ATLANTO-AXIAL SUBLUXATION IN RHEUMATOID ARTHRITIS

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With greater awareness of the complications and with improvement in radiographic techniques, atlanto-axial subluxation is being diagnosed more frequently. Its recorded incidence varies with the selection of patients and the radiographic techniques used.

Martel (1961), Sharp and Purser (1961), Conlon, Isdale and Rose (1966), Mathews (1969), Park and O'Brien (1969), Meikle and Wilkinson (1971), and Stevens, Cartilidge, Saunders, Appleby, Hall and Shaw (1971) have tried to establish the incidence of atlanto-axial subluxation in patients with clinical evidence of rheumatoid arthritis. Conlon, Isdale and Rose found an incidence of 25 per cent (eighty-four out of 333) in hospital patients, taking 2.5 millimetres separation of the dens from the anterior arch of the atlas in females, and 3 millimetres in males, to be the lowest limits of abnormality. Mathews found that of seventy-six consecutive patients seen as out-patients a quarter had 3 millimetres of separation or more in lateral tomographs of the neck taken in flexion. Despite improved methods of diagnosis, the natural history of atlanto-axial subluxation is still not clearly known. Isdale and Conlon (1971) reported a tendency for the degree of subluxation to increase with time. Despite sporadic reports of fatalities from this complication there are few detailed reports of neurological involvement.

MATERIAL

The patients included in this study fulfilled the diagnostic criteria of rheumatoid arthritis as defined by the American Rheumatism Association (Ropes, Bennett, Cobb, Jacox and Jessar 1959). They were assessed clinically, radiologically and neurologically by a team consisting of rheumatologist, orthopaedic surgeon and neurosurgeon. Between January 1969 and July 1971 all patients with rheumatoid arthritis admitted to the hospital, new out-patients with a diagnosis of rheumatoid arthritis and old patients with rheumatoid arthritis who complained either of neck pain or of neurological symptoms, had the cervical spine radiographed, with antero-posterior and lateral views taken in flexion and extension by a standard technique. In the later stages the radiographic magnification was corrected by the use of a standard known-distance device inserted into the mouth of the patient. Experience with this device showed that the error from magnification was 1 millimetre. This amount was deducted in the earlier cases in which the device had not been used. Antero-posterior and flexion and extension tomography was done when there was doubt about the clarity of the occipito-atlanto-axial complex.

Forty-one patients were found to have atlanto-axial subluxation of 3 millimetres or more (Fig. 1). Forty were sero-positive and one sero-negative. There were thirty-one women and ten men. Their ages ranged from twenty-eight to seventy-six years, with an average of fifty-seven (Fig. 2). The mean duration of rheumatoid arthritis at the time of diagnosis of subluxation was sixteen years, the range being four to thirty-nine (Fig. 3); the mean duration of neck or neurological symptoms was three and a half years, with a range of one month to twenty years. Thirty-six of the forty-one patients were taking a corticosteroid (5 to 7.5 milligrams of prednisolone daily, or the equivalent).

MEASUREMENT OF ATLANTO-AXIAL SUBLUXATION

In all patients the standard radiographs of the cervical spine in flexion and extension were examined with particular reference to the position of the dens. To estimate the percentage

narrowing of the spinal canal affected by the movement of the dens two measurements were taken: 1) the distance (a) separating the anterior surface of the dens from the posterior aspect of the anterior arch of the atlas in flexion (Fig. 4); and 2) the distance (b) separating the posterior surface of the dens from the anterior surface of the posterior arch in extension (Fig. 5). The percentage narrowing of the spinal canal could thus be calculated as $\frac{a}{b} \times 100$.

The amount of atlanto-axial subluxation varied from 3 to 14 millimetres (Table I).

FIG. 1

FIG. 2

Figure 1—To show the amount of atlanto-axial separation in flexion in the cervical spine in forty-one patients. Figure 2—The age distribution in the forty-one cases.

FIG. 3

To show the duration of rheumatoid arthritis in forty-one patients with atlanto-axial separation.

CHANGES IN THE DENS AND OCCIPITO-ATLANTO-AXIAL COMPLEX

In addition to atlanto-axial subluxation various radiographic abnormalities were seen. Two patients had a thin dens (Fig. 6). One patient had no visible dens at routine radiography

FIG. 4

FIG. 5

Figure 4—A typical example of separation of the dens from the anterior arch of the atlas in flexion of the cervical spine. Figure 5—The same case showing the separation reduced in extension.

