American Economic Review: Papers & Proceedings 2008, 98:2, 345–350
http://www.aeaweb.org/articles.php?doi=10.1257/aer.98.2.345

APPLICATIONS OF RECENT METHODOLOGY IN PROJECT EVALUATION[†]

Using Selection on Observed Variables to Assess Bias from Unobservables when Evaluating Swan-Ganz Catheterization

By Joseph G. Altonji, Todd E. Elder, and Christopher R. Taber*

Distinguishing causal effects from correlations is a key objective of research, regardless of field. Even in medicine, controlled experiments are not always practical or ethical. In economics, and we suspect in most other fields, identification strategies based upon observational data are rarely bulletproof.

In their landmark study, A. F. Connors et al. (1996) use propensity score matching methods with an extremely rich set of demographic characteristics and health status measures to assess whether Swan-Ganz catheterization (C), a widely used device for monitoring intensive care unit (ICU) patients, raises or lowers mortality. In their sample of ICU patients, they find that receiving C within the first 24 hours *raises* mortality rates, casting serious doubt on the value of this procedure for critically ill patients.

Jay Bhattacharya, Azeem M. Shaikh, and Edward Vytlacil (2007, 2008; hereafter, BSV) observe that since C recipients are sicker on many observed dimensions, propensity score matching, which ignores selection on unobservables, might overstate the negative consequences of C. They apply a set of bounds estimators, including an extension of Shaikh and Vytlacil (2004), that incorporates prior information that weekend admission to the hospital is a valid instrument for C. Their bounds include the possibility of a benefit over the first seven days, although their estimates suggest that C has either no effect or a harmful effect after 30 days.[1]

We revisit the issue using the methods of Altonji, Elder, and Taber (2002, 2005; hereafter, AET). First, we use a bivariate probit model to examine the sensitivity of the estimates to assumptions about the amount of selection on unobservables. We find that a correlation of roughly 0.15 between the unobserved determinants of C and mortality at 90 days would be enough to produce the harmful estimated effect when the true effect is zero. Second, we provide lower bound estimates ranging from -0.042 in the short run to -0.005 in the long run for the effect of C on mortality based on the assumption that the degree of selection on observed characteristics is the same as the degree of selection on unobserved characteristics. Given that mortality is caused by many factors that are more or less random at the time C is chosen, equality of selection is unlikely, and so the true mortality effect of C is likely to be larger (i.e., less negative) than the lower bound. Third, we find that selection on unobservables that is 0.8 times as strong as selection on observables could account for the positive probit estimate of the C effect if the true effect is 0. Finally, we discuss a possible extension of the analysis to a heterogeneous treatment effects model. Our main conclusion is that while

[†] *Discussant:* Hidehiko Ichimura, University of Tokyo.

* Altonji: Department of Economics, Yale University, Box 208269, New Haven, CT 06520, and NBER (e-mail: joseph.altonji@yale.edu); Elder: Department of Economics, Michigan State University, 110 Marshall-Adams Hall, East Lansing, MI 48824 (e-mail: telder@msu.edu); Taber: Department of Economics, University of Wisconsin-Madison, 1180 Observatory Dr, Social Sciences Building #6448, Madison, WI 53706, and NBER (e-mail: ctaber@ssc.wisc.edu). We thank Jay Bhattacharya for generously providing us with data for this study and Patrick Kline and Hidehiko Ichimura for helpful comments.

[1] Connors et al. (1996) provided the impetus for two large-scale experimental evaluations of the procedure, as well as several nonexperimental evaluations. The experimental studies find that C has no effect. See BSV for references. In this volume (see p. 357), Qi Li, Jeffrey Racine, and Jeffrey Wooldridge find that Connors et al.'s results are not robust to nonparametric approaches.

it is difficult to find evidence that C lowers mortality in the long run, Connors et al.'s data are not sufficient to draw strong conclusions about the efficacy of C for critically ill patients.

