Part I

Introduction

1

Classification of psychiatric disorders and their principal treatments

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A kind of thought compulsion, a logical and aesthetic necessity, insists that we seek for well-defined, self-contained, clinical entities; but unfortunately our subjective need is no proof of the reality of which we desire. (Hoche, 1910)

Despite many proposed candidates, not one laboratory marker has been found to be specific in identifying any of the DSM-defined syndromes. Epidemiologic and clinical studies have shown extremely high rates of comorbidity among the disorders, undermining the hypothesis that the syndromes represent distinct etiologies. Furthermore, epidemiologic studies have shown a high degree of short-term diagnostic instability for many disorders. With regard to treatment, lack of treatment specificity is the rule rather than the exception. (Kupfer *et al.*, 2002, p. xvii)

Introduction

Why, you may well ask, has a book about treatment found it necessary to begin with a section on diagnosis? Since the introduction of DSM–III (American Psychiatric Association, 1980), diagnosis has seemed to become the 'holy grail' of psychiatry. Yet currently, diagnosis is in the doghouse, as the quotations above, spanning nearly a hundred years, illustrate. But, even though we despair at regular intervals, we continue to want a nice clean system that allows psychiatric patients to be pigeon-holed by clever clinicians who then have both an explanation of a disorder and its solution. For the hope has always been that if a specific diagnosis is made correctly, that the proper and best treatment will follow almost automatically.

If only psychiatry were that easy! If it was, then this book would not be necessary, for from the proper diagnosis would flow the essential treatment. But in psychiatry, and we would venture to say in most other specialties as well (though perhaps not so readily acknowledged by other practising physicians as in psychiatry), the diagnosis not only does not point directly towards treatment but can become a source of major conflict between clinicians and patients, and lead to allegations of the generation of stigma, labelling and other counter-productive arguments.

Then why do we persist with trying to refine and rework diagnoses and diagnostic manuals, and furthermore, why give it pride of place in this first chapter? In psychiatry, the path from correct diagnosis to correct explicit treatment is neither straight nor unambiguous and probably depends upon a number of different factors. The first factor might be the way diagnoses have evolved or developed in psychiatry, especially since, but not solely because of, the development of the editions of the DSM or the ICD that were advanced in the mid 1970s and became established as the DSM–III in 1980 (American Psychiatric Association, 1980) and the ICD–9 (World Health Organization, 1975, 1978) in 1975 with its clinical modification in 1978. Both of these then new iterations of prior diagnostic manuals profoundly changed the way psychiatrists approached diagnosis on both sides of the Atlantic. Rather than making a diagnosis based upon a number of different factors that included not only the 'chief' or presenting complaint, the specific symptoms that surrounded or accompanied the chief complaint, and the associated physiological and somatic concerns and complaints, while also considering the patient's capacity for empathy, the nature of his rapport or stance or 'posture' in relationship to the interviewing physician, the congruence of his affect with the content of his speech, and his ability to step back and view and comment on his own behaviour, the psychiatrist using the 'new' post 1980s approach basically needed to consider only the chief complaint with its accompanying symptoms, since together they were thought to be part of the package of the patient's overall psychophysiological, i.e. biologically determined, disorder. Empathy, relationship to the interviewer or to other people, capacity for insight, even motivation to change took on less

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significance, if any at all, as the new psychiatry, determined to look and feel like the rest of medicine, moved rapidly down the one diagnosis-one treatment road. If you got the diagnosis right, the choice of treatment would be, as they say, a 'no-brainer'.

This process can be compared with travelling on an expanding limited-access highway system where all you needed to know was what exit to take. Get on the correct road and take the correct exit and you would speedily be led to your desired destination. Get on the right diagnostic road and you will soon arrive at your destination, the right treatment. If the things that you needed at a specific exit were not that close by and to make things even clearer and more precise with less chance for error, more exits leading to more highways could be built (or more diagnostic entities created) so that you could arrive at your destination even more rapidly and efficiently. Of course, one of the problems with the interstate highway system, at least in the USA, is that, from the highway, all the roads look the same; and even when you get off the highway, the interchanges, with their almost standardized or perhaps operationalized conglomeration of fast food chains and service stations (symptoms and checklists), all, at least from some distance, look the same as well. You get little impression of the people who live and work in that particular area. But venture perhaps no more than a mile from the interstate, and you will find towns and cities and country roads that carry with them the specific distinction, flavour, and even peculiarity of the people and the geographical areas that you are passing through. Such a diversion from the interstate may not get you to your destination sooner, and it may not even appear initially to lend any valuable information to the journey. Yet it may convey a completely different and more complex sense experience and, in turn, appreciation of the trip that the shorter, faster, more direct route, the route the map searches on the computer produce when queried, places little or no value upon.

