Hysteria: medicine, psychiatry and misdiagnosis*

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1 Fainting fits and Freud

In the second volume of her autobiography, *The Spiral Staircase*, the writer and former nun Karen Armstrong gives an account of the mysterious fainting attacks she began to suffer soon after she entered her convent in 1962 at the age of seventeen:

It began with the smell. It was a sweet and sulphury aroma, reminiscent of bad eggs and giving off an aura of imminent menace. Like any odour, it was also intensely evocative, I recognised it immediately. This was how it always started. In the convent I had several times been assailed by this strange smell, had looked around me for a cause and found the world splintering around me. The sunlight, the flickering candles of the altar and the electric light seemed to oscillate crazily; there would be a moment of pure nausea, and then nothing: a long, long fall into emptiness.¹

While she was still a nun Karen Armstrong had attacks like this on four or five occasions. She was reprimanded by her superiors for ‘emotional indulgence, exhibitionism … weakness of will … “You must pull yourself together, Sister,”’ Mother Frances had concluded, tight-lipped.’

It was never suggested that she should see a doctor. Armstrong recalls that ‘fainting meant only one thing: hysteria.’. She goes on to recall how she had imbibed this ethos at her convent school before she ever became a nun:

I assumed that even though I might not be feeling especially upset, I was displaying some subconscious need for notice, love or intimacy. The black-outs, I concluded, must be a bid for attention. And yet, I reflected wryly, my unconscious mind must be very slow on the uptake. You would think that by now it would have learned that, far from eliciting the tender concern I craved, the fainting simply inspired anger and disdain.²

Seven years later, after having been sent by her convent to Oxford University to read English literature, Karen Armstrong finally left the religious life. But although she had freed herself from its demands for narrow conformity, her new secular ‘liberation’ did not bring an end to her fainting fits.

Indeed, they seemed to become worse, and on one occasion the familiar stench was followed by a vision in which she felt a man was standing beside her – an aged, senile mask with empty eyes. She became gripped by fear:
I did not know who, what or where I was, but was aware only of my extreme terror, a cold sickening dread that made everything around me seem brown rotten and repulsive, because it had no meaning.³

This was not an isolated experience. On another occasion she lost contact with reality while shopping in Marks and Spencer and only came to her senses again after she had walked half a mile to Radcliffe Square where she found herself contemplating the perfect dome of Oxford’s Radcliffe Camera, ‘an image of wholeness and harmony’.

She was under no illusion that these mysterious episodes were supernatural visitations. She realised that something was wrong. As the episodes became more frequent she became frightened. What was happening was threatening to destroy her entire life. For the first time she consulted a doctor. Her GP agreed that she was not well and talked about how common ‘anxiety attacks’ are. He said he knew a good specialist at Littlemore Hospital – a psychiatric centre.

This was in 1970. After seeing the consultant, she was referred to a series of registrars. They attempted to probe her inner life and to talk to her about the emotional and sexual issues that they were convinced she was trying to avoid. She knew she could not expect a miracle overnight:

What did I know about psychiatry and the mysteries of the human mind? If I persevered I might well find that everything suddenly fell into place. But then I would be overtaken by a queasy sense of *déjà vu*. This was exactly the sort of reasoning that I had used in the convent, and look where it had got me.

The psychotherapy continued. But so did the fainting fits which became stranger and more worrying. Karen Armstrong began to ‘miss’ entire episodes in her life, as on one occasion where she went downstairs and made herself a cup of coffee and then, half an hour later, did the same thing again having completely forgotten the previous occasion.

On another occasion she found herself unexpectedly sitting in the English faculty library but had no recollection at all of how she had got there. ‘These were not bouts of forgetfulness, but lapses into a form of unconsciousness when I was quite *compos mentis* enough to boil a kettle or cross a road safely.’ For her psychiatrist at the Littlemore Hospital, however, the new symptoms were just another smoke-screen:

‘So in future,’ Dr Piet said pointedly, as he drew the session to a close, let’s not waste any more time. Let us have no more agonising about unimportant details. Let’s get right in there to the basic difficulties, which are the cause of all these symptoms. And do me a favour – let’s have no more histrionics about perfectly normal absent-mindedness. After all, Karen,’ he smiled to soften his bleak conclusions, ‘it’s not as if you ever did anything very dreadful at these times. Making a cup of coffee? Going to a library? Come on! You,
of all people, with your gothic imagination, can do better than that! Let’s see what you’re really like when you lose control. Surprise me!

A few weeks later, in the autumn of 1971 Karen Armstrong did just this; she woke up in hospital, having taken an overdose of the sleeping pills Dr Piet had prescribed for her insomnia. She could remember very little of what had happened and appears to have taken an overdose during the course of another of her mysterious ‘lapses’.

Before long she found herself once again at Littlemore Hospital, but not on this occasion as an outpatient:

A short, middle-aged man with a beer belly plonked himself down on the sofa beside me. ‘Cheer up, darling. It’s not so bad! In a day or two it’ll come to seem like home.’

That was precisely what I was afraid of. My new companion began to tell me a long convoluted story about how he had ended up in what he called the ‘loony bin’: headaches, doctors, inconclusive medical tests, and then six months in the Littlemore for depression, though, as he explained, he’d never really felt depressed. But that, the doctors had informed him, was really the cause of his trouble. If he’d allowed himself to feel depressed, he wouldn’t have had the headaches. And how were these headaches, after six months of group therapy, individual psychotherapy and Valium?

‘Shocking.’ He shook his head. ‘I don’t know. I really don’t. Nothing’s shifted them.’ He was plainly bemused by the whole psychiatric approach. How could he be depressed without realizing it? I wondered how he would get on with Dr Piet, and looked at him thoughtfully. Perhaps there was something physically wrong with his brain? But now he was stuck in here and branded a ‘nutter’ (his term), how would anyone ever find out? 4

Rescued by a friend from being shut up for weeks in a psychiatric hospital, Karen Armstrong returned to her lodgings in Oxford. She eventually weaned herself from her psychiatrist and moved to London to take up a university teaching appointment. But her fainting fits and her strange lapses of consciousness persisted. On one occasion she was meant to be at the theatre with friends but woke up in the Tate Gallery, having walked along the Embankment from Charing Cross. She had no memory of the previous three hours at all.

In February 1976, after three years in medical limbo and almost six years after she had originally consulted her doctor, she was on her way home to Finchley via Baker Street tube station. Just after she had gone through the ticket barrier she was hit by the familiar stench. But this time her experience was more intense and she lurched onto some railings. Then she suddenly felt all the pieces of a pattern fuse into a harmonious whole: ‘I had entered a new dimension of pure joy, fulfilment and peace: the world seemed transfigured, and its ultimate significance – so obvious and yet quite inexpressible – was
revealed. This was God. But no sooner had I realized this than I began to fall down that familiar dark tunnel into oblivion.’

Two hours later the doctor in the Accident and Emergency department of the Middlesex Hospital, where she had been taken, told her that she had suffered an epileptic seizure and that he had arranged an appointment for her in the neurology department:

I truly did not know whether to laugh or cry. I was an ex-nun, a failed academic, mentally unstable, and now I could add ‘epileptic’ to this dismal list … Even God, for whom I had searched so long, was simply a product of a faulty brain, a neurological aberration. I went to bed that night in despair.5

When she saw Dr Wolfe, the neurologist, he told her that the electro-encephalograph had clearly revealed a small but definite abnormality. He said that she had now had a grand mal epileptic fit and then elicited from her the story of her ‘fainting fits’ and how these had been dismissed by the nuns as due to some emotional disturbance, of how she had had frightening hallucinations of a grotesque face and had experienced lapses when she had done things without realising it.

In response to her saying that the blackouts had begun when she was eighteen, Dr Wolfe explained that ‘This condition quite often appears in late adolescence – with the hormonal changes, you see.’

Having explained that all the features she has described were ‘classic symptoms of temporal lobe epilepsy’, Dr Wolfe frowned slightly. ‘But forgive me, I’m puzzled: this must have been very alarming for you. Why on earth did you not take these symptoms to a doctor?’

I explained about Dr Piet, the hospitalization, the therapy and the drugs. Dr Wolfe covered his eyes with his hand, shook his head, and looked up again. ‘Do you mean to tell me,’ he asked, with devastating calm, ‘that you were treated by psychiatrists for over three years – men and women who were all fully qualified doctors – that you presented these symptoms, and that none of them, not one, in all that time suggested that you have an EEG?’

‘No, they didn’t.’ I was beginning to be invaded by an enormous astonishment, a confusion of feelings that included anger but also a relief so great that I was close to tears.

Dr Wolfe uttered that explosive sound that novelists used to transcribe as ‘Pshaw!’ ‘It’s not even as though temporal lobe epilepsy were an obscure condition,’ he snapped. ‘It’s the most common of all the focal epilepsies, and very well documented. And, as I say, you are almost a textbook case!’ He tailed off. Then his face cleared, and he wrote out a prescription for the drug that, he hoped, would eliminate the demons that had haunted me for so long.
As I got up to go, he looked at me sternly. ‘I don’t think you need waste any more of your time with these psychiatrists.’ He made the word sound like an obscenity. ‘No amount of talking about your problems will make the smallest impression on your condition, and I’m very sorry indeed that you have had to wait for so long before getting adequate medical help. By the way,’ he added, as I reached the door, ‘it’s interesting that you were once a nun. People with temporal lobe epilepsy are often religious!’
2 Freud and the diagnosis of hysteria

Karen Armstrong’s account of the ‘psychologisation’ and consequent misdiagnosis of a real organic illness is one of many comparable stories. The further back in history we go, the more likely it is that we will encounter similar medical mistakes – although it is perhaps only in the last four decades or so that such misdiagnoses have been at all likely to be recognised by the psychiatrists and neurologists who made them. Before that, many of the diseases from which patients were actually suffering had not even been identified by medical science.

The particular misdiagnosis suffered by Karen Armstrong may well have had a celebrated precursor. When Freud published his Studies on Hysteria in 1895 one of the cases he described was that of Katharina. Freud’s story of Katharina is one of the most simple and engaging of all his case histories. It is also the most unusual since Katharina was not one of Freud’s paying patients, but the daughter of an inn-keeper who approached him while he was on holiday in the Alps in 1893. Having deduced that Freud was a doctor from his signature in the visitors’ book, Katharina consults him while he is gazing at the mountain scenery:

‘The truth is, sir, my nerves are bad. I went to see a doctor in L— about them and he gave me something for them; but I’m not well yet.’

So there I was with the neuroses once again – for nothing else could very well be the matter with this strong, well-built girl with her unhappy look.

Freud’s diagnosis is made almost instantaneously. Having ruled out any physical ailment on the grounds of Katharina’s robust appearance, he elicits a description of her symptoms and learns that she suffers from periodic fits:

‘It comes over me all at once. First of all it’s like something pressing on my eyes. My head gets so heavy, there’s a dreadful buzzing, and I feel so giddy that I almost fall over. Then there’s something crushing my chest so that I can’t get my breath.’

‘And you don’t notice anything in your throat?’

‘My throat’s squeezed together as though I were going to choke.’

‘Does anything else happen in your head?’

‘Yes, there’s a hammering, enough to burst it.’

Katharina goes on to describe how these attacks are accompanied by anxiety and by a recurrent hallucination in which she sees ‘an awful face that looks at me in a dreadful way’. She tells Freud that the attacks began two years ago but that she has no idea where they came from. Freud identifies Katharina’s fits as ‘anxiety attacks’ and assumes that
they are hysterical manifestations of an emotional trauma. He claims that he then elicited from Katharina the story of how, two years ago when she was sixteen, she had chanced to observe her father making love to a young servant-girl in his room. She is then said to have recalled an earlier occasion on which her father had made sexual advances to her which she had repulsed. Although she had at first had no recollection of the origin of her fits she now reportedly accepts Freud’s suggestion that they were precipitated by the scene with the servant-girl. According to Freud’s account, she even claims to remember experiencing some of the characteristic symptoms of her fits immediately after observing the scene.

As those who are familiar with modern Freud scholarship will be aware, Freud’s reports of what his patients have told him should be treated with considerable scepticism; very often such accounts turn out on investigation to be based on Freud’s speculative ‘reconstructions’ rather than accurate reports of what the patient has actually said.

But even if we accept his report as accurate, there is every reason to doubt Freud’s dogmatic insistence that Katharina was suffering from ‘hysteria’. The most straightforward objection is that, during his chance encounter with her on a mountain top, Freud was simply not in a position to make any diagnosis. More importantly still, every one of the symptoms she describes would be compatible with temporal lobe epilepsy. Mild temporal lobe seizures do not involve loss of consciousness and the symptoms reported by those who suffer them sometimes include recurrent hallucinations involving a ‘spectral face’ just as they did in Karen Armstrong’s case. Indeed we might well compare Katharina’s description of what happened inside her head – ‘There’s a hammering, enough to burst it’ – with one of the sensations reported by Armstrong. In describing the first occasion on which she saw the grotesque face, Karen Armstrong recalls that ‘My brain felt as though a cosmic potato masher was pounding it’. In the earlier version of her memoir she writes of a ‘tight feeling around my head, pressing down on my temples like a steel clamp’. It may be noted also that the onset of Katharina’s symptoms during late puberty is also a common part of the clinical picture of this kind of epilepsy.

Even if Katharina had come to Freud’s consulting rooms in the 1890s as a paying patient and he had physically examined her he would have been in no position to make the diagnosis which now seems to be the most probable explanation of her fits. For temporal lobe epilepsy was not widely recognised until the 1930s. The electroencephalogram, which was the only way of positively testing for any form of epilepsy, was not invented until 1929, and was not in general use until the 1940s. For this reason temporal lobe epilepsy, with its strange psychological-seeming symptoms, was repeatedly misdiagnosed as hysteria throughout the nineteenth century and the early part of the twentieth.

If Katharina was one victim of such a misdiagnosis, it seems possible that the patient Freud referred to as ‘Miss Lucy R’ was another. She was sent to Freud after she developed recurrent olfactory hallucinations centring on the smell of burnt pudding.

In the latter part of the nineteenth century it is understandable that Freud should turn to a psychogenic hypothesis in order to explain the onset of hallucinations. Since Freud’s time, however, it has become clear that hallucinations are almost always the product of
organic factors rather than psychological ones. This applies just as much to smells as it
does to any other form of hallucination. By the end of the nineteenth century John
Hughlings Jackson, the British neurologist whose pioneering work on epilepsy paved
the way for the eventual recognition of temporal lobe epilepsy, had already come to the
conclusion that ‘a sudden and temporary stench in the nose with transient unconscious-
ness’ was an epileptic event. His conclusion has been confirmed by modern neurolo-
gists. Henri Gastaut, for example, describes olfactory seizures as consisting in ‘a sensa-
tion of an odour which the patient usually considers neutral or disagreeable – e.g., the
smell of gasoline, of gas, sulphur, formalin, bad eggs, faeces, etc.’ Doris Trauner, who
observes that there are almost as many types of complex partial seizures as there are
patients with epilepsy, comes even closer to describing Lucy R’s main symptom when
she writes that ‘Some patients complain of intense olfactory hallucinations that in most
cases are unpleasant (e.g., a smell of rotten eggs or burnt toast).’

Once again there is a resemblance to Karen Armstrong’s case and once again it is at least
possible that Lucy R’s hallucinations were manifestations of temporal lobe epilepsy.
Whether or not these hallucinations were, strictly speaking, epileptic in character, it
seems virtually certain that their explanation was to be found in neurology rather than
psychology. It may well be significant that Lucy R had developed her olfactory halluci-
nations only after having been treated for some time for a chronic nasal infection which
was associated with caries of the ethmoid bone – the structure between the nose and the
skull. She subsequently lost her sense of smell entirely and it was at this point that the
hallucinations began. One organic alternative to the diagnosis of temporal lobe epilepsy
is that she was suffering from parosmia (or phantosmia) occasioned by damage to one of
the olfactory nerves.

Characteristically, however, Freud did not allow for the many gaps in medical knowl-
dge which then existed. Instead he made an a priori assumption that Lucy R was suffer-
ing from hysteria and embarked upon his usual quest for the trauma which supposedly
precipitated her condition. By way of an extraordinarily elaborate chain of causality, he
eventually traced the smell of burnt pudding to her disappointment at being rebuffed by
the employer with whom she had fallen in love.

The cases of Katharina and Lucy R were by no means the most significant misdiagnoses
associated with psychoanalysis. For it is now clear that Anna O, the very first psycho-
analytic patient (who was treated by Freud’s colleague Joseph Breuer) was the victim of
the most important of all psychoanalytic misdiagnoses.

Anna O. was a young woman who had fallen ill while nursing her father, who was
suffering from a subpleuritic tubercular abscess. Not long before her father died she
developed a severe cough and Breuer was called in to help. Subsequently she developed
rigid paralysis of the extremities of the right side of her body, a convergent squint,
disturbances both in her vision and in her hearing. At a very early stage of her illness
she had begun to experience brief lapses of consciousness or ‘absences’. During these
spells she would stop in the middle of a sentence, repeat her last words and then, after a
short pause, go on talking. Gradually these spells of confused behaviour grew longer
and more severe, and during her ‘absences’ she would hallucinate and behave in a
disturbed manner, shouting abusively, throwing cushions at people and tearing buttons off her clothes. If anyone came into the room during these states, Anna would complain of having ‘lost’ some time and would remark upon the gap in her train of conscious thoughts. She also developed a number of disorders of speech; eventually she lost her ability to speak her native language and would converse in English instead, apparently without realising what she was doing.

Although Breuer briefly considered a diagnosis of meningitis, he eventually opted for a diagnosis of hysteria and began the search for the precipitating traumatic events which would lead eventually to the creation of psychoanalysis.

It is now clear, however, that Anna O, although she was evidently emotionally distressed by her condition, was not suffering from a psychologically induced illness at all. Each of her most significant symptoms corresponds to a specific kind of brain lesion or a recognisable pattern of brain pathology. More importantly still, many of these symptoms are typical components of complex partial seizures – which is to say a particular form of temporal lobe epilepsy.\(^{10}\)

It is in the psychoanalytic tradition of misdiagnosis, created by Breuer and Freud at the end of the nineteenth-century, that we will find the immediate ancestry of the kind of diagnostic error perpetrated on Karen Armstrong during the 1970s. However, it would be quite wrong to lay such errors purely at the feet of the founders of psychoanalysis.

For psychoanalysis would never have been created at all, and would certainly never have achieved any degree of medical credibility, had its own tradition of psychogenic misdiagnosis not been deeply rooted in mainstream medicine.

The purpose of the essay which follows is to examine how it was that a tradition of misdiagnosis actually became established within the profession of modern medicine, and how, in spite of significant challenges to it made by a number of physicians – including the psychiatrist Eliot Slater – this tradition has not only survived but has actually grown stronger in recent years.

If it is the case, as I argue in this essay, that modern medicine has indeed incorporated a tradition of misdiagnosis into its own orthodoxies, then the consequences are potentially very serious indeed. Examples of individual cases comparable to that of Karen Armstrong will be found throughout the essay. But in its closing sections I have turned to consider the systematic errors which appear to be associated with the way in which the medical profession – or very significant sectors of it – has tended to react to complex, widespread, and extremely serious medical conditions such as Lyme disease and ME/CFS (chronic fatigue syndrome). The accuracy of diagnoses made in relation to these illnesses potentially affects millions of people.

It is because of the very serious implications of my argument that I have felt it necessary to expand what began as a brief article into a much longer essay, and to retrace in this essay in a broader context some of the steps I originally took in writing about the history of hysteria in Why Freud Was Wrong: Sin Science and Psychoanalysis. In the course of doing
this I have revised some of my views and tried to correct some errors of emphasis and interpretation while preserving the underlying argument. I hope that by focusing on modern diagnostic dilemmas rather than on the history of psychoanalysis, the significance of this argument and its relevance to the practice of medicine will be more readily visible than it was in that earlier and much longer book.
3 The case of Ms A

FORTY YEARS AGO, in May 1965, the British Medical Journal published an editorial entitled ‘Eclipse of Hysteria’ in which it suggested that hysteria, since it is not an illness in the accepted sense of the word, or even a well-defined clinical syndrome, might soon become redundant as a diagnosis: ‘Will “hysteria” share the fate of “hypochondriasis”? In days past this was a common diagnosis, but it is hardly ever made today, though hypochondriacal behaviour is common in all types of patients.’

The editorial had been written in response to the celebrated attack on the diagnosis of hysteria by the psychiatrist Eliot Slater which appeared in the same edition of the BMJ in which Slater had described the diagnosis as ‘a disguise for ignorance and a fertile source of clinical error’.

In view of the trenchancy of Slater’s attack and its virtual endorsement by the BMJ, it might well be thought that by now the diagnosis in question might have been relegated to the pages of medical history. In fact this is not the case. Consider, for example, the following:

NEW APPROACHES TO CONVERSION HYSTERIA

Patients with hysterical conversion, now called conversion disorder by the main US psychiatric glossary, often present with striking neurological symptoms such as weakness, paralysis, sensory disorders, or memory loss, in the absence of any pathology that could be responsible. Most patients will be referred to a neurologist or psychiatrist after consulting their family doctor.

As many as 4% of those attending neurology outpatient clinics in the United Kingdom have been estimated to have conversion disorders. Similar rates have been reported for both in- and outpatient clinics in other European countries.

These words do not come from some obscure medical backwater. They are the opening paragraphs of an editorial which appeared in the summer of 2000 in the same journal which had published Slater’s attack on the diagnosis 35 years earlier.

The appearance of this editorial in the BMJ is just one sign of a remarkable development – namely that, during the last twenty years (and particularly the last ten), the diagnosis of hysteria, far from disappearing, has been all but rehabilitated. Indeed, only two years earlier, another editorial comment in the BMJ had appeared under the heading ‘Hysteria can be diagnosed with confidence’. This was in response to its publication of an article entitled ‘Slater revisited’, which appeared to show a fall in the rate of misdiagnosis in this area. The boldness of the headline was only partly qualified in the body of the editorial, which advised physicians that ‘after careful investigation hysteria can be diagnosed with confidence’.
The rehabilitation of the diagnosis is by no means complete for it would seem that the term ‘hysteria’ is now rarely used by doctors in order actually to formulate their diagnosis. Most doctors recognise that the term is offensive not only to their patients but also to many of their peers with the result that hysteria has sometimes been aptly described as ‘the diagnosis that dare not speak its name’. But the term is sometimes used out of the patient’s earshot (as the BMJ editorial illustrates) and the concept of hysteria is certainly alive and well, hiding beneath more neutral-sounding terminology such as ‘conversion disorder’ and ‘functional symptoms’.

Does this mean that, perhaps because of the extraordinary medical progress brought about by Magnetic Resonance Imaging and other forms of brain scan, the dangers of misdiagnosis alluded to by Slater now belong to medical history?

One tragic case history should suffice to indicate that this is not the case. This case history, reported in the American Journal of Psychiatry in April 2002 by H. Brent Solvason and his colleagues, concerns Ms A, a 49-year-old white American woman who worked as a senior communications engineer.

Ms A had grown up as the youngest of three children. She described her relationship to her family as ‘distant’ and her illness seemed to coincide with an almost complete estrangement from her parents, which had come about after she had entered into a sexual relationship with another woman:

She noted that she had always been sexually interested in women, but until just before the onset of the symptoms leading to hospitalization, she had never had a same-sex romantic relationship. The revelation of her homosexuality to her family was poorly received. She described it as ‘cold’, and she felt criticized and ashamed.

Her illness began with acute lower back pain, which did not respond to the attentions of a chiropractor. She then began to suffer from a tremor all over her body and weakness in the muscles of her legs which made it difficult for her to walk. At this point she was subjected to a battery of physical tests, including MRI scans of her brain and spine, a lumbar puncture and liver tests. Although these revealed some minor abnormalities there was nothing that seemed to explain the severity of her symptoms. A large number of other tests and investigations proved negative.

