Original Article

Acute Type A aortic dissection in the United Kingdom: Surgeons volume-outcome relation

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Disclosures: The authors have no conflicts of interest or any sources of funding to declare.

Word Count: 4,233

Glossary:

AAD: Acute Aortic Dissection

ATAD: Acute Type A aortic dissection

IRAD: International Registry for Acute Dissection

SCTS: Society for Cardiothoracic Surgery

NICOR: National Institute for Cardiovascular Outcomes Research

TAVI: Transcatheter aortic valve implantations

NACSA: National Adult Cardiac Surgery Audit

CCS: Canadian Cardiovascular Society

NYHA: New York Heart Association

ONS: Office for National Statistics

MAV: Mean Annual case Volume

IQR: Inter-Quartile Range

ABSTRACT

Objectives

Surgery for acute Type A aortic dissection carries a high risk of operative mortality. We examined the surgeon volume-outcome relation with respect to in-hospital mortality for patients presenting with this pathology in the United Kingdom.

Method:

Between April 2007 and March 2013, 1550 acute Type A aortic dissection procedures were identified from the National Institute for Cardiovascular Outcomes Research database. 249 responsible consultant cardiac surgeons from the UK recorded one or more of these procedures in their surgical activity over this time period. We describe the patient population and mortality rates, focusing on the relationship between surgeon volume and in-hospital mortality.

Results:

The mean annual volume of procedures per surgeon during the 6-year period ranged from 1 to 6.6. The overall in-hospital mortality rate was 18.3% (283/1550). A mortality improvement at the 95% level was observed with a risk adjusted mean annual volume >4.5. Surgeons with a mean annual volume over the study period ≥4 had significantly higher in-hospital mortality rates in comparison to surgeons with a mean annual volume <4 (19.3% vs. 12.6%; P = 0.015).

Conclusion:

Acute Type A aortic dissection patients who are operated on by lower volume surgeons experience higher levels of in-hospital mortality. Directing these patients to higher volume surgeons may be a strategy to reduce in-hospital mortality.

Keywords: aorta, dissection, aneurysm, surgeon volume-outcome, AAD

INTRODUCTION

Acute Type A aortic dissection (ATAD) is a lethal condition and a cardiac surgical emergency. The incidence of aortic dissection is 30-43 per million population per year and this is incrementally increasing[1,2,3]. Forty percent of patients with dissection are aged 60 to 74 but 27% are aged 17 to 59 years, thus all ages are affected[4].

In medically treated patients, mortality rates are 1-2% per hour after the initial event, with death due to coronary or other organ malperfusion, cardiac tamponade, acute heart failure due to aortic regurgitation or aortic rupture. Emergency surgery can convert a 90% mortality rate at 30-days to a 75-90% long-term survival rate[4]. Mortality rates may vary, the International Registry for Acute Dissection (IRAD)[5] and the UK Society for Cardiothoracic Surgery (SCTS) 'Blue Book'[6] have published operative mortality rates of 25.1% and 22.8% respectively. In contrast, the German registry GERAADA published their series with lower rates of 17%[7]. This variation in reported mortality might be due to the volume-outcome relationship that has been at the center of debate and discussion. The Mount Sinai group utilizing the Nationwide Inpatient Sample of North America reported that lower-volume surgeons and centers have approximately double the risk-adjusted mortality of patients undergoing repair by the highest volume care providers[8].

This study aims to report the national UK surgeon outcomes in the operated ATAD patient population and explore the relationship in this population between surgeon volume and adjusted in-hospital mortality.

METHODS

NICOR database

Prospectively collected data were extracted from the National Institute for Cardiovascular Outcomes Research (NICOR) National Adult Cardiac Surgery Audit (NACSA) registry (version 4.1.2) on 20th November 2014 for all adult cardiac surgery procedures performed in the UK. As described elsewhere, reproducible cleaning algorithms were applied to the database[9]. Briefly, duplicate records and non-adult cardiac surgery entries [including transcatheter aortic valve implantations (TAVIs)] were removed, transcriptional discrepancies harmonized and clinical and temporal conflicts and extreme values corrected or removed. Data summaries are returned regularly to each unit for local validation as part of the NACSA in the UK[10].

For this study, records were included that corresponded to the following criteria: procedure on one or more of the root, ascending or arch aortic segments with a recorded pathology of "Acute Dissection" that were performed in England and Wales between 1st April 2007 and 31st March 2013. Records missing responsible consultant cardiac surgeon data (recorded in the form of General Medical Council registration number) were excluded.

Baseline and operative variables

For each procedure, data are recorded on patient characteristics, comorbidities, surgical team, intraoperative factors and postoperative outcomes. For this study, we extracted data on patient age at the time of procedure (years), gender, body mass index [BMI, defined as weight (kg) / height^2 (m^2)], Canadian Cardiovascular Society (CCS) angina class, dyspnoea (dichotomized as New York Heart Association (NYHA) grade < III and NYHA grade ≥ III), recent myocardial infarction (defined as within 90 days of surgery), history of major cardiac surgery, diabetes (diet or insulin controlled), smoking status, history of hypertension, serum creatinine >200 μmol/l, history of renal dysfunction, history of pulmonary disease, history of neurological dysfunction, extracardiac arteriopathy, preoperative heart rhythm, left ventricular ejection fraction (classified as <30, 30–50 and >50%), IV inotropes prior to anaesthesia, preoperative ventilation, preoperative cardiogenic shock, operative urgency, concomitant CABG and valve procedures, cardiopulmonary bypass time, aortic cross-clamp time, and circulatory arrest time.

