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## Measurement and classification in psychiatry

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### Why measure and classify

The formulation and development of medical disease concepts requires an interaction between two essential components. One is reliable recognition and labelling of a cluster of physical and/or psychological characteristics, regarded as undesirable because of the distress or disability that accompanies them. Sometimes a single characteristic is enough. The other is the testing of hypotheses concerning the relationship of these characteristics to damage and dysfunction in underlying biological systems (pathology) and to their causes (aetiology).

The terminology of symptoms, syndromes and disorders implies a hierarchical link to biological causes which, even if currently unknown, will eventually be empirically demonstrated. This assumption has often proved unwarranted. Probably more such clusters have proved useless or misleading than have successfully survived the process of scientific testing.

The approach to the categorization of mental disorders adopted in the latest International Classification of Diseases (ICD-10; WHO, 1992) is therefore appropriately cautious. The term 'disorder':

is used to imply the existence of a clinically recognizable set of symptoms or behaviour that in most cases is associated with distress and with interference with functions, always at the individual level and often at the group or social level (but not the latter only).

To make an ICD-10 diagnosis of mental disorder is not, therefore, to specify the presence of a disease, but to recognise the presence of the designated syndrome. It does, however, allow hypotheses concerning a pathology or other biological abnormality to be tested. The epidemiology of the disorder can be investigated and may provide a basis for further hypotheses. Another obvious test of usefulness is whether making the diagnosis is helpful to the individual concerned. Does it accurately predict forms of treatment that reduce disability

without harmful side-effects? Does it give some idea of the future course and outcome? Are there means of primary, secondary or tertiary prevention? These are matters for scientific inquiry.

At the very least, can the person afflicted and family carers be given the consolation that the condition has a name, that there are other people with similar problems and that experiences and methods of coping can be shared? The many charitable organisations that have been set up to help those who have a named syndrome, such as those attributed to Alzheimer, Asperger, Down, Kanner and Rett, demonstrate the value placed on the recognition of syndromes even at times when there was no hard-and-fast knowledge about causes and no cure was firmly available. The name is not a mere label, but an indispensable basis for communication and investigation.

A further benefit from testing disease theories is that there is often a bonus, both in the form of a reformulation of the original clinical syndrome and in the emergence of new knowledge of pathological, physiological, biochemical or etiological mechanisms that hitherto had been unsuspected. It is possible to hypothesise, on the basis of recently acquired biological knowledge, the existence of new syndromes within the old concepts.

Successful disease theories tend to evolve over time; from an initial association between a syndrome and a biological abnormality, towards a sophisticated complex of interlocking dimensional criteria based on deviations from normal biological functioning. The amount of knowledge involved in such concepts is immense by comparison with that in the initial categorical description. By the same token, the power to relieve suffering derived from the application of the knowledge may also increase dramatically (Häfner, 1987; Scadding, 1990).

This does not mean that it would have been possible to reach such a satisfactory formulation without having gone through a stage of simple categorisation, nor that disease categories can now be dismissed. To argue this would be to misunderstand the nature and value of scientific classification. Tycho Brahe and Linnaeus were part of a progressive scientific tradition, no less valuable because their contributions, if frozen into orthodoxy, would have persisted as a static and sterile preoccupation with description and classification. In fact, astronomy and botany could not have developed without them. Kepler and Darwin, and their successors in turn, would have had no foundations upon which to build.

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The essentialist alternative to the empirical approach, in which disease entities are regarded as having an existence independent of the observer, was the main basis of medicine for 2000 years, notably in the Galenic humoral theory. But recent advances in knowledge have demonstrated the dimensional relationships underlying more and more apparently discrete clinical syndromes. Although we cannot avoid classifying, we can avoid reifying the resultant classes. Scientists should have no difficulty in passing from the categorical to the dimensional mode as it suits their purposes. Diabetes and hypertension are obvious examples.

International nosological systems, such as Chapter F of the new ICD-10, which provide standardised Diagnostic Criteria for Research (DCR; WHO, 1993) to guide clinical recognition of the syndromes of mental disorder, serve essential public-health and scientific purposes. It is necessary to use them sensibly and work to improve them. But they can only help to further knowledge if two conditions are met. First, and more important, the rules must be applied to a base of clinical observations ('symptoms and signs') that accurately reflect the condition of the patient. In other words, the application of standard rules does not of itself guarantee accuracy. Second, the resulting categories should not be regarded as disease entities, but as technical aids for testing clinical hunches and research hypotheses, and for providing good-quality records that can be used for public-health and epidemiological purposes.

This book describes the development and use of methods that help to ensure the fulfilment of both these essential conditions: the reliable and accurate description of symptoms and syndromes, and the testing of hypotheses concerning their relationship to damage and dysfunction in underlying biological systems.