When her symptoms persisted she was seen by a specialist who diagnosed atypical multiple sclerosis. Five months after the onset of her illness, however, she developed double vision, frequent urination and had difficulty in swallowing; this was in addition to her tremors, back pain and difficulty with walking. She was seen by another neurologist whose differential diagnosis included Creutzfeldt-Jacob disease. She was then seen by the colleague of a national authority on prion disease, who felt that Creutzfeldt-Jakob disease was a ‘low-probability’ diagnosis. It was recommended that her blood mercury level should be tested but this test was also negative.
The difficulties resulting from her illness led eventually to the breaking up of her relationship with her lover. It was at this point, with all the tests for organic illness returning negative results, that Ms A’s physician began seriously to consider a psychiatric diagnosis. Ms A was now subjected to a battery of psychiatric tests. Although the results of most of these were also negative, she did register as suffering ‘elevated depression’. Because her physical symptoms were difficult to explain, she was given the diagnosis of ‘major depression and conversion disorder’ and sent to a psychiatric hospital. Here she was seen by a specialist in dissociative disorders and psychosomatic illness. He hypnotised Ms. A and noted that she was highly hypnotisable and that, under hypnosis, her symptoms improved. This appeared to confirm the psychiatric diagnosis.

Eventually, with her physical symptoms persisting, and amidst new reports of hallucinations, the psychiatrists decided to proceed with ‘a behaviourally oriented rehabilitation program’ which involved ‘aggressive physical therapy, occupational therapy, and participation in groups and activities, with the focus on treating Ms. A’s functional disability’. In a deliberate and orthodox attempt to place this presumed somatizer in a double-bind, her psychiatrists told her (untruthfully) that if she did indeed have an organic disease, her condition would improve.

In fact her condition continued to deteriorate. She began to throw her head back while eating, causing herself to choke. She would sometimes smear her food on her face and when the nursing staff ignored her she would respond with low moans. Presented with Rorschach ink blots, she would tell long convoluted stories, using what appeared to be neologisms (for example ‘torcle bug’ and ‘buldar’). When questioned, she would laugh and say that she had made these terms up by combining words.

Her condition continued to deteriorate and, because of her severe difficulties with swallowing and chewing, the psychiatrists who were in charge of her case decided to feed her through a nasal tube. Within 24 hours she removed this herself and did the same with its replacement.

Because they did not want to engage in more invasive measures in order to feed ‘an individual with a psychosomatic illness’, the psychiatrists decided to try feeding her normally again. That day Ms A ate both lunch and dinner and did so without any difficulty that was noted by the nursing staff.

However, about an hour after she had finished her dinner, Ms A was heard choking. She was examined by nursing staff but it would seem that because they could find no sign of food in her throat, they presumed that her choking was functional or hysterical.

Half an hour later Ms A was found sitting rigid in her chair. Her face was blue. She was not breathing and had no pulse. She had suffered a cardiac arrest. Although she was resuscitated and taken to the Intensive Care Unit, an EEG found her to be brain dead, and after a conference with her family, her life support was switched off.
It was only at this point that psychiatrists read the result of an EEG which had been taken earlier in the week. This revealed abnormalities and specifically raised the possibility of a ‘rapid neurodegenerative process’.

A full autopsy revealed that Ms A had died as a result of choking on food. Her brain tissue appeared normal to the naked eye. But under the microscope it showed signs of extensive pathology caused by prion infection. Throughout her illness, it was clear that Ms A had in fact been suffering from Creutzfeldt-Jakob disease.
4 A letter to the editor

It might well be thought that that the psychiatrists who cared for Ms A would treat the story of what happened to her, and her eventual post-mortem diagnosis, as nothing more and nothing less than a shocking and salutary lesson about the dangers of making the diagnosis of ‘conversion disorder’. They might even use their experience to question whether this was a genuine diagnosis, or a pseudo-diagnosis as many, including Eliot Slater, have argued. However, from their published paper on Ms A, it is clear that this is not the case. While conceding that their patient was suffering principally from a neurological disorder they attempt to rescue their misdiagnosis and offer their error as a genuine insight into Ms A’s condition:

Given the changes seen in Ms. A’s personality, it would be expected that she would have had a diminished capacity to use her usual coping strategies at a time of extreme duress. Because of this, we believe that she communicated distress through an elaboration of symptoms (illness behavior) during the course of her illness. We base this idea partly on the psychometric findings but also on the important psychosocial stressors that preceded the onset of symptoms. In our opinion, this illness behavior as well as her somatization of distress contributed to a confusing clinical picture, especially given the lack of evidence of a medical or neurologic illness until just before death [italics added].

This attempt to rationalise a medical mistake and to present error as insight is by no means the end of the story. Soon after the ‘case-conference’ paper describing Ms A’s case was published in the American Journal of Psychiatry two English physicians began to prepare a response to it which would eventually appear in the journal in February 2003. The physicians in question were Jon Stone, a neurologist at the University of Edinburgh, and Michael Sharpe, Professor of Psychiatry at the University of Edinburgh. The letter which they submitted to the journal also carried the name of Martin Zeidler, a specialist in Creutzfeldt-Jakob disease.

When their letter is read carefully it is not clear that they accept that the diagnosis of conversion disorder which had been given to Ms A was a mistake at all. The mistake, it would seem, was the failure to recognise the presence of neurological disease as an aspect of her condition: ‘Clinical vigilance for a missed diagnosis of neurological disease,’ they write ‘in cases of conversion disorder is essential’ [italics added]. This reading of their position is reinforced by what they say elsewhere:

The authors [of the paper about Ms A] usefully highlighted how organic disease may be important in generating symptoms that are medically unexplained, either directly by effects on brain function or because of more complex behavioral responses to illness. They also showed how our current somatoform classification leaves little room for a dual diagnosis of organic and functional disorder. This unsatisfactory either/or philosophy is perhaps one rea-
son why doctors are reluctant to diagnose conversion disorder in the first place [italics added].

Their closing words do appear to concede that Ms A’s psychiatrists made a mistake. But the thought that this mistake might actually lead to physicians exercising more caution about making the diagnosis of conversion disorder is clearly one which Stone, Sharpe and Zeidler find unacceptable:

It would be a shame, however, if this case conference reinforced the erroneous idea that the development of neurological disease in such cases is the norm. Failure to make a positive diagnosis of conversion disorder can have serious adverse consequences. The patient may be denied appropriate treatment management that vitally depends on persuading him or her that the symptoms are reversible and not due to disease. We should not withhold the diagnosis simply because we occasionally get it wrong.

The fact that this letter was written at all is testimony to the continuing prestige and apparent usefulness of the diagnosis of hysteria – in some quarters at least. Yet it leaves unaddressed the most disturbing aspects of the original article.

What ought to be recognised as one of the most shocking features of the case history of Ms A is the inability of her original physician, and the psychiatrists to whom she was subsequently referred, to ‘see’ their patient, or the symptoms from which she suffered, in any but the most perfunctory sense of the word. Instead of relying on their own eyes and their own observations to register the reality of her illness, they evidently chose to place all their faith in the results of technologically sophisticated tests. When the machines they were relying on failed to show that their patient was ill, they concluded that she was physically well and that her symptoms were the product of some occult process taking place mysteriously somewhere inside her. Their belief that her various, increasingly acute physical symptoms could all somehow be ‘produced’ by the process of somatization or conversion was not a rationally tested belief, nor was it empirically verifiable. It depended entirely on their faith in an article of medical ‘knowledge’ which they had apparently accepted on trust from their elders.

It is this same article of received medical wisdom which informs the letter submitted by the three Edinburgh-based physicians in response to the case history.

The willingness of a group of editors to publish this response as a contribution to clinical medicine suggests that the vast legacy of medical error bequeathed to modern medicine by Charcot and Freud has still not been recognised for what it is. Eliot Slater’s 1965 paper ‘Diagnosis of ‘Hysteria’” may well have exercised a salutary influence on the profession, but it would appear that this influence is now waning.
5 Eliot Slater

Eliot Slater developed his sceptical attitude towards hysteria only after a great deal of research. This included a study of eighty-five young or middle-aged patients who had received the diagnosis of ‘hysteria’ at the National Hospital for Nervous Diseases in London during the years 1951, 1953 and 1955. The most important and the most surprising findings of this study were, as he himself put it, ‘the gravity of the after-history and the frequency of misdiagnoses’.

During a follow-up period which averaged nine years, twelve of the eighty-five patients had died, fourteen had become totally disabled and sixteen partially disabled. Most of these cases of death or disability were due to organic illnesses which had been mistaken for ‘hysteria’. Among the conditions which had been misdiagnosed either by neurologists or by psychiatrists – including Eliot Slater himself – were three cases of vascular disease, three of tumour and a number of cases where supposedly hysterical black-outs and fits were subsequently rediagnosed as epileptic.

Four of the deaths were due to suicide, but in two of these instances the patient had suffered from organic diseases which had not been diagnosed by doctors at the National Hospital. One was a man suffering from various symptoms, including pain in the legs, unsteadiness of gait and impotence. Although Slater himself had diagnosed ‘hysteria’, the man was later admitted to another hospital and found to be suffering from disseminated sclerosis. In another case a woman who complained of severe headaches and poor vision was held to be suffering from ‘drug addiction and hysteria’. She was transferred to the Maudsley Hospital, from which she discharged herself after two weeks, her illness having been diagnosed as ‘conversion hysteria’. Two years later she died of a brain tumour.

After discussing these and many less serious misdiagnoses and placing them in the context of medical history, Slater comes to the conclusion that the diagnosis of hysteria has no validity whatsoever:

Looking back over the long history of ‘hysteria’ we see that the null hypothesis has never been disproved. No evidence has yet been offered that the patients suffering from ‘hysteria’ are in medically significant terms anything more than a random selection. Attempts at rehabilitation of the syndrome, such as those by Carter and by Guze, lead to mutually irreconcilable formulations, each of them determined by their terms of reference. The only thing that hysterical patients can be shown to have in common is that they are all patients. The malady of the wandering womb began as a myth, and as a myth it yet survives. But, like all unwarranted beliefs which still attract credence, it is dangerous. The diagnosis of ‘hysteria’ is a disguise for ignorance and a fertile source of clinical error. It is, in fact, not only a delusion but also a snare.17.

It may be noted that one of the objections which Slater makes to the diagnosis of hysteria is what he terms ‘the logical indefensibility of making diagnoses by exclusion’. In this
connection he appeals for support to one of Charcot’s near contemporaries, the English neurologist Charlton Bastian. According to Bastian the diagnosis of ‘hysterical paralysis’ is ‘a negative verdict’. By making it, Bastian says:

We merely imply that we think we are warranted in saying that we think that the case before us is not one which has been caused by any gross organic disease of the nervous system, and that no causative changes therein would be detectable, even with the aid of the microscope. We leave entirely unanswered the other ordinary problems which go to constitute a complete diagnosis; nothing is implied, that is, as to the part of the nervous system which is at fault, or as to the nature of the process by which its functional activity has been impaired … [Moreover] the more slender and insecure is the practitioner’s knowledge of nervous diseases, the more prone is he to regard … strange or puzzling cases as instances of ‘hysterical paralysis’.  

Slater’s ultimate objection to the use of hysteria as a diagnosis rested not so much on the diagnostic mistakes he was able to show it had led to. It rested on the observation that the meaning of ‘hysteria’ seems constantly to fluctuate. Although he was prepared, with various caveats, to accept that it might be permissible to refer to a symptom as ‘hysterical’ he did not accept what he termed the ‘substantival view’ of hysteria:

… [T]o suppose that one is making a diagnosis when one says a patient is suffering from ‘hysteria’ is, as I believe, to delude oneself. The justification for accepting hysteria as a syndrome is based entirely on tradition and lacks evidential support. No clearly definable meaning can be attached to it; and as a diagnosis it is used at peril. Both on theoretical and practical grounds it is a term to be avoided.

This sceptical attitude towards hysteria undoubtedly exercised a profound influence on the entire medical profession throughout the next two decades. It is clear, however, that this influence is now waning. Indeed Professor Michael Sharpe, the co-author of the letter to the American Journal of Psychiatry, is one of a number of prominent medical figures who have consistently argued for a rehabilitation of the diagnosis of hysteria – under the modern rubric of ‘conversion disorder’.

In recent years Professor Sharpe has frequently collaborated with the neurologist Jon Stone, as he does in his AJP letter. In several papers, some of them published in the BMJ, Sharpe, Stone and their colleagues have thus argued that the rate of misdiagnosis reported by Slater for the ‘hysteria’ diagnosis has fallen dramatically in the decades since he wrote, and that therefore Slater’s views should be ignored.

In their paper ‘Functional Symptoms in Neurology: Diagnosis and Management’ (Advances in Clinical Neuroscience and Rehabilitation, Vol 4, Issue 6, Jan-Feb 2005, 8-11), to cite but one recent example, Sharpe, Stone et al consider one of the objections commonly made to the diagnosis of conversion disorder or ‘functional’ illness: ‘Perhaps many [patients so diagnosed] will develop a disease-cause for their symptoms’.
Their answer to this objection is as follows:

Neurologists don’t generally worry about this but psychiatrists do, largely because of an influential paper by Slater published in 1965. Slater was wrong and the misdiagnosis rate at follow up for patients with functional neurological symptoms in modern case series is consistently under 10% and usually around 5%. This is the same rate for other neurological and psychiatric conditions such as MS and schizophrenia.

It is indeed the case that a number of surveys carried out in the last decade have reported a much lower incidence of misdiagnosis than that found by Slater (see, for example, ‘Slater revisited: 6-year follow-up study of patients with medically unexplained motor symptoms’ by Crimlisk HL, Bhatia K, Cope H, David A, Marsden CD, Ron MA. BMJ 1998; 316: 582–6). One possible reason for this apparent fall in the rate of proven misdiagnosis is that some of these studies have used a much shorter follow-up period than the nine to eleven year period employed by Slater. But another possible reason is the influence of Slater himself.

In this respect it should be recalled that Slater’s main conclusion was that the label hysteria was unhelpful, that it generally served to disguise medical ignorance about real but unrecognised (or underrecognised) organic conditions. He was, in short, calling for much more care to be used in diagnosis.

It is generally agreed that his paper was extremely influential. To the extent that what he said was heeded by his colleagues it would almost inevitably follow that instances of misdiagnosis caused by the failure to exclude neurological diseases which were already recognised at the time, and for which diagnostic tests were available, would fall. Neurologists and psychiatrists would either refrain from using the label at all or they would be much more mindful when they did do so of the need to exclude organic diagnoses.

This tendency would have coincided both with the falling prestige of psychoanalysis and with the huge progress there has been in clinical knowledge and diagnostic techniques over the last twenty-five years or so. This latter factor would almost inevitably lead to a situation where more and more previously unrecognised organic symptoms would be correctly identified in the first instance. Inevitably there would be a residue of the hysteria diagnosis. But it is likely that it would be reserved for exceptionally subtle and unusual symptoms which did not register any positive pathology in any recognised diagnostic test and which seemed to be entirely outside the symptom range of recognised and identified illnesses.

The more care that was taken to exclude organic illness at the outset, the less likely is it that the diagnosis would subsequently be shown to be wrong.

However, the mere fact that the diagnosis was not discovered to be mistaken in the next few years by the physician who made it or by others does not mean that the diagnosis was correct, merely that it was unlikely to be shown to be false by the current state of medical knowledge.
In short, the state of affairs which Sharpe and Stone describe, in which the rate of acknowledged or proven misdiagnosis in this area has declined significantly is exactly what one would hope for if Slater’s conclusions were a) right, and b) widely heeded by the generation of psychiatrists and neurologists who trained in the years immediately following the publication of his 1965 paper.

It is possible, however, that there is another factor at work. For it is also almost inevitably the case that an initial diagnosis of conversion disorder will tend significantly to reduce the number clinical tests and investigations which are performed on a patient, particularly investigations of a non-routine kind such as SPECT (Single Photon Emission Computed Tomography) scans. Indeed, one of the reasons psychiatrists are sometimes urged to make the diagnosis of conversion disorder or hysteria is specifically in order to avoid expensive and supposedly unnecessary medical investigations. There is therefore a significant danger that the label of conversion disorder can become self-confirming as patients find themselves ‘trapped’ within a psychological diagnosis.

Any non-dogmatic treatment of the subject might at least be expected to consider these factors. It is interesting that Michael Sharpe, Jon Stone and their colleagues do not mention it. Instead they deliver themselves of the extraordinarily blunt claim that ‘Slater was wrong’.

Before saying this they do not even pause to remind their readers of what the central claim which Slater had made in 1965 actually was. Instead they give the impression that he had predicted that the rate of misdiagnosis in this area would remain high. He had, in fact, done nothing of the kind. He had implicitly implored his medical colleagues to do everything within their power to reduce it.

But even more importantly than this he had opposed the use of ‘hysteria’ as a diagnosis on the grounds that it is a term of such vagueness, which is attended by so little clinical understanding, that it is, as Bastian put it more than a century ago, ‘only a half-diagnosis’.20

To the extent that Sharpe, Stone and others who have criticised Slater have suggested that some forms of misdiagnosis once associated with the label ‘hysteria’ are a great deal less common than they once were, they are undoubtedly correct. It would indeed be remarkable if the advent of MRI scans and other sophisticated techniques had not eliminated a very significant number of such misdiagnoses. However, there remains a great danger, vividly illustrated by the fate of Ms A, that the delusions of human omniscience which have accompanied the history of medicine at practically every stage of its development, may increasingly be supplemented (or replaced) by a technological hubris, and by the equally dangerous belief that our machines are all-seeing and all-knowing. This is not, and almost certainly never will be, the case.
6 Leaping into the medical darkness

To make the suggestion that hysteria or conversion disorder is not a valid diagnosis is not, of course, to suggest that all patients who consult primary care physicians are suffering from organic illnesses. It is an accepted fact that a substantial proportion of such patients have psychological problems rather physical ones. Some of those who are suffering from depression or relationship problems may offer a physical presenting system which masks the true reason for their visit to the doctor (which is not the same thing as saying that the physical symptom has been produced by a process of ‘somatization’). Others may suffer from what used to be called ‘hypochondria’ but now tends to be referred to as ‘health anxiety’. Such patients may, by selective over-attention to certain bodily sensations, exaggerate the symptoms they report without any intent to deceive. Others may, by feigning illness for financial reasons, be guilty of deliberate deception or ‘malingering’. Still others may report factitious illnesses (either their own or their children’s) in an attempt to gain attention or sympathy. Doctors may also encounter patients who have, in effect, lost faith in their own wellness and who, perhaps on the basis of some past ailment, believe themselves to be ill when this is not in fact the case, and when they do not have the physical symptoms of illness.

It is not the patients who fall into any of these categories, however, who are most likely to receive the diagnosis of hysteria. This diagnosis was once applied to almost any physical symptom which could not be explained medically. Increasingly, though, it (or the euphemisms which replace it, such as ‘functional’ or ‘conversion disorder’) tends to be reserved for patients who suffer from neurological-seeming symptoms. As the neurologist Patrick Vuilleumier and his colleagues put it:

Hysterical conversion disorders refer to functional neurological deficits such as paralysis, anaesthesia or blindness not caused by organic damage but associated with emotional ‘psychogenic’ disturbances. Symptoms are not intentionally feigned by the patients whose handicap often outweighs possible short-term gains.

Patients who receive the diagnosis, it might be said, present with apparently real neurological symptoms which neurologists cannot explain or understand on the basis of present medical knowledge. However, it should be noted that Vuilleumier’s phrasing of the claim is subtly different from this reformulation of it. His unequivocal and absolute claim is that the patients in question have symptoms which are ‘not caused by organic damage’.

It need hardly be said that this claim, for all its seeming objectivity, is tendentious. For, just as Charcot did 120 years ago, Vuilleumier implicitly assumes the omniscience of modern medicine. It is this startlingly simple and evidently fallacious assumption which provides the insecure foundation for the diagnosis of hysteria now, just as it did in the time of Charcot and Freud.
There can be no doubt that one of the reasons the diagnosis of hysteria has continued to prove attractive to physicians is that a whole series of neurological deficits of the kind indicated by Vuilleumier, including severe sensory deficits such as deafness and blindness, and motor deficits such as paralysis or paresis of a limb, can be produced (or maintained) in the absence of any severe or distinct organic pathology. Experiments in hypnosis have sometimes furnished more instances of this.

However, the fact that such deficits may appear to be independent of severe pathology does not mean to say that they are entirely independent of organic pathology. Indeed this very point has increasingly been recognised is some of the recent experimental work associated with the diagnosis. Some physicians are clearly aware that the proposition that real physical symptoms (which are not figments of the patient’s imagination) can be produced from mental forces alone, without the participation of demonstrable physical processes, is scientifically eccentric.

They are presumably also mindful that medical science has recently discredited one of the most hallowed of all stress aetiologies – namely the theory that stomach ulcers are principally caused by stress. Here it took the pioneering work of the Australian scientist Barry J. Marshall, who first cultured the bacterium Helicobacter pylori, and then swallowed it in solution, to demonstrate that stomach ulcers have a bacterial cause. (Marshall’s webpage gives a link to a stimulating discussion of the inadequacy of stress aetiologies by Barry Spencer.)

This is not to say that stress cannot or does not have measurable physiological effects on the human organism and that such physiological changes cannot play a pathogenic role. This clearly is the case and there are a number of stress aetiologies which, because they can be related to specific physiological factors such as raised blood pressure, must be regarded as entirely legitimate. The problem with the hysteria diagnosis is that those who make it are, almost by definition, unable to point to any such factors.

This is evidently accepted by Peter Halligan, who notes, in the course of the *BMJ* editorial cited at the beginning of this article, that

> There is no generally accepted explanation for how a psychological stress can convert into (often highly selective) symptoms. In this respect, conversion hysteria retains ‘the doubtful distinction among psychiatric diagnoses of still invoking Freudian mechanisms as an explanation’. The crux of the problem is to explain how abnormal psychological states can produce specific, long term neurological symptoms and disability in patients (who claim not to be consciously responsible) in the absence of detectable pathology.

It should be noted that in this passage Halligan (who cites my own work at this point) is doing what psychiatrists and other proselytisers of the hysteria diagnosis almost always do: he is placing the cart of diagnosis before the horse of pathophysiology. By this I mean that the diagnosis of conversion hysteria is only a medically valid diagnosis if the
pathophysiological process it invokes – the conversion of feelings or ideas into neurological symptoms – can reasonably be assumed to exist.

The option of defining a disease entity clinically rather than pathogenically may well be open to physicians dealing with a disease which has a recognisable and reasonably constant cluster of symptoms. And for them to postulate a viral or bacterial aetiology in the absence of a specific identifiable pathogen is also legitimate, since it is well-known that such aetiologies are found in many diseases.

However, although offered as a diagnosis, ‘hysteria’ or conversion disorder is multivariate and has no recognisable cluster of symptoms. Neither does it depend on any pathophysiological process which has ever been shown to exist. That being so the diagnosis is wholly speculative. It is based not simply on one, but on two simultaneous leaps into the medical darkness. Yet, at the very moment that he obliquely acknowledges this, Halligan premises his entire discussion on the assumption that hysteria is a valid diagnosis.

One response to the radical insecurity which any self-respecting physician or psychologist ought to feel about making this diagnosis is referred to by Halligan when he mentions there have been a number of attempts to use ‘functional imaging’ in order to throw light on the pathophysiological process which hysteria assumes.