TABLE I

DETAILS OF FOURTEEN PATIENTS WITH ATLANTO-AXIAL SEPARATION BUT CENTRAL NERVOUS SYSTEM NORMAL

Case number	Age (years)	Sex	Neck pain	Relevant history	Atlanto-axial subluxation		Maximum subluxation below C.2
					Percentage canal narrowed	Dens displaced (millimetres)	
1	51	Female	+	Transient manual paraesthesia	27	8	1/6
2	60	Female	+	Manual paraesthesiae	40 Absent dens	8	Slight
3	54	Female	+	Episodic uselessness of arms	32	8	Nil
4	49	Male	Nil	(Impaired taste and smell)	17	5	Slight
5	47	Female	+	Awaits operation	23	6	Nil
6	72	Female	+	Urgency. Digital paraesthesiae	24	4	Nil
7	63	Female	+	Nil	52 Separated dens	13	1/6
8	59	Female	+	(Pain in hands)	13	3	1/3 at C.4-5
9	56	Female	Nil	Nil	25	6	Nil
10	55	Female	+	Nil	25	5	Nil
11	59	Female	+	Vertigo on neck extension	22	5	Nil
12	61	Female	+	Nil	25	7	Slight
13	59	Male	Nil	Nil	30	9	Slight
14	51	Male	Nil	Nil	17	4	Nil

(The percentage subluxation in Cases 2 and 7 was calculated by using the anterior and posterior borders of the body of the axis as reference points.)

(Fig. 7) but a separated attenuated dens was revealed by tomography (Fig. 8). Another patient had no visible dens either in routine views or on tomography, and it was considered that it had been almost completely destroyed (Fig. 9). Six patients had a "hook" erosion on the posterior surface of the dens (Fig. 10). A number of patients had erosions of the occipital condyles, lateral masses of the atlas and lateral articulations of the axis.

FIG. 6

An antero-posterior tomograph showing a thin dens.

FIG. 7

FIG. 8

Figure 7—A plain antero-posterior radiograph showing apparent loss of the dens. Figure 8—The tomograph reveals a distorted dens.

Sub-axial radiographic changes—Subluxations, apophysial erosions, narrowed disc spaces and end-plate erosions were seen at various levels (Fig. 11). Forward subluxation was seen most frequently at the level of C.3-4 and C.4-5. In only three cases was it of major degree. In one there was subluxation of half diameter at the C.3-4 level, and in two of one-third diameter at the C.4-5 level.

PATHOLOGY

Rheumatoid arthritis often affects the cervical spine (Sharp and Purser 1961; Bland, Davis, London, Van Buskirk and Duarte 1963). The synovial joints of the occipito-atlanto-axial region are liable to be involved (Davis and Markley 1951; Hauge 1958; Webb, Hickman and Brew 1968).

An excellent account of the morbid anatomy of atlanto-axial subluxation in rheumatoid arthritis was given by Ball and Sharp (1971). They maintained that insufficiency of the transverse ligament initiates the subluxation and that this is accompanied by excessive dynamic

loading of the other joints and ligaments in the vicinity. Various degrees of involvement of the transverse ligament are listed, varying from atrophy to complete destruction by rheumatoid granulation tissue. The transverse ligament, however, was found to be surprisingly healthy in a few cases of severe subluxation. They also found "hook" erosions of the posterior surface

FIG. 9

FIG. 10

Figure 9—An antero-posterior tomograph revealing a small residual dens. Figure 10—A lateral tomograph showing a deep posterior erosion of the dens due to granulation tissue between it and the transverse ligament.

FIG. 11

The radiographic findings in the cervical spine below C.2 in the forty-one patients with atlanto-axial separation.

of the dens due to granulation tissue between it and the transverse ligament. Synovial tissue in the apophysial and neurocentral joints may become affected by the rheumatoid inflammatory process and causes subaxial end-plate erosions, narrowing of disc spaces and subluxations of the cervical spine.

No autopsy material became available during the present study.

NECK PAIN

Of forty-one patients, thirty-one (76 per cent) had pain in the upper cervical or suboccipital areas with variable radiation to the mastoid, occipital, temporal or frontal regions. Among thirty-one patients with painful necks, ten were found to have no abnormality of the central nervous system, whereas six of ten patients without pain had abnormal signs.

