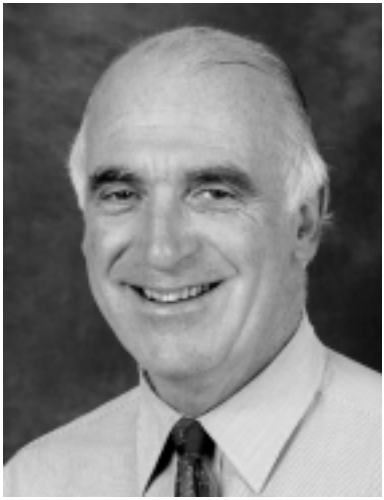
The World Health Organisation tells us that depression is the greatest cause of disability in our world. But how many depressions are out there, and what can be done about them? Read Gordon Parker's book and find out.

Gordon Parker is a Professor of Psychiatry at the University of New South Wales and was instrumental in setting up the Black Dog Institute which leads research into depression in Australia. His book is filled with careful clinical observations, wise recommendations on treatment and much more. His views on the increasing incidence of bipolar disorder are interesting and important. Anyone experiencing depression or managing it should read this book carefully.
John Ellard AM, Former editor Modern Medicine Australia

. . . compelling and compassionate . . . honest concerning available facts . . .
Anthony W. Clare, MD. St Patrick's Hospital, Dublin

. . . an excellent overview of the depressive illnesses, written by one of the leading authorities on the subject.
Kay Redfield Jamison, PhD, Professor of Psychiatry, The Johns Hopkins School of Medicine

Gordon Parker has style. The second edition of *Dealing with Depression* provides an outstandingly accessible account of how he sees the theory and practice of treating depression. While it is clearly written originally for the non-professional, the story is told from the cutting edge of research and treatment. It exemplifies the modern unifying approach to psychiatry–a synthesis of reliable knowledge and clinical judgment. It is a book both to read and to recommend to one's patients and their families.
Guy Goodwin, Professor of Psychiatry, Oxford University



Gordon Parker is a leading international expert on depression and mood disorders. He is Scientia Professor of Psychiatry at the University of New South Wales, and Executive Director of the Black Dog Institute (incorporating the former Mood Disorders Unit) at the Prince of Wales Hospital in Sydney.

DEALING WITH DEPRESSION

A commonsense guide to mood disorders

2nd edition

GORDON PARKER

with the assistance of David Straton, Kay Wilhelm, Philip Mitchell, Marie-Paule Austin, Kerrie Eyers, Dusan Hadzi-Paviovic, Gin Malhi and Sue Grdovic

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People seem to be able to bear or tolerate depression as long as there is the belief that things will improve.

Kay Jamison, *Night Falls Fast*

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PREFACE TO THE SECOND EDITION

Dealing with Depression was first published in early 2002. While the book was written for those with mood disorders and their families, it also proved to be of considerable interest to health professionals. In preparing this edition, the objective has been to maintain the strengths of the original publication but to give greater emphasis to topics that have generated questions from interested readers. In particular, the book now covers in much greater detail the bipolar disorders and the ways in which differing personality styles can contribute to the non-melancholic depressive disorders.

The non-melancholic disorders represent the most common depressive conditions in the community but they have proved difficult to define and differentiate from each other. It has therefore been difficult to determine the most appropriate treatments. Most systems classify them on a dimensional basis (i.e. severity, duration and persistence). We argue for an alternative model—that certain personality styles predispose individuals to develop these depressive conditions following germane stressful events. We also suggest that personality style shapes the clinical depressive pattern and, perhaps more importantly, influences the chance of responding to differing treatments. This is a central component of our approach, but it remains a field where much more research is required to determine its capacity to indicate the most appropriate treatments for different disorders.

Our Mood Disorders Unit team (incorporated within the Black Dog Institute) is working hard at developing the model and those

with non-melancholic conditions may be able to help us with parts of that research. Thus, this book has a unique feature. Those who have experienced a depressive disorder may wish to complete the Personality and Treatment Response Survey on our website (www.blackdoginstitute.org.au), a confidential approach where your anonymity is totally respected. As part of that survey, you will complete a personality measure, with scores on key dimensions being given to you at the end of the questionnaire. As noted, personality influences the chance of onset of non-melancholic disorders (in particular), as well as influencing how long they last and (possibly) response to differing treatments. Your personality profile will give you a 'snapshot' of how you may be at increased (or decreased) risk to a non-melancholic depressive disorder, and thus allow you to consider the material in Chapter 10 (Personality Styles and Non-melancholic Depression) more closely. It may assist your thinking about what might be the most appropriate treatment for you.

Since the first edition was published, three concerns about the current management of mood disorders have increasingly occupied our Institute's attention. First, we believe that—as a consequence of the current model of depression—a significant number of individuals with a more 'biological' depressive disorder are being undertreated. Second, and more commonly, a high percentage of people with the less 'biological' disorders (i.e. non-melancholic ones) appear to be receiving treatment that is excessively physical. It is not unusual for us to see people with so-called 'treatment-resistant' non-melancholic disorders who have received up to 20 different drugs and/or ECT, the doctor's view being that depression is a disease and that only a physical treatment approach is relevant.

Third, there is a distinct failure to detect bipolar disorder. We undertook three informal surveys in 2003 among people referred with depressive conditions and found that 30–50 per cent across the

samples had a bipolar disorder; 80 per cent of the subjects had not received such a diagnosis before. The average time between the onset of their bipolar disorder and receiving such a diagnosis was 15 years. Such issues concern us. There is no doubt that the mood disorders have been destigmatised considerably in the last few years and this is of major importance, as it is for people to seek help. However, if the help lacks sophistication, there is a real risk that many people will not only receive inappropriate treatment but feel that they have received a 'bouncing cheque'.

Our Black Dog Institute is clinically focused. We seek to ensure that people with mood disorders (and their relatives and friends) receive a rich and sophisticated message about multiple mood disorders and a rational model for their treatment. Many of the issues considered in this book can be pursued further by reference to our website (www.blackdoginstitute.org.au), where we try to keep you up to date with advances in dealing with mood disorders.

I remain indebted to my colleagues and to our many supporters who have assisted the preparation of this edition, and I remain indebted to Kerrie Eyers from our Institute and Rebecca Kaiser from Allen & Unwin for superb editing.

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ACRONYMS

CBT	Cognitive behaviour therapy
ECT	Electroconvulsive therapy
ITP	Interpersonal therapy
MAO	Monoamine oxidase
MAOI	Monoamine oxidase inhibitor (irreversible)
PMD	Psychomotor disturbance
RIMA	Reversible inhibitor of monoamine oxidase
SDA	Serotonin-dopamine antagonist
SNRI	Selective noradrenaline reuptake inhibitor
SSRI	Selective serotonin reuptake inhibitor
TCA	Tricyclic antidepressant
TMS	Transcranial magnetic stimulation

INTRODUCTION

> *Depression is … a noun with a bland tonality and lacking any magisterial presence, used indifferently to describe an economic decline or a rut in the ground, a true wimp of a word for such a major illness.*
>
> William Styron, *Darkness Visible*

> *Others imply that they know what it is like to be depressed because they have gone through a divorce, lost a job or broken up with someone. But these experiences carry with them feelings. Depression, instead, is flat, hollow, and unendurable.*
>
> Kay Jamison, *An Unquiet Mind*

The depressive disorders comprise our most common, and most commonly misdiagnosed, psychological illnesses. While we cannot claim to cover every aspect of depression or offer any miracle cures, *Dealing with Depression* is written for those suffering depression themselves, the families and friends of those with depression and professionals who want to know more about these conditions and their treatments.

In attempting to make people aware of the high incidence of depression and its impact on the community, definitions of depression have been progressively redefined and oversimplified in communications to patients and the public. The current dominant model views depression as an 'it'—that is, a single entity rather

than a set of conditions, a 'disease' varying only in severity, and having nothing to do with an individual's personality (and thus beyond the individual's control). 'It' is commonly thought to be brought about by chemical changes in the brain, thus requiring antidepressant medication—with all antidepressant drugs being equally effective.

By contrast, numerous psychotherapies and a range of quite different approaches are also held to be relevant to all expressions of depression, and of comparable effectiveness to antidepressant medication. But, by lumping multiple potentially different depressive disorders under unifying terms such as 'clinical depression' or 'major depression', by not respecting multiple causes and by testing treatments as if they had universal rather than specific application, we have a non-specific model for depression and an 'All roads lead to Rome' therapeutic paradigm. Treatments tend to be dictated more by the therapist's discipline, training or interest than by characteristics of the particular depressive condition.

Such a model is rather like viewing all cancers as the same and applying any one of multiple treatments to them, whereas we know that there are many types of cancer (some benign, some malignant), all triggered by different factors, some environmental, some genetic. We also know that there are many different treatments for cancers, each specific to the type of cancer diagnosed.

Research at the Mood Disorders Unit (MDU)—now based in the Black Dog Institute in Sydney—has challenged many of the current assumptions about depression. Those of us working at the Institute do not view depression as an 'it' but suggest that there are multiple expressions of depression that can represent diseases, disorders or reactions. For some depressive diseases, chemical changes in the brain may be a primary cause, with the depression occurring independently of personality and temperament; for other depressive disorders, the individual's personality style may be all-important.

We also argue against the view that each of the available

antidepressant drugs are equally effective. We also suggest that the most effective therapies are not the same for each principal depressive type, with some expressions of depression most likely to respond to antidepressant medication and others quite unlikely to respond. Similarly, we suggest that the usefulness of some of the psychotherapies (such as cognitive behavioural therapy) is not equivalent across the depressive types.

In journal papers as well as educational and training activities, our Institute staff seek to challenge the 'dumbed down' view of depression, and we also believe that it is important to communicate the challenge more widely. Such issues shape the content and objectives for this book.

A diagnosis of depression, therefore, is only half the answer. The first question should be 'What type?' Once the specific type of depression has been identified, patients (and their families) can be better informed about the specific causes of the depression and the treatments for it. Patients are thus offered a much better chance of managing, or beating, their mood disorder.

In a book titled *Malignant Sadness: The Anatomy of Depression*, Lewis Wolpert, a Professor of Biology in London, considered his experience of depression:

> I had never been seriously depressed before . . . I have to admit that I then rather sneeringly proclaimed that I believed in the Sock School of Psychiatry—just pull them up when feeling low. But that certainly does not work with serious depression . . . It was the worst experience of my life. (1999: vi–viii)

Wolpert wondered whether his depression had been caused by a recent social event, medication he was taking for his heart, a family tendency to depression, cultural factors or difficulties in his

childhood. Readers of his book might well conclude that the cause of his depression—whether genetic or environmental, biological or psychological—must therefore be the cause of other people's depressive disorders, a logical conclusion if depression is an 'it'. But, if depression has multiple expressions and causes—as argued here—it is wise not to assume that factors relevant to one person's depression are relevant to all. Similarly, a treatment that works—or does not work—for one individual may not cross-walk to any other individual's depressive condition.

This book provides an overview of the highs and lows of mood disorders and of available treatments. More importantly, it describes the principal depressive subtypes, together with their distinctive features, contributory factors or triggers and available treatments. We hope that *Dealing with Depression* will show patients and their families that there are preferential options available to them, and enable them to deal more proactively with their condition and ask more searching questions about their management.

This is not a self-help guide, however, and we strongly urge any significantly depressed person either to seek treatment or, at least, be assessed by a professional. Different approaches and treatments suit different people so we don't stipulate that you *must* consult a general practitioner/psychiatrist/psychologist/counsellor—everyone has access to different resources and facilities. Chapter 13 takes you through the assessment procedure and, we hope, enables you to ask the relevant questions of your practitioner. Our Institute's website (www.blackdoginstitute.org.au) also has a useful 'Getting Help' section with additional practical information.

We first define the various types and subtypes of depression and demonstrate the high prevalence of mood disorders. The World Health Organization has estimated that over 150 million people throughout the world live with clinical depressive disorders, and that's not counting all of us who feel blue from time to time! We then describe the principal clinical disorders, their triggers and

their biology, before discussing the assessment procedures used by professionals and what to do once the diagnosis has been made.

Finally, we look at ways of beating depression. After noting the various treatments, both pharmaceutical and psychological, guidelines for matching the treatment to the depressive conditions are provided. The final chapter considers the role of family and friends and how best to help—and cope with—someone living with depression.

chapter 1

WHAT IS DEPRESSION?

> *Depression is a disorder of mood . . . [which] remains nearly incomprehensible to those who have not experienced it in its extreme mode, although . . . 'the blues' . . . give many individuals a hint of the illness in its catastrophic form.*
>
> William Styron, *Darkness Visible*

The term 'depression' means different things to different people. All of us, at one time or another, have felt depressed, whether over bad news, a day-to-day problem or even for no reason at all. This is described as *normal depression*, or a normal *depressive mood state*. It may be experienced as a 'blue' mood, a drop in self-esteem or self-value, increased self-criticism, a lack of pleasure in life, feelings of wanting to 'give up' and pessimism about the future. Such feelings are not usually held at great depth, are transient (usually lasting only minutes to a few days) and are not disabling.

A person suffering from *clinical depression* holds these mood state features with more conviction than someone experiencing normal depression. The mood state and associated symptoms (described on pages 2–4) will nearly always have been present for more than two weeks and are associated with both social and psychological disability.

A representative list of features of depression is given below, some of which indicate particular depressive disorder subtypes (expanded on in later chapters).

Thus, somebody with depression may experience:

- *Lowered self-esteem*—that is, a loss of normal self-confidence, feelings of worthlessness and inadequacy, or guilt. Pessimistic and self-critical thoughts are common.
- *Change in sleep patterns*—that is, insomnia, or broken or fitful sleep. Some people might get off to sleep at the normal time, but wake at 2–3 a.m. and then either not get back to sleep or sleep fitfully, waking up several more times during the night. Others may take hours to fall asleep, or sleep fitfully all night.
- *Change in mood control*. Although the word 'depression' suggests that mood is always 'down', during a depressive episode all moods tend to be hard to control. Some people may feel unduly miserable and pessimistic, crying for little reason, and not feel any better after a good cry. Others may have difficulty controlling anger, flying off the handle at the slightest provocation. Irritability may be high, often followed by self-reproach and guilt. Anxiety can also get out of control and 'mountains made out of molehills'; worrying may become excessive. Some people develop panic attacks.
- *Change of mood through the day* (*'diurnal variation'*). Some depressed people feel most depressed in the morning and improve as the day goes on, while others experience the reverse pattern, or no mood variation at all.
- *Change in appetite and weight*. This may take two forms. In some people, especially if older, appetite may be reduced and weight lost. Constipation can also be a problem. However, for others, appetite and weight may increase. This pattern occurs especially in people who feel needy and respond to cravings for sweet foods by bingeing or by drinking more alcohol than usual. Many people crave cigarettes when depressed. These behaviours are not always psychologically driven. As detailed later, some may reflect biologically adaptive responses that

redress the neurotransmitter changes underpinning the depressive condition.

- *Change in capacity to experience and anticipate pleasure*. Typically, hobbies and interests 'drop off'. People suffering from this symptom, termed **anhedonia**, 'just can't be bothered' to do the things that previously gave them pleasure.
- *Change in the ability to tolerate pain*. Physical pains that are normally bearable may seem to get worse, or pains that cannot be readily explained by a physical problem may be experienced. This is because some types of depression actually lower the pain threshold, while other depressive conditions cause physical symptoms (e.g. headaches, chest pain, stomach churning) as well as pain.
- C*hange in sex drive*. Libido is commonly reduced or absent. Occasionally, there is an increase in 'needy' sex, perhaps because depression impairs the capacity to feel close to a partner.
- *Suicidal thoughts*. It is common to feel that there's just no point in going on. This may extend to thoughts of death, as well as to vague or specific suicidal thoughts or plans.
- *Impaired concentration and memory*, causing some people to believe they may be 'dementing' or going mad. These intellectual functions return to normal when the depression is relieved.
- *Loss of motivation and drive*. Everyday activities may seem meaningless.
- *Increase in fatigue*, feeling tired and lacking in energy. Some people may also find it hard to concentrate and may feel 'slowed down'.
- *Change in movement*. Some depressed people become physically slow or even immobile and experience slowed thinking ('retardation'). Conversely, others may become more agitated and be unable to sit still, with excessive and persistent worrying causing profound mental stress. In some cases retardation may alternate with agitation. Such obvious movement irregularity is called 'psychomotor disturbance', or PMD.

Table 1.1 Common features of clinical depression

Reduced:	*Changed from 'normal':*	*Varying:*
• self-esteem • motivation • sex drive • ability to enjoy things • pain tolerance • concentration and memory	• appetite and weight • sleep pattern *Other characteristics:* • movement disorder —either agitated or retarded, called 'psychomotor disturbance' • person becomes out of touch with reality —this is infrequent • suicidal thoughts • impulsiveness and recklessness	• mood and energy through the day • emotions (such as gloom, anger, anxiety) *Increased:* • tiredness • lethargy and fatigue • hopelessness • pessimism • apathy • impulsiveness • recklessness

- *Psychotic features*. A small percentage of people suffering depression may develop delusions (false beliefs such as 'I am totally worthless', 'I am so guilty, I should be punished') and/or hallucinations (hearing voices and seeing things that are not there). Some people may note changes in their hearing and their sense of smell (often such senses are sharpened) or changes to taste (e.g. food tastes metallic).
- *Disability*. Clinical depressive conditions are associated with disability, or impaired function. Unipolar depression has been identified as the most disabling disorder and bipolar disorder the sixth most disabling. Disability clearly stops a lot of people from going to work, but it is interesting to note that a greater economic burden comes from people getting to work but not being able to function effectively at work.

False positives

While all the mood state features listed above may indicate depression, most by themselves do not. For example, changes in sleep patterns can be attributed to a number of reasons. Older people generally require less sleep. Sometimes, early morning waking indicates a weak bladder or a snoring partner. Being stressed commonly disrupts sleep. Sex drive is liable to be reduced when there are relationship problems.

Changes in mood control could be the result of excessive use of alcohol or drugs, or a reflection of personality style. Anxiety and panic attacks often occur separately from depression, while changes in appetite and weight could be due to other medical conditions or a result of medication, stress or grief.

chapter 2

DEPRESSION, A COMMON EXPERIENCE

Depression is referred to as 'the common cold of the psyche'. Most people will experience episodes of normal depression. However, 25 per cent of women and 20 per cent of men will experience episodes of clinical depression during their lifetime. Having a depressive episode is therefore commonplace and certainly no cause for shame.

What *is* a shame is that clinical depressive disorders are so often undiagnosed and untreated (or undertreated). This sometimes happens because of the fear of disgrace associated with depression; or because doctors or health professionals don't recognise depression for what it is. It can also occur because individuals do not recognise their own depression. Depression may come on as a conviction that this is the way the world is or, more indirectly, physically as a series of illnesses, aches and pains.

Some clinical depressive disorders seem to run in families, with family members prone to depression, or mood swings, in the same way that other families have a tendency to stomach ulcers, diabetes or migraines. However, for many people there is no family history of depression.

If the depression is minor or transient, it may resolve by itself and not require any intervention. If it is more intrusive and persistent, professional help should be sought. When the disorder becomes intractable and debilitating, specialist treatment is required.

Those who don't respond to initial treatment may require expert review. Some people need to try several antidepressant medications, while others need to try quite different non-medication approaches. Such varying choices sometimes reflect the general preference of the treating therapist, rather than a more logical, commonsense approach that respects the importance of identifying and addressing the specific cause. At other times, the outcome reflects the nature of the differing depressive disorders, or the fact that the ideal or best treatment is not identifiable.

Research at our Institute suggests four principal causes of persistent and treatment-resistant mood disorders: 1. undertreatment of the more biological conditions such as melancholic depression; 2. excessive physical treatments for those with the less biological (e.g. non-melancholic) depressive disorders; 3. failure to diagnose bipolar disorder; 4. failure to identify an underlying contributory condition (whether medical or another psychiatric condition such as an anxiety disorder).

Each of these issues will be discussed shortly, and each *underlines the importance of ensuring that the depressive subtype is identified.*

Remember that depression can be biological in its origin, but psychological in experience. Those suffering from depression may have to push themselves or be encouraged by someone else to seek advice or treatment. They may feel that nothing much can be done about the way they feel but, in fact, most acute depressive disorders can be successfully treated.

The purpose of 'normal depression'

For most people, depression (even the commonly occurring normal depression) is an unpleasant experience that often interferes with day-to-day functioning.

What is the purpose of such a painful experience? This question can be linked to another one: What is the purpose of pain? Pain has one distinct advantage—the unpleasant side effects of pain mean that most of us will go to considerable lengths to avoid it. For example, if we did not find heat painful, we might get too close to a fire and suffer the consequences. It is for such reasons that many nerves in our bodies have heat receptors.

In a similar way, it could be argued that normal depression can be an automatic defence response or a response cued by certain situations. Such a proposition has been explored by the American psychiatrist Randolph Nesse (2000) whose thesis is considered below. He looks particularly at how normal depression may have offered a selective advantage to civilisation over time. To the extent that any of Nesse's interpretations have validity, they allow the individual to question the meaning of a 'depressed mood'—what is normal depression trying to say?

Is normal depression a cry for help?

It is unlikely that normal depression is a cry for help. If it is, then it is not a very useful or effective signal, as it is more likely to evoke negative responses from others.

Does depression help to conserve resources?

If someone is lethargic, has no appetite, lacks motivation and has no interest in conversation, might not such a state resemble hibernation in the animal world and be a way of conserving energy?

Nesse argues that depression is 'poorly designed' for such a

purpose—at least in humans. The argument might hold for animals, where an animal continues to forage for as long as there is an adequate food source. But, when the food source runs low and the animal has to use up more energy foraging than would be obtained from eating the food, it would be wiser for the animal to stand still—even if starving—and wait for some other food source to turn up. It would stretch credibility to suggest that depression has such an advantage for humans.

Can depression resolve competition with a dominant figure?
Is depression a signal to a more 'powerful' competitor that a threat no longer exists (thus ending the conflict and the depression)? Does it represent a true wish on the part of an individual to resolve a conflict and obtain reconciliation, or is depression designed to lull the competitor into a false sense of security? Again, while of clear relevance to animals, its pertinence to humans can be questioned.

Can depression help us to be more realistic in goal setting?
If a particular goal (e.g. a new partner, or a new job) is starting to look like 'mission impossible', somebody in a depressed mood state may feel compelled to reassess the situation before disengaging from the pursuit or escaping from the situation. To persist with a goal that looks unattainable requires considerable increase of effort from the normal, everyday pace of life and, if the goal is not achieved, the resultant depression will be even greater.

The argument is, then, that a depressed mood drives people away from tasks that will be unprofitable, or a waste of effort or dangerous. Failure to reach, or to renounce, a goal may be depressing in the short term, but the negative cost or pain may be less than the costs and pain of persevering with the task. If a setback occurs in pursuit of a major goal, it would make sense not to rush into chasing another significant goal. In such instances, moving into a

depressive state (with symptoms such as pessimism and lack of initiative) might, as Nesse (2000) notes, 'prevent calamity even while it perpetuates misery'.

Conclusions

There are several limitations to the interpretations considered by Nesse. First, they appear more relevant to animals than humans. Second, their benefits in contemporary society are not obvious. Even if true, such theorising is likely to have relevance only to normal depression and perhaps to some forms of non-melancholic depression.

The answer is perhaps best addressed at the individual level. Consideration of their own patterns of behaviour might prove more useful to a depressed person, especially when the episode is over. Questions that could be asked include:

- In what circumstances do I find myself getting depressed?
- What then is the message?
- Do I want to do anything about it?

Thus, while relief from depression may require treatment for some individuals, others may obtain more benefit from rearranging their 'ecological niche' or modifying their work and family systems to prevent the grating factors that may be lowering their sense of self-worth and driving the pain of depression.

In both normal and non-melancholic depression, some elements of the disorder may have homeostatic capacities, helping the body to return to normal. Thus, sleeping excessively (as many depressed individuals do) may be an adaptive behaviour by restoring slow-wave sleep during times of stress. Carbohydrate cravings and eating chocolate, in particular, have comforting effects that trigger the release of endorphins to create a 'feel good' state. Eating more

of certain foods may lead to an increase in the amine L-tryptophan, thus increasing the activity of serotonergic neurotransmitters in the brain, which may be disrupted during depression.

Just as a pregnant woman may develop an aversion to cigarettes and alcohol because of potential damage to the foetus, some people may lose pleasure in drinking alcohol during their depression. And while some people may no longer be interested in smoking, others develop a craving for tobacco (which might then increase the level of the brain neurotransmitter, dopamine, which may be decreased in some depressive disorders).

Thus, some symptoms in the less biological types of depression may be a response to painful psychological and social life situations; others may be adaptive attempts at normalising disturbed biological changes.

For the more biological types of depression, such as melancholia, it is difficult to believe that such disorders are primarily adaptive or functional responses. The British satirist and writer Spike Milligan observed:

> I cannot reassure myself that it has been worthwhile . . . I do not hold with this romantic view of depression, that it has some purpose . . . As far as I am concerned it is without a redeeming feature. (Milligan & Clare 1999: 41)

By contrast, the academic psychologist and expert in mood disorders Kay Jamison has stated (1995) that, if given the choice as to whether or not she would have manic-depressive illness, she would change nothing. If she had not had the disorder, she would not have:

> felt more things, more deeply; had more experiences, more intensely . . . laughed more often for

> having cried more often; appreciated more the springs, for all the winters . . . Even when I have been most psychotic—delusional, hallucinated, frenzied—I have been aware of finding new corners in my mind and heart.

Similarly, Andrew Solomon's description of his depression ('the noonday demon') captures a number of positive consequences of dealing with depression (Solomon 2001).

As with winter, biological depressions exist and test people to and beyond their comprehension of what is endurable. However, they can also provide a frame of reference for a new mood or a new season.

chapter 3

CLASSIFICATION OF DEPRESSION

The history of classification of diseases in medicine is like the history of maps and charts. In the sixteenth century, early map makers in Europe asked ministers of the Church to climb their bell towers and write down everything they could see. Maps were drawn from such recordings.

The development of the magnetic compass allowed more directional accuracy and made coastal navigation easier. Bearings could be taken from features and the position of ships calculated. The invention of the sextant permitted the measurement of latitude; that is, distance from the Equator. This was enough to allow Christopher Columbus to cross the Atlantic safely and return home.

But without the ability to measure longitude, whole areas of charts were left void and marked with the words, 'There be Monsters here!' The discovery of how to measure longitude was the major scientific breakthrough of the eighteenth century.

