

Correspondance

Acute muscle spasm

In a *CMAJ* research letter, Norman Epstein described 8 patients with acute muscle spasm.¹ The diagnosis appears to have been based on subjective measurements of range of motion and pain; Epstein did not state how range of motion and tenderness were measured.

There is no proof that pain symptoms or decreased range of motion result directly from abnormality or spasm of muscles. There is no gold standard for diagnosing paralumbar spasm. There is no electrodiagnostic test or other objective investigation to prove that muscles are abnormal. There is also no medical literature proving that muscle spasm, especially paralumbar spasm, can be diagnosed clinically with scientific validity or reliability.

As E.W. Johnson writes, "In spite of overwhelming evidence that skeletal muscle spasm is nonexistent, physicians are continually deluged with seductive ads to prescribe expensive muscle relaxants."² I agree that a randomized, blinded, placebo-controlled trial is needed to corroborate the findings in this case series.

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2. Johnson EW. The myth of skeletal muscle spasm. *Am J Phys Med Rehabil* 1989;68:1.

There is evidence that pain symptoms or limitations of movement can be attributed to muscle spasm: Zhu and colleagues recently concluded that "the close correlation among evoked potentials, paraspinal muscle spasm and clinical symptoms suggest that the measurement of muscle activity may be more important to low back pain than is commonly accepted."¹

My study was not based on a subjective diagnosis of pain and range of mo-

tion. As noted in my article, I measured pain using an authenticated visual analogue scale and I used a standard clinical examination to determine range of motion.² I did not measure "tenderness."

Involuntary muscle spasm (dystonia) has been extensively studied by neurologists and neurophysiologists. It has been treated with some success with anticholinergics and other therapeutics, particularly botulin toxin.³⁻⁶

On a purely practical and clinical level, muscle spasm remains an important cause of patient suffering. Alleviating that suffering, by non-narcotic means if possible, is the bottom line. Perry Rush and I are in agreement that this case series must be put through the rigours of a randomized controlled trial before benzotropine becomes the standard of care, as I suggested in my article.²

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Just a family doc

As a first-year family practice resident in British Columbia, I was not surprised by this year's residency match results.¹ Throughout my medical school training, family medicine was looked down upon as a career. Physicians in specialty areas would often dis-

parage the "family doctor" who did a poor job managing a case. Worse off were rural family physicians, who were often criticized openly because of delays in care or lack of knowledge. Sadly, when asked their specialty by medical students, family physicians would almost invariably reply, "I'm just a family doctor."

I chose this residency only because I entered medical school knowing I wanted to practise family medicine. Once I began my medical education I did not receive any encouragement to pursue this path until I actually spent time in a family practice elective.

Unless attitudes toward family medicine change in our academic training centres, we can expect an American-style system, where specialists outnumber family physicians. Perhaps then a better appreciation of primary care will emerge.

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Exploring the actions of vitamin C

Vitamin C is an antioxidant with significant physiologic actions. For instance, it has been reported to lower erythrocyte sorbitol concentrations, which may make it useful in treating diabetes.¹ Data from the 3rd National Health and Nutrition Examination Survey showed that mean serum vitamin C concentrations were significantly lower in people with newly diagnosed diabetes than in people who did not have diabetes,² lending support to the earlier belief that diabetes mellitus may be associated with decreased serum vitamin C concentrations. These results

support the contention of Sebastian Padayatty and Mark Levine that subclinical vitamin C deficiency is more common than is generally recognized.³ An inverse association has been reported between plasma vitamin C concentration and glycosylated hemoglobin,⁴ suggesting that measures to increase plasma vitamin C levels may help to reduce the prevalence of diabetes. A colleague and I reported that 1500 mg of vitamin C, when given orally, reduces plasma glucose levels in patients with type 2 diabetes.⁵

Other studies have suggested that vitamin C reduces blood pressure.^{6,7} It may augment prostaglandin F and nitric oxide synthesis,^{8,9} which could account for its beneficial actions in diabetes and hypertension. The interactions of vitamin C with eicosanoids, nitric oxide, platelets, leukocytes and endothelial cells, among other types of molecules and cells, may account for some of its hitherto-unexplained beneficial actions. As suggested by Padayatty and Levine, more studies should be conducted on vitamin C's actions in various tissues, including cancerous ones.³ Until further studies are completed, however, caution should be exercised in advocating its use as an anti-cancer compound.

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[The authors respond:]

There is no doubt that vitamin C is a powerful antioxidant in vitro. It is less certain that vitamin C has antioxidant actions at physiologic concentrations in vivo.

Smokers and people with diabetes often have an unhealthy lifestyle, including a low intake of fruits and vegetables; this could partly account for the low plasma vitamin C concentrations in these groups. The low vitamin C levels could also be due to low bioavailability, increased utilization or even laboratory artifacts owing to oxidation of vitamin C during sample processing. Many apparently healthy people also have low plasma, and probably tissue, vitamin C concentrations.

In addition to its antioxidant effect, Vitamin C has been demonstrated to have many favourable actions in vitro and under experimental conditions in vivo. However, it is uncertain whether plasma concentrations of vitamin C higher than that necessary to prevent scurvy have any clinical benefit, except to serve as reservoir to forestall scurvy. Some studies have shown an association between increased vitamin C concentrations and a beneficial clinical outcome.¹ However, most of these studies do not distinguish whether the beneficial effect is due to vitamin C or something else, such as other components of fruits and vegetables or lifestyle factors. When pure vitamin C is administered, benefits are hard to demonstrate. For example, small short-term studies have shown a reduction in systolic blood pressure with vitamin C treatment² but the findings of larger studies are inconsistent.^{3,4}

Despite intense public interest in vitamin C, widespread use of this vitamin and decades of experimental studies, its role in health and disease remains unclear. We believe that one path to clarity is to determine the effects of vitamin

C on targeted clinical outcomes in healthy people as well as in people with pro-oxidant states, such as smokers and patients with diabetes; the effects must be determined in relationship to the concentration of the vitamin. As in vitro studies and surrogate markers alone cannot predict a clinical outcome, practising physicians will expect us to show clear clinical benefit before they use vitamin C for prevention or treatment purposes.⁵

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The abortion issue

Having read the well-written article by Laura Eggertson,¹ I feel that her thrust (and that of Planned Parenthood, the Canadian Abortion Rights Action League, Health Minister Allan Rock, etc.) is that it is a scandal and surprise that a “medically necessary” operation — abortion — is not universally accepted like other procedures.

You do not hear most of the old debating arguments about abortion any more, but the one that will not go away