Talking Forget-ME-Not

Official Journal of the ME/CFS Society (SA) Inc

2007 Issue 2

Your Society forget-ME-not

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ME/CFS Society (SA) Inc.

The ME/CFS Society (SA) Inc. is a non-profit organisation (Registered Charity 698) which aims to:
promote recognition and understanding of the disease among the medical profession and the wider community
provide information and support for people with ME/CFS and their families

Patron

Her Excellency Marjorie Jackson-Nelson AC, CVO, MBE, Governor of South Australia.

Management Committee – 2007/2008

The Society is directly administered by a voluntary committee elected at the Annual General Meeting.

President: Peter Cahalan
Vice-President: (vacant)
Honorary Secretary: Peter Mitchell
Treasurer: Richard Cocker
Management Committee Members: Lynda Brett; Melanie Cocker; Adrian Hill; Spen Langman; Emma Wing

Membership

Annual membership is from July 1 to June 30, and includes subscription to the magazine *Talking Point*. Membership rates for first-time members are as follows (GST included):

New Members (cheaper rates apply for renewal):
Single membership.....$35
Single Concession $25
Professional.....$50
Family.....$40
Family Concession $35
Overseas – as above plus $10

(Family membership is designed for families with more than one person who will directly benefit from the membership at the same place of residence. Family Concession applies when the main breadwinners are concession card holders.)

Donations

Donations are an important source of income for the Society and are welcome at all times.

All donations of $2.00 or over are tax deductible and a receipt will be issued.

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Talking Point

Talking Point is the official journal of the ME/CFS Society (SA) Inc. It is published quarterly, and is financed primarily by member subscriptions.
Editor: Peter Scott (pmrscott@tpg.com.au).
Assistant Editor: Jenni Gay (jrgay@iprimus.com.au).

Talking Point Subscriptions

Professionals.....$35
Persons with ME/CFS.....$22
Overseas (Asia-Pacific).....$32
Overseas (Rest of World) ...$38

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Note: It is our policy to ignore anonymous correspondence.

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Wednesdays 10am to 3pm (subject to volunteer availability).
Our email address is: sacfs@sacfs.asn.au.
Our Web site address is: www.sacfs.asn.au.
Our youth Web site address: www.sayme.org.au.

Disclaimer

The ME/CFS Society (SA) Inc. aims to keep members informed about research projects, diets, medications, therapies etc.

All communication both verbal and written is merely to disseminate information and not to make recommendations or directives.

Unless otherwise stated, the views expressed in Talking Point are not necessarily the official views of the Society or its Management Committee and do not imply endorsement of any products or services (including those appearing in paid advertisements) or treatments.

Always consult your medical practitioners before commencing any new treatments.

Notice to Vendors

The ME/CFS Society (SA) Inc. does not permit direct marketing of products to our members. This includes distributing promotional literature, providing demonstrations of products or approaching members at any of our events.

If you have information about products which you wish to bring to the attention of the Society, you should direct it to the Information Officer GPO Box 383, Adelaide 5001.

In particular, you should note that members give their contact details to the Society in trust and misuse of those is a breach of confidentiality. Any use of member information for direct marketing will be investigated and dealt with appropriately.

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Queensland ME Quarterly, Queensland ME/CFS Syndrome Society, PO Box 938, Fortitude Valley Qld, 4006.
ChaMEleon, ACT ME/CFS Society, Shout Office, Collett Place, Pearce ACT 2607.
ME/CFS News, ME/CFS Society W.A. Inc., c/- WISH, PO Box 8140, Perth, WA 6000.
The CFIDS Chronicle, CFIDS Association, PO BOX 220398, Charlotte, NC28222-0398, USA.
Perspectives, ME Association, Stanhope House, High Street, Stanford le Hope, Essex SS17 OHA, UK.
Country Network, Journal of the Northern Rivers ME/CFS/FM Support Assoc. Inc. PO Box 6024 Lismore NSW 2480.

From the President

By Peter Cahalan, President ME/CFS Society (SA) Inc.

The last several months have been an exciting time for the Society. The great bulk of our energies have gone into organising the two events surrounding the visit to Adelaide of four internationally renowned researchers and physicians. A busy time but pretty satisfying.

The public meeting

Sunday nights are not a great time for many people with ME/CFS to come out for a meeting. But over 550 people gathered in the Norwood Town Hall on 25 March. They were there to hear four eminent international experts who had been brought to Adelaide for a research forum over the next two days.

Professor Kenny De Meirleir (Belgium/USA) was returning to Adelaide, having been the keynote speaker at a similar event two years ago. Professor Abijhit Chaudhuri (UK) has amongst other things campaigned for the establishment of tissue banks of material from people who have died from ME/CFS to help advance research into the condition. Dr Dan Peterson (USA) is renowned as a local GP who happened to find himself in the midst of an outbreak of ME/CFS in Incline Village, Nevada in the early 1980s and who became a persistent champion of more research and better treatment into the condition. And Emeritus Professor Malcolm Hooper (UK) is a feisty campaigner for people with ME/CFS, MCS (multiple chemical sensitivity) and Gulf War Syndrome.

The four each spoke briefly. This gave plenty of time for a long panel session with questions fired at them from the audience. We chose to take written questions only as often at these events some questions are rather longer and more time-consuming than they need to be. This way we got lots of questions and answers. Perhaps it was all a bit too relentless and I think many people in the audience were quite exhausted at the end of the two-hour meeting.

I want to warmly thank the many volunteers who helped to get the meeting organised, and those who came along to help on the night. We secured one sponsorship – of $500 from Eco Pest Control, a small firm to whom we are most grateful. And one member spent several thousands of dollars on two large advertisements in the Sunday Mail and Advertiser to ensure that we got the word out to the wider community. At least 50 people on the night indicated that they had come because of seeing the ads. So they were certainly worth it.

We set a differential charge for attendance with members paying a gold coin donation and non-members charged $5. We're aiming to keep enhancing member benefits and this was one time when we could do so. We also were able to distribute to members free copies of the short version of the Canadian Guidelines on ME/CFS. The ME/CFS Association of Australia – like other bodies overseas – has accepted that the Guidelines should be used as the normative description of the condition. If you weren't there on the night and are wondering whether you are going to get a copy too – you are. But it will take our small volunteer team at the office to get to that amongst other tasks.

We took over $2500 on the night. With the $500 sponsorship that enabled us to just break even. There are those who said we should have charged more on the evening. Perhaps so. But we persist with trying to keep the basic costs of such events (and membership fees) as low as possible to ensure that we enfold people with small incomes.

We filmed the evening with the aim of making a DVD of it. There was an instant demand for it and, as I write in mid-April, we have over 100 orders for it. If you were one of those who pre-ordered, you can either ring the office or go onto the website at www.sacs.asn.au to confirm payment and delivery.

Finally, the evening generated about 40 new members for us. This means that we have been able to reach a target which we set for ourselves – without fanfare – after a major overhaul of our membership lists. We had found that we had a fair number of non-financial members and the sifting showed that we were hovering around the 240-member (financial) mark in January. We set ourselves the aim of getting to 300 financial members by 30 June. Well, we've made it! Mind you – it's pretty astonishing that so few people with ME/CFS sustain our work financially. If you take the usual estimates of up to 7000 people with ME/CFS in South Australia, then perhaps as few as one in twenty of them are in there fighting with us for them. We'd like to get that number up.

The research forum

I was privileged to sit in on the two-day meeting of the four internationals with a group of leading Austral-

ian researchers and physicians. I came away with the same feeling as after Professor De Meirleir's forum in 2005. For two days the researchers traversed just about every aspect of the condition without having to go anywhere near psychiatry as a discipline which can explain the fundamentals of ME/CFS. There was in fact a palpable mood of frustration that in countries such as Britain scarce resources have been channelled to cognitive behaviour therapy and to psychiatric research. You can't listen to presentation after presentation without coming away both bewildered as a layperson with the blitz of medical terms and absolutely certain that it's a biological condition.

I don't intend to write further here about the seminar. A report is being prepared by the Alison Hunter Memorial Foundation which organised and raised funds for the forum and you will hear more about it via that report. It will be written by experts – and in this field I simply am not one of them!!

I want to thank Christine Hunter AM, Annette Leggo and their fellow Board members for their outstanding work. If you wish to donate to key efforts to help our cause – then go for either your local Society (us) or for the Foundation.

The Multiple Chemical Sensitivity campaign

As I write the Health Department Reference Group on MCS has met for its second time – but still without the two local government representatives whose nominations the Department had sought a mere nine months ago! Still, they have now been appointed and the committee will have held its first full meeting by the time you read this.

The spirit on it is not too bad at present. The proof will be in whether real progress is made in improving the situation for people with chemical sensitivities. We will keep you informed. But it would be true to say that from my own perspective I have not had much time to put into this aspect of our Society's work in the first months of 2007. The big events in March have dominated our time and energies and following through after them is itself a big job for the team. Still, there'll come a time when we can get back to keeping up the pressure for positive change.

Our communications strategy powers on

We seem to be reaching an increasingly wide audience. Our website statistics especially seem to keep moving upwards. From our base of 30 000 visitors a year in 2004 we have climbed to around 110 000 as of now with over 1.5 million hits. If we had the time we'd spend some of it working out whether we could make revenue from the website, which is such a wonderful asset for us.

We're learning how to use it all the time. We recorded several radio interviews which Dr Peter Del Fante, our honorary secretary Peter Mitchell and I all recorded in March. The level of interest in them – as recorded as actual downloads by people wishing to listen to them in full – has been remarkable. We'll try to do more of this in future.

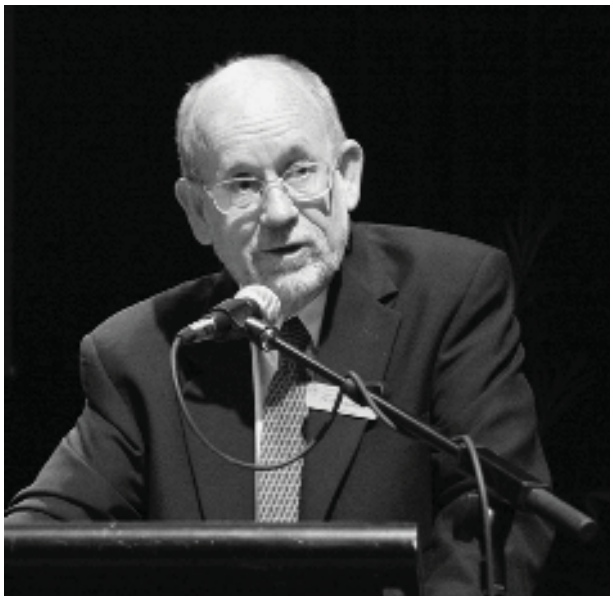
Our history

As I was writing the above an email came in from Jenni Gay. Jenni is a long-standing member and assists Peter Scott with the website and *Talking Point*. Jenni is very interested in us doing something to celebrate what she thinks is our 20th anniversary. You might recall that I asked people about the early history of the Society some time ago. I must say I had thought we might go back to around 1984. Jenni thinks the founding year was 1987 [*see pages 8 and 9*].

I strongly endorse Jenni's plea for us to dig for more about our history and to be able to send a good store of archival material to the Mortlock Library of South Australiana.

That's all for now. I hope you'll be able to join us for one or more of our seminars this year; that you enjoy the improved-look *Talking Point*; and that you get a chance sometime this year to beard a politician or influential person in their den and skewer them with lethal information about the importance of better treatment for people with ME/CFS. Good hunting!

1



Peter Cahalan

Photos from the 25 March meeting

The Society, in conjunction with the Alison Hunter Memorial Foundation, held a meeting at Norwood Concert Hall on 25 March 2007. It was entitled "ME/CFS: breaking news from around the world", and featured the following international speakers: Dr Abhijit Chaudhuri (UK); Prof Kenny De Meirleir (Belgium); Prof Malcolm Hooper (UK); and Dr Daniel Peterson (USA). Here are a few photos from the meeting:

Norwood Concert Hall foyer

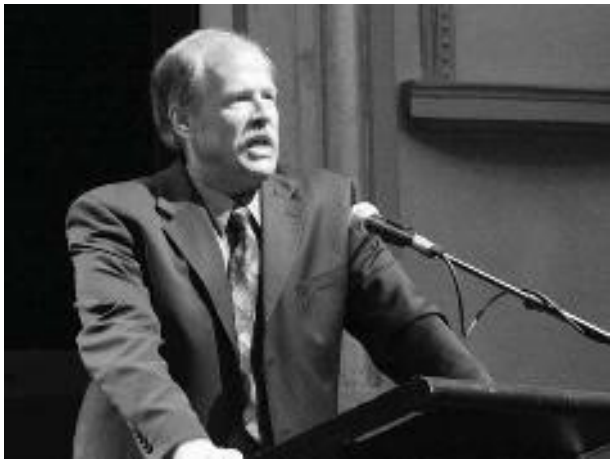
Audience building up before the event

Audience

Audience and speakers

The speakers (l to r):
Prof Kenny De Meirleir; Dr Daniel Peterson; Dr Abhijit Chaudhuri; Prof Malcolm Hooper; and Christine Hunter AM

Prof Kenny De Meirleir



Dr Daniel Peterson

Prof Malcolm Hooper

The speakers with Peter Cahalan

Adelaide Support Group closes

We regret to announce that the long-running Adelaide Support Group is closing shop for the present. We are very grateful to its leader, Darryl Turner for his hard work over the last few years.

Numbers had dropped to near zero and so Darryl understandably has decided to call it a day.

We'll let the dust settle for a while and keep an eye on whether there's a future increase in demand for a group such as this in the central metropolitan area.

