

# TECHNOLOGY DIFFUSION AND PRODUCTIVITY GROWTH IN HEALTH CARE

Jonathan Skinner and Douglas Staiger\*

**Abstract**—We draw on macroeconomic models of diffusion and productivity to explain empirical patterns of survival gains in heart attacks. Using Medicare data for 2.8 million patients from 1986 to 2004, we find that hospitals rapidly adopting cost-effective innovations such as beta blockers, aspirin, and reperfusion had substantially better outcomes for their patients. Holding technology adoption constant, the marginal returns to spending were relatively modest. Hospitals increasing the pace of technology diffusion (“tigers”) experienced triple the survival gains compared to those with diminished rates (“tortoises”). In sum, small differences in the propensity to adopt effective technology lead to wide productivity differences across hospitals.

## I. Introduction

THERE were large regional differences in per capita price-adjusted 2010 U.S. Medicare expenditures, ranging from \$6,911 in Lacrosse, Wisconsin, to \$13,824 in McAllen, Texas. Yet the evidence is mixed on whether more spending is associated with better outcomes (e.g., Fisher et al., 2003b; Skinner, Fisher, & Wennberg, 2005; Doyle, 2011; Doyle et al., 2015), and some have estimated the level of waste in health care spending equals 3% of GDP or more (Cutler et al., 2013; Fisher et al., 2003a, 2003b). This lack of association between spending and outcomes has sometimes been interpreted as “flat-of-the-curve” health care spending, or variations along a common production function with a very low or zero marginal value of health care spending.<sup>1</sup>

The flat-of-the-curve explanation is problematic for many reasons. Some studies find a negative association between state-level quality measures and per capita Medicare expenditures (Baicker & Chandra, 2004). Why should spending more be associated with providing worse quality care? Second, given results from Cutler et al. (1998), Berndt et al. (2002), and others that over time, survival and functioning have improved because of often expensive new medical technology, it would be surprising if the “wasted” health care spending equal to 3% of GDP should provide no benefit whatsoever.

In this paper, we draw on macroeconomic models of productivity to provide a better explanation for this empirical puzzle. That differential rates of technology adoption can explain long-term variations in per capita GDP across coun-

tries is by now well understood. Crespi et al. (2008) found as much as 50% of total factor productivity growth arises simply from the flow of knowledge across firms. Parente and Prescott (1994, 2002) showed that surprisingly small differences in the rates of technological adoption could imply large disparities in country levels of income, while Eaton and Kortum (1999) estimated that countries realized just two-thirds of the potential productivity gains because of the slow diffusion and adoption of ideas across borders (see also Hall, 2004; Comin & Hobijn, 2010; Comin & Mestieri, 2013).

A parallel literature in health care documents similar lags in adoption and with similar adverse effects on overall productivity. Despite powerful evidence from a 1601 experiment demonstrating the effectiveness of lemon juice in preventing scurvy, the British Navy did not require foods containing vitamin C until 1794 (Berwick, 2003).<sup>2</sup> Similarly, beta blockers, drugs costing pennies per dose, were shown during the early 1980s to reduce mortality by as much as 25% following a heart attack (Yusuf et al., 1985), yet by 2000/2001 median state-level use was still only 68% (Jencks, Huff, & Cuerdon, 2003).

We develop a general model in which hospitals and physicians seek to maximize the health of their patients by adopting new technologies in the face of financial and knowledge-based barriers. Variation across hospitals in these barriers leads to differences in the diffusion rate of new technologies. We apply this model to the treatment of patients diagnosed with an acute myocardial infarction (AMI, or a heart attack) during the period 1986 to 2004, a time of particularly rapid diffusion for new technologies.

We first consider three types of innovations that are highly effective and highly cost-effective in saving lives following AMI: aspirin, beta blockers, and reperfusion within 12 hours of the heart attack. (Reperfusion consists either of thrombolytic clot-busting drugs, or percutaneous coronary interventions, PCI, also known as angioplasty).<sup>3</sup> We also consider the diffusion of technology, first introduced in 2003, drug-eluting stents, to identify how changes over time in the speed with which new (and valuable) hospital innovations diffuse affect health outcomes. Finally, we test whether hospitals adopting these three highly effective treatments also adopted less cost-effective technologies

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\* Skinner and Staiger: Dartmouth College and NBER.

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<sup>1</sup> See, e.g., Fuchs (2004) and Enthoven (1978).

<sup>2</sup> In his 1601 voyage to India, Captain James Lancaster fed sailors in one of his ships three teaspoons of lemon juice every day, while in the other three ships, no lemon juice was provided. By the midpoint of the journey, 110 of the 278 sailors in the control group had died of scurvy (40%), while none of the sailors in the treatment group had been affected (Berwick, 2003).

<sup>3</sup> Strictly speaking, neither aspirin nor beta blockers were “innovations”; they had been in use for decades. The innovation was to use these drugs in treating AMI patients.

such as lidocaine, a drug with initially favorable results but whose effectiveness was questioned in the late 1980s, “late” surgical angioplasty (PCI) more than 24 hours after the AMI (again with less clear clinical effectiveness), and coronary artery bypass graft surgery (CABG).

The model is tested using a sample of 2.8 million heart attack patients drawn from the fee-for-service Medicare population from 1986 to 2004. Like Eaton and Kortum’s (1999) study of aggregate productivity, we find substantial differences in the extent to which some hospitals lag behind in the diffusion of highly effective technologies and that this differential lag can explain a nearly 3 percentage point difference in one-year survival between rapid-diffusing and slow-diffusing hospitals, almost one-third of the overall improvement in outcomes from 1986 to 2004. These productivity effects swamp the influence of differences in factor inputs, a result also found in the macroeconomics literature (Hall & Jones, 1999). We demonstrate that the “Asian tiger” hospitals, which between 1994–1995 and 2003–2004 demonstrated dramatic improvements in diffusion rates, also experienced above-average survival growth and three times the growth of the “tortoise” hospitals that experienced a decline in diffusion rates.

Finally, we found evidence that hospitals investing in highly effective medical innovations (aspirin, beta blockers, dropping the use of lidocaine) were quite different from those continuing the use of lidocaine and, most notably, adopting a mix of less cost-effective surgical innovations (early reperfusion, late PCI angioplasty, bypass surgery).<sup>4</sup> Consistent with Hall (2014), the survival benefits arising from the more effective medical innovations are estimated to be larger than those arising from the less effective mix of treatments. This evidence suggests that barriers to the diffusion of knowledge about the effectiveness of new technologies play an important role in explaining productivity differences across hospitals, rather than barriers to the adoption of new technologies per se.

These results can potentially reconcile two seemingly divergent views of the U.S. health care system. Technological progress has led to dramatic improvements in survival for heart attack patients (as in Cutler, 2004), but these improvements are largely associated with the adoption of effective new technologies rather than more factor inputs (Chandra & Skinner, 2012). Holding technology diffusion constant, however, we find modest improvements in outcomes associated with greater factor inputs, with a preferred estimate of between \$94,000 and \$155,000 per life-year. Chandra et al. (2013) found similar variation in productivity across firms in nonhealth industries as in hospitals, suggesting that the differences across firms in technology adoption may not be unique to health care. The real puzzle, therefore, is why many physicians and hospitals—and firms

more generally—fail to adopt highly efficient (or even modestly efficient) innovations.

## II. The Model

We focus on the “production” of survival following AMI. There are compelling reasons to focus on heart attacks. Nearly every AMI patient who survives the initial attack is admitted to a hospital, and ambulance drivers generally take the patient to the nearest hospital (although see Doyle et al., 2015). The outcome, survival, is accurately measured, and there is broad clinical agreement that survival is the most important end point, particularly in the elderly population. The measurement of inputs is also accurate, as is risk adjustment, including the type of heart attack. Finally, many of the studies focusing on the value of medical technology have used AMI as an example (Cutler et al., 1998; Cutler, 2004; Doyle, 2011).

