

## **INQUEST IMPLICATIONS?**

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The General Medical Council's "duties of a doctor" (2001) state that doctors must make the care of the patient their first concern and they must not 'give or recommend to patients any investigation or treatment which (they) know is not in their best interests, nor withhold appropriate treatments'. This was acknowledged on 15<sup>th</sup> June 2006 by Dr Susan Benbow of The Royal College of Psychiatrists in the Daily Telegraph.

The GMC stipulations are clear enough, so why then are sufferers from ME/CFS excluded from such protection?

There can be few people in the UK ME community who have not by now heard the results of the inquest into the tragic death from ME/CFS of 32 year-old Sophia Mirza, the beloved daughter of Criona Wilson from Brighton. Although severely sick with medically diagnosed ME/CFS, Sophia was abused by the doctors charged with her care by being wrongly sectioned under the Mental Health Act. Increasingly in cases of ME/CFS, the law which states that a person may be sectioned only if they represent a danger to themselves and / or to others is being swept aside by some influential but misinformed doctors involved with ME/CFS.

Sophia's mother recorded:

"In July, the professionals returned - as promised by the psychiatrist. The police smashed down the door and Sophia was taken to a locked room within a locked ward of the local mental hospital. Despite the fact that she was bed-bound, she reported that she did not receive even basic nursing care, her temperature, pulse and blood pressure (which had been 80/60), were never taken. Sophia told me that her bed was never made, that she was never washed, her pressure areas were never attended to and her room and bathroom were not cleaned."

It is only a few days since the Inquest was completed. Hesitant as we are to intrude on the renewed grief of a mother for the preventable death of her much-loved daughter, we believe that the issues raised must be confronted, otherwise there will be more unnecessary deaths, and more grieving.

## **INQUEST FINDINGS**

Although Sophia died in distressing circumstances in November 2005, the inquest was not held until 13<sup>th</sup> June 2006.

The first autopsy found no cause of death. Two weeks later, more tests were carried out and again, no cause of death was found.

Whilst we ourselves were instrumental in securing the release of Sophia from the locked ward, it was entirely through the personal intervention of Simon Lawrence of the 25% ME

Group for the Severely Affected (of which Sophia was a member) that permission was sought for a further autopsy and -- unusually -- was granted by the Brighton Coroner.

This time, the examination of Sophia's spinal cord showed unequivocal inflammatory changes affecting the dorsal root ganglia, which are the gateways for all sensations going to the brain through the spinal cord. These inflammatory changes affected 75% of Sophia's spinal cord.

At the inquest, one of the pathologists stated: "ME describes inflammation of the spinal cord and muscles. My work supports the inflammation theory because there was inflammation in the basal root ganglia".

Dr O'Donovan (the neuropathologist who, along with Dr Abhijit Chaudhuri, had examined the spinal cord) stated that psychiatrists were baffled by Sophia's illness, but that "it lies more in the realms of neurology than psychiatry, in my opinion".

Both Dr O'Donovan and the local pathologist, Dr Rainey, said that "ME" was the old-fashioned term and that new terminology --- CFS---has come in, so that was the term that would be used.

Dr Rainey also gave evidence that Sophia had a "fatty liver" (see below).

In Sophia's case, the Coroner was specific: the medical cause of Sophia's death was recorded as 1a) acute anuric renal failure; 1b) CFS. The second cause was recorded as including dorsal root ganglionitis. Sophia died as a result of acute renal failure arising as a result of ME/CFS.

This is in keeping with the medical literature that shows end organ failure to be a common cause of death in ME/CFS.

### **INQUEST SPARKS DEBATE**

Almost immediately, certain people were quick to assert that the Coroner's findings could not be taken to mean that death was caused by ME/CFS, suggesting that it was dehydration resulting in renal failure, not ME/CFS, that was the real cause of death, as Sophia was unable to drink for several days prior to her death.

She received no medical intervention such as intravenous fluid replacement – the medical intervention she did receive was to be forcibly removed from her home and incarcerated in a locked psychiatric ward. In her mother's opinion, this contributed to her death.