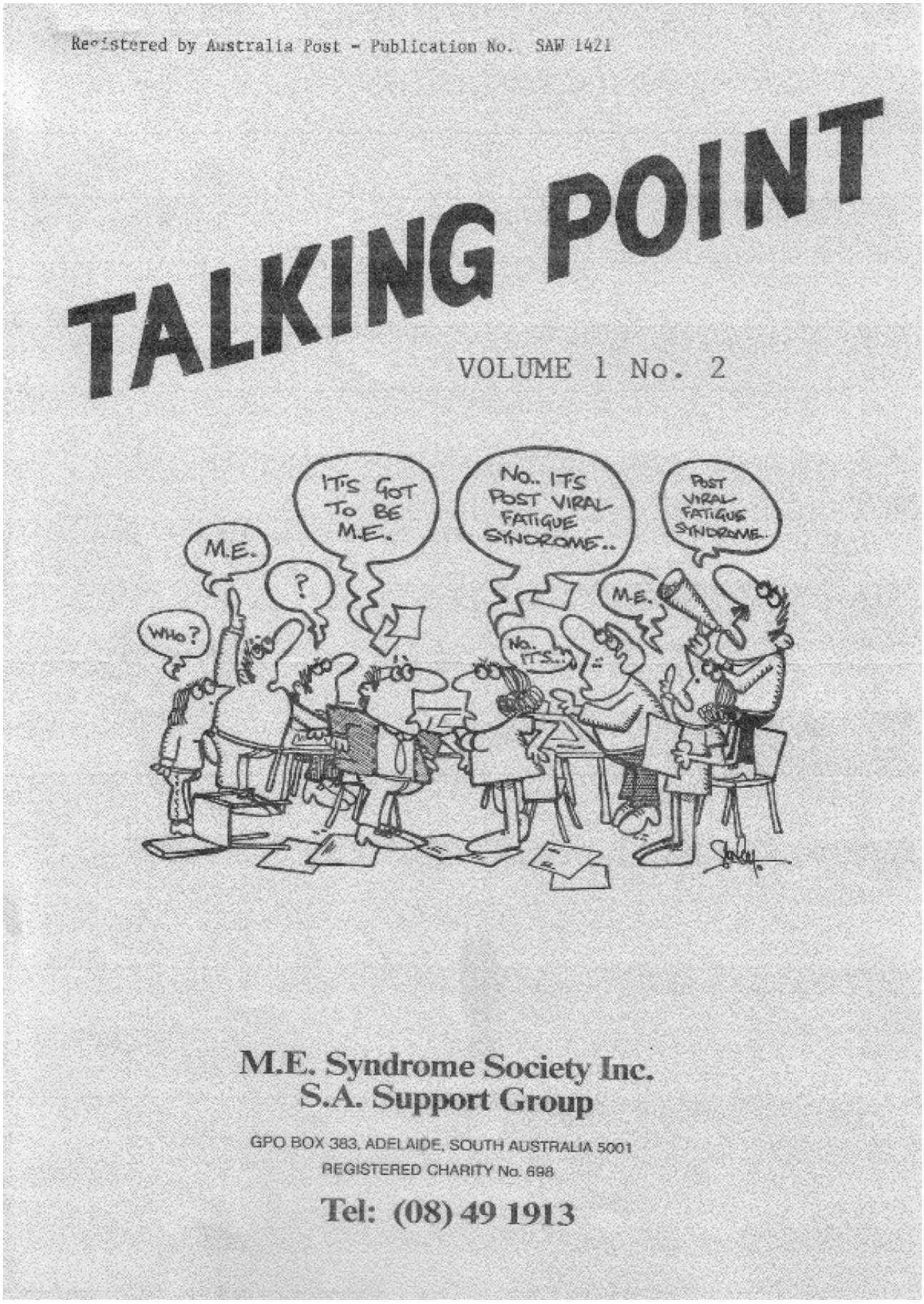
Meanwhile it's nice to note that efforts are under way to see if a support group can be set up in the Clare Valley area. And the active Yorke Peninsula Support Group had an excellent meeting addressed by Dr Ian Buttfield on 21 April. 1

Volunteers

Volunteers

It was twenty years ago today…

Here is the cover of the second ever issue of Talking Point*, published in 1987:*



Is it our 20th birthday this year?

By Jenni Gay.

I am keen to preserve our Society's history before it is too late and would appreciate hearing from people who were around in the early days. I joined the Society in late 1987 but left in mid-1993 and rejoined in Dec 2000.

I still remember the day I rang the President after a suggestion by Dr Kathleen Maros, my diagnosing doctor. I also have vivid memories of Lyn Drysdale telling me she was talking to me from her bed and that it was her office. (I couldn't understand this at the time, but little did I know that I would also be doing that in the future some of the time – in fact, most of this has been typed while sitting and lying in bed as I recover from a severe relapse.) It was just wonderful after 15 years of being unwell to find that it wasn't all in my head and to receive her precious support.

The first *Talking Point* in my collection is Vol 1 Issue 2. It contains the Financial statement dated 2nd Feb 1987 - 30th Jun 1987 for the AGM held in August that year. This information and the editorial has lead me to conclude that the Society officially started in February that year i.e. 20 years ago last February. In that first five months, the Society had grown from 4 members to 130 and had taken $674 in Membership fees and $1320 in donations. All done without email, the Internet or computers!

Our first president and founder was Lyn Drysdale of West Lakes Shore, and quarterly meetings were held at the Australian Mineral Foundation at Conyngham St, Glenside. They were very well attended by over 100 people each time. We all brought a plate to share, and raffle tickets were sold to raise funds. Lyn was a very dynamic lady despite her own ill health. The first committee consisted of:

President	Lyn Drysdale
Secretary	Simon Fisher
Treasurer	Kay Botroff
Committee	Brian Caire
	Colleen Harris
	Chris Hughes
	Phil Kirk
	Jeff Gregory

I would also like to compile a couple of full collections of *Talking Point* as they are an important record of our Society and its activities over the last 20 years. One collection would be held in our own Office and the other given to the State Library which already holds some issues from 1996. I will donate my early issues but need the following issues to complete this project.

Volume	Issue No.	Number needed
1	1	2
1	2	1
2	1	2
2	2	2
2	3	2
2	4	1
3	1	1
3	2	1
3	3	1
3	4	1
4	1	1
4	2	1
4	3	1
4	4	1
5	1	1
5	2	1
5	3	1
5	4	1
1992 (only have Dec)		2 of any issues
1993 (only have Jun)		2 of any issues
1994 - 2000		2 of all issues
2000 onwards		1 of all issues

Please contact me:

- by email (jrgay@iprimus.com.au);
- write to me c/- of the Office;
- phone the Office on Wed (8410 8929) and leave your number and suitable times and I will ring over the next week.

Kadina meeting

A report by Jane Gill of a CFS meeting at Kadina Town Hall on Saturday 21 April 2007 in which Dr Ian Buttfield was guest speaker.

Dr Buttfield's visit was very productive.

We had 30 people attend – we were hoping for at least 10 and Dr Buttfield thought only six would go – so 30 was significant.

He talked about the cell – the damage to it and the effect on patients. He talked about the mental health aspects of CFS, its social aspects, and mentioned his involvement on the Chemical Sensitivity board.

Because there were so many there and because there were so many questions – a lot of them personal – there wasn't really time for much discussion on the chemical sensitivity work.

He talked for about an hour, with question time following.

There were quite a few at the meeting I didn't know.

One chap – wasn't sure if he had it – but after hearing the talk decided to revisit his GP armed with this information. Others I keep referring to the same GP in Kadina, Dr Wood (who I go to), and when talking to him this week, I said I believe that by sending everyone to him, he will be hearing similar histories and gathering more information re CFS, and for that reason alone it's smarter to have at least one clued up GP in the area than a lot getting a little exposure to it.

Dr Ian Buttfield and David Shepherd

It was an extremely successful afternoon. We put on a very yummy afternoon tea – fresh fruits, dried fruits, fruit balls, salad/cold meat platter, tea, coffee etc. and fresh fruit juice/water when they arrived. We wanted to make every effort to make them truly feel valued.

Love and hugs and prayers for the CFS society. 1

DIRC wants you to tell your story

Telling stories can empower, raise awareness and improve understanding.

The Disability Information and Resource Centre (DIRC) wants people with a disability in South Australia to tell their stories and share their experiences.

DIRC has received a grant from Community Benefit SA to fund the establishment of the *History of Disability in South Australia* project. The project will include the history of people with a disability in South Australia since settlement in 1836. Over the past 170 years much has happened in the lives of people with a disability. DIRC will create a website that will explore and record this history.

We want your stories so we can acknowledge, commemorate and celebrate the lives of people with a disability. These stories will provide a lasting legacy for all South Australians.

Stories will be chosen to feature on our *History of Disability in South Australia* website.

To tell your story go to www.dircsa.org.au/history/submit, complete the form and submit it to us by 30th June 2007. 1

My relapse

ANZMES member, Heather Roberts, recently went through an exhilarating recovery, followed by a shattering relapse. In this article she shares some of what she has learned from the experience.

After just over three years in bed, on March 23 2006 I suddenly made a dramatic improvement. From being able to walk poorly with a walker that morning, in the afternoon I was able to walk confidently around 30m with the walker, to stay up for about four hours and even to do some weeding in the garden! I was so much better, that a few days later I dared to begin to suspect that I had fully recovered.

There followed a glorious and terrifying three months as my world expanded, then a sickening disappointment when I caught a cold and my energy continued to drain away long after the cold symptoms had passed. I was experiencing my first relapse. I don't think I initially handled it very well, and, now in November, there has still been no recovery of energy levels, but I've learnt a few things about coping with the new situation. Here are the main ones.

Accept what has happened and stop fighting. It took me some months to be able to bear to take this step, but I have had to learn that over-activity, however appealing or essential the activity in question seems to be, only makes me sick.

Establish a new routine. Whilst I was stronger, I was staying up for periods of four or even five hours at a stretch, and mostly just doing things when I felt like it. Carrying on with this policy when my energy was greatly diminished led to exhaustion, labile emotions, and failing to manage the basics like regularly showering or cleaning my teeth. I now have a routine to which I strive to adhere whereby in my first activity period of the morning I get the essentials done, leaving more pleasurable activities and those that could be completed by someone else to the lunchtime and afternoon activity slots. I have also found a simple timer with an alarm to be helpful with encouraging me to stick to this routine. I set it to go off when my daily schedule says I need to return to bed, so that I'm less likely to continue with what I'm doing until it's finished and simply wear myself out.

Re-learn how to rest. The reduction in my energy has been frustrating, so I've needed to develop strategies to enable me to rest. Otherwise I can get so agitated lying in bed that I simply have to get up and do something, no matter how much my body protests! For me, doing a calming activity as soon as I first lie down helps me to wind down. So I will often begin by playing something mindless like Solitaire on the laptop. If I get agitated, I'll also often put on either soothing music or a really interesting story. The music calms me and the story distracts me from thinking about all the things that need to get done.

Use activity periods for the good of my body, as well as my mind. For me, the main thing this has meant has been shortening my activity periods, but being more active in them. Initially I was focusing, once the basics were taken care of, on sewing Christmas presents and writing emails – both very sedentary activities. This meant that I was physically almost completely inactive, and, with the help of my doctor, I eventually made the connection between this and the hugely painful buildup of gas I was getting in my gut. I need to move if want my bowels to move! I have now tried to include some significant physical activity into at least one of my activity periods as well as slipping in small 'bites' of activity (such as pacing a little whilst waiting for the jug to boil) into the rest of my day.

Hope these tips are of use to someone! 1

Reprinted with permission from Heather Roberts. Originally published in Meeting Place, *Issue 86 December 2006.*

Relapses in CFS

Relapses are one of the most frustrating and difficult aspects of ME/CFS. Those of us with the disease are almost certain to experience a relapse at some time or another. There are things that we can do that will help with relapses though. In this article, Dr Rosamund Vallings examines ways to avoid or at least minimise and manage our way through relapses.

Typically, Chronic Fatigue Syndrome is an illness characterized by remissions and relapses. Even during the remissions most people say they never feel entirely well, but will often feel well enough to lead a near normal life. One of the greatest risk times however is when a person feels almost totally well, and after sometimes years of inactivity, may overdo it suddenly and become ill again. Human nature is such that when feeling well, there will always be a tendency to "get stuck in", take on extra commitments or catch up on all the tasks left undone. The CFS person is therefore treading a fine line and can easily risk relapse.

The relapses often occur quite unexpectedly and with no apparent cause, but often on careful consideration it is possible to see where the risk occurred and why the relapse followed. It really is a matter of vigilance and never overstepping the mark. It is like being on a knife edge and it is easy to fall off. Life can become very boring if a person never takes risks, and in CFS these risks may lead to temporary relapse, but sometimes it is worth it, and the price to pay maybe quite small if that person has structured their life to cope with the times of relapse. It is really a matter of learning how much one could or should do, keeping within the boundaries as far as possible and knowing how best to handle the difficult periods. Most with CFS learn to gradually restructure life to fit the illness and cope well. Sometimes those around them do not cope so well!

The commonest causes of relapse will be stress and/or overexercise. Life is full of stress and would be boring without it, but it is a matter of recognizing it and handling it well. There are plenty of helpful books, tapes, counsellors etc to advise about stress management, but much of it we already know. We just don't apply it. Acknowledging that stress aggravates any illness is the first essential, and then recognizing the individual stressors in one's own life, and being on the lookout for them. Some stresses can be dealt with and worked through, it may be possible to walk away from others, but it is the bottled up stress that causes trouble healthwise. Professional help is certainly sometimes needed and learning some good relaxation strategies such as visualization or self hypnosis can be helpful. Some stresses hit us suddenly and unexpectedly, but if good stress management techniques have been learnt. the risk of relapse is minimized. For young people exams and school pressures are often hard to handle. so extra understanding and support will be needed at these times.

Physically overdoing it in any way can be a risk in CFS. Any physical activity needs to be undertaken gradually in a paced fashion, be it exercise, housework or one's job. Sports people find this illness particularly frustrating and risk of relapse particularly hard to cope with. There is an urge to get back on the sports field and back into harsh training. All too often a sportsperson may suddenly decide to go back onto the rugby field or tennis court far too soon. Fitness has been lost and there has been no preparation, and this sudden burst of activity can put a person back many months. The so called "crash and burn" approach just does not work.

Obviously one can physically overdo things in many other ways too, but with caution and pacing carefully. People often surprise themselves by finding they can in fact do more than they thought. So testing oneself out cautiously from time to time is sensible, but not to extremes. There are many stressors on the body, as well as exercise, which can be risk factors in this illness. Accidents, surgical operations, chemical exposure, binge drinking, late night parties all pose hazards. Toxic reactions to herbal products, mega vitamins also occur quite frequently.

There will also be times in life when a person is more at risk of relapse than at others. In women, times of hormonal change can be difficult to manage. Some women notice they always go downhill premenstrually or during menstruation if it is heavy. Many with CFS notice a worsening of symptoms at adolescence or menopause. Environment may change from time to time and different environments can create difficulty and changes need to be thought through carefully. (eg when moving house, holidays). Weather too may make health differences, and some will relapse in heat, others in cold or damp conditions. Other medical conditions can put one more at risk too, and so continual surveillance is important, particularly if new

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ME – my story

By Mary Gibson.