One of the most frequently cited of these attempts was actually reported after the publication of Halligan’s editorial. This is a study which Patrick Vuilleumier conducted and which he and his colleagues reported in 2001 in their paper ‘The functional neuro-anatomical correlates of hysterical sensorimotor loss in seven patients using SPECT (single photon emission computerized tomography)’ (Brain 2001 124, 1077-90).

Studies were made of seven patients who were all suffering from paralysis or significant weakness of their arms or legs (or both) on one side of their body but who, after extensive physical examinations, were not found to be suffering from any recognised organic disease. Since they supposedly had troubled emotional lives it was assumed (without any positive evidence for this assumption) that their condition was the product of emotional factors and could therefore reasonably be labelled hysteria.

When the patients’ brains were minutely examined using SPECT (single photon emission computerized tomography), the scans revealed ‘a consistent decrease of regional cerebral blood flow in the thalamus and basal ganglia contralateral to the deficit.’ In the case of four patients who subsequently recovered it was discovered that this pattern of decreased blood flow was no longer present – a finding which tended to confirm that the symptoms of paralysis or weakness had been directly associated with the reduced cerebral blood flow revealed by the scans.

Vuilleumier’s paper describes a remarkable piece of medical detective work. However, it is by no means clear that he and his team of investigators actually found what they thought they had - a cerebral substratum for ‘hysterical’ symptoms. For there is no evidence at all that there was any correlation between the emotional tribulations which had supposedly been suffered by these patients in the past and the onset of their symp-
toms. We might do well to recall the words of Eliot Slater when he bleakly reminded his colleagues that ‘Unfortunately we have to recognise that trouble, discord, anxiety and frustration are so prevalent at all stages of life that their mere occurrence near to the time of onset of an illness does not mean very much.’

If we examine the extremely brief case histories which are contained in the appendix to Vuilleumier’s article, Slater’s words are likely to resonate even more insistently. It might conceivably be that the pattern of reduced cerebral blood flow was related to the patients’ emotional lives. But it might also have been caused by a process of undetected infection. Or it might have been caused by other factors. Are we really supposed to believe that the symptoms of the 21-year-old woman who was found not to be malingering, but who was unable to walk, stand or raise her painful right leg from the bed, were in some way a consequence of, or reaction to, her ‘misconduct at school during teenage’ some three or four years previously? It seems on the whole rather more likely that these symptoms were related to the operation she had undergone a few months previously for suspected appendicitis, or to the symptoms which had prompted this operation, or to the anaesthetic administered to her. Yet the details provided of this patient, and of the other experimental subjects, are so fragmentary that it is impossible to take any informed view.

Although Vuilleumier’s intriguing and unquestionably valuable paper is intended to illuminate the pathophysiological process which supposedly lies behind hysteria, the study he describes might equally well have been undertaken by a sceptical physician who wished to cast doubt on the diagnosis. For what it shows, or appears to show, is that the symptoms of the supposedly ‘hysterical’ patients do have an organic basis (or at least a cerebral correlate) – one which may well be completely unrelated to the psychological stresses the patients are said to have suffered.
7 Hystera and history

There is no medical evidence to confirm the centuries-old conjecture that emotional trauma can in itself give rise to the bewildering variety of neurological and other discrete physical symptoms which have been associated with the diagnosis of hysteria over the last two centuries. There is, however, a great deal of evidence to suggest that most of the symptoms which have in the past been held to be ‘hysterical’ can actually be explained medically by non-psychological theories.

This does not mean that emotions, stress and other ‘psychological’ factors do not play a significant part in our physical health. Since stress and all kinds of emotion are experienced by the body as physiological or bio-chemical events, it may very well be the case that there are unknown pathological processes associated with them (in addition to those which are already understood). It is also possible that these may be associated with some physical symptoms.

But unless a particular pathophysiological process involving emotional factors can be demonstrated to play a part in generating specific physical symptoms, there is no good reason to favour psychosomatic explanations in preference to alternative non-psychological theories.

If medicine was a journey of discovery which set out to illuminate the dark interior of an unchanging continent then, since knowledge is cumulative, we could count on the continual diminution of the mysteries and misdiagnoses which currently beset us. But the simple fact is that microbes, and the diseases that they bring, evolve much more rapidly than humans do. What this means is that new and ever-more complex medical mysteries are evolving even as some of the old mysteries are being solved.

In such circumstances, our delusions of omniscience and the belief that physical symptoms which do not accord with our current knowledge of physiology and neurology should be classified as ‘functional’ or ‘hysterical’ may well be just as dangerous in the future as they have been in the past.

What makes such an uncertain future even more hazardous is that medicine is a profoundly unhistorical or even anti-historical branch of knowledge. Although it eagerly conserves the memory of its great breakthroughs and successes, it tends to be amnesiac about its many errors. Yet it is sometimes just these errors which, carefully attended to, have the capacity to teach us most.

The greatest danger of failing to examine systematic errors which have been made by physicians in the past is that we may actually continue to use a tradition of error as the basis of our modern thinking about an entire branch of medicine.

It is exactly this, I believe, that has happened in the case of hysteria. The current revival of the diagnosis of hysteria suggests that the vast legacy of medical error bequeathed to modern medicine by Charcot and Freud has still not been recognised for what it is. But
it also suggests that it is this legacy which continues shape our current thinking and give rise to much of our contemporary confusion about ‘functional symptoms’, ‘conversion disorder’ and ‘somatisation’.

If we are to understand some of the most subtle problems which now confront modern medicine and modern psychiatry it is the medical mistakes contained in the legacy of Charcot and Freud we need most urgently to consider.

I have examined in detail the nature of the medical errors on which Charcot’s later work was based elsewhere. Suffice it to say here that, when Freud went to Paris to study under Charcot in 1885 he did so a full ten years before the X-Ray was invented, at a time when the discipline of neurology was only just beginning to elucidate some of the grosser mysteries of the brain and the nervous system.

By this point, however, Charcot had already made what is perhaps the most common of all medical mistakes: he had come to the conclusion that the work of neurology was all but complete and that he and his colleagues already understood almost all that there was to understand about cerebral pathology. It was with this dangerous mindset that he approached one of the most ancient of all medical mysteries, the problem of hysteria.

Charcot began by searching for the barely perceptible ‘dynamic lesions’ which he believed must underlie hysteria – which at this stage he imagined to be but another organically based neurological disease. However, in conditions where no EEG investigations were available, and where the cerebral pathology of many complex and subtle neurological conditions was undetectable even at autopsy, he was never likely to be able to locate such lesions. Instead he allowed himself to be guided by the work of Thomas Briquet who, in 1859, had published a treatise in which he had revived an idea first proposed in the seventeenth century by Thomas Sydenham. Sydenham had maintained that hysteria was a real clinical entity but that its prime characteristic was that it simulated epileptic fits and other organic disorders. He had further speculated that hysterical disorders were caused by ‘some great commotion of mind, occasioned by some sudden fit, either of anger, grief, terror or like passions’.

Charcot married Sydenham’s speculations with those expressed in a paper by the English physician, J. Russell Reynolds, which had appeared in the British Medical Journal in 1869. Reynolds had argued that in certain cases the paralysis of a limb could be produced merely by the patient’s continual contemplation of the idea of paralysis. Combining these ideas with some of the propositions of contemporary faith-healers, Charcot now embarked on the creation of a highly speculative and extraordinarily ambitious medical theory. In this theory practically any convulsive, seizure-like, or neurological-seeming symptom which he did not understand was deemed to arise not from detectable lesions in the brain but from ‘ideas’ which patients had formed in their minds – usually at the time of traumatic incidents.

According to Charcot these ideas had, because of their isolation from ordinary consciousness, been able develop in a non-conscious part of the brain until they were sufficiently powerful actually to produce physical symptoms of the kind for which
organic lesions would normally be held responsible. In this way an idea ‘would be removed from every influence, be strengthened, and finally become powerful enough to realise itself objectively through a paralysis.’

Charcot meanwhile continued to refine his description of what he had come to regard as the classic manifestation of hysteria. For, in spite of the protean character that some physicians attributed to the disease, there was at least one thread of symptoms which appeared to be continuous and which was related to the traditional explanation of hysteria – and indeed to its very name, which derives from the Greek word for uterus. From the time of Plato onwards, physicians had frequently explained a particular set of physical sensations reported by patients by suggesting that it was caused by the womb moving upwards through the body towards the head. This theory derived from the subjective experience of victims of ‘hysterical’ fits. For a woman suffering from such a fit would experience a sensation as if her womb were rising up through her body. As it travelled towards the head it would cause a feeling of tightness in the chest and on its arrival at the throat it would cause choking as if by a ball – the globus hystericus. Sometimes the woman would suffer only a slight lapse of consciousness. Sometimes she would fall down in a convulsive fit.

Charcot now applied his new views about pathogenic ideas both to ‘hysterical’ fits and to the whole realm of hysterical illnesses. It was these views which were then taken over by Freud whose theories of unconscious symptom-formation were based directly on Charcot’s supposed clinical breakthrough.

It was not until the middle of the twentieth century that a small number of clinicians, especially neurologists, began to recognise that at the very heart of Charcot’s work on hysteria there lay a fundamental medical error. Because of the great strides which had been made in clinical medicine, they could now see that Charcot’s classical ‘hysterical’ fit was not hysterical at all. The phenomenon he had described – that of an epigastric sensation rising upwards through through the body and causing choking as though by a ball in the throat – was something quite different. The great Czech neurologist Kamil Henner, for example, described the aura of temporal lobe epilepsy – a condition which was not recognised and defined until the 1930s – in terms which were almost identical to the old accounts of the hysterical fit. The patient experiences a feeling of constriction in the epigastrium, he wrote, often accompanied by anxiety:

This spasmodic sensation travels upwards through the thorax, into the throat and is then followed by unconsciousness or by psychomotor automatism. The symptoms described by the patients correspond exactly with Charcot’s boule hystérique or globus hystericus.

Henner himself went on to suggest that one of the tasks of modern neurology should be to correct traditional interpretations of globus hystericus, ‘for I presume that in these cases one is nearly always dealing with an epileptic seizure’. Although the sensations Charcot described and attributed to hysteria clearly corresponded to the classic aura found in many cases of temporal lobe epilepsy, Charcot himself always treated the presence of such an aura as a sign that any subsequent seizure was not epileptic.
Moreover, as other neurologists would recognise several decades later, the classic hysterical posture in which patients arched themselves backwards on the floor in the position known as *arc-en-ciel* (or *arc de cercle*) is also not hysterical. It is a characteristic manifestation of frontal lobe epilepsy, a condition which was not recognised until the closing decades of the twentieth century.
8 The case of the florist’s delivery man

Even later than this, other physicians have reviewed the case of Le Log——, whom Charcot had regarded as a classic example of a patient suffering from traumatic hysteria. The case of Le Log—— is a particularly interesting one because it demonstrates the manner in which Charcot felt justified in applying the diagnosis of hysteria not simply to patients who suffered seizures, or neurological deficits such as the paralysis of an arm or a leg, but to a complex illness involving a range of physical symptoms and with an unpredictable course.

Le Log—— was a florist’s delivery man in Paris. One evening, in October 1885, he was wheeling his barrow home through busy streets when it was hit from the side by a carriage which was being driven at great speed. Le Log——, who had been holding the handles of his barrow tightly, was spun through the air and landed on the ground. He was picked up completely unconscious although there were no signs of external injury. He was then taken to the nearby Beaujon hospital where he remained unconscious for five or six days. He was subsequently sent home but his convalescence was interrupted by nosebleeds which were so severe that they could only be stopped by plugging his nose. He eventually suffered a violent seizure, preceded ‘by a sensation of a ball rising in his throat’. During this seizure he lost consciousness and was taken on a stretcher to the Hôtel Dieu.

He stayed in this hospital for a total of two months and during the first week was in a state of continual coma. After coming out of this coma he was unable to speak for two days. He suffered from frequent severe nose-bleeds and the lower extremities of his body gradually became almost completely paralysed. It was at this point, in March 1886, almost exactly six months after the accident happened, that Le Log—— was transferred to Charcot’s hospital. On admission his memory and intelligence were ‘considerably affected’ and he stated that ‘at night-time he has flames before his eyes, and terrifying dreams, and all the while beating in the temples and dizziness in the ears.’ The left-hand side of his mouth was partly open due to a spasm of the muscles on the left side of the face which was accompanied by a twitching or tremor of the corner of the mouth. Throughout most of this period Le Log—— suffered from ‘a permanent headache of a constrictive character, producing the sensation of a heavy helmet pressing all parts of the head’.

What is most striking and most significant in its implications for the subsequent development of modern medicine is that Charcot, basing his view on the absence of external injury, felt quite able to explain all these symptoms – including the severe nosebleeds and seizures – by invoking the diagnosis of hysteria and by suggesting that Le Log—— was suffering not from the physical effects of his accident but from the idea which he had formed of it.

When the full story of Le Log——’s physical symptoms is told, however, and separated out from Charcot’s own diagnostic interpolations, the entire case is one which, rather than being a classic example of psychogenesis, is saturated in organicity. Most modern
physicians would unhesitatingly recognise in Le Log—‘s story a severe case of closed head injury. The long period of unconsciousness would itself be construed not as an indication of emotional trauma but as a sign of the severity of the intracranial injury. This would in turn suggest the likelihood of late epilepsy developing. This evidently happened in the case of Le Log—, and the exceptionally severe seizure he suffered appears to have resulted in further brain damage. The profuse nose-bleeds would merely confirm the picture, as would the facial spasm and the various other neurological signs which Charcot describes – including the terrifying dreams, the flames before the eyes and the noises in the ear which are forms of hallucination often associated with epileptic events.

Far from being a clear case of hysteria as Charcot claimed, Le Log—‘s illness is entirely consistent with a closed head injury resulting in lesions or even a basal fracture of the skull. His symptoms would be recognised by most modern physicians as those of brain damage, epilepsy and raised intracranial pressure. As I have put it elsewhere: ‘Le Log—, the classic example of a patient who supposedly suffered from traumatic hysteria, did not forget because he was frightened. He forgot because he was concussed. His various symptoms were not produced by an unconscious idea. They were the result of brain damage.’

At the centre Charcot’s work on hysteria we thus find two momentous medical mistakes. On the one hand Charcot’s acceptance of the pre-scientific medical conjecture that hysteria was a real disease entity which ‘imitated’ or ‘mimicked’ other illnesses led him to mistake cases of temporal lobe epilepsy (and frontal lobe epilepsy) for cases of hysteria. He failed to recognise that the reason his presumed hysterics had seizures which resembled epilepsy was because the seizures in question were themselves epileptic – manifestations of particular forms of epilepsy which had not yet been recognised.’ On the other hand Charcot mistook the kind of complex neurological illnesses which can result from closed head injuries for cases of traumatic hysteria.

In both cases he revived the kind of diagnostic reasoning which had dominated Western medicine in its pre-scientific era. For the view which Charcot effectively rehabilitated, namely that symptoms which cannot yet be explained organically must necessarily be psychogenic, is not a rational or a scientific view. It is a superstitious or pre-scientific view whose origins can be traced back many centuries.

Contrary to one of the most powerful of all current myths about the history of medicine, the view adopted by Charcot and, after him Freud, about the relationship of the mind to the body was very far from being a modern innovation. In many respects it was deeply traditional. From the time of Hippocrates onwards many physicians had put forward the idea that emotions not only influenced bodily functions but sometimes acted as causative, pathogenic factors. Galen, for example, included the passions among the causes of bodily disease, and the medical historian L. J. Rather has suggested that, as a result of Galen’s influence on European medicine down to the nineteenth century,

* It is also possible, of course, that some of Charcot’s presumed hysterics may have been simulating seizures.
physicians had given a great deal of attention to theories postulating the psychological causation of a variety of diseases.

Such theories were particularly attractive in the era preceding the formulation of the germ theory of disease when physicians remained unaware of the pathogenic role of bacteria and viruses. In these essentially pre-scientific conditions some extremely crude psychogenic theories of disease remained highly plausible. That such theories frequently embraced both contagious and epidemic diseases is made clear in a passage from Archer’s *Every Man His Own Doctor*, published in 1673:

> The observation I have made in practice of physick these several years, hath confirmed me in this opinion, that the original, or cause of most men and women’s sickness, disease, and death is, first, some great discontent, which brings a habit of sadness of mind ...

It was in the same century that Thomas Sydenham developed his influential account of hysteria in which he suggested not only that it *simulated* epileptic fits and other organic disorders but, as has already been noted, took the view that hysterical disorders were caused by ‘some great commotion of mind, occasioned by some sudden fit, either of anger, grief, terror or like passions’.

With the advent of new knowledge about the aetiology of disease brought by such researchers as Pasteur and Koch, such crude psychogenic theories had been rapidly eclipsed, and most physicians in the latter part of the nineteenth century were quite able to see them for what they were – ancient medical fallacies based on superstition and ignorance. Yet in some quarters of the medical profession, particularly those associated with exorcism and faith-healing, simplistic ideas about psychogenesis had been preserved. Such ideas were certainly held by the mesmerists and by later ‘miraculous’ healers such as Liébeault. It was Charcot who, by dint of the extraordinary authority he wielded in late-nineteenth century European medicine, succeeded in restoring these pre-scientific and superstitious ideas about psychogenesis to mainstream Western medicine.

By scientifically legitimating these ideas, by combining them with traditional notions about the nature of hysteria, and by investing them with a series of fundamental diagnostic errors of his own, Charcot effectively revived a tradition of misdiagnosis which had seemed on the point of disappearing from Western medicine.

Charcot’s errors, had they been made by a less influential physician, might be of merely academic interest. In practice, however, they constituted the very foundations on which a large part of modern neurology and modern psychiatry would be constructed.

The fact that Charcot’s medical mistakes almost immediately became the foundations on which Freud, the most influential of all Charcot’s pupils, built his theories of unconscious symptom formation and the entire edifice of psychoanalysis, greatly increased their influence.
Unfortunately for apologists of psychodynamic medicine, the work of Freud has not survived the relentless advance of clinical knowledge any better. For, as we have already seen, it is now clear that Anna O, the patient of Freud’s colleague Josef Breuer, whose case actually gave rise to psychoanalysis, was not suffering from hysteria any more than Charcot’s classic patients. Something similar may be said of practically every one of the series of ‘hysterical’ patients in relation to whom Freud developed his main psychoanalytic theories. On the evidence which Freud himself provides, it would seem that these patients were suffering from various ailments including temporal lobe epilepsy, Tourette’s syndrome, parosmia, rheumatism and pelvic appendicitis, all of which were misdiagnosed as hysteria.

It would be difficult to overstate the importance of these little recognised dimensions of the history of medicine. For we are not talking here about minor or peripheral mistakes of the kind which are bound to be made in every voyage of discovery. We are talking about fundamental errors born out of medical ignorance which actually provided the some of the foundations of modern Western medicine.

It is on those insecure foundations, and on the massive power of tradition associated with them, that the approach of modern physicians to the diagnosis of hysteria still rests. One of the principal reasons that the putative pathophysiology of hysteria – namely that real physical symptoms can be mysteriously produced by unconscious ideas or feelings – is still taken seriously today is that contemporary physicians, overcome by the sheer weight of medical tradition (just like those who resisted the bacterial aetiology of stomach ulcers) mistakenly believe that the diagnosis rests on secure historical foundations.

Studies of patients who have been held to be suffering from conversion disorder or hysteria which are based on follow-up periods of 6 years are all very well. But it is only if we view hysteria in the perspective of history over a period of a century or more that we are likely to recognise its true nature. Given the remoteness of the clinical data, we cannot ever hope place a precise figure on the rate of evident misdiagnosis associated with the concept of hysteria. But we can be sure that, at one time or other, a very large proportion of the complex organic illnesses currently recognised by medical science (and some simple ones as well) have, on some occasions at least, been misdiagnosed as, or mistaken for, hysteria. The list is practically endless but should certainly include temporal lobe epilepsy, frontal lobe epilepsy, closed head injuries, cerebral tumours, multiple sclerosis, Parkinson’s disease, Tourette’s syndrome, lupus, autism, syphilis, encephalitis, torsion dystonia, cervical dystonia, blepharospasm, spasmodic torticollis, chronic renal failure, lead poisoning, ataxia, chronic progressive chorea, myoclonus, progressive supranuclear palsy, restless leg syndrome, periodic limb movement disorder, reflux oesophagitis, hiatus hernia, pelvic appendicitis, rheumatism, peptic ulceration, pharyngeal pouch, hypoxia, hypoglycaemia, post-cricoid web, thyroid goitre, vascular disease, dystonia musculorum deformans (DMD), Wilson’s disease and, more recently, Lyme disease, Creutzfeld-Jakob disease, HIV aids, and calcium excess giving rise to chronic vomiting in a patient bed-ridden and immobile as a result of Guillain-Barré syndrome.
In view of the massive number of evident misdiagnoses which have been associated with the category of ‘hysteria’ since 1850 (not to speak of those which belonged to previous centuries or of those which have yet to become apparent), it might be thought that most modern physicians would now be prepared to recognise ‘hysteria’ for what it has in practice been: a term which is used by physicians in order to disguise ignorance as medical knowledge. They might, indeed, be inclined to recognise as prophetic the words of the French neurologist Paul Guirard. ‘When we have completed the clinical analysis of all the hysterical symptoms,’ wrote Guirard in 1914, ‘when we have given to each [organic] illness what belongs to it, who knows if anything will still remain of hysteria?’
9 Shell shock

It is, however, perhaps significant that Guirard made his sceptical pronouncement about hysteria in 1914, before the experience of the First World War and dramatic increase in diagnosed cases of hysteria this brought with it.

There can be no doubt at all that the First World War did produce an unprecedented number of such cases. The symptoms reported among British soldiers included paralyses, contractures, muscle rigidity; gait disorders involving the limbs, extremities, and spine; seizures, tremors, spasms, tics, and uncontrollable blinking; the radical narrowing of the field of vision and blindness; localized numbness and loss of sensitivity; localized pain, and hypersensitivity; mutism, aphonia, and stammering; deafness; fugue states, Ganser twilight state, amnesia, mental confusion, and extreme suggestibility; a persistent sensation of unpleasant smells and tastes or, alternatively, the loss of the sense of smell or taste.

It is interesting to note, however, that the term ‘shell shock’, which was first coined in 1915, was initially used by psychiatrists and neurologists to describe a syndrome which was presumed to have an organic origin and to be caused by the physical impact of a shell’s explosive force. There were, indeed, a number of cases where soldiers had been killed as the direct result of a shell exploding nearby even though they had no external injury on their body.

