29

Depression

- REACTIVE or NEUROTIC depression and ENDOGENOUS or PSYCHOTIC depression differ in their symptoms. BIOLOGICAL SYMPTOMS are more frequent in psychotic depression, whereas CAUSAL INSIGHT expressed by the patient into the origin of the depressive attack is more common in neurotic depression.
- If dogs are exposed to unavoidable shocks they develop the syndrome of LEARNED HELPLESSNESS, which shows many of the features of human depression, including decreased cerebral noradrenaline levels, which must be seen as *caused* by the depression rather than *causing* the depression.
- Depressive cognitions are marked by a diminished SELF-ESTEEM and NEGATIVE thoughts about the world, coupled with GUILT that the self-image is not as it should be.
- Melanie Klein described the early stages of attachment in which the child develops hatred and then AMBIVALENCE towards loved objects which have temporarily frustrated it by separation. Permanent loss can result in INTROJECTION of self-hatred, and vulnerability to depression.
- Brown and Harris' model of depression emphasizes the causal role of serious RECENT PROVOKING EVENTS in causing depression, modulated by VULNERABILITY and PROTECTIVE FACTORS. Early loss of a parent is a SYMPTOM-MODIFICATION FACTOR, loss by death producing psychotic symptoms and loss by separation producing neurotic symptoms.

Depressive symptoms occur in most of the population at some time during their lives. The principal abnormalities are of MOOD or AFFECT, with pervasive feelings of SADNESS, accompanied by TEARFULNESS or WEEPING, and varying from mild despondency ('the blues'), to utter DESPAIR, with SUICIDE a real possibility. Thought processes are slowed, and inability to concentrate or make decisions often show early as loss of interest in hobbies or activities; intellectual slowing down is accompanied by a more general RETARDATION, with slowed motor actions and speech, which shows long pauses, absent spontaneous utterances, and mono-syllabic replies to questions. Those thoughts that do occur are self-concerned, inward-looking and described by patients as 'painful', and concerned only with problems and difficulties,

and are self-deprecatory and self-accusatory, the individual taking upon themself the burdens and the responsibilities of the world. The NEGATIVE SELF-IMAGE is often associated with HYPOCHONDRIASIS, an unrealistic fear of serious physical illness, which can be precipitated by the so-called BIOLOGICAL SYMPTOMS of depression, which can be seen as physiological retardation, with loss of appetite and weight, reduced libido, and constipation (often misinterpreted as a symptom of bowel cancer), and disturbed sleep, in particular showing early morning wakening. Mood often varies during the course of the day, DIURNAL VARIATION.

Depression is classified in several ways. MANIA is the opposite of depression with excessive elevation of mood, an overwhelming sense of well-being and ability to carry out any task (without actually completing any), pressure of ideas, and flights of fancy. In BIPOLAR DEPRESSION or MANIC-DEPRESSIVE PSYCHOSIS, mood swings from mania to depression and back again, over days or weeks, passing through relative normality *en route*. Bipolar depression is relatively rare, and is probably genetic with a specific biochemical defect. It will not be considered further here. The more usual UNIPOLAR DEPRESSION shows mood changes between normality and depression, without a manic phase. Unipolar depressives can be divided into REACTIVE and ENDOGENOUS, each with several synonyms. REACTIVE, EXOGENOUS or NEUROTIC DEPRESSION apparently arises from a specific event in a patient's life, a loss or a failure, whereas in ENDOGENOUS or PSYCHOTIC DEPRESSION the patient is unaware of a precipitating factor, and lacks insight into the aetiology, the illness apparently arising from within without cause. Biological symptoms are more common in endogenous depression, and diurnal variation differs, moods being worst in the morning and improving during the day in endogenous depression, whereas in reactive depression mood becomes worse towards evening. Whether these are two separate conditions, or are merely extremes on a continuum, is still controversial.

This chapter will consider learning theory, personal construct theory, and psychoanalytic models of depression, and will also consider a sociologically based model to broaden the perspective.