The second factor is related to other specific ideas about psychiatry, diagnosis and biology that developed throughout the 1970s. There evolved a number of ideas that took hold beginning in the late 1960s that were to change psychiatry and the diagnostic process profoundly. There developed the belief that we could, with enough expertise and diagnostic rigidity, isolate very specific diagnostic entities in psychiatry; and further that these specific entities were separate and distinct from other diagnostic entities. For example, there evolved the idea that depression, i.e. mood disorders, could clearly, in many cases, be distinguished from anxiety disorders. There was even a specific test developed, the dexamethasone suppression test (DST), that was purported to be able to distinguish true melancholic depression from other entities. The title of that seminal paper in 1979, 'A specific laboratory test for the diagnosis of melancholia' conveys a good deal more about the wishes, not only of the authors, but of psychiatry in general, that there be specific laboratory tests that could help psychiatrists determine which patients had which diagnoses (Carroll *et al.*, 1981). If there was a biological or laboratory test that could help support that distinction, then psychiatry could have 'real' rather than imagined diagnoses (even though in most of medicine, there are actually very few diseases that have specific or pathognomonic tests that support their existence unequivocally). This is in no way to deny that the DST has gone on to become an important and useful measure of hypothalamic-pituitary-adrenal (HPA) activity and has led to many important areas of research and explorations into brain neurochemistry. And while the HPA axis is still thought to be overactive in some mood-disordered states, we now know that HPA overactivity may be a more general measure of an individual's reaction to stress and stressors rather than a specific laboratory test that reveals the presence of a specific mood disorder, or a mood disorder at all. What was originally proposed to be a specific laboratory test for a specific diagnostic entity turned out to be a laboratory test that cut across many of these so-called specific diagnostic groupings and appeared to be disordered across a number of conditions that all seemed to be linked together because of their relationship and reaction to stress. Stress certainly plays a role in many disorders, psychiatric as well as more purely medical.

Validity of psychiatric diagnoses

There has long been argument about the terms 'reliability', 'utility' and 'validity' of psychiatric diagnoses. Reliability, as a psychological construct, is the easiest of the three to resolve, as it is merely a measure of agreement between assessors of the same information. Thus for a diagnosis of a patient to be reliable, it is necessary for several people to see the same patient (preferable) or a set of proxy data (video recordings or transcripts) and show a level of agreement (measured by a standard measure of correlation) that is preferably above a level of 0.75 (Cicchetti & Sparrow, 1981). Confusion only arises when the lay interpretation of reliability is used (e.g. in a court of law counsel often asks if 'the evidence is reliable' when they really mean 'is the evidence valid', or can we really be confident that this evidence is a true record?). Validity is a much more difficult construct to achieve. Consider, for example, this quotation from two noted authorities on classification:

Table 1.1. Clinical Utility Total scores (CUTs) for some common psychiatric disorders

Diagnosis	Aetiology	Low comorbidity	Specificity of treatment	Natural history and course	CUT scores
Alzheimer's disease	−	+	+	+	3
Alcohol dependence syndrome	+	−	+	+	3
Generalized anxiety disorder	−	−	−	−	0
Adjustment disorder	+	+	−	−	2
Bipolar disorder	−	−	+	+	2
Schizophrenia	−	+	−	+	2
Dependent personality disorder	−	−	−	−	0
Bulimia nervosa	−	+	+	+	3
Social anxiety disorder	−	−	−	+	1
Obsessive-compulsive disorder	−	+	−	+	2

It is suggested that a score 0 or 1 renders the diagnosis suspect and ripe for reform.

We suggest, therefore, that a diagnostic category should be described as valid only if one of two conditions has been met. If the *defining characteristic* of the category is a syndrome, this syndrome must be demonstrated to be an entity, separated from neighboring syndromes and normality by a zone of rarity. Alternatively, if the category's defining characteristics are more fundamental – that is, if the category is defined by a physiological, anatomical, histological, chromosomal, or molecular abnormality – clear, qualitative differences must exist between these defining characteristics and those of other conditions with a similar syndrome. (Kendell & Jablensky, p. 7)

When current diagnostic practice is examined there can be only one, rather depressing, conclusion:

