Correspondence

INSTRUCTIONS FOR LETTERS TO THE EDITOR

Editorial comment in the form of a Letter to the Editor is invited; however, it should not exceed 800 words, with a maximum of 10 references and no more than 2 figures or tables and no subdivision for an Abstract, Methods, or Results. Letters should have no more than 3 authors. Full name(s) and address of the author(s) should accompany the letter as well as the telephone number and fax number (if available). Financial associations or other possible conflicts of interest should always be disclosed. To expedite receipt of letters, we encourage authors outside Canada to communicate by fax (416-967-7556).

Fibromyalgia and the Law

To the Editor:

Having read the case report by Mailis, *et al*[1] as well as the accompanying editorial by Wolfe[2] I am compelled to point out some things that were not made clear or were omitted from the discussions. With regard to the "psychosocial/personality factors" alluded to by the authors of the case report, conspicuous in its absence is the discussion of the personality and/or bias of the judge in the case. While not a Canadian I have read about personal injury cases in Canada and it is my understanding that personal injury cases are often adjudicated solely by a judge with no jury. I prefer the latter since in a jury trial a biased judge can obviously have influence over the case but not total control. A judge unwilling to accept the concept of fibromyalgia (FM) and the proposition that patients can have soft tissue pain many years after trauma will evaluate the medical evidence much differently than a disinterested third party (i.e., jury *and* judge). Furthermore, having to wait 9 years for a dispute resolution says a lot more about the inefficiency of the judicial system than about the vagaries of the case in question. I am confused as to exactly what happened in this case. Was there an award by the court, as suggested in the abstract, or a "settlement" as mentioned in the text?

Mallis, *et al* are also vague about the Vancouver Consensus Conference. I attended that conference in June 1994. When the attendees voted that "there are insufficient data at this time to establish a causal relationship between FM and trauma," that was in reference to what was known in 1994 as opposed to the year 2000 of the case report. In the ensuing years, however, several studies[3-5] have linked trauma and FM in a cause/effect relationship. If a vote were held today, I am certain that it would be different. One of the reasons I am so certain is that Dr. Wolfe, the Chairman of the Vancouver Consensus Conference, wrote in 1997 "... trauma may cause FM ..."[6], as opposed to the view in 1994.

The commentary by Dr. Wolfe[2] is certainly noteworthy but it raises more questions than answers. Dr. Wolfe described a "recent case" about a patient who was malingering and concluded that "everyone got it wrong," referring to the doctors who had evaluated the patient. No one doubts that there are dishonest people in this world and that we doctors are not immune to being victimized. However, isn't it also fraud when an insurance company does not fulfill its obligations regarding payment for needed health care? Aren't downcoding of bills for medical procedures and delays in payment fraud as well? Suspicion and/or blame placed on innocent patients whose behavior may not be easily understood is misdirected. Why are we so focused on people accused or suspected of being malingerers as opposed to turning our attention to individuals who truly exhibit criminal behavior? Governmental agencies try to adhere to budgets and insurers reap enormous profits while denying needed health care to sick and suffering individuals! According to a recent study, the average reimbursement for a CEO of a health care organization in the United States was $2 million[7]! Who is the bigger criminal? Who is responsible for hoodwinking more individuals, the malingerer who fools a few doctors or an insurance company that attempts to hoodwink an entire society?! To Dr. Wolfe I have to say: "Welcome to the real world!" There are sincere patients. There also are crooks who pose as patients. The best we can do is evaluate each patient and if necessary give an opinion based on our education, training, experience, and the facts of the case. No reasonable physician will deny that some patients suffer chronic pain often of a widespread nature after trauma[8]. To approach such individuals as if they were malingerers is a betrayal of the Hippocratic oath and makes us agents of the government and of the insurance companies. To whom do we owe our allegiance? Certainly it is to society as a whole, but it is also to our individual patients. It is my understanding that the legal standard is "to a reasonable degree of medical probability" with regard to causation. That is, is it more likely than not that the trauma caused or precipitated the patient's FM? To put it another way, were it not for the trauma would the patient have developed FM? Certainly no one can answer this question in the abstract except to comment on the possibility of such an association. As a Fellow of the American College of Physicians and a member of the American Medical Association I have reviewed the ethics manuals for both organizations[9,10]. To paraphrase them, if the physician has knowledge that can benefit the court, it is his duty to participate in the legal proceeding and be as objective as possible. Are doctors fooled sometimes? Do malingerers pose as patients from time to time? Do we live in an imperfect world? The answer to those questions, of course, is yes. It is important in reading Dr. Wolfe's editorial and the case report of Mailis, *et al* that we not throw the baby out with the bath water by placing blame on patients who may be injured in ways that we do not completely understand, but who are injured nonetheless.

Wheeling, West Virginia, USA. Thomas J. Romano, MD, PhD, FACP.

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To continue

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to next page

To the Editor:

The editorial by Wolfe[1] and case report by Mailis, *et al*[2] point out, at least in some extreme cases, how the tort system for motor vehicle injuries may be detrimental to the accident victim's health and recovery and encourage illness behavior, while compensation payments are subsequently consumed by the lawyers.

While the extreme cases reported by Mailis, et al draw attention to the problem because of the years of litigation with nothing to show, there is recent evidence that the concerns expressed apply also to the more typical claims of shorter duration, and with a less dramatic outcome.

Cassidy, *et al*[3] reported on the "social experiment" in Saskatchewan, Canada, that allowed observation of the effect on recovery from whiplash injury of changing the compensation system. In Saskatchewan, with a single insurer (Saskatchewan Government Insurance), the province's tort system for compensation changed on January 1, 1995, to a no-fault system. Under the tort system, of course, those in motor vehicle accidents could sue for compensation for pain and suffering in addition to treatment costs, lost wages, and other pecuniary expenses. With the change to a no-fault system, payments for pain and suffering were eliminated, and medical and income replacement benefits were increased. This resulted in very few lawsuits, although a suit was still possible in cases where medical costs exceeded a designated threshold or if the annual income replacement was over $50,000 Cdn.

Claims were evaluated and followed for the last 6 months of the tort period, and for 2 consecutive 6 month periods in the new no-fault period. Despite the fact that there were more collisions and more kilometers driven during the no-fault periods, there was a decrease in total whiplash claims by about 30%. Most striking, however, was that the median time from date of injury to recovery (as measured by the surrogate marker of claim closure), was reduced from 433 days in the tort period to about 200 days in the two 6 month periods of no-fault. In both the tort and no-fault periods, Cassidy, et al found, by using Medical Outcome Survey Short Form-36 and other measures of health, that the time of claim closure was consistently a close correlate for recovery from the injury.

Proponents of the tort system still argue that the potential for delay in recovery is "compensated" for by the increased delivery of monetary recompense to the client. This is likely a myth. Lemstra[4] reveals, for example, data from the Insurance Research Council (IRC) survey of claim costs and compensation to the claimant in the United States. The IRC compared the compensation and disability costs of claims in subjects represented by a lawyer versus those not represented. Looking at all motor vehicle injuries combined, represented clients had average economic losses of $6391 in comparison to $1755 for nonrepresented clients. Furthermore, average gross bodily injury payments for the represented claimants were $11,939 compared to $3262 for claimants without an attorney.

