Chapter 1

The General Neurotic Syndrome

As this book is primarily about the general neurotic syndrome (GNS), I need to be convincing in creating the groundwork to persuade the reader to continue to read. Some may feel this syndrome is a fictitious creation and so I will have to work even harder to persuade these sceptics; all I would ask at this point is for people to have an open mind. The general neurotic syndrome is not (yet) a familiar term, even though it should be. As it has been a subject I have had in my head for over 45 years – I hope not as an obsession but as a guiding light – I need to put my thinking about it into context.

1.1 Initial ideas

In the 1970s, when I was working in Southampton as a senior lecturer, my view of the general neurotic syndrome was very simple: 'If a person has both anxiety and depressive symptoms and some personality disturbance, the diagnosis of the general neurotic syndrome is the best way of defining the problem.' At this stage I was not certain how to define anxiety or depression or the exact nature of the personality disturbance. In defining it in this way I was also aware from my clinical practice that people with this syndrome tended to have poor outcomes.

This was a fairly limited definition, or rather an initial hypothesis that needed testing, and at the time I was not thinking of other disorders within the neurotic spectrum. Many people had theories about these and were prolix in expressing them. Alfred Adler, the well-known psychoanalyst, described most mental disorders as related to the desire to exercise power and overcome inferiority. He dwelt on this subject in writing about the 'neurotic constitution', an escape from the failure to become powerful. The neurotic symptoms each had a significant personal meaning for the patient; they became fictionalised and detached from reality (Adler, 1921). This led to the idea of a unified neurotic syndrome ('Die Einheitsneurose') but was not developed further and never really embraced in classification.

At this time, the view of 'neurosis' as an entity in clinical practice was an inchoate one in my mind. At its simplest level, it was manifest as a split between disorders in which common understandable symptoms are excessive but grounded in reality (neurosis) and those where bizarre inexplicable symptoms and behaviour are divorced from reality (psychoses). My view at this time conformed to this general idea but with the addendum that core neurosis was linked to personality.

But this was thrown into debate by what is now commonly called the Newcastle Group, a research mix of academics and clinicians together with a statistician, Roger Garside, who claimed that the common disorders of anxiety and depression could be separated using appropriate statistical methodology, and that clinicians should be able to make a primary diagnosis of anxiety or depression, with no need for both (Gurney et al., 1972; Roth et al.,

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1972). The authors were unequivocal in their conclusions: 'the first component extracted from a principal components analysis of the data was bipolar, with anxiety symptoms at one pole and depressive symptoms at the other; maladaptive personality traits were mainly associated with anxiety symptoms. This finding confirms that within an affective material there are two distinct syndromes corresponding to anxiety and depression' (Roth et al., 1972, p. 158). And again, 'using discriminant function analysis the bimodality of the patients' scores (i.e., clear separation between groups) indicated that there were two distinct groups, which, moreover, corresponded closely to the clinical differentiation into anxiety state and depressive illness, thereby confirming the hypothesis' (Gurney et al., 1972, p. 165).

But did these findings tell us that anxiety and depression were separate disorders? No. Anxiety and depression were identifiable as distinct entities but were they distinguishable in practice and did this have clinical meaning? Was the evidence of separation just a statistical method to separate symptoms but not patients? There has been debate about this ever since, increasingly unsupportive of this notion over the years. Perhaps the most economical summary was Dobson's, who made a full review of the literature and ended with a delphic summary. 'The distinction' between anxiety and depression 'may be more conceptually satisfying than empirically demonstrated' (Dobson, 1985).

But there is no doubt the Newcastle Group had stirred the neurosis pot from its position of quiet somnolence. Discussions about the anxiety/depression distinction and its value in selecting treatment (Kerr et al., 1972; Schapira et al., 1972) became very common in clinical practice. I recall arguments at ward rounds in Knowle Hospital near Fareham in Hampshire; vigorous discussions where junior colleagues were castigated for not committing themselves to a single diagnosis in a patient who had both symptoms of anxiety and depression. Sometimes the argument that as personality disturbance had been described in the presentation of the case this showed the diagnosis must be an anxiety one, 'as Professor Roth has said so'.

