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Schizophrenia

- Schizophrenia is characterized by HALLUCINATIONS, DELUSIONAL PERCEPTIONS and DELUSIONAL EXPERIENCES, as well as by THOUGHT DISORDER, in which thinking is illogical, language is disordered, and thought is concrete rather than abstract.
- Schizophrenia probably has a genetic component, although this is probably not primarily an abnormality of neurotransmission, but may instead be a deficit of ATTENTION or HABITUATION, which manifests early in childhood.
- Many of the abnormal behaviours shown by schizophrenics are the result of INSTITUTIONALIZATION and inappropriate reinforcement of behaviour.
- LOOSE CONSTRUING can be induced by SERIAL INVALIDATION, a process akin to the DOUBLE-BIND, early invalidation resulting in non-paranoid schizophrenia and late invalidation in paranoid schizophrenia.
- Psychoses cannot be treated by psychoanalysis due to a failure to form a TRANSFERENCE relationship with the analyst. This may reflect fixation at the PARANOID-SCHIZOID position and the inability to form proper OBJECT RELATIONS.

Schizophrenia is the most difficult of all the mental disorders. The clearest and most serious example of a psychosis, it is difficult to treat, difficult for families and relatives to cope with, and difficult for an outsider to understand, the utter lack of insight by the patient being complemented by an absence of empathy by outsiders for the patient's mental state; unlike neurosis or depression, where we all have suffered minor forms of the conditions, in schizophrenia we can draw on almost no personal experience that will help us to see the world through the patient's eyes.

Schizophrenia was first described systematically in 1896 by the German psychiatrist Kraepelin (1856–1926), who called it by the inappropriate name of DEMENTIA PRAECOX (precocious or premature dementia), inappropriate because there is no dementia as intelligence and memory are unimpaired. In 1911, Bleuler (1857–1939) coined the modern term SCHIZOPHRENIA, from the Greek *schizos* (a split) and *phrenos* (mind). Despite recurrent misusage in the popular press, schizophrenia is *not* a split mind or DUAL PERSONALITY (a 'Jekyll-and-

Hyde syndrome', an entirely separate condition which does occur, albeit rarely); rather the split is both between thought, feelings and activities, which no longer act in harmony, and also in the normal differentiation in space and time between self and not-self.

Clinical diagnosis looks for Schneider's FIRST-RANK SYMPTOMS of hallucinations, delusional experiences and delusional perceptions, any of which, in the absence of organic lesions, are sufficient for a presumptive diagnosis of schizophrenia. HALLUCINATIONS are experiences indistinguishable from sensory events that occur in the absence of sensations; they must not be confused with ILLUSIONS, which are distortions of sensation or perception, or DELUSIONS in which a *belief* is erroneous. Schizophrenic hallucinations are auditory (and other modalities indicate other conditions, typically being visual in *delirium tremens*, olfactory in temporal lobe epilepsy, and cutaneous in cocaine overdosage), and are either audible thoughts, voices arguing or voices commenting on the patient's actions (when they are grammatically in the third person, unlike the rare auditory hallucinations of severe depression which are in the first person). Auditory hallucinations while falling asleep or when waking, HYPNAGOGIC and HYPNOPOMPIC HALLUCINATIONS, are common and regarded as entirely normal. DELUSIONAL EXPERIENCES are disorders of the EGO BOUNDARY. Normally we have a precise sense of the extent of our own body which we control, compared with the rest of the world which is beyond direct control. If I put my hand on a table I know the fingers are mine, and I can move them and feel things touching them, whereas the table, only a micron beneath my fingers is not me, and I neither control it nor derive sensation from it. In schizophrenia the ego boundary shifts, patients either believing their body controlled by others (PASSIVITY), or that thoughts are inserted in their mind, or wishes, instincts or emotions are controlled externally, or that their thoughts are broadcast and affect other people directly. DELUSIONAL PERCEPTIONS (or just DELUSIONS or PRIMARY DELUSIONS) are abnormal beliefs about the world, often with a bizarre content involving ideas or threat or persecution (PARANOID DELUSIONS) or in DELUSIONS OF GRANDEUR of being a person of importance (e.g. Napoleon or Jesus Christ). Delusions are incomprehensible to an outsider and are characterized by:

