Letters to the Editor

Hyaluronic Acid Injections for Knee Osteoarthritis

To the Editor: When I read the FPIN's Clinical Inquiry summarizing the effectiveness of hyaluronic acid injections, the most commonly used form of viscosupplementation for adults with knee osteoarthritis, I was surprised by the authors' conclusion.[1] The authors accurately reported the data from the sources they cited; however, they did not mention the large amount of data from randomized trials. Randomized trials represent the least biased source of effectiveness data, overcoming most of the biases found in observational studies, yet they were not included. Instead, the authors reported data on outcomes from multiple retrospective and prospective cohort studies and from cost-effectiveness analyses based on observational studies.

A 2022 meta-analysis reported outcomes from 24 large placebo-controlled randomized trials (N = 8,997; 24 weeks median follow-up) evaluating viscosupplementation for knee osteoarthritis in adults.[2] Viscosupplementation was statistically more effective at relieving pain than placebo; however, the difference was not clinically meaningful (the equivalent of a 2-mm decrease on a 100-mm pain scale). Similarly, it found small, clinically meaningless improvements in function. Although serious adverse events were infrequent, they occurred more often in the adults who received viscosupplementation (3.7% vs 2.5%; number needed to harm = 84; 95% CI, 49-319).

Randomized trial data should always be included in a comprehensive assessment of the best available evidence for a therapeutic intervention. Based on the best evidence, the correct conclusion is that viscosupplementation provides little or no benefit and should not be recommended to our patients.

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Author disclosure: No relevant financial relationships.

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In Reply: I agree that if all else is equal, randomized controlled trials are the preferred type of evidence to guide clinical decision-making. However, the quality of randomized controlled trials varies, and a meta-analysis is only as useful as the studies it includes. Pereira, et al., note that there is "extreme between trial heterogeneity" in the studies they included, making their conclusions less certain.[1] Furthermore, of the 25 studies they used for their analysis, the five (20%) that were described as unpublished were less favorable to hyaluronic acid than the 20 published studies. They also state that their "findings represent summary estimates and do not exclude the possibility that selected osteoarthritis patient populations could benefit from viscosupplementation."

The specific question addressed by the FPIN's Clinical Inquiry was not how well hyaluronic acid treats pain and function, but whether the treatment successfully delays expensive knee replacements and thus decreases costs.[2] Knee osteoarthritis is an extremely common diagnosis with significant morbidity and limited treatment choices. Physical therapy and exercise are often not tolerated by, or effective for, these patients. When conservative management fails, options include steroids and hyaluronic acid injections, which have mixed evidence of utility; orthobiologics; "regenerative medicine," which is exorbitantly expensive and currently has very little supporting evidence; and surgery. Many patients want to postpone surgery as long as possible, so we sought to determine if hyaluronic acid can successfully do that.

The heterogeneity of the studies reviewed by Pereira, et al., suggests that some patients may experience significant benefit from hyaluronic acid injections and others may not.[1] This is where it is important to remember that patients are individuals, not statistical averages. Hyaluronic acid should not be recommended to all patients, but it can be a treatment option for select patients. These patients include those with comorbidities that prohibit surgery, older patients who could potentially delay a knee replacement for the rest of their lives, and younger patients who could delay a knee replacement long enough that when they have surgery, it is more likely to last for the rest of their lives without needing a revision surgery.

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Comments About Evidence-Based Vasectomy

To the Editor: Thank you for publishing the informative article "Vasectomy: Common Questions and Answers" in the September issue of *American Family Physician*.[1] I greatly appreciated the article and would like to offer a few updates reflecting practices that are becoming increasingly common as vasectomy gains popularity among patients.

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The article notes that vasectomy anesthesia typically involves 10 mL of lidocaine using a 25- or 27-gauge needle. In contemporary practice, the volume and needle size are often lower.[2] From my experience with training many physicians in performing vasectomies and observing numerous vasectomy clinics in the United States and internationally, the 30-gauge needle has become more widely used, and the volume of anesthetic administered is smaller. Our teaching emphasizes that precise placement matters more than high volume.

