Published in final edited form as:
J Vasc Surg. 2013 March ; 57(3): 627–634. doi:10.1016/j.jvs.2012.09.036.

Effect of hospital level variation in the use of carotid artery stenting versus carotid endarterectomy on perioperative stroke and death in asymptomatic patients

Theodore H. Yuo, MD[1,2], **Howard S. Degenholtz, PhD**[2,3], **Rabih A. Chaer, MD**[1], **Kevin L. Kraemer, MD, MSc**[2,3,4], and **Michel S. Makaroun, MD**[1]

[1]Division of Vascular Surgery, University of Pittsburgh School of Medicine

[2]RAND-University of Pittsburgh Health Institute, Pittsburgh, PA

[3]Department of Health Policy and Management, University of Pittsburgh Graduate School of Public Health, Pittsburgh, PA

[4]Department of Medicine, University of Pittsburgh School of Medicine

Abstract

Objectives—Perioperative stroke and death (PSD) is more common after carotid artery stenting (CAS) than after carotid endarterectomy (CEA) in symptomatic patients, but it is unclear if this is also true in asymptomatic patients. Further, use of both CEA and CAS varies geographically, suggesting possible variation in outcomes. We compared odds of PSD after CAS and CEA in asymptomatic patients to determine the impact of this variation.

Design of Study—We identified CAS and CEA procedures and hospitals where they were performed in 2005–2009 California hospital discharge data. Preoperative symptom status and medical comorbidities were determined using administrative codes. We compared PSD rates after CAS and CEA using logistic regression and propensity score matching. We quantified hospital level variation in the relative utilization of CAS by calculating hospital-specific probabilities of CAS use among propensity score matched patients. We then calculated a weighted average for each hospital and used this as a predictor of PSD.

Results—We identified 6,053 CAS and 36,524 CEA procedures that treated asymptomatic patients in 278 hospitals. PSD occurred in 250 CAS and 660 CEA patients, yielding unadjusted PSD rates of 4.1% and 1.8%, respectively (P<.001). Compared with CAS patients, CEA patients were more likely to be older than 70 (66% vs. 62%, P<.001), but less likely to have 3 or more Elixhauser comorbidities (37% vs. 39%, P<.001). Multivariate models demonstrated that CAS was associated with increased odds of PSD (OR 1.865, 95% CI 1.373–2.534, P<.001). Estimation of average treatment effects based on propensity scores also demonstrated 1.9% increased probability of PSD with CAS (P<.001). The average probability of receiving CAS across all hospitals and strata was 13.8%, but the inter-quartile range was 0.9%–21.5%, suggesting significant hospital level variation. In univariate analysis, patients treated at hospitals with higher CAS utilization had higher odds of PSD as compared to patients in hospitals that performed CAS less (OR 2.141, 95%

Contact information: Theodore H. Yuo, MD, 200 Lothrop Street, PUH A-1011, Pittsburgh, PA 15213, yuoth@upmc.edu.

Presented as a podium presentation at the Society for Clinical Vascular Surgery 2012 Symposium, March 14-17, 2012, Las Vegas, NV

CI 1.328–3.454, P=.002). Multivariate analysis did not demonstrate this effect, but again demonstrated higher odds of PSD after CAS (OR 1.963, 95% CI 1.393–2.765, P<.001).

Conclusions—CEA has lower odds of PSD compared to CAS in asymptomatic patients. Increased utilization of CAS at the hospital level is associated with increased odds of PSD among asymptomatic patients, but this effect appears to be related to generally worse outcomes after CAS as compared to CEA.

INTRODUCTION

Severe carotid artery stenosis is related to stroke, and carotid endarterectomy (CEA) is efficacious in both secondary[1,2] and primary[3,4] stroke prevention when compared to best medical therapy. Carotid artery stenting (CAS) is a newer modality for stroke prevention, but randomized controlled trials (RCTs) have not consistently shown equivalence between CAS and CEA [5,6,7,8,9,10]. Some authors have argued that for certain high-risk patients, CAS is equivalent to CEA[6], but multiple RCTs have shown that CAS has higher perioperative stroke and death (PSD) rates in symptomatic patients[7,8,9]. In asymptomatic patients, the comparison between CEA and CAS is less clear, with RCTs failing to find a significant difference in this subgroup[6, 10]. Some observational studies have not found a difference between CAS and CEA[11,12], while others show CEA to be superior[13,14]. Meanwhile, others have emphasized the use of best medical therapy alone for asymptomatic patients who cannot undergo CEA safely[15], and some have suggested that all asymptomatic patients should be treated with only best medical therapy and undergo neither CEA nor CAS[16]. Despite these contradictory findings, use of CAS is expanding, apparently at the expense of CEA[17]. In addition, there is wide variability in the use of both carotid revascularization (CR) techniques across the country[17,18]. Some authors have proposed variation in physician or hospital “enthusiasm” for CAS to be a key driver for this variability[17], but the clinical effect of this variation is not clear, as variation by itself does not address the issue of appropriateness[19]. We have two aims for our study. First, we sought to compare patient and hospital level PSD outcomes after CAS versus CEA in an administrative database to compare the rate of perioperative events after the two procedures. Second, we sought to determine if variation in relative hospital utilization of CAS is linked to PSD in asymptomatic patients after any carotid revascularization.

