premise that preoperative hypertension *is* a useful predictor of post-CEA hypertension.

The 1970 retrospective study of 27 patients quoted by the authors, found no correlation between mean preoperative blood pressure (BP) and the development of post-operative hypertension.[2] Similarly, Bove et al in a prospective study of 100 CEAs, did not find a correlation between mean preoperative BP and post-operative hypertension.[3] Both these studies defined post-operative hypertension as a rise in systolic BP of greater than 15 mmHg[2] or 40 mmHg[3] above preoperative levels. This definition, therefore, would overlook patients who were hypertensive preoperatively and who remained hypertensive post-operatively but did not develop a further rise in BP.

In contrast, several more recent studies have demonstrated a strong correlation between preoperative hypertension and the development of post-CEA hypertension.[4-8] Towne and Bernhard, retrospectively reviewed 253 CEAs and found that 79.6% of the patients who developed severe post-operative hypertension (defined as systolic blood pressure (SBP) greater than 200 mmHg) were hypertensive preoperatively.[4] Of these patients, 16.3% had controlled preoperative hypertension (SBP < 150 mmHg on medications) and 63.2% had uncontrolled preoperative hypertension (SBP > 150 mmHG with or without medications). Similarly, in a retrospective study of 166 CEAs, Asiddao et al reported that severe post-operative hypertension (BP > 200/110 mmHg) occurred more frequently in patients with poorly controlled preoperative hypertension (BP > 170/95 mmHg with or without medications) compared to those with controlled hypertension or normal blood pressure (52%, 35% and 17% respectively).[5] Overall, 78% of the patients in this study who developed severe post-operative hypertension had preoperative hypertension. Similar results were found in uncontrolled hypertensives in the retrospective study performed by Corson et al.[6]

Skudlarick and Mooring prospectively studies 41 patients undergoing CEA.[7] All hypertensive patients in this series were controlled on medications preoperatively and post-operative systolic BP was pharmacologically maintained between 100-150 mmHg. These authors found that 73% of the patients who required post-operative intervention for hypertension (SBP > 150 mmHg) were controlled hypertensives preoperatively. Similarly, Cafferata et al prospectively studied post-operative blood pressure control in 90 CEA procedures using a carotid nerve-sparing technique.[8] Although the incidence of post-CEA hypertension was low in this series (11%), these authors also found that patients with uncontrolled preoperative hypertension (BP < 140/90 with or without medications) were more likely to develop post-operative hypertension compared to controlled hypertensives and normotensives (24%, 14% and 5% respectively).

The statement by Shuaib et al suggesting that the development of post-CEA hypertension cannot be predicted on the basis of preoperative BP is, therefore, misleading.[1] This statement implies that there is not benefit associated with achieving preoperative blood pressure control. We believe that the bulk of available evidence supports the premise that patients with preoperative hypertension, particularly those with poorly or uncontrolled hypertension, are at substantially higher risk of developing post-CEA hypertension. Since patients who develop post-CEA hypertension are at higher risk for developing post-operative neurological complications adequate preoperative control of blood pressure is desirable.[1-5]

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REPLY FROM AUTHOR

It is a pleasure to respond to the letter of Drs Gelb and Herrick. They provide convincing evidence from three retrospective and two prospective studies that post-operative hypertension after carotid endarterectomy (CEA) can be predicted if patients have pre-operative hypertension. At the time of writing of our case report, we had conducted a literature search of articles on post-CEA "hemorrhage" and thus did not get access to the important literature on post-CEA "hypertension". We had based the statement regarding the unpredictability of preoperative hypertension as a predictor of post-CEA hypertension on the study of Lehv et al. As outlined in the letter of Gelb and Herrick, this study had shown no relationship between the mean pre-operative blood pressure and the development of post-CEA hypertension.

The clinical evidence summarized by Drs Gelb and Herrick shows that, in fact, a close relationship does exist between preoperative blood pressure and the development of post-CEA hypertension. We believe this information may have relevance to the development of post-CEA cerebral hemorrhage. in our case report, we postulated that the presence of long standing (and often poorly controlled) hypertension, leading to the development of small micro-aneurysms in the brain, may be a risk factor for the development of post-CEA cerebral hemorrhage. The sudden increase in blood pressure in the immediate post-CEA period may then lead to rupture of the microaneurysms and thus cerebral hemorrhage. Our review of the literature, at the time of writing, showed no reports where the presence of

pre-operative hypertension was considered a risk factor for the development of post-CEA cerebral hemorrhage. With the information summarized by Drs Gleb and Herrick, and the case-report we published, we hope the presence of uncontrolled hypertension will become recognized as an important risk factor for development of post-CEA cerebral hemorrhage.

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Erratum

The authors of the Letter to the Editor entitled "The Effect of Bile Duct Ligation-Induced Liver Damage on the Blood-brain Barrier" (Can J Neurol Sci 1989; 17: 451-452) should be listed as follows: P.A. Stewart, C.R. Farrell, J.A. Holash, D. Elliot (Department of Anatomy, University of Toronto), J. Neiman, L. Resch, P.L. Carlen (Playfair Neuroscience Institute, University of Toronto).

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