DR. GENOVESE I cannot tell you what would happen because we did not, in fact, look for this.

DR. L. E. GLYNN (*Taplow*) May I make a rather far-fetched contribution to the explanation how popliteal cysts may conceivably protect against cartilage damage? The erosion of cartilage takes place by the growth of pannus both over the cartilage and under the cartilage into the bone. With an expanding popliteal cyst into which inflamed synovial membrane can grow, the membrane has an alternative pathway in which to move and therefore less tendency to grow over and under the cartilage. This would therefore be a protective mechanism.

DR. DIXON May I put a clinician's rather simpler explanation for that phenomenon. When you try to aspirate a rheumatoid cyst you have considerable difficulty and have to use a very large-bore needle as the material is thick and cheesy and contains the remains of vast populations of cells. I am sure that, if it is enzymatic degradation which damages the knee, then this source of enzymes is being continuously lost from the joint.

References

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Inheritance of Primary Articular Chondrocalcinosis. By J. GEERARDS and J. K. VAN DER KORST (*Division of Rheumatology, University of Nijmegen, Holland*)

Three brothers and one sister, as well as a maternal cousin, presented with multiple joint complaints of varying duration. All five showed characteristic x ray signs of multiple articular chondrocalcinosis. No abnormalities of calcium and phosphate metabolism were found. Haemochromatosis, gout, and diabetes mellitus were excluded by laboratory investigations.

Among 108 relatives above the age of 30 years who were interviewed and examined, sixteen additional cases of articular chondrocalcinosis were found. Seven of these persons denied any joint complaint, and six of the others had not sought help for their articular symptoms. In the three remaining cases, as in four of the *propositi*, bouts of acute arthritis had occurred, involving predominantly the larger joints, and especially the knees and the shoulders, after the age of 30 years. Degenerative polyarthropathy, also involving the spine, arose after the age of 60 years in—what seemed to be—the most severely affected cases. No conspicuous aggregation of other pathological features was found in this family.

Eleven females and ten males were affected in the two generations of the pedigree that were examined. In the younger generation articular chondrocalcinosis was observed only in children of affected parents, including sons of affected fathers. The pattern of occurrence of articular chondrocalcinosis in this family strongly suggests the involvement of a single dominant autosomal trait.

Discussion

DR. E. B. D. HAMILTON (*London*) I have two questions: First, were all these patients of Dutch stock, or were any of them of middle European or Jewish ancestry? Secondly,

you have mentioned that the spine was involved in one or two of the patients. Did any of the relatives have back symptoms? Siťaj and Žitňan (1967), whose patients also had a strong family history, found disc calcification by the third decade. They also noted gradual loss of mobility in the lumbar and cervical spine. This contrasts with the findings in the non-familial cases who usually present later in life.

DR. VAN DER KORST As far as we have seen, this pedigree was of pure Dutch origin. The earliest cartilages to calcify are the menisci in the knees, the triangular ligaments in the wrists, the symphysis pubis, and the intervertebral discs, but we have rarely found symptoms from the spine below the age of 60 years.

DR. A. ST. J. DIXON (*Bath*) In the x ray that you showed us there was calcification in the supraspinatous tendon as well as in the articular cartilage. To me tendinous calcification seems to be a different clinical condition. Thus it is non-familial in contrast to chondrocalcinosis, yet some people believe that these two diseases are the same. Did your family study produce any other instances in which there was extra-articular tendinous calcifications as well as intra-articular chondrocalcinosis?

DR. VAN DER KORST We only found extra-articular calcification in the shoulder, but this was not very frequent.

DR. P. H. N. WOOD (*Manchester*) If one looks at the so-called inborn errors of metabolism, they are all, at least so far, transmitted as recessive or as sex-linked traits. When one thinks one has found a dominant pattern of inheritance, this is likely to exclude an underlying enzyme defect.

DR. VAN DER KORST I completely agree with you.

References

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Vertical Subluxation of the Axis in Rheumatoid Arthritis.

By D. R. SWINSON, E. B. D. HAMILTON, J. A. MATHEWS, and D. A. H. YATES (*King's College Hospital and St. Thomas' Hospital, London*)

This paper has been published in the *Annals* (1972) 31, 359.

Discussion

DR. J. K. VAN DER KORST (*Holland*) We still have only limited experience of prognosis of cervical involvement in rheumatoid arthritis, but the longer we know this complication, the more we realize that the prognosis can be good. The atlas can sublux either downwards or forwards. We studied a patient who died because of spinal cord involvement in whom forward subluxation occurred because bony outgrowth from the odontoid process pressed the anterior arch of the atlas forwards. I think this is one of the few occasions in cases of rheumatoid arthritis in which bony outgrowth can contribute to the symptomatology of the disease.