Reply to the Letter to the Editor

Editor's Spotlight/Take 5: CT Pulmonary Angiography After Total Joint Arthroplasty: Overdiagnosis and Iatrogenic Harm?

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I appreciate the comments and insight offered by Dr. Harris on my *Editor's Spotlight/Take 5* interview [4] with Dr. Leopold.

Dr. Harris asserts that the standard treatment for submassive or low risk pulmonary embolism does little for the thrombus that has already formed. Anticoagulation, so the theory goes, may help prevent further embolism, but does little for the clot that has already lodged in the pulmonary vasculature.

I agree with this assertion, but must acknowledge that there may be some theoretical treatment benefits of anticoagulation on an existing embolism. Heparin appears to inhibit the inflammatory cascade and prevent further leukocyte and platelet adhesion, which may limit in situ extension of the embolus [2]. This antiinflammatory effect of heparin may also prevent the chronic scarring within the pulmonary arterial lumen that can lead to pulmonary hypertension [5]. However, lacking from these theories are good data, and there is no

convincing evidence of which I am aware to support the clinical importance of these concepts. While the consensus is that immediate anticoagulation improves survival in patients with pulmonary embolism, there is a paucity of evidence from randomized trials to support this assumption. To my knowledge, the only randomized placebo-controlled trial of anticoagulation for acute pulmonary embolism that suggested a reduction in mortality was a 1960 study [1] that included only 35 patients. I am not aware of any prospective data on the mortality of untreated pulmonary embolism diagnosed following total joint arthroplasty.

It is clear that the potential benefits of anticoagulation come with a cost. I agree with Dr. Harris that identifying which subset of emboli that would benefit from treatment and justify the iatrogenic risks is critically important. I also agree that asymptomatic embolism following total joint arthroplasty is probably more frequent than commonly assumed; these silent emboli may not be clinically important or benefit from intervention. Had Dr. Harris used CT pulmonary angiography in his study in place of C¹⁵O₂ scans [3], the incidence of asymptomatic emboli would likely have been even higher given the increased sensitivity of the imaging tool. As Dr. Harris writes in his study, “the incidence of pulmonary embolization that does not lead to a positive C¹⁵O₂ scan is unknown.” [3].

I suspect that 50 more years of research will still not answer all of these questions. These issues are challenging to scientifically study due to cost, power, and ethical considerations. Furthermore, although the fundamental issues will remain, new imaging technologies and treatments will continue to introduce new concerns that will require critical evaluation. It is my hope that continued progress will be made in better defining the risks of iatrogenic intervention and the potential benefits of treatment of different subsets of pulmonary embolism following total joint arthroplasty.

(RE: Leopold SS. Editor's Spotlight/Take 5: CT Pulmonary Angiography After Total Joint Arthroplasty: Overdiagnosis and Iatrogenic Harm? *Clin Orthop Relat Res*. 2013;471:2733–2736.) The author certifies that he, or any members of his immediate family, have no funding or commercial associations (eg, consultancies, stock ownership, equity interest, patent/licensing arrangements, etc.) that might pose a conflict of interest in connection with the submitted article.

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