In online discussion groups, people asked if the acute renal failure was precipitated by dehydration, and one (non-medical) internet respondent was adamant: "Yes dehydration was the reason for the renal failure", but this was not an accurate representation of the Coroner's verdict. The same respondent, a prominent support group leader, asserted: "There is no way that the conclusions of this inquest can be seen to say that people can die of ME/CFS".

## **No evidence of brain swelling**

These people who seem so anxious not to impute Sophia's death to ME/CFS seem not to be aware that in dehydration, people die not from renal failure, but because the brain swells, and this was not found in Sophia's case: what was found as one of the causes of death was acute anuric renal failure, ie. a very sudden onset of lack of output of urine arising as a result of ME/CFS. Sophia's mother had stated that Sophia's head and neck "swelled up like a football" and Dr Rainey was asked specifically by the Coroner (Miss Veronica Hamilton-Deeley, Chief Area Coroner) if there was any evidence of brain swelling; he replied that there was nothing abnormal to be seen on the standard examination of the brain.

## **IT WOULD SEEM THAT SEVERAL SPECIFIC ISSUES ARISE FROM THIS INQUEST**

### **1. "FATTY LIVER" IN ME/CFS**

Dr Rainey gave evidence that Sophia had a "fatty" liver. This is notable, because there are reports in the literature that enlargement of the spleen and liver are not unusual. Published evidence shows infiltration of the splenic sinuses by atypical lymphoid cells, with reduction in white pulp, suggesting a chronic inflammatory process (see: Coincidental Splenectomy in Chronic Fatigue Syndrome. BJ Miller et al: JCFS: 1998:4(1):37-42).

There are reports of hepatic involvement in ME going back to 1977, including the following:

"Physical findings may include hepatitis" (BMJ 21<sup>st</sup> May 1977:1350)

"Enlargement of the spleen and liver is also not unusual" (Rev Inf Dis 1991:13: (Suppl 1):S39-S44)

"Typically, patients with major depressive disorder have no specific signs or symptoms. In contrast, CFIDS patients have been reported to have a multiple findings, including hepatomegaly (5 –20%)

(Psychiatric Annals: 27:5/May 1997:365-371)

In their evaluation of symptom patterns in patients with (ME)CFS who were ill for longer than ten years, Friedberg et al found hepatitis in 13.6% (J Psychosom Res 2000:48:59-68)

Abou-Donia (see below) has published evidence to show that a combination of stress and chemicals results in trauma to the brain via a breaching of the blood brain barrier (BBB) and that stress can intensify the effects of some chemicals, making them very harmful to the brain, nervous system and liver, resulting in abnormal fatty deposits that diminish the ability of the liver to rid the body of toxic substances (this evidence was also presented at the Sydney ME/CFS Conference in December 2001).

## **Neurodegeneration**

There is also evidence that organophosphate exposure produces apoptotic neuronal death and involves oxidative stress with a resultant neurodegenerative disorder (see the seminal published papers of Mohamed Abou-Donia, Professor of Pharmacology, Cancer Biology and Neurobiology, Duke University Medical Centre, Durham, North Carolina).

## **2. PSYCHIATRIC ABUSE OF ME/CFS PATIENTS**

What happened to Sophia is not an unusual occurrence: it was in 1990 that Peter Wakeford wrote in the UK ME Association newsletter: “Psychiatrists want the disease firmly in their domain. They’re gathering even now like locusts to redefine it. I have phone calls from isolated desperate people who say: The psychiatrists are trying to put me away in an institution. They’re taking away my day care. Help me” (see Perspectives, Summer 1990; pp 25-27). That same year, during the World First Symposium on ME/CFS held at the University of Cambridge, England, Dr Byron Hyde said ME/CFS is “a major health and economic threat second only to that of AIDS”.

No-one listened, so Sophia was one of many who have paid the price.

### **CMO’s Guidelines ignored**

She died almost four years after publication of the UK Chief Medical Officer’s Working Group Report on “CFS/ME”, which in January 2002 recognised that patients often feel “**severely overlooked**” by services and experience “**isolation, lack of understanding, and particular barriers to accessing all forms of care**”.

Medical arrogance and ignorance has not abated: in 2004, a typical quote from one physician was documented: “CFS is a lay diagnosis. I will not legitimise an illness that is not backed up by fact. CFS is not a fact”. (see Co-Cure MED 6<sup>th</sup> July 2005).