METHODS

Data source

After Institutional Review Board approval from the University of Pittsburgh, we obtained de-identified patient discharge data from the California Office for Statewide Health Planning and Development (OSHPD) for the years 2005 through 2009. The state of California is the most populous state in the United States, and as such, provides a convenient and economical single data source to obtain administrative data.

All patients discharged from a hospital in California, excluding federally funded hospitals like Veterans Affairs or military hospitals, are entered into an OSHPD database. Due to the de-identification process, variables are sometimes masked in order to ensure anonymity. International Classification of Diseases, 9th Revision, Clinical Modification (ICD-9-CM) procedure and diagnosis codes are included with each record along with a “present on admission” (POA) flag that we used to discriminate neurologically symptomatic from asymptomatic patients and to define PSD[11]. We first identified patients who underwent CR, as defined by ICD-9-CM procedure codes 38.12 and 00.63. Then, we assessed if the admitting or diagnosis codes, in any position, were associated with hemispheric cerebral ischemia or ophthalmic artery occlusion or embolism (Table 1). If the codes were POA, the

patient was identified as being symptomatic. Patients without such codes, or those where the codes were not POA, were defined as being asymptomatic. PSD was defined as the combined endpoint of death being the discharge disposition of the hospitalization or the presence of ICD-9 codes that suggested stroke, hemiplegia, or other manifestations of cerebral infarction that were not POA[11]. ICD-9 codes have demonstrated good specificity and positive predictive value for cardiovascular risk factors[20]. In addition, every discharge is also associated with a unique hospital code, which facilitates analysis at the hospital level.

Patient population

Using ICD-9-CM codes, we identified CAS and CEA procedures and hospitals where they were performed, along with preoperative symptom status. ICD-9-CM codes also provided evidence for patient comorbidities, which were then identified using the method of Elixhauser[21]. Records from pediatric patients and prisoners were excluded, in accordance with our submission to our local IRB.

Statistical analysis

We tested two null hypotheses: (1) Perioperative stroke and death are equally common after CEA and CAS among asymptomatic patients, adjusting for observable patient characteristics. (2) Perioperative stroke and death after carotid revascularization (both CEA and CAS) are unaffected by a hospital's relative utilization of CAS, adjusting for observable patient and hospital characteristics.

Estimates of the effect of CAS versus CEA on PSD were obtained using logistic regression on all available records of asymptomatic patients. Multiple models were generated, including single level models that assumed independence of the observations and multilevel models with random intercepts for each hospital that accounted for the nesting of observations within hospitals. Covariates were chosen using backwards elimination for dichotomous variables and Akaike information criterion, Bayesian information criterion, and the likelihood ratio test to determine the importance of the year that the procedure was performed. Patient age was retained to maintain clinical validity of the models. We also utilized a propensity score matching technique to examine the average causal treatment effect of CAS treatment. The propensity score is the probability of receiving CAS, as opposed to CEA, for a patient with specific prognostic factors that are available in the database. We computed the propensity score by using a probit model, with the dependent model being the receipt of CAS, and the independent variables being patient age, gender, ethnic background, and Elixhauser variables. Patients are then grouped into strata of similar propensity score with upper and lower limits constructed to ensure that covariates were similarly distributed and the average propensity score was approximately equal for CEA and CAS patients within strata. Finally, we used stratification matching, in which outcomes after CEA and CAS were compared within each stratum and then averaged across all strata, in order to identify the effect of CAS on PSD[22].

We then investigated the effect of the relative level of hospital utilization of CAS on PSD. We reasoned that the proportion of patients at a hospital that underwent CAS, as opposed to CEA, could serve as a quantitative measure of a hospital's preference for CAS. We used the strata from the propensity scores defined above and calculated the proportion that underwent CAS for each hospital within each stratum. Within strata, patients are similar based on observed characteristics, and if all hospitals treated such patients comparably, each hospital would have similar proportions that underwent CAS. Differences between hospitals therefore can be thought of as a form of geographic variation. We then calculated a weighted average across all strata for the hospital. This weighted average served as a measure of relative CAS utilization for the hospital.

We considered using the unadjusted proportion of CAS procedures as our metric for utilization, but this would unduly penalize hospitals that treated patients with more comorbidities, as those patients appeared more likely to be treated with CAS and independently also suffer PSD.

We then repeated logistic regression to estimate the association of different levels of relative CAS utilization on PSD using multilevel logistic regression models with random slopes and intercepts for each hospital. Building on our previous models, we included hospital level variables including the relative utilization metric, CAS and CEA volume, and number of patient discharges. Interaction terms between hospital level CAS utilization and CAS were also evaluated.

Data and statistical analysis was performed with StataMP (Version 11.2, StataCorp LP, College Station, TX).

RESULTS

Patient characteristics

During the analysis period from 2005–2009, a total of 47,598 discharge records were identified that recorded CEA or CAS. We removed records of patients who were under 18 years old, prisoners, or who underwent both CEA and CAS during their hospitalization (195 records removed). We then focused on asymptomatic patients only (4,826 records removed), yielding an analytic file of 42,577 CR records: 6,053 CAS and 36,524 CEA (Figure 1). In this group, the number of annual CEA was at its highest in 2005 and then dropped in each consecutive year, for a total 5-year reduction of 16% by 2009. Meanwhile, there was little net change in the number of annual CAS between 2005 and 2009 (Figure 2). Compared with CAS patients, CEA patients were more likely to be older than 70 (62% vs. 66%, P<.001), but were less likely to have 3 or more Elixhauser comorbidities (37% vs. 39%, P<.001) or carry a diagnosis of congestive heart failure (CHF) (11.5% vs. 7.5%, P<.001). Selected demographics and comorbidities are shown in Table 2 for patients with unmasked data. Of note, multivariate regression models used only patients with unmasked data (30,317 CR records: 3,476 CAS and 26,841 CEA).

