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ABSTRACT

Inefficiency in the U.S. health care system has often been characterized as "flat of the curve" spending providing little or no incremental value. In this paper, we draw on macroeconomic models of diffusion and productivity to better explain the empirical patterns of outcome improvements in heart attacks (acute myocardial infarction). In these models, small differences in the propensity to adopt technology can lead to wide and persistent productivity differences across countries -- or in our case, hospitals. Theoretical implications are tested using U.S. Medicare data on survival and factor inputs for 2.8 million heart attack patients during 1986-2004. We find that the speed of diffusion for highly efficient and often low-cost innovations such as beta blockers, aspirin, and primary reperfusion explain a large fraction of persistent variations in productivity, and swamp the impact of traditional factor inputs. Holding technology constant, the marginal gains from spending on heart attack treatments appear positive but quite modest. Hospitals which during the period 1994/95 to 2003/04 raised their rate of technology diffusion (the "tigers") experienced outcome gains four times the gains in hospitals with diminished rates of diffusion (the "tortoises"). Survival rates in low-diffusion hospitals lag by as much as a decade behind high-diffusion hospitals, raising the question of why some hospitals (and the physicians who work there) adopt so slowly.

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1. Introduction

There are pervasive regional differences in per capita U.S. Medicare expenditures, ranging from $5,877 in Salem, OR to $16,351 in Miami.[1] Yet there is little or no evidence that the higher spending in high cost regions lead to better outcomes, with estimates of inefficiency range from 20 to 30 percent of overall health care expenditures (Fisher et al., 2003; Skinner, et al., 2005). These estimates have 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.[2]

But this "flat of the curve" explanation is problematic for many observed patterns. First, Baicker and Chandra (2004) have documented that state-level quality measures are *negatively* associated with per capita Medicare expenditures. 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 has improved because of often expensive new medical technology, it would be surprising if 20 to 30 percent of health care spending (or between 3 and 4.5 percent of GDP) should provide *no* benefit whatsoever.

In this paper, we draw on macroeconomic models of productivity to provide a better explanation for these empirical puzzles. That differential rates of technology adoption can explain long-term variations in per capita GDP across countries is by now well understood. Crespi, et al. (2008) find as much as 50 percent 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

[1] Estimates are for 2006 (www.dartmouthatlas.org).

[2] See e.g., Fuchs (2004) and Enthoven (1978).

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 Hall, 2004).

There is a parallel literature in health care documenting similar lags in adoption, and with similar adverse effects on overall productivity. For example, 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).[3] Yet during the 18th century, more men in the British Navy died of scurvy than were lost to battle casualties (Lee, 2004). More recently, beta blockers, drugs costing pennies per dose, were shown during the early 1980s to reduce mortality by as much as 25 percent following a heart attack (Yusuf, et al., 1985). By 2000/2001, the median state-level use of beta Blockers among appropriate patients was still only 68 percent (Jencks, et al., 2003).

We develop a model in which output (survival) depends on factor inputs and the speed of technology diffusion. The hospital is assumed to maximize the present value of lives saved minus resource and learning costs. This in turn yields testable implications for the nature and extent of lags in total factor productivity among hospitals. We apply this model to the hospital-level treatment of patients diagnosed with an acute myocardial infarction (AMI, or a heart attack) using data on hospital-specific technology diffusion during 1994/95 for three inputs: aspirin, beta blockers, and reperfusion within 12 hours of the heart attack. (Reperfusion consists either of thrombolytic "clot-busting" drugs, or surgical angioplasty.) These treatments are: (a) proven to be effective in saving lives, (b) not so expensive as to cause financial barriers to diffusion, and

[3] In his 1601 voyage to India, Captain James Lancaster fed sailors in one of his ships 3 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 percent), while *none* of the sailors in the treatment group had been affected (Berwick, 2003).

(c) administered based on the decision of the physician, not by the supine heart attack patient. As well, we examine the diffusion of a much newer technology first introduced in 2003, drug-eluting stents, to identify *changes* over time in the speed with which new innovations diffuse in the hospital.

The model is tested using a sample of 2.8 million heart attack patients drawn from the fee-for-service Medicare population during 1986-2004. Hospitals are initially categorized into quintiles based on the diffusion of effective treatments in 1994/95. Like Comin and Hobijn (2004, 2008) who study country-level data, we find that hospitals with rapid diffusion in one highly effective technology are more likely to adopt other technologies. More importantly, we find that the 1994/95 quintiles of technology diffusion explain large variations across hospitals in risk-adjusted survival, and that these productivity effects swamp the influence of differences in factor inputs -- a result also found in the macroeconomics literature (e.g., Hall and Jones, 1999). And like Eaton and Kortum's (1999) study of aggregate productivity, we find substantial differences in the extent to which some hospitals lag behind, with an average gap of 3.3 percentage points in one-year survival between rapid-diffusing and slow-diffusing hospitals, nearly one-third of the overall improvement in outcomes during 1986-2004. Finally, we find that the "Asian tiger" hospitals which between 1994/95 and 2003/04 demonstrated dramatic improvements in diffusion rates also experienced above-average survival growth, and four times the growth in the "tortoise" hospitals that experienced a decline in diffusion rates.

These results can potentially reconcile the two 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 relatively inexpensive but effective treatments, rather than more factor inputs *per se*. Holding technology diffusion constant, however, we find modest improvements in outcomes associated with

spending more, with a preferred estimate of (at best) about $95,000 per life year during this period. At least for heart attacks, our results are inconsistent with the prevailing "flat of the curve" view of health care spending in the U.S.

The real puzzle is why many physicians and hospitals remain so far behind the production possibility frontier, contributing to a remarkable degree of productive inefficiency in health care. As we discuss below, our model suggests that some hospitals must either face very high barriers to the diffusion of effective health technologies, or must substantially undervalue the survival benefits of these technologies. In the conclusion, we speculate about why this might be the case for hospitals, and for technology diffusion more generally.

2. The Model

We focus on the "production" of survival following acute myocardial infarction (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. The outcome, survival, is accurately measured and there is broad clinical agreement that survival is the most important endpoint, 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).

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:

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

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 measures X_{it} more accurately than capital and labor inputs, we focus on the composite factor input rather than on capital and labor separately.[4]

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}

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

We adopt this special case to simplify the balanced-growth path of technological innovation, but in the empirical section allow for the more general translog production model (Christiansen, Jorgenson, and Lau, 1973), which allows for the marginal productivity of X_{it} to depend on technology.

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:

[4] In theory one could measure Part A hospital days and Part B physician resource-value units (RVUs), but the Part B data is available for just a 5 percent sample in earlier years, nor is hospital days always a good measure of health care intensity. See also Jacobs, Smith, and Street (2006) for an excellent discussion of measuring productivity in X.

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

In Equation (3), m_{ijt} 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 α_j is the return to adopting innovation j. The adoption rate in turn is written

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

In other words, this year's usage rate m is equal to last year's rate plus the institutional- and time-specific "core" diffusion rate π_{it} times the gap between best-practice (100 percent use) and last year's usage. We assume that each hospital chooses its adoption hazard across all new innovations at each point in time; we show below that this adoption rate is constant over time in steady-state.

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,

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

Thus, combining equations (3)-(5), the technology level at a given point in time can be written

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

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

Finally, we assume that there is a cost per patient of encouraging rapid adoption and diffusion, $C_i(\pi_{it})$, with $C' > 0$ and $C'' > 0$. The costs may include the obvious expenses of (e.g.) computerized information systems that prompt physicians when beta blockers or aspirin have *not* been administered, quality improvement initiatives, 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 other factors that affect the speed of diffusion (e.g., Rogers, 2003). This approach parallels other models in which physicians face different search costs and may hold different views about the value of new technology (see Phelps, 2000).

The Hospital Objective Function. There is considerable debate about the objective function of hospitals (e.g., Horwitz and Nichols, 2007); to avoid having to choose a specific model, we instead adopt a general objective function depending positively on survival and negatively on costs:

$$V_i = \sum_{t=0}^{\infty} \left[\Psi_i(a_{it} + \beta x_{it}) - \varphi_i P_{it} X_{it} - C_i(\pi_{it}) + K_{it}\right](1+r)^{-t} \tag{7}$$

where r is the discount rate, Ψ_i is the implicit social (dollar) value of improved health (assumed for simplicity to be constant over time), while K_{it} represents either fixed costs or subsidization from endowments or non-Medicare patient revenue. The provider-specific parameter φ_i reflects variation in the degree to which hospitals trade off the social cost of increasing X_{it} with the potential private benefits of doing more; for example when cardiac surgery generates profits, φ_i could be lower. Consider the special case where Ψ_i is equal to the social value of survival, and φ_i is one; for this case hospitals maximize social surplus. As we show in the Appendix, other models of hospital behavior reflecting the tension between financial profits and social welfare imply values of Ψ_i and φ_i below those corresponding to a social planner.

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;

$$\Im = V_i - \sum_{t=0}^{\infty} \lambda_{it}[a_{it+1} - a_{it} - \pi_{it}(a^*_{t+1} - a_{it})] \tag{8}$$

This model can also be written in continuous time as a current-value Hamiltonian, but we maintain a discrete time structure to help specify the empirical model. 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 π_i that is constant over time.

