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Mission Statement

The mission of the CRAJ is to encourage discourse among the Canadian Rheumatology community for the exchange of opinions and information.
A cursory look at the literature reveals an absence of pharmacoeconomic evaluations of disease-modifying antirheumatic drugs (DMARDs). This stands in stark contrast to the many cost-of-illness studies that have been conducted in recent years. One explanation for this imbalance is that cost-of-illness studies are concerned exclusively with costs. Pharmacoeconomic evaluations of DMARDs, however, must be founded in clinical reality because they will be used by decision-makers setting the therapeutic environment. The need to be clinically grounded, however, is linked to the many obstacles that stand in the way of the successful pharmacoeconomic evaluation of DMARDs. With new and possibly expensive treatments on the horizon, it will be necessary to understand these obstacles and make efforts to remove them.

WHAT ARE THE OBSTACLES, AND WHAT CAN WE DO ABOUT THEM?

For pharmacoeconomic evaluations, there must be a link between information and the individual patient. It is the individual patient who is the unit of evaluation in a pharmacoeconomic evaluation, which sounds simple, but it is not.

Many studies use the mean decrease in tender joint counts to measure the success of DMARDs. However, knowing that if a treatment decreases the tender joint count by, say, 7 on average, does not tell us how many patients benefited from the therapy. What do I tell the patient in the office who presents with 10 tender joints? Will they decrease by 7 if the patient receives the treatment? Any clinician would likely be careful not to make this sort of prediction. Preliminary response criteria have been established by the American College of Rheumatology (ACR),1 Paulus et al,2 and the European League Against Rheumatism (EULAR)3 that help solve this problem by defining response. With these definitions, we are now able to calculate the percentage of patients in whom their joint counts decreased by at least 20%, 50% or 70%. Tender joint count alone does not provide any information about the fate of individual patients and is therefore of no use in a pharmacoeconomic evaluation. However, response criteria are far from universally adopted; neither is there agreement on which criteria to use. What clinicians and researchers need to know is how many patients have adverse events that need some form of intervention and how many patients suffer from adverse events that have a significant impact on their clinical condition.

This provides practical information on how well the individual patient does when receiving a specific treatment, which is important not only for the clinician but also for a successful pharmacoeconomic evaluation. Curiously, it seems that the literature in the past decades has been guided more by statistical tools than by clinical considerations. Only now do we recognize that there is more information to be gained by classifying patients according to agreed-upon criteria that also make clinical sense than by chasing after p values alone. There is a long way to go, but with current efforts such as the Outcome Measures in Arthritis Clinical Trials (OMERACT) consensus meetings,4–6 there may be more and more clinical information that will be of use in the pharmacoeconomic comparisons of DMARDs and that will make them relevant to the clinician and policy-maker.

References
RE: SEPTEMBER EDITORIAL (VOLUME 9, NUMBER 3)

It is surprising that the editor-in-chief of an academic journal would miss important key words in a manuscript. Dr. Bookman, as an Ontario rheumatologist, was sent a clearly labeled draft copy of the guidelines for the medical treatment of musculoskeletal (MSK) diseases. More than 100 professional societies, groups, individuals and companies received this same draft copy, which explicitly requested their comments and suggested changes.

The MSK guidelines are one of a series of provincial guidelines developed over a number of years at arms’ length from government and industry. Those developed in the Centre for Evaluation of Medicines follow a rigorous, well-described process.1–3 In fact, seven eminent rheumatologists from around the province representing both academic and community practice joined family physicians, clinical pharmacologists, internists, pharmacists, epidemiologists and Arthritis Society consumer representatives to serve on expert panels to develop these guidelines.

The suggestion of undue influence by the Ministry of Health is misinformed because the ministry’s input is limited to stakeholder review along with the groups listed above. At this near-final stage, each comment received from any stakeholder is anonymously passed on to members of the panels for consideration.