My own view of the work of the Newcastle Group, given nearly 50 years of reflection, is that it was rather like shining a light into a dark corner for the first time. You are not quite sure what you are seeing but you carefully sketch what you can and report back. But you know you are missing a lot. It was a pity that the Newcastle Group's study only included inpatients (most patients with primary anxiety disorders rarely go into hospital) and did not test their hypotheses with another equivalent group of patients. They were also using statistical approaches that were relatively new to psychiatry but not fully understood, and in retrospect should not have been relied on to justify their arguments.

And what was the real purpose of the study? The exercise seemed almost like comparing two kinds of eating apples such as Worcester Pearmain and Cox's Orange Pippin. Visual examination reveals consistent differences between the two types of apple but when you take the broader picture the two apples are virtually identical. They come from a similar looking tree, belong to the same botanical species, and have very similar textures and flavours. Of course, it is possible to separate one from the other using the highly discriminant analysis of observation and so the two types of apple can be separated into different baskets and sold at different prices, but the fact remains they are eating apples with many more similarities than differences.

During this period I had also been involved in comparing the effectiveness of day hospitals and outpatient clinics for the treatment of anxious, phobic, and depressed patients (Tyrer & Remington, 1979; Tyrer et al., 1987). Our findings in these studies showed consistency of phobic symptoms but great inconsistency of anxiety and depressive ones over a two-year period. This reinforced the notion that the universal separation of anxiety and depression was not a useful clinical practice.

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It is reasonable to ask why this subject seemed so important to me at that time, as some might find the whole issue esoteric. I had been trained in medicine and had completed higher training in the subject and had always felt that diagnosis was a very important medical task – one that could only be taken on by a doctor at that time. If the most common conditions in mental health could not be properly diagnosed, what hope was there for psychiatry? (This, of course, was at a time when diagnosis was considered a critical part of psychiatric practice; the doubts that are being expressed today hardly existed at that time.) I do not want to give the impression I was moving towards a catastrophe in my thinking but the subject had to be addressed if I was to feel confident as a practitioner.

1.2 Later Stage (1980s)

A more coherent formulation of my view of the general neurotic syndrome was made in the early 1980s, but it was created as part of a concept not too different from 'Die Einheitsneurose'. In 1985, I wrote a paper on the subject in the *Lancet*. The Lancet always prides itself on being at the beginning of a medical story and also being there at the end, but not caring about what is in between, so they were very generous in publishing this paper before anyone else had mentioned the general neurotic syndrome, which to many others must have sounded bizarre. In this paper I wrote:

> It is more appropriate to regard many of these conditions (i.e., neuroses) as manifestations of one disorder, which may be termed the 'general neurotic syndrome'. To qualify for this diagnosis patients should show at least three of the following features:
>
> (a) two or more of the following symptomatic diagnoses are present together, either now or at times in the past: agoraphobia and social phobias, panic disorder, non-psychotic depression, anxiety, and hypochondriasis (including somatoform disorders);
> (b) at least one episode of illness has developed in the absence of major stress;
> (c) There are abnormal personality features of a passive dependent or an anankastic type;
> (d) There is a history of a similar syndrome in first-degree relatives. (Tyrer, 1985)

I also added: 'these symptoms can be placed in a "handicap hierarchy" depending on the degree of social impairment for the symptoms produced'. This was accompanied by a figure of concentric circles showing the different disorders with the outer ones showing the greater handicap (Figure 1). (Because 'handicap' is now felt to be pejorative it could be replaced with disability). Thus, generalised anxiety, which is associated with the least degree of social impairment, occupies less space than the other syndromes, and agoraphobia, social phobia and hypochondriasis occupies a larger space. But I was careful to emphasise that this was not a diagnostic hierarchy; it was merely to illustrate that there was more social impairment in the outer rings.

My justification of each of these four elements follows, and here I am updating and adding to my arguments, but not changing them in any fundamental way.

1 Simultaneous and Past Presentation of Two or More Symptoms of Six Conditions

I chose these six: agoraphobia, social phobia, panic disorder, non-psychotic depression, anxiety, and hypochondriasis (including somatoform disorders), as they are the most prevalent conditions in the neurotic group. At the time of writing in 1985 there was already much evidence that there was overlap between all these disorders. This was explained in different ways:

(a) there is a hierarchy of symptomatology (Boyd et al., 1984, Coryell et al., 1988);

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(b) they are a common group of disorders (Aronson, 1987);
(c) although the symptoms appear to overlap they are genuinely separate and successful intervention demonstrates their distinctiveness (e.g., pharmacological dissection, Klein, 1964, 1981).