i. Being held with absolute conviction.
ii. Being experienced as self-evident truths, typically of great personal significance.
iii. Not being amenable to reason or modifiable by experience.
iv. The content being fantastic or at best inherently unlikely.
v. Not being shared by those of a common social or cultural background.

Such criteria ensure that simple values or beliefs (for instance concerning political or religious questions) are not interpreted as

delusions (although delusions often have religious or political content). Leonard Woolf described a typical delusion when his wife Virginia, during a psychotic attack, believed the birds in the garden were singing in Greek; and he gave eloquent testimony to the folly of attempting to reason with the holders of such delusions.

Psychologically schizophrenia is characterized by THOUGHT DISORDER, a disorder of cognitive processing. Language is used unsystematically, with distorted grammar. Sentences are incomplete and rambling, phrases or sentences are not logically connected, and there are sudden changes of direction and irrelevancies. Thinking is illogical, with OVER-INCLUSION, conceptual and semantic boundaries being ignored, resulting in CLANG ASSOCIATIONS, where similar word sounds imply similar meanings, and the creation of NEOLOGISMS, words or phrases newly created from parts of other words or phrases (e.g. 'cosmetic neurosurgery', or 'placebionic' from 'placebo' and 'bionic'). Thought disorder can be demonstrated experimentally by sorting playing cards, schizophrenics being idiosyncratic in their placing, and ignoring conventional distinctions of suit and value. Thought is also CONCRETE, abstract ideas not being processed properly, as is shown clinically by asking a patient to explain a proverb such as 'A rolling stone gathers no moss'; they talk at length about moss being unable to stick, or grow, and ignore the metaphorical meaning of the proverb. Similarly, descriptions of pictures are concrete rather than abstract, emphasizing the physical objects present and ignoring the relationships between individuals or their motives. Schizophrenic thought can have a poetic flavour (as when a patient was asked the difference between a wall and a fence: 'A fence you can see through but a wall has ears'), and it is suggested that a milder variant, called the SCHIZOID PERSONALITY, is associated with artistic creativity.

Schizophrenia can be subdivided in several ways. Two particular distinctions are useful in practice. Paranoid patients typically present in middle age with mainly paranoid delusions of slow insidious onset, and little thought disorder; non-paranoid patients present between 15 and 35 years of age with an acute attack and relatively minor delusions but extensive thought disorder. Auditory hallucinations occur in both types, and in both types remission can occur, especially if treated with major tranquillizers such as chlorpromazine. Relapse can then follow, resulting in the chronic or 'burnt out' form of the disease found in patients institutionalized for many years. Recently, TYPE I schizophrenia, marked by 'positive' symptoms, such as hallucinations, delusions and thought disorders, which occur acutely and respond well to neuroleptics, has been distinguished from TYPE II schizophrenia, the chronic condition where symptoms are mainly 'negative', with cognitive impairment, poor response to neuroleptics, and increased ventricular size and reduced cortical substance seen on cerebral tomography. The different types of schizophrenia may have

different aetiologies, implying a heterogeneous disorder.