In 1942 I joined the Australian Army as a Nursing Sister. We had to be aged 25 if we wanted to go overseas. We eventually boarded the Queen Elizabeth and arrived in the Middle East. I was posted to the 2/2 AGH (Australian General Hospital) at El Kantara on the Suez Canal. After about two months I became quite sick with rubella and dysentery. I knew the doctors were wrong as I had been nursing children with rubella before I joined the army and caught it, too. Also, the Lab was closed as we had packed up the hospital to come home as the Japanese had declared war on Australia.

Over the years. I became more tired and by 1956 I discovered food allergies, particularly diarrhoea or aching cheeks or teeth after eating diary products, citrus, tomatoes etc. I heard a Dr. Richard Mackarness talking about food allergies on the radio and he was a top London psychiatrist. He had written a book called "Not all in the Mind" It was such a relief to know I was not imagining that diarrhoea was due to certain foods. So I started to avoid certain foods and felt much better.

Also I had frequently been in and out of the local hospital with diarrhoea and vomiting.

Every time influenza hit the community I would get very ill with it. My blood pressure and haemoglobin were always very low and the doctor would be very worried.

I tried many doctors and eventually found a doctor who helped me with my food allergies. An allergy specialist tested me and told me all I was allergic to were nuts. He was so wrong as I would get diarrhoea.

I didn't get much help until 2001 I found a Professor who sent me to a Pathologist who diagnosed Rickettsia Conorii (Mediterranean Tick Fever).

Veterans' Affairs said that it was easily cured with tetracyclines quite forgetting that they were invented 12 year after I was so ill in 1942 on the Suez Canal which is why mine is chronic

I have been on certain antibiotics and many vitamins since 2001 and am better than I was.

Age has caught up with me and I am now 91. Some weeks ago I thought I had a heart attack as my heart was racing. It was butter, and most drinks do it too, so I only drink rain or filtered water.

Certain chemicals, particularly formaldehyde, affect me. I am on oroxine and iodine and all help

My husband was a Japanese prisoner of war and we still live in our own home on the farm. We have been married 60 years and he is the same age. He has always been very supportive. Our son and daughter and spouses live near and keep and keep an eye on us.

1

Continued from page 12

symptoms develop.

Good sleep is crucial in the plan to avoid relapse. Many will relapse following sleep disruption. This maybe associated with environmental change (such as temperature), stress, shift work, wakeful baby, timezone change etc. Again many of these things can be predicted and planned for. Sleep medication will be needed for most people with CFS, and stopping medication suddenly because of running out or thinking one doesn't need it any more, can be pose major risks.

Understanding the illness and living within one's ability will give one the best chance of minimizing relapse. But if a relapse does occur, giving in to it is essential by treating oneself kindly and not feeling guilty to accept help or to take time out. We all need to learn not to take on too much and to know we do have the choice to use the most positive word in the English language: "NO".

1

Reprinted with permission from Dr Vallings. Previously published in Meeting Place*, Issue 86 December 2006.*

The Spoon Theory

By Christine Miserandino.

Editor's Note: Although this story relates specifically to Lupus, it is an excellent guide on how to pace oneself for any chronic illness, including ours.

My best friend and I were in the diner talking. As usual it was very late and we were eating French fries with gravy. Like normal girls our age we spent a lot of time in the diner while in college and most of the time we spent talking about boys, music or trivial things that seemed very important at the time. We never got serious about anything in particular and spent most of our time laughing.

When I went to take some of my medicine with a snack, as I usually did, she watched me with an awkward kind of stare instead of continuing the conversation. She then asked me out of the blue what it felt like to have Lupus and be sick. I was shocked, not only because she asked me the random question, but also because I assumed she knew all there was to know about Lupus. She came to doctors with me, she saw me walk with a cane and throw up in the bathroom. She had seen me cry in pain – what else was there to know?

I started to ramble on about pills and aches and pains, but she kept pursuing and didn't seem satisfied with my answers. I was a little surprised as. being my roommate in college and friend for years; I thought she already knew the medical definition of Lupus. Then she looked at me with a face that every sick person knows well – the face of curiosity about something no-one healthy can truly understand. She asked what it felt like, not physically, but what it felt like to be me, to be sick.

As I tried to gain my composure I glanced around the table for help or guidance, or at least to stall for time to think. I was trying to find the right words. How do I answer a question I was never able to answer for myself? How do I explain every detail of every day being affected and give the emotions a sick person goes through with clarity? I could have given up, cracked a joke like I usually do and changed the subject, but I remember thinking if I don't try to explain this, how could I ever expect her to understand? If I can't explain this to my best friend, how could I explain my world to anyone else? I had at least to try.

At that moment The Spoon Theory was born. I quickly grabbed every spoon on the table – hell, I grabbed spoons off the other tables. I looked at her in the eyes and said, "Here you go, you have Lupus." She looked at me slightly confused, as anyone would when they are handed a bouquet of spoons. The cold metal spoons clanked in my hands as I grouped them together and shoved them into her hands.

I explained that the difference between being sick and being healthy is having to make choices or to consciously think about things when the rest of the world doesn't have to. The healthy have the luxury of life without choices – a gift most people take for granted.

Most people start the day with an unlimited amount of possibilities and energy to do whatever they desire, especially young people. For the most part they do not need to worry about the effects of their actions. So for my explanation I used spoons to convey this point. I wanted something for her to actually hold, for me to then take away, since most people who get sick feel a 'loss' of a life they once knew. If I was in control of taking away the spoons, then she would know what it feels like to have someone or something else, in this case Lupus, being in control.

She grabbed the spoons with excitement. She didn't understand what I was doing, but she is always up for a good time, so I guess she thought I was cracking a joke of some kind like I usually do when talking about touchy topics. Little did she know how serious I would become.

I asked her to count her spoons. She asked why and I explained that when you are healthy you expect to have a never-ending supply of spoons. But when you have to now plan your day you need to know exactly how many spoons you are starting with. It doesn't guarantee that you might lose some along the way, but at least it helps to know where you are starting.

She counted out twelve spoons. She laughed and said she wanted more. I said, "No," and knew right away that this little game would work when she looked disappointed and we hadn't even started yet. I've wanted more spoons for years and haven't found a way to get more, why should she? I also told her to always be conscious of how many she had and not to drop them because she can never forget she has Lupus.

I asked her to list off the tasks of her day, including the most simple. As she rattled off daily chores or

just fun things to do, I explained how each one would cost her a spoon. When she jumped right into getting ready for work as her first task of the morning, I cut her off and took away a spoon. I practically jumped down her throat. I said "No! You don't just get up. You have to crack open your eyes and then realise you are late. You didn't sleep well the night before. You have to crawl out of bed and then you have to make yourself something to eat before you can do anything else because, if you don't, you can't take your medicine and if you don't take your medicine you might as well give up all your spoons for today and tomorrow too."

I quickly took away a spoon and she realised she hadn't even dressed yet. Showering cost her a spoon, just for washing her hair and shaving her legs. Reaching high and low that early in the morning could actually cost more than one spoon, but I figured I would give her a break. Getting dressed was worth another spoon. I stopped her and broke down every little task to show her how every little detail needs to be thought about. You cannot just simply throw your clothes on when you are sick. I explained that I have to see what clothes I can physically put on; if my hands hurt that day buttons are out of the question. If I have bruises that day I need to wear long sleeves and if I have a fever I need a sweater to stay warm and so on. If my hair is falling out I need to spend more time to look presentable and then you need to factor in another five minutes for feeling badly that it took you two hours to do all this.

I think she was starting to understand when she theoretically didn't even get to work and she was left with six spoons. I then explained to her that she needed to choose the rest of her day wisely since when your spoons are gone they are gone. Sometimes you can borrow against tomorrow's spoons, but just think how hard tomorrow will be with less spoons. I also needed to explain that a person who is sick always lives with the looming thought that tomorrow may be the day a cold comes, or an infection, or any number of things that could be very dangerous. So you don't want to run low on spoons because you never know when you truly will need them. I didn't want to depress her but I needed to be realistic and, unfortunately, being prepared for the worst is part of a real day for me.

We went through the rest of the day and she slowly learnt that skipping lunch would cost her a spoon, as well as standing on a train or typing on her computer for too long. She was forced to make choices and think about things differently. Hypothetically she had to choose not to run errands so that she could eat dinner that night.

When we got to the end of her pretend day she said she was hungry. I summarised that she had to eat dinner but she had only one spoon left. If she cooked she wouldn't have enough energy to clean the pots. If she went out to dinner she might be too tired to drive home safely. Then I also explained that I didn't even bother to add into this game that she was so nauseous, cooking was probably out of the question anyway. So she decided to make soup; it was easy. I then said it was only 7pm, you have the rest of the night but maybe end up with one spoon so you can do something fun or clean your apartment or do chores, but you can't do it all.

I rarely see her emotional so when I saw her upset I knew maybe I was getting though to her. I didn't want my friend to be upset but at the same time I was happy to think, finally, maybe someone understood me a little bit. She had tears in her eyes and asked quietly, "Christine,. How do you do it? Do you really do this every day?" I explained that some days were worse than others; some days I have more spoons than most. But I can never make it go away and I can't forget about it; I always have to think about it. I handed her a spoon I had been holding in reserve. I said simply, "I have learnt to live life with an extra spoon in my pocket in reserve. You need to always be prepared."

It's hard. The hardest thing I ever had to learn is to slow down and not do everything. I fight this to this day. I hate feeling left out, having to choose to stay at home or to not get things done that I want to. I wanted her to feel that frustration. I wanted her to understand that everything everyone else does comes so easily but for me it is one hundred little jobs in one. I need to think about the weather, the temperature that day and the whole day's plans before I can attack any one given thing. When other people can simply do things, I have to attack it and make a plan like I am strategising a war. It is in that lifestyle — the difference between being sick and healthy. It is the beautiful ability to not think and just do — I miss that freedom. I miss never having to count spoons.

After we were emotional and talked about this for a little while longer, I sensed she was sad. Maybe she finally understood. Maybe she realised that she never could truly and honestly say she understands. But at least now she might not complain so much when I can't go out for dinner some nights, or when I never seem to make it to her house and she always has to drive to mine. I gave her a long hug when we walked

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Managing stress and inflammation

Here are some basic practices you can follow to reduce the impact of stress on your immune system and to inhibit inflammation.

Shift the balance of fatty acids in your diet

Certain fatty acids in foods are less easily converted into the chemical components that aid inflammation. Omega-3 fatty acids (from fish and certain plants) help prevent inflammation while omega-6 fatty acids (from meat and dairy) bolster inflammatory responses. Switching from peanut and vegetable oils to canola and soybean oils, and eating more fish like tuna, mackeral, trout and salmon increases your omega-3 levels. Eating fewer beef and dairy products and giving preference to grass-fed animals rather than corn-fed ones can favourably reduce your levels of omega-6.

Increase your intake of antioxidants and natural anti-inflammatories

Oxidative stress can trigger the production of proinflammatory cytokines like interleuken-6. Antioxidants from green and black teas, oolong tea, garlic and fresh fruits may help. Vitamin D and the supplement turmeric have also been shown to have anti-inflammatory properties. But, of course, use of supplements and vitamins should be monitored closely for adverse reactions.

Bring laughter into your life

Sometimes laughter really is the best medicine. At the biophysical level, the convulsive motion of laughter moves lymph fluids through your body, helping your immune system clear toxins. Laughter also oxygenates the blood and increases circulation. Biochemically, it releases a host of healing agents. So consider bringing humour into your environment and embracing opportunities to laugh.

Other things to try

Other methods for boosting your immune system and reducing inflammation include: adequate sleep; daily stretching; gentle movement; and drinking plenty of clear fluids. Recent studies have also shown objective immune-enhancing effects from relaxation massage. 1

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out of the diner. I had the one spoon in my hand and I said, “Don’t worry. I see this as a blessing. I have been forced to think about everything I do. Do you know how many spoons people waste every day? I don’t have room for wasted time or wasted spoons and I chose to spend this time with you.”

Ever since this night I have used The Spoon Theory to explain my life to many people. In fact, my family and friends refer to spoons all the time. It has been a code word for what I can and cannot do. Once people understand the spoon theory they seem to understand me better, but I also think they live their lives a little differently too. I think it isn’t just good for understanding Lupus, but for anyone dealing with any disability or illness. Hopefully they don’t take so much for granted or their life in general. I gibe a piece of myself, in every sense of the word, when I do anything. It has become an inside joke. I have become famous for saying to people jokingly that they should feel special when I spend time with them because they have one of my spoons. 1

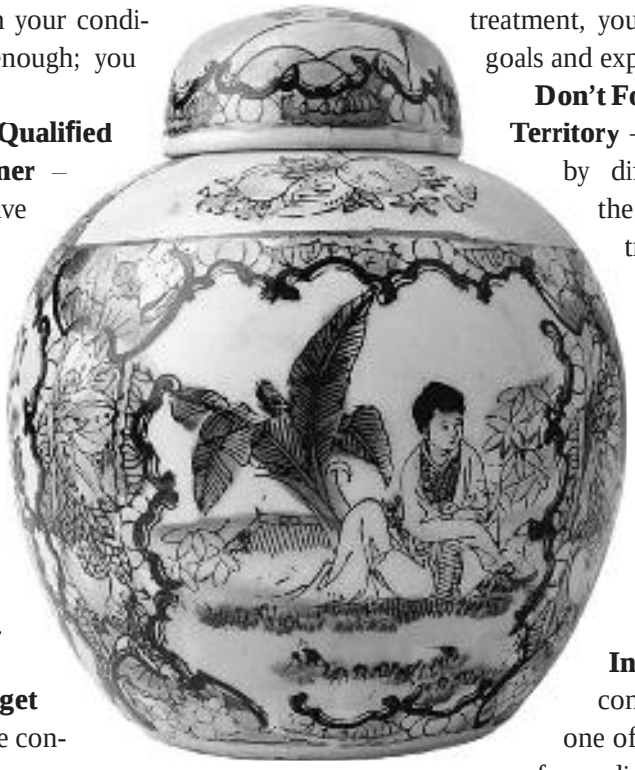
Alternative medicine – a sensible approach

By Mary Campbell.

