Lesson of the Week

Pretended paralysis requiring artificial ventilation

ANTHONY HOPKINS, CHARLES CLARKE

Pretended unconsciousness (psychogenic unresponsiveness) is an occasional problem in neurological practice.^{1,2} We report on two patients with pretended paralysis that was so profound that artificial ventilation was induced.

Case 1

A 19 year old nurse with a 12 hour history of shaking and weakness of the right hand presented to the accident and emergency department one morning. Flaccid paralysis of the right arm and hand was found. There was no sensory abnormality, but the reflexes were thought to be generally depressed. A history of two neurological episodes was obtained: she reported that at the age of 7 she had had a right Bell's palsy, and at age 17 she had had "polyneuritis" requiring ventilation for several weeks before recovery. The only residual symptom had been that she was "not always aware of passing water." Serial examination in the ward confirmed the right flaccid monoplegia and showed progressive weakness in her legs and absent ankle reflexes. She was recorded as being outwardly cheerful.

That evening, although she was fully conscious, the weakness in her legs had increased and respiratory movements were considered to be rather discoordinate. A cranial computed tomogram was normal. Lumbar puncture was undertaken in the usual left lateral position; as the needle went through the skin she said that she was about to faint and stopped breathing. Her jaw was tightly clenched, her eyes rolled up, but no convulsion was seen. Her pupils were dilated. Although her heart continued beating, the registrar undertaking the procedure gave mouth to mouth respiration. Twenty minutes later she "stopped breathing in expiration." She was given intravenous etomidate and suxamethonium and intubated and ventilated for about an hour. When seen shortly afterwards for the first time by one of us (AH) she was breathing spontaneously but rapidly and irregularly. The pupils were in the mid-range of diameter and reacted briskly to light; doll's eye movements were present. There was no motor response to calling her name or to any painful stimulus. The brachial tendon reflexes were just present, but no leg reflexes could be obtained. Although no spontaneous movements were seen, one of the nurses reported that she had observed the left hand being brought up to the endotracheal tube shortly before this examination. There were sufficient disparities in the history and examination to make a diagnosis of simulated unconsciousness likely, but the possibility of a brain stem plaque of demyelination was also considered.

The next morning the patient was wide awake with a right hemiplegia that was clearly inconsistent and hysterical; this resolved over the next few hours. A talk with her flatmate gave no indication of personal problems, and psychiatric consultation showed no obvious stress or anxiety, everything in her life being said to be "marvellous." Two days later, however, after intravenous administration of methylphenidate and sodium amylobarbitone, she told of considerable personal problems and feelings of guilt; the interview was interspersed with episodes of hyperventilation and apnoea.

After her discharge the notes from the hospital that she had attended two years before were obtained. These showed that at the age of 16 she had been admitted with a sore throat, flushed, and breathing rapidly and shallowly.

Pretended paralysis may be so severe that acute infectious polyneuritis may be mistakenly diagnosed and the patient given artificial ventilation

The notes stated: "After initial evaluation the patient gradually settled and became much calmer." Blood gas tensions on admission showed a pronounced respiratory alkalosis (PCO_2 1.93 kPa, pH 7.69). The morning after that admission she complained of "numbness over the right side of the face that did not fit any dermatome." Three weeks later she was seen at home by a consultant neurologist, who found right facial palsy, weakness of hip flexors and extensors, and depressed or absent tendon reflexes. He admitted her with a diagnosis of polyneuritis. During that admission the "generalised weakness became more severe, with the onset of bulbar symptoms and respiratory difficulties, necessitating a period of assisted ventilation." The notes were full of observations about doubts about her degree of cooperation and bizarre physical signs. After a "sudden inability to move her jaw or speak" she was intubated and ventilated for 10 days. Motor and sensory nerve conduction velocities proved to be entirely normal, as was the spinal fluid. Differences of opinion were recorded in the notes about whether this was an organic or hysterical illness, but, with the benefit of hindsight, it was probably simulated.

Case 2

A 33 year old bullion messenger was admitted through the accident and emergency department with a history of 24 hours' malaise followed by weakness and numbness of his legs. On admission he was barely able to stand. The strength of his arms was normal, but there was a global weakness of his legs. The reflexes were preserved. Although there was no objective sensory loss, he complained of numbness below the waist. No history of serious medical or psychiatric illness was obtained from the patient, his mother, his former wife, or, subsequently, his general practitioner. Postinfectious polyneuritis was diagnosed.

