Somatisation in neurological practice

The diagnosis
The average British neurologist, according to a recent survey,1 will fail to find an adequate physiological explanation for the symptoms of one in every five of his or her patients despite appropriate, often exhaustive, investigations. In some patients organic pathology may be present, but the symptoms and the ensuing disability cannot be satisfactorily explained as a result. According to Mace and Trimble,2 the authors of the survey, as many as 36 000 such patients may be seen by British neurologists every year. Surveys of neurological inpatients have produced equally striking results. In a recent study of 100 consecutive new admissions to a neurological ward an adequate organic explanation for the symptoms was only forthcoming in 40 of them, whereas in the rest organic pathology was absent or provided only a partial explanation.3 Reports from general medical settings echo these findings4 and it is generally assumed that psychological and social factors are important in determining these unexplained symptoms. The search for a psychiatric diagnosis wide enough to fit patients whose symptoms are partly explained by organic pathology as well as those in whom such explanation is not forthcoming, and precise enough to provide a pathophysiological explanation for such diverse symptoms continues to exercise psychiatrists and neurologists alike.

A brief definition of “somatisation”, a concept well discussed by Lipowsky,4 is appropriate here, as it provides a useful way to categorise these symptoms and to bypass fruitless diagnostic controversy. Somatisation can be defined as a tendency to experience and communicate somatic distress and symptoms unaccounted for by pathological findings, to attribute them to physical illness, and to seek medical help. It is usually assumed that somatisation occurs in response to psychosocial stresses, although this may not be recognised by the patient, or may be actively denied. Thus defined, somatisation encompasses a wide spectrum of symptoms referred to various organs or systems, appearing acutely or over many years and mimicking a variety of diseases. In many patients the unexplained somatic symptoms may be an integral part of depression or anxiety, whereas in others “classic” psychiatric symptoms are absent and the mental state may seem to be entirely normal. The categories of conversion disorder or hysterical neurosis, conversion type (DSM-III-R), or dissociative/conversion disorder (ICD-10), are included within the wider concept of somatisation. Briquet’s syndrome—a narrowly defined, chronic disorder with onset before the age of 30 and multiple symptoms pertaining to various systems—is also included here.

Within the wider concept of somatisation, hysteria, or conversion disorder, if narrowly defined, refers to those unexplained symptoms suggestive of neurological disease (loss of function or sensation) and has the doubtful distinction among psychiatric diagnoses of still invoking “Freudian” mechanisms as an explanation. The idea that a traumatic experience, usually of a sexual nature, could be rendered innocuous by being transformed into a somatic symptom5 is central to the diagnosis. The resolution of this unconscious conflict is the primary gain and the advantages resulting from the assumption of the sick role are known as the secondary gain. This legacy of the psychoanalytical era is still embodied in the current DSM-III-R criteria, which include a temporal relation with relevant psychological stressors and an “unconscious” motivation for the symptoms. In practice, however, these criteria are vague and difficult to establish. In the previously quoted survey of British neurologists6 relevant psychological factors were only evident in a third of the cases, suggesting that they may be difficult to elicit or have limited clinical relevance. Similarly, the degree of insight may vary depending on the duration of symptoms and contact with the medical profession.

Unexplained neurological symptoms are often part of a more widespread picture of somatisation and strict diagnostic boundaries are of little clinical relevance and difficult to delineate. Here the broad definition of somatisation is used to encompass neurological and other symptoms for which no obvious organic explanation is forthcoming. This is, in my view, and hopefully in that of the readers, more clinically sound than the artificial compartmentalisation proposed by diagnostic classifications. In doing so, however, caution has to be exercised in interpreting the results of studies with different definitions of what constitutes somatisation or conversion disorder. Pain will not be dealt with here for reasons of space, but much of what is contained in this editorial also applies to the symptom of pain when it appears as part of the clinical syndrome of somatisation.

Given the difficulties in establishing the diagnosis, it is not surprising that the reported prevalence of somatisation varies depending on the setting. Thus in the previously quoted survey of British neurologists7 20% of patients had non-organic symptoms, a similar rate to that quoted by United States neurologists.8 In a United Kingdom
survey of patients admitted to a neurological ward' an adequate organic explanation for the symptoms was forthcoming in only a third of the patients, whereas in a quarter, a purely psychological explanation was more appropriate. For the remainder, a mixture of organic and psychological factors seemed relevant and clinicians were often at a loss in trying to find appropriate diagnostic labels. A more recent study of consecutive acute neurological admissions in Denmark reported an adequate organic explanation for only 40 out of 100 patients. As can be expected the prevalence of Briquet’s syndrome, the most severe and chronic form of somatisation, is much lower (2 per 1000 in a United Kingdom general practice survey).

In children, unexplained physical symptoms are common and minor abdominal or limb pains and headaches without organic cause have been reported in up to 20% of school children, but account for less than 1% of children admitted to hospital. Unexplained neurological symptoms make up about 2% of the referrals to paediatric neurologists and this presentation is rare before the age of six. The symptoms children exhibit are not substantially different from those seen in adults and multiple symptoms are also common. In young children unexplained somatic symptoms are equally frequent in boys and girls, but in adolescence, females predominate.

