Disparate Care for Acute Myocardial Infarction
Moving Beyond Description and Targeting Interventions

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“A rising tide lifts all boats.”

Sen. John F. Kennedy, September 27, 1960

T he seminal Institute of Medicine (IOM) report, “Unequal Treatment,” established the definite penetration of disparate care throughout all of medicine. At first glance, any major differences in health outcomes between groups of patients might be declared as evidence of a health care disparity—applying that definition generically. But, this IOM report established a striking contrast between differences that are appropriate and are largely based on patient preference, disease severity, and treatment indication versus differences attributable to overtly nonphysiological influences that result in less than ideal outcomes. The differences attributable to this latter concern thus constitute true health care disparities and represent an appropriate target for interventions to either profoundly narrow or preferably eliminate those disparate outcomes that are nonphysiological in origin. Specifically, the root causes of disparate care include system level issues that largely reflect cultural competency or lack thereof (eg, language, geographic, and economic barriers to care); provider level concerns that focus on the pervasive influence of bias and stereotyping; and community level issues, especially the built environment and the social determinants of health. Given the steady focus on addressing and eliminating health care disparities, it is expected that many of us have grown weary over these messages. However, the data presented by Mathews et al require us to remain resolute in our focus. Disparate care persists in cardiovascular medicine and tempers our exuberance that we are making transformational progress in the treatment of heart disease.

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The authors evaluated outcomes from those patients enrolled in the CRUSADE [Can Rapid Risk Stratification of Unstable Angina Patients Suppress Adverse Outcomes with Early Implementation of American College of Cardiology/American Heart Association Guidelines] registry, and using probabilistic matching with the national Medicare claims database addressed outcomes by race. Disconcerting evidence of disparate care for non-STEMI (ST-segment–elevation myocardial infarction) emerges: less use of clopidogrel, less use of IIb/IIIa glycoprotein inhibitors, less use of diagnostic cardiac catheterization, and less use of revascularization strategies—either percutaneous coronary intervention or coronary artery bypass grafting. Recognize that all of these interventions in appropriate clinical scenarios, are class of recommendation I supported by level of evidence A. It is compelling to consider the required language for deployment of evidence-based guideline recommendations for these interventions—“is recommended; should be done.” Even the overarching intent of the CRUSADE registry/performance improvement initiative is “early implementation of ACC/AHA guidelines.” Clearly, we are failing. To any extent that we as clinicians and clinician scientists are not fully deploying the American College of Cardiology Foundation (ACCF)/American Heart Association (AHA) clinical practice guidelines for all patients, we are practicing heresy.

The tenor of this language is intended to be deliberate, because the findings reported by Mathews et al in this issue of Circulation are disquieting. However, before we fully impugn our discipline, we must carefully and fully consider the limitations of these data. First, identification of suitable patients was halted at those enrolled through 2006 and for whom follow-up data existed through 2008. Thus the data points are >5 years old compared with contemporary practice. Practice patterns have hopefully changed. Second, insurance coverage and census/zip code data were used as surrogates for socioeconomic status and more broadly the social determinants of health. These parameters are at best only crudely representative for a deeply complex variable impacting health outcomes and likely under-represent the true influence of the social and economic environment. And third, the populations were not balanced regarding the presence of important comorbidities, nor are we provided the necessary data regarding coronary anatomy which would allow for more idealized adjustment of these observational data. These limitations are major and mute, possibly even negate, the findings of this article. But importantly, if any of the findings from this article remain extant today, then our sober attention is required.

The 40,500 patients from 466 sites enrolled all had non-ST elevation MI. Of these, 3116 were black. All patients were aged >65 years, and notably the black patients had a higher prevalence of previous heart failure, stroke, diabetes mellitus, hypertension, renal insufficiency, and end-stage renal disease requiring dialysis. However, the natural history of non-STEMI would suggest a broad representation of multiple concomitant comorbidities and the likely presence of multivessel coronary artery disease. Thus, the magnitude of the differences between blacks and whites ought not be of major significance.

The current data, corroborated by other datasets as well, demonstrate that near-term outcomes (ie, 30-day mortality)
after hospitalization for blacks with cardiovascular disease are better than for whites despite either a higher burden of negative risk factors or lesser use of indicated therapies. Yet longer term outcomes at 1 year and beyond show not only parallel outcomes but worse outcomes for blacks versus whites. After adjustment for insurance beyond Medicare and income levels, longer term outcomes were no longer different. Readmissions at 30 days after the initial event were higher for blacks versus whites and remained significant at 6 months, 1 year, and 2 and 3 years. Once again, adjustments that included income thresholds eliminated those differences seen in rehospitalization.

The authors struggle to explain these incongruous findings. Mathews et al suggest that the reasons for less appropriate care for blacks versus whites “are multifactorial” and may relate to the risks of procedures, likelihood of incurring acute renal failure, or hesitancy by the provider because of the extent of comorbidities. Only as a lesser concern is provider bias mentioned. The authors further assert that the dissimilarity in short-term outcomes may be entirely physiological with the black patients experiencing myocardial infarctions of lesser severity with smaller infarct size. However, the sensitivity analyses removing such myocardial infarctions from the analysis still resulted in better short-term outcomes. The hypothesis that then follows is a survivor hypothesis (ie, for those black patients able to reach Medicare age, a “survival of the fittest” concept prevails). Finally, the fall off in longer term outcomes returns to the lack of a high level of subscription to evidence-based interventions for acute coronary syndromes. There is no logic here. Nonphysiological variables are clearly at play, and we will continue to struggle with outcomes such as those noted unless we fully account for the deeper social context of our clinical decision making.

How do we go forward? To the extent that any of these findings remain evident in contemporary medicine, it is time to change practice. There can be no apology given for poor application of evidence-based interventions regardless of the patient cohort. Our evidence base is not conditional and should not be applied capriciously. The authors struggle with explanations because the very dense social variables at play in these analyses are not imminently or easily modifiable and certainly are not quantifiable. But what we can modify is the application of indicated evidence-based medical therapy.

Thus, we must finally go well beyond simply describing disparate care. This article is of no relevance if it only serves to further entrench a disappointing finding that has already been well described. As professionals who are responsible for the care of all persons with heart disease and as persons who live in these complex social interactions, we must accept the realization that our decision making—critical to the outcome of those with heart disease—is modified by our social construct. None of us are overtly biased but all of us, this editori-alist included, are persuaded by our preconceived stereotypes and implicit bias. Implicit bias surveys of contemporary medical students indicate that ≈75% of all students portray a decided bias in favor of whites over nonwhites.6 We cannot reprogram our innate tendencies but we can manage our decisions. The landmark work by Cohen et al7 using the AHA Get With the Guidelines program as a quality improvement strategy demonstrated that the deployment of a process of care performance improvement initiative not only eliminated the evidence of disparate care for acute coronary syndromes but improved adherence for all cohorts. The asset here is that process of care improvement initiatives are race/ethnicity blind, gender blind, and age blind. In practice, then, a rising tide does lift all boats. It’s time for high tide in cardiovascular medicine. These data from the CRUSADE Registry call for action, and that intervention is the wider scale initiation of performance improvement strategies for all patients.

The moment for hand-wringing over disparate care has passed; let’s now embrace interventions that will have a meaningful impact.

**Disclosures**

None.

**References**


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