At present there is little evidence that most contemporary psychiatric diagnoses are valid, because they are still defined by syndromes that have not been demonstrated to have natural boundaries. This does not mean, though, that most psychiatric diagnoses are not useful concepts. In fact, many of them are invaluable. But, because utility often varies with the context, statements about utility must always be related to context, including who is using the diagnosis, in what circumstances, and for what purposes. (Kendell & Jablensky, p. 8)

Many, though not all, of the diagnostic concepts represented by the categories of disorder listed in contemporary nomenclatures such as DSM–IV and ICD–10 are extremely useful to practising clinicians, and most clinicians would be hard put to cope without them. Diagnostic categories provide invaluable information about the likelihood of future recovery, relapse, deterioration and social handicap; they guide decisions about treatment; and they provide a wealth of information about similar patients encountered in clinical populations or community surveys throughout the world. Diagnostic categories allow us to identify cohorts of like unwell people for whom we can collate their frequency in the population, their demographic characteristics, family backgrounds and premorbid personalities, their symptom profiles and the evolution of those symptoms over time found in the results of clinical trials of different therapies. Research can then take place on the aetiology of the syndrome. This is all very useful and often provides invaluable information, whether or not the category in question is valid. Its usefulness depends mainly on two things: (1) the quantity and quality of the information in the literature (which depends on how long the category has been recognized and provided with adequate diagnostic criteria and how much competent research the category has generated) and (2) whether the implications of that information, particularly about aetiology, prognosis and treatment, are substantially different from the implications of analogous information about other related syndromes. But in recognizing the merits of usefulness, we must not go too far and imply validity to the diagnostic edifice we have constructed; it is a pragmatic solution, not a real one, and new data may quickly sweep it away.

We then might consider the following in attempting to rate or score the strength or clinical utility of a given diagnosis.

Aetiology

A good diagnosis indicates the cause, preferably silently rather than expressing it in the diagnostic description. Our suggested requirement for a positive score (see Table 1.1) is that the diagnosis indicates clearly which aetiological factors, up to a maximum of three, are involved. Few psychiatric diagnoses attain a satisfactory level at present of this factor. The aetiological factors can include genetic, social and environmental ones, and at least two of the

main factors must be present in 90% of all cases. Conversely, the diagnosis cannot be made if these aetiological factors are absent.

Comorbidity

The original definition of comorbidity was 'the existence of two or more independent diseases in the same person at the same time' (Feinstein, 1970). In psychiatric classification this has been steadily eroded over the years so that now it is better defined as the presence of 'any distinct clinical entity that has existed or that may occur during the course of a patient who has the index disease under study' (Feinstein, 1970). Its absence indicates the 'zone of rarity that is considered to be essential for a valid diagnosis' (Kendell & Jablensky, 2003). Occasional comorbidity is to be expected even if a syndrome is clinically useful, but when it is extensive, it undermines the value of the diagnosis. We suggest that a lifetime comorbidity of 60% or more or a concurrent comorbidity of 40% or greater, the level required for extensive comorbidity among patients within a given diagnosis, raises serious questions as to the value of the diagnosis (and thus weighs against a positive score; see Table 1.1). The one exception is when the comorbid diagnosis is always secondary to the index one and can be claimed convincingly to be a consequence of its natural course.

Natural history and course

The natural history of most of the neuropsychiatric diagnoses is not known as therapeutic intervention is the rule and the interventions have, in most instances, modified that history (we hope in a positive direction). The study of the natural history of a given diagnosis might still be possible in lower middle income countries where treatment is in short supply. The course of any diagnosis, including its development over time in the presence of intervention (which may or may not be the same as the natural history), is now well recorded for most diagnoses. If we adopt a standard classification of the course of an illness (Frank *et al.*, 1991), this would include recovery, remission with episodic relapse, remission with frequent relapse, unchanged clinical state, intermittent deterioration, continuous deterioration and death. A good diagnosis should predict its course and help it to be separated from other conditions. We suggest that for a diagnosis to be useful (to score positively – see Table 1.1) at least 50% of cases should be allocated to one of the main groups of outcome after 5 years. A heterogeneous course is not a good diagnostic criterion.

Specificity of treatment

For the practising clinician a good diagnosis is an ideal treatment selector. It plots a strategy of management better than any other single item of information. This idea of specificity of treatment also had an important role in introducing new diagnoses. For example, the introduction of 'panic disorder' to DSM–III was influenced heavily by Klein's suggestion (Klein & Fink, 1962; Klein, 1964) that imipramine showed 'pharmacological dissection' in treating panic disorder successfully but in failing to treat generalized anxiety disorder. The term 'psychological dissection' can also be used similarly for psychological interventions. Not all diagnoses yet have successful treatments but they still allow a management strategy to be set in place, even if it is an inactive one, when a good diagnosis is made. For a diagnosis to be useful (and achieve a high score on Table 1.1), we suggest that it should lead to a specific intervention plan (SIP) in at least 70% of cases. A SIP should contain no more than three elements of intervention to retain the definition of specificity, and these should not be shared by other diagnoses.

