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Technology diffusion and health care productivity: angioplasty in the UK

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Abstract

The adoption of new medical technologies is argued to be a major contributory factor to the rising cost of health care although there is little empirical work devoted to exploring the mechanism of how this process works. This study builds on recent research by Cutler and Huckman to establish the degree to which a new technology, percutaneous transluminal coronary angioplasty (PTCA), substitutes for an older one (Cutler, D. and Huckman, R., 2003, Technological development and medical productivity: the diffusion of angioplasty in New York state, *Journal of Health Economics*, 22, 187-217). Using patient specific data over a 15-year follow-up period the mortality and morbidity impacts of PTCA relative to coronary artery by-pass grafting (CABG) are established. In considering the substitution process, hospital level data and control for medical management of CHD improves on the empirical specification suggested by the earlier research and the analysis explicitly controls for the endogeneity problems in estimating the process of substituting one hospital technology for another. Such improvements give robust estimates of the degree to which PTCA has substituted for CABG, as opposed to expanding surgical treatment to the potential patient population. Thus PTCA, although acting to reduce treatment costs through the process of substitution for the more expensive procedure is shown to increase overall costs through increasing the potential patient population that could be treated for CHD with surgery.

JEL classification: I1, O3

Keywords: Technological change, Medical productivity

1. Introduction

Advances in medical technologies generally expand what is possible and thus lead to increasing demand *and* supply of health care. The adoption of new medical technologies has long been argued to be a contributory factor to the rising cost of health care in developed countries around the world (Cutler & McClellan 1998; Cutler, McClellan, & Newhouse 1999; Newhouse 2002; Weisbrod 1991). There is little empirical work devoted to exploring the mechanism of how this process works however, with the majority of such studies focused on technology diffusion in the USA. Recently, in one of the rare quantitative papers to address this issue, Cutler and Huckman (2003) provided evidence on the impact that the diffusion of a specific surgical procedure for coronary heart disease, percutaneous transluminal coronary angioplasty (PTCA), had on treatment productivity in New York State. This is an interesting case as PTCA is generally considered a potential substitute for the more expensive surgical procedure, coronary artery by-pass grafting (CABG). Given that lower unit costs are associated with PTCA as compared to CABG it might be expected that total health care costs, or at least their rate of growth, would fall in this disease area. However as Cutler and Huckman (2003) (hereafter CH) show, while PTCA does act as a substitute for CABG for many patients, it also leads to treatment expansion as less severely ill patients are treated with the new technology. The impact is therefore to increase overall health care costs even though there is a process of substitution at work.

The aim of this paper is to revisit the empirical relationship between PTCA and CABG for a number of reasons. First, as CH note it is interesting to consider whether a similar pattern of diffusion exists in other health care environments to test the robustness of their findings. The data presented here relate to the UK where, through on-going collaborative research of trends, it has been established that the regulatory environment tends to lead to a different pattern of diffusion for these two technologies across a number of countries (Tech Investigators, 2001). In general, possibly reflecting stricter budgetary constraints and an associated greater regulatory control of new technology, the UK has had slower and lower up-take rates of both CABG and PTCA than the USA generally. The standardised rate of CABG for example within one year of admission for acute myocardial infarction (AMI), controlling for country specific demographic differences, was under 5% in the UK in 1998 compared to around 20% in the USA. The rate of PTCA one day after admission for AMI was less than 4% in the UK and, reflecting a more aggressive use of this technology, approximately 11% in the USA in

1998. Even acknowledging CH's observation that their data from New York state may differ from other states in the USA, the differences between the UK and the USA are of an order of magnitude that deserves an assessment of whether the relationship exposed between PTCA and CABG in the USA holds in other health care systems.[1] If there is a different relationship this will begin to give understanding of any different diffusion mechanisms at work across two major health care systems and inform debate over differences in expenditure rates across health care systems.