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

$$X_{it} = \Psi_i \beta / P_{it} \varphi_i \,. \tag{9}$$

Not surprisingly, factor inputs are greater when there is a higher implicit value by the hospital on saving a life-year Ψ_i, when the price of producing a factor input P_{it} is lower, and when financially motivated hospitals are reimbursed generously for care (φ_i is small). Optimal factor inputs are independent of the level of technology because the production function (Equation 2) assumes that the marginal product of factor inputs (β) does not depend on technology. In a more general specification that allowed for interactions between technology and factor inputs, optimal factor inputs would increase (decrease) if new technology increased (decreased) the marginal product of factor inputs.

For a constant growth rate α, it is straightforward to show that productivity in the steady state is given by:

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

Equation (10) states that the steady-state distance that a hospital lags behind the productivity frontier is a constant nonlinear function of the diffusion rate, in which small differences in diffusion can lead to very large differences in productivity (Parente and 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 α is annual productivity growth). Thus, a hospital with a 20% diffusion rate lags 4

years behind, and a hospital with a 5% diffusion rate lags 19 years behind. Equation 10 also implies that there is no convergence; productivity at all hospitals grows at the same rate as the frontier – α. This property has been noted in other papers as well (Eaton and 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:

$$C'(\pi_t) = \frac{\Psi\left(a_t^* - a_t\right)}{r + \pi} \tag{11}$$

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. Notice that the value of the incremental innovation decays both by the interest rate r, but also by the diffusion rate π; the value of adopting today is attenuated by the likelihood of adopting anyway sometime in the future, under than the status-quo π. Note that we can use Equation 11 to back out the implicit marginal cost of raising the underlying diffusion rate. We consider below plausible sets of parameters that satisfy this first-order condition.

3. Empirical Specification

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:

$$y_{it} = a_{it} + \beta x_{it} + u_{it} \tag{12}$$

Using the steady-state assumption from Equation (11), Equation (12) is rewritten

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

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 π_i. However, several challenges remain: x_{it} and y_{it} must be constructed from individual-level data; π_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.

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:

$$S_{lit} = Z_{lit}\Gamma + \sum_{i=1}^{H} \gamma_{it} + e_{lit} \tag{14}$$

The dependent variable, S_{lit} is a one-zero 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, Γ a vector of coefficient, γ_{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 (or DRG) weights across all hospital admissions, which reflect the Centers for Medicare and Medicaid Services (CMS) assessment of resources necessary to provide specific and detailed procedures. The hospital-year intercepts from Equation 14 (γ_{it}) are used in our subsequent estimation as risk-adjusted measures of survival and factor inputs.

Estimating each hospital's rate of diffusion. We use data on the diffusion of various innovations at a point in time to estimate the underlying diffusion rate at each hospital. In steady-state with a constant hospital-specific π_i, equation (4) implies that the cumulative adoption of

each innovation can be expressed as $m_{jit} = 1-(1-\pi_i)^{t-j}$, where m_{jit} is as before the (fractional) use of the jth innovation at hospital i and time t. In other words, the current rate of use of an innovation depends simply on the number of years it has been available (t-j) and the "core" speed of adoption at the hospital (π_i). Taking a first order approximation that $(t-j)\pi_i \approx 1-(1-\pi_i)^{t-j}$ and adding a stochastic term (v_{jit}) to allow for random fluctuations over time allows us to express m_{jit} as

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

Equation 15 describes a factor model, in which the dependent variable is the adoption rate of a given innovation by a given year, the common factor (π_i) captures the intensity of search for new innovations at hospital i, and the factor loading (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. One is to simply enter the common factor (which is proportional to π_i, but normalized to have mean zero and standard deviation one) linearly on the right-hand side of Equation (13). But Equation (13) implies a nonlinear influence of π on survival, and so we also create patient-weighted hospital-level quintiles of the common diffusion factor. Finally, in some specifications of equation (13) we include hospital fixed effects to proxy for each hospital's diffusion parameter. 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 π_i, resulting in estimates of β that are less subject to omitted variable bias due to unmeasured differences in diffusion.

Relaxing the assumption of a steady-state model. If hospitals are not in steady-state (e.g., because of changing costs of diffusion) then π_{it} will not be constant over time, and the equation for current survival becomes more complex. Using a first order approximation (valid for small π_{it}), equation (13) becomes :

$$y_{it} = \sum_{k=1}^{t} k\alpha\pi_{ik} + \beta x_{it} + u_{it} \tag{16}$$

where the summation captures the impact of the diffusion in each time period k on all k innovations that were available at that time. One approach to testing the model is to study survival rates of hospitals experiencing large changes in measured diffusion; from (16) one can see that changing rates of diffusion will shift health outcomes upward (the productivity "tigers") or downward (the "tortoises") to new steady-state output levels.

A semi-parametric approach to estimating the model. In some specifications, we estimate a flexible translog production function (Christiansen, Jorgenson, and Lau, 1973) to allow for diminishing returns to x_{it}, and interaction between diffusion and the productivity of x_{it}:

$$y_{it} = a_{it} + \beta x_{it} + \nu_1 a_{it} x_{it} + \nu_2 a_{it}^2 + \nu_3 x_{it}^2 + u_{it} \tag{17}$$

We estimate this model in a slightly more general formulation, by stratifying across quintiles of diffusion and allowing coefficients on x_{it} and x_{it}^2 to vary across quintiles.

The error term could be correlated with factor inputs. Estimates of the return to factor inputs (β) in Equation (13) may be biased by correlation between factor inputs (x_{it}) and the error term. There are two reasons to suspect such a correlation.

First, if there are interactions between technology and the return to factor inputs (as would be the case in the translog specification), then the optimal factor inputs will depend on the level of technology at each hospital. To the extent that our proxy for technology diffusion at each hospital is imperfect, the error term in Equation (13) will reflect some remaining technology

differences. This will bias the coefficient on factor inputs downwards (upwards) if higher levels of technology are associated with lower (higher) optimal use of factor inputs. To investigate the importance and direction of the bias arising from omitted technology differences, we present two types of evidence. First, we estimate Equation (13) with more and less detailed controls for technology diffusion, ranging from no controls to hospital fixed effects.[5] Second, we estimate the more general translog specification in Equation (17) to investigate whether the return to factor inputs varies with technological diffusion.

A second reason to suspect a correlation between factor inputs and 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 also tend to account for more spending. To address this issue, we also present estimates that replace x_{it} with lagged measures of factor inputs x_{it-1}, thus sampling the independent variable from the year t group of patients and the dependent variables from the year t-1 group. Instead, independent sampling error in x_{it-1} will bias the coefficient toward zero.

One approach to the endogeneity of factor inputs, which we do not take, is to estimate an instrumental variables model that seeks to express x_{it} in terms of "fundamentals." Note that we have already derived the first-order conditions for X in Equation (9); in log terms one can write:

(9') $$x_{it} = \ln(\Psi_i) + \ln\beta - \ln(P_{it}) - \ln(\varphi_i)$$

It is certainly possible to think of factors that might be associated with each parameter, for example for-profit or government status of the hospital (leading to a lower or higher φ_i), or higher state-level income (positively associated with Ψ). But all of the variables we considered

[5] Even with hospital fixed effects there may be bias because of changing technology diffusion over time.

are inappropriate instruments because they are likely to affect survival beyond their impact on x_{it}. Rather than use questionable instruments, we eschew the IV approach and interpret the estimate of β with caution.

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

$$CE = \frac{dC}{dX} \Big/ \left[\frac{dy}{dX} \frac{dL}{dy} \right] \tag{18}$$

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).[6]

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 often favor much larger estimates, of up to $250,000 per life year for older people (Hirth, et al., 2000, Murphy and Topel, 2006).

4. The Diffusion of Efficient Treatments for Acute Myocardial Infarction

Information on technology diffusion was measured in the Cooperative Cardiovascular Program (CCP) dataset, which involved chart reviews for over 160,000 AMI patients over age 65 during 1994/95, matched to the admitting hospital. We chose three measures of low-cost but effective innovations. The first, aspirin, reduces platelet aggregation and helps to limit clotting, thereby improving blood flow to the oxygen-starved tissue, and by 1988 it was included in

[6] The average inpatient cost of one year of inpatient treatment following AMI was equal to equal to $26,063 in 2004. These estimates ignore outpatient and physician costs, which are likely to add at least 15% to costs (and hence 15% to the CE ratio). The equation simplifies when we use expenditures rather than DRGs as inputs.

standard guidelines for care (ISIS-2, 1988). Heidenreich and McClellan (2001) viewed aspirin as the single most important factor in explaining why 30-day mortality rates declined during 1975-95.

The second, a beta blocker, is an inexpensive drug that by blocking the beta-adrenergic receptors reduces the demands on the heart. In a meta-analysis from 1985, Yusuf et al. summarized the existing literature as "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) By 1994/95, diffusion fell far short of ideal: average use among AMI patients was just 46 percent.

