

Arthritis: A Puzzle Solved?

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scleroderma patient for 24 yrs.; on AP for 17 years

The problem with arthritis, as I see it, is that with most treatments, remissions are rare and cures even more so. Each new drug, believed to be a “breakthrough” and the magic bullet that will finally bring lasting relief, over time becomes a disappointment. Even when symptoms improve, x-rays often show that the disease has progressed. And then there are the sometimes severe side effects, occasionally even fatalities, of many of the immune suppressing drugs.

Patients go to physicians for healing. With progressive diseases like arthritis, lupus, scleroderma and other rheumatoid diseases, remission is hoped for, and a cure the ultimate goal of the patient. But the failure of each new drug to provide either of these goals on any lasting basis puts both patient and physician in a difficult, sometimes adversarial basis.

This is not to say that nothing works. There are treatments, both natural and pharmaceutical, that do work for some. So why are they not more universally accepted and used? Perhaps the answer to that lies in the confusion or lack of understanding of the mechanism or cause of the disease itself.

This pursuit was the life’s work of Dr. Thomas McPherson Brown, a rheumatologist-researcher with a unique gift of seeing the puzzle pieces and being able to put them in place in the context of the body’s natural ability to fight disease.

“The body doesn’t do unnatural things,” he said when countering the prevailing theory that rheumatoid disease is the body turning against itself (*autoimmunity*). “If the body appears to be attacking itself, it’s probably trying to reach something that doesn’t belong there, something that might be hiding inside or attached to the cells of the body.”

His search for that mysterious something began in 1937 and continued until his death in 1989. A starting point and model in 1937 was rheumatic fever caused by *beta hemolytic streptococcus* (strep) and the hypersensitivity that followed, and was responsible for the eventual cardiac problems. However, upon deeper investigation into cases that seemed to deviate from this pattern, he began to search for something additional. That was when he encountered strep’s tiny traveling companion - mycoplasma.

Unlike chemical or drug sensitivities whose reactions go away when the offending chemical is removed, in rheumatic fever the hypersensitivity persisted. Why? His search and resulting discovery led him to study mycoplasma. As better scientific equipment became available, such as the electron microscope, Dr. Brown was finally able to photograph the tiny, elusive mycoplasma. Being both a treating physician and a researcher, he was able to apply his growing volume of information to his patients’ treatment and their response to his lab work.

Tetracycline antibiotics are the accepted treatment against mycoplasma and have quite benign side effects when compared to the stronger, more popular anti-rheumatic drugs. They work with few, if any side effects, and can be taken safely long-term.

Mycoplasma has a preference for mucous membranes and the joints. An infection elsewhere in the body: lungs, bladder, teeth, sinus, etc. could release an altered form of the causative organism (mycoplasma) which then migrates to another part of the body such as joints or connective tissue and remains there for perhaps years unnoticed. Then a stress, an injury, infection or exposure to an environmental toxin causes a small amount of antibody generated by the presence of the original mycoplasma. The resulting arthritis is a response to the hypersensitivity established in the body over a long period.

“The cyclical manner in which rheumatoid arthritis prefers to behave, the seasonal variations, the local influences of too vigorous physiotherapy (*or activity*) in flaring up the disease, infections and many miscellaneous factors doing the same thing would easily be explained on the basis of antigen release, and neutralization if you like, of antibody; removal of antibody temporarily until

another buildup begins - then another explosion (*occurs*) which could easily account for the cycle," Dr. Brown explained in a lecture to the Academy of Medicine.

Based on the basic premise that rheumatoid disease might be caused by a tissue hypersensitivity response to mycoplasma, Dr. Brown sought to test the theory in the lab and in patient response to treatment. A few other scientists shared his interest, and they learned from each other but most eventually followed other pursuits. Dr. Brown stayed the course and in doing so, gave thousands of rheumatoid patients a new lease on life - myself among them.

It took a few years of trying various antibiotics in differing dosages and frequencies before he found the one that gave the best results for the particular patient. Too high a dose, administered too frequently resulted in a Herxheimer flare (a temporary worsening of the disease). A reduced dose administered intermittently resulted in "increased strength, rising hematocrit, falling globulin, rising albumin and objective evidence of distinct improvement, particularly in those patients who had been going steadily downhill over the past ten years. This suggested to us that the work was worthy of being continued and second, that the constitutional elements of the disease were reversible." In the years that followed, the response of thousands of patients to antibiotic therapy bore this out again and again.

mycoplasma - a very small organism in which the ability to form a cell wall has been lost

hypersensitivity - abnormal sensitivity or reaction of the body to stimulus (*antigen*) of any kind.

antibody - the defender - a white blood cell that eliminates disease causing cells

antigen - the enemy - a protein marker on the cell that identifies the cell as self (*ok*) or non-self (*not ok*)

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