NEUROLOGICAL COMPLICATIONS

The association of rheumatoid arthritis with neurological abnormalities has been noted for many years. Peripheral neuropathy was recorded as a complication towards the close of the last century, and compression of the median nerve at the wrist is well known. Spontaneous subluxation of the atlanto-axial joint was reported as a complication of a throat infection by Bell in 1830. Involvement of this joint in rheumatoid arthritis was first reported in 1955 by Margulies, Katz and Rosenberg, and has been subsequently well documented. It may first present as a neurological syndrome requiring urgent treatment, or even as a cause of sudden death. In addition, neurological disorders may arise from occlusion of the vertebral arteries in their bony canals through the cervical vertebrae, or by intrinsic vascular disease in the brain or brain stem. Rheumatoid inflammatory tissue may also act as a compressive lesion within the spinal canal. It is possible for all these neurological complications to be present in one patient.

RESULTS

Fourteen of the forty-one patients had no abnormality of the central nervous system. Among these fourteen the range of narrowing of the spinal canal varied from 13 to 52 per cent with an average of 24 per cent. Nine of this group had no symptoms referable to a neurological disorder, but four had paraesthesiae or pain in the hands or fingers, and one complained of bouts of "uselessness" of the upper limbs. One also suffered vertigo on extension of the neck (Table I).

Trigeminal nerve involvement—Eight patients showed unequivocal signs of sensory impairment over the face, one of whom had previously suffered from ophthalmic herpes. Six had depressed pain sensation bilaterally in the territory of the first division, three also showing depression of light touch; of these three, one had impairment of light touch and pain sensation in the second division also, and another had unilateral depression of the corneal reflex. The remaining two patients had right-sided facial sensory impairment, one on the forehead (due to herpes) and the other in the form of a depressed corneal reflex.

Four of the six patients with bilateral sensory impairment showed neurological changes in the spinal cord, either demonstrated as hyper-reflexia alone or combined with spino-thalamic tract involvement. In no instance was the mandibular division of the fifth cranial nerve involved (Table II). The symptoms in three patients consisted of peripheral paraesthesiae, combined with vertigo in one patient. No others in the group shared a pattern of symptoms in common; their complaints were various and included urgency of micturition, giddiness on neck extension and a history of transient loss of consciousness two years previously. One patient had no complaint, and no patients had symptoms referable to the face. The range of narrowing of the spinal canal in this group varied between 20 and 40 per cent, with an average of 28 per cent.

Miscellaneous neurological abnormalities—A group of nineteen patients showed a variety of neurological abnormalities. Thirteen had pyramidal signs, consisting usually of an asymmetrical hyper-reflexia, the least affected showing a unilateral extensor plantar response. The range of narrowing of the spinal canal in this group was 13 to 52 per cent with an average of 31 per cent. Five patients had no symptoms referable to the central nervous system and only one patient had symptoms referable to spasticity (spontaneous clonus). Three patients had

TABLE II
DETAILS OF EIGHT PATIENTS WITH ATLANTO-AXIAL SEPARATION AND TRIGEMINAL NERVE INVOLVEMENT

Case number	Age (years)	Sex	Neck pain	Relevant history	Atlanto-axial subluxation Percentage canal narrowed	Dens displaced (millimetres)	Maximum subluxation below C.2	Neurological signs
15	50	Female	+	Dizzy on neck extension	25	6	Slight	Bilateral V ¹ hypalgesia Left upper limb } Hyper-reflexia Right lower limb }
16	64	Female	+	Digital paraesthesiae	48	10	Slight	Bilateral V ¹ hypalgesia
17	61	Female	+	Vertigo; upper limb paraesthesiae	30	6	Nil	Bilateral V ¹ hypalgesia and hypaesthesia Brown-Séquard { Hypalgesia right chest Left hyper-reflexia
18*	66	Male	+	Nocturnal extensor spasm	31	8	Nil	Bilateral V ¹ hypalgesia Progressively increasing spasticity
19	64	Female	+	Transient loss of consciousness two years ago	25	5	1/6	Right corneal reflex depressed
20	52	Female	+	Paraesthesiae hands and right foot	20	5	Slight	Bilateral V ¹ hypalgesia; depressed left corneal reflex; left upper limb hyper-reflexia; depressed pain sensation bilaterally L.4, 5, S.2
21	63	Female	Nil	(Whiplash injury two years ago)	23	6	Slight	Depressed pain sensation V ¹ (right)
22*	29	Female	+	Urgency; vertigo	40	10	1/5	Bilateral V ¹ , ² hypalgesia and hypaesthesia Right hyper-reflexia Bilateral spinothalamic impairment

* Operation.

symptoms referable to the nerves of the brain stem. One complained of urgency of micturition and two of paraesthesiae. The remaining patient suffered a sudden tetraparesis.

