DIAGNOSIS AND MANAGEMENT OF TRAUMATIC ATLANTO-OCCIPITAL DISLOCATION INJURIES

RECOMMENDATIONS

Diagnostic:

Standards: There is insufficient evidence to support diagnostic standards.

Guidelines: There is insufficient evidence to support diagnostic guidelines.

Options:

- A lateral cervical radiograph is recommended for the diagnosis of AOD. If a radiological method for measurement is used, the basion-axial interval-basion dental interval (BAI-BDI) method is recommended.
- The presence of upper cervical prevertebral soft tissue swelling on an otherwise non-diagnostic plain radiograph should prompt additional imaging.
- If there is clinical suspicion of AOD, and plain radiographs are non-diagnostic, CT or MR imaging is recommended, particularly for the diagnosis of non-Type II dislocations.

Treatment:

Standards: There is insufficient evidence to support treatment standards.

Guidelines: There is insufficient evidence to support treatment guidelines.

Options: Treatment with internal fixation and arthrodesis using one of a variety of methods is recommended. Traction may be used in the management of patients with AOD, but is associated with a ten percent risk of neurological deterioration.

RATIONALE

Although traumatic atlanto-occipital dislocation (AOD) was perceived to be an uncommon injury resulting in frequent death, improvements in emergency management of the

© 2001 The Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons. All rights reserved.

patient in the field, rapid transport, and better recognition have resulted in more survivors of AOD in the past two decades. Infrequent observation of patients with AOD and missed diagnoses may impair outcomes of patients with this unusual injury.(44) An assimilation of the reported experiences of clinicians evaluating and managing AOD may facilitate development of diagnostic and treatment options for this traumatic disorder. Specific questions that were evaluated include the sensitivity of plain radiographs, CT, and MR imaging in the diagnosis of AOD, as well as the safety and efficacy of various treatment modalities for AOD, including no treatment, traction, external immobilization, and internal fixation with fusion.

SEARCH CRITERIA

A National Library of Medicine computerized literature search of publications from 1966 to 2001 was performed using the following headings: "atlanto-occipital joint" and "dislocation". An exploded search of these headings led to 690 and 86,205 citations, respectively. A subset of 233 citations contained both headings. The references of the identified articles were reviewed to identify additional case reports. Since fewer than 100 cases of survivors of AOD were identified, even single case reports were considered, provided that basic inclusion criteria were met. The articles were reviewed using the following criteria for inclusion in diagnosis: human survivors, type of traumatic atlanto-occipital dislocation, and plain radiographic findings. The articles were also reviewed using the following criteria for inclusion in treatment: human survivors, type of traumatic AOD, management, and outcome. The observations from the reports were combined because the usual methods for analysis were precluded by the infrequent observation of this injury. The type of dislocation was classified according to Traynelis et al (51) into Type I (anterior), Type II (longitudinal), and Type III (posterior) dislocations. Lateral, rotational and multi-directional dislocations that could not be classified into one of these three

© 2001 The Spine Section of the AANS and CNS.

types were considered separately and are notated as "Other Type". The duration of follow-up ranged from several weeks to four years. Of the articles meeting the diagnostic selection criteria reported, 48 articles with 79 patients provided data on 29 Type I, 32 Type II, four Type III, and 14 other types of AOD. Two of these articles (10,44) included one patient each from two previously published individual case reports.(41,42) Of the articles meeting the treatment selection criteria, 43 articles with 62 patients provided data on 24 Type I, 23 Type II, three Type III, and 12 other types of AOD. Two of these articles (10,44) included one patient each from two previously published individual case reports.(41,42) All articles contained Class III medical evidence consisting of either single case studies or small case series with no report containing more than six patients. The information provided by these reports was compiled and scrutinized and make up the basis for this guideline. Summaries of these reports are provided in Evidentiary Table format.

SCIENTIFIC FOUNDATION

Diagnosis:

A variety of radiographic measurements has been proposed for the diagnosis of AOD on a lateral cervical radiograph (Figure 1). A displacement of more than ten mm between the basion and dens is considered abnormal by Wholey et al.(53) A ratio of the basion-posterior atlas arch distance divided by the opisthion-anterior atlas arch distance greater than one is considered abnormal by Powers et al.(43) A distance of more than 13 mm between the posterior mandible and anterior atlas or 20 mm between the posterior mandible and dens is considered abnormal by Dublin et al.(12) Failure of a line from the basion to the axis spinolaminar junction to intersect C2, or a line from the opisthion to the posterior inferior corner of the body of the axis

© 2001 The Spine Section of the AANS and CNS.

to intersect C1, are considered abnormal by Lee et al.(32) Finally, a displacement of more than 12mm or less than minus four mm between the basion and posterior C2 line, or a displacement of more than 12 mm from the basion to dens (two mm more than the Wholey recommendation) is considered abnormal by Harris et al.(24,25) A comparative study by Lee found a 50% sensitivity of the Wholey method, 33% sensitivity of the Power's ratio, and a 25% sensitivity of the Dublin method. The authors applied their X-line method with a 75% sensitivity.(32) Although neither the Power's ratio nor X-line method could be applied in nearly half their patients, a comparative study by Harris et al found a 60% sensitivity of the Power's ratio, a 20% sensitivity of the Lee method, and 100% sensitivity of the BAI-BDI method among those in whom the required landmarks could be identified.(25) Przybylski et al (44) reported failure to diagnose AOD in two of five patients with the Power's ratio, one of five patients with the X-line method, and in two of five with the BAI-BDI method. No radiographic method reviewed has complete sensitivity. The BAI-BDI method proposed by Harris et al (which incorporates the basion-dens distance described by Wholey) is at present the most reliable means to diagnose AOD on a lateral cervical spine radiograph.