One man had been in a dug-out when a shell exploded ten feet away. He then suffered tremors, general depression, and periods of crying. The next day, he was unable to talk. That evening, he entered ‘a state of acute mania, shouting “Keep them back, keep them back.” He was quite uncontrollable and … impossible to examine. He was quieted with morphine and chloroform, and got better and slept all night…. Next morning he woke up apparently well, and suddenly died.’ The second soldier was in an ammunition shed when it was hit by a shell. He became unconscious at once and died soon afterward.21

When the brains of these two soldiers were subjected to post mortem examination, microscopic haemorrhages and other vascular changes were observed. It was eventually acknowledged that the ‘overpressure effect’ caused by a shell’s explosion could rupture the internal organs of a human body and cause fatal internal injury, yet do so without leaving any external marks. It was naturally enough assumed that that it was this same effect (or possibly carbon monoxide poisoning associated with the explosions) which was also responsible for the neurological symptoms which some soldiers also developed. As David Marlowe has put it: ‘[S]hell shock was defined as a “commotional” illness: a physical ailment generated by the shock wave of exploding ordnance coming into contact with the head, producing microhaemorrhaging in the brain.’22

However, because some victims of shell-shock appeared not to have been in the immediate vicinity of exploding shells, and because post mortem examinations of some victims revealed no signs of brain pathology, shell shock tended increasingly to be viewed as a psychogenic condition. Today this view has been adopted almost as an
exclusive doctrine. At the time, however, there was considerable disagreement between the views adopted by different neurologists and psychiatrists, and some of the most zealous proponents of psychogenic explanations conceded what the medical evidence clearly indicated – that a significant proportion of the neurological symptoms of shell shock were ‘commotional’ rather than emotional, in that they were caused directly by the physical force of the explosion.23

Given the relatively crude methods of examination available at the time, the absence of visible pathology in the brains of many victims of shell shock was not conclusive and did not in itself rule out physical trauma as a cause of the symptoms. André Léri, a French neurologist who regarded hysterical symptoms without an organic basis as relatively common in wartime, himself went out of his way to stress the dangers of misdiagnosis. On the one hand he suggested that some conditions with a real organic foundation persisted unnecessarily for reasons which were psychological or neurotic but were not recognised as such. On the other hand, in an extended and crucial passage of his book Shell Shock, he pointed to the opposite mistake:

The contrary takes place in cases which are perhaps more frequent: the hysterical patient is not taken for an organic one, it is the organic patient who is considered to be hysterical; should he prove refractory, it must now be recognised that it is too often because he has real lesions which prevent his recovery. ‘By the side of the objective signs with which we are acquainted, there are others still unknown, the discovery of which will one day lead us to remove from the group of functional conditions states, which are now regarded as such’. [quotation marks as in original; the quote is not attributed] In this respect, war will have taught as much and will have served to teach the doctor a lesson in modesty.

In this way, since the beginning of the campaign, we have learned to separate from hysterical or pithiatic troubles a whole series of paralyses, and of reflex or physiopathic contractures. The discovery of blood in the cerebrospinal fluid enables us to group amongst organic paralyses certain paraplegias which, without that, would doubtless have been considered as hysterical … Thus certain contractures formerly reputed hysterical appear today to be due to the existence in the muscle of simple metallic powder or to a foreign body which has escaped radiography. Others depend on the partial irritation of a nerve at a distance, on the secondary retraction of the antagonists of the paralysed muscles themselves, etc; so that we now observe hardly any persistent contracture, which has not been tested as an organic determination … [Léri goes on to enumerate other formerly ‘hysterical’ conditions which were recognised during the war to be organic]

We have purposely insisted somewhat on this renovation of pathology that war has brought about. Unfortunately prolonged experience of the neurol-

* The adjective from ‘pithiatism’, an alternative term for hysterical introduced by Josef Babinski (see below)
ogy of war has caused us to think that perhaps there is too great a tendency to see refractoriness and recalcitrance where, really, there are sometimes disabilities of war, that are often organic but unrecognised. Frequent as we believe hysteria to be, and simple and rapidly efficacious as is its treatment; infrequent as we consider the so-called indefinitely refractory hysteries, which although well treated, retain for months or years the same fixed attitude, or the same tremor etc, we are more and more persuaded that in the majority of these patients, one day or another the chance discovery of some sign, either unknown or but little known so far, will give proof of their organic lesions.24

In spite of these cautionary words it was not the case that either Léri or any other neurologists active during the First World War, ever adopted a wholly sceptical attitude towards the diagnosis of ‘hysteria’. Léri himself, as the quotation above makes clear, held fast to the orthodox view that hysteria was ‘frequent’ in war and this represented the medical consensus at the time.

It would be quite wrong, however, to conclude from this that physicians in the First World War were actually intending to revive the same diagnosis which had been championed by Charcot some thirty years previously. For by this time Charcot’s version of hysteria had been effectively discredited. The presiding influence on neurologists during the period of the Great War was not Charcot but the man who had effectively dethroned him – his pupil, Joseph Babinski.

Babinski had observed Charcot’s clinical practice closely as the diagnosis of hysteria had become ever more capacious and ever more implausible. He had also been able to observe at first hand the extent to which Charcot’s own patients had responded – sometimes theatrically – to suggestions conveyed to them by the great clinician. The outcome of these and other observations was a pronounced scepticism. So profound was this scepticism, and so disillusioned was Babinski with his master’s favourite diagnostic category that he actually proposed that it should be dispensed with altogether and replace by a coinage of his own – ‘pithiatism’ – a term derived from the Greek words for ‘to suggest’ and ‘to cure’.

As this coinage implied, the new diagnosis was to be applied only to conditions which had been produced by the process of suggestion, and could be removed by counter-suggestion. Babinski specifically excluded a number of conditions from the diagnosis, maintaining, for example that ulcers, haemorrhages, fever and hemiplegia (paralysis of one side of the body) could not be produced by suggestion and therefore could not ever be considered to be hysterical – or ‘pithiatic’.

Babinski also attempted to exclude another group of symptoms, among them ‘[those] contractures, paralyses or paretic [partially paralysed] states which develop after traumatism’. Although he acknowledge that these had frequently been found in soldiers during the First World War, he specifically identified them as non-hysterical:
These phenomena may be entitled physiopathic – a term intended to express the idea that, on the one hand, neither hysteria nor any other psychopathic state can produce them, and, on the other, that while indicating a physical and material disorder of the nervous system they do not appear generally to correspond to any nervous lesion which can be detected by the methods at our disposal.25

It should immediately be clear that Babinski’s attempt to overturn the diagnostic edifice constructed by Charcot was far from being wholly successful. The term ‘pithiatism’, although it was adopted by some French neurologists, including Léri, never achieved real currency and some of the conditions Babinski attempted to keep out of any psycho‐genic diagnosis were still treated as hysterical by other physicians.

But Babinski’s attempted palace revolution remained surprisingly radical. Whereas Charcot had followed Sydenham by positing an entirely original pathophysiological process whereby hysteria could generate practically any physical symptom, Babinski severely restricted the diagnosis to conditions which could, in fact, be produced consciously and deliberately by an artful simulator (even though Babinski himself was not himself suggesting that the production of such symptoms in ‘pithiatism’ was in fact a conscious process). While Charcot’s hysteria demanded the existence of a complex pathophysiological process, it was not clear that Babinski’s ‘pithiatism’ assumed the existence of anything other than normal brain mechanisms.
10 Miraculous cures

Babinski’s new, rationalised version of hysteria influenced not only French physicians but also some of the British medical officers directly involved in the treatment of the thousands of neurological or psychiatric casualties which the war produced. One measure of his influence may be found in the fact that even W. H. Rivers, the most psychodynamically oriented of the British war physicians, specifically rejected the Freudian term ‘conversion neurosis’, on the grounds that it begged pathophysiological questions, and proposed instead that the term ‘suggestion-neurosis’ should be adopted.26

No less significantly, Babinski was himself partly responsible for training the British physician who was perhaps more successful than any other in bringing about apparent cures of soldiers with long-standing ‘hysterical’ symptoms – Arthur Hurst (later to be Sir Arthur Hurst).

Hurst, who briefly attended some of the lectures of Babinski in Paris during the immediate pre-war years, gained an apparently well-deserved reputation as a miraculous healer of soldiers suffering from shell-shock. Seale Hayne, the neurological hospital which Hurst set up in rural South Devon, became known as the place where the lame threw aside their crutches in order to walk, where those who were deaf recovered their hearing, and where men who had arrived blind learned to see.

Yet if we examine Hurst’s remarkable case histories, it soon becomes apparent that, even according to his own account, the various neurological deficits he cured were far from being independent of physical causes. In the majority of the cases he described, the original sensory loss had been caused by some physical insult – as in the case of the temporary blindness which can be induced by mustard gas. It was Hurst’s view that when the immediate physical causes of such neurological deficits receded, the deficits they had instituted could then be perpetuated by auto-suggestion:

In order to see it is necessary to look ... In the war of 1914-18 many cases of hysterical blindness followed the conjunctivitis caused by mustard gas and by sand-storms in the desert. The swollen lids remained closed. This was in part voluntary, as the patient knew that exposure to light would cause pain, and in part the result of protective spasms of the eyelids. The inflammation gradually subsided and the reflex spasms of the lids disappeared. At the same time the majority of patients realised they could now open their eyes without pain and complete recovery quickly followed. In men who had become abnormally suggestible as a result of the strain of active service, and especially those who for any reason were already anxious about the condition of their eyes, the natural results of the inflammation became perpetuated by auto-suggestion, especially if their anxiety was aggravated by the hetero-suggestion caused by injudicious treatment, such as the too prolonged use of eye-shades and dark glasses. The voluntary inactivity of the lid-raising muscles became perpetuated by hysterical paralysis, and the reflex spasm of the lids as hysterical spasm, which was exaggerated when the
patient attempted to open his eyes voluntarily or an attempt was made to pull them open ... Hysterical inability to open the eyes is ... often accompanied by hysterical blindness, the patient having so convinced himself that he cannot see that he makes no attempt to look when his eyes are at last open.\textsuperscript{27}

Hurst’s remarkable cures depended on a variety of strategies which he himself categorised as forms of suggestion. These included the administration of electric shocks, hypnotism and, perhaps most commonly, a form of rational persuasion, or explanation, wherein he imparted to his patient his own belief in the possibility of a rapid cure together with his medical rationale for this. Wherever possible Hurst attempted to effect his cures in a single session. In a remarkable number of cases he was apparently successful.

One of the cases he recounts concerned a 22-year-old soldier who had been looking over a parapet at Gallipoli in July 1915 when a shell struck the sandbags in front of him. The sand was thrown up into his eyes and he fell back on his head, knocking himself unconscious for 24 hours. On regaining consciousness he found that he could not open his eyes. When Hurst first saw him on September 17, 1915 his eyelids constantly flickered and he was quite blind. When his eyes were forcibly opened they were turned so far upwards that it was difficult to see even the iris:

The patient was easily hypnotised, and whilst asleep he was told that he would be able to see when he woke up. The moment he awoke the suggestion was repeated very forcibly, and his eyes were held open. He cried out that he could see, tears ran down his cheeks, and he fell on his knees with gratitude, as he thought that he was permanently blind and believed his sight had been restored by a miracle. When seen again on September 20, the external appearance of his eyes was normal and he said he was able to see as well as he had ever done.\textsuperscript{28}

Hurst’s theoretical account of what was happening to his patients is clearly constrained by the particular medical doctrine of hysteria which then held sway – which had been imparted to him by Babinski. It is for this reason that every aspect both of the cause and cure of hysteria is conceptualised in terms of ‘suggestion’. However some of Hurst’s own cases would appear to confute this theoretical model. In one of the most remarkable of all his case histories, he recounts the cure he made of a young man of eighteen who was not a casualty of the war at all, but who had been treated as a deaf mute since infancy, ever since he had fallen on his head at some time between the age of three and nine months.

When Hurst first saw this young man in May 1919 he appeared to be totally deaf. However, there were no signs of middle ear disease and the vestibular reactions were normal. Hurst recounts that he explained to the man by means of lip reading ‘that he could not hear because he had never tried to listen and that if he once made an effort to listen he would begin to hear’. During this very first session, according to Hurst,
he was taught to listen sufficiently to hear his name called close to his ear; this was, so far as he remembered, the very first word he had ever heard. The same day he heard his bicycle bell and a motor horn for the first time.29

During brief lessons given over the next three weeks he learned to hear a number of words. After a year he was still making slow but steady progress:

When I last saw him on July 20, 1920, he could hear a watch ticking three inches away from his left ear, and could repeat words he had never heard before if they were spoken several times distinctly, but not loudly, near his ear … Although his hearing is still far from normal, there is every reason to hope that considerable further improvement will take place.30

Although this young man’s deafness is described by Hurst as ‘hysterical’, it would seem that it was a product purely of a physical trauma and must, as Hurst himself writes, ‘have been due to some organic but evanescent lesion’ which had interrupted his development just at the point when he was learning to listen. Any notion that the young man’s later disability was a consequence either of neurosis or of psychological suggestion would be forced and implausible. A more credible hypothesis, which might be applied equally well to Hurst’s shell-shock patients, might tentatively propose that, as a result of a purely physical trauma, some of the cerebral connections on which sensory functioning depends had been ‘broken’ and needed to be ‘reconnected’.

Although it was apparently the case that Hurst was on occasion able to cure his patients by rational persuasion alone, it would be a mistake to regard this ‘talking-cure’ purely as a psychological matter, since what he was urging on his patients was, in effect, a form of cerebral reconfiguration.

Perhaps no less important an ingredient of his miracle cures was another factor which might mistakenly be regarded as having no physiological significance – namely the manner in which Hurst quite deliberately created in the minds of his patients the expectation of healing. In his view an ‘all important preliminary is the creation of a proper atmosphere of cure.’ As Ben Shephard has put it in his history of war psychiatry, ‘Patients arriving at Seale Hayne would be carefully primed by the nurses, told of the doctor’s miraculous record, shown other successful cases and the “trophies in the shape of discarded splints and crutches” which lined the hospital walls and given notice of when they would be “cured”.’31 As Hurst himself wrote:

By the following morning the patient is fully convinced that the hoped-for cure will take place: as the medical officer is equally convinced that he will cure the patient, the two essentials for recovery are present. The nature of the actual treatment is really immaterial, but simple persuasion has the great advantage of making the patient take an active part in his own cure and it removes any suspicion of charlatanism from the proceedings.32

Hurst’s own rationale for this theatrical stress on the notion of miraculous cures was, presumably, that it heightened the power of suggestion. But, because of what we now
know about the physiological basis of the placebo effect, it seems clear that one of the physical consequences of Hurst’s approach would be to release a significant surge of the neurotransmitter dopamine into the patient’s brain at the time of the anticipated cure.\(^33\)

It may well not be a coincidence that one of the physical therapies which has been successfully used in the treatment of patients suffering from post-operative hemiplegia [one-sided paralysis] is to flood the patient’s brain with dopamine; dopamine is used in combination with mannitol and large quantities of intravascular fluids in order to increase cerebral blood flow.\(^34\)

Although Hurst himself believed that the success of his talking cures ‘proved’ that the symptoms suffered by his patients were purely ‘functional’ or ‘hysterical’, his cures are in fact entirely compatible with the view that his patients’ undeniably physical symptoms were caused by subtle neurophysiological factors arising from physical trauma and compounded rather than caused by psychological factors.

Indeed, only if we had already achieved the kind of neurological omniscience which physicians have long arrogated to themselves, would it be possible to exclude such an organic explanation. Today it may well be that our understanding of neurology has progressed dramatically since the time of Charcot. But it remains incomplete. As Rita Carter writes in her book *Mapping the Mind*, ‘the vision of the brain we have now is probably no more complete or accurate than a sixteenth-century map of the world.’

Once we reject the false premise that our medical knowledge and our understanding of cerebral mechanisms is complete, then it automatically follows that we are not, any more than Charcot was at the end of the nineteenth century, in any position to make reliable predictions about the pathogenic processes which may give rise to unexplained symptoms. Since emotions are, as already noted, experienced by the organism as physical or bio-chemical events, we cannot and should not exclude the possibility that they may be implicated in some pathological processes. But there are a large number of other possibilities. These include many different kinds of organic pathology which can be traced by current methods of investigation as well as many more which cannot – including subtle deficits in the neurology of consciousness, cognition or volition which may well not be caused by emotions at all and may, to cite but one possibility, be one of the myriad consequences of the reduced cerebral blood flow which seems to feature so frequently in conditions which are deemed ‘hysterical’.\(^35\)
11 Treachery and deceit: the hidden impostor

The proposition that hysteria is a pseudo-diagnosis which has no justification in evidence-based medicine is by no means a novel one. This or a similar idea has been put forward repeatedly ever since the early twentieth century. Eliot Slater’s 1965 paper ‘Diagnosis of Hysteria’ is merely the most celebrated of numerous similar arguments.

Why then, in spite of these repeated attacks, does the diagnosis still survive?

One factor, which is directly related to the medical profession’s lack of any critical historical consciousness, is, as already noted, the sheer weight of tradition. It is clear that generations of physicians have passed on the doctrine of hysteria to the medical students they are training rather in the same way that doctrines or creeds are passed on by priests, without critically interrogating them or inquiring into the historical conditions under which they emerged. Here, for example, is a passage from one of the most highly regarded and commonly used British textbooks on clinical neurology, which was first published in 1989, and from which many future physicians learn the principles of neurological diagnosis. The textbook in question, C. David Marsden and Timothy J. Fowler’s, Clinical Neurology, contains the following discussion of hysteria during the course of a chapter written by a consultant psychiatrist:

Hysteria involves a state of dissociation or conversion, unconsciously determined for emotional gain ... The gain is usually not a simple desire to manipulate others or obtain a financial reward, it is often an attempt to reduce intolerable anxiety ...

Conversion is a concept whereby anxiety is ‘converted’ to a physical symptom and anxiety is relieved in the process ... Conversion symptoms can be motor, such as disturbance of gait, loss of speech, muscle weakness or paralysis and abnormal movements. Sensory symptoms include pain anaesthesias, blindness and deafness...

_Hysterical symptoms may mimic almost any medical condition_, and the diagnosis is even more difficult when there is an ‘hysterical overlay’ [italics added].

It would appear that what is happening here is that a creed is being recited, without any attempt to appeal to evidence, in the expectation that the ‘truth’ of what is being said will be accepted on trust. The extraordinary claim that ‘hysterical symptoms may mimic almost any medical condition’ derives ultimately not from any body of medical knowledge, but from centuries-old medical lore which goes back at least to the time of Sydenham in the seventeenth century and has no more evidential basis than astrology or tea-leaf reading.

The claim that hysteria may, by its alleged skill in imitating genuine organic diseases, actually have the effect of deceiving the unwary physician, comes in two different forms. It is notable, for example, that Sir Francis Walshe, in his robust reply to Eliot Slater’s
1965 paper, expressed a fervent belief in the potential deceitfulness not simply of hyste-
ria, but of the presumed hysterical, who was said to be given to untruthfulness and ‘pathological lying’:

for it has long been acknowledged that the hysterical is a master, or a mistress
of this upon occasion, and it may be an integral element in what is essen-
tially a psychical illness. Lhermitte has said that ‘hysteria is the mother of
deceit and trickery’.37

More commonly, however, it is hysteria itself which is seen as the potential deceiver,
against whose strategies of dissimulation good physicians should be for ever on their
guard.

An instructive example of the kind of consciousness which this may create both in
specialist and non-specialist physicians is provided by an article, ‘Pseudoneurologic
Syndromes: Recognition and Diagnosis’, written by two neurologists, Aziz Shaibani and
Marwan Sabbagh, which appeared in the American Family Physician in 1998. The abstract
of the article recites the same creed we have already encountered in Marsden and
Taylor’s standard neurological textbook:

Physicians may encounter patients with a collection of psychologic disor-
ders that present with neurologic symptoms or signs, yet have no identifi-
able structural or functional etiology within the nervous system. These dis-
orders comprise the so-called pseudoneurologic syndromes, which can mimic
almost any organic disease [italics added].

The opening paragraph of the article then sets out the doctrine on which everything that
follows is based:

When the mind-body relationship becomes unbalanced to the point of a dis-
eased state, the psyche can manifest several well-described disorders such as
somatization disorder, conversion disorder, psychogenic pain disorder
and factitious disorder. Perhaps the most common constellation of present-
ing symptoms and signs for these disorders are neurologic, and they consti-
tute the pseudoneurologic syndromes discussed in this review.

Once again there is no sign of critical reflection on the doctrines which are being ex-
pounded. The authority which is invoked in this case, the Diagnostic and Statistical
Manual of Mental Disorders (DSM-IV), is apparently treated as having the inerrancy of
scripture, and is deferred to accordingly.

The authors go on to consider a wide variety of allegedly psychogenic symptoms.
Although, as one would expect, they accept the organic aetiology of a variety of move-
ment disorders, including ‘tremor, parkinsonism, myoclonus, dystonia, tics and dyskine-
sia’, this is only the beginning of the story. For since they maintain, as an article of faith,
Sydenham’s seventeenth-century doctrine that hysteria ‘can mimic almost any organic
disease’ they are obliged to hold that every genuine neurological syndrome possesses a
kind of malign doppelgänger, an arch-deceiver which resembles its neurological counterpart in a number of crucial respects but which, if carefully interrogated by an artful physician, can be unmasked for the psychogenic impostor it is. Thus, we are told, ‘psychogenic parkinsonism’ can be distinguished from true neurological parkinsonism by various signs including subtle variations in the frequency and rhythmicity of the tremors by which patients are afflicted.

Although the two neurologists make no mention of psychogenic Lyme disease, or psychogenic HIV Aids (or, for that matter, psychogenic Creutzfeldt-Jacob disease), it presumably will not be long before these conditions too are detected by them or by their ever-vigilant colleagues.

One of the many features which is troubling about the article by Shaibani and Sabbagh is that the ‘clues’ to psychogenicity which they suggest are in some cases of proven unreliability. One such clue is that the conditions under scrutiny (which naturally include seizures) can be ‘precipitated by stress’. Yet research which was carried out as long ago as 1971 indicated that emotional stress could precipitate seizures in 21 per cent of patients with confirmed diagnoses of temporal lobe epilepsy.38

More specifically Shaibani and Sabbagh suggest that ‘Events that are related to either inducing or stopping seizures are suggestive of pseudoseizures’ and go on to note a case of supposedly psychogenic epilepsy induced by saline injection. They appear not to be aware of a paper by R. P. Lesser published in 1983 in which he described how he used a process of ‘suggestion’ (apparently the use of a saline injection as described in another of his papers) to induce what was presumed to be a psychogenic episode. This was subsequently confirmed as a genuine epileptic seizure whose underlying cause was a malignant brain tumour. Lesser and his colleagues recommended that the fact that seizure-like symptoms can be induced by suggestion should not be used to confirm a diagnosis of hysteria.39

Shaibani and Sabbagh also suggest that ‘pelvic thrusting’ may be indicative of a psychogenic seizure and appear to be unaware of a case history published by the neurologist Colin Binnie in 1988 in which rhythmic pelvic thrusting was found in association with frontal lobe seizures.40

Historically, as should by now be clear, the proposition that there are such things as hysterical or psychogenic seizures is deeply suspect. For it is founded squarely on Charcot’s belief that patients exhibiting the classic signs of temporal lobe epilepsy were actually having hysterical fits. It is founded, in other words, on a grave diagnostic error.

To say this, of course, is not to suggest that all neurological-seeming seizures are what they appear to be. There are undoubtedly cases where patients deliberately, or perhaps even without full knowledge of what they are doing, simulate epileptic fits for emotional or psychological reasons. But a simulated epileptic fit, which is by definition a product of a normal, pathology-free brain, cannot, or should not, be construed as a hysterical or psychogenic seizure, which is supposedly non-simulated and beyond the control of the patient afflicted by it.
Although the vast majority of modern neurologists would appear to be convinced of the reality of hysterical seizures (usually now referred to as pseudoseizures or non-epileptic events), this belief, like so many others associated with the diagnosis of hysteria, does not rest on any solid evidence. The clinical evidence is in fact equivocal and the belief rests principally on the weight of tradition and on the mistaken belief that Charcot’s original distinction between epileptic and hysterical fits was well-founded.

It remains possible that the episodes which are now classified as pseudoseizures consist entirely of a combination of: a) deliberately or unwittingly simulated seizures; b) episodes with other non-epileptic organic causes (such as, for example, syncope); and c) genuine epileptic seizures which cannot be detected by current techniques.

