

## Psychology of Dysfunctional Behaviour

### *SCHIZOPHRENIA*

Schizophrenia is one of the most serious psychiatric disorders. It is classified as a **psychotic disorder characterised by severe symptoms (that many would refer to as “abnormalities”) in the areas of judgement, emotions, perceptions and behaviour.** The name itself implies some kind of split: not, as popular opinion would suggest, a split personality, but a split between the mind and reality. It was the psychiatrist Bleuler who first labelled the disorder schizophrenia, deriving it from two Greek words meaning split (schizo-) and mind (-phrenia). The schizophrenic appears to retreat from reality into his or her own private world.

DSM-III-R identifies 3 major subtypes of schizophrenia:

1. **Catatonic schizophrenia:** disturbances of movement, usually either hyperactivity or inability to move at all.
2. **Paranoid schizophrenia:** typically suffer from delusions that people are out to get them or grandiose delusions that they are extremely important and influential people (e.g. Napoleon).
3. **Disorganised schizophrenia:** formerly called hebephrenic schizophrenics, these show disturbed behaviour that seem very inappropriate for the situation they are in and they are often socially withdrawn. This comes closest to ideas of true "madness".

### *Symptoms of schizophrenia*

Depends what you read and the date of the thing you are reading! In addition, not all schizophrenics exhibit the same symptoms. Nevertheless, the symptoms of schizophrenia affect all areas of mental life. Some examples:

### **EARLY THEORISTS**

**Kraepelin** in the late 19th century suggested "the weakening of judgement, of mental activity and of creative ability .... loss of energy .... and all remaining morbid symptoms, especially hallucinations and delusions would be regarded as more secondary accompanying phenomena". He first labelled it as dementia praecox which means premature mental degeneration. In the early 1900s, Bleuler believed, unlike Kraepelin, that schizophrenia did not always begin early in life. He distinguished four important features of schizophrenia, known as the four As: disturbances of **affect**, **ambivalence** in feelings, irrational mental **associations** and **autism** (self-absorbed withdrawal). Since these early theorists the symptomatology of schizophrenia has not really become any simpler and there is still a great deal of debate regarding the unitary nature of the disorder: is it a single disorder or is there really more than one type of disorder that should not come under the one heading of schizophrenia?

## LATER THEORISTS

### *Schneider's first rank symptoms:*

- Audible thoughts
- Voices discuss patient AND/OR Voices comment on patient's activity
- Thought invention AND/OR Thought withdrawal AND/OR Thought broadcasts
- Made feelings AND/OR Made impulses AND/OR Made volition

At one time, psychiatrists used these first rank symptoms according to Schneider's rule: *one first rank symptom indicates schizophrenia*. However, a World Health Organisation international study found that not all schizophrenics have a first rank symptom and also found that people who were not schizophrenics did have a first rank symptom (e.g. depressives and neurotics). Because of this, other symptoms were sought and other classifications were developed.

**Crow** (1980, 1985) suggests there are two types of schizophrenia, which he rather unimaginatively called **Type I** and **Type II** schizophrenia. There is a great deal of debate regarding whether these two types are both manifestations of the same disorder or whether there are, in fact, two different disorders. Do schizophrenics start with Type I and eventually go on to develop Type II or can they go straight into one or the other? Crow suggested that the two types of schizophrenia had two different pathologies (one biochemical, one structural - see Table 1 below) but felt that one could lead to the other. Type I, he suggested, does respond well to drugs, Type II does not. Perhaps this means that Type II is more severe and that people are less likely to be able to control Type II.