An idea of the peculiar and special flavour of schizophrenic thought can be given in a letter written by Ivor Gurney, a talented composer and First World War poet, who from the age of 33 spent the last fifteen years of his life in an asylum. He developed auditory hallucinations, and delusions that he was tortured by wireless waves and could converse with the great composers and had in fact written much of their music. This letter was written when aged 43, as if from Siegfried Wagner, the son of the composer:

> 'This is Franz Schubert's death day and enough for Kent Reach to think of — whose chimneys smoked grandly, but Nature said 'W.E. Henley Prague 1731' — So light dies down — But after saying that Europe should not stand war for its thought, by 137 degrees —
>
> Barnet won I think/especially about the South, most likely — but perhaps about the going North, where may Italy (North) and Rochester book-sellers flourish.
>
> The weeks and battles forgotten — (terrible to remember) need all the good of Artois and Picardy's poetry —
>
> Meanwhile the regrets and hopes of Highnam Manor float about the world — now vast in great victory, and as urgently as Sidgwick & Jackson cry out for the honour of provinces.
>
> Virgil is running 2/3, and is the master of business — music is at last in a very healthy state/and Rutland Boughton's group is announced — Rudford and Minster, that accepts such news, now forgets its threats and terrible fears, St. Maur gives news of pride every day, and seems to enjoy Welsh local poetry.
>
> It seems true that Anton Dvorak and Rochester Cathedral have truly the salvation of music and poetry — and one might want for Spring with 137 hopes — But desperate is Hebbel's need for Frankfort's urgencies.
>
> 'Lyrik, lyrik' in Schiller's name ever demanding, and let London lose its regrets and clear its history there with the brutality of Eaglefield-Hull, and as the name of Thames flows steadily to honour.
>
> Nature, now sombre, sometimes says tremendous things, and sometimes lightly, as of the biographer of F. P. Schubert, and waits for Florence City's reasons and reason —
>
> That London may honour St. Crispin — a saint glorious enough for Aquitaine/and to please William Cartwright, magister artis.
>
> I ask for some more money, please, and a small poetry book if there is one (modern if it can be got). It is painful and hard to write about modern anthologies though – and so better to rejoice (theoretically) about *Max Reger* —/ England is a South Place story...
>
> The glory of 1913 and 1914 is here remembered/as always worth the imagination of Petrograd and Moscow. (140–183); later

with the surpassing interest of men's music — like our hopes of Novgorod's earth.
with best wishes
(yours very sincerely)
Ivor Gurney
Oct 23 1933 Dartford. Kent.'

Reproduced with permission from
Hurd M, *The ordeal of Ivor Gurney* (1978)
Oxford University Press, 165

The letter shows clear signs of thought disorder, with disordered grammar, and incomplete rambling sentence structure; nonetheless it is clearly not just 'noise', but has a deep and complex meaning, albeit obscure, and at times its beautiful imagery is replete with associations, particularly for those who can perceive the musical, military and literary motifs.

Many models have been proposed for a disease as serious and complex as schizophrenia. As previously we will consider the learning, personal construct and psychoanalytic models, and will also consider the biochemical-genetic model as well.

THE BIOCHEMICAL-GENETIC MODEL OF SCHIZOPHRENIA

This model is not only extremely popular but also has several inherent fallacies of particular interest.

Schizophrenia undoubtedly has a genetic component. The life-time prevalence is 1% in the general population compared with 35% if one parent is schizophrenic, or 70% if both parents are schizophrenic. If both parents are normal then the risk is 10% with an affected sibling, 15% with an affected dizygotic twin, and 50–80% with an affected monozygotic twin. That these familial trends are genetic rather than environmental is shown in adoption studies, a child being more like biological than adoptive parents. Nevertheless, neither a single Mendelian gene nor polygenes explain the pattern of inheritance.

Genes produce proteins, particularly enzymes, and since enzymes metabolize neurotransmitters, schizophrenia has been argued to be due to abnormal neurotransmission. But which neurotransmitter? During the past four decades many candidates have been proposed: acetylcholine, noradrenaline, serotonin, GABA, dopamine, prostaglandins, and endorphins. Each has had its heyday, usually following an improved measurement technique, rather than strict argument. The most convincing hypothesis implicates abnormal dopamine transmission, and the logic of that specific argument will be considered here.

