Medical data is for informational purposes only. You should always consult your family physician, or one of our referral physicians prior to treatment.

The Journal Of The Academy of Rheumatoid Diseases

Volume 1 Number 2

Officers and Board Members:
President (Chairman): Gus J. Prosch, Jr., M.D.
Vice-Chairman: Sheldon Nelson, D.O.
Executive Director/Secretary: Wayne Martin, B.S.
Medical Journal Editor: Robert Bingham, M.D. (1986); Stephan Cooter, Ph.D. (1994)
Chief Editor/Treasurer: Robert Bingham, M.D. (1986)
Board Members:
Robert Bingham, M.D.; Warren M. Levin, M.D.; W. W. Mittelstadt, D.O., M.D.; Archimedes A. Concon, M.D.; Harley Robinson, D.O; Albert Jellen, M.D.
Research Advisory Board:
Harold Buttram, M.D.; Lazlo I. Belenyessy, M.D.; Dr. Paul K. Pybus

In Memoriam to Robert Bingham, the 1994 republication of *The Journal of the Rheumatoid Disease Medical Association* ©, is being made by The Arthritis Trust of America. Editorial and subscription office: c/o Perry A. Chapdelaine, Sr., The Arthritis Trust of America®, 7376 Walker Road, Fairview, TN. 1986 by The Rheumatoid Disease Medical Association, ISBN 0-930991-10-9.

Table of Contents

Volume 1 Number 2

The Rheumatoid Disease Foundation [The Arthritis Trust of America]

The Rheumatoid Disease Foundation was chartered as a non-profit, charitable organization in the State of Tennessee, October 13, 1982, and received its retroactive tax-exempt status from IRS March 29, 1983.

The purpose of The Rheumatoid Disease Foundation is:

1. To disseminate the scientific findings of Professor Roger Wyburn-Mason, that the *Limax amoeba* is the source-cause of most forms of Rheumatoid Diseases; [although it appears now that this theory was incorrect, the treatments developed from it have been successful]

2. To contract with professional scientific and medical organizations for research and develop mental studies related to the cure and/or remission of Rheumatoid Diseases;

3. To fund basic research with such professional organizations;

4. To provide free and/or contributory treatment to needy victims of Rheumatoid Diseases;

5. To solicit funds from the general public in support of the above programs.

Editorial

Too many patients have accepted verbatim the propaganda and money-raising line of the Arthritis Foundation — "There is no known cause for arthritis and no known cure." This may be a good emotional approach to the public in soliciting funds, but it gives an arthritis patient no hope in seeking a recovery from the disease. It may steer the person suffering from arthritis to the rheumatologist and to content him into accepting palliative treatment. But it is fatalistic to any hope of a permanent remission or "cure."

Those of us who have been offering our patients a "total arthritis program of treatment" with emphasis on improving their health and resistance to disease — in addition to the usual drug treatments when necessary — by improving the patient's diet, giving food and vitamin supplements, prescribing exercises and physical therapy, have seen many more recoveries from arthritis than the averages reported in the medical literature.

And those physicians who are bold enough and experienced enough to use alternative treatments and "approved drugs for unproven indications" have even higher percentages of improvement and remissions in their arthritis patients.

Examples of these are the use of therapeutic doses of minerals and vitamins, herbal extracts such as Yucca, non-specific vaccine immunizations to build up patient resistance to infections, high protein and complex carbohydrate diets, and the prescribing of anti-protozoal drugs rather than the anti-malarials.

A journal such as this will bring to the practicing physician the clinical results of many physicians in many countries who are on the forefront of **Arthritis Treatment**. They may be using "orphan drugs" whose patents have expired so that no pharmaceutical company has any interest in financing their clinical testing for "efficacy." They may have discovered some new use of an old remedy or treatment. They may have by serendipity observed a patient improve or recover with some new or different substance or method.

All of these can be reported in this publication. The test of a new method or treatment should be: "It must be beneficial to the patient, and it must do no harm." In other words, it is the **Risk/Benefit Ratio** that is important — not just that it has been "Approved" for advertising by the manufacturer or distributor by the Food and Drug Administration.

Why the medical profession has allowed the government and the legal professions to control our "standards of practice" is something I will never understand. Only physicians know what is best for their patients, and their judgments should not be controlled by laymen — only their peers.

The Academy of Rheumatoid Diseases solicits members who are open-minded and progressive in their arthritis treatment and in their own clinical experiences and investigations. We will welcome papers and case reports which will help and encourage other doctors in the management of this large family of diseases, "about which more is unknown than known." This *Journal* may play an

Medical data is for informational purposes only. You should always consult your family physician, or one of our referral physicians prior to treatment.
important role in reversing this void in medical knowledge.

ROBERT BINGHAM, M.D., EDITOR

Secretary's Report

Notes on the meeting of the elected officers of the new organization, the "Rheumatoid Disease Medical Association."

Meeting at the Miramar Sheraton Hotel, Santa Monica, CA, July 16, 1986.

Those present: Gus J. Prosch, Jr., M.D., President; Sheldon Nelson, D.O., Vice-President; Robert Bingham, M.D, Treasurer and Editor of the Journal; Warren M. Levin, M.D., Director.

Dr. Bingham reported that of 205 physicians invited to join the new association there are 43 who have paid their first two years' dues to become Charter Members. Many more are expected to join on an annual membership basis. More than $8,600 has been collected to date of which $2,600 has been paid for production and mailing of Volume 1, Number 1, of the new *Journal* which was distributed at the meeting and will be sent to all physicians on the referral list of The Rheumatoid Disease Foundation [now known as *The Arthritis Trust of America*].

Dr. Bingham reported that 3 of the doctors had either resigned or were being removed from the list for not using the treatment protocols of The Rheumatoid Disease Foundation.

A motion was made, seconded and passed (M.S.P.) that the new association be a separate entity from the The Rheumatoid Disease Foundation to avoid any conflict of interest between supporting the association and the Foundation just to receive referral of arthritis patients. The association will be a non-profit charitable and educational corporation, entirely and legally independent of The Foundation.