It should be added that this approach does not in any way suggest that environmental influences are irrelevant, or deny their common role in causing, exacerbating or otherwise influencing the expression of symptoms, syndromes and disorders.

### **The development of psychiatric syndromes**

The syndromes of 'schizophrenia', as described by Emil Kraepelin (1896) and Eugen Bleuler (1911), and the syndromes of 'autism', as described by Leo Kanner (1943) and Hans Asperger (1944), illustrate

the fitful progress made in the clinical description of two groups of severely disabling mental disorders.

*The importance of labels: early childhood autism*

The problems of defining and labelling syndromes are clearly illustrated in the case of 'early childhood autism'. Victor, the 'wild boy of Aveyron' first described with stunning clarity by J. M. G. Itard in his reports of 1801 and 1806 (Lane, 1977), provides an illustration. The phenomena delineated by Itard are as recognisable now as they were then, but, because he did not formulate the abnormal behaviours as symptoms, nor name them as a syndrome, it was not recognised that the techniques he used could be generalised to a class of children who were not simply mentally retarded in a global fashion but had highly specific impairments. It was not until Kanner described the phenomena and pointed out the similarities between them (only eleven children, but that was enough), and Asperger described in equally convincing manner a variant of the same set of problems in young men (Frith, 1991; Wing, 1981, 1996), that the syndrome and its boundaries could be investigated epidemiologically, and its relationship to diseases of known etiology and to intellectual disability more generally could be elucidated.

The development of the concept also illustrates the dangers that can follow the adoption of a name. Kanner used Bleuler's term 'autism' to label the syndrome. The confusion with 'childhood schizophrenia' still persists, but both DSM-III-R and ICD-10 now distinguish between the two types of syndrome. Autism and schizophrenia may yet prove to be linked (Frith and Frith, 1991; Frith, 1992) but the principle involved in separating them for classification purposes is important. It is simpler to link categories at a higher level than to distinguish between them once they are merged. It must remain possible to retrieve and study the elements right down to symptom level.

*The importance of labels: schizophrenia*

Schizophrenia is a condition at present defined only in terms of certain abnormalities of experience and behaviour. With only conjectural underpinning in biological knowledge, there is room for a

wide range of opinion as to which elements should be included or excluded. Two decades before Emil Kraepelin's views became influential, there was much the same discussion as now concerning the value of classifying severe mental disorders. The adherents of one school argued for the concept of a unitary psychosis; pointing out that there is an infinite variety of experience and behaviour and that to delineate boundaries between named classes is as fruitless as to try to classify the shapes of clouds. States of madness dissolved into each other, with or without a temporal sequence. Some proponents held that all mental illness began as melancholia and progressed through paranoia to dementia; others that virtually any sequence could occur.

Clouds can usefully be classified. The clarification in the fifth and sixth (1899) editions of Kraepelin's textbook brought to an end a period of chaos and introduced a simple, though crude, distinction between conditions characterised by mental deterioration, such as catatonia, hebephrenia and dementia phantastica, and the more periodic forms of mania and melancholia. He also hypothesised different causes for the two new 'disease entities'. The formulation was thankfully adopted because of its convenience.

The form in which dementia praecox has remained a dominant feature of psychiatric nosology is, of course, Eugen Bleuler's creation. The convenience of the new name, 'schizophrenia', must have played a large part in its acceptance, as did the fact that the connotations of the term 'dementia' seemed to have been dropped. Nevertheless, Bleuler's primary symptom was cognitive – loosening of the associations. This was his link to the biological origins of schizophrenia and also, through 'psychic complexes', to the disorders of affectivity, ambivalence, autism, attention and will. Catatonia, delusions, hallucinations and behavioural disturbance he regarded as accessory. These theoretical assumptions held for the largest sub-group, latent schizophrenia.

Bleuler's concept was subsequently used in markedly different ways. Under the influence of psychoanalysis in the United States, the least differentiated forms – latent and simple schizophrenia – dominated diagnosis to such an extent that descriptive psychopathology was derided and neglected. A similarly broad and vague approach to diagnosis in the Soviet Union, notably under the influence of the Moscow school, this time with a supposedly biological basis, was exploited for political purposes.

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At a symposium held on the occasion of the 600th anniversary of the University of Heidelberg, a later occupant of Kraepelin's Chair, Werner Janzarik, described the history and discussed the problems of the concept of schizophrenia. He began his paper with the incontrovertible observation that the history of schizophrenia is the history of the clinical syndromes that were 'only gradually, and at a relatively late period, grouped under the new designation after numerous differentiations and reclassifications.' He ended the lecture with the statement: 'So far, there is no conclusively defined disease known as schizophrenia. The history of the concept is a history, not of medical discoveries, but of the intellectual models on which the orientation of psychiatry is based' (Janzarik, 1987).

