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I never discovered why I had been given such toxic treatment

By SARAH WALLS

The registrar glanced down the list of symptoms that I had brought and handed it back. There was, he said, “no rational explanation” for them. The list included “a feeling of inflamed nerves” in my spine, cracking joints and tendons, marked tingling, electric jolts and constant activity in my nerves. Having entered hospital for investigation of a single symptom, I was shocked that this violent eruption of nervous activity should be dismissed so summarily. “No rational explanation” implied that if something was wrong, it was with the patient, not the treatment.

Over the next decade, as I pieced together the cause of my injury, the registrar’s comment frequently resurfaced and reinforced my determination to get to the bottom of the matter. When the puzzle fitted together in a way that challenged orthodox treatment, I witnessed how the medical, scientific and regulatory establishments respond when a new understanding of a drug’s action and of human biology emerges.

At the time of the incident in the Sydney outpatients’ clinic, I had been working as a journalist in New Caledonia, covering the French South Pacific mainly for *The Sydney Morning Herald* and *The Age* in Melbourne. Reporting on the return to peace of this Pacific island territory, where conflict over independence had led to four years of civil insurrection, was fascinating and demanding. My reporting skills, language skills and personal qualities were all tested to an unprecedented degree.

On 4 May 1989, I was present at a customary ceremony on the island of Ouvea when, in a shattering renewal of political violence, New Caledonia lost its two Kanak

independence leaders, Jean-Marie Tjibaou and Yeiweine Yeiweine, to an assassin's bullets. As I turned to gather my things at the end of the ceremony, shots rang out like fire-crackers in the dim tropical night. Moments later, Tjibaou lay, a bullet in his skull, his head cradled in the lap of an old woman rocking with grief. The carnage claimed six victims: Tjibaou's bodyguard shot dead the disaffected Kanak who had attacked the leaders, and three people survived bullet wounds.

This traumatic event occurred when I was chronically stressed and had persistent cystitis. Several weeks later, I developed a spasm in my urethra. It was painless but uncomfortable, and as the months passed, I had increasing difficulty urinating. In June 1990 doctors in Noumea advised me to return to Australia for a magnetic resonance imaging scan to clarify the cause of this chronic urinary retention.

The few days that I spent in hospital in Sydney had life-shattering consequences. To my horror, the urologist I saw told me that he thought I had multiple sclerosis. MS is an incurable disease in which the fatty myelin that insulates nerves in the central nervous system becomes inflamed and breaks down, impairing the transmission of nerve impulses and often leaving the patient severely disabled.

The urologist referred me to a neurologist who initially thought spinal cord compression more likely. He began investigation with a myelogram, a spinal X-ray in which contrast medium is injected directly into the cerebrospinal fluid. When the myelogram proved negative, I was given nerve conduction tests. The neurologist then concluded that I probably had MS after all. On the day I left hospital, he prescribed a five-day course of high-dose corticosteroid drugs.

The shock of the diagnosis paled in comparison with the agony of the investigation and treatment. Severe headache, nausea, neck pain and ringing in my head followed the myelogram. These symptoms were soon compounded by the highly unusual sensations detailed in the list I took to the outpatients' clinic a week later. There the neurologist prescribed four weeks of injections of adrenocortico-trophic

hormone (ACTH), a pituitary hormone that triggers production of the body's own corticosteroid hormones.

Four hours after the first ACTH injection, the symptoms of nerve vibration and tingling throughout my body flared again. The symptoms had abated when I stopped the steroids, and their renewed intensity aroused my first suspicion that the drugs might be aggravating my condition. I drew up a timeline, correlating my sensations with the medical interventions, and realized that the worst of the symptoms did indeed coincide with the drug treatment.

The following day, I showed my GP the timeline, and told her that I thought I was having an adverse reaction. She thought the symptoms more likely to be due to the underlying condition, and advised me to continue treatment. I was too sick to offer further resistance. Under medical supervision, I completed the treatment but with catastrophic results. A violent, persistent electrical storm spread throughout my entire central nervous system. Huge electric shocks exploded in my brain, spinal cord and genital nerves. I had a racing heart, flashing on my peripheral vision and loud noise in my head. My spinal cord and muscles went into severe spasm. I bruised severely, couldn't sleep and didn't menstruate for nearly two months. In seven months, I suffered five cracked teeth.

Even my fingernails and toenails ceased to adhere properly to the nail bed.