A new name was suggested for the association to emphasize its scientific and medical educational purposes: the "Academy of Rheumatoid Diseases." All of the officers and directors will be solicited for their comments and approval if agreeable.

It was M.S.P. that the by-laws and Articles of Incorporation, so well-researched by Perry Chapdelaine be sent to each officer and director for their recommendations and changes necessary for the new organization.

Discussion followed as to the purposes of the association, and it was generally agreed that they should be very broad, encompassing all of the rheumatoid and related diseases and not be limited to the amoebic aspect or the anti-protozoal aspect of treatment for rheumatoid arthritis.

It was decided that the officers and directors should meet together again, in about six months, at a convenient location, such as Nashville, TN, to plan the first annual meeting of the new association — to be held in conjunction with the Third National Seminar of the Rheumatoid Disease Foundation at a city yet to be selected. It was recommended that our new organization be very active in advertising and promotion of the next annual meeting, with greatest thanks and appreciation to Perry A. Chapdelaine, Sr., for the excellent program and fine speakers of this Second Annual National Seminar. With the permission of the Foundation, as many of the papers presented as possible will be printed in the quarterly issue of the new *Journal.*

Wayne Martin, B.S., Secretary
25 Orchard Court
Fairhope, AL 36532

Medical data is for informational purposes only. You should always consult your family physician, or one of our referral physicians prior to treatment.

The Free-Living Amoebic Causation and Cure of Activity in Rheumatoid Diseases

by Roger Wyburn-Mason

A reactive arthritis may occur with gastrointestinal infection with bacteria, such as Campylobacter jejuni, Salmonella, Shigella and Yersinia, in patients with HLA-B27 tissue types and disappear with successful treatment of the infection (Kosanen et al. 1980). Between 1922 and 1952, numerous publications described E. histolytica bowel infection with or without dysentery as associated with a condition practically identical with rheumatoid disease. Both conditions are cured by emetine without any exaggeration of the joint symptoms. Yet it is not unusual for sufferers from rheumatoid arthritis to show E. histolytica or its cysts in the feces and never in the rheumatoid lesions.

All terrestrial animals and plants, and those inhabiting fresh water and the sea, live in a world surrounded by many species of free-living amoebae. These may pass mammalian respiratory passages as cysts or trophozoites and may be present as trophozoites in the gastrointestinal tract of many animals, including man since they are found in their feces. As the organisms are motile, it would be reasonable to suppose that once they had entered the orifices of man or other warm-blooded animals, they would migrate under thermotropic influences into some body tissues. Since amoebae may be either non-pathogenic or pathogenic to animals, the same may apply should the organisms reach human tissues.[1]

In 1922, the eminent protozoologists, Kofoid and Swezy, in California, reported the presence in the bone marrow in cases of rheumatoid arthritis, without dysentery of E. histolytica in the feces, of a free-living amoeba distinguished from human cells by its mitotic processes which contained only 6 chromosomes as compared with the normal 46 of human cells. It showed a single blunted pseudopodium and numerous vacuoles. They suggested an aetiological relationship between the infection and the arthritic process.

In numerous recent publications, the speaker[14, 18] has shown that, using the property of thermotropism, free-living amoebae pathogenic or non-pathogenic, can be made to migrate in varying numbers out of human tissues, including those of the newborn and fetus (Figs. 1, 2). They are also found especially in the affected tissues of patients with rheumatoid disease, in malignant tumors, in normal feces, in uncooked butcher's meat, and in surface soil.

They may be recovered in small numbers from some tissues of apparently healthy humans, where they are presumably of non-pathogenic nature. They appear identical with those found by Kofoid and Swezy. These findings have been confirmed in various parts of the world, including Vanderbuilt University, U.S.A., the Oncological Research Institute, Bratislva, Czechoslovakia, Waikato Hospital, New Zealand, the Hospital for Special Surgery, and St. Vincent's Hospital, New York and in England.[1]

These organisms can be cultured in the laboratory in "amoeba saline" into which a culture of E. coli has been introduced. The effect of various antiamoebic substances in killing the organisms can then be studied by adding them to the cultured cells. Those which do so include one percent solutions of bile salts, 4-aminoquinolines, very dilute solutions of copper sulfate, metallic copper, gold salts, emetine, dehydroemetine, pentamidine, levamisole (which contains an imidazole group), and in particular the 5-nitroimidazole group of drugs, including metronidazole, clotrimazole, tinidazole, ornidazole and nimorazole. All of these possess a wide spectrum of antiprotozoal, including some antiamoebic activity.

The organisms have been recovered with great difficulty because they are not observed in affected tissues stained by ordinary methods. They are not numerous and look like macrophages or lymphocytes. This is a feature repeatedly observed in experimental infections with free-living amoebae in laboratory animals. (This recalls the situation in syphilitic lesions until stains for the treponema pallidum were discovered). The organisms in rheumatoid arthritis can, however, be demonstrated in tissue sections by immuno-fluorescent staining using sera containing appropriate antibodies to the organisms and by studying their mitosis and chromosome content in marrow biopsy material.

Rheumatoid Arthritis, a Generalized Disease

Rheumatoid Arthritis is not solely one of joints but may involve any tissue of the body. The same histological changes in the joint capsules may be found in extra-articular lesions and consist of inflammatory lymphocytic infiltration, formation of germinal follicles and often plasmocytosis accompanied by arteritis, arteriolitis, or endarteritis. Many of the extra-articular lesions constitute so-called "auto-immune diseases," but also include Sjogren's syndrome, bone marrow infiltrations, and thymic lesions with or without myasthenia, fever up to 40° C, sweating and raised ESR, typical of an infection. Any of the extra-articular of AI lesions may occur in any combination with or without arthropathy.