I. Data

We use the data of Connors et al. (1996), which contain detailed information on health status from medical charts and from interviews with patients and proxy respondents. They also provide demographic information and private insurance status. The study considers a number of outcomes in addition to mortality, but we follow BSV and focus on mortality in 7, 90, and 180 days. The estimation sample size is 4,572 patients.[2]

In AET (2008), we report means by C and by mortality status at 60 days. We find that most traits associated with higher mortality rates are also associated with catheterization. The raw difference in mortality rates between those with $C = 1$ and $C = 0$ is 0.038 at 7 days, 0.093 at 90 days, and 0.087 at 180 days. The question is whether the implied adverse effects of C simply represent selection in who is treated. As we have already noted, Connors et al. (1996) find that controlling for observed characteristics reduces, but does not eliminate, the adverse effect of C, and BSV motivate their attention to selection on unobservables by noting the systematic pattern in the observables.

II. The Sensitivity of Probit Estimates of Catheterization to Correlation in Unobservables

Following AET (2005), we assess the sensitivity of conclusions about C to unobserved heterogeneity by computing estimates under alternative assumptions about the degree of correlation in the unobservables that affect the choice of C and Y, where Y indicates death within t days after admission to the ICU. Consider the model

$$(1) \quad C = 1\,(C^* > 0) \equiv 1(\boldsymbol{X'\beta} + u > 0),$$

$$(2) \quad Y = 1\,(Y^* > 0) \equiv 1(\boldsymbol{X'\gamma} + \alpha C + \varepsilon > 0),$$

$$(3) \quad \begin{bmatrix} u \\ \varepsilon \end{bmatrix} \sim N\left(\begin{bmatrix} 0 \\ 0 \end{bmatrix}, \begin{bmatrix} 1 & \rho \\ \rho & 1 \end{bmatrix}\right),$$

where we leave the dependence of the parameters $\boldsymbol{\gamma}$, α, ρ, and ε on t implicit.[3] Since semiparametric identification requires an excluded variable, we treat ((1), (2), (3)) as if it is underidentified by one parameter—ρ. Each column of Table 1 reports estimates of α for specified values of t, with the rows corresponding to assumptions about ρ. Setting $\rho = 0$ corresponds to treating C as exogenous. Consider the case of death within 90 days. When ρ is 0, $\hat{\alpha}$ is equal to 0.231 and the average marginal effect on the mortality probability (ME) is 0.074. This confirms Connors et al.'s (1996) main result that C raises mortality. However, a correlation of 0.1 nearly eliminates the effect in the 90-day case, making α statistically indistinguishable from zero with an ME of only 0.021. A value of ρ equal to 0.2 is enough to shift the estimate to a negative value of -0.033.[4] Note that for mortality at seven days, a value of ρ of 0.1 is sufficient to reverse the sign of α. We obtain similar results if we relax the bivariate normality assumption.[5]

The results in Table 1 show that even a modest value of ρ could eliminate the positive (harmful) effect of C on mortality, but it is not clear what range of values of ρ are plausible. In the next section, we use the degree of selection on the observables as a guide.[6]

[2] To improve comparability, we again follow BSV and exclude patients with chronic obstructive pulmonary disease, cirrhosis, coma, lung cancer, and colon cancer. Our results are not sensitive to this exclusion.

[3] A hazard model would be a natural alternative framework to explore, but a proportional hazards model is not an attractive option because the relative effects of the covariates on the hazard are likely to vary with survival time.

[4] See Paul R. Rosenbaum (1995) or Rosenbaum and Donald Rubin (1983) for examples of this type of sensitivity analysis. Connors et al. (1996) present a related calculation, noting that to explain away their positive estimate of α, one would need an omitted variable that is roughly six times as powerful in predicting mortality as any observed covariate.

[5] We use the semiparametric specification

$$(4) \quad u = \theta + u^*,$$

$$(5) \quad \varepsilon = \theta + \varepsilon^*,$$

where the distribution of θ is unrestricted and u^* and ε^* are independent standard normals, which nests the bivariate probit model. We estimate the model using nonparametric maximum likelihood, as described in James J. Heckman and Burton Singer (1984), treating the distribution of θ as discrete with three points of support for θ.