Somatisation, including conversion disorder seems to be commoner in those of low socioeconomic status, and dramatic changes in prevalence have been associated with improving education and greater prosperity in communities studied prospectively. In these communities the overall psychiatric morbidity remained unchanged, with depression becoming commoner as somatisation waned, an indication of the complex link between these conditions.

Comorbidity

The association of brain damage and narrowly defined conversion hysteria has been recognised for a long time and the problems in separating the two have in the past led to passionate pleas to abandon the diagnosis. Slater et al. in their classic study found that only 21 out of 85 patients with the diagnosis of hysteria merited this diagnosis at follow up, whereas other patients in their series had gone on to develop neurological or psychiatric illness that accounted for the initial symptoms. Although diagnostic difficulties are likely to remain, the availability of new diagnostic techniques (for example, imaging) and the better characterisation of neurological disorders (for example, dystonia) have greatly improved the accuracy of diagnosis.

The prevalence of brain disease in those with unexplained neurological symptoms varies widely depending on the setting. Thus demonstrable organic pathology was reported in only 3% of those admitted to a psychiatric hospital, whereas that was the case for nearly half of those seen in neurological settings. The presence of brain pathology may both predispose and provide a model for the development of conversion symptoms. This complex interaction is best seen in epilepsy where nearly a quarter of patients may also have pseudo-seizures. The presence of cognitive impairment, early onset epilepsy, and anticonvulsant toxicity seem to predispose to pseudo-seizures, with the epileptic attack providing a model for conversion symptoms. Similar associations have also been described long ago in patients with head injuries.

The incidence of conversion symptoms in other neurological diseases has not been systematically studied. Claims made that this association was common in multiple sclerosis have not been substantiated and it remains to be explored whether damage to specific brain structures (for example, the frontal lobes with the attending impairment in executive and attentional functions) results in an increased vulnerability to develop conversion symptoms.

Association with other psychiatric diagnoses, especially depression, is common. A 50% lifetime prevalence for depression and 20% for panic disorder have been reported in patients with multiple somatic symptoms without organic pathology. In cross sectional studies major depression has been detected in over 50% of these patients. In those with narrowly defined conversion disorder, affective symptoms seem to be less common with a frequency of under 30% in cross sectional studies, although few reports separate these patients from those with multiple somatic symptoms. The frequent association of somatisation and affective disorder raises the possibility that affective disorder may be a predisposing factor and not simply a reaction to disability. Psychiatric symptoms are often overlooked in these patients, who tend to minimise or explain away psychological distress. This inability to acknowledge and express emotional nuances has been called ‘alexithymia’ and it is probably an important determinant in the somatic presentation of psychiatric symptoms.

Psychiatric comorbidity seems to be less severe in children and adolescents with unexplained somatic and neurological disorders. Good premorbid adjustment is common and when present emotional disturbances are only mild. Sexual abuse and bereavement are only relevant in a minority of children and overt family pathology has been found in less than a quarter of cases. On the other hand, sociocultural factors and physical or psychiatric illness in other family members are particularly relevant in these children.

Pathophysiological mechanisms

The mechanisms of hysterical symptoms remain to be determined. Symptoms tend to reflect the patient’s idea of disease, rather than patterns of neurological dysfunction. This has led to the belief that a “central” mechanism resulting in an altered state of awareness or dissociation may be the necessary facilitator. This theory originates from the apparent similarity between hypnosis—a state of altered awareness during which hysterical symptoms may be induced or removed—and dissociation. Neuropsychological studies performed in patients under hypnosis have yielded conflicting and inconclusive results. Changes in the amplitude of the P300 in response to suggestion have been interpreted as implicating attentional mechanisms and a role for the attentional systems involved in focusing (anterior cingulate) and arousal (frontal circuits) has been postulated. Most of these studies have been performed in normal subjects, however, and in the few that included patients with conversion disorder the results have been inconclusive. Magnetic stimulation of the motor cortex has shown normal latency and response amplitude in patients with hysterical limb weakness and it remains to be determined whether abnormalities of the readiness potential, a negative shift that precedes a self initiated action, are present in these patients.

A specific role for the non-dominant hemisphere has been suggested after the finding that conversion symptoms are commoner on the left side of the body and that they may resemble the unawareness and neglect found in
right parietal lesions. These old findings have recently received new impetus with the description of non-specific EEG abnormalities in the right hemisphere of patients with gross conversion symptoms.

Although these psychophysiological mechanisms may be necessary for the symptoms to appear, psychopathological abnormalities are still paramount. For patients with hysterical symptoms the sick role becomes attractive when the demands of life are too difficult to cope with, because of limited emotional resources or the clustering of adverse events. This learnt behaviour is in part determined by cultural factors and childhood experiences and is reinforced by the perceived advantages of the sick role (for example, financial gain, the attention of others). The patient's view of psychiatric illness as shameful and indicative of personal failure is also important in this context.

