Hospital Allocation and Racial Disparities in Health Care*

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Abstract

We develop a simple framework to measure the role of hospital allocation in racial disparities in health care and use it to study Black and white Medicare patients who are treated for heart attacks – a condition where virtually everyone receives care, hospital care is highly effective, and hospital performance measures have been validated. We report four facts. (1) Black patients receive care at lower-performing hospitals than white patients, even when they live in the same hospital market or ZIP code within a hospital market. (2) Over the past two decades, the gap in performance between hospitals treating Black and white patients shrank by over two-thirds. (3) This progress is due to more rapid performance improvement at hospitals that tended to treat Black patients, rather than faster reallocation of Black patients to better hospitals. (4) Hospital performance improvement is correlated with adoption of a high-return low-cost input, beta-blockers. Closing remaining disparities in allocation and harnessing the forces of performance improvement, including technology diffusion, may be novel levers to further reduce disparities.

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1 Introduction

Black Americans experience considerable disparities in health outcomes in the U.S. relative to other demographic groups. The overall age-adjusted death rate is 20% higher for non-Hispanic Black Americans than their non-Hispanic white peers (Murphy et al., 2015). The causes behind this gap are multifactorial and interrelated (Williams and Jackson, 2005). They include inequities existing prior to health care delivery related to living conditions, like income and education (Cutler, Deaton and Lleras-Muney, 2006; Braveman, Egerter and Williams, 2011); differences in the quality of care from a given health care provider due to communication and biases (Institutes of Medicine, 2003; Chandra and Staiger, 2010; Alsan, Garrick and Graziani, 2019); and differences in the health care providers patients use.

This last source of racial disparities derives from *allocation*: Black and white patients are often treated by different health care providers, some of which may have much higher (or lower) performance. Health care providers that serve Black patients tend to have poorer patient outcomes and report lacking the resources to deliver high-quality care (Bach and Schrag, 2004; Barnato et al., 2005; Skinner et al., 2005; Jha et al., 2007). The allocative forces that generate these disparities reflect historical and current inequities, including the long shadow of *de jure* hospital segregation, trust, and neighborhood segregation (Smith, 2005; Sarrazin, Campbell and Rosenthal, 2009; Chandra, Frakes and Malani, 2017; Alsan and Wanamaker, 2018). Differences in allocation may derive from decisions made by patients and by agents acting on their behalf, including physicians, ambulance drivers, and family members.

Over time, patients tend to reallocate towards higher-performing hospitals (Chandra et al., 2016). Whether the dynamics of allocative forces exacerbate or reduce racial disparities over time is ambiguous. Reallocation could be easier for better-resourced groups or it may be more feasible for groups receiving care from low-performing hospitals because there is more room to improve. The role of allocation in racial disparities is of central importance because it is a powerful driver of improving outcomes: Chandra et al. (2016) found that reallocation to higher-performing hospitals accounted for 1 percentage point of the increase in heart attack survival rates from 1996 to 2008, a similar magnitude to that of breakthrough medical technologies.

This study focuses on disparities in the performance of hospitals used by Black and white

patients in Medicare experiencing an acute myocardial infarction (AMI), or heart attack, between 1995 and 2014. We study AMI because nearly all of these patients receive hospital care, hospital intervention can be highly effective, and hospital performance measures have been extensively validated (Hull, 2020; Doyle, Graves and Gruber, 2019). Our work draws on these validated metrics to measure performance as the survival rate at the hospital adjusting for rich patient observables—the expected survival rate of a "typical" AMI patient at the hospital.

Taking an allocation perspective derived from the literature on firm productivity growth (Baily et al., 1992; Olley and Pakes, 1996; Foster, Haltiwanger and Krizan, 2001, 2006; Bartelsman, Haltiwanger and Scarpetta, 2013), we first show that during the 1990s, Black patients' were treated at lower-performing hospitals relative to white patients (Figure 1). Next, by reweighting white patients to have the same distribution of geography as Black patients, we show that in the 1990s differences in the hospital markets patients live in explain 44% of the Black-white gap (we use 306 hospital referral regions to describe hospital markets). Much of the 1990s disparity therefore derives from Black patients living in markets with low-performing hospitals, a feature that would also hamper reallocation as a tool for disparity reduction because it would require patients to travel long distances in search of better treatment. Differences in the ZIP codes patients live in (within hospital markets) explain a further 25% of the gap. This component of the disparity may reflect differences in where patients live in markets relative to high-performing facilities and may be more amenable to reallocation. The remaining 32% reflects differences in the performance of hospitals that white and Black patients are treated at despite living in the same ZIP code. This component could encompass differences by race in where patients live within small areas areas as well as differing beliefs about hospital performance on the part of patients or agents acting on their behalf.

By the 2010s we find that the gap in the performance of hospitals used by Black and white AMI patients had shrunk by 70% (Figure 2). By the 2010s, it was no longer the case that differences in the hospital markets that Black and white patients lived in contributed to the gap. These findings suggest that Black-white disparities in the performance of hospitals used in heart attack care no longer emanate from large-region differences in where patients live (e.g. the North versus the South, or even Nashville versus Memphis). Rather, differences in the hospitals used by patients living in the same hospital markets now drive the observed, albeit smaller, racial gap. The remaining gap is also potentially actionable through allocation policies because it would not depend on emergency

patients traveling long distances to access better hospitals. Still, it is important not to view a change in the hospital performance disparity as automatically reflecting the force of reallocation to higher-performing hospitals. For example, there may have been no change in where patients were treated, but greater improvement at the hospitals where Black patients tended to receive care. Further investigation is required to demonstrate whether reallocation or performance improvement is responsible for the narrowing disparity uncovered by the static analyses.

We next develop a dynamic decomposition framework to elucidate the economic forces behind these trends. We find larger performance gains at hospitals used by Black patients than those used by white patients at baseline. In fact, the entire closing of the gap results from *differential performance improvement* (Figure 3) – meaning that hospitals which tended to treat Black patients raised their performance more than other hospitals. Under one-third of the convergence can be attributed to *differential reallocation*, where Black patients increased their use of higher performing facilities at a faster rate than white patients. These findings show that in the AMI context, performance improvement has acted as a force to improve outcomes and reduce disparities, highlighting the potential importance of this channel in analyses of disparities in the health care system. The framework we develop also represents a methodological contribution that can be applied to study the role of allocation in disparities in many contexts such as education, employment, and criminal justice.

Finally, we investigate the role of technology in hospital performance improvement by analyzing the adoption of low- and high-cost AMI treatment technologies. Beta-blockers are inexpensive medications that were known to substantially improve outcomes at the onset of our analysis period, and we find that the use of these drugs grew more rapidly at hospitals that tended to treat Black patients at baseline. Thus, the adoption of beta-blockers and other technologies for which they are a surrogate, including better management, may have contributed to differential performance improvements (Bloom et al., 2012; McConnell et al., 2013; Skinner and Staiger, 2015; Bloom, Sadun and Van Reenen, 2016). At the same time, we find that the adoption patterns of capital-intensive technologies like cardiac catheterization are disparity-expanding. These findings allow us to connect the literature on racial disparities to literatures on allocative forces as well as the productivity of medical treatments.

Our results are robust to a host of alternative approaches to measuring hospital performance.

They hold under richer or coarser controls for patient observables, suggesting that endogenous selection of hospitals does not drive the results. Moreover, they are insensitive to whether we measure hospital performance using only white patients, thus ruling out that our findings are driven by performance improvements that are specific to Black patients. Rather, our results suggest that hospitals with high market shares among Black patients improved their overall performance across patient race groups.

The remainder of the paper proceeds as follows. Section 2 covers our data and performance measurement approach, Section 3 explains our static and dynamic decomposition frameworks, Section 4 presents results, Section 5 provides evidence on the role of technology adoption, and Section 6 concludes.

2 Setting, Data, and Measurement

2.1 Setting

We examine Medicare beneficiaries with Fee-for-Service (FFS) coverage hospitalized for an AMI between 1995-2014. This setting has a number of advantages for this study. One is that it essentially removes insurance coverage as a determinant of patient allocation to hospitals. FFS Medicare patients have access to the vast majority of hospitals and for most patients, there is no difference in the cost of going to a different hospital. Selection into the hospitalization data is also less of an issue because AMI is an emergent condition requiring immediate treatment at a hospital – there is little scope for patients to go untreated or undiagnosed. AMI also has validated hospital quality measures: hospital measures of quality using risk-adjusted survival and calculated using observational data correlate extremely well with quality measured from patients who are quasi-randomly assigned to facilities (Hull, 2020; Doyle, Graves and Gruber, 2019). This research suggests that the rich patient data in Medicare claims accounts for endogenous hospital selection well, yielding quality measures with a strong signal of true performance. The rich data on AMI treatment also provides observable measures of technology use like beta-blockers and cardiac catheterization that allow us to probe the mechanisms underlying changes in hospital performance. Finally, allocative forces have been shown to operate at a point in time and over time for heart attacks (Chandra et al., 2016), and there has been technological progress that is tracked in data, allowing us to understand why hospitals are

improving (e.g. McClellan, McNeil and Newhouse, 1994; Cutler et al., 1998; Chandra and Staiger, 2007, 2020). Given the aforementioned reasons, we do not study allocation to physicians rather than hospitals or allocation for common chronic diseases. While these sources of health care would be important to study, analyses would suffer from a lack of validated outcomes measures and the potential selection into an office visit or hospital stay.

We conduct point-in-time analyses during a baseline period (1995-1999) and an endline period (2010-2014). Our over-time analyses consider changes from the baseline to endline period. Thus, for each hospital, we measure allocation and performance in those two periods. In addition, we estimate allocation and performance for intermediate periods (2000-2004 and 2005-2009) when visualizing their evolution in figures. To conduct inference, we use a hospital-level bootstrap approach with 1000 replicates (Appendix Section D).

2.2 Data

Our primary data source is Medicare hospitalization records for 100% of beneficiaries between 1995-2014 linked to enrollment records with demographic information about these individuals. Together these data provide rich information about patients, including their race, location of residence, the hospital at which they receive treatment, and their diagnoses.[1] To define hospital markets, we use Hospital Referral Regions (HRRs) from the Dartmouth Atlas. This market definition uses the empirical patterns of where Medicare patients go for major surgeries to create 306 hospital markets for the U.S. Lastly, to observe use of beta-blockers, which is not tracked in the claims, we link to 1994-1995 patient records abstracted for the Cooperative Cardiovascular Project (CCP), a large-scale effort to measure hospital quality, and publicly reported process of care measures from CMS for 2010-2014 (see Appendix Section C.1).