Perhaps the most striking commentary on this question is that provided by Professor Alan Richens, one of the editors of the standard textbook on epilepsy in which Colin Binnie’s case history, referred to above, appears. In a letter in response to an inquiry which I made of him in December 1993, Professor Richens wrote as follows:

As you are well aware, our knowledge about epilepsy has advanced considerably since Charcot’s day, with the advent of EEG and various imaging techniques. However, there is still a small proportion of patients in whom the diagnosis remains elusive because the clinical presentation is atypical and the investigations are normal, even the EEG recorded during a seizure. The term ‘hysterical seizures’ is now seldom used for these patients, most neurologists preferring to call these attacks ‘non-epileptic seizures’. I suspect, however, that like Charcot we are failing to recognise organic pathology in the absence of sufficiently sensitive techniques. Colin Binnie’s patient in our A Textbook on Epilepsy was fortunate in showing an ictal abnormality in the EEG. I believe that a number of patients are less fortunate in that they have a deep-seated focal abnormality which does not reveal itself even in an ictal recording [i.e. one taken during a seizure] ... I do recognise, however, that occasionally feigned attacks can occur, often in those who have or have had true epileptic seizures, and who are attention-seeking in some form or other. But they should not be labelled ‘hysterical’ [italics added].

This balanced and reasonable appraisal of the situation by one of the leading authorities on epilepsy is far removed from the views which we find in Shaibani and Sabbagh’s article on ‘pseudoneurologic syndromes’. For these authors appear to approach their task in something of the same spirit as an exorcist who must first determine whether his subject is animated by a benign spirit (a genuine neurological disorder) or whether he or she is possessed by a treacherous demon (a psychogenic illness) – in which case the attentions of a psychiatrist are sought.

The fact that modern neurologists should resemble an exorcist casting out demons is perhaps not a coincidence. For it seems to fit entirely with the sacred nature of the various beliefs which surround the diagnosis of hysteria. Indeed it would seem that this diagnosis might well appeal powerfully to physicians who need to perceive themselves
as righteous crusaders, pitted in an almost apocalyptic battle against an arch-deceiver whose many disguises must be unmasked in order for the truth to prevail.
12 Mysterious mechanisms

Precisely because the current move towards rehabilitating the hysteria diagnosis resembles a revivalist religious movement, it seems unlikely that it will be easily deflected from its course. But if it is true that any religious movement is potentially dangerous, then it is almost certainly the case that crusading movements which secrete themselves within ostensibly rational institutions, such as the profession of medicine, and seek to advance their cause using the discourse of science, are doubly dangerous.

When we review the history of the concept of ‘hysteria’ itself in relation to the series of misdiagnoses perpetrated by Charcot and Freud, these dangers are painfully visible. It was, no doubt, partly for this reason, and partly because of the cogent and comprehensive attack which was launched on the diagnosis of hysteria by Eliot Slater in his 1965 paper, that the 1970s and the 1980s saw something approaching a terminological revolution. During these decades the use of the term ‘hysteria’ rapidly declined. In the United States in particular the diagnosis seemed, in theory at least, almost to disappear from mainstream psychiatry.

In fact the reaction against the traditional terminology associated with hysteria started long before Slater’s contribution. The German psychiatrist Kranz put forward this view in 1953:

[H]ysterical phenomena are only modes of reaction which fundamentally are available to everybody and are not in themselves abnormal, but become so in that they last unduly long, become fixed or are excessive ... It is reasonable to ask that we should at least drop the word ‘hysteria’ in favour of ‘hysterical reaction’, and in the end give up this term too, loaded as it is with moral value judgments: we can make ourselves understood by psychiatrists without it.42

A year earlier, in 1952, the American Psychiatric Association took a significant step when the term hysteria, as a separate diagnostic category, disappeared from their Diagnostic and Statistical Manual of Mental Disorders.

The shift away from traditional terminology has been consolidated in later editions of this manual, which is sometimes referred to as the ‘bible of American psychiatry’. But although ‘hysteria’ is now conspicuously absent from the list of recognised diagnoses, the manual does give criteria for the diagnosis of three disorders which are clearly derived from the traditional concept – ‘conversion disorder’, ‘somatization disorder’ and ‘histrionic personality disorder’. The research criteria for the diagnosis of ‘conversion disorder’ as given by the third edition (DSM III) in 1980 were as follows:

The predominant disturbance is a loss of or alteration in physical functioning suggesting a physical disorder. It is involuntary and medically unexplainable... ... One of the following must also be present:
(1) A temporal relationship between symptom onset and some external event of psychological conflict.
(2) The symptom allows the individual to avoid unpleasant activity.
(3) The symptom provides opportunity for support which may not have been otherwise available.43

There are at least two apparent advantages of this approach. In the first place the disappearance of the label ‘hysterical’, with its pejorative and morally censorious overtones, may seem to be a considerable gain. In the second place the insistence that the physical symptom should be involuntary has the effect of separating this putative psychiatric disorder from deliberately feigned or simulated illnesses – a category which the concept of ‘hysteria’ has, on some occasions at least, confusingly to embraced.

The DSM III definition of conversion order, however, is far from satisfactory. One major problem is that, although it excludes consciously simulated illness, it does not exclude the unconscious simulation of illness. What this means in practice is that patients with imaginary symptoms which have no apparent physiological basis have to be placed in the same category as patients whose symptoms seem real, but are not susceptible to medical explanation. The dangers of this approach should become evident if we consider the subsidiary indicators given for the disorder. Criterion (2) – that the symptom allows the individual to avoid unpleasant activity – is, it will be noted, scarcely specific to emotionally based disorders. Most forms of illness, from broken legs to acute appendicitis, create just such opportunities. Criterion (2) is thus rather like saying that a specific name may be given to a plant providing that its leaves are green. Though the restriction may create the illusion of rigour, the field of definition is not very much reduced. Something similar can be said about the next criterion. For since most illnesses provide an opportunity for seeking support – if only from a physician – criterion (3) is almost as empty as criterion (2). Among the subsidiary criteria this leaves only (1), which demands that there should be some kind of temporal relationship between the onset of the illness and ‘some external event of psychological conflict’. The most fitting response to this is perhaps Slater’s, in the words which have already been quoted: ‘Unfortunately we have to recognise that trouble, discord, anxiety and frustration are so prevalent at all stages of life that their mere occurrence near to the time of onset of an illness does not mean very much.’44

In view of the fact that the subsidiary criteria (1), (2) and (3) are objectively empty, or very nearly so, it would seem that, in DSM III, the diagnosis of ‘conversion disorder’ relies almost entirely on the main condition and that therefore the only strict criterion is that the patient’s symptoms were medically inexplicable.

It is difficult not to draw the conclusion that, in formulating its criteria in this particular instance, the American Psychiatric Association did little more than take an old diagnostic error and give it a new name together with a new aura of respectability. Since the very concept of ‘conversion’ is specifically psychoanalytic, and since it is historically indivisible from Freud’s own idiosyncratic theories of ‘hysteria’, it further seems that the creation of the category ‘conversion disorder’ was a politically astute way of preserving the old concept of ‘hysteria’ in euphemistic disguise.
To confer medical respectability on a label originally invented by a nineteenth-century nerve-doctor who put forward as a scientific fact an entirely fictional account of the pathology of ‘hysteria’ seems, on the face of it, an unsatisfactory way of dealing with medical uncertainty. The idea that conversion symptoms ‘represent painful emotion converted into physical innervations’, to cite one standard psychoanalytic formulation, was commented on robustly as early as 1926 by the Oxford psychologist William McDougall in his Outline of Abnormal Psychology. Having made critical observations about one aspect of the psychoanalytic theory of symptoms, he went on to write these words:

> Even less satisfactory is the language of those psychoanalysts who are content to postulate within the organism a ‘mechanism’ of conversion through which various mental entities are put, much as you put in a pound of pork at one end of a mechanism and get it out in the form of sausages at the other. It seems to me far better frankly to admit our ignorance than to resort to such mythological and mechanical devices.\(^{45}\)

Unfortunately McDougall’s trenchant views, if indeed they ever became known to the compilers of the Diagnostic and Statistical Manual, appear to have exercised no influence over them. Since 1980, DSM III has itself been revised and the definition of conversion disorder has been modified yet again. But the underlying concept has remained unaltered. At the same time, although relatively new terms such as ‘conversion disorder’ and ‘somatization’ gained more and more currency, they have not entirely succeeded in ousting the older terminology. As Aubrey Lewis predicted, the term ‘hysteria’ has outlived its obituarists, and is still sometimes used as a diagnosis.

Even where the concept of ‘hysteria’ has been discarded, there has usually been no appreciable gain, principally because of the confusion, or conceptual emptiness, associated with the terms which have been adopted in its place. If one example of this has been provided by the term ‘conversion’, another is furnished by the increasingly widespread use of the term ‘somatization’. In 1980 DSM III adopted ‘somatization disorder’ as a recognised psychiatric diagnosis, characterising the disorder as a syndrome of multiple somatic symptoms that cannot be explained medically. The revised edition of DSM III, produced in 1987, requires a history of several years’ duration beginning before the age of thirty. The patient must have at least thirteen symptoms from a list of thirty-five. According to the most recent edition of the most authoritative American psychiatric textbook, Kaplan and Sadock’s Comprehensive Textbook of Psychiatry, ‘A symptom need only be reported by the patient in order to be counted; it is not necessary to establish that the patient actually had the symptom.’\(^{46}\) Among the symptoms included in the list of thirty-five are diarrhoea, nausea, back pain, chest pain, trouble walking (sic), difficulty urinating, sexual indifference, and menstrual periods which are judged by the patient concerned to be more irregular or more painful than is normal.

As has already been acknowledged, there can be no doubt that physicians do frequently encounter patients who report multiple physical symptoms which they have imagined or exaggerated because of their anxiety, insecurity or need for attention, and that many
such patients believe themselves to be genuinely ill. The problem posed by such patients is an extremely serious one, partly because they can use up a disproportionate amount of a country’s health services, and partly because their tendency to take refuge in illness often masks serious psychological distress. But describing such patients as ‘somatisers’ or judging that they suffer from ‘somatization disorder’ merely adds another layer of confusion to a situation which is already confused enough. For the term somatization has at least two different, mutually contradictory meanings. In Kaplan and Sadock’s Comprehensive Textbook of Psychiatry V we are offered the following definition of the term in the section devoted to ‘Somatoform Disorders’: ‘Somatization is the tendency to experience, to conceptualise and to communicate mental states and personal distress as bodily complaints and medical symptoms.’ We are told that somatization is a general psychological disposition and that it is not in itself a psychiatric disorder although it can become one in extreme manifestations. We are further told that whereas the concept of a conversion reaction was elaborated in the psychoanalytic tradition, ‘somatization disorder originated in the phenomenological and descriptive approach’.

Yet if we turn from the section on ‘Somatoform Disorders’ to that devoted to classical psychoanalysis, we find that the concept of somatization makes its appearance in a list of ‘Immature Defence Mechanisms’. In somatization, we are told, ‘psychic derivatives are converted into bodily symptoms and there is the tendency to react with somatic rather than psychic manifestations.’ On this view, then, somatization, far from being distinct from conversion, appears to be cognate with the process of hysterical conversion which Freud himself postulated and which was adopted as a key aetiological assumption in the first edition of the DSM, which defined a conversion reaction as a functional symptom resulting from the conversion of anxiety into bodily sensations. The psychiatrist Z. J. Lipowski, who has had a major influence on popularising the term, actually confirms its origin in psychoanalytic terminology when he writes that the term ‘was introduced by Stekel early in this century to refer to a hypothetical process whereby a “deep-seated” neurosis could cause a bodily disorder.’ As Lipowski notes, the term somatization ‘was thus related to, if not identical with, the concept of conversion’. Having acknowledged its psychoanalytic origins, Lipowski then goes on to use the term in the non psychoanalytic sense given above.

The confusion over what somatization actually means, and where the concept comes from, is significant. For while it may well be the case that it has been redefined in terms of phenomenology, it must be suggested that its strongest appeal to psychiatrists, and the reason it has been adopted so widely, springs from the fact that it is both congruent with psychoanalytic assumptions and, ostensibly at least, independent of them. In its ‘strong’ sense, which also coincides with its etymological sense, the word ‘somatization’ refers to a process whereby real physical symptoms are supposedly created by transforming psychological or emotional energy into somatic form. In its ‘weak’ sense the word refers to a process in which patients use a multiplicity of physical symptoms, which may be imaginary or non-existent, in order to mask depression or anxiety or in order to establish a particular kind of relationship with doctors. A major problem stemming from this conceptual double-life is that the widespread use of the ‘weak’ form of the word actually tends to reinforce the psychosomatic fundamentalism of those wed-
ded to the ‘strong’ form of the word and to the psychoanalytic aetiologies associated with it.\footnote{51}

The greatest practical danger of this state of affairs is that it encourages physicians to entertain, in a somewhat inchoate form, the extreme Charcotian or Freudian assumption that almost any physical symptom can be produced psychosomatically. I have already cited one telling example of this assumption in the passage quoted earlier from C. David Marsden’s textbook, Clinical Neurology, where medical students are told that ‘Hysterical symptoms may mimic almost any medical condition’.

The capacity of such formulations to mislead is perhaps best understood if we place them alongside an extreme version of psychosomatics such as that of Freud’s follower Georg Groddeck. Groddeck believed (or sometimes behaved as though he believed) that illnesses performed a psychological function and that specific illnesses could actually be produced by the unconscious, which he called the ‘It’:

Sometime or other in the course of the treatment I am accustomed to call my patient’s attention to the fact that from the human semen there is born, not a dog, nor a cat, but a human being, that there is some force within the germ which is able to fashion a nose, a finger, a brain, [and] that accordingly this force, which carries out such marvellous processes, might well produce a headache or diarrhoea or an inflamed throat, that indeed I do not consider it unreasonable to suppose that it can even manufacture pneumonia or gout or cancer. I dare to go so far with my patients as to maintain that the force really does such things, that according to its pleasure it makes people ill for specific ends ...

In this particular case Groddeck writes that he never worries himself in the least ‘as to whether I believe what I am saying or not’. But he does appear to endorse the view that all diseases have a psychological function:

May I repeat what I am saying? Illness has a purpose; it has to resolve the conflict, to repress it, or to prevent what is already repressed from entering consciousness; it has to punish a sin against a commandment ... Whoever breaks an arm has either sinned or wished to commit a sin with that arm, perhaps murder, perhaps theft or masturbation; whoever goes blind desires no more to see, has sinned with his eyes or wishes to sin with them; whoever gets hoarse has a secret and dares not tell it aloud. But the sickness is also a symbol, a representation of something going on within, a drama staged by the It, by means of which it announces what it could not say with the tongue. In other words, sickness, every sickness, whether it be called organic or ‘nervous’, and death too, are just as purposeful as playing the piano, striking a match, or crossing one’s legs. They are a declaration from the It, clearer, more effective than speech could be, yes, more than the whole of the conscious life can give.\footnote{52}
It would be easy to dismiss Groddeck’s paeans to the purposefulness of disease as a historical curiosity with no relevance to the present. Yet Groddeck’s views are still taken seriously by many people today, including some mainstream physicians. Perhaps the most distinguished and reputable of all such physicians is the neurologist Oliver Sacks.

In his book *Migraine*, Sacks suggests that there is a certain kind of migraine which should be approached not only as a physical event ‘but as a peculiar form of symbolic drama into which the patient has translated important thoughts and feelings’. The symptoms of this kind of migraine can constitute ‘a bodily alphabet or proto-language’ and must be interpreted, he suggests, ‘as if they were palimpsests in which the needs and symbols of the individual are inscribed ...’ One of the main authorities invoked in order to justify this approach is Georg Groddeck. Sacks also explicitly draws a parallel between his own speculative account of ‘situational migraines’ and Freud’s theory of hysteria, which he appears to accept.53

It would seem that one of the reasons Groddeck’s theories continue to exercise an appeal some eighty years after they were first published is that, like Freud’s theories with which they are closely associated, they translate into a persuasive (and highly poetic) register a popular folk-theory of medicine which has a very wide appeal. It is from this perspective, I believe, that we should view the claim that hysteria may ‘mimic almost any medical condition’. When such careless claims are made by experienced physicians in textbooks which credulous medical students are expected to treat with respect, they tend to confer academic respectability on this kind of folk-lore. This, in turn, can all too easily result in dangerous or even fatal misdiagnoses.

In an article dealing with the tendency of doctors to misdiagnose real organic conditions as psychological disorders, Linda Gamlin relates the case of a woman who, by the time she was taken to hospital, was so ill that she nearly died. ‘For over two weeks she had been feverish and extremely weak, with typical signs of liver disease: yellow skin, dark brown urine, and putty-coloured stools.’ The woman’s general practitioner, however, had diagnosed post-natal depression and had associated her illness with an emotional breakdown which she had suffered seven years earlier. This view was repeated by no fewer than four other doctors in her group practice. Only when her husband rang a hospital consultant in desperation was the proper diagnosis of viral hepatitis made and the woman rushed to hospital.54

Another example, cited by the psychologist Ellen Goudsmit in her paper ‘The Psychologisation of Illness’, concerns a woman who developed anxiety, fatigue and intermittent cramp-like abdominal pains after changing her job. Her GP diagnosed irritable bowel syndrome, a condition widely held to be psychogenic. In his view her symptoms had been precipitated by stress at work, and he advised a short period of sick leave. As the pain did not subside, the woman continued to consult her GP but neither he, nor the other doctors who saw her, questioned the diagnosis. A few months later, she noticed that the pain was associated with ‘spasms visible through the abdominal wall’, but the doctors who examined her at the local casualty department continued to invoke irritable bowel syndrome. When the patient eventually consulted another physician (a woman)
she found ‘an easily palpable mass in the left iliac fossa’. An emergency operation confirmed cancer of the sigmoid colon.58

Goudsmit also cites the case of the late Jacqueline Du Pré, who was diagnosed with, multiple sclerosis in 1973 and died fourteen years later at the age of 42. She quotes the following passage from Carol Easton’s biography of the cellist:

There is no specific test for multiple sclerosis. Its early symptoms - fatigue, loss of sensation, weakness and visual changes - are frequently misdiagnosed as psychoneurosis or an even more severe psychiatric disorder, such as hysteria, particularly in women. When doctors could find no organic cause for her complaints, they prescribed a year’s rest, and referred her to a psychiatrist. When she consulted a doctor in Australia about her tenacious fatigue and occasional double vision in her right eye, he dismissed her symptoms as ‘adolescent trauma’ and suggested she take up a relaxing hobby.

Goudsmit notes that, according to the Multiple Sclerosis Society, such an experience is not rare and after months or years of having been told their symptoms are psychological or psychosomatic, many patients feel relieved when they learn that they have MS.56

Such examples can be multiplied almost indefinitely. A common feature of many of them is the credulous and perhaps not always fully conscious acceptance by some physicians of extreme theories of psychosomatic illness for whose correctness there exists no evidence whatsoever, and which are ultimately derived from ancient medical fallacies about the non-existent disease of hysteria.

The careless use of the terms ‘conversion’ and ‘somatization’, and, indeed, the very fact that these medically tendentious terms are used at all, almost inevitably contributes to sustaining this climate of credulity.
13 Mass hysteria or psychiatric error?

One of the problems with terms such as ‘conversion’ and ‘somatisation’ is that they refer to a putative physiological process for whose existence there is no real evidence. However, it would be quite wrong to imply that all the processes designated by the term hysteria are speculative or non-existent. If we turn from the realm of the individual patient to consider the physical symptoms which are sometimes exhibited by groups, then it becomes clear that term ‘hysteria’ can sometimes be applied to processes which are very real. The topic is an important one because it bears directly on the subject which is dealt with in the final part of this essay – the ‘mystery’ disease entity – or presumed disease entity – to which British doctors in the 1950s gave the name myalgic encephalomyelitis but which many physicians, both in Britain and America, have since come to regard as an example of ‘mass hysteria’.

The term ‘mass hysteria’ bears only a loose relation to the term ‘hysteria’ as it has come to be used by physicians. For whereas ‘hysteria’ has traditionally been used as a medical diagnosis, ‘mass hysteria’ has a much more informal significance and belongs as much to the vocabulary of journalists, sociologists and historians as it does to medical specialists. Some forms of collective behaviour such as those involving fainting schoolgirls or the physical symptoms which can result from imaginary gas leaks clearly do have a medical dimension. Other phenomena, such as witch-hunting, apocalyptic cults and prophetic trances, do not. Referring to phenomena such as mass ecstasy and outbreaks of self-punishment among the Flagellants and others, Gregory Zilboorg has written:

These epidemics, while definitely of a pathological order, are certainly psychosocial phenomena rather than manifestations of individual mental illness. The inaccurate terms applied to these phenomena, such as ‘mass hysteria’ or mass psychosis’, are merely descriptive literary phrases and not diagnostic terms, for the individuals who form a part of these mass reactions need not be and are not always mentally sick.57

Although Zilboorg’s words may seem to imply that the term ‘mass hysteria’ can be correctly applied to certain phenomena, it is not clear that this meaning is in fact intended. Even if mass hysteria is defined, as the psychiatrist Simon Wessely has suggested, as ‘an outbreak of abnormal illness behaviour that cannot be explained by physical disease’, we will not find any real medical tradition associated with the term as is the case with its individual counterpart; it is probably best regarded as a colloquial rather than a technical description.

Nevertheless, as I have already suggested, there can be no doubt that the processes designated by this term are, sometimes at least, very real. Perhaps the most common cases are those involving schoolchildren.

From a report which was subsequently published in the British Medical Journal, it would seem that one such case began in Blackburn in Lancashire on 7 October 1965. The case has a particular significance in the history of medicine. It so happened that it occurred
only a few months after the publication of Eliot Slater’s celebrated attack on the diagnosis of hysteria in the *British Medical Journal*. It was chronicled a year later in the same journal by a young psychiatrist who was destined to play a crucial role in rehabilitating the diagnosis which Slater seemed to have discredited for good.

Colin McEvedy, the psychiatrist in question, was the son of a Manchester surgeon. Educated at Harrow and Magdalen College Oxford, he did his clinical training at Guy’s Hospital, qualifying in 1955. After National Service during which he helped to conduct experiments on the effects of oxygen deprivation on high-flying RAF pilots, McEvedy joined the Maudsley Hospital as a fledgling psychiatrist where he so impressed the Director, Professor Aubrey Lewis, that he was invited to join his professorial unit.

Lewis himself had an interest in the diagnosis of hysteria which, as has already been noted, he predicted would ‘outlive its obituarists’. The distinguished professor of psychiatry, however, almost certainly did not realise, when he wrote these words in 1975, how much his young protégé would contribute to hysteria’s longevity. Nor would he recognise just how remarkable – in more ways than one – McEvedy’s contribution would prove to be.

As McEvedy himself recorded in his *BMJ* article (co-authored with local paediatrician Peter Moss), it was at midday on Thursday 7 October 1965 that the Blackburn Medical Officer of Health received a phone call from the headmistress of St Hilda’s, a Church of England secondary day school for girls. In the early part of the morning a few of the girls in the school had complained of feeling ‘dizzy and peculiar’ and some had fainted. Later that same morning the outbreak of illness had turned into an epidemic and the girls were, to use McEvedy’s words, ‘going down like ninepins’. A medical officer who immediately went to the school noted that the girls looked both frightened and shocked. Ambulances took 85 of the most severely affected pupils to hospital. The rest of the girls were sent home and told not to return until the Monday. Of the girls taken to hospital the majority – 51 in all – recovered sufficiently during the course of the afternoon to be sent home. But 34 remained ill and were detained in hospital. Over the weekend six of the girls who had been sent home had to be readmitted either because they had relapsed or because they had been discharged too soon. Three new cases were also taken in although there were reportedly no cases in the girls’ families or in the community at large.58

On Monday the school reassembled, but another epidemic now broke out very similar in character to the first. Fifty-four girls were taken to hospital and the school was closed for the rest of the week. Many of these Monday cases had previously been taken ill on the Thursday and this time just over half had to be kept in hospital overnight because of the severity of their symptoms. When the school finally reassembled on the following Monday, sixty girls said they felt ill again but none actually required hospital admission. The episode then came to a halt.