### A. The Hospital Production Function

We develop a simple model of hospital productivity that distinguishes between inputs that require substantial contributions of capital and labor (e.g. hospital bed-day or surgical procedures) and technology innovations where barriers are unlikely to arise solely from financial constraints. Suppose that medical care per patient (e.g., quantity of medical services) at hospital  $i$  in year  $t$  ( $X_{it}$ ) is produced with constant returns technology,

$$X_{it} = h l_{it}^{\xi} k_{it}^{1-\xi}, \quad (1)$$

where  $l_{it}$  and  $k_{it}$  represent labor and capital inputs per patient at hospital  $i$  in year  $t$ , and  $h$  is a constant measure of productivity in producing  $X$ . Letting  $r$  denote the cost of capital and  $w$  the wage rate, the efficient marginal expenditure per  $X$  (the implicit price) is  $P_{it} = h^{-1} \left( \frac{w_{it}}{\xi} \right)^{\xi} \left( \frac{r_{it}}{1-\xi} \right)^{1-\xi}$ .

Because our data measure  $X_{it}$  more accurately than capital and labor inputs, we focus on the composite factor input rather than on capital and labor separately.<sup>5</sup>

While it seems reasonable to assume constant returns for producing medical care services (doubling staff and beds at a hospital can produce twice the number of admissions), we assume that medical care per patient has declining returns in terms of patient survival (or quality-adjusted life years). We assume initially a simple production function that specifies a linear relationship between survival per patient ( $y_{it}$ ), the log of composite medical care inputs  $x_{it} = \ln(X_{it})$ , and the level of technology at hospital  $i$  at time  $t$ ,  $a_{it}$ ,

$$y_{it} = a_{it} + \beta x_{it}. \quad (2)$$

<sup>4</sup> The distinction between medical and surgical treatments for AMI follows Chandra and Staiger (2007).

<sup>5</sup> In theory one could measure physical inputs as hospital days and physician resource-value units (RVUs), but neither captures treatment intensity. See Jacobs, Smith, and Street (2006) for an excellent discussion of productivity in the quantity of medical services,  $X$ .

We adopt this special case to simplify the modeling of our balanced-growth path of technological innovation.

### B. The Diffusion of Technology

Technology is modeled as the sum of many separate innovations, and for simplicity we assume a model of certainty in which one new innovation becomes available each year. Letting  $j$  index the year the innovation first appeared yields

$$a_{it} = \sum_{j=1}^t \alpha_j m_{jit}. \quad (3)$$

In equation (3),  $m_{jit}$  is the fraction of appropriate patients at hospital  $i$  receiving treatment  $j$  (or the proportion of physicians who have adopted innovation  $j$ ) by time  $t$ , while  $\alpha_j$  is the return to adopting innovation  $j$ . The diffusion rate in turn is written as

$$m_{jit} = m_{jit-1} + \pi_{jit}(1 - m_{jit-1}). \quad (4)$$

This year's usage rate  $m$  is equal to last year's rate plus the institutional- and innovation-specific diffusion rate  $\pi_{jit}$  times the gap between best-practice (100% use among appropriate patients) and last year's usage.

The frontier technology available at time  $t$ ,  $a_t^*$ , is the technology that could be achieved if a hospital had fully adopted all innovations available:

$$a_t^* = \sum_{j=1}^t \alpha_j. \quad (5)$$

To solve the dynamic model below, we collapse the entire matrix of innovation-specific diffusion rates  $\pi_{it} = \{\pi_{1it}, \pi_{2it}, \dots, \pi_{kit}\}$  into a common "core" diffusion rate  $\pi_{it}$  for hospital  $i$ . Combining equations (3) to (5) to express the technology level at a given point in time is

$$a_{it+1} = a_{it} + \pi_{it}(a_t^* - a_{it}). \quad (6)$$

Equation (6) is the Nelson-Phelps (1966) partial adjustment model for productivity, where the diffusion rate ( $\pi_{it}$ ) determines the rate of partial adjustment in productivity toward the frontier that is achieved each year.

### C. The Hospital Objective Function

There is considerable debate about the objective function of hospitals (e.g., Horwitz & Nichols, 2007). To avoid having to choose a specific model, we instead adopt a general objective function depending positively on survival and profitability,

$$V_t = \sum_{t=0}^{\infty} [\Psi_i(a_{it} + \beta x_{it}) - \phi_i P_{it} X_{it} - C_i(\pi_{it}) + K_{it}] \times (1+r)^{-t}, \quad (7)$$

where  $r$  is the discount rate,  $\Psi_i$  is the implicit social (dollar) value of improved health (assumed for simplicity to be constant over time), and  $K_{it}$  represents either fixed costs or subsidization from endowments or non-Medicare patient revenue. If the hospital were acting to maximize social benefit,  $\phi_i$  would equal 1, but increasing certain inputs (e.g., cardiac surgery) could improve profitability; thus in general  $\phi_i \leq 1$ . As we show in the online appendix, other models of hospital behavior reflecting the tension between financial profits and social welfare imply values of either  $\Psi_i$  below the value corresponding to a social planner, or  $\phi_i < 1$ , or both.

We assume that there is a cost of diffusion, equal to  $C_i(\pi_{it})$ , with  $C' > 0$  and  $C'' > 0$ . The costs may include the obvious expenses of, say, computerized information systems that prompt physicians when beta blockers or aspirin have not been administered, quality improvement initiatives, fixed costs of new technologies such as catheterization laboratories, or higher wages and research time to help recruit smarter or more technically skilled physicians (Bero et al., 1998; Bradley et al., 2001). These costs (and marginal costs) are likely to differ substantially across hospitals and will likely reflect physician search costs, the quality of institutional leadership, and other factors affecting the speed of diffusion (Rogers, 2003; Bradley et al., 2001; Phelps, 2000).

### D. Solving the Dynamic Model

The maximization is subject to the equations denoting the evolution of technology over time and is expressed as a discrete-time Lagrangian:<sup>6</sup>

$$\mathfrak{L} = V_i - \sum_{t=0}^{\infty} \lambda_{it} [a_{it+1} - a_{it} - \pi_{it}(a_{t+1}^* - a_{it})]. \quad (8)$$

Under constant productivity growth, where  $\alpha_t = \alpha$  and  $a_{t+1}^* = a_t^* + \alpha$ , the first-order conditions (shown in appendix equations A.4a through A.4d) yield a dynamic steady-state path with an equilibrium (and stable) diffusion rate  $\pi_{it} = \pi_i$  that is constant over time.

From the first-order conditions, optimal factor inputs are given by

$$X_{it} = \Psi_i \beta / P_{it} \phi_i. \quad (9)$$

<sup>6</sup> This model can also be written in continuous time as a current-value Hamiltonian, but we maintain a discrete time structure to be consistent with the empirical data.

Not surprisingly, factor inputs are greater when there is a higher implicit value by the hospital on saving a life-year  $\Psi_i$ , there is a higher return to factor inputs  $\beta$ , when the price of producing a factor input  $P_{it}$  is lower, and when financially motivated hospitals are reimbursed generously for care ( $\phi_i$  is small).<sup>7</sup>

For a constant growth rate  $\alpha$ , it is straightforward to show that productivity in the steady state is given by

$$a_{it} = a_t^* - \alpha \left( \frac{1 - \pi_i}{\pi_i} \right). \quad (10)$$

Equation (10) states that the steady-state distance that a hospital lags behind the productivity frontier is a constant nonlinear function of the steady-state diffusion rate ( $\pi_i$ ), in which small differences in diffusion can lead to very large differences in productivity (Parente & Prescott, 1994). Note that the term  $(1 - \pi_i)/\pi_i$  can be interpreted as the number of years a hospital lags behind the frontier (since  $\alpha$  is annual productivity growth). Thus, a hospital with a 20% diffusion rate lags four years behind, and a hospital with a 5% diffusion rate lags nineteen years behind. Equation (10) also implies that there is no convergence; productivity at all hospitals grows at the same rate as the frontier  $-\alpha$ . This property has been noted in other papers as well (Eaton & Kortum, 1999) and is a consequence of the Nelson-Phelps (1966) partial adjustment model implied by equation (6).

Finally, the optimal diffusion rate is chosen to set its marginal cost equal to its marginal benefit:

$$C'_i(\pi_i) = [\Psi_i(a_t^* - a_{it})]/(r + \pi_i). \quad (11)$$

The numerator of the right-hand side of equation (11) measures the immediate benefit, in dollar terms, of moving to the frontier today, while the denominator converts this to the present value, as the value of today's innovation decays in the future. Equation (11) shows that differences across hospitals in rates of diffusion are implicitly determined by corresponding variations in the marginal cost of adopting those new technologies.

