

Vade MEcum

(a “vade mecum” is a small reference guide containing information that is frequently consulted)

Eileen Marshall Margaret Williams 28th June 2005

Two of the biggest problems currently besetting those with Myalgic Encephalomyelitis (ME) are (i) how to ensure that a physician accurately records the diverse and fluctuating symptomatology without dismissing such symptomatology as somatoform disorder and (ii) how to ensure that s/he understands that ME is not identical to “CFS/ME” as portrayed by psychiatrists of the “Wessely School”, whose papers purporting to address ME (under the umbrella of “CFS”) currently flood the literature but which bear little if any relationship to authentic ME.

For the benefit of the uninitiated, it may be worth briefly considering the historical facts. ME has been described in the medical literature for about 70 years, with first reports being dated to 1934, but it is now well recognised that the history goes back into the 19th century (see: SPECT Brain Imaging in Chronic Fatigue Syndrome. J Patterson et al. EOS – J Immunol Immunopharmacol 1995:vol 15: no.1-2:53-58). The disorder used to be called atypical polio until 1956 when it was first named ME (Lancet: Leading Article: May 26 1956:789-790). In 1959, Acheson (later to become Sir Donald Acheson, UK Chief Medical Officer) published his extensive review. It is now known that it was Acheson who wrote the Lancet Leading Article in 1956, in which he noted that in nearly every patient there were symptoms or signs of disease of the central nervous system. ME was first classified as a neurological disorder by the WHO in 1969 and was accepted as a specific disease entity by The Royal Society of Medicine in 1978. Things started to go badly wrong when in the 1970s certain psychiatrists became involved, notably McEvedy and Beard, who in a paper with no scientific merit whatever, dismissed ME as mass hysteria (see: BMJ 1970:1:7-11). The authors never examined a single patient: they simply used carefully selected old case notes as a vehicle for a PhD study. In 1988, following a spectacular increase in cases in the US, the term “chronic fatigue syndrome” (known as “CFS”) was introduced and doctors were encouraged to lump together all cases of “chronic fatigue” of whatever origin and to regard them as one syndrome; to facilitate this, all physical signs (including those that had been such classic features of ME) were expressly excluded from the new case definitions of 1991 (Oxford) and 1994 (CDC). (For references see: What is ME? What is CFS? by Professor M Hooper et al available on line at

http://www.meactionuk.org.uk/What_Is_ME_What_Is_CFS.htm).

Although they claim otherwise, “Wessely School” psychiatrists who advise Government and the medical insurance industry are not talking about authentic ME as listed in the WHO International Classification of Diseases (also listed as CFS, which is why it is sometimes referred to as ME/ICD-CFS) and as described by the late Dr Melvin Ramsay, but about chronic, medically unexplained tiredness that they unhelpfully refer to as “CFS/ME” and attribute to “aberrant illness belief”.

As Hyde noted in 1992: “This failure to return to the literature haunts the very basis of their definitions” (The Clinical and Scientific Basis of ME/CFS. ed: BM Hyde; The Nightingale Press, Ottawa, Canada 1992), because “Wessely School” psychiatrists glibly claim that the features documented in the medical literature about early outbreaks of ME have altered and are no longer seen. This is untrue: what seems to be true is that the psychiatric lobby fails to look for such features or to include people with such symptomatology in their studies. The enormous correspondence received by Professor Hooper and his team in response to their numerous papers in the public domain demonstrates this beyond doubt: people have written with profound relief and gratitude, confirming that they suffer from signs and symptoms documented by Hooper et al but have never seen such signs and symptoms mentioned by the psychiatrists in any of their copious literature and that such symptoms are not recognised by GPs as being part of the ME/ICD-CFS picture.

The following notes may help to highlight the features and symptomatology of ME/ICD-CFS and may hopefully help sufferers challenge the prevalent psychiatric dominance.

One of the most useful and important descriptions of ME is that of Dr Andrew Wallis as contained in his doctoral thesis (An Investigation into an Unusual Disease seen in Epidemic and Sporadic Form in a General Practice in Cumberland in 1955 and subsequent years. Andrew Lachlan Wallis. Doctoral Thesis, University of Edinburgh, 1957), from which some of the following notes are taken. [Please note that prior permission was sought and obtained from the University of Edinburgh before publishing these notes].