**Table 1: Crow's Type I & Type II schizophrenia**

	Type I	Type II
<i>Characteristic Symptoms</i>	Hallucinations, delusions, thought disorder (positive symptoms)	Affective flattening, poverty of speech, loss of drive (negative symptoms)
<i>Response to neuroleptics (schizophrenia drugs)</i>	Good	Poor
<i>Outcome</i>	Reversible	Irreversible (?)
<i>Intellectual impairment</i>	Absent	Sometimes present
<i>Postulated Pathological Process (i.e. cause)</i>	Increased dopamine receptors in brain	Cell loss and structural changes in the brain

This distinction between Type I and Type II schizophrenia has been gaining favour among clinicians in recent years because this classification seems better able to predict the outcome and most effective treatment of the disorder than other classifications.

The symptoms have also been grouped into three categories: positive symptoms, negative symptoms and psychomotor symptoms (Comer, 1995).

- **Positive symptoms** seem to represent pathological excess, i.e. additions to the “normal” repertoire of behaviours. They include **delusions**<sup>1</sup>, **disorganised thinking and speech**<sup>2</sup>, **heightened perceptions and hallucinations**<sup>3</sup> and **inappropriate affect**<sup>4</sup> (emotion).
- **Negative symptoms** include poverty of speech<sup>5</sup>, blunted and flat affect<sup>6</sup>, disturbances in volition<sup>7</sup> and disturbed relationships with the external world<sup>8</sup>.
- **Psychomotor symptoms** include loss of spontaneity in movement and the development of odd mannerisms (like Gerald’s hair-twisting in the video). Sometimes these disturbances take an extreme form so that the schizophrenic can no longer move at all (catatonia). Some show catatonic rigidity where they maintain a rigid position for hours and will not be moved, other show catatonic posturing where they assume awkward positions for long periods of time. Some subjects show a very odd condition called waxy flexibility where they maintain postures into which they have been placed by someone else (e.g. a nurse might raise the patient’s arm and they will maintain the position until moved again).

In general, the above cover all of the possible symptoms of schizophrenia. The disorder usually emerges between the late teens and mid thirties, although the course of the disorder may vary widely from person to person (APA, 1994). Many patients seem to go through three phases: **prodromal** (symptoms not yet prominent, but there is some deterioration of functioning including social withdrawal, acquisition of peculiar habits and difficulties in communication, thought and perception), **active** (more prominent

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<sup>1</sup> The most prominent delusions are being watched, commented on, delusions of persecution and delusions of grandeur. For a list of all the different types of delusional belief see Perrotto & Culin pg. 252. Some people with schizophrenia hold a single delusion that dominates their life and behaviour while others have many different delusions.

<sup>2</sup> Disorganised thinking and speech is a common feature of schizophrenia, shown typically by peculiar excesses of verbal expression which makes communication with others extremely difficult (like Gerald in the schizophrenia video). It may be that disorganised thinking and speech is one of the first signs of schizophrenia (Bilder et al, 1991; Harvey, 1991).

<sup>3</sup> Heightened sensitivity to sound and light is often reported by schizophrenics and has been supported in experimental studies in the laboratory (Cornblatt & Keilp, 1994). Perceptions that occur in the absence of external stimuli (hallucinations) are the most severe disturbance found in schizophrenia. They mostly take the form of auditory hallucinations where schizophrenics hear voices commenting on them or telling them what to do (Meuser et al, 1990). These voices can be so severe that the schizophrenic cannot get to sleep. Hallucinations involving the other senses are also experienced, e.g. tactile (tingling, burning), somatic (organs shifting inside the body, snake crawling inside the stomach), visual (seeing things or people that are not there).

<sup>4</sup> Many schizophrenics show emotions that are not appropriate to the situation. They might laugh when being told terrible news or cry when they are in a situation that should make them happy.

<sup>5</sup> Schizophrenics often display alogia (decreased command and productivity of speech: brief and meaningless replies) and some show blocking (thoughts disappear from memory so that they cannot complete sentences).

<sup>6</sup> Many show less anger, sadness, joy etc. much less than other people. Some show almost no emotion at all (Wechsler, 1972).

<sup>7</sup> Volition is voluntary actions and many schizophrenics feel drained of energy and interest in normal goals; they are unable to complete a course of action. This is particularly common in schizophrenics who have had the disorder for many years, perhaps supporting Crow’s notion of Type II schizophrenia being a continuation of Type I. Eventually, daily activities such as eating become impossible.