Regarding specific recommendations, neither the stakeholder draft nor the final draft advise NSAIDs alone at any stage of rheumatoid arthritis. Early assessment by a rheumatology expert for consideration of DMARD therapy is emphasized throughout and advice regarding prescribing and monitoring of DMARDs is provided. Dr. Bookman also expresses concern that subdued recommendations regarding intra-articular steroids (IAS) “flew in the face of standards of care”. Standards of care tend to be individually defined whereas guidelines are meant to be systematic. As our guidelines suggest, there are surprisingly few data on the benefit and harm of IAS in rheumatoid arthritis. Short-term low-dose oral corticosteroids show benefit in small trials compared with placebo in patients with rheumatoid arthritis, thus their current Grade B recommendation.

Anne M. Holbrook, MD, PharmD, MSc, FRCPC
Chair, Ontario Musculoskeletal Therapeutics Review Panel, Centre for Evaluation of Medicines, St Joseph’s Hospital. McMaster University, Hamilton, Ontario.

RESPONSE TO LETTER FROM DR. ANNE HOLBROOK:

Dr. Holbrook may be a guidelines authority, but she is not a rheumatologist. She indicates that a large number of panelists participated in the development of these guidelines. How were these panelists selected? There were “seven eminent rheumatologists” involved. Were they directors of the RDU’s, executive members of the CRA, appointees of the Ontario Medical Association? Were there external reviewers? The criteria of selection certainly were not transparent. As government and industry are both sponsors of the project and the Centre for Evaluation of Medicines respectively, it is naive to assume that this process is entirely “at arms length”.

The fact is that these guidelines were developed without the full knowledge or backing of the rheumatology community. Although there was wide consultation on the draft format, clearly the train was well out of the station by then and rheumatologists had to respond loud and forcefully. The guidelines were not so much the issue as was the perception that policy was being set without rheumatology leadership. We think that this is a mistake.

It appears that the guidelines will be endorsed by the Ontario Ministry of Health as a showpiece to place arthritis care in the hands of family practitioners. While we have no argument over the need for better rheumatology training for family doctors, the fact is that there are studies indicating that they cannot diagnose rheumatic diseases very well.1–3 The guidelines are a map for treatment without emphasis on appropriate diagnosis. There is growing evidence that early referral of rheumatoid arthritis is vital.4 There is danger that the guidelines, by ignoring the need for diagnosis will further delay timely referral of rheumatic diseases.

Rheumatologists think that this initiative, along with that of the Ontario “Arthritis Strategic Action Group” will be detrimental to their patients unless there is a proper medical perspective. Should a non-rheumatologist be heading up the development of guidelines for arthritis care? We do not intend to sit by in silence.

A.A.M.Bookman MD FRCP<br>C edición-in-Chief, CRA Journal

References
The iris, ciliary body and choroid make up the uveal tract. The uveal tract is the middle coat of the eye, lying between the white outer covering of the eyeball (sclera) and the retina, which lines the interior of the posterior segment of the globe (Figure 1). Uveitis is an inflammation of any part or all of the uvea. Panuveitis is when the entire uvea is inflamed. Iritis (iritocyclitis) inflammation of the iris and ciliary body. Iritis is characterized by a breakdown of the natural barrier between the blood vessels and the fluid (aqueous humor) that fills the space between the iris and the cornea (the anterior chamber). As a result, protein, fibrin, and white blood cells enter the aqueous. The cells and debris circulating in the anterior chamber can be deposited on the inner (interior) surface of the cornea (the corneal endothelium). These deposits, called keratic precipitates (KP), can be fine or take the form of larger greasy clumps called mutton-fat KP. A distinction is made between non-granulomatous iritis (with fine KP) and granulomatous iritis (with mutton-fat KP). Although more severe uveitis, including panuveitis, can be a manifestation of juvenile rheumatoid arthritis (JRA), it is the non-granulomatous iritis that is most characteristic of JRA.

