was noted with low doses of prednisone, however, in contrast with the dramatic improvement obtained in the original papers.^{5 6}

On the other hand, synovial biopsy in our case did not show malignant infiltration. Thus it is reasonable to suggest that the rheumatic manifestations belong to a paraneoplastic syndrome, as has been previously reported with other neoplasms.⁷

To our knowledge this is the first description of peripheral synovitis and pitting oedema as an initial manifestation of non-Hodgkin's lymphoma. We would like to emphasise the need to consider the possibility of an underlying malignancy in a patient with these clinical characteristics who is unresponsive to the usual medical treatment.

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Arthritis and carcinoma

Sir: We read with interest the paper by Chakravarty and Webley describing two cases of asymptomatic renal cell carcinoma causing an acute monoarthritis, previously unreported.¹ They suggested that this occurrence is both rare and underreported. In our experience this is not necessarily the case. We have recently seen two patients whose cases illustrate some interesting similarities.

Patient No 1, a 63 year old man presented to our hospital with a five week history of a painful, swollen knee joint and was unable to bear weight. Clinical examination confirmed an acute monoarthritis of the knee joint, with a tense effusion, warmth, and tenderness. He had been previously treated for a large cell solid temporo-orbital tumour by radiotherapy because the lesion was considered inoperable. The knee joint effusion was aspirated to rule

out sepsis. Bacteriology was negative, no crystals were found, but adenocarcinoma cells were seen in abundance. He made an excellent functional recovery after palliative irradiation to the knee.

Patient No 2, a 63 year old woman, was referred to the radiotherapy department of the same hospital with metastatic disease for which no primary tumour had been located despite extensive investigation. She had initially presented with a painful knee of one year's duration. Clinical examination showed an acute monoarthritis with a moderate effusion. Isotope bone scan disclosed a hot spot in the upper tibia. She had a synovial biopsy and cytological examination of the synovial fluid. The synovial biopsy sample showed evidence of infiltration with adenocarcinomatous cells as did the synovial fluid.

While we agree with the authors that in cases where there is doubt about the cause of a joint effusion, early examination of synovial fluid is important, and may prevent the need for an open or closed bone biopsy, we are of the opinion that the occurrence of malignant joint effusions is not rare but more likely to be underreported.

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Angiotensin converting enzyme in rheumatoid arthritis

Sir: Being interested in vascular endothelial cell transdifferentiation,¹ I read with interest the instructive articles by Veale *et al*² and Goto *et al*³ about the angiotensin converting enzyme production in rheumatoid arthritis. Although there is no doubt that vascular endothelial cells participate in this process, the role of macrophages remains questionable because the antibodies used for their identification (antigen CD14) are not macrophage specific and cross react with vascular endothelial cells.^{4 5} Moreover, undifferentiated vascular endothelial cells can transdifferentiate into macrophage-like cells and migrate into the extravascular space.⁶

It may be useful to re-evaluate the role of macrophages in rheumatoid arthritis in the light of the new knowledge about the transdifferentiation of vascular endothelium.⁶ For example, the mesenchymoid transformation cells responsible for joint destruction differentiate into fibroblasts in due course⁷ and are not, therefore, inflammatory cells. McCachren considers, however, any collagenase producing cells immunoreactive with Leu-M3 or HAM56 to be macrophages.⁸ Again, these antibodies are not macrophage specific and cross react with vascular endothelium.^{9 10} Mesenchymoid transformation cells remind me of undifferentiated vascular endothelial cells¹ by their character and aggressiveness.⁷ The facts that antiangiogenesis suppresses arthritis¹¹ and

that angiotensin II is an angiogenic factor¹² are in excellent keeping with that proposition.

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Endothelium damage and von Willebrand factor antigen

Sir: We read with great interest the recent paper by Blann *et al* on damage to the endothelium in Sjögren's syndrome.¹ The authors examined the relation of the auto-antibodies SSA and SSB to endothelium damage using serum levels of von Willebrand factor antigen (vWFAG) as an index of damage to the endothelium.

Damage of the endothelium may be present in Sjögren's syndrome, but there is no real evidence whether such damage results in high or low levels of vWFAG. The extent of the production of vWFAG in megakaryocytes in Sjögren's syndrome and other diseases is unknown; mainly because the plasma levels of the factor do not follow changes in the numbers of thrombocytes in peripheral blood and so this production site is no longer a part of the scenario. Further, in our opinion, endothelium damage cannot explain why increased concentrations of vWFAG are present in such varied diseases as proliferative glomerulonephritis, diabetes mellitus, systemic sclerosis, Sjögren's syndrome, and arteritis temporalis, as endothelial damage in these diseases is not a common denominator.

We have recently studied the immunohistochemistry in arteritis temporalis, in which