Comparison of Clinical Utility Total Scores (CUTs)

The comparison of clinical utility of some common diagnoses is illustrated in Table 1.1.

The current state of psychiatric diagnoses

The belief that psychiatry could define, isolate and separate specific diagnostic groupings appears to not have held up over time. Indeed we might argue that in many respects it has failed us in this task. The notion that one can clearly separate depression from anxiety might be possible in the most severe of instances of each of those states, but in general, there is a co-mingling of these states. Clinical experience would seem to suggest that the longer people remain depressed, the more that mantle of depression that they carry appears to be at least tinged with, if not layered throughout, with anxiety. Further, the more chronically anxious a person appears over time, the more depressed he also seems to become. In fact many patients with chronic states of mixed anxiety and depression often seem to drift into the large category that now bears the label of personality disorder.

Thus the specificity and separateness of current psychiatric diagnosis, upon which the current DSM and the ICD not only seemed to depend but also simultaneously helped to promote, appears to need some reworking. This

does not mean that psychiatric diagnoses are non-existent, useless or unconnected to treatment; rather it says that much more study and work needs to be done in this area before any confident conclusions can be reached. By attempting to define precise and distinct diagnostic categories, the current diagnostic manuals have established certain well-defined areas for us to carve out and examine to see if the boundaries and points of distinction are correctly placed. Much important research and many significant biological and neuroanatomical discoveries would have been impossible if some genuine, categorical, data-based and empirically enhanced diagnostic system had not been created.

Creating diagnostic manuals is an iterative process, and we need to remain open to what it is that we actually see and experience clinically. From those observations there will come improvements in how we understand and utilize diagnoses. We need to remember that in general our patients do not read the DSM before they present to us (although given the ubiquity of the Internet, it does seem to be getting more common).

The idea of specificity of diagnosis was further driven by the rapid development of biological ideas into the practice of psychiatry. There were a number of forces that came together in the late 1960s and early 1970s to strengthen biological psychiatry and to promote the idea that there were biological underpinnings if not for all, for at least most, psychiatric disorders. As we proceed with the discussion, we want to emphasize that we believe that all feelings, thoughts, actions, cognitions and behaviours are rooted in and mediated by biological processes, lest the following discussion give the impression that we are 'anti-biological'. The success of medications developed and brought to market in the late 1950s and early 1960s conveyed new hope and offered a wide array of compounds for psychiatric practice. While the discovery of chlorpromazine opened the path towards much more precise pharmacological treatment of psychiatric illness, leaving the previous compounds with their weak non-specific sedating and mentally dulling effects way behind, the additional discovery of the antidepressants shortly thereafter heralded a way of thinking that was to revolutionize psychiatric practice.

Prior to the discovery of the antidepressants, the concept of depression, as put forth primarily by the psychoanalytic school, was based upon the idea that depression was anger turned against oneself, the result of a negative introject, because of a loss of an ambivalently felt loved object (person) (Jacobson, 1971). Unable to admit consciously or openly anything that could be considered negative or hostile towards the lost or departed figure, the depressed person turned those negative feelings against oneself and was thus able to preserve a positive memory or posture to the loved object. (One never, in some broad metaphorical sense, wants to say anything bad about the dead.) Then when Kuhn (1958) put forth the idea of a medication that was an antidepressant, psychiatrists of the day must have wondered how a medication, a biological intervention, could reverse the process of turning the negative aspects of one's ambivalence about a lost object away from the self, because such a cognitive process, at that time, was thought necessary to occur if depression was to be 'resolved' more than relieved. (To put these ideas into perspective, it can be pointed out that many people in psychiatry at that time believed that the success of electroconvulsive therapy [ECT] in depression was due to the fact that the ECT was a form of punishment that ultimately helped relieve the guilt the depressed patient felt about her anger towards the ambivalently loved but now lost object. The depressed person had now, through the pain and suffering of ECT, paid penance, as it were, to her guilt and now was able to recover and move forward!)