### **ME/CFS patients forced onto gym machines**

In the UK, state-condoned cruelty towards those with ME remains rampant, even being reported to occur in the new Centres set up with Government funding of £8.5 million. “People with ME deem these Centres not only to be unhelpful, but to be working against their interests” (see letter dated 3<sup>rd</sup> June 2006 sent by Paul Davis of Research into ME [RiME] to Members of Parliament). In the Greater Manchester area, sufferers attending these Centres report being told during cognitive behavioural therapy (CBT) sessions -- the only intervention on offer at these Centres --- that they have a ‘fear of activity’ and have ‘motivation problems’. In London, one person describes how they were put on gym machines and finished up in bed for several months; in a letter to this person’s GP, psychiatrist Professor Peter White from St Bartholomew’s Hospital wrote that symptoms were a result of deconditioning and that fear and anxiety prevented them from further exercise and that psychological factors contributed to the illness (see the RiME Spring 2005 Newsletter).

### **“This may result in screams...it may feel punitive”**

In a letter dated 22<sup>nd</sup> November 2003, the mother of a young man severely affected by ME wrote:

“The consultant in charge wrote to Dr Wessely for advice. On my son’s hospital file is a document dated 07.03.01, a “Draft Action Plan Proposal following consultation with Trudie Chalder”. I find the action plan shocking, and I was particularly disturbed by the penultimate paragraph, which states:

“We expect (*her son’s name*) to protest, as well as the activity causing him a lot of pain. This may result in screams....it may feel punitive”.

“This plan has never been discussed with me. There were a number of painful incidents...he was found bleeding from the stomach (and) had surgery in September 2001. On 18<sup>th</sup> April 2001 I wrote to the consultant about the pain my son must experience in having a naso-gastric tube frequently inserted...it had been re-inserted 11 times in the previous 7 weeks. I have no record of receiving a reply.

“The action plan also accounts for the diagnosis of “elective mutism”. [*Many in the ME community will recall that this was what Simon Wessely wrote in a 1988 medico-legal report about Ean Proctor from the Isle of Man --- it seems that the extensive biomedical research findings that have emerged since then have had little impact on some psychiatrists and that their attitude has remained rigidly unchanged for the last 18 years*]. Community speech therapists have refused to work with him on the basis that he might “not be compliant”.

#### **“Pursue exercise to the point where he resists”**

“There is a record of a confidential meeting on 31<sup>st</sup> May 2001, which agreed to continue with the behaviour programme. It states that: “The Chronic Fatigue Service believe that this exercise programme is to pursue exercise to the point where he resists”. The service referred to above is the one at Kings College Hospital. I wrote to the consultant and complained that it was too much for my son. The response was to increase the programme further. I then discovered that in a referral letter, (the consultant) stated that my son was suffering from ‘pervasive refusal syndrome’. I complained to the Chief Executive of the hospital Trust. An investigation was promised but this never happened.

#### **“Dr Chalder’s position is extreme”**

“(My son) was not being treated with any respect. I believe that the action plan devised by Trudie Chalder was harmful and posed unacceptable risks. The approach of Dr Chalder and the Chronic Fatigue Service is diverging from Department of Health policies like the Expert Patient programme. It is not good practice to cause patients ‘a lot of pain’ (and) I question whether it is ethical, indeed it may be unlawful. Dr Chalder’s position is extreme and I hope the Department of Health will consider carefully whether it wishes the Chronic Fatigue Service, of which Dr Chalder is a member, to have any role in proposals for new services for patients with ME”.

#### **“Our patients suffer at the hands of a poorly informed medical establishment”**

That such acts approaching barbarity continue to occur is acknowledged by international experts: in her incoming IACFS Presidential Address, Professor Nancy Klimas, a world renowned immunologist from Miami, was blunt: **“Our patients are terribly ill, misunderstood, and suffer at the hands of a poorly informed medical establishment and society”** (see Co-Cure, 21<sup>st</sup> March 2005).

### **3. DEATHS IN ME/CFS**

An article in the current issue of the “New Scientist” (“First official death from chronic fatigue syndrome” by Rowan Hooper) claims that CFS has been given as an official cause of death apparently for the first time in the world, but Rowan Hooper is under a significant misapprehension. There are many recorded deaths from ME/CFS.