The still, silent transparency of my nervous system, which I had taken for granted, was obliterated. I felt as if I had been plugged into the 240-volt power system, and left in constant torture. Yet the only risks I had been warned of were a headache from the drop in pressure from the lumbar puncture, and a moon face and fluid retention from the steroids and ACTH. Back in New Caledonia, my Noumea GP told me he thought I had arachnoiditis. This, it transpired, was a disease in which the

delicate arachnoid sheath covering the brain and spinal cord becomes inflamed, often as a result of medical intervention.

Unable to credit that such an outcome was acceptable, I was determined to act. In December 1990 I sought a second opinion, lodged a complaint with the Health Department, and saw a lawyer about medical negligence proceedings. The second neurologist was sympathetic but accepted the diagnosis of MS. He attributed the electrical symptoms to a reaction to the contrast medium, though

"Their persistence to date", he reported, "is not easy to explain".

I realized that if I wanted an adequate explanation, I would have to find it myself. In March 1990 illness and dwindling finances forced me to return permanently to Sydney. In constant agony, I could no longer work as a reporter. I was consumed with anger at the loss of my career and of any hope of a normal life, and shamed and humiliated at having been conned. Journalists are supposed to be streetwise enough to spot when they are not being told the truth. Yet I had accepted assurances that it was safe to inject a foreign product into my spine, and had continued treatment despite the desperate signals of injury generated by my own body.

I found temporary work near the university medical library and began investigating. For someone suffering constant, violent bodily tension and noise in the head, the library was a blessed haven of peace and quiet. I soon learned that myelograms were hardly "perfectly safe", as the first neurologist had claimed.

Both the old oil-based myelographic contrast media and the more recent water based media were neurotoxic and known to cause severe damage.

Myelograms had numerous side-effects whereas magnetic resonance imaging (MRI) was painless, non-invasive and diagnostically superior. An MRI scan could have shown either spinal cord compression or the abnormalities suggestive of MS with no risk of pain or injury. Moreover, a 1985 study showed that the most common cause of

female chronic urinary retention was not spinal cord compression or MS, but anxiety or depressive illness, i.e. stress.

The information on steroids was equally disturbing. Among the first facts I discovered was that corticosteroids are stress hormones. I was astonished that stress hormones, or their synthetic analogues, could be used to treat inflammatory conditions, which are frequently stress-related. In the case of MS, a disease often associated with stress, evidence for the benefits of steroids seemed unconvincing. There was no evidence of benefits from long-term treatment or for treating chronic symptoms, and by the early 1980s steroids and ACTH were used in MS only in short courses to treat acute episodes. By the end of the 1980s, ACTH had gone out of general use, replaced by short-term (4-7 days) high-dose steroids. The treatment prescribed for me—five days of high dose steroids followed by four weeks of ACTH injections—was quite unorthodox.

In September 1991 the violent activity in my head was confirmed by an electroencephalogram. The "markedly abnormal" EEG, showing "prominent left fronto-temporal high voltage slow activity", was given to a third neurologist, who asked to see me immediately and ordered MRI scans. My brain scan was normal and my cervical scan showed only subtle changes in the mid-cervical cord. The neurologist concluded that it was unlikely that I had MS but offered no explanation in his reports for the persistent electrical abnormality in my brain, spinal cord and genital nerves.

Not until 1994 did I find the first significant clues to the puzzle. By then I was on disability support and retraining as a yoga teacher. Unwilling to risk further drug treatment, I coped with the agony by relying on yoga, meditation and dream-based psychotherapy, and by doggedly searching for an explanation. At school I had had little interest in science. Now what I was reading was of acute personal relevance. As I sifted through the literature, I was struck by how little questioning there was of the orthodox view of steroids as anti-inflammatory agents. How could supposedly anti-

inflammatory agents have an inflammatory effect, as apparently had happened with me? How could it be beneficial to treat stress-related conditions with agents well known to mediate the stress response?

In mid-1994 I stumbled on an article by a research scientist who was clearly asking similar questions. E. Ronald de Kloet was head of the Division of Medical Pharmacology at the Leiden/Amsterdam Center for Drug Research in Holland. In an article on "Corticosteroids, stress and aging", he challenged the conventional view of steroid action, arguing that "the action exerted by corticosteroids may be trophic or damaging, depending on the physiological condition". A new view was needed: "the progress in molecular biology of steroid action has made a re-evaluation of mineralocorticoid- and glucocorticoid responsive systems imperative".