<sup>8</sup> Schizophrenics often become obsessed with their own internal world and can no longer interact with the external world. One study found that 75% of schizophrenics were less knowledgeable about everyday social issues than were people with other psychological disorders (Cutting & Murphy, 1990).

schizophrenia symptoms perhaps triggered by some stress) and *residual* (return to prodromal level of functioning but with deterioration in emotions and volition). Each of these phases can last for days or years. The actual diagnosis of schizophrenia is not easy: for example, people with schizophrenia often exhibit severe mood changes and people suffering from bipolar disorder may have distorted perceptions and bizarre cognitive experiences. The difficulty is telling different disorders apart. DSMIV suggests schizophrenia should be diagnosed when:

1. The person has shown continuous signs of schizophrenia for 6 months or more. For at least one month the person should show two or more of the major symptoms of the disorder.
2. The person has deteriorated from a previous level of functioning in such areas as work and self-care.
3. A depressive or manic episode was brief in comparison with the duration of psychotic symptoms.
4. The symptoms are not due to substance abuse or a medical condition that could cause similar symptoms.

***Prevalence: what is the risk of developing schizophrenia?***

The risk of developing schizophrenia is called the **lifetime morbid risk** and it is based on the prevalence of the disorder in the population. The table below shows the lifetime morbid risk of developing schizophrenia, but note that it shows nothing about the pattern or distribution of schizophrenia in the general population. For example, schizophrenia seems to have a higher prevalence among poorer people (Perrotto & Culkin, 1993), suggesting that socio-economic factors may play a role in the aetiology of the disorder.

***Table 2: Prevalence of schizophrenia***

Category	% chance
General population	0.85
With one parent schizophrenic	13.90
With 2 parents schizophrenic	46.30
With mz twin schizophrenic	47.40
With dz twin schizophrenic	15.00
With sibling schizophrenic	10.20
Parents of schizophrenic children	4.40
With Uncles, Aunts schizophrenic	3.60
With nephews, nieces schizophrenic	2.80
Grandchildren of schizophrenics	3.50
1st cousins of schizophrenics	3.50

In the population at large, the prevalence rate of schizophrenia is around 1% and as shown in the table above, it seems that the closer your kinship to a schizophrenic, the

more likely you are to be a schizophrenic yourself. Does that suggest that schizophrenia is inherited? What is it that causes schizophrenia? Can we suggest that it is caused by genetic factors or is some other factor or combination of factors responsible?

### *Causes of Schizophrenia: the theories.*

There are a number of methods that have been used to try to discover the causes of schizophrenia: the main methods are behaviour genetics (using family studies, twin studies and adoption studies), correlational research (prospective and retrospective), experiments (usually only used in schizophrenia to investigate the effectiveness of treatments) and case studies. There will be a separate handout covering these methods, but bear in mind that many will have been used to develop the following theories regarding the causes of schizophrenia. More information on these methods can also be found in Perrotto & Culkin, pg. 19-25.

#### *[A] Genetic*

Genetic researchers believe that some people inherit a biological predisposition to schizophrenia. This viewpoint has been supported by family, twin and adoption studies where the basic conclusion is that there does appear to be some evidence for a genetic component to schizophrenia (e.g. Shields, 1976; Heston, 1966; Rosenthal et al, 1971). Family studies have investigated the relatives of schizophrenics to see what the incidence of schizophrenia is in close and not so close family members. It seems that the more closely related the relatives are the more likely they are to have schizophrenia (Kendler et al, 1994; see Table 2 above). This suggests some kind of family basis for schizophrenia but this does not have to be genetic: it could be that certain patterns of behaviour within a family are encouraged or copied by many members of the family. Family members are exposed, generally, to similar environments so you cannot separate out nature from nurture here. Twin studies may be more enlightening here, but also suffer from the same nature/nurture problem.

