frenzy and the breaking forth of sores and the like" (954a 21-26). In the case of a "man of genius", black bile, which is as such a pathogenic fluid, is in an optimum state. Such a person represents the normal within the abnormal or, in the words of Klibansky, Panofsky, and Saxl (1964, p. 40), an "eucrasia within an anomaly".

During the Renaissance, a time of revived interest in astrology, this association between melancholia and genius acquired a special meaning. In Antiquity, the planet *Saturn* had been associated with *Kronos*, the son of *Uranus* (the god of Heaven) and *Gaia* (the Earth goddess), who had been banished to the underworld after the castration of his father. Now, Saturn gradually turned into a symbol of the ambivalence of intellectual and artistic life. According to ancient astrology, Saturn was the planet of gloom, desolation, decline and old age. However, the Neo-Platonists claimed that Saturn, as the highest planet, was the planet of the elevated, of ecstatic release from earthly things and happenings (Klibansky, Panofsky, and Saxl 1964, pp. 133-195). *Marsilio Ficino* (1433-1499), Neo-Platonist of the Florentine school as well as a priest and physician, depicted Saturn as the heavenly body whose rays influenced the vital spirits of the brain (*spiritus animalis*), which were thought to connect the physical to the spiritual. This influence was described as a kind of saturation process, one to which those born under the sign of Saturn were especially sensitive. Although enjoying intellectual powers and creative talents far exceeding those of others, there was a tragic element to these people. They spent their lives teetering on the very brink of catastrophe and they were especially susceptible to melancholia. Black bile was identified with the earth, including the very center of the planet itself, which meant that such people tended to have a deeply penetrating understanding of existence. The association with Saturn, the highest of the planets, meant that they aspired to higher planes of thought (Klibansky, Panofsky, and Saxl 1964, pp. 241-274). The novel element here is the heightening of self-consciousness, the awareness of man's vulnerability to catastrophe and decline. Their view of life took on a nostalgic and tragic tint. This outlook was to recur in a variety of different forms in later periods, as, for example, in the Elizabethan literature of the 16th and 17th centuries (Elizabethan Malady; cf. Klibansky, Panofsky, and Saxl 1964, pp. 228-240).

3.4 The Renaissance and 17th and 18th Centuries

The Renaissance was not only an age of heightened self-awareness and the era of *Homo literatus*, with his knowledge of the classics, it was also the time of alchemy. It was this which lent impetus to the interpretation of disease in terms of chemical change, an approach which texts on the history of medicine refer to as *iatrochemistry*. Substances such as salt, sulfur and silver became the focus of attention. Paracelsus (1493-1541) was one of the first to apply the newly gathered knowledge to medicine, in an attempt to break down the hegemony of humoral pathology. He did not, however, renounce the doctrine of the temperaments and of the elements. Melancholia now became associated with the qualities of the chemical elements, which are sharp and acidic. Thomas Willis (1621-1675), renowned for his *Two Discourses Concerning the Soul of Brutes*, expounded the theory

that, in melancholia, the blood became "salino-sulphureous", causing the *spiritus animalis* to adopt a different pattern of motion (Jackson 1986, p. 112).

The high point of 17th century medical literature on melancholia, however, was Robert Burton's *The Anatomy of Melancholy*, published in 1621. This work, which may seem somewhat bizarre to the modern reader, offered a compilation of all contemporary knowledge on the subject of melancholia. Greatly indebted to ancient medicine and philosophy, Burton punctuated his arguments with references from ancient times. He subscribed to the dichotomy of the passions (derived from Thomas Aquinas), a doctrine which was generally accepted at the time. Here, the passions which predisposed one towards desire (*passiones concupiscibiles*) were distinguished from those which predisposed one towards rage (*passiones irascibiles*). 'Sorrow' and 'fear', emotions ('perturbations') belonging to the latter category, were described by Burton as being directed at the evil which crossed one's path. Sorrow was related to disaster in the present, fear, to disaster in the future. Burton considered sorrow to be a major cause of melancholia, as well as one of its manifestations. It was:

> "... an inseparable companion, the mother and daughter of melancholy, her epitome, symptom, and chief cause."
>
> (Burton 1621, p. 298)

The same was true of fear, the emotion most able to hold the imagination in thrall (*ibidem*, p. 302). The imagination was:

> "... *medium deferens* of the passions, by whose means they work and produce many times prodigious effects."
>
> (Burton 1621, p. 297)

Burton went on to add, however, that the converse was also true, since imagination serves to enhance the impact of emotions.
In discussing the symptoms of melancholia, Burton considered fear and sorrow to be just some of the phenomena affecting nearly all of those suffering from this condition. Although these emotions were relatively non-specific phenomena, this did not mean that they were unimportant.
Burton was acquainted with many of the forms of anxiety known today. He made reference to fear of death, to fear of losing those who are most important to us and to paranoid anxiety. He also cited anxiety based on ideas and delusions of reference, fear associated with depersonalization, delusional depersonalization and hypochondria. Other forms which are mentioned include agoraphobia (!) and many other kinds of specific phobias, such as fear of public speaking, fear of heights, claustrophobia, anticipatory fear and hyperventilation (Burton 1621, pp. 442-449).
The physiology of emotions was strongly emphasized in René Descartes' *Les Passions de l'âme* (The Passions of the Soul), which was published about a quarter of a century later (Descartes 1649; cf. Riese 1965; Rorty 1982). This emphasis on physiology had a distinctly mechanistic flavor, in keeping with the contemporary trend towards a general mechanization of the world view. A trend which, in medicine, would only really take off in the 18th century (*iatromechanics*; cf. King 1978).
According to Descartes, passions not only prepare the body but also predispose the soul to desire that for which the body is being prepared. The physical manifestations of emotion therefore provide some degree of insight into the function of emotions.
Fear and anxiety did not rank highly amongst the passions (cf. *Les Passions de l'âme*, articles 34-39, 58, 165, 174, 176). Indeed, Descartes considered the emotion of fear to be quite useless.

Nevertheless, his descriptions of the processes which give rise to fear are worthy of mention, since they are representative of the 17th and 18th century mechanistic view of disease. According to Descartes, the sight of a dangerous animal caused certain particles (the 'esprits animaux' or *spiritus animalis* referred to previously) to be released by the pineal gland. Although invisible, these rapidly-moving particles were quite material in nature. They traversed the neural pathways to reach the heart, the leg muscles and the circulatory system. This gave rise to the physiological component of the 'fight' or 'flight' response. The altered distribution of blood then caused a rush of these 'esprits animaux' to the brain. Here, the pores associated with fear were opened, directing the 'esprits' onwards, back towards the periphery. Mental influences were not, therefore, presumed to be involved in the generation of fear. Instead, fear was seen as a complex, but purely physiological, reflex.

Although emotional perception was secondary, consisting of the registration of pineal gland movements, Descartes believed that such registration had a purpose, namely evaluation. It facilitated the perception of objects in terms of their effects upon us, whether beneficial or otherwise[4]. Properly employed, passions predispose the soul to desire those things which are naturally good for us. In this resigned adaptation to the eternal laws of nature, one can detect the aftereffects of the *Stoa* - aftereffects which extend to the implicit morality of certain modern day forms of psychotherapy.

Descartes' emphasis on mechanics was not accepted by the medical world until the closing years of the 17th century. It enjoyed a brief flowering in the period around 1750 before giving way to other interpretations. Those associated with iatromechanics, as it was called, included men such as *Pitcairn*, *Hoffmann* and *Boerhaave* (whose work reveals traces of a humoral pathology, interpreted from the point of view of fluid dynamics). Boerhaave and Pitcairn favored a *vasocentric* approach, seeking the origins of melancholia in the modified flow patterns and viscosity changes of liquids in the blood channels. Hoffmann was one of the first to consider a *neurocentric* approach. He considered nerves to be hollow tubes containing a flowing liquid. Under normal circumstances, this neural fluid was thin and volatile, however if it thickened to an earthy, slimy consistency then melancholia developed.

Under the influence of Isaac Newton, men such as *Mead* and *Cullen* (who was the first to use the term neurosis) speculated that this neural fluid might also have electrical properties. Cullen and others thought of this neural fluid as a tenuous, highly mobile substance, which was related to ether. This ether-like substance did not actually flow, instead it transmitted motion to the various parts of the body by means of vibration. This caused the fluid hydraulics model of mental illness to be discarded. At the time, there was only a vague notion of electrical phenomena. These were linked to the vitalistic interpretation of disease, the central concepts of which were tone and irritability. Cullen considered the irritability of the nervous system to be responsible for tissue tone (cf. Lopez Pineiro 1983, p. 12).

Meanwhile, the clinical description of melancholia had remained virtually unchanged. Some still cited the Galenic trio of generalized, cerebral and hypochondriacal melancholia. However there were those, such as Boerhaave for example, who interpreted this classical triad merely as stages on a continuous scale of severity. Some authors were inclined to uncouple hypochondria from melancholia (of which it was the least serious form) and link it instead with hysteria. According to *Sydenham* and *Lorry*, for example, hysteria in women was equivalent to hypochondria in men.

The 18th century was a time bursting with tensions and contrasts, with shifts and changes. Humoralism gave way to solidism (the explanation of disease based on the properties of the organs and tissues). Iatrochemistry gave way to iatromechanics, which in turn was replaced by ideas such as tone and irritability. Vasocentric views were replaced by neurocentric ones. Meanwhile, vitalistic views of disease blended with speculation on the electrical properties of neural fluids. Each of these various approaches was considered to be compatible, incompatible or related to one other (cf. King

1978). The rational framework of the early 18th century clearly offered medicine of that time the requisite intellectual freedom in which to forge its principle concepts.
Towards the end of the century this all began to change. Pathological anatomy had expanded enormously, and greater emphasis was being given to clinical observation and description. It was a time of sensualism and of a fascination with sensory perception. Notions such as irritability bear witness to a preoccupation with the hypersensitivity of the nervous system and of the senses. The end of the 18th century saw the final demise of humoralism. During the same period, the notion that melancholia originated in the blood, or in the functions of the liver or spleen, was also dismissed. The central nervous system replaced blood and abdominal organs. Today, the idea of temperament is all that remains of humoralism. Although it has no place in the scientific view of character and personality, it still exists as metaphorical expression for the experiences of despondent people.