De Kloet and his colleague, Marian Joels, a neurophysiologist from Amsterdam University's Institute for Neurobiology, had pulled the rug from beneath the orthodox view of steroid action by showing that chronic elevation of corticosteroid levels reversed the effects seen during acute elevation. They termed these opposing effects "biphasic" because they occurred at two different phases of the steroid exposure.

What particularly caught my attention was that chronic steroid elevation caused substantial rises in intracellular calcium. Raised intracellular calcium, I knew, was a key feature of the inflammatory process, and Dr Kenneth Smith, a neurophysiologist at the University of London, had shown that it could cause demyelination. Calcium also played a key role in muscle contraction and in tissue hardening and scarring.

I realized that Joels and de Kloet's findings had a logical implication: if glucocorticoids caused an influx of calcium into the nerve cells, as they had shown, and if raised intracellular calcium caused demyelination, as Smith had shown, then excess steroid levels might cause demyelination. If acute steroid elevation was anti-inflammatory, as conventionally agreed, and if chronic elevation reversed the effects

seen during acute elevation, as was now plain, then logically chronic steroid elevation must be a potential cause of inflammation and hence of demyelination. If so, then large doses or prolonged treatment with steroids were unlikely to benefit people with MS. Such treatment could accelerate their progression into a wheelchair.

This issue seemed so important that I raised it with de Kloet and Smith. Dr Smith did not rule out the possibility. In January 1995 he said that while he believed that steroids generally had an anti-inflammatory effect, “this would not rule out a pro-inflammatory action in more exceptional circumstances”. De Kloet indicated that I was on the right track: “It shows once again,” he replied, “that steroids may control the myelination process.”

My research now became more focused, and the picture of steroid action that emerged cast light on the extreme physical and emotional distress that I was still experiencing. Glucocorticoid hormones, the principal class of corticosteroids, are produced in the adrenal glands and taken up by receptors in every tissue in the body, including the brain as US stress scientist, Bruce McEwen, had shown in 1968. Not only do these hormones regulate the stress and inflammatory responses, they also govern learning, memory, cognition, emotion, the ageing process and responses to toxicity. They do so by altering genes in the nucleus of every cell they enter, which is why their effects are so persistent.

I began to understand why I felt as if my very self had been invaded, as if overnight I had aged decades. Seeking refuge in the library, trying to concentrate while my brain roared, was a way of holding fast to my intellect, like someone grasping a log while being swept out to sea. Five years after the injury, I still could not get body to relax, no matter how much meditation or relaxation I did.

No wonder!

De Kloet's work was at the cutting edge of a field that straddled mind and body and cut across all medical disciplines. In 1985 he and his student, Johannes Reul, had shown that there were actually two types of receptor in the brain that bind naturally produced glucocorticoids: classical glucocorticoid receptors (GRs) and mineralocorticoid receptors (MRs). Then in a series of studies in the early 1990s, he and Joels had shown how the same hormone could produce diametrically opposed effects, depending on whether steroid exposure was acute or chronic and on the shifting ratio of MRs to GRs.

In early 1996, I found in one of their studies the answer that I had been looking for. The study showed that steroids control activity in the nerve cells: low physiological levels of steroid were associated with a healthy range of responses; chronic steroid elevation produced long lasting and damaging changes in excitability, due to calcium flooding the nerve cell when too many glucocorticoid receptors are activated.

At last, here was scientific research that could explain the violently abnormal electrical activity in my brain that had begun with the steroid treatment and been confirmed in every EEG done in the past five years, producing what one neurologist referred to as "mountains and valleys" rather than "the normal little fluctuations in the EEG tracing". So violent was this activity that my four-year-old niece once complained that my hand "crackled".

That steroids could disrupt the brain's electrical activity was not news; steroids and ACTH were both known to increase susceptibility to seizures. What was news was how the MR/GR cellular mechanism operated. I realized that the implication of Joels and de Kloet's findings was that the extent to which corticosteroids mediated an inflammatory (or damaging) rather than an anti-inflammatory effect was a direct result of the extent of GR activation and consequent calcium influx. Moderate GR activation invited in a little calcium and potassium to turn off the stress and

inflammatory responses; too much steroid, too much GR activation, opened the calcium floodgates.

I went through everything I could find on the Medline database about intracellular calcium and inflammation. Not only did calcium play a key role in inflammation, but there was ample evidence of abnormal calcium handling in various disorders known to be stress-related and thus involving increased steroid production. Why, I wondered, was there so little awareness that low steroid levels were associated with a healthy nervous and immune system, while elevated levels raised intracellular calcium and led to nerve damage?