One is tempted to interpret these figures as meaning that those accident victims using a lawyer had more severe injuries and were compensated more. Yet, once attorney fees, court costs, and economic loss to the client are subtracted, represented clients only received a net payment of $1608 in comparison to $1507 for clients without a lawyer. In other words, the client represented by a lawyer gained only an additional $101 for their pain and suffering, while the "system" (lawyers included) received the rest. Looking specifically at whiplash injury without fracture, the IRC data show that those represented by an attorney (and who should thereby benefit by receiving maximum compensation — as so often promised as one of the perks of representation) received less net compensation after expenses than those not represented by an attorney. This, of course, ignores the issue of whether increased financial compensation, even if present, can ever provide adequate recompense for the extra pain and suffering claimants go through to receive it[3].

Thus, representation by an attorney benefits the attorney and the "system," but does not substantively increase (and in many cases reduces) net compensation to the accident victim. If seeking representation has even a small effect on recovery (and the Saskatchewan data indicate that the effect is actually rather large), and if representation reduces net compensation after all the players have taken their piece of the pie, the accident victim truly becomes a victim — not so much a victim of the accident but more a victim of the system.

Edmonton, Alberta, Canada. Robert Ferrari MD, FRCPC; Anthony Russell, MD, FRCPC.

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Drs. Mailis and Taylor reply

To the Editor:

In his editorial, Dr. Wolfe concluded that uncertainty about fibromyalgia (FM) comes from things we do not know or cannot determine: full nature of trauma, science of kinetics, the variability of human system to trauma and stress, etc[1]. However, this is only partially true. Beyond the reasons he has cited, variability about FM comes from a multiplicity of sources. Accordingly, we take the position that future research is necessary to reduce the uncertainty:

FM describes a multisystem complex, but to date it has not been found to have definable patho or psychophysiology. Nevertheless, despite the phenomenal absence of etiological consensus, FM has been raised to a substantial cause of disability and has become the subject of multiple debates, even within the ranks of rheumatologists[2]. We feel that any chronic pain disorder defined solely by its symptom complex and distinct "tender points" defies the concept of good Pain Medicine. Since pain is a multidimensional phenomenon, in addition to biological factors, one should not ignore potential psychological/ personality and socio-environmental contributions. Only a concerted approach based on the biopsychosocial model of illness will help better define FM.

For example, it is interesting that a recent study reported 14–22% prevalence of chronic widespread pain in the general population by using the American College of Rheumatology criteria for FM (pain > 3 months, in at least 2 contralateral quadrants and the axial skeleton). The overall prevalence of mental disorder in the chronic widespread pain group was 16.9% versus 6.5% in those without diffuse pain[3]. Studies like these should drive research towards determining the interplay between psychological factors and chronic widespread pain.

Furthermore, the role of "coaching" and "modeling" effects that can be seen within family units (as found in our own case report of a family of 6[4]), support groups, etc, should be explored and properly researched.

Last but not least, the role of central factors (possible central nervous system sensitization, attentional issues, etc) merit special attention.

Moreover, new consensus criteria based on considerably more than just "tender point" detection should be established and their validity tested. Ten years after publication of classification criteria for FM[5], the need for new criteria is overwhelming. It is possible that stricter and tighter criteria can allow FM to move from a disease "without borders" to a more definable (hence more respectable) entity.

Unfortunately, the "manufacturing of victims" following minor traumas such as we reported[4] is not confined to FM. Indeed, many members of the health care community contribute. One example is psychologists who, as in

this case, diagnose persistent post- concussion syndrome or acquired brain damage, wherein ubiquitous symptoms pertaining to "loss of energy, reduced concentration and memory, depressive affect," etc, are attributed to "acquired brain injury." In the absence of any objective evidence (neurological examination, radiology), the mere reporting of these symptoms often suffices for litigation purposes, particularly when supported by (controversial) neuropsychological test data. Unfortunately, as with FM, cognitive disability frequently exceeds what should be expected based on medical parameters, e.g. uncomplicated whiplash, and can even worsen over time (contrary to true brain damage that may improve over time). Patients and lawyers informed of "brain damage" are then "entitled" to base lifestyle and compensation demands on a fragile thread, resulting in additional examinations and opposing views. Cases that could be quickly resolved become interminable and highly costly to patient well being, to the courts, and ultimately, to the public.

Our case is not a mere constellation of "our perception bias and beliefs." Dr. Wolfe stated that "we have no reasons to believe that the opposing psychiatrists and pain experts are any less secure in their biases and beliefs than Mailis, *et al*". We beg to disagree with this comment.

Unfortunately for the family and their lawyers, the court felt that a solid line of facts (overwhelming evidence of subjective symptoms, unusual family pattern of mimicked symptomatology, delayed onset of symptoms following the accident, good academic performance of the children through school records, and the ability of the parents to learn new skills) defied the diagnoses of FM and acquired brain damage.

However, we would like to agree with Dr. Wolfe that such a case should make us all think about the law and symptom defined complexes so that it is less likely that another such case will happen again.

Dr. Romano's letter is interesting because he takes the position that (a) FM per se is "victimized" by our report[4] and Wolfe's editorial[1], and (b) it constitutes a plea for physicians "to be nice to patients and give them the benefit of doubt." He is also questioning our legal process, considering that a biased judge can influence the case, and that the waiting time of 9 years for the case resolution shows the inefficiency of our judicial process. To this, we can say the following: (1) In the Ontario jurisdiction, personal injury plaintiffs have the option of choosing to be heard by a judge alone or a judge and jury. Obviously, since the family (through their legal counsel) chose a judge, they must have felt that this was going to result in less "bias." (2) The 9 year wait can very easily be explained not by the inefficiency of our judicial system but by the number of plaintiffs and "doctor shopping" in search of some medical condition to explain the family's host of physical, cognitive, and emotional symptoms. (3) In terms of the settlement, while we did not clarify this in our paper, it was not an "award" from the court at the end of a trial, but money granted when the plaintiffs' lawyers conceded during the actual trial and accepted a miserable settlement. (4) As far as our paper is concerned, the issue of malingering is moot. Our focus was on the "fostering of illness" through support of questionable diagnoses, which to date lack sufficient "evidence based data" to survive the adversarial process. (5) The discrepancy between subjective complaints and real-life function in the members of our reported family (which should have been "caught" years earlier by the physicians) obviously seemed unacceptable to the court. It is one thing to give patients "the benefit of the doubt" and another to see thorough documentation of complaints in work and school records (something patients' physicians do not usually seek, but which practitioners conducting independent forensic examinations have access to).