Association of CAS with perioperative stroke and death

PSD occurred after 250 CAS and 660 CEA, yielding unadjusted PSD rates of 4.1% and 1.8%, respectively (P<.001), which corresponds to univariate logistic regression that also estimated a higher odds of PSD among asymptomatic patients with CAS (OR 2.341, 95% CI 2.019–2.715, P<.001). In a single level logistic regression that adjusted for demographics, including race, age, and gender, and clinical characteristics, as defined by Elixhauser comorbidities, CAS was still significantly associated with PSD (OR 1.948, 95% CI 1.1.583–2.398, P<.001). The effect of CAS remained significant after stepwise backward-selection estimation for covariates, (OR 1.960, 95% CI 1.1.595–2.409, P<.001). Nested models with random intercepts and random coefficients that account for the relationship of patients within hospitals also demonstrate increased odds of PSD associated with CAS (OR 1.865, 95% CI 1.373–2.534, P<.001). Regression coefficients for the final nested model are presented in Table 3.

Death occurred after 181 CEA and 86 CAS, yielding raw death rates of 0.5% and 1.4%, respectively (P<.001). Perioperative stroke occurred after 550 CEA and 191 CAS, for rates of 1.5% and 3.2%, respectively (P<.001).

We also investigated the impact of year on PSD rates. In univariate analysis, CAS PSD rates appeared to increase from 3.5% in 2005 to 6.0% in 2009 (P=.002), while CEA PSD rates fell

from 2.0% to 1.8% for the same time period (P=NS) using a two sample test of proportions (Table 4). However, in multivariate analysis, the year of procedure did not improve model fit and was not included in the final model.

Propensity score (PS) matching results

Blocks of PS matched patients were generated in order to balance the demographics variables and Elixhauser comorbidities that were used as covariates. Ten blocks were created (Table 5). As designed, patients within blocks were better balanced on their demographic and observed clinical characteristics than in the original population, as illustrated for block 3 (Table 6). Estimates of the average effect of CAS using stratification matching again demonstrated that CAS was inferior to CEA; patients treated with CAS had a 1.9% higher probability of PSD compared to patients treated with CEA (P<.001).

Hospital CAS utilization analysis results

A total of 278 hospitals were identified that performed at least one CEA or CAS from 2005–2009, of which 5 did not have a utilization metric calculated due to a combination of very low volumes and missing demographic information, leaving 273 hospitals. Our metric for hospital level relative utilization of CAS, which is the average probability of performing CAS versus CEA across all propensity score matched strata, weighted by the number of procedures in each stratum, had an average value of 13.8%, median value of 5.2%, and interquartile range of 0.9%–21.5% (Figure 3). Most hospitals that perform carotid revascularization concentrate on CEA, with CAS being limited to a few centers. Univariate logistic regression demonstrated statistically significant increased odds of PSD with increasing hospital level utilization (OR 2.141, 95% CI 1.328–3.454, P=.002). Multivariate regression analysis was also performed, controlling for patient characteristics associated with PSD from the previous models. We also controlled for hospital level characteristics, including CEA and CAS volume, the total number of hospital discharges, and accounting for the clustering of observations within hospitals. The effect of hospital level utilization on PSD became non-significant (OR 1.407, 95% CI 0.497–3.980, P=.520), while the effect of CAS had approximately the same magnitude as in previous models (OR 1.963, 95% CI 1.393–2.765, P<.001).

In the final model, we failed to find evidence for a volume-outcome relationship at the hospital level for neither CEA (OR 0.974 for an increase in 100 CEA procedures, 95% CI 0.900–1.053, P=.502) nor CAS (OR 0.805 for an increase in 100 CAS procedures, 95% CI 0.615–1.054, P=.114). The interaction term between our metric for hospital utilization and use of CAS was also not significant (P=.581) and was dropped from the final model. Regression coefficients for the final model are presented in Table 7.

DISCUSSION

We have three principal findings. First, among the asymptomatic patients we identified, we found a PSD rate of 4.1% after CAS and 1.8% after CEA. Current guidelines for CEA in asymptomatic patients[15, 23,24] call for a PSD rate less than 3% in patients with asymptomatic carotid disease; CEA in our administrative data set meets this standard, while CAS does not. Second, after controlling for differences in patients treated with CEA and CAS with multivariate logistic regression techniques, we still found that CAS was associated with approximately twice the odds of PSD as compared to CEA. We supplemented this analysis with a propensity score matching technique to minimize the risk of confounding based on observed data and again found a statistically significant increased probability of PSD with CAS. Third, we found a univariate relationship between hospital level utilization of CAS and PSD, with increased relative utilization of CAS associated with increased PSD among

asymptomatic patients undergoing CR; this relationship is not significant in multivariate analysis.