From a study of the world literature, it seems that some of the extra-articular lesions may involve:

1. Exocrine glands often producing enlargement and dilatation of the ducts. It may involve the lacrimal and salivary glands (Sjogren's syndrome, R.A. in miniature), breast (cystic mastitis), pancreas (lymphocytic pancreatitis, which may exhibit calcification), liver (active chronic hepatitis, primary biliary cirrhosis), gall bladder and bile ducts (chronic cholecystitis and stenosing cholangitis), and kidneys (chronic nephritis, pyelitis).
2. Endocrine glands, including the thyroid (lymphocytic or Hashimoto's thyroiditis), adrenals, parathyroids, thymus (with or without myasthenia) and pituitary.
3. Polymyositis, myasthenia, bursitis, tenosinovitis, or rheumatoid nodules in any tissue.
4. Mucosal inflammation followed by atrophy, which may involve the gastro-intestinal tract producing atrophic stomatitis, pharyngitis, esophagitis, gastritis, or coeliac disease and ulcerative colitis, or in the respiratory tract leading to atrophic rhinitis, Eustachian salpingitis, laryngitis, or bronchitis.
5. Fibrosing alveolitis, pulmonary nodules, lung fibrosis, or pleuritis.
6. Peri-, myo-, or endo-carditis.
7. Bone marrow infiltrations with various disturbances of blood formation.
8. Paget's disease of bone, spondylitis.
9. Lymphadenopathy or splenomegaly with reactive lymphoid hyperplasia.
10. Choroiditis, uevitis, retinitis, scleritis.
11. Various skin lesions, including icthyosis, dermatitis, leukoderma, and melanoderma.

The serum may or may not contain RF, various auto-antibodies, ANF, increase in gammaglobulins and usually a raised ESR.

The Effect of Antiamoebic Drugs on Active Rheumatoid Disease

The author has shown that when any substance which kills the free-living amoebae *in vitro* is administered to cases of active rheumatoid disease or its extra-articular manifestations, it may cause a rapid disappearance of the inflammatory symptoms around the joints and elsewhere in the body.[1] This includes coeliac disease, ulcerative colitis, cystic mastitis, lymphocytic thyroiditis, etc. Myasthenia gradually disappears and in early cases[11], complete cures may be obtained. These drugs often induce an Herxheimer reaction that is a transitory exaggeration of the inflammatory changes

around the joints and elsewhere. This may be accompanied by influenzal symptoms, sweating, pyrexia, lymphadenopathy, rise in ESR, and eosinophilia.

This reaction has been reported by others in cases of active rheumatoid disease treated with gold salts[18] and levamisole[19], (which kill free-living amoebae). Such a phenomenon, first described by Herxheimer[20] in 1902, when cases of syphilis were treated with mercury, also occurs in syphilis treated with penicillin and in other diseases due to organisms more complex than bacteria when drugs which kill the causative organism in the tissues are administered. This reaction is due to liberation of irritant and antigenic substances from the dying and dead organisms. The Herxheimer reaction in cases of active rheumatoid disease treated with antiprotozoal substances has been confirmed in various countries, notably in U.K., U.S.A., Holland, and New Zealand.[1] It is not observed in healthy persons so treated or in rheumatoid diseases given anti-biotics. It's occurrence in rheumatoid disease, including "autoimmune" lesions during treatment with various antiamoebic drugs *proves* the presence in the *affected tissues* of a *causative pathogenic organism*.

By administration of antiamoebic drugs, especially 5-nitromimidazoles, to cases of rheumatoid disease for 3-6 months, the evidences of disease activity usually completely disappear in joints and extra-articular tissues. "Autoimmune" lymphocytic and humoral reactions are thus not the primary disturbance in rheumatoid and "autoimmune" diseases, but are the cellular-antibody response to the infection and its antigens and contribute to the tissue damage.

Practical Details of Treatment of Rheumatoid and Related Diseases

A simple method of treatment of rheumatoid disease is to administer 2 grams on 2 successive nights of one of the 5-nitroimidazoles for a 70 Kg patient. In some cases, the organism , as judged by the severity of the Herxheimer reaction, may prove to be susceptible to one or another of the 5-nitroimidazoles.

In order to prevent any severe Herxheimer reaction, the patient may be given an anti-inflammatory drug. The drug may remain at an effective level in the blood for four or more weeks while the inflammatory reaction is dying down.

If it is necessary for a further dosage of the antiamoebic drug to be given, wait until three months after the original doses to give the induced inflammatory response [time] to die down. Often only a single treatment is necessary and improvement may continue over the course of a year or more with return of a normal ESR and disappearance of all signs of disease activity.

References

1. Wyburn-Mason, R. *The causation of rheumatoid disease and many human cancers. A new concept in medicine.* Tokyo, Iji Publishing Co., Japan; 1978.

2. Editorial. Pathogenic free-living amoeba. *Lancet* ii: 165-6; 1977.

3. Craig and Faust. *Clinical parasitology*. Ed. by Faust, E.C., Russell, P.T., Jung, R.C. 8th Edition, Lea and Fibiger, London; 1970.

4. Dobell, C. *The amoebae living in man*. John Bale, Sons and Danielson, London; 1919.

5. Carter, R.F. Amoebic meningo-encephalitis. *Transactions of Royal Society of Tropical Medicine Hygiene.* 66: 195-7; 1972.

6. Nagington, J., Watson, P.G., Playfair, T.S., et al. Amoebic infection of the eye. *Lancet* ii: 1537-8; 1974.

7. Kofoid, C.A., Swezy, O. Mitosis in Endamoeba dysenteriae in the bone marrow in arthritis deformans. *Univ. Calif. Publ. Zool.* 20: 301-307; 1922.

8. Ely, L.W., Reed, A.C., Wyckoff, H.A. The amoeba as the cause of the second grade type of chronic arthritis. *Calif. State J. Med*. 20:59; 1922.

9. Kofoid, C.A., Swezy O. On the occurrence of Endamoeba dysenteriae in the bone lesions of arthritis deformans. *Calif. State J. Med*. 20: 59; 1922.

10. Kofoid, C.A., Swezy, O. Amebiasis of the bones. *JAMA* 78: 1602-4; 1922.

11. Kofoid, C.A., Boyers, L.M., Swezy, O. Endamoeba dysenteriae in the lymph glands of man in Hodgkin's disease. *Univ. Calif. Publ. Zool*. 20: 309-12; 1922.