Secondly, in the empirical specification used as part of their overall assessment of PTCA's productivity impact directed at quantifying the degree of substitutability between PTCA and CABG, CH acknowledge that there is inherent bias imbedded in their regression coefficients as unobservable factors may be correlated with the CABG and PTCA rates. As an example they cite varying rates of medical management for CHD across different localities which they can not control for due to data constraints. They argue that as their analysis of substitutability relies on examination of the *change* in the specific coefficient of interest over time, under the assumption that the bias is constant over time, their analysis is unaffected. While this may be true the assumption of constant bias is crucial to their analysis. As the authors point out, their analysis essentially assumes that "the unobservables have the same impact on technology utilization over time" (op.cite., p192). This implies that any unaccounted change in medical practice and productivity, for instance through changes in medical management arising from drug therapy or in medical preferences, has no differential impact on their estimates over the 18 years of their analysis. As described below, in this paper the econometric model will use hospital level data and control for medical management of CHD, proxied by statins prescriptions, improving the empirical specification. Further, the parameters of the substitution process are estimated by instrumental variables, thus explicitly controlling for the endogeneity bias and avoiding a constant bias assumption to interpret the parameter estimates.

Thirdly, as part of their analysis of the productivity impact of the newer technology CH also consider the differential effect that PTCA has on health outcomes as compared to CABG. This assessment is limited, however, by the fact that their data only allows estimation of the impact PTCA has on within hospital mortality over a constrained time period and does not allow inspection of long-term outcomes or morbidity data measured, for example, through hospital re-admissions. The data used here incorporates long-term follow-up and therefore allows a

1 The rates in New York state are acknowledged by CH (op.cite., p212) to be low compared to other USA states.

more extensive examination of this relationship.

As has been documented by the TECH Investigators (2000) both CABG and PTCA have diffused differently across different health care systems. Three basic patterns are discernible. The first, characterised by the USA, is one of early start-up and a quick rise in up-take in new procedures. The second pattern, characterised by Canada and Australia, involves a later start followed by relatively fast up-take. The last pattern, characterised by the Nordic countries and the UK, involves later start-up and slower diffusion. From these results it could be inferred that the process of substitution across these two procedures might vary markedly across the USA and the UK. Consistent with this earlier study, a simple comparison of procedure rates per 1000 population suggests that the trend rates were different when comparing absolute levels. However the trends were similar in an important aspect. Figure 1 presents the annual procedure rates of CABG and PTCA for the UK patient data used in this analysis. The procedure rates are expressed as the number of procedures per 1000 population aged 45 and over.[2] The *relative* pattern follows that shown by CH (op. cite. Fig. 1 in CH) almost exactly. Up until 1997 the CABG and PTCA rates rise together with the PTCA rate rising faster than the CABG rate. After 1997 the rates move in opposite directions.[3] The conclusion from this descriptive data follows therefore that of CH - the differing growth rates and general movement in opposite direction after 1997 indicates that PTCA, while it may begin to diffuse as a complementary procedure, is a growing substitute for CABG over time. The comparison between the data used here with that of CH reflects the different absolute diffusion up-take rates across these countries already noted in the TECH study (op. cite.), is that the New York state rates rise faster for both CABG and PTCA when compared to the UK. First indications would suggest therefore that while a similar substitution mechanism may be operating in both the USA and the UK the degree of substitution, as dictated by procedure growth rates, may well be different.