Chronic anterior non-granulomatous uveitis in children is most often caused by JRA (known as juvenile chronic arthritis [JCA] in the European literature). Roughly 6% of all cases of uveitis occur in childhood and of these, 80% are associated with juvenile rheumatoid arthritis. The incidence of iritis in patients with JRA ranges from 8% to 24%, but certain subgroups are at high risk. Of all cases of JRA-associated uveitis, 78% to 90% will have pauciarticular JRA (and 90% of these will be antinuclear antibody [ANA] positive and rheumatoid-factor negative), 7% to 14% will have polyarticular disease, and 2% to 6% will have systemic JRA (Stills’ disease). Young girls with pauciarticular onset, ANA seropositivity and rheumatoid factor negativity are most likely to develop uveitis. Many children who are rheumatoid factor positive and have polyarticular disease uncommonly develop uveitis. The majority of children who develop uveitis do so within four to seven years of the onset of JRA; the peak is within two years of onset. The disease is bilateral in 75% to 80% of children.

Common symptoms and signs of other forms of iritis are pain, photophobia, and red eye; however, the anterior uveitis seen in JRA is almost entirely asymptomatic. To detect uveitis, an ophthalmologist must examine the anterior chamber aqueous with a slit lamp to see if cells, keratic precipitates and/or proteinaceous exudate (called flare) are present. Because flare can persist despite treatment in some children with JRA, the focus of treatment is to eliminate cells. Even low-grade uveitis left untreated can lead to ocular damage including retinal swelling (macular edema), glaucoma and cataract. Up to 10% of patients with JRA will develop iritis before joint disease.
There is no correlation between joint activity and eye disease. To prevent ocular damage, children with JRA must be screened regularly by an ophthalmologist who is familiar with the disease and comfortable performing thorough eye examinations on sometimes-uncooperative children.

Guidelines have been suggested for screening children with JRA (Table 1). There remains controversy, however, over the frequency of screening; some suggest frequency should be independent of the risk of developing uveitis, and that screening should be at least every six months, regardless of the subgroup of disease. We prefer to err on the side of the more frequent follow-up examinations particularly in children at higher risk. Note that risk is determined by the number of joints affected in the first three to six months of the disease.

Even though many children with pauciarticular onset develop polyarticular disease, it is the original presentation as pauciarticular that confers a higher lifetime risk. Some authors have suggested decreasing screening frequency seven years after onset in children older than seven if an eye disease has never occurred.

The mainstay of treatment is corticosteroid medication, which can be administered topically, by regional injection, orally or parenterally. The first-line treatment is usually topical steroid drops or ointments. Administration as often as every hour may be needed to control the inflammation. Although many children will tolerate a short course of treatment followed by a rapid tapering of their eyedrops, approximately 10% to 15% of children with JRA iritis will require topical steroids to be tapered slowly. In our experience, tapering too quickly is the most common cause of recurrent chronic disease. Some children may require tapering that takes many months, continuing with doses as low as one drop per week. It is this unique feature of JRA that makes it essential that children be cared for by an ophthalmologist well versed in this disease.

If the adverse effects of steroids such as glaucoma do not develop, a periocular injection may allow for reduction in topical dose frequency. The injection, which in young children may require a general anesthetic, is given in the sub Tenon’s space. Tenon’s fascia lies outside the eyeball underneath the conjunctiva. In more resistant cases, oral or parenteral steroids may be needed. When steroids fail or are poorly tolerated, methotrexate (oral or subcutaneous) can be a valuable adjunct. Nonsteroidal anti-inflammatory medications appear to have a limited role both topically and orally. Other immunosuppressive medications might sometimes be necessary.

During active uveitis, a topical dilating drop is usually used to prevent scar tissue (posterior synechia) from forming between the iris at the pupil to the lens. These synechiae may cause the border of the pupil to stick down to the lens causing an irregular and poorly dilating pupil.

The main ocular complications of JRA-associated iritis are cataract, glaucoma and macular edema. Despite all the advances in the management of this condition, up to 22% of children with pauciarticular...
JRA-associated uveitis still develop legal blindness secondary to chronic low-grade intraocular inflammation. The prognosis is more favourable for males, for those with unilateral disease, for older children, for those in whom the disease is milder at presentation, and for children without posterior synechia or cataract at presentation. Cataract and glaucoma can both also be caused by steroids. When vision is threatened, cataract may require surgery, although this can further aggravate iritis. Therefore, attempts are made to delay surgery when possible until the iritis is well controlled. Unlike adults, intraocular lens implantation in children with JRA can have serious complications. Glaucoma can occur in children who have had cataract removal and in those who have not. It is difficult to treat and a combination of medication (topical and systemic) and surgery are usually needed. Glaucoma may be due to clogging of the eye’s drainage system (trabecular meshwork) by cells and protein in the aqueous, excessive posterior synechia blocking the flow of aqueous through the pupil, or synechia which cause the iris tissue to scar over the trabecular meshwork. Macular and optic nerve edema are very difficult to treat and often lead to irreversible loss of vision.