12. Kofoid, C.A., Swezy, O., Boyers, L.M. The coexistence of Hodgkin's disease and amoebiasis. *JAMA* 78: 1602-4; 1922.

13. Kofoid, C.A., Boyers, L.M., Swezy, O. Occurrence of Endamoeba dysenteriae in the lesions of Hodgkin's disease. *JAMA* 78: 1604-7; 1922.

14. Wyburn-Mason, R. *A new protozoon, its relation to malignant and other diseases*. Kimpton, London; 1964.

Arteriosclerosis
A Vital Message to My Patients

Gus J. Prosch, M.D., and Wyatt C. Simpson, M.D.

We sincerely desire that all our patients and their families enjoy the best health possible. During the past 2-3 years, there has been an explosion of research and knowledge that can vitally effect the health of each of us. This paper was written in an effort to teach you, inform you, and convince you to change some of your eating habits so that you and your family will enjoy a healthier life and fewer health problems. Some authorities may question some of our conclusions, but when we daily observe the health of our patients improve from following these suggestions, in the name of *Truth* we must speak out and share this information with you.

Today's number one health hazard is arteriosclerosis or hardening of our arteries. This causes heart attacks, strokes, and peripheral vascular disease (usually in the legs) which leads to more misery and suffering than any other disease known today. This does not count the expenditure of billions of dollars and the loss of millions of days of productive work for the American work force.

Before 1900, this disease was hardly known and was extremely rare. In fact, the first "heart attack" was described in the medical literature in 1910. Dr. Paul Dudley White (President Eisenhower's heart specialist) saw a heart attack for the first time in 1929. The disease began with the advent of hydrogenated oils (margarine) and the processing (refining) of our grain foods such as wheat, corn, rye, barley, oats, etc., where all the vital fatty acids are removed from these grains. The food companies must remove these fatty acids so that the grain foods do not turn rancid and spoil, otherwise the foods would not last long on the shelves of our super-markets. Our great-great grand parents and their parents had very little arteriosclerosis even though their diets included foods known to be high in cholesterol such as eggs, butter, lard, and "sow-bellies," etc. However, they did not eat any hydrogenated oils, and their grain foods were home ground and not processed.

We have known for 20 years that the dietary cholesterol cannot be the cause of arteriosclerosis for several reasons. First of all, the dietary cholesterol in the stomach is broken down into its tiny component parts and although some is absorbed through the intestinal wall, most of our cholesterol in our system is manufactured by our own body. The problem of arteriosclerosis develops because our bodies do not use the cholesterol properly that our bodies make. Also, Iceland Eskimos, whose diet by the way is ten times higher in cholesterol than our diet, have very little arteriosclerosis. They should be "dying like flies" if dietary cholesterol intake caused arteriosclerosis. But they do not suffer from heart attacks, strokes, and poor circulation in their extremities unless they move to more civilized areas of the world and begin eating as we do. The two things these Eskimos eat differently from us are: (1) they do not eat any hydrogenated oils and (2) they eat a great deal of cold water ocean fish which are very high in fatty acids.

In the early 1940s, when the Germans overran Norway, the incidence of arteriosclerosis, cancer, and schizophrenia was quite high in that country. The Germans took away all the margarine from the Norwegians, and the incidence of these diseases dropped significantly. After the Germans left and Norwegians again began to eat their margarine, the incidence of these diseases increased to their former levels.

In America, we are developing arteriosclerosis at earlier ages than ever before even though there is a greater effort on the part of most of us to decrease our cholesterol intake in our diets. Autopsies performed on soldiers killed in the Korean War showed approximately 30% of these young men suffered from advanced atherosclerosis. About twenty years later, in the Viet Nam War, autopsies performed on soldiers killed showed approximately 60% suffered from advanced arteriosclerosis. We must do something about this trend, and that is the purpose of this paper.

Recent research has proven that all hydrogenated oils block the chemical pathways that are necessary for our bodies to use the cholesterol that our bodies manufacture. Also, our bodies must have certain essential fatty acids (now being removed from our foods) to assimilate and use our cholesterol as well as to manufacture certain hormone-like chemicals called prostaglandins, our cells cannot function properly, and they will be subject to disease. We believe that this is one of the main reasons that we are seeing an explosion of many chronic degenerative diseases such as Arteriosclerosis, Arthritis, Diabetes, Lupus, Schizophrenia, Multiple Sclerosis, Asthma, and numerous others. Other conditions that can benefit from these dietary changes include hyperactivity and learning disabilities, premenstrual syndrome, systemic yeast infections as well as many skin disorders and allergies.

What You Can Do!

1. Totally avoid all hydrogenated oils as in margarine, cooking oils, deserts (doughnuts, cookies, cakes, etc.) and deep fried foods such as French fries, corn and potato chips, etc. You can use cold pressed oils found at health food stores for cooking, but do not use extremely high temperatures. If the cooking oil label doesn't state "cold pressed," it is probably hydrogenated. You should read all food labels and avoid those that have hydrogenated oils as ingredients. Don't cook foods with high temperatures as all oils over 350 degrees become hydrogenated. Cook longer at lower temperatures.

2. Increase the essential fatty acids in your diet.

   a. Eat cold water ocean fish 3-4 times per week such as salmon, cod, mackerel, sardines, (pour off hydrogenated oils), water packed tuna. Warm ocean fish (snapper, flounder, perch, etc.) is second best. Fresh water fish (cat fish, trout, etc.) contain the smallest amount of the fatty acids.

   b. Try to eat 3 teaspoons of 1 tablespoon of *virgin* (not pure) olive oil daily (as on salads) but keep refrigerated after opening bottle.

   c. As a snack food, walnuts are very high in fatty acids.

   d. Only eat breads and cereals that have "100% whole wheat or whole grain" written on the package. Most brown breads are not whole grain but have coloring added. Avoid processed or refined cereals or white flour products such as breads, crackers, macaroni, spaghetti, noodles, etc. You can get these foods as whole grain from health food stores.