2 The Triggering Effect of Stress

The requirement that at least 'one episode of illness has developed in the absence of major stress' was included to illustrate the importance of life events in creating mood disorders (Figure 1.1), as well as the relevance of what became known as adjustment disorders. But I specified the absence of major stress to exclude post-traumatic stress disorder, a completely separate condition, but left the option open for minor stresses at home, at work, and in relationships, to create a stepwise increment in symptoms.

So, when Mrs Bennet says to her daughter, 'Don't keep coughing so, Kitty, for Heaven's sake! Have a little compassion on my nerves. You tear them to pieces', she is illustrating the consequences of minor stress in creating symptoms, stresses which for the average person would go unnoticed. Here again personality is involved. Some years ago (Tyrer, 2007), I suggested the term 'personality diathesis' was a better one than personality disorder as the possession of such a vulnerability was a lifelong one that lowered the threshold to stress. I still think this is a better term than 'disorder' but as disorder is applied universally to all mental health conditions, we have to accept the common parlance (but see Chapter 8 for its implications).

3 There Are Abnormal Personality Features of a Passive Dependent or an Anankastic Type

I have to admit this was a partial guess at the time, but in retrospect it is justified. The reason for the choice of these two personality features emanated from a study of neurotic patients who also had a personality disorder (Tyrer, Casey & Gall, 1983). We found that anankastic and passive-dependent personality traits were most often found in those with neurotic disorder.

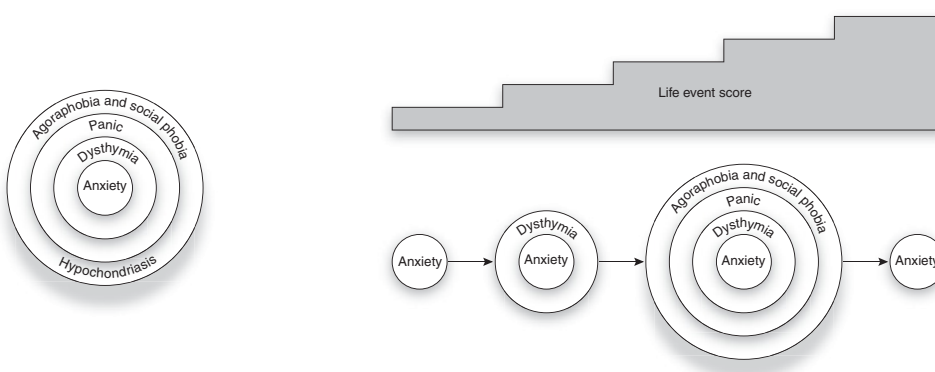

Figure 1.1 The initial formulation of the general neurotic syndrome and its components, including its variation over time in response to stresses
(From Tyrer, 1985, with kind permission of the publishers of the *Lancet*)

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The simultaneous presence of a clinical syndrome (cothymia) and personality disorder might best be described as a Galenic syndrome. Galen, in his 192 AD commentary, *De Temperamentis*, described how the four (personality) humours, melancholic, sanguine, choleric, and phlegmatic, were associated with specific diseases. His four humours dominated medicine for the next 1,500 years, accompanied by the dictum that too much of one humour led to disease. The sanguine person could accumulate too much blood and so needed leeches to reduce it, the phlegmatic person with lung disease created too much phlegm, and the choleric patient needed a purgative to remove an excess of bile.

Although Galen clearly had no good knowledge of the nature of bodily diseases, his linking of personality to them was novel and needs to be resurrected in modern psychiatry. A Galenic syndrome can therefore be defined as 'a combination of personality disorder and clinical symptom complex so frequently associated that the two conditions should be considered as a single disorder'.

4 History of a Similar Syndrome in First-Degree Relatives

This is a clear reference to the genetic elements of neurotic disorder, a subject that has attracted much attention. There is unequivocal evidence that there is a genetic contribution to all the disorders within the neurotic spectrum. There is much less agreement about the role of the family environment in causation. In the words of a recent meta-analytical review: 'Panic disorder, generalized anxiety disorder, phobias, and obsessive-compulsive disorder all have significant familial aggregation. For panic disorder, generalized anxiety disorder, and probably phobias, genes largely explain this familial aggregation; the role of family environment in generalized anxiety disorder is uncertain' (Hettema et al., 2001, p. 1568).

