Moral Hazard and Less Invasive Medical Treatment for Coronary Artery Disease: An Analysis of Smoking in the National Health Interview Survey

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September 6, 2013

Abstract

We use Medicare claims data linked to the National Health Interview Survey (NHIS) to study how changes in patient smoking behavior are related to three common treatments for Coronary Artery Disease (CAD): medical management, Percutaneous Coronary Intervention (PCI), and Coronary Artery Bypass Graft (CABG). We find that the more invasive a patient's treatment, the more likely he is to quit smoking. Patients undergoing CABG, the most invasive procedure, are 13 and 16 percentage points more likely to quit smoking than patients treated with PCI or medical management, respectively, in the one-year window surrounding their procedure. This and other behavioral responses may partially offset the risks inherent to a more invasive procedure and help explain why the longer term outcomes for CABG patients rival or even exceed those of similar patients receiving PCI or medical management.

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Acknowledgement: Research for this paper was supported by grant number 5R21 AG033876 from the National Institute on Aging and the Office of Behavioral and Social Sciences Research to the National Bureau of Economic Research. This paper was presented at seminars at the University of Chicago, the University of Illinois at Chicago, Johns Hopkins University, and the University of Melbourne. We would like to thank the participants in those seminars for helpful comments and suggestions. We also would like to thank Peter Cram for his medical insight and Sandra Decker for her advice on working with the linked NHIS/Medicare data. We are indebted to Jonathan Fisher, Research Data Center (RDC) Administrator at the New York Bureau of the Census RDC at Baruch College, and Frances McCarty, Senior Service Fellow at the National Center for Health Statistics RDC, for their assistance in helping us to gain access to and work with restricted files from the National Health Interview Survey. All errors are our own.

I. Introduction

Coronary Artery Disease (CAD) is a common and deadly disease. In 2010, over 350,000 people died of CAD in the United States, making the disease responsible for roughly one in seven deaths (Murphy, Xu, and Kochanek, 2012). CAD is caused by a buildup of plaque on the arterial walls leading to the heart, resulting in reduced blood flow. If the buildup is not checked, CAD can result in a myocardial infarction (MI) (aka "heart attack") due to insufficient oxygen reaching the heart.

A number of medical treatments are available to patients with CAD. First, and least invasive, is "medical management." Medical management involves non-surgical treatment including prescription medication, lifestyle modification, and frequent monitoring. The second treatment is a revascularization procedure known as Percutaneous Coronary Intervention (PCI). A doctor (usually a cardiologist) performing PCI makes a small incision and arthroscopically inserts and inflates a balloon at the site of the lesion to expand the vessel. PCI in the modern era usually involves the placement of a wire mesh stent at the blockage site, which assists in keeping the arterial walls expanded to maintain blood flow. The PCI procedure takes approximately 60 minutes and the patient usually spends one night in the hospital.[1] The third and generally most invasive treatment is Coronary Artery Bypass Graft (CABG)[2], a major surgical procedure that involves harvesting a section of vessel from a different area of the body (either vessels in the groin or chest wall), opening the chest cavity via a sternectomy, and connecting one healthy part of the diseased artery to another, surgically bypassing the lesion. CABG surgery takes approximately four hours and patients generally spend at least a week recovering in the hospital.[3]

[1] http://www.medicinenet.com/coronary_artery_bypass_graft/article.htm (accessed 5/31/12)
[2] Less invasive CABG procedures have been in development and increasing use in recent years, though these were very infrequent during the period we examine.
[3] http://www.medicinenet.com/coronary_angioplasty/article.htm (accessed 5/31/12)

Of the two procedures, PCI is the more recent, having been initially used in the late 1970s, over than a decade after CABG was first performed. Its use expanded rapidly upon FDA approval of the coronary stent in 1994 (Cutler and Huckman, 2003). Since the development of PCI, there have been numerous studies comparing the effectiveness of the two procedures in various populations (see Rodriguez et al. 2001 and Serruys et al. 2009 for two recent articles with a summary of prior research). While the results vary, our general interpretation is that PCI patients have lower perioperative mortality – due partly to fewer surgical complications from a less invasive procedure – but that CABG patients have similar or better long-term outcomes.

In this paper, we test one hypothesis that may explain why CABG patients have relatively good long-term outcomes, despite a higher surgical complication rate. Specifically, we expect that the more invasive nature of CABG – a patient's heart and lungs are bypassed during the surgery, he is in the hospital for a week, has a longer post-operative recovery period, and is left with a major scar and residual pain from the sternectomy – sends a stronger signal to the patient that he has a serious health problem. As a result, we hypothesize that a patient who undergoes CABG rather than PCI is more likely to change his behavior in a way that promotes good health and a longer life: he is more likely to quit smoking, begin exercising, improve his diet, and avoid excessive alcohol intake.[4]

This hypothesis is consistent with a prior economic research on moral hazard, showing that individuals change their behavior when their perceived risks change. Peltzman's (1975) study of the effects of automobile safety regulation is a classic and seminal example. He

[4] CABG patients might also quit smoking at a higher rate because they physically cannot smoke for the week they are in the hospital. Using our current data, we cannot distinguish between these two possible causes of smoking cessation: the health signal sent by the CABG surgery and the impact of "going cold turkey" for a week while in the hospital, though we find the former explanation to be more plausible. In future work, we plan to look at the impact of CABG surgery on the smoking behavior of spouses, which should not be influenced by the week at CABG patient spends in the hospital.

develops a model in which the legal mandate to install various safety devices on automobiles lowers the price of fast and reckless driving because it lowers the probability that the driver will die in an accident. Hence the demand for this activity rises. Empirically, he finds that the increase in this offsetting behavior (reckless driving) is so large that the regulations at issue had very little impact on highway deaths and actually increased pedestrian deaths. More recently, Dave and Kaestner (2009) investigate the impact of health insurance access on the health behaviors of the elderly, showing that access to Medicare at age 65 leads to a reduction in preventative behaviors and an increase in risky health behavior amongst the elderly. Peltzman (2011) demonstrates how medical technology breakthroughs can lead to offsetting behavior by showing that the age cohorts that benefited the most from the introduction of antibiotics experienced worse mortality rates from risky health behaviors.

In this study, we test one potential behavioral response to surgery – smoking – and see results consistent with patient offsetting behavior. Patients who undergo a more invasive treatment for CAD are more likely to quit smoking (or conversely, patients who undergo a *less* invasive procedure are *less* likely to quit smoking). Compared to CAD patients who are medically managed, patients who have PCI or CABG are three and 16 percentage points more likely to quit smoking, respectively, in the one-year window surrounding their surgery. Our results are robust to a number of different specifications, including a simple grouped-by-year regression using 11 observations, done in the spirit of Donald & Lang (2007).

II. Data

In this study, we use individual Medicare data merged with responses from the National Health Interview Survey (NHIS). The Medicare records identify those patients who have been

diagnosed with CAD and show which of them have undergone PCI or CABG, along with the exact date of each diagnoses and procedure. The Medicare data also allow us to control for disease severity and other conditions that might be correlated with procedure type and induce quitting, such as a myocardial infarction. The NHIS provides information on smoking and quitting behavior, as well as individual characteristics.