In psychiatry, early attempts at classification were a bit like the ministers climbing their bell towers: all they could do was look at their patients and write down what they saw. And while most of

medicine was able to progress through the 'coastal navigation' stage, psychiatry had a more difficult task.

In much of medicine, firm objective findings clearly demarcate one disease from another. These findings can be measured—such as a blood test that confirms diabetes, a biopsy that shows a particular type of cancer or a post-mortem that shows a clot in a coronary artery.

Psychiatric classification has had to operate, as it were, out of sight of land. There are no sharp-edged rocks or islands from which to take a bearing. So, despite the many efforts to identify specific causes of mental illnesses such as **schizophrenia**, none has been found. Even post-mortem findings, which resolve most diagnostic disputes in medicine, fail to help much in psychiatry.

As a result, maps of psychiatric disorders have been a bit vague, just as charts were before longitude could be measured. But that is changing now—modern statistics and computer-driven research are providing better ways of knowing where in the sea of psychological phenomena we are at a particular time.

The importance of depression subtypes

Does it matter that there are different depressive subtypes? This is a commonly asked question and the answer has to be 'Yes'! Just as an accurate position is necessary if you are going to drill for oil in the seabed, or find a good spot for fishing, so too is it very important to know that there are different types of depression.

A pigmented spot of skin may be a freckle or it may be a melanoma. Swollen ankles can reflect either heart failure or a kidney problem. Before doctors could tell the difference (and subtype the various expressions), successful diagnosis and, therefore, treatment was often due to chance.

Such is the risk of viewing depression as a single disorder, and why it is important that the principal subtypes be recognised.

Regrettably, many experts and classificatory systems still hold the view that depression is one condition, varying only in severity. It resembles the markings on the charts before longitude was discovered: 'There be Misery here'. Descriptive, but not specific!

The psychiatric classifications of depression that have remained beached in those shoals have provided unhelpful maps of depression. Particularly confusing has been the longstanding tendency to classify depression on the basis of severity. Thus, the recent North American DSM systems essentially distinguish between 'major' and 'minor' depressions, diagnosing along dimensional parameters of severity, persistence and recurrence.

For example, the DSM diagnosis of dysthmia requires a person to have a depressed mood and at least two associated mood disorder features 'for most of the day, for more days than not . . . for at least 2 years'. Including it in a diagnostic manual gives the impression that it is a firm diagnostic category. In reality, like major depression, it is a jumbled mosaic, capturing any number of more minor mood and other disorders. The capacity then to define the causes of and treatments for dysthmia as for major depression is logically flawed.

Similarly, the World Health Organization classifies depressive disorders as 'mild', 'moderate' and 'severe', which is also non-specific and unhelpful. Such dimensional models have more recently been extended to include 'subclinical' and 'sub-syndromal' expressions of depression, stretching the meaningful definition of depression and compounding the intrinsic limitations of a severity-based model. This model continues to hold back understanding and treatment. For example, in medicine, swollen ankles can be 'severe', 'moderate' or 'mild' but these descriptions are less important than identifying whether the swelling is due to heart problems or kidney problems. Such is the case in understanding 'depression'.

It is very important to concede that there are different types of depression, as treatments for each type vary in relevance and

usefulness—antidepressant medication might be better than psychotherapy for one type of depression, while the converse may hold for another depressive type.

It is also important to accept that, while social, psychological, biological and medical conditions can all influence the nature of depression, they do not necessarily provide an 'explanation' for any one individual. Although a family tendency to depression, difficulties in childhood, changing cultural trends and even evolutionary explanations should be considered, such factors are of quite varying relevance to differing depressive types. For some people, genetic factors may be the principal cause and life stressors of minor relevance; for others, the reverse may hold. And, to have experienced a traumatic event does not, necessarily, make it a 'cause' of depression.

There is a famous saying that 'the beating of tom-toms will always restore the sun after an eclipse'. This reminds us that, if two events occur together, we risk concluding that one must have caused the other. Thus, depression might occur for the first time in a menopausal woman—but the menopause may not itself be *the* cause. Depression may well seem a very logical outcome for someone who has experienced a long sequence of high-level stressors in their life (e.g. poor parenting, childhood sexual abuse, the break-up of a marriage and a severe medical illness). While such events may seem a total explanation of the depression, they may have contributed to it only partially or have no relevance at all.

The causes of depression may therefore be difficult to identify for a range of reasons. Ideally, professional assessment should clarify the relevance of possible causes and provide an accurate subtyping diagnosis.

chapter 4

CLINICAL DEPRESSION

Most people experience mood states. Such states are usually not of major intensity, last less than two weeks and don't interfere with the ability to function. The distinction between a normal mood state and a depressive disorder is crucial to understanding depression and is the focus of this chapter.

Depressive disorders

Depressive disorders are more severe than a depressed mood state, last for at least two weeks and affect functioning at home and/or work. There are three classes of clinical depressive disorders:

- non-melancholic depression;
- melancholic depression; and
- psychotic melancholia.

Melancholic depression and psychotic melancholia are less common depressive illnesses, affecting 1–2 per cent of Western populations, with the numbers being roughly equal for men and women.

Non-melancholic depression

This is not a 'pure' category, rather a residual category left after excluding the melancholic and psychotic disorders. It comprises a heterogeneous conglomerate of conditions that most usually reflect an interaction between stress and the individual's personality style. Not surprisingly, in comparison with the two other depressive classes, non-melancholic depression lacks specific defining features such as psychomotor disturbance (PMD), which helps to define melancholic depression, or psychotic features which, together with PMD, assist in defining psychotic depression. Thus, people with non-melancholic depression can usually be cheered up to some degree and are less likely to report significant concentration and memory problems.

As with the other depressive disorders, those with a non-melancholic depression have a mood disorder (feel depressed, have lowered self-esteem and are self-critical) and experience many of the associated symptoms (such as appetite and sleep disturbance) noted in Chapter 1.

Non-melancholic depression is the most common depressive disorder, affecting one in four women and one in six men in the Western world over their lifetime. It has a high **spontaneous remission** rate (i.e. getting better naturally, without intervention), making accurate assessments of specific treatments difficult. In fact, response rates to quite different treatment approaches (e.g. antidepressant drugs, psychotherapy and counselling) are very similar, making management recommendations difficult to develop from any evidence base.

Melancholic depression

The depressed mood state in melancholic depression is generally more severe than in non-melancholic depression and PMD is evident. This subtype has a low spontaneous remission rate and, before effective treatments were available, could last from months

to decades. Its response rate to physical treatments (e.g. antidepressant drugs) is high, but minimal to non-physical treatments such as counselling or psychotherapy.

Psychotic melancholia

The depressed mood state and PMD are even more severe in psychotic melancholia than in melancholic depression. As well, a feature unique to this disorder is present—psychotic phenomena (delusions and hallucinations), or near-psychotic symptoms (termed 'overvalued ideas'), most commonly involving pathological expressions of guilt. This condition has a very low spontaneous remission rate. It responds only to physical treatments.

The principal depression patterns

The common patterns experienced by those with the major depressive classes are described below, after noting normal mood swings and depression.

Normal mood swings and normal depression

Most people experience a pattern of fairly regular 'ups and downs' (e.g. being the life of the party one night and feeling glum and flat the next). This could simply be the result of following the fortunes of our favourite sporting team, or our mood reflecting the vicissitudes and weather changes of life like a barometer. In the past, when these mood swings were more a reflection of personality, the term 'cyclothymic' personality style was once used. In recent years, 'cyclothymia' has been incorporated into the definition of bipolar disorder, not necessarily a valid step.

If depression is defined as being blue, sad, hopeless and helpless, and with feelings such as wanting to give up and pessimism about the future, then more than 90 per cent of people will admit to such a state several times a year. While these states may range from mild

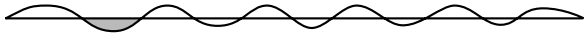


Figure 4.1 Normal mood swings

to troublesome and last from minutes to hours to a couple of days, most people expect them to settle by themselves or with the use of personal coping strategies. By definition, neither normal mood swings nor normal depression has any significant effect on day-to-day functioning.

Non-melancholic depression

In non-melancholic depression, a depressed mood is present for more than two weeks and is accompanied by social impairment (e.g. difficulty in dealing with work or relationships). There is no observable PMD and no psychotic features, and the features of the clinical mood state (as listed in Chapter 1) can vary. Spontaneous remission (i.e. getting better naturally and without intervention) is common.

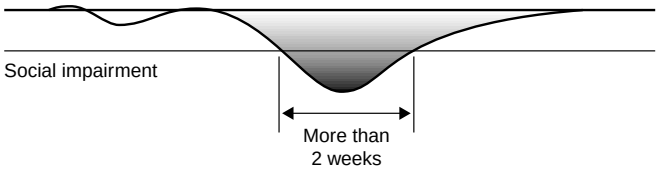


Figure 4.2 Mood disturbance in non-melancholic depression

There are many different expressions of non-melancholic depression, reflecting the contribution of the individual's personality style (detailed on page 59) and their response to stress, as well as any contributory anxiety disorder. Personality style characteristics also account for the variable mood state features and coping repertoires observed in non-melancholic depression, and indicate why quite contrasting therapies (e.g. those addressing predisposing factors, others directed at anxiety, and still others focusing on the depression) are claimed as effective.

We suggest that the more commonsense approach to choosing a treatment is first to identify the key cause (which could reflect personality style, an anxiety condition or the individual's coping responses) and focus on that cause, as well as addressing the depressed mood state.

Melancholic depression

The mood state in melancholic depression is more severe than in non-melancholic depression. It lasts more than two weeks and involves moderate to severe social impairment, as well as visible PMD (e.g. retardation or agitation).

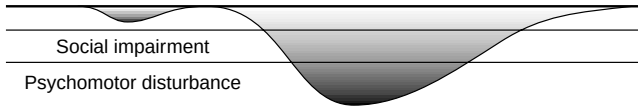


Figure 4.3 Clinical features in melancholic depression

Melancholic depression is primarily biological and spontaneous remission is unusual. It tends to run in families. The first few depressive episodes may develop in response to stress, but later episodes may appear 'out of the blue' or come on after a minor problem.

Melancholic depression may also develop as a result of exposure to certain drugs (licit or illicit) and some diseases. These can act like external stress, disrupting some of the brain's neural circuits (the basal ganglia and prefrontal cortex links) and causing depression, PMD and concentration problems. A similar disruption happens in Parkinson's disease.

For some people, especially those with no family history of melancholia, an episode of melancholic depression can occur for the first time late in life, as age changes in the brain disrupt relevant brain (or neural) circuits.

Active physical treatments are almost invariably needed but, as detailed later, different antidepressant drugs from various classes have a wide range of effectiveness in treating this subtype.

Electroconvulsive therapy (ECT) may be effective but is rarely needed. Psychotherapy and counselling may also be used in addition to physical treatments. However, as the main underpinning mechanism in melancholic depression is biological, not psychological, these treatments are not appropriate as primary therapies.

Psychotic melancholia

In psychotic melancholia, the depressed mood is extremely severe and present for more than two weeks. There is severe social impairment and PMD, and psychotic features (such as delusions) are evident, due to additional neural circuits in the brain being disrupted. As with melancholic depression, psychotic melancholia can first appear late in life.

Preferred treatments are biological and physical, with the older classes of antidepressant drugs appearing to be more effective than many of the newer ones (see Chapter 14). However, antidepressant drugs alone are usually less effective than combination antidepressant and tranquilliser treatments (**antipsychotic** or **neuroleptic** medication) or even ECT in some instances.

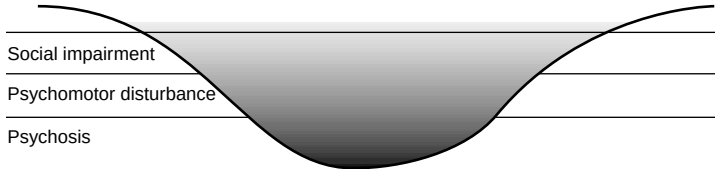


Figure 4.4 Clinical features in psychotic melancholia

Mapping the main depressive conditions

Figure 4.5 shows the main expressions of depressive disorders and the features that demarcate one subtype from another. The suggestion that psychotic depression is more 'weighty' or severe than melancholic which, in turn, is more severe than non-melancholic depression is generally true. However, subtype distinctions are driven more by the presence or absence of certain key features than by depression severity.

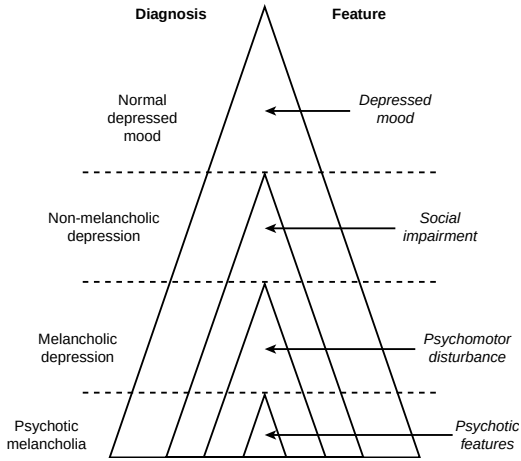


Figure 4.5 The four principal expressions of depression and their component features

Most current classifications of depression make distinctions on the basis of varying levels of severity. As noted earlier, we argue that three separate principal depressive clinical classes exist, with class distinctions reflecting different causes leading to differing clinical features requiring different treatments. The clinical disorders mapped in Figure 4.5 will be fleshed out in the following chapters.

chapter 5

UNIPOLAR AND BIPOLAR DISORDERS

Over time, people can exhibit quite varying patterns of mood swings. Within the clinical depressive disorders, it is important to make a distinction between unipolar and bipolar depression. The terms 'unipolar' and 'bipolar' originally referred only to the melancholic depressive subtype but have, in the last twenty years, been broadened to refer to all expressions of clinical depression. Thus, the term '**unipolar depression**', or 'unipolar disorder', is used to describe a pattern of episodes of clinical depression in which there are no 'highs'. For a clinician to tell a patient, however, that they have a 'unipolar depression' means little more than that they have a non-bipolar disorder and is, therefore, not particularly informative.

By contrast, the term '**bipolar disorder**' is used to describe patterns of manic or hypomanic behaviour (with elevated, expansive or irritable moods) that may or may not alternate with episodes of clinical depression. The word 'hypomania' comes from the Greek and means 'less than mania'. Occasionally, people show a mixture of both high and low features at the same time, or switch during the day, giving a mixed picture. Bipolar disorder is thought to be primarily caused by biological factors. It is strongly inherited.

It is now common to distinguish Bipolar I and Bipolar II disorders from one another, but distinctions between the two are not clear-cut, whether examined in formal diagnostic manuals or considered more informally in clinical practice. In general terms, Bipolar I disorder corresponds to the earlier term 'manic depressive illness'. Here, episodes of highs or lows are more severe and persistent, functioning is impaired and psychotic features may be present. In the past, people with severe bipolar disorder may have been admitted to an asylum where they could have remained manic for many months or depressed for many years and then spontaneously remitted, indicating that there is a pattern to even the most severe expressions of the condition.

Bipolar II is less severe (so that highs are more likely to be described as 'hypomanic' rather than 'manic'); it is not usually associated with psychotic features and tends to be briefer. The North American DSM-IV diagnostic manual specifies that hypomanic episodes must last at least four days, but many researchers and clinicians are confident that some individuals with true Bipolar II can have highs and lows lasting hours rather than days. The diagnostic manuals generally rule that mood and behavioural changes must be evident to others (e.g. family members) but there are many people with true bipolar disorder (I and II) who fail to show observable features—even when psychotic—so that imposing 'observable change' may miss some individuals. The term 'mixed state' refers to times when individuals meet the criteria for both manic and depressive episodes.

As noted in the Introduction, our Institute undertook three audits in 2003 in an attempt to estimate how common bipolar disorder is in those experiencing depression. Of those presenting with a depressive condition, up to 50 per cent were judged to have bipolar disorder (nearly all Bipolar II), 80 per cent had not had the diagnosis made previously and the average interval between onset and diagnosis was fifteen years. For most of those who had not

previously received such a diagnosis, questions pursuing such a diagnosis had not been asked—presumably, because the health professional was not aware of bipolar disorder or did not think it likely or worth pursuing.

To friends and family members, Bipolar II may appear to be merely normal mood swings or a reflection of personality style. Certainly, mild expressions can be very difficult to distinguish from a normal volatile or cyclothymic personality style. There are many people who swing from being the life of the party to being quiet, uncommunicative and even grumpy as part of their personality style and not because they have a bipolar disorder. Personality, however, tends to be persistent over time. By contrast, questioning will usually identify an onset or pattern change for those with true bipolar disorder, although we now recognise that bipolar disorder can also be present in quite young children.

There are other reasons why the diagnosis of bipolar disorder is not always straightforward. Creative people can experience self-induced highs when caught up by the Muse: a writer, for example, may describe feeling 'taken over' during a burst of creative planning, or writing in a state of excitement, needing only a few hours sleep. Some drugs (both legal and illicit) will induce a sense of being high. Nearly all antidepressant drugs (particularly the broader-spectrum drugs such as the tricyclic antidepressants [TCAs] and monoamine oxidase inhibitors [MAOIs]) can 'switch' a depressed person into a hypomanic or manic state—whether or not that individual had previously experienced a high or had a tendency to do so.

We used to think that manic-depressive illness affected about 1 per cent of the population. More recent epidemiological data suggest that the lifetime chance of Bipolar I is about 1 per cent, but the lifetime chance of Bipolar II could be up to 10 per cent of the population. Such a high percentage could reflect definitional or diagnostic error (falsely diagnosing bipolar disorder when it does

not exist), but our Institute's view is that Bipolar II is common and that its rate may have risen considerably in the community in the last decade.

Approximately 5 per cent of those suffering bipolar disorder experience only highs. However, the great majority of people with the disorder alternate between highs and lows and, commonly, experience intervals of quite normal mood states in between episodes. Each individual tends to have a distinct pattern. For instance, some people with bipolar disorder might have only one episode every decade, while others may have daily mood swings.

Before any individual settles into their general mood swing 'pattern', atypical patterns may be observed, particularly in adolescents. Thus, some may develop anxiety or eating disorders, or even have schizophrenia-like conditions for several years, before the more typical pattern takes shape. A family history of bipolar disorder can then be very helpful in establishing whether an atypical presentation is a variant or a forerunner of bipolar disorder.

Bipolar disorder

Bipolar disorder is biologically mediated and strongly inherited. When people with bipolar disorder develop depression, it is nearly always a psychotic or melancholic depressive episode. There are exceptions, however. For instance, an individual with bipolar disorder may have a sequence of melancholic depressive episodes and then, when faced with an unusual stressful event (such as a job loss), develop a normal depressed mood, a non-melancholic depressive disorder or a grief state.

How, then, can bipolar disorder be diagnosed with any confidence? At the Mood Disorders Unit at the Black Dog Institute, we explore a number of parameters with open-ended questions designed to establish whether, during any particular period, an individual has:

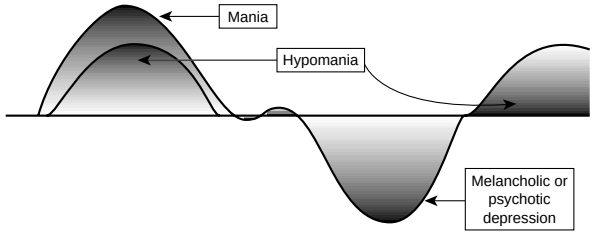


Figure 5.1 Mood swings and common clinical states in bipolar disorder

- experienced an elevated, euphoric, irritable or more confident mood;
- experienced reduced sleep, but without feeling tired the next day;
- had more energy or felt 'wired';
- spent more money than usual or wished to do so;
- made frivolous or unnecessary purchases;
- talked more and made more phone calls than usual;
- experienced increased libido;
- dressed more colourfully;
- been more verbally or behaviourally indiscreet than usual;
- found 'nature' more beautiful;
- been more creative;
- sung more than usual.

It is important to understand that such features are noted relatively consistently across different cultures, although cultural factors influence their actual expression.

If there is still doubt about the diagnosis after working through the list, then it is a good idea to talk to a family member to seek their observations and ask whether the individual has shown a pattern change at some time. It is also valuable to pursue the family

history to determine whether any family members have had bipolar disorder (even obtaining old hospital and medical records) and to ask to see the individual when the next 'high' is experienced.

Even after pursuing all diagnostic options, for a percentage of individuals the diagnosis will remain in doubt. Their situation should generally be reviewed after an interval; or, less commonly, mood stabilisers could be trialled to see whether they have any impact on mood state and functioning, although improvement does not, by itself, confirm such a diagnosis.

A number of reasons have been offered as to why bipolar disorder is commonly missed. A final reason is this—many patients with mild bipolar disorder enjoy their highs and prefer not to tell anybody about them. Once identified and diagnosed, they are concerned that medication will take away the pleasurable states and their creativity. Such concerns need to be respected and addressed.

chapter 6

GENERAL FEATURES OF DEPRESSIVE AND BIPOLAR DISORDERS: THE EXPERIENCE

Depression can be experienced in many different ways, reflecting the individual's personality, coping repertoires and mood state, as well as the type of depression. As there are no absolute rules, definitions of 'the experience' can only be imprecise markers of depressive subtypes.

For someone experiencing depression, their general mood state will be negative and marked by pessimism, lowered self-confidence and a sense of helplessness and hopelessness. They may want to 'walk away from things' (e.g. leave a difficult job or marriage), thus risking a drop in the social hierarchy. These features are generally more severe and pervasive in melancholic and psychotic depression. By contrast, in normal and non-melancholic depression, individuals may be able to 'bounce out' of the mood state, perhaps in response to support from others or to something pleasant occurring in their life.

Some people may detect certain gains from experiencing depression. For example, someone who has constantly won in life and who has taken things for granted may, for the first time, appreciate others and re-evaluate life's basic priorities. It has been argued that,

in such instances, the 'rosy glow' that non-depressed people can adopt to handle life's difficulties may be lost, thus ensuring issues are seen in a more objective way.

Coping repertoires in normal and non-melancholic depression

It is interesting to consider the coping repertoires used in dealing with or trying to overcome these expressions of depression. For example, some individuals describe depression as making them feel 'cold', so they respond by trying to warm themselves up, perhaps by taking a warm bath or sitting next to a window basking in the sun on a winter's day.

Some people may engage in self-consolatory behaviours, such as going shopping or 'pigging out' on certain foods, such as chocolate. These behaviours can reflect complex psychological processes—'He no longer cares for me. I will therefore care for myself. I will eat something I can really enjoy.' The link between being sensitive to rejection, the concept of atypical depression and having cravings for foods that influence the serotonergic system is detailed later.

Others may become reckless and impulsive and perhaps throw or smash things, or drive dangerously. While recklessness is more likely in men, women may also show this behavioural pattern, perhaps by socialising or relating to men in at-risk ways.

Some women may become careless and 'forget' about contraception, particularly after a break-up in a relationship. Reasons for doing so may include an attempt to restart the relationship or to 'keep part of him'.

A more common behaviour is to seek reattachment. Many people seek help from friends and professionals or in less direct ways such as praying. Some seek to distract themselves from the depressive thoughts by working harder or more repetitively, or by developing a 'depressive habit' such as painting the kitchen during

each episode. Others will attempt to block things out by drinking to excess, taking anxiety-relieving medication or sleeping tablets, or just going to bed to escape. Suicidal thoughts and actions may occur even in non-melancholic disorders, but are more likely to be countered by such notions as 'I wouldn't want to hurt my children'.

Those who have an 'internalising' personality style (see page 61) may retreat to their room to brood and ruminate about their hurt. They may believe themselves to be more inadequate than they actually are, ignoring their usual strengths.

Those with an 'externalising' style (see page 60) may be irritable and angry with those around them and start yelling and throwing or smashing things such as china and glassware (and can be subtyped into those who break one plate at a time versus those who prefer to break as much as possible in one go).

Thus, in normal depressive mood states and in non-melancholic depression, we see people using their inherent coping repertoires and revealing aspects of their personality as they try to cope with their mood state.

Coping repertoires in melancholic depression

The mood state in melancholic depression is more dominant than in non-melancholic depression. It is generally more severe and certainly more pervasive (there is nothing to look forward to, there is no pleasure to be found in the usual pleasurable events, interest cannot be maintained in activities) and will be present throughout the day, although it may be particularly severe in the morning. Concentration and memory function are commonly impaired. There is (in comparison to non-melancholic depression) an increased risk of suicide.

Observable PMD means that somebody suffering from melancholic depression can appear either retarded or agitated, or even

alternate between the states. Agitation may increase the suicide risk, while severe retardation may reduce the risk. Unfortunately, as treatment progresses and retardation decreases, those suffering melancholic depression can be at greater risk of suicide even though by all appearances they are recovering.

In retarded melancholic depression, actions slow down—those suffering from this disorder may walk or talk slowly, pause before moving or talking, use briefer sentences with reduced conversational richness. They are not able to brighten (at all, superficially or temporarily) at the introduction of pleasant topics. Many say that they find it hard to get out of bed and do such basic things as having a shower, as if there is some kind of mechanical failure to function. The normal 'light in the eyes' is diminished or lost, facial movements are less mobile, hair may become brittle and skin pale and even pasty.

The novelist William Styron, in *Darkness Visible*, captures the elements of this mood state:

> . . . my speech, emulating my way of walking, had slowed to the vocal equivalent of a shuffle . . . I'd feel the horror, like some poisonous fogbank, roll in upon my mind, forcing me into bed. There I would lie for as long as six hours, stuporous and virtually paralyzed. (1992: 56–8)

A description of Spike Milligan when he was experiencing an episode also captures aspects of retardation:

> He is markedly lacking in spontaneity, sitting quietly, responding to questions but initiating little conversation. There is a noticeable lack of facial expression and little extraneous movement. (Milligan & Clare 1994: 13)

And Milligan's own description:

> . . . this vital spark has stopped burning . . . I go to dinner . . . and don't say a word, just sit like a dodo. It must be a bit unbalanced at the table with me sitting there dead-silent . . . It is like a light switch. I feel suddenly turned off. There is a tiredness, a feeling of complete lethargy. (Milligan & Clare 1994: 15)

Those suffering agitated melancholic depression appear preoccupied with what are usually quite mundane things—which are blown out of proportion—and show considerable mental anxiety. They may pace up and down, wringing their hands, or even make little picking movements. Speech is rapid but superficial and without the usual richness—again dominated by mundane concerns and often guilt. Sufferers may look apprehensive or even fearful and their mental anguish is often visible to others.

