Can muscle hypertrophy cause entrapment neuropathy?

TO THE EDITOR: I read with interest the article by Wilson et al.8 (Wilson TJ, Tubbs RS, Yang LJS: The anconeus epitrochlearis muscle may protect against the development of cubital tunnel syndrome: a preliminary study. J Neurosurg [epub ahead of print February 12, 2016. DOI: 10.3171/2015.10.JNS151668]).

The cubital tunnel is formed by a groove between the olecranon and the medial epicondyle (ME), usually covered by a fascial sheet known as the cubital tunnel retinaculum (i.e., the Osborne fascia).5 Occasionally the cubital tunnel retinaculum is replaced by a muscle tissue—i.e., the anconeus epitrochlearis muscle that is often regarded as a cause of ulnar neuropathy at the elbow (UNE) In their report, Wilson et al.8 retrospectively examined the prevalence of this muscle found during surgery for the UNE and compared it to the prevalence in arms of control patients reported in the literature. In their series of 168 UNE patients, they found a prevalence of 5.4%, which was significantly lower (p < 0.001) than the 15.5% reported in 634 control arms from previous MRI and cadaver studies.8 The authors concluded that the presence of the anconeus epitrochlearis muscle seems an unlikely causative factor for UNE. Furthermore, they argued that its presence may actually have a protective role against the UNE.8

In our series of 221 patients we found UNE to consist of 2 focal neuropathies.3,4 The first occurs 2–3 cm distal to the ME. Because we found ulnar nerve constriction on ultrasonography in 38% of these arms,3 we concluded that this type of UNE is caused by ulnar nerve entrapment under the humeroulnar aponeurosis (HUA),3 which is the distal expansion of the cubital tunnel retinaculum. As we found UNE at this location mainly in dominant arms of older farmers, miners, carpenters, and so on,4 we reasoned that in these patients UNE is caused by decades of hard manual labor that transforms the HUA into a thick fibrous band. Because the anconeus epitrochlearis stretches 2–3 cm proximal to the HUA, it is difficult to attribute to it any role in the pathogenesis of this UNE variety, known in the literature as “a cubital tunnel syndrome.”5

The second, more common type of UNE occurring in 85% of arms in our series was found at the ME or up to 2 cm proximal to it in the retroepicondylar (RTC) groove.3

We observed it almost exclusively in the nondominant arms of younger administrative workers and students.4 As no nerve constriction was found in any of the arms affected at this location,3 we believe that in these arms, ulnar neuropathy is caused by exogenous nerve compression, probably most importantly while using a computer mouse with the dominant arm and leaving the nondominant arm lying pronated on the desk.4 Because the anconeus epitrochlearis stretches between the ME and the olecranon, it may well protect the ulnar nerve at this location.

I agree with Wilson et al. that “entrapment neuropathies commonly occur as nerves pass beneath rigid ligamentous structures,”8 as this is also the case with ulnar nerve entrapment under the HUA (i.e., the cubital tunnel syndrome). However, I am reluctant to accept their suggestion of “a much rarer scenario involving compression of a nerve by a hypertrophied muscle secondary to overuse.”8 My opinion is based both on my personal experience and on my general reasoning. Although at my institution over 8000 electrodiagnostic evaluations are performed each year, in more than 20 years of practice I have not come across a single patient with a nerve entrapment unequivocally proven to be caused by muscle hypertrophy (e.g., pronator teres syndrome). Moreover, I am reluctant to believe that a rather soft hypertrophic muscle may actually cause entrapment of a much tougher peripheral nerve.

In my opinion nerve entrapment within or beneath the muscle can only occur due to tough fibrous bands. As we did not find ulnar nerve entrapment proximal to the RTC (i.e., under the arcade of Struthers) or distal to the HUA (i.e., at the deep flexor pronator aponeurosis)3 in any of our 221 UNE patients, entrapment by tough fibrous bands is, at least for the ulnar nerve, probably much rarer than their frequent citation in the literature would suggest.

Returning to the role of the anconeus epitrochlearis muscle, it is easy to imagine that an additional layer of muscle overlying the ulnar nerve in the bony RTC groove might provide some degree of protection against the external compression, as the results of Wilson et al. suggest.8 However, strictly speaking, the anconeus epitrochlearis does not protect the ulnar nerve against cubital tunnel syndrome but, rather, against much more common external compression in the RTC groove.

It is interesting to note that the role of the anconeus epitrochlearis in the causation of UNE in many respects reflects a similar recent evolution in our understanding
of another factor traditionally believed to cause UNE, ulnar nerve dislocation (i.e., luxation) at the elbow. As for the anconeus epitrochlearis, recent studies also found a similar occurrence of ulnar nerve dislocation in arms with and without UNE. To underscore similarity even further, there are also some data indicating that complete ulnar nerve dislocation may even have a protective effect. It may function as a natural equivalent of surgical anterior transposition of the ulnar nerve, effectively relieving ulnar nerve strain during protracted elbow flexion.

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References

Disclosures
The author reports no conflict of interest.

Response
No response was received from the authors of the original article.

Cranioplasty after decompressive craniectomy


Decompressive craniectomy is a life-saving option for patients with medically refractory elevated intracranial pressure due to various pathologies. Cranioplasty using an autologous bone flap or artificial bone graft substitute is done for cosmetic, mechanical, and therapeutic purposes. The authors reported statistically significant higher rates of bone resorption in the cohort of patients who underwent cranioplasty using autologous bone graft compared to those in whom an artificial bone substitute was used. However, the authors reported a lower risk of complications when bone resorption was not taken into account as a complication. The authors concluded that younger age (<30 years of age), shunt dependency, and bone flap fragmentation are independent risk factors for bone resorption and need for reoperation, and they recommended initial artificial bone substitute in patients younger than 30 years of age.

Wound infection after cranioplasty is a known factor associated with bone flap resorption. The rate of infection following cranioplasty is quite high, up to 10%. Hence, it will be important to know the infection rates in the reported series to eliminate the confounding effect of infection. Resorption of the bone flap is a major issue in the pediatric age group following cranioplasty, especially in young children up to an age of 7 years. The authors described 3 age groups (0–30, 31–60, and 61–90 years) for comparison. It would probably be more helpful for the authors to describe the number of pediatric patients and offer a comparison between pediatric and adult patients.

The follow-up duration was 6 months in a majority of patients, and a long-term follow-up period is warranted to establish a true picture of the complications of autologous bone and artificial bone graft, because complications as late as 6 years postoperatively have been reported following cranioplasty.

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