The 'dopamine theory of schizophrenia' originated in the discovery

that major tranquillizers, or NEUROLEPTICS, such as CHLORPROMAZINE, not only alleviate the symptoms of schizophrenia but also block dopamine transmission, both *in vivo* and *in vitro*; hence it was argued that the antipsychotic effect occurred *because* of blocking dopamine transmission. That, however, is still controversial: degree of dopamine blocking and neuroleptic antipsychotic action are only moderately correlated; dopamine blocking occurs within minutes *in vivo* and yet antipsychotic action takes several weeks to occur; and those behavioural effects of neuroleptics related to dopamine blockade show TOLERANCE after several weeks, whereas no tolerance is shown in antipsychotic action. Nevertheless, despite such difficulties, 'the dopamine hypothesis of neuroleptic action' is generally felt to be correct. But that does not prove the 'dopamine hypothesis of the *cause* of schizophrenia'. Consider some clear counter-examples: Parkinson's disease is alleviated by anticholinergics, but is due to a dopamine deficit; and Huntington's chorea is treated with dopamine blockers but is due to a GABA deficiency. To push the argument to its logical absurdity, pneumonia is treated with penicillin, but in no sense is due to low levels of penicillin in the lungs or serum. More direct evidence is needed that excess dopamine is the cause of schizophrenia, but such evidence has not been forthcoming, dopamine levels not being convincingly raised in the brains of schizophrenics. A variant of the model therefore argues for increased receptor sensitivity to normal levels of dopamine, and that seemed to have been found in some post-mortem studies. However, most schizophrenics now receive treatment with dopamine blockers, which could actually be causing the receptor differences; and post-mortem examination of the rare brains of schizophrenics who are not on medication indeed suggests receptor sensitivity is normal.

I wish to argue that the biochemical model is overly simplistic, ignoring the inherent complexity of mental illness, and the interaction between brain function and environment. The simple model (Fig. 30.1a) is inadequate for the reasons given above. A more complex model (Fig. 30.1b) argues for a PRIMARY COGNITIVE DISABILITY caused partly by genetic factors; certainly longitudinal studies show that children subsequently developing schizophrenia were passive babies, had short attention spans, difficulties at school, poor control of emotions, and were disruptive in class. By age 15 they showed signs of early thought disorder. Such behaviour is not itself psychotic, but eventually results in a PRIMARY PSYCHOTIC DEFECT, which as a result of other disruptive factors such as social processes, or even environmental factors such as viral infections, results in the florid symptoms of schizophrenia, the SECONDARY PSYCHOTIC DEFECT, which then further impairs social interactions, and exacerbates the problem further. Treatment by neuroleptics diminishes the primary psychotic defect, but this alone is insufficient to alter the secondary defect, which is