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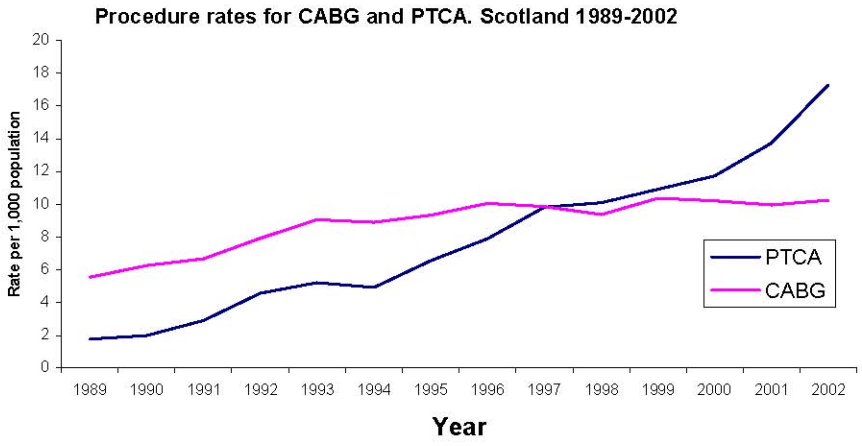
2 The population figures used to calculate these rates and for the Scottish health board regions are based on the Office of Population and Census and Office of National Statistics estimates.

3 The crossover year in the CH data are 1996. Of interest is the slight flattening of up-take in PTCA between 1992 and 1994 with subsequent growth, probably attributable to the introduction of stents.

Figure1.



The paper proceeds as follows. The next section provides some background material, as well as describing the data used in the analysis. Section 3 and 4 discuss issues of specification and estimation in the analysis of productivity and substitution respectively, together with the estimation results. Section 5 concludes.

2. Data

Data were retrieved from the Medical Record Linkage database held by the Information and Statistics Division (ISD) of the NHS in Scotland. This database holds linked data on all inpatient and day case hospital episodes from 1981 onwards within Scotland – excluding psychiatric and maternity admissions. The ISD Medical Record Linkage Database is detailed elsewhere (Kendrick and Clarke, 1993). It has been subjected to a number of reviews relating to its quality and ability to link hospital episodes (e.g. Kendrick and Clarke, 1993; Hartley and Jones, 1996). The database has been found to have a high level of accuracy as assessed by an internal audit of one per cent of the hospital returns annually. The accuracy of the linkage system is around 99 per cent overall, while reviews of individual diagnostic categories and surgical procedures returns an accuracy of 90 per cent and 94 per cent respectively. Moreover, the demographics of the Scottish population are advantageous as the population is stable and has low levels of annual migration. The recorded patient level linked data include the patient's age and sex, disease classification and co-morbidity data as based on ICD-9 and ICD-10 diagnostic codes, length of stay, operative procedures performed based on the UK

Office of Population Censuses and Surveys (OPCS, 1997) surgical and procedure codes (OPCS-3 and OPCS-4), whether the hospital admission was elective or emergency and discharge information.

The data used in this analysis relate to the linked patient level hospital records for individuals who had either a PTCA or a CABG performed within the Scottish region of the UK National Health Service (NHS), over the period 1989 to 2003. The start date for this analysis was dictated by the fact that data retrieval were based on OPCS surgical and procedure codes and PTCA was only assigned such a code in 1989 as it was at that time a new procedure. All coronary heart disease (CHD) events were therefore retrieved for the years 1989-2003 and the sub-set of patients who had either PTCA or CABG identified. All such individual patient records were used, as discussed in more detail below, to analyse the impact that PTCA had on long-term health outcomes over a total patient population of 58,842.

For the analysis of the potential substitution of PTCA for CABG the focus is on the hospital level. Hospital records, the appropriate level of decision-making in analyzing the up-take of new surgical technology, were confined to those hospitals that were the main providers of CABG and PTCA procedure within this NHS region. This gave a panel of 4 major hospitals over 11 years for the analysis of substitution between the two procedures. Data were also retrieved from ISD on prescribing patterns for statins by health board region and the calculated prescribing rate for the health boards was applied to individual hospitals[4] as a proxy for the level of medical management of CHD, and taken to be a substitute (at the margin) for surgical intervention. This allows control for a major unobservable factor noted by CH (op. cite.) in their analysis. These prescribing data were only available from 1992 onwards which is why the analysis of substitution between the two procedures is restricted to an 11-year period. Demographic information for the hospital catchment's area was based on the relevant health board population data which was gained from the UK Office of Population and Census and Office of National Statistics (www.statistics.gov.uk).

