ment of inflammatory cell infiltration (Figure 3) at the site of ligamentous insertions (enthesopathies), particularly at the posterior tip. Shadows need substance for their interpretation, and systematic pathology supplies that substance.

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Reply

To the Editor:

Trauma may affect the joints of rheumatoid patients whether those joints are themselves involved by the rheumatoid process or not. If they are already so involved, it may be difficult to distinguish the effects of trauma histologically. My article does not challenge Ball's hypothesis of primary rheumatoid inflammation in the neurocentral joints or its possible extension into the adjacent discovertebral joints.

Professor Bywaters' position is somewhat confusing. He apparently accepts the role of trauma but prefers to postulate that synovial cell transformation at moving interfaces occurs first and this becomes the site of rheumatoid inflammation. However, disc destruction is not always attended by significant inflammatory cell infiltration and I, like others (1), believe we should be cautious about interpreting any tissue damage in rheumatoid patients as being of rheumatoid origin. Moreover, microscopic observations of granulation tissue at

sites of cervical disc involvement do not preclude the possibility that trauma plays a more important role in causing the extensive cervical discovertebral destruction seen radiologically. Furthermore, this has more significance than the "which-came-first-chicken-or-egg" problem, for if abnormal cervical mobility is indeed an important pathogenetic factor, more rigorous cervical immobilization may be beneficial.

I did not argue that the process in the dorsal discs is traumatic simply because they have no neurocentral joints. In the cases I have seen, the radiologic features of the disc lesions in the dorsal and lumbar segments suggested a traumatic rather than inflammatory etiology and histologic findings appear to support this interpretation (1). Reference to the absence of neurocentral joints in the dorsal spine was intended to show that one need not predicate involvement of neurocentral joints to explain the discovertebral destruction. Professor Bywaters believes that such destruction in the dorsal spine represents extension from arthritis in the costovertebral joints. We too have recognized costovertebral joint involvement radiologically (2), but this is an uncommon finding. I can accept that such extension may involve the adjacent disc but do not believe that the usual cause of the disc lesions in the dorsolumbar spine seen radiologically is inflammatory. Severe disc destruction is relatively uncommon in the dorsolumbar spine possibly because the latter is relatively stable, whether the costovertebral or apophyseal joints are affected or not. Furthermore, not all would agree with Professor Bywaters' histologic interpretation of what constitutes a rheumatoid lesion in the dorsolumbar discs (1).

As for the reference to "shadows . . ." I admire Professor Bywaters but I believe that he, like the rest of us, deals with shadows whether they are on a viewbox or in a microscope. Furthermore, radiologists, like pathologists, require substance to form shadows and in both cases it is the interpretation that counts!

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