3. With any chronic illness at all, you should follow the above plus add the following supplements and follow the additional instructions.

   a. Purchase some salmon oil capsules (Maxepa) at a health food store and take 4-6 capsules daily. Extreme care must be exercised in locating this product as it is made only in England and many health food store products claiming this ingredient only contain soy oil. *Efamol* is one acceptable brand and *Nature's Way* is another.

   c. Decrease your red meat intake since red meats contain arachidonic acid which can provide too much of a bad prostaglandin plus a very bad substance called leukotrienes which will aggravate many disease conditions.

   d. Avoid all sugars, sweets, deserts, and all white flour foods.

   e. Get a good hypoallergenic, non-yeast multiple vitamin and mineral tablet and take 3-4 tablets per day. Be sure you get at least 500 mg. vitamin C, 50 mg. B-3 and B-6, 50 mg. zinc, 100 mcg. selenium, and 400 mg. of magnesium in your supplements. The above mentioned vitamins are necessary in the fatty acid chemical

reactions.

If you and your family will follow the above recommendations in your dietary habits, you will enjoy a longer, healthier life with much less chance of developing any chronic degenerative disease. You may pass the information on to friends and relatives to help them enjoy a happier and healthier life.

(

Anti-Amoebic Treatment of The Rheumatoid Diseases

Gus J. Prosch, Jr., M.D.

I was asked to speak on the anti-amoebic treatment of the Rheumatoid Diseases, and I even discussed this subject at last year's seminar to a degree. I also realize that the protocol is spelled out in detail in the information sent out by The Rheumatoid Disease Foundation [now The Arthritis Fund] to physicians, but one of our primary problems is physicians not using the protocol and instructions properly and therefore not getting good results.

Because of this, I feel it is appropriate at this time to go into detail about the present treatment and a tape-recording of this talk can be provided for any physician who wants to treat their patients properly.

To begin with, since the anti-amoebic treatment is controversial, I believe it is very important that all patients be completely informed as to what we do, and we should instruct the patient what to expect during the treatment. I believe that the more confidence a patient has in our treatment, the better results we will see. In my practice, I give every new patient a brochure that explains everything about the treatment so the patient will know exactly what to expect. Besides this, I have made a 45 minute videotape that all new patients are required to watch before I actually treat them. This not only develops confidence in the patient about the treatment, but it saves me considerable time when talking to the patients. Most all questions are answered on this videotape. In addition to this, I give each patient an audio tape recording of the videotape so they can relisten and review everything should they forget or get confused about the instructions.

One big problem that we all face with out patients is that orthodox or established medicine today convinces rheumatoid arthritis patients that they are going to have to live with their arthritis for the rest of their lives. When patients believe they are not going to get well, the brain produces more harmful chemicals that suppress the immune system, and that actually hinders the patient from getting well faster and better. We therefore in treating our patients, must give our patients hope, not a false hope but a belief that there's a good chance that they can get well. And I do believe that if we can rid the patients of amoebae in their bodies, they can and will get well. With newer and better drugs such as clotrimazole and tinidazole available in the future, I do believe we are going to be even more successful than we are now. I have patients tell me every day, "Dr. Prosch, you are the only doctor who has given me hope that something can be done for my arthritis." And those patients who don't have this hope do not respond as well to the treatment.

Now concerning anti-amoebic therapy, when a patient comes for my treatment, I usually begin therapy with prescriptions for Flagyl or Metronidazole and Allopurinol. The dosage for Allopurinol which inhibits the enzyme systems of the amoebae is 300 mg. tablets, three times daily for 7 days. If the patient weighs less than 100 pounds, I usually give one 300 mg. tablet twice daily and, if a child, I cut the dosage proportionately. In treating nearly 1000 patients, I have only seen 2 reactions to the Allopurinol, and they both consisted of a moderately severe hemorrhagic rash that was generalized. They both cleared up on discontinuing the Allopurinol and giving high doses of vitamin C and bioflavinoids. I do advise patients when taking any drug to call me if anything arises that I haven't told them to expect. I therefore do get extra calls when patients begin having the flu symptoms with the Herxheimer reaction, and I have one of my personnel talk to them first to screen out the Herxheimer symptom patients from the drug reaction patients.

With metronidazole, for patients weighing less than 150 pounds, I give two, 250 mg. tablets or 500 mg. after each meal, two

days in a row each week for six weeks. This means 1500 mg. each of the 2 days they are treated each week. For those above 150 pounds and up to 175 pounds, I give 1 extra tablet each day of treatment. For those patients weighing 175 to about 225 pounds, I give two tablets after meals and 2 at bedtime each day, making a total of 2000 mg. daily. For patients above 225 pounds, I give 3 tablets after each meal, making a total of 2250 mg. daily. I impress on these patients that when they have the flu symptoms, this is a good sign, and they should not discontinue the treatment. These amoebae can invade any tissue in the body, and patients may have Herxheimer symptoms wherever the germs are located. For example, with amoebae in the heart, the patients may notice their heart racing or skipping some. In the bladder, the patients may have cystitis symptoms such as urgency, frequency and burning on urination. In the brain, the patient may develop temporary depression or cloudiness or fogginess in their thinking. In the breast, areas of pain, tenderness or soreness may develop. These are all good signs and mean the amoebae are being killed in these areas. Of course, some joints may ache and hurt that the patient didn't know were arthritic and this is normal. Metronidazole has been known to cause paresthesias in the arms and legs or numbness and tingling feelings. The drug should be discontinued if this happens, and the paresthesias usually go away. I've had this happen only a couple of times, and I usually do not mention this beforehand as many patients are quite suggestible and will develop these symptoms; I usually tell them to take some Benadryl and continue the treatment for 1 more week and call me again. If symptoms persist, I discontinue the Flagyl, but usually the symptoms go away.

On the patient's first visit, I give 1 c.c. of Depo Medrol which counteracts the most severe Herxheimer symptoms the first week as more germs are killed then, and the Herxheimer reaction can be very severe that first week. Many patients have fairly severe flu-like symptoms the second and third week and this is good. If the Depo Medrol is not given, the flu-like symptoms can be so severe that the patients may discontinue the medications and will not respond to the therapy.