Sensory involvement was present in eight patients, and in one only was it unaccompanied by some other abnormality of neurological function. This patient showed loss of vibration sense only in the left lower limb. The remainder had impairment of light touch and pin prick sensation variously distributed over trunk and limbs in the patchy manner characteristic of cervical cord lesions. One showed fasciculation. Four also had circumferential superficial sensory loss in one or both lower limbs; a further patient showed this pattern in the upper limbs also, accompanied by diffuse facial hypalgesia. This syndrome was almost certainly not organic in origin. Only three patients in this group complained of paraesthesiae. Two further patients showed intention tremor, associated in one with hyper-reflexia (Table III).

Fasciculation—This sign, indicative of involvement of anterior horn cells by trauma or disease, was found in four patients. It was present or could be induced in the intrinsic muscles of one or both hands in all four and was diffuse in three. In one it was accompanied by facial hemispasm, in another by sensory impairment, presumably functional. In only one of the four was there associated hyper-reflexia. Another showed in addition sensory impairment in the lower limbs. In none was there depression or absence of deep reflexes. One patient showed diffuse neurological disease; she complained of paraesthesiae of the tongue and lips, vertigo and tinnitus, urgency of micturition and loss of consciousness when the neck was moved. Her case records indicated progressive loss of deep reflexes in all limbs, the right knee and left biceps jerks being the only reflexes present at the time of our examination. She had palatal paresis, deafness on both sides and sustained nystagmus. The dens was subluxated by 23 per cent. In four other patients one or two upper limb reflexes were absent.

Patients showing progressive or urgent neurological syndromes—There were three patients in this group. The first, a man of fifty-six, showed an increasing degree of spasticity over a period of three months. The cervical spine was immobilised in a plaster minerva splint, during which time bilaterally depressed sensibility of the first division of the trigeminal nerve disappeared. However, because of intolerance of the plaster and the appearance of increasing hyper-reflexia, sub-occipital fusion was performed (Newman and Sweetnam 1969). Skull traction was applied for six weeks after operation, followed by a moulded polythene collar. Four months after the operation there was virtually no neurological improvement and nocturnal extensor spasms, after complete remission during the period of skull traction, still persisted. Radiographs showed that the narrowing of the spinal canal had lessened from 31 per cent before operation to 2 per cent after spinal fusion. Further examination seven and fourteen months after operation revealed that the nocturnal spasm had ceased and that the hyper-reflexia was slowly lessening. The graft had consolidated (Fig. 12).

The second patient, a woman aged twenty-nine, had the spinal canal narrowed by 40 per cent, bilateral sensory impairment of pain and light touch of the first and second divisions of the trigeminal nerve, bilateral spino-thalamic involvement, and hyper-reflexia of the right upper limb. Her complaints were of urgency and giddiness. Fusion of the occipito-atlanto-axial complex was performed using cancellous bone chips. Skull traction was not applied. She remained in bed for six weeks, after which a minerva collar was worn for a further six weeks. Examination four months later showed that the facial sensory loss and reflex asymmetry had recovered and that only small patches of hyperalgesia remained. The urgency of micturition persisted. Screening showed that the cancellous bone grafts were being absorbed and that the degree of subluxation was unchanged. Five months after the first operation a further fixation was undertaken. There was now no sign of the bone grafts, or indeed of any new bone formation. The laminae of the first and second cervical vertebrae were wired, and after preparation of the laminae, spinous processes and the base of the occiput, fresh cancellous bone was packed into place. At assessment nine months later she had no complaints referable to the central nervous system, and only a patchy hyperalgesia of the trunk and lower limbs could be found.