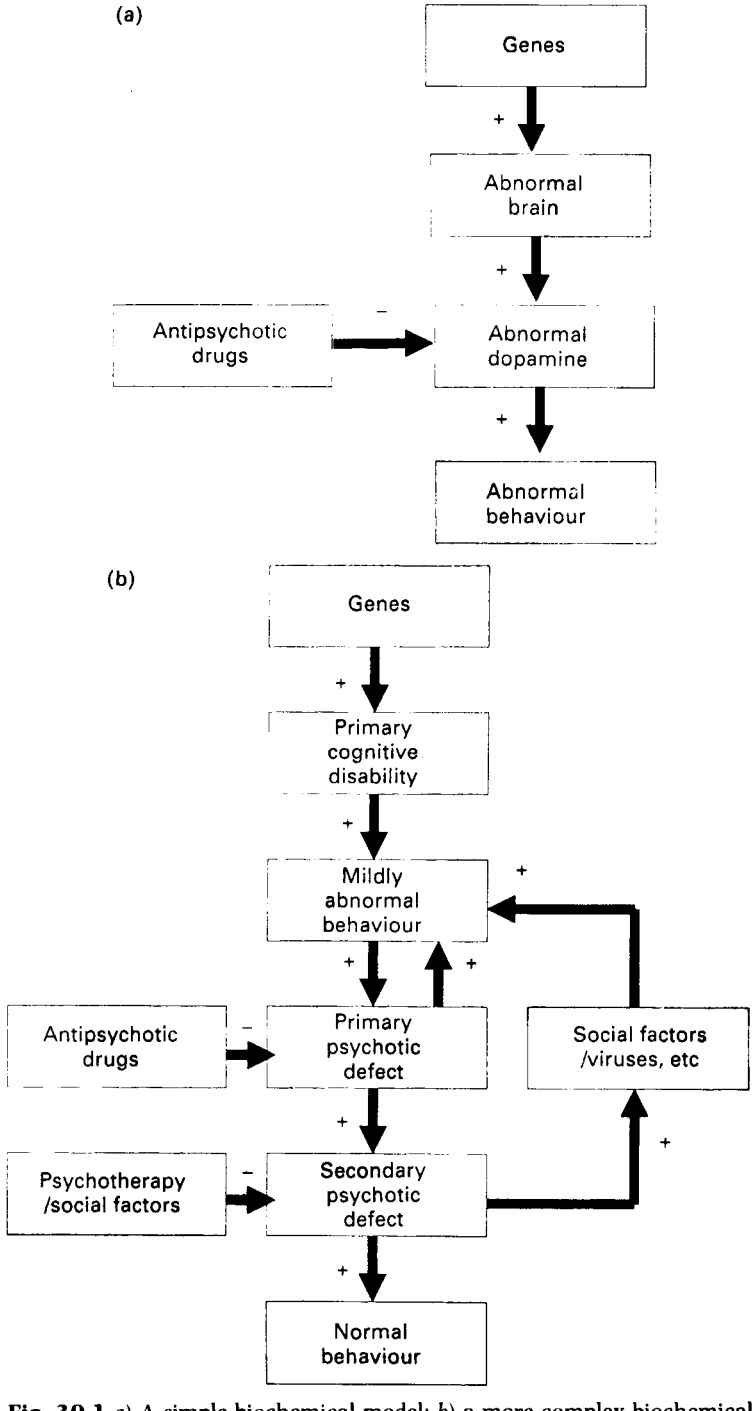

Fig. 30.1 *a*) A simple biochemical model; *b*) a more complex biochemical model. See text for further details.

maintained by social factors; however psychotherapy and social therapy, in combination with drugs, help remove the secondary defect, albeit after some time, and relatively normal behaviour is restored, leaving a system still vulnerable to future problems.

Such a model pre-supposes a primary cognitive disability present in early life, which might be a defect in ATTENTION. Normally we are bombarded by sensory inputs, most of which are consciously ignored until something interesting occurs, when consciousness is directed at them; thus as you read this you were probably unaware of the pressure of the chair upon your buttocks until it was mentioned, when suddenly it impinged upon consciousness. Much evidence suggests an ATTENTIONAL DEFICIT in schizophrenics, who are continually bombarded with sensations they are unable to ignore, both from external events, and from internal processes, such as memories, etc.; this partly explains the pressure of ideas and the clang associations in thought disorder. The other side of attention is HABITUATION, ignoring a frequently presented stimulus. In normal individuals, the GALVANIC SKIN RESPONSE (GSR) to a repeated stimulus shows a sudden rise in skin conductance at presentation followed by a return to normal levels. A slow return to normal indicates lessened habituation. Children of schizophrenic parents, who are at higher risk of becoming schizophrenic, show decreased habituation and less attention than controls. Chlorpromazine, in man and animals, decreases distractibility, and hence increases attention and habituation, perhaps explaining its role in treating the primary psychotic disorder of schizophrenia. Of interest is that hippocampal lesions in monkeys not only produce decreased attention and GSR habituation, but these can be treated by chlorpromazine. There is now growing evidence that human schizophrenics show structural abnormalities in the temporal lobe, particularly in the left hemisphere.