In the case of generalised anxiety, the hypothesis that an anxious mother can create an anxious child is a very persuasive one. Behavioural psychologists call this 'modelling'; the child observes the mother (sometimes the father) showing anxious avoidance and imitates it, is also often then taught to show anxious avoidance, and, hey presto, you have an anxious child who grows up to be an anxious adult. But the data do not give strong evidence in favour of this. One of the most influential researchers in the area, Roy Plomin, asked this question in 1987: 'why are children in the same family so different from one another?' (Plomin & Daniels, 1987). Twenty-four years later he admitted he did not have an answer. He summarises his dilemma clearly:

> It was reasonable to assume that the key influences on children's development are those that are shared by children growing up in the same family: their parents' personality and family experiences, the quality of their parents' marital relationship, their parents' educational background and socioeconomic status, the neighbourhood in which they are raised and their parents' attitude to school or to discipline. Yet to the extent that these influences are shared environmentally, they cannot account for individual differences in children's development because the *salient environmental influences are non-shared* [my italics]. The message is not that family experiences are unimportant but rather that the relevant experiences are specific to each child in the family, not general to all children in the family. (Plomin, 2011)

This is a subject highly relevant to the treatment called *nidotherapy* (Tyrer, 2009) that became a subject of particular interest in the Nottingham Study, and this will appear again in this book.

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1.3 Progress in Understanding the General Neurotic Syndrome since 1985

In a book I wrote in 1989, I formalised the definition of the general neurotic syndrome in the following words:

> The general neurotic syndrome is characterised by the simultaneous presence of various anxiety and depressive symptoms occurring in the absence of major psychological or physical trauma in individuals who have inhibited or dependent personalities. The diagnosis is made through a three-stage process:
>
> (1) Identification of the co-occurrence of anxiety and depressive symptoms in the absence of severe depressive illness or another significant psychiatric disorder;
> (2) Examination of environmental evidence of the symptoms and measurements of their severity;
> (3) Determination of the premorbid personality of the subject.
>
> (Tyrer, 1989, p. 154)

I also created the General Neurotic Syndrome Scale (Table 1.1)

This scale has deficiencies – not least that it was not formally tested – but the central elements are ones that I still think after a gap of 23 years are relevant to the diagnosis. I also

Table 1.1 The General Neurotic Syndrome Scale (GNSS)

Positive characteristics	Score	Negative characteristics	Score
Simultaneous presence of syndromal anxiety and depressive disorders (cothymia)	+2	Persistent phobic and obsessional symptoms	−2
Variation in the primacy of depressive and anxiety symptoms at different times	+3	Symptoms of anxiety and depression only occur in response to immediate life events	−3
If symptoms of panic, obsessive compulsive disorder, and hypochondriasis are present they do not last longer than three months	+1		
Premorbid anxious or dependent personality disorder	+3		
Premorbid anankastic (obsessive-compulsive) personality disorder	+1	Premorbid impulsive, borderline or antisocial personality disorder	−3
At least one parent has mixed anxiety depressive syndrome (cothymia)	+2		
TOTAL SCORE (no general neurotic syndrome)			0–3
TOTAL SCORE (likely general neurotic syndrome)			4–5
TOTAL SCORE (definite general neurotic syndrome)			≥ 6

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excluded phobic and obsessional symptoms by giving them a minus score, and some (that now includes me) would regard a negative score as inappropriate as these conditions, as suggested in my 1985 paper, could be regarded as extensions of the syndrome. On the other hand, the better definition and consistency of phobic and obsessional symptoms can make these conditions more amenable to diagnosis. The score needed to attribute the diagnosis of the general neurotic syndrome has not changed but now I feel more strongly that the higher score of 6 is the best threshold and that a GNS score of 4 only makes the diagnosis suspect. (In the rest of the book both versions of the GNS will often appear; this is of value in showing the linear transition of what is clearly a dimensional scale.

One important advantage of the general neurotic syndrome as a diagnosis is that it does not depend on time lines. The formal classifications of the neurotic group of disorders at that time had conditions varying from a few hours (acute stress reaction) to several years (dysthymic disorder) (Figure 1.2), which really made it impossible to make clear decisions when assessing a patient for the first time. All the requirements for making the diagnosis of the general neurotic syndrome are immediately clear in the scale; the examination of present symptoms and their precipitants (if any) are all that is needed.