### III. Empirical Specification of the Model

We now translate the theoretical model to a stochastic specification with measurement error. We rewrite equation (2) but add an error term  $u_{it}$  without yet making any claims for its statistical properties:

$$y_{it} = a_{it} + \beta x_{it} + u_{it}. \quad (12)$$

<sup>7</sup> Optimal factor inputs are independent of the level of technology because the production function, equation (2), assumes that the marginal product of factor inputs ( $\beta$ ) does not depend on technology. In a more general specification that allowed interactions between technology and factor inputs, optimal factor inputs would increase (decrease) if new technology increased (decreased) the marginal product of factor inputs.

Using the steady-state assumption from equation (10), equation (12) is rewritten as

$$y_{it} = a_t^* - \alpha \left[ \frac{1 - \pi_i}{\pi_i} \right] + \beta x_{it} + u_{it}. \quad (13)$$

This suggests a very simple estimation model, regressing survival ( $y_{it}$ ) on log inputs ( $x_{it}$ ), a linear trend (or year fixed effects) to reflect growth over time in the frontier  $a_t^*$ , and a variable reflecting the hospital-specific rate of diffusion  $\pi_i$ . However, several challenges remain:  $x_{it}$  and  $y_{it}$  must be constructed from individual-level data;  $\pi_i$  is not directly observable and must be estimated, and may change over time; the linear estimation equation may be too restrictive; and  $u$  could be correlated with  $x$ . We consider each of these issues in turn.

#### A. Creating Hospital-Level Survival and Input Measures

We create hospital-level measures of survival and factor inputs from the individual data in the Medicare claims data. Let one-year mortality following a heart attack be expressed as

$$S_{lit} = Z_{lit}\Gamma + \sum_{i=1}^H \gamma_{it} + e_{lit}. \quad (14)$$

The dependent variable,  $S_{lit}$  is a 1-0 variable reflecting whether the individual  $l$  who had an AMI in year  $t$  (and was admitted to hospital  $i$ ) survived for at least one year, with  $Z_{lit}$  a matrix of individual risk adjusters,  $\Gamma$  a vector of coefficient,  $\gamma_{it}$  a vector of hospital-year specific intercepts, and  $e_{lit}$  the error term. Similar equations are also estimated for two measures of total factor inputs in the year following the heart attack: hospital expenditures (in constant 2004 dollars) and the sum of diagnostic-related group (DRG) weights across all hospital admissions, which reflect the Centers for Medicare and Medicaid Services (CMS) assessment of resources necessary for specific services. The hospital-year intercepts from equation (14),  $\gamma_{it}$ , are used in our subsequent estimation as risk-adjusted measures of survival and factor inputs.

#### B. Estimating Each Hospital's Rate of Diffusion

We use data on the adoption of various innovations at a point in time to estimate each hospital's rate of diffusion ( $\pi_i$ ). In steady state, equation (4) implies that the current rate of use ( $m_{jit}$ ) of an innovation  $j$  depends simply on the number of years it has been available ( $t - j$ ) and the hospital-specific rate of diffusion ( $\pi_i$ ), where  $m_{jit} = 1 - (1 - \pi_i)^{t-j}$ . Taking a first-order approximation so that  $1 - (1 - \pi_i)^{t-j} \approx (t - j)\pi_i$  and adding a stochastic term ( $v_{jit}$ ) to allow for random fluctuations over time allows us to express  $m_{jit}$  as

$$m_{jit} = (t - j)\pi_i + v_{jit}. \quad (15)$$

The structure in equation (15) is consistent with a factor model in which the adoption rate of all technologies in a

given year depends on a common factor ( $\pi_i$ ) that captures each hospital's rate of diffusion, and the factor loading for each technology ( $t - j$ ) reflects the length of time the innovation has been available. Therefore, we fit a factor model to hospital-level data on the adoption rate of various innovations and use the prediction of the common factor as a proxy for each hospital's underlying diffusion rate.

There are two approaches to estimating the influence of this diffusion parameter on survival in equation (13). One is to simply enter the common factor, proportional to  $\pi_i$  (but normalized to have mean 0 and standard deviation 1) linearly, or to estimate the model by creating quintiles of hospitals according to their estimated  $\pi_i$ . Second, in some specifications of equation (13), we include hospital fixed effects to proxy for each hospital's specific diffusion parameters. Hospital fixed effects do not provide a direct estimate of how diffusion is associated with patient survival, but they avoid concerns about poorly measured estimates of  $\pi_i$ , yielding estimates of  $\beta$  less subject to omitted variable bias.

### C. Relaxing the Assumption of a Steady-State Model

If hospitals are not in steady state (e.g., because of changing costs of diffusion), then  $\pi_i$  will not be constant over time, leading to changing rates of diffusion. A rising diffusion parameter  $\pi_i$  for effective innovations will tend to accelerate health outcomes relative to its initial steady state, and the converse. We will therefore test empirically whether risk-adjusted survival rates of hospitals experiencing large improvements in measured diffusion (the productivity tigers) are higher than those at hospitals experiencing downward shifts (the productivity tortoises).

### D. The Error Term Could Be Correlated with Factor Inputs

Estimates of the return to factor inputs ( $\beta$ ) in equation (13) may be biased by correlation between factor inputs ( $x_{it}$ ) and the error term, whether because hospitals with a greater degree of unobserved efficiency (as reflected in the error term) may also experience greater skill in the use of  $x_{it}$  (Chandra & Staiger, 2007) or because of unobservable technological innovations that make inputs  $x_{it}$  more productive. The typical approach to this problem is to instrument for inputs, but we could not think of a plausible instrument that affected  $x_{it}$  but not productivity. Thus we interpret the estimate of  $\beta$  with caution.

A second cause for  $x_{it}$  to be correlated with the error term arises from our construction of  $y_{it}$  and  $x_{it}$  from individual data. Small numbers of people in each hospital-year observation could create a spurious positive correlation between  $y_{it}$  and  $x_{it}$ , given that (as we find in the data) people who live longer on average account for more spending. To address this issue, we also present estimates that limit the sample size to hospitals with at least fifty patients.

### E. The Cost-Effectiveness Ratio

To provide a basis for comparison with other studies, we also calculated the cost-effectiveness (CE) ratio, or the cost per life-year gained, defined as

$$CE = \frac{dC}{dX} \bigg/ \left[ \frac{dy}{dX} \frac{dL}{dy} \right], \quad (16)$$

where  $X$  measures DRGs (and  $dC/dX$  is the cost per DRG,)  $y$  is the probability of surviving one year,  $dy/dX$  is derived from the regression estimate, and  $dL/dy$ , the change in life expectancy conditional on surviving an extra year, is set to 5.25 based on estimates in Cutler et al. (1998).

There is some debate over the appropriate hurdle for whether a treatment is cost-effective. Generally values below \$100,000 per life year pass muster, although some clinical willingness-to-pay estimates are well below \$50,000 (King et al., 2005). Conversely, economists typically favor much larger estimates, of up to \$250,000 per life year for older people (Hirth et al., 2000; Murphy & Topel, 2006).

## IV. The Diffusion of Efficient Treatments for Acute Myocardial Infarction

### A. Data on the Treatment of Patients

The 1990s were marked by fundamental changes in the treatment of AMI. Technology diffusion was measured in the Cooperative Cardiovascular Program (CCP) data set, which involved chart reviews for over 160,000 AMI patients over age 65 during 1994–1995, matched to the admitting hospital (Chandra & Staiger, 2007). We consider three innovations resulting in major reductions in cardiovascular disease between 1980 and 2000 (Ford et al., 2007).

The first, aspirin, reduces platelet aggregation and helps to limit clotting, thereby improving blood flow to oxygen-starved tissue. By 1988 it was included in standard guidelines for care (ISIS-2, 1988).

The second, a beta blocker, is an inexpensive drug that blocks the beta-adrenergic receptors and reduces the demands on the heart. In a meta-analysis from 1985, Yusuf et al. summarized the existing literature: “Long-term beta blockade for perhaps a year or so following discharge after an MI is now of proven value, and for many such patients mortality reductions of about 25% can be achieved” (p. 335).

