Neurosis: another tough old word

In 1980 the American Psychiatric Association published a revised system of classification (DSM III) from which it had removed the category neurosis.1 Shortly afterwards the Lancet carried a leading article titled “Goodbye neurosis?”2 Subsequent events have confirmed the wisdom of the question mark: for, while the next revision of the American diagnostic system will continue to do without the class of neuroses,3 the term is still used frequently in everyday clinical practice in Britain and chapters on the neuroses continue to appear in British textbooks of psychiatry.4,5 Recently Sims has taken up the case for retaining neurosis in the diagnostic system, arguing that the disorders it denotes have so much in common that they should be kept together.6

This is not the first time that the obituary of neurosis has been written. In 1923 Schneider remarked that neurosis is “an erroneous and unfortunate expression,” arguing that all the conditions it denoted were abnormal reactions of the personality and should be classified as such.7 Schneider’s ideas have had some influence on psychiatric thought, but neurosis has lived on. What reasons are now being suggested for doing away with such a widely used term? There are three main arguments: firstly, it groups together conditions that would be classified better in other ways; secondly, it introduces unsubstantiated ideas about aetiology into the system of classification; and, thirdly, we can do perfectly well without it. Each of these arguments needs to be expanded a little.

The first point—that the class of neurosis contains some conditions that would be classified better in other ways—is not new. It has been with us ever since the term was proposed by Cullen in 1769. In its original meaning neurosis denoted “disorders of sense and motion” accompanied by evidence of general disease in the form of fever or wasting and seeming to be due to a “general affection of the nervous system” rather than a local cause.8 Cullen included in his category of neurosis conditions such as apoplexy, epilepsy, asthma, and mania, as well as those we would now call neuroses. As knowledge of pathology increased and the severe forms of mental illness were studied more carefully these other conditions were removed one by one from the neuroses and classified elsewhere. One of the conditions which the authors of DSM III now think is inappropriately classified under neurosis is neurotic depression, which they consider (with good reason) to be more closely related to major affective disorders. Instead of following historical precedent, however, by reducing by one more the conditions classified under neuroses they have removed the whole class from the system of classification.

As a result all other conditions that would otherwise be listed under neuroses have had to be found new homes. Some of these homes do not seem very happy: for example, depersonalisation disorders are now included with forms of hysteria as “dissociative disorders.” So one problem has been solved but at the expense of creating another.

The second argument is that the word neurosis is no longer a purely descriptive term: it has acquired an aetiological meaning—and a controversial one at that. This problem originated in the work of psychoanalysts, who extended the use of the word neurosis to include the psychopathological process through which the conditions were supposed to arise. Subsequently the aetiological use became more general; for example, a widely used textbook stated that as well as its descriptive connotation neurosis has a “more fundamental aetiological one.”11 This is an important problem, but all that is required to solve it is a more exact use of the word neurosis. Either the descriptive sense may be retained while the aetiological meaning is referred to in another way—for example, as “neurotic process”—or the descriptive sense may be referred to by a separate term as is done in the 9th revision of the International Classification of Diseases, which uses the phrase “neurotic disorder” for this purpose.

The third argument for abolishing the class of neuroses is that there is no need for this broad class, since the subclasses such as anxiety disorder, obsessional disorder, and hysterias are sufficient. When it has been said that a patient has, for example, an anxiety disorder, no useful extra information is conveyed by saying that this disorder is a neurosis. Furthermore, more, the subclasses are the only ones that epidemiologists or other research workers can use: neurosis is too general a grouping, and its boundaries with normality and with hysteria and personality disorder are difficult to draw.12 There are two main arguments in favour of retaining the class of neuroses. The first is that (with the possible exception of depressive neuroses) the conditions classified in this way have several factors in common. This is the argument developed by Sims in his paper, in which he draws attention to shared clinical features, such as difficulties with personal relations and bodily symptoms without an organic cause; and to common elements in treatment, notably psychological procedures to deal with poor self esteem and problems in relations with others.13 These features are certainly shared by the conditions we call neuroses, but they are not specific to these conditions, so the argument is not very strong.