The Medicare data are provided by the Center for Medicare & Medicaid Services (CMS). To identify CAD patients and the type of treatment they underwent, we use the Medicare Standard Analytical Files, including the Inpatient, Outpatient, Skilled Nursing Facility, Carrier, Durable Medical Equipment, Home Health Agency, and Hospice claims files. These files contain one or more records for each individual.[5] Each record contains the ICD-9-CM codes for all diagnoses made and procedures performed during that stay or claim. We identify CAD patients as those who have at least one diagnosis code beginning with 410, 411, 412, 413, or 414. We identify PCI patients as those CAD patients with at least one procedure code beginning with 0066, 3601, 3602, 3605, or 3606. We identify CABG patients as those CAD patients with procedure codes beginning with 361.[6] Finally, we identify medically managed patients as those patients who have been diagnosed with CAD, but do not have a concurrent or subsequent PCI or CABG procedure.[7]

The NHIS is an annual survey of approximately 85,000 individuals in over 30,000 U.S. households run by the National Center for Health Statistics (NCHS), part of the Centers for

[5] A single record in the Inpatient file corresponds to a stay in a hospital. A single record in the Skilled Nursing Facility file corresponds to a stay in a Skilled Nursing Facility. A single record in the Outpatient file corresponds to a claim by an institutional outpatient provider (Hospital outpatient clinic, rural health clinics, etc.). A single record in the Carrier claim file corresponds to a claim by a non-institutional outpatient provider (physicians, physician assistants, etc.)

[6] For both PCI and CABG, we exclude the small number of patients who do not have a concurrent or prior CAD diagnosis.

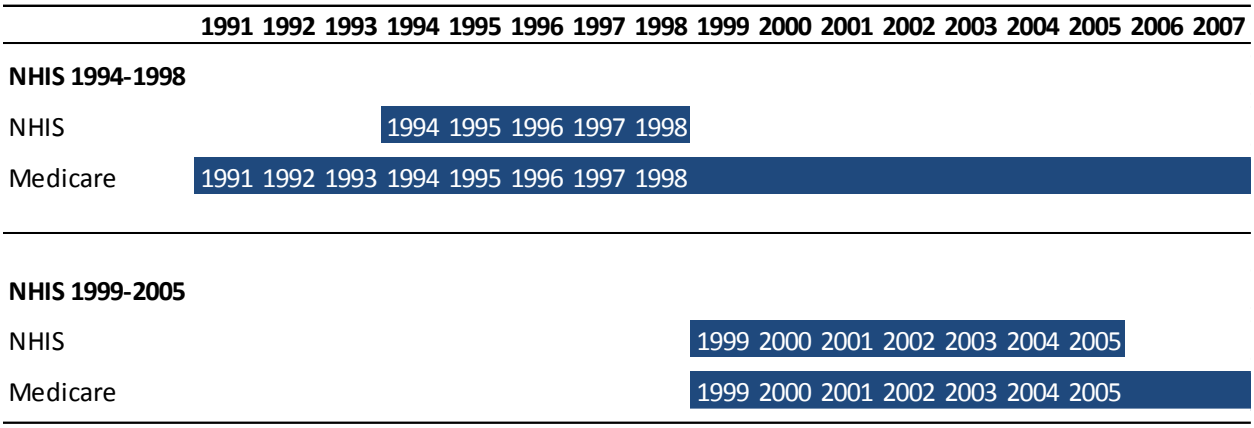
[7] A patient who is diagnosed with CAD before her NHIS interview date and has PCI or CABG after her NHIS interview date is counted as medically managed at the time of the NHIS survey.

Disease Control and Prevention (CDC). All participants are asked questions about their general state of health and disability. Each year, a subset of approximately 30,000 individuals is asked about their smoking habits. These respondents are asked if they have ever smoked 100 cigarettes in their life. For those who say yes, they are asked if they currently smoke every day, some days, or not at all. If they do not currently smoke, they are asked when they quit, a question they can answer in days, weeks, months, or years. We use the responses to these questions to create a synthetic panel, identifying whether a person smoked on each date prior to their NHIS interview. Each person is categorized as either an always smoker, a never smoker, or a quitter who smoked up to the day she reports quitting.[8]

The individual NHIS responses have been linked to Medicare data by the CDC and CMS and made available as a restricted-use dataset to researchers. The linkage is based on social security number, date of birth, and gender. To be linked, the data must match on all three fields. To date, the CDC and CMS have linked the 1994-1998 NHIS surveys to Medicare data from 1991-2007 and the 1999-2005 NHIS surveys to Medicare data from 1999-2007. The linkage is illustrated in Figure 1.

[8] This categorization vastly over-simplifies the complexity of smoking and quitting behavior, but still allows us to investigate our key question: what is the *difference* in quitting behavior between CAD patients undergoing medical management, PCI, or CABG.

Figure 1 – NHIS/Medicare Data Link



Note: the Medicare years labeled on the chart are potentially useful for our study because they represent a Medicare record that is linked to a later NHIS interviews. Medicare records linked to earlier NHIS interviews provide no information on quitting behavior after CAD treatment.

For those respondents who were diagnosed with CAD prior to their NHIS interview date, we have the ability to look at their smoking behavior before and after their diagnosis. For the subset of CAD patients who underwent PCI or CABG, we can also look at their smoking behavior before and after their procedure. For example, for individuals interviewed in 1994 who had PCI, we can look at their smoking behavior before and after their procedure *only if they underwent PCI between 1991 and 1994* (and within 1994, only if their procedure was before the date of the NHIS interview). If a person had PCI before 1991, then we have no record of their procedure. If a person had PCI after 1994, then we have no record of their smoking behavior *after* their procedure.

Each person in the linked dataset, therefore, has a "diagnosis window" within which they must be diagnosed with CAD to be included in our study. The longest window is for a person who was interviewed in 1998 – he will be included in our study if he was enrolled in Medicare and diagnosed with CAD at any point between 1991 and 1998. The shortest window is for a person who was interviewed in early 1999 – he will be included only if he was enrolled in Medicare and diagnosed with CAD on an earlier date in 1999 than the date of his interview.

III. Initial Analysis

In total, 12,265 NHIS respondents were linked to Medicare data and diagnosed with CAD during their diagnosis window. Of these individuals, between the date of their diagnosis and the date of their NHIS interview, 10,772 patients were treated only with medical management, 723 patients underwent PCI but not CABG surgery, and 770 patients underwent CABG surgery. Ninety-nine (99) patients underwent both PCI and CABG surgery. These patients are included in the CABG category, because that is the more invasive treatment. Our results are robust to including them in the PCI category or excluding them altogether.

Basic characteristics of these patients are shown in Table 1. Overall, when compared to patients undergoing medical management, patients who undergo a procedure (PCI or CABG) are more likely to be younger, male, and white. PCI and CABG patients appear to have largely similar demographic characteristics, though CABG patients are somewhat more likely to be male. When comparing medical conditions, both PCI and CABG patients are substantially more likely than medically managed patients to have had their first Acute Myocardial Infarction (AMI, a.k.a. "heart attack") within six months of initiating treatment. A number of other comorbidities – including congestive heart failure and valvular disease – show up most frequently in CABG patients, followed by PCI patients. In some of our regression specifications, we control for the covariates shown in Table 1.