The initial sample consists of patients who were hospitalized for AMI during the study period. We remove hospital stays by the same patient within one year of a prior AMI admission to exclude return visits. Following the literature, we call these hospitalizations index events. We limit our sample to patients age 66 and above with full FFS Medicare coverage during the year prior to

[1] Because hospital identifiers in Medicare data may change over time, we use a longitudinal crosswalk provided by Jonathan Skinner and the Dartmouth Institute for Health Policy and Clinical Practice to merge together all identifiers that ever refer to the same facility. Hospitals that change identifiers are tracked as one facility; hospitals that merge or de-merge are treated as one facility for the entire analysis period.

admission and the year subsequent to it (or until death if the patient dies during that year). Patients with Medicare Advantage coverage and those with a lapse in full (Part A+B) coverage are omitted because they are poorly observed in the data. We further restrict to events at short-term acute care or critical access hospitals, because these are the hospitals that treat heart attack patients. Because we use index events to estimate hospital performance, we require that the hospital have at least 25 events during the relevant 5-year measurement period (1995-1999, 2000-2004, 2005-2009, or 2010-2014). After imposing these restrictions, the result is the main analysis sample, which we use to estimate hospital performance. To analyze allocation, we focus on hospitals that met this patient volume threshold in the baseline period (1995-1999) and the endline period (2010-2014), yielding a balanced panel; we later show that our findings are robust to adding hospitals that only meet this restriction in one of the two periods.

2.3 Performance measurement

To measure hospital performance, we estimate adjusted survival rates for hospitals in each of the 5-year baseline and endline periods. Specifically, we calculate the 30-day survival rate of AMI patients at the hospital after adjusting for patient comorbidities and demographic factors. We describe our approach here and in more detail in Appendix Section A. Starting with the analysis sample, we calculate hospital performance by estimating the following equation:

$$s_{iht} = \alpha_t + \beta_t X_{iht} + \gamma_{ht} + \epsilon_{iht}, \tag{1}$$

where i indexes patients, h indexes hospitals, t indexes time period, s_{iht} is an indicator for whether the patient survived 30 days from their hospital admission date, X_{iht} is a vector of comorbidity and demographic indicators, γ_{ht} are hospital fixed effects, and ϵ_{iht} is a disturbance term. The comorbidities consist of indicators for whether a patient had diagnoses for any of 23 conditions during a hospital stay in the year prior to the admission and the demographic indicators are age-race-sex interactions. Race is included in the model in 3 categories: Black, white, and other (non-Black non-white). This regression is estimated separately in each period.

We then develop performance measures reflecting the expected 30-day survival for the average

Black and white patient at the given hospital and time period:

$$\hat{q}_{ht}^{B} = \hat{\alpha}_t + \hat{\gamma}_{ht} + \bar{X}_t^B \hat{\beta}_t \tag{2}$$

$$\hat{q}_{ht}^{W} = \hat{\alpha}_t + \hat{\gamma}_{ht} + \bar{X}_t^W \hat{\beta}_t, \tag{3}$$

where $\bar{X}_t^B$ and $\bar{X}_t^W$ are the average comorbidity and demographic indicators for Black and white patients in period t, respectively. Within a period, the measures from equations 2 and 3 are perfectly correlated because each is a level-shift of the other. This model pools patients of all races when estimating hospital performance. It allows Black and white patients to have different outcomes on average, but does not allow for race-specific hospital effects—such a model would be hampered by the fact that 20% of hospitals do not treat a single Black patient and many others treat too few Black patients to estimate a precise race-specific hospital effect (Barnato et al., 2005; Chandra, Frakes and Malani, 2017). Robustness analyses reported in Section 4.3 describe this assumption in more detail and show that our results are robust to an alternative approach to performance measurement that relaxes it. This approach highlights another key point about the forces we analyze: they are are distinct from those which reduce racial disparities in outcomes *within* a given hospital. This source of disparities is a crucial object of study but is not the focus of this work.[2]

2.4 Allocation measurement

Using these data, we measure the allocation of Black and white patients to hospitals during the baseline and endline periods. We define allocation as the national market share of the hospital among patients in the given race group in the given time period:

$$\theta_{ht}^{B} = N_{ht}^{B}/N_t^{B}, \quad \theta_{ht}^{W} = N_{ht}^{W}/N_t^{W}. \tag{4}$$

[2] A recent analysis at the frontier of research on how disparities could arise from differences in the quality of care within providers comes from Alsan, Garrick and Graziani (2019), who consider the role of physician race in the quality of health care for Black men.

2.5 Summary statistics

Table 1 presents summary statistics on the patients and hospitals included in our main analyses. Panel A describes Black and white patients in the baseline and endline periods. Overall, in both periods, Black patients were more likely to be female, had a greater burden of illness as measured by the comorbidities, and were younger. The number of heart attack patients decreased from baseline to endline. This decline reflects reductions in AMI incidence during this period as well as the rise of private insurance coverage in the Medicare program (Medicare Advantage), since these patients are poorly observed in our data and are thus excluded (Jacobson, Damico and 2018, 2018; Yeh and Selby, 2010). Black patients had higher survival rates in both periods than their white counterparts. This pattern is well-documented in AMI and other emergent conditions in the above-65 population, and may be due to unobserved health status differences by race among people who survive to older age as well as differences in the use of medical procedures with short-term risks of mortality (Barnato et al., 2005; Polsky et al., 2007, 2008; Thomas et al., 2011; Downing et al., 2018; Huckfeldt et al., 2019).

Panel B shows baseline and endline characteristics of the 2,712 hospitals in our main analyses. We note large variations in performance across hospitals as operationalized by 30-day survival adjusting for patient observables and measurement error (see Appendix Section A). In both periods the standard deviation of survival rates across hospitals is 4 percentage points. To benchmark this magnitude, we note that survival rates rose about 5 percentage points for both groups of patients between the baseline and endline periods. Thus going to a hospital with 1 standard deviation greater AMI performance at a point in time yields a similar benefit to the average rise in performance nationally over two decades.

The remainder of Panel B shows statistics about the allocation of patients to hospitals. The average hospital has hundreds of AMI patients during each 5 year period. Nearly every hospital treats at least one white patient, but due to the smaller numbers of Black patients as well as geographic segregation, 531 hospitals treat no Black patients at baseline and 592 treat no Black patients at endline.

3 Analytical Approach: Static and Dynamic Decompositions

Our main empirical analyses document and decompose disparities in the performance of hospitals that Black and white patients use. We first study these disparities at a point-in-time, yielding a static decomposition. Our approach draws on methods previously employed to study the firm-level drivers of sector-wide productivity growth (Foster, Haltiwanger and Krizan, 2001; Foster, Haltiwanger and Syverson, 2008; Baily et al., 1992). Our core insight is that just as one can decompose productivity growth in a sector into changes in allocation across firms and productivity growth within firms, so too can one decompose differences in AMI outcomes between Black and white patients into differences in allocation across hospitals and performance differences within hospitals. This method also uses re-weighting to document the role of geography in the gap, building on research that has decomposed the Black-white wealth gap as well as work studying the contributors to wage inequality over time (Barsky et al., 2002; DiNardo, Fortin and Lemieux, 1996). Finally, we further extend the static approach to study how Black-white disparities evolve over time, leading to a dynamic decomposition. We now review these decompositions in more detail.

3.1 Static decomposition

In the static decomposition, we quantify the degree to which Black patients' use of lower (or higher) performing hospitals reduces (or raises) their average 30-day survival relative to white patients in a given time period (baseline or endline). We can express the difference in survival rates between Black and white patients as:

$$\bar{q}_t^B - \bar{q}_t^W = \sum_{h \in H} \theta_{ht}^B \hat{q}_{ht}^B - \sum_{h \in H} \theta_{ht}^W \hat{q}_{ht}^W \tag{5}$$

$$= \sum_{h \in H} \theta_{ht}^W (\hat{q}_{ht}^B - \hat{q}_{ht}^W) \qquad \leftarrow \text{Within}_t \tag{6}$$

$$+ \sum_{h \in H} (\theta_{ht}^B - \theta_{ht}^W) \hat{q}_{ht}^B, \qquad \leftarrow \text{Between}_t \tag{7}$$

where $\bar{q}_t^B$ and $\bar{q}_t^W$ are the 30-day survival rates of Black and white patients in period t, q_{ht}^B and q_{ht}^W are the performance measures for hospital h in period t for Black and white patients (i.e. expected

30-day survival rates at the hospital for the average Black and white patient), θ_{ht}^{B} and θ_{ht}^{W} are the national market share of hospital h in period t among Black and white patients, and H is the set of hospitals.

Equation 5 illustrates that the difference in survival rates can be divided into two conceptually distinct components. The *within* (Within_t) refers to the difference in survival between Black and white patients resulting from disparities in the performance of a given provider for Black versus white patients. These race-specific performance differences could arise for a variety of reasons including biases or racism by providers and differences in the prevalence of underlying unobserved risk factors.

The *between* term (Between_t), or between-race gap, refers to the difference in survival resulting from hospital allocation, specifically, Black patients' use of lower or higher performing hospitals than white patients. In our analyses, this term is insensitive to the reference population we use for performance because $\hat{q}_{ht}^{B}$ and $\hat{q}_{ht}^{W}$ differ by a constant that drops from the summation. For ease of expression we omit the superscript in the remainder of the text.

In the empirical section we will decompose the between-race gap into three sub-components: (1) *between hospital market differences* that reflect differences in the hospital markets where Black and white patients live (defined as Dartmouth Hospital Referral Regions, or HRRs), (2) *between ZIP code differences* that measure differences in the small areas, or more colloquially neighborhoods, that Black and white patients live in within markets, and (3) *within ZIP code differences* in the hospitals Black and white patients use, even when they live in the same ZIP code. For ease of exposition, we detail this method later alongside the results.

3.2 Dynamic decomposition

We also develop a decomposition of the evolution of the between-race gap in hospital allocation over time, or the change in Between_t. This dynamic decomposition complements the static decomposition by showing the role of performance and allocation in driving changes in the gap that occur over time. Two forces could contract disparities over time: (1) stronger performance improvement at hospitals that historically tended to serve Black patients, and (2) stronger reallocation of Black patients than white patients to historically better performing hospitals. To assess the relative contribution of these two forces to a reduction of disparities in hospital performance, we note that the

first difference of Between_t can be expressed as:

$$\Delta\text{Between}_t = \sum_{h\in H} \left(\theta^B_{h,t-1} - \theta^W_{h,t-1}\right)\left(\Delta\hat{q}_{h,t}\right) \quad \leftarrow\text{Differential Performance Improvement} \tag{8}$$

$$+ \sum_{h\in H} \left(\Delta\theta^B_{h,t} - \Delta\theta^W_{h,t}\right)\hat{q}_{h,t-1} \quad \leftarrow\text{Differential Hospital Reallocation} \tag{9}$$

$$+ \sum_{h\in H} \left(\Delta\theta^B_{h,t} - \Delta\theta^W_{h,t}\right)\left(\Delta\hat{q}_{h,t}\right), \quad \leftarrow\text{Cross} \tag{10}$$

where Δ is the first difference operator. In practice, t is the endline period (2010-2014) and $t-1$ is the baseline period (1995-1999).