The field of alternative medicine is large, varied, and often very confusing. People with ME/CFS/FMS can find themselves confronted with unfamiliar concepts and strange treatments; navigating a course through this maze is not easy. To help make this journey as smooth as possible below are some points to consider.

Remain Positive – Always maintain a positive attitude, firm in the belief that you will eventually recover from CFS, regardless of what treatments you try.

Do Yourself No Harm – Avoid treatments which you think have the potential to harm you, or which do, in fact, worsen your condition. Having CFS is bad enough; you don't need extra problems.



Make Sure You See a Qualified and Registered Practitioner – The practitioner should have a qualification. Their education, additional training, licenses and certifications provide insight into their approach to treatment and patients. Check with the Complementary Medicine Associations for registered practitioners in your area: www.atms.com.au, www.anta.com.au, www.cma.asn.au.

Stay Within Your Budget – Alternative therapies, unlike conventional medicine are not government subsidised and therefore can be very expensive. Allocate an amount of money you are prepared to spend and stay within this budget. Over-expenditure can cause personal and family stress and is a burden that is especially heavy in a time of ill–health when a normal level of income may not be available to you.

Be Prepared – Familiarise yourself with treatment procedures, demands, impacts and effects. Have a list of questions to ask the therapist before committing to treatment. Make sure you are comfortable with the answers – if you are unsure of treatment, don't do it!

Have an Open Mind and Be Patient – Once you have decided to try a particular therapy, have an open mind about what the results are likely to be: don't believe the treatment won't work – let the results speak for themselves. Be prepared to be patient, alternative medicine is not about 'quick fixes'. It takes time, personal effort and commitment.

Communicate – Remember alternative/complementary medicine goes hand in hand with conventional medicine. Both your GP and other practitioners need to be kept up to date with any alternative treatments and vice versa. Provide your therapist with information such as blood test results, current treatment, your medical history and your goals and expectations for treatment.

Don't Forget You are in Uncharted Territory – Many claims will be made by different practitioners about the cause(s) of CFS and about treatments. Remember that no objective evidence has ever been produced to support any of these claims. If there was an effective treatment for CFS, we would soon know about it. A healthy dose of scepticism will help you keep things in perspective.

Be Aware of Conflict of Interest – There is potential conflict of interest when someone offering an untested treatment for a disease also earns his/her income from providing that treatment. In other words, the person offering the treatment may consciously or unconsciously make exaggerated or false claims about the treatment in order to induce you to spend money on the treatment. Being aware of this will also help you to keep things in perspective.

Remember, Time is the Best Healer – Most people with CFS make substantial improvement over time and recover completely from this illness. Time and good illness management are usually the major factors in recovery, rather than which treatment you tried.

Men with CFS

By Pamela Young.

Don Sprayberry is 58 years old, has been married for 36 years and was a senior loan officer at a local credit union before chronic fatigue syndrome (CFS) stopped him cold in his tracks. He went to work one morning feeling perfectly healthy, but before lunch became very ill with what seemed like flu. For the next two weeks he took medicine and tried to work but his health further deteriorated. After months of testing by many doctors, he was eventually diagnosed with CFS. That was 13 years ago and he hasn't been able to work since.

Sprayberry's story demonstrates similarities and diversity in the CFS experience. Though the way CFS entered and altered his life is not uncommon to the more than one million Americans currently suffering with the illness, the fact that he's a man puts him in the minority of the CFS community. In fact, some would say that men with CFS constitute an under-represented segment of an illness that is itself often overlooked by the media and the Centre for Disease and Control and Prevention (CDC) estimates that four times as many women have CFS as men. That suggests that CFS affects at least 200,000 men in the US. Yet many of the articles on CFS appear in women-oriented magazines, the majority of CFS support group members and leaders are women and even the National Institute for Health (NIH) trans-departmental working group on CFS is sponsored by the NIH Office of Research on Women's Health.

What is that like for the men who have this illness? How do their issues mesh with or vary from their female counterparts? In short, how does CFS affect the lives of men who have it?

Losing identity

Like it or not, society still moulds strong concepts of what a man is supposed to be and do in this world.

Provider, protector and competitor are still roles often ingrained in a man's sense of self. When CFS strikes, everything changes and many of the traditional measurements of those qualities crumble. Perhaps that is why, when asked to describe how CFS affects them, many men begin, not with the burdensome symptoms they experience, but with how their roles in life have changed.

Loss of work and career status is often one of the first things mentioned by men with CFS when describing their experiences. Says Matt Matherne, a Gulf War Veteran, "There's a sense of feeling worthless because you feel like you've let your wife and family down. If I didn't have CFS I would probably still be in the military supporting my country and my family."

Sprayberry echoes this sentiment, saying, "I think the most difficult thing for a man is not being able to earn a living. You feel you aren't taking care of your family like you should."

Even for men who continue to work, the career and financial impact weighs heavily. Patrick Venetucci, a corporate executive with a young family was an international businessman working in Tokyo when he became ill. He has since moved back to the US, curtailed his work travels, changed career paths and dramatically reduced his working hours. He says, "Economically, CFS puts more pressure on the house, especially when the man is the primary or sole breadwinner. All of a sudden the household income stream is at risk."

CFS often undermines a man's family and social identity as well. Says Matherne, "All the things my wife depended on me to do are mostly no longer possible. It eats away at me". Venetucci agrees, "I did so much more for my wife before I was sick. Now the burden is on her."

Venetucci also describes his changed role as father. "I have two small children. Right now, I can play with them lying down and they don't realise any difference. But I have to refrain from going to birthday parties and social outings with them to preserve my energy. I worry very much about the future and all the things I won't be able to participate in."

Additionally, many men construct their social network around business, athletics and other physical activities. No longer able to participate in these activities as they once did, their social supports dry up.

In a world where a man often defines who he is by what he does, this erosion of career, home life and social identity leaves many men with CFS experiencing a profound loss of self – further compounded by struggling with a disease that's considered primarily a women's illness.

And these identity struggles aren't just internal. Other people don't often understand or accept the fact that a man could be so disabled by this illness, especially when there are few outward signs of the physical devastation CFS wreaks on men and women alike. The outside world expects men – sometimes

more so than women – to 'suck it up' and get going. Sprayberry says, "I'm six feet four inches tall and weigh 230 pounds. Some people look at me and think I can't be that ill because I look so strong." Other men describe struggling with the same misguided expectations from others (and themselves).

Finding help

Because it's less common to associate CFS with males, many men with the illness are slow to seek medical help as symptoms emerge. They first try to explain away the fatigue or pain. As a result, they may enter an extended period of pushing themselves in a way that further compromises their health. It often isn't until symptoms are severe enough to interfere seriously with career and personal activities that a man may bring it up with his doctor. And then, many doctors are slow to consider CFS. This delays the opportunity to begin treating and managing the illness.

Dr Nancy Klimas, a respected medical authority on CFS, sums it up this way, "Men will deny they're ill longer than many women and not seek treatment until they're unable to do much of anything." She thinks that may help explain why clinic-based CFS studies show a fewer number of men with the illness, because fewer men are seeking help for their CFS.

Once men are diagnosed with CFS, some express having trouble identifying with the information they find on the illness because it so often speaks from a female perspective or is covered in predominantly women-oriented media. As Sprayberry explains it, "Very little is ever mentioned about men and CFS and it can make you have doubts about your experience." Venetucci describes feeling like 'a mutant among mutants' reading about an illness that's marginalised to begin with and then not seeing any male experiences reflected in the media.

For Andrew Mosmiller, a 20-year old Maryland man, seeking help from established CFS support groups has proved challenging as well. He says, "My method of coping seems different from most of the support group members I've encountered, which places me on the outside of such circles in many ways."

This experience appears to be reflected by other men with CFS as they attempt to take advantage of support group networks which often consist largely of women. It suggests that the nature of support for a man may be somewhat different than it is for a woman. Data shows that many women utilise supports groups to address the significant emotional strain and personal isolation brought on by CFS. Yet many men are more interested in exchanging experiences about ways to function better. The two desires aren't mutually exclusive, but they tend to align poorly when seeking and receiving support.

Mosmiller explains, "The group in my area is very caring and responsible, but when I post a comment about having trouble with a specific situation in my life, I'm looking for support that includes some practical suggestions. What I often get instead is lots of compassion but few recommendations. I appreciate this response, but I'm still left wondering what I can do."

While a handful of CFS information lines and support groups are led by men and some have active male members, currently no US support groups or websites are principally geared towards men with this illness.

Clinically speaking

It's common to hear stories from both genders about misdiagnosis and lack of assistance by the medical community. People are often sent from specialist to specialist and given a battery of tests – or worse, told there's nothing wrong with them – before finally reaching a diagnosis of CFS. With men, this problem again is compounded by the perception of CFS as a women's illness. Doctors may not initially think to explore the CFS diagnostic criteria for their male patients. Anecdotal feedback also suggests that physicians may not be as responsive to men's complaints of fatigue, though evidence suggests that gender bias may exist in a man's favour when it comes to specific pain.

Overall, though, the medical community doesn't benefit from significantly more or better information than the general public when it comes to men with CFS. Dr Lucinda Bateman, who has focused her practice on the diagnosis and care of CFS and fibromyalgia, explains, "We have a lack of information about CFS in general terms. Combine that with the fact that men make up a smaller percentage of patients diagnosed and that leaves us with very little knowledge on the subject." Kliman agrees about the shortage of gender-specific information. She says, "I'm surprised to see how little we know in this area."

So how do CFS-experienced physicians address the illness in their male patients and do they perceive any physiologic differences?

Bateman expresses seeing more similarities than differences. Klimas observes the most disabling features, like profound fatigue, exercise induced relapse

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The loneliness of the long-distance sufferer

In this article from the UK ME Association's ME Essential *magazine Pat Mathewson describes her 26-year experience of being an ME sufferer and the loneliness of such a long struggle with the illness.*

This is what it's like to have ME:

- Being too weak to wash or dress yourself.
- Being too weak to clean your own teeth.
- Being too weak to wash and dry your own hair so that your teenage son has to do it.
- Being too weak to do the cooking you love.
- Being too weak to chop a carrot.
- Being too weak to open a jar.
- It means listening to the laughter and chatter of your family in a distant room as they try to enjoy Christmas without you – as you lie in a darkened room wishing you could fall asleep and not wake up. You can't be with them because you can't sit up because every light is too bright, every sound too loud and every smell too strong.
- It means being afraid to move knowing that you will have to get up for a pee and that, when you do, you have to start the resting process all over again.
- It means being constipated for days because you haven't got the strength to push and you get exhausted with the effort.
- Having ME means being lonely and isolated. It means you can't read a book or watch TV or listen to the radio.
- It means that your friends can't visit or phone you.
- It means you can't write letters to them.
- It means feeling cut off.
- It means being in bed for so long you forget what the kitchen is like and all your pot plants are dead.
- It means, as you recover from a relapse, that your first trip out in a car is frightening as the speed feels too fast, even though it is only 20mph.
- It means that summer has gone since you were last outside and the leaves are falling from the trees.
- It means living through the embarrassment of using a wheelchair to have any life at all.

- It means hearing a 'friend' say, "Oh for Christ sake, get up and walk", or, to your husband. "Give her a good kick up the ass. That'll get her going."
- It means feeling hopeless as you hear pundits say 'it's all in the mind' and yet again a newspaper has called it 'yuppie flu'. I'm no yuppie and ME is NOTHING like flu.
- It means you can no longer help your family and friends as much as you would wish.
- It means that you look as if you're just lazy as you no longer jump up to help with the washing up.
- It means watching everyone dancing at your daughter's wedding while your heart, body and soul ache to join in but you have to hide the pain.
- It means endless longing to swim in the clear blue sea.
- It means having so much wanting, longing and yearning inside of you that you feel it must be visible to those around you. But no, they don't see it. They see you trying to be normal, putting a brave face on – because who wants to be with an old misery guts?
- It means refusing alcohol when old friends say, "Go on – one won't hurt you!"
- It means watching your friends grow while you stand still. You hear about their jobs, their latest exercise. "You should try it. A little bit of gentle exercise is what you need."
- It means feeling miserably cold because you can't move about and get warm.
- It means being overweight because you can't exercise.
- It means, whatever reason for going to hospital, if you mention ME you will encounter off-hand, impatient nurses having a go at you because they think you are a whinger. You lie there in despair because you don't have the strength to stand up for yourself.

- It means little grandson grabbing your hand saying, "Come on Granny." He doesn't know that you can't run after him.
- It means not being able to run up and hug someone you love.
- It means clinging on, as hard as you can, to all the skills that make up the person you are – only to find that, one by one, you have to give them up. You wonder who you are and what you stand for.
- It means anger, rage, disappointment, tears and more tears.
- It means clinging on to a fragile thread of hope that one day, some day, someone will find a reason for the ME and a cure.

But most of all the wishing and hoping comes down to needing understanding, knowing full well that it's a lost hope and people will STILL say, "Oh, we all feel tired!"

You try every crackpot remedy just because 'other people' want to see you make an effort. "Come on at least try it. You've got nothing to lose." Only hope… money…dignity…time…energy.

And then you are faced with failure yet again and someone is sure to say that you haven't tried it long enough or that it didn't work because you are 'blocking.' Blocking? Why would you block recovery when every fibre of your being aches to be the fit and well person you used to be?

The one who grew food and flowers, baked her own cakes and bread, delighted in cookbooks and new recipes, knitted for England, made her own and her children's clothes, walked everywhere – no school run for me; the one who thought she could change the world, joined CND, campaigned for Nicaragua, wrote letters for Amnesty and Survival and even now protests against the Iraqi war, wants social justice and fair trade in the world.

I was 34 when ME changed my life and made me a prisoner in my own body.

This year I will be 60. It's a long time to wake up every morning and feel the pain that will be with me all day and every day. It's a long time to feel such utter frustration that I feel that I'm going to explode with it and the letters of that word will float in the air for all to see. 1

Reprinted with permission from ME Essential*, quarterly magazine of the ME Association, UK, issue 97 (January 2006).*

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and cognitive dysfunction, as being shared by both genders. Dr Charles Lapp, a CFS pioneer, describes the clinical approach to men and women with CFS as being "very much the same".

But without drawing broader references, all three physicians have observed occasional differences. For example, Bateman reports that some of her most challenging cases of severe, widespread, postviral pain have been in men with CFS. Klimas has noted some gender differences in response to certain medications.

Prostatitis, an infection of the prostate gland, has also been observed in men with CFS and may parallel the recurrent urinary infections report by women with the illness.