[6] One could follow BSV and use weekend admission as a determinant of C, but the medical literature is mixed on whether this variable can be excluded from Y. It is not clear

TABLE 1—SENSITIVITY OF ESTIMATES OF SWAN-GANZ TREATMENT EFFECTS TO VARIATION IN THE CORRELATION OF DISTURBANCES IN BIVARIATE PROBIT MODELS

	Dependent variable: mortality		
ρ	7 days	90 days	180 days
0.0	0.137	0.231	0.219
	(0.058)	(0.046)	(0.046)
	[0.025]	[0.074]	[0.071]
0.1	−0.029	0.065	0.053
	(0.058)	(0.046)	(0.045)
	[−0.005]	[0.021]	[0.017]
0.2	−0.195	−0.103	−0.114
	(0.057)	(0.045)	(0.045)
	[−0.036]	[−0.033]	[−0.037]
0.3	−0.363	−0.270	−0.282
	(0.056)	(0.045)	(0.044)
	[−0.067]	[−0.086]	[−0.092]

Notes: Cell entries are estimated Swan-Ganz treatment effects from bivariate probit models restricting ρ to the value given in the first column. Standard errors are in parentheses and marginal effects are in brackets.

III. Estimates of the *C* Effect Using Selection on the Observables to Assess Selection Bias

Consider the linear projection of C^* onto $\mathbf{X}'\boldsymbol{\gamma}$ and ε, where ε is an index of the unobserved factors that determine mortality:

$$\text{(6)} \quad \text{Proj}\,(C^*|\boldsymbol{X}'\boldsymbol{\gamma}, \varepsilon) = \phi_0 + \phi_{\boldsymbol{X}'\boldsymbol{\gamma}}\boldsymbol{X}'\boldsymbol{\gamma} + \phi_\varepsilon\varepsilon.$$

The magnitudes of $\phi_{\boldsymbol{X}'\boldsymbol{\gamma}}$ and ϕ_ε summarize the relative strength of the dependence of C on the observed factors and unobserved factors that determine mortality. AET formalize the idea that "selection on the unobservables is the same as selection on the observables," as

$$\text{(7)} \qquad \phi_{\boldsymbol{X}'\boldsymbol{\gamma}} = \phi_\varepsilon.$$

Roughly speaking, (2) says that the part of Y^* that is related to the observables and the part related to the unobservables have the *same* relationship with C^*. The assumption that there is no selection on unobservables that matter for Y is $\phi_\varepsilon = 0$.

The precise conditions and formal model leading to (7) are given in AET (2002). Roughly speaking, it holds if (a) the elements of $\boldsymbol{X}$ are chosen at random from the full set of factors that determine Y, (b) the number of observed variables $\boldsymbol{X}$ and the number of unobserved variables are large and none of the elements dominates the distribution of C or Y, and (c) a condition holds that implies that the coefficient of the regression of C^* on $Y^* - \alpha C$ is equal to the coefficient of the regression of the part of C^* that is orthogonal to X on the corresponding part of $Y^* - \alpha C$.

These assumptions are unlikely to hold exactly. Although both Y^* and C do depend on a fairly large set of factors, information on medical charts is collected precisely because it is believed to be relevant for assessing health status and guiding treatment. Furthermore, health is a stochastic process, and future shocks (e.g., infection) that lead to mortality are unknown when C is chosen. Consequently, in the Swan-Ganz application, selection on observables is likely to be stronger than selection on unobservables, implying that $0 < \phi_\varepsilon < \phi_{\boldsymbol{X}'\boldsymbol{\gamma}}$. Below, we interpret estimates of α that impose (7) as a lower bound for α and single equation estimates with C treated as exogenous (which impose $\phi_\varepsilon = 0$) as an upper bound.

In the bivariate probit case, (7) may be rewritten as $\rho = cov\,(\boldsymbol{X}'\boldsymbol{\beta}, \boldsymbol{X}'\boldsymbol{\gamma})/var(\boldsymbol{X}'\boldsymbol{\gamma})$ and the inequality restrictions on ϕ_ε correspond to

$$\text{(8)} \qquad 0 \leq \rho \leq \frac{cov(\boldsymbol{X}'\boldsymbol{\beta}, \boldsymbol{X}'\boldsymbol{\gamma})}{var(\boldsymbol{X}'\boldsymbol{\gamma})}.$$