LEARNING THEORY MODEL OF DEPRESSION

When a dog is placed in a SHUTTLE BOX, a cage with a low barrier down the middle, and is given mild electric shocks through the floor of one side, preceded by a warning bell or light, then it rapidly learns to escape shock by jumping the barrier. If, previously, the animal has been given a series of UNAVOIDABLE SHOCKS, against which at first it struggles but then accepts passively, then in the shuttle box it does not try to avoid the shocks but instead sits meekly in one corner and

accepts them. Such animals also perform poorly in other learning conditions, and in many ways appear to be depressed, showing no spontaneous activity, impaired sleeping, etc. The animals suffer from LEARNED HELPLESSNESS, the unavoidable shocks having taught them that their actions have no effect upon the world, and hence all action is without effect and not worth making. The learning has generalized to the nature of learning itself. Of especial interest is that human depressives, especially suicides, have decreased cerebral noradrenaline levels; since the brains of animals with learned helplessness also show decreased noradrenaline levels, the implication is that biochemical changes are *secondary* to the depressed mood which followed behavioural events, rather than the biochemistry *causing* the depressed behaviour.

The theory of learned helplessness is similar to cognitive theories which say that depression results from diminished SELF-ESTEEM, an inability to see one's own actions as responsible for change in the world, an emphasis upon FAILURE rather than ACHIEVEMENT, a stressing of negative connotations of events, and ATTRIBUTION of success to external agencies rather than to the person themself.

Therapies derived from learned helplessness and cognitive models make the subject aware of controlling their own actions, and seeing the consequences of those actions. A dog with learned helplessness can be treated behaviourally by dragging it over the barrier while being shocked, demonstrating that its action will stop the punishment. (Interestingly the dog is also helped by electro-convulsive therapy and by antidepressant drugs, which are used in human depression.) Cognitive therapies aim to restrict depressed, negative thoughts, by identifying depressive cognitions, which are often AUTOMATIC THOUGHTS, reflex responses to particular situations (e.g. a friend in a hurry rushes off; the depressive thinks 'They are bored. Everyone finds me boring'), and consciously replacing them with alternatives ('Well they were in a hurry, I'll ring later and invite them to the cinema'). The treatment is coupled with EXPERIENCE OF SUCCESS; small easy tasks are successfully accomplished and rewarded, helping to show the fallacy of self-fulfilling cognitions such as 'What's the point in trying, I'm bound to fail?'

PERSONAL CONSTRUCT THEORY OF DEPRESSION

In depression the predominant emotion is GUILT, which arises from an awareness of contradictions or separation between the core constructs of 'actual self' and 'self as ought to be'; the separation is inversely related to SELF-ESTEEM, which is least in depressives and highest in manics, with normals in between. Consider the problem of depression in students. The construct of 'Student – not student' has other

constructs in parallel, perhaps:

Student	—	*Not student*
Active	—	Inactive
Interesting	—	Boring
Thinking	—	Unthinking
Vivacious	—	Inactive
Living for today	—	Living only for the future
Stimulating	—	Tedious
Always out at theatres, concerts, etc.	—	Sit at home working all the time

And if the construct 'Self as should be – self as really am' is also put on this system then it will appear as:

Student	—	*Not student*
Self as should be	—	Self as really am

Although the *image* of students is one thing, *reality* is different. If the terms on the left-hand side are also those associated with core constructs for important aspects of living, then guilt emerges. That guilt can be removed in several ways. The construct system could be rebuilt in a way that accepts that stereotypes often don't correspond to realities, and that one can live a life with components of stereotype and reality; such a change would however induce anxiety, and be threatening, particularly if imposed from outside, by problems with social life or exams. Alternatively, the number of constructs can be reduced so that contradictions disappear; but that is the route to the monolithic construct system of neurosis. The depressive response does not alter the structure of the system but instead reverses the components associated with the self, accepting that 'self as should be' is the same as 'self as really am', and hence concentrates only on work, perhaps seeing it principally as a way of getting on in life, etc. The problem with that solution is that the self is construed almost entirely in *negative* terms (uninteresting, boring, etc.), which makes further contradictions emerge, with further guilt; and so on. Contradictions can be minimized by construing less elements (by having less interests, meeting fewer people, doing less things, etc.) but that also exacerbates the problem.