The US-UK Diagnostic Project (Cooper et al., 1972) and the International Pilot Study of Schizophrenia (IPSS: WHO, 1973), in which the seventh and eighth editions of the Present State Examination (PSE: Wing, Cooper and Sartorius, 1974) were used, were set up as part of the reaction against the use of terms like 'schizophrenia' without any technical provenance. The studies demonstrated the extent to which such a diagnosis in the USA and USSR was broader and less definable compared with usage elsewhere. The strong resurgence of public-health psychiatry in the USA and the growth of biological psychiatry led to the creation of DSM-III and its subsequent editions, DSM-III-R (APA, 1987) and DSM-IV (APA, 1994), which provided top-down algorithms for classification that set real technical standards. ICD-10 (1993) moved in the same direction by providing international standards for Chapter F (WHO, 1993).

The limitations of these rule-based nosologies are obvious and accepted. A paper on possible future criteria for schizophrenia (Flaum and Andreasen, 1991) illustrates the problem. DSM-III-R, ICD-10 and three proposed options for DSM-IV were compared. It is clearly unlikely that one of these five sets of rules will be found to represent the clinical manifestations of a disease process, while the other four are not. 'State-of-the-art' rules formulated in the absence of external validating criteria are likely to be fragile and transitory unless there is a consensus among professional opinion that they should only be used in order to exploit the advantages for clinical comparability, professional education and scientific study provided by a reference classification.

Within these limits, the ICD-10 rules should remain the world standard until the next revision, to be used irrespective of whatever local

or hypothesis-based criteria are used in addition. However, there remains the necessity to define and measure the phenomena on which the rules should operate.

### **International Classification of Diseases, tenth edition, ICD-10**

The term ‘disorder’, as used in ICD-10, is a higher order equivalent to ‘syndrome’. The structure of Chapter F does not conform to Hempel’s ideal classification (1959), which is ‘mutually exclusive and jointly exhaustive’. Such a structure is based on unattainable Aristotelian verities. Chapter F is one of 21 chapters in ICD-10, each using several axes of classification; some based on aetiology, some on pathology, some on syndrome. ICD-10 represents a stage in a gradual evolution that will continue as long as distressing and disabling disorders persist. The top-down classifying criteria for Chapter F make diagnoses more internationally comparable, thus enhancing clinical, educational, public health and research functions. That is good progress. But it is essential that the rules are applied to a base of clinical observation that accurately reflects the condition of the patient.

### **Categories and dimensions**

As disease theories become more successful in providing a solid basis of knowledge about abnormalities of biological and psychological functioning, the dimensional aspects of measurement within and between clinical syndromes become apparent. Both modes of measurement are necessary for advance, and it should be possible to move from one to the other as appropriate, without any sense of incongruity. This is as true of mental disorders as it is of a condition like diabetes. A system of clinical measurement cannot be purely categorical or purely dimensional (Wing, 1995). The most obvious example of the dimensional approach is in defining severity of symptom types, whether for investigation, for treatment purposes or for the assessment of outcomes. But the symptoms themselves must first be defined.

### **Defining symptoms**

The formulation and development of medical disease concepts requires an interaction between reliable recognition and labelling of

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one or a cluster of physical and/or psychological characteristics (regarded as undesirable because of the distress or disability that accompanies them) and demonstrable damage and dysfunction in underlying biological systems (pathology and/or aetiology). Medical terminology tends to label many such characteristics as ‘symptoms’ if a hypothetical link has been suggested, even when the evidence for it is inconclusive. The usage is so universal that it is adopted here. However, the caveat stated at the beginning of this chapter, that many such hypotheses have proved false, and even harmful when acted upon, must be kept in mind.

Undesired or undesirable physical symptoms are easier to define and recognise than psychological or behavioural equivalents, but a physical characteristic (sweating for example) is not necessarily symptomatic of a disorder simply because it *is* clearly ‘physical’.

On the other hand, the closer the definitions of an abnormal subjective experience comes to what Lewis (1953) called a ‘psychological dysfunction’, because it is defined in terms of deviation from a standard of normal psychological functioning, the more easy it is likely to be to find a link to an abnormality of biological function. For example, a description of thoughts or impulses intruding into the mind against conscious resistance has long been familiar to psychiatrists who make a practice of listening in detail to the unpleasant experiences described by their clients. The name ‘obsession’ is a convenient label, which can be given a precise definition that differentiates it from other symptoms such as phobia or thought insertion (see Chapter 4). Thought insertion can similarly be differentially defined, and there are already testable theories of how it might be linked through a neuropsychological intermediary to neural processes (Frith and Frith, 1991; Frith, 1992).