In Table 2, we present MLE estimates of α and *ME* imposing $\rho = cov\,(\boldsymbol{X}'\boldsymbol{\beta}, \boldsymbol{X}'\boldsymbol{\gamma})/var(\boldsymbol{X}'\boldsymbol{\gamma})$. The standard errors assume that (8) holds for the particular $\boldsymbol{X}$ variables that we have. They ignore variation that would arise if the set of $\boldsymbol{X}$ variables is too small for such variation to be nonnegligible. When t is 7, the lower bound estimate of α is −0.231 (0.286) and the corresponding value for *ME* is −0.042, which is a substantial negative effect. Note that $\hat{\rho}$ is 0.221, indicating substantial selection on observables. The lower bound estimates at the other horizons are also negative, although point estimates are not significant and the *ME* are small. In particular, at 180 days the effect is essentially zero. Given that this is a lower bound, the point estimates suggest

that this variable is sufficiently powerful for identification in the Connors et. al. (1996) sample. When we use a linear probability model for Y and use weekend admission to estimate the effect of C by 2SLS, we obtain implausibly large positive estimates with huge standard errors. At 90 days, the marginal effect on mortality is 0.650 (0.296).

TABLE 2—ESTIMATES OF SWAN-GANZ TREATMENT EFFECTS ASSUMING EQUALITY OF SELECTION ON OBSERVABLE AND UNOBSERVABLE DETERMINANTS OF MORTALITY

	Dependent variable: mortality		
Estimate of:	7 days	90 days	180 days
α	−0.231	−0.044	−0.017
	(0.286)	(0.174)	(0.176)
	[−0.042]	[−0.014]	[−0.005]
ρ	0.221	0.165	0.142

Note: Cell entries are estimated S-G treatment effects from bivariate probit models restricting $\rho = cov(\boldsymbol{X'\beta}, \boldsymbol{X'\gamma})/var(\boldsymbol{X'\gamma})$. Standard errors are in parentheses and marginal effects are in brackets.

that C does not substantially lower mortality in the long run. However, the standard errors are large enough that we cannot reject moderate beneficial effects.

The following refinement to our framework is useful for thinking about ρ. Let $\boldsymbol{e}$ be a vector of all unobserved variables that affect mortality and are *known* to doctors when C is chosen. Let u^* be an index of other factors that affect choice of C. For simplicity, assume that they are unrelated to $\boldsymbol{e}$. Let v be an index of other contemporaneous determinants of mortality that doctors do not know about when choosing C and assume they are uncorrelated with $\boldsymbol{e}$ and u^*. Let v^* capture future variables that have an impact on mortality that are unknowable when C is chosen. Then,

$$C^* = \boldsymbol{X'\beta} + \boldsymbol{e'\beta_e} + u^*,$$

$$Y^* = \boldsymbol{X'\gamma} + \boldsymbol{e'\gamma_e} + v + v^*,$$

$$\rho = \frac{cov(\boldsymbol{e'\beta_e}, \boldsymbol{e'\gamma_e})}{var(\boldsymbol{e'\gamma_e} + v + v^*)}.$$

Let ϕ be selection on the unobservables known to doctors when they choose C. Because u^* affects C but not Y^*,

$$\phi \equiv \frac{cov(\boldsymbol{e'\beta_e} + u^*, \boldsymbol{e'\gamma_e})}{var(\boldsymbol{e'\gamma_e})} = \frac{cov(\boldsymbol{e'\beta_e}, \boldsymbol{e'\gamma_e})}{var(\boldsymbol{e'\gamma_e})}.$$

Let $var(\boldsymbol{e'\gamma_e}) = \theta\ var(\boldsymbol{e'\gamma_e} + v + v^*)$, where θ is the fraction of the unobserved determinants of variance in mortality that doctors know about at the time they choose C. Then $\rho = \phi \cdot \theta$. For illustrative purposes, suppose one were to assume that unobserved mortality determinants that doctors know have 0.7 as large an effect on C as the observed mortality determinants. Then ϕ would be $0.7 \cdot cov(\boldsymbol{X'\beta}, \boldsymbol{X'\gamma})/var(\boldsymbol{X'\gamma})$, which equals 0.1155 in the 90-day mortality case because $cov(\boldsymbol{X'\beta}, \boldsymbol{X'\gamma})/var(\boldsymbol{X'\gamma})$ is 0.165. If we assume that θ is 0.5, then $\phi_\varepsilon = \phi \cdot \theta = 0.1155 \cdot 0.5 = 0.0578$. Since $var(\boldsymbol{e'\gamma_e} + v + v^*)$ is normalized to 1 and in the 90 day case $var(\boldsymbol{X'\hat{\gamma}}) = 0.449$, the physicians' R^2 for Y^* is

$$\frac{var(\boldsymbol{X'\gamma}) + \theta}{var(\boldsymbol{X'\gamma}) + 1} = 0.655.$$

We lack the medical expertise and data on physicians' beliefs to speculate much further.