I try to have the patients return to the office for their second visit in 6 weeks. At this evaluation, I usually see one of three responses. They may have had no Herxheimer reaction, and usually in this case their arthritis is still quite active, but fairly often they are quite improved even without a Herxheimer reaction. With no improvement and no Herxheimer reaction, I then use another anti-amoebic drug. More commonly they have had a fairly severe Herxheimer and especially the 2nd, 3rd, or 4th week of treatment; and in many cases, many are asymptomatic with their arthritis. If they had no Herxheimer the 5th or 6th week but still have arthritic pains, I try another anti-amoebic drug. If they had a Herxheimer all six weeks of therapy, I continue them on the Metronidazole for another 4 to 6 weeks of treatment.

Whenever I see that the Metronidazole isn't working or maybe the amoebae have built up a resistance to the Metronidazole, I usually beigin other anti-amoebics. During the initial visit, I usually prescribe also copper aspirinate, 1 tablet after each meal. This is my own personal therapy as I believe the amoebae are strongly infested in the colon, and the copper has some effect on killing these colon germs. And if any of the copper is absorbed into the system, it should have an additional positive effect on the amoebae. I do not believe the copper is efficiently absorbed, however, as I personally took 80 mg. of copper daily for 3 weeks, and my serum level of copper was not increased at all.

I have also tried the copper beads sublingually that Dr. Sheldon Nelson developed, on about 150 patients, and I finally discontinued this method of copper administration as only about 20-25% of patients developed a Herxheimer reaction, and I became personally convinced that a good percentage of these patients had the Herxheimer reaction because I had suggested to them that they might get the flu symptoms. I came to this conclusion when I gave the beads to a dozen patients without mentioning the Herxheimer reaction, and none of them had any Herxheimer and their arthritis symptoms did not improve at all. However, I must say that I have had 3 or 4 patients that sincerely believe the copper beads helped them better than anything else. So I occasionally use the copper beads in resistant cases but only as a last resort and then as a desperation trial — just like I occasionally try bile salts in the form of Decholin, 2 tablets, 3-4 times daily. Rarely, a resistant patient will get some response to the Decholin, but this is the exception and not the rule.

My second choice of drugs is either Yodoxin or Furoxone or a combination of the two depending on the patient. I usually begin Yodoxin, 2 tablets 3 times daily for two weeks only. There have been some eye problems when given longer than this, so I usually limit the Yodoxin treatment to 2 weeks. I have seen some good responses and Herxheimer reactions to the Yodoxin.

When prescribing Furoxone, the dosage is 100 mg. or 1 tablet 3 times daily for patients under 125 pounds, and one tablet 4 times daily when over 125 pounds of weight. I usually write the prescription for a 30 day supply and write in 2-3 refills. I instruct the patient to call me in 30 days; and if they are having a Herxheimer reaction, I have them get a refill for another 30 days.

My next choice of an anti-amoebic drug is Rimactane or Rifampin. The dosage is 2,300 mg. capsules daily for 30 days with 2-3 refills given. If after 30 days, the patient is still having some Herxheimer reaction, I have them refill the prescription and take it another 30 days. This drug is normally used for tuberculosis, but it is a very good anti-amoebic. I have seen some very severe Herxheimer reactions with this drug, and I always impress on these patients to call me if anything unusual other than routine Herxheimer reactions develop. I have seen one patient develop fairly severe paresthesias in the toes and feet when he continued the medication after the numbness and tingling began. He developed weakness and minimal loss of function in his toes, but it went away after about 8 months. I discontinue the medication immediately now if paresthesias develop, and they usually clear up promptly. Also one patient developed double vision or diplopia which cleared up on stopping the Rimactane.

These medications or anti-amoebic drugs are the main ones available in the U.S. today; and sometimes when patients are not responsive satisfactorily to the methods or treatment I have discussed, I may then try combinations. For example, Furoxone and Allopurinol, or Metronidazole and Yodoxin, or Furoxone and Yodoxin, and have found in some cases good results. Occasionally I re-prescribe one of the above medications and add Potaba in the dose range of 12 grams daily in divided doses. Envelopes containing 2 grams of Potaba are available and can be mixed with water, 2 packages, 3 times daily with meals. Potaba, or potassium Para Amino Benzoic Acid is a vitamin but must be used in high doses to be effective as an anti-amoebic.

I have found that I get very good to excellent results in 8 out of 10 patients with treatments I have described so far. I'm not sure whether there is a different germ involved in the 2 out of 10 that don't respond, or possibly these patients' amoebae are just resistant to the available medications we have in the U.S.

When I have a patient that comes in with a very acute rheumatoid arthritis and numerous joints are very hot, that is, swollen, red, and very painful, these patients can be relieved very nicely by giving high doses of the enzyme Bromelain. I use a Bromelain tablet

containing 500 mg. per tablet. By giving 2000 mg. or 4 tablets a.m. and p.m., the acute inflammation usually subsides rapidly and these patients are very grateful. The theory is that the enzymes destroy the auto-antibodies in the inflamed sites that are responsible for the acute inflammation. I usually keep the patients on 4 tablets twice daily for 7 days then decrease the dosage to 3 tablets twice daily for 1 week, then 2 tablets twice daily for 1-2 weeks. I try not to give Bromelain if the patient is also given Depro Medrol or other cortisone like medications; and in these cases, I wait a week after the steroid is stopped and begin the Bromelain.

I might also mention at this point that when patients come in and are taking prednisone, gold injections, or penicillamine, they do not respond to any treatment as well as those patients not taking these drugs. Dr. Wyburn-Mason liked to get the patients off these drugs completely for at least 4 months before initiating the anti-amoebic therapy. I have found that much better results are achieved when his recommendations are followed.

