

The Journal of the



Canadian Rheumatology Association

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Volume 9, Number 2

June 1999

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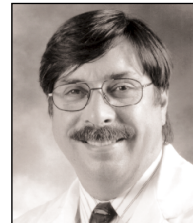
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*The mission of the CRAJ is to encourage discourse among the Canadian Rheumatology community for the exchange of opinions and information.*

## Rheumatology Clinical Fellowships— Whence the Support?

Recent assessments of human resource requirements across Canada suggest there is a shortage of rheumatologists.<sup>1</sup> This shortage is particularly acute in academic centres.<sup>2</sup> There are currently several academic positions available across the country including those in Halifax, Toronto, Quebec City, Ottawa and Saskatchewan.

Furthermore, utilization studies indicate that musculoskeletal complaints are primary reasons for attending a physician's office in Canada.<sup>3,4</sup> When planning human resource needs for the future, ministries and patient advocacy groups focus on societal needs which frequently exceed the capability of the supply of physicians active at the time.<sup>5</sup>

The current shortage of rheumatologists has resulted for a number of reasons:

1. With the aging of the population, there has been a greater demand for rheumatological care.<sup>3</sup> Badley and Wong suggest that by the year 2031, the prevalence

of arthritis diagnosed by a health professional as a long-term condition in Canada will increase from 10.7 to 15.7%—an increase of 124%. At the same time, the number of physicians to care for these patients is predicted to decrease if the current situation is allowed to continue.

2. Over the past two decades, there has been a greater recognition of rheumatic diseases in part due to better diagnostic ability for conditions already recognized (e.g., systemic lupus erythematosus) and the wider acceptance of other conditions such as fibromyalgia. The interventions available for other conditions such as osteoarthritis have also contributed to an increased number of consultations to rheumatologists.<sup>6</sup>
3. Eva Ryten, former director of research for the Association of Canadian Medical Schools, recently presented data to the Fraser Institute demonstrating that almost 50% of graduates of Canadian medical schools leave for the U.S. reducing the potential pool available for post-graduate training.<sup>7</sup> As well, a significant number of Canadian rheumatologists have emigrated to the United States.
4. The emigration of practitioners has not been balanced by the immigration of trained professionals. While extremely well qualified international medical graduates are available, there are government and licensing body restrictions which prevent these individuals from practicing in Canada.<sup>8</sup>
5. As in other specialty areas, there is a progressive greying of the active cohort especially those associated full-time with the universities.
6. There has been a progressive decline in the number of training positions in rheumatology. This has resulted in the reduction of Royal College certified rheumatologists. Whereas in 1983 there were 20 candidates for the rheumatology subspecialty qualification, in 1998 there were only nine. This decrease threatens not only the supply of clinical rheumatologists, but also the supply of potential academic rheumatologists committed to teaching, clinical, and basic science research.

Strategies to deal with this shortage of rheumatologists must be devised. As rheumatologists, we can do little more than disseminate the facts regarding issues one through five. However, we can be an effective advocacy group regarding issue number six.



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What then are the reasons for a progressive decline in the number of training positions in rheumatology?

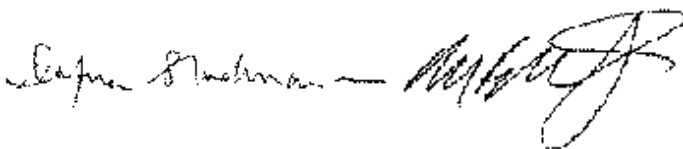
1. There has been an overall decrease throughout the 1990s in training positions funded by the Ministry of Health. This decrease affects all specialties and subspecialties.
2. The MOH's stated goal has been to train more specialists such as general internal medicine, general surgery, obstetrics and gynecology and psychiatry at the expense of subspecialties such as rheumatology. At the University of Toronto, for example, the number of ministry positions assigned to rheumatology has decreased from six positions in 1988 to three positions in 1994. Thus, in the past five years there has been a 50% reduction in training slots.
3. There has been a decrease in leverage previously offered by the Arthritis Society fellowships-residencies. In its matching program, the society offered to couple each university rheumatology training position with an Arthritis Society funded position. The society was accepted as a legitimate funding source by the MOH (one of few). This unique situation among subspecialties was in large part due to the vision and persuasiveness of the late Mr. Edward Dunlop, the first managing director of the society. As the Arthritis Society reduced its funding, universities have gladly said "you don't put one in and we won't either" and this has resulted in a drastic decrease in training positions. For example, in Toronto as the fellowships decreased from six to three, the total number of trainees decreased from 12 to six.

