in a stable phase of their disease and who have survived the acute event. They also caution against extrapolation of their data in determining prognosis during the acute phase of myocardial infarction or unstable angina phase.

Combining the data from tables 3, 4 and 7 brings up some interesting points. Examining table 3, 22 of 32 patients with single vessel disease had grade 2, 3 or 4 arteriographic score. Table 4 indicates that only 36 patients in the entire series had arteriographic scores between 1 and 4. Therefore, this group of 36 patients is made up of the above mentioned 22 patients with single vessel disease and 14 others. The features of this group are interesting in that only 28% had typical angina and 22% had no pain at all. The severity of pain is detailed in table 7. Of 28 patients with scores of 1–4, 20 had either no pain or pain only with severe exertion. (The “angina” in table 7 includes typical, atypical or uncertain forms and has been called “pain” in table 6.)

The patients with scores 1–4 in the series, therefore, not only are predominantly single vessel disease patients, but also have low incidence of typical or severe angina. It would be important to know how many single vessel disease patients had severe classical angina, because most of them apparently did not have it. It is pertinent to add that of the original 350 patients selected, 40 were excluded because they underwent surgery. If the indication for surgery was the presence of severe symptoms, then 40 of the most symptomatic patients were excluded from the entire study.

It is therefore better if the conclusion about single vessel disease is modified to indicate that the prognosis is good if the patients are in a stable phase, have already survived an event, do not have severe angina and pain is either absent or of mild nature and atypical character.

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The author replies:
To the Editor:

Dr. Mathur’s comments and suggestions are appreciated. His conclusions may be correct, but we do not believe that it is possible to be certain of these conclusions from our study.

Of the 22 patients with single vessel disease without total occlusion of that vessel (thus a score of 2, 3, or 4 in that vessel) only 17 had a total score of 1 to 4. The other five had trivial disease (score 1) in one or both of the other vessels.

Thus of the 36 patients in table 4 with a total score of 1 to 4 only 17 had single vessel disease. Review of our patients’ charts indicate that seven of these 17 patients (41%) had typical angina pectoris. Only one of these 17 patients had no pain at all. It is wrong to combine the columns labelled “Not angina” and “No pain” (see definitions, table 1).

The severity of pain (or limitations experienced by the patient) is known in only 13 of the 17 patients with single vessel disease and a total score of 1 to 4. Two of these 13 patients had pain at rest and two had pain on minimal exertion.

The frequency of typical angina pectoris and severely limiting chest pain is less in patients with single vessel disease than in patients with severe obstructive disease in two or three coronary arteries (tables 4 and 7).

It is probably true that patients with single vessel disease and a history of typical angina pectoris and/or severe limitations due to chest pain have a worse prognosis than patients with atypical angina or only mildly limiting chest pain, but it is not possible to be certain of this from the data presented in our study.

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STI in COPD

To the Editor:

We disagree with the conclusion reached in the recent article by Hooper and Whitcomb entitled “Systolic Time Intervals (STI) in Chronic Obstructive Pulmonary Disease (COPD).”1 Although these authors concluded that “the abnormalities of STI demonstrated in these patients are characteristic of left ventricular dysfunction and indicate that subclinical left ventricular dysfunction is frequently present in patients with moderate obstructive lung disease” they performed no hemodynamic studies to confirm their hypothesis. We believe that another explanation is not only possible but probable for the observed abnormalities in STI. Similar alterations in STI occur in patients with acute and chronic right ventricular failure secondary to pulmonary embolism and mitral stenosis where left ventricular function may be normal.2 Furthermore, abnormal STI are present in patients with COPD who have normal pulmonary artery wedge pressures and normal echocardiographic indices of left ventricular function.3 In these patients LV stroke volume is reduced and this observation is the likely explanation for the decreased left ventricular ejection times recorded in such individuals.4 Also, left ventricular filling pressures were reduced in most of these patients which may have contributed to the prolonged pre-ejection period index.5 Thus, our data support the