NATIONAL ME / FM ACTION NETWORK

Charitable tax no. (BN) 89183 3642 RR001
ISSN 1205-786X

Quest

Quest #59 May/June 2003

National ME/FM Action Network Celebrates 10th Anniversary

On June 16, 2003 the **National ME/FM Action Network** celebrated its 10th anniversary. We are very proud of our accomplishments since we started in June 1993. Anyone who would like a copy of our report on our accomplishments, please let us know and we will be happy to send it to you.

All of you deserve much credit for what we have been able to accomplish due to your support and assistance with our many projects. We must also mention our many doctor and lawyer contacts, both inside and outside Canada, who are always there to lend us a helping hand. We appreciate their help and we will to continue our close relationship with them.

We would also like to remind you to continue to send us names and addresses of doctors and lawyers you are aware of as well as those of your acquaintances so that we may send them information on the new ME/CFS Working Case Clinical Definition, Diagnostic and Treatment Protocols which was published February 1, 2003. The definition is of no use unless it is in the hands of the right people.

Sincerely,
Lydia E. Neilson, President CEO
NATIONAL ME/FM ACTION NETWORK

Chronic Fatigue: Looking Beyond Functional Somatic Syndrome

(An essay for the Newsletter of the National ME/FM Action Network) -
Dr. Abhijit Chaudhuri,

Introduction

Fatigue is a common, yet one of the poorly understood symptoms. It is estimated that fatigue accounts for 10-15 million physician office visits in USA alone every year [1]. After pain, fatigue is probably the second commonest symptom of patients seeking medical attention. Because fatigue is common, it is frequently trivialised. To add to the difficulty, fatigue, like pain, is largely subjective and cannot be objectively quantified. At the outset, however, it is important to distinguish between types of fatigue. Objective fatigue is a measure of muscle contractility and is associated with primary muscle diseases and disorders of neuromuscular transmission [2]. For the purpose of this article, we shall consider the subjective fatigue characterised not only by physical, but also, mental fatigue. This is non-neuromuscular fatigue or central fatigue [3] and cannot be quantified directly by muscle electrophysiology unlike the neuromuscular, objective fatigue.

Fatigue is a symptom, not a disease

All healthy subjects in their lifetime will experience short-term tiredness after exertion, infection or sleep deprivation. Most women in their reproductive life will be tired during their menstrual cycles or after childbirth. These are common examples of physiological fatigue that is self-limiting and recovers after rest or sleep. When fatigue is persistent or relapsing, is not the result of ongoing exertion and is not substantially relieved by rest or sleep, then it becomes a cause for concern. The physician at the primary care is likely to be contacted at this stage because sufferers will consider this symptom as being unusual or unfamiliar in comparison to their previous experience of "normal" tiredness. Just as most people will recognise simple headaches to be self-limiting and not see a doctor until the headache symptoms are too frequent, severe or have unusual characteristics, very few patients with persistent fatigue will be seeking help because of anticipated gain from medicalising their symptom. There is a long list of diseases associated with chronic fatigue. In organic diseases, fatigue is related to the underlying pathophysiological processes. Inflammatory and immunological disorders like rheumatoid arthritis provide excellent examples where cytokines may be responsible for fatigue. When injected in experimental animals, anti-inflammatory cytokines induce slow wave sleep. Cytokines given as drug therapy commonly produce fatigue and myalgia [4]. In a smaller proportion of patients, cytokines also induce neuropsychiatric symptoms and depression. Paroxetine, an anti-depressant that selectively inhibits serotonin reuptake, has been successfully used in patients for the primary prevention of depression induced by high dose interferon-alfa therapy [5].

The first task of the physician is to exclude diseases, medical and psychiatric, that may be associated with chronic fatigue. In psychiatric diseases, fatigue usually reflects depression, dysthymia or hidden anxiety that should be uncovered and addressed. Since in a number of medical disorders, physical and mental fatigue may appear long before other clinical symptoms declare themselves, a complete physical examination and appropriate screening investigations are always necessary [1]. However, a normal clinical assessment and negative test results do not diminish the importance of the problem nor does it automatically confirm that fatigue is psychological. A diagnosis of Addison's disease may be easily missed in the early stages. Similarly, many patients with Parkinson's disease or multiple system atrophy experience fatigue months before other neurological symptoms are clinically apparent.