IV. The Relative Amount of Selection on Unobservables Required to Explain the *C* Effect

AET also provide a different, more informal way to use information about selection on the observables as a guide to selection on the unobservables. Consider

$$(9)\quad \frac{E(\varepsilon|C=1) - E(\varepsilon|C=0)}{var(\varepsilon)} = \lambda \frac{E(\boldsymbol{X'\gamma}|C=1) - E(\boldsymbol{X'\gamma}|C=0)}{var(\boldsymbol{X'\gamma})}.$$

The equation says that the relationship between C and the mean of the distribution of the index of unobservables that determine mortality is λ times as strong as the relationship between C and the mean of the observable index $\boldsymbol{X'\gamma}$ after adjusting for variances. The parameter λ is the relative strength of selection on unobservables and selection on observables. Under the same assumptions as (7), $\lambda = 1$, which corresponds to the case of equality of selection on observables and unobservables.

A natural way to assess the strength of the evidence that C raises mortality is to ask how large λ has to be for bias to account for the entire estimate of α under the null that α is zero. Following AET, we ignore the fact that the model for the binary variable Y is a probit, and treat α as if it were estimated by a regression of the latent variable Y^* on X and C. Let $\boldsymbol{X'\beta}$ and $\widetilde{C}$

represent the predicted value and residuals of a regression of C on X. Then

$$Y^* = \alpha\widetilde{C} + \boldsymbol{X}'[\boldsymbol{\gamma} + \alpha\boldsymbol{\beta}] + \varepsilon.$$

If the bias in a probit is close to the bias in OLS applied to the model above for Y^*, then the fact that $\widetilde{C}$ is orthogonal to $\boldsymbol{X}$ leads to the usual bias formula:

$$\text{plim}\ \hat{\alpha} \simeq \alpha + \frac{cov(\widetilde{C},\varepsilon)}{var(\widetilde{C})}$$

$$= \alpha + \frac{var(C)}{var(\widetilde{C})}[E(\varepsilon|C=1) - E(\varepsilon|C=0)].$$

Given a value of λ, we can use (9) and an estimate of $E(\boldsymbol{X}'\boldsymbol{\gamma}|C=1) - E(\boldsymbol{X}'\boldsymbol{\gamma}|C=0)$ to estimate $E(\varepsilon|C=1) - E(\varepsilon|C=0)$, and then use the equation above to estimate the bias. Under the null hypothesis that C has no effect, we can consistently estimate $\boldsymbol{\gamma}$ and thus $E(\boldsymbol{X}'\boldsymbol{\gamma}|C)$ from a probit after imposing $\alpha = 0$. When $var(\varepsilon)$ is very large relative to $var(\boldsymbol{X}'\boldsymbol{\gamma})$, what one can learn is limited unless one is confident in the choice of λ, because even a small shift in $(E(\varepsilon|C=1) - E(\varepsilon|C=0))/var(\varepsilon)$ is consistent with a large bias in α. But this is also the circumstance with the greatest potential for large bias.

The results for mortality at various time horizons are in Table 3. In the 90-day case, the (unreported) estimate of $(E(\boldsymbol{X}'\boldsymbol{\gamma}|C=1) - E(\boldsymbol{X}'\boldsymbol{\gamma}|C=0)/var(\boldsymbol{X}'\boldsymbol{\gamma})$ is 0.211, which under (9) with $\lambda = 1$ implies 0.211 as an estimate of $E(\varepsilon|C=1) - E(\varepsilon|C=0)$ (recall that $var(\varepsilon) = 1$). Multiplying by $var(C)/var(\widetilde{C})$ yields a bias reported in the table of 0.288 (0.056). The unconstrained estimate of α is 0.231 (0.046), and the bottom row of the table reports the ratio $\hat{\alpha}/\{[var(C)/var(\widetilde{C})]\ [E(\varepsilon|C=1) - E(\varepsilon|C=0)]\}$, which in the 90-day case is 0.231 / 0.288, or 0.801. That is, one can attribute the entire positive C effect to bias if the normalized shift in the distribution of the unobservables is 0.801 as large as the shift in the observables ($\lambda = 0.801$). We would not want to rule out such a possibility, although we suspect that the true value of λ is lower for reasons discussed above. At seven days, the ratio of selection on unobservables relative to selection on observables need only be 0.289 to explain away the positive mortality estimate.