3.5 The 19th Century: The Further Disintegration of the Concept of Melancholia

The concept of partial insanity was popular in the nosographical schemes of the 18th century and the first decades of the 19th century (Starobinski 1960, p. 55). As has been noted, medical scholars of the late 18th century were fascinated with the sensitivity and irritability of the nervous system. This, together with the fast growing influence of faculty psychology (which sharply distinguished between intellect, will and imagination), may have contributed to the popularity of the concept of partial insanity. This was not particularly novel, it was more the emphasis of an element of meaning found in descriptions of melancholia dating from ancient times (e.g. those of Aretaeus and Galen). For centuries it had been observed that the thoughts and ideas of the melancholic were confined to a single theme, often to the extent that they became delusional. Melancholia was traditionally considered to be a delirium without fever, accompanied by fear, despondency and additional phenomena such as restlessness, insomnia, weariness and discomfort in many parts of the body. In addition, however, frequent reference was made to the monothematic content of the melancholia sufferer's ideas and thoughts (for a review, see Jackson 1983).
Late 18th century medicine had a neurocentric orientation, one which tended to focus upon sensitivity and cognitive capabilities. This may, perhaps, have influenced the classification of mental illnesses into disorders in which the powers of judgement were completely incapacitated, and those in which this was only partly the case (partial insanity). Melancholia was considered to fall within the latter category, a view which can be found, for example, in the *Traité médico-philosophique sur l'aliénation mentale* (Medical-Philosophical Treatise on Mental Disorder) by Philippe Pinel (1745-1826; Pinel 1801). Pinel also considered mania to belong to this 'melancholia with delirium'.
This (temporary) identification of melancholia with partial insanity completed the decline of a concept which for centuries had dominated the description of mental illness. Melancholia was divided up, and its various parts were classified under other disorders. A number of things contributed to this redistribution, such as resistance to humoral pathology and its terminology, more detailed observation (as a side effect of growing institutionalization) and rationally-inspired expectations regarding psychology's ability to influence mental illness (see Porter 1978, pp. 8-38; especially pp. 16-20).
The first line of demarcation was given with the idea of partial insanity itself, since some melancholics exhibit no signs of insanity (delirium) whatsoever. That is to say, none of their thoughts and ideas would be regarded as psychotic in the modern sense. In the *Manual of Psychological Medicine*, which he wrote in collaboration with Bucknill, *D. Hack Tuke* (1827-1895) distinguished between simple melancholia, in which the intellectual powers were intact, and complicated melancholia, in which they were not. The distinction applied by *Henry Maudsley* (1835-

1918) was essentially the same as Tuke's, if somewhat wider in scope. In addition to melancholia simplex (cf. Bucknill and Tuke's 'simple melancholia'), he distinguished not one, but two forms of melancholia, both falling within the category of ideational insanity. These were melancholia as form of general insanity and melancholia as a form of partial insanity. In the first case intellectual derangement is complete, whereas in the latter case it is only partial. There was also a parallel distinction in mania. However, Maudsley went to great pains to emphasize the provisional nature of this classification (cf. Jackson 1986, pp. 170-171).
Similarly, in France, the significance of the term melancholia declined considerably under the influence of a nomenclature introduced by *Jean-Etienne-Dominique Esquirol* (1772-1840) in his *Des Maladies Mentales* (1838). Since ancient times, the meaning of the term *melancholia* had encompassed both dejection and exaltation. Finding this an unsatisfactory state of affairs Esquirol substituted the term *monomania* for melancholia. Monomania, which became an equivalent of the term partial insanity, was subdivided as follows:

1. "monomania, properly so-called, which is indicated by a partial delirium and a gay or exciting passion"; this condition corresponded to "maniacal melancholy, maniacal fury, or (...) melancholia complicated with mania; in fine ... (to) *amenomania*"; and
2. monomania corresponding to "melancholy of the ancients, the *tristimania* of Rush and the delirium with melancholy of Pinel" (Esquirol, 1838, p. 404; English translation from Jackson 1986, p. 152).

Esquirol borrowed the terms amenomenia and tristimania from the work of *Benjamin Rush* (1745-1813), the father of North American psychiatry. It was Rush who had linked tristimania with hypochondria, alluding to the ancient hypochondriacal form of melancholia rather than to the diluted, 18th century meaning of the term. Confusingly, besides employing monomania in the broad sense mentioned above, Esquirol also used the term to denote the mania-like form of partial delirium. In this way, monomania became the equivalent of the first form of monomania, the partial delirium with the "gay or exciting passion". The second, melancholic, form of monomania was denoted by the term *lypemania*. In addition, Esquirol distinguished mania as a generalized insanity associated with excitement and exaltation. He occasionally tended to describe lypemania, monomania (in the strict sense of the word) and mania as having progressive degrees of severity, the greatest derangement occurring in mania and the least in lypemania.
Even in ancient times it had been frequently pointed out that melancholia and mania could occur in parallel, in sequence and in combination. However, it was not until 1854 that cyclical mood swings were specifically identified as the distinguishing criterion of a sub-category of manic and depressive patients. In the same year, *Jules Baillarger* (1809-1890) described 'la folie à double forme' (the insanity with two forms), ten days later followed by a commentary of *Jean-Pierre Falret* (1794-1870), in which he also discussed a 'folie circulaire' (circular insanity). Some forty years later, Kraepelin explicitly harked back to the work of these two French clinicians when he distinguished dementia praecox from manic-depressive psychosis.
Nonetheless, even by the middle of the 19th century, the terminological distinction between affective and schizophrenic psychopathology was by no means a fait accompli. Accordingly, *Richard von Kraft-Ebing* (1840-1902) declared that there were two forms of melancholia, namely simple melancholia and melancholia with stupor. The latter form, also known as *melancholia attonita* or *melancholia stupida*, was quite different from the partial delirium of French and English psychiatry. It was related to a condition which, a few decades later, Kahlbaum was to call *catatonia*, which could also be associated with reduced consciousness. With regard to simple melancholia, von Kraft-Ebing distinguished between a mild form of melancholia which was not associated with delusions, melancholia associated with precordial pain, and melancholia associated with delusions and

hallucinations. He placed strong emphasis on psychomotor inhibition in all forms of melancholia, stating that in cases of melancholia attonita, this usually had an organic cause, such as a blockage in the motor neuron pathways. Such inhibition could, however, also be psychogenically induced. In practical terms, there were all kinds of transitional states between the two major forms of melancholia. The difference between these states lay in the relative degree to which mental and organic components were involved in the origin of the inhibition.
This shows that the 19th century ups and downs of melancholia, as a concept, were determined by a variety of different factors. In addition to the attempts to distinguish new forms and the pursuit of ever more precise classification, there were also advocates of continuity, who searched for transitional forms between the diverse clinical pictures. An extremely clear-cut example of this is provided by Wilhelm Griesinger (1817-1868) who strongly defended the concept of a unitary psychosis (Einheitspsychose) in his *Die Pathologie und Therapie der psychische Krankheiten für Ärtzte und Studierende* (The Pathology and Therapy of Mental Diseases for Physicians and Students). In this work he cited both his mentor, Zeller, and the Belgian alienist Guislain. According to Griesinger, the various clinically defined forms of mental illness could be reduced to the different stages of one and the same disease. The first of these successive 'states of mental depression' was the *stadium melancholicum*, that is, the deterioration of normal emotions such as grief and jealousy. Next came *hypochondria*, the mildest form of insanity. This was followed by *melancholia in sensu stricto*, which, although not necessarily associated with psychotic phenomena, had a greater effect on the personality than did hypochondria. Finally there was *mania*, which caused the most pronounced mental derangement of any of the conditions listed here.

3.6 Emil Kraepelin

The debate about the classification of mood disorders, which continued on into the twentieth century, centered around the question of whether or not this group of disorders could be subdivided. However, after 1900, the matter of whether mood disorders constituted a separate category of mental illness was hardly ever discussed.
This remarkable fact can be largely traced back to the work of one man, *Emil Kraepelin* (1856-1926). It has often been pointed out that the term melancholia (along with related terms such as mania, partial delirium and monomania) can certainly not be identified with what we currently refer to as affective disorder. The clinical pictures concerned were always those incorporating a variety of phenomena which would currently be regarded as expressions either of schizophrenia or of a schizophreniform disorder. It is remarkable, to say the least, that this age-old intermingling of psychotic and affective symptomatology should have come to an end at the beginning of this century.
In his early years, Kraepelin worked with the neuro-anatomist Flechsig. For a lengthy period he was also a research worker in Wundt's psychological laboratory. Kraepelin cannot simply be portrayed as a materialist, or as a somatologist, his field of interest was much too comprehensive for that.[5] Nevertheless, the idea that every psychiatric clinical picture could ultimately be reduced to an organic substratum in the brain was kept alive by Kraepelin and many of his contemporaries. They were motivated by the discovery of the cause of dementia paralytica, the confrontation with many cases of alcohol dementia and the aftermath of the theory of degeneration, formulated by Morel in the middle of the 19th century. Kraepelin accordingly established an anatomical laboratory in Heidelberg. He also brought in the histopathologist Nissl, to assist him in the visualization of the cerebral cortex. Their collaboration eventually bore fruit, in the form of photographs (measuring from 50 to 75 centimeters) showing general views of the cerebral cortex, (Kraepelin 1983, p. 73). However, it were not these efforts which ultimately contributed to the lasting topicality of

Kraepelin's work. Possibly influenced by Kahlbaum, he became persuaded of the importance of systematic clinical observation and description. It was this conviction which inspired him to amass a database of all the patients registered in Heidelberg, something which required great perseverance. This database enabled him to follow the medical histories of his patients, in some cases for several decades. It formed the basis for the separation of manic-depressive illness ('das manisch-depressive Irresein') from schizophrenia (dementia praecox), which was first described in the fifth (1896) and sixth (1899) editions of his famous textbook (Kraepelin 1983, pp. 68-69; cf. Kraepelin 1905; 1913). Although there was always a bias towards localization, it gradually faded into the background and made way for a more functional and strictly empirical approach, in which classification had less to do with diagnosis (i.e. the cause) and more with prognosis (cf. Berrios & Hauser 1988). It was the course alone (rather than cause, symptoms or periodicity) which was to prove decisive demarcating dementia praecox from manic-depressive illness. Dementia praecox led, by definition, to personality decline (Verblödung; dementia), whereas manic-depressive illness did not.