Chronic fatigue as a clinical entity

Chronic fatigue as a medical problem has been known for centuries. Neurasthenia was a fashionable, but rather imprecise diagnosis in the 19th century [6]. Another condition, termed neuromyasthenia, was characterised by subjective nerve and muscle "fatigue" following the epidemic outbreaks of viral infections since the early part of the last century. The clinical characteristics of the viral epidemic that triggered neuromyasthenia were often similar to non-paralytic poliomyelitis ("atypical poliomyelitis") [7]. Epidemic neuromyasthenia had a distinctive clinical picture consisting of "headache, myalgia, myasthenia, encephalopathy, lymphadenopathy, morbidity and survivance" [8]. Residual symptoms years after included persistence of fatigue, muscle pain, nervousness and disturbance of skin sensitivity (allodynia). Myalgic encephalomyelitis (ME) was the preferred name given to these patients during the 1960s [9]. Sporadic cases of a similar syndrome were subsequently identified as post-viral fatigue syndrome (PVFS). PVFS was a diagnosis of the post-polio immunisation era and many patients had a history of Epstein-Barr virus or Coxsackie virus infection. ME or PVFS is qualitatively similar to a number of fatigue syndromes known as a sequel of systemic infections (poliomyelitis, Lyme disease, Q fever, sepsis) and post-infective neurological diseases (multiple sclerosis, Guillain-Barre syndrome).

Chronic fatigue syndrome (CFS) was introduced in 1988 as a broad term for persistent and relapsing fatigue of over six months' duration for which there was no known medical explanations [10]. ME/PVFS was subsumed within this new designation of CFS. The diagnosis of CFS is clinical. However, not all known psychiatric causes of chronic fatigue are reliably excluded by the present diagnostic criteria of CFS. As a result, it is difficult to know what proportion of patients receiving a diagnosis of CFS will have ME/PVFS as opposed to the cases with medically unexplained chronic fatigue due to anxiety, somatisation or affective disorders. This problem is also central to the interpretation of the research studies and therapeutic trials in CFS that have used different sets of criteria for the selection of patients. It appears that a strict application of the revised Centers for Disease Control (CDC) criteria [11] with exclusion of major psychiatric and somatisation disorders is the best possible way to identify ME patients (CFS/ME) at present. When appropriately defined, CFS/ME has a much lower prevalence (0.2-0.4% in adults and 0.07% in the paediatric age group of 5-15 years) as compared to the cases with medically unexplained chronic fatigue more commonly seen in the primary care (prevalence rate of 2% and above). The use of ME as a diagnostic term has been criticised because there is inadequate pathological evidence of inflammatory

changes in the brain and spinal cord of patients. In the United Kingdom, the conjoint term CFS/ME is preferred at present.

CFS: the nature of symptoms

Fatigue in CFS/ME is qualitatively similar to fatigue due to inflammatory or immune mediated diseases (rheumatoid arthritis and systemic lupus erythematosus), metabolic disorders (diabetes and liver disease) and neurological conditions (multiple sclerosis and Parkinson's disease). Some physicians may consider it significant that CFS/ME does not have a specific or sensitive diagnostic marker and CFS/ME patients may not have abnormal clinical signs. Psychiatrists consider CFS/ME as part of an overarching diagnostic category called "medically unexplained symptoms (MUS)" [12]. According to this view, patients without disease-specific diagnostic markers and suffering from CFS/ME to irritable bowel syndrome have a common condition (MUS) for which cognitive behaviour therapy (CBT) is the treatment of choice.