Similar mechanisms are also likely to operate in patients with more widespread somatic symptoms. Symptoms may result from magnification of physiological sensations (for example, hyperventilation, arousal) when the person is abnormally anxious or depressed and if the connection between the stimulus that causes arousal and the ensuing symptoms is not made, symptoms may come to be seen as primary. Personality factors such as introspection and a vigilant attitude to illness are also contributory. The contact with the medical profession at this stage may serve to consolidate the symptoms by paying undue attention to them or by providing a quasiscientific explanation. In this way, a symptom that initially may have a doubtful significance in the patient's mind becomes legitimised and the presence of anxiety or depression is explained away as an appropriate reaction to a disturbing physical symptom. The choice of symptom may be determined by the experience of illness in the patient or close relatives.

The management of somatisation in neurological practice
The exclusion of organic pathology, or the evaluation of its contribution to the overall clinical picture, has been greatly aided by modern laboratory techniques including imaging. It is, however, useful to remember that "classical features" of hysteria such as belle indifference, non-anatomical distribution of sensory loss and "give way" weakness need to be interpreted with caution as these signs are often present in anxious patients with well-documented organic disease.

A close collaboration between neurologists and psychiatrists is essential in the evaluation and treatment of these patients. It usually fails to the psychiatrist to elicit the relevant aetiological factors, to assess the presence of significant psychiatric symptoms, and to initiate treatment. The way neurologists approach patients in these early stages is all important. A clear message that credibility is not in doubt and that the symptoms are distressing whatever their cause should be coupled with an explanation that normal investigations augur well for the recovery of function. Equally important is to present the psychiatric referral as a crucial step in the diagnosis and treatment, not as a mysterious referral to a "colleague with an interest in your condition". In turn the psychiatrist needs to be familiar with neurological disease and with the somatic presentation of psychiatric illness. Initially, questions about the way the patient has coped emotionally with distressing physical symptoms are far more useful than standard ways of eliciting psychopathology, which often result in a defensive denial. The first psychiatric interview should have the double aim of establishing a common ground for further treatment and assessing whether significant psychiatric symptoms, deserving treatment in their own right, are present. Arguments as to whether psychological factors are the cause of physical symptoms are best avoided at this stage. Useful hints about the way to interview these patients and to gain their compliance have been clearly discussed by Creed and Guthrie.

The treatment of conversion disorder is often based on behavioural programmes specifically tailored to the patient, combining physiotherapy with cognitive interventions aimed at modifying the patient's interpretation and response to symptoms. In most cases this treatment can be carried out on an outpatient basis. A useful review of some of these principles is provided by Bass and Benjamin. Intravenous amytal has a limited part to play in dealing with these patients and it may be more useful in restoring memory in the amnesic patient than in removing paralysis or abnormal movements. A videotape recording of the procedure may be useful in demonstrating to the patient the normality of the affected limbs, speech, or memory and can be used as an encouraging first step in treatment. Hypnosis may be used for similar purposes but it is no longer a mainstay in the armamentarium of British psychiatrists who prefer to use better validated and more reliable behavioural techniques.

Treatment with the appropriate psychotropic medication when the symptoms of anxiety or depression warrant it is also an important part of treatment, as are the avoidance of unnecessary investigations and iatrogenic damage.

The outcome of treatment has seldom been monitored over long periods. Acute conversion symptoms are regarded as having a better prognosis and striking improvements have been reported with simple supportive measures in patients with hysterical paraplegia or gait disturbances after minor trauma.

Follow up studies of patients with more pervasive somatic symptoms have suggested that older subjects with overt psychological disturbance may fare worse. The type of symptom seems to carry less prognostic significance, although intermittent symptoms (pseudoseizures) or symptoms causing little functional disability may prove more difficult to remove.

When treatment fails, attempts to minimise the costs of these patients to the Health Service are still worthwhile. An approach centred on advising referring physicians on how to deal with patients with multiple somatic symptoms has proved a worthwhile cost cutting exercise in the United States, but unfortunately money savings were not followed by symptom reduction or increased patient satisfaction.

Conclusions
The management of neurological symptoms for which no satisfactory organic explanation can be found is a daily challenge for neurologists and psychiatrists. Modern diagnostic techniques make it likely that diagnostic errors are now much less common than previously reported, but diagnostic labels remain inadequate for many of these patients. A pragmatic, multidisciplinary approach to their management is required, keeping in mind that organic pathology and somatisation frequently coexist and that significant anxiety or depression are often part of the mental state. When all else fails, prevention of iatrogenic damage and unnecessary use of resources remain worthwhile aims.
NEUROLOGICAL STAMP

Jacobus Ludovicus Shroeder van der Kolk (1797–1862)

Kolk became Professor of Anatomy and Physiology at the University of Utrecht in 1826, and it was there he introduced microscopic and experimental techniques in the study of histological anatomy. He was very interested in the contemporary treatment of the insane and his most important contributions were in that field. He also studied the anatomy of the brain of the mentally deficient. Kolk became an inspector of lunatic asylums and upon the request of the Government, wrote a comprehensive report on the conditions of the insane asylums and their inmates. He exerted great influence on the Government and asylum directors. Philatelically he was honoured in 1960 in World Mental Health Year by The Netherlands (Stanley Gibbons 898; Scott 383).

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