Many of the case reports and case series in the literature do not describe the method(s) used for diagnosis of AOD. Since the most sensitive method was proposed by Harris et al (25) in 1994, this method was probably not used for many of the evaluations. Although retrospectively a diagnosis was possible on the first lateral radiograph in 60 of 79 patients, (sensitivity = 0.76) the diagnosis was actually made in only 45 of the 79 patients (sensitivity = 0.57) on the first lateral radiograph. Of the fifteen in whom the diagnosis could have been made on the first lateral radiograph, three were not stratified by type, whereas eleven of the remaining twelve were not Type II dislocations. A second lateral radiograph (nine cases), tomography (one

© 2001 The Spine Section of the AANS and CNS.

case), fluoroscopy (two cases), CT (two cases), and MRI (five cases) were required for diagnosis in 19 of 79 patients. The sensitivities stratified by type of dislocation are: Type I, 0.83 (24 of 29); Type II, 0.72 (23 of 32); Type III, 0.75 (three of four), Other Type, 0.71 (ten of 14). Since these data were obtained from case reports and small case series, comparison with the accuracy of plain radiographs in patients without AOD could not be performed. As a result, specificity, predictive values, and likelihood ratios can not be discerned from the available literature.

Of the fifteen patients in whom the diagnosis was missed on the initial plain radiographs, the initial neurological condition of three patients was unknown.(1) Of the remaining twelve patients, four were neurologically normal (one Type I, one Type III, two other type).(13,29) Two of those four patients originally reported as normal developed a monoparesis (one Type I, one other type).(7,49) Neither recovered completely. Eight of the remaining twelve patients had neurologic abnormalities from the outset, five of whom worsened. Four of the five transiently worsened, including one Type I injury patient with quadriparesis and Cranial Nerve IX, X, and XII palsies (9) who was only spastic at last follow-up. One patient with a Type I injury developed a hemiparesis that recovered.(27) One Type I injury patient developed quadriparesis who was hemiparetic at follow-up.(46) One lateral dislocation patient with paraparesis and toricollis recovered at last follow-up.(52) One patient (Type I) with a monoparesis initially experienced permanent worsening and was quadriplegic at follow-up.(54)

Although plain radiographs do not reliably identify AOD, the index of suspicion may be increased with the identification of prevertebral soft tissue swelling (STS). Although plain radiographs were obtained in all cases considered, the presence or absence of soft tissue swelling was described in only half. The sensitivity of soft tissue swelling is 0.90 (37 of 41 cases). Acute craniocervical CT imaging was performed in 40 of 79 patients with AOD. However, for 15 of

© 2001 The Spine Section of the AANS and CNS.

40 patients the authors did not report whether AOD was diagnosed by CT. The diagnosis of AOD was made by CT in 21 of 25 patients (sensitivity=0.84). Although no other CT findings were reported in 11 of 40 patients, 24 of the remaining 29 patients with AOD studied with CT had hemorrhages (19 subarachnoid hemorrhage, one subdural hemorrhage, four contusions). Five patients had no CT evidence of associated hemorrhage. Nine of fifteen patients in whom the diagnosis of AOD was missed on the first plain radiograph had subsequent acute CT imaging; eight had subarachnoid or other associated hemorrhage.(1,9,8,44) Craniocervical MR imaging was performed in 18 of 79 patients with AOD. The MR findings were not reported for four of the 18 patients studied. The diagnosis of AOD could be made in 12 of 14 cases studied with MRI (sensitivity=0.86).

In summary, physicians often miss the diagnosis of AOD on plain radiographs, (sensitivity=0.57) particularly in the circumstance of non-longitudinal dislocations (non-Type II). Although improved interpretation may increase sensitivity of plain x-rays to 0.76, additional imaging of the craniovertebral junction with CT or MRI is recommended in patients suspected of having AOD, given their superior sensitivity over plain radiographs. Other methods such as fluoroscopy, tomography, and myelography have also been used to confirm the diagnosis of AOD. Neurological abnormalities including lower cranial nerve paresis (particularly cranial nerves 6, 10, and 12), monoparesis, hemiparesis, quadriparesis, respiratory dysfunction including apnea, and complete high cervical cord motor deficits in the setting of normal plain spinal radiographs should prompt additional imaging with CT or MRI. The presence of prevertebral STS on plain radiographs, and subarachnoid hemorrhage on CT at the craniovertebral junction should prompt consideration of the diagnosis of AOD.

© 2001 The Spine Section of the AANS and CNS.

Treatment:

Ten patients in the literature did not receive initial treatment for AOD, nine of whom were not correctly diagnosed until neurological worsening occurred.(7,8,10,20,46,48,49,52,54) Five of nine had Type I injuries and four of nine had other injury types. Four of nine had persistent deficits at last follow up which were worse in comparison with their exams on presentation.(7,10,49,54) Two of these patients were normal initially. At last follow-up, one had a CN X deficit with spasticity (Type I) (49) and one a monoparesis.(7) The other two patients had mild initial deficits. One patient with an initial CN VI palsy had a hemiparesis at last follow-up, (10) whereas another with an initial monoparesis was quadriplegic at follow-up (54). Five patients who worsened initially without treatment eventually improved from their initial neurological condition. Finally, one quadriplegic patient with Type II AOD (56) who was not treated improved to quadriparesis at last follow-up. In summary, failure to treat AOD resulted in worsening of all patients with incomplete injuries. Nearly half of these patients failed to improve to their presentation examination baselines.