While Dr. Ferrari acknowledges that our paper draws attention because of the many years of fruitless litigation, he quotes recent evidence of similar encouragement of illness behavior and prolongation of recovery within the tort system for motor vehicle accidents in more typical claims of shorter duration and less dramatic outcomes. His well referenced message is that the presence of lawyers in a tort system can extend the time-to-case resolution in the absence of (comparative) financial gain. What Dr. Ferrari might not know, is that the first author of the recent Saskatchewan study[6], Dr. Cassidy, is now under direct attack. *The Medical Post*[7] quotes the May 22, 2000, issue of *National Law Journal*, which opposes the study. *The Medical Post* warns: "Meanwhile, Dr. Cassidy...worries that harsh fallout from studies with controversial conclusions could have a chilling effect on researchers.... And he does not want to see researchers cowed by pressure groups who don't like their findings."[7]

Our paper focused on the patient aspect of prolonged symptomatology, which ultimately "cost" years of suffering plus the additional cost of compensation failure. In our case report, the patients were victims. The community (courts, premium insurance costs, etc) was a victim. Fibromyalgia and acquired brain damage as entities were victims because their existence was (rightly so) not believed.

Dr. Ferrari's letter reminds us that research too (even conducted by reputable investigators and published in a renowned journal after thorough scrutiny[6]) has also become a victim. And when the research community is under attack, this is truly scary!

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Toronto, Ontario, Canada.

Angela Mailis, MD, MSc, FRCPC (PhysMed);
Ann Taylor, PhD (Psychol).

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To the Editor:

To me, Dr. Wolfe's recent editorial[1] read like a legal brief instead of a medical commentary. Dr. Wolfe is a rheumatologist, not an attorney; yet he describes his views on the medicolegal aspects of fibromyalgia (FM) and trauma and discusses legal proceedings and court cases. He concludes that answers regarding trauma and FM are not known, and perhaps doctors are being deceived by the patient/plaintiffs who report chronic pain following an injury. To support his opinions, he offers as "evidence" a tragic family of FM sufferers, the lack of a compensation system in Lithuania, and an amazing surveillance videotape that reputedly showed a healthy individual with no medical abnormalities whatsoever.

Dr. Wolfe surmises that medicolegal uncertainties are further clouded by physicians' perceptions, expectations, inclinations, and biases and concludes that rheumatologists, other physicians, and health workers are no more skilled than others in evaluating the relationship, subsequent illnesses, and allegations of disability. He will have us believe that given the lack of absolute proof that trauma causes FM, and the lack of any special skills among physicians, perhaps FM doesn't exist at all, any injured person who reports chronic pain is a malingerer, and any physician's opinion that FM from a trauma exists is no more credible than the medical opinions of our local mail carriers.

I'm not sure if Dr. Wolfe has seen actual patients in a medical office recently, but I feel compelled to point out that the practice of taking a patient history, performing an examination, forming impressions and conclusions, and treating patients is alive and well in the medical community. Every day, innumerable physician-patient interactions occur based on trust

and empathy. Treatments are prescribed, clinical patterns are appreciated, and research projects are undertaken completely independent of such things as US Supreme Court Case No. 97-1079, *ipse dixit,* and other legal matters Dr. Wolfe discusses.

As a physical medicine and rehabilitation specialist (Physiatrist), I have seen over 11,000 patients with FM in my 12 years of private practice, and over half of them report a trauma that led to their chronic pain. If key features of the clinical evaluation include: 1. No previous pain complaints before the trauma similar to those experiences since the trauma; 2. a history of trauma that caused pain to develop; 3. pain resulting from the trauma that has persisted ever since the trauma, i.e., an "unbroken chain" of pain; 4. pain persisting for at least 6 months, well beyond the usual soft tissue healing time; 5. the presence of characteristic painful tender points as defined by the American College of Rheumatology criteria[2], then I will diagnose FM caused by the trauma, i.e., posttraumatic FM. This diagnosis is independent of any medicolegal activities the patient may be involved with. In reality, less than 5% of my patients with posttraumatic FM are involved in litigation, and the true malingerer is rare. The vast majority of patients with FM are not disabled, and I have yet to see a patient whose chronic pain "disappeared" after resolution of a litigation process.

Our country has laws governing liability issues related to trauma, so sometimes patients find themselves involved in litigation matters. All injured patients have legal rights and the legal experts, the attorneys, advise them of such rights. My role as a physician is to provide medical advice and treatment, and help patients do as well as possible. I don't think we should let our (or Dr. Wolfe's) perceptions of the legal system ever influence how we practice medicine.

Dr. Wolfe mentions that there are few useful scientific data regarding FM and trauma. Yet, using the standards of "absolute proof" he seems to require, very little of what physicians diagnose and treat has ever been 100% scientifically proven. It is very difficult, if not impossible, to design research studies that will meet the standards of "absolute proof." In the case of trauma and FM, we can use our astute clinical observations and current useful scientific data, i.e., Dr. Buskila's study[3], to recognize that trauma is a cause of FM, more probably than not. At a recent FM convention, 5 FM panel experts opined that trauma can cause FM (FAME 2000 International Fibromyalgia Conference, Universal City, California, USA, May 20, 2000). Fortunately, medical philosophies and practices are based on probabilities and not absolute proof or else there would be very few medical conditions of any kind, not just FM, that physicians could treat.

Dr. Wolfe published a paper entitled "Post-Traumatic Fibromyalgia, A Case Report Narrated by the Patient" in 1994[4]. Yet less than 2 years later, he proposed the term "posttraumatic fibromyalgia" be eliminated altogether[5]. Undoubtedly, Dr. Wolfe's own biases and perceptions have changed, and now he wants us to think about FM and the law when we diagnose and treat patients so legal cases involving FM and trauma are less likely to happen.

If Dr. Wolfe is unwilling or unable to treat patients with FM from trauma, or if necessary, address issues involving causality and disability, he can rest assured that numerous physicians such as myself are willingly capable of providing such medical services. Because at the end of the day when the court recorders are silent and the judges and attorneys have gone home, our patients still have pain and need our help.

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Dr. Wolfe replies

To the Editor:

It is hard to reply to Dr. Romano's rambling letter on issues as remote as judges' personalities and the salaries of CEOs of health care organizations, except to observe that the art of the irrational and the non sequitur is not lost in West Virginia. Malingering is a very rare event in fibromyalgia (FM), but the legal crisis is not — a crisis that has been fueled by events such as the written assertions made by Dr. Romano in separate legal proceedings that FM was caused by prescription of high doses of corticosteroids and progesterones. Dr. Romano, his letters to editors notwithstanding, has added little light to this discourse.

Ferrari and Russell, on the other hand, splendidly summarize, and with evidence, the medical and social consequences of alterations in the compensation law. They show that it is possible to alter behavior and change the prevalence of disease. Their carefully thought out letter deserves detailed consideration. It would seem that the case described by Mailis, et al would have had a far different and far quicker ending if the Saskatchewan process had been available in Ontario.

