tion) over a treatment period of 5 weeks in patients with osteoarthritis of the hip. Initially, the manipulative therapy was more effective in reducing pain and increasing function, but it lost efficacy over time. Additional clinical studies are warranted to obtain information about the efficacy of manipulation therapy and to determine whether there are particular subgroups of patients who are most likely to benefit from this intervention.[1]

I agree with Beaty et al. that there are now a number of effective surgical options, before a total hip arthroplasty, that may be effective in patients with osteoarthritis of the hip. Arthroscopic or open operative techniques used for anterior femoral impingement resulting from acetabular dysplasia are reported to reduce pain and improve function and to prolong the time until total hip arthroscopy is required.[2,3] In addition, osteotomies of the femur, pelvis, or both can be performed to prolong the time to total joint arthroplasty. Although there are data on the efficacy of these techniques from case reports and reports on small case series, additional long-term follow-up is still required to determine which patient population with osteoarthritis of the hip is likely to benefit from these joint-saving techniques.

Nancy E. Lane, M.D.

University of California at Davis Medical School
Sacramento, CA 95817
nelane@ucdavis.edu

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More on HIV-Associated Kaposi's Sarcoma

TO THE EDITOR: The AIDS Malignancy Consortium, a multicenter cooperative group funded by the National Cancer Institute, has enrolled 442 patients who have human immunodeficiency virus (HIV) infection and measurable Kaposi's sarcoma in a series of therapeutic trials since 1996 (Table 1).

Our data suggest that persistent Kaposi's sarcoma despite apparently effective antiretroviral therapy is not a rare, isolated, or recent phenomenon, as suggested by Maurer and colleagues in their letter to the editor (Sept. 27 issue).[1] On the contrary, our findings indicate that since the introduction of effective antiretroviral therapy, many patients with AIDS-associated Kaposi's sarcoma have CD4 counts above the level typically associated with susceptibility to opportunistic diseases. Furthermore, although effective HIV suppression has been correlated with regression of Kaposi's sarcoma after antiretroviral therapy,[2] a substantial proportion of our patients had undetectable HIV viral loads.

These findings raise important questions about the mechanisms that control the progression of human herpesvirus 8 and Kaposi's sarcoma. They also suggest a need for studies to identify clinically relevant correlates that can distinguish between patients whose Kaposi's sarcoma responds to antiretroviral therapy and those who do not have such a response. These factors may include age, duration of HIV infection, human herpesvirus 8 viral load, and patterns of viral gene expression within tumors.

Table 1. Characteristics of Patients with Kaposi's Sarcoma Enrolled in AIDS Malignancy Consortium Trials, 1996–2007.

Variable	Value
Total no. of patients	442
Age — yr	
Mean	42
Median	40
Range	23–66
CD4 count — cells per mm^3	
Mean ±SD	329±379
Median	266
CD4 count ≥300 per mm^3 and undetectable HIV viral load — no./total no. (%)	96/332 (28.9)
Therapy with protease inhibitor or non-nucleoside reverse-transcriptase inhibitor, CD4 count ≥300 per mm^3, and undetectable HIV viral load — no./total no. (%)	78/248 (31.5)

Susan E. Krown, M.D.
Memorial Sloan-Kettering Cancer Center
New York, NY 10065
krowns@mskc.org

Jeannette Y. Lee, Ph.D.
University of Alabama at Birmingham
Birmingham, AL 35294-3300

Dirk P. Dittmer, Ph.D.
University of North Carolina at Chapel Hill
Chapel Hill, NC 27599-7290

for the AIDS Malignancy Consortium

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THE AUTHORS REPLY: With respect to the comments of Krown and colleagues: we note with interest the number of patients with a CD4 count of 300 per cubic millimeter or more and an undetectable HIV viral load who were enrolled in their therapeutic trials. We have not claimed that what we are observing in San Francisco is an isolated phenomenon and, on the contrary, have suspected it to be more widespread. However, Kaposi's sarcoma remains a striking rarity in published studies[1-5] and is not a well-described phenomenon in clinical practice.

It is difficult to compare directly the subjects in the trials by Krown et al. with our patients, since the correspondents have not provided data regarding the duration of the CD4 counts and viral loads, the duration of HIV infection, the CD4 nadirs of the subjects, the temporal relationship with the presentation of Kaposi's sarcoma lesions, and the response to antiretroviral treatment. Our patients have persistent Kaposi's sarcoma despite sustained high CD4 counts and undetectable viral loads for more than 2 years. The median CD4 nadir in our group is 340 per cubic millimeter, with a median duration of HIV infection of 18 years. We encourage Krown et al. and others to publish their data more fully.

Toby Maurer, M.D.
Maya Ponte, M.D., Ph.D.
Kieron Leslie, M.B., B.S., M.R.C.P.
University of California at San Francisco
San Francisco, CA 94110
maurert@derm.ucsf.edu

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Fluorodeoxyglucose PET in Relapsing Polychondritis

TO THE EDITOR: We report on a case of fever of unknown origin in which ^{18}F-fluorodeoxyglucose (FDG) positron-emission tomography (PET) was instrumental in depicting cartilage inflammation, which eventually led to a histologically confirmed diagnosis of relapsing polychondritis. Relapsing polychondritis is a rare inflammatory disease that may affect cartilage throughout the body, but the diagnosis may be challenging in the absence of typical auricular or nasal involvement, as in the patient described here.[1]

A 67-year-old man presented with a 1.5-year history of persistent cough with clear secretions, progressive shortness of breath, sore throat, bloated stomach, low-grade fever, malaise, and weight loss. Physical examination revealed tenderness on all sternocostal junctions and in the laryngeal region. Vesicular breath sounds were decreased. The erythrocyte sedimentation rate was 130 mm per hour, and the C-reactive protein level was 20.0 mg per deciliter (normal level, <0.5). The white-cell count was 15,000 per cubic millimeter, with 86% neutrophils. The lactate dehydrogenase level was 516 U per liter (normal range, 240 to 480). All cultures and serologic tests were negative. The antinuclear factor level was normal, and the level of c-antineutrophilic cytoplasmic antibodies was slightly increased.

The patient's condition improved with the administration of corticosteroids for the treatment of