Our first two findings regarding the higher PSD rate for CAS as compared to CEA have been reported previously by other investigators using different data sets and statistical methodologies[13,14]. In addition, the absolute PSD rate after CEA of 1.8% for all asymptomatic patients is in line with the approximately 1.4% PSD rate in both observational[25] and a recent contemporary RCT[10]. Given the all-inclusive nature of the OSHPD data, these rates likely reflect the true CEA PSD rates in the community in the time period of interest and the real world effectiveness of CEA. Meanwhile, the observed PSD rate for CAS was 4.1%, which is certainly above the 3% upper limit prescribed for CEA, and above the guidelines set by prominent insurance groups[26] for management of asymptomatic carotid disease. While such a rate may be acceptable for symptomatic patients, it is important to recognize that our analysis was restricted to asymptomatic patients in whom initial perioperative risk of stroke must be balanced against the modest stroke prevention benefit that operating on an asymptomatic patient implies[16]. By itself, this is noteworthy, and consistent with recent SVS guidelines that suggest that CAS should not be offered to asymptomatic patients. The higher odds of PSD persists after adjustment for the slightly higher, and clinically small, burden of medical comorbidities of patients who were treated with CAS at the time, as was described by other investigators[13,14].

Our third finding is the most novel; the difference in outcomes in CEA and CAS drives differences in overall carotid revascularization outcomes. Other published reports have detailed geographic variation in the use of CAS versus CEA[17,18] and have linked some of this variation to physician specialty[27], but have yet to describe a clinical effect from this variation. To our knowledge, we are the first to suggest that the increased hospital level utilization of CAS is associated with worse outcomes in a univariate analysis. However, in multivariate analysis, the effect of variation in hospital level utilization is diminished, suggesting that the hospital's utilization level does not affect the likelihood of the PSD outcome; rather, it is the generally worse outcomes after CAS that account for the effect seen in the univariate analysis.

Beyond the choice of CAS versus CEA, our findings must be interpreted in the light of recent studies that suggest that for many asymptomatic patients, especially those with short life expectancies, best medical therapy may be the optimal treatment modality[28]. Approximately 90% of patients who underwent carotid revascularization in this analysis appeared to be asymptomatic. Further, current Society for Vascular Surgery guidelines emphasize the importance of a minimum life expectancy of at least three years in order for there to be a benefit in asymptomatic patients undergoing CEA[15]. We look forward to future RCTs that may address the choice of CEA, CAS, and BMT in asymptomatic patients[29].

As with other observational studies using administrative databases, our analysis rests on the accuracy of the diagnosis codes, and significantly different coding of comorbidities or complications after CEA or CAS could affect our analysis. Further, residual confounding may still be present, despite our efforts. Clinically important variables like degree of stenosis, anatomy, or surgical history were not available to us and could not be included in our models. Similarly, our ability to distinguish symptomatic from asymptomatic patients is limited by the nature of administrative data. Finally, due to the structure of the database, longitudinal patient identifiers are not available, and so only outcomes related to the initial hospital stay can be analyzed.

CONCLUSIONS

In this analysis of a recent administrative database, odds of PSD after CAS is twice as high as odds of PSD after CEA. Furthermore, hospital level utilization of CAS is associated with PSD, but this effect appears to be fully explained by generally worse outcomes after CAS as compared to CEA. These results support recent guidelines that suggest that in asymptomatic patients with appropriate anatomy and lesion characteristics, CEA should be the first line therapy with best medical therapy offered to patients who are poor surgical risks or have short life expectancies. The role of CAS in this patient population needs further study before it can be recommended for broader use.

Acknowledgments

We wish to acknowledge Anthony Fabio, PhD from the Clinical and Translational Science Institute at the University of Pittsburgh for his invaluable statistical assistance (National Institutes of Health grants UL1RR024153 and UL1TR000005). Dr. Yuo was supported by a grant from the Agency for Health Care Research and Quality (grant T32HS019486-01).

References

1. North American Symptomatic Carotid Endarterectomy Trial Collaborators. Beneficial effect of carotid endarterectomy in symptomatic patients with high-grade carotid stenosis. N Engl J Med. 1991 Aug 15; 325(7):445–53. [PubMed: 1852179]
2. European Carotid Surgery Trialists' Collaborative Group. MRC European Carotid Surgery Trial: interim results for symptomatic patients with severe (70–99%) or with mild (0–29%) carotid stenosis. Lancet. 1991; 337:1235–43. [PubMed: 1674060]
3. Executive Committee for the Asymptomatic Carotid Atherosclerosis Study. Endarterectomy for asymptomatic carotid artery stenosis. JAMA. 1995; 273:1421–1428. [PubMed: 7723155]
4. Halliday A, Mansfield A, Marro J, Peto C, Peto R, Potter J, et al. MRC Asymptomatic Carotid Surgery Trial (ACST) Collaborative Group. Prevention of disabling and fatal strokes by successful carotid endarterectomy in patients without recent neurological symptoms: randomised controlled trial. Lancet. 2004 May 8; 363(9420):1491–502. [PubMed: 15135594]
5. Endovascular versus surgical treatment in patients with carotid stenosis in the Carotid and Vertebral Artery Transluminal Angioplasty Study (CAVATAS): a randomised trial. Lancet. 2001 Jun 2; 357(9270):1729–37. [PubMed: 11403808]
6. Yadav JS, Wholey MH, Kuntz RE, Fayad P, Katzen BT, Mishkel GJ, et al. Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy Investigators. Protected carotid-artery stenting versus endarterectomy in high-risk patients. N Engl J Med. 2004 Oct 7; 351(15):1493–501. [PubMed: 15470212]
7. Mas JL, Chatellier G, Beyssen B, Branchereau A, Moulin T, Becquemin JP, et al. EVA-3S Investigators. Endarterectomy versus stenting in patients with symptomatic severe carotid stenosis. N Engl J Med. 2006 Oct 19; 355(16):1660–71. [PubMed: 17050890]
8. Ringleb PA, Allenberg J, Brückmann H, Eckstein HH, Fraedrich G, Hartmann M, et al. SPACE Collaborative Group. 30 day results from the SPACE trial of stent-protected angioplasty versus carotid endarterectomy in symptomatic patients: a randomised non-inferiority trial. Lancet. 2006 Oct 7; 368(9543):1239–47. [PubMed: 17027729]
9. Ederle J, Dobson J, Featherstone RL, Bonati LH, van der Worp HB, de Borst GJ, et al. International Carotid Stenting Study investigators. Carotid artery stenting compared with endarterectomy in patients with symptomatic carotid stenosis (International Carotid Stenting Study): an interim analysis of a randomised controlled trial. Lancet. 2010 Mar 20; 375(9719):985–97. [PubMed: 20189239]
10. Brott TG, Hobson RW 2nd, Howard G, Roubin GS, Clark WM, Brooks W, et al. CREST Investigators. Stenting versus endarterectomy for treatment of carotid-artery stenosis. N Engl J Med. 2010 Jul 1; 363(1):11–23. [PubMed: 20505173]