How scientific is this idea? Perhaps very little, because the diagnosis of "MUS" disregards the underlying physiological principles of biological symptoms, and in doing so, overemphasises the role of psychogenesis. CFS/ME provides a very good example of this oversimplification. It has been reported that nearly half of all CFS/ME patients experience significant depression and anxiety, but yet no study has shown whether these symptoms were the cause or merely the effect of a disabling illness. The psychological paradigm does not explain how the non-psychiatric CFS/ME patients develop their symptoms; yet the psychiatric shoe- horn for CBT is recommended for all CFS/ME cases. Indeed, despite the fact that CFS/ME patients have distinctive neurobiological abnormalities (e.g. buspirone test [13] and regional brain proton magnetic resonance spectroscopy [14]) for which no psychological explanation is readily available, the psychiatric hypothesis for CFS/ME is the one that remains most popular with many physicians and journals like the *British Medical Journal* and the *British Journal of General Practice* in the United Kingdom. Readers of these journals are likely to assume that the biological signatures of CFS/ME are the sole consequence of physical inactivity and are maintained by illness beliefs that encourage activity avoidance. This, of course, is the view of the psychiatrists [15]. Indeed, psychiatric research also claims that membership of patients' groups is associated with poor treatment responses in CFS/ME because the members are encouraged to avoid activity [16]. The alternative explanation that fatigue, and any associated psychological changes may be related to an underlying neurological dysfunction is easily overlooked. Yet another reason for favouring the psychological paradigm of functional somatic syndrome in CFS/ME [17] is that more women than men experience CFS/ME symptoms [15].

Gender bias in fatigue

Community based studies have shown a consistent trend of more women than men experiencing chronic fatigue. CFS/ME is often denigrated as a problem of women "who are tired all the time (TATT)". An otherwise healthy woman will experience periods of fatigue during her menstrual phase, after childbirth or at the menopause. In an interesting study of healthy women of reproductive age, it was discovered that cortical excitability to transcranial magnetic stimulation was significantly altered during the menstrual period [18]. The depression of cortical excitability in the luteal phase was of the same direction and approximates magnitude as that observed after a single dose of benzodiazepines. The result of this unique research indicated that certain biological influence in women might increase the susceptibility to develop fatigue [18]. It therefore comes as no surprise that women with CFS/ME invariably experience worsening of their symptoms during the menstrual periods. In the ME literature, it is well recorded that women "whose menstrual phase had not been previously marked by hyperirritability and emotional tension complained that they could not control themselves and flew into rages at what they realised were really insignificant frustrations and annoyances"[19].

Developing a biological paradigm of CFS/ME

In any complex disorder like CFS/ME, it is important to pay attention to the biological principles underlying disease symptoms. Like CFS/ME, fibromyalgia is common in women and shares common features like fatigue, muscle pain and sleep disorder. Fibromyalgic muscle pain has been considered in the psychiatric literature as somatic dysthymia, somatoform pain disorder or, indeed, another example of medically unexplained symptoms. Recent data suggest that widespread and unpleasant skin sensitivity experienced by many fibromyalgic patients (similar to many CFS patients) may be due to central nervous system amplification of the nociception in general, not to a specific muscle disorder [20]. There is evidence at present that the central fatigue in CFS is influenced by neurochemical changes and result from altered cortical excitability due to a lack of limbic-motor integration of volitional activities [3].

Recent research has also uncovered that genetic or acquired deficits in norepinephrine inactivation may underlie hypoadrenergic states leading to orthostatic intolerance and fatigue [21]. Most patients with orthostatic intolerance are young women [22]. This syndrome, first described by Da Costa more than 100 years ago, has been variously called soldier's heart, neurocirculatory asthenia and mitral valve prolapse syndrome. As an example of fatigue syndrome similar to CFS, mental health professionals implicate psychiatric morbidity in orthostatic intolerance [15].

Mind, body and perception

The current emphasis on the cognitive-behavioural model of fatigue is rather narrow. According to the paradigm developed by the modern philosophers Gorovitz and MacIntyre on the nature of fallibility, ignorance and ineptitude are probably the likeliest reasons for misinterpreting the symptoms of chronic fatigue in many cases. There may be another, a third reason as to why it is difficult to grasp the neurobiology of fatigue. Gorovitz and MacIntyre used the words "necessary fallibility". It means that there may be some kinds of knowledge that science and technology will never unearth.