In *An Unquiet Mind*, Kay Jamison describes one experience of agitation:

> . . . I became exceedingly restless, angry, and irritable, and the only way I could dilute the agitation was to run along the beach or pace back and forth across my room like a polar bear at the zoo. (1995: 45)

Such stereotypical presentations (of observable retardation and agitation) are usually independent of the individual's personality, and suggest to the professional observer a biological disruption or disease process. In younger people, observable psychomotor disturbance may be less evident. On questioning, however, they will usually describe experiencing motor changes (e.g. an inability to

undertake basic things) and, in particular, describe distinct effects on thinking and concentration, perhaps finding it quite impossible to study or concentrate on reading.

The simple term 'depression' has been rejected by a number of writers as it is not capable of capturing the import of melancholic depression. William Styron suggested (1992) the term 'brainstorm' to describe 'a veritable howling tempest in the brain' (p. 38). The broadcaster Helen Razer (1999) felt bombarded by a 'broken head' and felt 'plagued . . . by the suspicion that my synapses were exploding . . . of phrenic crashing . . . I became immobilised by these potent little shocks . . . I would imagine that my poor battered lobes were rolling about yolklike in my feckless eggshell head' (pp. 132–3)

Coping repertoires in psychotic melancholia

In psychotic melancholia, the depressed mood is either extremely severe or, at times, denied. Where depression is denied, the individual may instead describe states of nothingness, of profound enervation, or even of the disturbance being felt at the physical level (with flu-like symptoms, physical agitation or pain). Such a mood state persists throughout and over the days and lacks the late-in-the-day lift experienced by many with melancholic depression.

PMD (whether retardation or agitation) is even more severe in psychotic melancholia than in melancholia, so that in the retarded state the individual's appearance may resemble that of someone with dementia. Concentration, attention and memory are generally impaired.

In agitated psychotic depression, the individual is rarely still except when asleep. A common speech pattern is that of repeated questions; for example, 'What is going to become of me?'

Delusions are far more common than hallucinations (unless the

individual has profound PMD) and many people suffer from 'mood-congruent' themes (e.g. 'I am so worthless that I deserve to be put in jail or punished in some other way'). Minor indiscretions of the past may serve as a focus for such delusional thinking and are generally much exaggerated. Thus, someone who did not disclose two dollars on their tax return twenty years before may genuinely believe that they will be jailed for this minor indiscretion. Hallucinations, if present, may be subtle (strange tastes or smells) or reflect the underlying mood state (voices belittling the individual and telling them to commit suicide).

A percentage of people with psychotic melancholia will experience mood-incongruent delusions (e.g. that their home is being gassed or their food poisoned). Some delusions effectively build in physical features that emerge during the episode. For example, psychotic melancholic people with constipation may believe that their bowels have turned to cement or that they have a bowel cancer. Many with psychotic melancholia have overvalued ideas (see page 22) that are just short of being delusional, usually associated with preoccupations of guilt, and cannot be relieved by reassurance.

Hallucinations can be brought on by noises, smells or tastes. Hearing may become highly acute with some people hearing distant traffic noise not heard by others. Helen Razer captures such nuances in her 1999 book *Gas Smells Awful*:

> The tiniest sound can make you start. Music is deafening . . . Taste is repugnant. Mildly offensive smells work your gut into a frenzy. Everything appears to have hideously sharp edges . . . you can smell colour, savour sound, feel invisible objects. (p. 130)

In such severe states of depression, many people feel they are a burden on others and may seek to kill themselves or, if they feel

that the whole world is a burden, may kill others to protect them. This explains the rare but tragic situation of a postnatal puerperal psychosis (see pages 41–2), where a caring mother may kill or harm her baby.

Coping repertoires in bipolar disorder

> *. . . an illness that is biological in its origins, yet one feels psychological in the experience of it.*
>
> Kay Jamison, *An Unquiet Mind*

People with bipolar disorder generally experience both highs and lows (although a small number get only highs). The lows are almost always of the melancholic or psychotic depressive type.

During the high, the individual feels terrific and very confident. Talk increases and is so much faster than usual that others are unable to keep up. The mind races with ideas and creativity is distinctly increased—certainly in the mind of the individual, but often in reality too. People with bipolar disorder have lots of energy and need less sleep than usual—perhaps getting up in the middle of the night to do housework or write 'The Great Australian Novel'! Sexual interest (and activity) commonly increase. Dressing is often more colourful and singing more common. The world looks brighter and more attractive (trees are greener, water more sparkling).

However, because the world is seen through 'rose-coloured glasses', judgment may be poor. Purchases, loans, affairs and other commitments can be undertaken without due regard for the consequences. Falling in love at such times, ignoring objectivity and consequences, can lead to a number of emotional hangovers.

While those with depression risk a fall in the social hierarchy, those in a manic or hypomanic mood may rise in the hierarchy. For instance, the shy schoolgirl may ask the captain of the rugby team

to take her to the school dance and behave that night with complete self-confidence. Others may persuasively ask for a salary rise or propose to 'Miss Impossible' and, at times, succeed.

Although highs make most people feel happy, friendly and amusing, as with alcohol, others can become irritable and aggressive. Experiencing bipolar disorder is as if your brakes have failed; whatever direction you are going in—whether gambling, shopping, driving, having sex, drinking, taking drugs or showing off—you are going too far and too fast.

Kay Jamison describes one of her highs:

> . . . everything seemed so easy. I raced about like a crazed weasel, bubbling with plans and enthusiasms . . . I felt great. Not just great, I felt *really* great. I felt I could do anything, that no task was too difficult . . . not only did everything make perfect sense, but it all began to fit into a marvellous kind of cosmic relatedness. (1995: 37)

In Chapter 5 we drew a distinction between Bipolar I and Bipolar II. In Bipolar I the mood state is more severe, tends to be more persistent and is often associated with psychotic features. Delusions are most commonly grandiose, reflecting the mood state. For instance, a clerk became convinced that he was a very successful businessman and bought four sports cars (of differing colours, of course). Or a woman, convinced she was the world's best designer, designed (in her head) a rotisserie for installing above swimming pools that would slowly rotate people to give them an overall sun tan. Following a cocktail party to which she invited Sydney's top businessmen (with many accepting), she was off (first class) to London to persuade Harrods to buy her concept.

In Bipolar II the mood state may still drive excessive or unnecessary shopping (e.g. a dozen sets of bed linen when only one

might have been needed) but, not uncommonly, individuals will say that the mood elevation was useful in stimulating them to buy things that were necessary or to do things (such as an annual spring clean, or completing the last six years' taxation returns) that they had been putting off.

Thus, there can sometimes be advantages in experiencing a high and a lot may be achieved as a result. Many of the world's top creative people have suffered bipolar disorder and, if you have bipolar disorder in your family, you have a distinctly increased chance of being in *Who's Who*. But it is an asset with liabilities. If most people were said to have four-cylinder brains, people with bipolar disorder have V8s. Unfortunately, they also have cheap drum brakes. Quite a dangerous combination!

chapter 7

POSTNATAL MOOD DISORDERS

The adjustments required for a new baby mean that most parents experience some stress and they certainly experience many common features of depression, including sleep deprivation, low energy and social withdrawal. Increased stress leads to a higher level of anxiety in most parents. The boundary between normal adjustment and the less severe postnatal disorders can be somewhat blurred.

As with the depressive disorders, there is no single condition of postnatal depression. Instead, there is a range of conditions, the main ones briefly summarised below.

Maternity blues

Most women experience normal mood changes following the birth of a child. They usually settle within the second week. So-called 'maternity blues' are therefore relatively normal and are likely to be caused by an extreme reduction in hormone levels (both oestrogen and progesterone) immediately post-partum (i.e. after the birth), showing a rather similar mechanism to premenstrual mood shifts.

Only a minority of women, even those with severe maternity blues, go on to develop a formal postnatal depressive disorder.

Postnatal depression

Postnatal depression refers to a clinical depressive disorder occurring within the first six months after the birth of a baby. The overall incidence is said to be about 1 in 10, but this 10 per cent figure may be somewhat inflated by the inclusion of conditions other than clinical depression (especially anxiety disorders). The chance of clinical depression occurring during this period is approximately three times the overall new episode rate for depression in women over their lifetime.

Most postnatal depressive states are non-melancholic in type and have similar depressive features. In addition, however, there is usually greater depressive rumination and obsession about the well-being of the baby, as well as the woman perceiving herself to be inadequate as a mother. A significant number of women fear they will harm their baby.

For those who are genetically or otherwise biologically predisposed to develop melancholic depression, the postnatal period is a high-risk time for developing the condition, whether as an initial episode or a recurrence. For women with a history of bipolar disorder, there is a very high chance of a significant mood disturbance (i.e. highs, depression or a mixed state) both during pregnancy (most commonly in the second trimester) and/or in the post-partum period (most commonly in the first four weeks). It is uncommon, however, for a new episode of melancholic depression to begin during pregnancy.

Puerperal psychosis

'Puerperal psychosis' is an all-encompassing term used to describe any psychotic condition occurring in the first month post-partum.

The psychotic features of depression (delusions and hallucinations) are usually extremely florid and therefore very disturbing to the woman and to family members. In addition, the woman may appear quite cognitively affected—that is, in terms of being aware of what is happening to her. A small percentage of women may have a first onset or a recurrence of a schizophrenic episode but, over the last few decades, we have come to realise that the majority of such episodes are actually mood disorders. Thus, the postpartum period provides a distinctly increased risk for women who are genetically disposed to develop any of the more biological mood disorders or who already have such a history or who carry the risk to their development. The risk appears highest for those who have a history of bipolar disorder.

Specifically, a woman with bipolar disorder has a 25 per cent chance of a puerperal psychosis compared to a general population risk of 0.1 per cent—a risk 250 times greater. The main manifestations are manic episodes, psychotic depressive episodes and, quite commonly, mixed states where both manic and depressive features are experienced. Onset is usually sudden, within the first two or three weeks after the birth of the baby. While episode symptoms are commonly bizarre and disturbing at the time, the outcome is usually good, with most women responding well to treatment. There is, however, an increased suicide risk for women during the first year of treatment.

Treatment options

Treatment options for the clinical condition are considered in more detail later, but there are some specific features to take into account when dealing with a diagnosis of postnatal depression. A family history of depression, or previous episodes of depression, increase the chance of a woman developing a postnatal mood disorder, as do a number of psychosocial factors, including low

self-esteem, exposure to poor parenting practices or difficulties with a spouse. Management of postnatal depression must address any such relevant risk factors. Allowing the woman to sleep (easy to recommend, not always easy to implement) is also of key benefit.

Management of postnatal depression should involve assessment by trained primary health care staff such as early childhood or mothercraft nurses, or antenatal midwives, all of whom have the experience to know when to provide management themselves and when to refer women for medical or psychiatric assessment.

Drug treatment during pregnancy and while breastfeeding is clearly an extremely important issue in terms of the health of the baby. If a woman is on antidepressant or mood-stabilising medication, she should consult an expert and attempt a drug-free conception. In the first three months of pregnancy certain medications should be avoided, but this is not always achievable. In such cases, the woman, her partner and her doctor have to work together to address cost–benefit issues. (See Reference section for papers by Professors Marie-Paule Austin and Philip Mitchell with guidelines for the use of pyschotropic drugs during pregnancy and when breastfeeding.)

chapter 8

GRIEF: THE EXPERIENCE

Grief differs considerably from depression. In depression, there is a drop in self-esteem and self-worth. In grief, there is distress over the loss of either another person or of an ideal. When grief is at its worst, such distress is usually experienced as overwhelming separation anxiety.

Grief is generally experienced in stages. The first stage, which may last from hours to days, is a phase of numbness, where the individual is in a state of disbelief or even denial. The second stage, which may last from weeks to several months, is when separation anxiety is at its most severe, and waves of grief, sadness and tears are experienced. During this stage, sleep and appetite disturbances are common, as are social withdrawal, a sense of guilt or a wish to blame others. The lost individual may be 'seen' or experienced in some way. The third stage may begin after weeks or months: social withdrawal ceases, distressful symptoms settle and happy or positive memories of the dead individual return. Only one-third of grieving people actually go on to develop distinct depression, but usually not until weeks or months after their loss.

This biphasic response of grief (first phase) and depression

(second phase) may be a built-in response designed to promote survival. An analogy from the animal kingdom will make the argument clear. Imagine a mother and infant monkey in the jungle. The mother disappears and, after an interval, the baby begins to emit high-pitched screams and run around in a seemingly erratic way. If it is not reunited with its mother, the infant is likely to assume a slumped over, immobile position. Why?

The first phase is designed to re-establish contact with the mother. Assuming that she has just wandered away, she is more likely to see a darting infant, hear its screams and come running back. If, however, the mother has been taken by a predator, it would be unwise for the infant to continue such behaviours—even if it were not taken by the same predator, it would soon become exhausted. The second phase of behaviour, therefore, protects the infant against both detection and dehydration or heat loss. In other words, first-phase anxiety is designed to promote reattachment, while second-phase depression promotes survival.

Thus, grief (separation anxiety) is a state distinct from depression (loss), although it may lead to a depressive state.

The following example illustrates a situation in which it is difficult to determine where grief ends and depression begins, as the two states seem to overlap to such a degree.

A 23-year-old woman became severely distressed when she found that her boyfriend had been unfaithful to her and had left her for another woman. In the first week she was unable to sleep for more than two or three hours a night, lost her appetite completely and admitted to a significant weight loss of 6 kilograms. She cried repeatedly. She described being hypervigilant, jumping at any loud noise, and on more than one occasion she thought she saw her boyfriend and his red sports car—only to find that it was a complete stranger. She felt insecure, jumpy and anxious.

Interestingly, at that time she noted a fantasy of being pregnant. She cannot remember any loss of self-esteem, and was

more distressed by the loss of her boyfriend and her sense that they had formed a couple.

In the second week, she felt less anxious and insecure and reported that her sleep, although still patchy, was improving, as was her appetite. However, she was aware that she had lost her boyfriend irrevocably and felt hopeless, helpless and depressed. Her self-esteem dropped, she became critical of herself and started blaming herself for having 'lost another bloke'.

In the third week, she stopped going to work and discussing the issue with her girlfriends. Instead, she spent the days lying in bed, pigging out on chocolates.

This example illustrates the biphasic process noted earlier, with grief being the driving condition in the first week. Her fantasies of pregnancy can be presumed to reflect her wish to be reunited with her boyfriend or, if she couldn't have him, to have at least part of him. It could also signify her desire for a surrogate relationship with another person she could care for—a baby.

She moves into depression in the second week. In the third week, she chooses a self-consolatory strategy as a way of dealing with her pain (eating chocolate in response to food cravings). She is 'caring' for herself in a surrogate way, with an unconscious motivation of 'I'll care for myself as I wish to be cared for, as no one else is caring for me'.

Perhaps the most severe examples of a grief–depression sequence are observed in mothers who have had a young baby die—for example, from sudden infant death syndrome (SIDS). Here the response pattern is so distinct that we can only wonder at how instinctive and characteristic the behaviours are. In the first week after the death of the baby, the mother might wake during the night and run through the house searching for her baby, often screaming. After days or weeks, the high arousal pattern is replaced by one in which the mother spends most of her time slumped, rarely responding to others and appearing almost robotic. The depressed state begins to overwhelm her.

The two phases of any biphasic response (i.e. high arousal and depression), following a significant loss, generally overlap rather than forming two discrete stages in time. This makes it difficult to determine whether an individual's current state is one of grief, depression or a combination of both.

As noted earlier, only one-third of people experiencing grief will go on to develop a distinct depressive phase. Most will experience a range of alternating and evolving grief stages before some resolution occurs.

Grief can be suppressed, unresolved and prolonged. In such cases the grief can be labelled 'pathological'. The most common causes of unresolved grief are blocked anger and suppressed emotions, fuelled by the excessive use of benzodiazepines or other sedatives or drugs that suppress grief and its processing. The role of antidepressants is not clear-cut. If grief has moved to a pathological state or to a formal depressive condition, antidepressants may have a direct role; or, in the case of the SSRIs (selective serotonin reuptake inhibitors), they may help to reduce any contributory excessive worrying for a period. In general terms, a range of proven counselling techniques is preferable to medication.

chapter 9

STRESS AND DEPRESSIVE SUBTYPES

It is possible that an individual's episode of depression may be caused entirely by a major stressful situation or event (stressor). For others, stressors may trigger an episode that was waiting to happen. Alternatively, a depressive episode may be completely unrelated to a stressful event. It is therefore not surprising that, in many written accounts of depression, the role of stressful events as a trigger is difficult to determine. Often, the explanations provided by therapists are just as speculative.

This chapter looks at two broad groups of stressful events—*distal events*, which may have occurred years previously, and *proximal events*, which occur close to the onset of a depressive disorder.

Distal stressors

Some distal risk factors are biological (e.g. genetic influences, brain damage from injury or alcohol abuse). Many of them increase social risk and appear to have particular relevance to the non-melancholic disorders. Sex (or gender) is a good example. A

consistent finding from studies of normal communities is that women are more likely to develop depression than men, although bipolar disorder and melancholia have similar lifetime rates in men and women. This would seem to suggest an overrepresentation of non-melancholic depression in women, and could indicate that the 'anxious worrying' (see pages 62–3) style is more common in women. In groups where there is social homogeneity (e.g. where men and women have the same occupations), the difference between men and women is so slight as to be non-existent (as established by Black Dog Institute researcher, Professor Kay Wilhelm). Anatomy is therefore not necessarily destiny.

The type of parenting received can also be a distal stressor. Low levels of care and lack of affection from a parent increase the chance of depression, as does exposure to a parent who is controlling and overprotective. Low levels of parental care may make the child insecure—acting as a direct stressor—which in turn can lead to a child developing a low sense of self-worth. This creates a vulnerability in the adult to stressful events that reflect on self-esteem. A controlling parent often effectively delays a child's normal progress to independence, with the result that the child is later ill equipped to handle the everyday tasks of adult life. This, in turn, makes the adult easily stressed and more likely to develop depression when faced with adverse events.

Socioeconomic levels can also act as distal stressors. The lower the social class in the early years, the greater the chance of non-melancholic depression in adulthood—presumably because of increased exposure to a range of stressful factors. Social class does not seem to have any clear relationship to melancholic depression, although bipolar disorder appears to be somewhat overrepresented in higher socioeconomic groups.

Most of the distal social stressors that dispose a person to non-melancholic depression are, however, modifiable. For example, someone exposed to uncaring parenting will be at much less risk of

developing depression if they subsequently have a caring partner who can act in a 'buffer' role, or who can modify the risk elements. Conversely, someone who has experienced caring parenting but is then demeaned by their spouse is more likely to develop depression than someone who has experienced only caring relationships.

Proximal stressors

Proximal stressful events are the presumed causes of depression. Some, such as substance abuse (excessive intake of alcohol or drugs, both prescribed and illegal), are more influential than generally conceded.

In non-melancholic disorders, depression is usually more a consequence of an interaction between an immediate stressor and the individual's temperament and personality style. The same stressful event can evoke a wide range of responses in different people. Some may ignore it, others worry about it. Some may feel that 'all is lost' and others that the ability to control life has slipped away. The individual's reaction to the stressor contributes to both the onset of the depression and its severity.

The most common causes of melancholic, psychotic and bipolar depression appear to be biological. In the past, melancholic depression was called 'endogenous' depression, meaning 'coming from within'. It was therefore considered to be independent of stress. However, stress may precipitate a biological reaction, thus bringing on depression. (A number of medical conditions, for example diabetes, can be similarly brought on by stress in those predisposed to the disease.) As the melancholic disorders are more likely to appear after the age of 40, an age effect on the brain must be conceded.

Some external factors may be relevant for the more biological disorders. For example, in comparison to non-melancholic disorders, the onset of manic, psychotic and melancholic

depressive episodes increases in spring, indicating a seasonal cause. The rapid increase in hours of bright sunshine is thought to trigger depression and mania by affecting the pineal gland.

It does seem that the principal depressive subtypes show varying susceptibility (or resistance) to certain life stresses. This idea is developed further on the following pages.

How does stress lead to depression in non-melancholic disorders?

Our research at the Black Dog Institute supports a model that shows the non-melancholic disorders to be mainly a consequence of an interaction between stress and the individual's personality. A central feature of depression is loss of self-esteem (i.e. thinking less of oneself or being increasingly self-critical). Any event, therefore, that impacts on an individual's sense of self-worth risks precipitating depression.

A common stress event that impacts on self-esteem is the break-up of an intimate relationship. The event itself is irrelevant—it is the individual's appraisal or perception of the event that is crucial. Consider a man who responds to a marital break-up with 'My wife has left me for another man. She thinks I'm a jerk, and everything recently just confirms what a hopeless human being I am'. Contrast this with somebody who says 'My wife—what a jerk!—has left me. Great. I can get on with life again'.

The chance of developing depression is greater for the first respondent than the second. This is because the event differed in the impact it had on each man's self-esteem level, and because they processed the event differently as a result of their differing personalities.

Stressful events can be acute (a marital break-up) or ongoing (a dysfunctional marriage), but both have an impact on an individual's self-esteem.

Many people who develop a non-melancholic disorder have such a low ongoing self-image, or their personality type is such, that any stressful event is likely to trigger depression. In a sense, some people actually create their own triggers. For example, a woman who thinks that everyone rejects her may misinterpret a remark at a party and become immediately and distinctly depressed.

Our research has suggested a 'key and lock' concept for understanding some links between stress and non-melancholic depression. Here, we argue that certain events in early childhood (e.g. a critical father) lay down (or create) a cognitive 'lock'. In adulthood, the individual may handle a series of extremely stressful events with equanimity but become suicidally depressed when a boss criticises his work—this 'mirroring event' becomes the key that opens the lock established in childhood. Determining the types of stressful events—and interpreting their meaning—can be extremely helpful to many people in increasing their resilience to episodes of non-melancholic depression.

How does stress lead to depression in melancholic and psychotic depressive disorders?

The brain is made up of anatomical sections and numerous circuits (like railroad tracks). If, for example, circuits linking the **basal ganglia** (the brain centres that refine motor performance) and the **prefrontal cortex** (a structural region at the front of the brain) are disrupted, there are three principal effects: depressed mood, observable psychomotor disturbance and cognitive processing problems giving poor concentration.

Disruption of these circuits can occur in response to stress or even spontaneously. We presume that certain neurotransmitters (which modulate mood and other mental states) have been 'turned

off'. Many factors can influence neurotransmitter function. In melancholic depression (and, less clearly, in psychotic depression) there is often a family history of depression, suggesting a genetic influence. People with melancholic depression commonly report a significant stress prior to their first episode, or first few episodes. Subsequent episodes tend to appear more spontaneously and are less clearly related to stressful events. Therefore, certain genetic influences may create a vulnerability that initially requires a stress event to trigger the depressive state.

Physics provides a useful analogy with Hooke's Law, which states that if elastic objects are stretched within their limitations they will bounce back to their previous state. If, however, they are stretched beyond a certain point, their elasticity is lost. In melancholic depression, for example, it seems that initial elasticity allows the vulnerable individual to be unaffected by stressful events—for a period, at least. However, once a formal episode has occurred, the elasticity is lessened and future episodes may occur without the individual necessarily reporting a precipitating stressful event.

Certain drugs and some diseases can also act like environmental stressors, in that they have the capacity to disrupt some of the brain's neural circuits linking the basal ganglia and prefrontal cortex. In older people, the effects of the ageing brain may disrupt the circuits structurally as well as functionally. This is why we sometimes undertake brain scans (CAT or MRI) to see whether there are any unidentified bright objects (UBOs) or 'hyperintensities' indicative of microvascular damage impacting on neural circuits.

There are parallels between these depressive conditions and Parkinson's disease (which causes changes in the basal ganglia and other parts of the brain), including depression and a movement disorder. These parallels provide some understanding of biological depressive disorders such as melancholia. In psychotic melancholia,

the disruptions in the brain's circuitry are more severe and extend to other brain circuits and regions, causing delusions and hallucinations as well as severe PMD.

A metaphor on the road

> *There was a neuronal pileup on the highways of my brain, and the more I tried to slow down my thinking the more I became aware that I couldn't. My enthusiasms were going into overdrive …*
>
> Kay Jamison, *An Unquiet Mind*

This overview of stressors suggests quite different principal causes and factors relating to the different depressive types, with distinctive contributions from a range of variables. In all depressive types, stress may have been experienced before an episode, and it is easy then to assume that the stress itself must have caused the depression. We seek now, however, to provide a metaphor illustrating how, for differing expressions of depression, a particular nominated stress may be the primary determinant, a substantive or minor contributing factor, or just a mere non-causal after-the-event interpretation.

Imagine a busy main road. People cycle along it to get to work. Large trucks also use the road, often travelling at speed. Sometimes, they travel very close to the cyclists. A number of cyclists have fallen off and the traffic police have asked why. All the cyclists gave the same response, indicating the same cause: 'A truck came too close to me and blew me off.' Further investigations, however, showed that, despite all the cyclists citing the same reason for their accidents, they differed from those who didn't fall off in quite definite ways.

The non-melancholic cyclists were of two main personality

types: internalising and externalising. The internalising non-melancholic cyclists, being inclined to anxious worrying, rode very slowly and cautiously. When a truck passed, they wobbled badly. Their slow speed meant that they had little equilibrium and therefore often hit the kerb.

On the other hand, the externalising non-melancholic cyclists rode rather too fast for safety. They got very angry at any truck that came too close—yelling their irritation and taking their hands off the handlebars to gesture. Their overreaction upset their equilibrium.

The melancholic depressive, psychotic melancholic and bipolar cyclists had all bought their bikes from the same shop and there were some individual design distinctions that made these bikes more demanding to ride.

The melancholic cyclists could travel along quite well, but if they encountered a truck and had to pull onto the road's shoulder, the loose gravel tended to deflate their tyres. Their bikes could only go a short distance before losing momentum, at which point the rider fell off.

When the psychotic melancholic cyclists swerved, the action sheared off a bolt that detached the front wheel! They were no longer able to direct their cycles.

The bipolar cyclists' faulty bikes had gears that sometimes got stuck, in either the low or high range. In addition, the brakes were not powerful enough for such high-performance machines. Any swerve to avoid a truck exposed this instability, though their bicycles were difficult to control even in good conditions.

The rest of the cyclists on the road were unaffected by trucks, although some wobbled a bit, but didn't fall off. Many didn't even notice the heavy traffic.

This metaphor also indicates how decisions about treatment should not necessarily be driven by what might look, at face value, like the cause. In the same way, an elderly man may trip and

fracture his hip but the trip may—in comparison to his underlying bone osteoporosis—make a minimal overall contribution to the cause of the fracture. So, considerations about the cause of depression need to be sophisticated.

