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ATLANTO-AXIAL DISLOCATIONS

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Abstract. The clinical problems, treatment and results are discussed in 23 cases of atlanto-axial dislocation.

Key words: Atlanto-axial dislocation.

INTEREST in the subject of atlanto-axial injuries and affections has grown over the years. A large number of papers have been published in the last 50 years. According to Washington (1959), Bell (1830) first described a case of infective aetiology while Blockley and Purser (1956) attributed the earliest description of traumatic cases to Corner (1907), Fritzche (1912) and Jefferson (1927). Reports of a series of cases have been published more recently by Blockley and Purser (1956), Lipscomb (1957), Sherk and Nicholson (1970), and Wadia (1967).

Anatomy

The most important features are:

1. The relationship between skull and the axis with the weak atlas lying between the two stronger bones. The stability of atlas on axis is dependent on transverse ligament aided by the alar ligaments.
2. The ossification of the odontoid process of axis may be incomplete with a plate of cartilage persisting at the base and acting as a weak point. The tip may also remain separate.
3. Congenital anomalies at this level are well known and may take the form of absence or hypoplasia of odontoid process, and poor formation transverse ligaments. Both these predispose to a dislocation at slight injury.
4. The position of oro- and naso-pharynx in front of vertebrae result in infective processes affecting these vertebrae.

Pathogenesis

Cases of atlanto-axial dislocations can be grouped in three groups: (1) traumatic; (2) infective; and (3) rheumatoid.

At the J.J. Group of Hospitals we have studied 40 cases in the last 20 years. Their age and sex distributions are shown in Table I. As only one case of rheumatoid variety-ankylosing spondylitis was seen, it has not been separately tabled.

Traumatic Group

There were 23 cases in this group subdivided into four groups (Table II). Burst fractures were produced by forces acting vertically with a double break in the ring of atlas bilaterally. As the fragments tended to move outwards, the chances of

TABLE I
Age and sex

Age range (yr)	Traumatic		Infective	
	M	F	M	F
0-10	1	3	1	2
11-20	9	2	3	1
21-30	6	—	1	4
31-40	2	—	2	2
Total	23		16	

TABLE II
Types of traumatic injury

Type	No. of cases
Burst fractures	5
Disl. atlas with fracture odontoid process and forward displacement	13
Disl. atlas with odontoid process in the normal position	3
Disl. atlas with congenital absence of the odontoid process	2

cord affection were less. The other three groups were classical flexion injuries with forces acting from above and behind, forwards and downwards. No case of extension injury has been recorded in our series. Where there was a congenital absence of odontoid or weakness of ligaments, the force required to produce the dislocation was much less than where odontoid was fractured. These occurred mainly in children. The following case histories illustrate these points.

Case 1: *Burst fracture.* A 16-year-old boy who fell on his head 1 month earlier. His complaints were of pain and stiffness in upper part of neck, but no C.N.S. changes.

Cases 2 and 3: *Dislocation of atlas with fracture odontoid process.* Case 2. A 14-year-old boy with history of a fall from 3 ft, 2-3 years earlier and progressive weakness in last 4 months. Examination showed spasticity, motor weakness and exaggerated jerk. Radiograms showed dislocation of atlas with fracture of odontoid and moderate mobility.

Case 3. A 22-year-old man who complained of pain and stiffness in his neck for 4 months following a fall from a swing but no neurological deficit.

Case 4: *Dislocation of atlas with odontoid intact.* An 11-year-old boy was brought to the hospital as a complete tetraplegic (C5 level) from a fall from first floor of his building. Complete C.N.S. recovery occurred under traction.

Case 5: *Dislocation of atlas associated with congenital absence of odontoid.* A 10-year-old boy came because of increasing weakness over 3 years from repeated minor trauma to the back of his head—school mates hitting him from behind. His muscle power was grade 3 with spasticity and exaggerated jerks.