Although the Wallis paper clearly deals with an infectious disease, in relation to the current and significant increase in the number of cases it is essential to be aware that Professors Vojdani and Lapp from the US have demonstrated that **ME/ICD-CFS may be either virally or chemically induced** (see: Interferon-induced proteins are elevated in blood samples of patients with chemically or virally induced chronic fatigue syndrome. Vojdani A, Lapp CW. Immunopharmacol Immunotoxicol 1999;21:(2):175-202) and that the multi-system symptomatology is consistent.

SUMMARY OF THE WALLIS THESIS

An infectious disease occurred in epidemic and sporadic form in Cumberland in 1955 and subsequent years. Both sexes and all age groups were affected, and the disease affected over 200 people. There were changes in the blood picture in 30% of cases. There was objective evidence of CNS involvement in 20% of cases, with subjective neurological phenomena in over 60% of cases. Characteristic features were the presence of muscular pain and a protracted outcome.

The disease was the cause of much disability and loss of working time. It was characterised by acute myalgia, disturbance of the reticulo-endothelial system and by CNS involvement, and also by psychological sequelae. Relapses were common: those with more severe involvement of the RES and CNS had more prolonged illness with greater liability of relapses. Some patients suffered a mild illness initially, only to be followed by more severe relapse at a later stage. Recurrence of symptoms became a well-marked feature. The causal agent was pan-trophic, with various

systems showing clinical evidence of disease. Sequelae included debility, depression and emotional lability. From the early stages of the epidemic, Wallis recognised that the disease was not conforming to any disease with which he was familiar; he was particularly struck by the stoicism of those affected.

Onset was either abrupt or insidious, with the latter being more common in adults. The more severe cases occurred in adults with insidious onset.

In the more severe cases a definite pattern emerged. The clinical picture varied from mild to one of considerable severity and duration. In a minority, the onset was precipitate diarrhoea with associated nausea: in this group, upper respiratory symptomatology was minimal. The costal margins were the site of considerable pain. Involvement of the liver and spleen was a typical finding: both were tender to palpation; in some cases there was actual enlargement. Morphological abnormalities were found in lymphocytes associated with an eosinophilia in 30% of cases.

The initial stage was followed by lethargy and weariness increasing rapidly in degree, with loss of muscular power, especially in lower limbs. Patients longed for bed, but it needed a considerable effort of will to get there: if stairs had to be climbed, the effort of doing so left them exhausted, with aching legs which felt leaden. Patients fell into a deep sleep at first, then became restless, with vivid, disturbing dreams and inversion of sleep rhythm. Headache (usually frontal, but not infrequently temporal) was severe, as was aching pain in back of neck, which was *always* present and sometimes severe. Myalgia was prominent, commonly affecting the para-spinal muscles, especially in the lumbar and shoulder girdle, aggravated by standing. The affected muscles were acutely tender. Photophobia was present, with blepharitis in some cases. Blurred vision was present, and it required a conscious effort to bring objects into focus; it was not due to refraction errors. Sudden eye movements caused acute stabs of pain. Diplopia was present in all severe cases. During the first week, vertigo was usually present, associated with lateral nystagmus (which was intermittent). Hyperaesthesia was present in the skin over affected muscles. Pins and needles commonly affected the extremities. Other abnormal skin sensations included prickling (accompanied by observable slow waves of contraction along a bunch of muscle fibres). Attacks of sweating were normal, with drenching sweats at night. There were abnormalities of taste and smell. A labile emotional state and outbursts of weeping proved remarkably persistent. Persistence in an activity was found difficult to maintain.

Wallis meticulously documented his findings in separate sections, for example, noting distinctions in symptomatology between those with acute onset and those with insidious onset. Whilst this inevitably leads to some duplication in these notes, some sections have been maintained for ease of reference.

