Schizophrenia, a splitting of the mind

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Dementia Praecox, the early term for schizophrenia was presented by Emil Kraepelin in 1898. Dementia Praecox included – dementia paranoids, catatonia and hebephrenia. Whilst these different entities are symptomatically very diverse, Kraepelin believed they shared a common core. Kraepelin noted several major symptoms in his patients, these included hallucinations, delusions, negativism, attentional difficulties, stereotyped behaviour and emotional dysfunction. Kraepelin focused on describing schizophrenia and made no attempt to categorise and explain what he saw.

Eugen Bleuler however tried to define the core of the disorder. Bleuler disagreed with Kraepelin on two points. Bleuler believed that the disorder didn’t necessary have an early onset and that the disorder didn’t necessarily lead to total dementia. Since he believed that the disorder didn’t lead to total dementia the term dementia praecox was no longer valid, so in 1908 Bleuler suggested a new term for the condition Schizophrenia. Bleuler had a great influence over the American concept of Schizophrenia. Whilst the European view of Schizophrenia remained relatively narrow.

The American view of schizophrenia broadened significantly during the 20th century, with 80% of patients in the New York State Psychiatric Institute being diagnosed with Schizophrenia in 1952. Adolf Myer argued that diagnostic categories where often to stringent and believed that a more flexible approach to defining Schizophrenia was necessary. Kansnin then found that some patients showed signs schizophrenia combined with symptoms from other disorders. The concept of schizophrenia was also

broadened by Hoch who believed that schizophrenia often disguises itself has other disorders. As a result a lot of people who would normally have been diagnosed with personality disorders or neurosis, where diagnosed has having schizophrenia. After the publication of DSM III the American definition moved away from the very broad definition of schizophrenia, to a more controlled approach that meant that less people are now wrongly diagnosed with schizophrenia.

The symptoms of schizophrenia cause suffers problems in several major areas these include: thought, perception, attention, motor behaviour and emotion. Many patients, who are diagnosed with schizophrenia, only have some of the symptoms. Unlike most disorders schizophrenia doesn’t have an essential symptom, which must be present for schizophrenia to be present. The symptoms of schizophrenia can be divided into two categories, positive and negative symptoms.

Positive symptoms include disorganised speech, hallucinations, delusions and bizarre behaviour. Disorganised speech also known has formal thought disorder. It refers to the problems that a patient has in organising speech in a manner the listener can understand. A patients speech can be made difficult because they are incoherent and whilst the patient makes repeated references to central ideas, the images and fragments of thought are not connected. Speech can also become disorganised through loose association or derailment, in which case the patient may have more success communicating to the listener but they have difficulty sticking to one subject. The disorganisation of thought seems more central to schizophrenia than the disorganisation of speech. Many schizophrenics are subject to

delusions, holding a belief that the rest of society would simply deem as false. Delusions generally take one of eight forms.

1. Somatic passivity – The patient believes they are the unwilling recipient of bodily sensations administered by an external force.

2. Thought insertion – The patient believes that thoughts that are not their own have been inserted to their head.

3. Thought broadcast – The patient believes their thoughts are being broadcast to others around them.

4. Thought withdrawal – The patient believes that some external force is stealing their thoughts.

5. Made feelings – They believe that some external force is making them feel a certain way, for example unhappy, when the patient believes they are not in fact unhappy.

6. Made volitional acts – When the patient makes a particular action for example pick up a cup, they believe they have no control over their movements.

7. Made impulses- Is when the patient believes they certain impulses are placed in their head by some external force.

8. Persecutory delusions – The patient believes they are the victim of a great plot against them.

Schizophrenics also often report that the world seems unreal in some way. The most dramatic distortions of perception are called hallucinations. There are many different types of hallucinations. Below there's a brief description of some types of auditory hallucinations.