JRA-associated uveitis is a chronic asymptomatic condition that can lead to severe blinding ocular complications. Early detection through routine screening and appropriate management is essential.

TABLE 1
SCREENING GUIDELINES FOR EYE EXAMINATIONS IN CHILDREN WITH JRA†

<table>
<thead>
<tr>
<th>Onset:</th>
<th>American Academy of Pediatrics ‡</th>
<th>Kanski †</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pauciarticular at onset &amp; ANA positive</td>
<td>3–6 months*</td>
<td>6 months</td>
</tr>
<tr>
<td>Pauciarticular at onset &amp; ANA negative</td>
<td>6 months</td>
<td>6 months</td>
</tr>
<tr>
<td>Polyarticular onset &amp; ANA positive</td>
<td>3–4 months*</td>
<td>6 months</td>
</tr>
<tr>
<td>Polyarticular onset &amp; ANA negative</td>
<td>6 months</td>
<td>6 months</td>
</tr>
<tr>
<td>Systemic onset</td>
<td>12 months</td>
<td>12 months</td>
</tr>
</tbody>
</table>

† Suggested frequency of visits for a child without a history of iritis.
‡ After four years and no eye involvement, decrease to 6 months.

References
Osteoarthritis Guidelines: A Step-wise Approach

In 1995 the American College of Rheumatology (ACR) published guidelines for the medical management of osteoarthritis in two parts: osteoarthritis of the hip\(^1\) and osteoarthritis of the knee.\(^2\) The guidelines state that the goals for the management of osteoarthritis of the hip are to control pain, limit disability and educate the patient.

Hochberg et al\(^1,2\) outline the use of pharmacologic agents (acetaminophen, NSAIDs, opioid analgesics) and nonpharmacologic modalities including patient education, physical and occupational therapy. An individualized plan for each patient is recommended. The guidelines do not include specific recommendations for surgical therapy.

Pharmacologic therapy is recommended for pain relief and acetaminophen (up to 4000 mg/day) is cited as the initial drug of choice. NSAID use is recommended if adequate pain control is not achieved with acetaminophen. The recommended use of NSAIDs is given in two steps, beginning with low-dose ibuprofen or nonacetylated salicylates and then if necessary using full-dose NSAIDs. NSAIDs are not advocated as first-line therapy primarily because of concerns about toxicity, specifically upper gastrointestinal (GI) bleeding and renal failure. Strategies for decreasing the risk of upper GI bleeding are not described in detail in the guidelines but misoprostol is cited as the only drug approved for prevention of GI complications.

If NSAIDs do not provide adequate pain control, consideration of referral for joint surgery is recommended. Opioid analgesics are only advocated for the short-term treatment of acute exacerbations of pain, and long-term use is cautioned.

The guidelines for the management of osteoarthritis of the knee are identical in many respects, including the goals of management and nonpharmacologic therapy modalities. Recommended pharmacologic therapy is similar to that of osteoarthritis of the hip and includes nonopioid oral and topical analgesics, NSAIDs and the careful use of intra-articular steroid injections.

Nonpharmacologic modalities such as patient education, weight loss, and physical and occupational therapy are recommended as initial interventions. Patients who have knee effusions or other signs of local inflammation may be considered for injection of intra-articular steroids.

As in the guidelines for OA of the hip, acetaminophen is the drug of choice for first-line therapy. For additional pain control, topical capsaicin cream is suggested. If these measures do not provide sufficient pain relief, low-dose and then full-dose NSAID therapy is recommended. Further therapy for pain unresponsive to medical therapy could include joint surgery or in those patients for whom surgery is contraindicated, joint lavage or arthroscopic debridement.

