NEUROVASCULAR compression of various cranial nerves and the development of clinical syndromes related to them was recognized many years ago, but the clinical significance of decompression was not really appreciated until 1967, when Peter Jannetta and his colleagues began to report their operative experience with a variety of cranial nerve syndromes. There has been abundant skepticism over the last two decades about whether transposing an arterial loop away from the trigeminal root in tic douloureux, from the cochlear-vestibular bundle in Ménière's disease and benign positional vertigo, and from the facial nerve in hemifacial spasm would do anything to relieve those conditions. Even greater skepticism developed when neurovascular compression of cranial nerves by venous structures was thought to account for the same compression syndromes when an arterial loop could not be found at surgery. Nevertheless, experience with neurovascular compression and the effects of decompression for all the conditions noted above have been reported by many investigators in thousands of patients over the last several years. Neurovascular decompression has proven effective to the satisfaction of most neurosurgeons and has become a part of the armamentarium of those who treat patients with tic douloureux, Ménière's disease, benign positional vertigo, and hemifacial spasm.

Despite the validity of arterial transposition as a treatment for syndromes of the trigeminal, facial, and vestibular nerves, skepticism continues in relation to the use of neurovascular decompression for spasmodic torticollis and essential hypertension. In the case of torticollis, the small number of patients afflicted with this condition probably accounts for the lack of experience with decompression as a treatment option. On the other hand, large numbers of patients with essential hypertension are available for treatment, which argues for the availability of greater experience and potential proof that arterial compression of the medulla is the cause of essential hypertension.

There is abundant scientific evidence that brain-stem structures regulate arterial blood pressure. Cushing made us aware of brain-stem mechanisms in hypertension at the turn of the century. Others have explored the basic mechanisms of hypertension in greater detail since then. Over the past three decades, Reis and colleagues have investigated both the anatomy and physiology of hypertension further, focusing on medullary interactions within the brain stem and nuclei of the cerebellum. It is now known beyond the shadow of a doubt that compression of the medulla (occasionally confirmed at operation when the brain stem is manipulated) elicits sympathetically mediated hypertension. Thus, there are physiological as well as anatomical reasons to suspect that neurovascular compression could cause essential hypertension and that decompression might treat it effectively. Scattered reports in recent years describe the beneficial effects of such treatment; however, the operation has not become popular.

The study by Naraghi, et al., in this issue now provides postmortem anatomical evidence to support the idea that neurovascular compression of the left side of the medulla causes arterial hypertension. The cause-and-effect correlation in their findings is, literally, perfect. In the same study, control groups consisting of patients with renal hypertension and patients with normal blood pressure during life showed no compression, whatsoever, of the left ventrolateral medulla at autopsy. Perfect correlations, while possible in biological systems, are rarely seen and, when observed, are often discounted as "too good to be true." Is this study an example of "too good?"

Where does neurovascular decompression of the ventrolateral medulla on the left side fit in the treatment of essential hypertension? The answer to the question awaits the test of time, as it did for the treatment of
other neurovascular compression syndromes of the cranial nerves. Today, hypertension of many varieties is treated fairly effectively with medications. Still, the appeal of a cure for hypertension remains, as demonstrated years ago when stenosis of the renal artery was recognized to cause hypertension and surgical treatment to cure it. Perhaps neurovascular decompression of the ventrolateral medulla will find a similar therapeutic role.

Basic and clinical research is still needed to define the role of surgery as a treatment for essential hypertension. Risks, benefits, costs, and criteria for treatment must be worked out. For those who remain skeptical about the efficacy of neurovascular decompression for hypertension, the medullary compression hypothesis supported by Naraghi, et al., deserves further scrutiny before it is proven unequivocally.

Reference


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