In January 1995, after being advised that no clinician in Australia was likely to testify on my behalf, I approached Dr Charles Poser of Harvard University, a world authority on MS and chairman of the 1983 committee that had developed diagnostic criteria for the disease. Dr Poser replied that while it was “quite clear” that I did not have MS, it “would be extremely difficult to find any neurologist anywhere in the world willing to testify against” the neurologist concerned.

In 1996 I decided to summarize what I had found on myelogram risks and the pro-inflammatory effects of excess steroids, and send the summary to potential experts. I also sent it to health authorities, doctors and scientists, including Dr Poser, Dr Smith and Professor de Kloet. In May I was thrilled to receive a reply from de Kloet saying that he was coming to Melbourne for a conference the following month.

On a mild winter’s day, I arrived, nervous but excited, for my appointment with him in the hotel lobby. The casually dressed, smiling man who greeted me soon put me at ease. I still had little external sign of damage, apart from being slightly deaf and unable to run. But de Kloet understood the gravity of my injury, and was curious to know how I was coping. Over lunch, we talked about his research, the clinical use of

steroids, and how he might be able to help me with my case. Steroids, he said, should be given for no more than a few days, as the aftermath was greatly underestimated: long-term exposure to high glucocorticoid levels increased vulnerability to degenerative events.

I particularly wanted to know whether he thought I was right in arguing that excess steroid levels could be pro-inflammatory. After all, nothing I had found in the literature explicitly stated this. As in his articles, de Kloet was cautious. But before leaving Melbourne, he gave me a letter of support to give potential clinical experts, and confirmed my point-form summary of MR/GR effects, including the fact that chronically high glucocorticoid levels could “increase pain and inflammation”. I felt a rush of gratitude to the first person in six years willing to speak out on my behalf.

Persuading clinicians to do likewise was another matter. In July a senior Brisbane neurologist declined to act as expert witness, but sent me the names of four New Zealand doctors to approach. All declined to be expert witnesses. However, one described my summary as “most impressive”, and suggested that I contact Professor W.H. McDonald, the leading British authority on MS.

I decided first to address a critical gap in my summary by looking for evidence of biphasic effects in the immune system. What I found suggested that the pattern that Joels and de Kloet had demonstrated in the nervous system, of contrasting effects from acute and chronic steroid exposure, applied here too, and again depended on GR-mediated calcium influx. The ramifications seemed monumental: if chronic glucocorticoid elevation could be pro-inflammatory, then a dysregulated stress system might not just affect the severity of autoimmune disease but actually trigger disease.

In other words, imbalances in the body's own glucocorticoids might set off inflammatory conditions by altering the genes via calcium flows. Steroid treatment would then be a case of "short-term gain, long-term pain": any relief obtained might be at the cost of exacerbating the condition by disrupting hormone levels that are normally self-regulating.

I wrote a new chapter discussing interactions between the stress and immune systems, and in early 1997 sent off the revised summary to de Kloet for comment. Then another lightning bolt struck when I belatedly read one of the two partial theories of steroid action on which US scientists were continuing to rely.

The hypothesis on acute steroid effects published by US physiologist Allan Munck in 1984 provided the theoretical underpinning for the clinical use of steroids as anti-inflammatory agents. At the time, Munck saw steroids as essentially suppressive, rather like neuroendocrine policemen stopping the troops of the immune system from running amok and damaging the body. In 1986 Sapolsky, Krey and McEwen published their glucocorticoid cascade hypothesis, which I had already read, on chronic steroid effects. They conjectured that chronic steroid elevation triggered a cascade of damaging events, starting with receptor down-regulation and ending in atrophy of the hippocampus, an organ in the limbic system governing the stress response. Both hypotheses were formulated before it was technically possible to distinguish glucocorticoid effects on the two types of steroid receptor in the brain.

In 1991 de Kloet published his own theory, the corticosteroid receptor balance hypothesis. Echoing the old yin-yang idea of health as a dynamic balance between opposing forces in the body, he argued that the balance between mineralocorticoid and glucocorticoid receptors in the limbic system was critical for homeostatic control. Where limbic MRs and GRs were out of balance, the result could be either "enhanced or reduced responsiveness to excitatory stimuli". In other words, in de Kloet's view,

steroids were not always suppressive; they could also have a stimulatory effect. Changing the idiosyncratic MR/GR balance altered an individual's susceptibility to stress and stress-related disease. The relationship between the neuroendocrine and immune systems was clearly more like that of partners in a waltz: as long as they are in close communication, the waltz flows. Once balance and timing are lost, disease ensues.