#### OUR ACTION PLAN IS CLEAR

We need to convince governments and universities of the societal need for more rheumatologic care. This means an immediate increase in training positions because it requires five to seven years to produce a rheumatologist.

To help universities and governments, we must reinvigorate the fellowship-residency Arthritis Society funding programs to help university programs increase their rheumatology training programs. The potential to train is there. We must ensure that the potential is utilized by providing training paths. Universities will be encouraged to increase training slots if they can automatically double their numbers with the society funding matched positions.

The society is therefore pivotal. As long as the Arthritis Society remains a legitimate funding source for residency training, we have the potential to solve our own problem. Let it not be said that we had the opportunity to determine our own destiny but didn't recognize that opportunity. We must therefore confirm and enhance the fellowship-residency support program.



*Dafna D. Gladman*

*Murray B. Urowitz*

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At the CRA's annual meeting, rheumatologists from across the country met to discuss topics pertaining to the field.

**D**r. Eric Rich covered the practical use of biologics in treating rheumatism. Over the last few years, advances in this field have progressed at a remarkable pace, and many trials have been conducted. Dr. Rich summarized five published studies on the subject.

**CHIMERIC MONOCLONAL ANTIBODY TO TNF ALPHA**  
Tumour necrosis factor (TNF) alpha is a critical inflammatory mediator in rheumatoid arthritis (RA). Chimeric monoclonal antibody to TNF alpha (cA2 or infliximab) has been studied for its effects in patients with active rheumatoid arthritis.

Elliott et al<sup>1</sup> presented a breakthrough short-term study comparing single-infusion cA2 to placebo in a randomized, double-blind, placebo-controlled, 73-patient trial.

Tender and swollen joint counts were reduced significantly. The American College of Rheumatologists' (ACR) criteria for 50% improvement in measures of disease activity (ACR 50% goal) was reached by 58% of patients taking high-dose (10 mg/kg) cA2. The ACR 20% goal was reached by 79% of patients.

Shortly after this trial, however, Elliott et al<sup>2</sup> were disappointed when they studied repeated treatment with cA2 in patients having disease flares. Although patients did well initially with their swollen joint counts, they flared upon redosing. And upon further redosing, patients experienced flares in closer and shorter increments.

"One explanation for this trend could be that up to 50% of patients receiving multiple dosing developed human antichimeric antibodies. This could be a factor for the reduced efficacy with repeated dosage," Dr. Rich said.

**COMBINATION THERAPY: cA2 AND METHOTREXATE**  
Because the antichimeric human antibodies the patients developed seemed to shorten the effect of cA2, investigators decided to lower the immunogenicity by adding an immunosuppressant. After conducting pilot studies, they tried combining cA2 with methotrexate.

The placebo-controlled, double-blind, 26-week study, conducted by Maini et al<sup>3</sup> examined the effects of multiple cA2 infusions given alone or in combination with low-dose methotrexate. The study involved 101 patients with active RA who were exhibiting an incomplete response or flare of

## WORKSHOPS

- Biologics
- Refractory ankylosing spondylitis
- Glucosamine
- Hepatitis C

The CRA met in Banff in February 1999.

disease activity while receiving low-dose methotrexate.

