Divided selves as nervous types

GORDON CLARIDGE *delivered the Hans Eysenck Lecture at the Annual Conference in Cardiff.*

WHEN I started to prepare my Hans Eysenck Memorial Lecture it suddenly occurred to me that it was more than 50 years since I first met Hans Eysenck. 'Met' is a slight exaggeration since the encounter was more one-sided: I was an undergraduate at University College London sitting listening to his lectures on personality. I recall that the experience was quite inspirational and, although I was not to know it at the time, it was eventually to set me on a career path which, in various ways and at various points, was much influenced by his ideas. What follows is a small tribute to that.

I have chosen as my topic a relatively modest part of Eysenck's extensive work: his writings on psychoticism and psychotic disorder. This is not with the intention of promoting the detail of Eysenck's theorising on the subject – indeed I have often been very critical of that (Claridge, 1981, 1983). My choice was more motivated by a wish to remind those interested in serious mental illness of some of the general principles of what he had to say, principles which have been largely lost in the current debate about the nature of psychotic disorder. Psychosis has also been the main focus of my own research over the past several decades and so I have a further special reason for acknowledging my debt to Eysenck for sowing the seeds of my interest in the topic.

The discussion here will be confined to schizophrenia because that is mostly where controversial debates about psychosis have occurred – and because it is a simpler way to present the arguments. However, all that follows applies equally well to other forms of psychotic illness. That at least is one point that I did agree with Eysenck about: his support for the notion of a unitary psychosis and a common trait of psychoticism (Eysenck, 1952; Eysenck & Eysenck, 1976).

The nature of serious mental illness has always been open to different interpretations, modern accounts of which routinely begin with mention of two eminent European psychiatrists: Emil Kraepelin (1856–1926) and Eugen Bleuler (1857–1939). Bleuler is well-known for coining the term 'schizophrenia', while Kraepelin had an even wider influence on psychiatry, in several ways. He introduced what still remains the basic framework of psychiatric classification and established the medical disease model as the paradigm for psychiatric illness. The latter, with regard to psychotic illness, has in recent times been cast as the 'broken brain' theory, proposing that conditions like schizophrenia are essentially neurological diseases akin to other degenerative disorders of the nervous system (Andreasen, 1984).

This medical model has come under regular attack. And here again we can identify two historically important figures who, like Kraepelin and Bleuler, were roughly contemporaries of each other: R.D. Laing (1927-89) and H.J. Eysenck (1916-97). Of the two, Laing was by far the better known, at least in society at large. His seminal book about schizophrenia, *The Divided Self* (Laing, 1960), became not just a statement for an alternative psychiatry; it was also adopted as the bible for a 1960s youth protest movement against the 'Establishment'. Eysenck (1960) was equally outspoken, but his voice was heard more within the corridors and lecture halls of academia. There, like Laing, he was certainly disliked by psychiatrists; but – and this is an important point to bear in mind for the later discussion here – he was

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also disliked by many psychologists, especially clinical psychologists persuaded by the message of *The Divided Self*.

Laing and Eysenck were of courses very different characters but it is instructive briefly to compare them a little further, in order to get some insight into the complexities of the issues we are trying to deconstruct here. The first point to make is that in a very important way they were both actually saying the same thing! Thus, in criticising the medical model they both argued for an inextricable connection between illness and health – that to understand the sick, one had to reach out to understand the healthy. But of course their proposed way of doing this was quite different. Eysenck's approach was statistical and biological; Laing's was social, psychoanalytical, and philosophical.

In its current form the debate about schizophrenia is usually represented as a simple contrast between two polarised views, disagreeing on the relevance of biology to its aetiology and treatment. On the one hand, there are those who emphasise psychological and psychosocial causes, who are interested in the subjective experience of the psychotic state, and who – among clinical psychologists – foster the use of cognitive behavioural methods of treatment. In contrast, others stress genetic causation of brain abnormalities for which drug treatments are the most appropriate therapy. This latter, so-called 'biomedical' model is somewhat oversimplified, but it brings me to the main theme of this article.

Attempts to conceptualise schizophrenia and other psychoses biologically have their roots in two relatively distinct theoretical traditions. One – on the personality side and forming the background to Eysenck's (1957) contribution to the field – has its origins in Kretschmer's (1925) dimensional description of personality, and in Pavlov's (1935) 'nervous type' formulation of temperament: that psychological and behavioural differences reflect naturally occurring variations in brain function. According to this perspective the starting-point of theory is *normal* personality, the abnormal (mental illness) being seen as an extension, exaggeration, or distortion of that.

The other theoretical tradition started at the illness end. Even the 'broken brain' model can accommodate a degree of dimensionality. This was articulated early on in the recognition of 'schizoid' as a mild variant of schizophrenia, an idea which, according to M. Bleuler (1978), was introduced into clinical discussions by his father around 1910. Modern equivalents of such dimensionality in the medical interpretation of schizophrenia are the notions of schizotypy (Meehl, 1962, 1990) and the schizophrenia spectrum.

Although superficially similar, these two – personality and medical – formulations of the continuity within psychotic disorder are actually quite different. Elsewhere (Claridge, 1997) I have tried to capture the distinction between quasi-dimensional and fully dimensional models of schizotypy. The former, it is argued, is predicated on the idea of schizotypy as a single-gene based CNS deficit, albeit of lesser degree than that found in full-blown schizophrenia, and sometimes compensated for in an illusion of normality. Fully dimensional theory, on the other hand, proposes that schizotypy is simply a multi-genic 'nervous type' personality dimension like any other, though occasionally doubling up as a predisposition to mental illness. As an analogy here we can compare trait anxiety and disorders of anxiety.