Physical examination revealed: tachycardia; low-grade fever or sub-normal temperature; clusters of petechiae on the palate; mouth ulcers; abdominal distension; tender nodules in recti muscles; tenderness without guarding was always present on palpation at the subcostal margins and frequently over the RIF; liver and spleen were very tender in the more severe cases, being enlarged in some cases; skin was affected by rashes (both maculo-papular and urticarial); eyes were puffy and upper lid tended to droop. The most obvious feature O/E was the degree of spontaneous muscular

pain: palpation of the affected muscles frequently revealed the presence of palpable and tender nodules in muscles, with a gritty sensation to the touch. Headache, myalgia, muscle cramps and waves of dizziness persisted, as did parasthesiae. “Easy fatiguability” was the rule.

In cases with insidious onset, Wallis particularly noted the signs and symptoms as being: excessive tiredness; sweating; cold hands and feet; dizziness and unsteadiness; headache; neuralgic pain; insomnia; cognitive problems; aching in back and legs; pins and needles in hands and feet; partial aphonia; blurred vision (this was a constant complaint); frightened by loss of power in legs (being almost unable to bring one leg past the other); joint pains (knee, ankle, elbow, wrist and MCP joints); skin over fingers was cyanosed and felt cold; pain in calf muscles; myalgic pain in back, neck and shoulders; hyperaesthesia of skin (this was commonly noted).

Neurological signs and symptoms in cases of insidious onset (and in later stages of abrupt onset) were: parasthesiae; hyperaesthesiae; impairment of taste and smell; vertigo; blurred vision; loss of concentration; poor recent memory; impairment of co-ordination and unsteadiness; general weakness (a frequent complaint: walking, lifting and carrying were all limited); inversion of sleep rhythm; pupils frequently sluggish in reaction to light and accommodation; ptosis of eyelid (not in all cases); hyperacusis (commonly found); nystagmus; neuralgic pain was commonly complained of; alteration in speech; nominal aphasia; ataxia; Romberg commonly positive; observable tremor; impaired judgment of distance; a variable reflex state was found during the course of the illness, being brisk in a large number of cases, with knee and ankle clonus.

Sensory disturbance complained of by most cases included: tingling / pins and needles; numbness / heaviness of limb; feeling of pressure on scalp as if wearing a tight skull cap; feeling of drops of water trickling on affected area; feeling of insects crawling over skin; abnormal perception of taste and smell.

Objective sensory disturbances noted: hyperaesthesiae; impairment of position sense; impairment of joint sense; impairment of vibration sense; some cases showed diminished sensation to touch and pin prick.

Autonomic disturbance: abnormal coldness in extremities; sweating / drenching nocturnal sweats; sluggish pupillary reactions.

Complications: myocarditis (heart rate was accelerated during the illness); dyspnoea on slightest exertion; orchitis; DVT.

Treatment: palliative only. General management: bed rest was the most important factor; early mobilisation resulted in relapses. Convalescence was slow in the more severe cases.

Prognosis: persistence of symptoms was a marked feature. In a few cases, there was permanent neurological involvement.

Notable features were the similarity of symptoms and clinical history with other outbreaks in Akureyri, Iceland (1948); Adelaide, Australia (1949); New York State (1950); Middlesex Hospital, London (1952); Coventry, England (1953); Durban, South Africa (1955); Royal Free Hospital, London (1955).

Post-mortem histopathology

There were no fatalities during the period January – August 1955, but one patient who had exhibited the illness died the following year. Death was found to be due to numerous small haemorrhages in the mid-brain. Post-mortem histopathology report from this (female) case stated:

“There are in the entire diencephalon, particularly round 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.”

One paragraph in Wallis’ thesis particularly stands out: “Consultant medical opinion rather clearly showed disinterest, an implication being that a hypothetical mountain was being erected on an imagined molehill, and that the cases could readily be explained on conventional grounds by a competent person. The psychogenic sequelae were considered to be affective disorder”. Wallis then adds: “I understand, however, since obscure illnesses have become respectable following the outbreak at the Royal Free Hospital, followed by reports and leading articles in the medical press, the consultant in question now makes such a diagnosis himself”.

It seems that medical arrogance was prevalent then as now.