3. Analysis of PTCA impact on health outcome

Following CH the analysis here first considers the impact on health outcome for those individuals receiving either PTCA or CABG. Three measures of health outcome are proposed;

4 The overwhelming majority of Scottish health boards only have one hospital represented in the sample.

in-hospital mortality, any mortality recorded over the (maximum) follow-up period of 15 years and a combined hospital readmission and mortality outcome measure. The latter is taken as an indicator of the impact of PTCA on both mortality and morbidity. Two basic models are analysed. First, a logistic regression is undertaken with the dependent variable defined as either in-hospital mortality or long-term mortality defined as observed mortality over the 15-year period of follow-up. Proxying both dependent variable definitions by $MORT_i$ the regression model is of the form:

$$\ln\left(\frac{\Pr(MORT_i=1|x_i)}{1-\Pr(MORT_i=1|x_i)}\right)=\alpha+\beta_1(PTCA_i)+(\beta_S-\beta_1)(PTCA_i\times\{year\in S\})+x_i'\gamma+v_i \quad (1)$$

where is an indicator variable (one for received PTCA and zero for received CABG), this indicator is then interacted with three-yearly time period indicators which captures any trend improvement in the performance of PTCA relative to CABG with respect to outcome. These year interactions are estimated relative to the initial data period of 1989-1991. Finally a vector of control variables is included that includes year dummies, hospital dummies, age, the diagnosis on presentation for the initial procedure, (coded 1 if AMI; 2 if ischaemic heart disease; 3 if stroke, and 4 otherwise) and further dummy variables indicating the presence or absence of co-morbidities and whether or not the admission was an elective or emergency.

Given that PTCA and CABG affect both mortality and morbidity an analysis was also undertaken allowing for multiple outcome measures; multiple end-points. As well as considering mortality within the period of follow-up, counts of hospital readmission based on three separate groupings of one, two or three or more readmissions over the 15-year period of follow-up, taken as indicators of morbidity, were estimated through a competing risk duration model. While it may be argued that unobserved heterogeneity is minimised across patients, given selection criteria for the use of these procedures, it is possible that it remains an issue. A frailty model was therefore specified. In fact a shared frailty model was proposed given that it is unreasonable to assume that the probability of any given outcome was statistically independent of any other for any given individual patient. If no account were taken of this correlation the underlying hazard rate being modelled would be underestimated. For estimation purposes an accelerated failure time model incorporating shared frailties across individuals for the pre-defined health outcomes was specified to have a lognormal survival function.

Time (*t)* to one of the four pre-specified endpoints (*k*) defines the probability of exit to one of the *k* multiple destinations and is dependent on the hazard function to destination *k*, for the cluster of patients, *i*, treated at hospital *j*, which is given as $\lambda_k\left(t_{ij}\middle|x_{ij}\right)$. The probability of exit to destination *k* is therefore given as:

$$P(k:t,t+dt)=\lambda_k(t_{ij}\middle|x_{ij})\exp\left\{-\sum_{j=1}^{K}\int_0^t\lambda_j(u)du\right\}$$

where the first term on the right hand side are the transition intensities based on the hazard function and the second term is the survival function to at least time *t*. Unobserved heterogeneity is allowed for by introducing a frailty as an unobserved multiplicative effect on the hazard function, which is clustered around the index variable *i*. The distribution function of the unobservable heterogeneity over the population is specified as Inverse Gaussian. Finally the unobservable heterogeneity is assumed to be shared across individuals when these individuals face the competing risks of attaining the various end-points. The confounding variables, the x_{ij}, are the same as used in the logistic regressions. This specification leads to a likelihood function which estimates the hazard function for each of the end-points for each individual relative to a baseline hazard for the population which takes account of unobserved heterogeneity constrained to be similar for each individual even when facing different end-points. The baseline hazard is assumed to be log-normal.[5] With these constraints the likelihood function simultaneously estimates the parameter coefficients, β, and the ancillary parameters. It is these β coefficients which are of interest, and in particular the coefficient on whether or not PTCA was performed as the sign will indicate whether receiving PTCA reduced the probability of reaching one of the multiple end-points: death or 1, 2, and 3 or more hospital re-admissions.