Administrative data were also extracted including: patient admission, procedure and discharge dates, responsible consultant cardiac surgeon and anonymized hospital identifier. Further details of variable definitions are available at: http://www.ucl.ac.uk/nicor/audits/adultcardiac/datasets.

Outcomes

The primary outcome for this study was in-hospital mortality, defined as death due to any cause during admission to the base hospital for cardiac surgery. The secondary outcome was mid-term mortality followed up to 5 years. Follow-up data up until the point of discharge was collected by the NACSA clinical registry system and post-discharge survival data was collected by linking the records via patient NHS numbers to the Office for National Statistics (ONS) death registry, which records all deaths in England and Wales. The final date of follow-up was 30th July 2013. Data on cause of death was unavailable. An attempt to back-fill missing in-hospital mortality data was made by record linkage to the ONS registry prior to applying the extraction criteria.

Case volumes

For analytical purposes, case volumes are presented both continuously and categorically. In the continuous analysis, the mean annual case volume (MAV) was calculated. This was achieved by taking the total number of procedures for each surgeon and dividing this by the number of years in which they contributed data to the registry. In the categorical analysis, the case volume was stratified into two groups (surgeon MAV of ATAD procedures <4 and ≥4 over the study period). The cut point was selected as being clinically meaningful after the introductory analysis showed it to be the approximate inflection point for improved mortality.

Statistical analysis

Categorical and dichotomous variables are summarized as absolute number and percentage. Non-normally distributed continuous data are summarized as median and inter-quartile range (IQR). The prevalence of missing data in the registry for baseline and operative measurements, as well as in-hospital mortality, are reported. Due to the relatively low number of missing data items for the majority of the variables, categorical variables with missing data were imputed with the baseline category and continuous variables were imputed with the mean value before calculations were performed.

Where categorical comparisons are made between groups the chi-squared test was used, for similar comparisons between continuous variables the Wilcoxon rank-sum test was used.

To quantify the relationship between MAV and in-hospital mortality we performed three separate multivariable regression analyses. Firstly, an initial balancing score was fitted for each patient using a mixed-effects linear regression model. The dependent continuous variable used was a log transformation of the surgeon MAV, with random intercepts for each hospital, and the following patient variables were entered as independent variables: age, gender, body mass index, smoking, renal failure, hypertension, pulmonary disease, neurological disease, neurological dysfunction, peripheral vascular disease, recent myocardial infarction, unstable angina, arrhythmia, New York Heart Association class, previous cardiac surgery, diabetes, ejection fraction, cardiogenic shock, pre-operative ventilation, operative urgency, cardiopulmonary bypass time, circulatory arrest time, surgery on the aortic arch and concomitant procedures. The volume-outcome relationship was then assessed by plotting estimated restricted cubic spline regression functions with three knots between the surgeon MAV and in-hospital mortality; these were then adjusted for patient case-mix by entering the balancing score into the regression model. The spline estimates were based on a standard, fixed effects logistic regression model. The balancing score therefore acts in a similar way to a propensity score[11,12], but is generalized beyond a dichotomous treatment assignment.

Secondly, in order to examine the contribution of hospital volume to outcome, a logistic regression model for in-hospital mortality was fitted including random intercepts for each hospital, with surgeon and hospital MAV entered into the model as continuous variables, along with the independent demographical and procedural variables listed above, interactional terms between hospital and surgeon MAV were also explored.

Thirdly, a similar approach was taken to assessing the contribution of MAV to mid-term mortality. Cox proportional hazards models were fitted, again with random intercepts for each hospital and with surgeon and hospital MAV entered into the model as continuous variables, along with the independent demographical and procedural variables listed above. In order to demonstrate any non-proportional effects of early mortality two models were fitted, one with a start time of the procedure date and the second with a start time of 90-days post-procedure.

To evaluate the categorical difference in volume, Kaplan-Meier charts were used to plot the actuarial 5-year survival, incorporating a landmark cutoff of 90 days where the groupwise mortality rates were rebased to zero. The log-rank test was used to assess the equivalence of death rates between groups in both phases of the analysis.

Statistical analyses were performed with SAS version 9.3 (SAS Institute, Cary, NC). In all cases a P value <0.05 was considered statistically significant.

RESULTS

Characteristics of the study population

1632 patients were identified from the NACSA database as having had surgery for ATAD during the time period. Patients who had surgery on the descending and/or thoracoabdominal segments of the aorta (n=63; 3.9%), and 19 (1.2%) records that lacked responsible clinician data were excluded from the analysis. The 1550 that remained were included in the study analysis dataset, however 162 (10.5%) records lacked follow-up mortality data and are excluded from that element of the analysis.