1. Audible thoughts – The patient may hear a voice from above their head repeating some of their thoughts.

2. Voices arguing – The patient hears voices arguing, often about the patient themselves.

3. Voices commenting – The patient hears voices commenting on their actions, often in a negative way.

Negative symptoms consist of behavioural malfunctions such as avolition, alogia, anhedonia, and flat effect. Avolition is when the patient has a lack of interest or energy when it comes to routine activities such has personal hygiene. They spend a lot of time sitting around doing nothing. Alogia has several components, these include a reduction in the amount of speech, a degrading of the content of the speech and whilst the person is coherent they convey very little information when speaking. Anhedonia is the inability to experience pleasure. Flat affect is when virtually no stimulus can get an emotional response.

There are other symptoms of schizophrenia that do not fit into either positive to negative symptoms. One of these is catatonia, which is when the patient adopts strange seemly uncomfortable poses for long periods of time. Some schizophrenics report a sudden increase in their overall level of activity.

Some also have inappropriate affect. This is when the emotional response of the sufferer is out of context. For example they laugh upon hearing their father died.

We know people suffering from schizophrenia differ from normal people in the way they think, perceive, speak and imagine. But we don't know why some people become schizophrenic. Below I'm going to outline some of research people have done into the causes of schizophrenia. There has been a large amount of research done on the genetic influence of schizophrenia. These include family studies, twin studies and adoptee studies.

In 1987 McGuffin and Farmer found that the closer a person in genetically to a schizophrenic relative, the more at risk they are to becoming schizophrenic themselves. Therefore there is support for the idea that schizophrenia can be transmitted genetically. But relatives of schizophrenics share not only genes but also the same environment. Therefore we can't rule out the influence the environment can have on the chance of someone developing schizophrenia.

The chance of MZ twins both having schizophrenia is considerably higher than that for DZ twins. Whilst the concordance rates for MZ twins is high it is not a 100% therefore we can assume that whilst genetics has a part to play it is not the only factor in determining whether a persons gets schizophrenia. The study of children of schizophrenic mothers raised by adoptive parents from an early age has helped provide more conclusive information on the influence of genes in the development of schizophrenia. In 1966 Heston found that the adopted children of schizophrenic mothers where more likely to become schizophrenic than adopted children not of schizophrenic

mothers. All data so far suggests that genetic factors play a part in the development of schizophrenia. We cannot however conclude that schizophrenia is caused just by genetics.

Speculation that schizophrenia was caused by chemical factors emerged as soon has the disorder was identified. Kraepelin believed it to be caused by a chemical imbalance. Carl Jung believed that schizophrenia was caused by “ toxin X” a mystery chemical yet to be identified. If genetics play a part in the development of schizophrenia it makes sense to look in to the biochemical aspect of schizophrenia. The theory that schizophrenia is caused by the neurotransmitter dopamine is a popular theory based upon the range of drugs that are effective in treating schizophrenia. If we know or can at least hypothesises how a drug affects the brains biochemistry, we can make an educated guess at the process responsible for the disorder. Whilst the effects of a drug may provide a clue to the causes of a disorder it cannot logically be used to prove the causes of a disorder. Indirect support for the dopamine activity theory comes from research into amphetamines. Amphetamines can induce a state that closely resembles paranoid schizophrenia. Research suggests that we can be pretty sure that the psychosis inducing effects caused by amphetamines comes from the amphetamines effect on the brains dopamine. Research into levels of homovanillic acid (HVA) the major metabolite of dopamine suggests that the dopamine releasing neurons or the dopamine receptors are overactive. Other research in to the levels of dopamine activity in schizophrenics has suggested that and excess in dopamine activity may not be applicable to all schizophrenics. For instance some studies have shown that amphetamines do not worsen the symptoms

of all schizophrenics (Kornetsky, 1976). One study (Kammen et al, 1977) found that symptoms lessen after amphetamines have been administered. A correlation of the results of studies into amphetamines and schizophrenia, suggests that amphetamines worsen positive symptoms and lessen negative symptoms.