Of twenty-one patients with AOD initially treated with traction, two worsened transiently and developed worsening quadriparesis and CN VI deficits. Both had resolution of their CN VI deficits but not of their quadriparesis. One patient had a Type II injury (40) and one patient had a rotational dislocation.(10) Four patients were initially normal and remained normal at follow up (2,4,16,33). The remaining fifteen patients had improved neurological function compared to their initial findings at last follow-up. Ten had Type I injuries, five had Type II injuries, two had Type III injuries, and two had other dislocations. In total, one of six patients with Type II injuries and one of three patients with other translational injuries had transient worsening with the use of craniocervical traction. In summary, traction for AOD has been reported to cause

© 2001 The Spine Section of the AANS and CNS.

occasional neurological worsening. In both circumstances, the worsening did not persist after discontinuation of traction. Because the frequency of neurological worsening with traction for AOD is approximately 10%, ten times higher than that for subaxial injuries, the use of traction should be considered with caution in patients with AOD.

Of nineteen patients initially treated with external immobilization excluding traction, eight were immobilized in anticipation of internal fixation and fusion and none worsened during the pre-surgical interval (one Type I, four Type II, three other type).(10,11,31,33,38,44) Of the remaining eleven patients treated with external immobilization alone excluding traction, four worsened transiently (three Type I, one Type II). (9,11,13,27) All subsequently underwent craniocervical fixation and fusion. Two of these patients were normal at follow up (one initially normal, one initially hemiparetic) and two were spastic (one initially quadriparetic and one hemiparetic). Of the remaining seven patients managed with external immobilization alone who did not worsen while in external immobilization, two patients managed in collars and one patient treated in a halo were unstable after six to 22 weeks of immobilization (one Type I, two Type II). Two were quadriplegic and one was normal. All three underwent internal fixation and fusion without change in their initial neurological condition at last follow-up. Only four patients with AOD were successfully treated with external immobilization alone (one Type I, two Type II, one other dislocation). Of the twenty-one patients initially treated with traction, six were subsequently managed with external immobilization and none developed neurological worsening. Two of the six (both Type I) remained unstable after three to five months of bracing and were subsequently treated with craniocervical fixation and fusion. Five of those six patients had improvement in their neurological condition at follow-up. The sixth patient remained normal.

© 2001 The Spine Section of the AANS and CNS.

In summary, five of thirteen patients with AOD who did not worsen neurologically while treated with external immobilization (with or without traction) failed to achieve bony union with stability without internal fixation and fusion. In addition, six patients transiently worsened with external immobilization (with or without initial traction). Factors affecting fusion or persistent non-union such as degree and type of displacement, patient age, and association with occipital condyle fractures could not be identified. Since eleven of forty patients (28%) managed with external immobilization either deteriorated neurologically or failed to achieve craniocervical stability without surgical internal fixation and fusion, treatment of AOD with external immobilization alone should be considered with caution.

Finally, nineteen patients in the literature were treated with planned early craniocervical fusion with internal fixation. Only one patient worsened neurologically following surgery. This patient with a Type II injury was normally initially and developed a CN X deficit which persisted at follow-up.(44) All but three of the remaining eighteen improved neurologically at follow-up. Four had Type I, ten had type II, and four had other types of dislocation. None of the patients treated with craniocervical fusion and internal fixation had late instability requiring reoperation or further treatment.

SUMMARY

AOD is an uncommon traumatic injury which is difficult to diagnose and is frequently missed on initial lateral cervical radiographs. Patients who survive often have neurological impairment including lower cranial neuropathies, unilateral or bilateral weakness, or quadriplegia. Yet, nearly 20% of patients with acute traumatic AOD will have a normal

© 2001 The Spine Section of the AANS and CNS.

neurological examination on presentation. The lack of localizing features may impede diagnosis in the patient with a normal cervical radiograph. A high index of suspicion must be maintained in order to diagnose AOD. Prevertebral soft tissue swelling on a lateral cervical radiograph or craniocervical subarachnoid hemorrhage on axial CT have been associated with AOD and may prompt consideration of the diagnosis. Additional imaging including CT and MR may be required to confirm the diagnosis of AOD if plain radiographs are inadequate. All patients with AOD should be treated. Without treatment, nearly all patients developed neurological worsening, some of whom did not recover. Although treatment with traction and external immobilization has been used successfully in some patients, transient or permanent neurological worsening and late instability have been reported more often in association with these treatments compared to surgical treatment. Consequently, craniocervical fusion with internal fixation is recommended for the treatment of patients with acute traumatic AOD.

KEY ISSUES FOR FUTURE INVESTIGATION

Although the use of external immobilization for AOD was often associated with late instability, several patients achieved stability without operative management. CT imaging with three dimensional reconstruction for more precise measurement of the magnitude of displacement and MR imaging for differentiation of partial and complete ligament tears from stretch injuries may be useful in identifying a subgroup of patients in whom stability might be achieved with external immobilization alone. Because AOD remains relatively infrequent, cooperative prospective collection of plain radiographic, CT and MR imaging data in patients with AOD is recommended to determine if a subgroup of patients with AOD can be treated with external immobilization alone with fewer occurrences of late instability.

© 2001 The Spine Section of the AANS and CNS.

