estimating myocardial infarct size. Circulation 65: 342, 1982

The authors reply:
To the Editor:
It is encouraging to learn that other groups are interested in performing the QRS scoring system and attempting to determine the correlation between QRS score and other aspects of their patients with acute myocardial infarcts. It is reassuring to learn that significant correlations have been obtained between QRS score and enzyme-estimated infarct size and predischarge exercise performance as well as with the variable we evaluated, left ventricular ejection fraction. Since any method for estimating infarct size has some limitations, it is important that several methods for estimating size be available for optimal evaluation of the effect of any therapeutic intervention aimed at limiting infarct size. Our group is currently completing a retrospective evaluation of the relationship between enzyme-estimated and QRS-estimated size of initial myocardial infarcts. We are also in the process of determining the relationship between the quantity of initial ST-segment elevation and both the final QRS score and the incidence of serious complications in hospital.

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Irreversible Coronary Occlusion and Ergonovine
To the Editor:
We read with great interest the report of irreversible coronary occlusion related to administration of ergonovine by Crevey and co-workers (Circulation 64: 853, 1981). The patient described had multivessel coronary artery disease and sustained a total occlusion of the right coronary artery after a small dose of ergonovine (0.05 mg). This occlusion could not be reversed by large quantities of sublingual nitroglycerin and an acute transmural myocardial infarction resulted. They suggested that the ergonovine caused irreversible spasm. Review of figure 1 in their article suggests an alternative explanation. This figure shows a large intraluminal filling defect extending distally from the severe proximal stenosis. This closely resembles the intracoronary thrombi described by Ganz et al.1 Rentrop et al.2 and others in transmural myocardial infarction. Vetrovec et al.3 described similar intracoronary filling defects in patients with intermediate syndrome and nontransmural infarction. We have seen identical lesions in these syndromes and documented their thrombotic nature by lysis with streptokinase.4 It is possible that coronary occlusion in this instance was related to thrombosis alone, or was superimposed on spasm, rather than to irreversible spasm. In addition to treating the patient with apparent irreversible spasm with intracoronary nitroglycerin, consideration could also be given to intracoronary streptokinase therapy.

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References

The authors reply:
To the Editor:
We also noted the angiographic similarities between our patient’s coronary lesion and recently published examples of lesions thought to contain thrombus. This observation suggests a link between the processes of coronary vasospasm and thrombosis. Vasospasm, especially that which occurs adjacent to fixed atherosclerotic lesions, may reduce coronary blood flow enough to promote local platelet aggregation and thrombus formation. Recurrent vasospasm could lead to progressive thrombotic accretions and finally to complete thrombotic occlusion. In our patient, the sequence of events strongly suggests that ergonovine-induced active vasoconstruction acutely precipitated the complete obstruction. Ergonovine may have temporarily compressed naturally occurring pathogenic phenomena. We agree that in similar circumstances fibrinolytic therapy may be a useful addition to efforts directed against vasospasm.

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