For example, if a perfectionistic individual develops a non-melancholic depressive 'fracture' after being 'tripped up' by his boss, then therapies that address his perfectionism may be of greater relevance than those that are limited to, or excessively focused on, the depressive 'fall'. This has advantages beyond the immediate situation, as addressing any underlying risk factor builds resilience against future stressors.

Returning to the cyclists on the road, those with internalising personalities disposing to non-melancholic depression may benefit from therapies that both reduce anxiety and mute their tendency to worry excessively or be insufficiently assertive. Those with externalising personalities may benefit from learning damage control when faced with a stressful event. For the other more biological types of mood disorders, physical treatments such as medication are almost invariably required to address intrinsic underlying biological causes, rather than viewing any proximal stressor as all-explanatory.

The suggestion that certain depressive subtypes are underpinned by a 'structural' or organic problem is seen by many as a very negative explanation, because it risks stigma and implies fault. Others are relieved by it (e.g. a member of the audience at a public lecture noted: 'I'm sick of being told that I just need to pull up my socks . . . I know I've got a structural problem and I want others to accept it'). The bottom line is that suffering biological mood disorders is similar to suffering from a medical condition such as diabetes. Accepting that model is not always easy.

chapter 10

PERSONALITY STYLES AND NON-MELANCHOLIC DEPRESSION

We all know what 'personality' is—but it remains difficult to define. Here is one definition: personality is the unique combination of qualities and traits (whether inherited or acquired) that characterises individuals in their social interactions. This places an emphasis on traits (or ongoing characteristics) and suggests that personality is best defined by observable social characteristics rather than by the individual's inner world or psyche. An immediate problem crops up with this last component. If you are intrinsically shy but socially gregarious, the definition given here would judge you as 'friendly' rather than as 'shy'. Is that the real you?

Personality emerges from temperament, which can be defined as those characteristics that are genetically determined and which effectively 'hard-wire' an individual's style. Environmental factors (particularly in early childhood development) may or may not modify the bedrock temperament style to create our personality. For example, a temperamentally confident child may—as a consequence of critical parenting—develop a personality style marked by a poor sense of self-worth. Personality is therefore potentially modifiable—encouraging news.

Models of the so-called 'normal temperament' emphasise five principal dimensions (the five-factor model) along which we all range (see Figure 10.1). First, we all span a dimension of *emotionality*—from anxious worrying through to emotional stability and resilience to stressful events. Second, we range across an *introversion/extroversion* dimension, from being shy and preferring our own company to being party animals seeking novelty, stimulation and excitement.

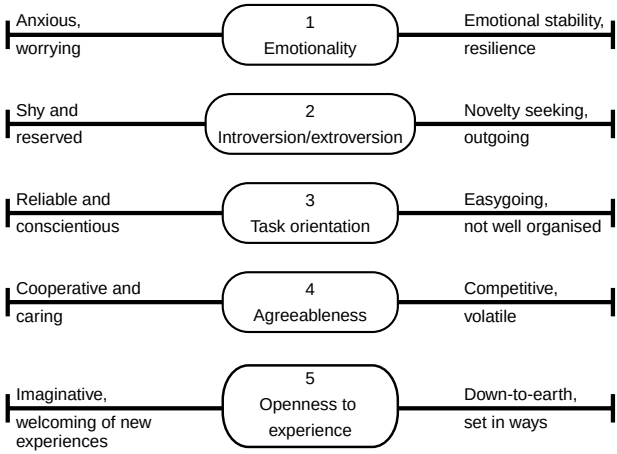


Figure 10.1 Five-factor model of normal temperament

Third, a dimension of *task orientation*, ranging from being highly reliable, conscientious, work-focused and even perfectionistic through to being easygoing, unreliable and feckless. The fourth dimension is described as *agreeableness*, where we range across a gradient from being cooperative and caring to being volatile and regarding other people as principally there to meet our needs. The fifth dimension is a rather unusual one, most commonly called *openness to experience*, ranging from welcoming new experiences to being set in our ways.

Dimensions of personality that influence risk to depression

Over the last few years our research team at the Mood Disorders Unit, Black Dog Institute, has tried to determine the contribution that each of these general personality dimensions makes to the onset and course of depressive disorders, and whether there are other personality dimensions that are relevant.

Our current model suggests eight dimensions of relevance to depression. You will see that some emerge from the five-factor model, but several others were also identified. The eight comprise:

1. *Anxious worrying*—reflecting high levels of internalised, ongoing anxiety.
2. *Irritability*—reflecting high levels of externalised, ongoing anxiety.
3. *Social avoidance*—individuals avoid socialising.
4. *Personal reserve*—individuals are uncomfortable sharing their feelings or opening up to others.
5. *Self-criticism*—reflecting ongoing low self-esteem and self-worth.
6. *Perfectionism*—reflecting self-imposed high standards.
7. *Interpersonal sensitivity*—individuals' sense of self-worth is highly dependent on how they see other people reacting to them.
8. A *self-focused* dimension, where individuals are focused more on their own needs than on the needs of others.

In addition to these eight dimensions of personality style, we suggest there are two important dimensions of personality functioning, or coping (cooperativeness and effectiveness), that are also of relevance to the onset and persistence of depression. As both constructs are dimensional (i.e. vary by degree rather than by type), it is rare for individuals to be placed at risk of depression in relation to only one component.

As noted, we are studying how these dimensions influence the risk and persistence of depressive disorders. After reading this chapter, you may wish to complete the anonymous and confidential Personality and Treatment Response Survey on our website (www.blackdoginstitute.org.au). This will provide you with a report on your own personality profile and you might then wish to reread some of the sections in this chapter.

Personality profiling versus cognitive theories of depression

Cognitive theories of depression argue that those who develop depression view themselves, their future and the world in a negative way and that this view is ongoing (i.e. it is part of their personality and thus makes them vulnerable to depression). A number of recent research studies, however, suggest that such perceptions are, in most cases, consequences—rather than causes—of depression. Their role in causing depression, therefore, has been somewhat downgraded in recent times.

Again, as a universal theory (one that seeks to explain all occurrences of depression), such a cognitive theory has immediate limitations. While it may be of relevance to those who score high on *anxious worrying* and *interpersonal sensitivity*, it may have no relevance to those who score as *self-focused*, and who tend to blame others rather than themselves when they become depressed.

A general principle of cognitive theory is, however, relevant when considering the role of personality—that it is the way in which we see the world, rather than the way the world actually is, that influences our judgments and may or may not lead to depression. Those who do not believe themselves to be particularly influential or masterful are more likely to develop depression. A related 'locus of control' theory suggests that those who have an *external* locus of control (i.e. they see themselves as a cork bobbing on the ocean,

prone to being moved around at the whim of others) are highly likely to develop depression. This is in sharp contrast to those who have a strong *internal* locus of control: that is, they view themselves as masters of their own destiny and have their hand on the tiller.

Thus, we think that personality profiling is more useful for modelling non-melancholic depression than general cognitive models, because it has greater potential to identify why a particular individual has become depressed in response to certain stressors. This is more useful than seeking to explain the cause of depression as reflecting a single stressful event by itself.

Risk of depression and implications for management

We now consider how such personality dimensions may increase the risk of depression. Any contribution of personality style to the onset of melancholic and psychotic depression remains unclear—hardly surprising when we view these conditions as essentially biological disorders. Thus, they are not caused by our personality—although, for certain individuals with these conditions, personality may make a secondary contribution. So, we focus in this chapter on the contribution of personality style and functioning to the non-melancholic disorders, which we see as reflecting an inter-action between personality style and meaningful stressful life events (stressors).

Emotional equilibrium

The concept of *emotional equilibrium* is useful in considering how personality may contribute to depression. Emotional equilibrium is a state of stable balance, such that any disturbance from outside tends to be corrected.

Let's assume that everyone has an internal regulating machine that needs to be reset after an upsetting event. Most people will

develop a depressed mood after a distressing event, but the great majority return to emotional equilibrium within days (i.e. they have a normal depressed mood state). Some people, however, are unable to reset their mechanism easily and thus lose their emotional equilibrium. They remain essentially 'stuck'. Their personality style and ways of dealing with events sustain the depression, rather than enabling them to get over it.

So how can equilibrium be lost? There are two main ways:

1. *The machinery can fail*; for example, if the keel on a yacht breaks off, the yacht will capsize. Thus, all individuals—irrespective of their personality style—may develop a non-melancholic depression when faced with certain enormities.
2. *A positive feedback loop can develop*. This means that two or more factors can influence each other to such a degree that a small disturbance leads to a further disturbance. This loop is also called a 'vicious circle'. An example of feedback occurs when a microphone is placed too close to a speaker. A small noise from the speaker is amplified into the mike, and further amplified by the speaker. While the feedback loop can be of use in creating musical effects, this reverberation (mulling over, rumination) is not useful for humans. For those with an 'anxious worrying' personality style, such a model is particularly relevant.

We now consider briefly how each of the eight identified personality dimensions influences the onset, duration and the actual clinical pattern of the non-melancholic depressive disorders. Ideally, the clinician should build these factors into the management plan.

Anxious worrying and irritability

We all vary in our general level of intrinsic anxiety, some high, some low, with most people in the middle. Those with high trait (i.e. ongoing) levels of anxiety tend either to internalise it or

externalise it. An *anxious worrying* internalising style is extremely common in the general community, and probably represents the largest personality risk group for non-melancholic depression. People with this personality style tend to have family members with a similar temperament style (which suggests a genetic contribution). They are at high risk of developing both anxiety and depressive disorders, which frequently appear in adolescence or early adulthood.

Why are they so at risk? Largely because their capacity to worry both drives and perpetuates depression. When they become depressed, they tend to worry more, cry, develop fatigue, withdraw (perhaps by going to their room) and keep their distress to themselves—although a percentage will seek a high level of support and reassurance.

Those who externalise their high levels of intrinsic anxiety tend to become irritable when stressed and during episodes of non-melancholic depression. Their risk of this type of depression is considerably increased by their personality style. Normally pleasant people, they become concerned about how 'crabby' and 'snappy' they are to those around them when they get depressed.

One of the main strategies for assisting individuals with non-melancholic depression, contributed to by *anxious worrying* or *irritable* personality styles, is to use SSRI antidepressant medication—both during the episode and subsequently—because it mutes the emotional disturbance and normalises anxiety. At too high a dose, this medication can turn down the worry level so that the individual does not worry about things that deserve to be worried about. If this happens, lowering the dose of the medication usually corrects the situation. Anxiety management courses and certain psychotherapies, such as cognitive behavioural therapy (CBT), can add value to SSRI medication; for some people it can be an equally useful alternative intervention to medication.

Social avoidance and personal reserve

The next at-risk personality style comprises the main characteristics of shyness and introversion. There are two somewhat overlapping dimensions of relevance here. First, there are those who show *social avoidance*, preferring their own company to the company of others; they avoid mixing and social events and tend to hold back when meeting people. They remember themselves in their younger years as being reserved, generally avoiding social interaction, and inhibited in their behaviour towards others.

Second, there are those who show *personal reserve*—preferring not to get too close to others and feeling uneasy about opening up to people. Behavioural approaches that promote their confidence in socialising and trusting others appear to be useful, with many people also benefiting from assertiveness training courses.

Self-criticism

The next at-risk dimension is expressed by individuals who have an ongoing low sense of self-worth. Sometimes termed a 'depressive personality' style, it describes those whose usual mood is gloomy and unhappy, whose self-concept is dominated by beliefs of inadequacy and low self-esteem, and who are often self-critical and negative. Such people often report lifelong depression. Their depression may be little more than an extension of these long-standing characteristics, so that such individuals often have difficulty in determining when episodes start and finish.

The most useful therapeutic strategies are those that build up their self-confidence. This can happen naturally (e.g. by forming a relationship with someone who genuinely respects and cares for them; by being successful at work), or by giving the individual direct 'survival strategy' advice on how to handle life's vicissitudes, or via a range of counselling and psychotherapeutic approaches (including narrative therapy) that promote a sense of mastery and personal control.

Perfectionism

The next dimension involves those who rate high on reliability and conscientiousness, and who are highly self-controlled and perfectionistic. They control their environment in direct and indirect ways so as to reduce the chance of being exposed to stressful events. Such individuals are less likely to seek clinical attention as doing so would involve the risk of surrendering control—a central issue of concern. They are most likely to develop depression when they lose control of an issue that they see as their responsibility (e.g. their child doesn't take up an expected career choice; they get into an unsatisfactory relationship). This group is highly valued as community members and we seek them out to be our doctors, lawyers or financial advisers.

Their vulnerability is the flip-side to their personality style—they lack flexibility and are less adroit in situations where they are required to move from an entrenched view. Their sense of pride—not only in their work but in many other matters—leaves them particularly vulnerable to depression (and even to suicide) when their reputation or judgment is challenged or impugned.

This is a particularly difficult personality style to recommend ideal therapeutic approaches for: when their depression starts to improve, they frequently go back to ruminating about the factors that brought on the depression. They mentally torture themselves about 'Why did I let it happen?' and have difficulty in maintaining emotional distance from the psychological pain. Often recipients of critical parenting in their early years, they tend to incorporate their own 'harsh voice', so that effective therapeutic strategies need to mute their censorious self-talk.

Interpersonal sensitivity

Individuals scoring high on the dimension of *interpersonal sensitivity* tend to worry what others are thinking about them (and try to second-guess such views). When in a relationship, they have

difficulty believing that their partner truly cares about them, and are excessively concerned that their partner will reject or abandon them. When they become depressed they tend to develop features of so-called **atypical depression**, sleeping more than usual (hypersomnia) rather than less and often experiencing increased appetite (hyperphagia) rather than losing it.

In a reappraisal of this concept that our research team published in the *American Journal of Psychiatry* (Parker et al. 2002), we argued that the food cravings—often for carbohydrates and chocolate—might reflect homeostatic mechanisms (attempting to restore equilibrium) rather than be depressive symptoms. The foods that are generally craved have a comforting effect, release endorphins and provide 'feel good' sensations—and some of the craved foods lead to an increased intake of L-tryptophan, which increases brain serotonin levels.

The hypersomnia (excessive sleeping) may also be a compensating strategy in that it restores slow-wave sleep during stress for many individuals.

Ideal therapeutic strategies remain unknown, but some individuals respond well to medication (SSRIs, MAOIs) and others to psychotherapeutic approaches that address their exquisite sensitivity to interpersonal situations.

Self-focus

The *self-focused* dimension is defined as prioritising one's own needs and not being very tolerant of others. When personal needs are not met, the individual can rapidly become angry, setting the scene for interpersonal clashes and subsequent depression. Such people tend to be dramatic, emotional, volatile and often erratic in their general functioning. As the psychiatrist Kay Jamison puts it: 'For those with a short wick . . . and impulse-laden wiring, life's setbacks and illness are more dangerous' (Jamison 1999, p. 49).

These individuals are more likely to externalise their depression by raising their voice, arguing or throwing things, or by being

reckless. By releasing their frustration and distress in these ways, they often recover rapidly from their depressed mood. Those around them, however, may end up distressed from the fallout.

As Jamison notes, for those who are impetuous and volatile, 'their . . . risktaking will make them generators and throwers of sparks as well [as] . . . high-wire acts and dealers in discord' (1999, p. 49).

Strategies for treating self-focused people generally require attention to their emotional 'trigger-finger' and include courses such as anger management (described later).

Personality functioning

Finally, as noted earlier, there are two important constructs defining personality functioning—*cooperativeness* and *effectiveness*. There is historical support for these two—when asked for a definition of mental health, Freud said 'to love and to work'. 'To love' did not just mean falling in love but making and maintaining intimate relationships; 'to work' did not just mean having a job but working effectively and with satisfaction.

Cooperative people are agreeable and affiliative, while effective people are self-directed. Those who score low on these parameters tend to show tenuous stability under stress, have difficulty adjusting to the environment, cause personal discomfort to themselves and others, and often get into self-defeating cycles where everything goes wrong for them. Such characteristics increase the chance both of developing a non-melancholic depressive disorder and having it persist, while they can also make for great difficulties in working productively with a therapist.

Clinical presentation of personality styles

Under stress, and during an episode of non-melancholic depression, personality styles can be magnified. To the clinicians, such 'pattern

analyses' can often be helpful in identifying the contribution of relevant at-risk personality styles.

Those who are *anxious worriers* develop many features of anxiety in addition to their depression. Those high on the *irritability* and *self-focused* dimensions become more hot-tempered, snappy and irritable, with those in the *irritability* group being more likely to be concerned and apologetic about their behaviour. Those who are shyer (*personal reserve* and *social avoidance*) tend to become even more asocial. *Self-critical* individuals feel even more self-critical and unable to win, and think that losses and depression are part of their destiny. Those high on *perfectionism* tend to feel a greater sense of loss of pride and often blame themselves excessively. People with *interpersonal sensitivity* tend to become preoccupied with the most recent hurtful interaction, feeling rejected or abandoned.

Conclusions

Non-melancholic depressions represent what happens when an upsetting event occurs to people with personality styles that increase the risk of depression and then sustain it. Those with internalising personalities may create their own internal feedback in response to a personally relevant stressor, whether minor or major. They brood and worry about the upsetting event, become increasingly self-critical and keep the mental image of the problem humming round their circuits. They operate like a feedback loop. Those with externalising personalities may sustain their depression by over-reacting to a disturbance and generating new incoming drama, rather like a guitarist standing close to a loud amplifier.

Rather than viewing depression as a disease that is due primarily to perturbed neurotransmitter changes in the brain and is

independent of personality, our model for considering the non-melancholic disorders (at least) is to propose an interaction between a meaningful stressor (i.e. an event of major severity or one that has 'key and lock' meaning [see page 52] to the individual) and certain personality styles. We have identified a number of at-risk personality styles and considered how they may put their owners at risk and also shape the clinical pattern of the depression.

This rather specific model has a number of treatment implications. Rather than argue that any treatment (an antidepressant drug, CBT, counselling or whatever) has universal relevance to all expressions of depression, we favour a 'horses for courses' model that respects differing personality components being more likely to respond to differing treatment approaches. If non-melancholic depression is to be treated effectively, the personality contribution that can both dispose to, and maintain, depressive episodes needs to be identified and modified—so decreasing the risk of future episodes and building up resilience.

As noted earlier, you may like to assess your personality on our web-based Personality and Treatment Response Survey (www.blackdoginstitute.org.au). Finally, while we argue for personality contributing to non-melancholic disorders, personality does not create 'pure types' of depression. Thus, most people who are at increased risk to non-melancholic depression are likely to score highly on several personality dimensions.

chapter 11

FOUR VIGNETTES

The four vignettes that follow consider the role of biological, social and other factors in depression.

Sue

Sue had been a shy and somewhat conservative child. She was 24 when her mother first took her to a psychiatrist, but had had depressive episodes since early adolescence. After losing a job she became increasingly withdrawn. She was anxious, worried and afraid to go out of the house. She was also tearful and would frequently retreat to her room and stay in bed, asking her family to say she wasn't home if a friend phoned.

Her family thought she had something on her mind, but she wouldn't tell them what it was. Instead she 'stewed' on her own. Sometimes they would find her crying. Normally very reliable, she was now letting responsibilities lapse, but felt guilty at the same time for letting her family down.

Sue told the doctor that she had avoided relationships to escape being criticised. She had just been 'dropped' by her first boyfriend,

so life wasn't worth living. Her normal worrying style had been blown out of all proportion.

Catherine

Catherine was brought into hospital by her husband, Bob. He said that in recent weeks she had been behaving as she had after the birth of their son, twenty years earlier. Although they had eagerly awaited the baby, after the birth Catherine had become so deeply depressed that she was unable to look after her son. There had been one other episode of depression since then, but it had lifted after some time on antidepressant medication.

She had been going through menopause, and Bob had at first thought that this was the reason for her constant tears and withdrawal. She said that she had no energy and, even though she was tired, she could not sleep through the night. She was lethargic, moved slowly and hardly spoke. Formerly an excellent hostess, she had stopped contacting friends, and lost both her interest in cooking and her appetite.

Bob found her late one night with a bottle of tablets and, although she assured him that she wouldn't harm herself, she confessed that she had been thinking what a relief it would be to be dead.

Jason

Jason was normally an extrovert, always on the go, moving from one interest to another and needing a lot of stimulation. He was the life of the party, eager to meet people and warmed quickly to new friendships. He had had a number of relationships, but was a bit fickle. He would fall intensely in love, but then find his partner boring after a while.

He became depressed after losing his job, a consequence of a few too many 'sickies'. He started to take his frustrations out on his girlfriend, snapping at her constantly. He also got into arguments with his family.

He told his doctor that he was aware of how irritable he had become and, while he regretted it, he said he couldn't control his feelings. He had recently smashed a DVD player to 'let off some steam'. It had made him feel somewhat better, temporarily.

Harry

Harry, a 67-year-old widower, was admitted to hospital after a concerned neighbour and his general practitioner had found him unkempt and very agitated.

In hospital he initially refused food, saying that his insides had rotted away. Preoccupied and distressed by this delusion, he was unable to be reassured by the staff. He paced constantly, wringing his hands and saying over and over, 'What will become of me?'

He told staff that he was being punished for his failures and indiscretions. His guilt was out of proportion to the incidents he then described: for instance, he felt he deserved to go to jail for failing to include a minor payment on his tax return twenty years ago.

A relative remembered that Harry had been like this before, once soon after he had returned from the war, and twice in the last few years. One episode came on after Harry thought he had sped through a police radar. He was convinced that this incident had caused his depression. An uncle had been hospitalised for a similar setback.

Interpretation

In the absence of specific markers for melancholic and psychotic depression, both Sue and Jason are more likely to have a non-melancholic depression. Sue's symptoms are indicative of an internalising *anxious worrying* personality style, while Jason's are of an externalising *irritable and self-focused* personality style. Both styles contribute to the onset and persistence of the depression following particular stressors.

Catherine's and Harry's depressions appear more biologically

based, with evidence of psychomotor disturbance (retardation and agitation respectively). Harry is also experiencing psychotic episodes. Catherine is likely to have melancholic depression and Harry, with his delusions, a psychotic melancholia. Their depressions appear unrelated to personality style—the biological stressors are hormonal in Catherine's case and genetic in Harry's.

Diagnostic subtyping is assisted by considerating type-specific clinical features such as psychomotor disturbance and evidence of psychosis, and further helped by other clues presented in the vignettes. Treatment recommendations would be expected to differ for each one.

You may care to consider the most appropriate management recommendations for each of the four before reading on.

chapter 12

THE BIOLOGY OF DEPRESSION

While our knowledge of the working brain is still limited, in most instances of clinical depression it is likely that neurotransmitter function is disrupted. Another term for neurotransmission is 'nerve conduction'—but what does this mean?

The human brain is extremely sophisticated; indeed, it is far more complicated and versatile than even the most powerful modern computer. It contains in excess of 100 billion brain cells, known as neurones, each of which is connected to many other neurones. If you look at them under a microscope, neurones appear as thin wires connecting little blobs of brain tissue. However, even with strong magnification the neurone-to-neurone connections, known as synapses, are not apparent. Synapses can be electrical but the majority are chemical. A signal from one part of the brain travels to another—as a series of electrical impulses—along neurones. Where two neurones meet, the signal is carried across the synapse by the release of a tiny balloon-like packet of 'neurotransmitter', in which a message-carrying chemical is carefully packaged. Once released into the synapse this balloon immediately ruptures, releasing its chemical contents, which are then free to quickly migrate

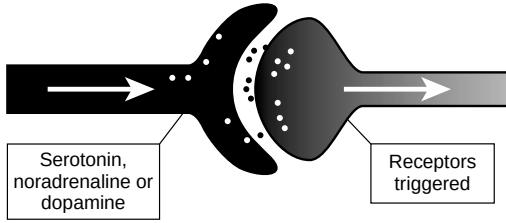


Figure 12.1 Neurotransmitters and the functioning of the brain

across the synapse and attach themselves to unique docking stations called receptors. Receptors are like light switches waiting to be switched on or off. When an appropriate chemical successfully docks with a receptor, the switch is thrown and this initiates a complex series of reactions in the next neurone that culminates in the reproduction of the original signal. In this manner, information is passed from one neurone to another and, in healthy brain function, the signal remains as strong in the second and subsequent neurones as it was in the first.

There are many different neurotransmitters in the brain, of which more than 100 or so have been identified. Each neurotransmitter serves a variety of purposes. Mood regulation involves many neurotransmitters, including some brain hormones such as cortisol. However, there are three neurotransmitters that are particularly important.

The first of these, serotonin, is the most well known and most often talked about. It is found in the blood, gut and almost all parts of the brain. Serotonin neurones have their 'home' in the base of the brain, and their connections travel upwards to other parts of the brain including the hypothalamus (the hormone centre of the brain), the hippocampus (the memory centre of the brain) and the amygdala (a region thought to be involved strongly in emotions such as fear and anger). Neurones containing serotonin make lots of

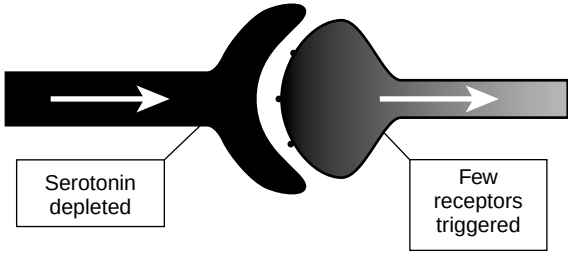


Figure 12.2 Serotonin neurotransmission in certain types of depression

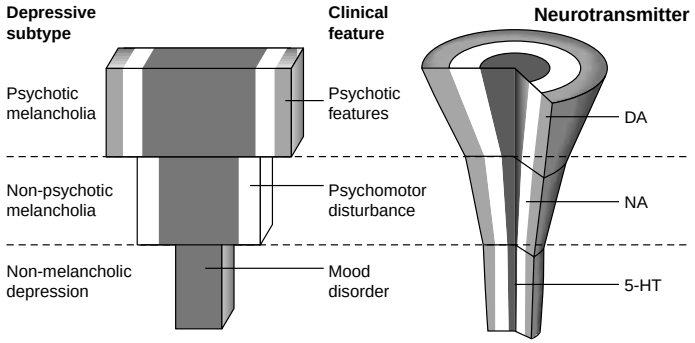
connections and therefore serotonin is involved in regulating blood pressure, temperature, sleep, appetite, sexual behaviour, pain, learning and, last but not least, mood. To serve these many functions there are several kinds of serotonin receptors into which serotonin 'docks', allowing the same signal to have different meanings depending on which kind of receptor a neurone possesses.