Mailis and Taylor's letter makes several points that are important and should be discussed. They write that "...one should not ignore potential psychological/personality and socio-environmental contributions. Only a concerted approach based on the biopsychosocial model will help better define fibromyalgia." But hasn't that already been done, almost from the beginning of FM? Is there any rheumatic disorder that better defines biopsychosocial than FM, or one in which biopsychosocial issues have been discussed more. Mailis and Taylor go on to cite the splendid article of Benjamin, et al, where it was found that 16.9% of persons with widespread pain had mental illness compared with 6.5% without widespread pain. The point that Mailis and Taylor would make is "about the interplay between psychological factors and chronic widespread pain." But there is another point to be made, and it is how few persons satisfied criteria for mental disorder and how slight a difference in mental disorder prevalence was found between those with and those without widespread pain.

Citing this as partial evidence, Mailis and Taylor approach the American College of Rheumatology (ACR) classification criteria for fibromyalgia[3]. "Moreover," they write, "new consensus criteria based on considerably more that just 'tender point' detection should be established and their validity tested. Ten years following the publication of classification criteria for FM, the need for new criteria is overwhelming. Maybe, stricter and tighter criteria can allow FM to move from a disease 'without borders' to a more definable (hence more respectable) entity." Just how this should be accomplished, however, they do not state.

The criticisms of the ACR FM criteria are many. Ideally, we would like to have criteria that reflect the major characteristics of the disorder ("more than just tender point detection"), that accurately distinguish persons with and without FM, and that are feasible in ordinary clinical practice. In addition, Mailis and Taylor call for "stricter and tighter criteria." They indicate that "...definition by symptom complex and distinct 'tender points'..." is wrong ("bad pain medicine" — whatever that means).

In developing the ACR criteria[3], one point the study committee considered seriously was who should interpret the symptoms. For example, should the patient's report of fatigue, sleep disturbance, and pain be taken at face value, or should the physician interpret the symptoms and decide whether they were "true" or "valid." There is an enormous literature on the

hazards of letting observers decide how much pain and distress other humans have, and the committee felt that the bias of the physician would not clarify the issue of diagnosis.

Although Mailis and Taylor want to define, and presumably to diagnose, FM using "a concerted approach based on the biopsychosocial model," what that means or how it could be implemented is unclear. Should life stresses, income, employment, education, family life, and psychological status be part of the diagnostic process? How could any of these items (1) be measured, (2) be measured accurately, and (3) be carried out in the clinic? And why should these factors, important in every human disease, be restricted to diagnosis of FM?

From my analyses of the ACR criteria data set, and in subsequent analyses of different data sets with a wider range of "biopsychosocial" variables, certain points emerge. In almost any set of variables, tender points best distinguish FM patients from those without FM. But if you change your definition of FM so that you require the presence of fatigue, sleep disturbance, and other symptoms, then those required variables will perform best. That is, if you change your definition of FM, then different criteria sets can be used. In the ACR criteria study[3] and in other analyses, however, the actual differences between criteria based on tender points and those based on symptoms were small. FM is a general syndrome with variable degrees of pain, fatigue, sleep disturbance, tenderness, and distress. You can't get around it, and like it as you may, it is defined by "symptom complex and distinct 'tender points'."

What's so bad about a syndrome defined by symptoms? Patients present with fatigue, headache, backache every day. Why should such symptom diagnoses be acceptable and the FM syndrome symptoms be "bad pain medicine"? I am not uncomfortable diagnosing FM in patients who do not satisfy ACR criteria and not diagnosing FM in those who do meet the criteria; nor would I be uncomfortable if the word fibromyalgia were stricken from my vocabulary. I do not treat patients differently if they have 9 tender points compared to 11, nor do I think Mailis and Taylor do, either.

When used for the purpose they were designed (studies), the ACR criteria have worked very well. If they are used rigidly in the clinic then, of course, they will not work well; but there the fault is with the clinician. If used in the medicolegal setting they are a disaster, for (1) they imply a diagnostic accuracy and meaningfulness that was never intended and, more importantly, (2) they are not needed, for it is symptoms and symptoms severity that are key issues. Eliminating the term fibromyalgia from the disability process might be a step in the right direction.

Given its superfetation of innuendo, selective quotation, misstatement, and *ad hominem* attack, Pellegrino in his letter doesn't seem even faintly to understand what I was writing about. Let me restate it briefly. It is the view that truth is not given to one individual, even a physician, but must be subject to scientific investigation and validation. Bertrand Russell in his Introduction to *Mathematical Philosophy* put it this way, "The Method of postulating [assuming] what we want has many advantages. They are the same as the advantages of theft over honest toil."

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Wichita, Kansas, USA. Frederick Wolfe, MD.

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The Epidemiology of Psoriatic Arthritis

To the Editor:

I must clarify an important misunderstanding of our case ascertainment methods stated by Dr. Veale in his recent editorial[1] of our article[2]. Dr. Veale suggests that interpretation of the results of our study should be "cautious," arguing that "it is also possible that only mild cases of psoriasis were entered into the community database; more severe cases being assessed in hospital may therefore bypass the system. This may explain the low incidence figure...." This is not the case. As described in the methods section of our paper, the Rochester Epidemiology Project (REP) data resources include all medical records (both inpatient and outpatient) from all medical care providers to county residents. As in all other REP studies, in this study of the epidemiology of psoriatic arthritis, we reviewed all the hospital records (as well as all outpatient records) for our study population.

Mayo Foundation,
Rochester, MN 55905, USA. Sherine E. Gabriel, MD, MSc.

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Dr. Veale replies

To the Editor:

I thank Dr. Gabriel and colleagues for their clarification with regard to the case ascertainment in their study[1]. Clearly from the point of view of an epidemiological study, case ascertainment is an important issue, as I mentioned in my original editorial. I accept that the authors have carefully collected data from the Rochester Epidemiology Project database, and I am certain that they made every effort to include all the medical records from the patients in Olmsted County. The caution that I recommended in the interpretation of the results of their study relates to a number of factors and not just the ascertainment methodology. I am sure the ascertainment methodology that they used was sound in that clearly it would have identified every one of the database who met their criteria. There are, however, possible confounders outside of their control that may lead to bias. The retrospective nature of the study, as in all such cases, is not helpful. The identification of cases of psoriatic arthritis from a cohort of patients entered into the database with an initial diagnosis of psoriasis may also create a bias. Finally, there is the matter of the low rate of disease progression as evidenced by the rate of development of erosions, which appears extraordinarily low at a rate of 11% in this study.

In conclusion therefore it is as a result of all of these potential factors for creating bias that I urge caution in the interpretation of the results, and not for any single issue relating to the case ascertainment methodology used in this study.

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Leeds, UK. Douglas Veale, MD, FRCPI, FRCP(Lond).

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Extensive Pigmentation Secondary to Minocycline Treatment of Rheumatoid Arthritis

To the Editor:

Minocycline has been documented to cause pigmentation of the skin, nails, bones, thyroid, mouth, and ears. Skin pigmentation is reported to occur in 4–15% of patients taking cumulative doses > 100 g[1,2]. With the recent interest in minocycline as a palliative treatment for rheumatoid arthritis (RA), a new population is experiencing this effect. Early recognition and discontinuation of the drug will minimize the degree of permanent color change. We describe 4 cases of minocycline induced pigmentation secondary to longterm treatment for RA.