The third measure is reperfusion within 12 hours of the AMI. Reperfusion, or restoring blood flow to the oxygen-starved heart muscles, can be effected using thrombolytics, drugs that help break down the clots blocking the blood, or a percutaneous coronary intervention (PCI) in which a “balloon” is threaded into the blocked artery and expanded, thus restoring blood flow. Each was considered a highly effective treatment strategy at the time (Ryan et al., 1993). Since 1995, cardiologists have increasingly adopted stents, cylindrical wire meshes, to maintain blood flow following

TABLE 1.—ASSOCIATION OF COMMON FACTOR FROM ONE-FACTOR ADOPTION MODEL WITH ADOPTION AND OTHER CHARACTERISTICS OF THE HOSPITAL

	Quintile 5 (quickest)	Quintile 4	Quintile 3	Quintile 2	Quintile 1 (slowest)	Overall
Fraction aspirin	0.90	0.86	0.81	0.77	0.65	0.80
Fraction $\beta$ blocker	0.66	0.53	0.46	0.41	0.31	0.47
Fraction reperfusion within 12 hours	0.22	0.20	0.19	0.18	0.15	0.19
Average hospital AMI volume <sup>a</sup>	91	104	94	86	67	89
Fraction major teaching hospital	0.45	0.30	0.24	0.16	0.06	0.24
Fraction for-profit hospital	0.04	0.08	0.07	0.09	0.17	0.09
Fraction government hospital	0.11	0.09	0.13	0.10	0.17	0.12
Average state income (1994–1995)	43,790	42,603	42,168	42,215	41,648	42,495
% of hospitals performing stents in 2003–2004	0.74	0.69	0.63	0.49	0.32	0.57
Of those, % drug-eluting stent 2003–2004	0.62	0.60	0.59	0.57	0.52	0.59

Each column of the table reports average hospital and patient characteristics by adoption quintile and overall (in the final column). All averages are weighted by number of AMI patients in each hospital. Adoption quintiles were defined based on the common factor estimated from a one-factor model of hospital use of aspirin, beta blockers, and reperfusion. All estimates except for stent data come from the Cooperative Cardiovascular Project (CCP), 1994–1995, with a sample of 139,847 AMI patients and 2,999 hospitals. Estimates for each quintile are based on samples of approximately 28,000 AMI patients. Stent data are derived from Medicare Part A (hospital) claims from 2003–2004 for the same sample of hospitals.

<sup>a</sup>Volume for Medicare patients only.

the angioplasty. While not all hospitals had the catheterization laboratories necessary for PCI, thrombolytics were a viable option for nearly every hospital.

#### B. A Factor Model of Diffusion

We estimate a factor model based on equation (15), in which the proportion of patients in each hospital receiving each of the three treatments depended on a single common factor. Hospital-level data on each of the three treatments was available for 2,999 hospitals in 1994–1995 from the CCP data.<sup>8</sup> Factor analysis normalizes the underlying factors to have a mean of 0 and variance of 1.

Table 1 summarizes the extent to which the common factor was associated with each of the individual variables. Quintiles across hospitals based on this common factor show clear differences in the use of aspirin (from 90% in the highest-adopting quintile to 65% in the lowest), beta blockers (66% to 31%), but with more modest differences in reperfusion (22% to 15%).<sup>9</sup> One could interpret these patterns as reflecting demand; patients in high-quintile regions ask for and get beta blockers, for example. However, elderly supine heart attack patients are unlikely to be requesting specific treatments, and hospitalized patients should not have to ask their physicians for these treatments given their clear benefits.

Table 1 also demonstrates that hospitals in the quintiles with quicker adoption also have higher patient volume, are much more likely to be major teaching hospitals and less likely to be for-profit hospitals, and are located in states with slightly higher average income. Teaching hospitals and those with higher volume are particularly likely to experience lower informational barriers to rapidly adopt

new technologies. Moreover, the positive association between patient volume and the speed of diffusion of highly effective technologies suggests that the market may reward rapid adopters with higher demand (Chandra et al., 2013).

We can use estimates from the randomized trials for aspirin, beta blockers, and reperfusion to make predictions about the gaps in mortality across our diffusion quintiles. Using the disparities in utilization from table 1, multiplied by treatment effects estimated in clinical trials,<sup>10</sup> yields a predicted one-year survival different of 3.9% between the highest and lowest diffusion quintiles. In the next section, we compare this estimate, based solely on clinical studies and the CCP diffusion data, with results from Medicare risk-adjusted mortality data.

The final rows of table 1 show patterns of diffusion a decade later for a quite different innovation: drug-eluting stents. In April 2003, the FDA approved new drug-eluting stents, which were coated with antibiotics to reduce the likelihood of the blockage reappearing at the site of the original stent.<sup>11</sup> We linked the hospital-specific measures of the diffusion of drug-eluting stents, as described in Malenka et al. (2008), to the earlier diffusion quintiles. Hospitals with the most rapid diffusion of cardiac technology in 1993–1994 were both more likely to implant stents in 2003–2004 and, conditional on having catheterization facilities, were more likely to have adopted drug-eluting stents. Knowing rates at two different points in time allows us to measure changes over time in hospital diffusion rates, which we consider in the next section.

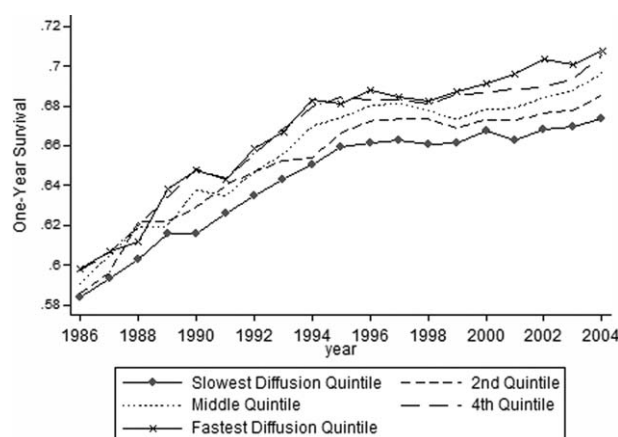
<sup>10</sup> The estimated effect of beta blockers is a 22% decline in one-year mortality arising from beta blockers (Phillips et al., 2000), times a baseline 33% mortality probability. For aspirin, mortality was 18% lower (Krumholz et al., 1995). For 12-hour reperfusion, we use as a lower bound the impact of fibrinolytic therapy, of about 25% mortality decline (FTT, 1994). Each of these was multiplied by the gap across quintiles (from table 1) in the corresponding diffusion measures.

<sup>11</sup> While there has been some controversy about the health benefits of drug-eluting stents (see Malenka et al., 2008), there was widespread consensus among cardiologists in 2003 that this new technology was better than the older bare-metal stents. Also note that the estimated diffusion rates are for all stenting patients, and not solely AMI patients.

<sup>8</sup> Hospitals in this sample were required to have remained in existence throughout the period 1986 to 2004 in the Medicare claims data, as described in section VI.

<sup>9</sup> These averages are for all patients and not for “ideal” patients, since it is often difficult in practice to define ideal or appropriate patients. While a high fraction of patients should receive beta blockers and aspirin, the optimal rate for revascularization is substantially lower.

FIGURE 1.—SURVIVAL RATES BY YEAR AND DIFFUSION QUINTILES, 1986–2004



The figure reports risk-adjusted one-year AMI survival rates annually from 1986 to 2004 for hospitals in each adoption quintile. Adoption quintiles were defined based on the common factor estimated from a one-factor model of hospital use of aspirin, beta blockers, and reperfusion (see table 1). Risk-adjusted survival was derived from Medicare claims data on 2.8 million AMI admissions from 1986 to 2004.

## V. The Relationship between Patient Survival and a Hospital's Diffusion Rate

### A. Data on Patient Cost and Survival

The primary data set is a 20% sample of the Medicare Part A (hospital) claims data for all heart attack (AMI) patients age 65 and over in the United States from 1986 to 1991 and a 100% sample from 1992 through 2004, with updated information on mortality through 2005. (We limit the follow-up period to ten years following the 1994–1995 CCP data on diffusion.) The original sample comprises 3.3 million people. We eliminated hospitals with fewer than five patients in any of the 100% sample years (and any hospital that closed during the period of analysis), resulting in a final sample of 2.8 million people.

