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Surgeon case volume, not institution case volume, is the primary determinant of in-hospital mortality after elective open abdominal aortic aneurysm repair.

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Abstract

OBJECTIVE: Studies analyzing the effects of volume on outcomes after abdominal aortic aneurysm (AAA) repair have primarily centered on institutional volume and not on individual surgeon volume. We sought to determine the relative effects of both surgeon and institution volume on mortality after open and endovascular aneurysm repair (EVAR) for intact AAAs.

METHODS: The Nationwide Inpatient Sample (2003-2007) was queried to identify all patients undergoing open repair and EVAR for nonruptured AAAs. To calculate surgeon and institution volume, 11 participating states that record a unique physician identifier for each procedure were included. Surgeon and institution volume were defined as low (first quintile), medium (second, third, or fourth quintile), and high (fifth quintile). Stratification by institution volume and then by surgeon volume was performed to analyze the primary endpoint: in-hospital mortality. Multivariable models were used to evaluate the association of institution and surgeon volume with mortality for open repair and EVAR, controlling for potential confounders.

RESULTS: During the study period, 5972 open repairs and 8121 EVARs were performed. For open AAA repair, a significant mortality reduction was associated with both annual institution volume (low <7, medium 7-30, and high >30) and surgeon volume (low ≤ 2 , medium 3-9, and high >9). High surgeon volume conferred a greater mortality reduction than did high institution volume. When low and medium volume institutions were stratified by surgeon volume, mortality after open AAA repair was inversely proportional to surgeon volume (8.7%, 3.6%, and 0%; P < .0001, for low, medium, and high-volume surgeons at low-volume institutions; and 6.7%, 4.8%, and 3.3%; P = .02, for low, medium, and high-volume surgeons at medium-volume institutions). High-volume institutions stratified by surgeon volume demonstrated the same trend (5.1%, 3.4%, and 2.8%), but this finding was not statistically significant (P = .57). Multivariable analysis was confirmatory: low surgeon volume independently predicted mortality (odds ratio [OR], 2.0; 95% confidence interval [CI], 1.3-

3.1; P < .001); low institution volume did not (P = .1). For EVAR, neither institution volume nor surgeon volume influenced mortality (univariate or multivariable).

CONCLUSION: The primary factor driving the mortality reduction associated with case volume after open AAA repair is surgeon volume, not institution volume. Regionalization of AAAs should focus on open repair, as EVAR outcomes are equivalent across volume levels. Payers may need to reevaluate strategies that encourage open AAA repair at high-volume institutions if specific surgeon volume is not considered.

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