Max Hamilton (1960) was to redefine the assessment of depression and at the same time, perhaps unwittingly, shift psychiatry's attention, even more than perhaps the discovery of chlorpromazine and imipramine did, away from seeing things in psychodynamic or psychoanalytic terms and towards viewing psychiatric illness as a disease. These diseases had specific accompanying psychophysiological symptoms that were rooted in biology and formed a package that we would call a disorder. Hamilton's rating scale for depression (HRS-D; Hamilton, 1960) had nothing about introjects, ambivalence about lost objects, or turning anger against oneself, though it did have ratings for the mood state of depression, guilt and suicidality. But more importantly, by using the HRS-D, depression was to be defined primarily in physiological terms such as sleep, appetite, energy, sexual function and physical manifestations (or experiences) of anxiety. Further, these items within the HRS-D could be, in most instances, directly measured and scored. Different raters or clinicians could learn the scoring system and achieve reliability between them. Thus it was thought one could, with appropriate training and experience, be able to rate (and define) the degree of depression in one's patient and have it be related to the degree of depression in another patient of another therapist if both therapists had achieved a good level of reliability between them in the scoring of the HRS-D.

It was not an insignificant change when the categories that were identified as reactions (i.e. 'schizophrenic reactions') in DSM–I (American Psychiatric Association, 1952) became disorders (i.e. 'schizophrenic disorders') in

DSM–II (American Psychiatric Association, 1968). The idea of psychiatric illness was changing significantly. No longer were *reactions* thought to occur because the id overwhelmed the ego or because the defences failed to keep unwanted ideas out of the conscious mind or that mental conflicts (ambivalence) led directly to anxiety or somatic symptoms or conversion reactions. Rather, psychiatric *disorders* occurred because of biological irregularities or failures that were either predisposed and constitutionally determined or arose *de novo* because external events upset to a significant degree previous biological balances and/or compromises that the organism (individual) had attained.

The last 15–20 years have seen an explosion of biological research in psychiatry that now includes the most sophisticated aspects of pharmacological challenges, molecular biology, neuroimaging and genetic techniques. We have come much closer to understanding some of the biological processes that appear to be disordered in different disease states, but the precise relationship of those biological processes to the actual clinical symptoms and affects that the patients' experience still remains elusive. Further, there has been an explosion of new psychopharmacological compounds in the last 15 years that promise effectiveness equal to the older drugs (those discovered and brought to market in the late 1950s and through the 1960s) but whose side effects are purported to be much milder and more tolerable than those found among their predecessors.

But another explosion has occurred, an explosion that in many ways makes the organizing of this section on diagnosis somewhat challenging. That explosion has been the explosion in the number of specific psychiatric entities now in each of the diagnostic manuals, whether it is ICD–10 or DSM–IV. There are probably many reasons for the increase in diagnoses. One reason may be that the splitters currently rule the psychiatric nomenclature process. These are people who honestly believe that in breaking psychiatric diagnoses into smaller and smaller categories, we may find more precise (and hopefully more successful) treatments for each of the categories. Another reason may have to do with the issue of promotion of drug treatments. The more diseases that are available, the more pharmaceutical companies have the opportunity to show effectiveness of their compounds, and the more marketing mileage they may be able to gain in their attempts to promote the uniqueness (or the broad applicability) of their particular compound(s) when compared with those of their competitors. A further reason may be that the greater the number of narrowly defined diseases for which there can be developed evidence for specific treatments, then the more readily these diseases and disease states will be accepted as legitimate by third party payers or by governmental agencies who want to know that their dollars or pounds or euros are being spent on the treatment of specific, well-defined entities rather than some global, somewhat nebulous concept of disease or unwellness rather than illness or disorder. Some more cynical observers note the exponential increase in diagnoses (and consequent increase in size of each new DSM volume) as a commercial matter, and regard the initials, DSM, as now standing for 'Diagnosis as a Source of Money' – for the American Psychiatric Association (Blashfield & Fuller, 1996).