> "(...) the universal experience is striking, that the attacks of manic-depressive insanity within the delimitation attempted here never lead to profound dementia (Verblödung, G.G.), not even when they continue throughout life almost without interruption. Usually all morbid manifestations completely disappear; but where that is exceptionally not the case, only a rather slight, peculiar psychic weakness develops, which is just as common to the types here taken together as it is different from dementias in diseases of other kinds."
>
> (Kraepelin 1913, p. 1185; translation from Thompson 1987, pp. 246-247)

At the onset of the illness, it can be extremely difficult to reach a correct diagnosis. Some things to go by are age at onset (younger than twenty or beyond middle age) and a confirmed family history. There is also the premorbid character which, in the case of manic-depressive illness, is weak, susceptible, dejected and lacking in self-confidence (Kraepelin 1913, p. 1387).
Meanwhile, the category of manic-depressive illness was very broad, encompassing much more than the bipolar disorder, as it is called today:

> "Manic depressive insanity (...) includes on the one hand the whole domain of the so-called periodic and circular insanity, on the other hand simple mania, the greater part of the morbid states termed melancholia and also a not inconsiderable number of cases of amentia. Lastly, we include here certain slight and slightest colorings of mood, some of them periodic, some of them continuously morbid, which on the one hand are to be regarded as the rudiment of more severe disorders, on the other hand pass over without sharp boundary into the domain of personal predisposition. In the course of the years I have become more and more convinced that all the above mentioned states only represent manifestations of a single morbid process."
>
> (Kraepelin 1913, p. 1183; translation from Thompson 1987, p. 245)

In this definition, the bipolar disorder of our time coincides with "periodic and circular insanity". Here, manic derangement is characterized by the triad of rapid association of ideas, elated mood and hyperactivity. Depression, on the other hand, is associated with the triad of dejection or anxious moods, inhibition of thought and reduced spontaneity. In addition to the circular and simple disorders, amentia and milder mood disorders, Kraepelin also refers to mixed pictures. These cases exhibit some characteristics of mania and others typical of depression. He also refers to the so-

called 'ground states' (Grundzustände; predisposing personality traits), which form the basis for the development of mood disorders (Kraepelin, 1913, pp. 1289 et seq. resp. 1303 et seq). Kraepelin distinguished four 'ground states': depressive, manic, irritable ('erregbare') and cyclothymic. Finally, a distinction was made between this group and the form of melancholia associated with a decline due to the effects of aging ('Rückbildungsalter'; involution). In the latter case, inhibition was often absent whilst anxiety and hypochondria were more prominent. Although Kraepelin was initially inclined to keep this (involutional) melancholia separate from the others, he abandoned this line of thought following the comprehensive study of this clinical picture by Dreyfus (1907). He subsequently included this form of melancholia within "das manisch-depressive Irresein" (manic-depressive psychosis). The debate about involutional melancholia would not finally be settled until the nineteen seventies, when this condition became just another form of depression.
Something which is common to all forms of manic-depressive illness is the absence of an immediate cause, or at least a course which appears to be independent of any possible causes.
A distinction should therefore be made between psychogenic depressions and the conditions referred to above (Kraepelin 1913, pp. 1389-1390; cf. pp. 1369-1370). The most fundamental cause of manic-depressive psychosis is an hereditary, morbid predisposition. According to Kraepelin, of the 900 cases which he studied in Heidelberg, he had managed to establish that about 80 percent involved an hereditary defect (Kraepelin 1913, pp. 1353-1354).
Despite opposition (Hoche 1910; Jaspers 1953, pp. 471-479), Kraepelin's interpretations nevertheless enjoyed great authority in German-speaking regions. This is demonstrated, for example, by *Eugen Bleuler's* textbook, which was published in 1916. In describing the manic-depressive illness as a distinct disorder, this work relies heavily on the questions which Kraepelin had raised on the subject (Bleuler 1916, pp. 345 et seq). The thirteenth edition of this textbook, which was published in 1975, exhibits some reservations about the heredity hypothesis and about the possibility of an airtight distinction between dementia praecox on the one hand, and psychogenic disorders on the other. Nevertheless it still contains references to the old Kraepelinian classification (Bleuler 1975, pp. 457 et seq).

3.7 Critique on Nosology: Reaction Type versus Endogeny

The situation was different in the United States where, in the first decade of this century, *Adolf Meyer* (1866-1950) expressed doubts about the value of the course criterion. He distinguished several forms of depression, such as constitutional depression, simple melancholia proper, other forms characterized by neurasthenic malaise or hypochondriacal complaints, depressive deliria, catatonic melancholia and the so-called 'delire de negation', in which the patient believes he is nothing (Winters 1951, p. 566-567). This was more than just a debate about classification. Meyer was particularly uneasy about the coupling of heredity (endogeny) and manic-depressive psychosis, in the broad, Kraepelinian sense. He viewed this link as nothing more than "neurologizing tautology", which might easily give rise to therapeutic nihilism. Moreover, it did not do justice to the fact that mental illness is an attempt at adaptation, a reaction to the demands of a changing situation. Only when disease is seen as an inadequate attempt at recovery, the search for what he called "modifiable determining factors" could begin. We are then "in a live field, in harmony with our instincts of action, of prevention, of modification and of an understanding doing justice to a desire for directness (...)" (Winters 1951, p. 599).
The question remains as to whether Meyer did justice to Kraepelin. In any event, Kraepelin cannot be accused of therapeutic nihilism. Under his direction, the enlarged baths at Heidelberg achieved international fame and were visited by many foreign guests. Nothing was too much trouble for him when it came to experimenting with new methods of treatment. Of greater importance is the

conceptual point at issue here. According to Meyer, the debate on classification should not be short-circuited by an appeal to such ideas as endogeny and heredity, about which nothing was empirically established. He did not presume that biological processes should enjoy a privileged position in the list of determining factors for mental illness. For this reason, Meyer substituted the term manic-depressive psychosis with the etiologically neutral term "affective reaction type". Within this reaction type, he distinguished a manic-depressive type, an anxious type and simple depressions (Winters 1951, p. 600).
Essentially the same view was held by Sir *Aubrey Lewis* who, in the nineteen thirties, published an authoritative study of 61 cases investigated at the Maudsley Hospital in London (1934b; cf. 1936, 1938). According to Lewis, a total reaction of the organism is involved, even in cases where the illness appears to be entirely without cause. Without doubt, there were

> "changes in the internal structure of the body, its chemical and vegetative regulation which play a great part in determining its course. But these are only part of the total reaction of the organism, and it is by no means in denial of their fundamental importance in the illness that one refuses them independent and preponderant significance, either aetiological or as part of the process of the illness."
>
> (Lewis 1934b; via idem 1967, p. 113)

According to Lewis, the more closely patients were studied, the less evidence there was for a nosological distinction between autonomous (endogenous) and reactive (psychogenic; situational) depressions. Incidentally, the very same view had already been expressed eight years earlier by Mapother, Lewis' predecessor, on the basis of impressions obtained in clinical practice.
This established the scope of a debate which, since then, has been repeated with endless variations and which, even now, continues to exert a hold on psychiatry. Ironically enough, the seeds of controversy were sown by Kraepelin, the very person who most wanted to distance himself from the speculative impetus within the debate. In spite of his empirical bias, Kraepelin continued to link the clinically descriptive course criterion with the etiological hypothesis of endogeny. Through this, the reference to heredity became an established fact.
In practical terms, Kraepelin's coupling of the course criterion with the idea of endogeny raised two mutually related issues, namely:

1. the role of exogenic (biological and non-biological) factors in the origin of depression; and
2. the demarcation of manic-depressive illness with respect to psychogenic depressions and milder variants within the manic-depressive spectrum.

(Kendell 1968, p. 2; cf. Heron 1965).

Both of these issues ultimately proved to be insoluble within the framework of the endogeny/exogeny dichotomy. In practice, it was quite impossible to distinguish, in each case, between depressions which were psychogenically induced and those in which psychological and situational factors merely played an instigating role. The responsiveness of the illness proved to be of only limited significance as a distinguishing criterion. Meanwhile, it should not be forgotten that, in Kraepelin's time, the term endogeny was also an expression of embarrassment. At the end of the 19th century, Möbius introduced the twin concepts of endogeny and exogeny. By around 1900, these concepts corresponded respectively to diseases whose causes were still unknown and those whose causes had been well defined. Causes whose existence was (still) uncertain were described as endogenous. These were attributed, more or less out of embarrassment, to innate personal qualities (Lewis 1971). Exogenous causes included bacteria, toxins and tissue injury resulting from

brain traumas. In 1910, Bonhoeffer devoted a monograph to these so-called exogenic reactive types, and his name has been linked to them ever since.

III.H. The Influence of Psychology and Psychoanalysis

After 1920, mainly due to changes in the use of the adjective 'exogenous', this debate became more complex still. Originally used only in relation to biological factors, this term was extended to include intrapsychic and situational factors as well, thus highlighting the demarcation between depression and neurosis. New dichotomies consequently arose, such as: endogenous / reactive (Lange); autonomous / reactive (Gillespie); endogenous / neurotic; psychotic / neurotic. After the Second World War, these were supplemented by: S (somatic) type / J (justified) type (Pollitt); and vital depression / personal depression (van Praag).
Of course, this shift in the debate cannot be dissociated from the influence of *Sigmund Freud* (1856-1939) and the psychoanalytic school. In one of his early works (Manuscript G; 1895d), Freud had already addressed the neurological explanation of melancholia. In 1917, he published an excellent, authoritative article on the link between grief and melancholia (*Trauer und Melancholie*; Freud 1917). In this article, he stated that, unlike grief, object loss in melancholia was associated with unresolved feelings of ambivalence and regression of the ego. Anger at being abandoned would, then, be directed toward the ego which remained narcissistically identified with the other. This internal 'other' was then destroyed. Indeed, Karl Abraham had already demonstrated self-destructive behavior and experiences in depressive patients (Abraham 1912). From here, a line can be drawn via the work of Melanie Klein to authors such as Lindemann (1944) and Bowlby (1969; 1973; 1981). Klein distinguished a depressive position as a phase in early childhood development. Lindemann wrote an influential article on reactions to grief. Bowlby, as is well-known, worked extensively on the relationship between psychopathology and the processes of attachment and separation.
One should also mention, in passing, the influence of the theory of emotions developed by the philosopher *Max Scheler* (1874-1928). Scheler distinguished between four emotional levels or strata: the level of sensory, physical (or vital), psychic and spiritual feelings respectively (Scheler 1916, p. 344). This theory constitutes part of Scheler's own moral philosophy with its emphasis on values as non-subjective realities that are expressed in the in the interaction between a person and his environment. Scheler's theory had the advantage to account for the fact that people are capable of experiencing more than one mood and/or emotion at a time. A person can be in a dejected mood whilst at the same time being angry with his neighbour. *Kurt Schneider* applied this feeling theory to depression by stating that depression is based on a disorder in the vital sphere. Psychic feelings, such as feelings of guilt and inadequacy, would then be an "understandable reaction" to this vital disorder. The vital feeling of being depressed would co-occur with psychic feelings of guilt and worthlessnesss. This theory of feelings was held, albeit temporarily, in high esteem. The adjective 'vital' for endogenous depression, which till recently was in use on the European mainland, represents the psychopathological remnant of this theory.

