Early Surgical Decompression: Too Early or Too Late?


Lindley B. Wall, MD

Initial evaluation of nerve compression involves determining longevity and severity of symptoms prior to proceeding down a treatment path. The surgeon must differentiate whether the presenting condition involves a transient onset of symptoms affecting a normal nerve, in which case relief of the compression should allow return to a normal physiologic state; or a nerve with an altered physiology resulting from a slowly developing subclinical, and possibly intermittent, chronic compression, that has a lower threshold of tolerance for nonphysiologic pressure changes. The latter nerve may return to its baseline condition once the offending agent is removed, yet remain physiologically abnormal and susceptible to subsequent pressure changes. A third category involves nerves in a symptomatic state of static chronic compression. Thus, the clinical question arises: Should a nerve with new-onset symptoms undergo surgical decompression acutely, or should it be allowed a trial of nonoperative treatment such as bracing, anti-inflammatory medications, and injections?

The study by Jung et al. has attempted to shed light on this clinical question involving chronic compression by means of a laboratory animal model. The authors conducted an elegant study investigating neurovascular changes utilizing a previously published murine model of chronic nerve compression1, comparing the effects of early surgical decompression (at two weeks) and late decompression (at six weeks). They demonstrated an injury response within the nerve after the time of compression. Interestingly, they identified evidence of irreversible physiologic changes seen after late decompression but not after early decompression at two weeks. This study demonstrates that decompression of an otherwise physiologically normal nerve can return the nerve to its previous normal physiologic state if performed within a certain early time period.

The model utilized for this study, constructed to mimic human nerve compression by producing a chronic compression scenario in the murine model without inducing structural change in the nerve, deserves mention independently from the current results. Although this model is well thought out, it should be noted that it has not been formally validated by comparison with known changes in human nerves experiencing compression. Thus, direct application to the clinical setting remains impossible. Second, the established murine model is a "chronic" nerve compression model; it is a stretch to consider the nerves in this study, decompressed at two weeks, to have undergone "chronic" compression. This state of compression would most logically be considered an early subacute compression. Therefore, the study by Jung et al. seems to be comparing a subacutely compressed nerve (two weeks) to a more chronically compressed nerve (six weeks).

Another consideration, and a limitation of the murine model used, is the initial state of the nerve. This study models a nerve experiencing a constant static compression; a mechanical stricture was placed onto a physiologically normal nerve. The study does not model the common clinical situation of the physiologically abnormal nerve with subclinical signs of compression causing intermittent symptoms over time. It is possible that a nerve with a lower threshold resulting from subclinical chronic compression would produce a different physiologic response to constant subacute or chronic compression than a normal nerve because of possible physiologic adaptation. Furthermore, such a nerve would respond differently to surgical decompression secondary to its previously altered physiology. Thus, there may be no timing of decompression that allows complete return to normal, as assessed by diagnostic tools, suggesting that no surgery may be early enough in these cases.

The overall question that remains is whether early surgical release for early signs of nerve compression is justified to prevent irreversible change and maximize long-term outcomes. Although it can be argued that this study provides laboratory support for early release of a nerve with evidence of constant static compression in a subacute setting, it does not provide evidence in favor of early decompression of a physiologically altered nerve that has experienced slow chronic subclinical compression, given that this condition was not modeled in the study. Notably, the study does raise our awareness of the potential early adverse influences of subacute and chronic nonphysiologic pressures on nerve function and provides new data establishing that a time frame does exist for physiologic recovery of compressed nerves. Lastly, the study supports the need for
additional research regarding improved neurodiagnostic testing to determine the benefit of early decompression prior to irreversible changes within the nerve.

Lindley B. Wall, MD*
Washington University, St. Louis, Missouri

*The author received no payments or services, either directly or indirectly (i.e., via his institution), from a third party in support of any aspect of this work. Neither the author nor his institution has had any financial relationship, in the thirty-six months prior to submission of this work, with any entity in the biomedical arena that could be perceived to influence or have the potential to influence what is written in this work. Also, the author has not had any other relationships, or engaged in any other activities, that could be perceived to influence or have the potential to influence what is written in this work. The complete Disclosures of Potential Conflicts of Interest submitted by authors are always provided with the online version of the article.

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