The obverse is also true. Symptoms should be defined as far as possible without recourse to purely social factors in the definition. Shoplifting and vandalism, for example, are undesirable behaviours, defined in purely social terms, and relatively easy to define reliably. Biological theories can be invoked to help explain some aspects in some people, but are not likely to account for a useful proportion of the variance. The more exclusive the social component in defining deviance, the less applicable is a symptom label.

The problems of defining individual symptoms (and problem behaviours, often called ‘signs’) are considered in Chapter 4, which describes the SCAN Glossary.



### Clusters or syndromes

Some symptoms are clearly members of a group because they have a core quality in common but manifest it in different ways. Obsessional symptoms, for example, have as a core quality the characteristic of intrusion into consciousness against the person's active willed resistance. But the content of the obsession varies widely. Many types of delusion, hallucination, phobia etc, are like this.

Another characteristic that helps, if present, to give solidity to a syndrome construct is when individual symptoms that are not apparently members of the same group nevertheless tend to occur together. The negative and positive symptoms 'of schizophrenia', for example, are so called because they do tend to coexist, and are thus thought likely to be related in some basic way.

Symptoms may also tend to occur in sequence rather than (or as well as) together, i.e. as a syndrome over time. There may be a recognisable 'natural' syndromatic course, episodic or developing, and also perhaps a characteristic outcome. Such syndromes have been refined over a century and a half of clinical observation, and elegantly described in such classics as Jaspers' *General Psychopathology*. On the whole, they have been supported by statistical analyses.

### The social context of diagnosis

Diagnosis and treatment are central, though by no means exclusively, to the medical role. That is why, ostensibly at any rate, patients consult doctors. But these functions provide the occasion for others. Doctors should be familiar with the range of human experience and behaviour. Many of them should be able to take on something of the role of counsellor, befriender, teacher, psychotherapist, social worker or advocate. Moreover, diagnosis is at least as much to do with ruling out disease explanations as with establishing that one or other of them would prove useful for helping a patient.

The problems brought to psychiatrists are therefore far from exclusively biological. Biological abnormalities can have social causes; many biological systems depend for their proper functioning on interaction with the psychosocial as well as the physical environment; and social influences can amplify biological impairments. The extent to which an individual is socially disabled depends, in addition, on

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psychosocial factors such as disadvantage and public, family and self attitudes. The resulting problems of classification and measurement of long-term psychiatric disorders have been discussed elsewhere (Wing, 1992, 1995; WHO, 1980).

Scientists must nevertheless try to keep the various factors contributing to social disablement theoretically separate, since they are likely to have different causes and practical effects and to require different types of intervention. This conclusion can be applied to most psychiatric syndromes, from phobias to dementia. The more clearly symptoms are described, and the more precisely the rules for grouping them into syndromes are specified, the more comparable will be tests of hypotheses of all kinds.

## References

- American Psychiatric Association (1987) *Diagnostic and statistical manual of mental disorders*, third edition – revised. Washington, DC: APA.
- American Psychiatric Association (1994) *Diagnostic and statistical manual of mental disorders*, fourth edition. Washington, DC: APA.
- Asperger H. (1944) Die autistischen Psychopathen im Kindesalter, *Archiv für Psychiatrie und Nervenkrankheiten*, 117: 76–136.
- Bleuler E. (1911) Dementia praecox oder die Gruppe der Schizophrenien. In: Aschaffenburg G. (Hgr) *Handbuch der Psychiatrie*, Spezieller Teil, 4. Abt. 1. Hälfte. Leipzig: Deuticke.
- Cooper J. E., Kendell R. E., Gurland B. J., Sharpe L., Copeland J. R. M. and Simon R. (1972) Psychiatric diagnosis in New York and London. London: Oxford University Press.
- Flaum M. and Andreasen N. C. (1991) Diagnostic criteria for schizophrenia and related disorders. *Schizophrenia Bulletin*, 17: 133–56.
- Frith C. (1992) *The cognitive neuropsychology of schizophrenia*. Hove: Erlbaum.
- Frith C. D. and Frith U. (1991) Elective affinities in schizophrenia and childhood autism. In: Bebbington P. (ed.) *Social psychiatry. Theory, methodology and practice*, pp. 65–88. New Brunswick and London: Transaction Publishers.
- Frith U. (ed.) (1991) *Autism and Asperger syndrome*, Cambridge University Press.
- Häfner H. (1987) The concept of a disease in psychiatry. *Psychological Medicine* 17: 11–14.
- Hempel C. G. (1959) Introduction to problems of taxonomy. In: Zubin J.