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## DISCUSSION

DR. EDWARD A. STEMMER (Long Beach): To date, we have performed 109 carotid endarterectomies, considerably fewer than Dr. Thompson. Like the authors, we have performed a sizable number (15% of the total) on patients with asymptomatic bruits associated with significant arterial stenosis. Usually, this operation was performed as a preventive measure prior to carrying out major vascular procedures in the same patient.

As the authors have stated, nonoperative treatment has little to offer these patients and we believe they are best treated with endarterectomy.

[Slide] This shows our results classified according to the technic of operation. The most significant observation was that the use of vasopressors to artificially elevate the blood pressure was distinctly detrimental to the outcome of operation. It resulted in high mortality and a high incidence of postoperative stroke.

We regularly employ hypercarbia which is carefully monitored to maintain a  $P_{CO_2}$  between 60 and 80 mm. Hg which is monitored during operation. We are convinced that hypercarbia is beneficial and it did improve our results.

Although it is probably true that most patients can be operated upon safely without a shunt, we agree with the authors that use of an internal shunt adds to the safety of the procedure in a significant number of patients. The advantages of a shunt certainly outweigh its disadvantages as our data indicates when we use  $CO_2$  with the shunt. The 1.4% mortality represents a single patient who died of myocardial infarction 10 days postoperatively. We had no neurologic deficits.

With long-term follow-up, 72% of our patients with transient episodes were either cured or markedly improved. The two patients indicated in parentheses died of unrelated causes long after the carotid endarterectomy. By contrast, only 30% of those with fixed neurological deficits preoperatively were significantly improved. No patient who had undergone a so-called prophylactic endarterectomy suffered any adverse effects from operation. Most of our problems have occurred in patients with multiple sites of cerebrovascular disease. Sixty-six per cent of our patients had more than one vessel involved.

[Slide] This summarizes our usual management of patients undergoing carotid endarterec-

tomy. We employ carefully monitored hypercarbia in the operating room with general anesthesia (preferably with penthrane to eliminate arrhythmias that can occur during hypercarbia). We use an internal shunt when the internal carotid artery is patent. We use systemic heparinization while the shunt is in place and we do not use vasopressors.

Finally, I would like to ask Dr. Thompson if he has long-term follow-up arteriography on those patients in whom a completely occluded internal carotid was reopened. We have also attempted to do this and have been able to open up about a fourth of the internal carotid arteries, but we have a serious question about how long they really stay open.

DR. J. ALEX HALLER (Baltimore): I wish to ask one question of Dr. Thompson.

The most difficult group to evaluate are those with the asymptomatic carotid bruits. If I understood your figures correctly, Dr. Thompson, there were 37 patients with bruits alone who subsequently developed difficulty and of this group, ten of the 37 developed frank strokes.

If I remember correctly, patients who develop strokes fit in the categories of approximately one-fourth having carotid disease and three-fourths having intracranial cerebral disease.

I this is true also in your group, this would mean that perhaps two or three of the patients who developed frank strokes had this on the basis of carotid occlusion. What I would like to ask is whether the autopsies on these patients confirm the same incidence of intracranial disease as in the general population? One of the problems in this type of palliative operation is whether there is such a thing as "preventive palliation." Therefore, it would be of considerable consequence to know exactly where the occlusion occurred in those patients with asymptomatic bruits whom you have carefully followed and who subsequently developed fatal strokes.

DR. CHARLES G. ROB (Rochester): I would wish to raise the question again of the asymptomatic bruit. Essentially, operations on these patients are "entirely prophylactic" and one must remember that the bruit comes and goes. It is heard one day and then perhaps not on the next day.

The next point is that we are still worried about operating on the asymptomatic bruit, but essentially our definition of asymptomatic has changed.

We have relaxed it considerably and we now feel that a symptomatic bruit is a patient with mild early forgetfulness, or early so-called senile mental changes and I believe that when you look carefully at these patients with so-called asymptomatic bruits as we do, extremely few of them are in fact asymptomatic.

DR. JESSE E. THOMPSON (Closing): I am glad to see Dr. Stemmer's results parallel our own. Hypercarbia is a satisfactory method of cerebral protection during operation but all the published statistics to date show that it is not any better than the use of a shunt; in fact, a shunt is a bit more effective than hypercarbia alone. The use of hypercarbia plus a shunt takes advantage of both these modalities of protection.

One should not use hypercarbia unless the patient can be monitored adequately in the operating room. The necessary monitoring equipment should be available in every operating room of every hospital in which the operation is performed. For this reason also we believe a shunt is probably the simplest and most practical way of achieving satisfactory cerebral protection during operation.

Dr. Stemmer has asked about total carotid occlusion. This is a vexing problem and we have not done routine arteriography postoperatively on all of the patients; it was performed on those of whom we were concerned at the time of operation since the back flow was poor, although not zero. In every instance of this sort in which we did arteriography the artery was found to be occluded.

There are two factors about total occlusions; namely, time and the nature of the stroke. Probably the nature of the stroke itself is most im-

portant in determining operability in this group of patients. There are not very many patients with total occlusions who are candidates for operation.

The remarks of Dr. Haller and Dr. Rob deal with the asymptomatic bruit and this, too, is a controversial problem. I think one has to be careful in his recommendations lest we create a monster in this regard.

First of all, in answer to Dr. Haller's questions, I would just like to point out that 74% of patients with cerebrovascular insufficiency syndromes do have at least one operable lesion in the extracranial vasculature. The intracranial vessels are often free of demonstrable disease.

The patients in our group have all had carotid bruits and many have been followed by arteriography, although not all. After they had frank stroke, they were studied and we found these patients had totally occluded carotid arteries in the neck. Most of the problem here relates to the extracranial lesion and not to an intracranial occlusion.

It is important to mention the recent work of Dr. Javid and his group in Chicago on the natural history of the atheroma. They have studied patients serially with arteriograms who had lesions in the neck. They found that only 38% of the lesions remain unchanged in size; 62% of these lesions changed as they followed them along and interestingly enough, 34% of these increased in size at a rate greater than 25% per year, whereas the remaining lesions, although increasing, did not increase that fast.

These studies of the natural history of atheromas coincide with our own clinical observations in this regard.

As Dr. Rob has pointed out, perhaps we are finding more of the patients with bruits who are not entirely asymptomatic and this gives us reassurance about operating on them.