Infective Group

These cases are usually of tuberculous aetiology in our country though pyogenic infection of the retropharyngeal lymphoid tissues can also occur; they may present in three ways as shown in Table III. Three case histories are presented in brief:

Case 6: A 32-year-old woman had pain in the back of her neck and stiffness from 7 months and acute retropharyngeal abscess formation for the final week, which required transpharyngeal drainage.

Case 7: A 30-year-old woman with history of tubercular adenitis of the neck 3 years earlier. Pain and stiffness of neck for 1 month now. No neurological deficit was present.

Case 8: A young child with a destructive lesion of C1-2 and a retropharyngeal abscess.

Rheumatoid Spine – Ankylosing Spondylitis

Subluxation of the atlanto-axial joint in patients with long-standing flexion deformities have been described. Conlon *et al.* (1966) stated that 25 per cent of their 337 cases of rheumatoid arthritis showed some evidence of subluxation while Crellin *et al.* (1970) treated 13 cases of affection at C1-2 level, nine of whom had neurological involvement. Our one case had no neurological deficit when first seen; patient has been lost to follow-up.

Neurological Aspects

The pattern of neurological involvement varies greatly (Table IV). It is essential to stress that along with motor signs many cases show a bizarre picture of sensory involvement unrelated to any root or segmental values. This is specially true of cases with spasticity and weakness rather than total tetraplegia. This bizarre picture of motor and sensory affection may lead an unwary clinician to a diagnosis of disseminated sclerosis, amyotrophic lateral sclerosis, etc. The vagueness of the injury or infection and a long time interval varying from immediate to 15 years in our series makes the diagnosis more difficult.

TABLE III
Types of infective cases

Type	No. of cases
Acute retropharyngeal abscess	3
Erosion of C1-2 vertebrae	3
Old history of sore throat, fever and torticollis	7

TABLE IV
C.N.S. involvement

	Traumatic group	Infective group
Tetraplegia	5	3
Weakness and spasticity	11	5
Normal	7	8

Blockley's series: immediate, 7; late, 10

TABLE V
Treatment

	Traumatic group	Infective group	Total	Mortality
Fusion only	13	7	20	—
Decompression and fusion	7	2	9	4
Conservative	2	5	7	—
Not treated	1	2	3	1

Treatment (Table V)

Our general approach for these cases is to carry out a posterior occipeto-cervical fusion with special exceptions. Where the first set of radiograms show the forward slip to be correctable on extension, an early fusion is done. Otherwise skull calipers are fixed and traction applied with gradual extension of the neck until correction is obtained in about 3 weeks. Fusion is then undertaken. Decompression of the cord is reserved now only for cases with persistent deficit associated with uncorrectable subluxation. The danger of decompression has been realised after three deaths from respiratory failure which occurred on the operating table just as the posterior arch of the atlas was being nibbled off; no recovery occurred in spite of the patient being on a respirator for several weeks.

For cases with infective lesions in the acute phase, the treatment is initially conservative, except where a large retropharyngeal abscess required drainage by the transpharyngeal route; after the acute phase was over, or if the patient has come late, fusion is the method of choice.

Conservative treatment with collars is reserved for children under 8 years of age or where the degree of forward slip is less than 3 mm in full flexion and there is no neurological involvement. It must be remembered that wearing the collar for months, or years, in a hot tropical climate is a near impossibility for a labourer or a farmer. Most discard them in a few weeks. Even in Blockley and Purser's (1956) series of 51 cases, ten showed late neurological involvement in spite of wearing collars.

Mortality

The causes of death are shown in Table VI; the cases of acute respiratory failure have been already referred to. Post-mortem examination showed evidence

TABLE VI
Mortality

	No. of cases
Menengitis	1
Respiratory failure	
On table	3
During transport	1

of recent as well as old haemorrhages in the medulla and upper cervical cord along with degenerative changes. Similar experiences have been described by Dastur *et al.* (1965).

Results

These are shown in Tables VII and VIII and are related to the duration of affection in regard to their neurological recovery. The long-standing cases often showed a residual deficit.