TABLE III
DETAILS OF NINETEEN PATIENTS WITH ATLANTO-AXIAL SEPARATION AND MISCELLANEOUS CENTRAL NERVOUS SYSTEM SIGNS

Case number	Age (years)	Sex	Neck pain	Relevant history	Atlanto-axial subluxation			Neurological signs
					Percentage canal narrowed	Dens displaced (millimetres)	Maximum subluxation below C.2	
23	55	Female	+	Nil	24	5	Nil	Mild right-sided hyper-reflexia
24	28	Female	+	Nil	29	7	1/5	Left supinator jerk exaggerated
25	57	Female	+	Brief attacks of diplopia	23	6	Nil	Left-sided hyper-reflexia
26	54	Male	Nil	Burning paraesthesiae face and legs	17	5	Nil	Fasciculation; right upper limb hyper-reflexia
27	50	Female	Nil	Nil	39	9	Slight	Bilateral upper limb hyper-reflexia
28	73	Male	+	Labial paraesthesiae	17	4	1/6	Fasciculation; depressed pain sensation all limbs and face
29	72	Female	+	Tinnitus	32	8	1/2 at C.3-4	Bilateral upper limb hyper-reflexia
30	58	Female	+	Paraesthesiae tongue and lips; tinnitus, vertigo, deafness, "blackouts" with neck movement; urgency	35	8	Nil	Progressive loss of deep reflexes. Nystagmus, nerve deafness, palatal paresis
31	64	Female	+	Digital paraesthesiae	22	5	Nil	Right upper limb hyper-reflexia
32	43	Male	Nil	Nil	23	6	Nil	Left upper limb hyper-reflexia; impaired pain sensation right lower limb
33	76	Female	+	Vertigo on neck extension	13	3	Slight	Depressed vibration sense left lower limb
34*	61	Male	+	Transient tetraparesis	44	11	Nil	Bilateral hypoaesthesia S.I. Right upper limb hyper-reflexia
35	54	Male	+	Spontaneous clonus	17	5	Nil	Generalised hyper-reflexia
36	56	Male	Nil	Nil	52 Thin dens	14	1/3 at C.4-5	Facial hemispasm: generalised fasciculation
37	64	Female	+	Vertigo; paraesthesiae of feet	25	5	Slight	Static and intention tremor both upper limbs
38	60	Female	+	Nil	46	12	Nil	Left extensor plantar
39	61	Female	+	Vertigo; occasional urinary incontinence	38	8	Nil	Left upper limb hyper-reflexia. Depressed vibration sense in lower limbs and depressed touch and pain sense in lower limbs and trunk
40	60	Female	Nil	Nil	33	6	Slight	Impaired vibration and touch sense in lower limbs. Fasciculation
41	58	Female	+	Urgency; occasional urinary incontinence	28	7	Slight	Right lower limb hyper-reflexia and extensor plantar; intention tremor left upper limb

* Operation case.

The canal was now narrowed by 17 per cent and the second bone graft appeared completely absorbed, again with no radiographic evidence of new bone formation (Fig. 13). The patient continues to wear a soft cervical collar.

The third and last patient had a transient bout of tetraparesis while in hospital. Radiographs showed that he had narrowing of the spinal canal by 44 per cent. Examination

FIG. 12
To show sub-occipital fusion obtained with chip
grafts only.

FIG. 13
Lateral radiographs taken in extension and in flexion, showing absorption of chip grafts and only
partial control of the atlanto-axial separation by wiring.

a few hours after the episode showed bilateral hypoaesthesia of the soles of the feet with hyper-reflexia in the right upper limb. Operative fixation was considered essential. The laminae of the first and second cervical vertebrae were wired by two loops and a prepared bone bed grafted. A further neurological episode two months later was probably due to low-pressure hydrocephalus and resulted in a transient right lower monoplegia. At examination

nine months after the operation he showed a right upper monoparesis with no sensory loss. There was now 25 per cent of canal narrowing. By July 1972, eighteen months after operation, there were no abnormal neurological signs. The atlas was securely fused to the occiput but there was slight movement between atlas and axis. One of the two loops had snapped. The amount of atlanto-axial separation was five millimetres.

After this review was completed, the patient in Case 7 presented with severe neck pain and paraesthesiae of the hands. Examination showed sensory ataxia with dorsal column involvement. Suboccipital fusion with cancellous graft and wiring was performed.

DISCUSSION

The patient (Case 30) showing diffuse neurological signs and symptoms is an excellent example of the complexity of the neurological picture that can confront the clinician. Her signs could imply the presence of peripheral neuropathy (progressive loss of reflexes), vertebral arterial insufficiency (loss of consciousness on neck movement) or intrinsic pontine vascular disease (palatal weakness, deafness, tinnitus, vertigo, facial paraesthesiae), and para-pyramidal involvement (urgency). It was difficult to ascribe more than the urgency to the subluxation alone.

