Neurological Abnormalities complicating Sub-foraminal Osseous Disease in Chronic Rheumatoid Arthritis. By N. A. RANA, D. O. HANCOCK, A. R. TAYLOR, and A. G. S. HILL (*Oxford Regional Rheumatic Diseases Research Centre*)

The purpose of the investigation was to find out the character and extent of neurological involvement in atlanto-axial subluxation (AAS) an in upward translocation of the atlas (UT), 49 patients were selected who showed one or other of these changes.

Routine antero-posterior and lateral flexion/extension neck x rays together with lateral tomography in flexion and extension were done in 24 cases in which the odontoid process was not clear, together with a routine neurological examination. The degree of spinal canal narrowing caused by backward subluxation of the odontoid process was expressed as a percentage of the sagittal diameter of the spinal canal posterior to the odontoid process.

41 patients had AAS: fourteen of them showed no neurological abnormality, but the encroachment of the canal varied between 13 and 52 per cent. Two patients with UT also had a normal central nervous system.

Trigeminal nerve involvement was found in eight patients with AAS, the first division being consistently affected; the third division was not involved in any. Three patients with UT had similar involvement, but one showed impairment in the third division.

Seventeen patients with AAS exhibited central nervous system abnormalities which would not be expected to interfere with normal function. Thirteen had hyper-reflexia, usually asymmetrical—their range of subluxation was 13–52 per cent. Sensory signs were present in eight of this group, and were the only abnormality in one.

Fasciculation was present in four patients with AAS and in one with UT. Of the remaining two patients with UT, one showed spontaneous clonus associated with loss of consciousness, the other diminished superficial sensation with hyper-reflexia.

Five patients with progressive or urgent syndromes had an operation with good early results.

It is concluded that the important central nervous system findings consist of:

- (1) Impaired sensation in the first two divisions of the trigeminal nerve.
- (2) Hyper-reflexia, usually monomelic.
- (3) Patchy superficial sensory loss.
- (4) Fasciculation.
- (5) Preservation of position sense.

Discussion

DR. E. N. COOMES (*London*) Can you tell us anything about the size of these patients' cervical canals and how they compare with those of people with ordinary spondylosis who develop myelopathy?

DR. HANCOCK No. The way we compared each case with the others was, as I have said, to obtain a percentage so as to avoid the problem of comparing the sagittal diameters of the canals. The presence of neurological change was in no way related to the amount of the canal that was occupied. In fact, there was no correlation between the degree of occupation of the canal by the peg and the presence of neurological signs.

DR. E. N. COOMES (*London*) What about the corneal reflex? As you know, the centre for this reflex lies at C1/2 and I noticed many years ago that this reflex is one of the first things to disappear in atlanto-axial subluxation. I am rather surprised that you detected it in only one patient.

DR. HANCOCK It was absent in one or two patients but this was certainly not invariable. Of course we only had seven patients with trigeminal signs.

DR. J. A. MATHEWS (*London*) The danger of neurological damage due to atlanto-axial subluxation is, as you say, not only related to the amount of subluxation, but also to the speed with which it happens.

DR. HANCOCK The most interesting thing in atlanto-axial subluxation is to observe the patient under television x ray. As they bend the head forwards, the axis does not move forwards slowly, but suddenly. I suspect that this is when the centre of gravity of the head moves in front of the peg. With repeated flexion cumulative damage occurs to the anterior cord.

DR. D. HENDERSON (*Bath*) In view of the widespread nature of the lesions, is it naïve to suggest that it develops on a vascular basis rather than a purely traumatic one?

DR. HANCOCK Very reasonable! I think the pathogenesis of the neurological changes is in doubt. There is not only direct trauma of the peg on the cord, but also the vertebral artery could be compressed in the intervertebral canal. There may also be intrinsic vascular disease.

DR. K. A. MEIJERS (*Holland*) It is very difficult to decide about the importance of the circulation. We have studied one patient in whom both vascular damage and also direct trauma to the cord appeared important.

DR. HANCOCK A common sign in subforaminal tumours is wasting of the intrinsic muscles of the hands and it is suggested that this has a vascular basis. I wonder if we are not seeing a prodrome of this when fasciculation occurs in the hand intrinsics. Is it due to the anterior cord being repeatedly traumatized or to direct trauma to the descending anterior spinal artery?

Treatment of Dislocations in the Cervical Spine in Rheumatoid Arthritis and Ankylosing Spondylitis complicated by Signs of Cord Compressions: A Follow-up Study. By K. A. E. MEIJERS, G. TH. VAN BEUSEKOM, F. DUYFJES, and W. LUIJENDIJK (*University Hospital, Leiden, Holland*) From 1961 until 1970 sixteen patients were treated with cervical cord lesions due to severe destructive changes in the cervical spine. In fourteen cases the diagnosis was RA, in one AS, and in another it remained obscure. A cord lesion was considered as soon as a patient started complaining about the following: severe neck pain, tingling or numbness in fingers and/or feet, severe loss of muscle power in arms and/or legs, problems with micturition, and jumping legs. At neurological examination the signs of a cord lesion were present.

Radiological changes were localized in the C1/2 area in twelve cases, below C2 in two, and in the whole spine in