Hysteria: a case for conservation?

Diagnostic concepts are discarded when they are no longer clinically useful. The survival of hysteria is therefore evidence of its continued value, and psychiatry and neurology would be hard pressed if the diagnosis, or something similar, were no longer available. Yet hysteria has had to endure many demolition attempts. The American Psychiatric Association’s current classification, DSM III, has abandoned hysteria but only by subdividing it and using new terms to describe familiar clinical presentations. One of the criticisms of hysteria is that follow up studies show the diagnosis to be unstable. About one third of patients diagnosed at a neurological hospital as having hysteria were later found to have organic disease that was presumed to have been present at the time of the original diagnosis. Others had developed psychotic illnesses. A similar study at a psychiatric hospital showed that only 13% of patients retained hysteria as the sole diagnosis. The term has also been criticised because of its multiple meanings, which include clinical syndromes, a personality type, and an epidemic phenomenon.

The fear of missing organic disease means that doctors have become more cautious about diagnosing hysteria. Several conditions previously regarded as hysterical are now thought to have an organic basis, including spasmodic torticollis, blepharospasm, and writer’s cramp. There may be some more to come. Nevertheless, there is a nucleus of patients for whom no diagnosis other than hysteria seems right.

The two main variants of hysteria are conversion disorder and Briquet’s syndrome. Conversion disorder is seen most often by neurologists and has been defined as loss or distortion of neurological function not fully explained by organic disease. Psychiatrists add that there should also be positive evidence that the symptom is linked to psychological factors. The commonest symptoms include gait disturbance, seizures, paralysis, and sensory disturbance. Although the symptom itself cannot be explained by organic disease, coexisting disease of the nervous system occurs in as many as half the patients in some series. Cerebral disease may facilitate the use of hysterical mechanisms and provide a model for the symptom—for example, pseudoseizures in epileptics. The prevalence of organic disease is much lower, however, in studies that have been confined to patients in psychiatric hospitals.

Briquet’s syndrome has been brought to prominence by psychiatrists in St Louis and is characterised by multiple and recurrent somatic complaints for which medical attention is sought; the history covers many years, and the condition begins before 30. It is almost exclusively confined to women. Guze et al have attempted to justify the diagnosis by showing a high degree of consistency over many years and a strong familial pattern. Female relatives of patients with Briquet’s syndrome have an increased risk of the syndrome and of antisocial personality, and male relatives have an increased risk of antisocial personality. The condition may affect 1% of American women but seems much rarer in Britain. Americans have easier access than Britons to specialists, and they might thus more easily acquire numerous diagnoses. Cultural and iatrogenic factors may therefore play a prominent part in starting the condition.

Like others before him Marsden has defended the use of hysteria to describe a symptom rather than a disease and has formulated a set of rules to help reduce diagnostic errors. He emphasises that those with hysterical symptoms may have associated physical or psychiatric illness that is recognised yet to be detected; they may exhibit abnormal illness behaviour, and they require further neurological and psychiatric exploration.

Conversion disorder and Briquet’s syndrome are two of several conditions characterised by somatic complaints without organic pathology. They are included by DSM III in the group of somatoform disorders with psychogenic pain, hypochondriasis, and dysmorphophobia, but these conditions are not clearly distinguished from one another on clinical or genetic grounds. They are classified according to the superficial presenting complaint, not on consistent differences in psychopathology, and they have little implication for treatment. They also merge with the factitious disorders, such as dermatitis artefacta and Munchausen’s syndrome, in which symptoms are started voluntarily by the patient. The distinction between somatoform and factitious disorders rests on the subjective judgment of whether symptoms are produced consciously or unconsciously. This judgment, which is notoriously difficult, is further complicated by the patient’s insight varying from time to time.

What all these conditions have in common is a desire to assume the sick role when there is no evidence of bodily disease. They do not fit neatly into the medical model of disease, and they may be understood best through learning...
theory.1 The steady flow of publications on hysteria suggests that the conservative are on top at the moment, but they may be conserving what is merely a façade and one of only several manifestations of abnormal illness behaviour. Hysteria should be regarded as a first stage in diagnosis. Doctors should then look carefully for neurological or psychiatric illness and particularly for factors in the patient’s social orbit that may be rewarding the sick role.