Biochemical research into the causes of schizophrenia has been one of discovery followed by failure to replicate. Methodological flaws plague research in this area, mean that many unrecognised factors could have affected brain chemistry. Therefore it would be sensible to remain cautious when looking at the dopamine activity theory.

The search for a brain abnormality as a cause for schizophrenia started as soon as the syndrome was identified. Until recently most of the research into brain abnormalities as been useless. But recent technological advances have meant that recently a number of interesting findings have been made. It is believed that some schizophrenics have observable brain pathology. The post-mortem analysis of the brains of schizophrenics consistently reveals brain abnormalities, although the specific problems reported vary from study to study (Weinberg et al, 1983). Recent studies using CAT scans and MRI have revealed that some schizophrenics especially males have enlarged ventricles (Andreasen et al, 1990).

So far I have looked at several different causes for schizophrenia. Data shows that general stress can often cause a relapse in a schizophrenic (Ventura & Nechterman, 1989).

A number of studies show a relationship between social class and the diagnosis of schizophrenia. The highest levels of schizophrenia are found in cities among the lower socio-economic classes. Some people believe that being in a low social class may cause schizophrenia. This is called the sociogenic hypothesis; it suggests that the degrading treatment the person receives from others, the lack of education may lead to severe stress and therefore schizophrenia. Another explanation for the large amount of schizophrenics in the low social classes is the social- selection theory. This suggests that a person will drift towards the lower social classes during the development of their psychosis.

Many theorists see the family relationship especially the relationship between mother and her son has very important in the development of schizophrenia. The term schizophrenogenic mother was coined for the dominant, cold and conflict-inducing mother that is said to produce schizophrenia. Another prominent theory is the double bind theory proposed by Bateson, 1956. This theory suggests that a person close to the patient makes contradictory demands of the patient. Whilst controlled studies looking at schizophrenogenic mothers and double bind theory have not yielded supporting evidence. These studies have shown that families of schizophrenics differ from normal families. Families of schizophrenics generally have a lack of communication between its members and there are often high levels of conflict. We cannot presume that these factors help cause the development of schizophrenia; they could be caused by having a young schizophrenic in the family. A series of studies have found that the family can have an important affect on patients after they leave the hospital.

Brown et al (1966) conducted a nine-month follow up study of a sample of schizophrenics that had left hospital to live with their families. Before the patient left the hospital the families where interviewed, and rated on there expressed emotion (EE). They where divided into families that showed a high level of EE towards the patient and those that showed low EE towards the patient. Nine months later it was found that 10% of the schizophrenics from the low EE families had returned to hospital, whilst 58% of the schizophrenics from high EE families had returned to hospital. This research has now been replicated many times (Vaughn & Leff, 1976; Leff, 1976; MacMillan et al, 1981; Koenigsberg & Hadley, 1986.). So whilst it is obvious that the behaviour of a family can have an effect on whether a patient is re-hospitalised.

It is extremely difficult to treat schizophrenia, where does one start to treat a disorder that has such a wide range of symptoms. The earliest treatments where radical and often harmed the patient. For instance Sakel (1938) used insulin to induce comas. At the time Sakel claimed that three quarters of the patients treated this way showed an improvement. Later findings by others suggests that insulin coma therapy presented a serious health risks to the patient including death. In 1935 Moniz introduced the prefrontal lobotomy. This involves destroying part of a patient’s brain. In his initial reports like Sakel he reported a high rate of success. Many patients that received psychosurgery did indeed calm down and could be discharged from hospital. But also many patients suffered from serious losses in their mental capacities, became dull and even died. Cerletti and Bini developed electro convulsive therapy (ECT) in 1938. ECT involves placing electrodes on a

subjects temples and applying a current of between 70 and 130 volts. This induces a seizure followed by unconsciousness. This resulted in the same outcome has psychosurgery.

