In 1972 while traveling through Germany with my wife, we were involved in a multivehicle accident on the Autobahn. Although we were not injured, many people were and during the melee that followed the accident I was approached by a German resident in medicine who asked me to look at one of the people delayed by the accident. He was a rotund German man who was sweating profusely. I attempted to take a history, but even without knowing German the fact that he was popping nitroglycerin told me a great deal. Within seconds he had arrested and I found myself performing cardiopulmonary resuscitation in the middle of the Autobahn. An ambulance came and away we went with me still doing chest compression and the German resident providing respiratory support. Looking around the interior of the ambulance I saw an early version of the defibrillator but could not convince the German attendants to use it. I grabbed the thing and cranked it up to full power, applied it to his chest without the ceremony of any lubricant and shocked him from ventricular fibrillation into sinus rhythm. Only after arriving at the hospital did I discover that the defibrillator had been installed that morning and no one had been instructed in its use. On entering the hospital, the emergency physicians drew up a syringe of a compound unknown to me and injected it into his IV. I asked what it was they were giving the patient. The physician responded, “It is streptokinase and we give that for heart attacks.” It was indeed, in 1972, a homeopathic dose and was a therapy I had never heard of. The hospital where this occurred was in Gottingen. Ironically, this was the same hospital where some years later Peter Rentrop pioneered acute myocardial infarction intervention with wire perforation of the thrombus and intracoronary lytic therapy.

Much has happened since that curious first experience of mine, and acute myocardial infarction intervention has become the poster child for the most dramatic success in interventional cardiology. Among the important discoveries about thrombolytic therapy and primary angioplasty has been the understanding that prompt intervention is associated with improved outcomes. As evidence has mounted, hospitals began instituting measures to decrease the time it took to get the patient into the cath lab and open the artery. Guidelines on acute myocardial infarction have routinely recommended the briefest possible time for restoring flow, and initiatives to reduce door-to-balloon time have been promoted by the American College of Cardiology, the American Heart Association, the European Society of Cardiology, and almost every other organization dedicated to the treatment of myocardial infarction. In recent years, the door-to-balloon time has become a performance measure that is used to grade hospitals and, in some cases, influence reimbursement. Not surprisingly, when the consequences are so strong, hospitals have taken this very seriously and the stated goals on short door-to-balloon times have generally been achieved. In fact, so many hospitals approach 100% performance on this measure that consideration has been given to abandoning it as a performance measure.

Surely this success of standardizing protocols and achieving very brief door-to-balloon times has been a worthwhile endeavor. However, some patients are not well served. Speed at any cost is not always productive. Once, at morning rounds, I encountered a case of a patient admitted after an episode of chest pain and near syncope in which the electrocardiogram showed ST-segment elevation in the V1, V2, and V3 leads. A diagnosis of ST-segment elevation myocardial infarction was made and the patient was rapidly transported to the cath lab. A discussion ensued about which artery to inject first in order to recanalize the obstruction. The catheterization revealed total occlusion of the circumflex artery and intervention was begun without wasting time on left ventriculography.
or other measures. Opening the circumflex turned out to be a bit difficult but it was finally accomplished and the patient was returned to the coronary care unit. Later that day, blood pressure began to drop precipitously and echocardiography revealed the true problem. The left ventricle was hypercontractile, the septum was displaced toward the left ventricle, the right ventricle was markedly dilated, and pulmonary artery pressures were elevated. Computed tomography angiography showed the massive pulmonary emboli that had been the etiology of the original problem. It has occurred too many of us that in the rush to achieve optimal door-to-balloon times, we have skipped some important cognitive procedures along the way. In retrospect, the electrocardiogram should have given a clue to the possibility of pulmonary embolus. A left ventricular end diastolic pressure of 5, which followed the recanalization procedure, should have been a clue, but the cardiologists were barking up the wrong tree by assigning the near syncope and chest pain to myocardial infarction.

Of course we always want to do the right thing for the right patient, but among the things that we also agree on is that standardization of protocols improves overall performance. However, a formulaic approach to patients without considering other possibilities may lead to disaster. This case illustrates the problem of having speed trump everything else, but it also raises the question of incentives in medicine. We are incentivized when the admitting diagnosis is ST-segment elevation myocardial infarction to achieve the short door-to-balloon time, and as mentioned, this is also tied to performance measures and scoring. Perhaps for balance there should be a scorecard for missing a not too difficult diagnosis of pulmonary embolism. As quality improvement measures are pursued, it will be important to evaluate quality measures that are much more nuanced.

Perhaps what Dr. Eugene Stead told his trainees for years, “What this patient needs is a doctor,” remains sage advice.

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