Biological principles are likely to be overlooked in fatigue research based on questionnaires dealing with the subjective and psychological attributes of fatigue. Because fatigue is perceptual, there has been an unfortunate trend in ignoring the neurobiological processes underlying fatigue. The British neurologist, W Russell Brain, had given an excellent example of how physiological principles influence human perception [23]. If one sits in a completely black room with the eyes closed, he or she sees a black expanse that is very near or behind the closed eyelids. When the eyes are opened in the same room, the black expanse appears to be outside at a finite distance. The sensory shift in the perception of the black expanse depends solely on the nervous impulses from the muscles that open and close the eyes, since nothing else has changed. Opening the eyes in the dark room causes an electrical change in the visual cortex simultaneous with the spatial change in the sensation of blackness. This simple example shows that simultaneous processing of information in the brain is responsible for changes in sensory perception. Subjective fatigue is a perceived sensation and this is higher in those with chronic fatigue. Changes in the neural information processing offers an approach to the perceptual basis of fatigue that remains to be explored in medical research.

Parallel information processing and central fatigue

Unlike the anatomy of peripheral fatigue that can be localised to the neuromuscular junction and muscles, the neuroanatomy of central fatigue is more complex. This complexity, however, is not unique to the symptom of brain generated central fatigue but applies to most higher order functions such as cognition and motivation. Although subjective feeling of fatigue is experienced as an instantaneous process, it combines several distinct processes such as perceiving, memory, motivation, thinking and formulating a response to prevent self-harm that are continuous, indivisible and requires parallel information processing components in several brain areas. Homeostatic processes such as temperature regulation, feeding and thirst correspond to motivational states and these homeostatic processes are, in turn, controlled by hypothalamic functions. Motivational states are also regulated by factors other than the physiological needs such as reward and pleasure sensations.

If we follow the principle of parallel information processing in multiple brain areas for higher order, perceptual functions in humans, then it is possible to identify some of the component regions that may be involved in fatigue. Brain derived chronic fatigue is a common in diseases that affect subcortical areas, basal ganglia, limbic system, thalamus, hypothalamus, reticular and autonomic systems and specific brain stem nuclei (medulla). In multiple sclerosis, severity of fatigue correlates with altered metabolism in the basal ganglia and dorsolateral prefrontal brain regions. In myotonic dystrophy, fatigue is a conspicuous symptom and the brain pathology is characterised by neuronal loss in dorsal raphe nucleus and medullary nuclei as well as cytoplasmic inclusion bodies in the thalamus, caudate and other brain stem nuclei. It appears that those brain regions that are responsible for integrating afferent traffic from unpleasant sensations and motivational values to the higher order cortical activities are relevant in the experience of fatigue. Dysfunction of this extended neural network at one or more levels may provoke the symptom of abnormal and persistent fatigue in brain diseases.

Looking beyond psychological paradigms in CFS/ME

CFS/ME must not be viewed in complete isolation from fatigue due to other medical or neurological disorders. Anxiety and depression are common and are often the first symptoms of brain diseases ranging from Huntington's disease to multiple sclerosis. Yet, it is only relatively recently that Huntington's disease and multiple sclerosis have been firmly established as neurological diseases and not being a problem "in the mind". In multiple sclerosis, fatigue is the only disabling symptom in 40% patients 24 and over 85% of patients experience fatigue in the course of their illness [25]. Anxiety is also common in these patients and the rate of depression in multiple sclerosis is nearly seven fold higher than the normal population. Studies have shown that fatigue in MS and CFS/ME is comparable and the cognitive deficits in CFS/ME cannot be explained by depressive psychopathology [26]. The basis of fatigue in multiple sclerosis or human prion diseases remains as unclear as in CFS/ME. It therefore comes as a surprise to many patients when fatigue in CFS/ME is viewed as a psychological problem similar to hysteria and their disability trivialised as nothing more than ordinary unhappiness or tiredness. Failure to recognise fatigue or its importance in a patient may undermine the physician-patient relationship that is vital for any successful therapeutic partnership. Normal or negative test results in CFS/ME only exclude other diseases and do not reduce the importance or the existence of fatigue; nor do these results positively identify chronic fatigue as a medically unexplained symptom.