EVIDENTIARY TABLE I - Imaging Diagnosis of AOD

AUTHOR	AOD TYPE	DIAGNOSIS MADE BY	XRAY FINDINGS	CT FINDINGS	MR FINDINGS
Grabb 1999	I II II	Plain Xray Plain Xray MRI	STS, Power+ STS, Power- STS, Power-	Unreported None performed None performed	Part tear tectorial Tear Post. AOL Part tear tectorial
Naso 1997	I/II	Plain Xray	No mention STS	Unreported	Delayed study
Sponseller 1997	I II	Plain Xray (missed) Plain Xray	No mention STS No mention STS	None performed Unreported	None performed Brainstem contusion
Przybylski 1996 Pang 1980	I II II I/Lateral I/Lateral	MRI Plain Xray (missed) 2nd plain Xray Plain Xray (missed) Plain Xray (missed)	Power/BDI/Xline- Power/BDI/Xline+ Power/BDI-,Xline+ Power/BDI/Xline+ Power/BDI/Xline+	SAH, - Dx SAH, +Dx SAH, +Dx Normal, Head only SAH, +Dx	BS contusion, +Dx BS contusion, +Dx None performed None performed None performed
Yamaguchi 1996	I	Plain Xray	No mention STS	SAH,+ tomo	BS Contusion,+Dx
Guigui 1995	I	Plain Xray	STS	+Dx	None performed
Ahuja 1994	I II II II I/II I/II	Fluoroscopy 5 Plain Xray (3 missed)	STS,Power- STS,Power+ STS,Power+ STS,Power+ STS,Power+ STS,Power+	SAH, unknown None performed SAH, +Dx SAH, unknown None performed SAH, +Dx	None performed None performed None performed None performed None performed None performed
Donahue 1994	I II II II	Plain Xray Plain Xray Plain Xray Plain Xray	STS STS, 5mm distract STS 6mm distract	None performed None performed None performed Intracerebral bleed	None performed None performed None performed None performed
Palmer 1994	II	CT	No mention STS	Unreported	CordContusion,+Dx
Dickman 1993 Papadououlos 1991	II Rotatory Rotatory II/Rotatory	Plain Xray CT MRI 2nd Plain Xray	15mm distraction STS STS STS	None performed + Dx No blood, - Dx +Dx	None performed None performed Epidural, +Dx Epidural, +Dx
Harmanali 1993	II	Plain Xray	No mention STS	None performed	- Dx
Hosono 1993	I	Plain Xray (missed)	STS	Edema, head only	Delayed study
Matava 1993	II II II	Plain Xray Plain Xray Plain Xray	STS No mention STS No mention STS	Delayed study None, +DX SAH, +DX	None performed None performed BS Contusion
Nischal 1993	II II	Plain Xray Plain Xray	STS STS	BS contusion,- Dx - Dx	None performed None performed
Bundshuh 1992	I I	Plain Xray Plain Xray	STS STS, Power/Xline-	SAH, +Dx SAH	SAH, + Dx - Dx
Farley 1992	I	Plain Xray	STS, Power +	None performed	Cord contusion
Belzberg 1991	II	2nd Plain Xray	STS	SAH, +Dx	None performed
Hladky 1991	II II	MRI MRI	No mention STS No STS	Contusion,head only Normal, Head only	+ Dx + Dx
Lee 1991	II I/Rotatory	Plain Xray Plain Xray	STS STS	SAH, +Dx + Dx	None performed None performed

© 2001 The Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons. All rights reserved.

AUTHOR	AOD TYPE	DIAGNOSIS MADE BY	XRAY FINDINGS	CT FINDINGS	MR FINDINGS
Maves 1991	II II III	Plain Xray Plain Xray Plain Xray	No mention STS No mention STS No mention STS	None performed None performed None performed	None performed None performed None performed
Montane 1991	I II II	Plain Xray 2nd Plain Xray 2nd Plain Xray	STS STS No STS	None performed None performed None performed	None performed None performed None performed
DiBenedetto 1990	I	Plain Xray (missed)	STS	ICH, +DX	None performed
Jones 1990	I	Plain Xray	No mention STS	+DX	Premedullary edema
Colnet 1989	Lat/rotatory	Tomography	Late study	SAH, +DX	Delayed study
Jevitch 1989	Lateral	Plain Xray (missed)	No mention STS	Delayed study	None performed
Hummel 1988	I	2nd Plain Xray	No mention STS	Subdural, Head only	None performed
Zampella 1988	II	Plain Xray	No mention STS	SAH, Head only	Delayed study
Georgopoulous 1987	I	Cineradiography	No mention STS	Delayed study	None performed
Bools 1986	I III	Plain Xray 2nd Plain Xray	STS No mention STS	SAH, +DX None performed	None performed None performed
Collato 1986	I/lateral	Plain Xray (missed)	No STS	SAH, Head only	Delayed study
Putnam 1986	I	Plain Xray	STS, Powers +	SAH, +DX	None performed
Ramsay 1986	I	Plain Xray (missed)	No mention STS	None performed	None performed
Roy-Camille 1986	I I	Late Plain Xray Plain Xray	No mention STS STS	Delayed study None performed	None performed None performed
Zigler 1986	I	Plain Xray	No mention STS	None performed	None performed
Watridge 1985	Lateral	Plain Xray (missed)	No STS	Delayed study	None performed
Banna 1983	Rotatory	Plain Xray	No mention STS	+ Dx	None performed
Kaufman 1982	II II	Plain Xray Plain Xray	STS STS	None performed None performed	None performed None performed
Woodring 1981	I I	Plain Xray Plain Xray (missed)	No mention STS STS	None performed None performed	None performed None performed
Powers 1979	I II	Plain Xray 2nd Plain Xray	Late study No mention STS	None performed None performed	None performed None performed
Rockswold 1979	II	Plain Xray	No mention STS	None performed	None performed
Eismont 1978	III	Plain Xray (missed)	No mention STS	None performed	None performed
Fruin 1977	I	Plain Xray	No mention STS	None performed	None performed
Page 1973	I	Plain Xray	STS	None performed	None performed
Evarts 1970	I	Plain Xray	No mention STS	None performed	None performed
Gabrielsen 1966	I	2nd Plain Xray	STS	None performed	None performed
Farthing 1948	III	Plain Xray	No mention STS	None performed	None performed