Table 1 – Characteristics by Treatment[9]

Demographic Characteristics	MM	PCI	CABG
Age			
< 55	5%	4%	2%
55-64	8%	8%	7%
65-69	25%	25%	28%
70-74	22%	25%	27%
75-79	20%	20%	23%
80-84	13%	12%	12%
85+	8%	5%	2%
Gender			
Male	42%	52%	59%
Female	58%	48%	41%
Race			
Asian	1%	1%	1%
Black	11%	8%	6%
Hispanic	8%	6%	7%
White	78%	85%	86%
Mult./Oth/Unknown	1%	1%	1%
Education			
Elem (K-8)	23%	18%	22%
HS (non-grad); GED	19%	23%	18%
HS grad	28%	29%	28%
Some col; AA deg.	17%	19%	18%
BA degree	7%	6%	8%
Grad. Degree	5%	5%	5%
Unknown	1%	0%	1%
Family Income			
$0 to $9,999	21%	16%	16%
$10,000 to $19,999	25%	23%	25%
$20,000 to $35,000	19%	23%	23%
$35,000 or over	17%	21%	20%
Unknown	18%	17%	16%
Count	10,772	723	770

Medical Conditions	MM	PCI	CABG
First AMI Within 6 Months of Treatment			
Yes	8%	42%	37%
No	92%	58%	63%
% With Commorbidity Within 6 Months of Treatment			
Congestive heart failure	14%	21%	34%
Valvular disease	11%	18%	26%
Pulmonary circulation disorder	2%	4%	4%
Peripheral vascular disorder	10%	18%	22%
Paralysis	1%	1%	3%
Other neurological	3%	3%	3%
Chronic pulmonary disease	15%	13%	21%
Diabetes w/o chronic comp.	14%	16%	17%
Diabetes w/ chronic comp.	5%	7%	11%
Hypothyroidism	7%	7%	5%
Renal failure	2%	4%	4%
Liver disease	1%	1%	0%
Chronic Peptic ulcer disease	0%	1%	1%
HIV and AIDS	0%	0%	0%
Lymphoma	0%	0%	1%
Metastatic cancer	1%	1%	1%
Solid tumor without metastasis	5%	5%	3%
Rheumatoid arthritis	3%	2%	2%
Coagulation deficiency	3%	4%	9%
Obesity	3%	8%	9%
Weight loss	1%	1%	2%
Fluid and electrolyte disorders	10%	13%	24%
Blood loss anemia	1%	3%	3%
Deficiency anemias	11%	15%	22%
Alcohol abuse	1%	1%	1%
Drug abuse	0%	0%	0%
Psychoses	2%	1%	2%
Depression	4%	7%	6%
Hypertension	37%	37%	36%
Count	10,772	723	770

Note: All data is unweighted. Age is as of diagnosis (CAD) or procedure (PCI / CABG). Comorbidities based on Elixhauser et al. (1998)

Table 2 shows the smoking status of each group of respondents – medical management, PCI, and CABG – as of the date of the NHIS interview. In looking at this table, two items merit

[9] Results presented in this paper include all Medicare participants, regardless of age. Results excluding those under 65, available upon request, are similar.

notice. First, CABG patients are more likely to have ever smoked than PCI patients, who were in turn more likely to have smoked than medically managed patients (i.e. the percentage of respondents who never smoked gets lower as one moves from left to right in the table). Second, most people who have ever smoked have quit smoking by the time of the NHIS interview, a trend that is most pronounced for CABG patients. While 61.2% of CABG patients in our study smoked at some point in their life, only 9.1% smoke as of the NHIS interview date. PCI patients have a lower proportion of quitters, followed by medically managed patients.

Table 2 – Smoking Status as of NHIS Interview Date

	Count			Percent		
Smoking Status as of Survey	Medical Management	PCI	CABG	Medical Management	PCI	CABG
Current	1,325	88	70	12.3%	12.2%	9.1%
Quit	4,477	342	401	41.6%	47.3%	52.1%
Never Smoked	4,970	293	299	46.1%	40.5%	38.8%
Total	10,772	723	770	100%	100%	100%

Note: This table shows the smoking status of every NHIS respondent who was diagnosed with CAD prior to their interview date. Data is unweighted.

The data in Table 2 are consistent with the broad hypothesis in our study – patients who undergo a more invasive treatment for CAD are more likely quit smoking. However, they could also be consistent with a story in which people who undergo CABG surgery are also more likely to quit smoking for reasons unrelated to surgery. If our hypothesis is true, we should see that the differential quitting behavior between CABG, PCI, and medical management is driven by quits that occur close to the date of the treatment.

Table 3 – Counts of Smokers Before and After Treatment

	Count			Percent		
	Medical Management	PCI	CABG	Medical Management	PCI	CABG
Smoke before	1,713	121	115	15.9%	16.7%	14.9%
Don't smoke before	9,059	602	655	84.1%	83.3%	85.1%
Total	10,772	723	770	100.0%	100.0%	100.0%
Smoke after	1,539	100	81	14.3%	13.8%	10.5%
Don't smoke after	9,233	623	689	85.7%	86.2%	89.5%
Total	10,772	723	770	100.0%	100.0%	100.0%
Change in smokers	-174	-21	-34	-1.6%	-2.9%	-4.4%
Percent Quit	10.2%	17.4%	29.6%			

Note: This table includes every NHIS respondent who was diagnosed with CAD in our data prior to their interview date. It shows their smoking status exactly six months before and exactly six months after diagnosis (CAD) or surgery (PCI/CABG). Data is unweighted.

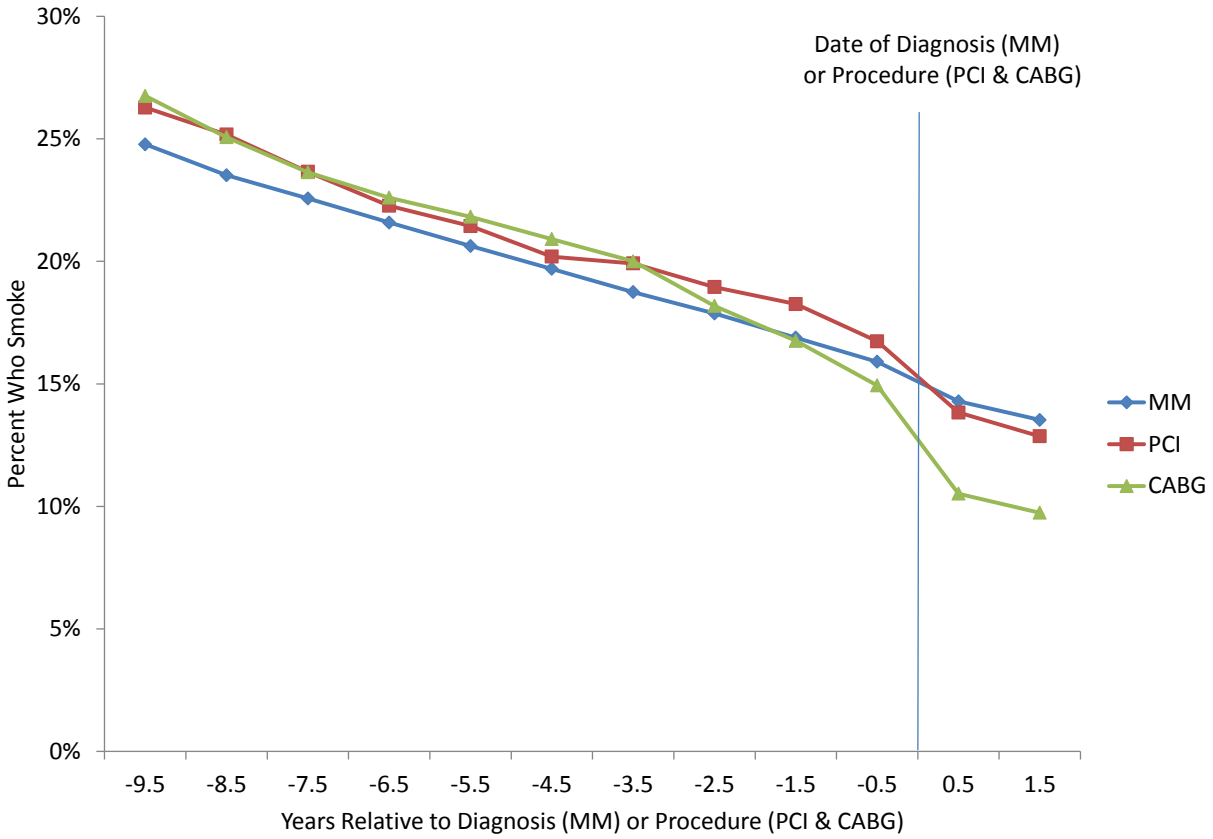
Table 3 focuses on those quits that take place immediately around the initiation of treatment, where the initiation of treatment is defined to be the diagnosis date for medically managed patients and the procedure date for PCI and CABG patients. The "before" period is exactly six months before the diagnosis/procedure date, while the "after" period is exactly six months after diagnosis/procedure date.[10] Among the 10,772 patients diagnosed with CAD who receive only medical management, 1,713 smoked six months before their diagnosis and 1,539 smoked six months after their diagnosis. The 174 who quit smoking represent a 1.6 *percentage point* reduction in the number of smokers and a 10.2 *percent* reduction. The corresponding numbers for PCI are a 2.9 percentage point reduction and a 17.4 percent reduction. For CABG, they are a 4.4 percentage point reduction and a 29.6 percent reduction.