Because of the neuroendocrine effects of CFS, all three doctors describe looking closely at the hormone levels in both groups. Says Lapp, "Many men with CFS develop low testosterone levels.. This is frequently overlooked by the primary care doctor, but replacement can improve libido, energy and quality of life."

The essence of a man

CFS drastically changes lives. That much is clear for both men and women. The panoply of symptoms, the isolation, diminished roles, frustration with the medical community and hope for improved treatments and a cure are all characteristics of the CFS experience. Yet for men, these dynamics may exact a different type of toll.

When asked to convey to the world what it's like to be a man with CFS, Mosmiller offers a perspective many men with the illness seem to share. He says, "It's a constant exercise in dealing with frustration. This is probably true for women as well, but men with CFS have to adjust to things that go very much against the grain of what a man thinks and feels he should be. It's as if it takes away some of the essence of what it is to be a man." 1

This article is reprinted from the CFIDS Chronicle*, Fall 2006. Pamela Young is Director of Publications for the CFIDS Association of America.*

IACFS Professional Conference report

The 8th International IACFS Conference on Chronic Fatigue Syndrome, Fibromyalgia, and Other Related Illnesses was held in Florida on 10-14 January 2007. It was hosted by P.A.N.D.O.R.A., the Patient Alliance for Neuroendocrineimmune Disorders Organization for Research and Advocacy.

The Conference was divided into two sections: one for Patients; and one for Professionals.

The following, by Cort Johnson, is the first part of the report on the Professional Conference. The report dedicated to the Patient Conference appeared in the previous issue of Talking Point *(2007 Issue 1).*

PART I: The Cardiovascular and Exercise Studies and Fatigue Overview

These overviews do not follow the conferences agenda (fatigue, pain, gender, sleep, etc.). Several of those sessions were undersubscribed and had papers on different subjects shoehorned in to fill them out. In order to obtain a more orderly presentation some new sections (Cardiovascular/Vascular, Exercise and CFS) are added in this overview while others are retained (Brain, Immune, etc.). Papers that I found most interesting are highlighted. Some overviews are found under more than one category.

The conference began on a high note with an overview on the state of fatigue science in Japan.

CONFERENCE HIGHLIGHT

A Model of CFS Emerges in Japan

Yasuyoshi Watanabe:
Introductory Overview to the Fatigue Section

After a short introduction by Nancy Klimas, the conference was started off by Dr. Watanabe, a member of the Japanese delegation. Dr. Klimas has said that the Japanese government is spending more on CFS right now than the U.S. Not only are the Japanese doing a great deal of work on CFS they are doing it in a cohesive and integrated manner.

The Japanese are not studying CFS out of the kindness of their hearts. The Japanese government has come to realize that chronic fatigue poses severe economic costs (>10 billion dollars/year) and that it is in their interest to resolve it. This has lead several government ministries to fund several large scale research projects including *"The molecular/neural mechanisms of fatigue and the fatigue sensation and the way to overcome chronic fatigue"* from 1999-2005 and another project from 2004-2009. They have three chronic fatigue research centers and are supported by laboratories in 26 universities and institutions. Among other things they are trying to find and/or develop anti-fatiguing foods and drugs.

The Japanese have been mostly focused on the central nervous system (CNS). Dr. Watanabe stated that lesions in several parts of the frontal cortex (Broadman's Area (BA) 10,11) may alter the operation of our sensory system and effect motivation in CFS patients.

The neuroendoimmune dysfunction in CFS appears to be most related to the abnormal activity of five substances two neurotransmitters – glutamate and serotonin, acetylcarnitine, transforming growth factor-b and interferon. Abnormal activation of the serotonergic system in the *anterior cingulate* appears to be particularly important. Dr. Watanabe believes problems in the prefrontal cortex and anterior cingulate play a key role in CFS. *We will see the anterior cingulate come up several times in the conference.*

The Japanese attempt to develop chronic fatigue animal models underscores how intensely they are studying chronic fatigue. *They are the kind of basic research study one would have thought would have been done long ago in a disease characterized by post-exertional fatigue!* They have been talked about for years in the U.S. These animal studies, which assess just what happens when organisms become fatigued, appear to be a kind of backdoor

way of getting at CFS.

The Japanese have found that fatigued animals exhibit reduced glucose uptake in the forebrain, mitochondrial dysfunction, cytokine upregulation (IFN, TGF-B and ?), changes in gene expression and serotonergic dysfunction. All of these have been found in CFS.

These researchers also measured about 100 biochemical factors and were able to use them to develop an "index of exhaustion". This included reduced glucose uptake in the brain, reduction of the dopaminergic fibers in the brain, degeneration of the pituitary melanotrophs, and Dopamine is an intermediate in tyrosine metabolism and precursor of norepinephrine and epinephrine (noradrenaline/adrenaline). Dopamine plays an especially important role in the basal ganglia; a part of the brain that has long been of interest in CFS. Norepinephrine/epinephrine are the central neurotransmitters of the sympathetic nervous system and play a central role in the stress response – another area of great interest. Pituitary melanotrophs produce melantropin, a hormone.

The reduction of dopaminergic nerve fibers in fatigued animals suggests that fatigue is associated with problems with basal ganglia and sympathetic nervous system functioning. There is evidence for both in CFS. But what might cause the brains of CFS patients to resemble those of exhausted laboratory rats? Dr. Watanabe will attempt to answer that question in his summary of the brain section.

They also had healthy volunteers undergo four hour exercise sessions and looked for biomarkers of fatigue. They noted that when fatigue reaches a certain state that motivation drops. They were able to relate decreased levels of the alanine amino acid to physical fatigue and reduced levels of the branched complex chain amino acids and tyrosine to mental fatigue. Tryptophan losses were correlated with a kind of complex fatigue associated with both physical and mental fatigue. *Tryptophan is a precursor of serotonin. Researchers have suspected it is involved in fatigue for some time and some evidence suggests tryptophan levels are altered in CFS.*

They have found, interestingly, that an aromatherapy substance called 'green odor' activated the anterior cingulate in the brain and seemed to relieve symptoms of fatigue. This was quite a presentation and a great way to start the conference. We will hear more from Dr. Kuratsune and the other Japanese researchers later.

CARDIOVASCULAR/VASCULAR SYSTEM

More and more studies suggest there is something the matter with the circulatory systems of CFS patients. There's evidence of low blood flows to the brain and muscles, abnormal skin blood flows, low blood volume and others. Dr. Hyde suggested many years ago that CFS is a kind of vasculitis (a disease afflicting the vascular system.) Several studies at this conference suggest that he may, at least in part, be right.

Dr. Spence's talk on arterial stiffness and inflammation in CFS was shoehorned into the pain section. Unfortunately Dr. Spence was only given half his allotted time (7½ minutes) to give a talk that was for me the highlight of the conference.

CONFERENCE HIGHLIGHT

Connecting the Dots – A Central Paper in CFS?

Vance Spence, F. Khan, G. Kennedy, C. Underwood and J. Belch:
Inflammation and arterial stiffness in patients with chronic fatigue syndrome

This talk actually raised the hairs on the back of my neck. Dr. Spence's interest in arterial stiffness was prompted by a single finding in an otherwise largely unnoticed paper that concluded that connective tissue problems are not present in adolescent CFS. This team did, however, find greatly increased arterial stiffness, an abnormality it had no explanation for. Dr. Spence said that this caught his attention – arterial stiffness is rarely found in adolescents and certainly not to this extent; these young CFS patients had higher levels of arterial stiffness than diabetes patients do.

Dr. Spence's study looked at several inflammatory factors (cholesterol, free radical by-products, c-reactive protein) that are often

associated with arterial stiffness. It found abnormally high levels of free radical by-products and C-reactive protein in CFS patients but not controls, as well as arterial stiffness that was increased, if my notes are right, by about 50% – an enormous amount. A further statistical analysis indicated that C-reactive protein was significantly correlated with increased arterial stiffness.

But how to explain this unusual finding? Dr. Spence noted that none of the usual suspects involved in arterial stiffness (increased age, diabetes, arteriosclerosis, etc.) could account for it in these CFS patients. So what could be stiffening the arteries of CFS patients? When he suggested neutrophil elastase my mouth gaped open. Elastase is a central factor in Dr. De Meirleir's RNase L paradigm and that he often finds it elevated in his patients. Dr. Baraniuk's fluid cerebrospinal proteome study suggests elastase is implicated in blood vessel problems in the brains of CFS patients. When Dr. Spence indicated that logical consequences of increased arterial stiffness are exercise intolerance and diastolic dysfunction the hairs stood up on the back of my neck. Talk about paradigms starting to meet up.

Elastase dissolves elastin, a substance which gives blood vessels, tendons and ligaments their elasticity. Monocytes, for instance, secrete elastase in order to allow them to travel through connective tissues and get at infections. A recent study found that increased serum elastase levels are associated with increased arterial stiffness (See Sources of Orthostatic Intolerance *for more on elastase).*

A Talk With Dr. Spence

This paper was the result of an alert researcher picking up on an obscure finding and putting the pieces together. I was able to talk to Dr. Spence later. The founder of the MERGE research/advocacy group, he is an enormously engaging and enthusiastic researcher with a severe case of CFS. If my notes are correct, he believes that the circulatory problems seen in CFS – which MERGE has been studying for some time now – may originate in a dysfunction of the endothelial cells lining the blood vessels. These cells are not only involved in opening and closing the blood vessels but in the immune response as well, and they are often attacked by pathogens.

Funding, funding, funding: MERGE has been able to turn out several small studies a year and I asked him about his funding. He said it was all from CFS patients giving what they could. He noted that MERGE is trying to fund seed studies that major funders will pick up on and expand.

Funding is, of course, a big problem for CFS researchers. Dr. Evengaard several times during the conference noted the almost non-existent rate of governmental funding of non-behaviorally oriented CFS research in Europe. She said that (aside from Japan) the U.S. is the only country willing to fund biologically oriented research studies. I asked Dr. Hanna about the possibility of the NIH funding studies outside the U.S. and she said there was no problem with that, that she is simply looking for multidisciplinary studies that seek to explain, not simply characterize CFS. I asked both Dr. Spence and Dr. Kerr if they were considering attempting to secure funding through the NIH. They both were but the process is long and demanding and Dr. Spence, as mentioned earlier, has a severe case of CFS. These creative researchers desperately need more money.

CONFERENCE HIGHLIGHT

Explaining CFS?

Tae Park:
Decreased renal function in CFS patients (poster)

In his poster Dr Park states that he believes that the unexplained bright spots ('unidentified bright objects') sometimes found in the MRI's of CFS patients brains are evidence of an 'arteriolar vasculopathy' or a blood vessel disease. Specifically Dr. Park believes that CFS is not a *'systemic micro-vascular inflammatory process'* – a process that would affect not only the brain or the heart or the muscles but potentially every organ system in the body. Dr. Park put his money where his mouth is by looking at blood flows in a different organ – the kidneys. He found that all of the CFS patients in this small study demonstrated markedly reduced renal blood flows (40-60% of norms). Dr. Park believes that the problems with medication intolerance/toxicity in CFS relate to poor drug clearance by the kidneys.

He is not alone in his interest in the microvasculature. Both the MERGE research team and Dr. Stewart have found evidence of microvasculature problems in CFS. Recent studies have found that women are more prone to microvascular problems in the heart than men and of course many more women than men have CFS. Is CFS a 'systemic micro-vascular inflammatory process'?

A Talk with Dr. Park: Dr. Park was gracious enough to explain his theory further in an e-mail. He has seen evidence of capillary inflammation and something called 'perivascular cuffing' in the autopies of CFS patients. *Perivascular cuffing refers to the accumulation of immune cells surrounding the blood vessels that have presumably been injured.* He notes that viruses such as HHV-6A and EBV are able to attack the endothelial cells lining the blood vessels. Then he stated something very interesting; that despite the low renal blood flows the typical CFS patient is not diabetic or hypertensive and shows no evidence of kidney disease. *Does this pattern occur elsewhere? We seem to have diastolic dysfunction without heart failure or heart enlargement and low muscle blood flows without metabolic dysfunction. Is CFS a disease in which chronically low microvascular blood flows impair organ functioning but rarely cause overt organ disease?* He believes the low renal blood flows he's finding reflect lowered brain and heart capillary blood flows as well. He noted that his treatment protocol incorporating IV gamma globulin resulted in both improved kidney blood blows and cognitive functioning. *Dr. Park's treatment protocol is discussed in the Clinical Trials overview.*

Dr. Park believes the increased rates of patent foramen ovale and diastolic dysfunction (see Dr. Cheney below) in CFS are caused by reduced capillary blood flows and upper airway obstructions that elevate the pressure in the pulmonary artery during sleep. Increased pulmonary artery pressure should elevate diastolic pressure and pop open the foramen ovale. In his sleep summary *Dr. Lappe stated he was intrigued by findings of upper airway resistance syndrome in some CFS patients. Dr. Cheney ascribes a different cause to elevated pulmonary artery pressures in CFS (see below). Dr. Park stated he has successfully treated heart problems in CFS using his protocol.*

ANOTHER Syndrome? Metabolic Syndrome and CFS

Elizabeth Maloney, DrPH, MS, James Jones, Chrisine Heim, Roumiana Boneva, William Reeves:
CFS is associated with high allostatic load in Georgia

This study extends the CDC work on allostatic stress in CFS. 'Allostatic load' is a measure of wear and tear on the body. The Wichita study showed that particularly with regard to cardiovascular factors CFS patients carry a high allostatic load. *In their Wichita studies the CDC took a broad brush approach to CFS research looking a wide variety of different factors. Intriguingly, given the recent findings from Vance Spence of MERGE and Dr. Cheney and others, cardiovascular factors jumped out.*

This study, which extended the CDC's look at these factors, found that both the CFS and CFS-like patients had significantly higher rates of allostatic load associated with the cardiovascular system than did the normal controls. My notes indicated Dr. Maloney found increased heart rate, SDB/DBP, and levels of albumin and, most importantly, c-reactive protein in

CFS patients compared to age, sex and BMI matched controls.

The CFS patients in the Wichita studies looked a lot like metabolic syndrome patients, an idea explored in an earlier edition of Phoenix Rising. Dr. Maloney explained that metabolic syndrome is characterized by high rates of abdominal obesity (waist-hip ratio), high triglyceride levels and high lipid levels. *Spence has shown that CFS patients have high levels of the oxidized form of the 'bad' cholesterol and low levels of the 'good' cholesterol as well as high levels of oxidized stress.* Dr. Maloney found that 33% of CFS patients (versus 14% of controls) met the criteria for metabolic syndrome and that females with CFS were particularly susceptible to doing so.