The first term, *differential performance improvement*, measures the relationship between the Black-white market share difference at baseline and the growth in hospital performance over time. Specifically, its left side shows whether the hospital was relatively popular among Black or white patients at baseline, while its right side captures the change in performance. Thus, the term captures the change in the between-race gap explained by hospital performance growth, fixing allocation at baseline values. A positive contribution from this term would imply that performance grew more rapidly at hospitals that were historically relatively popular among Black patients.

Differential hospital reallocation, which appears next, has two parts. Its left side is a difference-in-difference of Black vs. white market share at endline vs. baseline, and it shows whether Black patients re-allocated at a faster rate to the given hospital than white patients. The right expression is the hospital's performance during the baseline period. A positive reallocation term would imply that Black patients shifted towards historically better performing hospitals at a faster rate than white patients.

The first two terms associate a change over time with a baseline value. They thus fail to capture the fact that reallocation over time and performance improvement over time could co-occur. The cross term accounts for this final component of the change in the between-race gap. It captures the change explained by stronger performance improvement at hospitals that had stronger relative gains in Black patient market share over time.

4 Results

4.1 Static Decomposition

Table 2 displays the static decomposition results. Our first key finding, shown in column 1 of the table, is that Black patients used hospitals with significantly lower performance than white patients during the baseline period — that is, Between_t is negative. Specifically, Black patients used hospitals with 1 percentage point lower expected survival rates than white patients on average. Panel A of Figure 1 visualizes this result, plotting the distribution of hospital performance among Black patients and white patients at baseline. The vertical bars indicate the means of the distributions and are 1 percentage point apart, matching Table 2. To put the magnitude of this result into context, reperfusion therapy for AMI – developed in the late 1980s, still used today, and widely acknowledged to be a transformational treatment – increases survival by 2 percentage points (Fibrinolytic Therapy Trialists' Collaborative Group, 1994).

We next quantify the contribution of geography using an inverse probability weighting approach, where we re-weight white patients to match the contemporaneous geographic distribution of Black patients at the hospital market (as given by HRRs) and ZIP code level. This method allows us to account for differences in the geographic distribution of the populations and assess how allocation for Black and white patients compares among patients in the same market or neighborhood. We so by developing the weighting function (c.f. Barsky et al., 2002):

$$\omega(z) = \frac{\Pr(black|Z=z)}{\Pr(white|Z=z)} \cdot \frac{\Pr(white)}{\Pr(black)}, \tag{11}$$

where z is a vector of covariates. $\omega(\cdot)$ serves to reweight white patients such that their distribution of z is equalized with that of Black patients. Thus, letting z be a vector of indicators for HRRs or a vector of indicators for ZIP codes, we can define the reweighting functions $\omega_t^{HRR}(\cdot)$ and $\omega_t^{ZIP}(\cdot)$ which match distributions at the market and neighborhood level, respectively, in time t.

Using the weights given by the $\omega(\cdot)$ functions, we construct counterfactual hospital market shares for white patients. The resulting objects are $\theta_{ht}^{W,HRR}$, which is the national market share for hospital h in time t for white patients after reweighting them to have the same market-level distribution as Black patients, and $\theta_{ht}^{W,ZIP}$ which has the same interpretation except that the

reweighting occurs at the more finely-grained neighborhood level.[3]

We can decompose the between-race gap into three components:

$$\text{Between}_t = \sum_{h \in H} \left(\theta_{ht}^{W,HRR} - \theta_{ht}^{W} \right) \hat{q}_{ht} \qquad \leftarrow \text{Between Markets} \tag{12}$$

$$+ \sum_{h \in H} \left(\theta_{h,t}^{W,ZIP} - \theta_{ht}^{W,HRR} \right) \hat{q}_{ht} \quad \leftarrow \text{Between ZIP Codes} \tag{13}$$

$$+ \sum_{h \in H} \left(\theta_{ht}^{B} - \theta_{ht}^{W,ZIP} \right) \hat{q}_{ht}. \qquad \leftarrow \text{Within ZIP Codes} \tag{14}$$

The remainder of Table 2 presents this decomposition. Nearly half (44%) of this disparity is explained by differences in the hospital markets that Black and white patients live in, which means that just more than half (56%) of the disparity exists even when comparing patients in the same market. 25% of the total gap is explained by neighborhood differences in residence within a market. The remainder, reflecting differences in hospital choice among Black and white patients in the same neighborhoods, is 32% of the total. Panels B-D of Figure 1 visualize these findings by sequentially reweighting white patients to match the geographic distribution of Black patients at finer levels of geography and showing how the means, displayed as vertical bars, evolve. This approach visualizes the disparity explained at the market level, depicting between-market differences (Panel B); at the neighborhood level, showing between-neighborhood differences within the same market (Panel C); and the remainder, depicting the within neighborhood differences (Panel D).

By the mid 2010s, we find that these disparities have shrunk substantially – the racial disparity is less than one-third its level at baseline. Column 3 of Table 2 shows that at endline, black patients use hospitals with 0.3 percentage points lower expected survival rates than white patients (Figure 2 visualizes this closure of the gap). The subsequent rows of the table show that the role of geography also changes: only 5% of the disparity is now explained by market differences in where Black and white patients live, and the contribution is not statistically significant. ZIP code differences play a much larger relative role at this time period, explaining 46% of the disparity; the contribution is about half its absolute value in percentage points at baseline and is significant at the 10% level. The remaining half of the gap persists within market and ZIP code.

[3]Because the weighting function is only defined in the support of the white patient geographic distribution, the ZIP code weights for white patients are developed from the over 98% of Black patients who live in ZIP codes in which there was at least one white patient.

As expected, as the disparities in performance between hospitals used by Black and white patients were narrowing, the survival outcomes for Black patients relative to white patients were improving. Specifically, the unconditional survival rate for Black patients rose by 0.4 percentage points more than for white patients from baseline to endline (Table 1). This improvement is similar though slightly less than the change in the between-race gap in performance of 0.7 percentage points (Table 2). Thus most of the gains in performance at hospitals with high market shares among Black patients translated into improved outcomes at the patient level, too. That said, translation was not one-for-one. The difference suggests a small impact of countervailing forces on the relative survival rates of Black vs. white patients within the same hospital such as unmeasured changes in patient health or provider bias by race.

Column 5 of Table 2 confirms that we reject the null hypothesis of no closing of the gap: the change in Between_t from baseline to endline of 0.7 percentage points is highly statistically significant. Moreover, it is mostly accounted by the declining contribution of market-level differences. Given the difficulty patients would experience attempting to cross markets to access higher-performing facilities during an emergency like AMI, it is hard to explain the totality of these findings by patients re-allocating because they have become more elastic to hospital performance. In the next section, we formally investigate the role of reallocation in this result and contrast it with another mechanism we have identified: differential performance improvement.

4.2 Dynamic Decomposition

Table 3 displays the dynamic decomposition results. Here, we divide the total disparity reduction of 0.7 percentage points into the contributions of differential performance improvement and differential hospital reallocation. We find that the former channel fully accounts for the disparity reduction over time and its contribution is highly statistically significant. This result implies that hospitals that tended to treat Black patients at baseline experienced stronger performance gains than hospitals that tended to treat white patients. While we also find a role for the reallocation channel – Black patients reallocating to hospitals with better performance at baseline at a faster rate than white patients – it is quantitatively smaller than the role of differential performance improvement. Reallocation contributes 0.2 percentage points to the aggregate disparity reduction. Finally, the negative cross term implies that white patients reallocated more strongly towards hos-

pitals that improved performance over this time period. This term contributes -0.2 percentage points to the disparity.

We visualize these results in Figure 3. Panel A graphs the between-race gap at baseline, endline, and two intermediate periods. The change in the gap from baseline to endline is the quantity that we decompose. The subsequent panels display counterfactuals in which we assume hospital market shares (Panel B) or performance levels (Panel C) remain constant at baseline levels. The changes in the gaps from baseline to endline visualized in these two panels are algebraically identical to the differential performance improvement and reallocation terms, respectively. Specifically, in Panel B we fix market share but allow performance to evolve, isolating the component of the disparity reduction due to performance changes alone. Panels A and B look strikingly similar, reflecting that differential performance improvement can account for the entire reduction. Similarly, in Panel C we fix performance but allow market share to evolve, depicting the role of reallocation in disparity reduction. Here, the gap attenuates only slightly, indicating that reallocation plays a much smaller role than changes in performance.

4.3 Robustness

We report the robustness of both our static and dynamic decomposition results to several alternative specifications and measures to address concerns about selection, race-specific hospital performance, MA penetration, and the role of hospital entry and exit. Our findings are highly robust to these alternative approaches.

Selection Our measures of hospital performance could be biased if patients select hospitals on the basis of unobserved health status. If this selection is correlated with hospital volume, one could observe a spurious relationship between performance and allocation leading to biased estimates of the between-race gap. Though prior work has validated our approach (e.g. Hull, 2020; Doyle, Graves and Gruber, 2019), as an additional falsification test of the selection on observables assumption we consider the sensitivity of our findings to the richness of controls in the performance model (Altonji, Elder and Taber, 2005; Oster, 2019). We try two alternative specifications with coarser controls: no patient-level controls and demographic controls only (age/race/sex interactions); we also test two additions to the main specification: adding interactions between the race and comorbidity variables, and a control for the type of AMI (see Appendix Section A). Our findings using

these alternative specifications are qualitatively unchanged from the main specification (Appendix Tables A1 and A3), though the role of performance improvement is slightly larger when all patient controls are omitted.

Race-Specific Performance Our model of hospital performance allows for average differences in patient outcomes by race but assumes that the difference does not vary by hospital. This assumption will not bias our estimates if hospitals have idiosyncratic race gaps in their treatment quality or outcomes due to, for example, provider prejudice. However, if Black patients disproportionately choose hospitals that are (or become) relatively high-performing for Black patients, the decompositions would overstate the race gap in performance and potentially misstate how it has evolved over time. For example, if hospitals that were popular among Black patients were more likely to address *within*-hospital disparities, raising performance more for Black patients than white patients, our analyses might show a declining between-race gap that actually represents a declining within-hospital gap.