[10] Creating a "quit window" around the date of diagnosis/procedure is necessary for two reasons. First, it is unlikely that many individuals quit on exactly the day their treatment began. Second, our smoking data, which are based on individuals' recollections, are insufficiently precise to pinpoint the exact day of quitting. Our conclusions do not change with other reasonable definitions of the quit window.

Further evidence is provided by Figures 2 and 3. In Figure 2, we calculate the percentage of the population smoking each year at twelve points in time, measured in years relative to the date of diagnosis (in the case of medically managed patients) or procedure (in the case of PCI and CABG patients)[11]. In the CABG series, for example, the year -3.5 shows the percentage of CABG patients who were smoking exactly three and a half years prior to their procedure date. In the 10 years prior to diagnosis/procedure, the three series track each other reasonably closely. At the first point on the graph, 9.5 years before diagnosis/procedure, roughly the same share of eventual CABG and PCI patients are smoking, and this share is approximately 1.5 percentage points higher than the share of eventual medical management patients smoking. The CABG and PCI series track each other closely until 3.5 years prior to the procedure date, at which point the CABG series declines at a slightly faster rate. The differences between the three series emerge most starkly in the period immediately after diagnosis/procedure, when the PCI series drops below the medical management series for the first time, and the percentage of CABG recipients smoking falls at a substantially faster rate. Six months after surgery, the percentage of CABG recipients smoking is more than three percentage points lower than the corresponding percentage for either medically managed or PCI patients.

[11] Because we have data on only the most recent quit date for each individual, we assume that each smoker was smoking in all years before their quit date. Since we are using Medicare data for our analysis, most people are over 65 when they received their diagnosis or procedure, and it is unlikely that they started smoking for the first time in the ten years immediately prior. It is possible that individuals quit and restarted during this time period, and we do not distinguish them from continuous smokers.

Figure 2 – Smoking Rate by Year Relative to Diagnosis (MM) or Procedure (PCI & CABG)

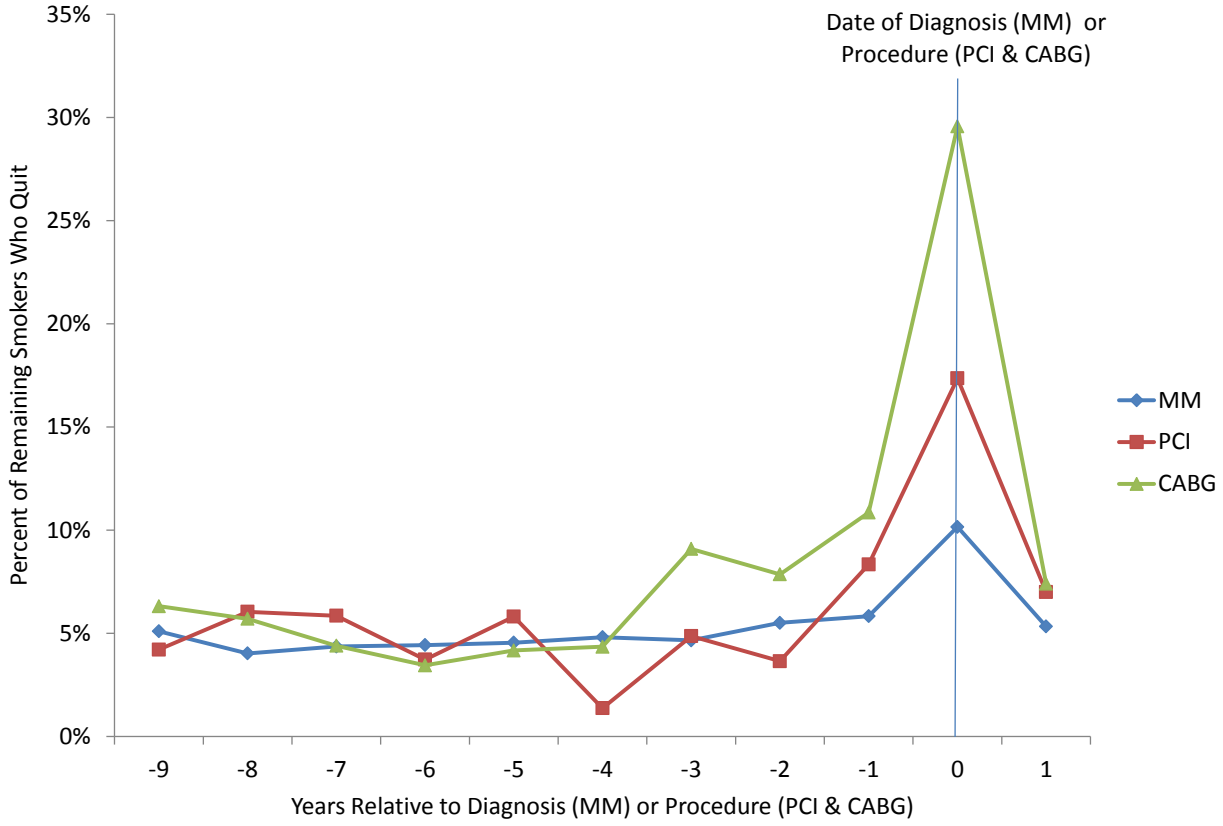


Note: the figure shows the percentage of respondents who smoked at each point relative to their diagnosis/procedure. Data is unweighted.