The second neurotransmitter important to mood regulation is noradrenaline. Its home is also in the base of the brain and, like serotonin connections, noradrenaline neurones extend to virtually all areas of the brain. Noradrenaline is, however, better known for its role in the 'fight or flight' response; it puts the brain and body on high alert in the face of imminent danger so that the individual prepares to either fend off any threat or escape it altogether. Noradrenaline therefore regulates arousal, the cardiovascular system and primes the brain to alarm signals in the environment. Interestingly, in addition to acting as a neurotransmitter, noradrenaline also regulates other neurotransmitters, in particular serotonin, and as such is thought to play an important role in regulating mood both directly and indirectly.

The third neurotransmitter of particular relevance to mood is dopamine. Dopamine is closely related to noradrenaline in that one chemical reaction converts dopamine into noradrenaline, and much of the noradrenaline in the brain is made in this way.

However, unlike noradrenaline and serotonin neurones, dopamine neurones are found in several locations in the brain and, although the connections are still quite widespread, they are much less diffuse. Like noradrenaline and serotonin, dopamine has many types of receptors and is involved in a range of processes such as reward, emotion and movement. Indeed, it is most well known for its role in Parkinson's disease, in which a lack of dopamine in a specific part of the brain eventually leads to disordered movement. It is interesting to note, then, that many patients with Parkinson's disease also develop depression. Furthermore, depression in Parkinson's disease often occurs before any movement changes become apparent, suggesting that dopamine is equally and perhaps independently critical in the development of depression.

It has been known for more than half a century that these three principal neurotransmitters play a role in regulating mood, a fact that has been convincingly demonstrated time and time again by experiments in animals and the successful use of antidepressants in humans. However, bringing together our knowledge of the brain's chemistry and our understanding of clinical depression has largely failed to respect their differential impact on key depressive subtypes. In general, and especially over the last decade, all types of depression have been equated to a lack of serotonin or disruption in its neurotransmission. This is reflected in the development of antidepressants (selective serotonin reuptake inhibitors or SSRIs) that act selectively on serotonin alone. By comparison, noradrenaline and dopamine have attracted much less attention, partly because they are judged to have greater contributions to anxiety and psychosis respectively. Consequently, antidepressants having specific noradrenaline or dopamine actions are far fewer and have only recently been developed and made available. The 'serotonin model of depression' has been useful in that many of the antidepressants it has spawned are much better tolerated and so many



DA = dopamine, NA = noradrenaline, 5-HT = serotonin.
(Malhi, Parker & Greenwood, Black Dog Institute)

Figure 12.3 Our 'structural' and 'functional' model for the principal depressive subtypes, with the functional model demonstrating the differing contribution of three neurotransmitters

more people have been treated. However, such a model is limited in terms of understanding and managing the varying types of clinical depression.

An alternate view, developed at our Institute, is illustrated in Figure 12.3, and suggests that there is a broad association between the three principal subtypes of clinical depression and the three neurotransmitters described above. In this model, we suggest that all three neurotransmitters contribute to each subtype of clinical depression, rather than one only. However, we also suggest that their proportional influence varies, such that dysfunction in one neurotransmitter clearly underpins the illness pattern in each subtype and so shapes the differing depressive symptoms and clinical pictures. There are major implications for treatment.

In non-melancholic depression, we suggest that the admixture of anxiety and depressive symptoms is largely the consequence of altered serotonin neurotransmission. This can be seen as the first line of chemical adjustment that takes place in a person, in terms of their mood, when clinically depressed. There may, in some instances, be evidence of additional noradrenaline and dopamine-related symptoms but, even then, these are often submerged within serotonin-driven abnormalities. Classic symptoms include changes in sleep, appetite and libido along with symptoms and signs of anxiety.

In melancholic depression, there are additionally clear psychomotor changes (or PMD as described earlier). Patients have difficulty initiating thoughts and movements which, if executed, are slowed. Pleasurable experiences are limited, and attention and concentration are often severely compromised. These symptoms are derived largely from noradrenaline neurotransmission disruption, although dopamine abnormalities also contribute significantly. Serotonin-driven symptoms may also be present but do not determine the clinical picture.

In psychotic melancholia, the presence of delusions and hallucinations along with marked melancholic symptoms signify the primary role of dopamine neurotransmission dysfunction. Serotonin and particularly noradrenaline still contribute, but the clinical picture predominantly reflects a dopaminergic contribution.

This model is, of course, limited in only touching on the chemistry and chemical treatment of depression. However, it is useful as it provides a model for the specific neurotransmitters, linking them to each other and to the key clinical subtypes. This allows the actions of various classes of antidepressants that act on neurotransmitters to be better explained and has ramifications for their use. Thus, the model allows a testable hypothesis (and richer clinical practice) in suggesting that while the SSRIs may be the most relevant drug class for certain non-melancholic disorders, they may

not be as effective for melancholic and psychotic depression. The model suggests that those who develop melancholic depression may require 'dual action' (i.e. serotonergic as well as noradrenergic drugs) and that those who develop psychotic depression may require an antipsychotic (dopaminergic) drug in addition to a broad-action antidepressant.

What's the solution?

As noted in Chapter 9, causes and triggers of depression can operate at different levels. So, while the differing types of depression can reflect or be underpinned by differing neurobiological processes (including neurotransmitter dysfunction), their onset may be triggered by psychological and social—as well as biological—factors. Causes, however, do not—and should not—necessarily entirely dictate treatments. Even if a depressive condition is primarily biological and requires a primarily biological approach, solutions also need to be considered at psychological and social levels. How can this proceed?

Biological solutions

Imagine all the synapses as leaky rain tanks running low on their particular neurotransmitter. The obvious solution is to find a way of refilling the tanks. This can be done by turning off the tap or by mending the leak.

Turning off the tap. This means, stop doing anything that is contributing to the problem. For example:

- address the trigger that caused the problem;
- sleep where you are unlikely to be disturbed;
- check whether any medication you are on can cause depression;
- get any thyroid or other medical problem treated;
- avoid caffeine as it contributes to insomnia and anxiety;

- as far as possible avoid major life changes or dramas;
- avoid alcohol and illicit drugs;
- treat chronic pain;
- declare a truce in major conflicts; and
- don't take up smoking.

Mending the leak. This means stopping the drain of neurotransmitters from the synapses by using an appropriate antidepressant medication. If our model is applied, the chance of someone with non-melancholic depression responding to an antidepressant is likely to be similar across all antidepressant classes. For someone with melancholic depression, the antidepressants most likely (but not necessarily required as the first drug of choice) would be those acting on noradrenaline and serotonin—and with possible dopamine action as well. The tricyclic antidepressants and MAOIs are the best examples of these drugs, with their modern equivalents including venlafaxine, duloxetine, reboxetine and mirtazapine. For those with psychotic depression, an antipsychotic drug would be required in addition to such an antidepressant. The roles and advantages of those drugs with specific noradrenergic and dopaminergic actions need to be clarified within this model.

Some of the antidepressants have the potential to influence other 'biological processes' that can increase the risk of depression. For instance, the SSRIs mute anxious worrying and irritability, personality traits that are most commonly observed in those with non-melancholic depression, and thus increase resistance to future depressive episodes.

Psychological solutions

Psychological treatments aim to support and counsel the individual during the depressive episode, build resistance to future episodes by increasing self-esteem, modify at-risk personality styles and address the triggering stressors. Cognitive behavioural therapy

(CBT) and interpersonal therapy (IPT) are the most common formalised approaches (detailed in Chapters 16 and 17). A number of recent studies have also provided evidence suggesting that when CBT is added to mood stabilising medication for those with bipolar disorder, the outcome is improved as compared to those receiving such medication alone.

Social solutions

Social interventions aim to decrease the occurrence and impact of stressful events and promote the socialisation of the individual. The depressed person is encouraged to take part in pleasant activities, counteracting any tendency to shun social contact and thus maintain the depression. A shy individual may be encouraged to develop a repertoire of socialising strategies (through social skills training) or to use assertiveness training to become more self-assured. Social engineering approaches are also relevant here; for example, encouraging someone in a dysfunctional relationship or work environment to make changes that will boost self-esteem. The point to be emphasised is that rather than the individual changing themselves, the objective is to determine a better or more appropriate 'ecological niche', so that the individual's self-esteem is boosted and their resilience increased.

chapter 13

PROFESSIONAL ASSESSMENT

You just can't seem to get good help these days.
Helen Razer, *Gas Smells Awful*

Most people with depression consult a professional willingly but some, particularly adolescents, may have to be forcefully encouraged or even coerced by others. If you find yourself in this position you should tell the professional that you are attending 'under sufferance' and then establish the rules that will apply to the relationship. Minors may be concerned that the assessment procedure will result in information being passed on to their parents, and adults may worry that their case will be discussed with their spouse.

Professionals have an obligation to respect confidences. Any concerns about how the interview data will be recorded, or who will have access to it, should be raised at the start of the interview. If the professional is able to provide reassurance about patient confidentiality, the patient should try to face the assessment with an open mind.

It is becoming more common for patients to bring a family member or friend to an assessment interview. In this case, the professional must establish whether the patient wants to be interviewed alone or with the other person present. It is usually the

patient's call; rarely will the professional make the decision. It may be best to start the assessment alone, as this gives the patient more control over private issues. A family member or friend can be invited to join at a later stage, to give their own observations and to be involved in the development of the patient's management plan.

If, however, you are a relative or a friend of someone with depression, the rules are less clear and guidelines are more difficult to draw up. If you are encouraging someone to see a psychiatrist, do not disguise the issue. Do not wait until the day of the appointment before telling them about it. Do not tell them that the appointment is with a 'doctor' or a 'counsellor' if it is with a psychiatrist.

If it is unlikely that your relative will tell the professional of any risks their disorder may pose, such as self-harm, inform the professional or the secretary/receptionist directly of your concerns. If your relative or friend has a very severe disorder such as psychotic depression or mania, try to accompany them to the appointment. If you are not asked to be present during the initial assessment, ask that you are at least briefed about the management plan. Ideally, however, you should be present while the management plan is being discussed as this will prevent or limit miscommunication, and you may have valuable contributions to make to its development.

What a detailed assessment might cover

At the initial assessment the following questions are the kind to expect:

- Is depression the principal disorder, or is it secondary to some other condition (e.g. anxiety or substance abuse) that should be assessed and treated?
- If depression is the principal disorder, what are its key features (in order to determine the depressive subtype)?

- Is the lifetime pattern unipolar or does the patient have bipolar disorder?
- What is the risk, if any, to the patient of self-harm, harm to reputation or even harm to others?
- What is the current level of disability?
- Were there any triggers (e.g. stressful events) to the episode?
- If there were triggers, did they entirely cause the depressive state, did they activate or worsen it, or were they merely coincidental?
- How did the patient interpret the triggers, and what thoughts did they activate?
- Is the patient part of a family network and, if so, what is the quality of the relationships?
- What can the patient remember about their childhood, including the level of parenting received, their interactions with other children and their experiences at school?
- Is there any family history of depression or other relevant medical problems?
- How many and what types of jobs has the patient had and what level of satisfaction, if any, was there?
- What is the quality of the patient's relationships with intimates, peers, workmates? Have those relationships been sustained over time?

The professional should also seek to establish:

- the patient's personality style and repertoire of coping responses, identifying particularly any **cognitive style** (i.e. any cognitive schema or at-risk thinking patterns) that may increase the patient's vulnerability;
- a drug and alcohol history;
- any medical/surgical problems, in particular any that may have contributed to the depression;

- whether or not the patient suffers from any allergies, especially to medication;
- any cognitive limitations affecting concentration, memory and intellectual functioning;
- the patient's life history of depressive episodes, previous treatments and perceived effectiveness or ineffectiveness of these treatments;
- any current 'sustaining' factors to the depression—for example, ongoing work problems or dysfunctional relationships;
- the patient's own views about the reasons for their depression, and their preferred treatments.

What the patient should be told

Just watch out for One Solution Fits All doctors.

Helen Razer, *Gas Smells Awful*

If the outcome of the assessment is that the patient is depressed, the professional should tell the patient the diagnosis and identify the likely depressive subtype. If the patient is suffering from another disorder—for example, anxiety or bipolar disorder—this should be formally acknowledged. If the diagnosis (or its causes) are unclear, this is worth stating—and how it is likely to be clarified (e.g. information from a family member, observation over time, response to treatment, second opinion).

The professional should also identify to the patient any other problems of significance, as well as any medical or emotional conditions that require investigation and/or treatment. It is important to provide the patient with a pluralistic explanation (i.e. one that takes all relevant factors into account) of why the disorder developed at this particular time. This involves integrating past environmental and developmental factors with genetic influences, stress levels and personality interactions.

The professional should also recommend an immediate management strategy and describe the lines of responsibility for those involved in the case. For example, the general practitioner is to handle X (e.g. testing for medical factors) and the non-medical professional to handle Y (e.g. counselling). There are great benefits in the professional making the goals of therapy quite explicit (perhaps at the first session, or after several meetings), rather than leaving them implicit or unnegotiated. The professional should always give the patient an accurate assessment of the costs involved and the likely advantages of the management plan.

Different professionals (psychiatrists, psychologists, general practitioners, nurses, social workers, occupational therapists, counsellors) have different training backgrounds and therapeutic orientations. Their therapeutic approaches may range from the very narrow to the very broad, each having advantages and disadvantages for the management plan.

chapter 14

DRUG TREATMENTS

The most common treatments for depression are drugs. This chapter looks at the three types of drugs used:

- antidepressants;
- tranquillisers;
- antimanic drugs, or mood stabilisers.

Antidepressants

There are several separate families of antidepressants and various antidepressant **drug classes** whose effectiveness differs across the depressive subtypes. For this reason, and for other reasons detailed in Chapter 12, antidepressants should not be discounted if one particular type does not work. Even if two antidepressants fail, a careful review should be undertaken. This could show that the patient would benefit from another antidepressant drug, or a combination of drugs.

Although there is no universal method of labelling drugs, antidepressants can be classified according to generation, chemical characteristics and the function of the drug. Drug 'generation' refers to

the period of the discovery of the drug and its formal release. Thus, the first-generation antidepressants emerged in the late 1950s and were extensively trialled in the 1960s. Second-generation antidepressants emerged in the late 1970s and early 1980s, while third-generation drugs have generally been available only since the 1990s.

The defining chemical characteristics of the drug classes are determined by their nuclear structure. 'Tricyclic' drugs have a three-ring structure, 'tetracyclic' four, and recently developed 'bicyclic' drugs have a two-ring structure.

The third element underpinning an antidepressant's label refers to the function of the drug. For example, drugs that block the enzyme, monoamine oxidase (MAO), are subdivided into those that block the enzyme irreversibly (the MAOIs) and those that do so reversibly (the RIMAs). While most antidepressants have multiple actions, many work by inhibiting the reuptake or reabsorption of one or more different neurotransmitters (including serotonin, noradrenaline and dopamine) at the nerve synapses, thus increasing the concentration of the neurotransmitter. First-generation antidepressants (the tricyclics and MAOIs) act on multiple neurotransmitters. The newer drugs are more selective in the neurotransmitters they target. This selectivity is captured in the drug class name, such as selective serotonin reuptake inhibitor (SSRI) or selective noradrenaline reuptake inhibitor (SNRI).

Are all antidepressants of equal benefit?

Formal trials comparing one drug class against another have allowed many scientific reviewers to conclude that all antidepressants are of equal benefit, and that their differences are due to varying side effects. We, however, argue strongly that the equal-benefit view is a myth, with some antidepressants being far more powerful than others in helping certain types of depression. The equal-benefit view was built largely on data from drug trials, where relatively 'pure' cases of depression are included (i.e. those with coexisting

other problems are disallowed) and the depressive disorders are generally less severe and more likely to remit spontaneously in comparison to patients seen in clinical practice.

Also, patients with melancholic and psychotic depression are rarely included in clinical trials. When data are collected from 'real world' clinical practice studies, a different picture emerges—one that shows that the different classes of antidepressants vary considerably in their effects on different types of depression.

Antidepressants and non-melancholic depression

In this subtype the role of antidepressant drugs needs to be clarified more precisely. All antidepressant classes bring about a significant improvement in depression severity (say, a 60% chance of the patient responding) but this is not as high as it might seem—people with many of these conditions also benefit from discussion with an empathic interviewer or from counselling, with such therapeutic ingredients contributing significantly to that 60% figure.

For non-melancholic depression the selective serotonin reuptake inhibitors (SSRIs) and dual action (i.e. serotonin and noradrenaline reuptake inhibitor—SNRI) drugs (perhaps at low dose) are probably the benchmark antidepressants. They are just as effective as the older first-generation antidepressants but better tolerated in terms of side effects; they may also have beneficial effects beyond treating the depression—for example, by decreasing worrying, brooding and irritability. These drugs have powerful anti-anxiety capacities and, as many of the non-melancholic depressive conditions are contributed to by the individual's anxiety levels, the drugs operate to address any underpinning anxiety.

People with an *anxious worrier* personality style commonly state that these drugs (and particularly the SSRIs) induce a sense of detachment from their problems. The problems are still there but are viewed as if by a non-worrier, so that sufferers feel they are swimming rather than sinking. This means that patients worry less,

or for briefer periods, so decreasing the chance of worry developing into a depression. If the patient is already depressed, these drugs may make the depressive episodes briefer and more manageable. They also seem to help a significant percentage of those who externalise their anxiety with bursts of irritability. Regrettably, the literature examining the impact on other personality styles is limited, and is being pursued in our research.

Antidepressants and melancholic depression

The older tricyclic drugs (TCAs) and irreversible monoamine oxidase inhibitors (MAOIs) appear to be more effective than the SSRIs for melancholic and psychotic depression, but tend to have more side effects. The effectiveness of a number of the other, newer antidepressants for these depressive subtypes is less clear. Some may be too refined in their action, unlike first-generation antidepressants, which impact on multiple neurotransmitters. Unfortunately, their impact on multiple neurotransmitters also increases the range of possible side effects. First-generation antidepressants have also been shown to be more effective in older patients, those who have had multiple episodes and when PMD is severe. In one of our research studies, for patients with melancholic depression and over the age of 60, we established that their chance of responding to a TCA was four times as great as for an SSRI. At our clinics we see many so-called 'treatment-resistant' patients with melancholic depression who have been trialled only on narrow-action antidepressants; often we are able to initiate a response merely by using a broader-action drug. Regrettably, many doctors have been scared off using broader-action drugs and have accepted the myth that all antidepressant drugs are equally potent, resulting in much undertreatment for those with melancholic depression.

It would be reasonable for an individual with a first episode of melancholic depression to be started on an SSRI or SNRI. If treatment is successful, it can be recommended for any subsequent

episodes. If this first-line treatment fails, it may need to be augmented by other drugs (see below) or a TCA or MAOI considered.

Antidepressants and psychotic depression

It is generally not enough to treat psychotic depression with antidepressants alone. Most cases of psychotic depression will require a combination of drug therapies—for example, an antidepressant and a tranquilliser (see pages 95–6). In some cases, electroconvulsive therapy (ECT) may be needed.

Augmentation of antidepressant drugs

The effectiveness of some antidepressants can be increased by the use of augmentation drugs such as thyroid hormones or lithium.

There is increasing evidence to suggest that the new atypical antipsychotic drugs (see pages 95-6) may also have augmenting effects on antidepressants. They often work rapidly and can also be ceased rapidly in many cases. Our research suggests that the antidepressant needs to be 'on board' first, that it has to be a potentially effective antidepressant (dual action or even broader in action) and that, if augmentation is to occur, the benefit will be observed within one week (but often in the first couple of days). If the augmenting drug induces a complete remission, we recommend it being ceased after a few days—and thus reserved for any further need for an augmenting drug.

While not investigated formally, the benefits of such augmenting drugs may be of greatest relevance to those with melancholic and psychotic depression or who have depression as part of a bipolar disorder.

How useful is St John's wort (hypericum) as an antidepressant?

Williams and colleagues (2000) considered the data collected from 14 trials of the antidepressant properties of the popular herbal remedy St John's wort, involving over 1400 adults. In eight

trials subjects were given St John's wort or a placebo; in the other trials they took St John's wort or a tricyclic antidepressant. The doses of St John's wort ranged from 300–1800 mg/day.

'Responders' were defined as those whose condition improved by 50 per cent or more. The aggregated studies indicated that 38 per cent of those receiving a placebo were responders, compared to 62 per cent of those receiving St John's wort and 61 per cent receiving the tricyclic antidepressant.

Such data would seem to indicate that St John's wort is an effective antidepressant (being significantly superior to the placebo) and of comparable effectiveness to tricyclic antidepressants. However, as trials were generally undertaken on those with mild depression, St John's wort is likely to be of possible assistance only to a percentage of people experiencing non-melancholic depression.

In 2002 a large comparative study of St John's wort, an SSRI and a placebo for treating 'major depression' was published in the *Journal of the American Medical Association* (Hypericum Depression Trial Group 2002), with neither drug being superior to the placebo over the eight-week trial. As suggested earlier and reviewed elsewhere (Parker et al. 2003), we feel that such findings are more likely to reflect the limitations of current clinical trial procedures and use of the non-specific category of 'major depression'.

The status of St John's wort as an antidepressant remains to be clarified, but its effectiveness is likely to be limited to milder expressions of non-melancholic depressive disorders.

St John's wort can have side effects and can interact with other drugs, and there are now several reports suggesting that it may have some toxic effects on reproductive functioning. As with any drug, care should be taken to consider its side-effect profile.

How quickly do antidepressants work?

Most treatment guidelines suggest that antidepressants may take many weeks to work. It is argued that, even if the current treatment

seems ineffective, it should be persisted with for several weeks or even months. Our Mood Disorders Unit research team at the Black Dog Institute interprets the evidence differently.

If medication is likely to be effective, evidence of at least some improvement should appear in the first ten days or so, whether it be an improvement in mood, sleep or other features. For melancholic and psychotic depression, the rate of improvement is generally slower (but relatively constant). It may, in fact, appear painfully slow.

If no improvement is noted in the first two weeks after commencing an antidepressant, the dose of that drug may need to be increased, a change to another class of antidepressant may be required, or augmenting strategies (the addition of quite different drugs) may need to be introduced. Unfortunately, when changing from one drug to another, days to weeks may pass before success can be established. It might also be the case that non-drug strategies will be more effective in bringing the depression to an end.

It is important to challenge the myth that antidepressants need to be trialled for many weeks or months so as to ensure that patients are not left on an antidepressant for an extended period with the view that it might start working after two or three months.

Antidepressant drugs for children and adolescents?

This is a topic undergoing close review. We have known for a long time that the older antidepressants (such as TCAs) were not beneficial for children and young adolescents. For much of the last decade, however, it was thought that the SSRIs were beneficial. The actual evidence now suggests a weak 'signal' of benefit at best, and it appears that young people are more likely to develop serious troubling side effects on SSRI medication. In adolescents, depression is most commonly expressed as irritability. It may be, then,

Table 14.1 Currently available antidepressant drugs

	Drug name
First-generation antidepressants	
Tricyclics (TCAs)	Amitriptyline Clomipramine Dothiepin Doxepin Imipramine Nortriptyline Trimipramine
Irreversible monoamine oxidaze inhibitors (MAOIs)	Phenelzine Tranylcypromine
Second-generation antidepressants	
Tetracyclics	Mianserin
Third-generation antidepressants	
Selective serotonin reuptake inhibitors (SSRIs)	Citalopram Escitalopram Fluvoxamine Fluoxetine Paroxetine Sertraline
Serotonin and noradrenaline reuptake inhibitors (SNRIs)	Duloxetine Venlafaxine
5-HT2 blockers	Nefazodone
Reversible inhibitors of monoamine oxidase-A (RIMAs)	Moclobemide
Other drug types	
Noradrenaline reuptake inhibitors (NARI)	Reboxetine
Dopamine-noradrenaline reuptake inhibitors (DNRIs)	Bupropion
Noradrenergic and specific serotonergic antidepressants (NaSSA)	Mirtazapine

that some adolescents are more likely to experience worsening of irritability when prescribed an SSRI, thus putting them at risk.

As we do see depressed children who benefit considerably from SSRI medication (perhaps more for its anti-anxiety effect) and who, when without this medication, suffer considerably (often requesting its recommencement), guidelines for proceeding will need careful thought.

Tranquillisers

Antidepressant drugs are significantly different from the class of drugs known as tranquillisers. This class can be divided into minor and major types.

Minor tranquillisers

Most of the minor tranquillisers belong to a family of drugs called benzodiazepines, examples of which are alprazolam, diazepam, lorazepam and oxazepam.

Benzodiazepines are addictive and may exacerbate depression or interfere with the normal grief process. In recent years they have been greatly overused, leading to an understandable suspicion of psychiatric medication. An unfortunate effect of this overprescription has been a misplaced fear of antidepressants, which are not addictive in the sense of the individual developing increased tolerance.

Major tranquillisers

The major tranquillisers (also known as neuroleptic or antipsychotic drugs) are of particular benefit in treating psychotic depression when administered together with an antidepressant.

The newer atypical antipsychotic drugs have a different side effect profile, generally making them more tolerable to patients. As well as being effective in treating psychotic depression, they may (as noted earlier) be of some assistance as augmentors of antidepressant drugs where, if effective, results are often rapid.

Antipsychotics are thought to work by blocking the action of the neurotransmitter, dopamine. There are two main groups of antipsychotics—typical and atypical.

The term 'atypical' has many meanings. As a class, these drugs are serotonin–dopamine antagonists (SDAs), interacting with key brain dopamine pathways. They have different side effects to older typical

antipsychotics; in particular, they are less likely to cause muscle stiffness, are generally easier to tolerate and somewhat safer—although not without side effects that need to be appreciated.

Antimanic drugs or mood stabilisers

As noted earlier, Bipolar Disorder I affects about 1–2 per cent of adults in the community, and Bipolar Disorder II perhaps another 5–10 per cent, with more than 50 per cent of both groups likely to experience the condition before the age of 30.

Most of our knowledge about the use of antimanic medications is in reference to Bipolar I. By decreasing both the frequency and amplitude (height and depth) of the mood swings, the antimanic medications lithium, valproate and carbamazepine (usually called mood stabilisers) are generally viewed as being equally effective in the treatment of bipolar disorder. (Lithium is also used to strengthen, or augment, antidepressants when they are ineffective in dealing with unipolar depression. Such a role appears to be of likely benefit only to those with melancholic depression.)

It has been suggested that each of those three drugs may have specific advantages, depending on their different side effects and the profile of the disorder. In the last two years a number of studies have been published indicating that the atypical antipsychotic drugs (see above) also have mood-stabilising properties, so that these are now somewhat more likely to be used in managing an inpatient with acute mania.