To create hospital-year risk-adjusted survival and (inflation-adjusted) expenditures, we estimated equation (14) at the patient level using identical specifications for three dependent variables: one-year survival, total Part A (hospital) Medicare reimbursements during the year following the AMI, and total DRG weights per patient during the year following the AMI as a measure of factor inputs.<sup>12</sup> These regressions included categorical variables indicating the presence of seven comorbid conditions, anatomical location of the MI, and full interactions of each five-year age bracket, by sex and race. The risk-adjustment regressions for one-year survival and one-year expenditures are shown in appendix table A.1.

### B. Descriptive Results

We begin by showing graphically the association between technology adoption and risk-adjusted survival in figure 1, which displays the weighted average of risk-

<sup>12</sup> For example, an AMI patient fitted with a drug-eluting stent would qualify for 3.12 DRG “units” in 2003, and this was common across all hospitals. Note that DRG weights may change slightly over time.

adjusted one-year survival ( $y_{it}$ ) by year and quintile of diffusion. The average gap in survival between the slowest and most rapid adopters is 2.7 percentage points, somewhat less than the predicted gap of 3.9 percentage points based on evidence from randomized trials discussed in the prior section. The lag in terms of years between the most rapid and slowest hospital adopters varies over the time period, but the average annual (horizontal) gap is roughly five to ten years. In our model,  $(1 - \pi_i)/\pi_i$  can be interpreted as the number of years a hospital lags behind the frontier. Thus, we would observe a gap of ten years if the most rapid adopters had a diffusion rate of 10% (nine years behind the frontier) and the slowest adopters had a diffusion rate of 5% (nineteen years behind the frontier). Diffusion rates of 10% and 5% are in line with the adoption rates of the quickest and slowest hospitals in table 1, again suggesting that differences in survival are broadly consistent with observed differences in diffusion.<sup>13</sup>

### C. Convergence

A key implication of the model is the lack of convergence; the low-diffusion hospitals are predicted to grow at the same rate as high-diffusion hospitals. This can be seen visually in figure 1. But we can also test another implication of the model: that the hospital-level variance in risk-adjusted survival is not predicted to narrow over time ( $\sigma$ -convergence). We do not find evidence of such convergence: our estimate of the (weighted) standard deviation of hospital fixed effects, correcting for estimation error (by subtracting the variance of the “noise” component of the fixed effect), is 0.043 in 1986 and 0.042 in 2004.

### D. Estimates of Technology Diffusion on Survival

Table 2 presents estimates of the regression model in equation (13). We begin with the simplest regression model in which survival is a function of the continuous diffusion index, log DRG inputs, and year fixed effects. Recall that the diffusion index is normalized to have a standard deviation of 1. Thus, the coefficient implies that a 1 standard deviation increase in the diffusion rate is associated with a 1.4 percentage point increase in patient survival, which yields a similar survival benefit as the predicted doubling of DRG inputs ( $0.022\ln(2) = 0.015$ ). Column 3 replaces the continuous diffusion index with dummies for the hospital-specific diffusion quintile and demonstrates again that controlling for DRG inputs, the most rapidly diffusing hospitals (quintile 5) experience a 2.7 percentage point higher survival rate compared to the slowest-diffusing hospitals.

<sup>13</sup> If aspirin began diffusing in 1975, beta blockers in 1985, and reperfusion in 1992 (all start dates), then by 1995 diffusion rates of 10% and 5% would generate aspirin use of 88% and 64%, beta blocker use of 65% and 40%, and reperfusion use of 27% and 14%, which are reasonably consistent with estimates in table 1.

TABLE 2.—REGRESSION ESTIMATES OF SURVIVAL ON DRG INPUTS AND THE EFFECTIVE TREATMENTS DIFFUSION FACTOR

Dependent Variable	One-Year Survival	One-Year Survival
Diffusion factor (continuous)	0.014 (0.001)	
Diffusion quintile 2		0.009 (0.002)
Diffusion quintile 3		0.016 (0.002)
Diffusion quintile 4		0.024 (0.003)
Diffusion quintile 5		0.027 (0.002)
log (DRG)	0.022 (0.004)	0.023 (0.004)
$R^2$	0.09	0.09

Each column reports estimates from a separate regression in which an observation is a hospital-year, with  $N = 49,937$  hospital-years. All regressions are weighted by the number of patients in each hospital-year. The dependent variable is the risk-adjusted one-year survival rate among AMI patients. The diffusion factor and quintiles are based on the common factor estimated from a one-factor model of hospital use of aspirin, beta blockers, and reperfusion. Log(DRG) is the log of the risk-adjusted total DRG weights per patient in the year following his or her AMI. Year dummy variables are included in all regressions. Risk-adjusted survival and DRG weights are derived from Medicare claims data from 1986 to 2004. The sample is limited to hospital/year observations with at least five observations per hospital. Standard errors (clustered at the hospital level) are in parentheses.

#### E. Estimates of $\beta$ , the Marginal Productivity of Inputs

Table 3 examines how the specification of the model affects estimates of  $\beta$  and interprets them in the context of the cost-effectiveness ratio. Column A of the table reports estimates for all hospitals, while column (B) is limited to hospitals with at least fifty AMI patients. Rows 1 and 2 show the traditional regressions of risk-adjusted survival on risk-adjusted expenditures and DRG inputs. These regressions show either a negative association between spending and survival or, in one case (with  $\ln(\text{DRG})$  on the right-hand side of the regression across all hospitals), a very small positive coefficient of  $\beta = 0.017$ , with a weak cost-effectiveness ratio of \$301,000.

As noted earlier, these estimates may reflect the fact that lower-spending hospitals were those with greater diffusion of effective technologies. When we control for either the continuous measure of diffusion (row 3) or quintiles (row 4), we find a positive and more favorable association between DRG inputs and survival, with the all-hospital sample yielding cost-effectiveness ratios of \$227,000 and \$216,000 per life-year (although the sample of hospitals with  $N > 50$  shows insignificant estimates). Finally, as shown in row 5, including hospital fixed effects (which potentially capture additional differences across hospitals in diffusion not measured by our diffusion index) raises the estimate of  $\beta$  to 0.053, with an implied cost-effectiveness ratio of between \$94,000 and \$155,000. The estimate of  $\beta$  is higher in the period 1986–1994 than in 1995–2004, when the estimated cost-effectiveness ratio ranges from \$115,000 to \$171,000.

This pattern of coefficients is consistent with our model if the return to DRG inputs is lower in hospitals with higher technology diffusion, as represented graphically in figure 3 for a given year. Consider just two hospitals, given by A (on the production function PF(1)) and B (on the produc-

tion function PF(2)). If the researcher does not control for technology adoption, she would estimate the dotted line connecting points A and B—effectively, flat-of-the-curve health care, as shown in row 1 or 2 of table 3. As we control with more accuracy for each hospital's technology level in table 3, the estimated (marginal) slope of the production function becomes steeper, to approximate aa' or bb' in figure 2.

#### F. Changes over Time in Diffusion Rates

A prediction of the theoretical model is that hospitals that manage to improve their diffusion parameters will, like countries such as Japan or Korea in the postwar period, experience rapid growth in outcomes (Parente & Prescott, 2002), and conversely. Table 4 further considers risk-adjusted survival among hospitals that were initially in the slowest diffusion quintile (1) or the highest diffusion quintile (5) during 1994–1995. For the slow-diffusion hospitals in 1994–1995 remaining a slow-diffusion hospital (in drug-eluting stents) in 2003–2004, one-year survival rates rose from 65.2% in 1994–1995 to 69.1% in 2003–2004, an increase of 3.9%. Similarly, hospitals initially in the highest-diffusing quintile (5) in 1993–1994 that remained in the highest-diffusing quintile by 2003–2004, increased survival by 3.5 percentage points—very similar to the stable low-quintile hospitals, as predicted by the model.

