16.1 Schizophrenia – symptoms and diagnosis

BACKGROUND

Some studies indicate that there is approximately a 1% life time risk of developing schizophrenia. Kraepelin (1902) described the symptoms of 'dementia praecox' (senility of youth) as being delusions, attention deficits, and bizarre motor activity, due to a form of mental deterioration that began in youth. Bleuler (1911) observed that deterioration did not continue and often began after adolescence, and so introduced the term 'schizophrenia' (split mind) to describe how psychological functions had lost their unity.

DIAGNOSIS

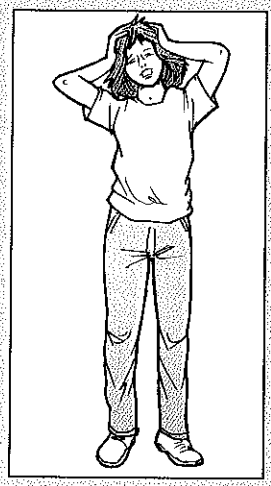
The DSM IV diagnostic criteria are:

1 **Two** or more of the following symptoms present for a significant amount of time in a one month period:
- **Hallucinations** (if there are extensive auditory hallucinations of voices, then no other symptoms have to be present)
- **Delusions** (if these are very bizarre, then no other symptoms have to be present)
- **Disorganised speech**, e.g. incoherent
- **Catatonic or disorganised behaviour**, e.g. repetitive movements or gestures
- **Negative symptoms**, e.g. emotional blunting

2 Disturbance must last for 6 months (including 1 month of the above symptoms).

3 The symptoms must have produced a marked deterioration in functioning at work, in social relations, and in self care (axis 5 of the DSM IV).

SYMPTOMS



EMOTIONAL

Emotions can be either:
- flat, unresponsive and insensitive, or
- inappropriate to the situation and changeable

BEHAVIOURAL

Somatic disturbance, e.g.:
- psychomotor agitation – fixed, repetitive gestures
- catatonic stupor – keeping the same position for long periods of time

PERCEPTUAL

- Auditory hallucinations, usually voices commenting upon behaviour and thoughts in the third person, are heard.
- Visual hallucinations, such as size, space, and colour distortions occur.

COGNITIVE

Disruption occurs to:
- **thought processes** – schizophrenics show **cognitive distractibility** (they are unable to maintain a consistent train of thought); **attentional deficits** (focusing on irrelevant stimuli); and **thought passivity** (where they think that others block, insert or withdraw the thoughts in their head).
- **thought content** – includes delusions, e.g. of persecution, control, or grandeur.

SUBTYPES OF SCHIZOPHRENIA

The DSM IV lists five sub-categories of schizophrenia, because of the huge variety of symptoms shown.

DISORGANISED SCHIZOPHRENIA

Symptoms mostly involve:
- **incoherent** thoughts and speech
- **bizarre** delusions and hallucinations
- **inappropriate** emotions and behaviour

UNDIFFERENTIATED SCHIZOPHRENIA

The classification for those whose symptoms are not classifiable under any of the other subtypes. This is the least useful of the diagnostic classifications.

CATATONIC SCHIZOPHRENIA

Involves alternating between:
- catatonic **stupor** and **negativism**, and
- catatonic **excitement** – prolonged, frenzied, even violent behaviour

PARANOID SCHIZOPHRENIA

Involves organised and complex delusions (often of persecution), mostly auditory hallucinations, and relatively few other symptoms.

RESIDUAL SCHIZOPHRENIA

Involves the gradual development of many minor problems, e.g. unusual behaviour, social withdrawal, emotional blunting, and apathy.

ALTERNATIVE TYPOLOGIES

- **Type 1 schizophrenia** – is characterised by positive symptoms, e.g. hallucinations and delusions
- **Type 2 schizophrenia** – is characterised by negative symptoms, e.g. emotional blunting and avolition

16.2 Explanatory theories of schizophrenia

BIOLOGICAL THEORIES

GENETIC CAUSES

Family studies – Children of two schizophrenic biological parents are around 46% likely to develop the disorder. These studies do not rule out environmental learning though.

Twin studies – Studies from many countries have produced different estimates, but Gottesman (1991) suggests that monozygotic identical twins (who have the same genes) have significantly higher concordance rates (48%) for schizophrenia than dizygotic non-identical twins (17%). Concordance rates refer to whether **both** twins develop the disorder.
However, identical twins also share more similar environments.