#### **Premature deaths in ME/CFS**

Even though the pathology precipitating death varies widely (*see below*), it is clear that there are premature deaths in patients with ME/CFS: it can readily be seen that very ill ME/CFS patients die if they are medically abused, or if their secondary complications of ME/CFS are medically neglected.

In 1992, Professor Hugh Fudenberg from South Carolina (a pioneer of clinical immunology and one of the most distinguished minds in the field, being awarded The Medal of the Institut Pasteur at the age of 32 and was also a Nobel prizewinner nominee) stated that there is “**a greater death rate than normals in the same age range**” (see: *The Clinical and Scientific Basis of Myalgic Encephalomyelitis Chronic Fatigue Syndrome*: ed. BM Hyde, published by The Nightingale Research Foundation, Ottawa, Canada, 1992: page 644).

There is a Memorial List of people who have died of ME/CFS on the National CFIDS Foundation website, (<http://www.ncf-net.org/memorial.htm>) and a scientific paper analysing the causes of death of people on this list is currently in press.

#### **ME death recorded, 1957**

It was in 1957 that Dr Andrew Lachlan Wallis reported the post-mortem histopathology on a female from Cumbria who had died of ME; the report can be found in Wallis’ Doctoral Thesis (held at the University of Edinburgh and essential reading for anyone with an interest in ME/CFS; see also “Vade Mecum” by E. Marshall and M. Williams; *Co-Cure ACT*: 29<sup>th</sup> June 2005, which contains a summary of the thesis). The histopathology report states:

“There are in the entire diencephalon, particularly around the third ventricle, numerous small haemorrhages, which extend into the adjacent parts of the mid-brain. Similar haemorrhages can be seen in the corpora mamillare. The haemorrhages are mostly around the small vessels but some are also to be seen in the free tissue. This is a significant finding”.

It was indeed a significant finding and remains so, given the long history of vasculopathy in ME/CFS that abounds in the medical literature (summarised in “Vade Mecum”).

#### **ME postmortem findings suppressed**

There have been countless recorded deaths from ME/CFS throughout the world: these include a UK consultant in the NHS who died from ME, whose next of kin was warned by the Official Solicitor that action would be taken against them if they divulged the post-mortem findings, to the extent that the next of kin was reduced to a state of chronic fear.

### **ME death, Leamington Spa, 1988**

There is the well-reported case of Joanna Butler, a young woman aged 24 from Leamington Spa, Warwickshire, who was severely affected by ME. She was nursed at home by her devoted parents and was bed-bound for the last two years of her life and required tube-feeding. Although she died of ME, her parents were suspected of having caused her death by administering too high a dose of a medically-prescribed morphine-related compound, and the local paper (Courier) reported that the Warwickshire County Coroner (Michael Coker) ordered a police investigation. This investigation cleared them of blame but they were hounded to such an extent that they were forced to move away from the area (see the press reports in The Observer, 19<sup>th</sup> March 1998: “Tragic death of young ME victim” and the reports in the local paper, including the Courier, which carried a report on the ‘many who die each year’ of ME).

### **Death of Alison Hunter, Australia, 1996**

Perhaps the best known ME death is that of 19-year old Alison Hunter in Australia, who died in 1996 from severe ME, suffering seizures, paralysis, gastrointestinal paresis, heart damage, massive ulceration to her throat, horrendous neurological problems and overwhelming infection, having courageously fought ME for ten years.

“Doctors projecting their own fears and inadequacies”

James Ibister, head of Haematology at Royal North Shore Hospital, Sydney, said:

“To be honest, I felt helpless towards the end. On many occasions I was extremely embarrassed about the way she was treated by the system. A lot of terrible things Alison went through were doctors projecting their own fears and inadequacies. How anyone could not think she had a major medical illness was beyond me”.

Alison, he said, suffered “terrible physical distress compounded by insults and inhumanity”.

In a lasting tribute to her brave daughter, Christine Hunter founded the internationally respected Alison Hunter Memorial Foundation for ME/CFS ([www.ahmf.org](http://www.ahmf.org)) which is renowned for the quality of its international conferences.

Christine Hunter was honoured for her outstanding work for those with ME/CFS in January 2006 when she was made a Member of the Order of Australia.