The effect of cA2 lasted much longer when methotrexate was used (regardless of the cA2 dose), and these patients developed much fewer anti-chimeric human antibodies. Methotrexate seemed to diminish immunogenicity.

## HUMAN RECOMBINANT FUSION PROTEIN

Moreland<sup>4</sup> conducted a short-term study on a new TNF antagonist. This was a recombinant fusion protein consisting of the soluble TNF receptor linked to the Fc portion of human IgG1 (TNFR:Fc). This double-blind trial involved 180 patients with refractory RA who had been previously treated unsuccessfully with disease-modifying anti-rheumatic drugs (DMARDs). The patients were given either placebo or subcutaneous TNFR:Fc twice weekly for three months.

In this trial, the ACR 20% goal was reached by 75% of patients who were given high doses of TNFR:Fc (16 mg/m<sup>2</sup> body surface area). A total of 57% of such patients reached the ACR 50% goal. However, when the twice weekly injections were stopped, patients relapsed.

## COMBINATION THERAPY: TNFR:Fc GIVEN WITH METHOTREXATE

Weinblatt et al<sup>5</sup> examined the effects of combining TNFR:Fc with methotrexate in a 24-week, double-blind, randomized, placebo-controlled trial. The trial involved 89 patients with persistently active RA despite at least six months of methotrexate therapy. They were prescribed either placebo or TNFR:Fc (etanercept 25 mg) twice weekly continuing to receive methotrexate.

The addition of TNFR:Fc to methotrexate resulted in rapid and sustained improvement. Seventy-one per cent of patients receiving combination therapy met the ACR 20% goal.

However, "additional study needs to be conducted to determine whether TNF receptor fusion protein is effective alone or whether there are added effects from a methotrexate combination," Dr. Rich said. cA2 and TNFR:Fc are initially very useful in treating RA.

Because of immunogenicity, cA2 must be given along with methotrexate in order to maintain the effect. Studies must be conducted to determine whether TNFR:Fc is effective alone or if there are added benefits from a methotrexate combination.

*Continued on page 11*

# Refractory ankylosing spondylitis

**D**rs. Walter Maksymowych and Glen Armstrong discussed refractory ankylosing spondylitis and new ways to treat this disease.

## ANKYLOSING SPONDYLITIS

For the last 30 to 40 years, the pathology of ankylosing spondylitis has been thought of in much the same way as rheumatoid arthritis (RA), with the final common pathway being synovitis. However, synovitis is not a predominant component of early ankylosing spondylitis. Instead, histopathologically, there is subchondral bone marrow inflammation with granulation tissue erupting through the iliac cartilage.

The x-ray is still used to diagnose ankylosing spondylitis. However, a change in x-ray signifies late-stage disease. By the time sacroiliac joint disease shows on the x-ray, the pathology is probably well established. A better assessment method is magnetic resonance imaging (MRI), which is useful for detecting pathologic processes such as bone marrow edema and inflammation, bone erosion and sclerosis of the bone.

## CONVENTIONAL TREATMENT AND NSAIDS

Nonsteroidal anti-inflammatory drugs (NSAIDs) have primarily been used to control symptoms of ankylosing spondylitis, but there is no evidence that these agents influence the disease course. About 25% of patients are refractory to NSAIDs.

## SULFASALAZINE (SALAZOPYRIN)

Dr. Maksymowych briefly discussed sulfasalazine's role in treating ankylosing spondylitis. He described a large multicentre sulfasalazine trial involving 350 ankylosing spondylitis patients. The sulfasalazine patients with polyarticular disease experienced a marked improvement over those taking placebo. However, within two years most patients had stopped taking the drug because of side effects or diminished efficacy.

## MESALAMINE (PENTASA)

5-aminosalicylic acid is a formulation of mesalamine that is bioavailable in both the large and small bowel. It is a breakdown product of sulfasalazine without the sulfapyridine and thus is useful in patients who are allergic to sulfur drugs.