11. Giacovelli JK, Egorova N, Dayal R, Gelijns A, McKinsey J, Kent KC. Outcomes of carotid stenting compared with endarterectomy are equivalent in asymptomatic patients and inferior in symptomatic patients. J Vasc Surg. 2010 Oct.524:906–13. 913.e1–4. [PubMed: 20620010]
12. CARESS Steering Committee. Carotid revascularization using endarterectomy or stenting systems (CARESS): phase I clinical trial. J Endovasc Ther. 2003 Dec; 10(6):1021–30. [PubMed: 14723574]
13. McPhee JT, Schanzer A, Messina LM, Eslami MH. Carotid artery stenting has increased rates of postprocedure stroke, death, and resource utilization than does carotid endarterectomy in the United States, 2005. J Vasc Surg. 2008 Dec; 48(6):1442–50. 1450.e1. [PubMed: 18829236]
14. Sidawy AN, Zwolak RM, White RA, Siami FS, Schermerhorn ML, Sicard GA. Outcomes Committee for the Society for Vascular Surgery. Risk-adjusted 30-day outcomes of carotid stenting and endarterectomy: results from the SVS Vascular Registry. J Vasc Surg. 2009 Jan; 49(1):71–9. [PubMed: 19028045]
15. Ricotta JJ, Aburahma A, Ascher E, Eskandari M, Faries P, Lal BK. Updated Society for Vascular Surgery guidelines for management of extracranial carotid disease. J Vasc Surg. 2011 Sep; 54(3):e1–e31. [PubMed: 21889701]
16. Abbott AL. Medical (nonsurgical) intervention alone is now best for prevention of stroke associated with asymptomatic severe carotid stenosis: results of a systematic review and analysis. Stroke. 2009 Oct; 40(10):e573–83. [PubMed: 19696421]
17. Goodney PP, Travis LL, Malenka D, Bronner KK, Lucas FL, Cronenwett JL, Goodman DC, Fisher ES. Regional variation in carotid artery stenting and endarterectomy in the Medicare population. Circ Cardiovasc Qual Outcomes. 2010 Jan 1; 3(1):15–24. [PubMed: 20123667]
18. Patel MR, Greiner MA, DiMartino LD, Schulman KA, Duncan PW, Matchar DB, Curtis LH. Geographic variation in carotid revascularization among Medicare beneficiaries, 2003–2006. Arch Intern Med. 2010 Jul 26; 170(14):1218–25. [PubMed: 20660840]
19. Birkmeyer JD. Primer on Geographic Variation in Health Care. Eff Clin Pract. 2001; 4(5):232–3.
20. Birman-Deych E, Waterman AD, Yan Y, Nilasena DS, Radford MJ, Gage BF. Accuracy of ICD-9-CM codes for identifying cardiovascular and stroke risk factors. Med Care. 2005 May; 43(5):480–5. [PubMed: 15838413]
21. Quan H, Sundararajan V, Halfon P, Fong A, Burnand B, Luthi JC, Saunders LD, Beck CA, Feasby TE, Ghali WA. Coding algorithms for defining comorbidities in ICD-9-CM and ICD-10 administrative data. Med Care. 2005 Nov; 43(11):1130–9. [PubMed: 16224307]
22. Becker SO, Ichino A. Estimation of average treatment effects based on propensity scores. The Stata Journal. 2002; 2:4:358–377. Available at http://www.stata-journal.com/sjpdf.html?article=st0026.
23. Liapis CD, Bell PR, Mikhailidis D, Sivenius J, Nicolaides A, Fernandese Fernandes J, et al. ESVS Guidelines Collaborators. ESVS guidelines. Invasive treatment for carotid stenosis: indications, techniques. Eur J Vasc Endovasc Surg. 2009 Apr; 37(4 Suppl):1–19. [PubMed: 19286127]
24. Chaturvedi S, Bruno A, Feasby T, Holloway R, Benavente O, Cohen SN, et al. Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Carotid endarterectomy--an evidence-based review: report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. Neurology. 2005 Sep 27; 65(6):794–801. [PubMed: 16186516]
25. Woo K, Garg J, Hye RJ, Dilley RB. Contemporary results of carotid endarterectomy for asymptomatic carotid stenosis. Stroke. 2010 May; 41(5):975–9. [PubMed: 20339122]
26. Angioplasty and stenting of the cervical carotid artery with embolic protection of the cerebral circulation. BlueCross/BlueShield Association Technol Eval Cent Asses Program Exec Summ. 2010 Aug; 24(12):1–3.
27. Nallamothu BK, Lu M, Rogers MA, Gurm HS, Birkmeyer JD. Physician specialty and carotid stenting among elderly medicare beneficiaries in the United States. Arch Intern Med. 2011 Nov 14; 171(20):1804–10. [PubMed: 21824938]
28. Ricotta JJ, Aburahma A, Ascher E, Eskandari M, Faries P, Lal BK. Updated Society for Vascular Surgery guidelines for management of extracranial carotid disease. J Vasc Surg. 2011 Sep; 54(3):e1–e31. [PubMed: 21889701]