The biochemical-genetic model has been examined in detail because it is a popular model of the aetiology and pharmacotherapy of schizophrenia. However, it tells us little of the *experience* of schizophrenia, and ignores many aspects of the disease, for which we must look to specifically psychological models.

THE LEARNING THEORY MODEL OF SCHIZOPHRENIA

Learning theory has no specific model for the *cause* of schizophrenia, but nonetheless explains many of the strange behaviours of schizophrenics. Being schizophrenic does not make one incapable of learning, and of responding to events in the world. If that world is bizarre, then bizarre behaviours can result. The world of a chronic schizophrenic, the long-stay mental hospital, is indeed strange and many behaviours result from that social environment due to inadvertent reinforcement

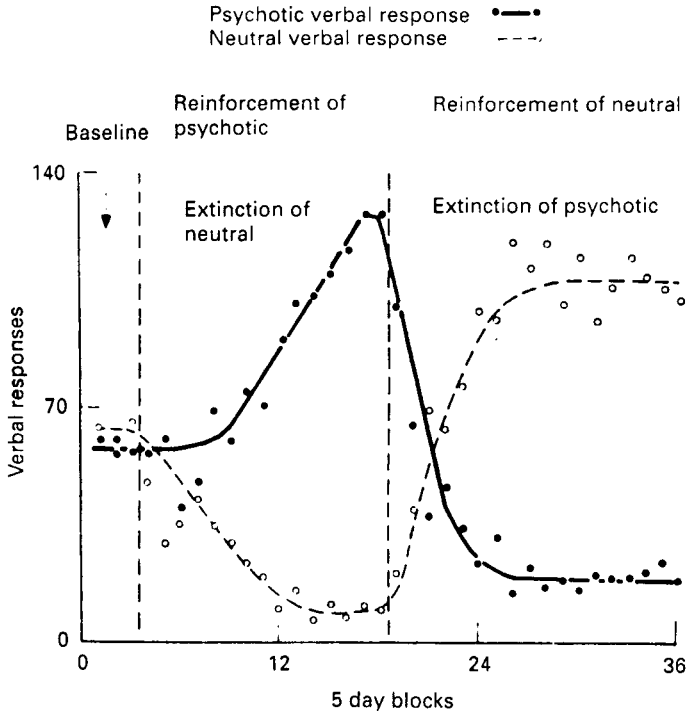

Fig. 30.2 The influence of differential reinforcement of 'psychotic' or 'normal' types of speech on the verbal behaviour of psychotic patients. Reproduced with permission from Allyn T and Houghton E (1964), Modification of symptomatic verbal behaviour of mental patients, *Behaviour Research and Therapy*, **2**, 87–97.

by staff and patients, and as a result of INSTITUTIONALIZATION. If patients are ignored by staff or told to be quiet then some will learn to be taciturn and uncommunicative; and if a well-meaning person listens to a patient's paranoid delusions, nodding their head repeatedly, but ignoring the content, then such speech and ideas are reinforced (see Fig. 30.2). Head-banging and tantrums are also examples of ATTENTION SEEKING behaviours, which are rewarded by attention from staff. Learning is itself exploited in TOKEN ECONOMIES; beneficial social behaviours such as self-care, good appearance and voluntary activities are rewarded with tokens which are exchanged for privileges or goods (see Chapter 31).