I've gone over the primary anti-amoebic drugs and how they are to be used to get the best results. I do not mean to insinuate that any physician must use this protocol exactly because I realize that some cases are different, but I get calls every week from patients who tell me that other physicians on our referral list gave them the drugs in a different manner. For example, some physicians prescribe the Metronidazole, 1-250 mg. tablet, 3 times daily for 10 days, which is the treatment for Trichomonas vaginitis but will not kill the amoebae. Or the Allopurinol is given only 1 tablet daily. These physicians simply have not studied the protocol, and when they do not get results, they have only themselves to blame — at the expense of the suffering patient. This can only give our treatment and protocol a bad result which will discredit every physician on our list plus our foundation. Let me make an urgent plea to any physician listening to me now or whomever listens to the tape recording of this talk to study the protocol and treat your arthritic patients in the proper manner. You owe it to yourselves and to your patients. I've had patients who have read *Rheumatoid Disease Cured at Last* go to doctors on our referral list and fully expect to receive the recommended treatment but instead receive treatment for allergies or just nutritional supplements, and occasionally they are given gold injections or the orthodox, non-steroid anti-inflammatory drugs. Heaven forbid! These physicians are charlatans who join up with us to use the anti-amoebic therapy, then treat the patient in the orthodox manner. We are learning who these physicians are who have been using us to get new patients, and we are kicking them out of our organization. In my opinion, it is the lowest character for any physician to represent himself as one of us and not treat a patient properly who came to him in the first place for that particular treatment.

Another large area of complaint is certain physicians who represent or claim on our physician referral list to give the intraneural injections and they know nothing about how to properly give the injections. This is unfair to our organization, the physicians themselves, but especially to the patients, and this is blatant deceit. These physicians also are being weeded out of our organization. Those physicians attending this seminar will observe a videotape that will show them exactly how the intraneural injections are to be given, and this tape is available from The Rheumatoid Disease Foundation [now The Arthritis Fund] to any physician who wants to learn. The videotape is overly repetitious to make sure the physician can properly give the injections after studying the tape, so really there is no excuse for not giving the injections properly.

Let me give a brief review about these intraneural injections because those of you not using the intraneural injections are really missing out on a tremendously beneficial technique to help all arthritic patients. I have patients who come to me from all over the United States who mainly just want the intraneural injections because they get so much relief. The injections help tremendously in rheumatoid arthritic patients but are even more effective in osteo arthritic patients. Even patients with acute back sprain and other muscle sprains often get immediate relief of their pains and muscle spasms. It is an excellent tool to add to any physician's armamentarium of treatments and skill.

The sincere and genuine physicians on our referral list can easily be the best doctors in their part of the country. These physicians are men of vision, they have taken off the blinders placed on them by orthodox medical training, and most of all, they have courage, fearless courage, to stand up for their convictions and give their patients the very best treatments available today. I'm proud of these pioneers in medicine and delighted to work with them.

The actual techniques of the intraneural injections were first pioneered by our chief medical advisor, Dr. Paul Pybus and Dr. Roger Wyburn-Mason. Dr. Pybus has further refined the art.

Medical data is for informational purposes only. You should always consult your family physician, or one of our referral physicians prior to treatment.

Boron in Medicine — Update

Rex E. Newnham, Ph.D., D.O., N.D.
Cracoe House Cottage
Cracoe, Skipton BD23 6LB
North Yorkshire, England

History. Boron as the sodium salt has been used by man for over 2500 years as a flux for welding gold and as an embalming agent by the Egyptians. As supplies became easier to get, namely from Italy, boric acid and borax became increasingly used as a mild antiseptic, especially for eyes and burns.

For the last 200 years, boric acid has often been used as a food preservative, but this use has been recently stopped because it tended to disguise food that was unfit for use as being in a reasonable condition for use. People must have ingested considerable quantities without any ill effect during this period. Much has been used as a simple home remedy for stings and burns, and as a powder to prevent rash.[1]

Antipathogenic Action. Boric acid and borax in a 2-3% solution will prevent the growth of most bacteria and will kill many fungi. A 1.5% solution has some stimulating effect on phagocytosis *in vitro,* but at 2% this ceases.[6]

Biochemistry.[10] Borates are active complexing agents for diol groups particularly in secchorides, and in some of the B-vitamins and ascorbate and can inhibit certain enzyme reactions. They can reverse gel formation.

Pharmacology. These substances are readily absorbed by damaged skin and by mucous membranes. 50% of borate is eliminated via the kidneys in the first 12 hours, and 90% of the remainder is gone within a week, in all but extreme doses.[7]

Borates are slightly astringent and will tend to allay the pain of burns and wounds. If the dry powder is introduced to the nose, it can bring on sneezing and lacrimation.[6]

Toxicology. These substances are not dangerously toxic, but large doses can be dangerous. The LD-50 for borax is 5.33 g/Kg for guinea pigs, and 2-3 g/Kg for Swiss mice. But for boric acid, it is greater than 4.1 g/Kg for mice.[9]

Rats and dogs were fed a diet containing 52.5, 117, 350, 525, and 1750 ppm boron as borate and as boric acid for up to 38 weeks. In this period, reproductive studies were possible. Only the highest level was there any toxicity with congestion of the kidneys, liver, small gonads, thickened pancreas, and a swollen brain. Even at 525 ppm, there was no adverse effect. Rats ingesting 350 ppm boron for 2 years showed no histologic changes at necropsy.[9]

Some workers have shown that 3 g boric acid or 5 g borax have no effect on the adult human, while others have reported symptoms at 1-2 g per day.[2] No one is likely to take too much in their food even if they do use a supplement that has only a few mg per tablet. Greater absorption is likely to come from a mouthwash or if a borate is applied to damaged skin.

Extensive laboratory studies on both man and animal have not shown the exact role of boron in their metabolism. Patients have been given 10 g/day for extended periods and were still excreting boron after 7 weeks. The LD-50 for the dog is 1 g/Kg and these dogs developed a violet red skin color with persistent vomiting, diarrhea, and meningismus. Acute intoxication can include hypothermia, depression, and ataxia.[5]

With daily doses of 100 mg/Kg, it takes 18 days for the dog to reach a plateau in boron excretion.[5]

The literature from 1848 to 1948 contains data of 86 cases of borax-boric acid toxicity and 42 of these died. Some were given doses of over 100g, yet many had no real confirmation of the cause of death. One 2 day old infant died and this was blamed on the mother who cleansed her nipples with a boric acid solution. A proper autopsy and analysis should have been used to prove the cause of death. Many of the deaths were due to absorption of borax/boric acid through damaged skin. Granulating skin will readily absorb these substances and so will mucous membranes.[5]

The acute toxic dose for an adult is from 20 to 60g in a single dose, but infants have died with 5g, yet others lived after being given 9g boric acid. There is a great individual variation with these substances. A 50% plasma — Ringer's solution IV is the best antidote and will increase the LD-50 for mice by 250%.