Many patients who do not respond to or tolerate a particular antimanic medication will usually do well with one of the others, while patients with severe cases of bipolar disorder may require combination therapies. Particularly severe cases may require the additional use of one of the major tranquillisers.

Antimanic medications are effective in treating episodes of mania and also in preventing relapses into mania and depression. When

unsuccessful, combinations of mood stabilisers, or the introduction of newer mood stabilisers (e.g. lamotrigine) may be tried.

When someone is experiencing a manic episode, all mood stabilisers generally take several weeks or more to work. For this reason, it is usually also necessary to prescribe a calming medication, such as a tranquilliser, which is withdrawn once the mania settles. A person experiencing mania often has no awareness or insight into the changes of behaviour that are occurring. The patient cannot understand the need for treatment—a need that is obvious to friends and family. Recovery takes at least several weeks, sometimes months. For someone with a first-onset episode, six months of ongoing treatment may be sufficient to prevent a relapse. The more severe or frequent the episodes, the more compelling the argument for long-term treatment.

Some people with bipolar disorder find it difficult to take a mood stabiliser regularly. This may be due to a reluctance to accept the diagnosis or the need to take medication regularly, or to see whether they can manage without tablets. Called 'poor compliance' or 'poor adherence', it is a common reason for relapsing into a new episode. (In such cases, it is important to understand why the medication was stopped.) Others, however, experience further episodes despite adherence to medication. Patients and families need to understand that most people who experience a manic episode will have further episodes of mania and/or depression.

As noted earlier, we are now recognising that Bipolar II is becoming far more common, although it is unclear whether there is a true increase in the condition or detection is improving. Rules for management may not necessarily be the same as for Bipolar I disorders.

First, it might be useful merely to have the individual chart their moods for several months and rate the impact and disability of their condition before deciding to begin medication.

Second, observations at the Black Dog Institute suggest that many people with Bipolar II will report improvement in their

highs as well as their lows on SSRI or SNRI drugs alone. We are now investigating whether these drugs might have mood-stabilising (or modulating) potential.

Third, many patients with Bipolar II conditions do benefit from orthodox mood stabilisers such as lithium and valproate, although the dose level does not always need to be as high as for those with Bipolar I disorders.

Frequently asked questions about drug treatments

Three of the most common questions asked by patients when discussing their drug treatments are:

- Must I take medication?
- How long will I need to stay on medication?
- Which drugs should I consider and what are their side effects?

Must I take medication?

There should be no general rule applied to any individual about the need to take medication. Someone who presents with a recent non-melancholic depression that came on after a major stressor, and who has few clinical symptoms, will often do well without the need for antidepressants.

If, however, depression came on for no good reason, sleep patterns are affected, lack of emotional control is making the condition worse, or the doctor thinks the patient might have a melancholic depression, it would clearly be better to trial an antidepressant.

With non-melancholic depression, there is no one treatment. It's a bit like deciding what to do when a car runs off the road into a ditch. If advice to rev the engine (counselling, psychotherapy) moves the car back towards firm ground, fine. But if all that is happening is that the wheels are spinning, it's better to put a sack

under the tyres to aid grip or call a tow-truck (medication) as well. As noted on page 90, many antidepressants, particularly the SSRIs, act on a patient's predisposing and perpetuating personality traits (such as worrying). They may therefore indirectly prevent the onset—as well as shorten the duration—of depressive episodes.

When deciding to stop taking an antidepressant, the patient must check how to taper the dose. Suddenly ceasing all medication without slowly decreasing the dose can lead to severe emotional and physical reactions including anxiety, agitation, insomnia, severe sweats and racing heart. For some medications, even missing one dose can initiate a withdrawal reaction.

For melancholic and psychotic depression, and bipolar disorder, the advantages and need for physical treatments are distinct, with recovery quite unlikely to occur otherwise. While physical treatments may be the mainstay, complementary counselling and psychotherapies do increase the chance of improvement (now also shown for bipolar disorder). While patients 'need' and appreciate a combination approach, it is important to note that combination approaches have now been clearly demonstrated to be superior to physical treatment (medication, ECT) alone.

How long will I need to stay on the medication?

There are many treatment guidelines that have strict rules, including views that patients should remain on medication for life if they have experienced either one severe depressive episode or a number of episodes. Such rules are too prescriptive and restrictive for many of those affected. However, there is an argument for extended medication treatment if a single episode of depression

- was extremely severe;
- put the patient at considerable risk and compromised their functioning for an extended period; or
- was extremely difficult to treat.

In contrast, however, a patient may have had numerous depressive episodes precipitated by social factors. At some stage in the recovery process, these social factors become irrelevant. In such cases, it is difficult to argue that antidepressant medication should be taken for an extended period.

Decisions on how long a patient should receive medication should therefore be made on a case-by-case basis. The prescribing clinician needs to decide what medication is required to ensure recovery from a particular episode, as well as whether there is a need for continuation or maintenance medication to prevent subsequent episodes. Large samples of data are available to help the clinician on such issues, but the very fact that the analyses are based on grouped data means that they should be used only as a guide.

Once a patient has recovered, the decision whether or not to alter the dose or stop the drug will depend on the individual case.

Which drugs should I consider and what are their side effects?
The decision on which drugs to take is best made between the individual and the treating doctor.

Most drugs generate long lists of possible adverse effects, even though the probability of a patient developing any of them is in most cases negligible. However, some antidepressant drugs potentially have very severe side effects, particularly if taken in conjunction with other drugs. It is the responsibility of the treating clinician to address these issues at the individual level.

For those seeking reference material on the side effects of drugs, drug interactions and drug safety during pregnancy and breast-feeding, two publications are recommended: *Psychotropic Drug Guidelines* and *The Maudsley Prescribing Guidelines*.

Most pharmaceutical companies provide quite detailed product information leaflets that cover side effects and frequently asked questions about individual drugs. If in doubt about a side effect or the possible complications of combining certain medications,

contact the relevant pharmaceutical company by phone—most provide a responsible and helpful service. Bimonthly and yearly *MIMS Manuals* (Monthly Index Medical Specialty) give detailed and abbreviated information on drug side effects and related issues. (See the Reference section at the end of this book.)

chapter 15

ELECTROCONVULSIVE THERAPY AND TRANSCRANIAL MAGNETIC STIMULATION

Some depressive states, especially severe psychotic depression and melancholia, may require non-medication intervention. While there are several possible strategies, we consider two in this chapter—electroconvulsive therapy and transcranial magnetic stimulation.

Electroconvulsive therapy

Electroconvulsive therapy (ECT) is a modern psychiatric treatment that is effective for a range of psychiatric disorders, not only melancholic and psychotic depression, but also (in certain circumstances) manic disorders.

A recent sample of patients assessed at the Mood Disorders Unit at the Black Dog Institute, who had recovered from severe depression, rated ECT as the most effective treatment for their illness. More than 80 per cent of patients who have undergone ECT are willing to receive the treatment again. That said, many patients are reluctant to start a course of ECT.

While the evidence indicates that ECT is a highly effective treatment for severe expressions of all depressive subtypes, its low initial

acceptability by patients and its side effects during the treatment argue for it being used only for a limited set of depressive conditions.

Who might need or benefit from ECT?
Generally, those who haven't responded to other treatments may benefit from ECT. However, ECT is occasionally used as a first-line treatment for patients who have responded well to it previously, or who have the following conditions:

- severe psychotic depression;
- severe melancholic depression (where the patient is too ill to eat or drink, is unable to take antidepressant or antipsychotic medications, or presents an immediate risk of suicide);
- life-threatening mania (with exhaustion and delirium); or
- severe postnatal depression.

How long does ECT take to work?
Most ECT is given at a frequency of three treatments a week. Evidence of improvement usually occurs in the first seven to ten days (during the first four treatments), but recovery from the depression and lasting improvement may require many more treatments, with the standard course lasting three to four weeks.

Improvement is best judged on the days when ECT is not given.

What's the procedure?
Patients are given a general anaesthetic to bring about sleep. They are given oxygen, and medication to relax their muscles. Next, electrical stimulation is applied through electrodes attached to one or both sides of the scalp. This causes a brief convulsion. The resulting activity in the nerve cells helps to release chemicals that restore normal functioning to the brain. The changes in the nerve impulses and neurotransmitters that occur are similar to those seen during antidepressant treatment.

There are two forms of treatment—unilateral and bilateral. Unilateral ECT involves placing both electrodes on the scalp over the non-dominant hemisphere. In bilateral ECT, electrodes are put on both sides of the head. Bilateral ECT is more effective and produces a more rapid response than unilateral ECT but, as it has some side effects, unilateral ECT is the preferred treatment.

Two doctors (one trained in psychiatry, one in anaesthetics) and a number of nurses remain with the patient in the treatment suite, a specially equipped area of a hospital's psychiatry department. Treatment is usually given in the morning after the patient has fasted from midnight the night before.

Patients are linked to both an ECG (electrocardiogram) to measure electrical events in the heart, and to an EEG (electroencephalogram) to measure brain waves; their heart rate and blood pressure are carefully monitored during the procedure. There are also 'stimulus dosing' procedures that determine and control the most appropriate level of electrical stimulation.

The patient wakes in a recovery room about twenty minutes after the end of the treatment under the observation of trained staff.

How safe is ECT and what are the side effects?

ECT is a very safe treatment, even for those with other physical problems or illnesses. There is no definitive evidence that it harms the brain, but a small percentage of people do describe some memory changes (e.g. needing to take a shopping list) following a course of ECT, so that the cost–benefit ratio in deciding on ECT should always be respected.

Procedural side effects include headaches (relieved with paracetamol) and some confusion, which usually clears within hours. Memory for recent events, dates, names of friends and so on may not be as good for up to a week (occasionally longer) after treatment, but many patients find that their memories from the time of their illness are somewhat hazy anyway.

ECT still attracts adverse publicity, but it is a very effective and sometimes lifesaving treatment. Abrams (1992) is a useful technical reference.

Transcranial magnetic stimulation—an alternative to ECT?

Transcranial magnetic stimulation (TMS) is a procedure used by neurologists, both as a treatment and as a diagnostic procedure. A coil is held near the patient's head and a magnetic field created to stimulate relevant parts of the brain. Unlike ECT, there is no need for a general anaesthetic nor is a convulsion induced.

A number of studies have been completed to assess the effectiveness of TMS when compared to ECT. One TMS study undertaken at our Mood Disorders Unit suggested that it was unlikely to be as effective as ECT.

Considerable development work is being done on TMS, comparing bilateral and unilateral TMS and varying the relevant stimulus parameters. No clear statement about its utility is expected for a number of years. If TMS is demonstrated to be as effective as ECT, this would be a distinct advance in the treatment of many mood disorders.

For those wishing to learn more about TMS and its technical aspects, the text by George and Belmaker (2000) is recommended.

Vagal nerve stimulation

Several international studies have been undertaken to determine whether vagal nerve stimulation (VNS) (which involves a surgical operation to insert a stimulating device into the vagal nerve) is of benefit for those with treatment-resistant depression as it has been for some people with epilepsy. Results have generally been disappointing, failing to identify any clear-cut short-term benefit.

chapter 16

COGNITIVE THERAPIES

People who develop depression—particularly those who develop non-melancholic depression—often have an ongoing negative view of themselves, even when they are not depressed. They distort their experiences through a negative filter and develop thinking patterns that are so entrenched they don't even notice the errors of judgment caused by thinking irrationally.

Depressed people tend to focus on their shortcomings and ignore their positive points. They may also read rejection and criticism into events that are, in fact, neutral. If things go wrong, they assume that it is their fault, and the future often seems packed with potential disasters for them and their families. Even pleasant things can be interpreted in negative ways, leading to feelings of distress. So, people who think in this way often end up feeling low, having talked themselves into this frame of mind.

To deal with this, many cognitive therapies have been developed that seek to correct such thinking patterns—and extend that thinking into new behavioural patterns. This chapter details the key underpinnings of cognitive behaviour therapy (CBT) and notes some other cognitive strategies.

Here is an example of the cycle of *event-faulty thinking-reaction*:

- Your best friend forgets your birthday. *(Event)*
- You think, 'If she really liked and cared for me, she wouldn't have forgotten my birthday. I guess no one really cares about me. I'm worthless.' *(Faulty thinking)*
- Depression, loss of self-esteem. *(Reaction)*

CBT brings a problem-solving approach to the identification of thoughts and behaviours that precipitate and perpetuate depression.

In the sequence *event-thought-reaction*, most people aren't consciously aware of the step in the middle: thought. Habits of mind aren't a problem unless we find ourselves constantly feeling bad. When we reach that stage, we need to look at the way we are thinking and see whether we need to change some of our thinking habits.

The objective of CBT is to make patients aware of their thinking habits and how these habits contribute to feelings of depression. It aims to change negative thinking habits so that people can stop themselves feeling so bad. It is also something they can learn to do for themselves.

Either in one-on-one therapy sessions or in small groups, the therapist teaches people to look logically and rationally at the evidence for their negative thinking, thus helping them adjust the way they view the world around them.

The therapist will provide homework to be done between sessions. Patients may be asked such questions as:

- What do you think of yourself?
- What do you make of your experiences?
- How do you view the future?

This is really extended self-therapy. By using CBT tools and

observing the way they think about their world, patients will find out how to examine the truth, or otherwise, of their everyday assumptions and interpretations. Thinking negatively is a habit and, like any other bad habit, it can be broken.

Schemas, and learning to reframe them

The concept of *schemas* comes from the idea that children evaluate themselves and their environment, through interaction with significant others, in order to construct their own 'reality'. The automatic thoughts and other evaluations of day-to-day experiences that tend to reinforce or support this 'reality' are known as 'underlying assumptions'.

Underlying assumptions may be adaptive and lead to positive attitudes (e.g. 'I will have a go at most things and do a reasonable job' or 'People are basically helpful'). They may also be dysfunctional (e.g. 'I have to be the best at everything' or 'People generally let me down') and leave a person vulnerable to the onset of depression, or at risk of subsequent relapse.

People can learn to reframe and modify their existing schemas or deeply entrenched assumptions, as well as adjust their approach to problem-solving and the life values they hold as a result of early experiences. There are four steps to achieving this:

- *Step 1 Appraise the situation leading to depression.*
  What is going on? What is the pattern? What are my reasons for reacting this way? Are they justified? Where is this getting me?
- *Step 2 Evaluate the thoughts and beliefs triggered by events.*
  What is the evidence for how I am thinking? What would it take to make it change? How strongly do I believe in these thoughts and feelings? How much do I have invested in this?
- *Step 3 Substitute helpful thoughts.*
  How else can I view this? What is another way of looking at this?

What has worked for me in the past? What would the helpful part of me advise?

- *Step 4 Try out new ways of thinking and behaving.*
  What new idea or option can I try? If, by some miracle, things were different, how would they be different? What is required to make this difference? What option will I try first?

Becoming aware of our thoughts

Whenever we feel upset, depressed, guilty or angry, we should stop what we're doing and try to become aware of what we are thinking, or what images are going through our minds. If we find ourselves saying 'I felt that . . .' or 'I thought that . . .', we are probably about to express a thought and not a feeling. For example, 'I felt that he had no right to say that to me' is a thought, not a feeling.

We can all identify some of the familiar put-downs we use against ourselves, or the negative thoughts we have about ourselves; for example:

- 'I'm hopeless at . . .'
- 'Everyone is smarter than me.'
- 'I'll never get over what he said to me . . .'

The aim is to gather more accurate information about our thoughts so that we can pinpoint and counteract distortions.

Faulty thinking habits

Let's look at a character called Bill who, unfortunately for him, illustrates all of the following faulty thinking habits.

- *Black and white thinking*
  Bill sees an event either as a success or a total failure. He evaluates

everything in extremes. Take, for example, his thoughts about this week's tennis match: 'I've got to be perfect this week, I was so lousy last Sunday'.

- *Generalising*
  Bill needs only one example of behaviour to make a general rule for all times and all places. He forgets that behaviour is very much determined by a specific situation, such as the other person's mood. For example, Bill's tennis partner, Ted, is not very talkative today. (Bill doesn't know that Ted's dog died last night.) Bill thinks, 'Ted doesn't like playing tennis with me—nobody has ever really enjoyed my company'.
- *Getting things out of proportion*
  Bill focuses on an event that would be unpleasant (for anyone), but builds the situation up to an extreme. For example, Bill has made a mistake at work. He thinks, 'How incompetent of me. That's blown my chances with the boss. No one else would have made such a stupid mistake. I'm hopeless'. He also treats criticism as total rejection.
- *Personalising a situation*
  If someone is angry or upset, Bill thinks it is his fault or his responsibility. He feels that things are happening this way because of him. For example, tennis has been rained out. Ted was looking forward to the exercise, and now he is grumpy. Bill thinks, 'Ted is mad at me because this is the second Sunday that I've booked for tennis and it's rained. I'm embarrassed—it's rained two Sundays in a row!'
- *Setting unrealistic expectations*
  Bill believes that it is essential to be perfect and in control at all times. For example, he finds taking his two-year-old, Sam, to a restaurant an irritating experience. He loses his temper with Sam because he is messy and noisy. Bill thinks, 'I should be able to control Sam better. I'm a hopeless parent, always yelling at him'.

- *Arbitrary inference*
  Bill often draws conclusions or inferences from situations where there is no evidence to support his conclusions. He then uses these inferences to put himself down. For example, 'Everyone else looks happy all the time. I should be happy all the time. I'm a failure if I feel unhappy'.
- *Selective abstraction*
  Bill is sensitive, always on the lookout for signs of rejection or criticism. He dwells on things that others have said or done and interprets them as critical of himself. 'Ted rushed off straight after tennis today. I think he finds me boring. He didn't want to stop for coffee.' (Ted was, in fact, under orders to be home on time after tennis to put on the barbecue for guests.)

Learning rational thinking

Rational thinking is realistic thinking, not simply positive thinking. Sometimes, positive thoughts can be irrational and leave a person feeling just as down. For example, 'This time I *will* succeed. It'll be different from every other time'.

Such a self-instruction is irrational because it sets up unrealistic expectations that will only lead to feelings of failure and despair if things don't work out. Of course, we should all allow for faults, mistakes, bad moods and unpleasant feelings over the course of an ordinary day. The trick is not to let such feelings overcome us.

The way we talk to ourselves influences the way we feel and behave. For example, 'I feel upset about what just happened, but I can't do anything about it. I'll distract myself by keeping busy. I won't let myself dwell on it' instead of, 'Oh no! How can I face the rest of the day after this! I'm too upset to work. Today is a disaster'. People who continually tell themselves 'I can't cope' will end up believing it. This will prevent them from learning new ways of coping.

Here are some of the common irrational beliefs that can have a very negative effect on thought patterns.

- It is essential that people who are important to me should like/love/approve of me all the time. If they don't, it must mean that I'm bad/worthless.
- I must be good at what I do and always try to improve myself.
- Some people are bad/wicked/evil and I should be very upset by their behaviour.
- It is a total disaster if things don't work out the way I want.
- Fate/destiny controls us. We have little to do with causing our own sorrows or unhappiness.
- If something is, or may be, dangerous or frightening, I must be concerned about it and worry about it happening to me.
- If things are too hard it's better to avoid them than fail.
- We must have others to rely on. We all need someone stronger than ourselves in order to cope with life.
- What happened in my past will always affect me—both now and in the future.
- I should be very upset and dwell on other people's problems and crises if I really care for them.

Changing the way we think, by being aware of our thoughts, involves three stages:

- *Anticipation*—before the event.
- *Reaction*—during the event.
- *Analysis*—after the event.

How someone looks forward to an activity is very important in laying the groundwork for their emotional response. For example, if an invitation to a party brings an emotional response such as,

'I won't know anyone there. I'll look so stupid', then negative thinking has to be stopped before it starts. It is completely self-defeating. More self-encouraging statements would be: 'I might find the party a bit of a strain but I'll get myself a drink and stand in the kitchen. I might be able to offer some food around and get to talk to people that way'.

Self-talk in any situation makes a difference to the way we cope. Negative thoughts after an event make it harder to face that activity in future. Develop the habit of thinking logically rather than emotionally. For example: 'I'm shy at parties, but they are an important part of meeting people. What can I do to cope better? Which bits worry me most?'

Narrative therapy

Narrative therapy is about listening to how people describe their problems as 'stories'. By listening and attempting to understand these stories, the therapist helps the patient consider how the stories may restrict them from overcoming their present difficulties. The focus of therapy is on highlighting the significance and resolution of certain life events in terms of their impact on the future life course of the individual.

Therapists using narrative therapy are instructed to listen actively to the stories without reading in or interpreting undesirable meanings, a technique known as 'deconstructive listening'. This is usually accomplished by clarifying the meanings of stories and asking 'What if . . . ?' or 'Could it be that . . . ?' questions. Patients may be encouraged to write down their thoughts about the events they relate and what they learned from those experiences. The therapist may also use a series of probing questions to encourage the patient to look critically at how their behaviour may be adversely affecting the way they are perceived by others and by themselves, and how these behaviours may ultimately stop them from achieving their goals in life.

Component techniques therefore include:

- 'deconstructive questioning' or getting patients to re-examine their stories from a different perspective and realise how their stories may influence their thoughts about their future;
- listening and asking about alternative stories;
- helping patients develop new stories;
- questioning patients on how they arrived at a particular conclusion;
- asking for more detail in order to get patients to analyse the logic embedded within their stories; and
- asking for the meaning of particular points in the stories.

Thought-stopping

Thought-stopping is a useful technique to interrupt negative thinking.

- Say 'STOP! STOP!' to yourself, as loudly as you can, and simultaneously imagine a stop sign.
- Immediately distract yourself by concentrating on regulating your breathing, relaxing, thinking of something positive or imagining something pleasant.

Cognitive therapies—are they specifically effective?

When depressed individuals consult a professional their depression may improve because their therapists activate a remission as a consequence of their interest in and understanding of their patient—so that improvement emerges from 'non-specific' factors, or the personal qualities of the therapist. If, however, improvement relates to a specific therapeutic component (e.g. prescribing the right antidepressant drug, providing appropriate counselling), we can conclude that the component itself is the therapeutic agent.

In the past, most psychotherapies have been criticised for bringing about improvement only as a consequence of non-specific therapeutic components—in other words, the patient's improvement relates more to the therapist's interpersonal skills than to therapy itself. How do cognitive behaviour therapy (CBT) and other cognitive approaches stand up to this criticism?

CBT has been a manualised therapy (i.e. prescribed via manuals) for more than two decades. Because it is based on a seemingly logical theory, has a reasoned treatment model and extensive scientific evaluation, it is held to be superior to all other psychotherapies as a strategy for treating depression. It is the mainstay approach for many mental health professionals who manage depression, and general practitioners are being increasingly encouraged to use it. In 2003, our research team at the Black Dog Institute published a review of CBT in the *American Journal of Psychiatry* (Parker, Roy & Eyers 2003). We suggested that the very large database of randomised controlled studies (where CBT was compared with other therapies or placebos) did not provide strong support for it having a 'specific' therapeutic effect.

Why could we not find strong support for its specific benefits? Two reasons suggest themselves. First, the current nature of randomised controlled studies—where treatments are tested for their 'universal' effect (i.e. as if having application across all types and expressions of depression—an uncommon model in medicine). Second, CBT was tested in relation to the non-specific diagnosis of 'major depression', not against specific depressive subtypes. We suggested earlier that a 'horses for courses' model—for both the depressive disorders and their treatments—should be used to test all interventions. This model suggests that individual treatments (CBT, other psychotherapies, antidepressant drugs) will be specifically effective for certain subtypes of depressive disorders—as against having universal application. We need further studies to define the effectiveness of each separate treatment for the

various depressive conditions. As yet, those who might be likely to benefit specifically from CBT have not been identified.

If we had to speculate on the probable uses of CBT, we would suggest that it is likely to be of benefit for subsets of those with non-melancholic disorders, but of no specific benefit to people with melancholic or psychotic depression. Those who appear to benefit from it most are people with generally good coping skills (with self-esteem issues that make them vulnerable to depression only in certain circumstances), who are highly responsible, and who find CBT theory intellectually appealing and sensible, and who relate well to the therapist and practise their assigned tasks.

We don't suggest that CBT is necessarily better than any other psychotherapy or approach to managing depression. Each method may have specific benefits in certain circumstances, but defining these circumstances is not easy. It is, however, one of our Institute's current research priorities. Regrettably, the prevailing models for defining the depressive disorders (eg 'major depression') disallow such information to be obtained from the published literature.

There are many versions of CBT, and practitioners range considerably in their competence and in the extent to which they provide a detailed CBT program or merely components of it. If you wish to receive CBT, try to find a professional who is skilled in the therapy.

Author note: Much of the material in this chapter has been adapted from a treatment manual prepared by our clinical psychologist colleagues Susan Tanner and Jillian Ball. For futher details and practical exercises, see Tanner and Ball (1999). Additional material has been provided by our Institute psychologist, Vijaya Manicavasagar.

chapter 17

INTERPERSONAL THERAPY

Like cognitive behaviour therapy, interpersonal therapy (IPT) is of greater relevance to the non-melancholic disorders. Formally, however, it makes no assumption about the origin of the depression, and uses the connection between the onset of depressive symptoms and current interpersonal problems as a treatment focus. The underlying assumption is that depressive symptoms and interpersonal problems are interrelated.

Clinical depression is seen as having three components.

1. *Symptom formation*, that is, the development of depressive **affect** based on psychological and biological origins;
2. *Social functioning*, that is, where social interactions are derived from childhood experience, current social reinforcement and/or personal attempts at competence and mastery of social and interpersonal situations; and
3. *Personality*, including the handling of guilt and anger. These enduring traits constitute a person's unique pattern of reactions and functioning; some of them can contribute to depressive symptomatology.

IPT deals with current rather than past interpersonal relationships and focuses on the patient's immediate social context. It attempts to intervene in symptom formation and in the social dysfunction associated with depression, rather than changing enduring aspects of personality.

The onset of any depression occurs in a social or interpersonal context, which may cause and maintain the depression, particularly the non-melancholic disorders. IPT works on the premise that understanding the context can help the individual to identify depression, master it, deal with it and prevent it recurring.

The goals in IPT are:

- to demystify depression (the causes and triggers of depression can be identified, and depression can be mastered);
- to provide strategies for dealing with depression by making a 'diagnosis' about some of the causes, by educating the individual to establish links between symptoms and feelings, by exploring the interpersonal context, and by identifying the problem areas and making them explicit; and
- to improve the individual's interpersonal functioning.

Once the problem area has been identified, the therapist can give the patient a framework explaining where therapy is going and what might be achieved. IPT doesn't aim to change personality, but it helps to deal with the current stressful situation.

