numbers have changed little, but others have been superseded and the pattern of change has varied from joint to joint.

In the knee (433 procedures), the indications for arthrodesis (8) and osteotomy (19) are clear-cut and have not changed: mould arthroplasty (40) had its vogue; synovectomy (143) reached a peak of thirty a year and subsequently fell to ten to twenty, which now seems likely to remain fairly constant; synovectomy has only recently been recognized as an essential complement to excision of a popliteal cyst.

In the hip (266 procedures, 112 major), the incidence of most minor operations has not changed, with the exception of iliopsoas release (85) in vogue for 3 years, reflecting the continual search for an effective remedy for chronic hip pain and deformity. Excision arthroplasty is now obsolete and total hip replacement (Charnley) provides eighteen to twenty new hips annually without making excessive demands on nursing care and physiotherapy. There has been no significant change in the number of osteotomies (24) during the 15 years.

In the wrist (231 procedures), arthrodesis (22) has been largely superseded by excision of the lower end of the ulna (84) providing relief of pain and a strong grip in addition to increased mobility.

In the elbow (116 procedures, 71 major), synovectomy has replaced excision of the head of radius as the main indication for operation. Operations on other joints have been important for individual patients but have made little impact on the surgical treatment of rheumatoid arthritis. Cervical fusion has saved four patients from devastating disability or death.

Discussion

DR. L. HOLT (London) Many of our cases are dealt with as out-patients. This has many benefits because the patient is not worried, and there is less fuss and less separation from home.

DR. F. ANDREWS (Reading) This creates a serious administrative problem. We are now devoting two operating lists a week to out-patients and more than two-thirds of our rheumatic beds are being used for surgery. Nevertheless, it appears that we are still only recognizing a quarter of those who require surgery.

DR. HILL We already do some minor operations on a day-case basis but we have not yet done a synovectomy. This procedure is usually performed on four or five joints at a time and it seems too much to expect anyone to accept this as an out-patient.

Cellular Hypersensitivity to Salivary Blood and Liver in Sjögren's Syndrome. By H. BERRY and P. A. BACON (St. Bartholomew's Hospital)

Sjögren's syndrome is the association of keratoconjunctivitis sicca and xerophthalmia (sicca syndrome or SS) with arthritis or other collagen disorder. SS has recently been described in association with certain 'auto immune' liver diseases, such as primary biliary cirrhosis and chronic active hepatitis.

Patients with Sjögren's syndrome show immunological disturbances with hypergammaglobulinaemia, a range of non-organic specific autoantibodies, and depressed reactivity to *D.N.C.B.* The histology of the salivary glands

shows infiltration with small lymphocytes and Sjøborg (1968) has shown cellular reactivity to parotid extracts in some patients with Sjögren's syndrome.

We have investigated cell-mediated immune reactivity to liver and parotid gland extracts, using the leucocyte migration test (IMT) in three groups of patients:

- (1) With auto-immune liver disease;
- (2) With Sjögren's syndrome with arthritis;
- (3) With rheumatoid arthritis with no evidence of SS;
- (4) A control group of healthy persons and patients with other diseases.

The results may be tabulated as follows

Group	1	2	3	Controls
No. of patients	34	14	44	40
+ve LMT to liver	26	0	1	1
+ve LMT to parotid	11	12	10	0

Reactivity to liver was seen in 77 per cent. of Group 1, but only one patient with rheumatoid arthritis and one control. Reactivity to parotid was seen in 33 per cent. of Group 1, 90 per cent. of Group 2, and 22 per cent. of Group 3. A labial biopsy was performed in five patients in Group 2 and in five positive and five negative reactors in Group 3.

This study confirms that SS is seen in certain liver diseases in the absence of arthritis. Cellular reactivity to liver is practically confined to autoimmune liver disease but reactivity to parotid gland is seen in patients with SS associated with arthritis disease. Parotid reactivity is also seen in some patients with rheumatoid arthritis alone. This may be evidence of early or subclinical SS.

Discussion

PROF. I. M. ROITT (London) This is an encouraging start, but I think that you must take into account the studies of Brostoff (1970) suggesting that leucocyte migration inhibition with mitochondria may occur in all sorts of unexpected conditions, such as Hashimoto's disease, pernicious anaemia, and other autoimmune diseases. Your extracts are extraordinarily crude and I think that you must try to characterize the principle in your parotid gland extract which specifically reacts with the Sjögren's syndrome patients.

DR. BERRY I should like to thank Professor Roitt for his helpful suggestions, which we would like to look into.