### **Death of Casey Fero, 2005**

There is also the recorded death from ME/CFS of 23 year-old Casey Fero in the US on 4<sup>th</sup> July 2005, where the forensic pathologist’s report of September 2005 revealed his shock at the state of Casey’s heart, which showed fibrosis indicating a long-term infection.

### Countess of Mar presentation to CMO, 1998

As long ago as March 1998, a deputation consisting of the Countess of Mar, Dr Betty Dowsett (Honorary Consultant Microbiologist with a professional lifetime's experience of authentic ME) and Mrs Doris Jones MSc was granted an 80 minute private interview with the then Chief Medical Officer (Sir Kenneth Calman) at which a 74 page document was presented. It was entitled "The Organic Basis of ME/CFS" and included five sections, one of which documented illustrative case histories of deaths from ME/CFS.

Those case histories provided details of 63 people with ME/CFS known to have died, including 20 cases of suicide (age-range 14-50). Reasons given included unbearable pain and inability to cope with disability. Professions included medical doctor, nurse and teacher, as well as mother.

There were 43 cases of non-suicide death (age-range 19-70), which included 10 cases of ME/CFS complications, including progressive ME/CFS (a 19 year old female), very high levels of HHV-6, mitral valve prolapse, and EBV infection.

These included seven cases of cardiac failure, including cardiac arrhythmias and cryptogenic myocardial fibrosis, as well as heart attacks.

There were four cases of those with ME/CFS developing cancers, including brain tumour.

There were nine other cases, which included ulceric gastritis, atypical pneumonia, toxoplasmosis and bacterial infection of the brain, seizure and haematomas.

There were cases of pancreatic cancer, a further case of cryptogenic cardiac failure, plus endomyocardial (Leo Eslers) fibrosis.

There were cases of hepatic and renal failure, and of intravascular coagulation (that particular patient had only 2 months earlier been 'treated' at a London Teaching Hospital and told she suffered from severe depression).

Other studies showed additional cases where ME/CFS patients died from cardiac failure.

### Male / female causes of ME/CFS death

The document detailed general observations on deaths; information provided by Dr Dowsett confirmed that in ME/CFS, males die predominantly from cardiac failure and females die predominantly from neurological complications, sometimes manifesting as tumours, and both sexes may die from pancreatitis.

Professions included a photographer; a computer systems analyst; a radiologist; an air-line pilot and nurses.

### US Neurological Findings in ME deaths, 1993

The presentation to the CMO also provided a copy of Dr Paul Cheney's Testimony before the US FDA Scientific Advisory Committee on 18<sup>th</sup> February 1993, in which he testified that "we have lost five patients in the last six months". Cheney continued: "The most difficult thing to treat is the severe pain. The most alarming is the neurologic elements of this disease. Half have abnormal MRI scans. 80% have abnormal SPECT scans. Most have abnormal neurological examinations. The most severe cases have neurological findings which are striking".

Details of this presentation to the CMO were made available to the subsequent CMO's Working Group on "CFS/ME" during its three-year existence, but were ignored.

Let there be no misunderstanding about deaths 'as a result of ME/CFS': they occur far more commonly than many would care to acknowledge.

#### **4. Inflammation in ME/CFS**

Clearly, Rowan Hooper did not do the requisite research regarding previous deaths from ME/CFS before publishing his article.

His "New Scientist" article does, however, note that Dr Jonathan Kerr from St George's, University of London, (author of important gene research studies in ME/CFS) says he is not surprised that inflammation was found in Sophia's spinal cord, as it is known to be associated with ME. Kerr said that the known over-activation of the immune system may underlie the inflammation of the neurological tissue, but that "people have been reluctant to subscribe to the biological side because of the power of the psychiatric lobby".

It is not the case that inflammation in ME/CFS cannot be diagnosed: if people who are thought to have the disorder are permitted to be correctly and appropriately investigated, modern medical imaging techniques can detail subtle changes in tissues, including inflammation.

#### **Long research history of inflammation and CNS abnormalities in ME/CFS**

Mindful of the recent paper by Dr Mark Demitrack (for a summary of which see "A gleam of light at last?" by E. Marshall and M. Williams: Co-Cure ACT:RES: 13<sup>th</sup> June 2006), it is salutary to recall that it was in September 1988, in conjunction with the University of Pittsburgh, that the US NAIAD held a large research workshop called "Consideration of the Design Studies of Chronic Fatigue Syndrome", at which there were participants from the CDC and from the NIH.