In summary, the guidelines provided by the ACR give a step-wise approach to medical management and outline the important role of nonpharmacologic treatment modalities while emphasizing the need for an individualized approach for each patient.

References
he results of many new osteoarthritis (OA) studies were presented at the American College of Rheumatology (ACR) meeting in Boston in October 1999. In the past, we have “inverted the pyramid” in the treatment of rheumatoid arthritis (RA); perhaps it is time to invert the pyramid in the treatment of OA. Guidelines often recommend treatment with nonpharmacologic agents in symptomatic OA, which is quite appropriate. However, most guidelines describe first-line treatment with acetaminophen, up to 4 g per day. Data are lacking on the long-term efficacy and safety of such large doses of acetaminophen. The long-term compliance at these high doses is also unknown.

Many studies presented at the ACR meeting demonstrated the preference of nonsteroidal anti-inflammatory drugs (NSAIDs) over acetaminophen. One study was conducted on patients with moderately symptomatic OA who had only partially responded to acetaminophen (1.2 g to 4 g per day). When these patients switched from acetaminophen to NSAIDs, including rofecoxib, they showed greater improvement. Patients with OA who were not “acetaminophen treatment failures” were randomized to receive either acetaminophen, 4 g per day, or other NSAIDs. The preference was for NSAIDs. Even more striking were the results of other studies.

Dr. Roy Altman presented the results of a study in which patients were randomized to either ibuprofen (1.2 g per day) or acetaminophen (4 g per day) for six days. The preference and all outcome measures supported the use of ibuprofen. In a crossover trial of OA, patients were randomized to receive either the NSAID Arthrotec (75 mg bid) or acetaminophen (1 g qid). For the 180 patients who completed the study, Arthrotec provided a better quality of life.

Wolfe et al surveyed patients with fibromyalgia, OA or RA to determine whether patients preferred NSAIDs or acetaminophen. Even when factoring the frequency and degree of side effects, OA patients preferred NSAIDs over acetaminophen most of the time.

Many guidelines do not discuss the use of intra-articular injections, in particular the use of viscoelastic substances. Sripada et al presented results that suggested that viscoelastic with higher molecular weights injected intra-articularly into the knee performed better than non-cross-linked substances.

When selecting treatment for OA, it is imperative to look at factors that prevent progression of the disease. Exercise and weight loss certainly can improve symptoms over time, but recent studies have been looking for drugs that can modify disease. Preliminary results of a trial of doxycycline in the treatment of OA were presented. However, the potential disease-modifying drug properties are too preliminary at this point in time.

Reginster et al presented exciting new three-year data on glucosamine sulfate in the treatment of symptomatic knee OA. In this large multicentre double-blind randomized controlled trial, glucosamine sulfate (1.5 g per day) was compared with placebo. Weight-bearing antero-posterior (AP) knee joint radiographs were performed at 0 and 3 years. The total knee joint space widths were measured by radiologists who were unaware of the treatment allocation. (There is debate about whether this is the best way to measure cartilage thickness in a noninvasive fashion. However, this was the state of the technology when the trial began.) There was no average progression of OA in the glucosamine-treated group (and in fact there was a mean improvement in joint space in the AP knee standing) compared with placebo. In the placebo group there was worsening on average of 0.08 mm to 0.1 mm per year of joint space. In the glucosamine group there was actually improvement of joint space. The concomitant medications in both groups were similar (NSAIDs and acetaminophen).
However symptoms were slightly better in the glucosamine group than in the placebo group. At the end of three years there was slight improvement in OA (as measured by joint space improvement) in patients receiving glucosamine. These results should revolutionize our treatment of OA in the prevention of disease progression. A study of chondroitin a couple of years ago demonstrated equally impressive results.7

The use of intra-articular steroids in knee OA have often been reserved for patients with prolonged morning stiffness, joint effusions, warmth and gelling (all features of an inflammatory component of their OA). However, it may very well be that intra-articular steroids also decrease metalloproteinases, which are damaging to cartilage. Studies in humans are necessary to determine if intra-articular steroids have great efficacy in noninflammatory OA. My clinical observations suggest that this is certainly the case.