Comparison of the Wallis findings with other published findings

The post-mortem histopathology report in Wallis’ thesis was particularly interesting, given the long history of vascular abnormalities and impaired blood flow in ME/ICD-CFS, for example, references in the literature to vasculopathy include the following:

“lymphocytes in the cerebrospinal fluid congregate in the perivascular (Virchow Robin) spaces of the brain...these findings do suggest that the disease may involve the perivascular spaces of the brain”

“dilatation of the Virchow Robin spaces could also suggest intracranial arterial or periarterial pathology, in particular, one would expect to find a congregation of lymphocytes in the perivascular spaces around the central nervous system arteries...(Wallis) revealed an artefact that is in an anatomical position similar to that suggested by MRI studies”

re: the Los Angeles 1934 epidemic: “The blood vessels throughout the nervous system were distended with red blood cells...the most characteristic change was infiltration of the blood vessel walls” (see: The present consensus on MRI in

ME/CFS. Royce J Biddle. In: The Clinical and Scientific Basis of ME/CFS. ed: BM Hyde; The Nightingale Press, Ottawa, Canada 1992).

From the earliest reports of ME, autonomic vasomotor instability has been noted (eg. AM Ramsay, Update: September 1976:539-541), as has impaired bloodflow in the microcirculation (eg. LO Simpson, NZMJ:1984:698-699).

Vasculitis of the liver has been seen on biopsy and LFTs also sometimes signify a vasculitis of the liver (see: ME: The Epidemiological and Clinical Observations of a Rural Practitioner. John Richardson. In: The Clinical and Scientific Basis of ME/CFS. ed: BM Hyde; The Nightingale Press, Ottawa, Canada 1992).

Vasculitic skin lesions are documented in ME (see: Myalgic Encephalomyelitis – A Persistent Enteroviral Infection? EG Dowsett et al. In: The Clinical and Scientific Basis of ME/CFS. ed: BM Hyde; The Nightingale Press, Ottawa, Canada 1992).

Vascular headaches are well-documented in ME from the earliest reports (eg. Leon-Sotomayer) and are recorded as “long-term residuals” in 100% of ME patients (see: Cardiac and Cardiovascular aspects of ME/CFS. BM Hyde, A Jain. In: The Clinical and Scientific Basis of ME/CFS. ed: BM Hyde; The Nightingale Press, Ottawa, Canada 1992).

Temporal lobe perfusion defects may indicate primary inflammatory changes or secondary vascular impairment in (ME)CFS patients: “the diminished uptake of this oxine can be interpreted as due to a) diminished rCBF b) inflammatory regional changes present in 71% of patients studied” (see: Study of Cerebral Perfusion by NeuroSPECT in Patients with Chronic Fatigue Syndrome. Ismael Mena. In: The Clinical and Scientific Basis of ME/CFS. ed: BM Hyde; The Nightingale Press, Ottawa, Canada 1992).

In 1994, Schwartz et al documented the following: “As with any chronic inflammatory condition affecting the central nervous system, the T2-bright foci on MR in (ME)CFS may represent perivascular cellular infiltrate and / or reactive demyelination of the surrounding white matter....these abnormalities may reflect the result of a vasculopathy specifically involving the small vessels of the cerebral white matter; indeed, the distribution of lesions on MR in (ME)CFS is similar to that observed in occlusive arteriolar disease of any origin. The cortical defects measured with SPECT may result from decreased flow through cortical arterioles owing to vasculitis. **Specifically, on the basis of our observations, the white matter abnormalities seen on MR images may represent chronic demyelination, which appears to be irreversible**” (see: Detection of Intracranial Abnormalities in Patients with Chronic Fatigue Syndrome: comparison of MR imaging and SPECT. Schwartz RB, Komaroff AL et al. Am J Roentgenol 1994:162:935-941).

Despite the dismissal by “Wessely School” psychiatrists of any possibility of demyelination in ME/ICD-CFS, there is persistent reference in the literature to its presence in ME/ICD-CFS, for example, at the Third Annual Symposium on Chronic Fatigue Syndrome and the Brain in Los Angeles in 1992, Dr Thomas McNamara noted that UBOs in ME/ICD-CFS represented focal oedema in the perivascular Virchow Robin spaces and they might also represent areas of demyelination.