III.I. Towards the Twin Pillars of DSM-III

Psychoanalysis enjoyed considerable influence for several decades, to such a degree that there was barely any perceptible movement in the classification debate. However this began to change with the discovery of the mechanisms of action of antidepressants, towards the end of the nineteen fifties. At that time, the effects of Lithium had been known for ten years whilst ECT had been in use

for more than twenty years. The advent of these new therapeutic drugs, raised the question of whether the biological active site of these drugs could be linked with specific target symptoms of depression (or mania). Gradually, it was accepted that this was indeed the case. Target symptoms, it was assumed, pointed to a given core disorder in the spectrum of depressive symptomatology. The increasing use of advanced statistical methods also had considerable impact on the debates. It enabled larger groups of patients, taken from adjacent diagnostic categories, to be investigated for clustering of symptoms (discriminant function analysis) and underlying factors or dimensions (factor analysis).

The outcome of these developments was not immediately obvious (cf. Akiskal and McKinney 1975). In summary, the classification debate gradually split into two separate debates after 1960. These concerned

(1) the distinction between manic-depressive psychosis and other forms of depression; and
(2) the distinction between 'endogenous' and neurotic depression.

The considerable overlap between these two debates served only to complicate matters, since the classificatory status of endogenous depression was central to both.

When highlighting some moments of this debate, mention should first be made of the clinical and genetic studies of Perris (1966) and Angst (1966). Both workers found that depressives with a previous history of mania had different hereditary profiles from those with no such previous history. On the basis of these findings, they concluded that the distinction between unipolar and bipolar depressions, which was already defended on clinical grounds by Leonhard in 1959, was valid. This distinction became one of the basic assumptions of the classification of mood disorders in the DSM-III (APA 1980) and its subsequent editions (APA 1987; 1994).

Kendell (1968), meanwhile, carried out a retrospective investigation of 1080 patients who had been admitted to the Maudsley Hospital. He could find no evidence of a bimodal distribution in the symptom profiles of a heterogeneous group consisting of manic-depressive patients, patients with neurotic depression and patients with involutional melancholia. However, just such a distribution was found in the so-called Newcastle study. Something common to both studies was the relatively high loading of a 'bipolar' factor.

As Kendell himself observed, his investigation did not negate the distinction between psychotic and neurotic depression. These could represent two poles of a continuum, with the psychotic pole displaying greater homogeneity than the neurotic pole. The simple fact that the symptoms of the neurotic side are milder and fewer in number contributed substantially to the reduced tendency towards clustering (Kendell 1968; 1976).

The lack of homogeneity at the neurotic depressive pole of the spectrum also found expression in the tri- and tetra-partite divisions of Klein (1974) and Paykel (1971) respectively. Klein distinguished an 'endogenomorphic' depression, as well as a reactive and a (chronic) neurotic form. Paykel distinguished psychotic depressives as well as three other groups: anxious depressives, hostile depressives and young depressives with personality disorder. Again, remarkably, some degree of consensus existed with respect to the psychotic or 'endogenomorphic' end of the spectrum. The limited extent of this consensus was due to the fact that, for the above-mentioned authors, the central issue was not bipolarity (cf. Perris, Angst and Kendell), but rather vital phenomena and psychotic symptoms.

It is, for this reason, not surprising that some clinicians opted for a centre-periphery instead of for a continuum model. Mendels and Cochrane (1968), for example, observed that:

> “the so-called endogenous factor might represent the core of depressive symptomatology, whereas the clinical features of the reactive factor may represent phenomenological manifestations of psychiatric disorders other than depression which 'contaminate' the depression syndrome”.

Ultimately, however, with the advent of DSM-III(R), neither the centre-periphery model nor the continuum model was to gain the upper hand. Instead, the winner was a twin pillar model, centering around the pillars of bipolar disorder and depressive disorder. In an article which appeared in 1974, Klein articulated an important consideration regarding this shift. He pointed out that 'endogenomorphic' depressions (those which give the impression of having arisen 'endogenously') occur particularly frequently in the group of neurotic depressives. As a result of epidemiological studies less emphasis was placed upon the heterogeneity of neurotic depressions. Conversely, greater attention was paid to the chronicity and severity of the disease. Finally, from the eighties, genetic investigations not only favoured the further demarcation of bipolar disorder, something which had already been advocated on the basis of factorial analysis studies, but also the distinction of dysthymia (neurotic depression) as a separate category.

In summary, it can be said that a consensus began to emerge, in which the most important demarcation line was drawn between bipolar disorder and unipolar depression. In addition, within the group of unipolar depressive disorder, a subdivision was created, roughly corresponding to the distinction between endogenomorphic and chronic neurotic depression, as described by Klein. Once again, ironically enough, the old concept of melancholia was even called upon for assistance, namely in the definition of the endogenomorphic (vital) form of 'major depression'.
It should be noted, by way of a cautionary remark, that this consensus was by no means universal. For example, it has still not been decided whether or not the categorical distinction between cyclothymia and dysthymia on the one hand, and personality disorders on the other, is an artifact. Besides, the debate about the demarcation between affective and anxiety disorders has become of particular relevance in the past decade. Longitudinal, familial and epidemiological studies have demonstrated that there is a high degree of co-morbidity of affective and anxiety disorders, both in the course of the illness and in relatives. However, it is beyond the scope of this chapter to pursue that particular story at this point.

I switch now to a discussion of some highlights from the conceptual history of anxiety and anxiety disorders.

4. HIGHLIGHTS FROM THE CONCEPTUAL HISTORY OF ANXIETYAND ANXIETY DISORDERS

4.1 The Demarcation of Agoraphobia

Prior to about 1850, as mentioned in section II, anxiety was not considered to be a distinct form of psychopathology in the medical literature. This is of particular importance to the recent debate on the demarcation between affective and anxiety disorders. For hundreds of years, the symptoms of anxiety had simply been seen as part of melancholia. In the course of the past century and the present one, the various forms of anxiety came to be distinguished from depressive disorders, on a variety of grounds. In the light of the century-old merging of anxiety and depression, a reconsideration of these grounds is therefore a matter of considerable topical interest. What is it, that explains the urge to accord the various forms of anxiety independent status within the total spectrum of psychopathological symptoms?
Historically, attention focused initially on phobias. Phobic anxiety, like other symptoms of anxiety, had been described in another terminology, already in the Hippocratic texts. Burton described agoraphobia, claustrophobia and fear of public speaking. Errera (1962) cites le Camus' *Médecine de l'Esprit* (Medicine of the Mind; 1769, pp. 259-265) and de Sauvages' *Nosologie Méthodique*

(Methodical Nosology; 1770-1771) as being the first medical studies in the field of phobia. The former includes a section on *'Des aversions'* (avoidance behavior) whilst the latter lists many different types of phobias. The term phobia was occasionally used in a diagnostic context even before 1850, as in the work of Benjamin Rush (1798) for example.
However, three publications which appeared around 1870 became particularly authoritative. The first of these was a short article by *Benedikt* entitled *Über Platzschwindel* (On dizziness on squares). Here, the author focused on a form of dizziness which, because of its characteristic symptomatology and treatability, in his opinion, merited a separate classification amongst the various forms of giddiness (Benedikt 1870). This article was to go down in medical history as one of the first descriptions of agoraphobia. As just noted, this is in fact historically inaccurate. Furthermore, the term *'Platzschwindel'*, was invented by Griesinger, not Benedikt. Nevertheless, by according a separate classification to the series of phenomena currently referred to as agoraphobia, Benedikt's article does mark a turning point.
Although Benedikt had observed anxiety in the patients he described, he believed this to be secondary to the dizziness:

> " ... however, as soon as they enter a wider street or (especially) a square, they are overcome by dizziness. They either become terrified of collapsing mentally or else they are gripped by such tremendous fear that they will never dare to pass through such a place again."
>
> (Benedikt 1870, p. 488; translation by the author)

Two years later, *Westphal* (1872) challenged the view that the dizziness was of primary importance. Westphal, who was the first to use the term agoraphobia in a technical sense, believed that anxiety, rather than dizziness, was at the root of this phobia. It was anxiety which caused the dizziness, not the other way around. Westphal's observation was the forerunner of a debate which was to be in full swing rather more than a century later. This debate centers around the provocative role of bodily sensations and of their interpretation, in the origin of panic attacks. Westphal based his hypothesis upon clinical observation, rather than on theoretical considerations. He imputed Benedikt's interpretation to the incompleteness of his investigations (Westphal 1872, p. 153). Interestingly enough, Westphal himself was very much aware of the fact that the three patients he described were certainly not afraid of streets or squares, as such. He stressed the unfounded nature of their anxiety. Their's was rather a fear of anxiety itself, an anxiety which only much later is linked to particular situations. Modern day authors who point out that agoraphobic anxiety is not a fear of streets or squares and that it occurs under all sorts of other circumstances, find an ally in Westphal.

4.2 Anxiety under Circumstances of War

In the same period *Da Costa* (1871) published an article on cardiac symptoms in exhausted infantry soldiers during the American Civil War. Da Costa, himself a cardiologist, spoke of an 'irritable heart'. Observations of more than 300 patients led him to believe that this condition was caused by a heightened nervous irritability of the heart. This, in turn, was caused by prior over-activity such as long marches, for example, or physical illness. During auscultation of the heart, Da Costa heard a weak and sometimes split first sound, a pronounced second sound and sometimes a systolic murmur. This systolic murmur has recently been related to the mid-systolic 'click' of mitral valve prolapse. Da Costa spoke of a sound "like the sudden motion of an only slightly elastic or cartilaginous substance" (*ibidem*, p. 26).
This classic article by Da Costa continued to stir things up amongst cardiologists, neurologists and

psychiatrists well into the middle of this century (Skerritt 1983). The debate became particularly intense during and after both World Wars, when, once again, tens of thousands of those departing the front lines were afflicted with the syndrome described by Da Costa. Thomas Lewis gave a figure of 70,000 such cases amongst British soldiers in the First World War, 44,000 of whom subsequently received a war pension (Lewis 1940). In addition to a report on this subject, which he drew up for the Medical Research Committee in 1917, Lewis also published a monograph in 1940, on the same theme. By that time many agreed with Lewis that the term 'irritable heart' was incorrect, since this placed a one-sided emphasis on heart palpations and on pain in the region of the heart. This suggested, wrongly, that the complaints were cardiac in origin. However, despite this consensus, there was still no unanimity about what actually lay behind the syndrome.