We investigate this concern in two ways and find little support for it. First, we estimate a model of hospital performance limiting only to white patients. If the performance improvements we observe at hospitals with high market shares among Black patients are driven by improvements in performance specific to those patients, our results would attenuate under this approach. However, when we present this sensitivity analysis in Appendix Tables A2 and A4 the findings are qualitatively unchanged. We continue to see a large decline in the performance gap, one that is more than completely explained by differential performance improvement. This result reinforces the interpretation that performance improvements at hospitals tending to serve Black patients were not specific to Black patients alone.

Second, we estimated an alternative model without limiting to white patients, but allowing hospital performance to differ by race. Augmenting the fixed effects model to permit hospital-race effects raises measurement issues because only a handful of hospitals treat large numbers of Black *and* white patients in both periods – under one-fifth treat at least 25 Black and 25 white patients at baseline and endline – and many hospitals treat no Black patients at all. Thus, we estimate a random effects model in which hospital performance is drawn from a trivariate normal distribution: one hospital random effect and two hospital random slopes (for Black patients and non-Black non-white patients). This model emits an estimate of the underlying variance-covariance matrix of the

distribution which is robust to measurement error from small samples of Black or white patients at the hospital level. It also emits best linear unbiased predictions (BLUPs) of hospital performance for Black and white patients. Appendix Section 2.3 describes the approach in more detail.

The findings of this second sensitivity analysis of performance by race also support our main approach and results. In Appendix Table A5, we show that hospital performance for Black and white patients is highly correlated: 90% and 83% during the baseline and endline periods, respectively. Such a high correlation limits the potential scope for selection on race-specific performance. We use the BLUPs to re-estimate the decompositions with the performance measure $\hat{q}_{ht}$ replaced with predicted performance for Black or white patients. The results, presented in Appendix Tables A2 and A4, are similar to those of our main approach regardless of whether we use performance for Black or white patients. While the disparity at baseline is somewhat smaller, it continues to attenuate substantially over time and this decline is due to differential performance improvement.[4] As in the previous sensitivity analysis by race, these findings are inconsistent with declining within-hospital gaps driving the over-time changes, since they are so similar for Black patient performance and white patient performance.

Attrition to Medicare Advantage Patients who enter Medicare Advantage (MA), the private insurance system in Medicare, are not observed in our data. MA penetration was increasing during our study period and MA enrollees are disproportionately white and more likely to be healthy (Brown et al., 2014); if marginal MA enrollees make different hospital choices than those in fee-for-service Medicare, particularly if they tend to allocate toward high-performing hospitals, their attrition from the sample could generate the declining race gaps we observe. To address this concern, we repeat our analyses in markets with below median MA penetration in 2014 to limit the potential for attrition to MA. The results are reported in Appendix Tables A2 and A4 and are very similar to our main findings, suggesting that selection into MA is not driving the patterns we report.

Entry and Exit To this point, all results have analyzed a balanced panel of hospitals, omitting any role for hospital entry and exit. The productivity decomposition literature has emphasized the

[4] A similar pattern obtains from a random effects model without race-specific random slopes and is presented in the same tables. Unlike the main model of performance, BLUPs from random effects models are shrunk toward the mean. These results suggest that shrinkage drives the smaller disparity at baseline in the race-specific performance model, rather than the race-specific slopes.

importance of this channel (Foster, Haltiwanger and Syverson, 2008; Melitz and Polanec, 2015), and the entry of higher-performing facilities and the exit of lower-performing ones drives some of the improvement in aggregate hospital performance over time (Chandra et al., 2016). To test whether net entry expands or contracts disparities, we relax the requirement that hospitals treat at least 25 patients at baseline and endline and return those that only meet the threshold in one of the periods to the sample. Accounting for entry and exit requires a more complex decomposition approach, which we discuss in Appendix Section B. To summarize, an entry term reduces the Black-white gap when entering hospitals are relatively popular among Black patients and have higher than average performance; an exit term reduces the gap when, for example, exiting hospitals were relatively popular among Black patients but have lower than average performance.

We find results similar to those of our main analyses. The static decomposition in Appendix Table A2 shows that at each point in time, the between-race gap is slightly larger with the unbalanced panel; the reduction in the gap over time is also slightly larger. Decomposing this change, Appendix Table A4 shows that net entry plays a role in reducing disparities where, by design, it previously could not (entry exacerbates the disparity but is is more than offset by exit). Differential performance improvement continues to make the largest contribution to disparity reduction, though the magnitude is somewhat less than in the main specification.

5 Technology Adoption as a Mechanism

Earlier, we noted that differential performance improvement can explain the totality of the narrowing between-race gap in hospital performance. To understand this finding we explore the adoption of new treatment technologies as a mechanism for improvement. The adoption patterns of these technologies could play a key role in determining hospital performance, and through this channel, racial disparities in care outcomes. Our framing draws on work showing that a disproportionate share of survival gains come from the diffusion of low-cost technologies that benefit virtually all patients rather than high-cost interventions which exhibit diminishing returns (Chandra and Skinner, 2012; Skinner and Staiger, 2015). In the years prior to the study period, the technology frontier of AMI treatments moved forward dramatically as both types of interventions diffused through hospitals. Beta-blocker drugs provide an example of low-cost, high-benefit, TFP

raising AMI technology. Their value was established in a series of trials in the 1980s, and by the beginning of the study period, guidelines recommended their use in nearly all AMI patients (Yusuf et al., 1985; Lee, 2007). Cardiac catheterization, in contrast, is an example of a capital-intensive intervention that requires costly and specialized labor inputs (Jencks et al., 2000; Jencks, Williams and Coleman, 2009; Cutler et al., 1998). It enabled improved imaging of the heart as well as a procedure called percutaneous coronary intervention (PCI) to open blocked blood vessels.

Rapid adoption of these technologies by hospitals with that were relatively popular among Black patients could reduce the between-race gap in hospital performance. To test this hypothesis, we repeat the dynamic decomposition replacing the hospital performance outcome with the use of beta-blockers, and separately, cardiac catheterization. We consider whether the results follow a pattern similar to our main results: that Black patients tend to use hospitals with lower levels of technology adoption at baseline compared to white patients; that this gap attenuates from baseline to endline; and that the attenuation is explained by faster technology adoption at hospitals with relatively high Black patient shares *ex ante*. Such patterns would support technology adoption as a mechanism for the findings of the previous section.

We develop and describe our two measures of adoption in more detail in Appendix Section C.1. Briefly, we measure beta-blocker take-up as the share of AMI patients at the hospital who were prescribed beta-blockers at discharge. CMS and accreditation agencies have used this metric as an indicator of hospital quality. Like CMS, we do not risk-adjust this metric because the drugs are thought to be appropriate for the vast majority of patients. We take it as a surrogate for hospital use of low-cost technologies that generate large benefits including but not limited to beta-blockers themselves. To measure high-cost technology adoption, we examine the use of cardiac catheterization on the day of admission, adjusted for patient risk-factors – the clinical literature suggests that only these catheterizations have a meaningful impact on survival (Hartwell et al., 2005; Hochman et al., 2006; Likosky et al., 2018). We re-run the model specified in equations 1-3 with catheterization as the outcome, yielding hospital-level estimates of adoption at baseline and endline.

Table 4 presents decompositions of the measures. Takeup of both technologies rose substantially from the baseline to endline periods. In column 1, we focus on beta-blockers, where the Black-white gap was substantial at baseline. Compared to white patients, Black patients receive treatment at

hospitals with 1.7 percentage point lower rates of beta-blocker use at baseline but 0.2 percentage points lower use at endline. The dynamic decomposition shows that over 90% of the closure of the gap is due to differential performance improvement: hospitals relatively popular among Black patients were also faster to adopt the drug. Reallocation also contributes about 40% of the closure; the excess over unity is offset by a negative cross term: there was some reallocation away from hospitals that grew their use of beta-blockers.

In contrast, the findings for catheterization in column 2 are the reverse: at baseline, Black patients received treatment at hospitals with slightly higher cardiac catheterization rates than white patients. This gap attenuates and flips over time – at endline white patients use hospitals with higher cardiac catheterization rates. We found similar results when we measured high-cost intervention in other ways: counting catheterizations on any day, counting catheterization or bypass surgery, or counting bypass surgery alone. Results for aspirin, a low-cost technology with a racial disparity at baseline that matched the high-cost measures in that it favored white patients, are also similar to the findings for high-cost technologies (Appendix Table A6).[5]

These results suggest that diffusion of beta-blockers and technologies for which they serve as surrogates attenuated disparities in the performance of hospitals used by Black and white patients. On the other hand, the patterns of diffusion for capital-intensive technologies like catheterization expanded disparities. Our findings highlight the potential role of technology adoption in the evolution of performance gaps between hospitals used by Black and white patients. These findings do not, however, preclude other potential causes for performance improvement that, for example, come from better treating other comorbidities that may increase mortality risk.

6 Conclusion

We quantified the role of patient allocation to hospitals of differing performance in Black-white disparities in AMI treatment and outcomes by analyzing nearly two decades of Medicare patients. We first showed that in the 1990s, Black patients tended to use hospitals with substantially worse

[5] We also investigated if hospitals that tended to treat more Black patients experienced greater gains in the returns to catheterization. Gains could occur if hospitals improved their skill at using the procedure or if they came to better target it at patients who were likely to benefit. Hospitals with larger Black patient shares experienced lower returns to catheterization at baseline. However, this gap persisted through endline, suggesting that differential gains in returns do not drive the findings we observe.

patient outcomes during the 1990s – the survival rate of a typical patient was a percentage point lower at hospitals used by Black patients than at hospitals used by white patients. Our work next highlighted the role of geography in accounting for the racial gap. While this point has been recognized in the literature (Baicker, Chandra and Skinner, 2005; Skinner et al., 2005), we add to it by developing a novel reweighting approach to decompose the contributions of markets and neighborhoods. Differences in where Black and white patients live between markets drive a nontrivial share, nearly one-half, of the gap in the 1990s. Yet by the same token, just over half of the disparity persists even within markets. Of this gap, about half reflects patients living in different ZIP codes and the other half reflects different hospital choices among patients in the same ZIP code. These findings reinforce the research on place-based and person-based policies by highlighting the performance of hospitals as important component of place and thus a potential focus of intervention (Bartik, 1991; Glaeser and Gottlieb, 2009). Our work complements literature recognizing the importance of local areas in impacting many economic outcomes such as educational attainment, intergenerational mobility, and life expectancy (Chetty et al., 2016, 2018).

Next, we found that between the 1990s and 2010s, the Black-white gap shrank by two-thirds. We developed a dynamic decomposition framework to determine whether this trend was driven by stronger reallocation of Black patients to better performing hospitals or stronger performance improvement among hospitals treating disproportionately Black patients. Our framework can be generalized to study the evolution of allocation-related disparities in other health care settings, like physicians or nursing facilities, as well as non-health contexts like schools, employers, and judges.

