The term ‘comorbidity’ was introduced in medicine by Feinstein (1970) to denote those cases in which a ‘distinct additional clinical entity’ occurred during the clinical course of a patient having an index disease. This term has recently become very fashionable in psychiatry to indicate not only those cases in which a patient receives both a psychiatric and a general medical diagnosis (e.g. major depression and hypertension), but also those cases in which a patient receives two or more psychiatric diagnoses (e.g. major depression and panic disorder). This co-occurrence of two or more psychiatric diagnoses (‘psychiatric comorbidity’) has been reported to be very frequent. For instance, in the US National Comorbidity Survey (Kessler et al., 1994), 51% of patients with a DSM–III-R/DSM–IV (American Psychiatric Association, 1987, 1994) diagnosis of major depression had at least one concomitant (‘comorbid’) anxiety disorder and only 26% of them had no concomitant (‘comorbid’) mental disorder, whereas in the Early Developmental Stages of Psychopathology Study (Wittchen et al., 1998) the corresponding figures were 48.6% and 34.8%. In a study based on data from the Australian National Survey of Mental Health and Well-Being (Andrews et al., 2002), 21% of people fulfilling DSM–IV criteria for any mental disorder met the criteria for three or more concomitant (‘comorbid’) disorders.

The use of the term ‘comorbidity’ to indicate the concomitance of two or more psychiatric diagnoses appears incorrect because in most cases it is unclear whether the concomitant diagnoses actually reflect the presence of distinct clinical entities or refer to multiple manifestations of a single clinical entity. Because ‘the use of imprecise language may lead to correspondingly imprecise thinking’ (Lilienfeld et al., 1994), this usage of the term ‘comorbidity’ should probably be avoided.

However, the fact remains that the co-occurrence of multiple psychiatric diagnoses is now more frequent than in the past. This is certainly in part a consequence of the use of standardised diagnostic interviews, which helps to identify several clinical aspects that in the past remained unnoticed after the principal diagnosis had been made—a development that is obviously welcome because it is likely to lead to more comprehensive clinical management and more reliable prediction of future disability and service utilisation.

But this is only one part of the story. The other part is that the emergence of the phenomenon of ‘psychiatric comorbidity’ has been to some extent a by-product of some specific features of current diagnostic systems. Artificially splitting a complex clinical condition into several pieces may prevent a holistic approach to the individual, encouraging unwarranted polypharmacy, and may represent a new source of diagnostic unreliability because clinicians may focus their attention on one or other of the different ‘pieces’, especially in those clinical contexts in which coding of only one diagnosis is allowed.

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‘PSYCHIATRIC COMORBIDITY’ AS A BY-PRODUCT OF RECENT DIAGNOSTIC SYSTEMS

A powerful, usually unrecognised, factor contributing to the emergence of the phenomenon of ‘psychiatric comorbidity’ has been ‘the rule laid down in the construction of DSM–III (American Psychiatric Association, 1980) that the same symptom could not appear in more than one disorder’ (Robins, 1994). This rule (never made explicit, to my knowledge, in DSM-related publications), probably explains why the symptom ‘anxiety’ does not appear in the DSM–IV criteria for major depression, although the text of the manual acknowledges that patients with major depression frequently present with anxiety. Lee Robins, the only author who, as far as I know, has mentioned the above rule in the literature, stated: ‘I thought then, as I still do, that the rule was not a good one’ (Robins, 1994). Actually, DSM–IV does not allow the presence of anxiety in a patient with major depression to be recorded either as a symptom or, as allowed for delusions, a specifier for the diagnosis. The concomitant diagnosis of major depression and panic disorder is encouraged (being one of the most common forms of ‘psychiatric comorbidity’), whereas the concomitant diagnosis of major depression and generalised anxiety disorder is not allowed (unless generalised anxiety occurs also when the patient is not depressed). The latter exclusion criterion seems to be an acknowledgement of the implausibility of the idea that anxiety and depression, when they occur simultaneously, are two separate clinical entities, but it actually contributes to leaving the presence of anxiety in a patient with major depression (with its significant prognostic and therapeutic implications) totally unrecorded.

Not surprisingly, both the elimination of the above exclusion criterion (Zimmerman & Chelminski, 2003), which would be consistent with the logic of the system but would multiply the cases of ‘psychiatric comorbidity’, and the introduction of a mixed depressive–anxiety diagnostic category (Tyrer, 2001) have been proposed. A second, obvious, determinant of the emergence of the phenomenon of ‘psychiatric comorbidity’ has been the proliferation of diagnostic categories in recent classifications. If demarcations are made where they do not exist in nature, the probability that several diagnoses have to be made in an individual case will obviously increase. The current classification of anxiety and personality disorders is a good example of this. It is rare to see a patient with a diagnosis of an anxiety (or a personality) disorder who does not fulfil the criteria for at least one more anxiety (or personality) disorder. The fact that ‘neuroses and abnormal personalities’ do not have clear boundaries either among themselves or with normality was clearly recognised by Jaspers (1913; see below), and would argue in favour of a dimensional approach to their classification. Paradoxically, the attempt by the DSM to

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1 See pp.190–196, this issue.
characterise ‘pure’ disorders in these areas seems to be the first step towards the identification of several ‘dimensions’. However, how a dimensional approach would actually work in clinical practice (e.g. in what cases a disorder would finally be diagnosed, and how the diagnosis would be expressed) remains unclear.