Figure 3 displays the same data in a different format, showing the annual quit rate for patients in each of the three treatment groups relative to the date of diagnosis/procedure. For the group that receives only medical management, roughly 5% of smokers quit each year in the nine years prior to being diagnosed, a rate that doubled to 10% during the year of their diagnosis with coronary artery disease. The PCI and CABG series show a similar trend, though they represent fewer individuals and are somewhat noisier. In the years prior to their procedure, roughly 5% of smokers quit each year, though this percentage began to rise between one and three years before the procedure date. During the procedure year – defined to be the six month window on either side of the procedure date – the quit rate jumped to 18% for patients receiving PCI and 30% for

patients receiving CABG. In the year following diagnosis/procedure, the quit rate for all three groups dropped back to approximately 5%. Figures 2 and 3 provide reasonably compelling evidence that at least a portion of the increased quit rate for more invasive treatments observed in Table 2 is related to treatment received, and not simply a spurious correlation.

Figure 3 – Quit Rate by Year Relative to Diagnosis (MM) or Procedure (PCI & CABG)



Note: the figure shows the percentage of remaining smokers who quit at each point relative to their diagnosis/procedure. Data is unweighted.

IV. Results

To further explore the relationship between treatment for coronary artery disease and smoking behavior, we fit three related models of quitting smoking. The first is a single-period quit model using individual data. Conditional on a person smoking exactly six months prior to

treatment, we predict her likelihood of quitting based on the treatment received – medical management, PCI, or CABG – and other control variables.[12] The second is a multi-period quit model using individual data. The regression that we estimate takes the form of a discrete time linear probability hazard function with 11 periods: 9 before treatment and 2 after. It allows us to control for events that occur before treatment. For example, some CABG and PCI patients are diagnosed with CAD before they undergo treatment, and conditions reflected by the Elixhauser indicators can emerge before treatment. The third model is a multi-period quit function using grouped data, inspired by Donald and Lang (2007). In this model, we build a synthetic panel, grouping the individual data by period and treatment type into 33 cells (3 treatment types and 11 periods), and run difference-in-differences regressions with 11 data points.

Before the results of these models are presented, it is useful to point out the relationship between a smoking participation function and a quit function. As an identity[13]

$$\frac{s_t}{s_{t-1}} \equiv 1 - q_t, \qquad (1)$$

where s_t and s_{t-1} are the smoking participation rates in periods t and t-1, respectively, and q_t is the quit rate in the window defined by periods t and t-1. All rates are defined as fractions and can be

[12] For this model, the "after" period is exactly six months after the diagnosis / procedure date.

[13] Let S_t be the number of smokers in period t, and let S_{t-1} be the number of smokers in period t-1. Let Q_t be the number of quitters in period t (the number who smoke in period t-1 but do not smoke in period t). Assume as is the case in our data that there are no starters or re-starters. Then

$$S_t \equiv S_{t-1} - Q_t.$$

Divide both sides of the identity by N, the size of the population:

$$\frac{S_t}{N} \equiv \frac{S_{t-1}}{N} - \frac{Q_t}{S_{t-1}} \frac{S_{t-1}}{N}.$$

Solve the last identity for $\frac{\frac{S_t}{N}}{\frac{S_{t-1}}{N}}$ to obtain equation (1).

interpreted as probabilities at the individual level. Take natural logarithms of the identity to obtain

$$\ln \frac{s_t}{s_{t-1}} \equiv \ln(1-q_t) \cong -q_t. \qquad (2)$$

Strictly speaking, the approximation in the last part of equation (2) holds for $q \leq 0.2$. But even for a quit rate as large as 0.3 (the largest rate in our data), ln (1 – q) = - 0.350, which is close to 0.3. Equation (2) indicates that a regression in which the first difference of the log of smoking participation is the dependent variable should have approximately the same coefficients with the signs reversed as one in which the quit rate is the dependent variable. It also suggests that it is useful to begin with a log smoking participation function to arrive at a specification of a quit function. In particular, if the log smoking participation function contains individual fixed effects, these effects are eliminated by taking first differences to obtain the quit function. We make use of that insight in more detail in developing multi-period quit models.

Model 1: Single Period Quit Model Using Individual Data

We first fit a single-period quit model using only those respondents who smoked before their diagnosis or surgery. We begin with two-period log smoking participation functions for each of the three types of patients (c = CABG, p = PCI, m = MM). The first period (t = 1) is the six month period before treatment in the case of CABG and PCI or diagnosis in the case of MM The second period (t = 2) is the six-month period after treatment or diagnosis. The model for the i^{th} individual in group g (g = c, p, m) is

$$\ln s_{igt} = f_{ig} - \beta_g a_{igt} - \phi h_{igt} - \gamma e_{igt} - \eta a_{igt} x_{ig}. \qquad (3)$$

Here f_{ig} is a person-specific fixed effect, a_{igt} is a dichotomous variable that equals 1 in the period after diagnosis/procedure and is equal to 0 before, h_{igt} is a dichotomous variable that equals 1 if

the patient had his or her first acute myocardial infarction (AMI) in period 2, e_{ig} is a vector of 29 dichotomous indicators for the Elixhauser comorbidity conditions diagnosed for the first time during period 2, and x_{ig} is a vector of demographic and socioeconomic characteristics. Since individual fixed effects are included in equation (3), the demographic and socioeconomic variables can be included only if the interact with period 2, which from now on is termed the after period.

Multiply the CABG participation equation by c, the PCI equation by p, and the MM equation by (1 – c – p). Then take first differences to obtain

$$- (\ln s_{i2} - \ln s_{i1}) \cong q_i = \beta_m + (\beta_p - \beta_m)p_i + (\beta_c - \beta_m)c_i + \phi h_i + \gamma e_i + \eta x_i. \quad (4)$$

We estimate equation (4) as a linear probability model so that q_i is an indicator equal to 1 if person i quit smoking during the quit window and 0 otherwise. To count each person once in the regression, we assign individuals who had both PCI and CABG surgery to the CABG category, as that is the more invasive treatment.[14]

The results appear in Table 4. The first column shows the simplest specification, with quitting predicted only based on the treatment dummies. According to this specification, a patient undergoing PCI is 7.3 percentage points more likely to quit than a patient diagnosed with CAD who is medically managed, a result that is significant at the 0.10 level. A patient undergoing CABG is 20.0 percentage points more likely to quit smoking than a medically managed patient, a result that is significant at the 0.01 level. The p-value testing the equality of these two coefficients is approximately 0.03.

[14] For people who had both PCI and CABG, we cannot simply give them a 1 for both the PCI and CABG indicators, since they may have had the surgeries on different dates, requiring a different quit indicator (q_i) for each surgery. Our conclusions are not sensitive to assigning these patients to the PCI group or excluding them entirely.

Table 4 – Single-Period Quit Model

	(1)	(2)	(3)	(4)
Constant	0.101***	0.092***	0.068***	0.124*
	(0.008)	(0.008)	(0.011)	(0.066)
PCI	0.073*	0.034	0.015	0.010
	(0.039)	(0.041)	(0.042)	(0.041)
CABG	0.200***	0.169***	0.135***	0.115**
	(0.046)	(0.047)	(0.046)	(0.045)
AMI		0.096***	0.095***	0.098***
		(0.029)	(0.030)	(0.030)
P-Value: PCI = CABG	0.030	0.022	0.041	0.071
Controls				
Elixhauser			X	X
Demographics				X
Observations	1949	1949	1949	1949

Note: Robust standard errors in parentheses, clustered by PSU. All specifications have 765 clusters. AMI is an indicator for a patient having his first AMI during the treatment window. Specifications 3 and 4 include 29 dummy variables for the Elixhauser commorbidity conditions first diagnosed during the patient's treatment period. In specification 4, demographic controls include gender, race, education dummies, and income category dummies (including a dummy for missing income data). Observations are weighted by the NHIS probability weights. "P-Value: PCI = CABG" is the p-value on a test of equality of the PCI and CABG coefficients. * significant at the 0.10 level; ** significant at the 0.05 level; *** significant at the 0.01 level.