Another salient difference between the two models is their view on the possibility of a truly healthy form of schizotypy (McCreery & Claridge, 2002) – or what McCreery (1993) labelled 'happy schizotypy': logically impossible in the quasi model but an intrinsic, even defining, feature of the fully dimensional model.

The advantage of the fully dimensional model is that it can accommodate quite disparate outcomes for schizotypy. Thus it can reconcile the idea that the cognitive and emotional qualities to be found in highly schizotypal individuals can, it is true, predispose them to the devastation of serious mental illness: disorganised thought, threatening hallucinations, delusional beliefs, negative mood and disturbed affect. Yet it can also allow for the possibility that these same traits will lead to rewarding and adaptive psychic experiences and be associated with heightened creativity, non-threatening perceptual aberrations (e.g. out-of-the-body experiences), and paranormal beliefs and reported experiences.

What determines which of these fates befalls a particular individual? Table 1 makes what is hopefully an informed guess about some important variables. There is nothing particularly original about the suggestions: the table is merely meant to underline the fact that schizophrenia, like any other psychological disorder, results from a genetically determined disposition,

TABLE 1 High schizotypy, healthy or sick outcomes: possible influences

Genetic loading

Profile of schizotypal traits

- Unusual experiences (e.g. hallucinations) not sufficient…
- Negative (anhedonic) traits more disabling

Protective factors

- High intelligence
- Socially syntonic personality traits (e.g. social acceptability)
- Happy family
- Healthy context for deviant beliefs/ behaviour

Vulnerability for illness factors

- Absence of the above
- Early abuse and neglect
- Biological hazard (e.g. birth complications)
- Drug abuse

Chance factors!

interacting with family and social influences, and modified by other personality and cognitive factors.

Probably the most important determinant of a healthy or sick outcome is the presence or absence of anhedonic lack of emotional responsiveness. Although it might seem counterintuitive, the hallucinatory-type experiences that help to define the more positive symptoms of psychosis do not in themselves constitute a strong risk factor. They are only likely to do so when negative emotionality is also part of the profile. Thus, it is significant that healthy individuals reporting strong aberrant perceptual experiences (e.g. out-of-body experiences) rarely show high scores on measures of more negative (anhedonic) aspects (e.g. McCreery & Claridge, 2002). Supplementing this effect and perhaps partly a reflection of it are 'syntonic personality traits', e.g. socially acquired acceptance and therefore lack of anxiety about the experience of 'psychotic' perceptions and thoughts. It is said that Jean Paul Sartre, when he told his mother as a child that he heard voices, was reassured that it was nothing to worry about.

The inclusion of 'chance factors' perhaps also deserves further comment. This 'Darwin's nose' element in human experience and development is not, in my view, emphasised as much as it might be. Yet the course of a person's life – and of those around them – can be strongly influenced by apparently random events that suddenly become salient. This is true universally, of course, but can be poignantly so in the case of serious mental illnesses, such as schizophrenia, or extreme deviation on schizotypal, schizoid, or borderline traits. There, as many clinicians will attest, a chance encounter, say, with another caring human being can often make the difference between a life of fulfillment and one of misery. Accidents of birth could also be said to come into this same category, under 'protective factors' listed in the table. For example, a background of privilege or exposure to a particular phase in shifting social norms can profoundly shape the definition and consequences of deviance. Lewis Carroll and John Ruskin would hardly have survived as comfortably as they did in a different place, at a different time, and with a different heritage.

The crucial message to be taken from Table 1, however, concerns biology, and is twofold. First, that nervous system functioning plays a significant (and discoverable) role in the processes that lead to differing outcomes for those vulnerable to serious mental illness. And secondly, as I have tried to explain, admitting to that fact does not in any sense mean buying into a traditional medical disease model.

For those committed to neither side of the debate about schizophrenia, its construction as a psychobiological disorder will seem uncontroversial, almost self-evident. Yet in some quarters of cognitive clinical psychology there still seems some difficulty in throwing off the more constricting shackles of Laingian heritage and opening up to genuinely alternative views of schizophrenia: alternative, that is, to those of both Laing and biological psychiatry. Consider the following, from Richard Bentall's (2003) book, *Madness Explained*:

> *…Gordon Claridge's revised biomedical model – in which madness is regarded as the dysfunctional manifestation of an extreme variant of normal personality – seems threatened by the apparent association between madness and genius. (p.115)*

Surprisingly (given his involvement in definitive research on the structure of schizotypy: Bentall *et al*., 1989), Bentall has evidently misunderstood both dimensional models of schizophrenia and variants of the so-called 'biomedical' explanation. (It is of course precisely the connection between madness and genius that gives strength to the fully dimensional model described here). So it would seem that in their eagerness to persuade us that things biological are not relevant to their enterprise, cognitive clinical psychologists have rejected some ideas which, ironically, support their argument against 'broken brain' theory. A case, I would venture, of throwing out the brain in the brainwashing.

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DISCUSS AND DEBATE

How convincing is the evidence for a connection between creativity and madness?

Are the schizophrenias and bipolar affective disorder simply variants of a unitary psychosis?

How might cognitive clinical psychology benefit from consideration of a revised biomedical model of psychosis?

Have your say on these or other issues this article raises. E-mail 'Letters' on psychologist@bps.org.uk or contribute to our forum via www.thepsychologist.org.uk.

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