A third relevant characteristic of current diagnostic systems is the limited number of hierarchical rules. A consolidated tradition in psychiatry was to establish a hierarchy of diagnostic categories so that, for example, if a psychotic disorder were present, the possibly concomitant neurotic disorders would not be diagnosed because they would be regarded as part of the clinical picture of the psychotic condition. One could argue that the current possibility of diagnosing a panic disorder in the presence of a diagnosis of schizophrenia represents a useful development, because this additional diagnosis provides information that may be useful for clinical management. But are we sure that the occurrence of panic attacks in a person with schizophrenia should be conceptualised as the ‘comorbidity’ of panic disorder and schizophrenia? Is the panic of a person with agoraphobia, of a person with major depression and of a person with schizophrenia the same psychopathological entity that simply ‘co-occurs’ with the other three? I am not aware of any research evidence on this issue.

A fourth relevant feature of our current diagnostic systems is the fact that they are based on operational diagnostic criteria. Because of this, they are regarded as more precise and reliable than the traditional ones based on clinical descriptions. However, the old clinical descriptions provided a gestalt of each diagnostic entity, which is often not provided by current operational definitions. This was probably due in part to the different emphasis laid on the various clinical aspects (whereas in current operational definitions the various clinical features are usually given the same weight), as well as to the inclusion of some aspects regarded as essential (e.g. autism in the case of schizophrenia) that do not appear in current diagnostic systems because they are regarded as not sufficiently reliable. Traditional clinical descriptions encouraged differential diagnosis, whereas current operational definitions encourage multiple diagnoses, probably in part because they are less able to convey the ‘essence’ of each diagnostic entity. Is this an intrinsic limitation of any operational definition, or a remediable flaw of our current operational definitions? Was the above-mentioned gestalt (for instance, in the case of schizophrenia) a fact or an illusion? Are we sure that we have used all the resources of the operational approach in typifying, for instance, the disorder of social and interpersonal functioning in schizophrenia?

‘PSYCHIATRIC COMORBIDITY’ AND THE NATURE OF PSYCHOPATHOLOGY

Most of the recent debate about psychiatric comorbidity has been remarkably atheoretical, focusing on the practical usefulness of one or the other approach in terms of treatment selection and prediction of outcome and service utilisation. However, the emergence of the phenomenon of ‘psychiatric comorbidity’ has obvious theoretical implications. The frequent co-occurrence of the mental disorders included in current diagnostic systems has recently been regarded as evidence against the idea that these disorders represent discrete disease entities (e.g. Cloninger, 2002). The point has been made that the nature of psychopathology is intrinsically composite and changeable, and that what is currently conceptualised as the co-occurrence of multiple disorders could be better reformulated as the complexity of many psychiatric conditions (with increasing complexity being an obvious predictor of greater severity, disability and service utilisation). From the psychodynamic viewpoint, the idea seems to be reinforced that the interaction of congenital predisposition, individual experiences and the type and success of defence mechanisms employed may generate an infinite variety of combinations of symptoms and signs. From the psychobiological viewpoint, the hypothesis seems to be supported that ‘noxious stimuli…perturb a variety of neuronal circuits’. The extent to which the various neuronal circuits will be involved varies individually, and consequently psychiatric conditions will lack symptomatic consistency and predictability’ (van Praag, 1996). From the evolutionary viewpoint, the concept seems to be corroborated that mental disorders are the expression of preformed response patterns shared by all humans, which may be activated simultaneously or successively in the same individual by noxae of various nature – a view endorsed by Kraepelin himself in one of his later works, in which he dismissed the model of discrete disease entities even for dementia praecox and manic–depressive insanity (Kraepelin, 1920).

However, the emergence of the phenomenon of ‘psychiatric comorbidity’ does not necessarily contradict the idea that psychopathology consists of discrete disease entities. An alternative possibility is that psychopathology does consist of discrete entities, but these entities are not appropriately reflected by current diagnostic categories. If this is the case, then current clinical research on ‘psychiatric comorbidity’ may be helpful in the search for ‘true’ disease entities, contributing in the long term to a rearrangement of present classifications, which may involve a simplification (i.e. a single disease entity may underlie the apparent ‘comorbidity’ of several disorders), a further complication (i.e. different disease entities may correspond to different ‘comorbidity’ patterns) or possibly a simplification in some areas of classification and a further complication in other areas.

There is, however, a third possibility: that the nature of psychopathology is intrinsically heterogeneous, consisting partly of true disease entities and partly of maladaptive response patterns. This is what Jaspers (1913) actually suggested when he distinguished between ‘true diseases’ (such as general paresis), which have clear boundaries among themselves and with normality; ‘circles’ (such as manic–depressive insanity and schizophrenia), which have clear boundaries with normality but not among themselves; and ‘types’ (such as neuroses and abnormal personalities), which do not have clear boundaries either among themselves or with normality. Recently, it has been pointed out (Nesse, 2000) that throughout medicine there are diseases arising from a defect in the body’s machinery and diseases
arising from a dysregulation of defences. If this is true also for mental disorders – for example, if a condition such as bipolar disorder is a disease arising from a defect in the brain machinery, whereas conditions such as anxiety disorders, or part of them, arise from a dysregulation of defences – then different classification strategies may be needed for the various areas of psychopathology.

**DECLARATION OF INTEREST**

None.

**REFERENCES**


