In fact, unlike A B Garrod, Gosse used *rheumatism* with the general meaning of aglic fluxion not restricted to the joints, a meaning in line with that used by Galen, which today remains in parallel with the nosographical nomenclature. He emphasised the relationship of such congestive changes with cold (particularly a rapid passage from warm to cold) and with the nervous system. His 'ideas' of rheumatism were the result of an extension of the above mechanisms to a large panoply of conditions including practically all those which are painful, congestive, or febrile. His study is obsolete regards today's nosology, and was obviously expressed in the words of his day, before knowledge of the aetiological data that were revealed by advances in bacteriology. However, it foretold the modern investigations into the diffuse role of non-specific vascular disturbances and of neurotransmitters in neurogenic inflammation.

While each of these meanings of *rheumatoid* had its own logic, a long term follow through confirms that the term had a useful place in the progress of an already established nosography, though it was abandoned when it served only to extend an already unclear pathological entity. As a term the modern biology is providing a plethora of basic information in rheumatology, the origin of the term *rheumatoid arthritis* must not be forgotten. In extensions such as *rheumatoid nodules* or *rheumatoid factor*, the adjective *rheumatoid* is elliptical, as the changes which are thus named cannot be directly explained by the etymology, but are indirectly related to it through a nosological entity.

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**Interleukin-6 in clinical relapses of polymyalgia rheumatica and giant cell arteritis**

Polymyalgia rheumatica and giant cell arteritis are related diseases associated with increased concentrations of acute phase reactants. Clinical symptoms and acute phase reactants respond promptly to corticosteroid treatment in most patients, and a decision to withdraw prednisone may be recommended after two years of evolution of the disease. This may represent a critical phase for patients with polymyalgia rheumatica, who are exposed to relapse with threatening symptoms of giant cell arteritis. At present there are no signs or tests that distinguish patients with evolving disease from those in complete regression: changes in the acute phase reactant concentration (ESR) and C reactive protein (CRP) are used as aids in the management of the disease, but their values remain normal in about 50% of patients with clinical relapses of polymyalgia rheumatica and giant cell arteritis who are receiving treatment.

Furthermore, diagnosis of relapse is frequently based on the clinician's experience in detecting clinical symptoms and signs of disease. This would be of help in enabling more precise monitoring of patients receiving treatment, and might also make possible a reduction in the duration of steroid treatment.

Interleukin-6 (IL-6) mediates the acute phase response by hepatocytes. Its concentration is increased in patients with polymyalgia rheumatica and giant cell arteritis before corticosteroid treatment, and while this increase is not specific to these diseases, IL-6 could be a biological marker of their disease activity. To determine the value of IL-6 as such a marker in clinical relapses of polymyalgia rheumatica and giant cell arteritis, we studied the IL-6 concentrations, with those of acute phase reactants, during the reduction of prednisone dosage in patients with polymyalgia rheumatica and giant cell arteritis.

Twenty patients (15 women, five men: 17 with polymyalgia rheumatica (criteria of Bird et al) and three with giant cell arteritis (American College of Rheumatology 1990 criteria)) were included in the study. Additional clinical evaluations were made by a consultant rheumatologist and blood samples were taken before and one month after 50% reduction of the prednisone dose, and at clinical relapse. The latter, defined as the reappearance of typical morning pain and stiffness of the shoulder and pelvic girdles, was identified by a rheumatologist blinded to the results of acute phase reactant and IL-6 assay.

When IL-6 concentration in blood was taken, clinicians took the decision to increase the dose of prednisone to that given before relapse. ESR was measured by the Westergren method; fibrinogen (NV < 4 g/l) and C reactive protein (NV < 10 mg/l), orosomucoid (α-2-glycoprotein; NV < 1.2 g/l), and haptoglobin (NV < 4.1 g/l for men, < 2.6 g/l for women) were measured by immunonephelometry; fibrinogen (NV < 5.5 g/l) was measured by a chromometric method. Plasma samples were stored at −80°C until required for determination of IL-6 concentration by radioimmunoassay (Medgenix).

Statistical analyses for unpaired and paired data were performed using the Mann-Whitney test and the Wilcoxon test, respectively. The level of significance was set at p < 0.05.

At entry to the study, all patients were in remission and were receiving a stable dose of prednisone less than 10 mg/day (mean 4.4 mg/day, range 2–12 mg). Nine patients had a clinical relapse within one month after their prednisone dose was tapered. No subsequent evaluation was made, so the occurrence of late relapse could not be determined in this study. The clinical characteristics of patients who did or did not experience clinical relapse were not different (table 1), and there was also no difference in concentrations of IL-6 and acute phase reactants between the two groups before...