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Lassa fever

Lassa fever is endemic in many parts of rural west Africa. The virus is transmitted to man by a rodent ( Mastomys natalensis ) which is the natural reservoir of infection and which may enter dwellings and contaminate the environment with virus infected secretions, particularly urine. Among patients admitted to hospital in west Africa mortality ranges from 15% to 20%. Nevertheless, considerable evidence now shows that infection may be mild, particularly in children.3 Thus seroepidemiological studies in parts of Sierra Leone suggest that about 6% of the population a year may acquire immune responses to the virus of Lassa fever without there having been any clearly defined disease outbreaks ( J B McCormick, personal communication). In African hospitals infection has been transmitted to other patients and hospital staff through parenteral exposure to blood or blood stained secretions, which usually contain high concentrations of virus. Among patients variable standards of nursing or sharing equipment (including needles and syringes) were responsible for hospital acquired infections; among hospital staff accidental inoculation or contamination of broken skin or the mucous membranes were likely portals of entry for the virus. Outside Africa Lassa fever has not been transmitted from infected patients to other people, either in hospital or in the community. Nevertheless, cases of severe and fatal laboratory acquired infection have occurred.14

Several viruses with different modes of transmission cause viral haemorrhagic fevers in the tropics, but Lassa fever is the only one which has been imported into the United Kingdom: 10 confirmed importations have been reported since 1971. With the exceptions of a British nurse flown home from Sierra Leone in 1985 1 and a patient who was travelling to the United States, all reported cases have been admitted initially to hospitals in inner London. As about 800 passengers arrive daily in this country from west Africa, not surprisingly patients frequently present with febrile illnesses in accident and emergency departments on return from parts of the tropics in which viral haemorrhagic fevers are endemic.9

In 1976 the Department of Health and Social Security issued a memorandum on Lassa fever which provided guidelines for the isolation of patients and surveillance of their contacts.7 Although updated, these recommendations were influenced by the high mortality among patients admitted to hospital and appreciable secondary attack rates among their contacts in outbreaks in west Africa. Much more is now known about the clinical course and route of transmission of Lassa fever as well as other viral haemorrhagic fevers, and this is reflected in the DHSS’s recently published memorandum on the control of viral haemorrhagic fevers.9

In contrast with the previous memorandum, not only Lassa fever, but Congo/Crimene haemorrhagic fever and Marburg and Ebola fevers are also covered, since, although they have not yet been imported to Britain, they are potentially capable of person to person transmission in hospital by routes similar to that of Lassa fever.

Many of the amendments are welcome, in particular restricting surveillance to only close contacts—that is, those in direct contact with patients, their blood, excretions, and secretions, or clothing, bedding, or fomites which may have been contaminated by them. Also included are those who have cared for patients during their illness or who have handled their specimens. The new recommendations now obviate the need to follow up other contacts. Hitherto there has been considerable over-reaction to the risks of the spread of infection within the community, and this had imposed a considerable workload on the public health services, which has been costly in terms of time, energy, and resources. Indeed, it has often been counterproductive, creating alarm among the general public and focusing attention away from the dangers of hospital and laboratory infection.9 This change in policy makes sense, since no secondary cases have occurred in Britain in about 1500 people placed under surveillance after the importation of the 10 confirmed cases of Lassa fever.

The clinical features of Lassa fever as well as of other haemorrhagic fevers are often specific in the early stages. Most febrile patients who have recently been in parts of the tropics in which viral haemorrhagic fevers are endemic are much more likely to have infections such as malaria or typhoid. Hence the recommendation that appropriate specimens should be taken to exclude malaria before transferring sick patients to a strict security isolation unit is welcome. A positive diagnosis will reduce the necessity for transporting sick patients to such hospitals, and may even save lives as a result of prompt diagnosis and treatment.

The memorandum also emphasises the impracticality of instituting stringent isolation precautions for all patients who present in Britain after developing febrile illnesses while in Africa or after returning from areas where viral haemorrhagic fevers are endemic. Levels of isolation are recommended according to whether there is strong, moderate, or only minimal risk of infection. Patients who have recently come from major cities with a negligible prevalence of viral haemorrhagic fever may be admitted to standard isolation facilities in district general hospitals or to infectious disease hospitals. On the other hand, febrile patients who have recently been in a rural area where viral haemorrhagic fever is endemic or who have