Given Chandra et al. (2016)'s findings that reallocation played an important role in improving overall patient outcomes over time, one might have suspected that reallocation to better hospitals acted to shrink the disparity, too. However, we did not find first-order evidence for that view, finding instead that differential performance improvement among hospitals that disproportionately treated Black patients led to the narrowing between-race gap in hospital performance. Lastly, we demonstrated that diffusion of a low-cost technology, beta-blockers, followed a similar, disparity-reducing path. This result suggests that take-up of beta-blockers and performance-improving strategies correlated with them may have been responsible for this progress. We note that our results do not establish that the diffusion of beta-blockers per se was responsible for improving outcomes at these hospitals; instead, we follow Chandra and Skinner (2012), Skinner and Staiger (2015), and other

work in viewing beta-blockers as a surrogate for a variety of TFP-improving changes that can include superior management and leadership in the hospital (Bloom et al., 2012; Tsai et al., 2015; Bloom, Sadun and Van Reenen, 2016).

More generally, our research connects a large literature on racial disparities in health care with the economics of productivity (Baily et al., 1992; Foster, Haltiwanger and Krizan, 2006; Melitz and Polanec, 2015) and the economics of medical innovation and diffusion (Chandra and Skinner, 2012; Chandra and Staiger, 2007; Skinner and Staiger, 2015). These connections raise the question of whether performance improvement and reallocation could be harnessed to address the disparities highlighted for AMI as well as disparities in other parts of the health care system with similar features. The power of the differential performance improvement channel over the past two decades suggests that direct efforts to increase the performance of hospitals in markets that serve a large share of Black patients can close the gap. These efforts would not change where patients receive treatment but could directly resource the hospitals they use. This approach aligns with Baicker, Chandra and Skinner (2005), who argued that place-based quality improvement efforts could address racial disparities.

While we showed that reallocation was a smaller force for stemming racial disparities in AMI, there is still scope to use this channel going forward. In AMI, for example, about half of remaining racial disparities originate from patients who live in the same ZIP code receiving treatment at different hospitals. This finding points to the potential for reallocative policies like efforts to change referral patterns or provider networks to close ongoing gaps. Hospital closure and entry, which are powerful forces for improvement in other sectors, could also play a role here – though the emergency nature of AMI necessitates more study of this channel.

Our approach focused on hospital performance for the typical patient, abstracting away from racial disparities in outcomes within the facility. By taking this approach, we are able to uncover a surprising decline in the gap in the performance of hospitals used by Black and white patients. While encouraging, this approach measures only one of the multitude of structural forces deteriorating health outcomes for Black patients at the point of care. Another key force comes from biases and discrimination on the part of practitioners. Regardless of whether they originate inside or outside the hospital, these forces can persist even as "between" hospital disparities close. The dynamics of disparities, whether arising within hospitals or from patient allocation between them, are a crucial

area for future research.

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Tables

Table 1 - Summary Statisics on Patients and Hospitals

Time period	Baseline (1995-1999)		Endline (2010-2014)	
Race	Black	White	Black	White
Panel A: Patients				
Survival, 30 Days	81.7%	81.2%	86.7%	85.8%
Age	76.8	77.6	77.7	79.3
	(7.5)	(7.3)	(8.2)	(8.2)
Female	59.8%	49.5%	58.7%	48.2%
Comorbid Conditions				
Heart Failure	18.2%	12.5%	24.2%	15.0%
Stroke	2.1%	1.2%	1.2%	0.6%
COPD	7.6%	7.5%	10.4%	8.9%
Count (out of 23)	1.3	0.9	1.9	1.3
	(2.0)	(1.7)	(2.6)	(2.2)
Patients	66,092	1,018,234	60,204	710,068
Panel B: Hospitals				
Survival, Risk-Adjusted, Std Deviation	(3.8pp)		(4.3pp)	
Patients per Hospital (Total)	411.2		297.4	
	(369.5)		(310.1)	
Patients per Hospital (by Race)	24.4	375.5	22.2	261.8
	(47.2)	(347.6)	(45.8)	(283.0)
Hospitals (≥1 Patient in Race Group)	2,181	2,711	2,120	2,706
Hospitals (Total)	2,712			

Notes: Panel A displays statistics about the sample of patients used to measure allocation and performance. Panel B shows statistics about the hospitals in our main analyses. All cells report averages or rates except those in parentheses which report standard deviations. See text for more details.

* Reports the standard deviation across hospitals of expected survival rate of the average Black or white patient, the measure of hospital performance described in equations 2 and 3 (whether measured for the average Black or white patient, this standard deviation is always equal in the model in our main specification). Adjusted for measurement error.

Table 2 - Static Decomposition of Racial Disparities in Hospital Allocation

	(1)	(2)	(3)	(4)	(5)	(6)
Time period	Baseline (1995-1999)		Endline (2010-2014)		Change	
	Value	Share	Value	Share	Value	Share
Between-Race Gap (Black-White)	-0.96	100%	-0.29	100%	0.66	100%
	(0.11)		(0.11)		(0.13)	
Components of Between-Race Gap						
Between Hospital Markets	-0.42	44%	-0.01	5%	0.40	61%
	(0.07)		(0.07)		(0.09)	
Between ZIP Codes (Within	-0.24	25%	-0.13	45%	0.10	15%
Hospital Markets)	(0.07)		(0.07)		(0.10)	
Within ZIP Codes	-0.30	32%	-0.15	50%	0.16	24%
	(0.05)		(0.05)		(0.06)	

N=2,712 hospitals. All values are percentage points (i.e. 1 indicates 1 percentage point) except for shares indicated with a % symbol. Notes: This table displays the between-race gap, or the difference in the average performance of hospitals used by Black and white patients, during the baseline period (columns 1 and 2), endline period (columns 3 and 4), and the change from baseline to endline (columns 5 and 6). Hospital performance is measured by the risk-adjusted survival rate. The decomposition uses reweighting to successively equalize the geographic distribution of Black and white patients at the hospital market (HRR) and ZIP code levels. It separates the between-race gap into a between hospital market component, a between ZIP code component, and a within ZIP code component. See text for more details. Bootstrapped standard errors using 1,000 replicates in parentheses.

Table 3 - Dynamic Decomposition of Racial Disparities in Hospital Allocation

	(1) Value	(2) Share
Change in Between-Race Gap from Baseline to Endline	0.66 (0.13)	100%
Components of Change		
Differential Performance Improvement	0.68 (0.15)	102%
Differential Hospital Reallocation	0.19 (0.08)	29%
Cross	-0.21 (0.09)	-31%

N=2,712 hospitals. All values are percentage points (i.e. 1 indicates 1 percentage point) except for shares indicated with a % symbol. Notes: This table displays and decomposes the change in the between-race gap from the baseline period to the endline period. The decomposition separates the change into differential performance improvement, or changes in performance associated with baseline Black-white allocation differences; differential hospital reallocation, or changes in Black and white patient allocation associated with baseline performance differences; and cross, or changes in allocation and performance co-occurring. See text for more details. Bootstrapped standard errors using 1,000 replicates in parentheses.

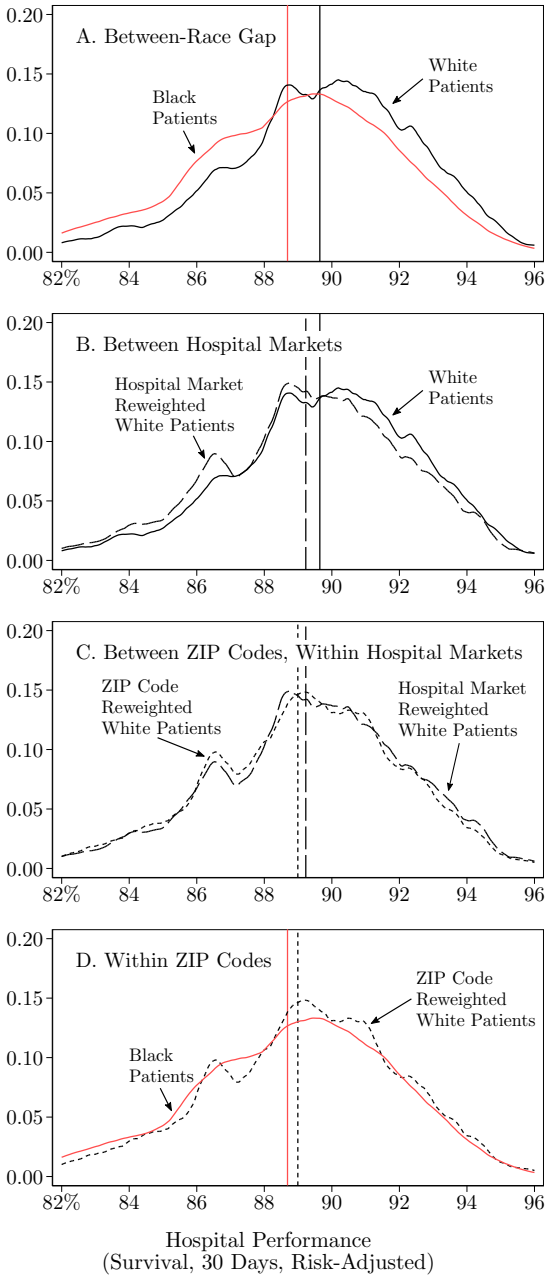
Table 4 - Decomposition of Hospital Technology Adoption

	(1) Beta-Blockers (Low-Cost)	(2) Cardiac Cath (High-Cost)
Avg at Baseline (All Patients)	37.3%	16.4%
Avg at Endline (All Patients)	98.5%	41.9%
Between-Race Gap (Black-White)		
Baseline	-1.70	0.65
	(0.56)	(0.35)
Endline	-0.23	-0.93
	(0.09)	(0.46)
Change	1.46	-1.58
	(0.56)	(0.39)
Components of Change		
Differential Performance Improvement	1.35	-0.64
	(0.58)	(0.32)
Differential Hospital Reallocation	0.59	-0.16
	(0.34)	(0.25)
Cross	-0.48	-0.78
	(0.36)	(0.21)
Hospitals	2,602	2,712

All values are percentage points (i.e. 1 indicates 1 percentage point) except for shares indicated with a % symbol. Notes: This table displays and decomposes the change in the between-race gap from the baseline period (1995-1999) to the endline period (2010-2014) for hospital technology adoption and use. The between-race gap is the Black - white rate; thus a negative value would indicate that Black patients receive treatment at less technology intensive hospitals than white patients. Column 1 analyzes the use of a low-cost technology, beta-blockers at discharge. Column 2 analyzes the use of a high-cost technology, cardiac catheterization, on the same day as admission. See text for more details. Bootstrapped standard errors using 1,000 replicates in parentheses.