All 4 patients were females taking 100 or 200 mg of minocycline for RA (Table 1). They progressively developed blue to black discoloration, predominately on their arms (Figure 1) and lower legs, especially in areas of previous trauma such as the shins and knees. The pigmentation was often bilateral, ranging from small well circumscribed macules to more diffuse coalescent patches. One patient also had a bluish-gray discoloration involving the proximal 2/3 of all nail beds.

Skinner, *et al* conducted in 1971 a double blind study evaluating the effectiveness of 250 mg tetracycline daily for the treatment of RA in 27 patients[3]. They found that low dose tetracycline has no statistically significant benefit compared to placebo. However, 3 double blind, placebo controlled studies recently addressed the efficacy of minocycline for the treatment of RA[4-6]. All 3 studies showed minocycline (200 mg daily) to be superior to placebo, especially in improving joint tenderness, joint swelling, and laboratory measures[4-6].

Pigmentation is the most frequently observed adverse reaction to longterm high dosage minocycline. It was first reported in 1978[7]; however, since then many other reports have documented skin darkening as well as pigmentation of sclerae (Figure 7), conjunctival cysts, oral mucosa (Figure 5), ears (Figure 6), thyroid, teeth, and nails[8,9] (Figure 4). It is classified into 3 different types based on the site of pigmentation and the pathological findings. Type I pigmentation is characterized by blue-black macular pigmentation localized to areas of scarring, inflammation[10], or bruising. These sharply marginated macules are most frequently seen on the face within resolving acne lesions (Figure 2). Type II pigmentation presents as blue-black, brown, or slate gray macules on healthy skin primarily on the shins (Figure 3), ankles, and arms[11]. Type III pigmentation appears as generalized and symmetrical muddy brown macules on healthy skin[11]. This pigmentation resembles an off-color suntan and is generalized all over the body, sometimes accentuated in sun exposed areas. With Type I pigmentation, risk of development is independent of duration and total cumulative dosage; however, type II and type III predominately develop in patients who are treated for prolonged periods at high dosages[11]. Dwyer and Cuddihy[2] studied 54 patients being treated with minocycline for rosacea or acne for a mean duration of 17 months. They found 15% developed pigmentation. Patients treated for rosacea were more likely than patients treated for acne to develop minocycline associated hyperpigmentation. This may be a consequence of the older age of the rosacea patients, and that they had greater sun related purpura. They also usually receive higher cumulative dosage of minocycline compared to patients treated for acne. Goulden, *et al* also reported an increased incidence of pigmentary side effects in patients over age 35 (27%) compared to patients younger than 35 (11.8%)[1]. In addition to age and longterm sun exposure, patients with RA may develop prominent skin thinning due to prior or concomitant use of oral corticosteroids. All these factors lead to compromised connective tissue and allow for easy bruising and subsequent pigment deposition.

Patients receiving longterm treatment with minocycline should be informed of the pigmentation side effects and screened for its development. Both type I and II cutaneous pigmentation may resolve upon cessation of the minocycline. However, this may take months to years before complete resolution is achieved. In contrast, the muddy brown pigmentation seen in type III may persist indefinitely, but fortunately this is the rarest of the 3 types.

A more rapid alternative to simple observation is laser treatment, which has been shown to effectively remove pigmentation especially in patients with localized areas of involvement. Any of the Q-switched (ultra short pulse) therapeutic lasers that are commonly used to remove tattoos work equally well with no residual scarring or hypopigmentation[12]. As minocycline becomes a popular regimen for RA, more pigmentation side effects will be observed in this older, more susceptible population. Tetracycline has a better side effect profile and similar immunomodulatory properties compared to minocycline. In dermatological immune mediated disorders such as bullous pemphigoid and pemphigus vulgaris, tetracycline is effective at high doses (500 mg QID)[13-15]. In contrast to Skinner and co-workers' negative experience with only 250 mg daily[3], new studies should be done using doses of at least 1 g daily.

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Table 1. Four patients with RA presenting with dramatic minocycline induced pigmentation.

Patient	Sex	Age, Yrs	Dose, mg/Day	Duration of Treatment, mo	Examination Findings
1	F	44	200	12	Bluish pigmented macules on the shins, knees, left dorsal hand (Figure 1), and under both eyes.
2	F	66	100	30	Diffuse gray pigmentation over both arms from the elbows to the hands and patchy gray macules on the shins.
3	F	55	200	24	Bluish-gray pigmentation involving the proximal 2/3 of all nail beds.
4	F	63	200	60	Multiple well circumscribed blue to black macules on the shins bilaterally.

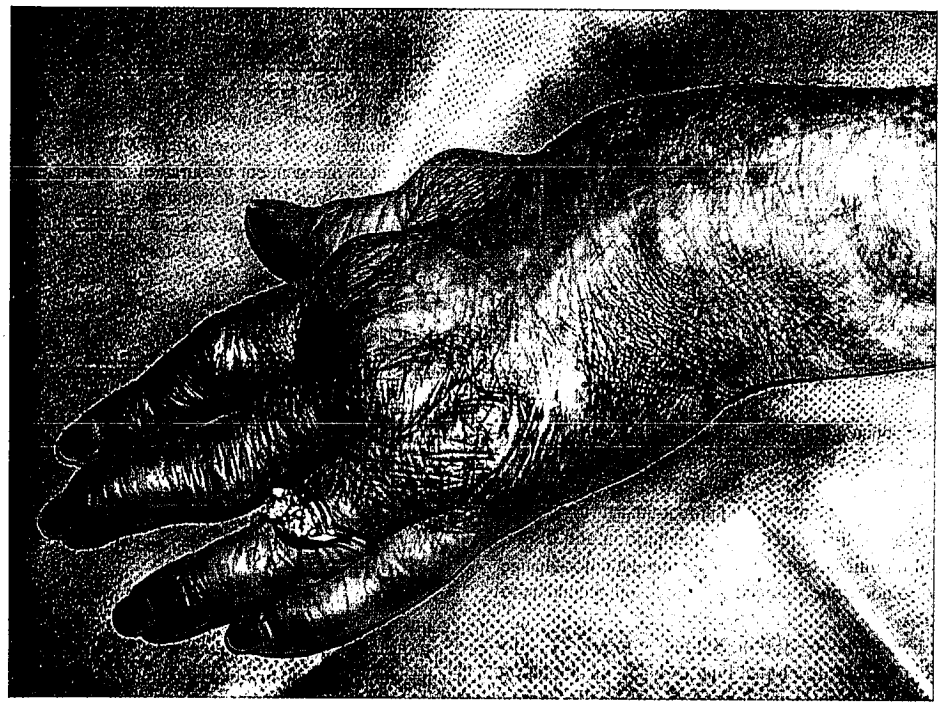

Figure 1. Bluish pigmentation of the left dorsal hand, Patient 1.