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 either by using thrombolytics, drugs which help break down the clots blocking the blood, or angioplasty, in which a "balloon" is threaded through a vein into the blocked artery and expanded, thus restoring blood flow. Since 1995, cardiologists have increasingly adopted stents, cylindrical wire meshes, to maintain blood flow following the angioplasty. The two treatments (thrombolytics and angioplasty/stents) are substitutes because thrombolytics reduce the patient's ability to clot after invasive surgery. Randomized trials have shown both to be effective, but with most studies showing slightly larger benefits for primary angioplasty. By 1994/95, many larger hospitals had catheterization laboratories, but thrombolytics were a viable option for nearly every hospital.

The factor model (Equation 15) was estimated using the proportion of patients receiving each treatment for each hospital in 1994/95, and assuming a single common factor. Factor analysis normalizes the underlying factor to have a mean of zero and variance of one, so the units of the estimated factor have no particular interpretation. Table 1a presents the correlation coefficients among the three variables (aspirin, beta blockers, and reperfusion) and the estimate

of the common factor. The correlation of each input with the common factor ranges from 0.87 for beta blockers to 0.30 for reperfusion, demonstrating that hospitals that adopt one innovation early are also more likely to adopt other innovations. Note that the correlation between beta blockers and reperfusion is only 0.03, reflecting in part the specialization of some hospitals into surgical treatments for AMI (Chandra and Staiger, 2007).

In Table 1b, we show that the quintiles based on this common factor show clear differences in the use of beta blockers (from 65 percent in the highest adopting Quintile 5 to 31 percent in the lowest Quintile 1) and aspirin (90 percent to 65 percent), with more modest differences in reperfusion (21 percent to 15 percent).[7] One could interpret these patterns as reflecting demand; patients in high quintile regions ask for and get beta blockers, for example. But this seems unlikely; elderly heart attack patients are unlikely to be requesting specific treatments, with few knowing the value of beta blockers or aspirin. More to the point, hospitalized patients should not *have* to ask their physicians for these treatments given their clear benefits.

Table 1b also demonstrates that hospitals in the quintiles with quicker adoption also have higher patient volume, are more likely to be major teaching hospitals, and are located in states with slightly higher average income (which proxies for a higher social value per life year). These hospitals are likely to experience both a lower marginal cost of diffusion and place a higher value on more rapid diffusion.

The 1993/94 diffusion measures provide two independent predictions on steady-state differences in productivity across quintiles of hospitals. The first arises from the implications of

[7] 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 β blockers and aspirin, the optimal rate for revascularization is substantially lower.

the model that annual adoption rates are predictive of differences in output. Assume that aspirin use in 1980 for each quintile was equal to the national average of 6 percent (Heidenreich and McClellan, 2001). Based on Table 1b, the implicit adoption rate in the slowest quintile is $\pi = 6$ percent, and in the quickest adopting quintile $\pi = 14$ percent. Plugging these adoption rates into Equation 10, Quintile 5 survival is predicted to be 8.5 years ahead of Quintile 1 survival. Under reasonable assumptions, the corresponding estimates for beta blockers and for reperfusion are similar, implying a 5-20 year gap in the survival rates between Quintiles 1 and 5.

A second approach predicts the difference in survival probabilities rather than with regard to the number of lagged years.[8] Based on estimates from randomized trials, the differences in the use of aspirin, beta blockers, and reperfusion from Table 1b together imply about a 3.9 percentage point gap between the highest and lowest diffusion quintiles.[9]

The final rows of Table 1b shows patterns of diffusion for a quite different innovation: drug eluting stents. As noted above, stents are used to maintain blood flow following angioplasty. 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. [10] We linked the hospital-specific measures of the diffusion of drug-eluting stents, as described in

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[8] One can also multiply the average number of lagging years between Quintiles 1 and 5 times the average annual productivity gain to infer the long-run differences in terms of survival.

[9] Multiplying the 22 percent decline in one-year mortality arising from beta blockers (Phillips, et al., 2000), times a baseline 30 percent mortality probability and a 34 percentage point gap in beta blocker use between quintiles 1 and 5 implies leads to 2.2 percentage point lower mortality. For aspirin, the equivalent estimate was 1.5 percentage points (based on 18 percent lower mortality from aspirin, as in Krumholz et al. 1995). Much smaller effects are estimated (0.2 percent) from the gap across the quintiles in 12-hour reperfusion (FTT, 1994).

[10] While there has been some controversy in 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 patients, and not solely AMI patients.

Malenka et al. (2008), to the earlier diffusion quintiles. Hospitals with the most rapid diffusion of cardiac technology in 1993/94 were both more likely to implant stents in 2003/04 – many hospitals do not have cardiac catheterization laboratories – and conditional on having catheterization facilities, were more likely to have adopted drug-eluting stents, with 61% diffusion rates compared to 53% in the slowest diffusing quintile (Table 1b). Knowing rates at two different points in time allow us to measure *changes* over time in hospital diffusion rates.

5. Data and Estimation in a Panel of AMI Patients, 1986-2004

The primary dataset is a 20% sample of the Medicare Part A (hospital) claims data for all heart attack (AMI) patients age 65 and over in the U.S. during 1986 – 1991, and a 100% sample from 1992 through 2004, with updated information on mortality through 2005.[11] The original sample comprises 3.3 million people. We eliminated hospitals with fewer than 5 patients in any of the 100% sample years (and any hospital that closed during the period of analysis), resulting in a final sample limited to 2.8 million people. The Medicare claims data includes detailed information on comorbidities (i.e., preexisting conditions), as well as the location and type of heart attack. We use these data to test several implications of the model, particularly the predictions on survival differences based on the CCP data described above.

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: 1-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

[11] One concern is bias resulting from out-of-hospital AMI deaths which do not appear in our sample. For example, a positive correlation between the quality of emergency medical services and hospital technological diffusion would bias our results towards zero since sicker patients are more likely to survive to the emergency room.

measure of factor inputs.[12] These regressions included categorical variables indicating the presence of seven comorbid conditions, anatomical location of the MI, and full interactions of each 5-year age bracket, by sex and race. The initial risk-adjustment regression is shown in Table A.1 along with relevant means of the independent variables for the entire sample, for both one-year survival and one-year expenditures.

A first look at the data. We begin with summary statistics showing the time-trend in risk adjusted survival – in other words, the weighted averages of y_{it} across hospitals within each time period. Figure 1 shows these risk-adjusted one-year survival and one-year expenditures by year. Survival rose rapidly during the late 1980s and early 1990s (the period of analysis in Cutler et al., 1998), but since then has flattened out, particularly in the late 1990s, before assuming a more modest upward trend in the 2000s.[13] And while the 1997 Balanced Budget Act legislation led to a pause in the rapid cost growth, expenditures have since resumed their upward trend.

We show graphically the bivariate association between technology adoption and risk-adjusted survival in Figure 2. These display the weighted average of risk-adjusted one-year survival (y_{it}) by year and by quintile of our diffusion index (the common factor described in Section 4). The average gap in survival between the slowest and most rapid adopters is more than 3 percentage points, with the difference widening to 3.5 percent by 2004. The magnitude of these differences are similar to the estimates we suggested in Section 4 based on what clinical

[12] 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.

[13] What can explain this pattern of diminishing returns to technology after the mid-1990s? One possibility is that by this time, aspirin had largely diffused across all patients, and while stents grew also during this period, they "have not been associated with important reductions in mortality." (Brody, et al, 2003, p. 777) But it is less clear why beta blockers, which were diffusing during this period, didn't lead to more improvement in outcomes; Masoudi et al. (2006) suggested a secular decline in "ideal" patients for such treatments.

trials imply would result from the difference between quintiles in the use of aspirin, beta blockers, and reperfusion. 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 ten years, which is again within the range predicted by the diffusion calculations above.

Figure 3a displays Medicare reimbursements again by quintile of adoption. There are modest differences in expenditures, with Quintile 5, the most rapidly adopting hospitals, consistently higher. However, this difference is based primarily on higher reimbursement rates rather than more inputs *per se*. Figure 3b shows no difference in expenditures by quintile using a normalized "price" per DRG weight based on the national average. In sum, there are large long-run differences in total factor productivity across hospitals, and these do not appear to be associated with higher rates of factor inputs.

Estimates of the productivity parameter. 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 a linear time trend. The coefficient on the diffusion index is 0.017 (s.e., 0.001). Recall that the diffusion index is an estimate of a common factor that is proportional to the diffusion rate in each hospital, but normalized to have a standard deviation of one. Thus, the coefficient implies that a one standard deviation increase in the diffusion rate is associated with a 1.7 percentage point increase in patient survival – or has approximately the same impact as doubling DRG inputs (e.g., ln(2)*.031=.021, or 2.1%). Adding year effects (Column 2) has little impact on the coefficient. Columns 3 and 4 replace the continuous diffusion index with dummies for the hospital-specific diffusion quintile, with Quintile 1 (the slowest diffusion quintile) serving as the reference category. The coefficient on Quintile 5 relative to Quintile 1 is 0.033 (s.e. 0.002), implying that survival for hospitals in the fastest diffusion quintile was 3.3 percentage points higher than

hospitals in the slowest quintile. This difference in survival corresponds to the fastest quintile being 11 years ahead of the slowest quintile (the coefficient on the linear trend estimates that the annual survival improvement was 0.3 percentage points). Again, the differences between Quintiles 1 and 5 are within the range predicted by the diffusion calculations in Section 4.