Some theorists argue that schizophrenia itself is a learned response to the social environment of the family. Bateson's DOUBLE-BIND THEORY suggested that parents place their children in situations where they cannot possibly do right. A mother might complain that the child hasn't kissed her, but withdraw and show dislike when kissed; whether affection is shown or not the mother's behaviour shows dislike, and since the situation is inescapable, the child learns that all behaviours

result in punishment, and instead the child withdraws into itself and learns to ignore the outside world. Although poorly supported by empirical evidence, the theory is popular and the phrase SCHIZOPHRENOGENIC MOTHER is now a part of psychiatric terminology. The theory influenced ANTI-PSYCHIATRISTS, such as R D Laing, who said schizophrenic symptoms were meaningful responses to an intolerable situation in which the entire family was abnormal. Although also not supported by adequate data, such family studies have inspired specific studies on the problems of relapse in schizophrenic patients discharged home on neuroleptic medication, which show unequivocally that high levels of EXPRESSED EMOTION in the families, usually over-involvement, hostility or criticism, result in higher relapse rates; patients should be discharged into such families with care and FAMILY THERAPY, treating the whole family to reduce deleterious communication, can benefit the patient.

THE PERSONAL CONSTRUCT MODEL OF SCHIZOPHRENIA

In personal construct theory, the most important empirical observation is that schizophrenics have loose construing, with many unrelated constructs; as such the construct system is similar to that of an obsessional but without the obsessional's single monolithic construct system — and it is of interest that obsessive-compulsives are at higher risk of subsequent schizophrenia. How does this system develop? As in neurosis, the driving force is anxiety resulting from invalidation and hence threatened change of the system, particularly due to SERIAL INVALIDATION, which shows similarities to Bateson's theory of the double-bind. Imagine that some person, X, is nasty to Y, the patient. X is construed by Y as 'hating'. Next time however X is nice to Y, and Y therefore changes their construction of X to 'loving'. The next occasion though X is nasty again, Y's construing changes back to 'hating'; and so on, X never behaving consistently, so that Y cannot construe them successfully, and Y's construct system is invalidated every time. There are two ways to make the system work in some degree. The relationship between constructs can be altered to produce IDIOSYNCRATIC CONSTRUING, the links between 'loving-hating' and other constructs such as 'considerate-inconsiderate' being reversed from the normal, so that, say, 'loving' and 'inconsiderate' are linked; when X is nasty they are then construed as 'loving and inconsiderate', and the system is not actually invalidated (although it predicts almost nothing, and is almost incapable of falsification). The alternative solution is LOOSE CONSTRUING, where 'loving-hating' exists in isolation from other constructs, so that X's nastiness predicts nothing else about X. Since the system predicts nothing, it also cannot be invalidated.

Loose construing is typically found in thought disordered non-paranoid schizophrenia, whereas idiosyncratic construing is typical of paranoid schizophrenia. Serial invalidation is thought to occur in childhood for the non-paranoid schizophrenic, producing an inadequate construct system that cannot cope with adult life, so that schizophrenia develops at a young age. By contrast in paranoid schizophrenia a normal childhood allows development of an adequate construct system, but serial invalidation in middle life then causes idiosyncratic construing. Serial invalidation does not produce generalized disordered construing, since schizophrenics have relatively normal construing of *physical objects*, but are especially loose at construing *people*, emphasizing the role of the social environment in causing schizophrenia.

Personal construct theory suggests therefore that schizophrenic thought is not meaningless noise but has a sense and meaning, albeit known principally to the patient themself, but which is accessible through careful analysis. Figure 30.3 summarizes diagrammatically the structures proposed by personal construct theory in neurosis and psychosis.

THE PSYCHOANALYTIC MODEL OF SCHIZOPHRENIA

Freud recognized early in his career that psychoanalytic therapy worked only with neurotics, and not with psychotics. In neurosis, psychotherapy works by the patient developing strong emotional attachments to the therapist (the TRANSFERENCE) and the therapist forming a relationship to the patient (usually attachment, but sometimes a dislike), COUNTER-TRANSFERENCE. The transference induces conflict between the patient's wishes and the analyst's wishes producing RESISTANCE and forming the TRANSFERENCE NEUROSIS, whose working through is therapeutic for the neurosis proper, for which the transference neurosis has acted as a surrogate.