THE PSYCHOANALYTIC MODEL OF DEPRESSION

In 1917, in his *Mourning and Melancholia*, Freud noted the similarities between grief and melancholia (an old-fashioned name for depression), and discussed the processes of ATTACHMENT and LOSS. Freud said that attachment involves a discharge of emotion (the LIBIDO) on to an external object (a person or thing) and the creation of a mental image

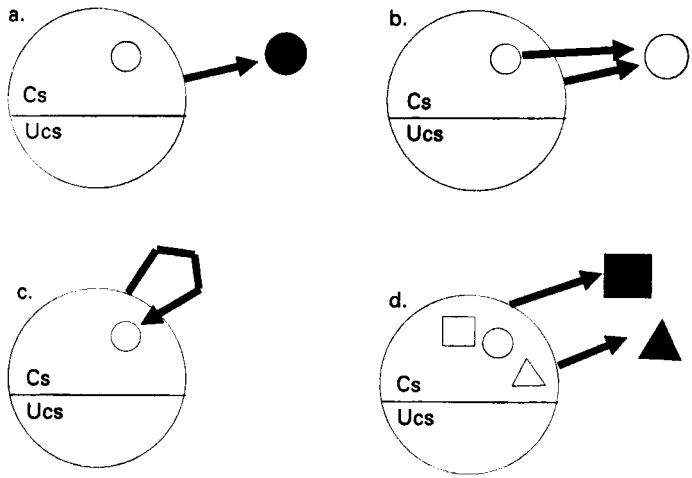

Fig. 29.1 The process of attachment and loss in adults. *a*) The object (solid circle) has emotions projected on to it, and a mental image (open circle) is formed in the conscious mind (Cs). *b*) After loss the mental image of the object firstly causes an hallucinatory external image of the object (large open circle) to occur in the outside world. *c*) The normally outward directed emotions directed inwards to the mental image of the object. *d*) The emotions are transferred to other objects, for which mental images are formed. The mental image of the original object still continues to exist.

of that object (Fig. 29.1a). After loss we firstly hallucinate an object on to which we discharge the emotion (Fig. 29.1b), but then the ego's REALITY TESTING confirms the object does not exist, and the emotions are directed inward to the mental image of the object (Fig. 29.1c). Finally CATHEXIS occurs when the emotion discharges onto new objects, albeit while retaining the mental image of the old object, which perpetually remains a part of us (Fig. 29.1d).

Freud gave no detailed account of how this process resulted in depression, and for that we must look at the work of the British psychoanalyst Melanie Klein (1882–1960) who undertook psychoanalytic explorations of infancy and childhood, and formulated a theory of OBJECT RELATIONS, of early attachment. In earliest infancy, the child relates to objects (either people or things) by expressing love; the archetypical object is the mother's breast, the fount of all good things. The love initially is expressed just onto the object (Fig. 29.2a) but then with INTROJECTION a portion of the object is taken into oneself as a mental image, and love is directed at the image as well as the object (Fig. 29.2b), and the object thereby acquires new properties by PROJECTION. Inevitably temporary SEPARATION from the object occurs, as also do frustrations when the object does not reciprocate love; then hate is directed both at the object (or its absence) and at the mental image (Fig. 29.2c). The child learns not only to direct love but also

