CORONARY SPASM AND ORGANIC STENOSIS

MacAlpin found that in five of six cases of spontaneous or drug-induced dynamic coronary occlusion that caused attacks of variant angina, the obstruction occurred at the site of a well defined organic stenosis,1 and also that in 90 percent of cases spasm occurred at the site of an organic lesion.2 The existence of spontaneous coronary spasm or constriction is no longer doubted by anyone. During the last 10 years or so I have treated patients who manifested a spasm of accommodation with various concomitant symptoms of imbalance in the autonomic nervous system. Even young people who are otherwise completely healthy often have very severe pains in the region of the heart. I have found that when the spasm is released, such pains disappear. This observation has obliged me to ponder the possibility that severe constriction of lung duration could disturb nutrition of the blood vessels and that the resulting tissue damage could produce the preconditions for sclerotic change.

I am prompted to ask whether the organic lesion itself may not be the consequence of a primary spasm at the same site. In other words, may not the spasm be the etiologic cause of (coronary) sclerosis? The next stage will be to ask "why are some people's coronary arteries so apt to vasoconstrict?" 3

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References

REPLY

Having never previously heard of spasm of accommodation, I looked it up and found it described as attacks of convergence, accommodation and miosis of the eyes resulting in faulty vision. It is apparently an uncommon functional disturbance usually occurring in psychoneurotic persons.1 That Vilkari has encountered severe chest pains in some of his patients in association with spasm of accommodation is interesting. Electrocardiograms and esophageal motility studies on these patients during attacks of chest pain would be helpful in assessing the significance of the chest pain. I cannot find a reasonable explanation to connect spasm of the ciliary body and the coronary arteries except for an imbalance of autonomic nervous system function, which is apparently common in patients with spasm of accommodation but which I have not been able to detect in any of my patients with proved coronary spasm. To the best of my knowledge, none of the 70 patients with variant angina that I have seen have had a history of spasm ofaccommodation, although they were not asked about that specific entity.

The possibility that coronary spasm could injure the vessel wall and hence might itself be a cause of organic coronary artery disease was hinted at by Friedrich Kreyeig in 1816,1 and this hypothesis was more recently presented by Gertz et al. and by Marzilli and co-workers.2,3 The merits of this hypothesis were discussed by Lown and DeSilva.4 It is certainly not unreasonable to suppose that if coronary spasm occurs at the site of a preexisting pliable atheromatous lesion, the plaque could be disrupted and subintimal hemorrhage or endothelial rupture result—both of which could initiate acute coronary occlusion. It is known that severe spasm of cerebral arteries in human beings and of coronary arteries in dogs can result in damage to the vascular endothelium and smooth muscle. It is unknown whether spasm of a normal human coronary artery can also result in damage to the vessel wall, and whether this is in reality one cause of organic coronary artery disease and an explanation for the commonly observed relation of coronary spasm with sites of organic arterial stenosis.

Little is known of the normal determinants of vasomotion of large coronary arteries. Why some people's coronary arteries are so prone to vasoconstriction which can produce myocardial ischemia is still unknown.

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References

INABILITY OF A CHEST PAIN HISTORY QUESTIONNAIRE TO PREDICT CORONARY DISEASE IN THE EXERCISE LABORATORY

We have been using a Bayesian approach in our evaluation of the exercise test for the diagnosis of coronary artery disease. In an attempt to standardize our typing of chest pain, we
Table I: Questionnaire Scores for the Angiographic Groups

<table>
<thead>
<tr>
<th>Angiographic Score</th>
<th>Questionnaire Score</th>
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<tbody>
<tr>
<td>0–3 (16)</td>
<td>8.4 ± 9.2</td>
</tr>
<tr>
<td>2–5 (7)</td>
<td>9.9 ± 4.4</td>
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<tr>
<td>&gt;6 (23)</td>
<td>0.7 ± 10.0</td>
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</tbody>
</table>

Figures in parentheses indicate number of patients.

Table II: Pretest Likelihoods for Coronary Disease for the Angiographic Groups

<table>
<thead>
<tr>
<th>Angiographic Score</th>
<th>Pretest Likelihood for Disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–3 (16)</td>
<td>40.6 ± 92.3</td>
</tr>
<tr>
<td>2–5 (7)</td>
<td>48.3 ± 19.4</td>
</tr>
<tr>
<td>&gt;6 (23)</td>
<td>62.5 ± 27.9</td>
</tr>
</tbody>
</table>

Figures in parentheses indicate number of patients.

References

PULMONARY VALVE ECHOGRAM

The article by Starling et al. validated our earlier report that the presence of a B shoulder in the tricuspid valve echocardiogram is associated with elevation of right ventricular end-diastolic pressure. This observation is of particular importance in the assessment of total right heart function and in the interpretation of the pulmonary valve echo. The pulmonary valve echo will show loss of the A dip in the presence of pulmonary hypertension and sinus rhythm. However, the depth of the pulmonary valve A dip is not dependent on any absolute level of pulmonary arterial pressure, but probably reflects the relative right ventricular to pulmonary arterial end-diastolic gradient. A significant pulmonary valve A dip should not occur in the presence of significant pulmonary hypertension unless the right ventricular end-diastolic pres-