In Case 2, the location of the lesion is most unusual. The medical literature failed to reveal another case of giant-cell tumor of the temporal bone. The cystic character of the lesion and the fact that many giant-cell tumors of bone are manifestations of hyperparathyroidism stress the need for blood chemical studies in similar cases.

Giant-cell tumors arise from resorptive processes in cartilaginous bone with the possible exception of the type that Geschickter and Copeland6 refer to as subperiosteal. This theory fails to account for the occurrence of such a tumor in the squamous portion of the temporal bone (membranous bone), a fact that has been commented upon by Keegan and Baker8 in their report of a giant-cell tumor of the frontal bone.

REFERENCES

NON-TRAUMATIC ATLANTO-AXIAL DISLOCATION
REPORT OF CASE WITH RECOVERY AFTER QUADRIPLEGIA

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In 1830, Bell1 described the occurrence of dislocation of the atlas because of destruction of the transverse ligament that holds the odontoid process of the axis in its normal anterior position. This particular instance was associated with a neck infection in the patient. Greig3 demonstrated that during a cervical infection, a diffuse tissue hyperemia occurs which results in adjacent bone decalcification with loosening of the intervertebral ligaments, especially between the atlas and axis, so that the skull and 1st cervical vertebra dislocate anteriorly. In 1932, after a thorough study of this disease process, Jones5 emphasized specifically that dislocation results because the decalcified arch of the atlas no longer will permit a firm attachment of the

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transverse ligament which is chiefly responsible for holding the odontoid process anteriorly against the atlas.

At this time, Jones\textsuperscript{10} reviewed 14 cases in the literature and added 2 personal instances. By 1934, Jones and Roberts\textsuperscript{11} presented 5 additional cases bringing the total to 21. It was learned that this disease occurred most frequently between the ages of 6 and 12 years and was distributed equally between the sexes.\textsuperscript{12} Almost all of the individuals had a precedent inflammatory condition of the neck such as: abscesses, tuberculous cervical glands, or syphilitic pharyngeal ulceration. Fitzwilliams\textsuperscript{7} had an instance occurring with acute rheumatism; Böger,\textsuperscript{8} Ely,\textsuperscript{9} Stammers and Frazer,\textsuperscript{13} and Evans\textsuperscript{10} reported additional cases associated with rheumatic fever. Trauma is not a principal factor. The type of infecting organism made no difference since the hyperemia throughout the cervical tissues was directly responsible for the decalcifying process. However, Berkheiser and Seidler\textsuperscript{14} believed that the streptococcus was the most common organism in their 5 cases, which are included with the series of Jones and Roberts.\textsuperscript{11} Approximately 10 days after the throat infection, a sudden neck pain or torticollis was usually experienced. At this time, cervical x-rays revealed a forward dislocation of the atlas on the axis with vertebral decalcification, especially of the arch of the atlas.\textsuperscript{15} The usual treatment has been manipulative reduction and traction fixation or plaster immobilization for approximately 10 weeks.\textsuperscript{15} By this time, recalcification has occurred.

Jones and Roberts\textsuperscript{11} provided a logical clinical classification of this syndrome:

\textit{First Type.} This is the most common. The atlas is dislocated forward and rotated slightly but enough to produce torticollis with painful movement and deep cervical muscle spasm.

\textit{Second Type.} The displacement is more marked, with spinal cord compression but with survival of the patient. Jones and Roberts\textsuperscript{11} reported the first case of this type, which was that of a quadriplegic child who made a full recovery after reduction of the dislocation. Our adult patient, who had complete recovery of quadriplegia following surgical spinal cord decompression, fits into this group.

\textit{Third Type.} The dislocation is complete, with sudden death from spinal cord compression, such as in the cases of Bell,\textsuperscript{1} Greig,\textsuperscript{8} Reid,\textsuperscript{12} and Jones.\textsuperscript{16}

In 1935, after a thorough search of the literature, Hess, Bronstein, and Abelson\textsuperscript{10} found a total of 22 cases and added 2 of their own. Although this is mainly a disease of children, they found mention of it in only one pediatric journal.\textsuperscript{14} They emphasized the avoidance of excessive rotation and manipulation of a child’s head during surgical drainage of cervical infections since the relaxed anesthetic muscles may not protect against vertebral dislocation. These same authors\textsuperscript{9} reported in 1942 a 5-year follow-up of a 12-year-old boy who had an unreduced spontaneous atlanto-axial dislocation. Their suggestion was that although occasionally spontaneous reduction and normal calcification may occur, some of the persisting spinal deformities in older persons may have arisen as in their described case. Martin\textsuperscript{11} added 4 personal cases to the literature in 1942. These instances were in children without neurological complications. In the same year, Englander\textsuperscript{4} presented a girl with this syndrome accompanied by neurological damage from an injured spinal cord.

Our case reported here is of an adult who developed quadriplegia from spinal cord compression and recovered after surgical decompression.

\textbf{CASE REPORT}

Mrs. R. R., a 40-year-old white housewife, was first admitted to the Northwestern Hospital on Sept. 20, 1946. She had had a sore throat and stiff neck since Aug. 1, 1946. Polio-