If both members of a pair of twins have a particular trait (like schizophrenia) they are said to be concordant for that trait. For a trait that is transmitted genetically identical twins will show a higher concordance rate than non-identical twins. It does seem that there is a higher concordance rate for schizophrenia in identical than in non-identical twins (e.g. Gottesman & Shields, 1982). You could, of course, argue that identical twins will be even more likely than non-identical twins to experience similar upbringings, so some researchers have investigated concordance in twins (and siblings) who have been adopted into separate families at birth.

These studies do seem to point to a genetic basis for schizophrenia (e.g. Gottesman, 1991; Kety, 1974). For details of the Kety study see Comer, pg. 535. However, most researchers would argue that the development of schizophrenia relies on an interaction with the environment too. It seems that a degree of vulnerability to exhibit schizophrenic symptoms might be the factor inherited, and then the rest depends on your environment. Provided your experiences throughout your life are stable and

untroubled then you will not necessarily show schizophrenic symptoms at all. This aspect that is inherited is called a "**predisposition**": you might be genetically set up to get the disease, but it will not necessarily develop. It is not possible, therefore, to claim that schizophrenia is a purely genetic disorder.

### **[B] Biochemical**

It appears that many of the symptoms of schizophrenia are very similar to those shown by people taking hallucinogenic drugs, such as LSD. Therefore, a number of researchers suggested that schizophrenia could be caused by some kind of imbalance in the chemicals in the brain. One neurotransmitter, **dopamine**, seems to be very active in the influence of drugs such as LSD. This led Iversen & Iversen (1975) to argue that schizophrenia might occur because the levels of dopamine in the brain are too high. This is the so-called "**dopamine hypothesis**". What evidence is there to support the dopamine hypothesis?

1. **Phenothiazine** (a drug) is often effective in treating schizophrenics and it appears to work by blocking the dopamine receptor sites in the brain, so that dopamine cannot be used as a neurotransmitter. This means that for someone taking phenothiazine there will be greatly reduced amounts of dopamine in the brain. It is argued that excess dopamine is a potential cause of schizophrenia, if reducing it reduces schizophrenic symptoms.
2. People who suffer from **Parkinson's disease** show stiffness and muscle tremors which can be reduced, in the short term, by using a drug called **L-dopa**. L-dopa increases the amount of dopamine in the brain. It has been noticed that schizophrenics who have been taking phenothiazine for a long time and hence have greatly depleted levels of dopamine in the brain, start to show tremors and muscle stiffness like Parkinson's patients.
3. Post-mortem studies of schizophrenic patients have shown that their brains contain extremely high levels of dopamine and an excess of dopamine receptors (e.g. Owen et al, 1987).

The idea that an excess of dopamine causes schizophrenia is an appealing hypothesis, partly because dopamine-receiving neurons have been found to play an active role in guiding and sustaining attention (Cohen et al, 1988). People whose attention mechanisms are impaired might be expected to show abnormalities of attention, perception and thought that characterise schizophrenia.

Generally, though, the evidence for the dopamine hypothesis is pretty inconclusive. Some studies (e.g. Gelder, 1989) suggest that schizophrenic brains do not contain overly high levels of dopamine. Also, there is no way of telling whether schizophrenia is caused by high levels of dopamine, or whether a **result** of schizophrenia is an increase in the level of dopamine. Also, recent research into the most effective schizophrenia drug Clozapine have suggested that another neurotransmitter, serotonin, is important in schizophrenia (Owen et al, 1993).

