The factors suggested by Dr. Conti that influence the outcome of the patients with ST-segment myocardial infarction are all valid. Unfortunately, they are not adequately addressed in the trials where influence of DTB time on outcomes has been studied.

Our goal in treating myocardial infarction should be both to decrease mortality and preserve left ventricular function (prevent the development of heart failure). Because the time from symptom onset to reperfusion has been shown to decrease infarct size, it is only natural now to target it as representative of the total ischemic time. We should start measuring it routinely.

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Out-of-Hospital Cardiac Arrest Patients With ST-Segment Elevation on Electrocardiogram

Don't Rush Patients for Emergent Percutaneous Coronary Intervention in the Era of Aggressive Door-to-Balloon Time

As an institution that has a great interest in and has studied cardiac arrest and ST-segment elevation myocardial infarction (STEMI) (1), we read with great interest and agree with Dr. Kern (2) that we should provide "operators and medical centers the opportunity to do what is best for the individual STEMI patient, without fear of unfair inflation of their overall reported mortality figures."

We agree that "such a change cannot come too soon" for those cardiac arrest STEMI victims comatose on arrival to the hospital. However, we now fear that with aggressive door-to-balloon-time initiatives and our prior report on STEMI and out of hospital cardiac arrest (OHCA), that operators are performing emergent percutaneous coronary intervention (PCI) too often in comatose patients when STEMI does not in fact truly exist.

A recent abstract from our institution by Abraham et al. (3), presented at the most recent American College of Cardiology Scientific Sessions, noted that aggressive catheterization was performed in patients with noncardiac causes of ST-segment elevation on electrocardiogram. Many of these patients had OHCA and the catheterization delayed the diagnosis and treatment of the primary etiology. Subsequent work-up showed that the cause of mortality was varied and included sepsis, hyperkalemia, intracranial hemorrhage, aortic dissection, left ventricular aneurysm, and pulmonary embolism.

We are now victims of our own success in that we had concluded in 2009 that "resuscitated patients with STEMI in the ED should be seriously considered for emergent revascularization regardless of neurologic status. These patients should be treated with the same urgency as patients with acute STEMI without cardiac arrest (1)."

We agree with Dr. Kern and advocate aggressive evaluation and treatment of comatose OHCA patients found to have diagnostic STEMI with emergent PCI and therapeutic hypothermia. However, we emphasize not to push every arrest patient to the catheterization laboratory without appropriate evaluation in the emergency department to ensure coronary obstruction as an etiology for the event. Not infrequently, these ST-segment changes are concurrent with another catastrophic event that leads to ischemic changes on the electrocardiogram.

Though, the mantra has always been "time is myocardium," a 5-min delay in door-to-balloon time to practice sound clinical medicine is unlikely to cause higher mortality and morbidity. However, delaying treatment of the true etiology of arrest by performing emergent catheterization may, in fact, do so.

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Reply

I appreciate the comments by Dr. Hosmane and colleagues on my paper (1), particularly their support that now is the time to provide "operators and medical centers the opportunity to do what is best for the individual ST-segment elevation myocardial infarction (STEMI) patient, without fear of unfair inflation of their overall reported mortality figures." This change in public reporting of outcomes data will require the main body of interventional cardiologists and their societal leaders to join together in championing this cause for the benefit of patients who suffer cardiac arrest. Reporting outcomes among the cardiac arrest population itself is warranted and needed, but such data should be separated from the general population undergoing percutaneous coronary intervention (PCI) to realistically compare apples to apples, not apples to oranges.

These clinical investigators, who recently published their experience supporting the performance of emergent coronary intervention post-cardiac arrest, now acknowledge their fear "that with aggressive door-to-balloon time initiatives and our prior report on STEMI and out-of-hospital cardiac arrest, that operators are performing emergent percutaneous coronary intervention too often in comatose patients when STEMI doesn't in fact truly exist." They express concern that not all ST-segment elevation indicate an acute myocardial infarction, and that coronary angiography might delay the true diagnosis and appropriate treatment. Theoretically this is possible, but the alternative diagnosis they note (sepsis, hyperkalemia, intracranial hemorrhage, aortic dissection, left ventricular aneurysm and pulmonary emboli), when severe enough to cause cardiac arrest are associated with very poor outcomes, with the possible exception of timely treatment for hyperkalemia. I believe the real issue is who post-resuscitation can truly benefit from emergent catheterization, can we prospectively identify them, and do our efforts help or harm them? Finding the cardiac arrest victim with an acutely occluded or unstable culprit lesion is the goal. We know that post-resuscitation ST-segment elevation is not definitive (2), with a 20% to 30% 'false negative' rate (3), and as noted by Dr. Hosmane and colleagues some degree of 'false positives' as well. That is why I argue to extend emergency coronary angiography to all successfully resuscitated with a likely cardiac etiology, regardless of their post-arrest electrocardiographic findings. I prefer to include some who ultimately do not have a culprit lesion found, in order not to miss those whose acute coronary lesion is only detected at emergent angiography. The literature suggests that approximately 50% of the successfully resuscitated without an obvious noncardiac cause of their arrest will have an acute culprit coronary lesion (3,4). One out of two is enough to convince me to perform emergent coronary angiography whenever someone is lucky enough to arrive at the hospital after being successfully resuscitated from out-of-hospital cardiac arrest.

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