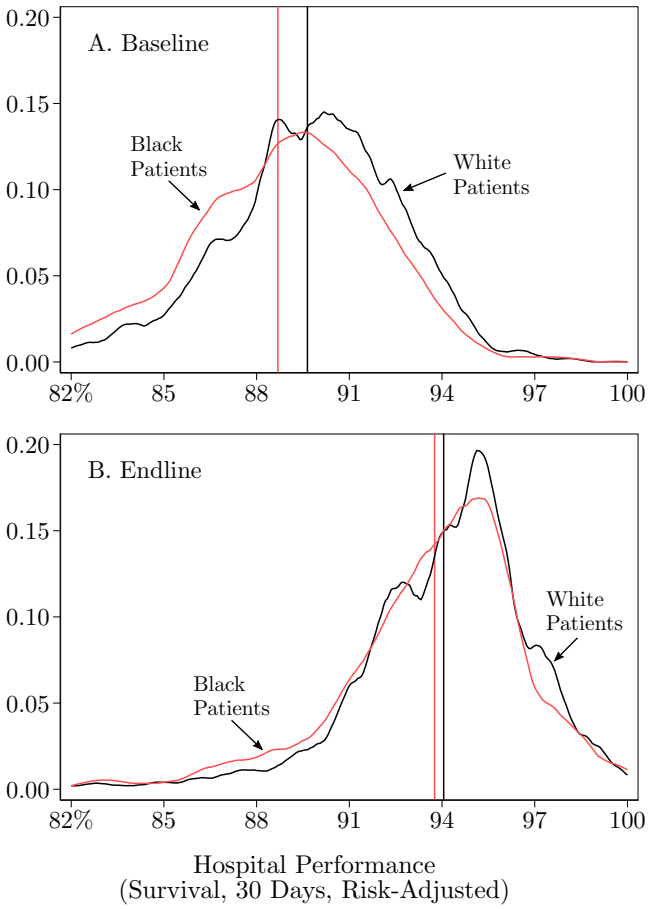
Figures

Figure 1: Decomposition of Between-Race Gap in Hospital Performance at Baseline



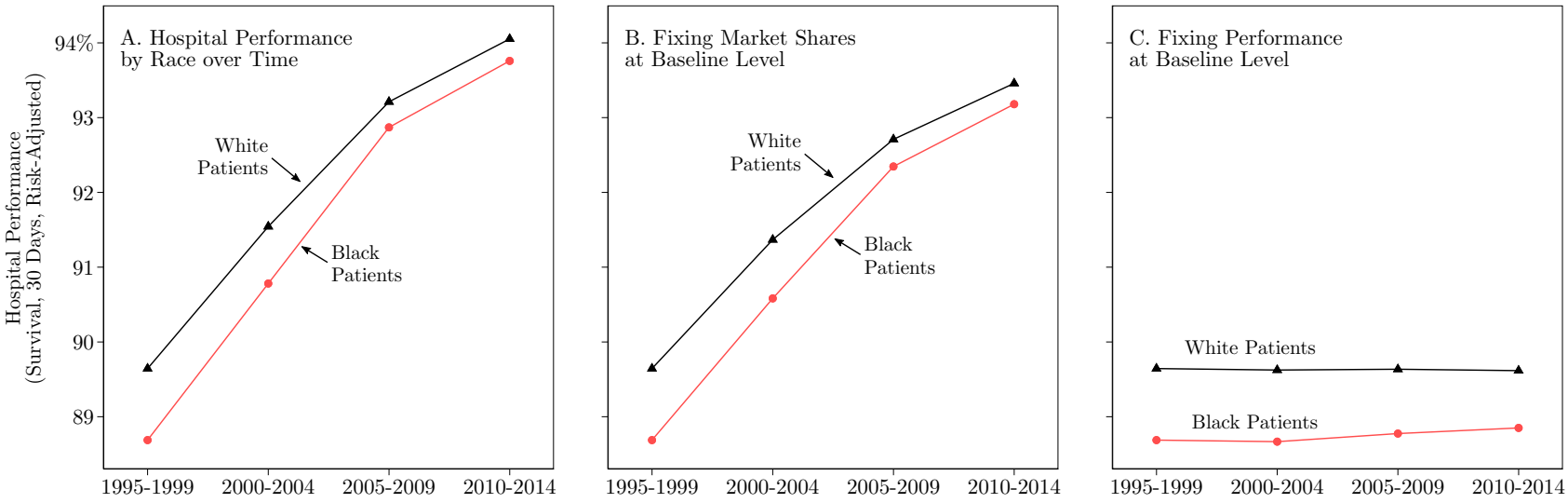
Note: This figure visualizes the static decomposition during the baseline (1995-1999) period. Each panel presents kernel densities of hospital performance for Black and white patients, with the vertical lines representing means. Panel A compares the actual densities for white and Black patients (the between-race gap). Subsequent panels reweight white patients to have the same geographic distribution as Black patients. Panel B compares densities for actual and market-level reweighted white patients (the between hospital markets term). Panel C compares densities for market- and ZIP code-level reweighted white patients (the between ZIP codes, within hospital markets term). Panel D compares the density for ZIP code-level reweighted white patients with the actual density for Black patients (the within ZIP codes term). See text for more details.

Figure 2: Between-Race Gap in Hospital Performance at Baseline and Endline



Note: This figure visualizes the between-race gap term of the static decomposition during the baseline (1995-1999) and endline (2010-2014) periods. Each panel presents kernel densities of hospital performance for Black and white patients, with the vertical lines representing means. See text for more details.

Figure 3: Decomposition of Between-Race Gap in Hospital Performance Over Time



Note: This figure visualizes the dynamic decomposition. Panel A shows the average hospital performance for Black and white patients in each period. The change in the Black-white gap from baseline (1995-1999) to endline (2010-2014) equals the change in between-race gap decomposed in Table 3. Panel B shows a counterfactual in which market shares remain at baseline levels but hospital performance evolves, depicting the differential performance improvement term. Panel C displays a counterfactual in which hospital performance remains at baseline levels but market shares evolve, depicting the differential reallocation term. See text for more details.

Appendix To:

Hospital Allocation and Racial Disparities in Health Care

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A Performance Measurement

Our main dataset is developed from 100% Research Identifiable Files (RIFs). Specifically, we use inpatient stays form the MEDPAR file and capture patient enrollment status and demographic factors from the Denominator and Master Beneficiary Summary Files from 1995-2014. We identify patients admitted to a hospital with AMI as MEDPAR records with a principal ICD-9 diagnosis code of 410, 410.X, 410.X0 - 410.X1, 410.X3-410.X9. Patients must be at least age 66 and carry full FFS (not Medicare Advantage) Part A and B coverage during the year prior to admission and up to one year following admission or death, whichever comes first. Only short-term acute care and critical access hospitals are included. Stays which were preceded by an AMI stay by the same patient in the year prior are omitted. As noted in the main text, we call the hospitalizations that satisfy these criteria index events.

Hospital performance is given by our estimates of risk-adjusted survival, which we define by estimating the following equation period-by-period:

$$s_{iht} = \alpha_t + \beta_t X_{iht} + \gamma_{ht} + \epsilon_{iht}, \tag{A1}$$

where s_{iht} is 30-day survival following hospital admission for patient i at hospital h in each period t, X_{iht} is a vector of patient controls, and γ_{ht} is a hospital fixed effect. In the main analyses, the vector contains indicators for patient comorbidities and demographics. The comorbidities are indicators for the presence of 23 conditions as given by diagnoses in inpatient stays in the year prior to the index event, counting principal and secondary diagnosis codes. The conditions captured are: unstable angina, chronic atherosclerosis, respiratory failure, hypertensive heart disease or heart failure, valvular heart disease, arrhythmia, hypertension, stroke, cerebrovascular disease, renal failure disease, dialysis, COPD, pneumonia, diabetes, protein calorie malnutrition, dementia, paralysis and disability, peripheral vascular disease, metastatic cancer, trauma, substance abuse, major psychiatric disorder, and chronic liver disease. The demographic indicators are age/race/sex interactions, where the age groups are 66-69, 70-74, 75-79, 80-84, 85-89, 90-94, and 95+, the race groups are Black, white, and other (neither Black nor white), and the sex groups are male and not male.

By extracting estimates of the fixed effects $\hat{\gamma}_{ht}$, this model yields performance measures for each hospital h in each period t:

$$\hat{q}_{ht}^{B} = \hat{\alpha}_t + \hat{\gamma}_{ht} + \bar{X}_t^{B}\hat{\beta}_t \tag{A2}$$

$$\hat{q}_{ht}^{W} = \hat{\alpha}_t + \hat{\gamma}_{ht} + \bar{X}_t^{W}\hat{\beta}_t, \tag{A3}$$

where $\hat{q}_{ht}^{B}$ and $\hat{q}_{ht}^{W}$ are expected 30-day survival rates for a Black and white AMI patients, respectively, at the given hospital-period, for a patient with control variables at the average for those in their race group and period ($\bar{X}_t^{B}$ and $\bar{X}_t^{W}$).

The standard deviation of $\hat{q}_{ht}^{B}$ (or equivalently $\hat{q}_{ht}^{W}$, since the two measures differ only by a constant) is expected to be greater than the standard deviation of true hospital performance q_{ht}^{B} due to measurement error. Table 1 reports a measurement error-adjusted standard deviation based on Chandra et al. (2016) and Sacarny (2018) to address this issue. We first form an adjusted variance estimate by taking the variance of the noisy performance measure $\hat{q}_{ht}^{B}$ and subtracting the average of the squared standard errors of the $\hat{\gamma}_{ht}$. The standard deviation reported in the table is the square root of this estimate.