Estimates of β, the marginal productivity of factor inputs. Table 3 examines how the specification of the model affects estimates of β, and interprets them in the context of the cost-effectiveness ratio. Column (A) of the table reports specifications that do not include year effects. These regressions are in the spirit of Cutler et al. (1998) who relied solely on variation over time to identify the average impact of changes in both technology and factor inputs on survival. In the upper-left corner, we first consider the coefficient estimates from regressions of survival just on log expenditures, without controlling for technology or year. The coefficient on log of expenditures, 0.028, is highly significant and implies a cost-effectiveness ratio of $177,000. The second row, which uses log DRG inputs, delivers a much larger estimate of 0.076, implying a far more favorable cost-effectiveness ratio of $65,000 per life-year. The bottom two rows suggest diminishing returns to factor inputs over time; the estimated β is larger in the earlier period (1986-94) than the later period (1995-2004). The cost-effectiveness ratio in the earlier period is $41,000, which is similar to that obtained by Cutler et al. (1998) using data from this period.

However, once one introduces year effects – the second column of Rows 1 and 2 – the coefficient becomes negative (for expenditures) or 0.014 (when using DRGs as factor inputs), implying at best a cost-effectiveness ratio of $355,000. By controlling for year effects, one ends up with the small or negative cross-sectional correlations found in Fisher et al. (2003) and Baicker and Chandra (2004). The final column, using lagged expenditures, finds an even more negative relationship between factor inputs and survival.

Note, however, that the specifications in the first two rows of Table 3 could be biased because they omitted any control for technological adoption. As we control successively more for the influence of technological adoption, the coefficient estimates for β rise, and the cost-effectiveness estimate declines. For example, for the specifications in column (b) that include year effects, introducing the linear diffusion parameter increases the coefficient from 0.014 to 0.019 (a cost-effectiveness ratio of $261,000) and the quintiles of diffusion yields similar results, with a coefficient of 0.020. 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 β to 0.052, with an implied cost-effectiveness ratio of $95,000.

This pattern of coefficients is consistent with our model if the return to factor inputs is lower in hospitals with higher technology diffusion, as represented graphically in Figure 4 for a given year. Consider just two hospitals, given by A (on the production function PF(1)) and B (on the production 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 the estimated (marginal) slope of the production function becomes steeper, to approximate aa' or bb' in Figure 4.

As mentioned in Section 3, year-to-year sampling fluctuations in both factor inputs and survival may cause an upward bias in estimated β when people who live longer account for more costs. Limiting the sample to hospitals with more than 50 AMI patients (not reported in Table 3) implied a cost-effectiveness of $160,000 instead of $95,000 (for a specification with hospital fixed effects) – still not "flat of the curve." Alternatively, we also estimated models using lagged

x_{it-1} instead of contemporaneous x_{it}, with estimated coefficients in the last column. In general, these estimated effects are near zero.

For the specification with hospital fixed effects, which controls most completely for technology diffusion, the estimated coefficient on factor inputs is slightly positive (.003). But this estimate most likely represents a lower bound on the slope of the production function for three reasons. First, there is still likely to be some within-hospital variation in technology diffusion over time, generating negative bias as illustrated in Figure 4. Second, the lagged factor input now contains independent sampling error, attenuating the coefficient toward zero. Finally, since estimates with hospital fixed effects rely on data that have been demeaned at the hospital level, the demeaned lagged factor inputs will be negatively correlated with current factor inputs, leading to a negative bias analogous to the lagged dependent variable bias in panel data.

In the hypothetical production functions shown in Figure 4, the slopes of each production function differ, both across hospitals and with respect to how much they spend. In Table 4, we report estimates using the translog production function, stratified by technology quintile. In order to most completely control for difference in technology, we also include hospital and year fixed effects in these specifications. Coefficient estimates along with corresponding CE ratios are reported for the 25^{th}, 50^{th}, and 75^{th} percentile of the hospital-level distribution; these in turn are displayed in Figure 5 centered on 2004 data. There are diminishing returns to expenditures; the CE ratio in the median quintile of adoption ranges from \$103,000 (25^{th} percentile) to \$310,000 (75^{th} percentile). Furthermore, the hospitals with the most rapid diffusion experience the *poorest* return to further expenditures. This result implies that hospitals with high levels of technology adoption will optimally choose lower levels of expenditure – which is consistent with the pattern of findings in Table 3. In other words, once aspirin, β blockers, and primary reperfusion have been adopted, hospitals use less of factor inputs because the incremental returns to further inputs

such as surgery are modest, a result also found in the clinical literature (Stukel, Lucas, and Wennberg, 2005). And like Hall and Jones (1999), the rate of technology diffusion explains far more variation in survival outcomes across hospitals than variations in factor inputs.

Convergence. As noted above, 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 2 by noting that the range of Quintile 1 and Quintile 5 is not narrowing; if anything the range is widening. 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 (σ-convergence). We do not find evidence of such convergence: our estimate of the (weighted) standard deviation of hospital fixed effects, correcting for estimation error, is 0.043 in 1986 and 0.042 in 2004. [14]

Changes over time in diffusion rates. A prediction of the theoretical model is that hospitals which manage to improve their diffusion parameters will, like countries such as Japan or Korea in the postwar period, experience rapid growth in outcomes (Parente and Prescott, 2002), and conversely. Table 4 further considers risk-adjusted survival among hospitals which were initially in the slowest diffusion quintile (1) or the highest diffusion quintile (5) during 1994/95. For the slow-diffusion hospitals in 1994/95 remaining a slow diffusion hospital (in drug-eluting stents) in 2003/04, one-year survival rates rose from 64.5 percent in 1994/95 to 68.8 percent in 2003/04; an increase of 3.7 percent. Similarly, hospitals initially in the highest diffusing quintile (5) in 1993/94 which remained in the highest diffusing quintile by 2003/04,

[14] The correction is done by subtracting the average variance due to estimation error in the hospital fixed effects (the "noise" component). The estimation variance for each hospital's fixed effect is equal to σ^2/N_h, where σ^2 is the variance of the error in the patient-level risk-adjustment equation and N_h is the number of AMI patients at hospital h.

increased survival by 3.1 percentage points – very similar to the stable low-quintile hospitals, as predicted by the model.

Hospitals initially in the lowest diffusion quintile during 1994/95 but which moved up to the highest diffusion quintile for drug-eluting stents in 2003/04 (the "tigers") experienced a gain of 5.5 percentage points. By contrast, the hospitals experiencing a decline in diffusion rates from quintile 5 in 1994/95 to quintile 1 in 2003/04 (the "turtles") showed a survival gain of just 1.8 percentage points, significantly below those of the "tiger" hospitals.[15] Thus, observed changes in technology diffusion are strongly related to changes in hospital productivity as measured by patient survival.

5. Conclusion

In this paper, we have attempted to peer inside the black box of hospital productivity changes both 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 1986-2004. The hospital quintile with the most rapid propensity to adopt these new innovations experienced survival rates 3.3 percentage points above the lowest quintile hospitals, or nearly one-third the entire improvement in survival since 1986. While we focused on just three innovations at a point in time, aspirin, beta blockers, and reperfusion in 1994/95, we view the results, and the non-convergence of the quintile outcomes, as supportive of the view that these hospitals have continued to innovate since then. Indeed, the "tiger" hospitals, those which increased their diffusion rates for new innovations, experienced far

[15] One hypothesis is that hospitals that were early adopters of surgery in 1994/95 would also be early adopters of drug-eluting stents in 2003/04, 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 1994/95 than beta blockers or aspirin in 1994/95. Note also that the baseline risk-adjusted survival rates for the stable hospitals were similar to those for the hospitals which subsequently moved (either up or down), lessening a potential concern that selection bias drives these results.

more rapid growth in survival outcomes between 1994/95 and 2003/04 than did the "turtle" hospitals whose speed of diffusion slipped from the highest to the lowest quintile.

Our model of health care productivity reconciles both the dramatic improvements in life expectancy for AMI patients over time (e.g., Cutler, 2004) and the apparent "flat of the curve" inefficiencies at a point in time (Fisher et al. 2003). 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 the single most important factor reducing the number of AMI-related deaths between 1980-2000 was the increased use of aspirin, followed by beta blockers and ACE Inhibitors (pharmaceutical treatments to reduce hypertension).

But at a point in time, it might appear that greater levels of factor inputs did not result in improved outcomes. We argue that, at least in the treatment of heart attacks, this "flat of the curve" association between factor inputs and outcomes may be more apparent than real, arising because we have not adequately controlled the diffusion parameters that largely determine hospital productivity. Furthermore, we find the marginal productivity of factor inputs is higher in the low adoption hospitals, suggesting that the high-cost factor inputs may be substitutes for the low-cost innovations (Chandra and Staiger, 2007). Of course, these estimates are sensitive to the specification of the model, so we cannot rule out "flat of the curve" spending entirely, particularly for the higher-intensity hospitals. Nor do these results necessarily apply for the treatment of diseases other than heart attacks, where technological gains have been far less prevalent.