DR. R. N. MAINI (London) We are by no means clear about the significance of inhibition of leucocyte migration and whether it is necessarily a test of cell-mediated immunity, *i.e.* thymus or T-cell dependent immunity (Federlin, Maini, Russell, and Dumonde, 1971). We must also ask ourselves whether the results that you obtained were in any way related to the pathogenesis of the disease. The question arises because release of parotid tissue antigens may follow injury from disease, and may therefore be of secondary importance. Have you looked at patients who have parotid damage from something like an infection, where immune mechanisms are not of primary aetiological importance, to clarify this point? Therefore, does this test tell you that pathogenetic significance is reflected in the reactivity to the 'antigens' you have used?

DR. BERRY This is a very small series but there was a very nice correlation with the degree of lymphocyte infiltration which might tie in with the idea that it is in fact showing a cellular immune mechanism.

DR. P. FOWLER (*Macclesfield*) A very few rheumatoid patients develop parotid gland enlargement when treated with Pyrazoles. It is interesting to speculate that these patients might be the ones in your rheumatoid group with sensitivity to parotid antigen, but have never produced any clinical signs of this until they have been treated with this group of drugs. It would be very interesting if you could look into this.

DR. BERRY I should be very interested in doing something along these lines.

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Proteolytic Activity of *Mycoplasma arthritidis*. By J. W. CZEKALOWSKI, D. A. HALL and P. WOOLCOCK (*University of Leeds*)

It has been claimed that mycoplasmas are associated with certain symptoms of arthritis, both in man and in animals. One of the reasons for the development of arthritic lesions could be that changes may take place in the protein-polysaccharide complexes of joint tissue. Bearing this in mind, we have explored the effects of various mycoplasmas and their metabolic products on proteins which are likely to be encountered in joints either in native or modified forms. Of several mycoplasmas examined, only two strains, namely PG6 and PG27, of *Mycoplasma arthritidis* showed liquefying effect on gelatin, a property examined in our screening programme, and the factors responsible for this activity appeared both intra- and extracellularly.

Two enzymes one of high and the other of low molecular weight, have been isolated by processes of ammonium sulphate and acetone precipitation and gel filtration of the filtered growth medium. Although both enzymes attack gelatin, neither is active against intact undenatured collagen, and hence they should be classified as gelatinases rather than collagenases. However, since neither of them has any effect on other soluble proteins such as casein, they must be differentiated from enzymes displaying generalized proteolytic activity such as trypsin and chymotrypsin, which also attack gelatin. Both enzymes release free amino groups and oligopeptides from gelatin, but only the heavier molecule does this with the simultaneous liberation of free hydroxyproline. It may,

therefore, be assumed that the two enzymes attack different parts of the gelatin molecule and, in view of their high specificity of action, we consider that they may represent a new class of enzyme.

The association of these enzymes with strains of *Mycoplasma arthritidis* offers a suggestion that these organisms may participate in the further degradation of joint collagen after its initial denaturation by other so-far unknown factors.

Discussion

DR. A. G. S. HILL (*Stoke Mandeville*) You mentioned that PG27 came from a human case. I don't think you were very specific about a case of what?

DR. CZEKALOWSKI PG6 was isolated from a rat and PG27 from an arthritic patient, but not from the arthritic lesion. Over the years, either PG27 has become contaminated with a rat strain, as may happen in laboratories, or antigenic degradation has taken place during propagation. Serologically they are very close to each other, but it is still possible to show the difference between PG27, the original human strain, and PG6, the rat strain.

PROF. D. A. WILLOUGHBY (*London*) What is the pH activity of the enzymes that attack gelatin? They were tested at pH 6; what is the shape of the curve of activity of the enzymes?

DR. HALL They both appear to have a rather broad range of activity stretching from pH 6 to 9.

DR. M. WILLIAMS (*Liverpool*) A word about the confusion between *Mycoplasma arthritidis* and *Mycoplasma hominis* type 2: the latter was originally isolated from the female urethra and it was not associated with human rheumatoid arthritis until Jansson (1971) published her paper on the subject. The original confusion probably arose through PG27—the human strain of PG6—being isolated in separate laboratories and being passed from one laboratory to another. I do not think there is now very much doubt that the two strains are immunologically and serologically identical. With regard to the pathogenesis of mycoplasmas, they do appear to be quite 'host-specific' in terms of the pathology that they can produce. I have isolated *Mycoplasma fermentans* from human early effusions but have been unable to induce a similar arthritis with this organism in any other sort of experimental animal. From that point of view, it is comforting to know that *Mycoplasma arthritidis* is the only species which has been shown to produce gelatinases, since this organism appears to be pathogenic only for the rat.

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