According to McEvedy the most striking features of the girls who were admitted to hospital on the first Monday ‘were swooning, moaning, chattering of teeth, hyperpnoea [abnormally fast breathing] and tetany – the general picture of gross emotional upset.’
However, the symptoms did not subside as rapidly as had been expected, which was why more than thirty girls were kept in hospital that night. By day 4 twenty of these girls were still in hospital. They continued to faint and, as McEvedy puts it ‘overbreathe to the point of tetany’.

Having set out the basic facts of what happened, McEvedy goes on to give his own interpretation of events:

From the start it was appreciated that most of the girls were hysterical – the psychiatric opinion was obtained on day 2 – but it was thought that some of the school population had probably been genuinely ill, and that it was the sight of these that had triggered off the hysterical reaction. The search for this organic nucleus proved fruitless. Apart from the hyperventilation and tetany physical examination was essentially negative; there were a few cases there were a few cases of altered sensation of the classical glove or stocking type [often associated with hysteria] and considerably more showed patches of altered sensation on the limbs, but there were no other objective findings. Pyrexia [fever] was almost always absent; a small number of girls (fewer than 10) had a low fever but none had a temperature of over 100º (37.8º C).

Having detailed a number of other tests to investigate the possibility of food poisoning or viral infections, all of which proved negative, McEvedy goes on to list the complaints made by the girls in order of incidence. The most common complaint of all was of dizziness (reported by 98 girls), closely followed by fainting and headache. These were followed in turn by ‘shivering; ‘felt cold’, pins and needles, nausea, pain in back or abdomen, ‘felt hot’, overt hyperventilation, general weakness, teeth chattering and tetany. 48 girls complained of numbness in their face, 39 of feelings of panic, 27 of pain in the neck or chest, and 14 of difficulty in speaking. Fewer than ten of the girls actually vomited.

McEvedy suggests that ‘the list as a whole is obviously that of hyperventilation associated with emotional reaction’. He goes on to write that at a clinical conference held on the Wednesday after the second phase of the epidemic

one of us [who] has been interested for some time in the phenomenon of mass hysteria [ie McEvedy] … thought that there was a prima facie case for regarding the outbreak as purely hysterical: the symptoms were predominately subjective complaints; those affected were adolescent females, a notably susceptible group; and there were features in the epidemiological pattern which were difficult to explain on an organic basis. A possible cause was to be found in the polio epidemic which the town had suffered earlier in the year and which could be regarded as a significant neurogenic stress.

A more immediate precursor of the epidemic was a ceremony which had been held in the Anglican cathedral on the previous day. The ceremony had been attended by a member of the Royal Family and because of a delay the girls had been kept waiting
outside the cathedral for some three hours. During this period 20 of them had felt faint enough to have to break ranks and go and lie down:

The whole outing must have made a powerful impression, and next day on the morning bus to school one mistress noted (in retrospect) an air of excitement and a great deal of talk about fainting – exactly who had fainted and how many times. The stage was set.

At assembly there was one faint. The prevailing rate for assembly faints in the school is two or three a week so there was nothing unusual in this. However, after assembly two or three girls said they felt dizzy, and when a fourth was asked to get a glass of water for the original fainter she immediately felt faint herself. To this nucleus a further half-dozen girls were added during the course of the first two periods. These girls were sat on chairs in a corridor running through the centre of the building; a mistress thought that rather than run the risk of girls hurting themselves by falling from the chairs in a second faint, they had better lie on the floor. In this state they were on view during the mid-morning break, and at this point the phenomenon became epidemic. 60

McEvedy goes on to drawn the conclusion that ‘what became epidemic was a piece of behaviour consequent on an emotional state … Once learned, this self-reinforcing piece of behaviour restarted spontaneously whenever the school was reassembled.’

The argument put forward in the paper is a compelling one and it seems to place beyond question that what happened at the school was an episode of ‘mass hysteria’ triggered by feelings of panic among the girls.

The description ‘mass hysteria’, however, like the individual medical diagnosis of ‘hysteria’, has sometimes been applied in error. Wessely, who has himself applied the description to several forms of illness, is obliged to admit that errors have been made:

It has been argued that rapidly dissipating, volatile airborne organic compounds, or a mixture of low levels of industrial air pollutants, coupled with incomplete environmental investigations … could have triggered short-lived symptoms erroneously attributed to mass sociogenic illness. Some researchers conclude that sick building syndrome is attributable, in whole or part, to polluted air… Indeed, a cursory environmental probe leading to the diagnosis of mass sociogenic illness among a group of mostly female garment-makers in Puerto Rico was later traced to toxic fumes that had caused respiratory and degenerative diseases, and some deaths … Hamilton concluded that ‘epidemic hysteria’ at a rayon plant in the 1930s was actually caused by carbon disulphide exposure … An outbreak of abdominal pain, nausea and vomiting at a British school in 1990 included classic features of mass sociogenic illness: a high female attack rate, rapid onset and recovery, hyperventilation and line of sight transmission. Tests later revealed cucumber pesticide contamination … [italics added]. 61
McEvedy’s himself rejects not only a purely organic explanation but also any ‘mixed’ theory according to which a core of organic illness among some of the girls gave rise to a much larger penumbra of emotional panic. It is by no means clear, however, that he is right to do so.

In this respect one of the most curious of McEvedy’s omissions in the article he wrote with local paediatrician Peter Moss, was his failure to report the fact that, as well as claiming a large number of pupils as victims, the epidemic at Blackburn also struck down one of the teachers. We know this because at the end of the first week Moss had himself advised the BMJ that the outbreak of illness had been restricted to girls aged 11-15 ‘with the exception of one case, a middle-aged woman teacher’. Although this information was recorded clearly enough in the BMJ for 16 October 1965, no reference was made to it in McEvedy and Moss’s paper 13 months later. Nor does this paper make any reference to the provisional diagnosis which had been made before the hysteria hypothesis had been adopted. The same BMJ report that mentions the middle-aged teacher notes that the illness, which had at that point affected about 80 girls, ‘is thought to be an unusual type of encephalitis’:

The onset is sudden, often with fainting, violent shivering, hot and cold feelings, and hyperventilation. Tingling in the face, hands and legs follows, and tetany may develop. Within a few minutes or up to half an hour or so the symptoms pass off, but the child may experience further attacks over the next few days. Other features of the illness noted in some cases are pain in the abdomen, which may extend to the back and shoulders, paraesthesiae in the limbs, and areas of altered sensation – notably behind the shoulders, on the thenar eminence [on the palm beneath the thumb], at the tips of the fingers, and in front of the forearms, thighs and shins.62

Here it will be noted that the hyperventilation is seen as being triggered by inflammation of the brain rather than as being the cause of the girls’ condition.

McEvedy’s failure to mention the middle-aged teacher is particularly odd. It is noted in an interesting, and evidently pseudonymous essay written by ‘P R Celsus’ in 1997 and posted on the University of Michigan’s website:

One might interject here that in the numerous school ‘mass hysterias’ examined, almost always there is the (analytic) embarrassment of one or more teachers falling along with the ‘hysteria-prone girls’. Psychogeneicists don’t quite know what to do with these adult ‘hysterics’ so, more often than not, they simply handle them as McEvedy did his adult embarrassment at Blackburn: omit any mention that there had been an ill-fitting adult victim.63

Celsus goes to note that, in the same edition of the BMJ in which the paper on the Blackburn school had appeared, McEvedy had also published his conclusions about another school epidemic which had taken place at a girls’ secondary school in Portsmouth.
In this case half a class of 13-year-olds fell ill in the late afternoon one Thursday and five were taken to hospital complaining of vomiting and abdominal pain. The next day there was an explosive epidemic which started at assembly and involved every class except the fifth during the course of the day. 72 girls had to be sent home though they seemed less ill than cases on the first day, with most complaining of faintness and feeling peculiar. A small number of cases occurred over the weekend. There was then another outbreak on the Monday when 47 girls were sent home and a trickle of new cases during school hours for the remainder of the week.

The results of all tests were negative and the medical officers concerned came to the conclusion that what was in question was a mixed epidemic in which the initial cases were caused by some mild gastro-intestinal illness but that subsequent cases were ‘functional’ – in other words psychogenic. In spite of this, however, McEvedy once again put forward a view of the epidemic as a case of mass hysteria, describing it as ‘probably functional’.  

In the introduction to this second article McEvedy and his fellow authors note that attention had been drawn to outbreaks of illness in schools in other parts of the country by the press publicity which had been given to the Blackburn epidemic. It was evidently in this way that the Portsmouth epidemic had come to McEvedy’s notice. But what he omits to draw attention to is the remarkable fact that the two putative outbreaks of mass hysteria had both begun on the same day – Thursday 7 October 1965.

What is perhaps even more remarkable is that Thursday 7 October 1965 was a date which also appeared in the BMJ in a third context, soon after the initial report of the Blackburn outbreak, whose principal feature was ‘dizziness’, associated with fainting and feelings of nausea. In a letter to the BMJ Dr Doreen Bull, a general practitioner from Leamington Spa in Warwickshire, about a hundred miles from Blackburn, reported that

While conducting surgery on 7 October, I suddenly felt extremely ill, sweating and collapse, and dizziness with nausea. Fortunately I managed to telephone my [practice] partner and recall fumbling to replace the receiver, before transient loss of consciousness. I was taken home somewhat ashen in colour and recovered sufficiently in early evening to see my patients.

In her letter Dr Bull also reported that four days later, on 11 October, her 11-year-old daughter woke from sleep with severe vomiting. The following day she had a temperature of 99°F (37.2°C), slight anorexia and dizziness. She returned to school and was well the remainder of the week until 17 October when she was sent home from school with feelings of dizziness. In the evening of 18 October she complained of severe ‘high epigastric pain going through to the back’. She then collapsed and was pulseless, sweating and ashen, remaining unconscious for possibly half a minute. A consultant surgeon and a paediatrician were called and, after discussion, it was felt that this acute episode was perhaps one of ‘winter vomiting’.

What is perhaps most interesting here is that, although fewer than 10 of the girls in the Blackburn school were reported to have vomited and although a similar number suf-
fered low-grade fever, they experienced almost all the other symptoms noted in Dr Bull’s letter including, to cite Moss’s initial report to the BMJ, ‘pain in the abdomen, which may extend to the back and shoulders’. It is also the case that the pattern of relapse experienced by the eleven-year-old daughter was not dissimilar to that found in the Blackburn girls, many of whom fell ill again four days after the initial symptoms had appeared.

One further perspective on the Blackburn and Portsmouth epidemics is provided by John Wray, a medical officer of health based in Wiltshire. In a letter which appeared in the BMJ in February 1970, Wray wrote:

One of the commonest epidemic diseases seen by family doctors is winter vomiting. Usually only of one day duration, it is so benign that less notice is taken of it than the common cold. Sometimes when it affects schools the medical officer of health is informed, as food poisoning may be suspected. Although no virus has been isolated, it is undoubtedly viral in origin. When I wrote an article on it I found it much commoner in females, as did Gray and Bradley. In other reported epidemics meningeal symptoms have been present. The same virus may produce vertigo and collapse

...When it affects boarding-schools it is put down to ‘epidemic hysteria’ because no physical cause can be found, and the incubation period is so short (sometimes less than one day according to my research) that all the pupils are affected on the same day. But when you follow an epidemic in the general population, as I have, you can definitely rule out hysteria. Without knowing what has happened at the school, parents have reported children suddenly vomiting in the middle of the night. The vomiting is so sudden and unexpected that I am convinced the virus affects the central nervous system (hence the variations of vertigo, collapse or meningeal symptoms).66

The fact that ‘winter vomiting’ appears to have been endemic during this period and that its symptoms show an overlap with those reported both at Blackburn and at Portsmouth, is, of course, far from conclusive. Even the fact that an experienced medical officer later reported that outbreaks of ‘winter vomiting’ were frequently mistaken for hysteria leaves room for argument, not least because vomiting itself, though prominent in the Portsmouth epidemic, was uncommon at Blackburn – where hyperventilation played a key role. At the same time, however, it must be noted that it is in the very nature of viruses that they mutate and change, and subtly alter their symptomatic presentation. It may well be that the overt hyperventilation observed among the Blackburn schoolgirls was brought on by feeling of panic, but a sudden outbreak of real organic disease in a school might itself be thought rather more likely to give rise to feelings of panic than a polio epidemic (however severe) which was already some months in the past.

What these various considerations call into question is the reliability of McEvedy’s reporting and of his verdict that both Blackburn and Portsmouth were episodes of mass hysteria. For all the information cited here, with the exception of Wray’s letter, was available to McEvedy before the publication of his articles, yet none of it featured in
them. What is particularly striking is his omission of any mention of the middle-aged teacher at Blackburn who was also struck down during the course of an epidemic which he portrays as afflicting only pubescent (and therefore ‘hysteria-prone’) girls.

When this omission is set alongside the many others, and McEvedy’s oblique confession that he had for some time been actively seeking cases of ‘mass hysteria’, it is difficult to avoid a particular suspicion. For it would seem that we are here confronted by a case in which the zeal of the investigator to arrive at a wished-for conclusion has overwhelmed his scientific scepticism and led him, at one point at least, to omit a crucial piece of evidence which might tend to cast doubt on this conclusion.

If all that was at issue was the question of whether or not two secondary schools succumbed to mass hysteria on the same day in October 1965, the seeming unreliability of McEvedy’s reporting would be of relatively little moment. However, as we will see, McEvedy was soon to become involved in one of the most important episodes in the history of the hysteria diagnosis, one which would affect the entire course of modern medicine.
14 The Royal Free epidemic

[This section unwritten]
15 Final diagnosis: the case of Carol Terry

If it were the case that that the tradition of misdiagnosis associated with the concept of hysteria applied only to well-described and well-recognised diseases such as viral hepatitis or multiple sclerosis, the implications for modern medicine would be serious enough. However, as should already be evident, the dangers are far greater than this.

There are perhaps two main categories of misdiagnosis associated with hysteria. In the first place it is clear that there is a strong modern association between the diagnosis and various kinds of ‘simple’ neurological deficit. Patients who have lost movement or sensation in a limb, or who have a sensory deficit, such as blindness or deafness which is not accompanied by the normal signs of aural or ocular disease, are, as we have seen, frequently diagnosed as suffering from ‘hysteria’ (or one of its modern aliases such as ‘conversion disorder’). Principally because it is apparently possible for such deficits to be produced by hypnosis, or maintained without there being serious organic lesions – as is demonstrated, for example, by many of Arthur Hurst’s case histories – this particular application of the hysteria diagnosis is perhaps the most plausible of all. However, it bears very little relationship to the historical roots of the concept of ‘hysteria’, which was originally applied not to ‘simple’ neurological deficits but to a pattern of recurrent fits or seizures. One reason why it is unhelpful to perpetuate this label is illustrated well by Hurst’s patients whose neurological deficits could almost always be traced by to some physical trauma and which, although they may have had a significant psychological dimension, were evidently not purely psychological in their origin. A further reason is actually provided by some of the very research which has been undertaken in order to support the diagnosis of hysteria. For what that research seems clearly to demonstrate is that there is an association between some neurological deficits and reduced cerebral blood-flow. Once again this finding suggests an organic rather than a psychological origin for the deficit in question.

The other main category of misdiagnosis associated with the concept of hysteria is found when this label (or one of its modern synonyms) is applied not to a simple neurological deficit but to a complex illness – an illness which has its own varying course and which may be associated with a whole series of physical symptoms. Historically the clearest instances of a complex organic illness being mistaken for a case of hysteria are those provided by patients such as Josef Breuer’s Anna O, and by Charcot’s patient Le Log——, whose cases have already been described.

The reason that cases such as these are so important to the history of Western medicine is that it is that they have given rise to what I have already referred to as a tradition of misdiagnosis. The existence of this tradition means that almost any complex organic illness which is unfamiliar, unrecognised or imperfectly understood is liable to be categorised as a manifestation of hysteria, or understood in some other way as a psychiatric disorder. This is particularly so when the illness in question involved the brain and gives rise to neurological symptoms.
One disturbing modern example of the psychologisation of a relatively uncommon organic illness has already been given in the account of Mrs A, who eventually died of the choking caused by her undiagnosed Creutzfeldt Jacob disease. In this case there can be little doubt that one of the factors which led to and sustained the misdiagnosis of ‘conversion disorder’ was the relative rarity of the disease from which she was actually suffering. In this respect it instructive to place alongside the story of Mrs A a different medical narrative whose ultimate import is very similar.

This narrative, which has been related by the medical writer Berton Roueché, concerns a woman he calls Carol Terry. She was thirty-two when Roueché first interviewed her about her story in 1978 in her office at the Department of Energy in Washington DC, where she worked as an auditor.67

The first manifestations of Mrs Terry’s illness had come in the late summer of 1971, when she had just turned twenty-four. At this time she had been married for two years to a young Italian called Pasquale (whom she called Pat) and had just begun to take fertility pills as they wanted a baby. Around the middle of July she began to feel a little shaky and sometimes her mouth felt tight. Then one day she fainted at work. The insurance firm who employed her at that point sent her home and told her to take a week off. Her local family doctor diagnosed fatigue and gave her a prescription for Valium. She then drove with her husband from the Los Angeles area where they then lived to Salt Lake City to spend her week off at her parents’ house. When she told her mother that she felt shaky and nervous her mother laughed and told her she was simply growing up.

At the end of the week they drove back, but on the way they quarrelled about something and they ended up not speaking for the entire seven hundred-mile journey. They went to bed not speaking. Then at five o’clock she got up, feeling obscurely that she had to do something. She went into the bathroom, took down her husband Pat’s razor, took out the blade and began to slash her wrists – first one and then the other. ‘I didn’t want to die,’ she recalled, ‘I just wanted to take some action. I don’t know why.’ When Pat woke up he screamed at the sight of the blood streaming from her wrists and rushed her to the casualty department of the nearest hospital where they gave her ten stitches.

Pat telephoned her parents who flew down and made arrangements for her to see a psychiatrist. She then spent a month in a psychiatric hospital in the Los Angeles area and, as she gradually became aware of what she had done, she began to feel both guilty and depressed. At the end of a month she began to feel a little better and was discharged. But the psychiatrist felt that she should not be on her own so she left Pat in Los Angeles and went to live with her parents. The psychiatrist she had been referred to in Salt Lake City held regular consultations with her in which she talked and he listened:

Whenever I asked a question, he simply threw it back at me: Why do you ask me that? He seemed to have made up his mind about me before I ever walked into his office. I realize now that I already had a permanent label, a category. And those people, the psychiatrists, deal entirely in labels. I was
an attempted suicide. That translated into a case of hysteria. Everything I said was interpreted according to my category.68

Meanwhile Mrs Terry was becoming frightened because she was finding that it was becoming more and more difficult to do what she wanted with her hands, which felt shaky and clumsy. She found it hard to do quite simple things like buttoning a blouse or washing her face. Whenever she told her psychiatrist this and asked him why she couldn’t make her hands stay still, he would just say ‘Why do you think?’

Eventually the psychiatrist seems to have tired of her and persuaded her to go into a psychiatric hospital where she was given Thorazine (an anti-psychotic sedative), group therapy and encouraged to work with her hands, even though, by this point, she could, she says, ‘barely get a button through a buttonhole’. Although one of the usual effects of Thorazine is to induce dryness of the mouth, Mrs Terry now started to drool.

At this juncture the hospital did perform various physical tests. Discovering that her liver was enlarged, they decided this was an effect of the Thorazine so they took her off the drug and gave her electroshock therapy instead. In January she was discharged and sent home to her parents again.

Although she felt less ‘dopey’, her hands still refused to obey her and kept shaking:

   Everything was getting harder and harder for me – tying my shoes, opening jar lids, setting my hair, writing, even signing my name. But every time I tried to discuss it with the psychiatrist he simply shrugged and said what he had always said: This was my way of showing resentment toward my husband … I was at a loss. All of my symptoms were worsening, and some new ones were coming on. It was getting hard for me to chew and swallow. My right leg was acting funny, I was beginning to limp. My balance was off. I don’t know how many times I tipped over and fell down. I was covered with bruises. But, worst of all, I began to have trouble speaking. I couldn’t seem to articulate; everything came out slurred. Even my family had trouble understanding me.69

At this point she and her husband decided to split up and they started divorce proceedings. Mrs Terry felt strongly though that the only part of her which was still functioning normally was her mind, so that perhaps the trouble wasn’t emotional after all as the psychiatrists maintained but physical.

She arranged to see a neurologist. But after four consultations in which he examined her and checked her reflexes and her walk he told her that her problems were psychological and that such physical problems as she did have were the side effects of Thorazine. He then put her on L-dopa, the drug which is used to control spasms in Parkinson’s disease, and referred her to a new psychiatrist.

As her physical condition deteriorated her new psychiatrist told her that the cause of all her troubles was an unhappy marriage and that her shaking hands were an expression
of her repressed desire to hit her husband. By this time Mrs Terry was spending practically all her time lying down – she found that she could speak better and swallow more easily in that position. Her hands shook so much that she could not hold a book steady enough to read. She found it difficult to swallow even water and began to lose weight.

By December 1972, eighteen months after the signs of her illness had begun to make themselves felt, Mrs Terry seemed to be rapidly worsening. Her parents had become so anxious about her condition that they had begun to consider alternative therapies and her father, in desperation, had taken her to a hypnotist. For about half a minute after she was hypnotised her hands stopped shaking. Then the shaking began all over again.

In January 1973 her divorce from Pat was granted. Now that her unhappy marriage was over her psychiatrist offered a new account of her condition. Her tremor and her other symptoms were no longer related to her marriage. The cause of all her trouble was that she didn’t want to go back to work and was trying to evade responsibility.