This lack of unanimity was also reflected in the nomenclature. Lewis introduced the term *effort syndrome* in order to emphasize both the intolerance to physical exertion and the syndromal character of the picture described by Da Costa. Whilst the clinical picture was indeed determined by cardiac symptoms, he found that only one sixth of the patients actually suffered from heart disease. Other terms which came into use were *soldier's heart* (MacKenzie 1916), *war neurosis* (Mackenzie 1920), *Da Costa syndrome* (Wood 1941), *neurocirculatory asthenia* (Oppenheimer 1918) and *heart neurosis*. Following rejection of the cardiac hypothesis, the controversy over etiology mostly centered on whether this picture was determined by psychic factors (Culpin 1920; Wood 1941) or whether it was a syndrome which could develop in more than one way (MacKenzie 1916; 1920; Jones and Lewis 1941; Jones 1948).

Mackenzie (1920) for example, blamed the confusion surrounding the condition (which he termed 'soldier's heart') on a consideration of some individual symptoms to the exclusion of all else and on the fact that this disorder had been named after its most prominent symptom (in this case, heart palpitations and pain in the region of the heart). Because of this, less prominent symptomatology tended to be disregarded. Mackenzie states that, according to the "law of associated phenomena", local disorders are usually accompanied by so-called reflex symptoms (see section IV.D) in other parts of the body. Moreover, long before the formulation of the attribution theory, he already emphasized the influence of medical terms on the way in which disease is perceived. By designating the systolic murmur associated with effort syndrome as an aortic valve defect, or a mitral valve defect, physicians could turn their patients into invalids. Intolerance to exertion would then wrongly be labelled as heart disease.

The debate was settled provisionally by two collaborative studies by a number of specialists from a section of the Maudsley Hospital which was rehoused at Mill Hill School during World War Two. A special unit had been set up at the school for the purpose of studying patients with effort syndrome. One of these studies was an (award-winning) work by *Maxwell Jones*, who was later to achieve fame as a protagonist of the therapeutic community (Jones 1948). In the other study, by the cardiologist *Paul Wood*, the author concluded that the symptoms of Da Costa Syndrome were also prevalent in peace time and that they closely resembled the symptoms of anxiety neurosis (Wood 1941). Although constitution, heavy exertion and previous infectious diseases could all be precipitating factors, effort syndrome was ultimately explicable in terms of (and was maintained by) a neurotic mechanism. It affects those who, in their youth, "clung too long to their mothers' skirts" (Wood 1941, p. 846) and who, either due to parental overconcern or to comments by their physician, learned to interpret various (normal) physiological changes as signs of physical impotence or even of danger.

Like Mackenzie, Wood placed great emphasis on the suggestibility of the patients, particularly on their capacity to interpret, in a negative way, the normal physiological changes which occur during physical exertion. Wood's interpretation was partly based upon physiological experiments which had demonstrated that peripheral sensation in effort syndrome was founded neither upon local abnormalities, nor upon a hypersensitivity of the central nervous system (such as in

hyperadrenalism), nor upon hyperventilation. There was therefore, in his opinion, no specific pathophysiological mechanism which could be held responsible for the physical symptoms. However, there did exist a specific psychological mechanism, which involved the association of exertion with all sorts of imaginary physical catastrophes. Wood believed that this association was mediated by an emotion, usually anxiety, although patients were generally not consciously aware of this. The therapy therefore consisted of a form of psycho-education in which people were informed of the physical manifestations of emotions and of the fact that physical sensations were not, by definition, indicative of a disorder in any particular organ.
Maxwell Jones concurred with this. He developed a form of group psycho-education, using groups of about 100 patients, which later evolved into the first therapeutic community. The aim of this therapy was to teach patients to adopt a different attitude towards their symptoms. In addition, Jones stressed the reality of this intolerance to exertion. Extensive physiological studies failed to provide an explanation for this intolerance, he therefore spoke of an effort phobia. Jones' investigations demonstrated namely that effort syndrome patients quit exerting themselves long before they reached their physiological limit, as expressed by a slightly smaller increase in the lactate level relative to a normal control group, following the subjective maximum of exertion.
The debate can be summarized by stating that the variation in nomenclature was determined not only by all sorts of theoretical views concerning the causation of physical sensations, but also by the immediate military importance of identifying and treating those suffering from war neurosis. Wars have contributed greatly to our knowledge, not only of anxiety disorders but also, for example, of traumatic neuroses and terror psychoses (cf. Bonhoeffer 1919; Kleist 1918). In addition, they have propelled this knowledge in a specific direction. Lewis' choice of the term effort syndrome was significant, as was his involvement in the development of exercise programs to increase the exertion tolerance of the soldiers he was treating. Both can be seen as a direct reflection of the military importance of the capacity to deliver physical effort (Lewis 1918). Lewis' effort syndrome is a splendid example of social influences affecting psychiatric diagnosis.
Regarding subsequent developments, it can be noted that the special relationship between anxiety phenomena and the heart persisted even after 1950. Whilst consideration was being given, in the sidelines, to the psychiatric mode of interpretation, the somatic approach continued to play a dominant role. The nineteen sixties saw the development of somatic concepts such as 'hyperkinetic heart syndrome' and 'hyperdynamic beta-adrenergic condition'. Recently, the relationship between anxiety disorders and mitral valve prolapse has been the subject of debate, as has the so-called fatigue syndrome (White 1989; Wessely 1990).

4.3 Anxiety psychosis

The frequent occurrence of anxiety with psychotic symptoms did not, of course, go unnoticed by 19th century psychiatry. *Wernicke*, however, was the first to use the term anxiety psychosis. In Wernicke's opinion, frightening cognitions, hallucinations, delusions and delusory ideas were the result, rather than the cause, of the emotion of anxiety. He explained the psychotic phenomena seen in anxiety psychosis by the intensity of the anxiety itself. The reverse was true of alcohol hallucinosis, where anxiety was the result of the hallucinations. Melancholia also differed from anxiety psychosis. So-called agitated melancholia, on the other hand, which was actually a variant of anxiety psychosis, was totally unrelated to melancholia (Wernicke 1895, p. 1020). The motor expressions which appeared in anxiety psychosis were interpreted by Wernicke as complications, rather than as a direct consequence of the anxiety.
In the ensuing debate, criticism was levelled at the term anxiety psychosis (Specht 1907; Forster 1910). Some questioned the worth of a classification which was based on the content of cognitions.

Anxiety-dominated cognitions were not restricted to anxiety psychosis, they also occurred in a variety of other psychiatric disorders. Wernicke himself had already made a distinction between anxiety psychosis on the one hand, and anxiety in paranoid delusions and in delusions of reference on the other. The distinguishing criterion - namely, that the cognition in anxiety psychosis must be an direct consequence of anxiety - proved not to be unequivocally applicable in practice.
Meanwhile, Specht's interpretation differed considerably from that of Forster. *Specht* felt that anxiety psychosis was a mixed (Kraepelinian) form of manic-depressive psychosis, in which motor agitation was an expression of the manic component. Like Wernicke, he drew a sharp distinction between anxiety and agitation, on the basis that the former frequently occurred in depression whilst the latter was commonly a feature of mania. Specht's view was that anxiety psychosis involved motor agitation as an expression of the manic component, accompanied by the anxiety and inhibition of thought as a result of the depressive component. For Wernicke, anxiety was the central characteristic of anxiety psychosis, for Specht it was agitation.
Forster, on the other hand, felt that the symptoms of anxiety psychosis could best be seen either as a variant of melancholia (melancholia agitata) or as the early stage of another disorder. Not only did Forster not want to separate anxiety from agitation, he also resisted Kraepelin's separation of manic-depressive psychosis from dementia praecox. In his opinion, anxiety was not so much an emotion which was difficult to define, but more a complex series of cognitions which cannot be expressed in words. This formal characteristic, i.e. a special type of cognitive complexity, was determined by the 'fundamental disorder' which Forster placed at the level of the organic cerebral substratum (Forster 1910, pp. 252-260).
In the ninth edition of Kraepelin's textbook, anxiety psychosis was still included under the 'Emotionelle Symptomenkomplexe' (emotional symptom complexes). It was defined as a dysphoric condition which cannot be sharply distinguished from depression. It was associated with anxiety, motor restlessness and psychotic symptoms (Kraepelin and Lange 1927, pp. 611 et seq). As a symptom complex, it could occur in all types of psychiatric disorders, including manic-depressive psychosis and dementia praecox. It could also be age-related, as in presenility and senility.
Two questions dominated this somewhat confusing debate surrounding anxiety psychosis. These concerned the relationships between emotion and cognition, and between anxiety and (psycho)motor agitation. On the first point Wernicke considered the anxiety emotion to be dominant, whereas Forster placed the cognition in the leading role. Specht's reference to the Kraepelinian idea of the manic-depressive mixed condition suggested a preference for the view that the affective component was dominant. On the second point both Wernicke and Specht made a sharp distinction between anxiety and agitation. As regards a distinguishing criterion for anxiety psychosis, Wernicke emphasized the anxiety, Specht the agitation. Forster allowed anxiety, agitation and desperation to intermingle, since he believed that there were insufficient empirical grounds for a sharp distinction between anxiety disorders, manic-depressive disorders and psychotic disorders. The background to this was Forster's rejection of the traditional classification (derived from faculty psychology) into thinking disorders, feeling disorders and disorders affecting the function of the will (Forster 1910, p. 260).

In the post-1910 literature, there are two publications which are also worthy of mention. These are G.E. Störring's *Zur Psychopathologie und Klinik der Angstzustände* (On the psychopathology and treatment of anxiety states) (Störring 1934) and K. Conrad's *Die beginnende Schizophrenie* (incipient schizophrenia) (Conrad 1958). Although neither work includes the term anxiety psychosis, both point out the fundamental significance of anxiety in the origin of psychosis. Both go on to describe a period of depersonalization, anxiety and anxious moods which often precedes the onset of psychosis. *Conrad* used the term *trema* to denote this anxious delusory mood. *Störring* described how this anxious delusory mood could lead to so-called 'objectivation' of anxiety. This refers to what

nowadays is called projection. Feelings of anxiety are no longer experienced internally, but transform into perceptions of a dreadful and mysteriously changed world. The background to this symptom, in the case of psychosis, is a disorder which affects the sense of identity. Psychotics are no longer able to perceive themselves as the source of meaningful experiences and activities. Feelings loose their natural bond with the I. As a consequence, they take on an enigmatic and indeterminate character. Whilst the patient does not necessarily experience anxiety subjectively, the world nevertheless changes in an obscure way, and appears to be terrifying, threatening and gruesome. Sometimes anxiety is experienced in flashes, in which case, according to Störring, it makes sense to speak of a delusory affect rather than of a delusory mood.
Meanwhile, with the virtual disappearance of the term anxiety psychosis from clinical usage, interest in anxiety symptoms in the context of psychosis had also faded (Schmidt-Degenhard 1986). However, studies pertaining to the occurrence of panic attacks in schizophrenia and in schizophreniform disorders are still published from time to time.