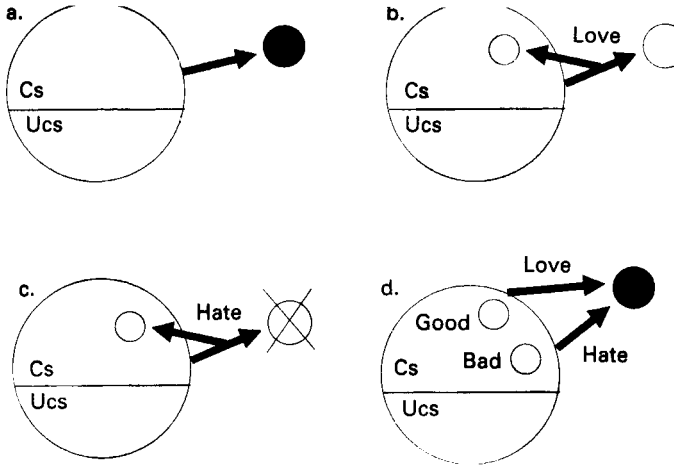

Fig. 29.2 Development of the paranoid-schizoid and depressive positions in infants. *a*) Emotions are projected outwards onto the object. *b*) A mental image of the object is formed and the emotion of love is projected both outwards onto the object itself and inwards onto the mental image of the object. *c*) The image outside either disappears temporarily or otherwise frustrates the infant so that the emotion of hate is directed both outwards onto the object and inwards onto the mental image of the object. *d*) The infant accepts that the same object has both good and bad aspects, towards which love and hate can both be directed, and separate good and bad mental images of the object are formed.

to direct hatred. At times that hatred is directed at the same object that previously had been loved (SPLITTING), resulting in the PARANOID-SCHIZOID POSITION.

At first a child fails to integrate the separate parts of an object, so the mother's breast, face, arms, etc., are different PART OBJECTS, and separate emotions are directed at them. As the child understands that one object has several facets, so GUILT develops at the hatred, directed at different parts of the now complete object, especially the mother, resulting in the Kleinian DEPRESSIVE POSITION; it is realized that loved and hated objects are the same, having both good and bad aspects, to which love and hate are separately directed (AMBIVALENCE), and two separate internal images develop, one good and one bad (the 'good breast' and the 'bad breast' in Kleinian terminology).

Early LOSS (i.e. a permanent loss, rather than normal, inevitable temporary separations) produces a situation in which after the expression of love (Fig. 29.3a), hate is then expressed at the object and its mental image (Fig. 29.3b). However, the child cannot explain the object's disappearance, and therefore blames itself for the loss, and introjects hatred against both the object and itself (Fig. 29.3c), the self-hatred being unconscious, and associated with GUILT at driving away the loved object. This vulnerability means that subsequent losses

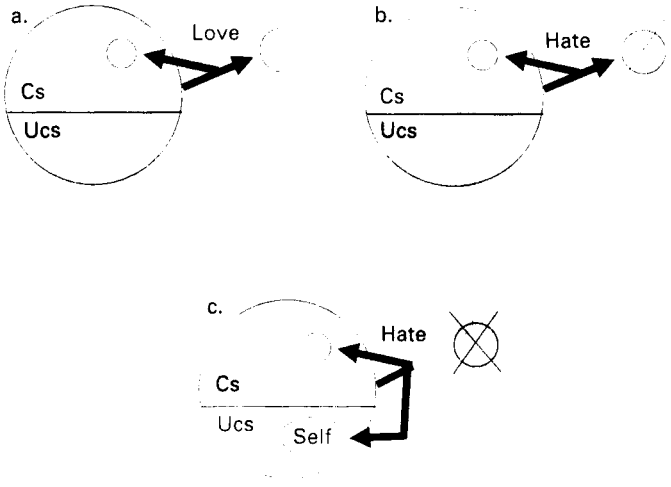

Fig. 29.3 The effects of loss in infancy. *a*) As in Figure 29.2, the infant projects love onto both the external object and also onto its own internal representation of it. *b*) The object disappears permanently (rather than temporarily as in Figure 29.2), and at first the infant acts as in the case of temporary separation, projecting hate onto the object and its image. *c*) The object fails to reappear and infant blames itself for the loss, directing the hate inwards onto both the conscious representation of the object and onto the unconscious (Ucs) representation of the self, which was felt to be responsible for the loss.

in adult life produce a reversion to infantile self-hatred resulting from guilt at responsibility for the loss, particularly when the adult loss involves ambivalence, with feelings of love and hate towards the lost object. Although the precipitating loss in adulthood may be fairly trivial it can result in a severe depressive attack in a vulnerable psyche.