The four broad areas where IPT can be helpful are:

- *unresolved grief* (either fresh or from the past): for example, difficulty in grieving over the death of a parent because of unresolved anger;
- *disputes* (which could be marital, with children, or at work);
- *transitions*: for example, when a child leaves home, when there is a change of job, separation, divorce, or on retirement; and

- *interpersonal shortfalls*: for example, if a person lacks assertiveness, is feeling lonely or bored, or has difficulty initiating or sustaining relationships.

Techniques

While the techniques of IPT are generic to psychotherapy (i.e. to listen, empathise, deal with a person's feelings and help them to talk freely about their worries and concerns), the therapy occurs in three main phases.

First, the therapist makes a diagnostic evaluation of the patient after obtaining a psychiatric history. Then, a review is made of the patient's current social functioning and close relationship patterns, as well as the mutual expectations and changes involved in these relationships prior to the onset of depression. This review provides a framework and defines the focus of treatment.

In the second phase, the therapist explores with the patient the identified interpersonal problem area, and a contract for treatment is discussed. The contract should outline the probable number of sessions the patient will need and the therapeutic goals, as well as touch on what each party might expect of the other.

The final phase of therapy encourages the patient to recognise and consolidate what has been learnt, and develop ways of identifying and countering depressive symptoms in the future. A standard course usually involves 12–16 sessions.

Session therapy outline

1. An explanation of IPT and an assessment of the patient's background and details. The therapist will elicit information using an interpersonal inventory.

2. The contract—the therapist makes explicit what the therapy hopes to achieve and the length of time the therapy is expected to last.
3. An exploration of past and present relationships in order to explore the facets of the patient's personality that led to the current difficulties.
4. A closer exploration of interpersonal incidents with questions such as: How did this situation occur? Emphasis is placed on what this exploration reveals to the patient about himself/herself.
5. A discussion of other possible behaviours in response to the situation.
6. An exploration of important incidents in and around the current situation and of what the patient's legitimate expectations might be.
7. An exploration of decision-making; working out future directions and expectations for the future.
8. Assignment of 'homework' that will help to clarify the problem.
9. Feedback to the therapist about the outcome of the homework.
10. Closure. A discussion with the patient about what has been learnt.

As for CBT, we would argue that IPT is likely to have differential benefits across the differing depressive subtypes and patterns. Again, the published literature disallows clarification as to what expressions of depression are most likely to benefit.

chapter 18

PSYCHOTHERAPIES AND COUNSELLING

There are many kinds of psychotherapy and counselling, all with varying emphases and approaches. Each may be useful in specific situations.

Psychotherapy

By definition, psychotherapy comprises a working relationship between a trained therapist and a patient. Psychotherapy emerged from psychoanalytic techniques that included encouraging patients to 'free associate'. The therapist would then progressively clarify and interpret links between the past and the present.

Brief psychodynamic psychotherapy seeks to overcome the intensive and extended nature of psychoanalysis. While this form of psychotherapy might involve exploring issues such as 'transference' (feelings about significant others in the past being projected on to the therapist), it seeks to focus on a particular problem, such as depression. Goals might be to explore how an individual developed depression (perhaps, by pursuing aspects of current relationships) or developed a propensity to depression (by considering aspects of

the patient's background such as low self-worth as a consequence of harsh and punitive parenting or childhood abuse). The direct aim of brief psychotherapy is to assist the development of insight. However, the non-judgmental support offered by psychotherapy can also be of distinct help to many depressed individuals. When this is the principal aim, the approach may be called *supportive psychotherapy*.

There are limitations to the brief psychotherapies. Treatment may, in fact, not be brief, but often extend for months and sometimes years. There may be a lack of structure, so that even after months in therapy the patient may be quite unclear about the goals and objectives. Dependency on the therapist can also develop. While this may be appropriate on a short-term basis, if it is not resolved or 'worked through' the patient's intrinsic capacity to deal (or not deal) with situations and thus build up a level of resilience may be taken away. The capacity for therapy to drift when applied by less skilled therapists has led to the development of quite structured psychotherapies, with cognitive behavioural therapy and interpersonal therapy being perhaps the best examples (see Chapters 16 and 17).

Effective therapists tend to be empathic (good listeners) and non-judgmental (trained to listen without being critical), and do not dictate what people should do. Their attitude, which is often one of unconditional positive regard for the patient, should encourage patients to 'ventilate' (talk about things that are concerning and preoccupying them).

Therapists may use the trusting and accepting relationship that should grow between them and the patient (the therapeutic relationship) as a microcosm (a smaller, safer version of the outside world) to understand how the patient acts in everyday life. Therapy may be structured to a particular pattern or follow the priorities set by either party.

As there are so many types of psychotherapy, effectiveness

depends on the skill of the therapist and the readiness and ability of the patient to embark on such a venture. Psychotherapy may be particularly helpful for those with non-melancholic depression, but it is not a first-line treatment for melancholic or psychotic depression, or for bipolar disorder.

Counselling

There are many techniques and applications for counselling. These are generally most useful when patients are either stressed or in crisis, as it is at this time that they are most amenable to changing set behaviours.

Much counselling focuses on problem-solving. The counsellor may, therefore, be of particular help in listening to a wide range of issues, clarifying and ranking key problems, identifying those that may require or benefit from action, encouraging the individual to act and then considering the results of such actions. Marital counselling and career advice are examples of the type of problem-solving a counsellor may undertake. Career advice identifies an important aspect of counselling—the provision of practical well-informed help.

Crisis counselling may involve problem-solving but will also seek ways to reduce an individual's stress levels. This can involve strategies to relieve any potential or actual post-traumatic stress reaction. While self-help is encouraged, the counsellor may also provide practical help.

Counsellors may range from those who are very directive to those who encourage patients to determine their own options. Such contrasting styles have their own predictable advantages and disadvantages. Whatever approach is taken, a counsellor should interact in a way that inspires trust and confidence.

Psychotherapy and counselling can, for a proportion of those with depression, be the principal therapies. For certain depressive

conditions, especially the more biological ones, they are better viewed as adjunctive treatments. This is not to minimise their impact or their potential benefits, but to suggest—as for all other treatments for depression—that we have yet to define the expressions and types of depression for which psychotherapy and counselling are most appropriate as principal therapies.

chapter 19

ANGER MANAGEMENT

In writing about her depression, the broadcaster Helen Razer (1999) introduced the concept of Van Pelt's Disorder. Named after Lucy Van Pelt in the Peanuts cartoon strip, this disorder invokes an image of someone 'snatching their paltry little Linus blankets and tripping over them relentlessly and kicking them in their adipose Charlie Brown arses' (p. 69).

Anger is clearly a powerful and complex emotion. It can be tangled up with depression and is difficult to deal with effectively. Anger can arise from situations or life experiences that have caused disappointment, hurt or fright. It may also occur in response to violation of a person's physical or psychological 'territory'. Individual physiological and psychological differences mean that some people get angrier than others, and some find it harder to control their temper than others. Those who are 'placid' by temperament have less of a problem, but the rest of us have to deal with uncomfortable levels of anger from time to time.

Most people find anger difficult to manage, but we can learn to use the energy that anger provides in constructive ways, rather than 'bottling it up', 'stewing', turning the anger inwards (acting in),

being hostile, volatile or irritable, or 'exploding' (acting out).

There are multiple strategies for handling anger but here is a useful process.

1. Recognise that you are angry.
2. Identify what made you angry.
3. Establish what you can do about these feelings of anger.
4. Release your anger in more constructive ways.

Recognise that you are angry

Many people don't recognise anger. Their feelings come out as 'hurt' or 'fear'. They feel powerless, belittled or humiliated and don't want to dwell on these feelings. Others ruminate on the experiences that have made them angry and nourish grudges. Some become sour, vindictive or depressed.

It is common to fear anger's destructive force. In many families, therefore, displays of anger are not tolerated: they may be stifled in the children, and repressed in parents. This can happen regardless of whether or not the anger is justified, leading to an added level of confusion and discomfort for those who are feeling angry. There is generally a taboo on expressing anger, or even feeling it.

We should consider our own behaviour. Are we being deliberately provocative or irritable? Perhaps passive-aggressive? It may be that the person who is making us angry reminds us of the less attractive parts of our own character. If we recognise *why* we are angry, this can help us to control the anger.

Don't shy away from anger—acknowledge it.

Identify what made you angry

Anger is a signal that all is not well. We should give shape to our feelings; to do so judiciously is neither bad nor childish, but

mature. We should identify the situations that make us particularly angry. Are we being overlooked or taken for granted? Have we had to compromise something we believe in? Has someone taken or violated something precious to us or to someone we value?

Once we have cooled off a little, we should ask whether our anger is 'reasonable'. Is it in proportion to the situation that caused it? An intense reaction probably means that the immediate cause of the anger has triggered off energy from older, unresolved hurts and fears—perhaps from as far back as childhood.

Establish what you can do about these feelings of anger

We can use our understanding of what gave rise to our anger to learn about ourselves. There are certain actions we can take that will ameliorate our feelings somewhat. Try to work out what these are and ask a (disinterested) friend what they think. Sometimes, past hurts, injustices or indignities can be resolved with those responsible for them. If this is not possible, a skilled therapist can help heal the bruises. Sometimes we can forgive, or at least move on. Situations or conflicts that are unresolvable should be avoided. It is difficult to change others' attitudes, but we can improve our own. Each of us has control over ourselves.

Release your anger in more constructive ways

Expressing anger can be tricky, which is why it is better to be at some distance from the raw emotion. So, don't 'see red', don't 'let it all out'; instead 'cool it' and get back to the rational self. Go for a brisk walk, beat up a cushion, or scream long and loud while face down in a pillow. Alternatively, write a 'bottom drawer' letter

expressing exactly what you would say if the situation occurred all over again. (*Just don't post it!*) Read it the next day, laugh at it and work out an alternative course of action for the next time. When you have expressed yourself, you will be calmer and make better decisions about what to do.

This doesn't mean, however, that we should not express our anger, just that it is often wise not to do so in the heat of the moment. We are often inarticulate when we feel indignant, so attempting to communicate while very angry will probably be counterproductive.

Communication with the person who caused the feelings of anger will only help if we can express clearly what has angered us, and what we see as the solution to the problem. Try to be direct: sarcasm, scorn, casting blame or character analysis will only make a messy situation messier. We may even be able to listen to the other person's point of view. We are not responsible for their feelings, so such approaches should not be threatening.

There may be a compromise that suits all parties, and this is far better than maintaining a 'cold war' of silences and passive aggression, which is wearing for everyone involved, solves nothing and can turn us into victims. Things don't change overnight, however, and we sometimes have to state our point of view firmly and objectively, more than once, and 'stick to our guns'.

Sometimes, we are unable to state our anger because the person or situation that provoked it is out of reach. This may be because of a professional relationship (boss/employee), illness, a closeness where frankness may be a risk, or where intimacy is not desired. In each case there are solutions, although it may take a chat with a friend or therapist to find them. However, such chats should never be used simply to reiterate your original anger.

Some solutions take a lot of courage. For example, if you are continually angry with your boss, consider that you have genuine cause for such feelings and that it is not just unrealistic expectations on your part, then the only solution may be to change jobs.

Finally, what about that unexpressed slow-burning anger, resentment? Resentment may arise from a feeling of being exploited. For example, in today's busy marriages, a partner can feel worn out from time to time, and resent that the other seems not to be pulling their weight. While this may not necessarily be the case, in such a situation both partners need to be direct with one another and seek creative solutions to the problem so that each gets a little spare time. Go somewhere quiet and try to nut out a solution. If the situation cannot be changed, strategies will have to be developed so that resentment doesn't rule you.

In earlier chapters, we noted that a significant percentage of those with non-melancholic depression feel more irritable and angry with others when depressed, as quantified on our measure's 'irritability' and 'self-focused' personality dimensions. These are, in fact, the most common expressions of non-melancholic depression in young people. Although they overlap, irritability and anger often have different origins.

Irritability is best viewed as the externalised expression of anxiety. It is thus likely to be episodic, and expressed when a person is stressed or depressed, often to their later embarrassment. ('I'm really embarrassed at how I've been behaving recently—biting my partner's head off and criticising the children, when they haven't really done anything wrong.') Thus, the expression of irritability during a non-melancholic depressive episode favours anxiety management rather than an anger-management program.

By contrast, those who get angry tend to have a more ongoing volatile personality style, and are less tolerant of and more frustrated by stressors. They reduce stress by blaming others rather than themselves and are less likely to be embarrassed at involving others in their 'hissy fits'. When expressed directly, anger may settle the individual—but the collateral damage to others may be irreversible. If the person is motivated, an anger-management program is generally one of the best initial strategies.

chapter 20

MATCHING THE TREATMENT TO THE DEPRESSION

> *I cannot imagine leading a normal life without both taking lithium and having had the benefits of psychotherapy . . . lithium . . . diminishes my depressions . . . gentles me out . . . But, ineffably, psychotherapy heals.*
>
> Kay Jamison, *An Unquiet Mind*

As noted in the Introduction, depression is commonly viewed as an 'it', as if there is just one condition that varies dimensionally—whether by severity, persistence or recurrence. Defining depression in such a way (with official systems listing 'major' and 'minor' depression as if such distinctions are illuminating) has consolidated the 'it' model. Acceptance of this non-specific model has had three main results.

The first is that patients are likely to receive a treatment favoured by their practitioner rather than a therapy tailored to their condition on a demonstrated and rational basis. Second, as a consequence there is a 'one size fits all' model for viewing treatment for depressive disorders. Evaluative studies have shown a similar response rate for non-melancholic depression across most therapies tested (whether antidepressant drugs, cognitive behaviour therapy or counselling), and so the non-specific model has been allowed to proliferate unchallenged.

As psychiatrist John Ellard (personal correspondence, 2002) has observed, classifying depression by severity and not by cause, and

then randomly assigning people to a treatment, is about as rational as randomly allocating people in pain 'to spend a month in a pain program or to have an appendectomy'.

A third result of the non-specific model is that treatment tends to be prioritised along disciplinary lines. Doctors may view depression as a disease and therefore treat patients with only antidepressant drugs. Clinical psychologists, on the other hand, may see cognitive behaviour therapy as the only appropriate treatment for 'it'.

We have argued that there are multiple expressions of depression. In a sense, depression is, at the first level, a non-specific signal like pain—but one requiring analysis and interpretation. The diagnosis of pain is not as important as diagnosing the cause of the pain, for then treatment becomes more rational and predictable. To prescribe an analgesic for pain may be helpful (and, at times, sufficient), but it may also be less important than determining what is causing the pain and addressing that cause.

Similarly, it is important to distinguish the three principal classes of depression—psychotic, melancholic and non-melancholic. Such classifications reject the non-specific model, with psychotic and melancholic depression responding to quite different treatment approaches.

Recommendations for managing non-melancholic disorders are less clear; it is not a pure class and, as noted, published studies suggest similar levels of success across contrasting treatment approaches. This unsatisfactory finding reflects current limitations in the way that multiple expressions of these conditions are grouped into pseudo-entities or 'things', such as 'major depression' and 'dysthymia', rather than acknowledging that non-melancholic disorders are probably better seen as a reflection of the painful impact between life stresses and a person's personality or temperament.

The management of non-melancholic depression might be

advanced by developing a specific model that identifies and favours certain treatments above others, depending on the mix of any contributing personality dimensions. Such a model might, for instance, suggest antidepressant medication as being more beneficial for certain temperament styles (say, anxious worrying) than cognitive behaviour therapy, and deliver the converse finding for those with a self-critical temperament style. Thus, there is a need to develop a 'horses for courses' model, as occurs in much of medicine, and our Institute research pursues the development of such a template.

However, until depressive disorders are conceptualised with some specificity, such a model cannot be developed beyond the broad suggestions put in this book. There are reasonably firm data for specific benefits of physical treatments for psychotic and melancholic depression—and as primary treatments. For the non-melancholic depressive disorders, much research work needs to be undertaken to determine the best treatments for the differing patterns and conditions.

A template identifying the specific effectiveness of differing treatments for differing conditions would allow depressed individuals to receive substantiated treatment recommendations based on the type of their depression, and a probability estimate of their likely response to quite contrasting treatments. This is what patients expect for their general medical problems. It is regrettable that such a situation does not currently hold for the management of depression. While recognising current limitations, some provisional recommendations are now put for the principal depressive classes.

Non-melancholic clinical depression

For non-melancholic clinical depression, there is a significant chance (say 20–60 per cent) that the depressive episode will remit spontaneously. That outcome is increased if the individual receives:

- appropriate professional assessment;
- a comprehensive and clear formulation of the problems or likely causes from the assessing professional; and
- basic counselling strategies for handling the episode and its consequences.

This response rate will be lifted further (perhaps by an additional 10–30 per cent) with specific treatments for the depression, which could involve:

- a course of an antidepressant drug;
- psychotherapy; or
- a combination of both.

Our current research will allow clarification of the extent to which the eight personality styles described in Chapter 10 respond to differing or specific treatment approaches.

Currently, an SSRI drug would be the most common first-line antidepressant prescribed, both because of its efficacy rate and because it may modify some of the 'drivers' of the episode (e.g. anxiety, irritability, anxious worrying).

A course of psychotherapy might also have a direct antidepressant effect, either by providing the patient with strategies for addressing the causes of the episode and/or through the support of the therapist. Psychotherapy can also address episode 'drivers', such as by using a CBT focus on faulty thinking patterns. The patient's personality style will influence the degree to which a treatment alliance is established or, if alliance is achieved, will activate the chance of disorder remission.

A combination of drug treatment and psychotherapy (either commencing together or, as is more common, with psychotherapy beginning after initial drug treatment) appears to offer better results than either treatment on its own.

If one SSRI fails because of unacceptable side effects, it may be appropriate to try another SSRI or a different antidepressant drug type. If two antidepressant drug types have been tried at an adequate dose and both have failed, it is best to have the situation reviewed by a depression specialist. This is preferable to the traditional approach of trialling numerous antidepressants one after the other or in combination.

A review with a depression specialist might involve an assessment of the patient's physical condition (e.g. medical problems, medication being taken), but would focus particularly on the identification and treatment of predisposing features (e.g. personality style) and depression maintenance factors, such as a dysfunctional marriage or problems with parents. The individual is more likely to benefit if the treatment approach addresses any predisposition to depression that arises from personality or temperament.

Melancholic depression

For melancholic depression, the chance of a spontaneous remission—with or without a professional assessment—is slight (perhaps 5–10 per cent). A physical treatment is almost always required—and rapidly. If the patient has had previous episodes and has always responded to a particular antidepressant medication then, subject to the acceptability of any side effects, the same antidepressant would probably be recommenced. If drug side effects have caused problems before, it would be wise to consider using an alternative antidepressant.

If the episode is the patient's first, it would be reasonable to try an SSRI initially. If an SSRI fails to be effective, a move to a dual-action or broader-action drug (e.g. an SNRI) would be the second option. (This would also be the first step with a patient who had had previous episodes that had failed to respond to an SSRI.)

At least 50 per cent of first-episode patients would be expected

to respond to such a treatment, showing some evidence of improvement (but not necessarily recovered) in the first one to three weeks.

If there is no evidence of any improvement with a dual-action antidepressant over three weeks, it may be wise to move to another drug class rather than accept the view that an antidepressant must be trialled for up to eight weeks. Other drug classes include a tricyclic or an irreversible MAOI (where higher effectiveness would be anticipated, but with certain worrying side effects and some management difficulties).

If there is no evidence of improvement after trialling two or three antidepressant drugs of differing classes, **augmentation** strategies (such as introducing lithium) may be of benefit. Sometimes, and increasingly, an atypical antipsychotic drug might be used as an adjunctive drug if the patient has failed to respond to several antidepressants—to boost the effectiveness of the antidepressant ('like jump leads to a battery'). If such a drug is going to work, its benefits will be evident in a few days and the dose of the antipsychotic should ideally then be reduced rapidly.

If a patient has had previous episodes and received antidepressants, it is vital for the practitioner to review the individual effectiveness of these drugs before deciding whether to follow the above strategy or design an alternative procedure.

ECT is highly effective. This treatment should be considered if a patient with melancholic depression has failed to experience any improvement after trialling several antidepressant drugs, or is at grave medical risk (e.g. from poor nutrition or dehydration), or highly suicidal and unable to be readily protected. Patients who have previously had ECT may request it instead of drug treatment.

In some cases where a patient has been maintained on an antidepressant drug over a period of time, the drug can become increasingly ineffective. American psychiatrists have termed this a 'poop-out' effect. It may reflect stressful life events overriding the

beneficial medication (which argues for an increased dose) or a true loss of effectiveness (suggesting an increased dose or a transfer to a new drug type, alone or in combination).

Non-physical treatments such as counselling or psychotherapy are best viewed as adjunctive. They may focus on physical treatment issues (e.g. drug adherence, side effects and the stigma of illness) or address issues in a patient's life that may be linked to the depression or quite independent of it. Non-physical treatments on their own are ineffective against melancholia.

If progress is less than satisfactory after two or three treatments have been tried, the patient should be assessed by a depression specialist.

Psychotic depression

For psychotic depression the spontaneous remission rate is negligible (less than 5 per cent). The two most effective treatments (with an effectiveness rate of about 80 per cent) are a combination of an antipsychotic drug and an antidepressant drug, or ECT (with bilateral ECT being slightly more effective than unilateral ECT). In most cases, it is advisable to start with drug treatment, but ECT could well be considered initially for patients who have previously responded well to it, or who are at grave medical risk or highly suicidal. For those with severe agitation, a sedative medication such as a benzodiazepine can be extremely helpful in settling the mental distress until the more definitive and specific treatments have started to work. Use of a sedative should be for brief or intermittent periods only, because of the risk of becoming dependent on these medications.

As for melancholic depression, non-physical treatments are important adjunctive strategies but, by themselves, are ineffective and inappropriate primary treatments.

If progress is unsatisfactory, assessment by a depression specialist should occur.

A note on counselling and psychotherapy

While counselling and psychotherapy have been recommended, their usefulness and importance depend to a great extent on the skills and interpersonal style of the practitioner. Their potential to help should not be minimised, however. While a wide range of strategies is available (not easily summarised here), a good practitioner will set goals, keep the patient informed about expectations and working hypotheses, and move comfortably from addressing problems in the past to trying to prevent such problems in the future. Practitioners with good interpersonal skills will make a patient feel understood, cared about and supported, but not overly controlled.

These matters of style are vitally important, yet it is difficult to define them accurately. Kay Jamison explains:

> The debt I owe my psychiatrist is beyond description . . . It was all the stupid, desperately optimistic, condescending things he didn't say that kept me alive . . . all the compassion and warmth I felt from him that could not have been said . . . and his granite belief that mine was a life worth saving . . . Most difficult to put into words, but in many ways the essence of everything. (1999: 118)

Treatment-resistant patients

At the Mood Disorders Unit at the Black Dog Institute we are commonly asked to assess patients with 'treatment resistance'. This term is used to describe the situation when a depressed patient has failed to show either an adequate response or a maintained response after receiving at least two quite different antidepressant

drugs or physical treatments and, thus, a definition essentially assuming that resistance has emerged from biological factors and that management is restricted to physical treatments. We view this model as narrow and unsatisfactory in failing to concede psychological and social factors.

Patients presenting to us with treatment resistance comprise:

- those with a non-melancholic depression whose treatment has been excessively biological, with numerous drugs being tried, but without enough attention being paid to issues that need to be worked through;
- those with melancholic depression who have been trialled on the newer narrow-action antidepressants only and who need a broader-action drug, augmenting strategy or drug-combination approach;
- those who do not have a primary depressive disorder but, instead, an anxiety condition, a medical problem, personality difficulties or substance dependence as their primary condition;
- those who have undiagnosed bipolar disorder or psychotic depression;
- those who have a primary organic problem (e.g. thyroid dysfunction); and
- those whose physiological configuration leads to them 'excreting' antidepressant drugs.

Situations such as these emphasise the importance of diagnosing depression accurately and specifying its subtype. By clarifying the true diagnosis and making treatment more rational, or by prescribing augmenting and combination treatments, most patients with treatment-resistant depression receive benefit.

chapter 21

LIVING WITH SOMEONE WITH DEPRESSION

> *People seem to be able to bear or tolerate depression as long as there is the belief that things will improve.*
>
> Kay Jamison, *Night Falls Fast*

Depression and hopelessness are infectious and can bring out the worst—and the best—in families. Families can slip into ways of behaving that are meant to protect the depressed person, but which, in fact, lead to overprotection or to feelings of resentment in other family members. Family members, carers, friends and the general practitioner can all become frustrated.

It is important to establish clear and effective communication within the family. This may require the whole family meeting with a professional for a few sessions. A depressive episode may provide an opportunity for family members to re-evaluate what is important in their relationships and to try to resolve 'unfinished business' such as grief, relationship difficulties, dormant regrets or guilt.

The first few weeks of treatment are often crucial. This is a time for patience, care and encouragement. The patient may not have wanted to see the psychiatrist in the first place. During the early stages of treatment, it is very common for the patient to want to give up—the drugs can throw up side effects without providing any obvious benefits. While patients may be told that their

condition *is* treatable, their depression often leads them to forget or dismiss this message. Family members should reinforce it.

It does not help to suggest that the depressed person should 'pull up their socks'. Unfortunately, this common response from family and friends can only be counterproductive, as it reinforces feelings of depression and guilt. If the depressed person is suicidal, it is important that risks be reduced and that protective (but not over-protective) support systems be set up.

Family members should never suggest that drug treatment be discontinued once a particular episode is over; that is up to the individual to discuss with the treating doctor.

It is important to realise that psychotherapy can have treatment-related effects. It can put all sorts of demands on the rest of the family. It is not uncommon for depressed people to 'bottle up' their feelings, especially anger. As therapy helps them unblock some of these old feelings, there can be emotional scenes. It may appear that the patient is attacking family members, but it is important that they do not take it too personally. If there is some validity to the complaints, family members should respond appropriately. If the real issues belong further back in childhood, let the patient blow off steam. The family should stick around to be understanding as the storm abates.

Counterattacking is not helpful. Family members who feel uneasy about what therapy is doing to the patient should ask to be included in a session with the patient and the therapist. In some cases, the spotlight may be on relationships and family members may find themselves being invited into therapy sessions anyway.

Partners may be asked to resurrect 'fun' in the depressed person's life. It is at this stage that families can talk openly about the effects of living with somebody suffering depression. This can be seen as the start of a relationship resuming a more equal pattern rather than maintaining the carer/patient roles that are common in the early days of depression.

It is important for all those involved in the management of depression to take steps to prevent any **recurrence**. Depressed people and their families need to recognise early warning signs and act quickly. Attention should also be paid to lifestyle changes, including diet, exercise, social life and interests.