On reading Munck's 1984 hypothesis, I suddenly realized the enormous significance of de Kloet's theory: it was the only one suggesting a cellular mechanism that accounted for the entire range of glucocorticoid effects, ranging from acute to chronic, physiological to pharmacological and deficiency to excess. De Kloet had presented his hypothesis as an explanation for steroid effects on ageing, but it seemed to me to offer dramatic potential for a better understanding of inflammation and autoimmune diseases. It explained how steroid levels could shift between states of steroid excess and deficiency, which is precisely what happens in autoimmune disease.

Most important, de Kloet's hypothesis had tectonic implications for drug treatment, for as he and Joels had reported in 1994, the factors known to alter the MR/GR balance were stressful and toxic insults, denervation and drug treatment. If drugs could alter this critical receptor balance, that implied that far greater caution needed to be exercised in prescribing them, for they might increase vulnerability to stress and stress-related disease. And since synthetic steroids bind only to GRs, then whenever they enter the brain, they must alter the MR/GR balance.

I was struck by how little discussion there was of de Kloet's hypothesis in the scientific literature. If these implications were plain to an injured patient, surely they must be plain to scientists in the field. If I was wrong, and de Kloet's hypothesis was wrong, one would expect critical analysis of the hypothesis explaining why it was

wrong. Instead, apart from the occasional peripheral reference showing that scientists knew it existed, there was no critical analysis. The hypothesis was simply ignored. De Kloet, in contrast, consistently acknowledged the hypotheses of his peers.

I wrote a new introduction discussing the three hypotheses, and with de Kloet's single-word verdict – "splendid" – sent off my summary to Professor McDonald, to the Australian Federal Minister for Health, the Research Advisory Board of the local MS Society, the Royal Australasian College of Physicians (RACP) and to doctors and scientists in Australia, New Zealand, the US, the UK and Europe. In all, I circulated some 40 copies, including one to US stress scientist Robert Sapolsky, whom de Kloet was confident would "respond in a constructive way".

Work done in isolation by an injured patient with no scientific qualifications could easily have been ignored. That did not happen. The RACP passed my summary on to the Australian Rheumatology Association, which in turn forwarded it to the Australian Association of Neurology (AAN). The AAN secretary, Professor John Willoughby, sent the RACP a three-page report on it.

"To deal with the issues at an appropriate depth," he said "[…] a committee of expert individuals would be required", though he did not think that appropriate as my report was "non-scientific". While critical of the "mixture of good and questionable biological science", Professor Willoughby accepted my central point that steroids could have pro-inflammatory effects: "It is quite possible that when properly understood," he commented, "these ideas will alter our ideas and use of steroids."

The head of the Research Advisory Board for the local MS Society, Professor John Pollard, confined his remarks to the issues raised by my treatment: "It is not the practice of Australian neurologists to prescribe long term corticosteroids for patients with MS. The vast majority of neurologists would follow the international practice

which is to give a three day course of intravenous methylprednisolone in a patient with a significant acute relapse."

Alas, Professor Willoughby, Professor Pollard and Professor McDonald all declined to be expert witnesses. I was devastated, consoled only by the systemic changes and the shift in the literature that followed circulation of my research summary. For the first time in nearly 20 years, the process by which MS was diagnosed was re-examined. The McDonald diagnostic criteria, published in April 2001, eliminated the "probable MS" category into which I had been put in 1990, and permitted drug treatment only once the MS diagnosis was confirmed.

From late 1997, journal articles appeared in which steroid therapy in MS and other chronic inflammatory conditions was fundamentally questioned, and the view of steroids as exclusively anti-inflammatory agents was tested and found wanting. The most significant change came in articles by the three American scientists who had published the earlier hypotheses, Allan Munck, Robert Sapolsky and Bruce McEwen.

In March 2000 Sapolsky and Munck published, jointly for the first time, a landmark review in which they overturned the conventional view of steroid action. Munck's 1984 hypothesis, they said, no longer reflected the current state of knowledge about glucocorticoids: far from being exclusively suppressive, glucocorticoids "actually mediate the 'backbone' of the generic stress response". They said that while the most thoroughly investigated areas of glucocorticoid action were the anti-inflammatory and immunosuppressive actions, glucocorticoids also have "permissive, stimulatory and preparative actions", investigation of which had been comparatively neglected.