Conclusions

It is easy to dismiss subjective fatigue as a largely psychological phenomenon and CFS/ME as a "non-disease"[27]. However, it still remains open to debate what precisely constitutes the psychological process responsible for the chronic disability in CFS/ME. From a neurologist's viewpoint, fatigue may be considered as a psychological problem provided "psyche" is defined as the collective force of integrative cortical function driven by the physiological principles of neuroscience. On the other hand, if the psychological process in fatigue is equated with the unsubstantiated hypotheses of illusory perception of effort, wrong body image, avoidance behaviour for physical activity or functional somatic syndrome, then it is a tragic fallibility of science. Lest we forget, Charcot, one of the founders of modern neurology and psychiatry, had emphasised more than a century ago, " When a patient calls on you, he is under no obligation to have a simple disease just to please you". Nothing provides a better example than CFS/ME.

[Ed Note: Dr. Chaudhuri is a Senior Lecturer in Clinical Neurosciences, University of Glasgow, and Consultant Neurologist, Institute of Neurological Sciences, Southern General Hospital, 1345 Govan Road, Glasgow G51 4TF Scotland, United Kingdom. Fax: 0141 201 2993 - E-mail: ac54p@udcf.gla.ac.uk]

Acknowledgement

AC is supported by the Barclay Research Trust held at the University of Glasgow.

References

1. Sharma OP. Fatigue and sarcoidosis. Eur Respir J 1999; 13:713-4.
2. Layzer RB. Asthenia and chronic fatigue syndrome. Muscle Nerve 1998; 21: 1609-11.
3. Chaudhuri A, Behan PO. Fatigue and basal ganglia. J Neurol Sci 2000; 179:34-42.
4. Krupp LB, Pollina DA. Mechanisms and management of fatigue in progressive neurological disorders. Current Opinion in Neurology 1996; 9:456-60.
5.Musselman DL, Lawson DH, Gumnick JF,et al. Paroxetine for the prevention of depression induced by high dose interferon-alfa. N Engl J Med 2001; 344: 961-6.
6.Gowers WR. Neurasthenia. In: A manual of diseases of the nervous system. London: J&A Churchill, 1893. Volume II, pp1045-50.
7. Ramsay AM, O'Sullivan E. Encephalomyelitis simulating poliomyelitis. Lancet 1956; 1: 761-4.
8. Holt GW. Epidemic neuromyasthenia: the sporadic form. Am J Med Sci 1965; 249: 98-112.
9. Chaudhuri A, Behan PO. Neurological dysfunction in chronic fatigue syndrome. J Chr Fatigue Synd 2000; 6(3/4): 51-68.
10. Holmes GP, Kaplan JE, Glantz NM, et al. Chronic fatigue syndrome: a working case definition. Ann Intern Med 1988; 108:387-9.
11. Fukuda K, Straus SE, Hickie I, et al. The chronic fatigue syndrome: a comprehensive approach to its definition and study. Ann Intern Med 1994; 121:953-59.
12. Sharpe M. The report of the Chief Medical Officer's CFS/ME Working Group: What does it say and will it help? Clinical Medicine 2002; 2: 427-9.
13. Bakheit AMO, Behan PO, Dinan TG et al. Possible upregulation of hypothalamic 5-hydroxytryptamine receptors in patients with post-viral fatigue syndrome. BMJ 1992; 304: 1010-2.