In the second specification, we add an indicator for patients who experience their first AMI during the treatment window. We see that an AMI is a strong predictor of smoking behavior, correlated with a 9.6 percentage point increase in the probability of quitting. Moreover, the estimated coefficients on the PCI and CABG variables have each declined by between three and four percentage points. While the coefficient on PCI is positive, it is small and no longer statistically significant. The coefficient on CABG, however, remains large and statistically significant at the 0.01 level. In the third specification, we add 29 dummy variables corresponding to the Elixhauser comorbidity conditions, which control for secondary diseases first diagnosed during the quit window (Elixhauser et al., 1998). The PCI coefficient drops to

1.5 percentage points, while the CABG coefficient is estimated to be 13.5 percentage points. Finally, in the fourth specification, we see that the PCI coefficient drops to 1 percentage point and the CABG coefficient drops below 12 percentage points when we add demographic covariates, including gender, race, education, and income.

Compared with patients undergoing medical management, PCI patients are only slightly more likely to quit smoking, once we control for heart attacks, other co-occurring medical conditions, and demographic characteristics, and the difference is not statistically significant. CABG surgery, however, continues to be associated with a 12 percentage point increase in the probability of quitting. This increase is statistically significant at the 0.05 level when compared to medical management and the 0.10 level when compared to PCI.[15] Moreover, the association between quitting and CABG is larger in magnitude than the association between quitting and an AMI.

Model 2: Multi-Period Quit Model Using Individual Data

In our second set of regressions, we fit a multi-period quit model using individual data. This allows us to improve on our single period model in several ways. First, we can check whether information conveyed through a patient's diagnosis with CAD, rather than through the PCI or CABG procedure, induces a patient to quit smoking. Since 40% of PCI patients and 43% of CABG patients in our sample were diagnosed with CAD at least six months prior to their procedure, we can separately identify a "diagnosis effect" and a "treatment effect." Second, we can use the time series data to improve our estimate of the impact of an AMI on quitting. Since many patients have their first AMI outside of the treatment window, we can make use of these

[15] Since medical management and PCI have similar point estimates, the difference in statistical significance when they are compared to CABG is due primarily to the larger number of patients treated with medical management.

additional incidents to better estimate the impact of a heart attack on smoking. Third, we can follow the same procedure with regard to each of the Elixhauser comorbidities, since some of these conditions are first reported more than six months prior to treatment.

To implement the multi-period model, we develop a synthetic 12-period panel based on the time periods shown in Figure 2. For CABG and PCI patients, there are 10 periods prior to treatment (from 9.5 years before to 0.5 years before) and two periods after treatment (0.5 years after and 1.5 years after). For medically managed (MM) patients, there are the same 10 periods before diagnosis and the same two periods after diagnosis. To focus on the key aspects of the model, we ignore the socioeconomic and demographic variables for the time being, assume a single Elixhauser comorbidity, and suppress the subscript i for an individual. Let a_t be a dummy variable that equals 0 in each period before treatment for PCI or CABG and equals 1 in each period after treatment. Specifically, a_t equals 1 in periods 11 and 12. This variable is not relevant for MM patients (see below). Let d_t be a dummy variable that equals 0 in all periods before diagnosis and equals 1 in all periods after diagnosis. Let h_t be a dummy variable that equals zero before an AMI and equals 1 after an AMI. Finally, let e_t equal 0 in each period before an Elixhauser comorbidity is reported and equals 1 in each period thereafter.

The log smoking participation model for PCI and CABG patients (g = c or p)

$$\ln s_{gt} = f_g - \beta_g a_{gt} - \phi h_{gt} - \gamma e_{gt} - \alpha d_{gt} - \lambda t, \qquad (5)$$

where we assume a linear trend in the absence of treatment. The model for MM patients is the same except that a_{mt} coincides with d_{mt}, so that we constrain β_m to equal zero. After pooling and taking first differences, one obtains

$$-(\ln s_t - \ln s_{t-1}) \cong q_t = \lambda + \alpha(d_t - d_{t-1}) + \beta_p p(a_t - a_{t-1}) + \beta_c c(a_t - a_{t-1}) + \phi(h_t - h_{t-1}) + \gamma(e_t - e_{t-1}). \quad (6)$$

Strictly speaking, time-invariant individual characteristics, such as formal schooling, can only be added to equation (6) by assuming that they interact with the linear trend in equation (5). Our results are not affected by allowing the trend to be nonlinear or by allowing individual characteristics to interact with the indicator for the period after treatment in addition to their interactions with a linear trend.

We fit equation (6) as a discrete time linear probability hazard model. We include only individuals who smoke in the first period for which we compute smoking participation (9.5 years before treatment or diagnosis), dropping everyone who never smoked or quit prior to that period. Each person is assigned a q_{it} variable that is equal to one in the period in which they quit after they quit and zero in all other periods. Individuals are deleted once they quit. The model in equation (6) has at most 11 observations per person corresponding to the 11 time periods in Figure 3. Individuals who smoke in all periods are the censored observations. The first period is defined by the window starting 9.5 years before treatment and ending 8.5 years before treatment. The last period is the window from 0.5 years after treatment to 1.5 years after treatment. The key window is period 10 and spans the dates from half a year before treatment or diagnosis to half a year after. That is the only period in which $a_t - a_{t-1}$ is equal to 1. Since there are repeat observations on all persons except those who quit in period 2, we cluster standard errors at the individual level. Standard errors that ignore clustering are, however, very similar to those that take account of it. This would be the case if the unspecified disturbance term in the log smoking participation function in equation (5) is a random walk. In that case, we eliminate serial correlation by taking first differences.

Results are shown in Table 5. Note that ΔAfter stands for $a_t - a_{t-1}$, ΔDiagnosed stands for $d_t - d_{t-1}$, and ΔAMI stands for $h_t - h_{t-1}$ in the table. In column 1, we exclude the AMI, Elixhauser

variables, and the individual characteristics. In addition, we do not distinguish between the period in which CAD was diagnosed and the period in which treatment began. Since those two periods are the same for MM patients, the coefficient of ΔAfter in column 1 reflects the increase in the quit rate of those patients in the first period after diagnosis (the one-year window from six months before diagnosis to six months after diagnosis). This allows us to replicate the results from the single period quit model (Table 4, column 1) with one change. In column 1 of Table 4 – the single period regression – the constant term of 10.1 percent reflects the quit rate for MM patients during the period when they receive their diagnosis (which for them, we defined to be the start of their treatment). In column 1 of Table 4, we have taken advantage of the time-series data to decompose this number into two parts. The coefficient on the constant term, 4.9%, reflects the average quit rate for patients in periods when treatment is not being initiated. The coefficient of 5.2% on the $\Delta After$ variable reflects the increase in the quit rate for MM patients in the period when they are diagnosed. The coefficients on $PCI * \Delta After$ and $CABG * \Delta After$ in Table 5 are identical to the corresponding coefficients in Table 4. This suggests that the exclusion of period dummies or a nonlinear trend prior to treatment in the smoking participation equation is appropriate.[16]

[16] Additional evidence in support of this proposition is contained in the multi-period quit model estimated with the aggregate data in Table 7.