29. Reiff T, Böckler D, Böhm M, Brückmann H, Debus ES, Eckstein HH, et al. Ongoing randomized controlled trials comparing interventional methods and optimal medical treatment in the treatment of asymptomatic carotid stenosis. Stroke. 2010 Dec; 41(12):e605–6. [PubMed: 21030708]

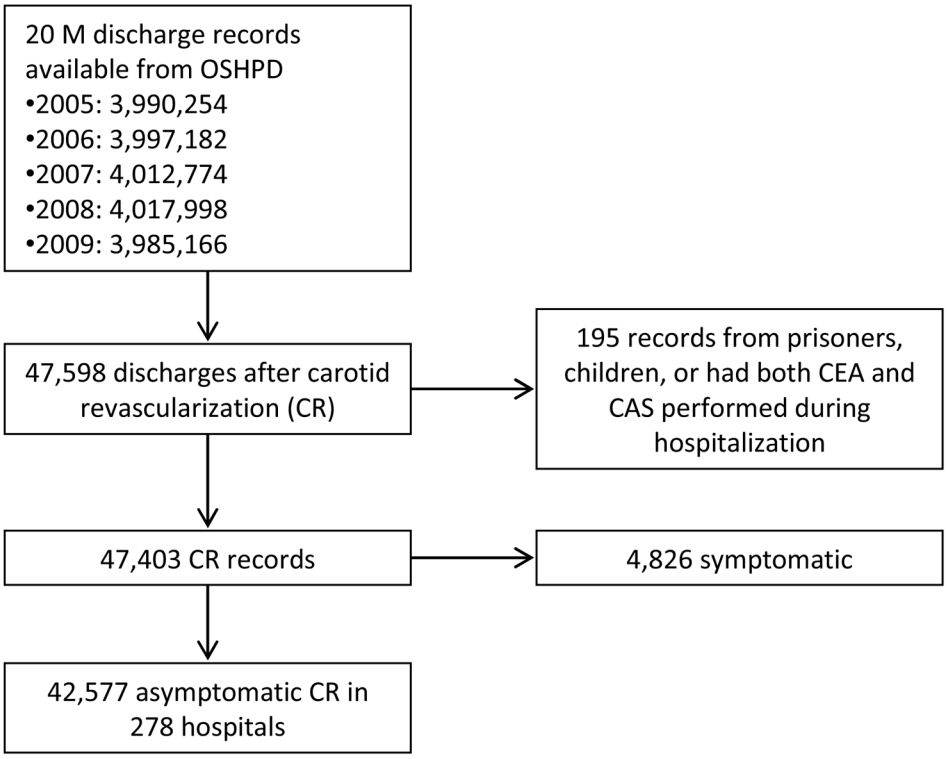

Figure 1.
Selection of hospital discharge records for analysis.

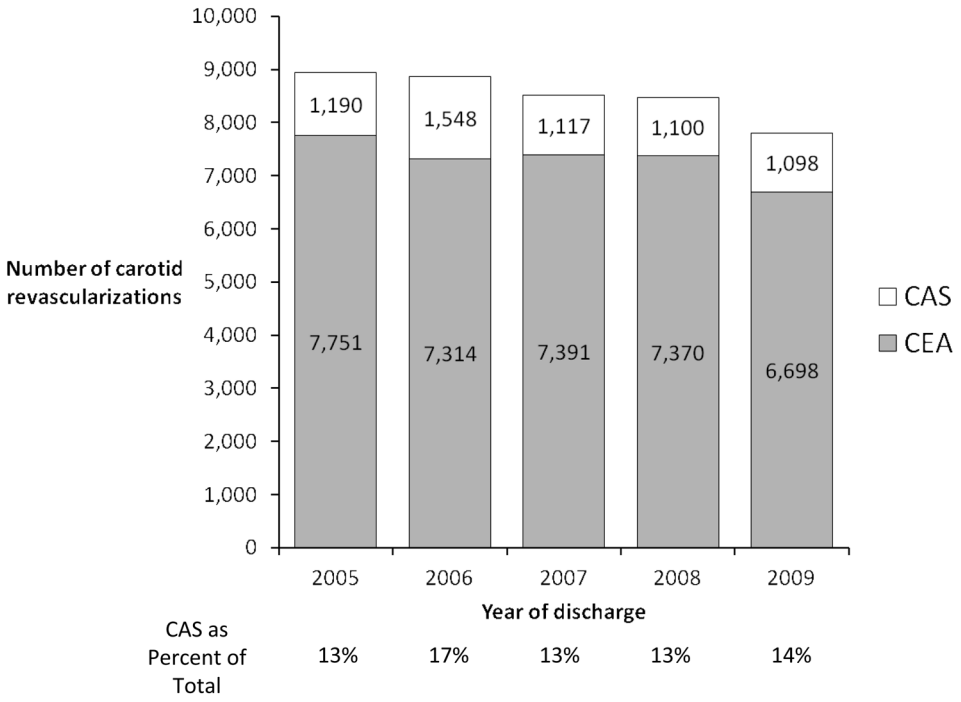

Figure 2.
Annual asymptomatic carotid revascularization procedures in California.