14. Chaudhuri A, Condon BR, Gow JW, Brennan D, Hadley DM. Proton magnetic resonance spectroscopy of basal ganglia in chronic fatigue syndrome. NeuroReport 2003; 14:225-8.
15. Wessely S, Hotopf M. Sharpe M. Chronic fatigue and its syndromes. Oxford: Oxford University Press, 1998.
16. Bentall RP, Powell P, Nye FJ, Edwards RHT. Predictors of response to treatment for chronic fatigue syndrome. Br J Psychiatry 2002; 181: 248-52.
17. Wessely S, Nimnuan C, Sharpe M. Functional somatic syndromes: one or many? Lancet 1999;354:936-9.
18.Smith MJ, Keel JC, Greenberg BD, et al. Menstrual cycle effects on cortical excitability. Neurology 1999; 53: 2069-72.
19. Deisher JB. Benign myalgic encephalomyelitis (Iceland Disease) in Alaska. Northwest Medicine 1957; 56: 1451-6.
20. Mense S, Simons DG, Russell IJ. Muscle pain: understanding its nature, diagnosis and treatment. Philadelphia: Lippincott Williams & Wilkins 2001.
21. Shannon JR, Flattem NL, Jordan J, et al. Orthostatic intolerance and tachycardia associated with norepinephrine-transporter deficiency. N Engl J Med 2000; 342:541-9.
22. Low PA, Opfer-Gehrking TL, Textor SC, et al. Postural tachycardia syndrome (POTS). Neurology 1995; 45:Suppl 5:S19-S25.
23. W. Russell Brain. Mind, perception and science. Oxford: Blackwell 1951.
24. Murray TJ. Amantadine treatment for fatigue in multiple sclerosis. Can J Neurol Sci 1985; 12: 251-4.
25. Krupp LB, Alvarez LA, LaRocca NG, Schienberg LC. Fatigue in multiple sclerosis. Arch Neurol 1988; 45: 135-7.
26. Daly E, Komaroff AL, Bloomingdale K, Wilson SI, Albert MS. Neuropsychological function in patients with chronic fatigue syndrome, multiple sclerosis and depression. Appl Neuropsychol 2001; 8: 12-22.
27. http:/bmj.com/cgi/content/full/324/7334/DC1.

U.S. DOCTORS' ROSTER - ADDITION

RICHARD N. PODELL, M.D. **Diagnosis & treatment for ME/CFS and FM**
105 Morris Avenue Tel: **(973) 218-9197** Fax **(973) 218-1199**
Springfield, NJ 07081 U.S.A. Web: **DrPodell.org**

NATIONAL LAWYERS' ROSTER - ADDITION - INITIAL CONSULTATIONS FREE

NORM CUDDY Tel. **(204) 944-3253** - Fax **(204) 947-2593**
TAPPY CUDDY
1000 - 330 St. Mary Ave. E-mail: **Mac@tcwpg.com**
Winnipeg, MB R3C 3Z5 Web: **www.tcwpg.com**

BARRY L. EVANS Tel. **(905) 433-1200 -** Fax **(905) 433-2555**
419 King St. West, Suite 208
Oshawa, ON L1J 2K5 E-mail: **blevans@interlinks.net**

ASHLEY R. GNYS
SHARPE, BERESH & GNYS Tel**. (905) 357-5555 -** Fax **(905) 357-5760**
4700 St. Clair Avenue
Niagara Falls, ON L2E 3S8 E-mail: **gnys@on.aibn.com**

ANDREW KERR Tel. **(705) 722-6393 -** Fax **(705) 725-7359**
McCARTHY RASTIN
48 Alliance Blvd., Suite 201A
Barrie, ON L4M 5K3 E-mail: **andrew.kerr@mbrbarrie.ca**

ROBERT TOCCHET Tel. **(902) 566-3400 -** Fax **(902) 566-9266**
CAMPBELL LEA
15 Queen St., Box 429

Charlottetown, PEI C1A 7K7 E-mail: **rtocchet@campbelllea.com**

ANDRÉ BOURDON Tel. **(705) 268-4242 -** Fax **(705) 264-1646**
GIRONES & ASSOCIATES
16 Cedar Street South E-mail: **abourdon@girones.on.ca**
Timmins, ON P4N 2G4 Web: **www.girones.on.ca**