TABLE VII
Follow-up

	No. of cases
0 to 6 months	13
6 months to 1 year	5
1 year to 2 years	8
2 years to 3 years	4
3 years onwards	2

2 cases refused treatment and never reported for follow-up. 5 cases died.

TABLE VIII
Results

	No. of cases
Complete neurological recovery with good fusion	24
Incomplete neurological recovery	4
Died	5
Unknown (including 3 who did not take treatment)	6
Total	39

SUMMARY

Of the 40 cases of atlanto-axial dislocation 23 were traumatic aetiology, 16 of infective aetiology and one was secondary to rheumatoid arthritis. Their clinical problems, treatment and results are discussed.

RÉSUMÉ

Parmi les 40 cas de dislocation Atlanto-axiale, 23 étaient d'étiologie traumatique, 16 d'étiologie infectieuse et 1 provenait d'une arthrite rhumatoïde. On discute leurs problèmes cliniques, ainsi que leur traitement et les résultats obtenus.

ZUSAMMENFASSUNG

23 von 40 Fällen von atlanto-axial dislokation zeigten traumatische Aetiologie, 16 waren infektiös und einer war sekundär von rheumatoid arthritis. Ihre klinischen Probleme, Behandlung und Resultate werden diskutiert.

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General Discussion

DR HARDY (*Chairman*). Thank you Professor again for a very comprehensive survey of an injury which we don't see very much of in our Units. I think fractures and displacements of the atlas and axis and occipital junctions are not common in our Units although, of course, we do see them.

DR GROGONO (*Canada*). I've been interested in this condition for some 20 years. I would like to ask you about the indications for fusion. I think there are different aspects to it, particularly in children. Mr Butler of Addenbrooks Hospital, Cambridge, some years ago collaborated in a paper and he showed that he could get satisfactory fusion between the atlas and axis for displacements in children. That was one question. What were your indications for fusion and whether you had to do this sort of radical operation? The second one was in the congenital absence of the odontoid is there any special problem? I know Dr Welply in Winnipeg and a case like this where he tried anterior and posterior fusion. Finally, have you studied patients brought in dead on admission? There is quite a high incidence of atlo-axial displacement if you take X-rays of people who've died in violent accidents. Is it a common complication of a violent car accident?

DR MASALAWALA. The fusion, as I've briefly indicated, we like to do in every patient. Where we can demonstrate radiologically appreciable forward dislocation of the atlas, the dislocation can be reduced either by extending the neck or by skull traction.

After about 3 weeks we like to do a fusion alone. In those cases where the dislocation cannot be reduced, in order to decompress the cord we like to remove the posterior arch of the atlas and half-an-inch of the foramen magnum and then fuse them. We feel that in our patients, especially those who come from the poorer classes of our country, it is not possible for them to wear collars nor is it possible for them to remain under continuous medical observation, and something which can give a permanent cure, or as near permanent as human resources can give, it is better rather than to go on with a long drawn out treatment. Hence we try to do fusion in all these cases. I fully agree that we can do a fusion in children, what I really meant was that if the child's dislocation was not very much we might try to treat them conservatively. Children will wear collars perhaps because they need not be out in the heat so much as a working man, but I have shown a small boy who had a marked immobility of the atlas on the axis and we did fuse successfully. As far as seeing cases which come in dead, they probably go straight to the post-mortem room and I'm afraid I've no answer to that. Anterior fusion is being done in our hospital by our neurosurgical colleagues, but personally I feel that it is a more difficult operation and one where you are working at a great depth. Unless you have got very good lighted retractors etc. it is not as easy as one would like to imagine and I found the posterior fusion perfectly satisfactory. Therefore I would not ban it, but I do know that Andre and MacNab described it long back, it has been described in cases where a posterior sort of laminectomy or removal of the posterior arches has been done to further stabilise the upper end of the cervical spine. I have fused C2 to C4 by the anterior root but I have not done occipital cervical anterior fusion.