A SOCIOLOGICAL MODEL OF DEPRESSION

The previous models have suggested that depression is a response to events in the immediate world, which affect the way that world is construed and are greater if the psyche is already damaged by loss at an early age. The British sociologists, Brown and Harris, have integrated those ideas into a comprehensive model, which is impressive in its elucidation and its testing against empirical data, and provides a general model for the influence of social phenomena upon disease, physical or psychological. The hypothesis (Fig. 29.4) says the immediate cause of depression is RECENT PROVOKING EVENTS, which produce the condition. However, since provoking events do not *always* cause disease, some individuals must have VULNERABILITY FACTORS, which

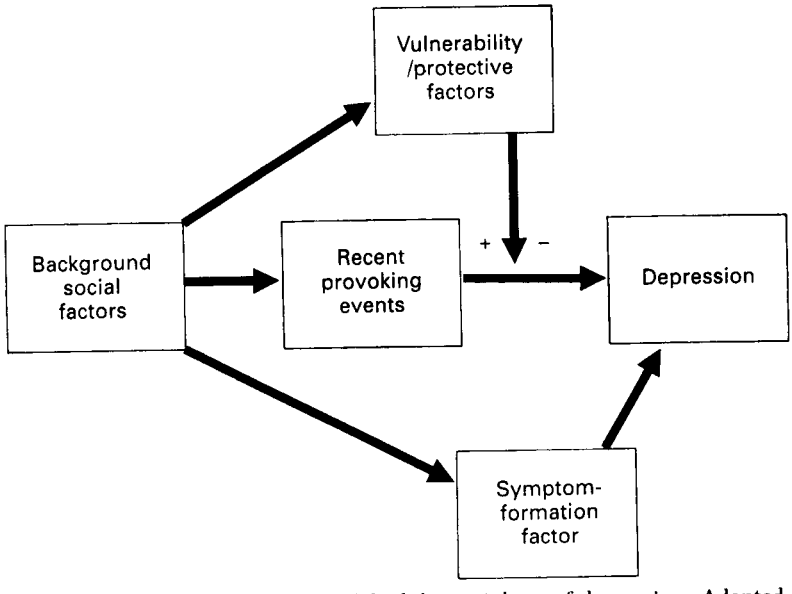

Fig. 29.4 Brown and Harris' model of the aetiology of depression. Adapted from Brown G W and Harris T (1978). *Social origins of depression*, London, Tavistock, 48.

exaggerate the provoking events, or PROTECTIVE FACTORS, which immunize against provoking events. Not all individuals who develop the condition have identical symptoms, and SYMPTOM-FORMATION FACTORS determine the precise nature of the disease, without necessarily affecting the likelihood of disease itself. Finally each factor, be it provoking event, vulnerability or protective factor, or symptom-formation factor, is influenced by background factors, such as social class, early experience, etc.

The core of the model says that recent provoking events *cause* depression; proof requires two steps, demonstration of a correlation and evidence that the relation is causal rather than mere association. Brown and Harris took careful systematic histories from a large population of women to assess 'events' in their lives over the previous year. Severity of events was assessed by independent judges. Depressed women had more *severe* events during the previous year, but *non-severe* events were as frequent in controls as in depressives. Therefore, not all events cause depression, only those perceived as severe. Severe events occurred throughout the previous year, but particularly in the six weeks prior to the illness (Fig. 29.5). Correlation had therefore been demonstrated. Causation is more difficult. Either events may be FORMATIVE, the event causing the depression (e.g. husband leaves and woman then becomes depressed) or TRIGGERING, in which depression occurs spontaneously and then causes the event (e.g. woman becomes depressed and husband leaves *because* she is depressed). These situ-