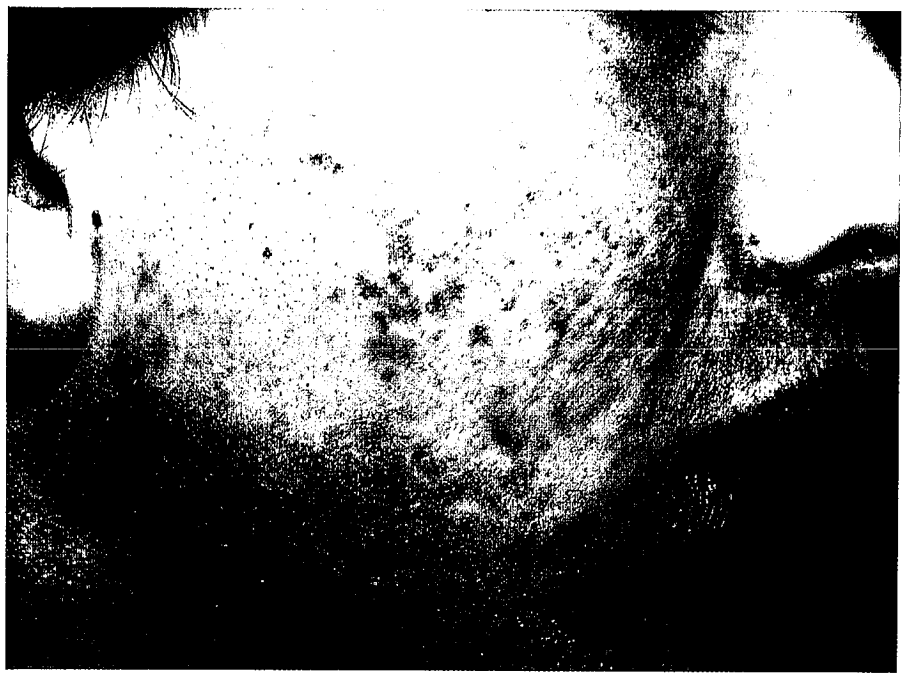

Figure 2. Type I minocycline induced cutaneous blue to black pigmentation localized to areas of inflammation—acne lesions.

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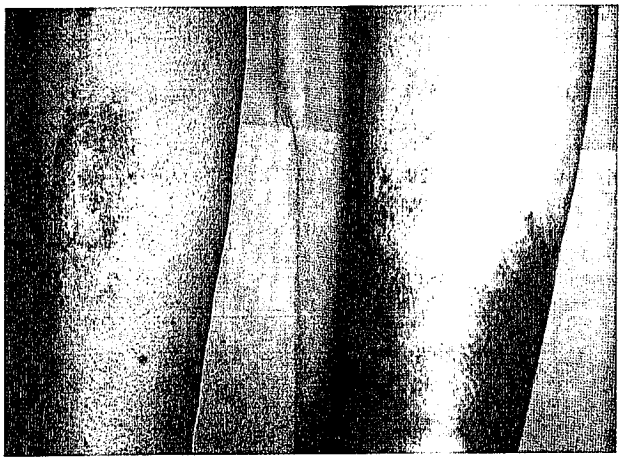

Figure 3. The shins are common areas of involvement in Type II cutaneous minocycline induced pigmentation.

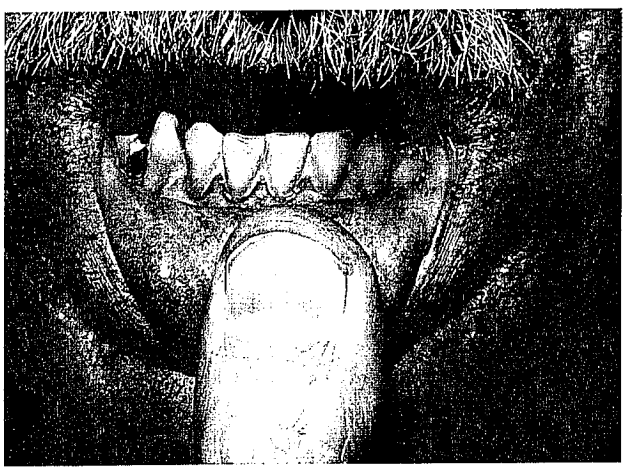

Figure 4. Minocycline induced pigmentation of the nails and teeth. This patient developed blue to black discoloration of the distal third of his permanent teeth and a slate gray discoloration of the proximal nail bed.

Figure 5. Minocycline induced pigmentation of the oral mucosa.

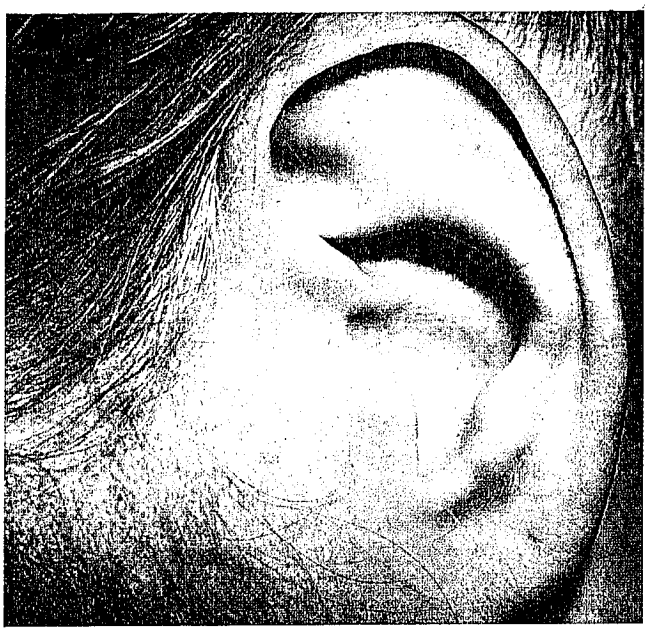

Figure 6. Bluish discoloration of the helix cartilage. When seen in association with scleral pigmentation, as in Figure 5, ochronosis should be suspected.

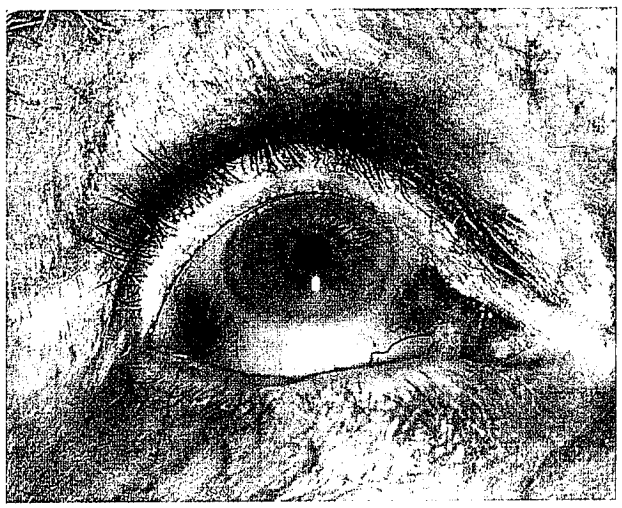

Figure 7. Blue pigmentary changes of the sclera after longterm monocycline treatment.

