Hysteria
Medicine, psychiatry and misdiagnosis

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INTRODUCTION

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 blackouts, 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 very well that 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. ‘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 electroencephalograph 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!’

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 unconsciousness’ was an epileptic event. His conclusion has been confirmed by modern neurologists. Henri Gastaut, for example, describes olfactory seizures as consisting in ‘a sensation 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 psychol-
ology. It may well be significant that Lucy R had developed her olfactory hallucinations 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 phantomsia) occasioned by damage to one of the olfactory nerves.9

Characteristically, however, Freud did not allow for the many gaps in medical knowledge which then existed. Instead he made an a priori assumption that Lucy R was suffering 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 psychoanalytic 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
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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 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.
1 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 sum-
mer of 2000 in the same journal which had published Slater’s attack on the diagnosis 35 years earlier.13

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 ‘Hystera 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’.14

The rehabilitation of the diagnosis is by no means complete for it would seem that the term ‘hystera’ 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’.15 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.16

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 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 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.
2 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 not 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 reason 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
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.
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.\textsuperscript{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:

\begin{quote}
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’.
\end{quote}

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 psychiatrists who have consistently argued for a rehabilitation of the diagnosis of hysteria – while favouring euphemistic formulations of the diagnosis such as ‘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 2005 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’.²¹

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.
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.
18 Bastian, Various forms of Hysterical or Functional Paralysis (1893), cited Slater, p. 1395
19 p. 1396
21 Bastian, quoted by Slater, p. 1395