Additionally, readers could infer that excising a segment of the vas deferens is recommended during vasectomy. However, the 2015 American Urological Association (AUA) vasectomy guideline states: "The panel found no consistent evidence indicating that division with excision of a short vas segment (less than 4 cm) are preferable to division without excision of a vas segment."[3,4] Excision does not improve the effectiveness of vasectomy and adds procedural complexity, including following protocols for tissue disposal and increasing cost without benefit to the patient.

Thank you for considering these clarifications to ensure that *American Family Physician* readers have the most up-to-date, evidence-based information.

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In Reply: Thank you for your comments. We agree that less lidocaine volume and a smaller needle gauge is preferable for most patients. We did highlight the mini-needle technique that uses 2 mL of lidocaine 2% with a 30-guage needle and noted that its effectiveness is similar to more traditional techniques.[1,2] The lead author of our article has trained residents in vasectomies for 20 years during his time in the military, and he has found that 2 mL of lidocaine 1% or 2% per side was enough when the overlying skin and vas deferens were appropriately targeted with palpation. However, it is helpful for learners to have extra lidocaine readily available when it is noted that initial anesthesia was incomplete during dissection of the vas deferens.

We also agree that removal of a vas deferens segment and simple excision are both acceptable options; there is no difference in sterility rates, especially with the addition of mucosal cautery and/or fascial interposition.[3,4] We would like to note that the 2012 AUA guidelines were published in print, whereas the 2015 update is available only online (the AUA notes that the 2012 published document should be cited when the 2015 update is referenced). Both versions note the same literature review time frame in their methods and make the same recommendations, but there is more detailed literature discussion in the published version. As such, both guidelines note that there is inconclusive evidence to recommend segment removal or simple incision, with the 2012 guidelines noting that a 1-cm segment is probably adequate if a segment is removed. Both guidelines recommend against removing longer segments (greater than 4 cm) because it requires further dissection of the spermatic cord and may have higher complication rates, even if it decreases failure rates.[3,4] In December 2025, the AUA released a new guideline that reviewed the same studies we addressed in our article.[5] The new guideline recommends techniques using fascial interposition and mucosal cautery together (category B rating) and used studies where segment excision (1-2 cm) was included. The AUA continues to recommend against using only ligation (with suture or clip) and segment removal due to high failure rates (category A rating).[5] We would also like to acknowledge that segment removal requires additional procedural complexity, and disposal may be cost prohibitive in some settings. However, for learners, the further dissection needed to obtain a segment allows for better understanding of the vas deferens anatomy.

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Benefits vs Harms of More Intensive Systolic BP Targets

To the Editor: We appreciate the thoughtful editorial by Drs. Ebell and Slawson on blood pressure (BP) targets and the importance of shared decision-making.[1] However, additional

context and more recent evidence are needed. Challenging a recommended systolic BP goal of less than 130 mm Hg risks adding confusion and hesitancy at a time when clarity and forward momentum are urgently needed.

The authors cited a 2020 Cochrane review in their editorial, but multiple randomized controlled trials have since been published.[2] A more recent 2025 meta-analysis incorporating data from four randomized controlled trials published after the 2020 Cochrane review further strengthens the evidence supporting lower systolic BP targets.[3] Compared with standard BP control, intensive control is associated with statistically significant reductions in myocardial infarction, stroke, heart failure, and cardiovascular mortality. The 2025 meta-analysis showed a number needed to treat of 58 to prevent one major cardiovascular event.[3] In time-to-benefit analyses, intensive BP control prevented one major cardiovascular event per 100 patients at 34 months.[4]

Adverse events were more common in the intensive BP control group in the 2025 meta-analysis, with a number needed to harm of 55, but they were generally mild and manageable with medication adjustments.[3] Older adults who are frail, those with limited life expectancy, and those unable to tolerate lower systolic BPs remain appropriate candidates for individualized targets. However, for most patients, the benefits of a systolic BP goal less than 130 mm Hg outweigh the risks.