Low activity levels and weight gain can contribute to metabolic syndrome. I asked Dr. Maloney if she thought the inactivity and the weight gain seen in CFS was the cause of the metabolic syndrome found? She thought that inactivity could contribute to it but was more focused on the high levels of the inflammatory marker c-reactive protein (CRP). The fact that this substance also showed up in Vance Spence's study interested her greatly.

(The CDC is careful to have body mass index matched CFS patients and controls in its research studies. Since both CFS and healthy controls were equally overweight it appears that something other that weight gain is causing the increased rates of metabolic syndrome in these patients. The higher waist/hip ratio's in the CFS patients compared to the controls suggests they have a metabolic problem that causes greater fat deposition in the abdominal area. Fat in this area appears to be unusually active, pumping out increased pro-inflammatory cytokines such as Il-6.)

A key player in metabolic syndrome may be a pro-inflammatory cytokine called IL-6. Fortuitously a paper examining IL-6 levels in CFS was presented by another CDC researcher. This study was a highlight not only because of its findings but because of its innovative approach to this issue.

CONFERENCE HIGHLIGHT

A New Way to Research CFS?

Brian Gurbaxani, Kristin Singletary-Meadows, Andrew Miller, Dimitris Papanicolaou, Suzanne Vernon and William Reeves:
Elevated pro-inflammatory IL-6 in patients with Chronic Fatigue Syndrome

Dr. Gurbaxani gave an engaging and at times hilarious presentation. He represents the kind of innovative younger researcher that has been attracted to the CDC CFS research program in the last few years. His resume is most unusual; he has a B.S. in Applied and Engineering Physics and a Ph.D. in Molecular Biology and Bioinformatics. His background includes writing 'satellite mission control algorhythms and large scale simulations of space stations' (!). He is currently involved in writing data mining algorhythms for large data sets in CFS and other diseases… he is not your typical CFS researcher.

Dr. Gurbaxani has been studying an important but puzzling aspect of CFS – pro-inflammatory cytokines. If CFS has an inflammatory component then the levels of these cytokines should be increased but study results – and there have been many CFS cytokine studies – have been mixed. If Dr. Gurbaxani is right then he may have found an explanation why.

Dr. Gurbaxani's initial analysis indicated that IL-6 levels were increased in CFS patients but not significantly so. Ordinarily this would be the end of the study; it would be deemed a failure, simply another study indicating that cytokine up regulation does not play a role in CFS. But Dr. Gurbaxani knows his numbers and he thought that something was not quite right; he felt the CFS data was different in a ways that eluded standard statistical tests and so he did some unusual analyses on them. These indicated that IL-6 levels in the CFS patients were indeed different – significantly different.

Then he looked at several of the measures we have been talking about; c-reactive protein levels and waist to hip ratio and symptom severity, and found that IL-6 correlated with all three. Through the course of his work IL-6

went from an insignificant variable to a potentially central factor in not only the inflammation but in the increased rates of metabolic syndrome found as well.

Could Dr. Gurbaxani's techniques shed light on the mixed results of prior cytokine studies? I ask Dr. Gurbaxani about this and he stated he had seen data sets in which he thought the same process might be occurring.

Why would cytokine studies or studies on other subjects for that matter in CFS come up with these unusual results? Why would a CFS researcher need to go to such lengths? One reason could be the heterogeneous group of CFS patients he or she is studying. Most diseases are well defined; when breast cancer researchers study breast cancer patients they know they're studying breast cancer not prostrate or brain cancer. Researchers don't know that with regard to CFS; they know they are researching a disease characterized by extreme fatigue and some other symptoms but they don't know if the source of that fatigue – the disease process – differs from patient to patient. Dr. Gurbaxani stated that after hearing CFS patient stories at the conference he believes there are probably many triggers for CFS.

Dr. Gurbaxani's abstract ended with a sentence that will be well to keep in mind not only with regard to Dr. Levine's study later in the conference but with other puzzling CFS research results. *"..information theory analysis shows that the distribution of IL-6 in CFS is different than control(s) in all of its aspects (not just mean values) suggesting a different process is at work in CFS"*.

This different distribution could simply reflect the presence of subsets in CFS. If one set of CFS patients had high IL-6 levels but others didn't then a means test will look interesting but it will not pass the rigorous criteria required for it to be called significant. I asked Dr. Gurbaxani if he thought that subsets could be driving these weird distributions? He did. Dr. Spence of MERGE has made this point as well. Dr. Gurbaxani believes identifying subsets is the biggest problem facing CFS research.

Paul Cheney and N. Lucki:
Evidence for diastolic dysfunction in the Chronic Fatigue Syndrome enhanced by tilt echocardiography: a study of ninety consecutive cases.

Dr. Spence indicated that a logical outcome of increased arterial stiffness was diastolic dysfunction, a subject Dr. Cheney has focused on for the last three years.

Again (see Keynote Speech, Patient Conference) Dr. Cheney drew our attention to the fact that "preserved ejection fraction heart failure in women is epidemic, unexplained and deadly." *Preserved ejection fraction heart failure refers to cases of heart failure in which the pumping action of the heart (systole) is preserved but the filling phase (diastole) is impaired.* He believes that his scenario of impaired cellular energy production – which should, as he noted in the patient conference, affect diastolic functioning long before it affects systolic functioning – could explain the abrupt recent rise in diastolic heart failure.

He described a range of observed abnormalities in diastolic functioning found in his CFS patients during Tilt table tests. In a small study he found that echocardiography during Tilt best differentiates patients from controls. *There is some disagreement about how well some of these measures reflect cardiac functioning but one of the measures Cheney presented called E/e' ratio is well correlated with cardiac functioning.* Dr. Cheney also found a high degree of atrial cavitation (50% of CFS patients), reversal of blood flow into the pulmonary vein, and abnormal E/A ratio's.

He believes the striking similarities in diastolic dysfunction in CFS patients and young men exposed to low oxygen environments indicates that CFS patients exist in a state of 'functional hypoxia'.

A Layman's Speculations (pay it no special mind): *In his keynote address Dr. Cheney pointed out that he first conceived that diastolic dysfunction (DD) was present in CFS when he saw that the greatest difference in cardiac output between CFS and healthy controls in the Peckerman study occurred when they were lying down. It is my understanding that the filling defect in DD is most evident at times when the most blood is present in the heart, i.e. when one is lying down. Cheney is finding, however, that the DD is greatest in CFS patients when they are tilted, a period when paradoxically, blood flows to the heart, especially in CFS patients with low blood volume, are at their lowest – and when pressure on the left ventricle to expand to fill with blood, is at its lowest. This would at least at first blush seem to be an odd time to exhibit increased diastolic dysfunction.*

How to explain this conundrum? This a very complicated subject that I am ill qualified to speak on but for whatever its

worth one school of thought believes DD is more a function of extracardiac factors than with heart disease; that is, these researchers believe the problem with DD didn't start with the heart – it started elsewhere and that DD is more a function of increased wear and tear on the heart that it is with a disease of the heart muscle itself. That most problems in CFS are at their most severe when one is standing could perhaps suggest that the DD dysfunction in CFS is a function of them, not of a damaged heart.

Another Cardiac Abnormality in CFS

Paul Cheney and L. Nucki:
Evidence of increased frequency of patent foramen ovale (PFO) in the Chronic Fatigue Syndrome and enriched oxygen modulation of the PFO (poster)

We noted that Dr. Cheney believes that CFS patients exist in a state of 'functional hypoxia' or low oxygen levels relative to their needs (see Keynote Address, Patient Conference). He believes that a left-shift on the oxy-hemoglobin dissociation curve should raise pulmonary artery pressure and that this, in combination with the low blood pressures often found in CFS, would result in increased rates of the patent foramen ovale. Poor diastolic functioning would then increase the severity of the PFO.

Looking at 41 consecutive CFS patients he found 81% of them had PVO – the highest percentage yet reported in a disease. Two thirds required a Valsalva maneuver to open them and one third exhibited it without doing the maneuver. *The Valsalva maneuver consists of blowing hard with the mouth and nose closed. This raises venous pressure and is used to investigate some aspects of cardiac functioning including decreased filling, i.e. diastolic dysfunction. Some PFOs only open during strain such as lifting, during sex, etc. That most PFO's opened only during the Valsalva maneuver would appear to suggest that they are usually closed in 2/3rds of CFS patients (?).* Dr. Cheney graded the PFOs in severity – most were of low to intermediate severity. *This finding appears to be very similar to that of diastolic dysfunction in CFS – it is very common in CFS but is usually of a mild to intermediate level of severity.* Giving oxygen to these patients closed the PFO in about a third of them.

Dr. Cheney believes that these PFOs are the source of the 'unidentified bright objects' seen on MRI scans.

It's hard to tell how important these PFOs are. Two recent large Mayo Clinic studies concluded that PFOs are not a risk factor for stroke, ischemic attack or a 'cerebrovascular event' (Petty et. al. 2006, Meissner et. al. 2006). Several moderately sized studies do suggest, however, that PFOs are a risk factor for migraine. We will see in the Gene Section of the Conference that a study indicated that migraine is much more commonly found among relatives of CFS patients than would normally be expected. Some researchers believe that both migraine and CFS are diseases of central sensitization; one causing pain and the other fatigue. Of course migraine is temporary in nature while the fatigue in CFS is chronic. Large scale studies examining the PFO/migraine connection are underway. Aside from this possible connection, PFOs are not, at least this point, associated with any known symptoms in healthy people including fatigue.

A Summary – Cardiovascular/Vascular Implications in CFS: *What does all this mean? Are the increased rates of abdominal fat, elastase, arterial stiffness, IL-6, c-reactive protein, metabolic syndrome and diastolic dysfunction in CFS related? Are these researchers converging on each other from different directions? At least at this point it does appear so. High Il-6 levels, c-reactive protein levels and arterial stiffness are found in metabolic syndrome and all three may be risk factors for diastolic dysfunction. This is a complex subject, one that Phoenix Rising will be covering in more detail as these studies are published.*

EXERCISE AND CFS

I loved the understatement made by the Snell group in one of their abstracts "*For many patients these problems are exacerbated following physical activity and adversely affect their work, social and even family lives. For such a prominent symptom of CFS research in this area has been limited. . .*"

Yes, it is amazing how little research has been done on what many CFS patients consider their most unique symptom – the crushing fatigue and/or pain and cognitive dysfunction, etc. that occurs after (too much) exercise. If someone had asked me what to study in CFS 20 years ago I would have said whatever you do find out what happens to me in the hours and days after I exercise. Give me an exercise test and start taking blood, start scanning my brain, etc.

There are some things we do know. One study suggested immune activation (complement system) occurs after exercise and a gene expression study found evidence of altered ion channel transport activity. *Please note that nothing in this section implies that CFS patients should not exercise to the extent that they can without relapsing.*

The CFS Brain on Exercise – Not a Happy Place

A. Garcia Quintana, A. Garcia-Burillo, J. Alegre-Martin, I Mena, J. Garcia-Quintana:
Brain SPET quantification in Chronic Fatigue Syndrome: comparison of basal and post-stress studies.

The enterprising Garcia Quintana group from Spain used a SPET scan to examine brain activity before and after an exercise session. This is the first time, to my knowledge, that this has been done. It found that one area in particular, the Wernicke area, showed evidence of reduced activity after exercise. This area, which is found in the cerebral cortex, is thought to be 'essential for understanding and formulating coherent . . . speech.' Other areas in the temporal lobe were affected. Interestingly it was difficult to tell if the anterior cingulate region – a region we will see mentioned time and time again with regard to CFS – was affected, because the activity in this region was too low to begin with.

Immune Activation During Exercise

J. Alegre-Martin,T. Soriano Sanchez, C. Javierre, J. Quintana, E. Ruiz, T. de Sevilla, K. De Meirleir, and A. Quintana:
Study of biological markers, ergometric parameters and cognitive function in a cohort of patients with Chronic Fatigue Syndrome / Associations between biological markers, ergonometric parameters and cognitive function in a cohort of patients

These studies found that the majority of the CFS patients had increased rates of RNase L activity (83%), RNase L fragmentation (88%) and a whopping 95% had increased elastase levels. They also have decreased 'functional reserve' and peak aerobic power. The high elastase readings were, of course, intriguing given Dr. Spence's supposition that elastase was driving the high levels of arterial stiffness he found. These researchers suggested that the RNase L abnormalities could contribute to the muscle symptoms found.

Dr. De Meirleir's research indicates that elastase is a key player in the responsible for the RNase L fragmentation found in CFS. High elastase levels alone will not fragment the RNase L enzyme; that process also requires that the RNase L enzyme be left in an unprotected state. I asked Dr. De Meirleir if high levels of a protease like elastase were, however, ***a necessary*** *component of RNase L fragmentation and he said that it was. The question, then, becomes what is driving the increased elastase production?*

The study that employed an exercise period found that RNase L activity in CFS was correlated with lactate concentrations, an intriguing finding given the increased lactate levels in the brain found earlier. *Blood lactate research in CFS has, however, had a decidedly mixed history with some studies showing elevated lactates and others not. This could suggest that a subset of CFS patients have elevated lactate levels.*

As we have and will see in this conference some researchers (Gurbaxani, Levine, Natelson, Lange, Klimas/Fletcher, De Meirleir) are starting to look for these subsets hidden within their study results. One can only imagine how much more significant research findings would be if more researchers tarted looking for and identifying 'anomalies' or subsets in their data – and then focusing on that set of patients.

Klimas, N., Rosenthal, M and M. Fletcher:
Immune effects of an acute exercise challenge in Gulf War Illness (poster)

This is a preliminary report from a large study focusing on immune markers and gene expression before and after exercise in GWI, CFS patients and healthy controls. It found that NK cell counts went way up following the exercise period but that T-cell counts remained the same. Unfortunately they don't tell us if this pattern was different in the controls versus the CFS or GWI illness subjects but this study does show that NK cells are sent into a tizzy after exercise and given the NK cell abnormalities found in CFS this is an interesting finding.