4.4 Neurasthenia

In the second half of the 19th century a new concept, neurasthenia, gained ground. *George M. Beard*, the American advocate of this idea, considered neurasthenia to be a functional disorder characterized by a deficiency of 'nervous force'. This deficiency could express itself in a multitude of symptoms, particularly at the level of the central nervous system, the digestive tract and the reproductive tract (Beard 1884; 1890). Although not highly prominent amongst these symptoms, morbid fear and phobia were nevertheless ranked amongst the most difficult symptoms to cure (Beard 1884, pp. 52-53; Beard 1890, pp. 50-67). Beard used analogies for nervous exhaustion such as that of a furnace lacking in fuel and of a battery losing its charge (Beard 1884, pp. 60-61). Central to the concept of neurasthenia was the lack of the strength and reserve to fight the disturbances of nervous function caused by stress. Beard's neurasthenia concept was closely linked with his vision of American society, which supposedly caused much greater over-excitation of the central nervous system than did European society. 'American nervousness', one of Beard's favorite synonyms for neurasthenia, was a typical product of an industrial society in which the upper classes were doomed to a hectic lifestyle.
Beard experienced just as little difficulty with the conceptual difference between the physical depletion of energy and the psychic feeling of exhaustion as did Freud, a decade later (cf. MacMillan 1976). He had observed that not only did neurasthenia patients tend to survive their own physicians, but that they were also capable of considerable mental effort. However this did not cause him to reconsider the difference between subjective feelings of exhaustion and an actual deterioration in achievements resulting from a lack of physical reserves. On the contrary, he stressed that, in a functional sense, there was actually something amiss, such as a hyperaemia of the cerebrum, the stomach or the prostate, for example.
Due to a lack of resistance, the functional disorder (which initially occurred locally) became transmitted to other regions of the body (irradiation). It therefore had no opportunity to develop into a permanent local abnormality. In Beard's opinion, this was not the case in healthy people where, occasionally, local over-excitation could even result in death. In the case of neurasthenia, the local functional disorder never exceeded the threshold of intensity beyond which permanent defects could develop. Irradiation not only explained the variable and migratory course, but also the multiplicity of symptoms. Amongst the symptoms included by Beard were the 'irritable heart', all kinds of phobias, compulsions, impotence, hyperaesthesia and an huge range of physical sensations. The irradiation of the local functional disorder occurred reflectively, and Beard thought that the sympathetic nerve played an important role here. This hypothesis of reflective nerve impulse

transport was one of the three basic assumptions in Beard's concept of neurasthenia. In addition to the reflex theory, there was the idea of the electrical nature of nerve excitation and the law of conservation of energy (cf. Rosenberg 1962).
Beard himself believed that it was open-minded observation which led him to the discovery of neurasthenia, and his descriptions do indeed bear testimony to his extraordinary attention to detail. He would take even the most idiosyncratic, subjective sensations quite seriously. The fact that neurasthenia had not been previously described was, in his opinion, due to the fact that neurasthenic patients are not to be found in hospitals or mental institutions. They should instead be sought elsewhere, neurasthenia being a disease of the street (Beard 1890, pp. 25-35).
Nevertheless, Beard overestimated his inductive powers, as is demonstrated by the above-mentioned three basic assumptions and the role which they played in his work. These assumptions constituted the guiding principle on which he based his attempts to forge a whole out of the positively exorbitant diversity of observations. Moreover, it was quite common in those days to think of psychic disorders in terms of an excess or a deficiency of (nervous) energy. Furthermore, as we have previously seen, ideas such as asthenia and irritability were already fashionable a century earlier (cf. Berrios 1990; Lopez Pineiro 1983, pp. 64 et seq). In 1848 W.B. Carpenter explicitly suggested the idea of a close relation between nervous energy and electricity. Thinkers such as Spencer, Fechner and Darwin subsequently elaborated this idea still further. Meanwhile, in the therapeutic sphere, the process of 'electrification' became quite popular (Russelman 1983, pp. 119-154). Nor was Beard the first to see a connection between lifestyle and functional changes in the central nervous system. The previously-mentioned theory of degeneration, which was very popular on the European continent at the time, provides still more far-reaching examples.
It is true to say that this fascination with the relationship between nervous energy and electrical phenomena was not unconnected with developments in the natural sciences. Its origin, however, lay in the Romantic Period. The intellectuals of the Romantic Period are known to have been strongly captivated by the living world's organic urge to develop and evolve. Early in the 19th century, the concept of natural force encompassed not only physical forces, such as motion and heat, but also biotic and psychic forces, such as the life force, growth energy and the urge towards further development. It is therefore not the case that the concept of physical energy was initially discovered by physicists and only later applied, in a metaphorical sense, to psychic symptoms. In the second half of the 19th century, the prevailing climate of thought, which was still dominated by the influence of the Romantic period, swung in a materialistic and mechanistic direction. This transformation, which was associated with such names such as *H. Helmholtz*, *E. du Bois-Reymond*, *E.W. Brücke* and *C. Ludwig*, was triggered by the discovery of the law of conservation of energy by *Robert Mayer* in 1842. It resulted in a differentiation being made between physical and psychic force, which suddenly breathed new life into the psycho-physical problem. At the end of the 19th century there was yet another swing, this time back in a (neo)romantic direction, whereby all sorts of vitalistic concepts gained ground anew. Beard's concept of 'nervous force' seemed to fit in with this neo-romantic pattern of a vitalistic mixture of psychic and physical forces.
In summary, it can be said that both Beard's description of neurasthenia, as well as the temporary popularity of this concept, cannot be understood from a purely medical perspective. Instead, one must consider the interaction between medical observations, theoretical opinions, philosophical traditions of thinking and various social changes. However, the fact that medicine concerned itself with neurasthenic patients at all was, to a great extent, a social phenomenon. When social pressure became too much for an individual's resilience, neurasthenia offered a medical excuse for taking it easy.

4.5 Anxiety Neurosis

The history of the classification of anxiety disorders since the time of Beard can be seen as a peeling-away of layers of the concept of neurasthenia. Anxiety neurosis was the first stratum to be laid bare under its surface. Next came all sorts of classificatory sub-divisions within anxiety neurosis (Tyrer 1984).

Kahlbaum's successor, *Hecker*, initiated the above-mentioned process in a classic article on anxiety states in neurasthenia (Hecker 1893). He had noticed that the anxiety attacks experienced by many neurasthenia sufferers were not accompanied by any subjective feeling of anxiety. There were also patients who did not show anything like the full range of physical symptoms. Hecker used the term *'larvirt'* (larval; larva-like) to denote this absence of a feeling of anxiety. The term *'abortiv'* was indicative of the interrupted, incomplete nature of the attack in terms of the somatic symptomatology. The picture described by Hecker bears a strong resemblance to the so-called 'limited symptom attacks' in present day literature on panic disorder. Citing Lange, a Dane who had formulated an interpretation of emotions which was practically identical to that of William James, Hecker stated that the absence of subjective anxiety in the attack was based on a kind of misperception. The physical symptoms were simply not recognized as expressions of anxiety. However, it was also possible for an attack to commence with just one of the somatic symptoms, before radiating to other parts of the body. The way in which Hecker described this irradiation betrays a relationship with Beard's reflex theory.

In 1895, *Sigmund Freud*, with reference to Hecker, joined the critics of Beard's broad concept of neurasthenia. However, in being more explicit about pathogenesis, Freud went a step further than Hecker (Freud 1895a; 1895c). He believed that demarcation of neurasthenia was essential since anxiety neurosis, because of its different pathogenesis, required different treatment. Neurasthenia was a disorder of the way in which the so-called somatic-sexual excitation was released, whereas anxiety neurosis was primarily a disorder in the psychic processing of such excitation. In the case of anxiety neurosis, Freud imagined that there was a build-up of pressure on the walls of the male seminal vesicles. When this pressure exceeded a given threshold, it was transformed into somatic energy and transmitted, via neural pathways, to the cerebral cortex. Under normal conditions, sexual 'fantasy groups' became charged with this energy, leading to sexual excitement (libido) and the pursuit of release. Anxiety neurosis involved a blockage in the psychic processing of this somatic sexual tension. Such a blockage might arise through abstinence, for example, or due to the use of coitus interruptus, or because sexual fantasies had simply failed to take shape. Somatic sexual tension was thus deflected away from the psyche (the cortex) and directed to subcortical paths, finally expressing itself as 'inadequate actions'. These 'inadequate actions' most characteristically occurred during an anxiety attack.

The pioneering article in which Freud detached anxiety neurosis from neurasthenia, includes a description of the symptomatology of the various forms of anxiety which is still valid today (Freud 1895a). Freud cited anxious expectation as the core symptom of anxiety neurosis. He also distinguished between specific phobias, agoraphobia, free floating anxiety and anxiety attacks. The latter were spontaneous in nature and were described as a purely somatic phenomenon (Freud 1895c, pp. 368-369). The aforementioned distinctions anticipated the now generally accepted classification of specific phobias, agoraphobia, generalized anxiety and panic disorders. Freud was not alone in anticipating DSM-III(R). As the authors of DSM-III(R) have acknowledged, striking similarities are also to be found in the sixth edition of Kraepelin's textbook (Kraepelin 1899; cf. Spitzer & Williams 1985). Furthermore, it is interesting that Freud considered agoraphobia to be characterized by a fear of panic attacks, and not by fear of streets or squares per se: "... ce que redoute ce malade c'est l'événement d'une telle attaque ..." ("... what the patient fears is the occurrence of such an attack ..."; Freud 1895b, p. 352).

Freud's reputation was not based upon his interpretations of anxiety neurosis, however. His second

theory of anxiety, in which anxiety was interpreted as a signal of inner threat, would ultimately have much greater influence (Freud 1926).
This second theory had already announced its arrival by around 1895, albeit in a somatic guise. Freud asked why non-processed sexual excitation should express itself specifically in the form of anxiety. In answering this question, a glimpse is afforded of something which would later be more explicitly developed as a theme. Unlike real anxiety, which was based on the perception of an external threat, neurotic anxiety was a reaction to inner threat. The core of this inner threat was an inability to process 'endogenously' created (sexual) excitation (Freud 1895a, p. 338). On another occasion Freud put it as follows:

> "Anxiety is the sensation of the accumulation of another endogenous stimulus, the stimulus to breathing ..." (Freud 1894, p. 194).