Table 5 – Regression Results for Multi-Period Quit Model with Individual Data

	(1)	(2)	(3)	(4)	(5)	(6)
Constant	0.049***	0.049***	0.049***	0.049***	0.047***	0.046***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)	(0.011)
ΔTreated	0.052***					
	(0.008)					
PCI * ΔTreated	0.073*	0.090**	0.097***	0.054	0.034	0.033
	(0.039)	(0.038)	(0.037)	(0.040)	(0.040)	(0.039)
CABG * ΔTreated	0.200***	0.225***	0.241***	0.195***	0.160***	0.155***
	(0.045)	(0.045)	(0.051)	(0.045)	(0.044)	(0.044)
ΔDiagnosed		0.050***	0.052***	0.042***	0.025***	0.025***
		(0.008)	(0.008)	(0.008)	(0.008)	(0.008)
PCI * ΔDiagnosed			-0.013			
			(0.033)			
CABG * ΔDiagnosed			-0.031			
			(0.040)			
ΔAMI				0.083***	0.075***	0.076***
				(0.023)	(0.024)	(0.024)
P-Value: PCI * ΔTreated = CABG * ΔTreated	0.031	0.021	0.023	0.016	0.030	0.034
Controls						
Elixhauser					X	X
Demographics						X
Observations	26658	26658	26658	26658	26658	26658
Individuals	3065	3065	3065	3065	3065	3065

Robust standard errors, clustered at the individual level, in parentheses. Regressions are weighted by NHIS probability weights. ΔAMI is an indicator for a patient having his first AMI during a particular period. Specifications 5 and 6 include 29 dummy variables for the Elixhauser commorbidity conditions first diagnosed during a particular period. In specification 6, demographic controls include gender, race, education dummies, and income category dummies (including a dummy for missing income data). "P-Value: PCI * ΔTreated = CABG * ΔTreated" is the p-value on a test of equality of the PCI * ΔTreated and CABG * ΔTreated coefficients. * significant at the 0.10 level; ** significant at the 0.05 level; *** significant at the 0.01 level.

In column 2 of Table 5, we add a diagnosis effect. The $\Delta Diagnosed$ indicator switches from zero to one when MM patients, PCI patients, *or* CABG patients are diagnosed, whether the diagnosis occurs in the same period as the treatment or not. Being diagnosed with CAD is

associated with a 5.0 percentage point increase in the probability of quitting, on top of the typical yearly quit rate of 4.9 percent.[17] Being treated with PCI or CABG is associated with an incremental 9.0 or 22.5 percentage point increase in the quit rate, respectively. This assumes that diagnosis and treatment occur in the same period. If not, the diagnosis coefficient must be subtracted since ΔDiagnosed equals zero for PCI and CABG patients diagnosed before treatment but equals one for all MM patients. That results in an incremental 4.0 percentage point increase for PCI patients and an incremental 17.5 percentage point increase for CABG patients. Since 40 percent of PCI patients and 43 percent of CABG patients are diagnosed before treatment, the average percentage point increases in the quit rates are 7.0 and 20.4, respectively.[18] Regardless of how these computations are made, the difference between the coefficients on PCI and CABG treatments is significant at the $\alpha = 0.05$ level.

In column 3, we test for a differential diagnosis effect of PCI and CABG patients. The estimated coefficients are relatively small and not significantly different from zero. In the remaining specifications, we assume that there is a single diagnosis effect that does not vary by treatment. In column 4, we control for one's first AMI. In column 5, we further control for the Elixhauser comorbidity conditions. And, in column 6, we add controls for demographic characteristics. An AMI proves to be a strong predictor of quitting smoking, increasing the predicted probability of quitting by between 7.6 and 8.4 percentage points, depending on the specification. As with the single period regression, once we add AMI, the estimated size of the PCI coefficient declines and is no longer statistically significant at conventional levels. In our

[17] The 5.0% diagnosis effect is similar to the 5.2% coefficient on $\Delta After$ in column 1, which reflects the diagnosis effect for all medically managed patients and for PCI and CABG patients diagnosed in the same period as treatment.
[18] For MM patients, the increase in the quit rate is 0.0501. Since 60 percent of PCI patients are diagnosed in the same period as treatment, the average predicted increase in the quit rate for these patients is 0.0897 + 0.60*0.0501 = 0.1198. The difference between that increase and the increase for MM patients is 0.0697 or 7.0 percentage points. Since 57 percent of CABG patients are diagnosed in the same period as treatment, the average predicted increase is 0.2250 + 0.57 * 0.0501 = 0.2536. The difference between that increase and the increase for MM patients is 0.2035 or 20.4 percentage points.

final specification, we find that a PCI procedure is associated with a 3.3 percentage point increase in the probability of quitting for patients who were diagnosed in the same period in which they were treated. However, given the imprecision of our estimate, we cannot rule out the possibility of no association. While the coefficient on CABG also declines somewhat, it remains large and strongly statistically significant, both in comparison to medically managed patients and PCI patients. In our sixth specification, undergoing CABG surgery in the same period in which CAD was diagnosed is associated with a roughly 15.6 percentage point increase in the probability of quitting smoking. The increased quit rate associated with CABG is substantially larger than the increase associated with less invasive treatments for CAD, and approximately twice the increase associated with an AMI.

Model 3: Multi-Period Quit Model Using Grouped Data

To illustrate that our results are not sensitive to a flexible specification of period effects and to account for clustering of disturbance terms by group and period at the individual level, we aggregate the data into 12 periods for each of the three groups of patients. There are 36 cells in the aggregate sample. For each patient group, there are 12 smoking participation rates, ranging from the rate 9.5 years before surgery to the rate 1.5 years after surgery. Along the same lines, there are 11 quit rates computed from the smoking participation rates for the current and prior period.

Table 6 – Grouped Data on Smoking Participation and Quit Rate

A. Unadjusted Quit Rate

	Quit Rate			Difference in Quite Rate			
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Period	CABG	PCI	MM	CABG - PCI	CABG - MM	PCI - MM	ΔAfter
-9	6.3%	4.2%	5.1%	2.1%	1.2%	-0.9%	0
-8	5.7%	6.0%	4.0%	-0.3%	1.7%	2.0%	0
-7	4.4%	5.8%	4.4%	-1.5%	0.0%	1.5%	0
-6	3.4%	3.7%	4.4%	-0.3%	-1.0%	-0.7%	0
-5	4.2%	5.8%	4.5%	-1.6%	-0.4%	1.3%	0
-4	4.3%	1.4%	4.8%	3.0%	-0.5%	-3.4%	0
-3	9.1%	4.9%	4.7%	4.2%	4.4%	0.2%	0
-2	7.9%	3.6%	5.5%	4.2%	2.4%	-1.9%	0
-1	10.9%	8.3%	5.8%	2.5%	5.0%	2.5%	0
0	29.6%	17.4%	10.2%	12.2%	19.4%	7.2%	1
1	7.4%	7.0%	5.3%	0.4%	2.1%	1.7%	0