1 patient was eliminated because the plain radiograph interpretation was not reported. Ferrara (1)

2 articles (11 patients) were eliminated because the type of dislocation was not reported. Cohen (1), Georgopolous (2/3), Hladky (1/3), Naso (1/2), Sun (6/6)

1 article (5 patients) was eliminated because individual patient data was not reported. Bulas (5/5)

© 2001 The Spine Section of the AANS and CNS.

EVIDENTIARY TABLE II - Treatment of AOD

AUTHOR	TYPE	INITIAL EXAM	TREATMENT	OUTCOME
Naso 1997	Mixed I/II	Quadriplegia	Supportive	Death five wks
Sponseller 1997	I	Normal	None (neuro worse), Traction, Fusion+Brace	Spastic, CN 10
	II	Normal	Brace failed (6wk), Fusion	Normal
Przybylski 1996	I	Quadriplegia	Collar + Fusion	Quadriplegia
Pang 1980	II	Quadriplegia	Halo failed (22 wk), Fusion	Quadriplegia
	II	Normal	Fusion + Collar	CN 10
	Mixed I/Lateral	Hemiplegia	Collar + Fusion	Monoparesis
	Mixed I/Lateral	Quadriparesis, CN6/7/12	Fusion + Collar	CN 12
Yamaguchi 1996	I	Quadriplegia, CN 10,11,12	Brace failed (10wk), Fusion	Quadriplegia, CN 10,11,12
Geigui 1995	I	Normal	Fusion+Brace	Normal
Donahue 1994	I	Hemiparesis	Halo distracted (temp neuro worse), Fusion	Hyperreflexic
	II	CN6	Halo + Fusion	Normal
	II	Quadriplegia, CN7/10	Collar/Traction + Fusion	Quadriparesis, CN7/10
	II	Quadriparesis, CN3/7	Fusion	Quadriparesis
Palmer 1994	II	Quadriparesis,CN6	Traction (neuro worse), Brace+Fusion	Quadriparesis
Dickman 1993	II	Quadriplegia, CN9/10	Brace	Unchanged (sepsis
Papadououlos 1991		Quadriparesis,CN6		death at three mo)
	Rotatory		Traction (neuro	Quadriparesis
		CN6	worse),Fusion+Halo	
	Rotatory		None (neuro worse),	Hemiparesis
		Hemiparesis,CN3/6	Fusion+Halo	
	Mixed II/Rotatory		Halo+Fusion	Normal
Harmanali 1993	II	Hemiparesis,CN12	Fusion+Brace	Normal
Hosono 1993	I	Hemiparesis	Brace(neuro worse), Fusion+Brace	Normal
Matava 1993	II	Hemiplegia,CN6/12	Fusion + Brace	Spastic, CN 6
	II	Hemiparesis,CN6	Fusion + Brace	Normal
	II	CN6/9/10	Fusion + Brace	Spastic
Nischal 1993	II	Quadriparesis, CN3,6,9,10	Brace+Fusion	Hemiparesis, CN3,6,9,10
	II	Quadriplegia, CN9,10	Brace+Fusion	Hemiparesis
Bundshuh 1992	I	Quadriparesis CN6,9,10,12	Traction+Fusion	CN6,12
Farley 1992	I	Quadriplegia, CN10	Traction+Brace	Quadriplegia
Belzberg 1991	II	Quadriparesis, CN6,9,10	Traction+Brace+ Fusion	Monoparesis, CN6
Lee 1991	II	Normal	Traction+Fusion	Normal
	Mixed I/Rot	CN6	Brace+Fusion	CN6
Montane 1991	I	Hemiparesis	Fusion+Brace	Spastic
	II	Quadriparesis	Traction, Fusion+Brace	Normal
	II	Quadriplegia	Fusion+Brace	Quadriplegia
DiBenedetto 1990	I	Quadriparesis, CN9,10,12	Collar (neuro worse, 6wk), Fusion+Brace	Spastic

© 2001 The Section on Disorders of the Spine and Peripheral Nerves of the American Association of Neurological Surgeons and the Congress of Neurological Surgeons. All rights reserved.

