Letters to the Editor

Neurovascular Contact of Cranial Nerve IX and X Root-Entry Zone in Hypertensive Patients

To the Editor:

Neurovascular contact, consisting of looping vascular structures comprised of the vertebral, posterior, or anterior-inferior cerebellar artery, has been identified to impinge on the medulla oblongata in many patients with "essential" hypertension. Hohenbleicher et al.1 have shown data that cast doubt on the relevance of neurovascular contact in the pathophysiology of hypertension, because the incidence of left-sided neurovascular contact seems to be equally frequent in normotensive and hypertensive patients (16% versus 23%, P = 0.12). Although we appreciate the attempt to perform this large-scale trial, we believe that the inclusion criteria of normotensive and hypertensive patients and the presentation of the results by Hohenbleicher et al.1 give some cause for the following critical comments.

The title of the presented study misleading the reader to assume that hypertensive patients in general have the same incidence of neurovascular contact as normotensives have, but in fact, the authors saw no difference in left-sided neurovascular contact, observing a group of only mildly hypertensive patients. In control subjects, the authors ruled out hypertension by repeated blood pressure measurements documenting resting blood pressure of <140/90 mm Hg. There should be no discussion about the 24-hour blood pressure measurement is the only standard to rule out hypertension and to make sure that no patient of the normotensive group has a mild form of hypertension. In hypertensive subjects, the authors based the inclusion criteria on the prescription of antihypertensive drugs and the average 24-hour blood pressure level of ≥140/90 mm Hg. Surprisingly, in the presentation of the results, the authors show the resting blood pressure, which is different from the 24-hour measurement. The resting blood pressure in hypertensive patients depends very much on the conditions of measurement (eg, time, frequency), which are not described in the methods. Based on the average daytime and average nighttime blood pressure shown in Table 3, we read that lots of patients with very mild hypertension (φ144/88 mm Hg) must have been included in the hypertensive group, 36% untreated patients and 50% treated only with 1 or 2 antihypertensive drugs. But shifting a few patients from one group to another would result in substantial changes.

We conclude that the difference in blood pressure (mm Hg) between normotensive patients and hypertensive patients was not very clinically important in the presented study of Hohenbleicher et al.1 We agree with the authors that the pathophysiology of hypertension and the role of neurovascular contact is still not fully understood, but from all recent work we know, neurovascular contact is not a morphological abnormality that reaches clinical importance at the borderline of 140/90 mm Hg. In our understanding of neurovascular contact, one should make sure that at least patients with severe hypertension are compared with normotensive patients to get a realistic chance in finding morphological differences.

Furthermore, in setting the primary end point of their study, the authors have excluded the right-sided neurovascular contact from the potential pathophysiological linkage to hypertension. Regarding both, the left- and the right-sided neurovascular contact, Hohenbleicher et al.1 find a significant difference in the incidence of neurovascular contact between the normotensive and the hypertensive group (25% versus 39%, P < 0.05). Other data2 indicate that the responses to pulsatile compression of the unilateral medulla oblongata are similar on both. Therefore, until we do fully understand the pathophysiologic relationship, we should be careful to not neglect neurovascular contact on the right side because of unclear clinical relevance. Further studies are necessary to clear up the situation.

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Response

Menzel and Geiger take objection to several aspects of our paper.1 These include the professedly misleading title, our definition of normotension, the severity of hypertension, and our focus on left-sided neurovascular compression.

It is not clear to us why the title of our study should be misleading. The title simply states what was done, namely, a study on neurovascular contact of the cranial nerve IX and X root-entry zone in hypertensive patients. The outcome of the study was not mentioned in the title.

Our definition of normotension was based on the repeated documentation of normal blood pressure values in controls, measured under resting conditions by trained personnel. We are not aware of any official guidelines suggesting that the definition of normotension must be based on 24-hour blood pressure measurements. Although we cannot rule out that some individuals may have inverted circadian rhythms or other aberrations of blood pressure that can only be detected with this technique, it is highly unlikely that this possibility should have had a substantial effect on the outcome of our study.

We certainly agree that the prevalence of neurovascular compression may be higher in patients with more severe hypertension (ie, patients requiring ≥3 antihypertensive drugs). Nevertheless, our hypertensive patients included a wide range of hypertension, from moderate to severe (WHO stage III), and we found no suggestion of a relationship between severity of blood pressure and the presence or absence of neurovascular compression (Table 3). It is therefore very unlikely that shifting a few patients from one group to another would have substantially altered our findings. We do, however, concede that similar studies in patients with more severe hypertension may need to be done.

As discussed in our paper, our primary hypothesis was focussed on the presence of left-sided findings, as this was the lesion originally described by Janetta and Gendell.2 We did, however, observe a significantly higher incidence of neurovascular contact when both sides were considered (as noted in our paper). As this was not the primary "end point" of our study, we were cautious not to overinterpret this observation.

Currently, apart from some experimental evidence,3,4 the idea that neurovascular compression of the brain stem results in hypertension is largely based on anecdotal reports on improved blood pressure following surgical decompression, a body of literature to which Geiger et al.5 have made important contributions. As succinctly pointed out by Johnson and Coley6 in correspondence related to this recent work by Geiger et al., "protagonists of neurogenic hypertension will continue to be critical of the MR technique and selection bias of studies that do not show significant differences between hypertensives and controls." We agree with Johnson and Coley that even when this finding is present, it can have no diagnostic or therapeutic impact, because the prevalence of similar vascular findings in normotensive individuals is remarkably high.

The strong placebo effect of surgery7 has been documented in a variety of settings, including angina pectoris, where sham ligation of the internal mammary artery resulted in a striking improvement in clinical signs and symptoms.8 On the other hand, we are well aware of the important ethical concerns revolving around the use of sham surgery in clinical research.9 Fortunately, this means that the question as to whether or not surgical decompression of the brain stem should be considered a therapeutic option in patients with severe hypertension is unlikely to be resolved soon.


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