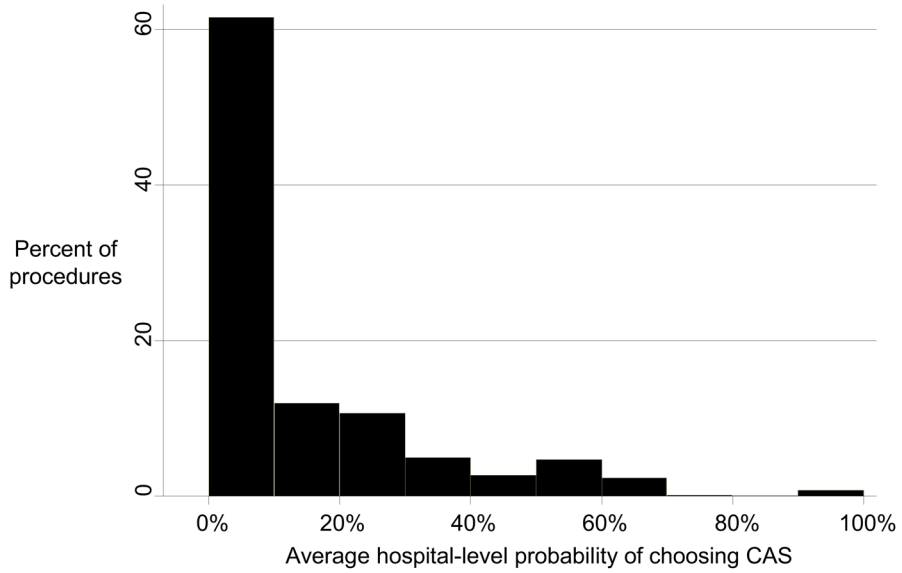

Figure 3.
Histogram of weighted average hospital-level probability of performing CAS versus CEA across all propensity score matched strata ("relative CAS utilization").

Table I

Definition of hemispheric cerebral ischemia or ophthalmic artery occlusion based on International Classification of Diseases, 9th Revision, Clinical Modification codes

ICD-9-CM Diagnosis code	Label
362.30	Retinal vascular occlusion, unspecified
362.31	Central retinal artery occlusion
362.32	Retinal arterial branch occlusion
362.33	Partial retinal arterial occlusion, Hollenhorst plaque, Retinal microembolism
362.34	Transient retinal arterial occlusion, Amaurosis fugax
362.84	Retinal ischemia
433.11	Occlusion and stenosis of carotid artery with cerebral infarction
433.31	Occlusion and stenosis of multiple and bilateral arteries with cerebral infarction
434.01	Occlusion of cerebral arteries (Cerebral thrombosis) with cerebral infarction
434.11	Cerebral embolism with cerebral infarction
434.91	Cerebral artery occlusion, unspecified, with cerebral infarction
435.8	Other specified transient cerebral ischemias
435.9	Unspecified transient cerebral ischemia: impending cerebrovascular accident; intermittent cerebral ischemia; transient ischemic attack

Table II

Selected demographics of all unmasked asymptomatic patient records

Variable	CEA	CAS	*P*
Total records	26,841	3,476	
Male	57%	56%	0.509
White Race	90%	83%	<0.001
Hispanic ethnicity	8%	9%	0.014
Age over 70	66%	62%	<0.001
Number of Elixhauser comorbidities	2.24	2.29	0.019
3 or more Elixhauser comorbidities	37%	39%	<0.001
Congestive Heart Failure	8%	11%	<0.001
Chronic Pulmonary Diseases	20%	17%	<0.001
Complicated Diabetes Mellitus	5%	4%	0.103
Renal failure	9%	10%	0.004
Obesity	8%	5%	<0.001
Hypertension, complicated	10%	11%	0.016

Note: Unmasked patients were used in all multivariate regression analyses.

Table III

Regression coefficients for multi-level model predicting PSD using demographic and clinical variables

Variable	Odds Ratio	Standard Error	95% CI	P-value
CAS (CEA as baseline)	1.865	0.292	1.373 – 2.534	<0.001
White race (non-white as baseline)	0.864	0.117	0.662 – 1.127	0.281
Hispanic ethnicity (non-Hispanic as baseline)	1.485	0.209	1.127 – 1.958	0.005
Male gender (female as baseline)	0.824	0.071	0.696 – 0.976	0.025
Age demographics (Age 70 to 74 years as baseline)*				
Under 50 years old	1.514	0.516	0.776 – 2.952	0.224
50 to 54 years old	1.125	0.330	0.634 – 1.998	0.687
55 to 59 years old	1.178	0.262	0.761 – 1.822	0.463
60 to 64 years old	1.436	0.241	1.034 – 1.996	0.031
70 to 74 years old	1.005	0.163	0.730 – 1.382	0.977
75 to 79 years old	1.191	0.171	0.900 – 1.578	0.222
80 to 84 years old	1.233	0.184	0.920 – 1.653	0.160
Over 85 years old	1.634	0.265	1.189 – 2.246	0.002
Elixhauser comorbidities				
Congestive heart failure	1.941	0.267	1.483 – 2.541	<0.001
Peripheral vascular disorders	1.333	0.141	1.083 – 1.640	0.007
Paralysis	6.937	1.661	4.339 – 11.09	<0.001
Other neurologic disorders	2.450	0.424	1.746 – 3.438	<0.001
Metastatic cancer	2.288	1.056	0.926 – 5.655	0.073
Solid tumor without metastases	1.887	0.519	1.101 – 3.236	0.021
Weight loss	3.542	1.097	1.930 – 6.500	<0.001
Fluid and electrolyte disorders	1.469	0.282	1.008 – 2.142	0.045
3 or more Elixhauser comorbidities	1.030	0.036	0.961 – 1.104	0.401