Adoption studies – When adopted subjects' environments are matched, the rates of schizophrenia are higher for adoptive children with schizophrenic biological parents compared to adoptive children with non-schizophrenic biological parents (Kety *et al.*, 1975). Ideally, identical twins with schizophrenia, raised apart in different adoptive environments, would be the best evidence for genetic causes, but obviously these cases are extremely rare.

Genetic factors do not account 100% for schizophrenia, however. People probably inherit a genetic predisposition for schizophrenia, which **may** be triggered by environmental factors.

BIOCHEMICAL CAUSES

A very popular theory of schizophrenia was the dopamine hypothesis – that over-activity of the neurotransmitter dopamine in the synapses of the brain caused type 1 positive symptoms of schizophrenia.
Evidence for the hypothesis included the findings that:

- large doses of amphetamines (which increase dopamine activity) can create amphetamine psychosis, which closely resembles acute paranoid schizophrenia. Small doses can trigger symptoms in schizophrenics.
- anti-schizophrenic drugs like chlorpromazine work by blocking the post synaptic receptor sites of dopamine, thereby reducing its activity. If schizophrenics are given too much of these drugs, they develop symptoms similar to Parkinson's disease (caused by too little dopamine).
- post-mortems and Positron Emission Tomography scans have found higher amounts of dopamine and dopamine synaptic receptor sites.

However, the dopamine hypothesis is an over simplistic explanation, since new anti-schizophrenic drugs (e.g. clozapine) work by affecting other neurotransmitters, especially serotonin.

BRAIN STRUCTURAL CAUSES

Enlarged ventricles – research has found that these fluid filled cavities in the brain are larger in schizophrenics due to brain cell loss. Cell loss in the temporal lobes of the brain (responsible for cognitive and emotional functions) has been associated with negative symptoms. However, the evidence is correlational – enlarged ventricles may be a symptom not a cause, and non-schizophrenics can also show them.

Brain area activity – schizophrenics' brain scans do not show the usual prefrontal activation of the cortex when given problem solving tasks. Brain scanning can not yet predict the presence of schizophrenia.

PSYCHOLOGICAL/ ENVIRONMENTAL THEORIES

PSYCHOLOGICAL CAUSES

A variety of theories have sought to explain schizophrenia at the psychological level, including:
Psychoanalytic theory – Freud suggested that regression to a state of 'narcissism' in the early oral stage could be responsible, where there is no developed ego to test reality. Psychotic thought resembles the id's primary process thinking, and is untreatable through psychoanalysis because the narcissistic person has given up any attachment to the outside world (preventing transference, for example).

Existential theory – Psychiatrists, such as Laing, have proposed that people withdraw from reality as a normal response to the pressures of a mad world. Schizophrenia is a social and interpersonal experience which can be regarded as a potentially beneficial journey of self discovery.

Labelling theory – Scheff (1966) has argued that schizophrenia may be largely a social role that, once assigned by diagnosis, is conformed to and becomes a self-fulfilling prophecy. The internalisation of the schizophrenic role is strengthened by the reactions of other people and hospitalisation. Szasz has taken these ideas further to argue that schizophrenia is a myth created by society to control those who are different.

Cognitive theory – Frith (1979) proposes that disruption to an attentional filter mechanism could result in the thought disturbance of schizophrenia, as the sufferer is overloaded with sensory information. Studies on continuous performance and eye-tracking tasks indicate that schizophrenics do show more attentional problems than non-schizophrenics. Perhaps reduced short-term memory capacity could account for some schizophrenics' cognitive distractibility.

SOCIAL/ENVIRONMENTAL CAUSES

Social or environmental factors could act to trigger schizophrenia in those with a genetic predisposition.

Family stresses – Faulty interpersonal relationships in the families of schizophrenics have been found by Fromm Reichmann (who proposed the idea of the 'schizophrenogenic mother'); Bateson (who discovered ambivalent 'double bind' communication between schizophrenic children and their parents); and Lidz and Fleck (who described 'schism' and 'skew' in the families of schizophrenics).
However, the evidence is correlational – perhaps schizophrenics cause stress and disturbance in their families.

Environmental stresses – Some studies have found schizophrenia is 8 times more common in the lower socio-economic groups. However, this could be a cause (providing greater stress) or a result (of downward social drift) of schizophrenia.

Viruses – Many viruses, e.g. influenza have been proposed to trigger genetic causes of schizophrenia.