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Termination of Disease Modifying Antirheumatic Drugs in Psoriatic Arthritis

To the Editor:

We read with great interest the recent paper of Lacaille, *et al*[1] describing their experience with the longterm treatment of psoriatic arthritis (PsA). There are only a few controlled studies in patients with PsA requiring more than just nonsteroidal drug treatment.

Since 1988 there has been a center for psoriatic arthritis at our institute. In and outpatients from all over the country are treated. We examined the treatment time of gold sodium thiomalate (GST), methotrexate (MTX), and sulfasalazine (SSZ) to get information in clinical practice about the therapeutic effectiveness and safety of these disease modifying antirheumatic drugs (DMARD).

The diagnosis of PsA was based on the presence of psoriasis and peripheral arthritis. We analyzed data of 123 patients who were taking DMARD in 147 cases. Patients were not randomly allocated to the therapies; indication for the drug and causes of withdrawal depended on the physician's judgment. Records from each patient visit were reviewed according to a standardized protocol and data were abstracted. Data included medication, dosage, disease duration at the beginning of therapy, duration of treatment, reason for discontinuation, side effects. All patients with DMARD were taking concurrent nonsteroidal antiinflammatory drugs and some of them also received systemic corticosteroids.

GST was started with an initial dose of 10 mg and 25 mg, followed by 50 mg for 20 weeks, then it was tapered to a monthly 50 mg injection. Oral MTX was started at 7.5 mg once a week. SSZ was increased gradually from 500 mg to 2000 mg/day. Higher or lower dosages of drugs were used, as clinically appropriate. Patients were included in this study more than once if they received more than one DMARD. Patients were followed until June 1997.

Forty patients received GST, 63 MTX, and 44 SSZ treatment. Clinical characteristics and the mean and median survival time of the different DMARD treatments are shown in Table 1. Statistical analysis was performed using a cumulative survival analysis of termination (Kaplan-Meier test) and a test of comparison between survival curves (log-rank test). No statistical difference was found between the cumulative survival curves by log-rank tests: MTX-SSZ: $p = 0.817$, GST-SSZ: $p = 0.158$, MTX-GST: $p = 0.201$ (Figure 1). Four patients stopped GST treatment due to flare of their psoriatic skin disease. Two GST, one MTX, and 4 SSZ treated patients withdrew from treatment due to complete clinical remission.

The use of life table techniques to audit therapies offers an easy, graphic presentation of an important measure of therapeutic outcome in clinical practice. In addition, a larger number of patients can be studied over a longer time interval than usual in traditional, double blind controlled studies.

Longterm followup studies examining the clinical response and the proportion of treatment discontinuation had results similar to Lacaille, *et al*[2-4].

In the only life table analyses of PsA patients, Gomez-Vaquero, *et al* found a median survival time of 6 months for GST and 16 months for MTX[5].

Table 1. Patients' characteristics.

	GST	MTX	SSZ
No. of patients	40	63	44
Male/female	21/19	32/31	26/18
Mean age at the start of treatment, yrs (range)	39.1 (22–73)	40.1 (15–72)	40.0 (15–69)
Mean disease duration, yrs	4.9 (0–16)	7.29 (0–30)	4.4 (0–17)
Mean treatment time, mo	21.4 ± 4.7	26.8 ± 4.2	43.5 ± 9.8
Median treatment time, mo	12.0 ± 2.5	12.0 ± 3.1	17.0 ± 2.5

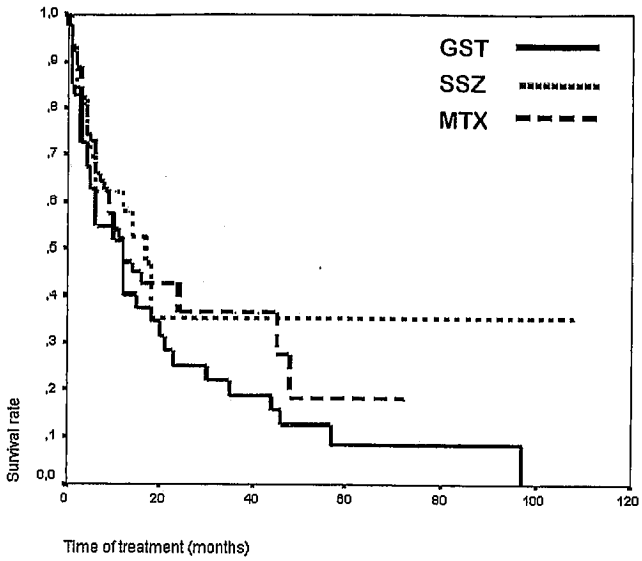

Figure 1. Lifetable analysis of treatment discontinuation with GST, MTX, and SSZ.

We did not find MTX superior to intramuscular gold, and although SSZ had the longest mean and median survival time, the log-rank test did not show significant differences among the 3 drugs. That some patients received MTX after having failed GST or SSZ, and that the subjects were not randomized are potential sources of differences from the earlier reported results.

We agree that both MTX and GST are effective DMARD in the treatment of PsA, and in cases where MTX failed or is contraindicated, GST is a good alternative. But we would emphasize the importance of SSZ, which is an effective and at the same time a safe drug.

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Beaver Fever — A Rare Cause of Reactive Arthritis

To the Editor:

We read with interest the case report of Tupchong, *et al*[1] describing giardiasis associated with reactive arthritis (ReA). We observed a patient with ReA induced by *Giardia lamblia* infestation. We describe our patient as a further case with an additional reference.

A 37-year-old man presented with malaise, pain, and swelling of the wrists, hands, ankles, and right knee. His history was unremarkable except that he had had diarrhea 6 weeks before, on his visit to a rural village. The diarrhea, which was foul smelling and watery, resolved spontaneously in a few days. Twenty days later he developed pain and swelling in his wrists, hands, ankles, and right knee. Before presenting to our hospital, he was treated with nonsteroidal antiinflammatory drugs (NSAID) by general physicians but no improvement was observed. He was a nonsmoker, heterosexual, with no significant family history, no rash, and no genitourinary symptoms. On examination there was tenderness and swelling in his wrists, ankles, and proximal interphalangeal joints of both hands and right knee. A mild diarrhea was observed during his hospitalization. Laboratory investigations revealed erythrocyte sedimentation rate 29 mm/h, C-reactive protein 16.7 mg/l (normal 0–5), white blood cell count 9050/µl (with mild lymphocytosis), and hemoglobin 15.3 g/dl. Routine biochemical tests and urinalysis were normal. Serum immunoglobulins and complements were also in normal ranges. Rheumatoid factor, antinuclear antibody, and anti-dsDNA were negative. Serology for hepatitis A, B, C, human immunodeficiency virus, *Brucella*, COREM and ASOT was negative. Repeated cultures of urine and throat for local infection were all negative. The stool was free of erythrocytes and leukocytes, but *G. lamblia* cysts were detected in the stool on the 5th hospitalization day. HLA-B27 was negative. Radiographs of hands, wrists, ankles, and right knee were normal.