Some recent research (Frith, 1988) suggests that there may be different brain dysfunctions, depending on the kind of schizophrenia the patient has. This suggests that there are two types of schizophrenias (Crow's Type I & Type II). Type I is, Frith claims, caused by an excess of dopamine, whilst Type II is caused by **structural degeneration** in the brain's tissues. This theory is still in its early stages, but could explain why *some* schizophrenic brains do not show high levels of dopamine. Researchers have found that many schizophrenics have enlarged ventricles in the brain (brain cavities that contain cerebrospinal fluid), in particular on the left side (e.g. Hyde et al, 1991; Losonczy et al, 1986). This has been found using CAT scans or MRI scans on live schizophrenic patients. Most of these patients show few positive and many negative symptoms of schizophrenia. However, it should be noted that many people who show these biochemical and structural abnormalities do not go on to develop schizophrenia and generally the evidence for biochemical theories of schizophrenia remain equivocal. This could be because biological factors merely provide the conditions for schizophrenia to develop and it is psychological factors that provide the trigger for the disorder to proceed.

### ***[C] Psychodynamic***

Freud believed that schizophrenia involves a two-part psychological process:

1. Regression to pre-ego stage
2. Restitutive efforts to re-establish ego control

He believed that it stems from a basic conflict between a person's self-gratifying impulses and the demands of the real world. When the real world is difficult, stressful, anxiety-producing then the person will regress to an earlier period in their functioning, even to the point of infancy. When they reach this stage, the schizophrenic will then try to re-establish ego control and try to interact with reality (e.g. auditory hallucinations may represent an unconscious attempt to substitute for a lost sense of reality). Of course, as with a great deal of Freudian theory, these views have almost no empirical support.

### ***[D] Behavioural***

Operant conditioning has been used to explain schizophrenia (e.g. Liberman, 1982). The idea is that most people learn from their environment to respond to social cues. When the response is socially acceptable then it will be reinforced. Some people, however, do not receive these reinforcements and they will stop attending to these social cues and focus instead on other, often irrelevant cues. As they do this more and more their behaviour will become more and more bizarre which means that their behaviour will eventually receive a lot of attention: this attention will act as a reinforcer for the behaviour. There has been some support for this because it has been shown that schizophrenics can learn appropriate responses if they are rewarded with cigarettes, food, attention etc. (e.g. Belcher, 1988). However, the behavioural explanation is

usually only considered to be, at best, a partial explanation of schizophrenia (Comer, 1995)

### *[E] Social*

Some studies (e.g. Srole et al, 1962) have suggested that schizophrenia is not evenly distributed throughout the population and that the lower socio-economic status you investigate then the more likely you are to find schizophrenics. Clark (1948) showed that low status occupations have much higher rates of schizophrenia than high status. Kohn (1973) suggests that this could be something to do with personal strategies for coping with stress. He argues that working class people emphasise conformity more than middle class people and that this might mean that they have fewer personal resources when faced with stresses. Of course, these ideas have caused a great deal of controversy. There is a great deal of argument about whether schizophrenia is the cause of social disadvantage (the **social drift hypothesis**) or whether schizophrenia is the result of social disadvantage (the **social causation hypothesis**).

### *[F] Existential Psychiatry*

This approach represents rejection of a medical model of mental disorder and a rejection of the notion of schizophrenia as a disease at all. Instead, within this model, schizophrenia is seen as simply behaviour which doesn't conform to the dominant model of social reality (e.g. **Laing**, 1959). More details about Laing's ideas can be found in Gross (1992; pg. 951-952).

Laing claims that schizophrenics *do* make sense if you can relate to their experience of existence (hence the existential approach). He claimed that unless we understand how the schizophrenic views the world we cannot possibly hope to reach them in order to treat them. The problem in schizophrenia, according to Laing, is that the person has a "**divided self**". By that he did not mean that the schizophrenic has a split personality, but that they had developed a false self in order to cope with the impossible demands of the world and the conflicting demands of others they were in contact with; this false self had become alienated from the real self so that the schizophrenic feels unreal and loses contact with reality. This split from reality causes the behaviours that we label "psychosis".