B. Adjusted Quit Rate

	Adjusted Quit Rate			Difference in Adj. Quite Rate			
Period	CABG	PCI	MM	CABG - PCI	CABG - MM	PCI - MM	ΔAfter
-9	6.2%	4.1%	5.1%	2.2%	1.2%	-1.0%	0
-8	5.1%	6.0%	4.2%	-1.0%	0.8%	1.8%	0
-7	4.2%	5.7%	4.3%	-1.5%	-0.1%	1.4%	0
-6	2.6%	3.8%	4.6%	-1.2%	-2.0%	-0.8%	0
-5	4.4%	7.6%	4.4%	-3.1%	0.1%	3.2%	0
-4	4.4%	1.3%	5.1%	3.1%	-0.6%	-3.8%	0
-3	7.7%	5.4%	4.5%	2.3%	3.2%	0.9%	0
-2	6.8%	3.6%	5.2%	3.2%	1.5%	-1.7%	0
-1	10.1%	8.0%	5.1%	2.2%	5.0%	2.9%	0
0	22.6%	10.6%	8.4%	12.1%	14.3%	2.2%	1
1	5.6%	6.7%	4.1%	-1.1%	1.6%	2.6%	0

Note: adjusted quit rate adjusts for diagnosis, AMI, and Elixhauser comorbidities

We obtain two quit series. The first, shown in Panel A of Table 6, is unadjusted for covariates. It is identical to the quit rate that appears in Figure 3. The second quit series, shown

in Panel B of Table 6, adjusts for effects due to diagnosis, AMI, and Elixhauser comorbidities. It is obtained from the individual data by estimating a discrete time hazard function for the probability of quitting that includes 11 period dummies interacted with each of three treatment dummies (one for CABG, one for PCI, and one for MM), and the diagnosis, AMI, and Elixhauser variables defined in equation (6). The 33 coefficients associated with the period-treatment interactions are quit rates by group and period adjusted for the effects of the last three variables just mentioned.[19]

In the spirit of Donald and Lang (2007), we use this data to perform simple difference-in-differences regressions with 11 observations. To illustrate the model that we estimate, consider a log smoking participation function for two groups (g = c or p, c = CABG, p = PCI):

$$\ln s_{gt} = \mu + \rho c + \beta c a_t + 11 \text{ period dummies} + \varepsilon_{gt}. \qquad (7)$$

Here a_t, as defined in equation (5), is an indicator that equals 1 in each of the two periods after treatment and ε_{gt} is the error term. Take the difference between each group in a given period to eliminate the intercept (μ) and the period dummies. Then take first differences to eliminate the group effect (ρ):

$$\ln s_{ct} - \ln s_{ct-1} - (\ln s_{pt} - \ln s_{pt-1}) \cong q_{ct} - q_{pt} = \beta c(a_t - a_{t-1}) + \text{error}. \qquad (8)$$

Equation (8) is a regression forced through the origin with 11 observations. The dependent variable is the difference between the quit rate of CABG patients and the quit rate of PCI patients in each period. The independent variable, $(a_t - a_{t-1})$, equals 1 in the window spanning the period from 6 months before treatment to six months after treatment (period 10) and equals 0 in each of the other 10 periods or quit windows.

[19] We do not adjust for demographic and socioeconomic characteristics since the inclusion of these characteristics has a very minor impact on the estimates in Table 5.

This approach has a number of attractive features. First, aggregation accounts for clustering in the disturbance term in an individual-level log smoking participation or quit equation by group and period. Second, if the error term in equation (7) is a random walk, then serial correlation is eliminated once first differences are taken. Third, the regression specified by equation (8) implicitly controls for a full set of period effects. Finally, by focusing on the difference in the quit rates in each period, we are asking whether this difference during the treatment period is sufficiently unusual compared to past and future values that it is unlikely to have arisen by chance. If the quit rates in the two series normally track one another but do not during the treatment year, we would expect that there is something unusual about the treatment year. On the other hand, if the quit rates in the two series often diverge wildly, then a substantial divergence in the treatment year might simply be due to chance.

Six aggregate quit regressions are contained in Table 7. The three in the top row employ the unadjusted quit series, while the three in the bottom row employ the adjusted quit series. In column 1, the dependent variable is the difference between the CABG and PCI quit rates; in column 2, it is the difference between the CABG and MM rates; and in column 3, it is the difference between the PCI and MM rates. Three separate regressions are obtained for each series because of evidence that the residual variance is not the same for each dependent variable.[20] To be consistent with the notation in Table 5, the variable $a_t - a_{t-1}$ is termed ΔAfter in the table.

[20] Consider the following two regressions

$$q_{pt} - q_{pt-1} - (q_m - q_{mt-1}) = \beta_p(a_t - a_{t-1})$$
$$q_{ct} - q_{ct-1} - (q_{mt} - q_{mt-1}) = \beta_c(a_t - a_{t-1}),$$

where m denotes medical management. Estimates of β_p, β_c, and $\beta_c - \beta_p$ could be obtained from a pooled regression of the form

$$q_{gt} - q_{gt-1} - (q_m - q_{mt-1}) = \beta_c c(a_t - a_{t-1}) + \beta_p(1 - c)(a_t - a_{t-1}).$$

We do not follow that approach because the residual variance in the first regression is not equal to the corresponding variance in the second regression.

Table 7 – Quit Rate Regression with Grouped Data

	CABG - PCI	CABG - MM	PCI - MM
ΔAfter	0.122***	0.194***	0.072***
(no adjustments)	(0.025)	(0.025)	(0.018)
ΔAfter	0.121***	0.143***	0.022
(with adjustments)	(0.022)	(0.022)	(0.022)
N	11	11	11

Note: Each cell represents the coefficient on ΔAfter from a separate regression. The dependent variable is the quit rate for one group of patients minus the quit rate for another. The independent variable is a dummy for the treatment year (ΔAfter). In the top row, the quit rate is unadjusted. In the bottom row, the quit rate is adjusted for diagnosis, AMI, and Elixhauser comorbidities. Regressions are forced through the origin. OLS standard errors are in parentheses.
* significant at the 0.10 level; ** significant at the 0.05 level; *** significant at the 0.01 level.

Focusing on the first top row of Table 7, one sees that the increases in quit rates of CABG patients and PCI patients compared to MM patients in the treatment period (19.4 percentage points and 7.2 percentage points, respectively) are practically identical to the corresponding estimates in column 1 of Tables 4 and 5. Of course, the same holds for the 12.2 percentage point differential between the quit rates of CABG and PCI patients. The standard errors associated with each of these estimates are smaller in Table 6 than in Tables 4 and 5, suggesting that those in the latter two tables are conservative lower-bound estimates. The coefficients from the regressions based on the adjusted series in the bottom row of Table 7 tell a similar story. Once we control for diagnosis, AMI, and the Elixhauser comorbidities, the CABG-PCI differential remains at 12.1 percentage points, while the PCI-MM differential falls to 2.2 percentage points and is not statistically significant.

V. Conclusion

Coronary Artery Disease is a frequently occurring and deadly disease. There are several common treatments – including medical management, PCI, and CABG – and each has benefits and costs associated with it. In this paper, we have examined one previously unexplored benefit of more invasive treatment: those who undergo a procedure, particularly the more invasive CABG surgery are more likely to quit smoking. In our preferred regression model, we estimate that CAD patients who undergo PCI rather than medical management are three percentage points more likely to quit smoking in the one-year window surrounding their surgery. Patients who undergo CABG are nearly 16 percentage points more likely to quit smoking during this timeframe. These results are robust to a number of alternative specifications.

While we do not have data on behaviors other than smoking, we suspect that patients undergoing more invasive surgery are also more likely to improve their diet, limit excessive consumption of alcohol, and (when recommended) exercise more. Taken together, these behavioral responses may offset the inherent risks in more invasive surgery and help explain why the longer term outcomes for CABG patients rival or even exceed those of similar patients receiving PCI or medical management. Our findings also highlight the importance of emphasizing healthier behavior to those patients who have less invasive medical treatment.

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