Twelve hours after admission he had severe weakness of the arms and legs and of flexion and extension of the neck. His voice was weak and he became breathless. Measurements of vital capacity showed a progressive fall from 3.5 litres to 2.5 and later 0.9 litre. The tendon reflexes remained. Ventilation was started because of the falling vital capacity and profound weakness. During intubation he sat up and grasped the endotracheal tube in both hands. Despite these movements, which clearly indicated that peripheral nerve function was present, he was sedated and ventilated for some 12 hours and plasma exchange carried out. Muscle power returned rapidly during the subsequent 24 hours, after which he was extubated. Results of peripheral blood studies and a chest radiograph were normal. Urinary porphyrin excretion and results of serial viral studies were negative. Nerve conduction studies, including measurements of sensory action potentials and F wave latency, also gave normal results. During the period of recovery his behaviour was inappropriate and aggressive towards the nursing staff.

Discussion

Both these patients caused considerable anxiety to the hospital staff, and, although we now are sure that these episodes of illness

Department of Neurological Sciences, St Bartholomew's Hospital, London EC1A 7BE

ANTHONY HOPKINS, MD, FRCP, consultant neurologist
CHARLES CLARKE, FRCP, consultant neurologist

Correspondence to: Dr Hopkins.

were simulated, this was not so easily apparent to the admitting physicians during the early part of their hospital stay. Anxieties were compounded by the fact that one of the patients (case 1) was a member of staff of the hospital to which she was admitted. Traditional neurological teaching emphasises that it is a mortal sin not to be well prepared to ventilate those who have a reversible acute neurological illness such as myasthenia gravis or acute postinfectious polyneuritis, which was the diagnosis first considered in each of these patients. It is in this context that the overenthusiastic management of these patients must be seen.

Previous workers have emphasised that the best pointer to the simulation of illness is inconsistencies on clinical examination, not any special investigation.¹² In case 2 the tendon reflexes were present throughout, which would have been unlikely if the global paresis was due to acute postinfectious polyneuropathy. The ankle reflexes, however, may be retained in porphyric paralysis.³ In case 1 the normally responsive pupils and doll's eye movements in the presence of total unresponsiveness were important clues, but by the time these observations had been made the patient had been intubated. After intubation both patients were observed to bring "paralysed" hands up to their endotracheal tubes; even so, the admitting physicians continued to express concern about organic illness.

Neither of these bizarre illnesses occurred in patients with a history of recognised psychiatric illness. One of the patients (case 1) was considered to be a competent student nurse by her superiors, and to have no particular problems by her flatmate. The mother, former wife, and general practitioner of the other patient thought that he was functioning normally in society. The psychiatrist who interviewed the student nurse (before abreaction), however, thought that her history was "too marvellous—like peaches and cream" and was not surprised by the extent of stress and guilt shown by abreaction. It is now recognised that conversion symptoms may arise in a wide variety of psychological (and organic) disorders and are one way of communicating distress.⁴⁵ A hysterical personality is not a prerequisite for conversion symptoms, though a histrionic personality or role playing ability may make the simulation more convincing.⁶ Marsden expressed a simple but convincing view of why patients adopt neurological conversion symptoms: "Perhaps the commonest is the patients' desire to convince or help the doctor to determine the real cause of their problems. Their anxiety leads to elaboration or exaggeration of their real deficit."⁷ Similar views have been expressed in an editorial review.⁸

Much has been written about "gain" in simulated illness. If the gain is pecuniary or some obvious evasion of duty then the simulation is said to be malingering, but if the gain is seen to be the communication of psychological distress then the illness is said to be hysterical. Another way of distinguishing the two entities is to state that malingering is a conscious act but a hysterical phenomenon is unconscious. Naish rebutted this division robustly: "How can [one] possibly tell? Since there is no test to distinguish between conscious and subconscious acts of will, how can anyone make any statement other than that the limb is disabled by an act of will? . . . What test can be applied? What measurements can be made to estimate the degree of malingering? . . . There is no test to distinguish hysteria from malingering. Since differentiation is impossible, the decision of a doctor that a person is subconsciously deceiving and therefore ill is an entirely arbitrary one which depends more upon the doctor's prejudice than his reason."⁹ We support these criticisms and for this reason prefer to say that our patients had pretended paralysis, in two of the strict senses of pretended—from *praetendere*, to hold before, to put forward; and, according to the *Oxford English Dictionary*, to offer for action, consideration, or acceptance, to profess or claim to have a right. In this sense the use of pretended makes no judgments about the subject's stance towards his illness. We acknowledge, however, that current everyday use of pretending carries connotations of feigning and defrauding, which are unfortunate. Equally, however, the use of "malingering" and "hysteria" immediately polarises the issue with no acknowledgment that there may be, and are likely to be, intermediate positions. There is an urgent need for further consideration of these aspects in "functional" disabilities, particularly those arising after accidents. We concur with Naish's

view that "a new form of words is required to express the view that a disability is not simply 'due' to an accident, but is dependent on an idea about the accident which has entered and become established in the claimant's consciousness."⁹