Concerns about BP measurement techniques in trials such as SPRINT (Systolic Blood Pressure Intervention Trial) are legitimate but should not eclipse the broader evidence.[5] In the 2025 meta-analysis, most of the trials included protocols with staff-attended BP measurements, which is closer to routine practice. Newer guideline targets already incorporate a pragmatic buffer by recommending systolic BP less than 130 mm Hg instead of less than 120 mm Hg, which is used in most trials.[3,6]

With increasing evidence, most national and international organizations have updated their recommendations to favor lower systolic BP targets. The 2025 American College of Cardiology/American Heart Association hypertension guideline is cosponsored by a broad coalition of 13 professional societies.[6] In addition, guidelines from the US Veterans Affairs, Kidney Disease Improving Global Outcomes, American Diabetes Association, Hypertension Canada, Japanese Society of Hypertension, European Society of Cardiology, and International Society of Hypertension endorse systolic BP targets of less than 130 mm Hg for most adult patients.

Shared decision-making remains essential, but for most adults with hypertension and elevated cardiovascular risk, contemporary evidence supports systolic BP less than 130 mm Hg as a default treatment target.

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In Reply: Shared decision-making around BP targets is appropriate when relevant potential benefits and harms are present, especially when those harms and benefits may vary based on a patient's risk factors, age, personal values, and comorbidities. Although Drs. McDaniel and Jenkins dislike "confusion and hesitancy" and prefer "clarity and forward momentum," caution is appropriate when we are helping patients make critical decisions about lifelong treatment.

They cite a 2025 individual patient data meta-analysis of two randomized trials from the United States and four randomized trials from China.[1] It is notable that 83% of the data comes from studies set in China, including 43% from a cluster randomized trial in rural Chinese villages.[2] In the latter study, the final systolic BP in the intensive care group was 126 mm Hg; in the usual care group, it was 156 mm Hg. In effect, the studies compared intensive therapy with inadequate therapy, inflating any apparent benefit. Reporting of harms also appears to have been inconsistent in the Chinese trials. For example, acute kidney injury occurred in 4.1% of intensively treated patients in the US SPRINT trial, compared with only 0.1% of patients in two large Chinese randomized controlled trials reporting this outcome.[3-5] The same pattern is seen for syncope and worsening kidney function.

Drs. McDaniel and Jenkins acknowledge that patients "…unable to tolerate lower systolic BPs remain appropriate candidates for individualized targets," and we agree. They cite the Hypertension Canada guideline in support of broad use of the 130/80 mm Hg target; however, that guideline recommends the more intensive target primarily for patients with high cardiovascular risk who are able to tolerate more intensive therapy. In fact, the guideline states: "The committee recognizes that the benefit of pharmacotherapy initiation in people at lower risk (i.e., systolic BP 130-139 without any high-risk conditions) is uncertain."[6]

In addition, six of the nine physicians on the Hypertension Canada panel were hypertension specialists.[6] Studies have shown that the composition of a panel reflects the intellectual biases of its members, with subspecialists generally favoring more intensive treatment and, as we noted in our editorial, primary care physicians taking a broader perspective of the whole patient that more fully balances benefits, harms, and individual patient values.[7]

The American College of Cardiology/American Heart Association guidelines included 13 professional societies with an impressive alphabet soup of acronyms, but this panel was dominated by subspecialists and did not include representation from the two most important primary care groups: the American Academy of Family Physicians and American College of Physicians. Family physicians should not only be at the table, they should hold the leadership role when guidelines are developed to inform our practice.

Regarding measurement of BP, the SPRINT, ESPRIT (Effects of Intensive Systolic Blood Pressure Lowering Treatment in Reducing Risk of Vascular Events) trial, and BPROAD (Blood Pressure Control Target in Diabetes) study use intensive and time-consuming office protocols that do not reflect the real world of clinical practice, with SPRINT being most relevant to a US primary care setting. The notion of a buffer between the desired target of less than 120 mm Hg and the stated target of 130 mm Hg is a novel concept but does not change the fact that treatment decisions should be based on the actual BP experienced by patients during their daily life and is not likely to cause harm due to overtreatment.

In conclusion, we stand by our core assertions: a 130/80 mm Hg target is not appropriate for all patients, and if pursued after shared decision-making, it should be based on the BP measured at home or office BP measured with protocols used in the SPRINT and BPROAD studies.

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Editor's Note: Dr. Ebell is deputy editor for evidence-based medicine for *AFP*.

