scientists who meet in various places, holding a never-ending series of seminars; rather painfully reminiscent of some of our activities in medical working parties and the like.

Among his biographical works is his description of the tragedy of Paul Kammerer in *The Case of the Midwife Toad*, where the scientist committed suicide when his Lamarckian beliefs that pigmentary characteristics could be acquired and passed on genetically turned out to be due to subcutaneous Indian ink. But the better known political side of Koestler is still topical. His *Spanish Testament*, in which he describes his experiences when locked up by the Franco regime, reminds us of the immense and hopeful changes that have taken place in Spain recently; and *Darkness at Noon* is still the best description of the ways in which people may be manipulated within a totalitarian society. Both his scientific and political messages, as expressed in these works, appear to me to have great relevance to us as physicians entering the 1980s. It is as doctors as well as individuals that, I feel, we are threatened by a host of events exemplified by the suspension of *The Times*. Let us hope that its reappearance is an augury of better things generally.

Are there any recent advances in the diagnosis and treatment of the collagen disorders? What is known about possible aetiological factors? If steroids are given is it possible to add azathioprine as a means of keeping steroid dosage to a minimum, and what treatment is advised to counter the side effects of steroids?

The term "collagen disorders" incorporates many diseases. Most doctors now include in this category such diseases as rheumatoid arthritis, systemic lupus erythematosus, polyarteritis nodosa, dermatomyositis, and Sjögren's syndrome. These disorders are characterised by a chronic inflammatory process in the affected tissues such as the joints, muscles, skin, and kidney, accompanied by a wide range of serological disorders, of which the most characteristic are hypergammaglobulinaemia, various autoantibodies, and evidence of complement consumption. Much is known about the mechanisms that lead to tissue damage in these disorders.[1] Much of the tissue damage results from the inflammatory response provoked by the deposition of immune complexes in different organs. These complexes result from the combination of the provoking antigens with antibody. Circulating immune complexes are seen transiently in various conditions including the normal host response to many microbial infections. Various defects may allow these complexes to accumulate to the point where these cause tissue damage including complement deficiencies and impaired function of the reticuloendothelial system. In addition, several defects in the regulation of the immune response have been described in diseases such as systemic lupus erythematosus, which may permit the uncontrolled persistence of initially appropriate immune responses that are switched off in normal individuals. The provoking antigens have not been discovered in most patients with collagen disorders, and until this is done the aetiology will remain unknown. It is suspected, however, that a variety of antigenic stimuli can provoke connective tissue disorders in susceptible individuals as, for example, hepatitis B virus in some patients with polyarteritis nodosa and drugs in some patients with systemic lupus erythematosus.

The best means of countering the side effects of steroids in treating these diseases depends on two major principles. Firstly, these drugs should be used only when the severity of the disease makes such treatment unavoidable. In most patients with systemic lupus erythematosus, for example, steroids are unnecessary. Secondly, the dose should be kept to the minimum needed to control the activity of the disease; it is often possible to use steroids on alternate days rather than daily, and this has allowed the resumption of normal growth in some children—for example, receiving steroids for juvenile chronic polyarteritis. The daily regimen for taking steroids is also important and resorting to a single morning dose of steroids, for instance, lessens the risk of producing adrenal suppression. Various therapeutic measures are available to permit a reduction in total steroid dosage.[2] These include the cytotoxic drugs, such as azathioprine, cyclophosphamide, and chlorambucil; plasma exchange; and intermittent infusions of steroids in high dosage. There is little controlled evidence that such measures are helpful, but most clinicians treating patients with severe connective tissue disorders resort to such measures in managing individual patients.

[1] *Immunopathogenesis of Rheumatoid Arthritis*, ed G S Panayi and P M Johnson. Chertsey, Surrey, Reedbooks, 1979.
[2] Hurd, E R, in *Immunology in Medicine*, ed E J Holborow and W G Reeves, pp 1067-1098. London, Academic Press, 1977.

Is hormone replacement therapy of any value in the treatment of menopausal hair loss?

The mechanism of this disorder is unknown, and it is not even clear whether it is a separate entity; for example, a clear relation to the menopause itself has not been shown, and the disorder could equally represent several non-related conditions, including that of simple age-related loss presenting first in those who have always had sparse hair. Although some believe it to be hormone dependent and analogous to male-pattern baldness, there have been no satisfactory trials of antiandrogens.