Section 4.3 discusses results using several alternative approaches to estimate hospital performance. Four of these alternatives are modifications to the risk-adjustment approach. The **no risk adjustment** approach replaces X_{iht} with the null vector while the **age/race/sex adjustment** approach includes only the demographic (but not comorbidity) indicators in it. The **race-comorbidity interactions** approach interacts the 23 comorbidity indicators with two indicators for patient race (Black and other, i.e. non-Black non-white, with white race the omitted group), adding 46 indicators to X_{iht} in addition to the comorbidity main effects. Finally, the **AMI type** approach adds an indicator for ST-segment elevation myocardial infarction or not (STEMI vs. NSTEMI), a key marker that can inform treatment decisions.[6]

The remaining alternatives investigate whether our main results are driven by race-specific performance improvement at hospitals tending to serve Black patients. One specification estimates

[6] We measure NSTEMI events by the presence of an ICD-9 diagnosis code of 410.7x on the patient's index MEDPAR record. Prior to October 2005, these codes were not explicitly labeled NSTEMI but were still typically used to indicate such events (Steinberg et al., 2008).

hospital performance using white patient observations. Two additional alternatives use random effects rather than fixed effects. The first **random effects** model repeats our baseline fixed effects analysis treating γ_{ht} as a random effect and extracting BLUPs $\hat{\gamma}_{ht}$. The second estimates an alternative model with **race-specific hospital performance**. Specifically, we estimate the following specification:

$$s_{iht} = \alpha_t + \beta_t X_{iht} + \gamma^0_{ht} + Black_{iht} * \gamma^1_{ht} + other_{iht} * \gamma^2_{ht} + \epsilon_{iht}, \tag{A4}$$

where $\left(\gamma^0_{ht}, \gamma^1_{ht}, \gamma^2_{ht}\right)$ is a vector consisting of the hospital random effect and two race-specific hospital random slopes which are distributed Normal $(\mathbf{0}, \Omega)$. The model yields an estimate of Ω, the variance-covariance matrix of the random terms. Appendix Table A5 reports on relevant terms from elements Ω: the standard deviation of hospital performance for white patients (the γ^0_{ht} term in the equation), the standard deviation of the Black-white differential (the γ^1_{ht} term), and the correlation between these two elements. We also report the correlation between hospital performance for white and Black patients (Corr $\left[\gamma^0_{ht}, \gamma^0_{ht} + \gamma^1_{ht}\right]$). To construct race-specific performance, we extract BLUPs $\hat{\gamma}^0_{ht}$ and $\hat{\gamma}^1_{ht}$. Equations A2 and A3 are modified to replace race-agnostic performance $\hat{\gamma}_{ht}$ with race-specific performance $\hat{\gamma}^0_{ht} + \hat{\gamma}^1_{ht}$ (Black patients, equation A2) and $\hat{\gamma}^0_{ht}$ (white patients, equation A3).

B Decomposition with Entry and Exit

We assess the robustness of the decompositions to several alternative specifications in Appendix Section A, including one approach in which we drop the restriction that hospitals appear in both the baseline and endline periods. This approach permits hospitals to enter and exit the sample. The static decomposition only changes in that the market shares and summations are taken over all available hospitals in the period, H_t. The dynamic decomposition requires more extensive modification which yields terms that account for the role of net entry in the evolution of the Black-white gap. This approach is based on Foster, Haltiwanger and Krizan (2001) and yields the

following augmented decomposition:

$$\Delta\text{Between}_t = \sum_{h\in B}\left(\theta^B_{h,t-1} - \theta^W_{h,t-1}\right)\left(\Delta\hat{q}_{h,t}\right) \quad \leftarrow\text{Differential Performance Improvement} \tag{A5}$$

$$+ \sum_{h\in B}\left(\Delta\theta^B_{h,t} - \Delta\theta^W_{h,t}\right)\left(\hat{q}_{h,t-1} - \bar{q}_{t-1}\right) \quad \leftarrow\text{Reallocation} \tag{A6}$$

$$+ \sum_{h\in B}\left(\Delta\theta^B_{h,t} - \Delta\theta^W_{h,t}\right)\left(\Delta\hat{q}_{h,t}\right) \quad \leftarrow\text{Cross} \tag{A7}$$

$$+ \sum_{h\in N}\left(\theta^B_{h,t} - \theta^W_{h,t}\right)\left(\hat{q}_{h,t} - \bar{q}_t\right) \quad \leftarrow\text{Entry} \tag{A8}$$

$$- \sum_{h\in X}\left(\theta^B_{h,t-1} - \theta^W_{h,t-1}\right)\left(\hat{q}_{h,t-1} - \bar{q}_{t-1}\right), \quad \leftarrow\text{Exit} \tag{A9}$$

where B is the set of hospitals open in both periods, N is the set of entering hospitals, X is the set of exiting hospitals, and $\bar{q}_t$ is the market-share weighted average performance of hospitals open in t. The entry term captures the narrowing (widening) in the Black-white gap explained by Black patients using higher (lower) performing newly entering hospitals than white patients. The exit term enters negatively and captures the narrowing (widening) of the gap due to Black patients using lower (higher) performing exiting hospitals than white patients.

The baseline dynamic decomposition approach is not sensitive to the race group for which we measure performance because in our main specification, the measures $\hat{q}^B_{h,t}$ and $\hat{q}^W_{h,t}$ differ by a constant within a period and that constant drops from all summations. When the decomposition is augmented to allow for entry and exit, the findings are no longer necessarily algebraically identical for $\hat{q}^B_{h,t}$ and $\hat{q}^W_{h,t}$ because the constant no longer drops out of the summations. In practice, however, we found that the results were essentially unchanged with either performance measure.

C Measurement of Technology Adoption

C.1 Main analysis

We measure low- and high-cost technology adoption in Section 5 by tracking hospital use of beta-blockers and cardiac catheterization, respectively. The beta-blockers measure is given by the share of AMI patients receiving beta-blockers at hospital discharge among those for whom it is

appropriate. Unfortunately standard hospital administrative data does not provide information on most prescribing. Instead, we track use at baseline with a dataset of medical chart abstracts of Medicare AMI patients nationally in 1994-1995 called the Cooperative Cardiovascular Project. To observe their use at endline, we draw on public 2010-2014 hospital quality data from Medicare Hospital Compare.

We sought to match the CCP exclusion criteria to the Hospital Compare criteria to the greatest extent possible. Specifically, the Hospital Compare measure makes the following patient exclusions: <18 years of age, length of stay >120 days, enrolled in clinical trials, discharged to another hospital, died in hospital, left against medical advice, discharged for hospice care, documented to receive only comfort measures, or documented reason for not providing a beta-blocker at discharge (Centers for Medicare and Medicaid Services, 2020). Since the CCP data includes only Medicare FFS patients, we make no age exclusion in that data. To best match the remaining Hospital Compare criteria, we make the following CCP patient exclusions: length of stay >120 days, documented terminal illness, died during the hospital stay, discharged to another facility, or documented reason for not providing a beta-blockers. The aspirin measure presented in Appendix Table A6 uses the same approach and exclusions with the exception of the final one, which is changed to patients with a documented reason for not providing aspirin. More details on acceptable reasons for which a patient may be exempt from receiving beta-blockers (or aspirin) can be found in Marciniak et al. (1998).

The information needed to produce the cardiac catheterization measure is observable in Medicare claims. Specifically, we identify a patient as having received cardiac catheterization if one of the following ICD-9 procedure codes appeared in the MEDPAR record for their index admission: 37.21, 37.22, 37.23, 88.5, 88.5X, 00.66, 36.0, 36.00-36.02, 36.05-36.07, 36.09. In the main text, we analyze same-day catheterization, which only counts procedures that occurred on the patient's admission day. To produce the risk-adjusted measures, we estimate equations 1-3 with the catheterization indicator on the left-hand side rather than survival.

Appendix Table A6 considers an alternative measure that includes catheterization procedures performed on any day of the stay (i.e. the presence of any catheterization ICD-9 procedure on the index MEDPAR record). It also includes two additional measures that more broadly capture intensive intervention by including bypass surgeries, otherwise known as coronary artery bypass graft (CABG), performed on any day. We assess CABG intervention using the following ICD-9

procedure codes on the patient's index admission MEDPAR record: 36.03, 36.10-36.19,36.2, 36.3, 36.31-36.39.

D Inference in static and dynamic decompositions

We use a bootstrapping approach to estimate standard errors for all decomposition results. Specifically, we estimate standard errors using 1000 bootstrap replicates. All study analyses are based on summations of interactions between hospital market shares and hospital performance measures (or the first differences thereof). Thus, to construct each replicate sample, hospitals are drawn with replacement from the full sample. Then, in each sample, we re-scale all market shares so that they sum to 1 within-period while keeping the performance measures from the original patient-level sample.

Appendix Tables

Appendix Table A1 - Robustness of Static Decomposition to Alternative Risk Adjustment

Specification	A. Analysis in main text			B. No risk adjustment			C. Age/race/sex only		
Time period	Baseline	Endline	Change	Baseline	Endline	Change	Baseline	Endline	Change
Between-Race Gap (Black-White)	-0.96	-0.29	0.66	-0.63	-0.10	0.53	-1.04	-0.47	0.57
	(0.11)	(0.11)	(0.13)	(0.11)	(0.12)	(0.13)	(0.11)	(0.10)	(0.12)
Components of Between-Race Gap									
Between Hospital Markets	-0.42	-0.01	0.40	-0.32	-0.02	0.30	-0.48	-0.12	0.36
	(0.07)	(0.07)	(0.09)	(0.08)	(0.08)	(0.10)	(0.07)	(0.07)	(0.09)
Between ZIP Codes (Within	-0.24	-0.13	0.10	-0.05	0.04	0.09	-0.23	-0.16	0.07
Hospital Markets)	(0.07)	(0.07)	(0.10)	(0.07)	(0.08)	(0.09)	(0.07)	(0.07)	(0.09)
Within ZIP Codes	-0.30	-0.15	0.16	-0.26	-0.12	0.14	-0.34	-0.19	0.14
	(0.05)	(0.05)	(0.06)	(0.05)	(0.05)	(0.06)	(0.05)	(0.05)	(0.06)

Specification	D. Add race-comorbidity interactions			E. Add AMI type		
Time period	Baseline	Endline	Change	Baseline	Endline	Change
Between-Race Gap (Black-White)	-0.95	-0.31	0.64	-0.97	-0.42	0.54
	(0.11)	(0.10)	(0.13)	(0.12)	(0.10)	(0.14)
Components of Between-Race Gap						
Between Hospital Markets	-0.41	-0.01	0.40	-0.41	-0.09	0.32
	(0.07)	(0.07)	(0.09)	(0.07)	(0.07)	(0.09)
Between ZIP Codes (Within	-0.24	-0.14	0.09	-0.24	-0.14	0.10
Hospital Markets)	(0.07)	(0.07)	(0.09)	(0.07)	(0.07)	(0.10)
Within ZIP Codes	-0.30	-0.15	0.15	-0.32	-0.20	0.13
	(0.05)	(0.05)	(0.07)	(0.05)	(0.05)	(0.07)

N=2,712 hospitals. All values are percentage points (i.e. 1 indicates 1 percentage point). Notes: This robustness table displays estimates of the between-race gap (Table 2) under alternative approaches to risk-adjusting hospital performance. See main text and notes to Table 2 for more details on the decomposition and see Appendix Section A for more details on the risk-adjustment approaches. Panel A repeats the approach of the main text. Panels B and C present more limited approaches to adjustment while Panels D and E augment the model in the main text with additional covariates. Bootstrapped standard errors using 1,000 replicates in parentheses.