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What Do Guidelines Say About First-Line Treatment of Acute Low Back Pain?

To the Editor: We enjoyed reading Dr. Earwood and colleagues' excellent and thorough article regarding acute low back pain.[1] The cost of treating low back pain in the United States is $134.5 billion annually and increasing.[2] Despite rising costs and increasing awareness, patients continue to receive care that is inconsistent with clinical guidelines, including an overreliance on passive modalities.[2]

We worry that the article overstates the evidence supporting routine use of acupuncture, dry needling, trigger point injections, and transcutaneous electrical nerve stimulation—all of which increase initial cost and have inconsistent supporting evidence.[2-4] Best evidence and guidelines for acute nonspecific low back pain recommend reassurance, advice to stay active and resume normal activities, and education on self-management and seeking care only when needed.[3,4] If pain or functional disability persists, then passive modalities such as those mentioned above may be considered. However, much of their benefit may derive from the placebo effect and is unlikely to change the natural history of low back pain.[5]

We also applaud the authors' reference to the OPAL (opioid analgesia for acute low back pain and neck pain) trial that demonstrated a lack of improvement in low back pain with an opioid prescription.[6] However, this trial has limitations when informing opioid recommendations. The OPAL trial treated patients with up to 12 weeks of pain, including those outside of typically defined "acute" periods; used a sustained-release oxycodone-naloxone formulation as opposed to an immediate-release opioid; and measured outcomes starting at 2 weeks instead of immediately after administration, when pain relief is needed most and opioids are likely to show an effect. Thus, although it is an important addition to low back pain research, the OPAL trial may not have actually addressed whether immediate-release opioids are effective at improving acute low back pain. In patients with contraindications to other medications (eg, nonsteroidal anti-inflammatory drugs), a short

course of opioids for severe acute low back pain at the lowest effective dose is reasonable and consistent with guideline recommendations.[3,4]

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The views expressed in this publication are those of the authors and do not necessarily reflect the official policy or position of the Department of the Navy, the Department of War, or the US government.

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In Reply: We appreciate your comments. We are also concerned about the increasing cost of lower back pain in the United States. The vast majority of the cost estimate you quote ($134.5 billion annually) is from the management and disability of chronic lower back pain, not acute.[1] The guideline on management of acute lower back pain has largely remained unchanged for decades. Despite that, you correctly identify that costs are increasing. In 2019, low back pain in general was in the top 10 leading causes of disability-adjusted life-years worldwide in all age categories, a 46.9% increase since 1990.[2] As physicians, we tend to blame nonadherence to guideline-directed therapy for this increasing cost, which has some merit. Sadly, this lament has not stemmed the increase.

In an era of decreasing patient trust in the health care system and physicians, reassurance is not enough. There is a link between trust in the physician and health outcomes.[3] Additionally, there are comorbid conditions that increase a patient's likelihood of developing chronic lower back pain (eg, smoking, obesity, mood disorders, sedentary lifestyle, certain occupations).[4] Management of acute lower back pain in isolation is efficient in clinic but is likely missing the mark.

In addition to managing comorbid conditions, using modalities that have evidence of reducing acute lower back pain and are unlikely to harm the patient is a reasonable approach. Similarly, avoiding modalities with evidence of harm or no evidence of improvement is a reasonable approach. There is a large body of evidence of harm from use of opioids in treatment of chronic lower back pain. Similarly, there is evidence that opioid use for acute lower back pain leads to chronic opioid use.[5,6] Although there are situations where opioid use should be considered for acute lower back pain, it deserves a healthy reconsideration if other modalities can be used with a reasonable expectation of success.

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Corrections

Incorrect units. "DASH Diet for the Primary and Secondary Prevention of Cardiovascular Diseases" (Cochrane for Clinicians, February 2026, p. 127) included incorrect units from the Cochrane Review and subsequent incorrect conversions to conventional units in the Practice Pointers section related to lipid reductions. The online version of the article has been corrected.

Incorrect formulation. "Menopause Management: When Hormone Therapy Is Appropriate" (Editorials, February 2026, p. 115) referred to the incorrect formulation of estradiol in Table 2. The online version of the article has been corrected. ■