No More Messing Around – Taking Exercise Stress Tests to the Next Level

Rudi Vermeulen, Ruud Kird, Hans Scholte:
A standardized test for post-exertional malaise in CFS? (poster)

This Dutch group carried the exercise testing regime one step or rather one day further by having CFS patients and healthy controls exercise three times over four days. It found that all the healthy controls and half the CFS patients improved their aerobic functioning in the second exercise trial. This would be enough for some CBT practitioners to say I told you so – that group is not sick! – and then instruct them to exercise their hearts out (perhaps literally). When these researchers extended the exercise test one day further, the test scores (VO2 max) of a big chunk of the CFS patients (25% more) declined - now 75% of the CFS patients were hurting. If they kept giving this test day after day they would surely find that all the CFS patients started falling apart.

This suggests what seems to be a rather obvious idea in retrospect, that one way to differentiate people with CFS from people with something else is through multiple day exercise testing. If post-exertional fatigue is a true marker of CFS and that fatigue is reflected by measures of aerobic output, then this test should differentiate 'true' CFS patients from others,

or at least from any subgroup of CFS patients who do not exhibit post-exertional fatigue. Of course this test would have to be limited to people who are not completely disabled by CFS.

CONFERENCE HIGHLIGHT

Finally! A Stress Test That Works – Hope For Disability Seekers

Margaret Ciccolella, Christopher Snell, Staci Stevens, Travis Stile, Mark Van Ness. Pacific Fatigue Lab, University of the Pacific, Stockton, Ca:
Chronic Fatigue Syndrome and the Abnormal Stress Test.

Documenting disability has been a real problem for CFS patients. The government believes that given their fatigued nature CFS patients should be unable to pass a stress test. Unfortunately CFS patients have shown a disturbing reluctance to fail them. Dr. Ciccolella reported one CFS patient who had four physicians testify as to his/her disability whose request for disability was nevertheless denied because he/she had passed a stress test. (This finding was overturned at the appellate level).

Dr. Ciccolella's theory was that since a single stress test would probably be insufficient to document disability in a disease characterized by post-exertional fatigue, they would do two, one 24 hours after the other. They found that while the control group did equally well at both tests, the exercise measures (Peak VO2, VO2 max) declined about 25% in CFS patients, far more than in other significant diseases such as COPD and heart failure (8%). This two-day stress test then provides objective evidence of disability in CFS and should provide, if it is accepted, major assistance to CFS patients attempting to get disability. This could be a real breakthrough.

The Snell Exercise Studies

The Snell group, which is associated with the Pacific Fatigue Lab in the University of the Pacific in California, has focused closely on the post-exercise period. They are sponsored by the CFIDS Association of America.

Christopher Snell, Staci Stevens, Lucinda Bateman, Travis Stiles and J. Mark Van Ness:
Using a reaction time paradigm to assess Neurocognitive Function in CFS.

So far we've found that if you test CFS patients after an exercise challenge, they exhibit declines in aerobic functioning and their NK cells proliferate wildly. Now the Snell group looks at cognition: CFS patients often complain that they have more trouble thinking after exertion. This study put that idea to the test; they measured a very simple measure of cognition – reaction time to both simple and complex stimuli - before and after an exercise test. It found that while CFS patients were slower, the results were not statistically significant. When they looked at all the test results together, however, there was a statistically significant difference. They also noted that variability within the CFS group was high while it was not in the control group. This suggests that some CFS patients did fairly well on these tests while others did not.

Perhaps the Dutch study noted above is informative in this regard. That study showed that a significant number of CFS patients who tolerated that first exercise test declined during the next one. Would a follow-up exercise test made their results more definitive?

It Hurts So Good? – No, It Doesn't

Staci Stevens, Christopher Snell, Lucinda Bateman, Travis Stiles and Mark Van Ness:
Post-exertional malaise following an exercise test (poster)

This was not an earth shattering study but it demonstrated very starkly how severely exercise affects CFS patients. After sedentary healthy controls and CFS patients exercised to capacity they were given SF-36 tests that characterize functioning in eight different areas: physical functioning, reduced activity levels because of physical problems, emotional problems, bodily pain, perception of general health, vitality (energy), social functioning and mental health. This study found that CFS patients reported significantly more problems in all eight areas compared to the flabby but

still healthy controls. Only one of the 21 CFS subjects had recovered to baseline within 48 hours while all the healthy controls had. This, of course, indicates that the exercise problems in CFS are not due to simple inactivity – the healthy controls were sedentary as well – but are an integral problem of the disease itself.

No Immune Activation During Exercise?

J. Mark Van Ness, Christopher Snell, Staci Stevens, Lucinda Bateman and Travis Stiles:
Metabolic and immune responses to exercise testing

This was an attempt to examine immune, metabolic (blood glucose, lactate) and sympathetic nervous system (nasal rhinometry) functioning during exercise and, with regard to the immune measures (RNase L fragmentation, elastase), after the exercise period in CFS patients and controls.

In contrast to the others this study failed. While CFS patients had significantly lower cardiopulmonary scores (VO2 max, etc.), rates of RNase L fragmentation and elastase activity were not increased after the exercise test. Nor were the measures of sympathetic nervous system functioning or metabolism different.

This is, unfortunately, rather familiar territory for CFS. Here we have one study showing increased rates of RNase L fragmentation and lactate and one showing no difference. Which is correct? Do the large numbers of CFS patients in the Garcia-Quintana study trump the control group in the Snell study? Or vice versa? I have no idea.

Summary: *We're seeing some real breakthroughs in the ability of extended exercise studies to reveal the disability present in CFS. These studies should give CFS greater credibility and new ammunition to use when applying for disability. Researchers are having more difficulty understanding what occurs during and after exercise in CFS. Explaining this problem seems to be one of the more difficult questions facing CFS researchers.*

The brain scan study indicated reduced brain activity after exercise and the aerobic tests indicated similar declines in aerobic functioning. The role RNase L plays in exercise intolerance, however, is up in the air and tests of sympathetic nervous functioning and metabolism were negative. 1

Reprinted with permission from Phoenix Rising Special Edition: A Layman's Guide to The 8th IACFS Conference.

Media Watch

Lyn Wilson has a blunt message for anyone who doubts that chronic fatigue syndrome is a debilitating physical illness. "Why would anyone want to give up their life and their income, for nothing? People who think we're not sick don't have a clue."

This was the opening paragraph of an excellent, long and well-balanced article on CFS/ME in *The Australian* on 29 January 2007 with the heading *Drained by the Brain*. This was a reference to Professor Andrew Lloyd's contribution in which he stated that, "The current thinking is that it is a disorder of the brain. We just don't know where in the brain or exactly what sort of brain chemical disturbance occurs."

Colin Neathercoat, a director of the ME/CFS Association of Australia, said that CFS organisations want Australian doctors to follow the Canadian Guidelines and the National Association's aim is for the Government to adopt them. The National Association is also moving towards asking the medical profession to use the name myalgic encephalomyelitis instead of chronic fatigue syndrome.

Also interviewed for the article were Peter Del Fante, Adelaide GP and CFS/ME specialist, and Jim Chambers, Vice President CFS/ME Victoria, who was able to represent the seriously affected sufferers.

As a result of this article, Jim Chambers was interviewed on the radio by 2HD (Newcastle) the following morning.

The article in *The Australian* was written by their health reporter, Clara Pirani. 1

Reprinted with permission from Emerge, Autumn 2007.

Cardiac symptoms

A letter to InterAction *56, June 2006.*

What's causing my cardiac symptoms?

I'm a severely affected 27-year-old woman, and whilst I have severe pain and sleep disturbances, it's my cardiac symptoms that I'm desperate to get some help with, or insight into. I suffer bouts of tachycardia or racing heartbeat (about 120 beats per minute, rather than the usual 60-100) once or twice a week, The attacks come on fairly randomly, but I've noticed that they're particularly bad after standing, making me wonder if there is a connection there.

I also get palpitations and arrhythmias where my heart skips beats and then flutters before regaining its rhythm. Again this occurs sporadically, and usually lasts a few minutes. I have worn a 24-hour ECG monitor but – of course! – I didn't experience these symptoms during that time.

However, the most common and intrusive of all my heart symptoms is what can only be described as a pounding or thudding heart beat. This bears no relation to my anxiety levels and the symptom is at its worst when I have gone over my baselines, or when my sleep is particularly awful. The problem is so bad that it prevents me from resting and sleeping, and often wakes me up: my heart beats so hard that it causes my whole body to shake. You can even see the pulsation through four layers of clothing! This can last from an hour or two, to several days or weeks and I've spoken to others with ME who also experience this distressing phenomenon.

I'd be grateful for any light you might be able to shed on my problems, and especially any suggestions be eased or treated.

AfME's principal medical adviser, Professor Tony Pinching, writes:

It's very understandable that symptoms that relate to the heart are especially worrying and distressing, and once one is aware of them, they can be hard to ignore – unless you know them to be harmless.

Many such symptoms are indeed benign expressions of altered body function in CFS/ME. However, if they are intrusive, frequent or unusual, they may merit specialist cardiological assessment. This is partly because they could be due to a separate co-existing condition, not related to CFS/ME.

Some of the infections that can trigger CFS/ME may also occasionally affect heart tissue. These are usually only present in the acute stage and are minor, but rarely they may leave some residual effect that could in theory coexist with the illness.

Your letter mentions several distinct symptoms, and I note that you are severely affected, which may be relevant to the reason for some of these.

Bouts of rapid regular heartbeats may occur in response to changes in physical activity, or in other body functions (including the effect of standing up, as the body reflexes that control blood pressure in this condition can be a bit sluggish), and in patients whose severe illness and disability has inevitably caused some cardiovascular deconditioning (unfitness), Gradual changes in body position, which physiotherapists or occupational therapists can advise on, may help minimise this effect. Anxiety, whether justifiable or due to co-existing anxiety states, can also trigger rapid heartbeats.

Skipped beats are amongst the commonest cardiac-type symptoms that I have come across in patients with CFS/ME. They can sometimes feel quite alarming, and may be worrisome if frequent. However, investigation of these typically shows them not to signify any serious hazard. An electrocardiogram (ECG) and 24-hour ECG tape can be helpful. Even if the symptoms don't occur during the 24-hour monitoring, minor versions can often be seen that help diagnosis.

Longer monitoring periods can be used if warranted.

If the skipped beats are frequent and intrusive, beta-blockers may reduce these and other forms of palpitations, but as side effects can include tiredness and weakness, these drugs seem to be poorly tolerated in many CFS/ME patients.

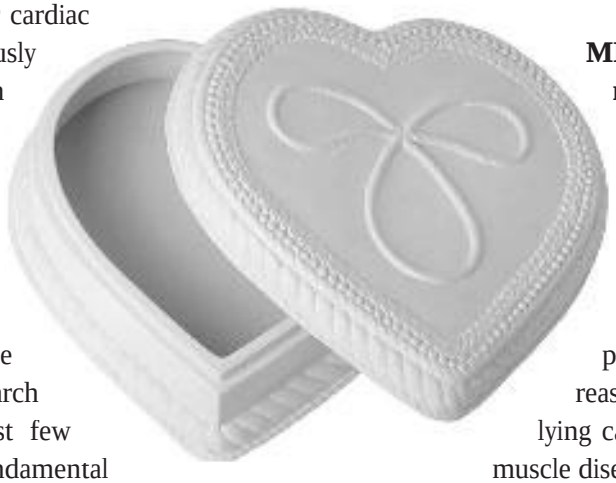
The pounding or thudding symptom sounds as if it is different, and is probably not actually cardiac in origin, It is hard to tell for sure however, without taking a more detailed direct history and preferably also examining you while this is happening.

I do wonder if this symptom may be part of the trembling, jumping or pounding that many patients with CFS/ME seem to get from the body muscles, which may affect the chest, back, abdominal wall or limb muscles, It can be quite rhythmic and certainly can cause the body to shake; some patients find that this pulsing is, or seems to be, in time with the heart rate.

This symptom may also be associated with lesser, irregular muscle twitches, and/or with cramps, and can often occur alongside muscle pain. If this is the case then it's one of the symptoms that may be helped by the use of muscle relaxants, such as Baclofen.

Liverpool-based Consultant Physician specialising in ME/CFS, Alastair Miller, adds:

I'm sorry to hear about your cardiac symptoms which are obviously very distressing especially in addition to severe CFS/ME.



Most NHS practitioners involved in the management of this illness would regard it as a 'medically unexplained syndrome' because despite the intensive research that's gone on over the last few years, we don't yet have a fundamental mechanism to explain what's going on at a molecular, cellular, biochemical or physiological level that can account for the complex range of symptoms seen. There are numerous theories about potential abnormalities of cellular workings, of the nervous or immune systems; or relating to the role of chronic infection.

CFS/ME has much in common with other medically unexplained syndromes such IBS, fibromyalgia, chronic pain syndromes, and chronic tension headaches etc. It's a frequent finding in studies that patients with CFS/ME are statistically more likely to suffer from other medically unexplained syndromes and once again the reason for this is so far unclear.

Medically unexplained cardiological symptoms are a common problem in the general population and are also common among those who have CFS/ME. From your description of the symptoms there are no features that cause me alarm and indicate they are suggestive of major underlying cardiac disease. However, they do merit careful evaluation and it's probably worth your GP or local cardiologist examining the symptoms further with a detailed clinical history and physical examination followed by some simple, safe, non invasive tests. These could include a chest x-ray, ECG, 24-hour ECG (repeat) and possibly an exercise ECG and echocardiogram. If the cardiological evaluation cannot identify a cause for the symptoms then it is reasonable to label them as medically unexplained cardiac symptoms and associate them with your CFS/ME. If this is the case they should respond to the same therapeutic approach taken in managing the illness generally (e.g., pacing).

If the sensation of tachycardia and dropped beats is particularly unpleasant, you're not asthmatic and have no other reason to avoid beta blockers then they may be helpful in gaining symptomatic control, although they worsen tiredness in some people.

However, they may be worth a try if other approaches haven't helped.

ME/CFS specialist and Secretary to the British Society for Ecological Medicine, Dr Sarah Myhill, concludes:

Heart problems of all descriptions are common in people with this illness. The reason for this is that the underlying causes of ME/CFS and heart muscle disease are I believe linked.

The first piece of evidence comes from a paper by Dr Arnold Peckerman who was commissioned by the National Institutes of Health in America to work on developing a test for CFS.

Peckerman looked at the research literature showing that CFS patients suffer from low blood pressure, low blood volume and are poor at maintaining their blood pressure when they stand up. Being a cardiologist, he diagnosed heart malfunction and went on to revive a test that was very popular in the 1970s called impedance cardiography, which measures cardiac output in a very non-invasive way.