Hospitals initially in the lowest-diffusion quintile during 1994–1995 that moved up to the highest-diffusion quintile for drug-eluting stents in 2003–2004 (the tigers) experienced a gain of 5.5 percentage points. By contrast, the hospitals experiencing a decline in diffusion rates from quintile 5 in 1994–1995 to quintile 1 in 2003–2004 (the turtles) showed a survival gain of just 1.5 percentage points, significantly lower than the tigers ( $p = 0.03$ ).<sup>14</sup> These patterns are consistent with the view that changes in technology diffusion can be directly linked to changes in hospital outcomes as measured by patient survival.

### VI. Did the Rapid-Diffusion Hospitals Adopt Every New Innovation?

Thus far, we have considered only three of the many new innovations for heart attack patients during the period 1984 to 2004. Did the rapidly innovating hospitals also adopt other technologies across the board? Or does the adoption of these highly effective innovations reflect specific skills of the hospital management, as in the Lucas (1978) model of managerial skills? In this section, we consider three addi-

<sup>14</sup> One hypothesis is that hospitals that were early adopters of surgery in 1994–1995 would also be early adopters of drug-eluting stents in 2003–2004, and so the improved survival of the tiger hospitals was simply the consequence of surgical innovations paying off in the 2000s. However, drug-eluting stents were no more correlated with surgical procedure rates in 1994–1995 than beta blockers or aspirin in 1994–1995.

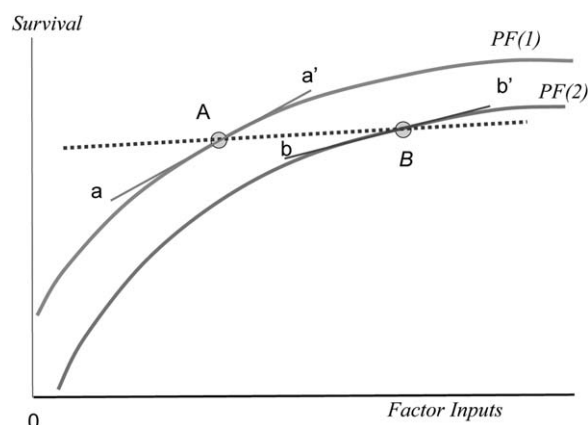


TABLE 3.—REGRESSION ESTIMATES OF SURVIVAL ON INPUTS FOR ALTERNATIVE SPECIFICATIONS

	Input	Period of Analysis	Adjusted for Diffusion	(A) All Hospitals	(B) Larger Hospitals ( <i>N</i> > 50)
1	Log(Expend)	1986–2004	No	–0.015 (0.003)	–0.026 (0.004)
2	Log(DRG)	1986–2004	No	[Undefined] 0.017 (0.004)	[Undefined] –0.016 (0.006)
3	Log(DRG)	1986–2004	Continuous measure	[\$301,000] 0.022 (0.004)	[Undefined] –0.009 (0.006)
4	Log(DRG)	1986–2004	Diffusion quintiles	[\$227,000] 0.023 (0.004)	[Undefined] –0.009 (0.006)
5	Log(DRG)	1986–2004	Hospital fixed effects	[\$216,000] 0.053 (0.003)	[Undefined] 0.032 (0.005)
6	Log(DRG)	1986–1994	Hospital fixed effects	[\$94,000] 0.069 (0.004)	[\$155,000] 0.044 (0.013)
7	Log(DRG)	1995–2004	Hospital fixed effects	[\$72,000] 0.043 (0.004)	[\$113,000] 0.029 (0.005)
				[\$115,000]	[\$171,000]

See notes to table 2. Each row of the table reports the coefficient on a measure of inputs from separate regressions of risk-adjusted one-year survival on inputs using different specifications as indicated. Column A reports estimates for all hospitals, while column B is limited to hospitals with at least fifty AMI admits in the year. All regressions control for year dummies. The cost-effectiveness ratios are in brackets and assume 2004 average costs of \$26,063 and an average length of life among incremental survivors of 5.25 years.

FIGURE 2.—INTERPRETING THE EVIDENCE ON SURVIVAL AND HEALTH OUTCOMES: FLAT-OF-THE-CURVE VERSUS PRODUCTIVITY DIFFERENTIALS



This figure illustrates the idea that when two health care systems are on different production functions PF(1) and PF(2), trying to make inferences about the marginal value of spending from a comparison of the two health systems can lead to biased results. In the graph, even though the marginal value of additional spending is positive for both health systems (with slopes of the marginal value given by *aa* and *bb*, respectively), a regression line based on a cross-sectional comparison (the dotted line passing through AB) yields a biased estimate of the true returns.

tional innovations with less cost-effective and more heterogeneous benefits.

The first is lidocaine, a drug used to prevent ventricular fibrillation in AMI patients. While this was viewed as a promising approach in the early 1980s, by the late 1980s, a consensus had emerged that the use of lidocaine for uncomplicated AMIs could actually increase mortality (Hine et al., 1989). As of 1994–1995, average rates of use were 20.4%.

The second is late PCI, or the use of angioplasty more than 12 hours after the index AMI. Beyond a small group of patients, its use was supported by either weak evidence or was contraindicated (Ryan et al., 1993). Within a year of

the AMI, 16.4% of all patients received such treatment. The third is coronary artery bypass graft surgery (CABG). The 1991 American College of Cardiology/American Heart Association (ACC/AHA) guidelines suggested strict criteria for CABG following AMI, and for many broad classes of patients, guidelines are not supportive.<sup>15</sup> On average, 14.6% of AMI patients received CABG within a year of the AMI.

With these three additional technologies, we have six diffusion measures created by averaging across all AMI patients in each hospital. We then estimated a factor model allowing for more than one factor using a varimax rotation, which facilitates interpretation of the factors by identifying each treatment rate with a single factor to the extent possible. The BIC goodness-of-fit criterion indicated just two distinct factors, labeled factor A and factor B. Note that we have not imposed any a priori restrictions on how these factors are estimated. As before, factor analysis normalizes the underlying factors to have a mean of 0 and variance of 1.

Factor A is nearly identical to our simple diffusion measure above; the correlation between the two is 0.98. To facilitate comparisons between factors A and B, we present in table 5 the difference between quintile 5 (fastest) and quintile 1 (slowest) innovations, with the factor scoring weights (these weights are used to combine the six measures to form predictions of each factor). Factor A loads heavily on medical treatments that are highly effective such as aspirin (a weight of 0.41), beta blockers (0.35), and

<sup>15</sup> For example: “The coronary artery bypass operation probably has little place in the management of most patients with uncomplicated acute Q wave myocardial infarction, but the matter remains arguable” (Kirklin et al., 1991, p. 1141).

TABLE 4.—GROWTH IN RISK-ADJUSTED SURVIVAL BY DIFFUSION QUINTILES IN 1994–1995 AND IN 2003–2004

	Survival: 1994–1995		Survival: 2003–2004		Change in Survival: 1994–1995 to 2003–2004	
	2003/04 Quintile 1 (slowest)	2003–2004 Quintile 5 (fastest)	2003–2004 Quintile 1 (slowest)	2003–2004 Quintile 5 (fastest)	2003–2004 Quintile 1 (slowest)	2003–2004 Quintile 5 (fastest)
1994–1995 quintile 1 (slowest)	0.652 (0.008)	0.655 (0.019)	0.691 (0.009)	0.710 (0.012)	0.039 (0.009)	0.055 (0.016)
1994–1995 quintile 5 (fastest)	0.686 (0.006)	0.684 (0.005)	0.701 (0.007)	0.719 (0.006)	0.015 (0.008)	0.035 (0.006)

The table reports patient-weighted risk-adjusted one-year survival rates in 1994–1995 and 2003–2004 and the change in survival rates from 1994–1995 to 2003–2004. Each cell identifies hospitals according to whether they were in the fastest or slowest adoption quintile of effective treatments in 1994–1995 and whether they were in the fastest or slowest adoption quintile of drug-eluting stents in 2003–2004 (see table 1). All standard errors are clustered at the hospital level.