So Laing rejected the idea that there could be an objective psychiatric diagnosis of schizophrenia: he claims that we need to address this notion of labelling someone as mentally disordered and instead of seeking an objective diagnosis, look to the assumptions that are made about patients by the psychiatric profession and look at the nature of interactions within the family that may have led to this "schizophrenic" label being applied. Overall, Laing's work on schizophrenia was concerned with describing the structure and meaning of experience and challenging the conventional border between madness and sanity.

## *[G] Family Influences*

The genetic theory about schizophrenia suggests that it runs in families. Of course, just because it runs in families does not mean that it is necessarily genetically inherited: it could be that there is something amiss with the way the family operates. **Bateson** (1956) suggested that in some families schizophrenia can be induced by putting the individual in intolerable situations. Bateson called these situations "**double-binds**". A double bind is where a person involved in a sequence of interactions becomes trapped between two equally unacceptable sets of social expectations or demands: whatever they chose to do they cannot win. In some families this double bind is imposed repeatedly on one member of the family, who might develop schizophrenia as a result. An example of a double bind might be a situation such as this:

*Parent to child:* "Don't hit your brother or I will punish you"

*Parent to child:* "You are so weak, never standing up for yourself and letting your brother bully you all the time"

*Parent to child:* "Don't keep running away from your brother like that, stay and play with him"

In this situation the child does not know what to do, she cannot win. She is told not to hit her brother or she'll be punished and then she is told that she is weak for not standing up to him. Finally she is not allowed to escape from the situation. She is trying to cope with a situation that is placing too many demands upon her and she cannot deal with it, no matter what she does. If this situation continues and particularly if other family members do the same thing, then the child could well become schizophrenic. Bateson called such families **schizophrenogenic**: they have disturbed patterns of interaction that makes them particularly liable to induce schizophrenia in their members. Lidz (1975) supported this notion and said that there are two kinds of schizophrenogenic families: those where the family is divided between two parents who are conducting a power game (**schismatic families**) and those that are dominated by one family member who ignores the emotional needs of all other members of the family (**skewed families**).

## *[H] Cognitive*

This is one of the newest explanations of schizophrenia and it incorporates the biological view. It suggests that the schizophrenic will have some biological abnormality which will lead to strange sensory experiences. Then, the schizophrenic will attempt to understand and explain their unusual experiences and this will lead to the further features of schizophrenia emerging. So, according to Comer (1995) the schizophrenia when first confronted by voices or visions will turn to relatives, friends etc. to help them understand what is happening. When these people deny the existence of these visions or voices that the schizophrenic knows they are experiencing then they will come to believe that their friends and relatives are trying to hide the truth from them and they will eventually reject all feedback from others. They will eventually

believe that they are being manipulated or persecuted (Garety, 1991). This theory reflects the more general trend to see schizophrenia as a multiply determined disorder.

### ***[II] Vulnerability model of schizophrenia***

Modern theorists tend to argue that schizophrenia is not caused by any one factor, rather it is caused by a combination of factors, including genetic, family, biochemical and social. In the vulnerability model no one single factor is seen as causing schizophrenia: it may be that the individual has a genetic predisposition which makes them particularly vulnerable to social, family and interpersonal stressors. If all goes well, the individual may be able to cope without any problems ... but they are vulnerable. If anything goes wrong, if they are faced with any kind of excessive stress, they will be unable to cope and schizophrenia will develop. Strauss and Carpenter (1981) argue that this kind of model is the best fit to all the different research findings concerning schizophrenia.

### ***Treatments for Schizophrenia***

According to Comer (1995) “The symptoms of schizophrenia might seem by their very nature to defy treatment. What possible help can there be for people whose thoughts and perceptions are so profoundly confused and distorted?”. There is no doubt that schizophrenia is not always treatable and when it is, a number of different treatments are often required. This is one of the reasons that there are a number of alternatives suggested for the treatment of schizophrenia. The major and most widely used treatments (and probably the most successful) are those involving antipsychotic drugs.