The Position in Australia. In 1981 or soon after, the various states scheduled boron compounds in any concentration, and this is an extreme case of bureaucracy because an apple can contain over 10mg of elemental boron. Many fruits and vegetables contain over 50 ppm boron and when these are grown on a really good soil, they will have up to 160 ppm boron. Should these foods be scheduled?

Yet at the same time, a mouth-wash containing 68% borate was acceptable for OTC sales. A good mouthwash with this substance would put many mg of boron into the blood. To become dangerous, the solution would have to be held in the mouth for many hours. Strong solutions or the powder when introduced into other body cavities have proved fatal. That legislation was introduced because a product called Bor-ex containing 5% boron was having remarkable results with both rheumatoid and osteo arthritis. Without advertising, the sales of this product went from zero to 10,000 bottles a month in 5 years. No unwanted side-effects were noticed during these 5 years.

A properly organized trial of Bor-ex is being carried out in one of the country's bigger hospitals. This started 3 years ago, but very regrettably is still not completed.

Carnarvon has 0.2 ppm boron in the water supply and people do go there from 1000 miles away in the Southwest to enjoy the good climate and get relief from their arthritis. It is really the good water and not the good climate that helps them. Yet some people in Carnarvon never drink local water. A survey was conducted there in 1981 that brought these facts to light.

The Position in the Rest of the World. West Germany stopped the use of boron compounds in medicine three years ago on the assumption that there were other drugs that would do everything that boron would do and that they would do it better.

In many other countries, a boron supplement is being used as a food supplement, and no claims are made, but satisfied users soon tell other people who need it. Over 250,000 people have used this supplement, and it corrects between 80 and 90% of all arthritis. No untoward side effects have been noted, but there are some useful side-effects, such as would be noticed if boron were the limiting factor in a person's well-being. Cardiopathies have been corrected, vision has been improved, psoriasis has been much improved, balance has been corrected. Arthritis in horses, cattle, dogs, deer, and goats have all been corrected.

As we use more and more superphosphate on our food crops, the availability of soil boron is decreased. It is estimated that most people in western societies ingest about 2 mg boron daily. This is based on the analysis of school meals in the U.S.A,[3] but analyses earlier in this century put the figure at 8 mg.[9]

The prevalence of arthritis seems to follow inversely the availability of boron in the soil. Jamaica has the least boron and 70% with arthritis. Mauritius has very little and has arthritis. Northern Thailand is very short of boron and much arthritis, but no figures are available. In Fiji, the Indians have much more arthritis than do the Fijians, and the reason is that Indians eat mostly rice while Fijians eat mostly starch root vegetables. Monocotyledons have a much less need for boron than do the dicotyledons.

The Theory Behind Boron Metabolism. Based on work done at Oxford in the Agriculture Faculty[4] it is believed that at the cellular level mineral metabolism is similar with both plants and man. If this can be relied on, then boron is a membrane catalyst which allows various ions to pass through the cell membrane, particularly phosphates to support synthesis of ATP. This will give energy for efficient repair. It is obvious that in osteo arthritis the cartilage is worn out, if it is because it lacks the necessary energy for cell division, it explains the action of boron. Then in rheumatoid arthritis, there is an autoimmune reaction for no known reason. It is suggested that the reason is that certain collagen fibers are overage and cannot repair themselves, due to lack of energy-rich compounds within the cells.

Other Boron Compounds. Boranes are hydrides of boron and are very toxic. They are used as solid rocket fuels and can be used to prevent bacterial decontamination of diesel fuel.

Boron analogues of many of the amino acids have been made and tested in North Carolina. The original research was to find carcinostatic compounds of boron, but some of these are also anti-arthritic, anti-inflammatory, anti-tumor and anti-hyperlipidemic in their action on test animals. The amino carboxyboranes are relatively non-toxic, but the cyano boranes are very toxic. More will be heard about these compounds.[7]

Some of the analogues of amino-acids have an LD-50 of 1800mg/Kg so they do not present any problems.

The Future of Boron. When the aforementioned trial is completed, it is likely that many people will require the boron supplement so as to relieve their arthritis and the health departments over-reaction will have to be reversed. Farmers will also have to look more to quality instead of quantity, and will have to add trace elements to their soil so as to give good quality crops.

Bibliography

1. Fisher, R.S. Boron — its occurrence in nature, foods, and humans. *Dept. of Medicine, Univer. of Maryland*; 1959.

2. Levinskas, G.J. *Metallo-Boron Compounds and Boranes*. Ed. Adams, R.M. Interscience Publishers, New York; 1964: Ch. 8.

3. Murphy, E.W., Watt, B.K., & Page, L. Regional variation in vitamin and trace element content of type A school lunches. *Trace Substances in Environmental Health* IV, Univ. of Missouri; 1971: 194-205.

4. Parr, A.J. & Loughman, B.C. *Boron and Membrane Function in Plants. Metals and Micronutrients: Uptake and Utilization by Plants*. Academic Press; 1983: Ch. 6.

5. Pfeiffer, C.C. & Jenny, E.H. The pharmacology of boric acid and boron compounds. *Bulletin of National Formulary Committee* xviii: 57-80; 1950.

6. Solis-Cohen. *Pharmacotherapeutics*. 1928: 583-585.

7. Spielvogel, B.F. *Synthesis and Biological Activity of Boron Analogues of the Alpha Amino Acids and Related Compounds*. Duke University. Durham Press: 119-129.

8. Underwood, E.J. *Trace Elements in Human and Animal Nutrition*. Academic Press; 1971: 434-436.

9. Weir, J.R. & Fisher, R.S. Toxicologic studies of borax and boric acid. *Toxicology and Applied Pharmacology* 23: 351-364; 1972.

10. Zittle, C.A. *Reaction of Borate with Substances of Biological Interest. Advances in Enzymology*, vol. 12, Interscience Publishers; 1951.