If family members are concerned that the treatment plan is not addressing the patient's needs, they should ask their general practitioner for assistance or contact other community resources (see 'Resources' on pages 159–67, or our website: www.blackdoginstitute.org.au). It may be difficult to obtain appropriate help—but persist.

Suicide

Depressive disorders can be associated with the feeling that life isn't worth living any more. Predicting just who is at risk is difficult, but statistics can give a general indication of the higher-risk groups.

Psychotic depression and Bipolar Disorder I pose a serious risk, as do symptoms of severe anxiety and agitation, or any serious physical illness. Being single, divorced, widowed, unemployed, or having a substance abuse habit increases risk. A suicide attempt is more likely when people have improved from their worst stage, often as they have greater 'energy' to engage in harmful activities. Those who have already attempted suicide are more likely to try it again.

Some warning signs of suicide are:

- repeated expressions of hopelessness or desperation;
- out-of-character behaviours, such as recklessness in a normally careful person;
- an abrupt mood change to an uncharacteristic calmness (a suicide plan may offer relief and lift mood);
- giving away prized possessions to relatives and friends;

- making out a will or taking out insurance;
- making remarks about death, dying and suicide.

Families can help by removing potentially harmful items such as tablets and firearms, and by seeking treatment as a matter of urgency. Local mental health services have 24-hour freecall helplines to provide advice and assistance. Our Institute's website has emergency details for those in Australia (www.blackdog institute.org.au). Information on child and adolescent mental health services is available from local area health services. If in doubt, ring the nearest hospital or the emergency services.

Are there any prizes?

One of the things that makes psychiatry a rewarding profession is that, not only do most depressed patients get back to their 'old selves', but it is quite common for them to learn from the experience:

> Mysterious in its coming, mysterious in its going,
> the affliction runs its course, and one finds peace.
> (Styron 1992: 73)

William Styron also noted (p. 84) that being 'restored to the capacity for serenity and joy . . . may be indemnity enough for having endured the despair beyond despair'. Margo Orum (1999) indicated that 'in the end, I opted for building more meaning into my everyday life, by doing the things I love, so that now my everyday life is . . . precious . . . the pay-offs are worth it'. Penelope Rowe (1999) could say 'I can "thank" my illness for shaving off some prickly edges and giving me greater tolerance (although I wish there had been an easier way!)'. In fact, some people who have dealt with depression end up emotionally stronger than they ever

were before. Strategies developed to manage depression can help in the management of general life situations. This optimistic thought should be kept in mind. For some depressed people and their families, it can be a long and hard road to recovery. There is, however, a signpost at the beginning that is worth reading:

> For most people, depression is treatable. The end-point is recovery, not merely improvement.

APPENDIX

MOOD DISORDERS, THE ARTISTIC TEMPERAMENT AND WORLDLY SUCCESS

In her book *Touched with Fire*, Kay Jamison provides a lengthy list of writers, musicians and artists who have had either a depressive or bipolar disorder. They include Hans Christian Andersen, Charles Dickens, F. Scott Fitzgerald, Leo Tolstoy, Graham Greene, William Blake, Robert Burns, Lord Byron, Adam Lindsay Gordon, Emily Dickinson, Sylvia Plath, Irving Berlin, George Handel, Noel Coward, Cole Porter, Paul Gauguin, Michelangelo and Georgia O'Keefe.

While her focus is on the reasons for the link between creativity and mood disorders, Kay Jamison's list also speaks to the reality that people with mood disorders can achieve and succeed in the most competitive and demanding careers when not restricted or disabled by their condition. As depression becomes less stigmatised we can expect more and more 'successful' people, be they professionals, business people or politicians, to discuss and identify how they have dealt with their mood disorder.

As an academic psychologist and author of the most respected and informative text on manic-depressive illness, Kay Jamison's description of her own bipolar disorder in *An Unquiet Mind* serves as an inspiration.

GLOSSARY

adherence (as in treatment adherence) Following the instructions of the health professional in regard to medication, non-physical treatments or basic advice. Synonymous with 'compliance' but less likely to suggest coercion.

adjunctive treatment A treatment administered alongside another treatment. This may be for an effect independent of the original treatment but is more often designed to strengthen the effect of another treatment.

affect An individual's mood state as observed by others (e.g. 'His affect was one of sadness').

affective disorder The group of psychiatric disorders (including both manic and depressive disorders) where an affective or mood disturbance is primary. 'Primary' indicates that this is the first-ranking condition and contributes the greatest symptom distress.

anhedonia Lack of pleasure. A common symptom experienced by depressed people. *Anticipatory anhedonia* is the inability to look

forward to a normally pleasurable activity; *consummatory anhedonia* is a lack of pleasure after engaging in any such activity.

antimanic Medication supplied to prevent, or help stabilise, the highs of bipolar disorder.

antipsychotic Medication to prevent or ameliorate a psychotic episode.

atypical depression A possible subtype of depression where the symptom pattern is in contrast with the usual depression profile (e.g. appetite increase rather than loss, hypersomnia rather than insomnia) and where the individual is likely to have a predisposing personality style of 'interpersonal hypersensitivity' (i.e. expecting that others will not like or approve of them). Alternatively, it may be an expression of depression in those with high anxiety. Atypical depression was held to be more likely to respond positively to MAOI (monoamine oxidase inhibitor) antidepressant drugs, but is now known to respond equally well to SSRIs and to CBT.

augmentation Increasing the effectiveness of one medication by adding another.

basal ganglia Brain centres controlling and refining motor performance.

bipolar depression An episode of depression in an individual with bipolar disorder. It is almost invariably melancholic or psychotic in its expression.

bipolar disorder Episodes of mania (or hypomania) alone or with depressive episodes at other times.

Bipolar I disorder Episode(s) of mania, with or without a history of depressive episodes.

Bipolar II disorder Episodes of both hypomania and depression (but with no experience of mania).

catecholamines The neurotransmitters noradrenaline and dopamine are catecholamines and have been implicated—like the indoleamine serotonin—as underpinning depression. The original *catecholamine hypothesis* was that depression resulted from a deficit of noradrenalin in the central nervous system, and that antidepressants acted by increasing the levels of catecholamines at brain synapses.

cognition The process of thinking. Cognitions refer to thoughts, understanding and reasoning.

cognitive limitations The individual's capacity to think or reason is impaired for some reason (e.g. in severe depression or dementia).

cognitive style The characteristics of an individual's habits of thought, how they organise their thoughts, what intentions they attribute to others and how they communicate with them.

combination therapy The use of two or more therapeutic agents for two or more different symptoms (e.g. an antipsychotic drug plus an antidepressant drug for psychotic features and for depression in psychotic depression).

compliance adherence To the therapy suggested, whether by finishing a prescribed course of medication or by following advice.

continuation therapy Therapy (usually drug treatment) that is implemented after the acute phase of an episode has been managed. Designed to control the last stages of the current episode. Contrast with *maintenance therapy*, used in the next phase, where the objective is to prevent new episodes.

cyclothymia A temperament style where the individual has mood

swings that are part of their personality, ranging from very sociable and talkative to quiet and solitary (thus a 'cyclothymic personality'). In recent years, the term has been more used to define a subtype and milder expression of bipolar disorder.

delusional depression Where the depressed individual experiences delusions (or false beliefs) during a depressive episode, and/or hallucinations or false perception (whether of sight, sound or other senses such as taste and smell). It is termed *psychotic depression* in this book. Its status remains unclear, being either a subtype of melancholic depression or a separate subtype.

depressogenic A stressful life event that is likely to cause depression in a vulnerable individual (e.g. loss of job, serious family dispute).

distal stressors Stressful situations that occurred many years before the onset of depression but which have disposed the individual to depressive episodes.

diurnal variation A change in depressive mood and energy at certain times of the day. Classically, those with melancholic depression report improvements in mood and energy later in the day, those with non-melancholic depression describe the opposite, and those with psychotic depression report no diurnal variation—the depression remaining persistent and unrelieved throughout the day. These differences are, however, not entirely specific to the different depressive subtypes.

dopamine A catecholamine neurotransmitter.

double depression Episodes of acute and more severe depression (major depression) superimposed on chronic depression (dysthymia).

drug classes 'Families' of drugs; that is, drugs derived from different chemical backgrounds.

drug trial In the drug development and research context, a drug trial is a study to determine whether a drug is efficacious (i.e. it works in comparison to a placebo or another active drug) and tolerated by subjects (i.e. quantifying the side effects). In the clinical context, a trial of a drug determines whether it is effective for a particular patient.

DSM (Diagnostic and Statistical Manual) The official diagnostic classificatory system of the American Psychiatric Association. It classifies depressive disorders largely on a dimensional basis but with secondary assignment (for those who meet the criteria for major depression) to melancholic and psychotic depressive classes.

dysthymia A diagnosis introduced into recent North American DSMs to describe non-psychotic, non-melancholic depression that is of 'minor' severity but present for two years or more.

endogenous depression An old term for *melancholic depression*, reflecting the view that this depression was not related to stress but came more from within the individual. While those with melancholic depression are less likely to report previous life stressors (and their depression is usually out of proportion to any such stressor), the lack of specificity of 'stress' to depressive subtypes argues for rejecting this term.

episode A bout of depression. To qualify as clinical depression, the episode should be present for most of the day and for two weeks or more.

euthymia A normal mood state, neither depressed nor manic.

externalising personality A style of dealing with stress by reflecting its impact behaviourally (e.g. raising one's voice, driving recklessly).

high The abnormal upswing in mood that is characteristic of hypomania or mania.

hypersomnia Excessive sleeping (the opposite of insomnia). This may occur in melancholic, non-melancholic and atypical depression.

hypomania Highs that are less severe than manic episodes and without any psychotic features such as misinterpretation of events.

ICD–10 The current official International Classification of Diseases prepared by the World Health Organization. This system categorises depressive disorders principally on the basis of severity.

improvement When the clinical condition is somewhat better but not sufficiently improved to be regarded as remission or recovery.

internalising personality Dealing with stress by not discussing things openly. Individuals with this personality style become quiet, go to their room, cry quietly and exhibit other withdrawal behaviours.

maintenance therapy Drug therapies that are either in place, or are put into place, after a patient's episode has been brought under control. These therapies are designed to prevent the onset of a new episode.

major depression A DSM diagnosis describing an episode of depression, with five or more specific features (e.g. depressed mood, loss of interest, sleep disturbance) present for two weeks or more and associated with social impairment.

mania A high mood of distinct severity, where the individual is commonly psychotic (i.e. with delusions and/or hallucinations).

melancholia/melancholic depression The quintessential biological depressive subtype, it has been variously described as: likely to emerge without any antecedent stressor; having certain specific clinical features (such as observable PMD) and relatively specific features (e.g. non-reactive mood, anhedonia, mood worse in the

morning); having genetic determinants; providing strong evidence of biological determinants; unlikely to respond to placebo medication; and being highly likely to respond to physical treatments.

minor depression DSM category for disorders not severe enough to meet the criteria for major depression. Includes dysthymia as well as several brief depressive disorders.

mixed state A bipolar disorder with features of both mania and depression at the same time.

mood Personal description of how an individual feels (contrasting with *affect*, or how the individual appears), or a reference to a more persistent emotional state than an affect.

mood stabilisers Drugs that help to control the fluctuations of mood that typify bipolar disorder.

neuroleptic Drug used in the treatment of any disorder with psychotic phenomena.

neurotic depression A dated term used to describe a depressive subtype contrasted with endogenous depression. A disorder in individuals with personality styles (e.g. neurotic, highly anxious, shy and unassertive) that disposed them to a greater risk of depression when faced with a life event stressor.

neurotransmitters Chemical substances present in the brain that help to carry signals between nerve cells. They can become depleted, or otherwise out of balance, and so lead to mental disorders.

noradrenaline A catecholamine neurotransmitter.

pharmacotherapy Drug therapy.

physical treatment A treatment that has physical concomitants (e.g. drug or ECT treatment) as opposed to psychological interventions.

postnatal depression Any type of depression in the first 9–12 months following the birth of a baby.

post-partum blues A mild postnatal depression that usually occurs in the first few weeks after having a baby.

post-partum or **puerperal psychosis** Any psychotic condition occurring in the first few weeks after having a baby, and most commonly a depressive (or manic) disorder rather than a schizophrenic disorder.

prefrontal cortex Frontal lobe region of the brain. Of suggested relevance to depression because neural activity (as measured by blood flow or glucose use) is more likely to be reduced in this region of the brain when individuals are depressed. This finding, however, lacks specificity (neither establishing the presence of depression nor by distinguishing depression from other conditions).

primary and **secondary conditions** The word 'primary' (as in 'primary depression') indicates that this condition provides the first, major or largest input to the presenting problems. Secondary depression generally indicates that the depression follows, or is otherwise related to, another major medical condition, whether psychiatric (e.g. schizophrenia), medical (e.g. a stroke) or other (e.g. alcohol state).

proximal stressor A stressful situation occurring immediately before a depressive episode, possibly causing its onset.

psychomotor disturbance (PMD) Decreased movement (i.e. retardation) or increased or perturbed movement (i.e. agitation) observable in melancholic and psychotic depression, but absent in non-melancholic depression.

psychopharmacology The science of drug therapy for psychiatric disorders.

psychosis Impairment of mental functioning in which the individual loses touch with reality and usually experiences delusions and/or hallucinations.

psychotherapy A non-physical treatment in which the therapist adopts a particular structure (e.g. analytic, interpersonal, cognitive, cognitive-behavioural) to address symptoms and/or personality problems experienced by an individual. It has a number of non-specific components (e.g. empathy) that may be therapeutic in themselves.

psychotic depression Either a subclass of melancholic depression or a separate subtype altogether. Its clinical features involve those most commonly overrepresented in melancholic depression, including observable PMD, although these features are generally more severe, with the added presence of psychotic symptoms (i.e. delusions and/or hallucinations).

reactive depression An old term for a depressive episode that can be explained as a consequence of having experienced a major stress, and with the implication that its clinical pattern is not melancholic in type. Now less popular, as life event stress has been found to have minimal specificity to any depressive subtype.

recovery When a depressive episode has completely resolved for a defined period.

recurrence A new episode of depression, after an individual has been completely well from depression for a defined period.

relapse The return of a full episode of depression when the individual has not completely recovered from an earlier episode.

remission When an episode of depression (or mania) has resolved completely but the period has been so brief that it is not clear whether actual recovery has occurred.

schizo-affective disorder A clinical pattern combining some features of schizophrenia in conjunction with those of a mood disorder (either mania or depression), most commonly used when the exact diagnosis remains unclear.

schizophrenia A psychiatric condition where delusions and hallucinations are common; thinking, insight and attention are commonly impaired, and behavioural problems frequent. It is quite distinct from the mood disorders, although a significant percentage of those with schizophrenia develop a superimposed depression.

seasonal affective disorder (SAD) Depressive episodes that occur on a reasonably regular basis in certain seasons (especially autumn and winter) and remit in the alternate seasons (spring and summer). The clinical feature pattern may differ from classic depression (e.g. by hypersomnia or appetite increase). Phototherapy (bright-light therapy) may be effective. This condition is more common in the northern hemisphere, but it does exist in Australia.

secondary depression Depression that follows a physical condition (e.g. a stroke) or substance abuse; in some cases, it is a by-product of another psychiatric condition (such as schizophrenia), or coexists with it.

serotonin A neurotransmitter (an indoleamine rather than a catecholamine, otherwise known as 5-HT or 5-hydroxytryptamine); widely distributed in the body, as well as in the brain. The *serotonin hypothesis* views depression as reflecting a functional deficiency in the serotonin system; antidepressant-drug effects are produced by increasing 5-HT in the brain.

side effects Unintended effects of medication that can exist alongside the positive effects of the drug; more discernible in the older-style antidepressants. If side effects are too disruptive, medication dosage may have to be lowered or that medication

ceased. Side effects are usually most pronounced in the first few weeks of treatment, but settle down after that.

somatic To do with the body—for example, a somatic (physical) illness.

spontaneous remission An episode of depression (or mania) that seems to resolve completely without medical intervention.

stigma A sense of shame felt by people as they attempt to fulfil their accustomed roles in the face of discrimination practised by the wider community. There are programs aimed at destigmatising depression so that people can feel positive about seeking help.

stressor An event or interpersonal interaction that causes distress. Stressors can be acute (e.g. the immediate aftermath of an accident) or chronic (e.g. poverty, a poor marriage).

stupor A state of reduced consciousness.

unipolar depression Originally used to subclassify the behaviour pattern of those with melancholic depression who had experienced only depressive episodes over time (in contrast to those with bipolar depression). Now used to describe the longitudinal course of any non-bipolar condition.

vegetative features Symptoms (e.g. reduced appetite and weight loss) that are more somatic than psychological, and commonly reported by those with depressive disorders.

RESOURCES

Medication

http://www.RxList.com
RxList is an Internet drug index that provides non-technical information on more than 4500 drugs in common usage. It has a medical encyclopaedia, and bimonthly and annual MIMS (*www.mims.com.au*).

Australian websites of interest

Depression

http://www.blackdoginstitute.org.au
The website for the Mood Disorders Unit, Black Dog Institute; provides a range of further references not listed here, and rapid access to other key sites.

http://www.healthinsite.gov.au
A listing of Australian health sites that have been assessed as credible by the Department of Health and Aged Care.

http://www.beyondblue.org.au
The 'beyond blue' national depression initiative.

http://mentalhealth.anu.edu.auhttp://bluepages.anu.edu.au
BluePages is a depression website developed by the Centre for Mental Health Research (ANU, Canberra) and the CSIRO Division of Mathematical and Information Sciences.

http://moodgym.anu.edu.au
Developed by the Centre for Mental Health Research (ANU, Canberra), this website provides a free online cognitive behavioural therapy program, an Internet-based CBT intervention.

http://depressioNet.com.au
DepressioNet aims to be a one-stop depression information resource for Australians. The site hosts a message board and chat rooms. It includes information about new research and opportunities to contribute to this research.

Bipolar disorder

http://www.mentalhealth.asn.au/dmda
The website of the Depression and Mood Disorders Association of New South Wales. Its objective is to inform and help people with bipolar disorder, depression or mood disorders and their families and friends.

http://members.iinet.net.au/~fractal1/fhello.htm
Fyreniyce refers to itself as 'The Australian Bipolar Website', a site dedicated to those who suffer µfrom bipolar disorder, and who

experience its 'fire' and 'ice'. The goal is to learn about the disorder, develop constructive ways to deal with it and provide support for those affected.

http://www.blackdoginstitute.org.au
Our website provides key information on bipolar disorders and some information to assist people in obtaining professional help.

Anxiety disorders

http://ada.mentalhealth.asn.au
The website of the Anxiety Disorders Alliance, a New South Wales-based organisation providing support and services to people with obsessive compulsive disorder, social phobia, specific phobia, panic disorder, agoraphobia, generalised anxiety disorder and post-traumatic stress disorder. It provides information and resources and runs a number of support groups.

General

http://www.sane.org
Run by SANE, an Australian charity helping people affected by mental illness through lobbying, education and research. It offers an information and referral service.

http://www.mentalhealth.asn.au
The website of the Mental Health Association NSW has publications, maintains an extensive database of mental health services and provides links to support groups.

http://healthnetwork.com.au
The Health Network is an Australian and New Zealand resource centre. Its health information database is separated into life stages but also allows browsing by condition and treatment.

http://www.health.gov.au
The website of the Australian Department of Health and Aged Care has information on policy initiatives, current campaigns and publications.

http://www.healthinsite.gov.au
HealthInsite is a Commonwealth Government site; it provides links to Australian health sites that have been assessed as credible by the Department of Health and Aged Care. The site is searchable by condition.

http://www.health.nsw.gov.au/policy/chm
The website of the unit within the NSW Health Department that deals with mental health services delivery in New South Wales. A range of useful publications, divided into topics, is available through the website.

http://www.betterhealth.vic.gov.au
The Better Health Channel is a website established by the Victorian Government. It aims to provide reliable, accurate, quality-assured and locally relevant information. Searchable by topic.

http://www.justask.org.au
Just Ask is Lifeline's rural mental health information service. It provides a telephone counselling service and information.

http://auseinet.flinders.edu.au
Auseinet, the Australian Network for Promotion, Prevention and Early Intervention for Mental Health, is a Commonwealth Government funded organisation. Its website provides a searchable database of resources relating to mental health and wellbeing.

http://www.infrapsych.com
InfraPsych Australia is a company that provides online support to psychiatric patients, caregivers and professionals. Its website contains information on a range of mental health topics.

http://www.mmha.org.au
Multicultural Mental Health is a national body dealing with multicultural mental health and suicide prevention. Its website provides information and resources in a large number of community languages.

For young people

http://www.reachout.asn.au
Reach Out! is a website designed specially for young people, providing support, information, referrals and inspiration.

http://www.kidshelp.com.au
Kids Help Line is a website with information and support for young people aged 5–18 years. The organisation provides an Australia-wide telephone counselling service as well as online counselling.

New Zealand websites of interest

http://www.mentalhealthorg.nz/
The Mental Health Foundation of New Zealand is a non-profit organisation providing a comprehensive mental health information resource.

http://www.balance.org.nz/
The New Zealand Bipolar Network, providing support, education and advocacy to those affected by bipolar disorder.

South African website of interest

http://www.mentalhealthsa.co.za
The site of the Mental Health Information Centre, at the University of Stellenbosch, Cape Town, promoting public awareness and general education.

US websites of interest

Depression

http://www.mentalhealth.org
The Substance Abuse and Mental Health Services Administration for the US Department of Health and Human Services. Provides information about mental health via 600 publications, its website and a toll-free telephone number.

http://www.psycom.net/depression.central.html
Dr Ivan's Depression Central, as this site is called, operates as a clearing house for information about major depression, manic depression (bipolar disorder), cyclothymia, dysthymia and other mood disorders and their effective treatment.

http://www.human-nature.com/odmh/depression.html
The online Dictionary of Mental Health. It also provides links to other sites that enlarge on topics discussed.

http://www.sciam.com/1998/0698issue/0698nemeroff.html
This site for *Scientific American* carries a comprehensive article on the neurobiology of depression.

http://www.depression.org/
National Foundation for Depressive Illness (NFDI) provides information about depression, referral and support groups.

http://www.nami.org
National Alliance for the Mentally Ill (NAMI) provides support, information and referral.

http://www.mgh.harvard.edu or *http://healthcare.partners.org/depression/*
The Depression Clinical and Research Program at the Massachusetts General Hospital provides information on depression and how to become involved in research studies.

http://www.depressionafterdelivery.com
Depression After Delivery is a national not-for-profit organisation providing support for sufferers of ante-partum and post-partum disorders. It provides education, support, information and referral.

Bipolar disorder

http://www/med.jhu.edu/drada/creativity.html
DRADA, Depression and Related Affective Disorders Association, looks at creativity, depression and manic depression, and provides studies of writers and artists.

http://www.mgh.harvard.edu or *http://www.massgeneral.org/allpsych/bipolar/index.html*
The Harvard Bipolar Program offers information on living with bipolar disorder, care providers and getting involved in research.

http://www.ndmda.org
The aim of the National Depressive and Manic Depressive Association is to inform the community that all depressive and manic-depressive illnesses are treatable medical diseases, as well as to foster self-help, improve access to care, and advocate for research to eliminate these illnesses.

General

http://www.nimh.nih.gov
The National Institute of Mental Health is a component within the US Government's National Institutes of Health. It conducts and supports research into mental disorders, supports the training of scientists and provides information to scientists, the public, service providers and the media about mental disorders. Its website has information on mental disorders, news of clinical trials, statistics and research fact sheets.

http://healthfinder.gov
Healthfinder is a service of the US Department of Health and Human Services, aiming to provide reliable health information.

http://mayohealth.org
This website, connected with the Mayo Clinic, has information about general diseases and advice on taking charge of your own health, plus news items of health interest and answers to questions put to specialists.

http://www.onhealth.com
This website describes itself as a 'new way to look at everything' and covers everything from diseases to alternative health and lifestyle advice for women, family and babies.

http://www.nmha.org
National Mental Health Association (NMHA) addresses all aspects of mental health and mental illness through advocacy, education, research and service.

http://www.mentalhelp.net
Mental Help Net is a privately established website providing free information to the worldwide mental health community. It has resources and information on a wide range of mental health topics.

http://www.aagpgpa.org
The American Association of Geriatric Psychiatry is a national organisation with the objective of enhancing knowledge and practice standards in geriatric psychiatry through education and research. Its website has information and resources for adults coping with mental health issues and ageing.

http://www.mentalhealthcom
Established by a Canadian psychiatrist, offering information on mental disorders and medications.

http://www.bu.edu/cpr
An interesting research, training and service organisation with a rehabilitative focus.

Canadian websites of interest

http://www.fhs.mcmaster.ca/direct/
Depression and Anxiety Information Resource and Education.

http://www.depressioncanada.com
Provides information on symptoms of depression, treatment and medications.

http://www.mentalhealth.com
This site aims to improve understanding, diagnosis and treatment of mental illness, and offers information about effective, well-researched treatments, including bipolar disorder.

UK websites of interest

http://www.psychiatry.ox.ac.uk/cebmh/guidelines/depression/depression1.html
A systematic guide for the management of depression in primary care from the Centre for Evidence-Based Mental Health in Oxford.

http://www.nhsdirect.nhs.uk/conditions/search_display.jhtml?id=145
This National Health Service facility has information on depression and support organisations.

http://www.depressionalliance.org
Useful information about different types of depression, medical treatment and self-help.

http://www.mhrc.cc
The Mental Health Resource Centre for England is a mental health and social care website search directory. Allows searching by National Health Service (NHS) region and identifies NHS Trusts, Strategic Health Authorities, Social Services, User & Carer Support Groups and Children & Adolescent Mental Health Services by region.

http://www.mind.org.uk
Mind is a leading mental health charity in England and Wales. It offers a telephone helpline and referrals to an established network of associations, conducts campaigns, and produces publications on a range of mental health topics.

http://www.scmh.org.uk
The Sainsbury Centre for Mental Health is a charity affiliated to the Institute of Psychiatry, Kings College, London. It aims to improve the quality of life for people with severe mental health problems by conducting research into services, developing services and training. Its website provides publications and information on service delivery issues.

Accessing other websites

There are numerous websites providing information on depression and mental health. They can be accessed via search engines, by

entering search terms such as 'depression' or 'mental health'; if required for a particular country, include the name (or abbreviation) of that country in the search term (e.g. uk depression). For more specific searches related to depression, include the topic of interest in the search term (e.g. uk depression treatment; uk depression medications; uk depression symptoms; uk depression self-help).

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