drop in mean aortic pressure from 92 to 78 mmHg, accompanied by a corresponding tachycardia (97-104 bt/min) within 10 min of the IV bolus. Since nine of their 12 patients were in the early stages of acute myocardial infarction, a combination of hypotension and tachycardia could provoke further ischemia. We therefore plead for caution in the use of beta, agonists and, particularly, isoxsuprine in acute pulmonary edema because of its potential harmful effects on circulatory dynamics and, possibly, myocardial function.

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REFERENCES


To the Editor:

In their report, Fallen and Rambhar found it difficult to implicate isoxsuprine alone as the cause of pulmonary edema. They found it likely that their seven patients suffered from “hemodynamic and/or volume-loaded pulmonary edema” due to the combination of volume expansion (under scoring our results) and isoxsuprine, with or without myocardial depression. Their patients received 2.1 to 5.7 L (mean, 3.57 L) of intravenous fluids in the 24 hours preceding the onset of pulmonary edema. This, they claimed, is 13 percent higher than average. Their patients received 633 to 3,912 mg (mean, 1,834 mg) of isoxsuprine, as compared to the 10 mg we gave in our study.

During vasodilator therapy of congestive heart failure complicating acute myocardial infarction, the intraventricular pressure and ventricular volume decreases, thus the overall myocardial oxygen demand tends to decrease, based on La Place’s principle. Some reduction in systemic arterial pressure usually occurs during vasodilator therapy, but if the drugs used are titrated properly, prolonged and significant hypotension can be avoided while obtaining the desirable effects.

It would be ill-advised to advise patients in pulmonary edema with parenteral fluids, if only to prevent a drop in systemic arterial pressure. These patients are already on a much flatter Frank-Starling curve, so that any increase in preload would add to the heart’s workload and myocardial oxygen consumption. This may lead to further myocardial ischemia in those patients with acute myocardial infarction.

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To the Editor:

I read with interest the case report by Thomas J. Quinn et al (Chest 1984; 86:781-82) in which the authors drew attention to the phenomenon of widened QRS complexes, simulating ventricular tachycardia, which occurred in their patient with extensive acute myocardial infarction. The authors claim that extensive literature search of the English language failed to reveal a similar case as theirs. However, the electrocardiographic features presented by the authors have been previously reported by Krikelis and me,14 and extensive animal laboratory work has focused on their electrophysiological mechanisms.14 Similar changes have been noted at the onset of variant angina. In a recently reported group of 16 patients with episodes of variant angina,1 monophasic QRS complexes resembling ventricular tachycardia were noted transiently in three patients. I believe that this is not an uncommon phenomenon, probably often misinterpreted by physicians.

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REFERENCES


To the Editor:

Dr. Madias correctly pointed out that ischemia can lead not only to marked ST segment elevation, but also to alterations in R wave amplitude and widening of the QRS complex. After reviewing Dr. Madias’ references, and despite QRS prolongation noted in multiple leads on multiple patients, the use of a 12-lead electrocardiogram enabled the clinician to identify a normal QRS duration in at least two of the 12 leads and identify the underlying rhythm.

Our brief article, to the best of our knowledge, is the first noting QRS prolongation in all 12 leads simultaneously. This led to the mistaken diagnosis of ventricular tachycardia, and therapeutic intervention was made on the basis of that diagnosis. I agree that this is probably not an uncommon phenomenon and is often misinterpreted by physicians.

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Communications to the Editor