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

two. Skull traction was applied for a few days to 10 weeks before operation. Neurological disturbances disappeared before operation in most cases. Owing to poor general health two patients were not operated upon. A stabilizing operation was performed in fourteen cases and a wire bone graft spondylosis was performed using the posterior approach. Postoperatively skull traction was continued for 2 to 5 months (average $2\frac{1}{2}$). The results are tabulated below.

Operation	Neurological symptoms directly after operation	Follow-up period (yrs)	Neurological complaints at follow-up
On C 1/2 7 RA 2 RA	None to few None to few	2-8 1½* 4*	Unaltered Many
1 RA	Died 15 days post-operatively of pulmonary embolism		
1 AS 1 not known	None None	9 6	None None
Below C2 1 RA	None	1*	Many
Both on and below C1/2 1 RA	Few	1	Unaltered

The postoperative neurological symptoms were none or few in all cases. In three patients (marked *), a relapse of the neurological complaints occurred. In two cases this happened 1 and 1½ years later due to an unstable graft; in the other case operated on for a slip C1/2, this was due to a new slip of the C7-T1 level 4 years after operation. Eight patients died 15 days to 8 years after operation. One was probably due to a cord lesion due to an unstable graft, and the others to miscellaneous causes.

Six patients are alive 2 to 9 years postoperatively, but one has a neurological relapse due to a slip of C7-T1.

At follow-up x ray we have found, in two RA cases, a progression of the destructive lesions and in two others ankylosis at lower levels. Except for the three patients with relapse, all were happy with the results of the operation. We think that the results of this type of treatment are gratifying and shall therefore continue with it.

Discussion

PROF. G. CHAPCHAL (*Holland*) I think it is most important to immobilize and reduce the condition. This is followed by fusion. We immobilize the head and neck in a 'halo' traction; this allows operation without any movement between the head and cervical spine and enables the patient to walk immediately after the operation.

DR. R. M. BENNETT (*London*) It has often occurred to me, in more flippant moments, that patients with erosions of the odontoid process may possess one peculiar advantage over the rest of us—namely, an immunity from judicial execution by hanging. The cause of death in hanging is usually attributed to a 'pithing' of the spinal cord by the odontoid peg.

DR. L. E. GLYNN (*Taplow*) I am probably the only person here who has ever done a *post mortem* on persons who have been hanged, and I did this on two who were executed in Pentonville Prison some years ago. Neither had dislocations of the odontoid process; their cervical spines were broken much lower down.

DR. R. L. F. NIENHUIS (*Holland*) In patients with rheumatoid arthritis undergoing operations there may be cervical lesions without neurological symptoms or signs. This is especially important when they are going to receive endotracheal anaesthesia. Preoperative x rays of the cervical spine must therefore be made.

DR. MEIJERS It is the custom in our unit to do that. We also ask the E.N.T. surgeons to examine these patients as there may be difficulties due to crico-arytenoid joint involvement. Regarding Prof. Chapchal's comment, our patients are so very disabled that a plaster jacket for them is a burden—so heavy that they are unable to stand it. This is why we have not used 'halo' traction.

DR. J. A. MATHEWS (*London*) I should like some practical advice. We try to protect the cervical spine of these patients by giving them restrictive collars of one sort or another. It seems to me that the more restrictive the collar, the more dangerous it may be to the upper cervical spine, as when we effectively immobilize the occiput and chin the only way the patient can open his mouth to eat is by flexion and extension of the upper cervical spine. There are some very complicated ways of overcoming this but I have no practical experience of success with these. Have you?

DR. MEIJERS Collars are a very difficult subject; the more complicated they are the more difficult they are to wear, and I think a simple cardboard collar is the best. Many patients patiently wear the collars in hospital but discard them at home. On one occasion we had a patient with a C3/4 subluxation who was immobilized in a collar and has worn this faithfully for 3 years. Now these vertebrae have fused.

Effects of Phenylbutazone on the Metabolism of ¹⁴C-Cortisol. By W. W. DOWNIE, G. REID, and M. C. K. BROWNING (*Departments of Pharmacology and Therapeutics, and Clinical Chemistry, Dundee University*)

It is known that drugs such as phenobarbitone and diphenylhydantoin alter the metabolism of endogenous cortisol by their effects on steroid-metabolizing enzymes in the liver. Phenylbutazone has been shown in animals to induce such enzymes, and as this drug is frequently used for long periods in the treatment of rheumatic disorders, it was considered of value to investigate its effects on endogenous cortisol metabolism in man. A number of indices were studied before and after one month's ingestion of phenylbutazone at therapeutic dose levels in normal volunteer subjects. These indices included the disappearance rate of ¹⁴C-cortisol from plasma, plasma 11-hydroxycorticosteroid levels, cortisol secretion rate, 6-hydroxycortisol, and the distribution of radioactivity in the urinary metabolites. A significant fall in the half-life of ¹⁴C-cortisol in plasma was noted, although there was no significant rise in the cortisol secretion rate nor in the excretion of 6-hydroxycortisol. An increase in the percentage of radioactivity associated with the urinary polar compounds, cortol and cortolone, was found, suggesting that phenylbutazone had produced enhanced activity of the enzyme 20-hydrogenase.