By the beginning of February she had lost twenty-five pounds and she was also becoming dehydrated. In a desperate attempt to get the proper medical treatment she felt she needed she insisted on being admitted to hospital again, stipulating on this occasion that it should be the best hospital in town – the University of Utah Medical Center. Eleven days after she had been admitted, however, it was discovered that her medical insurance had run out and she was discharged. Her ‘Discharge Summary’ gave her entire history. It noted that when she had been referred to a neurologist for an evaluation of muscle-tightening on her right side and drooling, this had been interpreted as a reaction to Thorazine. Later this view had been revised by one of the psychiatrists and, ‘without evidence of neurological disease, hysterical-conversion syndrome was considered’. The evaluation of her mental status read:

Alert, oriented. Patient speaks in a high, squeaky, almost unintelligible voice, which makes questioning impossible. Speech is slow, with short phrases. Follows commands readily. Psychomotor – rigid posturing of hands and fingers, hyperextended and stiff jaws. Patient moves in a very mechanical fashion.70

The results of a routine medical examination, which included pulse, blood pressure, temperature, eyes, chest, heart and abdomen were noted as being essentially normal. So, with one exception, were the results of laboratory tests. The exception was a SGOT test which measures the amount of the enzyme glutamic-oxaloacetic transaminase (GOT) found in the blood. An excess of this enzyme indicates liver damage and Mrs Terry’s blood gave a concentration of a hundred and ten units as opposed to the normal figure of forty. Her discharge summary, however, ignored this finding principally because the psychiatrists had concluded that she did have control over her bodily movements, her tremor and her voice but that she was declining to exercise this control. The report concluded with this brief paragraph:

Final diagnosis: Hysterical neurosis, conversion type. Disposition: The patient is transferred to Granite Community Mental Health Center. The patient
did not receive any medication during hospitalisation nor on transfer. Prognosis: Fair.71

That same day Mrs Terry was admitted to the Community Mental Health Center as a welfare patient. This transfer proved a stroke of good fortune. For one of the doctors on the staff of the Center, David Reiser, was struck by her appearance. She reminded him of a patient he had seen while at medical school and this prompted him to take a second look at her file. This quickened his suspicions and he referred her almost immediately to John Shields, a physician at a neighbouring clinic, for tests. Eventually he called Mrs Terry in. She later recalled what happened:

I knew something important had happened … It was the way Dr Shields and the ophthalmologist stood there looking at one another and nodding. I waited and finally Dr Shields turned to me. He wasn’t exactly smiling but he certainly looked pleased. He said they thought they knew what was wrong with me. He said they thought my trouble was basically not psychiatric. He said they thought I had a disease called Wilson’s disease. He said he understood that it was treatable … I heard what Dr Shields was telling me. I drank in every wonderful word of it. But the thing that mattered most – the thing that put me up in seventh heaven – was that I had a real disease. I wasn’t a psychiatric case. I wasn’t crazy.72

In fact, far from suffering from some disorder of the mind, Mrs Terry’s body was slowly being poisoned. Wilson’s disease is a genetic disorder of copper metabolism. Copper is one of the elements which is essential in tiny quantities to the normal functioning of the body. But if the body fails to excrete copper the result is a form of chronic copper poisoning. Wilson’s disease was first described by the British neurologist Kinnier Wilson in 1912. Although he suspected a toxic process, the association of the symptoms with copper was not established until 1948 when it was recognised that it upsets the natural balance of copper ingestion and copper excretion. At first the surplus of copper is stored in the liver and at this stage the patient will often be symptom-free. However, when the capacity of the liver to store copper is exceeded, it passes into the bloodstream and thence to the cornea of the eye and to the brain. Characteristic neurological disturbances associated with Wilson’s disease include slurred speech, failing voice, excessive salivation, drooling, difficulty in swallowing, tremor, incoordination and muscular rigidity progressing to a state of complete physical helplessness. It can also significantly affect people’s emotions and can lead to moodiness, anxiety or depression.

The tendency of Wilson’s disease to cause copper to accumulate in the cornea of the eye produces its most distinctive marker, which is known as the Kayser-Fleischer ring. This consists of an almost complete ring of brown pigmentation – a copper sediment – around the rim of the cornea. Occurring in approximately 95% of patients with late-stage Wilson’s disease, the Kayser-Fleischer ring is diagnostic of the condition. A failure to recognise its significance is critical since, unless the disease is treated, it is invariably fatal.
Carol Terry’s eyes were dark hazel, which made her Kayser-Fleischer rings difficult to notice at a glance. But when she was seen by Dr George E Cartwright, one of the foremost international experts on the condition, who happened to be based in Utah, he found her appearance characteristic:

I’ll never forget the look of her. She looked like Wilson’s disease. She had the typical masklike face and the fixed and twisted smile. She had what we call the wing-flapping tremor in her arms. Her fingers were constantly moving in what we call a pill-rolling tremor. She was drooling. I thought her Kayser-Fleischer rings were grossly visible. When I said hello to her, she answered me in that distinctive slurred speech, and in that typically squeaky voice. And then she gave that laugh they have. It’s the damndest laugh – it doesn’t come in the usual way, it comes on inhalation. It was all there. She was practically a textbook presentation. I think my secretaries could have made the diagnosis.

Once the diagnosis was made, and confirmed by laboratory tests, Mrs Terry was treated with penicillamine, a non-antibiotic derivative of penicillin which has the ability to bind with metal in the human body and to cause it to be excreted in the urine. Because the diagnosis had been made just in time, before there was significant irreversible damage, she made a slow but almost complete recovery over the next year. A slight limp, some mild slurring of her speech and an occasional tremor (which was apparently associated with anxiety) were the only residual signs of the illness. She also made an excellent emotional recovery. She returned to college, gained a degree and took a new job where she met and married Michael Terry.

‘Well yes,’ Mrs Terry told Berton Roueché on the last occasion that they talked. ‘I’ve thought about a malpractice suit. I’m told I have a good case. Those psychiatrists – I wouldn’t wish them on my worst enemy. But then – I don’t know. I guess they’re only human. So I think I’ll live and let live.’

There can be little doubt that one of the reasons Carol Terry’s condition was not diagnosed correctly during her first two years of medical care was the relative rarity of the illness from which she was suffering. It is estimated that Wilson’s disease has an incidence of one in 30,000 live births in most parts of the world. Although it has a much higher incidence in central America (and particularly in El Salvador where an incidence of 1 in 186 has been reported) this means that, at any one time, there may be no more than 10,000 cases in the whole of the United States and 1,500 in the United Kingdom. As a result, relatively few physicians will have the chance to observe the symptoms at first hand. The fact that Mrs Terry was eventually diagnosed by a physician who had encountered a patient with the disease is an indication of just how important such first-hand experience can be.

One of the other reasons the diagnosis can sometimes be so difficult is the tendency of copper poisoning to manifest itself in subtle emotional forms which do not resemble the symptoms of a ‘physical’ illness at all. Sometimes the initial presenting symptom may be mistaken simply for a form of adolescent ‘moodiness’ or emotional withdrawal. It has
been estimated that one fifth of patients present with purely psychiatric symptoms and that two-thirds of patients presenting only with neurological symptoms are routinely misdiagnosed. The rate of misdiagnosis in patients with purely psychiatric symptoms is even higher.76

There are a number of cases where patients have initially been diagnosed as suffering from schizophrenia. One such case concerned a young man who first developed psychiatric symptoms at the age of thirteen. In 1992, at the age of nineteen, he made his first suicide attempt by an overdose of tablets, as he felt tyrannized at work and deceived by his girlfriend. Around this time aggressive episodes and frequent changes in mood increased. He made another suicide attempt when he failed his driving test.

In 1994 the patient was admitted to a ward for the first time to clarify the cause of several symptoms: loss of appetite, intermittent wakefulness, tendency to withdraw, lack of drive, loss of interest, and ‘apathetic change of personality’. He later expressed fears of having AIDS, of his flat being burned down, and of his family being murdered.

In December 1994, because of feelings of pursuit, thought insertion, and sleep disturbance, he was readmitted to the hospital. He felt he was being controlled and poisoned. This time the diagnosis was ‘delusional disorder, persecutory type with organic influence.’

In October 1995 the patient was taken to a neurology department to clear up any organic causes of his symptoms which had included for some time a Parkinson-like tremor. Because his aggressive outbursts, compulsive acts, and ‘nihilistic delusions’ grew progressively worse he was transferred to a psychiatric ward after twelve days. Only at this stage was the diagnosis of Wilson’s disease made. He was treated with penicillamine but it was too late to improve his symptoms significantly.

Commenting on this case, Jürgen Müller writes as follows:

When confronted with an unclear, [puzzling] illness, thinking of Wilson’s Disease is the most important thing for a neuropsychiatrist to do. Diagnosis is seldom missed because of a lack of knowledge; mostly, it is clinical routine that prevents us from seeing atypical courses. Case reports could help us to be more aware of Wilson’s Disease.

The comment is a significant one in its somewhat curious claim that ‘diagnosis is seldom missed because of a lack of knowledge’. From the context it is reasonably clear that what is meant here is ‘lack of abstract knowledge about the existence and nature of Wilson’s disease’. The implied assumption – that that clinical experience and case reports do not themselves constitute ‘knowledge’ – sums up a great deal which is wrong with modern medicine, where clinical knowledge has been progressively devalued and laboratory tests have been accorded more and more weight in the making of a diagnosis.

Beyond this, however, there is a larger problem, which George Cartwright, the specialist who eventually treated Carol Terry, describes in relation to her case:
The tragic thing is that her liver involvement had been noted in all her hospital stays. Noted, but either ignored or misinterpreted. The trouble is, of course, that cases like [Mrs Terry’s], which present both psychological and neurological symptoms, are almost always diagnosed as psychiatric problems. At least fifty per cent of the cases I’ve seen or know something about were first diagnosed as functional [ie psychogenic]. And a psychiatric diagnosis tends to stick. I fault psychiatry on that. The psychiatrist should not assume that every patient in the six-to-thirty-two age group who seems to be in need of psychiatric help is actually a psychiatric patient. He should stop and consider the possibility of somatic illness. And in a referral he shouldn’t simply accept the previous diagnosis. He should see for himself. I’ll go further. I would suggest that all individuals admitted to psychiatric wards between the ages of six and thirty-two be screened for Wilson’s disease with a cerulo-plasmin test. It’s a simple test – all you need is a blood sample. That would save a lot of misery, even lives.77

Dr Cartwright’s analysis, it should be noted, was made in 1978 and it is probably true to say that awareness of the dangers of misdiagnosing Wilson’s disease has increased significantly since that time. To his analysis, however, it seems necessary to add one further observation. This is that one of the main reasons why an initial psychiatric diagnosis may still prove resilient and difficult to challenge in cases like this is that a whole series of neurological symptoms have effectively been ‘appropriated’ by psychiatry without there being any sound medical basis for this appropriation. To cite but the most obvious example, Mrs Terry exhibited, almost from the outset, a tremor which should in itself have announced the neurological basis of her illness. This, indeed, took a distinctive form, namely that of the ‘pill-rolling tremor’ (in which the thumb and forefinger move constantly against one another as though rolling a pill) which is characteristic both of Parkinsonism and Wilson’s disease. It is only because the medical mythology surrounding hysteria has persuaded both psychiatrists and neurologists that tremors can be psychogenic, with no organic pathology, that what should have been regarded as a clear sign of neurological disease became effectively invisible.
16 The foulest and the nastiest of creatures

Wilson’s disease may tend sometimes to be rendered difficult to recognise because of its rarity, but it can never be said that it is invisible in the laboratory. As the words of Dr Cartwright, quoted above, make clear, there is a simple blood test, the cerulo-plasmin test, which can be relied on to detect the disease. Although the problem of misdiagnosis posed by Wilson’s disease is likely always to remain significant, the existence of a reliable diagnostic test means that that the scale of this problem will always be limited.

Where the persistence in modern medicine of the mythology associated with hysteria poses a much more serious problem is in the case of neurological illnesses where there are no definitive and reliable tests.

In 1998 when eighteen-year-old Sally Russo graduated from high school in Cape Cod, Massachusetts, she thought she had her life planned out. An outstanding sportswoman, she had gained a scholarship to the local university. The first setback occurred when she had to leave university after one semester for personal reasons. But she resumed her education after winning another scholarship to Miami University on Ohio in 1999. Here she encountered another setback when she injured her back. This ended her career as a star hockey player and forced her to undergo three operations. However she carried on with her education and thought nothing of what she describes as ‘a weird bite’ she noticed on her arm during the summer of 2003. Two days later she found that she could move her arm and so she went to the emergency department of her local hospital where a doctor diagnosed her with shingles and gave her prednisone. She felt better, and soon forgot all about it. Soon, however, she found herself suffering from a series of symptoms and illnesses, including mouth sores and persistent bladder and sinus infections.

After fighting these symptoms valiantly for a few weeks she says she finally ‘lost it’:

I was coming home one day from work … I came in the door and I said to my mother, ‘This is not a sinus infection. Something is wrong. I don’t know what it is. I’m exhausted and have all this muscle and joint pain.’ I was just tired in a way that wasn’t like I needed a nap. It was a physical tired.76

In February 2004, while alone at work, Sally Russo collapsed on the floor and couldn’t get up. She was rescued by her mother who happened to call by and was able to summon an ambulance. After spending a week in Cape Cod Hospital, doctors suspected she might have lupus or rheumatoid arthritis, but they could not settle on a diagnosis.

These symptoms, however, proved to be but the beginning of the illness. That summer she began to have severe cramping in her foot and painful ‘charley horses’ or leg cramps that quickly turned into spasms, first in her legs and eventually throughout her whole body. Both her primary care physician and a muscle specialist in Worcester referred her to the main regional hospital.
She arrived in a wheelchair, paralyzed from the waist down and stayed a week as the spasms turned into full-blown seizures lasting from 10 minutes to an hour. The doctors were perplexed. ‘They did every test under the sun from a spinal tap to brain MRIs. They even checked my back again because they thought something was up with my nerves,’ she recalled later.

At that point doctors were wondering about Parkinson’s disease, multiple sclerosis and amyotrophic lateral sclerosis (ALS), which is a progressive neurodegenerative disease that affects nerve cells in the brain and the spinal cord. At her mother’s urging, they also tested her several times for Lyme disease, but the results all came back negative with one test indeterminate.

Russo left hospital and spent a week in a rehabilitation centre undergoing physiotherapy. The doctors now began to suspect a conversion disorder, and explained to her that they thought her psychological problems might be manifesting themselves as physical symptoms. On this occasion, however, to their credit, and to the credit of the psychiatric profession as a whole, three psychiatrists agreed she did not have conversion disorder.

The reasons which led them to reject this diagnosis are not recorded, but it meant that Russo was free to seek further opinions. A specialist from one clinic diagnosed a possible dopamine disorder and prescribed a medicine that would treat the dopamine levels in her body. The same week she saw Dr. Sam Dontha, a specialist in infectious diseases who is also an international expert on Lyme disease, a bacterial illness spread by the bite of infected ticks which are particularly prevalent in some rural areas of Europe and North America. Dontha immediately quizzed her on the ‘weird bite’ she recalled having shortly before the onset of her symptoms. For although sometimes the painless bite of ticks leaves no visible mark, in about half or more cases an infected bite gives rise to a red rash, known as erythema migrans. The rash begins with a lesion at the site of the tick bite and slowly spreads outwards. Often it does so in a circular pattern with a red rim, so that, although it can assume almost any shape or size, in its most distinctive form it resembles a bull’s eye. The ‘weird bite’ which Sally Russo had noticed on her arm, which she had dismissed at the time as a spider bite, had indeed been in the form of a bull’s eye. This was this was decisive for Dontha, as indeed it would be for any Lyme specialist or ‘Lyme-literate’ physician. ‘I was literally in his office for five minutes and he said, “You have Lyme disease,”’ Russo recalled.

There are a large number of reasons why the diagnosis of Lyme disease tends often to be missed. One is that the clinical tests for the disease are far from being 100% reliable. The Elisa test, which had been given to Russo, frequently returns both false positives and false negatives. So too does the more sophisticated Western blot test (which at this stage she had not even been given). Both tests depend on detecting the presence in the blood of antibodies to particular infections. They are both used in other contexts, the best known being in the diagnosis of the HIV aids. But in the case of Lyme disease the results they give are much less clear-cut.

The other main reason for the difficulties which surround the diagnosis arises from the complexity of Lyme disease and the multiplicity and variability of the symptoms to
which it gives rise. The history of the disease under its modern name is itself revealing, for when the name was first used the illness it designated was known as ‘Lyme arthritis’. The medical pioneer who first drew attention to the illness was not a physician but a mother, Polly Murray, who lived in Lyme, a wooded hamlet at the mouth of the Connecticut river. In 1972 two of Ms Murray’s four children developed painful arthritis in the knee. Their knees became so swollen that they could only walk with crutches. By talking to her friends and neighbours she discovered that a number of other children in the region had developed similar pains in their joints. She knew enough about arthritis to realize that a cluster of cases like this, affecting young children, was unusual. She got in touch with the state health authorities and in 1975 a group of medical scientists from Yale University, headed by rheumatologist Allen Steere, investigated. Mrs Murray was able to hand them a list of dozens who were affected. The team led by Steere, who had previously trained as an epidemiologist with the Centers for Disease Control, America’s medical detective agency, contacted each of the families directly and eventually compiled a list of 39 children who had similar symptoms.²⁹

Having identified what appeared to be a new clinical entity, Steere’s task now was to identify its cause. Some local residents blamed a new nuclear power station further up the river, others blamed the drinking water, the local swimming pool or food that they had eaten. After these and other possibilities had been carefully eliminated Steere began to favour an insect hypothesis, based on the observation that all the symptoms seem to have begun in the summer or the autumn. But only a quarter of the children remembered being bitten and their descriptions of the bite did not always match. None of them had actually seen an insect.

It was two years before the mystery was solved. This happened by chance when an ecologist walked into Dr Steere’s office with a vial containing an insect which he said had tried to bite him. The insect, which was so small that it was barely visible, turned out to be *Ixodes scapularis* (otherwise know as *Ixodes dammini*), a black-legged tick carried on the bodies of deer and field mice. It so happened that this tick was a newcomer to the region and for this reason local insect census takers were tracking its gradual progress across Connecticut. The dramatic moment of discovery came when Steere laid their surveillance maps over his own team’s maps for juvenile rheumatoid arthritis and found that there was an almost exact geographical match between the progress of the tick and the distribution of reported cases.

It was at this point that Dr Steere and his team, disregarding the protests of local civic leaders, decided to name the disease after the locality in which it had been first identified, and coined the term Lyme arthritis. This was in spite of the fact that the ‘new’ illness appears to have been cognate with a condition known by some of the older locals on nearby Long Island as ‘Montauk knee’. Indeed DNA analysis of museum tick collections has documented the presence of spirochete-infected ticks on Montauk Point, Long Island at least 50 years ago.³⁰

The name oddly localised a disease which, although it seems originally to have been concentrated along the New England coast, had by the late 1990s been reported in all states except Alaska and Montana. The three main foci of Lyme disease in the United
States are along the northeastern coast from Maine to Maryland, in the midwest in Wisconsin and Minnesota, and on the west coast in northern California and Oregon.

Though generally carried by a different species of tick outside North America, the disease is also found in many parts of Europe, including France, Germany, Austria, Scandinavia and Eastern Europe. Cases have been reported of Lyme disease infection that has occurred in northern forested regions of Russia, in China, and in Japan. It has not been found in tropical areas or in the southern hemisphere but it has been reported in many parts of the UK, especially Exmoor, the New Forest, the South Downs, parts of Wiltshire and Berkshire, Thetford Forest, the Lake District, the Yorkshire moors and the Scottish parks including Richmond and Bushey.81

Ticks are not, strictly speaking, insects. They are arthropods and are members (along with spiders, scorpions, mites and the horseshoe crab) of the class Arachnida. Because of their noxious, parasitic nature, it has sometimes been said that human detestation of the tick, which lives on the blood of other animals, including deer, dogs, rats and mice, surpasses that aroused by snakes and spiders. ‘Ill-favoured ticks’ were, in the view of Pliny the Elder, in his first-century Natural History, ‘the foulest and nastiest creatures that be.’82

The tick which is the main carrier of Lyme disease in North America, *Ixodes scapularis* is dependent at different parts of its life-cycle principally on deer and field-mice and without deer in particular it could probably not survive. Its seemingly sudden spread through North America towards the end of the twentieth century is almost certainly associated with the recolonisation of countryside by deer which had been hunted almost to extinction by European colonisers but which are now a protected species.

*Ixodes scapularis* is sometimes said to be about the size, and shape and colour of a poppy seed or a sesame seed, though Roueché, who has written interestingly about Lyme disease just as he has about Wilson’s disease, felt that this description was an exaggeration. The particular specimen of *I. dammini* which once nearly bit his wife was, he writes ‘about the size, and shape and colour of the period [full-stop] that ends this sentence.’ It may well be, however, that Roueché was describing a larval tick. Immature ticks at the biting stage, though still tiny, are usually significantly larger and it is said that an engorged female, after having feasted on the blood of its host continuously for as long as seven days, may reach the size of a small pea.83

After Steere had named the disease and identified the agent of infection, another team of scientists set about trying to define its pathophysiology. Most of the laboratory work was done at the Rocky Mountain Laboratories in Hamilton, Montana by a team under the direction of Willy Burgdorfer, of the National Institute of Allergy and Infectious Diseases. They conducted their study using ticks collected on Shelter Island to the east of Long Island and, in 1982, succeeded in isolating, from the midgut of the ticks, a previously unknown organism – a spirochetal bacterium of the genus *borrelia*. By showing that these bacteria reacted to serum taken from patients who had been diagnosed with Lyme disease (and whose blood would therefore carry antibodies to it) Burgdorfer and
his team were able to establish that this was the causative organism; it was duly named *Borrelia Burgdorferi* after its discoverer.

One of the immediate implications of Burgdorfer’s discovery was that Lyme disease, in that it had a bacterial cause, should be susceptible to antibiotic treatment. Prescribing a course of antibiotics immediately became standard procedure. In some patients, particularly when the disease was caught in the early stages, this proved to be an effective cure. In other cases, however, symptoms persisted even after extensive treatment.

Lyme disease is, of course, far from being the only disease to be caused by the slender, spiral and exceptionally mobile organisms known as spirochetes. Syphilis is also caused by a spirochete and the two diseases have some features in common. Perhaps the most important point is that in both cases the spirochete in question has the capacity to penetrate the blood-brain barrier. What this means is that, although the initial symptoms of Lyme disease affect principally the skin, the muscles and the joints (as well as giving rise to headaches, fatigue and flu-like episodes), it can also be associated with a wide variety of neurological symptoms. The most common of these, which can occur within weeks of the initial infection, is facial palsy, in which the muscles on one side of the face become temporarily weak or paralysed. Other early neurological symptoms can include mild forms of encephalitis or meningitis.

Sometimes, however, (as is also the case with syphilis) it appears that the infection lies dormant within the organism for months or even years only to emerge in the form of one or more of a bewildering variety of neurological symptoms which can include neuropathy (taking the form of numbness, tingling, burning, itching or pins-and-needles), tremor, muscle twitching, paresis (weakness) of one side of the body, urinary incontinence, vision problems such as diplopia (double vision), excessive sensitivity to light, sound or vibration, vestibular symptoms associated with balance and the inner or middle ear, seizures, severe startle reactions, panic attacks, short-term memory loss and other cognitive deficits.

Although some of Sally Russo’s symptoms, especially her joint pain and intense fatigue, are characteristic of Lyme disease, others are encountered much less frequently. But there is a sense in which the idiosyncratic presentation is itself a common feature of Lyme. Because it is a multi-system disease it can affect practically any part of the human body. Like the syphilis spirochete, it would appear that *borrelia Burgdorferi* can live within the central nervous system for periods of ten years or more and, as is the case in syphilis, the multivarious signs of the disease can be mistaken for a whole series of other disorders including lupus, multiple sclerosis, rheumatoid arthritis, fibromyalgia, or even, indeed, cancer. Whereas syphilis became known as ‘the great imitator’ because of its many presentations and the ease with which it was confused with other pathologies, so Lyme disease has frequently been dubbed ‘the new great imitator’.  

Lyme disease is also like syphilis in another respect. For one of the most frequent misdiagnoses received by patients suffering from syphilis, especially during the period before the middle of the twentieth century, when the pathophysiology of this complex disease was gradually pieced together, was that of hysteria. Precisely because of the unpredict-
able, ill-understood and complex nature of Lyme disease the same thing has happened in this case.

It may well be that in Sally Russo’s case, perhaps because of the very severity of her physical symptoms, three psychiatrists agreed _not_ to adopt the diagnosis of conversion disorder. Other patients, however, have not been so fortunate. As the psychiatrist and Lyme specialist Robert Bransfield has written

There are an increasing number of patients with chronic Lyme disease (neuroboreliosis) presenting in psychiatric offices. Lyme disease does not begin as a psychiatric illness. Other symptoms occur in early-stage disease. Late in the progression of this disease neurological, cognitive, and psychiatric symptoms predominate. If not well understood, these symptoms are sometimes viewed as non-specific and bizarre. Actually the symptoms can be quite specific with a clear physiological basis, but far too often a routine evaluation is insufficient to adequately evaluate these patients. When the evaluation is not properly targeted, key symptoms can be overlooked and these patients may be mistakenly diagnosed with chronic fatigue syndrome, fibromyalgia, MS, lupus, Epstein Barr, as well as many other medical and psychiatric conditions. They are considered by some to be ‘hypochondriacal’ or ‘crazy’. As a result, many of these patients feel alienated from the mainstream of the health care system. 