Table 3—The Amount of Selection of Unobservables Relative to Selection on Observables Required to Attribute the Entire Swan-Ganz Effect to Selection Bias

	Dependent variable: mortality		
	7 days	90 days	180 days
Mean of outcome	0.136	0.419	0.475
Univariate probit estimate	0.137 (0.058) [0.025]	0.231 (0.046) [0.074]	0.219 (0.046) [0.071]
Implied bias under equality of selection	0.475 (0.111)	0.288 (0.056)	0.288 (0.056)
Ratio of estimate to bias	0.289	0.801	0.759

Notes: Cell entries are estimated Swan-Ganz treatment effects from bivariate probit models restricting ρ to the value given in the first column. Standard errors are in parentheses and marginal effects are in brackets.

V. Heterogenous Treatment Effects

A disadvantage of our analysis is that it implicitly assumes that the response of Y^* to C does not vary with X and ε. Connors et al. (1996) provide some evidence that the adverse effects of C on 30-day mortality vary with observed patient characteristics and that they are largest for patients with a relatively high predicted two-month survival probability. AET (2002) provide a speculative discussion of the possibility of extension of their methods to consider treatment heterogeneity. A threshold crossing model with heterogeneous effects may be written as

$$C^* = \boldsymbol{X}'\boldsymbol{\beta} + u, \qquad C = 1(C^* > 0),$$

$$Y_c^* = \boldsymbol{X}'\boldsymbol{\gamma_c} + \varepsilon_c,$$

$$Y_{nc}^* = \boldsymbol{X}'\boldsymbol{\gamma_{nc}} + \varepsilon_{nc},$$

$$Y = 1(C \cdot Y_c^* + (1-C)Y_{nc}^* > 0),$$

where the latent variable Y_c^* determines Y if $C = 1$ and Y_{nc}^* determines Y otherwise. Apart from an intercept shift, our previous specification imposes $\boldsymbol{\gamma_c} = \boldsymbol{\gamma_{nc}}$ and $\varepsilon_c = \varepsilon_{nc}$. However,

doctors choose C at least in part to minimize mortality, so if they know $\boldsymbol{\gamma_c}$ and $\boldsymbol{\gamma_{nc}}$, then $\boldsymbol{X'\beta}$ is negatively related to $[\boldsymbol{X'\gamma_c} - \boldsymbol{X'\gamma_{nc}} + \varepsilon_c - \varepsilon_{nc}]$. The basic reasoning and conditions similar to those underlying (7) lead to

$$\frac{cov(\boldsymbol{X'\beta},\boldsymbol{X'\gamma}_c)}{var(\boldsymbol{X'\gamma}_c)} = \frac{cov(u,\varepsilon_c)}{var(\varepsilon_c)} \equiv \rho_{u\varepsilon_c},$$

$$\frac{cov(\boldsymbol{X'\beta},\boldsymbol{X'\gamma}_{nc})}{var(\boldsymbol{X'\gamma}_{nc})} = \frac{cov(u,\varepsilon_{nc})}{var(\varepsilon_{nc})} \equiv \rho_{u\varepsilon_{nc}},$$

$$\frac{cov(\boldsymbol{X'\gamma}_c,\boldsymbol{X'\gamma}_{nc})}{var(\boldsymbol{X'\gamma}_{nc})} = \frac{cov(\varepsilon_c,\varepsilon_{nc})}{var(\varepsilon_{nc})} \equiv \rho_{\varepsilon_c\varepsilon_{nc}}.$$

Since there is clear evidence that the sickest patients receive C, in light of our discussion of bounds above, one might want to impose

$$\frac{cov(\boldsymbol{X'\beta},\boldsymbol{X'\gamma}_c)}{var(\boldsymbol{X'\gamma}_c)} > \frac{cov(u,\varepsilon_c)}{var(\varepsilon_c)} \equiv \rho_{u\varepsilon_c} > 0.$$