Table 1 presents the results of the patient level analysis measuring the impact of PTCA on health outcomes. The first two equations relate to the logistic regressions using in-hospital mortality and any mortality recorded within the 15-year follow-up period as dependent variables. The third equation presents the results of the competing risks model described above.

Table 1. Results of PTCA on health outcomes

	Logistic: In hospital deaths	Logistic: All deaths	Competing risk: All deaths, 1, 2 & 3 or more hospital re-admissions
	Coeff. (s.e.)	Coeff. (s.e.)	Coeff. (s.e.)
Received PTCA	-1.22** (0.031)	-0.443** (0.072)	-0.358** (0.016)
Age	-4.275** (1.227)	-4.783** (2.603)	-0.005 ** (0.0001)
Age squared	9.212** (0.976)	8.982** (1.966)	
Elective admission	-0.573** (0.041)	-0.121** (0.076)	0.470** (0.023)
Type of CHD/CVD	0.224** (0.019)	0.389** (0.032)	-0.189** (0.012)
Presence of co-morbidity	0.509** (0.029)	1.075** (0.072)	-0.3324** (0.016)
Constant	1.478** (0.928)	0.508 (1.231)	7.0695** (0.441)
Year effects	Yes	Yes	Yes
Hospital effects	Yes	Yes	Yes
Pseudo R^2	0.121	0.171	
Log likelihood	-23163.59	-6806.28	-307105.66
# observations	58842	58842	58826

Notes: the Age variable has been re-scaled, and the age squared term was not included in the competing risk analysis.
** significant at 1% level

The results of all three equations confirm the CH finding, as the coefficient on the PTCA indicator is negative and highly significant in each case, that patients who received PTCA are less likely to die in hospital or over the follow-up period than those receiving CABG. The competing risks model infers that morbidity, as modelled through multiple hospital re-admissions, is also likely to be improved in those receiving PTCA rather than CABG. Indeed the results tend to support the suspicion noted by CH that given their data limitations, particularly that the definition of mortality in their analysis is confined to in-hospital mortality

which is rarely associated with PTCA or CABG (generally post-operative death rates are less than 2% even for CABG), their regression results should only be taken as indicative of the impact.[5] The signs on all the other coefficients are consistent across the logistic equations with the probability of death increasing with age, emergency admissions and the presence of co-morbidity. The coefficient signs in the competing risk model are more complex to interpret as the effect relates not only death but also to re-admission rates. The negative sign on the type of CHD/CVD points to an association of higher re-admission rates for individuals who initially suffered an AMI or IHD rather than stroke. Moreover the positive sign on the type of admission may also merely be picking up higher re-admission rates of survivors, proxied by initial elective rather than emergency admissions, over time. This may also explain the negative sign on the co-morbidity variable. Notwithstanding the more complex interpretation of these coefficients in the competing risks model, all results are highly supportive of PTCA leading to improved outcomes over CABG.

4. Analysis of the substitution of PTCA for CABG

To formalise their analysis of the degree to which PTCA has substituted for CABG, CH specify the following model using county as the unit of analysis:

$$\left(\frac{CABG}{Pop}\right)_{it} = \alpha_i + \delta_t + \beta_1\left(\frac{PTCA}{Pop}\right)_{it} + \left(\beta_S - \beta_1\right)'\left(\left(\frac{PTCA}{Pop}\right)_{it} \times \{t \in S\}\right) + x_{it}'\gamma + \varepsilon_{it} \qquad (2)$$

where the dependent variable, the CABG rate per 1000 population aged 45 and over, is regressed against county fixed effects (α_i), year fixed effects, (δ_t) the PTCA rate per 1000 population aged 45 and over, an interaction term of the PTCA rate per 1000 population aged 45 and over with $\{t \in s\}$, a vector of indicators of 3-year periods, and a vector of demographic controls x_{it} , including the percentage of a counties population that falls into each of three age categories: under 45, 45-64 and over 65, and the rate of total hospital discharges per 100,000