One of the great sources of resistance that has prevented acceptance of the general neurotic syndrome is the antipathy to joining anxiety and depression together as a single diagnosis. It is difficult to know why the resistance has been so strong. David Goldberg set the scene many years ago when he and colleagues produced the first standardised interview schedule for common mental disorders (Goldberg et al., 1970). In the second part of the schedule, the nine most common symptoms of mental illness are listed: I have placed them in Table 1.2 in terms of the most prominent mood associated with each.

I am sure everybody looking at this table will agree the overlap between anxiety and depression is massive and cannot be ignored in any sensible classification.

The schedule described by Goldberg et al. (1970), 12 years later changed its name to the Clinical Interview Schedule (Lewis, 1992). The major symptoms were the same as in 1970 but

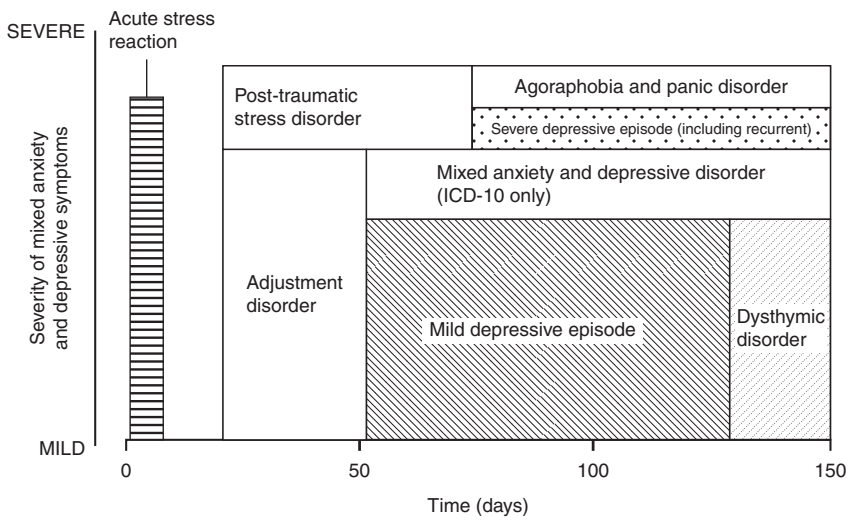

Figure 1.2 The confusing relationship between duration of symptoms and diagnosis in DSM-III and ICD-10

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Table 1.2 The association of depression and anxiety with the symptoms of common mental illness

Nature of symptom	Associated mood
Somatic symptoms	anxiety and depression
Fatigue	anxiety and depression
Sleep disturbance	anxiety and depression
Irritability	anxiety and depression
Lack of concentration	anxiety and depression
Depression	depression
Anxiety and worry	anxiety
Phobias	anxiety
Obsessions and compulsions	anxiety and depression

had two more added – worry about physical health (which might now be called hypochondriasis or health anxiety) and depressive ideas – and all of these were rated reliably. The first addition was understandable and a clear omission from the first schedule. The section on depression was split into frequency and severity of depression (depression per se) and related symptoms of depression such as hopelessness and guilt (depressive ideas) (Lewis, personal communication, 2021). With so many mixed symptoms, it was therefore not at all surprising that mixed anxiety and depression became an important feature in the responses. Still, only the revised Clinical Interview Schedule (CIS-R) reported this finding; no other scales addressed it. Its importance to public health was highlighted by Das-Munshi et al. (2008) who found, through analysis of data from the National Psychiatric Morbidity surveys, the one-month prevalence of Mixed Anxiety and Depressive Disorder (MADD, a very unfortunate acronym) was 8.8 per cent and accounted for 20 per cent of all days off work in the population.

The authors made a strong case for including mixed anxiety and depression in epidemiological surveys as this condition was also associated strongly with health-related quality of life. They concluded, 'our findings strongly support the inclusion of a dimensional perspective, without which the population burden of psychological morbidity is markedly underestimated' (Das-Munshi et al., 2008, p. 176).