Merskey emphasised the concept that the manifestations of hysteria correspond to an idea in the mind of the patient.⁴ This idea may be comparatively unsophisticated, so that an idea of paralysis of an arm or leg may not extend to all possible muscle contractions in that limb.¹⁰ These ideas of illness may extend to the vegetative system. Both pulse and respiration may be affected in hysterical women, as originally noted by Galen.¹¹ Swooning remained a legitimate response to fairly trivial distress until after Victorian times in the United Kingdom. Personal experience suggests that this remains a common, culturally acceptable method of showing distress among middle aged Middle Eastern women. During the first world war shell shock was a common diagnosis; this is now recognised as an understandable psychological reaction to conflict.¹² Owing to different army medical policies in the second world war shell shock was not seen. The hysterolepilepsy and bizarre hysterical contortions of the body described by Charcot are now not seen.^{13 14} All these are examples of how the ideas of simulated illness agree with the medical ideas of the time. We live in an age of high technology medicine, so it is not surprising that these apparently severe illnesses precipitated a "high tech" response from the medical staff. Our two cases underline the potential risks of simulating illness in an energetic medical environment.

What practical advice can be given to doctors who are presented with patients like ours? The first point is that they are most likely to see patients with acute infectious polyneuritis whose initial rapidly advancing weakness, when tendon reflexes are still preserved, is diagnosed as functional. The main clinical guide is inconsistencies on serial examination; this should ideally be performed by one person with experience of neurological illness. Views about mental state are irrelevant in the acute stage as many patients with acute infectious polyneuritis are understandably agitated by their rapidly advancing illness. Once pretended paralysis has been diagnosed speedy resolution is more likely if all the hospital staff in contact with the patient adopt a cheerful and confident manner. The diagnosis should be kept under constant review.

References

- 1 Plum F, Posner JB. *Stupor and coma*. 3rd ed. Philadelphia: F Davis, 1980.
- 2 Hopkins AP. Pretending to be unconscious. *Lancet* 1973;ii:312-4.
- 3 Ridley A. The neuropathy of acute intermittent porphyria. *Q J Med* 1969;38:307-33.
- 4 Merskey H. *The analysis of hysteria*. London: Baillière Tindall, 1969.
- 5 Snaith P. *Clinical neurosis*. Oxford: Oxford University Press, 1981.
- 6 Ziegler FJ, Mayer E. Contemporary conversion reactions: a clinical study. *Am J Psychiatry* 1960;116:901-10.
- 7 Marsden CD. Hysteria—a neurologist's view. *Psychol Med* 1986;16:277-88.
- 8 Anonymous. Functional signs [Editorial]. *Lancet* 1983;ii:891-2.
- 9 Naish JM. A concept of hysteria. *Health Trends* 1982;14:15-7.
- 10 Head H. An address on the diagnosis of hysteria. *Br Med J* 1922;i:827-9.
- 11 Veith I. *Hysteria: the history of a disease*. Chicago: University of Chicago Press, 1965.
- 12 Hurst AF. *Medical diseases of war*. London: Edward Arnold, 1918.
- 13 Charcot JM. *Clinical lectures on disease of the nervous system*. London: New Sydenham Society, 1889.
- 14 Massey EW, McHenry LC. Hystero epilepsy in the nineteenth century: Charcot and Gowers. *Neurology (NY)* 1986;36:65-7.

(Accepted 3 February 1987)

Can antihistamines prevent susceptible children from developing asthma after a cold?

I do not think that antihistamines are useful in preventing asthmatic children from developing wheezy symptoms in the context of upper respiratory tract infections. Indeed, antihistamines have little part to play in the management of asthma. I much prefer to rely on bronchodilators, inhaled sodium cromoglycate, or inhaled topical steroids. It seems useful sometimes to use salbutamol or terbutaline when wheezy children are affected by coryzal symptoms, giving the treatment by whatever route the child is most used to. For those already taking cromoglycate, increasing the dose at the appropriate time, during or at the beginning of infection, seems to have an appreciable and useful affect.—C B S WOOD, professor of child health, London.