Essentially what he found was that in severely afflicted CFS patients their hearts were failing to work efficiently and this explained many of their symptoms. The work of the heart is much less if you're lying

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down because it's much easier to push blood around on the flat than up and down hills!

Secondly, when cardiac output falls, the body shuts down the blood supply to less important areas in order to maintain vital organs such as the kidneys, liver and gut.

Therefore, we see decreased blood flow to skin (poor temperature regulation, cold hands and feet), to muscle (early switch to anaerobic or non-oxygen fuelled metabolism), to the brain (foggy brain and difficulty thinking clearly) and so on. People with ME/CFS who are not so severely afflicted also have a degree of heart malfunction which manifests as poor exercise tolerance.

The second clue came from a book published earlier this year called The Sinatra Solution. This was also written by a cardiologist who started asking why heart disease arose. There's no doubt that some of it is caused by poor blood supply to the heart. However, Dr Sinatra demonstrated that a large amount of heart malfunctions are caused by a muscle problem arising from a failure to supply energy at a cellular level.

We are all made up of cells: heart cells; muscle cells; brain cells; and so on.

All have a different job to do and rely on the energy supplied to them by mitochondrial (energy-making) cells in the form of ATP (adenosine triphosphate). In converting to ADP (adenosine di-phosphate) energy is released and then the mitochondrial cells convert ADP back to ATP again. Healthy people recycle a molecule of ATP about every ten seconds.

What Sinatra has demonstrated elegantly in patients with heart disease is that they have slow recycling of ATP – that is to say they suffer from mitochondrial or 'energy cell' failure.

He's put together a package of nutrients with which he treats his patients with reportedly excellent results. His daily 'awesome foursome' is made up of 200mg Co-enzyme Ql0, 2g of Acetyl L-carnitine, (a peptide which transports fuel for energy production across mitochondrial membranes) 15g. D-ribose (an essential compound in energy metabolism) and 600mg magnesium.

In ME/CFS I believe there is a generalised mitochondrial cell failure.

I've submitted a paper to the Journal of Nutritional & Environmental Medicine on this subject, which I hope will be published shortly. What this means is that energy supply to all cells in the body is impaired, causing them to 'go slow'. This theory provides a possible explanation for the multiplicity of symptoms that patients suffer from. Mitochondria can be damaged in lots of different ways: by viruses, chemical poisoning, or overwhelming stress, which would tie in with the different paths individuals have enroute to succumbing to ME.

This illness model has huge implications for treatment, which can then be tackled on a very logical basis.

Firstly, people with ME. must pace mental and physical activities so that they don't further stress their mitochondria.

Secondly, they must get a good night's sleep because it's during sleep that the body heals and repairs.

Thirdly, diet is such a common cause of fatigue, either as a result of low blood sugar or food intolerances, that it's vital to trial a 'stoneage' diet avoiding the major allergens such as gluten, dairy and sugar, instead choosing low glycaemic index foods like brown rice which release carbohydrates slowly.

Fourthly, everybody should try taking a good range of vitamins, minerals and essential fatty acids in order to give mitochondria the raw materials to function properly.

I see many patients improve on the above regime.

Especially crucial in this is the magnesium, because this mineral is necessary for energy to be released from ATP and for ATP to be re-made from ADP. 40% of resting energy simply goes into driving the mechanism which pumps calcium out of cells and magnesium into cells. Therefore when mitochondria start to fail they lose the energy that they need to draw magnesium into cells, further impairing their ability

to function well. For this reason intracellular magnesium deficiency is both a cause and a symptom of ME/CFS and mitochondrial failure (with many CFS studies demonstrating red cell magnesium deficiency). Some people need to have magnesium by injection in order to get their levels up.

I hope that this summary sheds some light on what could be going wrong with this member's heart.

Tachy-arrhythmias are likely to be due to poor energy supply to the heart's natural pacemaker, or possibly an adrenaline response from the body as cardiac output falls. The heart is simply not working efficiently as a pump, which I suspect is why she gets the severe thudding heartbeat, or again this could be an adrenaline response to low cardiac output.

Clearly she is stressing the heart (for the above reasons) and if some of these aspects could be tackled then there is great scope for improvement.

The problem for so many severely afflicted people is that they don't have the physical, mental or emotional resources to put in such major lifestyle changes, nutrional treatments and detox regimes. all at once.

However, if one can gradually chip away at them and start to see some improvements then this gives energy for further interventions to be put into place. 1

Ref1: American Journal of Medical Sciences, *Aug 2003; see www.cfids-cab.org/cfs-inform/Coicfs/peckerman.etal.03.pdf.*

Further information: Dr Myhill has a website with more detailed information about her views on treating cardiac problems in ME/CFS. Visit: www.drmyhill.co.uk.

Reproduced from InterAction, *the quarterly magazine of Action for M.E. Further articles on this and other subjects may be found on their website at www.afme.org.uk.*

Adelaide ME/CFS Research Forum report

A report by Colin Neathercoat of the ME/CFS Research Forum, The University of Adelaide, 26-27 March 2007.

Clinical and research specialists from the USA, Europe and Australia attending the ME/CFS Research Forum have begun the process of developing an international post-mortem protocol for the disease ME/CFS. The protocol will guide the retrieval and storage of key tissues and organs affected in ME/CFS, in particular the brain and spinal cord, for future pathological examination.

Dr Abhijit Chaudhuri, Consultant Neurologist, Essex Centre for Neurological Sciences, UK: *"Disease is characterised by pathological changes in the tissues of those affected. We have previous evidence of brain and spinal cord abnormalities in ME/CFS patients and establishing protocols and international collaboration is a logical next step in furthering our understanding of the disease."*

Participants examined research evidence for the role of persistent complex intracellular pathogens including Q fever, Ross River Virus, Rickettsia and HHV-6/7 in the development and perpetuation of ME/CFS.

Dr Daniel Peterson, Medical Director of the Whittemore Peterson Neuro Immune Institute, Nevada USA: *"Clear cut evidence now exists of antecedent infection with these agents in sub-groups of ME/CFS, presumably through mechanisms of immune perturbation."*

Evidence was presented of severe gastrointestinal compromise in ME/CFS including loss of mucosal surface integrity and bacterial colonosis.

The evidence base for ME/CFS research has been seriously distorted, greatly hindering understanding of the disease. Amorphous definitions have contaminated study cohorts and corrupted research data leading to misdiagnosis, false psychiatric attributions and the use of inappropriate treatments. The research group strongly recommended the international adoption of the 2003 Canadian ME/CFS Definition and Guidelines for Medical Practitioners.

Professor Malcolm Hooper, Emeritus Professor of Medicinal Chemistry, University of Sunderland, UK: *"There is a compelling need to identify and characterise sub-groups of ME/CFS associated with immunological, inflammatory, neurological and other biological markers to accelerate our understanding of the disease and the development of appropriate treatment protocols specific to each of these groups."*

Participants were also briefed on the establishment of the Whittemore Peterson Institute in Nevada, a unique dedicated ME/CFS research and treatment development centre offering comprehensive care for people affected by the disease. 1

Information about ME/CFS

What is ME/CFS?

Myalgic Encephalopathy/Chronic Fatigue Syndrome (ME/CFS) is characterised by severe, disabling fatigue and post-exertional malaise. Fatigue is just one symptom – there are a multitude of others. ME/CFS is a not uncommon medical disorder that causes significant ill health and disability in sufferers.

Myalgic Encephalopathy/Chronic Fatigue Syndrome (ME/CFS) is also known by other names such as Post Viral Fatigue Syndrome, Chronic Fatigue and Immune Dysfunction Syndrome (CFIDS) and Myalgic Encephalomyelitis.

It is now officially recognised by the World Health Organization International Classification of Diseases and by recent international and Australian guidelines on ME/CFS.

Prevalence

ME/CFS affects all social and ethnic groups. There is a predominance of females (2 to 1) and a bimodal distribution with peaks between 15-20 year olds and 33-45 year olds. The prevalence of ME/CFS varies between 0.2% and 0.5% of the total population. In South Australia this translates to between 3,000 and 7,000 cases at any one time.

Main characteristics of ME/CFS

Disabling fatigue for at least 6 months, along with cardinal symptoms such as:

- muscle aches and pain;
- unrefreshing sleep or altered sleep patterns;
- neuro-cognitive dysfunction (e.g. poor concentration and memory);
- gastro-intestinal symptoms (e.g. irritable bowel);
- orthostatic intolerance (e.g. low blood pressure);
- and unusual headaches.

A hallmark of the condition is that symptoms are usually worsened with minimal physical and mental exertion.

Definition

The Canadian Expert Consensus Panel published the first diagnostic ME/CFS criteria for clinical use in 2003. In contrast to earlier sets of criteria, this new definition made it compulsory that to be diagnosed with ME/CFS, a patient must become symptomatically ill after minimal exertion. It also clarified other neurological, neurocognitive, neuroendocrine, autonomic, and immune manifestations of the condition. The Canadian Consensus criteria are wholly supported by ME/CFS SA and by the National Board of ME/CFS Australia. Copies are available from the ME/CFS SA website.

Diagnosing ME/CFS

Note that there are many other conditions which may need exclusion by your doctor before a diagnosis of ME/CFS may be made. These include, Hypothyroidism, Hyperthyroidism, Diabetes Mellitus, Addison's disease and Multiple Sclerosis, just to name a few.

ME/CFS may also co-exist with or mimic symptoms associated with: fibromyalgia; multiple chemical sensitivity; Irritable Bowel Syndrome; depression; anxiety disorders; and somatoform disorders.

This can make the diagnosis of ME/CFS and any coexisting conditions difficult.

How is ME/CFS treated?

All treatment should be patient-centred and involve supportive counselling, lifestyle management and the setting of realistic goals. There is no known cure for ME/CFS. Management is geared at improving functionality and symptom control through an effective therapeutic alliance between the patient and their GP.

Therapy for ME/CFS is intended primarily to relieve specific symptoms. It must be carefully tailored to meet the needs of each patient. Sleep disorders, pain, gastrointestinal difficulties, allergies and depression are some of the symptoms which may be relieved through the use of medications and other interventions.

Lifestyle changes including appropriate rest, reduced stress, dietary measures/restrictions and nutritional supplementation may be of benefit. Supportive therapy, such as counselling, can help to identify and develop effective coping strategies.

There is still a great deal of controversy surrounding the issue of whether people with ME/CFS should undertake intentional exercise. Most ME/CFS patient groups recommend that sufferers pace themselves by starting with gentle exercises and slowly increasing levels of exercise without causing a significant relapse of symptoms. It is important to maintain physical fitness if possible, but we recognise that exercise is not always the best possible use of sufferer's limited energy reserves.

Prognosis

The prognosis for ME/CFS patients is variable. Most will generally improve in functionality to some degree over time, usually 3 to 5 years. However, symptoms may fluctuate or relapses may occur from time to time. Early intervention and positive diagnosis often result in a better prognosis. However, a significant proportion of patients will remain quite debilitated for longer periods of time.

TALKING POINT – 2007 ISSUE 2

The Official Journal of the M.E./C.F.S. Society (SA) Inc.

Proposed Meeting Programme for 2007

Saturday 21 April: *Doctors' overview of the March research forum.*
NOTE: MEETING POSTPONED.

Saturday 12 May: *Q Fever, Rickettsia and ME/CFS.*
Emeritus Professor Barrie Marmion.
2:00 pm DIRC Building, 195 Gilles Street, Adelaide.

Saturday 7 July: *Alternative therapies and ME/CFS.*
Kinesiologist Tim White and Bioresonance Therapist Dr Andrew Barrie.
12:00 pm DIRC Building, 195 Gilles Street, Adelaide.

Saturday 4 August: *A Guide to Managing ME/CFS.*
Psychologists Liana Taylor and Liz Vaskin.
1:00 pm DIRC Building, 195 Gilles Street, Adelaide.

Saturday 8 September: *Diet, Weight Management, Food Sensitivity, Low GI, Fructose Malabsorption.*
Dietitian Melanie Reid.
12:30 pm DIRC Building, 195 Gilles Street, Adelaide.

Saturday 6 October: *Physiotherapy and ME/CFS.*
Physiotherapists Julie Peacock and Nathan Strempel.
12:30 pm DIRC Building, 195 Gilles Street, Adelaide.

Saturday 17 November: *Annual General Meeting.*
Speaker to be announced.
DIRC Building, 195 Gilles St Adelaide.

Contact numbers

Miscellaneous Support Contacts

North Eastern	Julie	8264 0607
North Eastern	Pat	8264 9328
SAYME	Emma	8276 5353
SAYME Parents	Marg	8276 5353

Country Support Contacts

Auburn	Kay Hoskin	8849 2143
Barossa Valley	Dennis	8563 2976
Mt. Gambier	Di Lock	8725 8398 or 0438 358 398 (mobile)
Port Lincoln	Jade and Pauline	8683 1090
Port Pirie	Marj	8633 0867
Riverland	Kathy Southeren	8586 3513
Victor Harbor	Melanie	8552 0600
Whyalla	Peter	8644 1897
Yorke Peninsula (central)	Caroline	8837 4335
Yunta	Gloria	8650 5938

Support groups

Glenelg Support Group

The Glenelg Support Group meets on the third Wednesday of each month.

Venue: Cinema Centre Coffee Lounge, Jetty Road, Glenelg.
Time: 1:00 pm.
Contact: Marion Hansen.
Phone: Marion on (08) 8234 2342.

Northern Yorke Peninsula CFS Support Group

Venue: Community Health Centre Wallaroo.
Phone: David on 8862 1665.

YOUTH SUPPORT: SAYME

South Australian Youth with ME/CFS

The idea behind having a Youth group is to get young people with Chronic Fatigue Syndrome together at the same place at the same time to relax, chill out, and to have a bit of fun within the limits of their condition and to develop a network of friends with Chronic Fatigue Syndrome that understand the issues we face. Together we can help each other through the tough times.

The Youth group is open to young people up until the age of 30.

Please contact Emma Wing in the office on Wednesdays on 8410 8929 for a program of events or if you would like to receive our quarterly magazine. We would love to meet you.

Disclaimer

Please note that meeting times are subject to change.

If you are attending a meeting for the first time please call the contact or the Information and Support Line for confirmation of meeting days and times: 8410 8930 or 1800 136 626.

If undeliverable return to:
ME/CFS Society (SA) Inc
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