Appendix Table A2 - Robustness of Static Decomposition to Alternative Specifications

Specification	A. Analysis in main text			B. Performance estimated from white patients only			C. Random effects			D. Race-specific perf. model, white patients		
Time period	Baseline	Endline	Change	Baseline	Endline	Change	Baseline	Endline	Change	Baseline	Endline	Change
Between-Race Gap (Black-White)	-0.96	-0.29	0.66	-1.09	-0.58	0.51	-0.66	-0.26	0.40	-0.60	-0.25	0.35
	(0.11)	(0.11)	(0.13)	(0.18)	(0.17)	(0.20)	(0.09)	(0.08)	(0.10)	(0.08)	(0.07)	(0.09)
Components of Between-Race Gap												
Between Hospital Markets	-0.42	-0.01	0.40	-0.39	-0.01	0.39	-0.30	-0.02	0.28	-0.28	-0.02	0.25
	(0.07)	(0.07)	(0.09)	(0.07)	(0.07)	(0.09)	(0.06)	(0.06)	(0.07)	(0.06)	(0.05)	(0.07)
Between ZIP Codes (Within	-0.24	-0.13	0.10	-0.26	-0.30	-0.04	-0.15	-0.12	0.04	-0.14	-0.12	0.02
Hospital Markets)	(0.07)	(0.07)	(0.10)	(0.10)	(0.10)	(0.12)	(0.05)	(0.06)	(0.07)	(0.05)	(0.05)	(0.07)
Within ZIP Codes	-0.30	-0.15	0.16	-0.44	-0.28	0.16	-0.20	-0.12	0.09	-0.19	-0.11	0.07
	(0.05)	(0.05)	(0.06)	(0.12)	(0.08)	(0.13)	(0.04)	(0.04)	(0.05)	(0.04)	(0.03)	(0.04)
Hospitals		2,712			2,706			2,712			2,712	

Specification	E. Race-specific perf. model, black patients			F. Below median MA penetration			G. Unbalanced panel		
Time period	Baseline	Endline	Change	Baseline	Endline	Change	Baseline	Endline	Change
Between-Race Gap (Black-White)	-0.57	-0.16	0.41	-1.15	-0.39	0.77	-1.07	-0.32	0.75
	(0.08)	(0.07)	(0.09)	(0.16)	(0.13)	(0.18)	(0.11)	(0.10)	(0.13)
Components of Between-Race Gap									
Between Hospital Markets	-0.27	-0.02	0.25	-0.64	-0.16	0.48	-0.46	-0.03	0.43
	(0.06)	(0.05)	(0.07)	(0.12)	(0.09)	(0.13)	(0.07)	(0.07)	(0.09)
Between ZIP Codes (Within	-0.14	-0.07	0.07	-0.30	-0.11	0.19	-0.27	-0.13	0.13
Hospital Markets)	(0.05)	(0.05)	(0.07)	(0.11)	(0.09)	(0.13)	(0.07)	(0.07)	(0.09)
Within ZIP Codes	-0.17	-0.08	0.09	-0.22	-0.11	0.10	-0.34	-0.15	0.18
	(0.03)	(0.03)	(0.04)	(0.07)	(0.05)	(0.09)	(0.05)	(0.05)	(0.07)
Hospitals		2,712			1,199		3,998	2,877	

All values are percentage points (i.e. 1 indicates 1 percentage point). Notes: This robustness table displays estimates of the between-race gap (Table 2) under alternative approaches to estimating hospital performance or developing the sample of hospitals. See main text and notes to Table 2 for more details on the decomposition and see Appendix Section A for more details on the alternative approaches. Panel A repeats the approach of the main text. Panel B only uses hospitalization events from white patients (this excludes 6 hospitals with no white patients in either the baseline or endline period). Panel C uses random effects to estimate hospital performance. Panels D and E use performance measures derived from a random-effects model with race-specific performance. Panel F restricts to markets with below median Medicare Advantage penetration at endline. Panel G uses an unbalanced panel with all hospitals for which performance could be estimated in each period. Bootstrapped standard errors using 1,000 replicates in parentheses.

Appendix Table A3 - Robustness of Dynamic Decomposition to Alternative Risk Adjustment

	(A)	(B)	(C)	(D)	(E)
Specification	Analysis in main text	No risk adjustment	Age/race/sex only	Add race-comorbidity interactions	Add AMI type
Change in Between-Race Gap	0.66	0.53	0.57	0.64	0.54
from Baseline to Endline	(0.13)	(0.13)	(0.12)	(0.13)	(0.14)
Components of Change					
Differential Performance Improvement	0.68 (0.15)	0.92 (0.15)	0.67 (0.14)	0.66 (0.14)	0.64 (0.16)
Differential Hospital Reallocation	0.19 (0.08)	0.03 (0.09)	0.17 (0.08)	0.19 (0.08)	0.15 (0.08)
Cross	-0.21 (0.09)	-0.42 (0.098)	-0.27 (0.09)	-0.21 (0.08)	-0.24 (0.09)

N=2,712 hospitals. All values are percentage points (i.e. 1 indicates 1 percentage point). Notes: This robustness table displays and decomposes the change in the between-race gap (Table 3) under alternative approaches to risk-adjusting hospital performance. See main text and notes to Table 3 for more details on the decomposition and see Appendix Section A for more details on the risk-adjustment approaches. Column A repeats the approach of the main text. Columns B and C present more limited approaches to adjustment while Columns D and E augment the model in the main text with additional covariates. Bootstrapped standard errors using 1,000 replicates in parentheses.

Appendix Table A4 - Robustness of Dynamic Decomposition to Alternative Specifications

	(A)	(B)	(C)	(D)	(E)	(F)	(G1)	(G2)
Specification	Analysis in main text	Perf. est. from white patients	Random effects	Race-Specific Perf. Model		<Median MA penetration	Unbalanced panel	
				White Patients	Black Patients		q^B	q^W
Change in Between-Race Gap	0.66	0.51	0.40	0.35	0.41	0.77	0.75	0.75
from Baseline to Endline	(0.13)	(0.2)	(0.10)	(0.09)	(0.09)	(0.18)	(0.13)	(0.09)
Components of Change								
Differential Performance	0.68	0.63	0.41	0.38	0.38	0.64	0.46	0.45
Improvement	(0.15)	(0.21)	(0.10)	(0.09)	(0.09)	(0.19)	(0.16)	(0.10)
Differential Hospital	0.19	0.19	0.15	0.14	0.14	0.11	0.10	0.10
Reallocation	(0.08)	(0.11)	(0.05)	(0.05)	(0.05)	(0.11)	(0.07)	(0.08)
Cross	-0.21	-0.31	-0.16	-0.17	-0.11	0.02	0.08	0.10
	(0.09)	(0.12)	(0.05)	(0.05)	(0.05)	(0.13)	(0.12)	(0.07)
Entry							-0.12	-0.13
							(0.02)	(0.02)
Exit (enters negatively)							-0.22	-0.22
							(0.04)	(0.03)
Hospitals	2,712	2,706	2,712	2,712	2,712	1,199	4,163	4,163

All values are percentage points (i.e. 1 indicates 1 percentage point). Notes: This robustness table displays and decomposes the change in the between-race gap (Table 3) under alternative approaches to estimating hospital performance or developing the sample of hospitals. See main text and notes to Table 3 for more details on the decomposition and see Appendix Section A for more details on the alternative approaches. Column A repeats the approach of the main text. Column B only uses hospitalization events from white patients (this excludes 6 hospitals with no white patients in either the baseline or endline period). Column C uses random effects to estimate hospital performance. Columns D and E use performance measures derived from a random-effects model with race-specific performance. Column F restricts to markets with below median Medicare Advantage penetration at endline. Columns G1 and G2 use an unbalanced panel with all hospitals for which performance could be estimated in each period, emitting entry and exit terms; this approach is sensitive to the reference group and so we show it using performance for Black and white patients derived from the main text (not the random-effects model), respectively. Bootstrapped standard errors using 1,000 replicates in parentheses.

Appendix Table A5 - Race-Specific Performance Model

Model statistic		Baseline (1995-1999)	Endline (2010-2014)
Standard deviation of performance for white patients	$\mathrm{SD}(\gamma_{ht}^{0})$	2.94	2.63
Standard deviation of Black-white performance differential	$\mathrm{SD}(\gamma_{ht}^{1})$	1.31	1.50
Correlation between performance for white patients and Black-white differential	$\mathrm{Corr}(\gamma_{ht}^{0}, \gamma_{ht}^{1})$	-0.21	-0.32
Correlation between performance for white patients and Black patients	$\mathrm{Corr}(\gamma_{ht}^{0}, \gamma_{ht}^{0} + \gamma_{ht}^{1})$	0.90	0.83

Units for standard deviation estimates are percentage points (i.e. 1 indicates 1 percentage point). Notes: This table presents statistics from a random effects model with race-specific performance. The model includes a hospital random effect (yielding performance for white patients) and two race-specific random slopes (yielding performance differentials between the race groups and white patients). These statistics are derived from estimates of the model's variance-covariance matrix. See Appendix Section A for more details.

Appendix Table A6 - Decomposition of Additional Technology Adoption Measures

	(1) Cardiac Cath (Any Day)	(2) Cath or CABG (Any Day)	(3) CABG (Any Day)	(4) Aspirin
Avg at Baseline (All Patients)	53.8%	54.4%	9.9%	58.4%
Avg at Endline (All Patients)	78.9%	79.7%	8.8%	98.9%
Between-Race Gap (Black-White)				
Baseline	3.53	3.74	0.80	1.75
	(0.68)	(0.72)	(0.28)	(0.61)
Endline	0.20	0.41	-0.02	-0.15
	(0.57)	(0.61)	(0.17)	(0.06)
Change	-3.33	-3.33	-0.81	-1.90
	(0.57)	(0.57)	(0.23)	(0.60)
Components of Change				
Differential Performance	-1.56	-1.63	-0.60	-1.84
Improvement	(0.50)	(0.5)	(0.21)	(0.59)
Differential Hospital	-1.01	-0.96	-0.13	-1.05
Reallocation	(0.45)	(0.46)	(0.18)	(0.38)
Cross	-0.76	-0.75	-0.08	1.00
	(0.32)	(0.33)	(0.13)	(0.37)
Hospitals	2,712	2,712	2,712	2,535

All values are percentage points (i.e. 1 indicates 1 percentage point) except for shares indicated with a % symbol. Notes: This table displays decompositions of additional technology measures beyond those presented in the main text. See text and notes to Table 4 for more details. Columns 1-3 present alternative measures of intensive intervention: receipt of cardaic catheterization on any day of the index admission, receipt of cardiac catheterization or coronary artery bypass graft (CABG) on any day, and receipt of CABG on any day, respectively. Column 4 presents adoption of aspirin at discharge, which is calculated simularly to the approach for beta blockers. Bootstrapped standard errors using 1,000 replicates in parentheses.