There are a variety of optimizing economic models where rational agents 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 and 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, 2006). Finally, there may be differences in education across workers which affect their propensity to adopt (Nelson and Phelps, 1966) or technology may be most complementary with skilled workers (Caselli and Coleman, 2006). None of these models provides a good explanation of the non-adoption of inexpensive beta blockers, aspirin, and reperfusion by highly educated physicians. [16]

Because prices do not play an important role here, we instead look to informational or search barriers as an explanation for why physicians don't adopt. Recall Equation (11) which posited a first-order condition in which the marginal cost of speeding up diffusion C'(π), was set equal to the marginal benefit of innovating more rapidly. Using plausible parameters for measuring the social value of more rapid adoption yields a very high cognitive barrier: the implicit cost facing each physician of moving up just one diffusion quintile must be $11,200 annually. [17] Alternatively, the implicit value placed by hospitals and physicians on a life-year must be well below $25,000 per life-year to generate "reasonable" equilibrium conditions to explain observed slow diffusion rates.

One might also appeal to models of social norms to explain why innovations diffuse more rapidly in some regions than others, whether hybrid corn in the 1930s and 1940s or beta blockers in the 2000s (Skinner and Staiger, 2007), but the direction of causality is not well understood. The quality of management, including staff "opinion leaders," is clearly central to the rapid diffusion of beta blockers (Bradley, et al., 2001, 2005). Thus the diffusion parameters could as well be *symptomatic* of managerial efficiency, which has shown in non-health industries to be an

[16] The distinction between "inefficient" barriers to adoption, and the slow, but optimal, adoption of technology for a variety of reasons, was made by Coleman (2004).

[17] 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, $r = .05$, baseline $\pi = .10$, π must increase by 0.016 to shift to the next quintile (one-fifth the range between 6 and 14 percent, the implicit aspirin diffusion rates), and there are 10 AMI patients per physician.

important determinant of productivity (Bloom and van Reenan, 2007). But even after accounting for the lower productivity in hospitals compared to other industries (Bloom, Seiler, and van Reenan, 2007), one is still left with a puzzle of why individual physicians need management or opinion leaders to convince them to adopt *aspirin* for their heart attack patients.

Leibenstein (1966) used the term "X-efficiency" to describe residual differences in firm-level productivity which could not be readily explained by measured inputs or other factors. In many respects, the puzzle of slow diffusion for efficient AMI treatments provides a textbook case of X-inefficiency, because here at least we can observe directly several productivity measures rather than infer them as residuals, as Leibenstein did. While informational barriers are indeed important –there may be no one in the hospital to provide the "tactile" learning when reading an article just isn't enough (Keller, 2004) – there has historically been 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 (Lee, 2007).

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. Health care markets are notoriously imperfect. If patients both 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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Table 1a: Characteristics of Factor Model of Adoption: Correlation Structure

	Common Factor	Aspirin	β Blocker	Reperfusion
Common Factor				
Aspirin	0.871*			
β Blocker	0.792*	0.429*		
12 Hour Reperfusion	0.300*	0.189*	0.031	

Notes: The table reports correlations at the hospital level (N = 2765) weighted by number of patients in each hospital. Data from the Cooperative Cardiovascular Project (CCP), 1994/95, with a sample of 139,847 AMI patients. * denotes $p < 0.001$

Table 1b: Characteristics of Factor Model of Adoption: Association with Characteristics of the Hospital

	Quintile 5 (Quickest)	Quintile 4	Quintile 3	Quintile 2	Quintile 1 (Slowest)	Overall
Aspirin	0.90	0.85	0.80	0.76	0.65	0.80
β Blocker	0.65	0.53	0.46	0.40	0.31	0.47
Reperfusion within 12 hours	0.21	0.20	0.19	0.18	0.15	0.18
Average hospital volume*	95	101	94	88	67	89
Major teaching hospital	0.43	0.30	0.23	0.17	0.05	0.24
Average State Income (1994/95)	43,790	42,603	42,168	42,215	41,648	42,495
% Admitted to Hospital Performing Stents in 2003 /04	0.73	0.70	0.60	0.50	0.31	0.57
Of those, % Drug-Eluting Stent 2003/04	0.61	0.62	0.39	0.55	0.53	0.59

See notes above in Table 1a. *Volume for Medicare patients only. Weighted by number of patients in each hospital. Estimates for each quintile are based on samples of approximately 28,000 AMI patients. Stent data are derived from Medicare Part A (hospital) claims.

Table 2: Regression Estimates of Survival on Technology Diffusion and Factor Inputs

Input	1	2	3	4
Diffusion (continuous)	0.017 (0.001)	0.017 (0.001)		
Diffusion Quintile 2			0.012 (0.002)	0.012 (0.002)
Diffusion Quintile 3			0.020 (0.002)	0.020 (0.002)
Diffusion Quintile 4			0.028 (0.002)	0.028 (0.002)
Diffusion Quintile 5			0.033 (0.002)	0.033 (0.002)
Log (DRG)	0.031 (0.004)	0.019 (0.004)	0.031 (0.004)	0.020 (0.004)
Year Trend	0.0030 (0.0001)	Fixed effects	0.0030 (0.0001)	Fixed effects

Notes: N = 49,937 hospital-years. All regression weighted by the number of patients in each hospital-year. Sample limited to hospital/year observations with at least 5 observations per hospital. Standard errors (clustered at the hospital level) in parentheses.

Table 3: Regression Estimates of Survival on Factor Inputs for Alternative Specifications

	Input	Period of Analysis	Adj. for Diffusion	(A) No Year Effects	(B) Year Effects	(C) Year Effects Lagged Input
1	Log(Expend)	86-04	No	0.028 (0.002) [$177,000]	-0.010 (0.003) [Undefined]	-0.015 (0.003) [Undefined]
2	Log(DRG)	86-04	No	0.076 (0.003) [$65,000]	0.014 (0.004) [$355,000]	-0.015 (0.004) [Undefined]
3	Log(DRG)	86-04	Continuous Measure	0.078 (0.003) [$64,000]	0.019 (0.004) [$261,000]	-0.010 (0.003) [Undefined]
4	Log(DRG)	86-04	Diffusion Quintile	0.078 (0.003) [$64,000]	0.020 (0.004) [$248,000]	-0.010 (0.003) [Undefined]
5	Log(DRG)	86-04	Hospital Fixed Effect	0.102 (0.002) [$49,000]	0.052 (0.003) [$95,000]	0.003 (0.003) [>$1 mill.]
6	Log(DRG)	86-94	Hospital Fixed Effect	0.122 (0.004) [$41,000]	0.068 (0.004) [$73,000]	-0.004 (0.004) [Undefined]
7	Log(DRG)	95-04	Hospital Fixed Effect	0.047 (0.004) [$106,000]	0.043 (0.004) [$115,000]	0.002 (0.004) [>$1 mill.]

Notes: See notes to Table 2. Each entry in the table is the coefficient on factor inputs from a different specification as indicated in the table. Models with lags drop data from 1986, and have N=46,098. Cost-effectiveness ratios in brackets.

Table 4: Regression Estimates Stratified by Quintile of Diffusion

	Coefficient Ln(DRG)	Coefficient Ln(DRG)^2	CE Ratio: 25th Percentile	CE Ratio: 50th Percentile	CE Ratio: 75th Percentile
Quintile 1 (Slowest) N = 13,968	0.233 (0.033)	-0.060 (0.011)	0.071 (0.006) [$70,000]	0.057 (0.006) [$87,000]	0.045 (0.007) [$110,000]
Quintile 2 N = 10,132	0.214 (0.038)	-0.058 (0.014)	0.058 (0.007) [$86,000]	0.045 (0.007) [$110,000]	0.032 (0.009) [$155,000]
Quintile 3 (Middle) N = 8,923	0.243 (0.037)	-0.073 (0.013)	0.048 (0.006) [$103,000]	0.031 (0.007) [$160,000]	0.016 (0.008) [$310,000]
Quintile 4 N = 8,183	0.219 (0.043)	-0.061 (0.016)	0.053 (0.057) [$94,000]	0.039 (0.008) [$127,000]	0.026 (0.009) [$191,000]
Quintile 5 (Fastest) N = 8,731	0.259 (0.038)	-0.080 (0.014)	0.044 (0.007) [$113,000]	0.025 (0.007) [$199,000]	0.009 (0.009) [$552,000]

Notes: See notes to Table 2. Each row reports results from estimating a regression of survival on ln(DRG) and ln(DRG)^2, controlling for hospital and year fixed effects. Cost-effectiveness ratios in brackets.

Table 5: Growth in Risk-Adjusted Survival by Diffusion Quintiles in 1994/95 and in 2003

	Survival: 1994/95		Survival: 2003/04		Change in Survival: 1994/95 to 2003/04	
	2003/04 Quintile 1 (Slowest)	2003/04 Quintile 5 (Fastest)	2003/04 Quintile 1 (Slowest)	2003/04 Quintile 5 (Fastest)	2003/04 Quintile 1 (Slowest)	2003/04 Quintile 5 (Fastest)
1994/95 Quintile 1 (Slowest)	0.651 (0.008)	0.656 (0.013)	0.688 (0.004)	0.712 (0.012)	0.037 (0.009)	0.055 (0.015)
1994/95 Quintile 5 (Fastest)	0.684 (0.006)	0.690 (0.005)	0.701 (0.007)	0.721 (0.006)	0.013 (0.008)	0.031 (0.006)

Notes: All standard errors are clustered at the hospital level.