Clearly the degree of subluxation bears no relationship to the signs or symptoms and reflects the variation in tolerance of neural tissue to repeated trauma (Cregan 1966).

Conduction studies were not performed in any of the patients, and it is difficult to be certain that sensory changes are not due to peripheral neuropathy when the trunk is not involved and the sensory impairment is circumferential. It has been assumed that all the changes were intramedullary when unequivocal signs of involvement of the spinal cord were also present. Two patients showing signs of peripheral neuropathy had normal blood sugar levels, no glycosuria, and normal B12 and folic acid serum levels. One patient showed an incomplete Brown-Séquard syndrome. No patient had impairment of position sense, emphasising that trauma to the cervical cord from atlanto-axial subluxation is sited anteriorly.

Hyper-reflexia was the most reliable sign of pyramidal tract involvement and was occasionally accompanied by an extensor plantar response. The significance of slight loss of power was often hard to establish in the presence of the accompanying polyarthritis. Increased reflexes were most commonly confined to one limb. This was seen in eight patients; two had hyper-reflexia in both limbs on one side, two in both upper limbs, and one in all four limbs.

All patients showing fasciculation had this sign in the intrinsic muscles of the hands, a finding reminiscent of the wasting of these muscles seen in patients with sub-foraminal tumours.

Trigeminal nerve impairment was invariably in the first division. This strongly suggests involvement of the descending tract of the nerve, which reaches as low as the second cervical segment and could thus be damaged by a displaced dens. The trigeminal nerve may also be affected by vascular disease of the pons, but other signs may be present to elucidate the site of the lesion.

In our opinion the presence of central nervous system signs alone does not necessarily warrant operative intervention (Hamblen 1967; Crellin, Maccabe and Hamilton 1970). We believe that rapid progression, especially if it occurs in spite of efficient immobilisation, and dangerous signs, such as limb paralysis too severe to be ascribed to the muscle involvement of advanced rheumatoid arthritis, are indications for surgical intervention. However, it is interesting to note in the first patient in that group the four months of nocturnal spasms after operation, and the fact that having ceased during skull traction, they recurred when he was up and about only to cease seven months later. This suggests that the fixation obtained at operation was not initially effective and that his abnormal neurological signs disappeared only after consolidation had occurred. In this patient the cervical laminae were not wired.

The second patient demonstrated clearly that bone chip grafts alone may not suffice to

maintain stability of the atlanto-axial joint in patients affected by rheumatoid arthritis. Even wiring of the laminae had not completely controlled the subluxation, though we hope that it will have done so sufficiently to prevent reinvolverment of the central nervous system. For the moment we favour wiring together with bone chip grafting.

Light collars were recommended for all patients with troublesome pain. Neck pain in patients with atlanto-axial instability was most commonly situated at the lower end of the mastoid process, radiating upwards towards the vertex and forward to the temporal and frontal regions. All patients were recommended to wear a protective collar when travelling by road. However, we could not expect a regression of the central nervous system signs following such light immobilisation; indeed in the one patient treated with a minerva plaster some worsening occurred.

We are also inclined not to recommend the test for instability described by Sharp and Purser (1961). The palm of one hand is placed on the patient's forehead and the thumb of the other on the top of the spine of the axis. The patient then flexes the neck. By pushing with the palm, backward movement of the head on the neck can be appreciated. This may well cause an exacerbation of the neurological damage.

The important neurological signs present in atlanto-axial subluxation were:—1) within the territory of the first two divisions of the trigeminal nerve; 2) in the pyramidal pathways; 3) in the superficial sensory pathways; and 4) fasciculation.

SUMMARY

1. This paper presents the neurological findings in forty-one patients with rheumatoid arthritis complicated by atlanto-axial subluxation.
2. A method of radiological measurement of the subluxation is described. The extent of neurological involvement was found not to correspond with the degree of subluxation.
3. Fusion of the atlanto-axial complex or of the occipito-atlanto-axial complex is indicated only in patients with progressive neurological signs and in those suffering from acute neurological episodes.
4. Difficulty in obtaining fusion with cancellous bone grafts alone was encountered and we recommend wiring of the laminae C.1 to C.2 in addition.

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