It is sometimes forgotten that elements of the above hypothesis also appeared in Freud's signal theory. There also the basis of all anxiety was biological helplessness, i.e. the helplessness of the child with respect to its own drive impulses (cf. Freud 1926, p. 68). Although the signal theory also concerns the satisfying of needs, it does not relate primarily to sexual needs but rather to those associated with the instinct for self-preservation (cf. Freud 1933, pp. 100-101). Object loss, the most clear-cut threat recognized by this instinct, becomes the psychological prerequisite for inducing the ego to give off a small quantum of anxiety in order to restore a favourable balance of pleasure and displeasure. The threat of object loss remains linked to the biological state of being at the mercy of one's drive impulses. This linkage is mediated by remembrance-symbols which, via separation and birth, ultimately refer to an archaic inheritance of hereditary anxiety responses. In anxious patients, the symptom of gasping for air is no longer seen as a mitigated orgasm but rather as the rudiment of the cry of a newborn child (Freud 1926, p. 168).
In a negative sense, Freud's second theory of anxiety was of great significance within the classification debate. His statement that anxiety was the 'loose change' of the neurotic conflict, illustrates the nosologically non-specific character gradually adopted by anxiety in his interpretations.
This was partly the reason why the classification of anxiety symptoms did not exactly receive top priority in the period between 1930 and 1960.
This is not, of course, meant to detract from Freud's exceptional merits. In the field of anxiety theory, these merits lay particularly in the concept of anxiety as a reaction to an inner threat. This idea, which was without precedent in Freud's days, permanently changed the face of psychiatry. Freud thereby gave a wholly individual treatment to the fundamental distinction between (object-less) anxiety and (object-linked) fear, a theme which for the rest was to find its way into psychiatry via another route.[6]
Freud's approach was not limited to the psychoanalytic school. In its interpretation of anxiety, cognitive psychology (in the style of Beck) built upon Freud's pioneering concept of anxiety as an inner threat (Beck 1976; Beck, Emery & Greenberg 1985).

4.6 Clinical Studies

After 1900, despite the almost universal recognition of the central position of anxiety in psychopathology, relatively few psychiatric monographs were devoted exclusively to anxiety and anxiety disorders. One exception was the exhaustive study by Störring, mentioned previously. Several authors occupied themselves with conceptual questions, based on clinical observations.

Goldstein (1929) and Kronfeld (1935) produced splendid articles which incorporated some fundamental concepts. Some other names which should be mentioned in this context are those of Hoche (1911), Kornfeld (1902) and Oppenheim (1909). Janet's *Les obsessions et la psychasthénie* (Obsessions and psychasthenia; 1903) and his *De l'angoisse à l'extase* (From anxiety to ecstasy; 1926) are worthy of special mention.
Next, reference should be made to several studies arising from particular theoretical points of view. These include not only the psychoanalytical studies by Stekel (1932), Bitter (1948) and Riemann (1961), but also the anthropological studies of von Gebsattel (1954a, 1954b, 1954c) and Tellenbach (1976). In addition, studies exclusively devoted to a particular form of anxiety, such as the innumerable publications on *'Schreckneurosen'* and *'Schreckpsychosen'* (from the German Schreck: terror), were carried out in the periods around both World Wars (cf. Bonhoeffer 1919; Kleist 1918; Panse 1952).
Seen from a broader perspective, the conceptual framework within which the debate on pathological anxiety took place was specifically determined by the intellectual legacy bequeathed by scientists and philosophers such as *Charles Darwin, W.B. Cannon, William James, Sigmund Freud* and *Max Scheler*. It is no simple matter to illustrate just how the various interpretations and schools of thought influenced one another. Accordingly, I will restrict my discussion to two themes, the *role of bodily perceptions in the origin of anxiety* and the further determination of *the distinction between anxiety and fear*.

With regard to the first theme, there seemed to be a significant resistance amongst clinicians to the *James-Lange* theory of emotions. Bodily changes, according to this theory, instead of resulting from subjective feelings, were actually the cause of the emotional coloring of sensory perceptions. Perception became emotion via the awareness of bodily changes (cf. James 1884, pp. 189, 204; 1890, p. 450). James himself was responsible for the subsequent confusion as to whether a temporal-causal relationship existed between bodily changes and emotional perceptions, or whether both occurred simultaneously. It was usually assumed (probably not entirely correctly) that James was postulating a temporal sequence between bodily changes and emotional perceptions. Clinicians, who also adopted this interpretation, criticized him on this point by pointing out the immediacy of the experience of anxiety. According to Störring, the experience of anxiety was not mediated by prior bodily perception. However, he was not entirely logical on this point since he consistently spoke of anxiety as a processing of, or a reaction to, sensations associated with specific organs (Störring 1934, 24, 32). Both *Kornfeld* (1902) (not to be confused with Kronfeld, who has been mentioned previously) and *Hoche* (1911) lodged objections, on descriptive grounds, against the idea of a temporal sequence between bodily change and emotional perception. Kornfeld, who himself suffered from neurasthenic complaints and panic attacks, enlivened his article with a description of one of his nightly panic-attacks. On awakening, he would first experience a severe feeling of anxiety without really knowing why he was frightened. Shortly afterwards he would become aware of bodily sensations such as a feeling of constriction in the region of the heart, difficulty in breathing, paraesthesia and cold *akra*. This led him to conclude that a sharp distinction should be made between feelings of anxiety and the perception of bodily changes. Although Hoche thought along similar lines, like Hecker he formulated a sort of interoceptive explanation for anxiety, in which anxiety was based on the misinterpretation of bodily sensations. Kraepelin and Lange's handbook, ultimately rejected James' theory of emotions on theoretical grounds, both because of its psycho-physical dualism and its disregard for central regulatory processes. An emotion such as fear of suffocation could be both somatic and psychological in origin. According to Kraepelin and Lange, the origin of this fear of suffocation (whether lack of oxygen, hypercapnia, acidosis or frightening events) was irrelevant to the quality of the emotion itself. In all cases, the central issue was a threat to the patient's existence as a biological entity rather than any perception

of bodily changes (Kraepelin and Lange 1927, p. 470). It should be noted, however, that James was too much of a Darwinist to be accused of psychophysical dualism.
In summary, it can be said that clinical psychiatrists mainly resisted the James-Lange theory because of their support for the primacy of clinical observation. Nevertheless, the anti-theoretical sentiment in these convictions sometimes led to all sorts of non-explicit theoretical views being smuggled in through the back door.

In discussing the second theme, that of the further determination of the distinction between anxiety and fear, consideration should first be given to *Kurt Goldstein's* observations of patients with organic brain damage. The majority of Goldstein's patients were victims of the First World War. He observed (1929) that, when faced with overly-complex tasks, these patients displayed a catastrophic reaction consisting of a wide range of physiological and psychomotor symptoms. Goldstein believed that, even though it was not subjectively experienced as such, this condition could best be interpreted as an expression of anxiety.
Whilst Goldstein's patients were unaware of the fact of their anxiety, the appearance of their physical symptoms coincided with the failure to accomplish their tasks. Strictly speaking, their anxiety was neither a reaction to failure nor a reaction to an awareness of failure. Anxiety - and this was the essence of Goldstein's interpretation - was quite literally the actual manifestation of failure. Goldstein concludes that generally spoken anxiety was the expression of a frustrated urge for self-realization.
This reference to the urge for self-realization was particularly popular amongst those contemporary authors who drew their inspiration from vitalism. Although similar references can also be found in Freud's later work (Freud 1933), it was actually the colossal presence of *Charles Darwin* behind the scenes, which inspired this line of thought. However, Goldstein was not thinking of the survival of the species, or that of the individual, in purely Darwinian terms. The urge towards self-realization was more than a purely biological reality. It also found expression, for example, in the productive creativity shown by children and adults in mastering the world. Anxiety was referred to as the "Erschütterung (des) Bestandes der Persönlichkeit" (Disruption of the stability of personality; cf. Goldstein 1929, pp. 415-416). Ultimately, however, Goldstein failed to fully clarify the conceptual status of the propensity for self-realization.
The task of formulating, more explicitly, things which Goldstein merely hinted at, fell to other authors. Here we find a scheme in which personality was divided into an impersonal substructure and a personal superstructure. The substructure was described in vitalistic terms whilst the superstructure was analyzed in terms derived from existentialist phenomenology. Examples of this can be found in work by *Arthur Kronfeld, Felix Krueger, Philipp Lersch*, and, to a lesser extent, *H.C. Rümke*. According to Kronfeld (1935), anxiety was based upon a deterioration of the personal superstructure. In extremely succinct terms, this disintegration was expressed as psychotic anxiety. However, the type of anxiety which Kronfeld initially had in mind was existential rather than psychotic:

> "Anxiety is the mental expression of the existential annihilation of the integrity ('Einheitsform') of the person. Its archetype is the fear of death, the anxiety related to vital destruction."
>
> (Kronfeld 1935, p. 378; translation by the author)

Such statements only become comprehensible when it is realized that Kronfeld rejected the link between anxiety and threat. Anxiety, in the true sense of the word, is not the counterpart of safety, but of meaning, of being a person, of 'one-ness', of living. The anxious person need not necessarily have a subjective awareness of this, however. Death has significance long before it becomes a

subjective reality. According to Kronfeld, the biological aspect of anxiety consisted of life fleeing from death. Such flight is in vain, however, and therein lies the source of all fear of life and of anxiety about fate. Therefore, with anxiety, it is necessary to pose the question of meaning (*ibidem*, p. 384). Kronfeld's criticism of the biologically inspired interpretation of anxiety was that it failed to do this.
In uncoupling anxiety from threat, Kronfeld may have gone a little too far, but his remark that indescribable anxiety often has the quality of fear of living, receives daily confirmation from the mouths of borderline patients.