He was given metronidazole for 10 days and indomethacin 150 mg/day was added. Within a few days he improved significantly and the mild diarrhea was resolved. One month later he was asymptomatic without joint inflammation and no recurrence was observed.

Although it has been described, the association between ReA and *G. lamblia* infestation is not common. Musculoskeletal manifestations of giardiasis in adults were first described by Barton, *et al*[2] in the English literature. Subsequent reports by Shaw, *et al*[3], Layton, *et al*[4], and recently Tupchong, *et al*[1] supported this clinical syndrome in adults. Invariably the arthritis was refractory to treatment with NSAID but responded to antimicrobial treatment of the giardiasis. Despite the asymptomatic gastrointestinal infection in our patient, except for a mild diarrhea, he responded to metronidazole promptly. Although HLA-B27 positivity was suggested to support a genetic predisposition to reactive polyarthritis[3], our case was HLA-B27 negative. There have been 2 case reports of ReA due to *G. lamblia* in the absence of HLA-B27[2-4]. There is not sufficient information to determine the relationship between HLA status and the risk of developing ReA following giardiasis[4]. *G. lamblia* is commonly transmitted by contaminated water, especially in rural areas[5]. In our patient the history of travel to a part of the country where giardiasis is relatively common supported the diagnosis. As there was no clinical or laboratory evidence of connective tissue disease or infective focus other than *G. lamblia*, and rapid improvement of arthritic symptoms was observed with metronidazole therapy (although he did not respond to NSAID), the infectious pathogen that caused reactive polyarthritis seemed to be *G. lamblia*. We think our case indicates the necessity of taking a detailed history not to overlook the diagnosis in an asymptomatic patient presenting with ReA. Our case and that described by Tupchong, *et al*[1] emphasize the importance of considering *G. lamblia* infection in patients with resistant arthritis, especially after an episode of diarrhea.

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Book Reviews

Diseases of Skeletal Muscle

Robert L. Wortmann, Editor, Philadelphia: Lippincott Williams & Wilkins; 1999, 400 pages, price $99.00 US.

This is a multi-author up-to-date review of the different skeletal muscle disorders. The text is logically organized into 3 main sections. Section I provides a detailed description of the structure, physiology, and function of normal muscle, including the effects of aging on muscle function. Section II describes various disorders of skeletal muscles including inflammatory myositis, metabolic and genetic disorders, and muscle infections. Included in this section are excellent, well illustrated chapters on childhood myositis, metabolic diseases of muscle, congenital myopathies, skeletal muscle disorders in sarcoidosis, and rhabdomyolysis. The reasons for including fibromyalgia in this section are not entirely clear. There is no convincing evidence that fibromyalgia is a skeletal muscle disorder, and muscle biopsies have not revealed any specific or consistent microscopic, ultrastructural, biochemical, or metabolic abnormalities.

The grouping of polymyositis/dermatomyositis with such unrelated diverse entities as posttraumatic heterotopic ossification (mislabelled posttraumatic myositis ossificans), orbital myositis, and eosinophilic myositis serves no useful purpose. Sections on the natural history, course, prognosis and treatment of polymyositis and dermatomyositis, drug induced myositis, malignancy associated myositis, and pyomyositis are briefly discussed, and to be truly useful to the clinician, these require further expansion.

Section III succinctly examines diagnostic studies including measurements of serum muscle enzyme levels, myositis-specific autoantibodies, electrophysiologic evaluation, skeletal muscle imaging, and muscle biopsy. Overall, these chapters, particularly the one on autoantibodies in myositis, provide a readable, concise, and thorough approach to the diagnosis of muscle disorders. It would be useful to expand the section dealing with the differential diagnosis of proximal muscle weakness to include causes other than myositis of elevated serum creatine kinase levels.

In summary, these caveats aside, *Diseases of Skeletal Muscle* is an excellent resource monograph, dedicated to the structure, function, and various disorders affecting skeletal muscles. Its greatest strength is in the excellent quality of many of the individual chapters. The book is a welcome contribution to the scientific literature. It provides a valuable, user friendly resource, not only for clinical rheumatologists, but also for internists, neu-

rologists, and researchers who are interested in all aspects of skeletal muscle disorders.

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Pediatric Rheumatic Diseases

(vol. 3 of the Rheumatologic Rehabilitation Series) Jeanne L. Melvin, MS, OTR, FAOTA, and F. Virginia Wright, MSc, BScPT, Editors. Bethesda, MD: American Occupational Therapy Association; 200, 316 pages, price $58.00 US.

It has struck me over the years (as a physician who spends much time involved in the rehabilitation of children with arthritis) how valuable it would be to have a comprehensive reference to guide therapy. Children are not just little adults, and the pediatric rheumatic illnesses differ in many ways from their adult counterparts. Indeed, children provide additional challenges; issues of compliance, schooling, and pain must all be approached with a child's developmental capabilities in mind. This new book will help physicians and therapists gain a broad understanding of the elements necessary for rehabilitation of childhood arthritis.

The editors, 2 highly respected therapists, have drawn together a number of outstanding authors. The first 4 chapters are devoted to an overview of the most common pediatric rheumatic conditions. These sections are very well written and will bring therapists and other health professionals up to speed. The later chapters will be of value for all members of the health care team. Their focus is on understanding developmental stages, enhancing compliance, dealing with pain, factoring in schooling, and the measurement of outcomes. The real strength of the book is the expertise of the authors; each of these chapters is written by world leaders in the field. Finally, there are 3 chapters devoted specifically to the occupational therapy, physical therapy, and surgical therapy for children with juvenile rheumatoid arthritis. These are complemented by a number of helpful case studies that put the information in an overall context.

I have 2 suggestions to the editors for future editions. First, there are a few errors in the text, including medication dosage errors in the early chapters, and these should be corrected. Second, research in the field of pediatric rehabilitation is in its infancy; while the authors occasionally acknowledge that their recommendations are not based on high quality data, it would help readers to be explicitly shown which treatments are supported by evidence.

This is an important work — I believe that the book will be very helpful in my clinical practice. It will appeal to physicians, nurses, and especially therapists who deal with children suffering from rheumatic illnesses.

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Clinical Chief, Arthritis Team, Bloorview MacMillan Centre,
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Correction

Rischmueller M, Limaye V, Lester S, et al. Polymorphisms of the interleukin 10 gene promoter are not associated with anti-Ro autoantibodies in primary Sjögren's syndrome [letter]. J Rheumatol 2000;27:2945–6. Authors' names should have been listed in the following order: Vidya Limaye, MBBS; Sue Lester, BSc (Hons); Sarah Downie-Doyle, PhD; Kevin Pile, MD, FRACP; Peter Bardy, FRACP, FRCPA; Tom P. Gordon, MD, FRACP, FRCPA; Maureen Rischmueller, FRACP. We regret the error.