But we also need to be clear that the mixed anxiety and depression label in the Das-Munshi et al. paper was not a diagnosis. It was a sub-syndromal condition that was immediately disqualified once a patient had symptoms of sufficient severity of either anxiety or depression to qualify for one of these disorders. So here we had a 'non-diagnosis' of sufficient severity to create major problems in living that was wiped out once one of the thresholds of formal diagnosis was reached, when a single anxiety or depressive diagnosis took over.

The paper by Das-Munshi et al. (2008) has been well cited (over 100 times) but its subject matter remains isolated in research. It is difficult to understand why there has been so much resistance to defining a mixed anxiety depressive syndrome despite all the evidence of the last 50 years. The question has to be asked: 'If a very common combination of symptoms creates so much pathology in the population at a sub-syndromal level, why is it not recognised at a syndromal one?'

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Looking at the way it is dealt with in the psychiatric literature reminds me of the reluctance people now have about referring to the British Isles. There is a tremendous degree of affinity between England, Scotland, Wales, and Ireland but for reasons that are primarily political we do not use 'British Isles' very often. It is felt to be a colonial expression, a hangover from the time of Irish oppression. The people from the island of Ireland are even denoted separately in ethnic population studies; 'the Irish' are a separate group. It is the same with anxiety and depression. We keep them separate because it seems politically correct to do so; depression is a mood disorder; anxiety is a conurbation of like states all associated with high levels of arousal (Craske & Stein, 2016) and so differs from typical depression. So many like to keep them apart, and when those annoying epidemiologists keep reminding us that they are joined together, we just wish they would go away.

In a separate study, Lewis (1991) compared the level of agreement between anxiety and depressive symptoms using a well-known scale (Hospital Anxiety and Depression Scale: HADS) and the Clinical Judgement Scales of the CIS-R. The correlations between anxiety and depression scores in the HADS were 0.59 but only 0.29 in the Clinical Judgment Scales. It could be argued that the psychiatrists were better assessors of anxiety and depression than the patients but in a separate study (described in the same paper) Lewis also found that when psychiatrists (all Maudsley trainees) rated their own anxiety and depression they showed a similar poor correlation. It was therefore reasonable to conclude that psychiatrists were showing bias in finding a degree of separation between anxiety and depression that simply wasn't there.

Lewis therefore concluded from these findings that 'the use of neuroses as, in part, a unitary concept, may be useful and is certainly a legitimate way of describing the current empirical data' (1991, p. 272). Jay Das-Munshi (personal communication) has also suggested that the tendency in insurance-based national systems (e.g., USA, Germany) to bill individually for anxiety and depressive disorders also exaggerates the separation. You could say, if you so wished, that these studies constituted one up for the general neurotic syndrome early in the history of this concept.

1.4 Gavin Andrews and the General Neurotic Syndrome

The general neurotic syndrome suddenly achieved a measure of respectability, if a limited one, by a publication by Gavin Andrews and colleagues in 1990. In this paper, they generously acknowledged and referenced my 1985 publication but Gavin admitted to me (in 2012, when I visited his amazing home close to Botany Bay) that he did not mention that the essential three words in the title were in my 1985 paper. This really did not matter but it was generous of him to confess.

His data came from a study of 15,000 twins involved in the Australian National Health and Medical Research Council Twin Registry, who were also interested in participating in medical research. (It is a pity there was not a UK equivalent as my brother and I would have been keen to be involved). In Andrew's study of 892 twins, and a separate clinic sample of another 165 twins attending for treatment of panic and agoraphobia, there was no evidence of diagnostic stability over time, or to use the words of the authors, no suggestion of 'patterns of co-occurrence of diagnoses being associated with particular syndromes' (Andrews et al., 1990, p. 6). Of more seeming relevance was the background presence of personality vulnerability, so the author had

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captured the essential parts of the 1985 paper. He took the personality factor one stage further in a later paper:

> In all three domains of information, a general vulnerability factor, associated with personality trait measures of high trait anxiety and poor coping, emerges as a principal cause of these symptoms or disorders, and accounts for the majority of the variation in the comorbidity of symptoms or disorders. This vulnerability factor is shown to be under substantial genetic control. (Andrews, 1996)

He also offered prospects for treatment.

> As these vulnerability factors can be measured, treatment programmes for anxiety and depressive disorders should ensure that they are reduced if relapse is to be inhibited. Prevention programmes, aimed at people with high levels of this personality vulnerability which increases their risk of developing anxiety and depressive disorders, would appear to be practical.