Drs. Maksymowych and Armstrong conducted an open-label trial on 5-aminosalicylic acid involving 30 patients with active spondylarthropathy. Subjective measures such as severity of stiffness, night awakenings and severity of pain all improved significantly. There was also a statistically significant decrease in the number of affected peripheral joints and an average reduction in the erythrocyte sedimentation rate (ESR) from 36 to 20.

At the end of the 16-week study, 5-aminosalicylic acid therapy was stopped. Within four to eight weeks, most patients became clinically worse and their ESR rates deteriorated. When these patients were put on long-term therapy, they improved continuously.

## PAMIDRONATE

Bisphosphonates such as pamidronate possess anti-inflammatory properties and selectively localize to sites of active bone turnover. They are useful for treating early spondyloarthritis.

Pamidronate is suitable for close examination because it is taken intravenously and thus exposes body tissues to high circulating levels. The drug is very well tolerated with few side effects. Dr. Maksymowych's team conducted an open-label study on pamidronate.<sup>1</sup> Fourteen men and two women with ankylosing spondylitis were given varying doses of the drug for three or six months.

The group used the Bath Disease Activity Index (BASDAI), the functional index (BASFI), and metrology index (BASMI) outcome measures, as well as hemoglobin and ESR to assess disease progression. Significant improvements in these outcome measures were observed in most patients.

## CASES FROM THE STUDY

*Case 1:* A patient presented last year with joint pains, especially in the right knee. He was unresponsive to oral antibiotics, diclofenac and sulfasalazine. After a few months of high intensity pamidronate therapy, the synovitis in his right knee had completely disappeared.

*Case 2:* A patient presented in 1982 with hip and knee pain. After treatment with NSAIDs, aspirations and local injections, he developed active spondylitis with knee synovitis. When treated with systemic steroids and 5-aminosalicylic acid, he developed hyperthyroidism. After being given escalating doses of methotrexate, he still had ongoing knee synovitis. Last year, he was put on high-intensity pamidronate and his methotrexate dose was gradually decreased. By the end of last year, his knee synovitis was completely resolved.

## FUTURE TREATMENT OF ANKYLOSING SPONDYLITIS

According to Dr. Maksymowych, the way these patients are managed must be re-evaluated. Perhaps a combination approach should be followed, in which different aspects of the pathophysiology of the disease are addressed.

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## Glucosamine

Most studies examining the effectiveness of glucosamine and chondroitin sulphate have suggested there is an overall improvement in symptom score. Most of the data have been derived from European and Asian literature. Most of the available clinical data are difficult to interpret because of serious deficiencies in study design. More studies are therefore required.

In his presentation on the topic, Dr. Joseph Houpt mentioned three excellent articles in a current issue of the *Annals of Pharmacotherapy*.<sup>1-3</sup> He said the article by da Camara was of particular interest and that data from short-term human trials suggest that glucosamine sulfate administered orally, intramuscularly and intra-articularly may produce a gradual and progressive reduction in joint pain and tenderness as well as improved range of motion and walking speed.

*Glucosamine is the key precursor to all the various modified sugars found in GAG. Glucosamine also makes up 50% of hyaluronic acid, the backbone on which the other GAG, like chondroitin sulfates are added.*

There have been a number of other articles dealing with chondroitin sulfate. Leeb and coworkers looked at 12 published trials using chondroitin sulfate and found that only four of them were good enough to summarize. They conducted a meta-analysis on these. Leeb and his group looked at the Lequesne's Index. The VAS for pain and consumption of comedication; they regard these as the main efficacy parameters. All four trials showed that chondroitin sulfate treatment was superior to placebo.