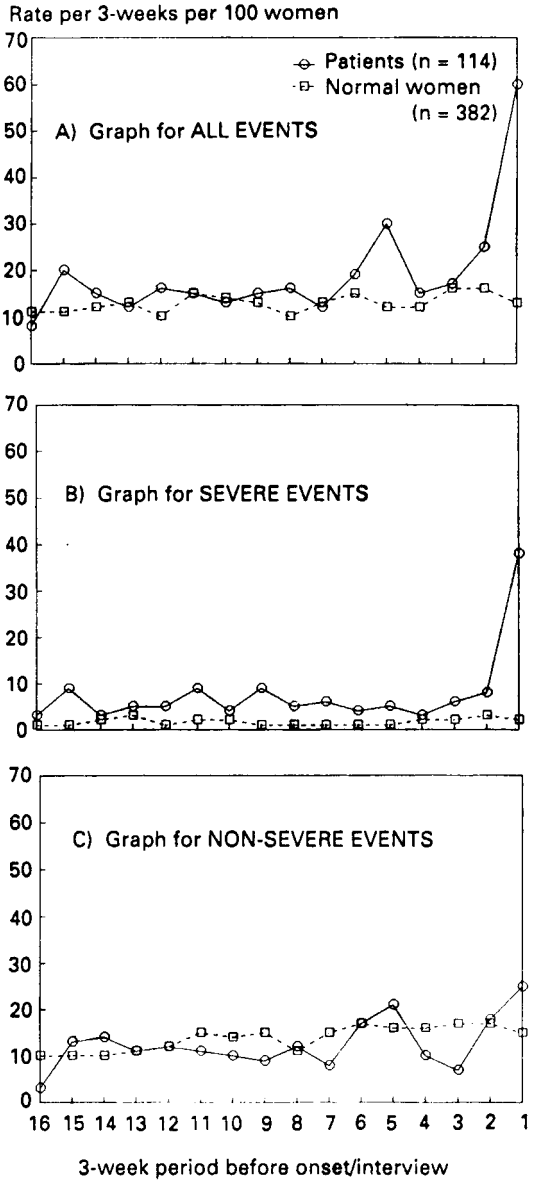

Fig. 29.5 The rate at which events (A: total; B: severe; and C: non-severe) had occurred in the 16 three-week periods prior to the onset of depressive attacks in patients, or prior to interview in a group of normal, control women. Adapted from Brown G W and Harris T (1978), *Social origins of depression*, London, Tavistock.

ations are distinguishable by the TIME BROUGHT FORWARD INDEX. Imagine a clock that strikes the hours. If an event occurs at random then there will be a certain delay until the clock next strikes. Some events occur just before the hour, and others long before, with an average delay of 30 minutes before the clock struck. If an event (such as a finger inside the works) *caused* the clock to strike then the delay before

the next strike would be less than if event and strike were independent. If striking was caused by events then they should occur sooner than expected, and the strikes are said to be brought forward in time as compared with chance expectations. It is measured by the TIME BROUGHT FORWARD INDEX, which if short indicates triggering, whereas if long indicates formative influences. Attacks of depression are brought forward nearly two years by severe events, indicating a formative or causal influence. Non-severe events bring forward attacks by only ten weeks or so, suggesting triggering, with illness causing event. When applied to acute schizophrenic attacks, where previous work had suggested social events were unlikely to be causal, the time brought forward index for severe events was only ten weeks, indicating triggering, and providing a 'control' for depression, where events are formative.

Vulnerability and protective factors manifest not simply by correlating with depression, but by modulating the relationship between events and depression. Considering *only* individuals experiencing severe events in the past year, 10% of those with high psychological intimacy with their husband became depressed, compared with 26% with medium intimacy and 41% with low intimacy. Intimacy, the ability to express feelings freely, without self-consciousness, with trust, effective understanding, and ready access, is therefore a protective factor. Intimacy was less common in women of lower social classes, showing the pervasive role of background factors and partly explaining the social distribution of depression. Husband's employment, lack of employment for the woman outside the home, having three or more children at home, and loss of a mother before the age of 11 were all vulnerability factors. The latter is particularly important in relation to psychoanalysis, especially as loss of a mother only made women more vulnerable when it occurred before 11 years of age in conjunction with severe events; loss of mother was unrelated to depression after non-severe events, and loss of a *father* (before or after age 11) did not alter vulnerability.