It is 17 years since one of the presenters (Dr Sandra Dougherty) reported MRI scans on patients demonstrated abnormalities consistent with demyelination and cerebral oedema in 57% of ME/CFS patients studied.

In 1995, Dr Charles Shepherd, medical adviser to the UK ME Association, wrote a letter to the BMJ in which he said:

" Although the precise pathoaetiology of myalgic encephalomyelitis remains the subject of debate, Shonagh Scott and colleagues are incorrect in asserting that 'no evidence exists' of encephalitis. Buchwald et al carried out a large cohort study in which neurological

symptoms, results of magnetic resonance imaging, and lymphocyte phenotyping suggested that the patients were experiencing ‘a chronic, immunologically mediated inflammatory process of the central nervous system (ref: *Ann Intern Med*: 1992:116:103-113). More recently, Schwartz et al, who used single photon emission computed tomography, described abnormalities that were consistent with the hypothesis that ‘a chronic viral encephalitis’ may be present (ref: *AJR*: 1994:162:943-951). Furthermore, in the only postmortem study to have been published, the polymerase chain reaction showed enteroviral sequences in samples from the hypothalamus and brain stem (ref: *Ann Intern Med*: 1994:120:972-3), indicating that viral persistence within selective parts of the central nervous system may also play a part” (*BMJ*:1995:310:1330).

Despite the intransigent refusal of the Medical Research Council and members of the “Wessely School” to acknowledge it, there is significant documented evidence of inflammation in ME.

It was in 2001 that a booklet called “[What is ME? What is CFS? Information for Clinicians and Lawyers](#)” by Professor Malcolm Hooper et al pointed out the following:

#### **“Evidence of abnormalities in ME**

“Despite beliefs and assertions to the contrary, in ME there is evidence of inflammation of the central nervous system (CNS); that is what helps to differentiate ME from other forms of CFS. There are many references in the medical literature to inflammation of the CNS in ME and in ICD-CFS (37),(38),(39),(40),(41),(42) but such CNS inflammation is not found in all variants of CFS. It is incorrect to deny the existence of CNS inflammation in ME / ICD-CFS. In some cases of ME, as in multiple sclerosis, there is evidence of oligoclonal bands in the cerebrospinal fluid. (43),(44)

“It is accepted by the most experienced ME clinicians that some degree of encephalitis has occurred both in patients with ME and in those with post-polio syndrome: the areas chiefly affected include the upper spinal motor and sensory nerve roots and the spinal nerve networks traversing the adjacent brain stem (which is always damaged). (45) In nearly every patient there are signs of disease of the central nervous system. (46) Recent research continues to support neurological involvement. (47),(48),(49),(50),(51),(52),(53)

References:

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(41) Encephalomyelitis resembling benign myalgic encephalomyelitis. SGB Innes *Lancet* 1970: 969 971

(42) Prevalence in the cerebro spinal fluid of the following infectious agents in a cohort of 12 CFS subjects: Human Herpes Virus 6 & 8; Chlamydia Species; Mycoplasma Species, EBV; CMV and Coxsackie B Virus. Susan Levine *JCFS* 2001: 9: 91-2:41-51

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### **THE RELENTLESS GRIP OF THE PSYCHIATRISTS AND THEIR MODEL OF ME/CFS**

To deny the existence of inflammation in ME/CFS is to deny reality, for which some UK psychiatrists (and those members of the medical profession who support their ill-founded notions without bothering to consider the actual evidence) are notorious.

The only way forward is biomedical research, but it seems that in the UK, science and humanity count for nothing when dealing with those blighted by the devastation of ME/CFS.

This was concisely exemplified by Professor Peter White's remarks to Dr Vance Spence at the third Oral Evidence Session of the Gibson Parliamentary Inquiry into ME/CFS on 7<sup>th</sup> June 2006, which were words to the effect that:

“If WE hadn't got the money, do you really think that the MRC would have given any money to YOU?”.

It seems inevitable that there will be many more cases like that of Sophia Mirza.