Behind the diagnostic difficulties which Bransfield describes here, there lies a full-blown split within the medical profession. Because this split helps to illuminate the hysteria diagnosis and the problems associated with it, it is necessary to consider it briefly here.
17 A professional disagreement

Alan Steere, the epidemiologist and rheumatologist who named Lyme disease, and identified it as a tick-borne infection, was also perhaps the most significant figure in the medical dispute which followed.

In April 1993 he and three colleagues from the New England medical centre in Boston published what proved to be an explosive clinical paper in the Journal of the American Medical Association (JAMA). The paper, which was uncompromisingly entitled ‘The Overdiagnosis of Lyme Disease’, reported the results of a retrospective study of 788 patients referred to Steere’s Lyme disease clinic who were thought by the referring physician (or sometimes by the patients themselves) to have a diagnosis of Lyme disease.

Of the 788 patients, Steere and his colleagues came to the conclusion that 180 (23%) had active Lyme disease, usually manifesting itself as arthritis, or in the form of neurological symptoms. They also reported that, in their view, 156 patients (20%) had previous Lyme disease ‘and another current illness, most commonly chronic fatigue syndrome or fibromyalgia’. In 49 of these patients, these symptoms began soon after objective manifestations of Lyme disease.

These findings, and the manner in which they were reported, were controversial enough. But the truly explosive claim made in the paper was that the remaining 452 patients (57% of the sample) did not have Lyme disease at all and had not ever had it. The majority of these patients, they said, had the symptoms of chronic fatigue syndrome (CFS) or fibromyalgia; the others usually had rheumatic or neurological diseases. To this finding Steere and his colleagues added the comment ‘... psychiatric disorders such as anxiety, depression or somatization clearly played a role in the illness of some of these patients ...’

They went on to say that, of the patients who supposedly did not have Lyme disease, 45% had had positive serological test results for Lyme disease in other laboratories, but all were found to have negative results in Steere’s own university laboratory. Prior to referral, just over half of the 788 patients had been treated with antibiotic therapy. In 322 (79%) of these patients, the reason for lack of response was held to be they had been incorrectly diagnosis and were not in fact suffering from Lyme Disease at all.

The paper duly concluded that ‘Only a minority of the patients referred to the clinic met diagnostic criteria for Lyme disease. The most common reason for lack of response to antibiotic therapy was misdiagnosis.’

The publication of this paper in 1993, carrying as it did Steere’s authority as the physician who had originally named the disease, would cause a storm among both physicians and patients the ripples of which can still be felt now, almost 15 years later.
In view of this the first thing that perhaps needs to be said about Steere’s suggestions was that there was almost bound to be some degree of truth in them. Wherever there is in existence a complex disease with many varying symptoms, for which diagnostic tests are not reliable, then it is likely that some people will be diagnosed as suffering from this disease who do not in fact have it at all. It may also be the case that some patients will actively seek out such a diagnosis either because they are attempting to validate symptoms which they have imagined or exaggerated, or because they have quite genuine symptoms and are seeking to escape from an alternative diagnosis which seems to offer no clinical certainty and no prospect of cure. Chronic Fatigue Syndrome is one example of an uncertain and unhopeful diagnosis which patients may seek to exchange for the more medically respectable label of Lyme disease. Steere himself would eventually offer a very similar view during an interview with the *New York Times*. In a profile of him which appeared in 1999 the *Times* said this of his views on overdiagnosis:

> More recently he has enraged many patients and their doctors with his contention that Lyme disease has become a junk-drawer diagnosis, covering maladies ranging from fibromyalgia to hypochondria. Many people receiving antibiotic treatment, he argued, are being done more harm than good.\(^7\)

The paper goes on to quote Steere as saying ‘I suppose Lyme disease is one of the few diseases that some people want to have, because it’s defined. I think it’s very difficult to have something that is not well understood.’ In these undeniably perceptive words, which seem to contain a clear reference to poorly understood illnesses such as chronic fatigue syndrome and fibromyalgia, Steere seems quite clearly to be suggesting that Lyme disease was in effect being used as an ‘asylum diagnosis’ by medical refugees who were in flight from less desirable diagnoses.

Yet although the kind of process described by Steere certainly does take place to some degree, the scale on which this happens is difficult if not impossible to quantify. Any attempt to estimate such a trend would, apart from anything else, need to be balanced by the opposite argument. For it seems almost certain that a significant number of patients who have received the diagnosis of CFS are indeed Lyme sufferers who have been misdiagnosed. In seeking out a diagnosis of Lyme disease such patients would not be refugees but exiles returning to their true homeland.

The problem with Steere’s paper was not that the kind of misdiagnosis he described does not take place. It is that he sought to quantify it without having any rational or scientific basis on which to do so.

As some other physicians and patient advocates pointed out at the time, one of the most mysterious aspects of Steere’s paper was the faith which he was evidently placing in the superiority of his own laboratory testing methods to those that had been used elsewhere. If Steere’s estimate of the extent of misdiagnosis was correct, wrote Carl Brenner, Marc C. Gabriel and John S. O’Donnell from the patient advocacy group Lymenet:

> it means [that he and his colleagues] have developed an extremely sensitive and specific group of tests that vastly outperform any of the existing anti-
body tests or testing protocols that have been reviewed in the Lyme literature. Indeed, the authors state that of the 452 patients in the study who were determined to have never had Lyme disease, 203 (45%) had obtained ‘false’ positive results from another laboratory.

It is difficult to accept uncritically the authors’ [implicit] claim that their antibody testing protocols are superior to others currently in use. Independent reviews of a wide variety of today’s antibody detection tests … have indicated generally dismal performance, marked by significant interlaboratory and intralaboratory variability. The authors offer no independent evaluations or persuasive arguments to distinguish their tests from others in current use. Instead, it appears that Steere and his co-authors are relying inappropriately on imperfect testing techniques – from a single laboratory – to make the diagnosis of Lyme disease. The presumption seems to be that their tests are better than others because the correlation between seropositivity and actual Lyme disease is highest; on the other hand, the definition of ‘actual Lyme disease’ in the study is derived almost exclusively from their test results. The reasoning is entirely circular.88

The same authors went on to question further the supposed infallibility of Steere’s testing methods by citing an extract from a study of the manner in which pregnant mothers can transmit the Lyme spirochete to the babies they are carrying:

A 24-year old white woman was admitted in February 1985 in labor at term of her pregnancy. Ultrasound examination showed that the fetus was dead when she arrived at the hospital. Following the delivery of her stillborn infant and completion of the fetal autopsy, a retrospective interview established that she had acquired Lyme borreliosis in the first trimester of her pregnancy outside of Salt Lake City, Utah. Postpartum serological studies yielded conflicting results because the Centers for Disease Control found strongly reactive results by IFA and ELISA, as did the New York State Department of Health; however, the Yale University laboratory of Dr. Allen Steere could detect no evidence of specific antibodies for B. burgdorferi. Fetal viscera showed B. burgdorferi in the liver, adrenal, brain, heart and placenta [italics added].89

What this passage shows is that, although autopsy findings clearly confirmed the presence of the Lyme spirochete, Borrelia burgdorferi, in the body of the dead baby, and although this finding was in accord with the results of tests carried out on the mother’s blood by two reputable laboratories (and therefore confirmed the accuracy of these tests), Dr Steere’s laboratory had conducted tests whose negative results were evidently false. As the Lymenet authors go on to write:

The implications of this finding for the patients studied in ‘The Overdiagnosis of Lyme Disease’ are obvious and profound, given that over a quarter of the study population was determined by the authors to have received ‘false positive’ test results from other laboratories. False negatives in Dr. Steere’s
laboratory may very well account for a significant portion of the discrepancy among results.

The authors went on to point out that, although it had been established that Lyme disease was sometimes resistant to antibiotic treatment, Steere and his colleagues, while implicitly accepting this, were simultaneously using a lack of responsiveness to antibiotics as an indication that the patients had been misdiagnosed and were not in fact suffering from the disease at all. ‘That this is offered as “science” in a leading medical journal,’ they conclude, ‘is appalling’.

Interestingly, the criticisms of Steere’s paper made here by a well-informed group of patient advocates were echoed by the true discoverer of the original Lyme cluster, Polly Murray, who had by this time written a book, The Widening Circle, about her family’s struggle with the disease. ‘I am dismayed about Dr. Steere’s position,’ she told the New York Times. ‘He feels that it’s overdiagnosed and overtreated, but I see people in the area who are having a real struggle with getting over Lyme disease. And some of them have responded to longer-term treatment.’

When Steere’s paper is read in the light of the criticisms subsequently made of it, and placed in the context of the times, it is difficult to avoid the impression that he had become anxious and embarrassed that ‘his’ well-defined illness might be being colonised by patients with an ill-defined syndrome – chronic fatigue – sufferers from which were already at this point being widely perceived within the medical profession as hypochondriacs, hysterics or malingers. Indeed, two years earlier, in 1991, an essay had appeared in a major medical journal entitled, ‘From the Centers for Fatigue Control (CFC) Weekly Report. Lyme disease – United States’, in which patients with unexplained persistent fatigue were mocked for wondering whether or not they had Lyme disease, suggesting that they were clinging to a stylish diagnosis unable to accept that they actually suffered from a fictitious ‘Lime Disease’. As the psychiatrist Brian Fallon and his colleagues wrote in 1998:

This sarcastic essay resulted perhaps from a sense that the medical scientific community had full knowledge about this disease. Such was (and is) clearly not the case. The observation that fatigue after Lyme disease is a significant problem, that many of these patients would meet clinical criteria for chronic fatigue syndrome (CFS), and that a substantial portion of these patients appear to have signs in experimental cerebro-spinal fluid studies of persistent infection with B. burgdorferi has led the National Institute of Health to fund major research studies investigating the extent to which ongoing symptoms are due to persistent infection [or to] a ‘post-Lyme’ syndrome.\(^0\)

In Steere’s overhasty leap to the conclusion that Lyme disease was being misdiagnosed on a massive scale, we may see at work once again the tendency of modern medicine to assume omniscience about a disease or a set of symptoms at a stage when few certainties exist and a huge amount remains to be discovered. This injudicious leap would almost certainly never have been made at all had not Steere, like so many of his medical colleagues, absorbed a series of traditional but false beliefs about hysteria and somatization.
which are the common inheritance of all modern physicians and which are themselves one of the chief sources of misdiagnosis.

The overall effect of Steere’s 1993 paper was to open up a lasting rift within the medical profession over the whole question of Lyme disease and its diagnosis. On one side, made up mainly by academic researchers and clinicians who accepted the position of the Centers for Disease Control, was the view that Lyme disease could usually be eradicated by a standard course of antibiotics lasting a matter of weeks. Adherents of this view believed that persistent Lyme disease was relatively rare and that when symptoms did not disappear with treatment this was usually because of a ‘post-Lyme syndrome’. On the opposing side, made up both of clinicians and patient advocates (often associated with ILADS, the International Lyme and Associated Diseases Society), was the view that Lyme disease was sometimes highly resistant to antibiotic treatment, that chronic Lyme disease was therefore relatively common and that long courses of antibiotics, lasting for six months or more, were sometimes both beneficial and necessary. The ILADS view is that the potential harm to patients in letting a persistent Lyme infection go untreated far outweighs the potential side-effects of long-term antibiotic use.

Although the dispute has not yet been fully resolved, the weight of the evidence appears to be on the side of those who have resisted Steere’s arguments. These arguments, indeed, which were clearly intended to warn against the possibility of misdiagnosis, may actually have increased the likelihood of a particular kind of misdiagnosis being made. In a paper published in 1998, Brian Fallon and his colleagues put forwards just such a view. Their paper, whose title, ‘The Underdiagnosis of Neuropsychiatric Lyme Disease in Children and Adults’, echoes and inverts that chosen by Steere, contains the following passage:

Whenever a disease exists for which serologic tests are unreliable in determining the presence or absence of the disease process, frustration and anxiety rise among both patients and doctors. When controversy exists even among the leading academic researchers as to the validity and reliability of these tests, a battleground is then set in which doctors dispute amongst themselves over the diagnosis while the patient is left with an uncertain clinical syndrome in which treatment recommendations vary widely depending on the physician chosen. If this particular disease process also has psychiatric manifestations that lower the patient’s frustration tolerance, increase irritability, and impair cognitive functioning, then the stage is set for a referral to a psychiatrist to address a presumed psychogenic or functional disorder. Such is the situation when dealing with Lyme disease. 91

To cite these words is not to suggest that there was never any truth at all in Steere’s arguments. The dangers of diagnosis-seeking to which he points are, as has already been observed, real ones, and are likely to be ever-present.

But when patients are suffering from severe physical and neurological symptoms the quest for a diagnosis is not irrational, as Steere comes dangerously close to implying; it
is entirely rational and should be recognised as such. Providing diagnoses, after all, is one of the things which medicine is for.

Because of the subtle and polysymptomatic nature of Lyme disease there is always the danger that it will be overdiagnosed. Indeed it seems quite likely that an over-enthusiastic dedication to the diagnosis of Lyme disease on the part of some specialist doctors will give rise to a significant number of misdiagnoses. This is because such specialists, like all physicians, tend to have an incomplete knowledge of the many subtle disease-states to which the human body is prone.

Such dangers, however, are only compounded when ‘orthodox’ physicians continually take refuge in the notion that we have a complete understanding a disease whose many complexities have so far eluded exact description.

An equally dangerous form of medical error takes place when doctors seek to resolve their uncertainty by offering not a diagnosis but a pseudo-diagnosis based on superstitious and traditional beliefs about the non-existent disease of hysteria and entirely mythical processes such as somatisation and conversion.

The case of the three psychiatrists who, faced with Sally Russo’s unexplained leg cramps, spasms, seizures and paralysis, gave serious consideration to making a diagnosis of conversion disorder, illustrates that the danger of such irrationality is a real one. Although these particular psychiatrists showed good judgment in rejecting the diagnosis, other psychiatrists in similar situations have not, and the consequences for their patients have sometimes been severe.

So far as Sally Russo herself is concerned it would be reassuring to report that after she had received her diagnosis her medical problems were over. Lyme disease, however, particularly when diagnosis has been delayed, is a difficult and sometimes intractable condition. Once Dr Sam Donta had made his diagnosis he immediately followed the ILADS protocols and put her on antibiotics. Russo began to feel better but at this stage she was still being treated for the dopamine disorder which her previous doctor had diagnosed, and still didn’t know which diagnosis was the correct one. She then tapered off the dopamine medication and began to feel that the antibiotics were helping.

However, that summer she hit a plateau where she wasn’t getting better or worse. When Donta put her on another antibiotic she had a spasm that lasted 12 hours that landed her back in the hospital for a week. Then, in November 2005, she went into a spasm that lasted for 22 hours. During another week-long stay at the hospital, her heart stopped for nearly a minute. At this point Russo decided to stop taking the medicine to let her body recover. ‘I had been on the medicine for 10 months and physically I was just too tired and I just wanted to give my body a break,’ she said.

It was only at this stage, that the diagnosis of Lyme disease was actually confirmed by positive Western blot tests. But when Russo decided to have a port put into her arm so that the antibiotic rocephin could be delivered faster, the complications increased. ‘Literally within 12 hours my body went out of control. I have never been so sick in all
my life … I went in [to hospital] to have that port put in and I was supposed to be home within four hours. I stayed for five weeks.‘

She eventually came home again. But when she recently asked her doctor when she would get better the answer was, ‘We don’t know. You’ll be better when you feel better and we can’t tell you when that’s going to be.’ While waiting for her condition to improve, and while still able to walk only with the help of crutches, Russo felt the need to share her story. She went to her local newspaper who published a long article about her case. The article ends with Russo’s own assessment of Lyme:

It is a brutal, brutal disease and I think people just don’t have any idea what it can do. I don’t even know what it would feel like to feel good again … If even one person reads this article and says, ‘I’m going to be more careful. I’m going to get checked because I got bit by a tick,’ I’ll be happy. If I could help one person, that would be unbelievable to me.92

The severe reactions which sometimes accompany long-term antibiotic treatments illustrate why such treatments should only be prescribed after a careful risk-benefit analysis has been conducted. Because the possible benefits of such treatment are attested to mainly by the clinical experience of individual physicians, and because few double-blind trials have yet been conducted, the controversy is not likely to be resolved soon. Until it is, many physicians will continue to maintain the view that the very severity of the symptoms Lyme can give rise to, and the potentially grave consequences of leaving the disease untreated, make it ethically irresponsible to withhold such treatment unconditionally.

What almost all physicians, on both sides of the Lyme divide, are agreed on, is that therapeutic success is best guaranteed by early diagnosis and treatment. No doctor, of course, can ever hope to avoid all misdiagnoses and nor should they be expected to. But what every doctor should recognise is that preserving diagnostic uncertainty when such uncertainty is demanded by the complexity or ambiguity of a patient’s symptoms can itself be essential to the making of a correct diagnosis. To attempt to resolve such uncertainty by applying the pseudo-diagnosis of hysteria or conversion, or by declaring symptoms to be ‘functional’, can leave patients with a life-time of disability ahead of them and can, indeed, be ultimately fatal.

[SECTION UNWRITTEN]
18 Conclusion

But it is not for a non-physician to pass final judgment on the validity of what purports to be a medical diagnosis. It would be more fitting to recall the verdict which was uttered by Eliot Slater in his 1965 paper:

Looking back over the long history of ‘hysteria’ we see that the null hypothesis has never been disproved. No evidence has yet been offered that the patients suffering from ‘hysteria’ are in medically significant terms anything more than a random selection. Attempts at rehabilitation of the syndrome, such as those by Carter and by Guze, lead to mutually irreconcilable formulations, each of them determined by their terms of reference. The only thing that hysterical patients can be shown to have in common is that they are all patients. The malady of the wandering womb began as a myth, and as a myth it yet survives. But, like all unwarranted beliefs which still attract credence, it is dangerous. The diagnosis of ‘hysteria’ is a disguise for ignorance and a fertile source of clinical error. It is, in fact, not only a delusion but also a snare.

The evidence of medical history, when critically reviewed, suggests that Eliot Slater was not ‘wrong’ as has recently been claimed. It suggests that his assessment of the hysteria diagnosis was both just and necessary.

Forty years on it is time that we heeded his words.
NOTES

2 same, p. 67
3 same, p. 75
4 same, p. 157
5 same, p. 206; the version of events here is that given in *The Spiral Staircase* (2004). In the original version of this memoir, *Beginning the World* (Macmillan, 1983) the description quoted here is applied to an earlier seizure which took place much more dramatically in a lecture theatre. It would appear that a number of seizures have been conflated into a single episode in the later version of the book. It should also be noted that Karen Armstrong was not in fact a failed academic, but a successful university teacher whose academic career was destroyed when her Oxford doctoral thesis was unexpectedly failed by an external examiner whose judgment was subsequently called into question.
6 same, p. 209
7 same, p. 74; *Beginning the World*, p. 124.
10 A somewhat more detailed account of Anna O’s illness is given in my *Freud* in the Great Philosophers Series, Weidenfeld 2003, the relevant sections of which will be found in ‘Freud, Charcot and hysteria’ on my website: http://www.richardwebster.net/freudandcharcot.html. A much more comprehensive account is contained in Chapter 4 of Why Freud Was Wrong: Sin Science and Psychoanalysis, Harper Collins, 1995, pp. 103-135).
11 *BMJ*, ‘Eclipse of Hysteria’ 29 May 1965, p. 1390
12 same, p.1399
14 *BMJ*. 1998 February 14; 316(7131): 0.
16 ‘Psychological Versus Biological Clinical Interpretation: A Patient With Prion Disease’, H. Brent Solvason, Ph.D., M.D., Brent Harris, M.D., Penelope Zeifert, Ph.D., Benjamin H. Flores, M.D., and Chris Hayward M.D. *Am J Psychiatry* 159:528-537, April 2002.
17 Slater, p. 1399
18 Bastian, Various forms of Hysterical or Functional Paralysis (1893), cited Slater, p. 1395
19 p. 1396
20 Bastian, quoted by Slater, p. 1395
28 See, for example the work of the French neurologist André Léri, Shell Shock: Comnootional and Emotional Aspects, University of London Press, 1919, passim.
29 Léri, Shell Shock, pp. 165-8. I have rendered the somewhat creaky published translation into normal English by changing one or two words, most significantly replacing the mistranslation of ‘ignoré’ (given as ‘ignored’ in the translation) by ‘unknown’ and rendering ‘sinistroses of war’ as ‘disabilities of war’.
33 The Psychology of the Special Senses, p. 98
34 The Psychology of the Special Senses, p. 74
35 p. 75
36 Ben Shephard, A War of Nerves: Soldiers and Psychiatrists in the Twentieth Century, Jonathan Cape, p. 79
41 C. David Marsden and Timothy J. Fowler (ed.), Clinical Neurology, Edward Arnold, 1989, p. 428. The section on hysteria from which I quote is part of the chapter entitled ‘Psychiatric Disorders’ which was written by Paul Bridges, Consultant Psychiatrist at Guy’s and the Brook Hospital.
46 Alan Richens, Letter to RW, December 1993

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48 Kaplan and Sadock, p. 1009.
49 Kaplan and Sadock, p. 375.
50 Lipowski, as above, p. 1359.
51 A striking, book-length example of this process at work is provided by Edward Shorter’s *From Paralysis to Fatigue: A History of Psychosomatic Medicine*. The main authority which Shorter cites in support of his own arguments is Lipowski. Yet whereas Lipowski promotes a complex and ‘weak’ form of the concept, which eschews psychoanalytic notions of conversion, Shorter invokes his authority in support of his own theories, according to which the unconscious mind freely converts the ‘stress of psychological problems into physical symptoms’. The relationship between Shorter’s ideas and Lipowski’s is discussed acutely by Theodore M. Brown in his review of Shorter in *Journal of the History of the Behavioral Sciences*, vol. XXIX, 1993, pp. 243–5.
56 As Goudsmith notes, this point is confirmed by Ian Robinson in his study of multiple sclerosis, in which he quotes another researcher to the effect that that ‘the discovery of the diagnosis gave back to many respondents their credibility and legitimised their strange behaviours which had previously been labelled as neurotic, hypochondria, malingering or drunk.’
57 Gregory Zilboorg, *A History of Medical Psychology*, Norton, 1941, p.XXX; see Simon Wessely, *Mass Hysteria: two syndromes*, Psychological Medicine, 1987, 17, pp. 109-10. Wessely quotes from the passage I cite here but manages to truncate the final sentence by omitting, without any ellipsis, the last clause: ‘for the individuals who form a part of these mass reactions need not be and are not always mentally sick’. As a result the quotation fits Wessely’s thesis rather better than it would have done had he quoted the full sentence.
59 Moss and McEvedy, p. 1295
60 Moss and McEvedy, p. 1299

Mysterious Illness at Girls’ School, British Medical Journal, 16 October, 1965, p. 950

P R Celsus, CFs and the Cerebellum, http://www-personal.umich.edu/~prcelsus/csf.html, 1997. I am indebted to ‘Celsus’ in many of the comments which follow.


The Medical Detectives, p. 347

The Medical Detectives, pp. 349-50

The Medical Detectives, p. 352

The Medical Detectives, p. 353

The Medical Detectives, p. 354

The Medical Detectives, p. 357

The Medical Detectives, pp. 359-60


The Medical Detectives, p. 357

See http://www.townonline.com/barnstable/localRegional/view.bg?articleid=497474&format=text

Laurie Higgins, 18 May 2006.


David France, NY Times, as above, The Medical Detectives, pp. 391


David France, NY Times, as above.


Fallon et al, as above

David France, NY Times, as above.