"I found that if you look at the data from 0 to 60 days, the numbers don't look very good. It's only when you get to 180 days, at the six-month data point, that it looks as if there's a separation between the groups," Dr. Houpt said. "So those individuals who say, 'Hey, I went out and I bought chondroitin sulfate and within a week I felt great,' they just don't fit the data." The results suggest that chondroitin sulfate may be efficacious.

Felson's group looked at studies of glucosamine and studies of chondroitin. They did Medline and manual searches of manuscripts in journal supplements, they contacted authors, they looked at what they called study quality and they looked for *p* values and size of treatment effect (the difference between the treated and placebo groups divided by the mean or the pooled standard deviation). They treated Global Pain Score, or the Lequesne's Index, as a primary outcome.

The quality scores weren't very good. The deficiencies were related to the descriptions of randomization, blinding and completion rates. Again, all eligible studies reported positive results and showed large effects. Glucosamine sulfate and chondroitin had a score reduction that was comparable to placebo.

Clinical trials of chondroitin and glucosamine show substantial benefits in the treatment of osteoarthritis (OA) but provide insufficient information about study design and conduct to allow definitive evaluation. Additional studies are required.

Glucosamine is the key precursor to all the various modified sugars found in glycosaminoglycan (GAG). Glucosamine also makes up 50% of hyaluronic acid, the backbone on which the other GAGs, like chondroitin sulfates, are added.

"Some patients say, 'Gee, you know, I got better and it didn't take three months; I got better in a couple of weeks,'" Dr. Houpt said. "There is speculation that perhaps the shunting of glucosamine to produce hyaluronic acid to whatever hyaluronic acid does in synovial fluid may be the reason patients are improving rapidly."

Dr. Houpt said he carried out a ten-week trial but had problems using WOMAC as a measurement instrument. WOMAC scores worsened during the two-week washout period whether or not the subject had previously been on NSAIDs.

Using global WOMAC scores after eight weeks of study, the placebo group improved 9% and the glucosamine group improved 21%. But standard deviations were very large, and after adjusting for multiple comparisons, none of the 24 *p* values was significant at the 0.05 level.

However, analyzing a daily diary of pain versus pain and reports of yesterday, we have data showing a significant difference between the two groups.

Uric acid metabolism research was much easier than this. At least we had something to measure! Clearly, meta-analyses have their problems. They attempt to derive precise statistical data from studies using imprecise measurement instruments.

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# Hepatitis C

**M**ark Wener, associate professor of medicine in the division of immunology and rheumatology at the University of Washington, gave an overview of the rheumatologic manifestations associated with hepatitis C.

Hepatitis C causes a mild acute infection that develops into chronic infection in 85% of cases. The rapid mutation of the RNA encoding the coat proteins of the virus causes chronic stimulation of the immune system, which contributes to autoimmune and rheumatologic sequelae.

## RHEUMATOLOGIC MANIFESTATIONS

Dr. Wener cited a number of studies that showed correlations between hepatitis C and rheumatologic manifestations.

Pawlotsky et al<sup>1</sup> conducted a study on 59 patients with chronic hepatitis C and found rheumatoid factor in 71% of those studied. "Therefore, rheumatoid factor is clearly associated with chronic hepatitis C infection," said Dr. Wener.

Lovy et al<sup>2</sup> described 19 patients with positive rheumatoid factor or various forms of arthritis who were subsequently found to have hepatitis C. Fifteen of the patients met the American College of Rheumatology criteria for rheumatoid arthritis (RA). The patients responded to low doses of prednisone and hydroxychloroquine.

Buskila et al<sup>3</sup> examined 90 hepatitis C patients who had never received interferon which can induce rheumatoid symptoms. Rheumatic manifestations were found in 31% of patients, and included arthralgias (9%), arthritis (4%), cryoglobulinemia (11%) and sicca symptoms (8%). Myalgia was reported by 24% of patients, and 16% were diagnosed with fibromyalgia. Of all the patients, 69% possessed auto-antibodies. The most prevalent of these was rheumatoid factor, found in 44%.

