Medial Epicondylectomy for Ulnar Palsy*

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Neuropathies of the ulnar nerve at the elbow produced by traction, compression, or percussion of the nerve against the unyielding medial epicondyle require treatment to eliminate the harmful nerve-bone relationship. The currently popular procedure is to transplant the ulnar nerve to some site on the flexor side of the medial epicondyle. All transplantation operations require exposure of a considerable length of the nerve, with risk to its branches, merely to transfer the nerve to the other side of a bony prominence which itself has no important function. In its new location, the nerve lies subcutaneously in an artificial bed or in traumatized muscle where fibrosis, cicatricial ischemia, angulation against the edge of the intermuscular septum, or traction in elbow extension may produce further interference with its function.

King and Morgan12 pointed out 28 years ago the logic of removing the offending medial epicondyle, as one would a tumor or foreign body, to permit the nerve to find its own optimal position. We are now reporting our experience with 14 patients treated by King and Morgan's medial epicondylectomy. We believe this method gives results superior to anterior transposition operations.

Historical Review

The susceptibility of nerves to harm by subtle physical influences was recognized in 1876 by Erb8 who wrote that "even a slight mechanical action may so change the molecular constitution of the motor nerves as to abolish their power of contraction." One year later, Panas19 published his observations on four cases of chronic ulnar palsy. One patient had a cubitus valgus deformity from a distal humeral fracture for 13 years before his ulnar symptoms began; the nerve of another patient was compressed by a sesamoid bone and relieved by excision of the bone; a third patient strenuously rowed a storm-tossed boat and began to have symptoms 6 months later; the fourth patient had ulnar nerve compression due to spurs on the distal end of the humerus.

At the end of the 19th century Mouchet16 described another case of delayed ulnar palsy in a patient with elbow deformity produced by fracture of the external condyle, and Broca and Mouchet3 reported that nine of the 78 fractures of the lower end of the humerus and of the forearm observed during a 20-year period resulted in nerve malfunctions; there were 40 external condyle fractures, three of which had nerve deficits, and 38 supracondylar fractures, six of which had nerve injuries. In these nine patients with nerve injury, the ulnar nerve was involved five times, the median five, and the musculospinal three. Broca and Mouchet also distinguished between lesions of primary, secondary, and delayed onset and recognized the latter as something to which the ulnar nerve is especially liable after external condyle fractures of childhood. These may result in dubitus valgus deformity which in turn leads to repeated stretching and percussion of the nerve against hard objects such as table tops and chairs. "Tardy ulnar palsy," the American term for what the continent knew as "Maladie de Mouchet," was first used in 1914 by Mouchet17 and in America in 1916 by Hunt.11 During the two decades between World Wars I and II, a number of reports and reviews of tardy ulnar palsy appeared.1,5,6,7,9,12,15 All have been in substantial agreement as to the cause, diagnosis, and treatment of this syndrome.

If cases of ulnar nerve deficit due to direct trauma are excluded, the remaining mechanically-produced ulnar neuropathies of rapid or slow onset and fast or gradual progression are found to have widely diverse causes. Over 50% have been said to result from fractures that produced valgus deformity, nerve-displacing callus, irritating ununited

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medial epicondyle, or varus deformity with nipping of the nerve between the medial epicondyle and the olecranon in elbow extension. About 20% of tardy ulnar neuropathies have been attributed to arthritis or exostosis that displaced the nerve from its groove and made it more vulnerable to bumps as well as to harmful tension when the elbow was flexed. Occupational trauma alone with no anatomical abnormality of the elbow has been blamed for about 4% of the cases. Abnormal development, adhesions, foreign bodies, tumors, blood vessel aberrations, shortening of the nerve after anastomosis of lacerations in the forearm, and unknown causes account for the rest.

A most interesting and increasingly important group first identified by Gowers in 1907 are those patients with presumably normal elbows who, after a period of enforced bed rest, developed progressive ulnar neuropathy despite resumption of normal activity and diligent avoidance of further pressure on the nerve. Four of the 15 cases in our series were of this sort. This mechanism seems akin to that in patients with occupation-produced palsies who repeatedly pummel the nerve against window sills, desks, and the like. These patients too often get worse after seeming avoidance of further direct compression, and deserve surgical relief just as certainly as those with fixed anatomical abnormalities.

**Pathogenesis**

Ischemia, anoxia, edema, fibrosis, and hemorrhage have seemed significant among the intraneural effects of repeated stretching of the nerve around the fixed medial epicondyle, of pummeling it between the bone and unyielding objects in the environment, and of compressing it against padded or unpadded surfaces. Lewis, et al., compressed the upper arm with an inflatable cuff and observed acral loss of touch before pain and pain before motion with centripetal progression of all three. When the cuff pressure exceeded the systolic blood pressure, paralysis consistently began in approximately 25 minutes. If, after paralysis appeared, a second cuff inflated to an equal pressure was applied distal to the first and the first cuff then removed, recovery from the paralysis occurred only to be followed after a further latent period by its reappearance. This seems to indicate that this paralysis is due to ischemia of the compressed segments of nerves rather than to general peripheral stasis. The centripetal and sequential progression of the deficits appears due to the greater sensitivity of the larger nerve fibers to anoxia.

Direct pressure applied by Waller to his own ulnar nerve at the elbow produced paralysis and anesthesia of slower onset and with greater variability than when a cuff was used. Recovery was also faster upon release of this pressure. Waller, after once compressing the nerve for 45 minutes, observed recovery of motion and sensibility to be complete only after 11 days. Anatomical variation is one possible reason for variability in the deficits produced by a pressure which may leave certain small vessels patent between blanched nerve bundles. The significance of this may be greater when pressure is applied to a short segment of nerves. Pressure applied over a long segment, as by a cuff, may compensate for anatomical variation and produce more specific deficits. Since in clinical compressions of the ulnar nerve only a short segment is affected, the amount and duration of pressure required to produce neuropathy is variable.

Experimentally applied pressure of less than 2 hours' duration has produced impairment of sensation lasting for a few hours and of motor function for from 2 to 18 days. This completely reversible palsy is attended by no definite histological change. Denny-Brown and Brenner categorized the effects of pressure on nerve conduction as: 1) undetectable; 2) paralysis with rapid, complete recovery on release of pressure; 3) paralysis with delayed recovery without degeneration; and 4) complete anatomic lesion with degeneration. The complete lesion was characterized by interruption of axis cylinders and myelin sheaths at the site of compression, and Wallerian degeneration of the parts of the nerve distal to the compression; connective tissue and vessels suffered lesser degrees of damage.

Of greatest relevance to our report is the intermediate degree (grade 3) characterized by delayed recovery but no Wallerian degeneration in the distal nerve segment. In this type of lesion the compressed segment shows slight vacuolation of myelin with swelling