In the future, cartilage may very well be regenerat-ed and, using arthroscopic surgery, routinely implant-ed back into young patients who have detectable early OA. At this point in time though, this is not a standard early treatment of knee OA.

It is definitely time for the guidelines for the treatment of symptomatic OA of the hip and knee to be revised to include potentially disease-modifying drugs that may reduce disease progression including glucosamine and possibly metalloproteinase inhibitors.

References
An Interview with Dr. Maurice Campbell

The Journal of the CRA is looking at the history of rheumatology in Canada through the eyes of its elders. We will reflect on the history of this specialty by interviewing the most experienced members of our community.

Dr. Campbell, what encouraged you to choose rheumatology as a career when so little was known about the specialty?

I had been a general practitioner for six years and my friend, an internist and cardiologist, had been encouraging me to become a specialist. I had two children, and one on the way, and I began to seriously consider this option.

In 1953, I applied to the Hôtel Dieu Hospital in Montreal, Quebec requesting permission to complete the requirements of the Royal College of Physicians (two years of training in internal medicine). I knew that if I wanted to become a specialist, this was my opportunity. I applied and was accepted. My interest in rheumatology was sparked by the doctors at the Hôtel Dieu and I have since had a wonderful career, I have thoroughly enjoyed my decision and I have no regrets.

What was the state of rheumatology when you first started?

Rheumatology was very new and was not yet recognized by the Royal College of Physicians. Certification was therefore not available to rheumatologists. This changed in Quebec in 1955. Our numbers were increasing and we were forecasting an increase in patients as well. It was a very exciting time for rheumatology. When we received certification, there were more than 15 rheumatologists. My practice had initially been equally divided between rheumatology and internal medicine. I received an influx of referrals once I was certified and this breakdown rapidly shifted to about 90% rheumatology and 10% internal medicine. For 24 years, I was the only rheumatologist in Trois Rivières, Quebec. My territory covered a radius of approximately 60 km with 250,000 people. It kept me very busy and truthfully, a little lonely at times. It can be difficult to be the only rheumatologist. I did not have colleagues to discuss my work with or toss around ideas.

How has the practice of rheumatology evolved (in academic or community settings) since you started?

The practice of rheumatology has made tremendous progress. When I first started training, 75% of patients became disabled or completely incapacitated. The inverse is true today. I have been fortunate to witness this evolution. Developments in treatment options have been particularly exciting. Every five years there are amazing changes in medications and treatments. The first non-
teroidal anti-inflammatory drug, for example, became available in 1954 when I was training at the Hôtel Dieu. Cortisone was being used as well and I was involved in some of the first clinical trials of prednisone. It was a fantastic time.

Osteoarthritis of the hip used to make rheumatologists feel useless; there was nothing we could do. And then in the late 1950s and early 1960s, news of reconstructive surgery was released. New treatments in physiotherapy were also developed. New lab tests came out and we were suddenly able to detect diseases such as arthritis or lupus. We could rely on procedures to help find our way around the jungle of rheumatic diseases.

**Q** How do you see the future of rheumatology evolving in Canada?

Rheumatology is a poorly understood specialty and it is not fully appreciated by other physicians and internists. Rheumatology is also poorly understood by the public and most people do not know what rheumatologists do.

Rheumatology requires superior interpersonal skills and a lot of brain work. Patients must be thoroughly questioned and treatment options must be determined on the basis of their answers. Rheumatologists are not sufficiently compensated for this work. I believe this is why there are not enough rheumatologists in Canada. Rheumatology is a widely misunderstood profession and unless something is done about this, the trend will continue.