5 The lognormal survival function is represented by a hazard that first increases from zero and then falls towards zero and parameterised as $h_{ij}(t|x_{ij}) = 1 - \Lambda\left\{\frac{\ln(t_{ij}) - \beta X_{ij}}{\sigma}\right\}$. The Inverse Gaussian distribution is commonly applied to model unobserved heterogeneity in such models because of it's analytical tractability. Fuller discussion of frailty models is found in Lancaster (1990, ch. 6)

population to control for shifts in overall hospitalisation rate. They also include the different form of payment mechanism (Medicare, Medicaid and HMO), which are not relevant to this analysis. [6]

To initiate the analysis performed here the same specification is used with subtle differences. As noted above, instead of the county being the level of analysis, the hospital is used as the unit of observation in this study. This reduces sample size but better reflects the level at which decisions are made concerning the substitution of PTCA for CABG. The total hospital discharge rate is also specified at the hospital rather than county level. All population rates are defined with respect to the relevant Health Board population level.[7]

In the CH analysis, the coefficients of greatest interest are represented by the vector $(\beta_S-\beta_1)$. By using time-varying coefficients the degree of substitution between the procedures is allowed to change over time as PTCA matures. CH consider this specification in levels and, through differencing the variables CABG, PTCA and total discharges only, also with respect to trends in the growth rate. The latter is referred to by CH as a changes specification. CH note that unobservable factors ε_{it} will be correlated with both CABG and PTCA and therefore the OLS estimator for β_1 and the β_S will be biased and consequently the value of substitution for any given period. By assuming that the bias in any given period is constant, they argue that $(\beta_1-\beta_S)$ can be estimated without bias. By then assuming that $\beta_1=0$, CH obtain the substitution rates over time.

We endeavour to deal with the endogeneity problem in two ways. First, in the specification considered here an important unobservable, namely the medical management of CHD, is controlled for. This is done through the inclusion of a variable which quantifies the proportion of the relevant Health Board population who were prescribed statins to proxy the use of medical management within the at risk population.

Secondly, we estimate the parameter β_S' directly taking account of the endogeneity of by the

6 In fact CH do not use the results in their main productivity calculations but rely on results from randomised clinical trials which compare the two procedures.

7 There are 15 Health Boards in Scotland, nine have one hospital per Health Board; four have two; and one have five and three hospitals respectively. With very little cross-Board flows for these procedures, the Health Board population therefore represents a relatively good proxy for each hospitals population draw.

method of Instrumental Variables (IV). As there are two sources of endogeneity, the correlation between $PTCA_i$ and the hospital effects α_i and the time varying unobservables ε_{it}, we first take first differences of model (2) which eliminates the hospital effects α_i :

$$\Delta\left(\frac{CABG}{Pop}\right)_{it} = \Delta\delta_t + \beta'_S\left(\Delta\left(\frac{PTCA}{Pop}\right)_{it} \times \{t \in S\}\right) + \Delta x'_{it}\gamma + \Delta\varepsilon_{it}. \qquad (3)$$

.

(3)

As $\left(\frac{PTCA}{Pop}\right)_{it}$ will be correlated with the unobservables ε_{it}, we instrument the differences $\Delta\left(\frac{PTCA}{Pop}\right)_{it} \times \{t \in s\}$ by lagged levels $\left(\frac{PTCA}{Pop}\right)_{it} \times \{t \in s\}$, along the lines of the standard panel data estimator of Arellano and Bond (1991). We estimate this model by Two-Stage Least Squares (2SLS), resulting in a consistent estimator for β'_S itself, i.e. no longer in deviation of β_1.