### ***Biological Treatments***

#### **Description of Treatment**

Drug treatment is obviously based on a biological model of schizophrenia, assuming at least a partial biochemical cause for schizophrenia. The antipsychotic drugs revolutionised the treatment of schizophrenia in the 1950s. Previously the only way to treat schizophrenia had been to institutionalise the patient, but the discovery of effective drugs to control schizophrenic symptoms meant that institutionalisation was very often not necessary. The antipsychotic drugs called phenothiazines (which are antihistamine drugs) were the widest used drugs. These drugs control the symptoms of schizophrenia by reducing the brain’s dopamine activity (see dopamine hypothesis above) and they appear more effective against the positive symptoms than the negative symptoms.

The most widely used of these is chlorpromazine. It was first used on schizophrenia patients in 1952 by Delay & Deniker who reported a sharp reduction in the symptoms displayed by their patients. It was first marketed in the USA in 1954 under the trade name Thorazine. These drugs not only reduce schizophrenic symptoms but can improve attention and information processing (Silverman et al, 1987). Many other drugs have been developed since the discovery of chlorpromazine and they are called, collectively, neuroleptics because they often produce effects similar to neurological diseases.

A drug introduced more recently is Clozapine (trade name Clorazil) has been effective in patients who have not responded to the phenothiazines. Clozapine acts in the same way as chlorpromazine, in that it blocks the action of a neurotransmitter, but rather than acting on dopamine receptors it blocks the action of serotonin and norepinephrine (Kane et al, 1988).

### **Effectiveness of treatment**

Antipsychotic drugs seem to reduce schizophrenic symptoms in many patients (Strange, 1992) and they appear to be the single most effective treatment for hospitalised patients (May et al, 1981). If schizophrenics stop taking the drug too soon then symptoms do return (Davis et al, 1983). However, these treatments do seem only to alleviate the positive symptoms of schizophrenia and have little effect on the negative symptoms (Leff, 1992). Also, there are some side effects.

For the phenothiazines, their dopamine blocking effects is responsible for their effectiveness but it is also responsible for many of their side effects. Common movement side effects resemble symptoms of Parkinson's disease (tremors, spasms and an inability to sit or stand still). There is also a side effect called Tardive dyskinesia which is a movement disorder characterised by involuntary facial movements, like grimacing or chewing as well as disruptions in breathing. These latter effects are problematic for around 20% of patients (Yassa et al, 1990).

Clozapine has been found to be even more effective than the phenothiazines, helping approximately 80-85% of schizophrenics as compared to the 60-75% helped by phenothiazine (Kane, 1992). Also, it causes less side effects because it does not block as many dopamine receptors (Chengappa et al, 1994). In addition, Clozapine does seem effective in reducing some of the negative symptoms (Breier et al, 1994). However, it has side effects of its own! There is a 1-2% chance of getting agranulocytosis (life-threatening drop in white blood cells), so schizophrenics taking this drug undergo regular blood tests. Patients may also experience dizziness, drowsiness, excessive weight gain or occasionally seizures. Despite all these possible side effects, drug treatments offer the most successful treatment for schizophrenics that is currently available and many are helped by them.

### ***Psychological Treatments***

- ***Psychotherapy***

There are a number of different kinds of psychotherapy and there is certainly not room here to cover all of them. The main kinds are **insight therapy** (e.g. psychodynamic, cognitive, humanistic), **social therapy** and **family therapy**.

Studies into insight therapy suggest that therapist with most experience of schizophrenia are those most likely to have successful outcomes (Karon, 1988) as are those who play a more active role, setting limits, making suggestions, challenging and guiding (Whitehorn & Betz, 1975). This suggests that cognitive

therapy (which is more active) may be more successful than humanistic therapy (which is more passive). However Torrey (1988) suggests that insight therapies in general are unlikely to work for schizophrenia because they are “analogous to directing a flood into a town already ravaged by a tornado”. Basically, the suggestion is that insight therapy may overstimulate or distress the already distressed patient.

