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Reply

To the Editor:

We thank Dr. Raskin for his thoughtful and provocative comments regarding computed tomography (CT) of the craniocervical junction in rheumatoid arthritis. We believe the difference between his work and ours, particularly regarding flexion and extension computed tomographic views, may be explained on the basis of patient selection. In Raskin's work, CT scans were performed on all rheumatoid arthritis patients in whom there was atlantoaxial subluxation. Other clinical features were apparently not a consideration in patient selection.

In our study, patients were retrospectively reviewed because they were possible neurosurgery candidates, regardless of the presence or absence of anterior subluxation. We also considered posterior subluxation, pseudo-basilar vagination, and other abnormalities in evaluating these patients.

In any population such as this, in whom there are neurologic deficits to begin with, we elect not to perform flexion and extension views for several reasons. First, symptoms in these seriously ill patients might be exacerbated by performance of flexion. Second, the patients are unable to flex fully due to their clinical status, and are unable to maintain a position of flexion. Third, different patients could maintain different degrees of flexion.

Thus, we believe degree of flexion was not reproducible from patient to patient. Indeed, there may not have been reproducibility between consecutive examination results in the same patient. Therefore, we elected to evaluate standardized neutral examinations. Even on plain radiographs, the reproducibility of flexion in the lateral projection is an important consideration in the evaluation of flexion and extension radiographs.

We concur with Dr. Raskin and his colleagues that computed tomography should not be a screening examination, but it may be useful in selected cases. In our patient population, not only were we unable to standardize the positioning of the patients and flexion, but flexion itself may have been potentially hazardous.

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Connective tissue disease after augmentation mammoplasty

To the Editor:

We read with great interest the report by Kumagai et al on 18 patients who developed connective tissue disease after cosmetic surgery (1). We have reported 3 patients in Singapore who developed connective tissue disease after augmentation mammoplasty (2). One patient had features of systemic lupus erythematosus. Another had features of progressive systemic sclerosis. Unlike the patients described by Kumagai, this patient responded to steroids and not to nonsteroidal antiinflammatory drugs. The third patient had features of idiopathic thrombocytopenic purpura. Steroids and cytotoxic agents were used without success, and splenectomy was performed but yielded no improvement. Since there was no report of this clinical manifestation in Kumagai's large series of patients, this may represent a new clinical finding.

In the course of our review of the literature on "human adjuvant disease," we noted 2 reports which suggest that there may be an acute form of the condition as well. Uretsky et al (3) in 1979 described a patient who developed arthritis, renal failure, and bilateral pulmonary infiltrates shortly after bilateral silicone transplant. Chastre et al (4) in 1983 described 3 patients in France who developed acute pneumonitis 1–3 days after subcutaneous injection of silicone. The patients developed acute respiratory failure. Known infective agents were excluded as the cause. Silicone was found in the cells and supernatant from bronchoalveolar lavage. These observations would suggest that silicone could cause an acute disease in humans.

Although a causal relationship between the cosmetic surgery and subsequent development of connective tissue disease in humans is difficult to prove, a study involving male transsexual patients who have undergone sex change operation and received silicone implants or injections may shed some light on this difficult issue. We propose to study the problem in such patients in Singapore, with particular emphasis on the material injected and possible host factors. We regret the omission of HLA typing results in Kumagai's 18 patients and the 28 others they reviewed from the Japanese literature.

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