TABLE 5.—ASSOCIATION OF FACTOR A AND B FROM TWO-FACTOR ADOPTION MODEL WITH ADOPTION AND OTHER CHARACTERISTICS OF THE HOSPITAL

	Factor A			Factor B		
	Quintile 5 (fastest)	Quintile 1 (slowest)	Factor Scoring Weight	Quintile 5 (fastest)	Quintile 1 (slowest)	Factor Scoring Weight
Aspirin	0.90	0.65	0.41	0.82	0.75	0.07
β blocker	0.65	0.31	0.35	0.41	0.51	−0.15
12-hour reperfusion	0.19	0.15	0.05	0.26	0.12	0.27
Lidocaine	0.17	0.21	−0.08	0.32	0.11	0.34
PCI after 12 hours	0.22	0.12	0.13	0.23	0.10	0.20
CABG	0.18	0.12	0.10	0.18	0.10	0.15
Average hospital volume <sup>a</sup>	103	64		83	77	
Fraction major teaching hospital	0.50	0.05		0.11	0.28	
Fraction for-profit hospital	0.04	0.16		0.14	0.08	
Fraction government hospital	0.10	0.17		0.14	0.13	
Average state income (1994–1995, thousands)	43.99	41.77		41.37	43.65	
Fraction of hospitals performing stents 2003–2004	0.82	0.28		0.75	0.31	
Of those, % drug-eluting stent 2003–2004	0.62	0.51		0.55	0.61	

Each column of the table reports average hospital and patient characteristics in the fastest and slowest adoption quintiles and factor scoring weights for defining each factor, based on the factors estimated from a two-factor model. The two factors were estimated from a factor model of hospital use of aspirin, beta blockers, reperfusion, lidocaine, PCI after 12 hours (late PCI), and CABG (coronary artery bypass graft surgery). All averages are weighted by number of AMI patients in each hospital. All estimates except for stent data come from the Cooperative Cardiovascular Project (CCP), 1994–1995, with a sample of 139,847 AMI patients and 2,999 hospitals. Estimates for each quintile are based on samples of approximately 28,000 AMI patients. Stent data are derived from Medicare Part A (hospital) claims from 2003–2004 for the same sample of hospitals.

<sup>a</sup>Volume for Medicare patients only.

avoiding lidocaine (−0.08). By contrast, factor B loads more heavily on surgical innovations that tend to be less cost-effective, such as reperfusion (0.27), PCI after 12 hours (0.20), and CABG (0.15), as well as lidocaine (0.34). One interpretation of the two factors is that factor A identifies hospitals that are able to accumulate new knowledge more quickly and thereby more rapidly identify highly effective technologies, while factor B identifies hospitals that are able to accumulate new technology more quickly (whether or not it is effective). Thus, factor A seems to identify “smart” hospitals, while factor B seems to identify “aggressive” hospitals.

There are other differences between the factor A and B hospitals, also shown in table 5, that are broadly consistent with this interpretation of the two factors. Unlike factor A, there were no differences in patient volume across quintiles of factor B, suggesting that the market may be rewarding “smart” adoption rather than the adoption of any new technology. Factor B hospitals also exhibit a higher fraction of for-profit hospitals (0.14 in the highest quintile versus 0.08 in the lowest) and a lower fraction of teaching hospitals (0.11 versus 0.28), suggesting that factor B adoption is less related to knowledge of the medical staff. In sum, these two

factors identify very different types of hospitals with very different strategies of technology adoption.

We first estimate the equivalent of equation (13) with both factors, shown in table 6; survival is a function of the continuous diffusion indices, log DRG inputs, and year fixed effects.<sup>16</sup> As before, hospitals in the top quintile of factor A are associated with 2.6 percentage point higher survival. By contrast, hospitals in the top quintile for factor B exhibit only 0.9 percentage point higher survival. Thus, adoption of the types of technologies associated with factor B has less of an impact on survival than the highly effective technologies associated with factor A.

Finally, one might expect the return to factor inputs to be higher in hospitals that adopt technologies associated with factor B, since these tend to be more expensive surgical interventions. We therefore estimate the more flexible “translog” production function (Christiansen, Jorgenson, &

<sup>16</sup> We show in the appendix that a model in which there are two types of innovations that appear in each year, a higher-value and lower-value innovation, leads to an estimation equation that is identical to equation (13), except to include both of the diffusion parameters for factor A,  $\pi_{A_i}$ , and factor B,  $\pi_{B_i}$ .

TABLE 6.—REGRESSION ESTIMATES OF SURVIVAL ON DRG INPUTS AND ON BOTH DIFFUSION FACTORS FROM THE TWO-FACTOR MODEL OF DIFFUSION

Dependent Variable	One-Year Survival	One-Year Survival
Factor A	0.014 (0.001)	
Factor A: Quintile 2		0.008 (0.002)
Factor A: Quintile 3		0.013 (0.002)
Factor A: Quintile 4		0.023 (0.002)
Factor A: Quintile 5		0.026 (0.002)
Factor B	0.005 (0.001)	
Factor B: Quintile 2		0.005 (0.002)
Factor B: Quintile 3		0.007 (0.002)
Factor B: Quintile 4		0.012 (0.003)
Factor B: Quintile 5		0.009 (0.003)
Log (DRG)	0.020 (0.004)	0.021 (0.004)
R <sup>2</sup>	0.09	0.09

Each column reports estimates from a separate regression in which an observation is a hospital-year, with  $N = 49,937$  hospital-years. All regressions are weighted by the number of patients in each hospital-year. The dependent variable is the risk-adjusted one-year survival rate among AMI patients. The diffusion factors and quintiles are based on the two-factor model described in table 5. Log(DRG) is the log of the risk-adjusted total DRG weights per patient in the year following their AMI. Year dummy variables are included in all regressions. Risk-adjusted survival and DRG weights are derived from Medicare claims data from 1986 to 2004. The sample is limited to hospital-year observations with at least five observations per hospital. Standard errors (clustered at the hospital level) are in parentheses.

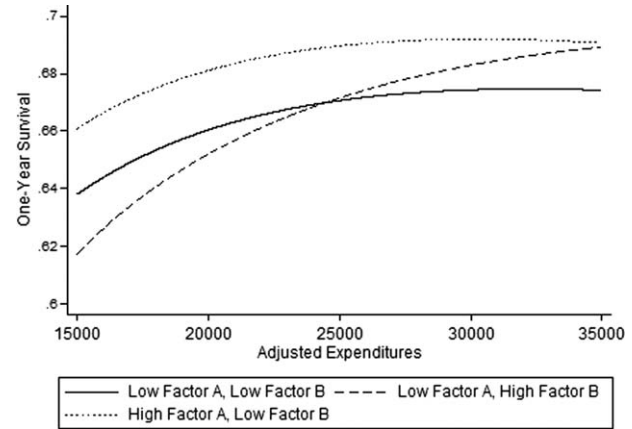
Lau, 1973) to allow for diminishing returns to  $\pi_k$ ,  $x_{it}$ , and interaction between diffusion and the productivity of  $x_{it}$ :

$$y_{it} = \omega_A \pi_{Ai} + \omega_B \pi_{Bi} + \beta x_{it} + \gamma_1 a_{Ai} x_{it} + \gamma_2 a_{Bi} x_{it} + \gamma_3 a_{Bi} \alpha_{Ai} + \gamma_4 a_{Ai}^2 + \gamma_5 a_{Bi}^2 + \gamma_6 x_{it}^2 + u_{it}. \quad (17)$$

This allows, for example, a higher marginal return to the level of spending  $x_{it}$  when hospitals invest more heavily in B-type innovations than A-type innovations.