So we, on opposite sides of the globe, had come to the same conclusion. All that remained was for others to follow this up. It was noted in further studies (e.g., Duggan et al., 1996) as a research finding but never appreciated at the clinical level until just recently. Now, after realising that treating resistant depression and anxiety with more and more of the same, it looks at last as though the penny is beginning to drop and the focus will change to examining the personality component (Berk et al., 2018).

1.5 Developments and Changes in Classification since 2010

In the last 15 years, the notion of independent specific psychiatric disorders has received quite a beating. The debates about endogenous and neurotic depression (Kiloh et al., 1972) have all disappeared, and there is increasing knowledge and acceptance of the dimensional nature of psychiatric disturbance. We also have studies that show poor reliability of anxiety and depressive diagnoses as currently described (Andrews et al., 2010) and a host of pharmacological studies that show the terms antidepressants and anxiolytics are inappropriate and 'drugs for depression' and 'drugs for anxiety' are now preferred (Haddad & Nutt, 2020). So why is there such a reluctance to talk about mixed anxiety and depression?

I suggest two reasons. The first is that there has been a gradual separation of anxiety and depression research groups in the last 30 years. The anxiety research groups never study depression and vice versa so there is an innate tendency for each to ignore the other. The second reason is related to treatment. Officially, diagnosis should be made independently of treatment. In the case of anxiety and depression there has been a growing trend for all practitioners to believe that anxiety disorders should be treated by behavioural means and depressive ones by pharmacological ones. So psychiatrists who treat mixed anxiety and depression make the diagnosis a depressive one when they prescribe drugs and an anxiety one when they prescribe cognitive and behavioural methods of treatment. There has also been the influence of training that could be called the Lewis Prediction after his 1991 paper – expressed as 'when symptoms of anxiety and depression are both present the psychiatrist is trained to separate them even when they are of equal importance'.

In this mess of contradiction, the admission that we are mistaken in separating these conditions is almost an admission of failure. The fear that most people might diagnose mixed anxiety and depression and use their discretion as to what treatments they offer is too much to bear. It implies that we know much less about mood disturbance that we think we do.

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At the risk of getting too tied up in minutiae these points are worth expanding in the context of the latest classifications.

1.6 Identification of the Co-occurrence of Anxiety and Depressive Symptoms

There has continued to be dispute over the relationship between these symptoms ever since, with only a very slight shift towards the acknowledgement of mixed symptoms as a useful concept. The *International Classification of Diseases* ICD-10 and ICD-11 (world classifications) have allowed mixed anxiety and depressive disorder to be used as a diagnosis but at a very low level when other anxiety and depressive disorders have been excluded. This is the wording in ICD-11:

> Mixed depressive and anxiety disorder is characterised by symptoms of both anxiety and depression more days than not for a period of two weeks or more. Depressive symptoms include depressed mood or markedly diminished interest or pleasure in activities. There are multiple anxiety symptoms, which may include feeling nervous, anxious, or on edge, not being able to control worrying thoughts, fear that something awful will happen, having trouble relaxing, muscle tension, or sympathetic autonomic symptoms. *Neither set of symptoms, considered separately, is sufficiently severe, numerous, or persistent to justify a diagnosis of another depressive disorder or an anxiety or fear-related disorder* (my italics). The symptoms result in significant distress or significant impairment in personal, family, social, educational, occupational or other important areas of functioning. There is no history of manic or mixed episodes, which would indicate the presence of a bipolar disorder. (World Health Organisation, 2018)

The clinician has many other diagnostic options that are considered more acceptable. Dysthymic disorder, a chronic depressive condition, is described clearly in ICD-11 (and essentially the same in DSM-5):

> Dysthymic disorder is characterised by a persistent depressive mood (i.e., lasting 2 years or more), for most of the day, for more days than not. In children and adolescents depressed mood can manifest as pervasive irritability. The depressed mood is accompanied by additional symptoms such as markedly diminished interest or pleasure in activities, reduced concentration and attention or indecisiveness, low self-worth or excessive or inappropriate guilt, hopelessness about the future, disturbed sleep or increased sleep, diminished or increased appetite, or low energy or fatigue. During the first 2 years of the disorder, there has never been a 2-week period during which the number and duration of symptoms were sufficient to meet the diagnostic requirements for a Depressive Episode. There is no history of Manic, Mixed, or Hypomanic Episodes. (World Health Organisation, 2018)

What is most odd about this description is that anxiety symptoms are not mentioned at all – yet another example of the Lewis Prediction that airbrushes every diagnostic conjunction of anxiety and depression away.