There are a number of other studies that show hepatitis C to be associated with cryoglobulinemia, arthritis and sicca syndrome. Probable associations include non-deforming polyarthritis, fibromyalgia and autoimmune thyroid disease. RA has also been reported to be associated with hepatitis C.

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*Continued from page 5*

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## TREATMENT OF HEPATITIS C

The Canadian Consensus Conferences in 1993, 1994 and 1995 stated that patients with chronic hepatitis C should be treated with interferon for six months. In 1995, the American Gastroenterology Association updated this recommendation to 12 months to ensure better results with the drug.

Unfortunately, 90% of patients taking interferon experience side effects, which can include depression, neurocognitive disorder, arthralgias, radial arthritis and sometimes autoimmunity.

There is a new guanosine-like nucleoside analogue on the market called ribavirin. This drug exerts an antiviral effect for many DNA and RNA viruses. Ribavirin has shown success in combination therapy with interferon. McHutchison et al<sup>4</sup> randomly assigned 912 hepatitis C patients to either interferon or a combination of interferon and ribavirin for 24 or 48 weeks. For both time periods, the rate of sustained virologic response was higher in patients who received combination therapy. "Although this study did not focus on the arthritis population, the results were still impressive," said Dr. Werner.

## CONCLUSIONS

"Hepatitis C clearly causes cryoglobulinemia," said Dr. Werner. It is also associated with RA and fibromyalgia. Because of these correlations, "rheumatologists must determine whether they should become more acquainted with hepatitis C and the drugs used to treat it."

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## The Future of Rheumatology in Canada

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In our last issue, we looked at the history of rheumatology in Canada. In this issue, we will look at the future of the specialty. This section of the journal examines rheumatology by hearing from the most experienced members of our community.

**R**heumatology must be examined nationally and internationally. This examination must be performed objectively without emotional, local or national biases. Only then can appropriate remedies be discovered.

At first glance, rheumatology in Canada appears to be in good health, especially in terms of research and academia. But on closer examination, it becomes evident that the spectrum of rheumatologic activities in clinical practice is relatively limited and aimed mainly at inflammatory and systemic diseases. In Canada and the U.S., we practice a kind of “non-interventionist” rheumatology for which there are few diagnostic and therapeutic techniques.

European rheumatologists, those from France in particular, care for the whole musculoskeletal system. They use more diagnostic and therapeutic techniques. They are more interventionist.

In our country, the Canadian Rheumatology Association (CRA) has made great strides since the beginning of the independent annual meetings (starting with the Royal College). The number of participants has more than quadrupled and the financial position is 100 times better than it was.

Although the CRA has evolved, we have maintained good relations with the Royal College (our father). The difference is that we now make independent professional decisions. This was our first step toward adulthood.

The time has come for us to take another step and leave our parents’ house. This involves moving away from the mother, the Arthritis Society (AS), as well. Our professional objectives are not the same. The AS is a charitable agency for patients, a granting foundation for research and a source of information. We must exert our independence and stand on our own. We, the members of the CRA, must now live in a separate house.

As professionals, it is not our role to increase the wealth of the Royal College and the AS. We must collaborate with them, but our primary goal is not the development of public information, paramedical professions or parallel medicines nor is it to collect funds from the public. Our primary aim is to develop rheumatology to make it a winning specialty on all fronts, including pay scale.

It is a well known fact that in Canada rheumatology is the lowest paid specialty (or one of the lowest). Recruits take this into consideration when choosing a specialty.

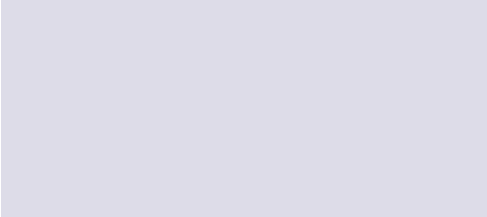
This must be one of the primary objectives of the CRA. But is certainly not the objective of the AS. The AS is doing its job, now we need to do ours.

