wine for the first correct answer!) In rodents, neonatal depletion of SP from afferent joint nerve endings markedly reduces the induction of arthritis with Freund adjuvant—the classic experimental model of autoimmune arthritis.[2,5,6]

The neurogenic theory of arthropathy might explain why anticonvulsants often appear to exert a positive effect on so-called autoimmune diseases. Patients with epilepsy taking phenytoin seem to be at lower risk for autoimmune disorders.[7] Scleroderma, very difficult to treat, was reported to respond to anticonvulsant drugs in studies conducted in the 1970s.[8,9] Moreover, scleroderma might not afflict paralyzed limbs; a similar observation has been made with psoriasis.[10]

Our treatment of the so-called inflammatory cascade is passable: it provides pain relief and some disease modification—but it does not deal with the underlying cause of the cascade. Animal experimentation points clearly to the direct effect of a neurogenic influence, which I would humbly suggest is the initial stimulus. All of our attention in the past has been focused on the inflammatory cascade—its effect on blood chemistry and its response to therapeutic agents, such as steroids or nonsteroidal anti-inflammatory drugs—but we continue to ignore the cause.

Perhaps the greatest defect in modern medicine is that once we have gone down the wrong path, it is difficult to change direction or admit failure. Better sooner than later! Most have forgotten that for centuries we got the circulation of the blood wrong. A modern example is the hilarity and disdain that greeted the scientists who dared to propose that infection with *Helicobacter pylori* was the fundamental cause of ulcers. To quote a popular song—When will we ever learn?

The critical test is to electrically stimulate the afferent nerves innervating joints in animals to see if this can trigger inflammatory arthropathy—a very fast, very cheap experiment. Would anticonvulsant drugs provide protection also?

—Alan Russell MD MB MRCP
Brampton, Ont
by e-mail

References

1. Glick EN. Asymmetrical rheumatoid arthritis after poliomyelitis. *BMJ* 1967;3:26-9.
2. Levine JD, Dardick SJ, Roizen MF, Helms C, Basbaum AI. Contribution of sensory afferents and sympathetic efferents to joint injury in experimental arthritis. *J Neurosci* 1986;6(12):3423-9.
3. Holzer P. Local effector functions of capsaicin-sensitive sensory nerve endings: involvement of tachykinins, calcitonin gene-related peptide and other neuropeptides. *Neuroscience* 1988;24(3):739-68.
4. Levine JD, Moskowitz MA, Basbaum AI. The contribution of neurogenic inflammation in experimental arthritis. *J Immunol* 1985;135(2 Suppl):843s-7s.
5. Maggi CA, Meli A. The sensory-efferent function of capsaicin-sensitive sensory neurons. *Gen Pharmacol* 1988;19(1):1-43.
6. Niissalo S, Hukkanen M, Imai S, Tornwall J, Konttinen YT. Neuropeptides in experimental and degenerative arthritis. *Ann N Y Acad Sci* 2002;966:384-99.
7. Bobrove AM. Possible beneficial effects of phenytoin for rheumatoid arthritis. *Arthritis Rheum* 1983;26:118-9.
8. Morgan RJ. Scleroderma: treatment with diphenylhydantoin. *Cutis* 1971;8:278-82.
9. Neldner HK. Treatment of localized linear scleroderma with phenytoin. *Cutis* 1978;22:569-72.
10. Sethi S, Sequeira W. Sparing effect of hemiplegia on scleroderma. *Ann Rheum Dis* 1990;49(12):999-1000.

Competing interests?

The article by Flook et al[1] on management of undiagnosed chest pain suggests a therapeutic trial of a proton pump inhibitor. While this approach sounds wholly reasonable, this particular article raises a serious issue of credibility. The authors state, under competing interests, "None declared." At the same time, Dr Karlson (one of the contributing authors) discloses that he works for AstraZeneca, who manufacture one of the proton pumps recommended. I do not doubt that the good doctor *declared* that he had no competing interests, but I would suggest that, absent some good evidence to the contrary, it would certainly appear otherwise.

In recent years, increasing attention has been paid by journal editors to ensuring the scientific accuracy and validity of articles published under their banner. I fully appreciate that it is simply impossible for

FOR PRESCRIBING INFORMATION SEE PAGE 1248

editors to verify the truth and accuracy of every declaration made in the hundreds of articles which cross their desks, but when the information available conflicts, perhaps they have an obligation to question the statements. In this instance, if indeed there be a valid explanation for the apparent discrepancy, perhaps even the unusual step of an editorial reassurance that the accuracy of the declaration has been verified would serve to re-establish the credibility of the content of the article. The phenomenon of "ghost-written" medical literature is well established. As it stands, it appears that you have been used by the pharmaceutical industry to publish very well crafted advertising in the guise of science. I sincerely hope that this appearance is unfounded.

—David Maxwell MD CCFP(EM)
Halifax, NS
by mail

Reference

1. Flook N, Unge P, Agréus L, Karlson BW, Nilsson S. Approach to managing undiagnosed chest pain. Could gastroesophageal reflux disease be the cause? *Can Fam Physician* 2007;53:261-6.

Response

The team of primary care authors that worked together to write the chest pain article[1] was supported by consultation with a cardiologist and research associate, Dr Karlson, who now works for AstraZeneca.

Our literature search was thorough and complete. The article is properly referenced, and it took us 3 years from the time we started writing until the article was published. The evidence quoted in the article has been rated for its value, and we are satisfied that the conclusions are evidence-based and accurate. We are happy to discuss any concerns that readers might have about the content or conclusions contained in the article.

Dr Karlson's employee relationship is clearly documented in the list of authors' affiliations at the start of the article and was clearly written on the disclosure form sent to the journal before publication. The authors do not know why the publication did not also list Dr Karlson's employee relationship in the competing interest section as it did in the authors' section.

—Nigel Flook MD CCFP FCFP,
Edmonton, Alta
—Peter Unge MD PhD, Stockholm, Swed
—Lars Agréus MD PhD, Stockholm, Swed
—Björn W. Karlson MD PhD,
Mölndal, Swed
—Staffan Nilsson MD, Norrköping, Swed
by e-mail

Reference

1. Flook N, Unge P, Agréus L, Karlson BW, Nilsson S. Approach to managing undiagnosed chest pain. Could gastroesophageal reflux disease be the cause? *Can Fam Physician* 2007;53:261-6.

Response from the Editor

Canadian Family Physician has a policy that all authors of a manuscript submitted to the journal are required to declare in writing any competing interests. Competing interests are published in the journal to allow readers to "make an informed decision about the existence and impact of potential conflicts of interest or bias."[1] We also ask reviewers and editors to reveal any potential conflicts of interest when reviewing a particular manuscript. If there is a conflict of interest, the reviewer or editor is asked to decline to comment on the manuscript.

Dr Flook and the other authors of the article in question[2] had completed the competing interest forms as requested. Inadvertently, the declared competing interests were omitted from the published version of the paper. It is our error, and we apologize for this omission. Please see the correction below.

—Diane L. Kelsall MD MEd CCFP FCFP
Editor, Canadian Family Physician

References

1. Scott-Lichter D; Editorial Policy Committee, Council of Science Editors. *CSE's white paper on promoting integrity in scientific journal publications.* Reston, Va: Council of Science Editors; 2006.
2. Flook N, Unge P, Agréus L, Karlson BW, Nilsson S. Approach to managing undiagnosed chest pain. Could gastroesophageal reflux disease be the cause? *Can Fam Physician* 2007;53:261-6.

FOR PRESCRIBING INFORMATION SEE PAGE 1248