**Q** Would you choose to become a rheumatologist today?

Yes, definitely. Even with the problems today of government involvement in medicine and medicare. I would probably be very impatient and angry because when I started there was free enterprise and we did not have to deal with all of these headaches. I would choose to become a rheumatologist again because I have such pleasant memories, I would want to repeat them. I have thoroughly enjoyed my career and I would highly recommend this profession to others choosing a specialty. I was very fortunate to have devoted competent teachers at the Hôtel Dieu. Their enthusiasm had a profound influence on me and I hope that I will have the same effect on others.
ANNUAL GENERAL MEETING AND SCIENTIFIC PROGRAM
The CRA AGM and Scientific Program will be held at the Chateau Lake Louise, Lake Louise, Alberta, February 24–26, 2000. Dr. Dianne Mosher is the Chair and can be reached by email at seca089@ibm.net. The Annual General dinner and awards night will be held on Thursday evening and the Guest Lecturer will be speaking on Saturday morning. Please mark the AGM on your calendar and watch for more details.

Highlights of the meeting will be the presentation of the CRA Awards, including Distinguished Rheumatologist, Distinguished Investigator and Young Investigator. Symposia organized by our Diamond sponsors will be held pre- and post-meeting: Hoechst Marion Roussel will sponsor a pre-meeting symposium on Wednesday, February 23, 2000; Searle Pfizer will be organizing a post-meeting symposium to be held on Saturday, February 26, 2000. Further details will follow in separate mailings.

Be prepared for a more balanced program of education and alternative activities; attendees will be invited to participate in a Celebrity Ski Race with proceeds to the Ogryzlo Fellowship.

The next AGMs will be held in February 2001 at Mont Tremblant, Quebec, and in conjunction with ILAR, in August 2001 in Edmonton, Alberta.

REGISTER EARLY FOR CRA MEETING 2000
All CRA members who pre-register for the Lake Louise meeting prior to February 1st will be eligible for a $500 draw at the CRA meeting at Mont Tremblant in February 2001.

FELLOWS IN TRAINING
Reminder: sponsorship to attend the AGM and Scientific Program is available. Please ask your Arthritis Centre Director for details; they have been sent the guidelines. Also, apply for your CRA “Member-in-Training” status. Applications for membership must be submitted on an annual basis and are not renewed automatically.

ROYAL COLLEGE UPDATE
The CRA Executive is requesting input from its membership on two issues concerning the RCPS.

1. Should the CRA, through the sub-specialty (chaired by Carol Yeadon), initiate a process to establish rheumatology as a “core specialty?”

2. What is the role of the CRA in continuous professional development (CPD) and program development accreditation?

   Please contact Paul Haraoui (paulharaoui@ibm.net) or Paul Davis (PaulDavis@ualberta.ca) with your comments or suggestions.

ARTHITIS CARE STRATEGY UPDATE
The “Arthritis Strategic Action Group” (discussed in the September 1999 issue of CRAJ) has not yet published its report and recommendations to the Ontario Minister of Health.

REINSTITUTION OF RADIOACTIVE SYNOVECTOMY YTTRIUM
Elizabeth Witmer, Ontario Minister of Health, has responded in a letter to the CRA, that she believes the withdrawal of radioactive synovectomy yttrium is a “local issue” for the Hamilton Health Sciences Corporation. In a follow-up, the minister was reminded that the cost of supplying the product to more than 300 Canadians with arthritis in the year preceding the withdrawal of product was approximately $70,000. At the current price from a foreign source, the cost is approximately $325,000 per year. The Executive awaits a response from the minister.

COMMITTEES
The following are the CRA committees with corresponding contacts.

Scientific Program Committee—Dr. Dianne Mosher (tel: 902-422-1170; e-mail: seca089@ibm.net)
Communications—Dr. Arthur Bookman (tel: 416-603-5404; e-mail: a.bookman@torhosp.toronto.on.ca)
Economics & Manpower—Dr. Michel Zummer (tel: 514-252-3804; e-mail: zummer@ibm.net) and Dr. Jamie Henderson (e-mail: jhenderson@health.nb.ca)
Therapeutics—Dr. Barry Koehler (tel: 604-273-8085; e-mail: bkoehler@ibm.net) and Dr. Janet Pope (e-mail: janetpope@lhsc.on.ca).

Please feel free to contact members of the executive if you have any questions or concerns. Information about the CRA can be obtained from Christine Charnock, administrative secretary, at ccharnock@ibm.net.