AUTHOR	TYPE	INITIAL EXAM	TREATMENT	OUTCOME
Colnet 1989	Mixed lat/rotatory	Hemplegia, CN 6,9,10	None (neuro worse), Traction+Shunt+ Decompression	Hemiparesis
Jevitch 1989	Lateral	Normal	Traction+Brace	Normal
Hummel 1988	I	Hemiparesis	Fusion+Brace	Normal
Zampella 1988	II	Quadriplegia, CN5-12	None	Quadriplegia, CN6
Georgopoulous 1987	I	Normal	None (neuro worse), Fusion+Brace	Normal
Bools 1986	III	Normal	Traction, Fusion+Brace	Normal
Collato 1986	Mixed I/lateral	Normal	None (neuro worse), Fusion+Brace	Monoparesis
Putnam 1986	I	Quadriplegia, CN 6	Brace	Death (sepsis eight mo)
Ramsay 1986	I	Quadriparesis	None (neuro worse), Traction+Brace	Hemiplegia
Roy-Camille 1986	I I	CN6,11 Quadriplegia, CN6,9-12	None, Brace failed (3 mo), Traction+Fusion Traction+Fusion	CN6 Quadriplegia
Zigler 1986	I	Quadriplegia, CN11	Traction+Brace +Fusion	Quadriplegia
Watridge 1985	Lateral	Paraparesis	None (neuro worse), Traction+Fusion+ Decompress+Brace	Normal
Banna 1983	Rotatory	Normal	Traction (2 wk)	Normal
Kaufman 1982	II II	Quadriplegia Monoparesis	Brace+Fusion Brace	Quadriparesis, CN9,10 Normal
Woodring 1981	I I	Hemiparesis, CN6 Monoparesis	Traction None (neuro worse), Traction+Fusion	CN6 Quadriplegia
Powers 1979	I II	Hemiparesis, CN6 Hemiparesis, CN7	Traction+Brace Traction+Brace	Hemiparesis Normal
Rockswold 1979	II	Hemiparesis, CN6	Traction, Brace+Fusion	Ambulates
Eismont 1978	III	Normal	Collar(neuro worse) Fusion+Brace	Normal
Fruin 1977	I	Hemiparesis CN6,9-12	Traction+Fusion	CN6,11
Page 1973	I	Quadriplegia CN10,12	Traction, Brace failed (5mo),Fusion	Quadriparesis,CN10
Evarts 1970	I	Hemiparesis CN 6,9,10,12	Traction, Brace+Fusion	CN6
Gabrielsen 1966	I	Hyperreflexic,CN6	Traction, Brace failed (3mo),Fusion	Numb scalp
Farthing 1948	III	Normal	Traction+Brace	Normal

3 articles (15 patients) were eliminated because the type of dislocation was not reported. Cohen (1), Georgopolous (2/3), Bulas (5/5), Naso (1/2), Sun (6/6)

2 articles (8 patients) were eliminated because the initial exam was not reported. Grabb (3), Ahuja (5)

2 articles (6 patients) were eliminated because the treatment was not reported.
Maves (3), Hladky (3)

1 article (2 patients) was eliminated because the outcome was not reported.
Jones (1), Bools (1/2)

© 2001 The Spine Section of the AANS and CNS.

REFERENCES

1. Ahuja A, Glasauer FE, Alker GJ, Klein DM. Radiology in survivors of traumatic atlanto-occipital dislocation. Surg Neurol 41:112-118, 1994.
2. Banna M, Stevenson GW, Hamilton, Tumiel A. Unilateral atlanto-occipital dislocation complicating an anomaly of the atlas. J Bone Joint Surg 65A:685-687, 1983.
3. Belzberg AJ, Tranmer BI. Stabilization of traumatic atlanto-occipital dislocation. J Neurosurg 75:478-482, 1991.
4. Bools JC, Rose BS. Traumatic atlantooccipital dislocation: two cases with survival. Am J Neuroradiol 7:901-904, 1986.
5. Bulas DI, Fitz CR, Johnson DL. Traumatic atlanto-occipital dislocation in children. Radiology 188:155-158, 1993.
6. Bundschuh CV, Alley JB, Ross M, Porter IS, Gudeman SK. Magnetic resonance imaging of suspected atlanto-occipital dislocation. Two case reports. Spine 17:245-248, 1992.
7. Collato PM, DeMuth WW, Schwentker P, Boal D. Traumatic atlanto-occipital dislocation. J Bone Joint Surg 68A:1106-1109, 1986.
8. Colnet G, Chabannes J, Commun C, Rigal MC, Alassaf M. Luxation occipito-atloidienne et syringomyelie deux complications rares de la traumatologie cervicale. Incidences diagnostiques et therapeutiques. A propos d'un cas. Neurochirurgie 35:58-63, 1989.
9. Dibenedetto T, Lee CK. Traumatic atlanto-occipital instability. A case report with follow-up and a new diagnostic technique. Spine 595-597, 1990.

© 2001 The Spine Section of the AANS and CNS.

10. Dickman CA, Papadopoulos SM, Sonntag VKH, Spetzler RF, Rekate HL, Drabier J. Traumatic occipitoatlantal dislocations. J Spinal Disorders 6:300-313, 1993.

11. Donahue DJ, Muhlbauer MS, Kaufman RA, Warner WC, Sanford RA. Childhood survival of atlantooccipital dislocation: underdiagnosis, recognition, treatment and review of the literature. Pediatr Neurosurg 21:105-111, 1994.

12. Dublin AB, Marks WM, Weinstock D, Newton TH. Traumatic dislocation of the atlanto-occipital articulation (AOA) with short-term survival. J Neurosurg 52:541-546, 1980.

13. Eismont FJ, Bohlman HH. Posterior atlanto-occipital dislocation with fractures of the atlas and odontoid process. Report of a case with survival. J Bone Joint Surg 60A:397-399, 1978.

14. Evarts CM. Traumatic occipito-atlantal dislocation. Report of a case with survival. J Bone Joint Surg 52A:1653-1660, 1970.

15. Farley FA, Graziano GP, Hensinger RN. Traumatic atlanto-occipital dislocation in a child. Spine 17:1539-1541, 1992.

16. Farthing JW. Atlantocranial dislocation with survival. A case report. North Carolina Med J 9:34-36, 1948.

17. Ferrera PC, Bartfield JM. Traumatic atlanto-occipital dislocation: a potentially survivable injury. Am J Emerg Med 14:291-296, 1996.