The results of the fully interacted regression analysis are in appendix table A.2, with illustrative examples shown in figure 3. We consider three types of hospitals. The first eschews all types of technology adoption; it lies 1 standard deviation below the mean for both factors A and B; the second is for hospitals like the first, except that the rate of diffusion for factor A treatments is +1 standard deviation above the mean. As shown in figure 3, the rapid factor A-adopting hospitals exhibit substantially better outcomes for their patients at all levels of spending, with marginal returns to additional inputs falling off rapidly above \$25,000 per enrollee, roughly the mean value of spending in 2004.<sup>17</sup> By contrast, the marginal returns to spending are strongly posi-

FIGURE 3.—ESTIMATED 2004 PRODUCTION FUNCTION FOR ONE-YEAR AMI SURVIVAL: LOW DIFFUSION (SOLID), HIGH DIFFUSION FOR FACTOR A (DOTTED), AND HIGH DIFFUSION FOR FACTOR B (DASHED)



The figure plots predicted one-year survival, based on regression analysis reported in appendix table A.2 with hospital fixed effects, for three hypothetical hospitals: a hospital that is 1 standard deviation below average on both adoption factors (low factor A, low factor B), a hospital that is 1 standard deviation below average on factor B but 1 standard deviation above average on factor A (high factor A, low factor B), and a hospital that is 1 standard deviation below average on factor A but 1 standard deviation above average on factor B (low factor A, high factor B). While the slope and shape of the production function come directly off the estimated coefficients, the intercepts for each of these three curves are derived from a between-hospital regression of the hospital fixed effects regressed on the two factors, their squares, and their interactions.

tive for hospitals with factor B at +1 standard deviation above its mean. This is consistent with hospitals spending more if they are more rapid adopters of factor B inputs, since the technologies typically entail billing more to Medicare. This is also consistent with Chandra and Staiger (2007), who find that hospitals specializing in surgical treatments can attain similar levels of outcomes to those specializing in medical treatments, albeit at higher costs, and with Doyle, Ewer, and Wagner (2010), who find that patients treated by physicians from a lower-ranked medical school attain similar outcomes but require more resources to do so.

## VII. Conclusion

In this paper, we have attempted to peer inside the black box of hospital productivity changes over time and across hospitals. We found that varying rates of adoption for low-cost but highly effective treatments explained a large fraction of the persistent differences in risk-adjusted survival during the period 1986 to 2004. Hospitals with the most rapid propensity to adopt these new innovations experienced survival rates nearly 3 percentage points above the lowest-quintile hospitals, or nearly one-third the entire improvement in survival since 1986.

We also found distinct differences across hospitals with regard to which kinds of technologies were adopted. Some hospitals were far more likely to adopt beta blockers and aspirin, highly effective and inexpensive treatments, and these experienced consistently better survival outcomes than hospitals that invested more heavily in surgical treatments such as PCI and CABG. While both types of hospitals experienced better overall outcomes than hospitals that failed to adopt any of the new technologies that became available during the 1990s, hospitals that rapidly adopted

<sup>17</sup> To facilitate the interpretation of the regression, we multiplied the DRG inputs times a constant, the national average reimbursement per DRG in 2004, leading to a dollar amount that corresponds to the DRG inputs.

beta blockers and aspirin had higher patient survival. Hall (2014) found similar results; regions adopting the cost-effective innovations like screening for colon cancer had better outcomes, while those with more rapid diffusion of low-effectiveness treatments, like breast cancer screening for women under age 50, did not.<sup>18</sup> Our evidence suggests that knowledge about which technologies are most effective is a larger contributor to variation in productivity across hospitals than the adoption of new technology writ large.

Our model of health care productivity reconciles both the dramatic improvements in life expectancy for AMI patients over time (Cutler, 2004) and the mixed evidence on the efficiency of spending at a point in time (e.g., Fisher et al., 2003a, 2003b; Doyle et al., 2015). Much of the dramatic growth in survival occurred as remarkably cost-effective treatments diffused across hospitals during the past few decades. For example, Ford et al. (2007) found aspirin and beta blockers to be among the most important factors reducing the number of AMI-related deaths between 1980 and 2000, followed by PCI and CABG. Of course, these estimates are specific to heart attacks, where the quality of clinical evidence is particularly good and may not apply to other diseases where clinical guidelines are much weaker.

Chandra et al. (2013), using similar data on AMI patients, developed an explicit model of productivity differences across hospitals (log survival minus log costs) and demonstrated that firms in nonhealth industries exhibit similar degrees of variation in productivity as do hospitals—in other words, that health care is not uniquely inefficient. Economists have identified a variety of optimizing economic models by which some firms adopt new innovations and others do not, which naturally lead to such productivity variations. For example, rational agents may adopt slowly because they are waiting for the price to decline (e.g., flat-screen TVs) or because of expertise in the older technology (Jovanovic & Nyarko, 1996). Alternatively, heterogeneity in production functions may lead to profit-maximizing differences in rates of diffusion (Griliches, 1957) or the presence of liquidity constraints may slow diffusion (Suri, 2011). Finally, there may be differences in education across workers (Nelson & Phelps, 1966) or technology complementary with skilled workers (Caselli & Coleman, 2006). While these theories may explain productivity differences in nonhealth sectors (and perhaps differences in adoption of new surgical innovations), they are less successful in explaining the slow diffusion of inexpensive beta blockers and aspirin by highly educated physicians.

Perhaps informational or organizational barriers explain the slow diffusion in both health and nonhealth sectors of the economy. Recall equation (11), in which the marginal cost of speeding up diffusion  $C'(\pi)$  was set equal to the

marginal benefit of innovating more rapidly. Using plausible parameters for measuring the social value of more rapid adoption suggests a very high equilibrium cognitive barrier facing physicians equal to \$11,200 annually to move up one diffusion quintile.<sup>19</sup> While this may appear to be implausibly high, previous research has shown that the quality of management, and in particular the presence of staff “opinion leaders,” can exert a disproportionate influence on individual physician adoption (Bradley et al., 2001, 2005). This points to a more nuanced model of diffusion in which organizational support and “tactile” learning from peers (as in Keller, 2004) are critical for rapid diffusion. For this reason, we expect organizational inefficiencies, whether in nonhealth industries (Bloom & van Reenan, 2007) or cardiac health centers for AMI patients (McConnell et al., 2013), are central to explaining why some hospitals were quick to adopt beta blockers and aspirin and others were not.

Leibenstein (1966) used the term *X-efficiency* to describe residual differences in firm-level productivity that could not be readily explained by measured inputs or other factors. In many respects, the puzzle of slow diffusion for beta blockers and aspirin provides a textbook case of X-inefficiency. The underuse of aspirin and beta blockers was allowed to persist for so long because there was so little pressure exerted by markets or management to change old habits and adopt the new innovations. It is telling that the increased public hospital-level reporting of beta blocker use for AMI patients has been central to its nearly universal diffusion in the last decade, so much so that it is no longer used as a quality measure (Lee, 2007). More difficult is to explain why adoption behaviors are correlated between nonhealth sectors and health sectors, as in the close correlation across states in the adoption of hybrid corn in the 1930s and beta blockers in the 2000s (Skinner & Staiger, 2007).

There are several limitations to this study. First, our unit of analysis is the hospital, which by necessity aggregates up the adoption decisions of the physician. While some studies have used overlapping physician-hospital affiliations to identify hospital diffusion patterns (Sacarny, 2014), we are unable to determine which physician initiated specific treatments inside the hospital. Second, while we have direct evidence on why the rapidly diffusing hospitals experienced better outcomes in 1994–1995 from the CCP, we know much less about subsequent adoption patterns of the newer technologies such as the use of angiotensin receptor blockers (ARB) and angiotensin-converting enzyme inhibitors (ACE inhibitors). Finally, our data are limited to Medicare enrollees age 65 and over, and there is little evidence on how, for example, beta blocker use in this population correlates with its use in the under-65 population.

<sup>18</sup> She also found that high-adoption hospitals tended to adopt all new technologies (Hall, 2014). In the principal component model that she uses, the first component splits out regions that adopt anything (either factor A or factor B) versus those that do not. When we estimate a principal components model, we find results similar to hers.

<sup>19</sup> We assume that the average lag from the frontier,  $a_t^* - a_t = 0.02$ ,  $\Psi = \$100,000$ , the one-year survival following AMI translates to an additional 5.25 life-years,  $r = .05$ ,  $\pi_A$  must increase by 0.016 to shift to the next quintile, and there are ten AMI patients per physician.

Parente and Prescott (2002) provide a ready explanation for why some countries lag so far behind frontier countries: government restrictions and monopoly restraints that interfere with the benefits of efficient technology adoption. If patients knew about the benefits of aspirin, beta blockers, and reperfusion and were sensitive to published and reliable information about hospital quality, physicians would be forced to respond rapidly to new innovations or face the loss of patients. But when quality measures are limited, patients are not well informed, and markets are distorted, remarkably large inefficiencies can persist across hospitals and over time.

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