Figure 1: One-Year Risk-Adjusted Survival Rate and One-Year Inpatient (Part A) Hospital Expenditures Following AMI (2004$)

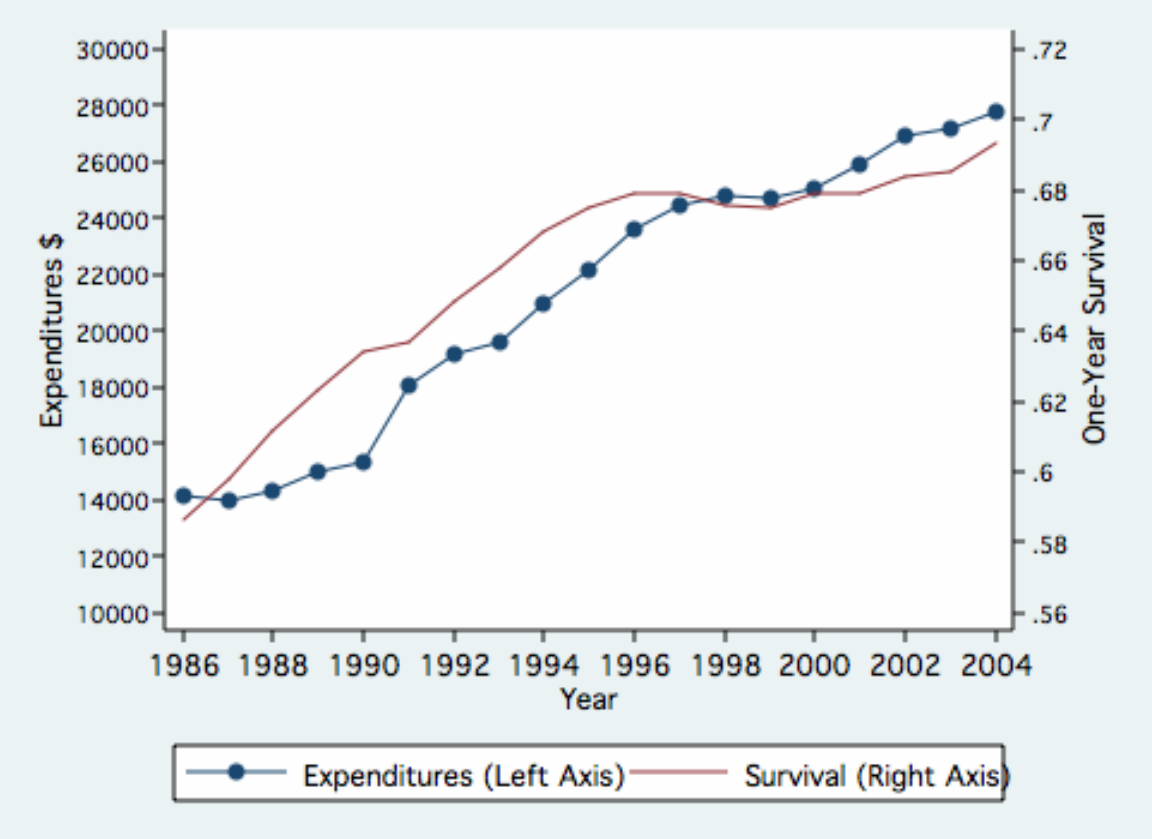


Figure 2: Survival Rates by Year and Diffusion Quintile, 1986-2004

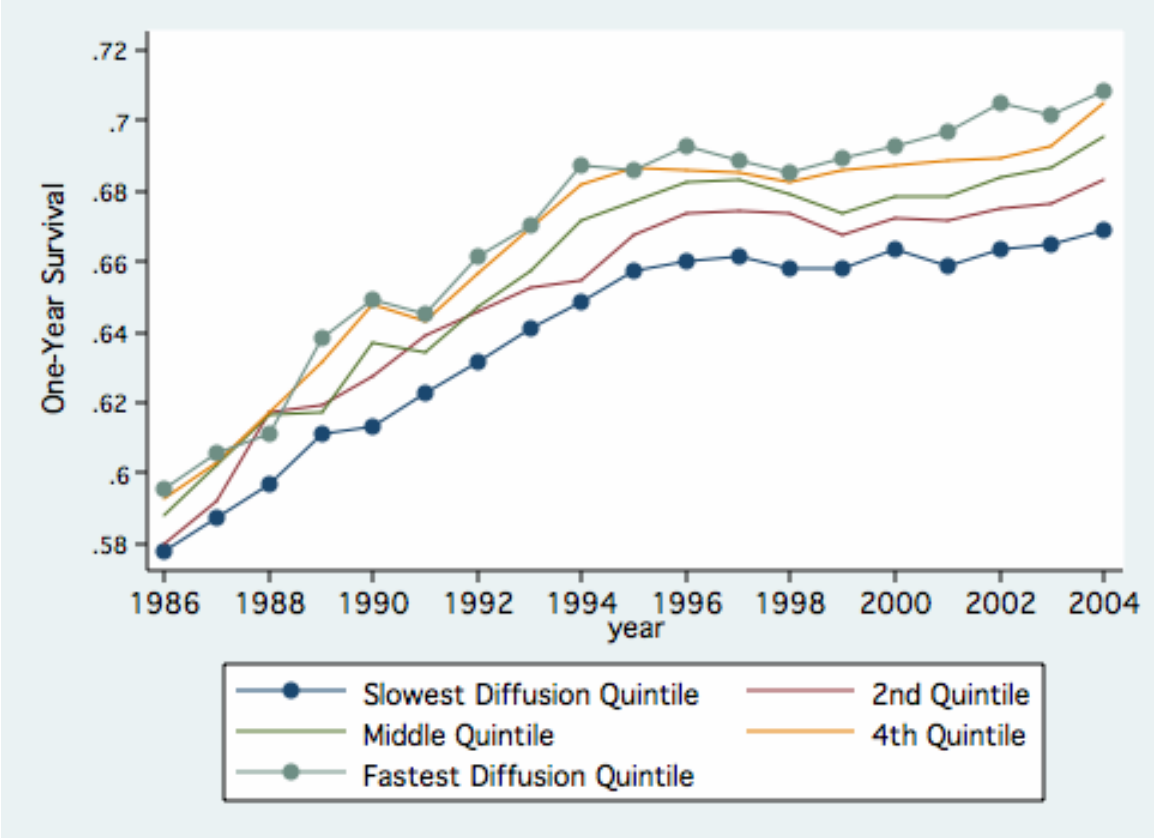


Figure 3a: Medicare Reimbursement by Year and Quintile of the Propensity to Adopt