In December 2002 Sapolsky's team published a major review of "Glucocorticoids and central nervous system inflammation", saying that the concept of glucocorticoids being universally immunosuppressive might be oversimplified. A review of the literature had shown that "under certain circumstances GCs might fail to

have anti-inflammatory effects and sometimes even enhance inflammation"—a result described as "quite unexpected". Bruce McEwen, too, commented on the "unexpected clinical ramifications" of the effects of acute and repeated stress in an article published in December 2000. Steroids, he said, were "now recognized as having biphasic effects on immune function".

A paradigm of steroid action that has been dogma for half a century is crumbling. De Kloet's MR/GR balance hypothesis, which best accounts for the biphasic effects, is still rarely cited, though his team's 1998 review on "Brain Corticosteroid Receptor Balance in Health and Disease" has attracted wider attention. By then de Kloet had embarked on a new challenge: identifying the steroid-controlled genes likely to determine the form of stress-related disease.

The picture now emerging of steroid action is extraordinarily complex. Not only do steroids exert opposing effects, depending on whether exposure is acute or chronic, but they may regulate the entire onset and termination of the stress and inflammatory responses. Scientists now recognize that steroids play a role both in turning on and turning off the acute inflammatory response. But they rarely acknowledge that chronic steroid exposure can be pro-inflammatory. This information is vital for patients suffering chronic inflammatory disorders, who are treated with the very hormones whose imbalance may trigger or exacerbate their condition.

Clinical understanding lags dangerously behind current scientific knowledge. Most clinicians continue to regard steroids as exclusively anti-inflammatory, immunosuppressant agents. Few patients are aware that they are being prescribed stress hormones that are potentially pro-inflammatory. Yet in 1998, when I went through ten years of reports to Australia's Adverse Drug Reactions Advisory Committee (ADRAC), the most common adverse reactions reported to steroids were inflammatory responses. A decade after diagnosis, it was clear that I did not have MS. I had suffered an "insult" to my central nervous system from investigation and

treatment. The dose of contrast medium used in my myelogram exceeded the maximum recommended; the initial dose of the corticosteroid prescribed, then the most potent corticosteroid on the market, exceeded the maximum recommended; the type of ACTH prescribed had gone out of general use in the 1970s; and the four-week dosage regimen had ceased to be orthodox practice by 1970.

The complaint I lodged with the Health Department was dismissed in 1993. Clearly, its substance was noted, for in 1994 the information on adverse reactions for both drugs was radically extended, and in 1995 the steroid prescribed was taken off the Australian market. In 1996 my adverse reaction was finally reported to ADRAC. Two months after my reaction was reported, the form of ACTH prescribed for me was taken off the Australian market. It was withdrawn from the UK market and placed on limited access in the US. In 2001, with no clinical expert witness, I lost my court case. The day the appeal period expired, the drug was returned to general distribution in the US.

My injury is no longer invisible. My legs are semi-paralyzed and I need a stick to walk. The invisible torture remains. Never again will I know bodily peace and silence. I wake and sleep imprisoned in the noisy vibration of nerves strung taut through my body like electrified barbed wire. To survive and stay sane, I had to learn to let go of anger, stop “stressing” and listen to my body. With my body in permanent physical stress, I could not tolerate psychological stress as well.

Gradually, I came to terms with loss of a normal life. It was some solace to an ex-journalist to find a rational explanation for the violent electrical symptoms that I had first reported at the outpatients’ clinic: they were classic symptoms of severe toxic exposure. What I never discovered was why I had been given such toxic treatment.

2 May 2006.

Sarah Walls

Sarah Walls Biography

Former Australian journalist Sarah Walls worked for 15 years in radio, television and newspapers, until she was severely injured by investigation and treatment with a myelogram and high doses of corticosteroids in 1990. At the time she was a correspondent for The Sydney Morning Herald and the Melbourne Age in New Caledonia, covering the French Pacific. In 1994 she retrained as a yoga teacher and spent the rest of the 1990s doing library research into her treatment, lobbying on patient safety issues and pursuing legal action. For the last decade, she has worked from home as a translator. Since the early 2000s she has gradually lost mobility and can no longer walk or stand unsupported. In 2006 she wrote this account of her investigation and treatment and of what she tried to do to ensure that no-one else would suffer similar injury.