The second reason for keeping an overall category of neurosis as well as the specific syndromes is that not all the conditions that would generally be thought of as neuroses fit neatly within the diagnostic criteria for particular syndromes such as anxiety disorder, obsessional disorder, or hysteria. There are many intermediate cases—especially among the minor emotional disorders seen so frequently in general practice, conditions characterised by a mixture of anxiety, depressive, obsessional, and hypochondriacal symptoms. These minor conditions have been called subclinical neurosis syndrome,14 though in recent years the name “minor affective disorder” has often been used.15 It is not enough to give these conditions a name, however: the epidemiologist requires a clear definition of the boundaries of this group of cases and a single place for them in the system of classification. This problem needs to be addressed in the next revisions of the main diagnostic systems—whether or not these common disorders are included in a class of neurotic disorders.

Are we ready to follow the Americans and say goodbye to neurosis? None of the arguments for or against the abolition of neurosis is compelling. On balance it seems best to be conservative and make changes only when there are strong reasons for doing so—and preferably when any new classification can be based more firmly on aetiology. Knowledge of the causes of anxiety disorders has increased substantially in recent years, but much uncertainty remains about the aetiology of all the conditions we have been considering. For the present there seems to be enough common ground among these disorders to keep them together in our system of classification, either as neuroses (or neurotic disorders) or under some broader rubric such as "neuroses and adjustment"
disorders." In any case, clinicians are unlikely to stop using the word "neurosis" in their everyday work as a useful collective term for a common group of problems. Indeed, these current disputes about the term neurosis are reminders of a debate some years ago about another controversial word, hysteria. At that time Sir Aubrey Lewis wrote "a tough old word like hysteria dies very hard. It tends to outlive its obituaries." Though neurosis is not as old a word as hysteria, it is likely to prove to be no less tough.

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Regular Review

Back to the future: So what will fibrinolytic therapy offer your patients with myocardial infarction?

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"... shall dissolve... and leave not a rack behind."

_The Tempest_ iv.i.

Our ancestors expected blood to clot when it left the body or when it stayed in the blood vessels after death. They were, therefore, puzzled by the incoagulability of menstrual blood and by Hunter's finding in 1794 that in "animals killed by lightning or electricity... or who are run very hard and killed in such a state" the blood also remained incoagulable. The next century saw the unravelling of these mysteries and the provision of important clues to an entirely new approach to the management of thrombosis.

If shed clotted blood is incubated its fibrin coagulum slowly dissolves and the addition of the powerful procoagulant, thrombin, will not produce any further clotting. By the beginning of the twentieth century it had been shown that not only was the blood from victims of violent death incoagulable and free of fibrin but that it would also render normal blood incoagulable by digesting its fibrin. This "fibrinolytic activity" was shown to reside in the globulin fraction of the plasma. During this century we have seen the clarification of the lytic pathway by the identification of the inactive β globulin precursor plasminogen, which is capable of being converted to the active serine protease enzyme, plasmin. Plasmin is non-specifically proteolytic and so not only attacks fibrin but also breaks down other proteins, including fibrinogen and members of the clotting cascade. The debates about the part played by "natural fibrinolysis" in the normal economy of the body need not concern us here, but it is still far from clear whether there is a balance between the coagulation and the fibrinolytic pathways which maintains our blood in an appropriate state of fluidity and, if so, whether disturbances of this balance make any contribution to thrombotic disease. Of more immediate relevance is to ask ourselves whether we can harness the clot dissolving properties of the plasminogen-plasmin system in the treatment of thrombi which have already formed and are about to produce infarction.

Harnessing the lytic system

Early attempts to capitalise on the fibrinolytic system were rather basic; in 1936 Judine "put post-mortem fibrinolysis to practical use by designing a corpse blood transfusion service, the donors being those who had died suddenly and whose blood, consequently fluid, required no anticoagulant." At the same time Tillett and Garner, at the Rockefeller Institute, were reporting that filtrates of haemolytic streptococci could cause rapid liquefaction of human fibrin, and by 1945 Christensen and MacLeod had shown that the bacteria were producing an activator of the fibrinolytic system, which they called streptokinase. We now know that this is a single chain protein with a molecular weight of 48,000 daltons, that it does not activate plasminogen directly but complexes with it, and that this complex is then converted into active plasmin. When we come to consider how to get fibrinolytic activity to the place where we want it (the harmful thrombus) and not to the places which we wish to spare (the helpful haemostatic mechanisms) the relevance of this will become apparent. Streptokinase is antigenic so that its administration may cause fever and allergic reactions, but of more importance is that it stimulates antibody production, so that treatment for more than a few days is impracticable.