18. Fruin AH, Pirotte TP. Traumatic atlantooccipital dislocation. A case report. J Neurosurg 46:663-665, 1977.

19. Gabrielsen TO, Maxwell JA. Traumatic atlanto-occipital dislocation with case report of a patient who survived. Am J Roentgenol 97:624-629, 1966.

© 2001 The Spine Section of the AANS and CNS.

20. Georgopolous G, Pizzutillo, Lee MS. Occipito-atlantal instability in children. A report of five cases and review of the literature. J Bone Joint Surg 69A:429-436, 1987.

21. Grabb BC, Frye TA, Hedlund GL, Vaid YN, Grabb PA, Royal SA. MRI diagnosis of suspected atlanto-occipital dislocation in childhood. Pediatr Radiol 29:275-281, 1999.

22. Guigui P, Milaire M, Morvan G, Lassale B, Deburge A. Traumatic atlantooccipital dislocation with survival:case report and review of the literature. Eur Spine J 4:242-247, 1995.

23. Harmanli O, Koyfman Y. Traumatic atlanto-occipital dislocation with survival: case report and review of the literature. Surg Neurol 39:324-330, 1993.

24. Harris JH, Carson GC, Wagner LK. Radiologic diagnosis of traumatic occipitovertebral dissociation: 1. Normal occipitovertebral relationships on lateral radiographs of supine subjects. Am J Radiol 162:881-886, 1994.

25. Harris JH, Carson GC, Wagner LK, Kerr N. Radiologic diagnosis of traumatic occipitovertebral dissociation: 2. Comparison of three methods of detecting occipitovertebral relationships on lateral radiographs of supine subjects. Am J Radiol 162:887-892, 1994.

26. Hladky JP, Lejeune JP, Leclercq F, Dhellemmes P, Christiaens JL. La dislocation occipito-atlodienne traumatique. Neurochirurgie 37:312-317, 1991.

27. Hosono N, Yonenobu K, Kawagoe K, Hirayama N, Ono K. Traumatic anterior atlanto-occipital dislocation. A case report with survival. Spine 18:786-790, 1992.

28. Hummel A, Plaue R. Diagnostik und behandlung atlantokzipitaler rupturen. Unfallchirurgie 14:311-319, 1988.

© 2001 The Spine Section of the AANS and CNS.

29. Jevitch V. Traumatic lateral atlanto-occipital dislocation with spontaneous bony fusion. A case report. Spine 14:123-124, 1989.

30. Jones DN, Knox AM, Sage MR. Traumatic avulsion fracture of the occipital condyles and clivus with associated unilateral atlantooccipital distraction. Am J Neuroradiol 11:1181-1183, 1990.

31. Kaufman RA, Dunbar JS, Botsford JA, McLaurin RL. Traumatic longitudinal atlanto-occipital distraction injuries in children Am J Neuroradil 3:415-419, 1082.

32. Lee C, Woodring JH, Goldstein SJ, Daniel TL, Young AB, Tibbs PA. Evaluation of traumatic atlantooccipital dislocations. Am J Neuroradiol 8:19-26, 1987.

33. Lee C, Woodring JH, Walsh JW. Carotid and vertebral injury in survivors of atlanto-occipital dislocation: case reports and literature review. J Trauma 31:401-407, 1991.

34. Matava MJ, Whitesides TE, Davis PC. Traumatic atlanto-occipital dislocation with survival. Serial computerized tomography as an aid to diagnosis and reduction:a report of three cases. Spine 18:1897-1903, 1993.

35. Maves CK, Souza A, Prenger EC, Kirks DR. Traumatic atlanto-occipital disruption in children. Pediatr Radiol 21:504-507, 1991.

36. Montane I, Eismont FJ, Green BA. Traumatic occipitoatlantal dislocation. Spine 16:112-116, 1991.

37. Naso WB, Cure J, Cuddy BG. Retropharyngeal pseudomeningocele after atlanto-occipital dislocation: report of two cases. Neurosurg 40:1288-1291, 1997.

38. Nischal K, Chumas P, Sparrow O. Prolonged survival after atlanto-occipital dislocation:two case reports and review. Br J Neurosurg 7:677-682, 1993.

© 2001 The Spine Section of the AANS and CNS.

39. Page CP, Story JL, Wissinger JP, Branch CL. Traumatic atlantooccipital dislocation. Case report. J Neurosurg 39:394-397, 1973.

40. Palmer MT, Turney SZ. Tracheal rupture and atlanto-occipital dislocation: case report. J Trauma- Injury Infection Crit Care 37:314-317, 1994.

41. Pang D, Wilberger JE. Traumatic atlanto-occipital dislocation with survival:case report and review. Neurosurgery 7:503-508, 1980.

42. Papadopoulos SM, Dickman CA, Sonntag VKH, Rekate HL, Spetzler RF. Traumatic atlantooccipital dislocation with survival. Neurosurgery 28:574-579, 1991.

43. Powers B, Miller MD, Kramer RS, Martinez S, Gehweiler JA. Traumatic anterior atlanto-occipital dislocation. Neurosurgery 4:12-17, 1979.

44. Przybylski GJ, Clyde BL, Fitz CR. Craniocervical subarachnoid hemorrhage associated with atlanto-occipital dislocation. Spine 21:1761-1768, 1996.