The first Panamerican Congress (Panlar Congress), held in Toronto 25 years ago, was organized by Edward Dunlop and the AS. The AS at that time was supporting the professional activities of rheumatologists. At the XII Panlar Congress last year in Montreal, the organization and financial responsibilities were assumed by rheumatologists without the financial or technical support of the AS.

A member of the AS asked me (in my capacity as



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president of the organizing committee) if I would allow the AS to organize a dinner for their 50th anniversary. I replied that would be like shooting ourselves in the foot. During our childhood, the AS helped us grow, but hanging on to our mother's apron strings will not help our specialty and it prevents us from becoming adults.

*One of our major problems is that we have been dreamers. Other medical specialties which were less idealistic and more pragmatic and business oriented have taken over techniques that we should be managing.*

My intention is not to throw stones at the AS, far from that. Members of the CRA must take charge and be prepared to make the changes required for a bright future.

It is important to distinguish academic and university activities from the activities of the practice of rheumatology in Canada. The academic and research activities are in good health, thanks in part to the AS. But university rheumatologists are responsible for limiting our research to the inflammatory field of rheumatology instead of teaching the entire musculoskeletal system.

The council of Rheumatic Disease Unit (RDU) directors should be held at the CRA as a part of that organization or as an independent one. The grants from pharmaceutical companies should go directly to the RDU Council.

The CRA web site should not be part of the Arthritis Society's site. This is a family house. We will keep good relations with the parents (or web links) but will not let them dictate our professional objectives or activities.

Registering patients (RA and OA) for clinical pharmaceutical trials should be governed by the CRA not the AS. If we let go of our privileges and relinquish control of our professional activities, we will find ourselves at the mercy

of technocrats, as has happened in the hospitals.

One of our major problems is that we have been dreamers. Other medical specialties that have been less idealistic, more pragmatic and more business oriented have taken over techniques that we should be managing. We can change that without throwing away our idealism; let's take back our share of profitable techniques (bone densitometry, arthroscopy, echography, paravertebral, epidural and peridural injections, and so forth). To do this we may need to fight our colleagues in other specialties.

Money will be needed to achieve this. Requests for CRA grants for teaching and training will have to compete with those sometimes mercantile, down-to-earth aims of the medical specialty. The Ogrzlo fellowship could even be put under the control of the CRA since it comes from rheumatologists' donations. There will definitely be a need to incorporate a foundation within the CRA.

There is certainly a need for more rheumatologists. In Europe there are three to four times more rheumatologists than there are in North America.

*Let's change that without throwing away our idealism, let's be realistic and get back our share of profitable techniques...*

The training for residents in rheumatology should cover a larger spectrum of musculoskeletal diseases and should teach more diagnostic and therapeutic techniques. This will make specialty consultations more useful to general practitioners and will make the specialty more attractive for residents.

This may also appeal to the university rheumatologist who, because of faster or more profitable techniques, may be able to work only one or two days a week in clinic setting allowing more time for research and teaching.

Let's adapt and modify our specialty to make rheumatology a winner in the 21st century.

The 53rd Annual of the Canadian Rheumatology Association held on February 24–27, 1999, in Lake Louise and was a tremendous success. The total attendance was 275—our best ever. For the first year all fellows in training were offered a sponsorship.

The 54th Annual Meeting of the Canadian Rheumatology Association will be held again at Lake Louise, on February 23–26, 2000. Mark your calendar and watch for additional information including abstract deadlines, booking dates and so on. Watch our website address for details about the agenda (the success of last year's meeting precluded our usual rotation to Mont Tremblant for the year 2000).

Dr. Dianne Mosher will again serve as Scientific Program Chair with the assistance of Co-chair, Dr. Paul Haraoui and the Scientific Program Committee: Dr. Walter Maksymowych, Dr. Michel Zummer, Dr. Barry Koehler and Dr. John Thompson. If you have any suggestions for the program, please contact these individuals.