If there is a gap in the presence of depressive episodes but there are still many of them, the diagnosis becomes 'recurrent depressive disorder' in ICD-11:

> Recurrent depressive disorder is characterised by a history or at least two depressive episodes separated by at least several months without significant mood disturbance.

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> A depressive episode is characterised by a period of almost daily depressed mood or diminished interest in activities lasting at least two weeks accompanied by other symptoms such as difficulty concentrating, feelings of worthlessness or excessive or inappropriate guilt, hopelessness, recurrent thoughts of death or suicide, changes in appetite or sleep, psychomotor agitation or retardation, and reduced energy or fatigue. There have never been any prior manic, hypomanic, or mixed episodes, which would indicate the presence of a Bipolar disorder. (World Health Organisation, 2018)

But again there is no mention of anxiety in any form in recurrent depressive disorder, so it will not surprise anyone to read that the descriptions of anxiety disorders do not include even a smidgeon of depressive symptomatology. Generalised anxiety disorder is 'characterised by marked symptoms of anxiety that persist for at least several months, for more days than not', but are 'not a manifestation of another health condition' (presumably including depression), and panic disorder is described by more serious symptoms of anxiety, 'palpitations or increased heart rate, sweating, trembling, shortness of breath, chest pain, dizziness or lightheaded-ness, chills, hot flushes, and fear of imminent death', but again excludes any other health conditions. Similarly, social anxiety disorder is 'characterised by marked and excessive fear or anxiety that consistently occurs in one or more social situations such as social interactions', again with no mention of any depressive components.

1.7 Categories and Dimensions

In trying to set the criteria for the diagnosis of the general neurotic syndrome, I need to emphasise that I am not pretending that this is a very clear category. Almost all psychiatric disorders are best seen as a spectrum from totally absent to strongly present – a continuum with points of diagnosis along the way. The points we choose are ones that are useful to clinicians, not ones that are clear and unambiguous. Bob Kendell emphasised this point many times during his career. Diagnoses are not set in stone, especially in psychiatry; they are merely functional abbreviations that help communication and decision-making (Kendell, 1975a; Kendell & Jablensky, 2003). The diagnosis of the general neurotic syndrome has no status or usefulness unless it helps practitioners to treat their patients. The last chapter of this book explains how this is possible.

It is useful to take an example from medicine to illustrate this, not least as medical diagnoses are often held up as real conditions, as opposed to the fanciful ones of psychiatrists. In the late 1950s, there was a vigorous debate about the status of hypertension in medicine. On the one hand there was Robert Platt, a scion of clinical excellence, who argued that severe hypertension was a genetically determined disease that was completely separate from other conditions in which there was high blood pressure. On the opposite side was George Pickering, an epidemiologist as well as a clinician, who argued that hypertension was a continuously distributed physiological trait, with some people having blood pressure at the upper end of the scale and others further down, but with no clear distinction between the two. It was therefore inappropriate to refer to two groups of people as having either 'normal blood pressure' or hypertension. At the time, there was gladiatorial combat in the *Lancet* between the two antagonists (Platt, 1959; Oldham et al., 1960; Pickering, 1960), all carried out with the utmost politeness, and most commentators, including the editorial staff of the journal (Lancet, 1959), supported the Platt argument.

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We now know that Pickering was right and Platt was wrong. There is no genetic basis to hypertension and no clear dividing line between the different hypertension diagnoses, but clinicians, understandably, still find it useful to use higher blood pressures as markers of severity. Even these have come under criticism as it is only night-time recorded (preferably during sleep) hypertension that predicts future cardiovascular events (ABC-H Investigators, 2014).

The hypothesis stated here is that the general neurotic syndrome is similar to hypertension. It represents the extreme of a range and whether it is regarded as clinically useful or a recondite reminder of the past depends on how useful it is in practice. Of course, as all psychiatrists know to their cost, when talking with medical colleagues, we do not have an independent measure of the syndrome like blood pressure to decide what level is pathological, but we do have other assessments that are reliable and sound.

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