**Social therapy** involves making practical advice and life adjustment as the central focus of treatment. Therapy will mainly be directed to problem-solving, decision-making and the development of social skills. Therapists might also help patients find jobs and housing. It seems that social therapy is successful at keeping patients out of hospital (Hogarty et al, 1986), but it is successful mainly in conjunction with drug treatment.

**Family therapy** involves helping families of recovered schizophrenics to cope with the schizophrenic and try to prevent relapse. It does seem that released schizophrenics who have families with high levels of expressed emotion (i.e. criticise a great deal, emotional overinvolvement) are more likely to relapse than those who return to less emotional relatives (Fox, 1992). Family members are often greatly affected by the behaviour of the schizophrenic (Creer & Wing, 1974), being disturbed by their socially embarrassing behaviour and not knowing how to cope with some of the more bizarre behaviours schizophrenics produce. This is why clinicians often involve the whole family in the treatment of schizophrenia. The therapy provides family members with practical advice, emotional support and helps them to become more realistic in their expectations and more tolerant of the schizophrenic behaviour. This approach often succeeds in improving communication and reducing tension within the family and so help relapse rates reduce (Zastowny et al, 1992).

- ***Behaviour Therapy***

Although there is no compelling behavioural explanation for schizophrenia, there have been some fairly successful attempts at adapting behaviour therapy for the treatment of schizophrenia. One of the first behavioural strategies for its treatment was token economy used in inpatient settings. This involves the patient earning rewards for exhibiting desirable behaviours and punishment for unacceptable behaviours. Token economy programmes have little effect on the primary symptoms but they can encourage improvements in the patient’s interpersonal behaviour (Paul & Lentz, 1977). Other behaviour change programmes have been developed (e.g. The Social and Independent Living Skills - SILS - programme, Wallace et al, 1980): these emphasise the development of specific skills but their use with schizophrenia has not been widely researched.

- ***Group & Milieu Therapy***

Group therapy has often been used in institutional settings as a treatment for schizophrenia but it seems to add little to drug therapy and may actually do more harm than good (May et al, 1984). Residential treatment programmes that

emphasise group involvement, patient responsibility and social interactions are called milieu therapy. This focuses on helping schizophrenics to acquire practical, everyday problem-solving skills and it can be beneficial, helping patients leave hospital quicker (Cumming & Cumming, 1962).

Hospitalisation of the schizophrenic is, obviously, sometime necessary and when this occurs a combination of many of the above treatments will be tried. An important alternative to institutionalisation (or, perhaps, subsequent to it) is care in the community. This is particularly important to consider with the closure of so many mental institutions in this country. It involves society taking responsibility for those with any kind of disorder and involves the patient taking some responsibility for their own treatment.

To manage in the community the schizophrenic needs ***medication, psychotherapy, help in handling daily pressures and responsibilities, guidance on making decisions, training in social skills, residential supervision and vocational counselling and training***. According to research, those patients who live in a community who systematically address these needs make greater progress than those living in other communities (Hogarty, 1993). This suggests that society needs to take care of those who need it and be active in this if successful care in the community is wanted. The consequences of inadequate care in the community is that all responsibility either falls on the family or on the schizophrenic him/herself. This could lead to too much strain being placed on the family or, in the absence of family help, the schizophrenic could end up homeless and basically helpless. Many of these schizophrenics report feeling relieved when they are able to return to hospital (Drake & Wallach, 1992). If care in the community is of a high standard though it can be extremely successful and is today a major aspect of treatment for recovering schizophrenics (Lieberman, 1994). The question is, has this country got it right as yet?

The best kind of treatment for the schizophrenic involves a combination of biological and psychological treatments, preferably in a community setting even if initially the schizophrenic has to be hospitalised to establish treatment. However, there is still a long way to go: not least regarding attitudes towards schizophrenia. There is a great deal of ignorance about the disorder and ignorance in this case leads to fear. Most people consider the schizophrenic to be violent, unpredictable and basically scary! Education is needed if we are ever to manage care in the community.