IME/FAE REGISTRY SUBMISSIONS

The **National ME/FM Action Network** continues to urge everyone who has attended an Independent Medical Examination (IME), Functional Abilities Evaluation (FAE) or any other form of assessment at the request of an insurance company or Canada Pension Plan (CPP) or Workmen's Compensation Board to act now by filling out our 7 question, confidential, independent medical examination Registry Submission Form so that the names of the doctors and healthcare professionals who evaluated you can be put on record.
To receive a copy or copies of the simple, confidential 7 question Registry Submission Form, please contact: **Mary Ellen**, Manager Special Projects, Mail: **P.O. Box 66172, Town Centre Postal Outlet, 1355 Kingston Rd., Pickering, ON L1V 6P7** - Phone or Fax: **(905) 831-4744** - Email: **marye@pathcom.com** - Or download the Form from our website at **www.mefmaction.net/medexac.html**.

OUR WORLD: FAVOURITE GAME SHOWS: The Wheel of Misconceptions - Medical Jeopardy - Symptoms Squares - Beat the Symptoms. Thank you to Lorraine Legendre, Ottawa.

RESOURCE BOOKS:

• ***Myalgic Encephalomyelitis / Chronic Fatigue Syndrome: Clinical Working Case Definition, Diagnostic and Treatment Protocols (Journal of Chronic Fatigue Syndrome, Volume 11, Number 1, 2003)*** is available for **U.S. $14.95** per copy plus $5.00 Shipping and Handling. To order, contact **The Haworth Press, Inc.,** 10 Alice Street, Binghamton, NY 13904-1580 USA**.** Telephone in US/Canada: **(800) 429-6784,** Telephone outside **US/Canada: (607) 722-5857, fax: (607) 771-0012,** email:**orders@haworthpressinc.com Online: http://www.haworthpressinc.com/store/product.asp?sku=4958**

• **QUEST COLLECTION" BOOK - FIVE YEARS (1993 TO 1998) :** By popular request, the **National ME/FM Action Network** has published an easy to read book consisting of a **collection** of important articles which have appeared in our **'QUEST'** newsletters over the years. For easy reference, these articles have been grouped into sections, according to their focus i.e. medical, legal etc. We have kept the **cost** of the book to a minimum of **$20.00** each which includes shipping and printing.
**QUEST Collection for 1999 to 2002 will be available in the Fall.

• **TEACH-ME - Sourcebook for Teachers.** We are proud to announce that the hard-copy of **our Sourcebook for Teachers**, an educational resource book full of information and teaching strategies for teachers and parents of children and youth who have ME/CFS and/or FM, is now available. Price **$22.00** (includes S & H) – Discount on bulk orders.
Cheques payable to the **National ME/FM Action Network**.and let us know how many copies you would like.
Please also see our youth and parents' pages on our website at: **www.mefmaction.net**

• **LEGAL/RESEARCH PACKAGE** - **Medical** and **Legal** Information on ME/CFS and/or FM, a resource for lawyers, advocates, doctors and patients. Please make **cheque payable to Marj van de Sande** in the amount of **$25.00** (our Director of Education) to cover photocopying, postage charges etc., **151 Arbour Ridge Circle NW, Calgary, AB T3G 3V9 - Tel/Fax: (403) 547-8799.**
E-mail: mvandes@telus.net

MEMBERSHIP: **$25.00 per year which includes bi-monthly newsletters** – Payment can be made by **CHEQUE, VISA or MASTERCARD** - **NATIONAL ME/FM ACTION NETWORK, 3836 Carling Ave., Nepean, ON K2K 2Y6 Canada**

Tel/Fax: (613) 829-6667
E-mail: ag922@ncf.ca - Web: http://www.mefmaction.net

DISCLAIMER: The NATIONAL ME/FM ACTION NETWORK serves as a clearing house for information on Myalgic Encephalomyelitis / Chronic Fatigue Syndrome and Fibromyalgia. Some of the information contained herein is intended to help patients and their physicians make informed decisions about their health. However, the NATIONAL ME/FM ACTION NETWORK does not dispense medical advice or endorse any specific medical hypothesis or product and assumes no responsibility for any treatment or action undertaken by its readers.