45. Putnam WE, Stratton FT, Rohr RJ, Stitzell W, Roat G. Traumatic atlanto-occipital dislocations: value of the Power's ratio in diagnosis. J of AOA 86:798-804, 1986.

46. Ramsay AH, Waxman BP, O'Brien JF. Injury 17:412-413, 1986.

47. Rockswold GL, Seljeskog EL. Traumatic atlantocranial dislocation with survival. Minnesota Med 62:151-152, 1979.

48. Roy-Camille R, Benazet JP, Saillant G, Henry P, Mamoudy P, Leonard Ph. Luxation traumatique occipito-atloidienne. Interet de nouveaux signes radiologiques (A propos de deux cas). Revue de Chirurgie Orthopedique 72:303-309, 1986.

49. Sponseller PD, Cass JR. Atlanto-occipital fusion for dislocation in children with neurological preservation. A case report. Spine 22:344-347, 1997.

© 2001 The Spine Section of the AANS and CNS.

50. Sun PP, Poffenbarger GJ, Durham S, Zimmerman RA. Spectrum of occipitoatlantoaxial injury in young children. J Neurosurg (Spine 1) 93:28-39, 2000.

51. Traynelis VC, Marano GD, Dunker RO, Kaufman HH. Traumatic atlanto-occipital dislocation. Case report. J Neurosurg 65:863-870, 1986.

52. Watridge CB, Orrison WW, Arnold H, Woods GA. Lateral atlantooccipital dislocation: a case report. Neurosurgery 17:345-347, 1985.

53. Wholey MH, Bruwer AJ, Baker HL. The lateral roentgenogram of the neck (with comments on the atlanto-odontoid-basion relationship). Radiology 71:350-356, 1958.

54. Woodring JH, Selke AC, Duff DE. Traumatic atlantooccipital dislocation with survival. Am J Radiol 137:21-24, 1981.

55. Yamaguchi N, Ikeda K, Ishise J, Yamashita J. Traumatic atlanto-occipital dislocation with long-term survival. Case report. Neurol Med Chir 36:36-39, 1996.

56. Zampella EJ, Duvall ER, Langford KH. Computed tomography and magnetic resonance imaging in traumatic locked-in syndrome. Neurosurgery 22:591-593, 1988.

57. Zigler JE, Waters RL, Nelson RW, Capen DA, Perry J. Occipito-cervico-thoracic spine fusion in a patient with occipito-cervical dislocation and survival. Spine 11:645-646, 1986.

© 2001 The Spine Section of the AANS and CNS.

Figure 1

Mid-sagittal diagrams of the craniocervical junction show the various methods for identifying AOD on a lateral cervical radiograph including the Wholey measure (A), Power's ratio (B), Dublin measure (C), X-line method (D), and BAI-BDI method (E).

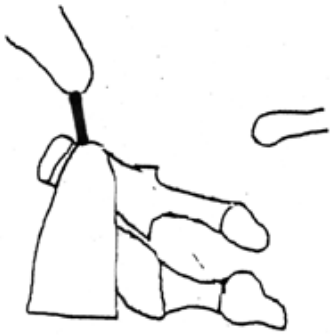

Figure 1A

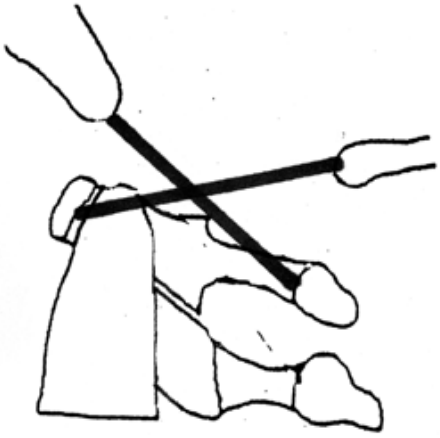

Figure 1B

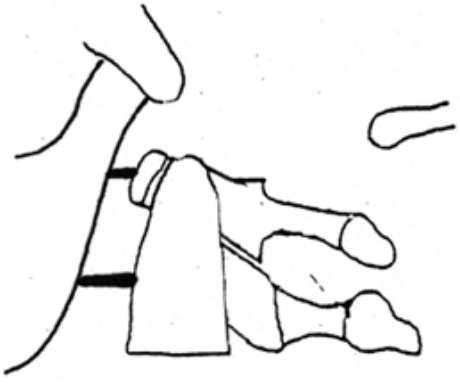

Figure 1C

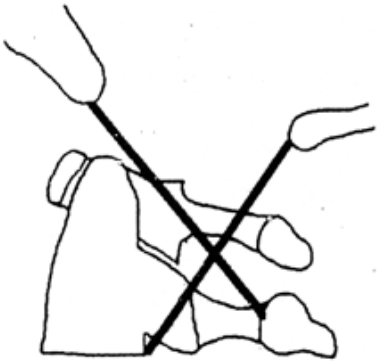

Figure 1D

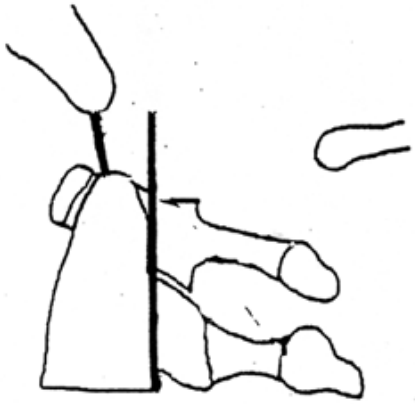

Figure 1E

© 2001 The Spine Section of the AANS and CNS.