scarring and interstitial fibrosis in the subendocardial regions of the anterior and apical left ventricle, without significant cellular infiltrate. The pathologic findings were thought to be consistent with chronic ischemia in the distribution of the terminal circulation (left anterior descending artery territory). The authors of this earlier paper concluded that this portion of the heart had a combined ischemic blood supply coupled with an excessive demand on the limited circulation. Perhaps a similar pathophysiology contributed to the chest pain symptoms reported in their 59-year-old female patient.

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Reply

We thank Dr. Symanski for his interest in our article describing a rare coronary anomaly (1). We are grateful that he brings to our attention the post-mortem results of Pete Maravich's heart (2). We agree that this anomaly, unbeknownst to us, has been previously described.

It was interesting to read the post-mortem examination that described a dilated heart weighing 650 g and having a left ventricular wall thickness of 15 mm. Equally interesting were the findings of myocardial fibrosis consistent with chronic ischemia. Dr. Symanski questions whether a similar pathophysiology may be contributing to our patient's chest pain.

At follow-up, our patient's atypical chest pain had completely resolved. On further review, our patient also had normal left ventricular volumes, mass, and ejection fraction. Cocker et al. (3) recently described the finding of myocardial fibrosis in elite athletes using magnetic resonance imaging. We wonder whether Pete Maravich's history as an elite athlete in combination with this rare coronary anomaly partially explains his post-mortem findings.

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Vertebral Artery Stenting
Not Quite Ready for Prime Time!

Atherosclerotic vertebral artery (VA) stenosis is a significant cause of vertebrobasilar ischemia. However, vertebral artery stenting (VAS) has not received the detailed scientific study that has been accorded to carotid artery stenting (CAS).

In their series, Jenkins et al. (1) show excellent results. These results add to the growing body of nonrandomized studies that demonstrate the feasibility and relative safety of VAS (2). Based on their outcomes, the authors recommend a more liberal use of VAS. However, several issues remain unresolved that beg for a more cautious approach.

No recent study of sufficient size has investigated the impact of optimal medical regimen on the natural history of VA disease or compared it with VAS (3). Further, there are several unresolved issues regarding optimal endovascular strategy. Bilateral VA stenosis presents a clinical challenge. Unlike anterior circulation ischemia, vertebrobasilar ischemia symptoms are difficult to lateralize to one side. It is not known whether unilateral VAS will resolve the symptoms or whether bilateral VAS is indicated. The authors report that 54.3% patients had bilateral VA disease, although only 6.3% of the subjects received bilateral stents. It is unclear how the stented side was chosen and whether symptoms resolved completely.

Subclavian artery stenosis without coexistent VA stenosis can cause vertebrobasilar ischemia (4). In the current study, 29.2% of the subjects had concurrent subclavian artery disease. It will be useful to know whether the subclavian artery was also stented concomitantly.

The present study did not use distal embolic protection, although this is the standard in CAS. This is an important issue in VAS with only limited data available (5).

Another important issue is restenosis. Unlike CAS, which has a low risk of restenosis, VAS has a significantly higher restenosis rate (6,7). Little information is available regarding the use of drug-eluting stents, although initial reports indicate a lower restenosis rate (8).

Jenkins et al. (1) demonstrate that VAS is relatively safe and feasible. However, before more widespread use, VAS should undergo the same meticulous investigation as CAS has been accorded. This will involve a direct comparison with optimal medical therapy and use of current endovascular standards (distal embolic protection and perhaps drug-eluting stents).

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Reply

We thank Dr. Gupta for his comments on our paper (1) describing our experience treating symptomatic patients in whom medical therapy had failed with vertebral artery stenting.

We are in complete agreement that this field will benefit from more data and larger clinical trials. Despite the fact that both vertebral arteries supply a single basilar artery, conventional wisdom has been that a single vertebral artery is adequate for posterior circulation perfusion. Clearly there are infrequent clinical exceptions to this rule. Clinical practice has been to preserve the dominant vertebral artery whenever possible.

In our series of patients, coexistent subclavian artery disease was demonstrated in 29.2% of patients, one-half of whom were treated concurrently with a vertebral stent and the other half of whom were hemodynamically insignificant.

There are several reasons for a lack of clinical trial data. One is the unwillingness of providers to pay for vertebral artery stenting or to reimburse for care performed as part of an investigational trial that has benefited carotid stenting significantly. The second issue is that the vertebral arteries are well treated with off-label balloons and stents, which diminish the enthusiasm of medical device manufacturers to support additional investigations.

As mentioned by Dr. Gupta, posterior circulation symptoms are more difficult to localize than anterior circulation symptoms. Patients with medically refractory vertebral basilar symptoms carry a 5-year stroke rate of 30%, and if the disease is intracranial in location, a 1-year stroke rate of 50% far exceeds the risk of anterior circulation disease (2). Posterior circulation strokes account for a minority of ischemic strokes (20% to 25%) (3), making it more difficult to enroll a sufficient number of patients in a clinical trial.

The effect of drug-eluting stents on vertebral artery restenosis and the utility of emboli protection devices have not been studied in the vertebral circulation. Going forward, these are important questions that will need to be answered. Until then, we must do our best to treat patients using the best available data.

The encouraging results of our current series of vertebral stenting, along with those of other single-center series (4,5), strongly support a strategy of percutaneous catheter–based therapy for medically refractory symptomatic patients with posterior circulation ischemia related to vertebral artery disease.

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