Yet, while trying to reify these concepts of specific diseases and specific treatments, we found an opposite effect to that expected. Rather than finding that specific treatments worked only in specific diseases, we found the opposite, that specific treatments seem to work across a wide array of diseases. We turn once again back to the idea that in the 1970s there was the belief that in most instances depression or mood disorder was separate and distinct from anxiety disorder. One would have assumed from those studies in the 1970s that eventually we would find that the treatments, especially the biological treatment, for each of those large groups of disorders (or disorder categories) would be separate and distinct as well. But the opposite has happened. We now use the selective serotonin reuptake inhibitors (SSRIs) for a wide array of conditions that fall under the categories of both depression and anxiety. And further, the older tricyclic antidepressants (TCAs) that, as their name indicates and as Kuhn in 1958 so identified them, were thought to be specific for depression, are now used in a wide array of anxiety disorders as well. And the purported biological neurotransmitter activity is not the same between the TCAs and the SSRIs, for one is thought to interact primarily (though not exclusively) on the noradrenalin (norepinephrine) neurotransmitter system (TCAs) while the other is thought to interact primarily on the serotonin neurotransmitter system (SSRIs). This does not mean that these two neurotransmitters systems do not interact, perhaps very intimately and subtly, with one another. But the idea of one disease, one medication, or the idea of pharmacological specificity to accompany diagnostic specificity (described as pharmacologic(al) dissection by Klein, 1964), is being eroded as we learn more and more about various treatments and their tendency for their use to spread far beyond their original diagnostic targets.

Matching diagnosis, syndrome and treatment

Reflecting on these issues about diagnosis and specificity, we must arrive at some way to associate the current plethora of psychiatric diagnostic categories to provide a

coherent and useful guide to treatment, if we are really going to live up to the title of this book. Our goal is, once again, to look at treatments in their broadest sense and to consider how each of them can be used to their maximum value. We do not want to be constricted too much by a diagnostic system that, while well intended, has turned out not to have fulfilled the promise of clarity and specificity all of us once hoped for. But we also do not want to give an unfair description of a treatment as effective when it is clearly ineffective if given for the wrong condition.

For this reason the evaluation of the general effectiveness of each of the main categories of treatment (and the more specific variations of treatment within the category) in psychiatry are presented in Part II. We leave the discussion of the specific value of each treatment to Part III, where we describe each treatment, sometimes in the context of a diagnosis and sometimes not, and summarize the evidence for each diagnosis in a concluding table for each chapter. Despite our criticisms of diagnosis, it is still the norm for clinicians, and indeed patients, to identify disorders and then to compare the treatments for these. Thus in practice we are pitting each treatment against all others in the relevant diagnostic group even when the diagnosis is of limited value. This is our best judgement as to how to approach the best way of testing effectiveness given all the limitations and gaps that we have in our knowledge base. Both practitioners and patients are keen on getting the best possible deal from the treatments available, and require their problems categorized in some way, so the competition is as much between treatments by diagnosis as by treatment in general. In choosing a treatment we also have to take into account other factors, particularly adverse effects, which may be complex to interpret as one person's poison may be another's elixir of life. Other relevant factors include comorbid medical illness, age and costs. Indeed, cost-effectiveness is rapidly becoming the watchword by which every treatment is being evaluated.

So we are left with a bit of a mishmash when it comes to fixing the current place of a treatment in psychiatry. Sometimes the treatment is so clear in its description that its diagnostic 'tag' appears to be unimportant; for others the treatment may be specifically linked to one diagnosis only. Thus in medicine penicillamine is used specifically to treat Wilson's disease (as this drug is a chelating agent that reduces the absorption of copper whose accumulation in the body becomes the manifestation of the disease). But a drug such as the benzodiazepine diazepam, despite being a sedative drug that reduces anxiety, can be used in many different ways because it is also an anticonvulsant as well as a muscle relaxant, so it can, therefore, be used for a wide number of disorders. And if we turn to the symptom of anxiety, we find that it is such a prominent component of so many disorders, its main treatments can appear again and again and sometimes be in danger of duplication.

In deciding on which chapters should be confined to describing treatments only (Part II) and which to diagnoses and their treatments (Part III), we hope we have chosen correctly. Part II is concerned with the main modalities of treatment whereas Part III describes individual therapies, sometimes closely linked to standard diagnoses (e.g. panic disorder), and some by groupings that reflect experience in practice but which are not necessarily in DSM–IV and ICD–10 (e.g. the section of organic disorders). The choice of these has not necessarily been an easy process, and we and our editors for each of the diagnostic areas have not always met agreement in how we defined our sections. This lack of agreement again reflects the fact that while much has been accomplished in psychiatric diagnosis in the last 25–30 years, much still resides in the realm of opinion. Much more needs to be done in order to understand the relationship between what we frame as categories of psychiatric diagnosis and the treatment or treatments that for some of the patients within those categories appear to be effective.