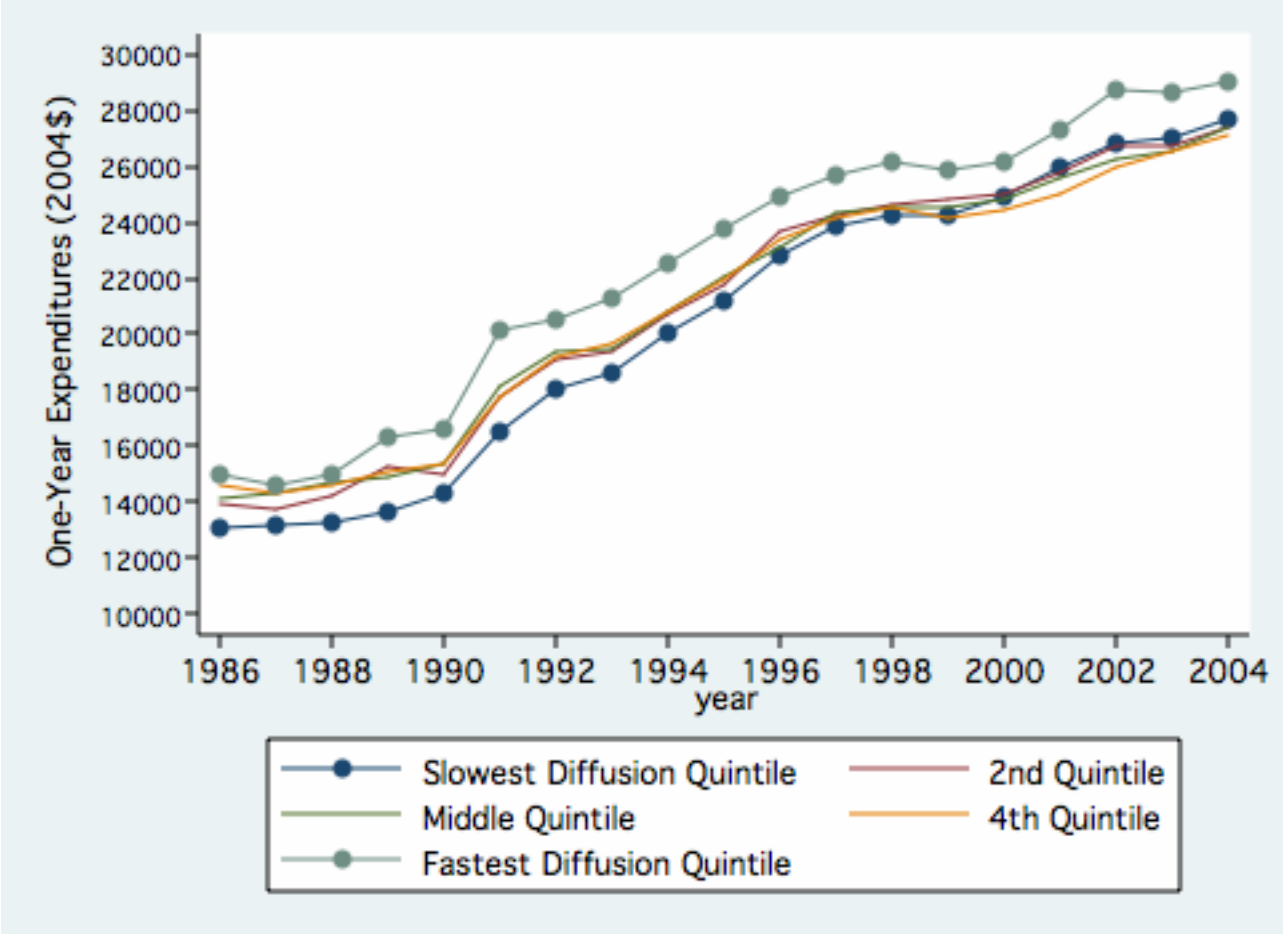


Figure 3b: Normalized Medicare Reimbursements (DRG Weights Multiplied by Common Reimbursement Rate)

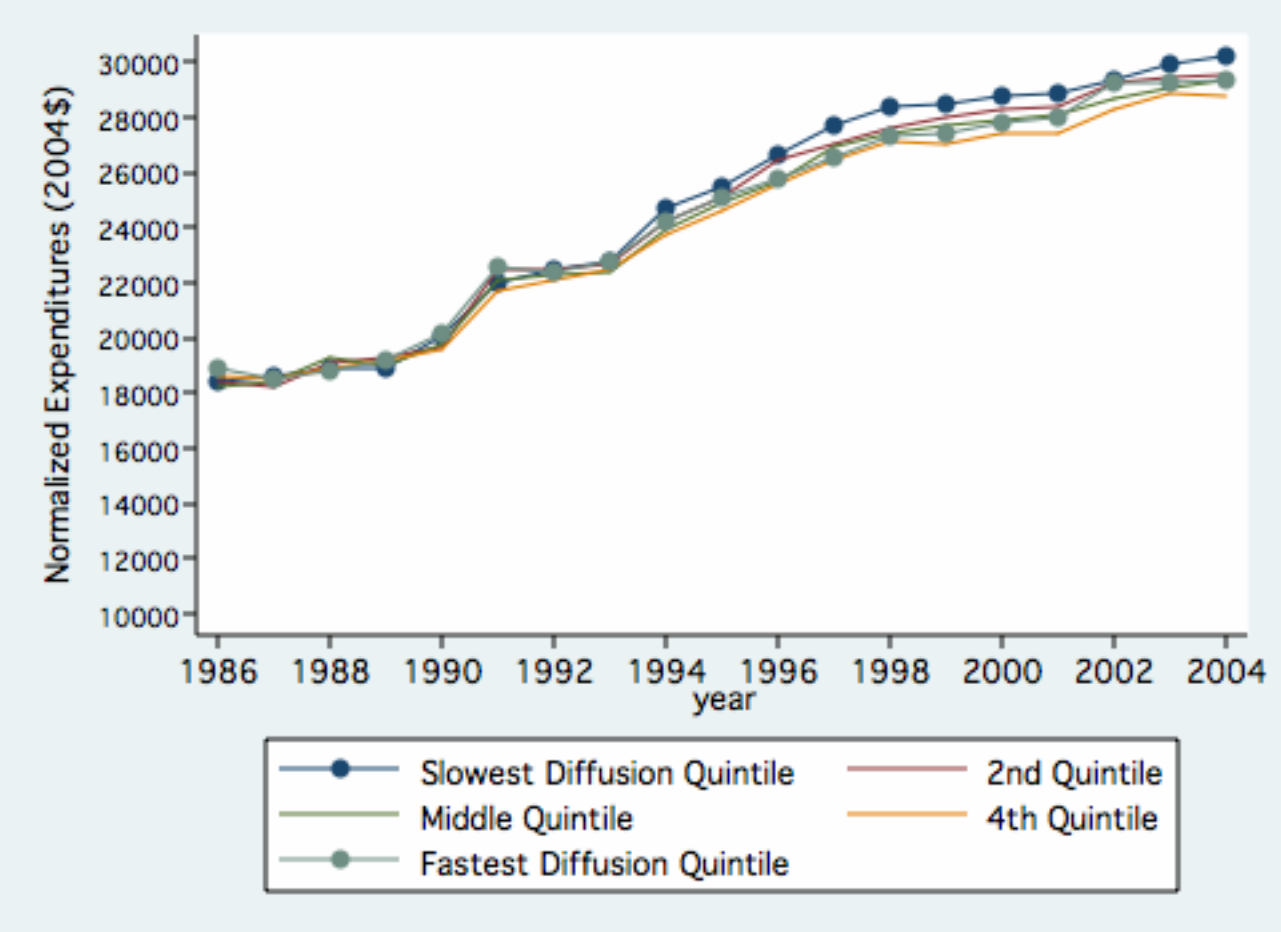


Figure 4: Interpreting the Evidence on Survival and Health Outcomes: "Flat of the Curve" vs. Productivity Differentials

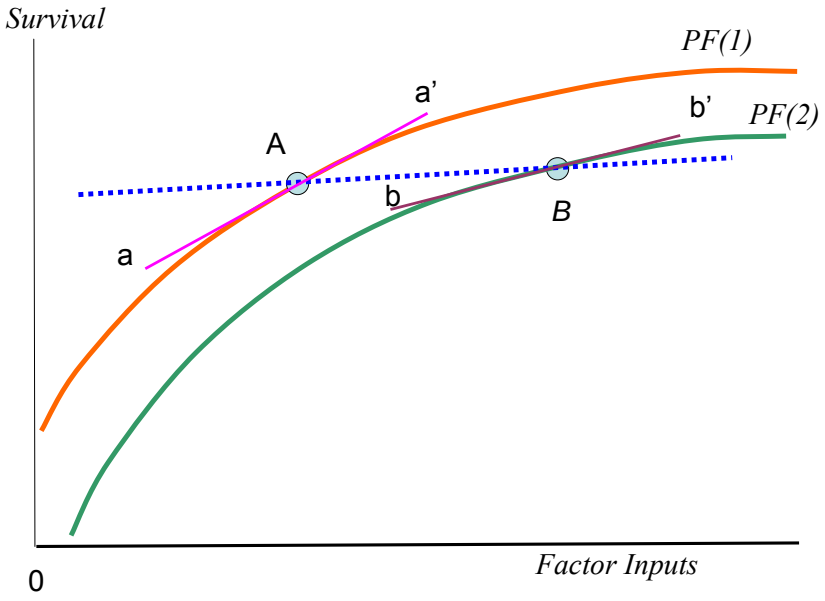
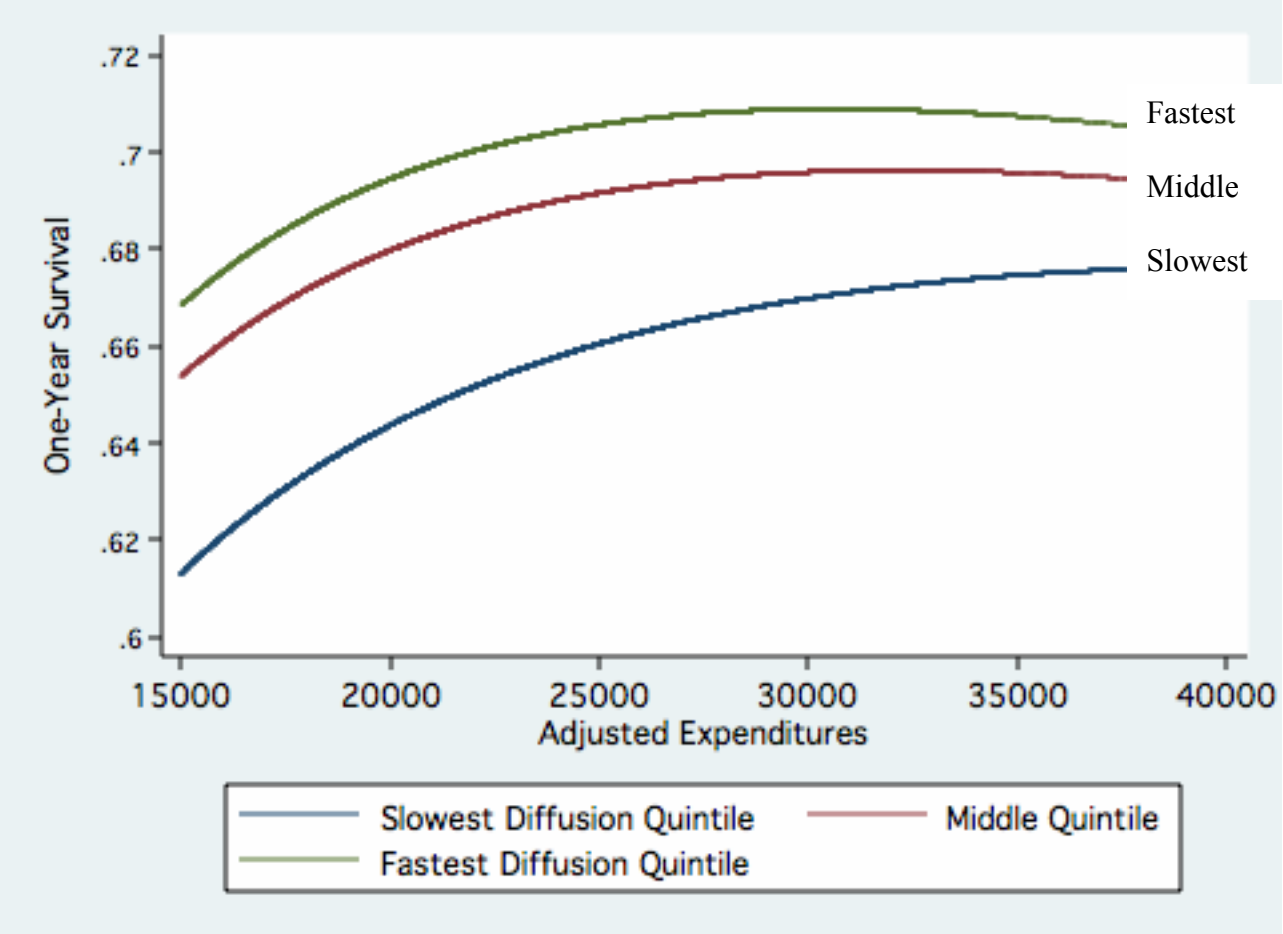


