LETTERS TO THE EDITOR

Differential Effect of Thrombolytic Therapy

Ritchie et al. (1) recently examined limitation of infarct size, estimated by thallium-201 tomography, after thrombolytic therapy with intravenous streptokinase in a subgroup of 207 patients participating in the Western Washington intravenous streptokinase trial. The authors report that the greatest benefit of thrombolytic therapy occurred in patients with anterior infarction who were treated within 3 h after the onset of symptoms. There was little benefit in patients with inferior infarction or in patients with anterior infarction admitted after 3 h. These findings are in agreement with the subgroup analysis of the trial on intracoronary streptokinase, conducted by the Netherlands Interuniversity Cardiology Institute (2). In that analysis the beneficial effects of thrombolytic therapy on infarct size (estimated from myocardial enzyme release), global left ventricular function and survival were related to two factors: the delay between onset of symptoms and therapy and the extent of myocardial ischemia estimated from the total amount of ST segment elevation in the admission electrocardiogram (ECG). When the admission ECG was taken into account, infarct location was not related to the effect of thrombolytic therapy. Generally, anterior infarctions are larger than inferior wall infarctions. The effect of thrombolytic therapy appeared to be related to the size of the ischemic area and not as much to the site of the infarction. It would be of great interest to know whether similar effects were observed in the Western Washington intravenous streptokinase trial.

It is now evident that thrombolytic therapy is useful in certain subgroups of patients with acute myocardial infarction and certainly not in all types of infarction. More studies, such as the report by Ritchie et al. (1), are needed to further delineate those patients who do and those who do not benefit from thrombolytic therapy. The use of threshold values such as anterior infarction within 3 h after the onset of symptoms may appeal to clinicians because of their simplicity. However, such selection criteria for thrombolytic therapy in our opinion do not reflect the more complicated clinical reality. It may be more appropriate to apply a set of inclusion criteria combining the delay between onset of symptoms and the total amount of ST segment elevation. For example, from the data in the trial by the Netherlands Interuniversity Cardiology Institute a so-called rule of four was developed. According to that rule, thrombolytic therapy is warranted in patients admitted within 1 h after the onset of symptoms who exhibit a sum of ST segment elevation in all 12 leads of 0.4 mV. In patients admitted between 1 and 2 h, the ST segment elevation should be 8 mm, 0.8 mV; between 2 and 3 h, 0.12 mV and between 3 and 4 h, 0.16 mV.

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References

1. Ritchie JL, Cerquiera M, Maynard C, et al. Ventricular function and infarct size: the Western Washington Intravenous Streptokinase in Myocardial Infarction Trial. J Am Coll Cardiol 1988;11:689–97.
2. Vermeer F, Simoons ML, Bär FW, et al. Which patients benefit most from early thrombolytic therapy with intracoronary streptokinase? Circulation 1986;74:1379–89.

Reply

We did not collect ST segment data and cannot comment on their utility, and they have not been reported in most other trials. We thus cannot ascertain from our data whether infarct size alone, or possibly other contributing coronary anatomic/physiologic features, can explain the differences between the anterior and inferior infarction groups. We agree that further information is needed.

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Dipyridamole-Induced ST Segment Depression by Collateral Steal?

Chambers and Brown (1) propose as determinants of dipyridamole-induced ST segment depression an increased rate-pressure product and the presence of "good" collateral vessels at coronary angiography. They found that among patients with (visually estimated!) high grade stenosis (≥95% luminal diameter narrowing), dipyridamole-induced ST segment depression was more common in patients with than in patients without good collateral vessels. The authors suggest that the collateral vessels are therefore responsible for the observed ST segment depression. In my view, ST segment depression after dipyridamole infusion in the presence of collateral vessels indicates that the stenosis at angiography is also *physiologically* significant. Therefore, the relation between collateral vessels and ST segment depression must be an indirect one. Furthermore, it is difficult to judge the functional significance of collateral vessels by the coronary arteriogram and their appearance may vary considerably in time and among various injections of contrast medium in the same patient.

For their methods of quantitative analysis, the authors refer to the excellent article of Wackers et al. (2), which deals with *exercise* thallium-201 testing. Did the authors use test-specific normal profiles for dipyridamole thallium-201 testing derived from subjects with low likelihood of coronary artery disease?

References

1. Chambers CE, Brown KA. Dipyridamole-induced ST segment depression during thallium-201 imaging in patients with coronary artery disease: angiographic and hemodynamic determinants. J Am Coll Cardiol 1988;12:37–41.

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