Models of treatment for mental disorder

This subject is relevant when it comes to choice of treatment for any disorder. The selection of treatment will depend to some extent on the model each practitioner uses for mental disorders. These have been summarized as the disease, psychodynamic, cognitive-behavioural and social models (Tyrer & Steinberg, 2005), and they can be viewed as a hierarchy (Figure 1.1). Each of the models on its own is unsatisfactory but together they can be very useful. The disease model is the equivalent of the well-established medical model of common parlance and is well-suited to organic disorders as it is associated with demonstrable (physical) organic pathology, either gross or accessible by microscopic means. Once a disease is clearly present it allows four elements to be identified relatively clearly:

(1) The description of symptoms and main features of the disorder (the clinical syndrome matching the underlying pathology).
(2) Identification of the specific pathology (i.e. the structural or biological changes created by the illness).
(3) Study of the course (natural history) of the syndrome.
(4) Determination of its cause or causes.

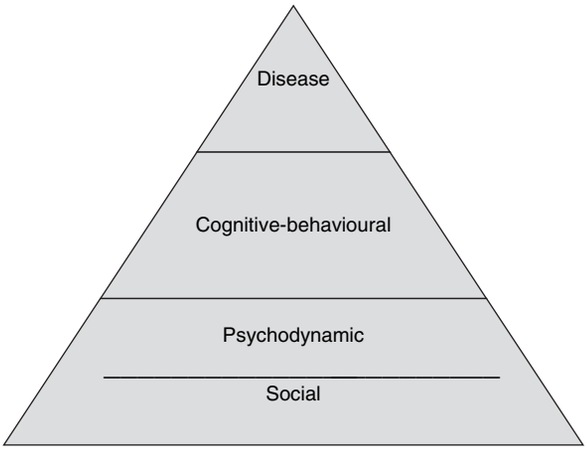

Fig. 1.1. The hierarchical model of mental disorders. Like all hierarchical models each level includes all disorders at that level and subsumes all those below (from Tyrer & Steinberg, 2005).

Although some disorders (e.g. Korsakoff's psychosis, Huntington's chorea) can satisfy all these requirements, most conditions encountered in psychiatry do not, and many do not get beyond the first element. The psychodynamic model does not even accept this element and maintains that the presentation of the complaint is a coded message that requires much further analysis (psychoanalysis) before a real understanding of the problem (or conflict) can be understood. The cognitive behavioural model blurs the distinctions between the disorders and examines the extent to which cognitive misinterpretations and distortions are present in the condition, so that, for example, in generalized anxiety disorder and obsessive–compulsive disorder the error may be in thinking, whereas in personality disorder the misinterpretation may be at the level of fundamental beliefs or schemas (Davidson, 2000; Tyrer & Davidson, 2000; Young *et al*., 2003). The social model abhors all diagnosis as stigmatic labelling, and that any advantages that they enjoy in terms of professional communication are more than offset by the depersonalization of diagnosis.

In practice most psychiatrists like to claim they are eclectic (i.e. they choose whichever model most fits the problem), but in the absence of clear guidelines this just looks like opportunism. However, by following diagnostic procedures and selecting treatment by diagnosis they are often accused by other practitioners and patients of following the 'medical model', a rather broader definition of the disease model described above. The proponents of alternative models are often dismissed or ignored but should not be. The simple fact is that a treatment that is not perceived by either therapist or patient to be in the right 'frame of management' will rarely be effective in practice because it will not be followed. This is very important when considering the evidence base of different treatments. If a psychological treatment is marginally inferior to a drug treatment, but the patient and day-to-day therapists concerned are violently opposed to drug treatment, then it is desirable, one might say essential, for the psychological therapy to be chosen. It may not be the best treatment in an ideal world but pragmatic decisions are the best for such situations. Time and again in the ensuing pages the reader will come across treatments that are *likely from present evidence* to be very similar in efficacy (the reason for putting the words in italics is that so many treatments deriving from different models are seldom compared in randomized controlled trials). Under such circumstances the treatment that best fits the patient's perception of the correct treatment is probably the one that should be chosen.

Choosing treatments from diagnosis: the example of mood disorders

In setting forth the details of specific treatments in Part III, we have acknowledged the significance of diagnostic practice and have followed the standard order of classification in numbering our chapters. So we begin with the organic disorders, the true repository of the disease model, and move through substance disorders, to the schizophrenias, mood disorders and neurotic (yes, we do still use this word occasionally), anxiety and stress-related disorders to eating disorders and the rest, ending with child psychiatric disorders. Only intellectual disability is a little out of the standard order, with only Chapters 10 and 44 addressing specific treatments for this group, and unfortunately the evidence base here is very thin.