Psychosis is untreatable by psychoanalysis because there is no transference, the patient being unable to form relationships. Freud said that in psychotics a breakdown in EGO-FUNCTIONS caused impaired REALITY TESTING and hence delusions and hallucinations, with loss of personal identity and failure to differentiate the internal world from the outside world: 'In a neurosis the ego... suppresses a piece of the id... whereas in a psychosis the ego, in the service of the id, withdraws from a piece of reality'. To Freud psychotic symptoms were a 'waking dream', PRIMARY PROCESS THINKING (akin to latent dream content) being unacted upon by censorship to produce SECONDARY PROCESS THINKING (the dream's manifest content), and emerging unchanged in unconsciousness producing conflicts and terrors. Freud merely described schizophrenia, but did not explain its origins, except in suggesting a failure

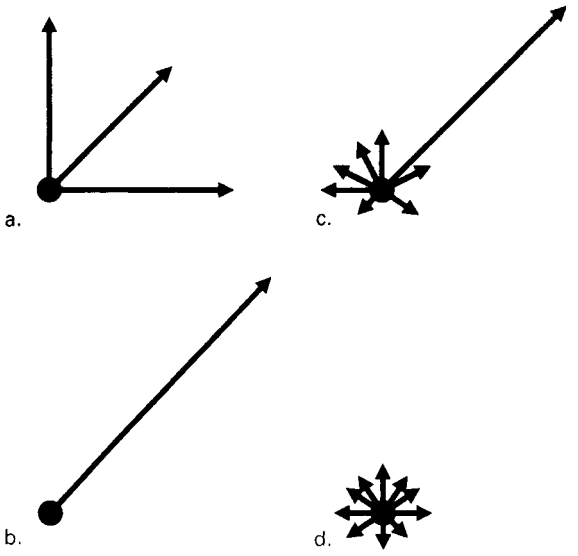

Fig. 30.3 A diagrammatic summary of the personal construct theory models of neurosis and psychosis. Each arrow represents an independent dimension in construct space (and the space can be multidimensional). The length of an arrow is proportional to the degree to which that dimension accounts for the relationships expressed in the repertory grid. In normal individuals (*a*) there are typically 3 or 4 principal dimensions underlying constructs. In individuals with phobias there is a single construct dimension (*b*), and in individuals with obsessional neurosis there is a single predominant dimension, but also a large number of small, independent dimensions (*c*). In schizophrenia there are large numbers of independent dimensions, each of which accounts for only a small proportion of the information in a repertory grid (*d*), and this looks similar to the subsidiary portion of the construct space found in obsessional neurosis.

of development early in childhood.

Melanie Klein said that schizophrenia resulted from problems at the very earliest recognizable age of psychological development, the PARANOID-SCHIZOID POSITION, early in the oral stage. Internal and external reality are not yet properly differentiated, introjection and projection meaning that the child confuses its own actions with those of objects in the world, a failure of ego differentiation. Objects therefore acquire by projection the ego's emotions, becoming perhaps loving or hating, and their properties are also introjected within the mind. Normal transition to the depressive position is helped by a mother who is neither confusing nor contradictory (the GOOD ENOUGH MOTHER in the jargon), and allows the child to develop a sense of object realities, and to dissipate the hatred developed in the paranoid-schizoid position. If

his process fails then emotions are not projected onto external objects ut back onto the child itself, resulting in NARCISSISM, or self-love, nd the development of either MEGALOMANIA (grandiose thinking) if ccompanied by positive emotions, or PARANOIA if accompanied by egative emotions.

This chapter should have made it clear that at present no complete escription of schizophrenia is possible; such a description cannot be ouched solely in terms of abnormal neurochemistry, although that nust be a part of it, but must also explain the especial mental life, nd the early infantile experiences of the schizophrenic, so that an ntegrated picture can help make possible adequate and humane reatments.