The initial results relating to the process of PTCA substitution for CABG are presented in Table 2 and replicate the CH model in levels, (model (2) above), but include the influence of the potentially important missing proxy relating to medical management (proxied through the level of statin prescription). The first two columns present the OLS estimation results with and without fixed hospital effects. The final column is specified in differences. The results for the fixed effects specification are remarkably similar to those of CH, as presented in their Table 2 (op. cit. CH p.201)). Using their assumption that the bias is constant over time and that substitution away from CABG accounts for none of the increased PTCA volume in the 1993-1995 period, we also find a substitution rate of around 40-50% by the end of the period. Clearly, the quite large positive estimate for the 1993-1995 period, in the fixed effects specification, may be due to endogeneity bias under these assumptions, $\hat{\beta}_1 = 0.58$ and the inclusion of the number of statins prescriptions perhaps does not control enough for this endogeneity problem.[8]

8 Interestingly, when the Statins variable is excluded from the fixed effects regression, the coefficients (βs) are not individually and jointly significantly different from zero.

Table 2. OLS estimation of the substitution impact of PTCA on CABG

CABG	Levels	Fixed effects	First Differences
	Coeff (rob s.e.)	Coeff (rob s.e.)	Coeff (rob s.e.)
PTCA rate (β_1)	0.1521 (0.421)	0.5756** (0.2254)	0.2817 (0.3212)
1995-1997 (β_2-β_1)	-0.1099 (0.0727)	-0.1761 (0.1350)	-.2806 (0.4204)
1998-2000 (β_3-β_1)	-0.3622 (0.4710)	-0.4029 (0.3224)	-0.009 (0.4988)
2001-2003 (β_4-β_1)	-0.0246 (0.0789)	-0.5090* (0.2611)	-0.4582 (0.4018)
Statins	-0.0157 (0.0133)	-0.0636** (0.0267)	-0.0358 (0.0358)
Discharge per 1,000 population	0.0064 (0.0064)	0.0045 (0.0103)	-0.106** (0.0122)
% population<45 years old	-0.1088 (0.1146)	-0.2756** (0.1445)	-0.1968 (0.1895)
% Pop. 45-64	0.1829** (0.1046)	-0.1366 (0.2245)	-0.9130** (0.2337)
Year effects	Yes	Yes	Yes
Hospital effects	No	Yes	No
# observations	44	44	44
# hospitals	4	4	4

* significant at 5% level;; ** significant at 1% level

Table 3 presents the results of the instrumental variables estimation procedure for model (3) in first differences. As can be seen for the full model with minimal instruments, reported in the first column of results, the overall level of significance is poor for the PTCA variables and for the variable proxying medical management; although the sign on this latter variable is in line with a priori expectations. To gain more parsimony the statins variable and age profile variables were dropped as regessors but retained as instruments, retaining their effect albeit indirectly. The estimation results are presented in the second column of Table 3. The third results column further places some exclusion restriction on the year effects. Indeed with restrictions placed on the year effects the model becomes relatively well-behaved, with the

instruments validity not rejected as indicated by the results of the *J*-test. Again, we find a significantly positive effect for the 1992-1994 period, as in the fixed effects model presented in Table 2, but now the results suggest that there is strong substitutability of PTCA for CABG by the end of the period, without having to make any assumptions about the constancy of bias and that there is no substitution in the first period.[9] This is consistent with the TECH (2000) results which show a much slower pattern of up-take of PTCA in the UK compared to the USA. This conservative pattern of up-take in the UK is consistent with PTCA initially being introduced in a complementary fashion in the UK, at a time when, according to the results of CH it was already acting as a strong substitute for CABG in the USA. However, reflecting the basic trends shown in Figure 1 above, PTCA begins to be a substitute procedure for CABG in the mid-1990s within the UK, although not strongly so until the last period of analysis. Moreover this degree of substitution is around the level estimated for CH with respect to their final period (the late 1990s). In this respect the findings here, that PTCA substitutes for CABG by 30%, support their hypothesis that PTCA does substitute for CABG at the margin by around 25 to 35% depending on the specification used by CH. This further supports the notion that, although PTCA is effective and less expensive than CABG, through expanding the potential treatment population overall health care costs are increased.