In addition, unless there are very large interactions,

$$\frac{cov(\boldsymbol{X'\beta},\boldsymbol{X'\gamma}_{nc})}{var(\boldsymbol{X'\gamma}_{nc})} > \frac{cov(u,\varepsilon_{nc})}{var(\varepsilon_{nc})} \equiv \rho_{u\varepsilon_{nc}} > 0.$$

We do not have a presumption about whether $cov(\boldsymbol{X'\gamma_c},\boldsymbol{X'\gamma_{nc}})/var(\boldsymbol{X'\gamma_{nc}})$ would be greater than or less than $cov(\varepsilon_c,\varepsilon_{nc})/var(\varepsilon_{nc})$, although both are very likely to be positive. Thus, $\rho_{\varepsilon_c\varepsilon_{nc}}$ would have to be estimated or a sensitivity analysis conducted. It would be interesting in future research to carefully explore the possibility of using these restrictions to help bound estimates of γ_c and γ_{nc} in a way that is analogous to our use of (8) to obtain bounds in the homogeneous effects case.

VI. Conclusions

We estimate the effects of Swan-Ganz catheterization using the methods of Altonji, Elder, and Taber (2002, 2005). Our main conclusion is that while it is difficult to find evidence that C lowers mortality in the long run, Connors et al.'s data are not sufficient to draw strong conclusions about the efficacy of C for critically ill patients.

REFERENCES

Altonji, Joseph G., Todd E. Elder, and Christopher R. Taber. 2002. "Selection on Observed and Unobserved Variables: Assessing the Effectiveness of Catholic Schools." http://www.econ.yale.edu/~jga22/website/research_papers/cathrs31te.pdf.

Altonji, Joseph G., Todd E. Elder, and Christopher R. Taber. 2005. "Selection on Observed and Unobserved Variables: Assessing the Effectiveness of Catholic Schools." *Journal of Political Economy*, 113(1): 151–84.

Altonji, Joseph G., Todd E. Elder, and Christopher R. Taber. 2008. "Evaluating Swan-Ganz Catheterization Treatment Effects." Unpublished.

Bhattacharya, Jay, Azeem Shaikh, and Edward Vytlacil. 2007. "Treatment Effect Bounds: An Application to Swan-Ganz Catheterization." Unpublished.

Bhattacharya, Jay, Azeem Shaikh, and Edward Vytlacil. 2008. "Treatment Effect Bounds: An Application to Swan-Ganz Catheterization." *American Economic Review*, 98(2): 351–56.

Connors, A. F., T. Speroff, N. V. Dawson, C. Thomas, F. E. Harrell, Jr., D. Wagner, N. Desbiens, et al. 1996. "The Effectiveness of Right Heart Catheterization in the Initial Care of Critically Ill Patients." *Journal of the American Medical Association*, 276(11): 889–97.

Heckman, James, and Burton Singer. 1984. "A Method for Minimizing the Impact of Distributional Assumptions in Econometric Models for Duration Data." *Econometrica*, 52(2): 271–320.

Li, Qi, Jeffrey S. Racine, and Jeffrey M. Wooldridge. 2008. "Estimating Average Treatment Effects with Continuous and Discrete Covariates: The Case of Swan-Ganz Catheterization." *American Economic Review*, 98(2): 357–62.

Rosenbaum, Paul R. 1995. *Observational Studies*: *Series in Statistics*. New York: Springer.

Rosenbaum, Paul R., and Donald Rubin. 1983. "Assessing Sensitivity to an Unobserved Binary Covariate in an Observational Study with Binary Outcome." *Journal of the Royal Statistical Society Series B:* 45(2): 212–18.

Shaikh, Azeem M., and Edward Vytlacil. 2007. "Limited Dependent Variables Models and Bounds on Treatment Effects." Unpublished.

This article has been cited by:

1. Qi Li, , Jeffrey S. Racine, , Jeffrey M. Wooldridge. 2008. Estimating Average Treatment Effects with Continuous and Discrete Covariates: The Case of Swan-Ganz CatheterizationEstimating Average Treatment Effects with Continuous and Discrete Covariates: The Case of Swan-Ganz Catheterization. *American Economic Review* **98**:2, 357-362. [Citation] [View PDF article] [PDF with links]