Since the 1950s drugs have been used to treat schizophrenia. One of the most common drugs is phenothiazine an antihistamine. Laborit a French surgeon found that antihistamines reduce surgical shock, make patients drowsy and can reduce anxiety about the impeding operation. After this discovery phenothiazine was found to also have a calming effect on schizophrenics. It is thought to work by blocking the brains dopamine receptors. By 1970, 85% of all patients in US mental hospitals where receiving some sort of phenothiazine (e. g. chlorpromazine). In recent years two other drugs have been given to schizophrenics, butyrophenones and thixoanthenes. These seem to have the same effect as phenothiazines. All the drugs seem to reduce positive symptoms but have very little effect on negative symptoms. Phenothiazines are definitely not a cure for schizophrenia. Phenothiazines have serious side effects including dry mouth, blurred vision, grogginess and constipation. This often means it is difficult to get patients to stick to a course of medication. Phenothiazines can also cause what is known has extrapyramidal side effects. Side effects that come from dysfunctions of the nerve tracts that descend from the brain to the spinal motor neurons. These extrapyramidal side effects resemble neurological diseases such has Parkinson’s disease or akasthesia (the inability to remain still). Recently a new drug clozaphine seems to produce therapeutic effects in patients that do not respond to other drugs. It also appears has if clozphine works without blocking dopamine receptors and

doesn’t have extrapyramidal side effects. Unfortunately clozphine weakens the immune system, can produce seizures and is very expensive. Despite their short falls drugs are an indispensable source of treatment for schizophrenics and is more humane than ECT or psychosurgery.

Freud did not believe psychoanalysis would be useful in treating schizophrenia. A number of others have proposed adaptations of psychoanalysis to try on schizophrenics. An American psychiatrist Harry Sullivan tried using psychoanalysis on schizophrenics in 1923. He reported that his approach was generally a success. Unfortunately a long-term follow-up on the schizophrenics that received psychoanalytic treatment wasn’t positive. Stone hypothesised that gaining a psychoanalytical insight into their disorder actually made schizophrenics worse.

Earlier we looked at how the level of expressed emotion (EE) in a family can affect a schizophrenic’s condition. Since high levels of EE seem to increase a chance of a relapse it makes sense to try and reduce the levels of expressed emotion. This involves family sessions where the importance of taking medication was empathised and the family was introduced to ways of expressing positive and negative emotion in a constructive way. This treatment was compared with patients receiving therapy by themselves at a clinic. Over a two year period it has been found that the patients that received the family therapy where considerably less likely to experience complications after being discharged from hospital. This could have been because these patients where encouraged to take their medication regularly. But other studies have found that medication rarely halts a patient’s

deterioration. Therefore one can assume that EE orientated therapy is successful.

Overall I believe that schizophrenia is a complex and frightening disorder. Because the symptoms are so varied maybe schizophrenia is not one but many disorders that have been conveniently categorised under one heading. All the symptoms of schizophrenia seem to suggest to me that schizophrenia is caused by a malfunction to fear or anxiety caused by a persons view of themselves. We know that schizophrenia could possibly be caused partly by a faulty gene. If a person is put under stress they deal with it through one of the ego defence mechanisms. If a person with the schizophrenia gene is put under stress then there ego defence mechanism may malfunction or not work. We can guess that stress can trigger schizophrenia because of the EE studies that look at patient relapse rate. Also if we believe that the symptoms are a reflection of the cause it is also not unreasonable to think stress can trigger schizophrenia. Stress is a persons perceived inability to cope with a situation. Therefore they are blaming themselves for their failure to perform. Many schizophrenics experience hallucinations commenting on their actions. This could be caused because they have a problem with their self-image or self esteem caused by stress. If you imagine yourself very stressed maybe at the scene of an accident, you need to do something about the accident, but your finding it difficult to remember what you should do. To avoid panic some people break down complex actions like first aid at the scene of an accident into simpler instructions. For instance at the scene of an accident you would take deep breath then look for danger, check the airways of the injured etc. When you are in a stressful situation you comment on

yourself. Maybe schizophrenic hallucination is the same response caused involuntary by a brain malfunction.