We do not have space to go through the relationship between treatments and every diagnostic group in this section as we would repeat ourselves, but it might be helpful if we concentrated on one group as an illustration. We have chosen the category of mood disorders for this exercise. We begin with a discussion, actually a form of text tabulation, of the ways the ICD and the DSM diagnose the mood disorders. We elucidate the categories and the subcategories, and we then try to point out the similarities and differences between the two systems. We then go on to discuss the various limitations to those diagnostic categories and subcategories, limitations and qualifications that will influence the practitioner and patient when deciding on effective treatments for this diagnostic group.

We could do this for each of the diagnostic categories, but that would, in essence, be another book in itself, and that book would be on the process of diagnosis rather than on the effectiveness of treatment, which is the focus of this text. We have chosen depression as an example because it is fairly ubiquitous, appears in many different forms and intensities or severities, and can be viewed in some people as arising endogenously from some internal dysregulated (we presume) source, and in other instances appears as a most human and natural reaction to loss, failure, pain, humiliation and disappointment.

The ICD–10 classification of mood disorders encompasses the major subcategories of manic episode, bipolar affective disorder, depressive episodes, recurrent depressive disorder, persistent mood disorder, other mood (affective) disorder, unspecified mood (affective) disorder. In a number of the major subcategories, the word 'mood' can be substituted with 'affective', which implies more of mood change or fluctuation and can include mood depression and elevation, and is not necessarily reserved for a 'negative' or 'depressed' state. Each of these major subcategories has further categories embedded or subsumed under them, but for purposes of economy, we will not list or review the categories labelled 'other' or 'unspecified', but we assume that in every instance such non-specified groupings are available to the diagnostician.

- **Manic episode** also includes mania with and without psychotic symptoms, and hypomania.
- **Bipolar affective disorder** includes a current episode of hypomania, episodes of mania with and without psychosis, a current episode of mild to moderate depression (with and without a somatic syndrome), severe depression with or without psychotic symptoms (and if psychotic symptoms are present, the psychotic symptoms can be classified as mood congruent or incongruent), a current mixed episode, or bipolar disorder currently in remission.
- **Depressive episodes** includes mild and moderate episodes (each with or without somatic syndrome), or a severe episode with or without psychosis (and if psychotic symptoms are present, the psychotic symptoms can be classified as mood congruent or incongruent).
- **Recurrent depressive disorder** includes recurrent episodes with the current episode being defined as mild or moderate (each with or without a somatic syndrome), or severe with or without psychosis (and if psychotic symptoms are present, are the psychotic symptoms mood congruent or incongruent), or a history of recurrent episodes now in remission.
- **Persistent mood disorders** include cyclothymia, and dysthymia.

The DSM–IV(TR) (TR stands for 'text-revised) category of mood disorders has two large subcategories, depressive disorders and bipolar disorders. For each of these categories, there are codings for subgroups, and the subgroupings have to do with severity (mild, moderate, severe with the severe category being divided into with or without psychotic features and the psychotic features being mood-congruent or incongruent), and remission (subcategories that would specify partial or complete remission).

- **Depressive disorders** include a single or recurrent depressive episode(s) and dysthymic disorder that can be further broken down into early or late onset dysthymia and whether there are any atypical features within the dysthymic disorder.
- **Bipolar disorders** include bipolar I disorder which then includes both single manic episode (with the opportunity to delineate whether the current and or the most recent episode is purely manic or mixed with depressive symptoms), bipolar II disorder (with the opportunity to delineate whether the current and or the most recent episode is purely hypomanic or mixed with depressive symptoms), cyclothymic disorder, and mood disorders secondary to either a general medical condition or substance-induced. In either of these two 'secondary' mood disorders, there are opportunities to describe the affective episode in terms of whether or not the episode has primarily depressive features including features so severe that they resemble a major depressive episode or manic features or features that are mixed. Included within the substance-induced mood disorder category is whether the affective features begin during the process of, including reaching the state of, intoxication or appear to begin during withdrawal.

But how are we to consider these multiple categories in a book on treatment? How do we define or cluster these various categories in order to make some sense as we proceed to discuss treatments, and to consider, in general, what treatments apply to these clusters that we determine.

Both the DSM and the ICD have probably brought a number of disparate, but consolidated through the common symptoms of either mood depression, mood elevation (mania or hypomania) or mood lability. They have also tried to classify or subclassify each of the mood presentations into the following:

- Is the disordered mood episodic or chronic?
- Is the episode or the disordered mood state mild, moderate or severe?
- If mild or moderate is there a preoccupation with bodily symptoms and function (though such a preoccupation might lead one to think about anxiety disorders as well)?