## CRA WEBSITE

The CRA website is located at [www.arthritis.ca](http://www.arthritis.ca). Dr. Steve Edworthy of Calgary is developing the website to meet the needs of the membership along with Dr. Kam Shojania and the committee. The website is currently closed for "construction". Dr. Edworthy would appreciate any input from the membership as to content, appearance and so forth.

## ARTHRITIS CARE IN A CMAJ SERIES

Dr. John Esdaile, Arthritis Centre Director at the University of B.C., is recruiting authors and topics on arthritis care for publication in the CMAJ. This series will be reviewed by members of the Therapeutics Committee chaired by Dr. Barry Koehler and Dr. Janet Pope. We anticipate that these series of articles will increase awareness, knowledge and perhaps skill in the management of musculoskeletal conditions.

## CRA EXECUTIVE

At the 53rd meeting of the CRA, the following members were elected to the executive. Dr. Glen Thomson, President ([ciads.novl@ibm.net](mailto:ciads.novl@ibm.net)); Dr. Dianne Mosher, Vice President ([seca089@ibm.net](mailto:seca089@ibm.net)); Dr. Simon Carette,

Past President ([scarette@ibm.net](mailto:scarette@ibm.net)); Dr. Carter Thorne, Secretary–Treasurer ([cartho@home.com](mailto:cartho@home.com)); Members at large: Dr. Arthur Bookman ([abookman@torhosp.toronto.on.ca](mailto:abookman@torhosp.toronto.on.ca)), Dr. Steve Edworthy ([edworthy@mccaig.ucalgary.ca](mailto:edworthy@mccaig.ucalgary.ca)), Dr. Paul Haraoui ([paulharaoui@ibm.net](mailto:paulharaoui@ibm.net)), Dr. Janet Pope ([janet.pope@lhsc.on.ca](mailto:janet.pope@lhsc.on.ca)), Dr. Michel Zummer ([zimmer@ibm.net](mailto:zimmer@ibm.net)), Dr. Bianca Lang, ex-officio member, President, CPRA ([blang.iwkgrace.ns.ca](mailto:blang.iwkgrace.ns.ca)).

Please feel free to contact members of the executive if you have any questions or concerns. Information about the CRA may be obtained from our administrative secretary, Christine Charnock, at [ccharnock@ibm.net](mailto:ccharnock@ibm.net).

## CANADIAN ARTHRITIS NETWORK

Dr. John Esdaile, Arthritis Centre Director at the University of B.C., is proposing the establishment of a National Registry through the Canadian Arthritis Network. He will be contacting Arthritis Centre Directors and other academics. He is also looking for interested community rheumatologists who might like to participate. Dr. Esdaile can be contacted by email at [jesdaile@bc.arthritis.ca](mailto:jesdaile@bc.arthritis.ca), or by fax at (604) 871-4501.

## CRA EXECUTIVE MEETING IN WINNIPEG, APRIL 10, 1999

Members of the CRA Executive gathered in Winnipeg with host Dr. Glen Thomson, CRA President. Deliberations included the strengths and weaknesses of the CRA, the CRA relationship with the Arthritis Society; standards of arthritis care; interactions of the CRA and the Royal College of Physicians and Surgeons; and the structure of the CRA.

## CRA SUPPORT "DECADE OF THE BONE AND JOINT 2000–2010"

Rheumatology organizations, professional societies and journals are gearing up for the decade of the bone and joint, a multidisciplinary campaign for improving the health and quality of life problems associated with musculoskeletal disorders, in particular, joint diseases, spinal diseases, osteoporosis and trauma. For information, visit [www.ort.lu.se/bjd/](http://www.ort.lu.se/bjd/) or [www.ilar.org/](http://www.ilar.org/).