4.7 From Dimension to Category

As has already been noted, the study of anxiety was not a high priority in the period from 1930 to 1960. In addition to the previously mentioned influence of psychoanalysis, which described anxiety as a non-specific phenomenon, the assumption that anxiety occupied a low position in the hierarchy of psychiatric symptoms also had a part in this (cf. Tyrer 1984). According to this line of thought, not only did anxiety occur in practically all psychopathological syndromes, it also marked the lower boundary of psychopathology, where this bordered on normality.
Jablensky (1985) adds to this that classification had traditionally been an area of interest for institutional psychiatry. An explanation for the relative neglect of the classification of anxiety disorders was that, as a rule, patients with neurotic anxiety were never hospitalized.
This status quo gradually changed towards the end of the nineteen fifties. I shall summarize a number of these developments.
The psychophysiological investigation of emotions continued along the lines of the James-Lange theory. In this context, Ax (1953) attempted to draw a distinction between the emotions of anxiety and anger, on the basis of their peripheral physiological symptoms.
The anxiolytic effect of benzodiazepines was discovered, resulting in a flood of research into the effects of these chemicals on the central nervous system (Sternbach 1980).
J. Wolpe (1958) introduced systematic desensitization as a form of behavior therapy, thereby giving new impetus to the treatment of people with anxiety disorders.
The British psychiatrist M. Roth (1959) described a form of depersonalization associated with severe anxiety and phobic phenomena. This was the so-called 'phobic anxiety-depersonalization syndrome'. Although it usually developed in the wake of a psychotrauma, this picture could sometimes occur spontaneously. The EEG's of just under one sixth of all patients revealed the presence of temporal-epileptic symptoms.
Finally, at the end of the nineteen fifties, D.F. Klein (who has been referred to previously) discovered that panic attacks in agoraphobic patients could be blocked using Imipramine (Klein 1964; 1980). This marked the beginning of a great flood of experimental, pharmacological, clinical, longitudinal, epidemiological, genetic and familial research into the existence and course of panic disorder.
With this boom in psychopharmacological research, increasingly stringent criteria for the definition of psychiatric syndromes were drawn up. This was principally for the sake of comparability between research groups. Thus, psychopharmacological and biological psychiatric research constituted a powerful impetus for the development of the *Feighner Criteria* (Feighner *et al*. 1972). These, together with the *Research Diagnostic Criteria* (1975; Spitzer, Endicott and Williams 1978) formed the basis of the DSM-III(R) (APA 1980; 1987). The emphasis on descriptive precision lead to the demarcation of various forms of anxiety and to an abandonment of the concept of neurosis, which was considered to be too vague. The 'depressive neurosis' of DSM-II (1968) became 'dysthymia' in DSM-III, falling under the affective disorders. Two types of hysterical neurosis, hypochondria and depersonalization, were classified under somatoform and dissociative disorders, respectively.

Neurasthenic neurosis was discarded. Anxiety neurosis, phobic neurosis and obsessive-compulsive neurosis were combined under the heading of anxiety disorders. Post traumatic stress disorder, a newcomer, was added to the anxiety disorders. The anxiety neurosis was subsequently split up into panic disorder and generalized anxiety disorder, whilst the phobic neurosis was divided up into agoraphobia, simple phobias and social phobia (cf. Spitzer & Williams 1985).

In spite of the non-theoretical nature of DSM-III, this change nevertheless heralded in a fundamentally different approach to the psychopathology of anxiety. DSM-III bode farewell, not only to the psychodynamic conflict model, but also to a broader tradition in which anxiety was associated with disorders in personality structure. It was replaced by a finely grained description and classification of more superficial symptomatology. Anxiety was no longer regarded as a consequence of the personality structure but primarily as a symptom in itself. This resulted in a switch from the predominantly dimensional or dispositional approach which characterized the neurosis model, to a typological or categorical approach to psychopathology.
Panic disorder represents the most outstanding example of this development, becoming "a microcosm of many of the classic controversies surrounding psychiatric research" (Gorman *et al*. 1989). Biological, psychoanalytic, behavioral and cognitive explanatory models now competed for priority in this debate (cf. Tuma and Maser 1985; Marks 1987; Barlow 1988; Glas 1991). At the moment, the question of what exactly should be classified (patients? theoretical constructs? diseases?) is once again highly relevant (cf. Blashfield 1986).

5. Summary

The preceding chapters cover only part of the history of anxiety and depression. Whilst this is partly attributable to the introductory nature of this review, the main reason is that this discussion has been limited to concepts alone.
Behind these concepts lies individual suffering, the often ineffective concern of those around the patient, and the numerous therapeutic efforts of physicians and paramedics. Although not covered here, this history is at least as worthy of our consideration as the one described above.
In the Introduction, it was stated that the history of anxiety and depression should be interpreted as the interplay of cultural changes and of changes not only in psychopathological symptoms per se, but also in their scientific interpretation. This interplay has, to some extent, been reflected in the preceding chapters.
Viewed in the context of the microcosm - macrocosm theme, which was current both in Antiquity and in the Renaissance, the mentally ill appeared as the personification of a disruption in the subtle balance of cosmic forces. Such people were different, but not so different as to warrant either ostracism or confinement.
The moralizing and allegorizing of humoral pathology in the Middel Ages raised the question of where disease ended and sin began. But it was difficult to know where to draw the line, as the 19th century theory of degeneration made clear. According to this theory, a transgression (due to alcoholism, for example) in one generation could lead to increasingly serious forms of psychopathology in succeeding generations.
The idea that melancholiacs show traits of the exceptional and of genius is a common thread running throughout history. Starting with Aristotle, it can be traced via Marsilio Ficino, through Burton and the poets of the Elizabethan Era, and onwards into the present century, in the work of William James. The fact that depressives' weaker perceptual defenses allow them a more adequate perception of reality may, perhaps, be a pale reflection of this very idea.
The rationalization and mechanization of the worldview in the post Cartesian Era finally resulted in a

view of the mentally ill in which the irrational and uncontrollable elements of their behavior received special emphasis. The bestial side of human nature revealed itself in such people. Right up to the present day, domination and control have been key words in understanding the motivation behind medical concern for the psychiatric patient. It is this very contrast with earlier treatments which illustrates the extent to which the medical domination of previously uncontrollable emotions has become both the motive and guiding principle for current theoretical and therapeutic activity. The current fascination of clinicians and researchers with the biological approach therefore comes as no surprise. Since this approach seems to bring the promise of control and of tangible results, in contrast to the unpredictable and much less concrete results obtained by psychological and social intervention.

It is appropriate to issue a warning at this point, however, since history has shown us how intractable psychopathological reality can be. It was not without reason that clinicians repeatedly demonstrated an astonishing eclecticism concerning the theoretical explanation of various insights. Clinicians showed reticence when it came to reasoning from preconceived theoretical points of view. I would remind you of the reservations expressed against the James-Lange theory of emotions. This reticence cannot simply be attributed to an anti-scientific attitude. It also stems from healthy clinical skepticism. The history of the classification of anxiety and depression serves to emphasize the fact that such skepticism was often quite appropriate. Whenever attempts were made to refine a given theory, or combination of theories, clinical reality always proved to possess an over-abundance of elusiveness and unpredictability. Broadly speaking, longitudinal clinical observation advanced the classification of anxiety disorders, and especially that of mood disorders, more than did any classification based on preconceived theoretical assumptions. In view of the controversy between the unitarians and the separatists, a combination of longitudinal and interdisciplinary (bio-psycho-social) research would seem to hold out particular promise for the future.

Finally, what has been said above should also be exphasized on epistemological grounds. As scientific disciplines, neurobiology and pharmacology tackle problems abstractively and objectively. This implies that there is, by definition, a gap between the research findings in these disciplines and clinical reality. Furthermore, scientific constructs never relate to this reality in its entirety, but merely to aspects of the whole. Reification, i.e. the identification of these constructs with reality, can only lead to distortion. The conceptual history of anxiety and depression illustrates the repeated recurrence of forgotten ideas. These were eliminated in the process of abstraction, only to return via the back door. Scientific interpretations only bear fruit when the tension-filled gap separating them from clinical reality, rather than being short-circuited, is kept open.

NOTES

1. Klibansky, Panofsky, and Saxl (1964, pp. 64-66 and 98-102) point out an irregularity in the classical interpretation of melancholia; this irregularity occurs in the theory of temperaments.
In the strict *humoralist* tradition, temperaments are reduced to disturbances in the balance between the bodily fluids. These disturbances initially appear to fall within the normal range, like the usual variations of character dispositions. However, changes in interpretation over the centuries become obvious: only blood is now considered to be an integral part of the body, with the other three fluids regarded as degeneration products. The significance of these degeneration products (yellow bile, black bile, and phlegm) would, especially in the Middle Ages, take on a negative connotation, as a result of which they would be considered to be responsible for all kinds of character abnormalities and immoral behavior.
The *Galenic* and *Neo-Galenic* theories of temperament, on the other hand, were originally oriented towards the primary qualities (heat, cold, dryness, and wetness), and not so much towards the humors. The Galenic temperaments are not in fact real temperaments, that is to say temperaments in a biological (humoral) sense, but rather disturbances in the balance between the primary qualities. Health was considered by Galen to be an ideal state; every relative excess of a particular bodily fluid was, to his mind, a disturbance and therefore also a disease. Thus the Galenic tradition had little influence on the development of a proper theory of temperament: it could not deal with normal variation of character dispositions.
Behind this irregularity in the theory of temperaments lies a difference of opinion about disease: the strict humoralistic tradition took a concrete and natural view of normality; whereas the Galenic tradition established itself on more abstract ideas, such as the primary qualities.

2. In Scholastic medicine, the emotions of anxiety and rage are diametrically opposed. In rage, heat is generated and floats to the periphery, the arms and legs display movement, the face becomes red, the pulse strengthens and the brain also comes to life again. In anxiety, the heat drains inwards, peripheral parts become cold and pale, the pulse rate decreases and the patient feels cold.

3. Plato distinguished four forms of godlike mania, namely, mania as the art of fortune telling (cf. the etymological relation between mania and *mantikè*, i.e. fortune telling); mania as ritual purification and consecration leading to the relief of disease and grief; mania in the sense of ecstasy inspired by the Muses; and finally mania in the sense of being emotionally moved by memories when looking at beautiful things.

4. Cf. Descartes, 1647, article 52: "... qui nous importent ..."; and Guéroult 1953, p. 253.

5. Kraepelin remained a dedicated experimental psychologist throughout his life. During his years in Heidelberg (1891-1903), the work which he carried out included experiments on the function of will (task performance, level of fatigue), cognitive capacities (distractibility, attention, memory, ideational association) and expressive functions (motor activity, handwriting, and language performance) (Kraepelin 1983, p. 71). During his Münich years (1903-1922), much to his regret, he was no longer able to find the time for experimental psychology, although he did give refresher courses for colleagues (*ibidem*, pp. 144, 145, 149).

6. The distinction between object-less anxiety and object linked fear which already had been formulated by the philosopher *Kierkegaard* (1844), was to be introduced by *Karl Jaspers* in 1946, in

the fourth edition of his *Allgemeine Psychopathologie* (General Psychopathology). In the ensuing years, it was developed as a theme in the anthropological school, which was oriented towards existential phenomenology.

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