Figure 5: Estimated Production Functions for Quintiles 1 (Fastest) 3 (Middle) and 5 (Slowest) Hospitals, 2004



Note: Estimates based on regression analysis reported in Table 4.

Appendix: The Derivation of the Dynamic Model

We rewrite the objective function (7) for hospital i at time t as Equation A.1:

$$V_i = \sum_{t=0}^{\infty} \left[\Psi_i(a_{it} + \beta x_{it}) - \varphi_i P_{it} X_{it} - C_i(\pi_{it}) + K_{it}\right](1+r)^{-t} \quad \text{(A.1)}$$

The basic model – in which saving lives is good, and spending more is bad – is consistent with a range of models trying to capture what it is that hospitals maximize. For example, consider a model in which there exists a tension between two goals of the hospital: to maximize social welfare – the value of lives saved less resource costs – and the desire to maintain financial stability by maximizing profits. Suppose that the relative weight between the two objectives is given by μ_i, which is hospital-specific and ranges between one (the hospital maximizes social welfare without regard to its own financial position) and zero (the hospital cares solely about maximizing profit). Thus the objective function is

$$V_i = \sum_{t=0}^{\infty} \left[\mu_i[\Psi^*(a_{it} + \beta x_{it}) - P_{it} X_{it} + K_{it} - C_i(\pi_{it})] + (1-\mu_i)[\omega_i P_{it} X_{it} + K_{it} - C_i(\pi_{it})]\right](1+r)^{-t} \quad \text{(A.2)}$$

in which Ψ^* is the true social value of survival, ω_i the marginal proportional contribution to profitability of an incremental X_{it}, and K_{it} , the fixed subsidization or fixed costs, and P_{it} the factor cost, are defined as above. It is straightforward to show that (A.1) is a "reduced-form" version of (A.2), where $\Psi_i = \mu_i \Psi^*$ and $\varphi_i = \mu_i - \omega_i(1-\mu_i)$. Note that $\varphi_i > 0$ for the solution to exist (since the marginal productivity of X_{it} in this log-linear production function never turns negative), so the temptation to provide a highly profitable procedure must be tempered by at least some desire to curb allocative inefficiency.

The discrete discrete-time Lagrangian based on A.1 is written

$$\Im = V_i - \sum_{t=0}^{\infty} \lambda_{it}[a_{it+1} - a_{it} - \pi_{it}(a^*_{t+1} - a_{it})] \quad \text{(A.3)}$$

This model can also be written in continuous time as a current-value Hamiltonian, but we maintain a discrete time structure to help specify the empirical model. The first-order conditions are written:

$$\frac{\partial \Im}{\partial X_{it}} = \left[\frac{\Psi_i \beta}{X_{it}} - \varphi_i P_{it}\right](1+r)^{-t} \quad \text{(A.4a)}$$

$$\frac{\partial \Im}{\partial \pi_{it}} = -(1+r)^{-t} C_{it}' + \lambda_{it}(a^*_{t+1} - a_{it}) \quad \text{(A.4b)}$$

$$\frac{\partial \Im}{\partial a_{it}} = \Psi_i(1+r)^{-t} + \lambda_{it}(1-\pi_{it}) - \lambda_{it-1} \quad \text{(A.4c)}$$

(A.4d) $$\frac{\partial \Im}{\partial \lambda_{it}} = a_{it+1} - a_{it} - \pi_{it}(a^*_{t+1} - a_{it})$$

In addition to these four first-order conditions, we also add a solvency constraint:

(A.4e) $$\sum_{t=0}^{\infty} \left[\varphi_i P_{it} X_{it} + \mathrm{K}_{it} - C_i(\pi_{it})\right](1+r)^{-t} \geq 0$$

to ensure the present value of profits is non-negative (although hospitals can lose money in a given year). While hospitals do go out of business, we seek to avoid these more complicated issues by focusing solely on hospitals that remain in the panel during the period of analysis.

We first characterize the equilibrium, and demonstrate that a steady-state solution exists: $\pi_{it} = \pi_i$. To show this, we first solve for λ_{it}. Dropping the i subscript and assuming a constant π, (A.3c) is written

(A.5) $$\lambda_0 = \lambda_1(1-\pi) + \mu\Psi(1+r)^{-1}$$

$$\lambda_1 = \lambda_2(1-\pi) + \mu\Psi(1+r)^{-2}$$

....

and by progressive substitution:

(A.6) $$\lambda_0 = \lambda_k(1-\pi)^k + \mu\Psi(1+r)^{-1}\left[1 + \frac{1-\pi}{1+r} + \left(\frac{1-\pi}{1+r}\right)^2 + ...\left(\frac{1-\pi}{1+r}\right)^{k-1}\right]$$

Solving for the infinite series (which converges given that π and $r > 0$) and assuming that the transversality condition is met, so that $\lambda_k(1-\pi)^k$ converges to zero as k gets large, $\lambda_0 = \Psi/(\pi + r)$. From A.5,

$$\frac{\Psi}{\pi + r} = \lambda_1(1-\pi) + \mu\Psi(1+r)^{-1}$$

By successive substitution, $\lambda_t = \lambda_{t-1}/(1+r)$. This in turn implies a steady-state solution for $X_{it} = X_i$ (since both λ and the objective function decay at the rate r).

By rearranging A.4b and substituting for λ_t, one can derive Equation (11) in the text. By further substituting $(1-\pi)/\pi$ for $(a_t^* - a_t)$, which comes from the steady-state expression in Equation (10), we can also write the solution for π as:

(A.7) $$C'(\pi) = \Psi_i \frac{\alpha(1-\pi)}{\pi}\left[\frac{1}{r+\pi}\right]$$

Simulations of this key first-order condition suggests a high degree of dynamic stability.

Appendix Table A.1: Basic Risk Adjustment Model

		One-Year Survival	One-Year Expenditures
	Mean	Coefficient	Coefficient
Vascular Disease	0.070	-0.028 (0.001)	1658 (55)
Pulmonary Conditions	0.187	-0.081 (0.001)	1368 (37)
Dementia	0.026	-0.135 (0.002)	-4919 (84)
Diabetes	0.246	-0.039 (0.001)	1948 (33)
Liver Disease	0.003	-0.240 (0.005)	-2019 (269)
Renal Disease	0.024	-0.278 (0.002)	1256 (94)
Cancer	0.042	-0.164 (0.001)	-2716 (70)
Location of MI			
Anterolateral	0.042	-0.003 (0.002)	685 (122)
Anterior Wall	0.165	0.033 (0.002)	1117 (107)
Inferolateral	0.029	0.062 (0.002)	331 (130)
Inferior Posterior	0.021	0.073 (0.002)	1033 (140)
Inferior Wall	0.178	0.102 (0.002)	333 (106)
Lateral (NEC)	0.021	0.066 (0.003)	-114 (139)
True Posterior	0.007	0.073 (0.004)	825 (194)
Sub-Endocardial	0.414	0.119 (0.002)	2092 (103)
AMI (NEC)	0.022	-----	-----
AMI (NOS)	0.100	-0.093 (0.002)	-1783 (111)
Constant (for non-black male age 65-69		0.733 (0.004)	24308 (166)
Age-Sex-Race-Year Categorical Variables		Yes	Yes
Sample Size	3,185,837	2,808,171	2,808,170
Note. Standard error of estimate in parentheses.			