*Overall P = .0879

Table IV

Number of CEA and CAS procedures and associated PSD by year

Year	2005	2006	2007	2008	2009
Count of CAS	1,190	1,548	1,117	1,100	1,098
CAS PSD count	42	50	48	44	66
CAS PSD rate	3.5%	3.2%	4.3%	4.0%	6.0%
Count of CEA	7,598	7,197	7,267	7,225	6,577
CEA PSD count	153	117	124	145	121
CEA PSD rate	2.0%	1.6%	1.7%	2.0%	1.8%

Table V

Propensity score matching results

Stratum	CAS records	CEA records	Propensity score			
			1st quartile	Median	3rd quartile	Average
1	56	1,253	4.4%	5.0%	5.3%	4.9%
2	545	6,262	7.8%	8.7%	8.8%	8.3%
3	1,095	9,601	10.0%	10.2%	10.5%	10.2%
4	432	3,269	11.1%	12.0%	12.3%	11.8%
5	393	2,457	12.9%	12.9%	13.2%	13.0%
6	123	527	14.4%	14.9%	15.3%	14.9%
7	421	1940	16.7%	17.6%	18.2%	17.4%
8	323	1288	19.9%	21.3%	22.0%	21.0%
9	87	243	26.9%	29.0%	32.7%	30.1%
10	1	1	50.8%	52.7%	54.7%	52.7%
Total	3,476	26,841				

Note: Strata were constructed to ensure that covariates were similarly distributed and the average propensity score was approximately equal for CEA and CAS patients.

Table VI

Selected demographics of patients in propensity score stratum 3

Variable	CAS	CEA
Total records	1,095	9,601
Male	60%	61%
White Race	99%	99%
Hispanic ethnicity	3%	4%
Age over 70	77%	78%
Number of Elixhauser comorbidities	1.78	1.77
3 or more Elixhauser comorbidities	24%	23%
Congestive Heart Failure	1%	1%
Chronic Pulmonary Diseases	5%	6%
Complicated Diabetes Mellitus	2%	3%
Renal failure	7%	7%
Obesity	2%	1%
Hypertension, complicated	8%	7%

Table VII

Regression coefficients for multi-level model predicting PSD using demographic, clinical, and hospital variables

	Odds Ratio	Standard Error	95% CI	P- value
Hospital level variables				
Weighted average probability of performing CAS across all propensity score matched strata (Utilization of CAS)	1.407	0.746	0.497 – 3.98	.520
Number of CAS procedures performed *	0.805	0.110	0.615 – 1.054	.114
Number of CEA procedures performed *	0.974	0.039	0.900 – 1.053	.502
Number of patient discharges **	1.011	0.015	0.982 – 1.040	.465
Patient level variables				
CAS (CEA as baseline)	1.963	0.343	1.393 – 2.765	<.001
White race (non-white as baseline)	0.881	0.121	0.674 – 1.152	.355
Hispanic ethnicity (non-Hispanic as baseline)	1.453	0.205	1.102 – 1.916	.008
Male gender (female as baseline)	0.825	0.071	0.696 – 0.977	.025
Age demographics (Age 70 to 74 years as baseline)				
Under 50 years old	1.498	0.511	0.768 – 2.922	.236
50 to 54 years old	1.120	0.328	0.631 – 1.988	.699
55 to 59 years old	1.169	0.260	0.756 – 1.809	.483
60 to 64 years old	1.429	0.240	1.028 – 1.985	.034
65 to 69 years old	1.006	0.164	0.731 – 1.384	.970
75 to 79 years old	1.194	0.171	0.901 – 1.581	.216
80 to 84 years old	1.237	0.185	0.923 – 1.658	.154
Over 85 years old	1.641	0.266	1.194 – 2.255	.002
Elixhauser comorbidities				
Congestive heart failure	1.936	0.267	1.48 – 2.534	<.001
Peripheral vascular disorders	1.334	0.141	1.084 – 1.642	.006
Paralysis	6.934	1.661	4.342 – 11.073	<.001
Other neurologic disorders	2.451	0.424	1.747 – 3.439	<.001
Metastatic cancer	2.320	1.056	0.940 – 5.727	.068
Solid tumor without metastases	1.885	0.519	1.100 – 3.231	.021
Weight loss	3.456	1.097	1.884 – 6.339	<.001
Fluid and electrolyte disorders	1.462	0.282	1.003 – 2.13	.048
3 or more Elixhauser comorbidities	1.029	0.036	0.96 – 1.102	.424

* Per 100 procedures

** Per 10,000 discharges