9 The coefficients $\beta_1,\ldots \beta_4$ are jointly significant with a p-value of 0.0000.

Table 3. IV estimation of the substitution impact of PTCA on CABG

CABG	First Differenced Models		
	Coeff (rob s.e.)	Coeff (rob s.e.)	Coeff (rob s.e.)
PTCA rate			
1992-1994 (β_1)	0.3501** (0.1015)	0.3704** (0.0887)	0.4243** (0.0674)
1995-1997 (β_2)	-1.1187 (1.3646)	-0.0783 (0.2275)	-0.1638 (0.1131)
1998-2000 (β_3)	0.9580** (0.3620)	-0.0985 (0.3934)	0.1108 (0.4233)
2001-2003 (β_4)	-0.4839 (0.3835)	-0.3532** (0.1777)	-0.2988** (0.0773)
Statins	-0.0265 (0.0472)		
Discharge per 1,000 population	-0.0099 (0.0089)	-0.0181** (0.0083)	-0.0159** (0.007)
% population<45 years old	-0.0155 (0.3001)		
% Pop. 45-64	0.5275 (0.5219)		
Year effects	Yes	Yes	Restricted
# observations	44	44	44
# hospitals	4	4	4
J-test: p-value (dof)	0.239 (4)	0.4 (7)	0.728 (7)
Instruments	$\left(\frac{PTCA}{Pop}\right)_{it-q} \times \{t \in s\}$, $\forall q = 2,3$	$\left(\frac{PTCA}{Pop}\right)_{it-q} \times \{t \in s\}$ $\forall q = 2,3$ Statins, Age	$\left(\frac{PTCA}{Pop}\right)_{it-q} \times \{t \in s\}$ $\forall q = 2,3$ Statins, Age

5. Conclusions

This study has replicated the approach adopted by Cutler and Huckman (2003) to establish the degree to which new technology, specifically PTCA, substitutes for an older one, in this case CABG in a health care system which is known to have generally low levels of technology diffusion. Using patient specific data over a 15-year follow-up period the mortality and morbidity impacts of PTCA relative to CABG are clearly established. In considering the substitution process using hospital level data and using estimation methods to control for endogeneity an improved empirical specification is suggested in comparison to earlier work. Such improvements quantify the degree to which PTCA has substituted for CABG, as opposed to expanding surgical treatment to the potential patient population. It has been shown, as represented by the data here that by the end of the period of study the UK witnessed degrees of substitution between PTCA for CABG that were of the same magnitude witnessed in the U.S. study. However caution must be exercised when making such direct comparisons on the process of substitution across the CH analysis and this analysis as slightly different time periods are analysed. CH consider 1982 through to 2000 and cover the early establishment of PTCA, while this study considers 1992 through to 2003 and therefore relates to a more mature period of up-take. Notwithstanding this caveat the orders of magnitude in the estimated value of substitution imply that the technology diffusion process is similar in the two countries. The international comparison of operative procedure rates of up-take in this treatment area analysed by the TECH Investigators (2001) concluded that the USA was characterised by early-start and quick up-take, while the UK was characterised by late-start and slow up-take. This current analysis would suggest that, if direct comparison is made to the CH results, consistent with this earlier TECH finding the UK catches up with US levels of substitution with some lag.

While specific productivity calculations are not pursued here clearly more work at this level of investigation is required. Further work to establish the degree of substitution between these technologies in other countries, or applying a similar analysis to other technologies would be of interest. More fundamentally establishing the impact that the regulatory environment and the payment system has on the diffusion pattern of new health care technology is vital to the understanding of the mechanisms through which such technology impacts on health care costs generally.

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