On becoming depressed it is symptom-formation factors that determine the degree and type of the symptoms. The SEVERITY of a depressive attack related to the number of severe events suffered (but not their type), and was greater in those experiencing loss of mother or sibling before age 11. Loss of mother is therefore *both* a vulnerability factor *and* a symptom-formation factor. Depressions also vary in symptom type, some having a neurotic pattern, with a specific cause apparent to the patient, and others are psychotic, without causal insight and with 'biological' symptoms. Depression type did not relate to the number, severity or type of provoking events, but was related to age (older patients having more psychotic symptomatology) and to experience of loss. Not only was previous loss more common in psychotic depression, but the manner of loss related to depression

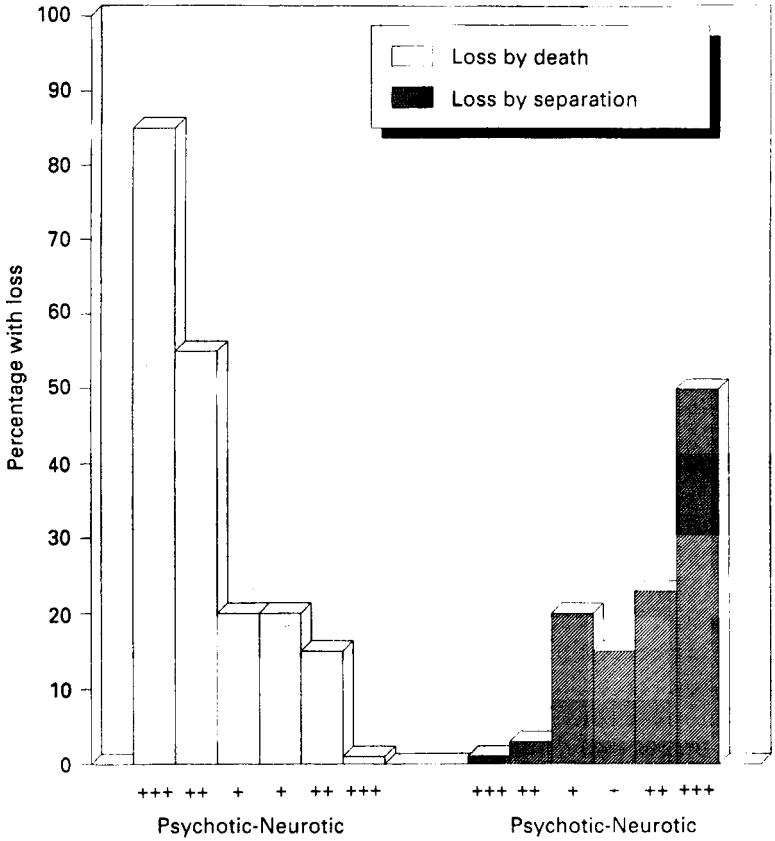

Fig. 29.6 The proportion of depressed patients with a history of loss either by separation or by death, according to the type of depression (psychotic or neurotic), and its severity (low, medium or high). Adapted from Brown G W and Harris T (1978). *Social origins of depression*, London, Tavistock.

type; loss of parent or sibling before age 11 by DEATH was associated with psychotic symptoms, whereas loss by SEPARATION (e.g. divorce, etc.) was related to neurotic symptoms (Fig. 29.6). Childhood loss therefore affected the symptoms seen in adult depression two, three or four decades later. The child's implicit explanation of family loss subsequently related to understanding of their adult illness, either being explicable and comprehensible in the case of separation, particularly if associated with prior arguments and discord, or being inexplicable, arbitrary and the blows of incomprehensible fate in the case of death due to illness or accident. The Brown and Harris model therefore says that the distinction between endogenous and reactive depression is aetiologically inappropriate (for they propose *all* depressions are causally reactive on prior severe events) but is important symptomatically, indicating differing cognitive explanations of illness in relation to early experience.

This chapter has tried to emphasize how depression can be analysed at many levels, from the behavioural model in laboratory animals, to the interpretations of the analyst's couch, and to large-scale sociological models, with each having common components in its emphasis upon *loss*, its explanation and understanding, and the subsequent generalization to future losses. No single model can be correct; rather all emphasize different aspects of a common problem.