[For other documented references to demyelination and cerebral oedema in ME/ICD-CFS, see Appendix below].

In 1999, Watson et al reported that perfusion defects seen in thallium cardiac scans of (ME)CFS patients were unlikely to be explained by occlusive coronary vessel disease and that in their studies (as well as in other independent studies), cardiac thallium SPECT scans were shown to be abnormal in the majority of patients with (ME)CFS and perfusion defects were common. Cardiac SPECT scanning is a nuclear medicine technique used to identify regions of under-perfused myocardial tissue (see: A Possible Cell Membrane Defect in Chronic Fatigue Syndrome and Syndrome X. Walter S Watson et al. In: Kaski JC (Ed). Chest pain with normal coronary angiogram: pathogenesis, diagnosis and treatment. Kluwer Academic Publishers, London 1999: chapter 13:143-149).

To this day, symptoms such as those documented by Wallis are still to be found in authentic ME/ICD-CFS patients and no amount of dismissal or denial will make them disappear.

Given that there is such a massive literature dating back to at least 1934 documenting the multi-system organic disruption in ME/ICD-CFS, how can “Wessely School” psychiatrists be allowed to retain credibility when they persistently ignore this evidence-base? How can they credibly claim to be “world experts” when they continue to disregard such long-standing factual evidence? How can they be permitted to continue to advise that only the most basic screening be performed on those with ME/ICD-CFS? Does this not amount to criminal negligence?

Spin-doctoring is no substitute for the real thing: what does it tell us about the integrity of Government -- the same Government that was so keen to promote the notion of “the patient-centred approach”; “shared decision-making”, not forgetting the much-publicised “Expert Patient Programme” in which patients living with chronic medical conditions were acknowledged to be well-informed about their own condition? (see: The Expert Patient: A New Approach to Chronic Disease Management for the 21st Century. Department of Health website: <http://www.doh.gov.uk>).

APPENDIX of some other references to demyelination and cerebral oedema in ME/ICD-CFS

1988

Anaesthetics and ME/CFS. Meeting Place 30, (Journal of the Australian and New Zealand ME Society (ANZMES) 1988:29

Research Workshop, National Institute of Allergy and Infectious Diseases. S. Daugherty, 15th September 1988. In co-operation with the University of Pittsburgh, the NIAID held a large research workshop entitled Consideration of the Design of Studies of Chronic Fatigue Syndrome. There were participants from the Centres for Disease Control (CDC) and from the National Institutes of Health (NIH). At this conference, it was recommended that the term CFIDS (pronounced seefids) be used instead of the term CFS on the basis of the immune dysfunction which has been observed in the disorder. One of the presentations was by Dr Sandra Daugherty, who reported that **MRI scans on patients demonstrated abnormalities consistent with demyelination and cerebral oedema in 57% of patients studied.**

1989

Detection of Viral Related Sequences in CFS Patients using the Polymerase Chain Reaction W. John Martin *The Nightingale Research Foundation 1989: 1-5*

1990

Chronic Fatigue Syndrome and the Psychiatrist. SE Abbey, PE Garfinkel. *Can. J. Psychiatry 1990:35:7:625-626*

1992

A Chronic Illness Characterized by Fatigue, Neurologic and Immunologic Disorders, and Active Human Herpesvirus Type 6 Infection. D Buchwald, PR Cheney et al. *Annals of Internal Medicine 1992:116:2:103*

“Magnetic resonance scans of the brain showed punctate, subcortical areas of high signal intensity **consistent with oedema or demyelination in 78% of patients**”

1994

Detection of Intracranial Abnormalities in Patients with Chronic Fatigue Syndrome: Comparison of MR Imaging and SPECT. RB Schwartz, BM Garada. *American Journal of Roentgenology 1994:162:935-941*

1995

Pathophysiology of a Central Cause of Post-Polio Fatigue Richard Bruno et al *Ann New York Acad Sci 1995:753:257-275*

1997

A 56 year old woman with chronic fatigue syndrome Anthony L Komaroff *JAMA 1997:278:14:1179-1184*