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Lateral Mass Fusion in Atlanto - Axial Dislocation

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Congenital atlanto-occipital dislocations cause symptoms by compression of the neuraxis at the craniovertebral junction. The aim of treatment is to relieve the pressure on the nervous tissue and fix the spine so that future compression of the neuraxis is avoided. This can be done in a variety of ways, and each case has to be assessed individually, as to which method is best suited for him. For instance, it is fairly a common occurrence of basilar invagination with atlanto-axial dislocation In such cases it is obviously not sufficient to reduce the dislocation and do an anterior fusion. Posterior decompression of the basilar invagination together with a posterior graft would be the obvious method of choice.

There are cases where the lateral atlanto-axial joint is absent. The normal superior articular facet of the axis lies more or less horizontal and on this the lateral mass of the atlas rests to form the atlanto-axial joint. We have found patients where the lateral mass of the atlas is well developed but the axis has no superior articular facet in which it can rest. In these cases the superior articular facet of the axis is not developed and the body of the axis is without the buttress that forms the superior articular facet. In such cases an anterior fusion is not possible. In four cases where we found this anomaly, an attempt was made to fuse the lateral mass of the atlas to the anterior surface of the body of the axis, but none of the grafts took successfully and a subsequent posterior fusion was necessary. If careful tomograms fail to show the A-A joint in the AP view, we now proceed to do a posterior fusion. It is also not possible to do an antero-lateral fusion in very young children where the articular surfaces are still cartilagenous. In cases where the odontoid is not developed, or is rudimentary, transoral excision of the odontoid process is obviously not the method of choice.

The subject which I have been asked to talk about is lateral mass fusion in A -A dislocation. In the early stages of recognition and treatment of A -A dislocations, we decompressed the cranio-vertebral junction by doing a laminectomy of C1 and C2 and enlarging the foramen magnum, without preliminary skull traction. This resulted in producing a haematomyelia and death in a number of patients. Since then we always put the patient on skull traction preoperatively to reduce the dislocation. In most cases we obtain a good reduction and then grafting is done. Redislocation is prevented by maintaining the traction till the graft fuses.

In cases of a fixed variety of dislocations where a perfect reduction is not obtainable, but the clinical condition of the patient improves with traction even with a partial reduction, fusion is done in this partially reduced position. In some patients who come at a very late stage and who do not improve with traction, indicating irreparable neural damage, surgery is not advised. In our earlier cases following posterior fusions, there were instances of redislocation either due to trauma to the neck or spontaneously due to traction of the weight of the head during flexion of the neck.

Mechanically, a compression graft is always stronger than a distraction graft and we started to use an anterior route to fuse the lateral atlanto-axial joints. Initially, we approached this joint, retracting the pharynx medially and the carotid sheath with the sternomastoid laterally. This was the method of ant. fusion. There was a high incidence of post-operative difficulty in swallowing and many patients required a tracheostomy. Therefore a more lateral approach was devised where the sternomastoid was retracted laterally and all other structures were retracted medially. This is the anterolateral approach to the A-A joint presently practiced.

Material

One hundred and twenty cases with atlanto-axial dislocations have been operated upon, 14 required reoperation, making a total of 134 operations.

Table 1 - Operative treatment

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Antero Lateral Fusion Operative Technique

The patient is supine, with the skull traction applied. The incision starts at the tip of the mastoid process and curves downwards and forwards below the angle of the mandible almost to the midline. The platysma is cut in the same line. The external jugular vein may need to be ligated and divided. The carotid sheath is dissected away from the sternomastoid muscle and retracted medially. The parotid gland may need to be pushed up out of the way and lymph glands along the jugular vein may need to be dissected out. The transverse process of the Atlas is easily felt, and traced medially, leads to the lateral mass of the Atlas. The longus capitus muscle covers the lateral atlanto-axial joint which can often be felt as it is opened up by the traction. The muscle is cut across and subperiosteally dissected off the lateral mass of the atlas and the body of the axis. At this stage there may by troublesome oozing from the bone and muscular vessels, which is, however, easily controlled. The joint is then opened and with a dental drill the adjacent articular surfaces are rawed, as also the anterior surfaces of the axis and the lateral mass of the Atlas Previously taken bone graft from the iliac crest or from a bone bank is now packed into the joint space and a piece of bone put over the roughened surfaces of the Atlas and the axis. A stitch through the cut edges of the longus capitis is passed over the graft and holds it firmly down against the bone bed. The strenomastoid and the pharynx are allowed to fall together and the platysma and skin are stitched in layers. A Jackson-Pratt drain is put in the prevertebral space for 24 hours. The patient is returned to the ward with the skull traction on and this is maintained, with reduced weights, until bone fusion occurs. Injection Dexamethasone 4 mg 6 hourly helps to prevent oedema.

Check X-rays are taken every 15 days. At the end of two and half to three months, the traction is removed and a cervical collar is given and physiotherapy is started to mobilise the patient.

Results

The recovery of neurological function depends entirely on the stage at which the patient presents himself for treatment. The longer the compression has been present, the more is the permanent damage in the cord, where no improvement is obtained by the traction, we presume the damage to be irrecoverable and now we do not operate on such cases. We have graded the results as follows:

Grading of results

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Table 2

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As you see seven patients with lateral mass fusion died. One patient who had post-fusion which redislocated and then had an Ant. fusion done died of pulmonary infection one month after the fusion. In this case one of our earliest anterior approaches, tracheostomy was too long delayed following pharyngeal oedema.

Of the anterior fusion, 2 patients died. One died with sudden cardiac arrest. Autopsy was not permitted. The other had several bilateral pulmonary infections.

Two patients with Antero lateral fusion died. One had a coronary infarct seen at autopsy and one died of pulmonary infection two months after surgery

Early in the series we did anterolateral fusion and post-decompression at the same session in patients who did not improve with traction. Two died, both with severe pulmonary infection. This highlights the incidence of poor respiratory capacity in patients who come with severe long standing compression and extremely weak intercostals and diaphragm. The use of a Jackson-Pratt drain for 25 hours to drain accumulated blood ooze and also the use of injection. Dexamethasone 4 mg 6 hourly for 5 days post operatively to reduce tissue oedema, has helped to prevent respiratory and swallowing difficulties in our more recent cases.