The 1550 patients were admitted to 41 different hospitals throughout England and Wales and were under the care of 249 different consultant cardiac surgeons. The mean surgeon MAV was 2.6 (SD = 1.2; median (IQR) = 2.3 (1.6, 3.3), with 199 of 249 surgeons (79.9%) performing fewer than 10 procedures overall. A total of 41 surgeons performed a single ATAD procedure. The highest number of procedures performed by a single surgeon during the study period was 33. The mean hospital MAV was 9.6 (SD = 4.6; median (IQR) = 8.7 (6.0, 13.2). The highest number of procedures performed by a single hospital during the study period was 107.

Pre-operative and operative differences between the two categorical groups are shown in Tables 1 and 2. Surgeons in the lower MAV group were more likely to operate on patients who had a recent MI, whilst being less likely to operate on patients with a history of pulmonary disease or patients who required surgery on the aortic arch. Surgeons in the lower MAV group also reported significantly longer circulatory arrest times.

In-hospital mortality and case volume

The overall in-hospital mortality rate for all ATAD patients was 18.3% (283 patients). Figure 1 plots the observed in-hospital mortality against the adjusted surgeon MAV. Somewhat counter-intuitively, the curve begins below the national mean rate at the lowest volumes then rises and peaks between 2 and 3 procedures per year, before decreasing in an approximate linear trend in higher volume surgeons. Significant in-hospital mortality improvements can be observed beyond a surgeon MAV of 4 to 4.5.

The unadjusted in-hospital mortality rate decreased from 19.3% in the group of surgeons who had a MAV <4 during the study period to 12.6% in the group who had a MAV ≥4; P = 0.015 (Table 2). Figure 2a illustrates the groupwise trends in 5-year follow up mortality rates, including a landmark rebasing at 90 days. The early difference in mortality rates is significant at the 0.05 level (log-rank test P value = 0.028), however this difference is not sustained in the second era, from 90 days to 5 years (P = 0.97). (Figure 2b is a detail from Figure 2a which charts the 90 day mortality only, included for clarity).

The logistic regression model shown in Table 3 demonstrates a similar in-hospital mortality advantage for surgeons who perform a greater number of operations, after adjustment for casemix and hospital volume, increasing surgeon MAV (assessed as a continuous variable) was associated with a significant reduction to in-hospital mortality (adjusted OR=0.853 (95% CI 0.733 to 0.992) P = 0.039). Other associated variables were: increasing age, previous cardiac surgery, peripheral vascular disease, left ventricular ejection fraction <30%, cardiogenic shock, salvage priority, concomitant CABG procedure, and increasing cardiopulmonary bypass time. Hospital MAV was not associated with a difference in in-hospital mortality (adjusted OR=1.005 (95% CI 0.956 to 1.057) P = 0.84).

Figure 3 includes four charts which show the interaction between surgeon and hospital MAV with regards to in-hospital mortality. The predicted probabilities of in-hospital mortality over the range of surgeon MAV are shown for the 20^{th}, 40^{th}, 60^{th} and 80^{th} percentiles of hospital MAV (which are, respectively, 5.8, 7.0, 10.2 and 14.3 cases per year). Visual inspection of these allows us to infer that there are no substantial differences in the relationship between surgeon MAV and in-hospital mortality as hospital MAV increases. The associated interaction test P value = 0.88.

Follow-up mortality and case volume

The results of the two Cox proportional hazard models are shown in Table 4. In the 'Start time = procedure date' model, higher surgeon MAV (assessed as a continuous variable) was again associated with a significant reduction in death (adjusted HR=0.882 (95% CI 0.801 to 0.972) $P = 0.011$). Other variables that were associated with a greater hazard of death were: increasing age, left ventricular ejection fraction <30%, cardiogenic shock, salvage priority, surgery on the aortic arch, concomitant CABG procedure and increasing cardiopulmonary bypass time. Hospital MAV was not significantly associated with a difference in death (adjusted HR=1.029 (95% CI 1.000 to 1.059) $P = 0.050$).

In the 'Start time = 90 days post-procedure' model, higher surgeon MAV was not associated with a significant reduction in death (adjusted HR=0.920 (95% CI 0.779 to 1.088) $P = 0.33$). Other variables that were associated with a greater hazard of death were: increasing age and left ventricular ejection fraction <30%. Hospital MAV was not significantly associated with a difference in death (adjusted HR=1.020 (95% CI 0.972 to 1.072) $P = 0.42$), suggesting that the significant effect observed in the 'Start time = procedure date' model is both non-proportional and also greatly reliant on the large early mortality burden.

DISCUSSION

It has been shown that out of every 1,000 emergency department patients presenting with acute back, chest, or abdominal pain, three patients with ATAD are diagnosed[13]. This is a fatal condition with a dire prognosis unless the patient receives immediate surgical intervention. The IRAD has published outcomes from multiple centres worldwide with an average in-hospital mortality of 25.1% in 2005[14]. European registries in the United Kingdom and Germany have published operative mortalities of 23.1% and 17% respectively[15]. A recent publication from Mount Sinai Medical Centre, using the Nationwide Inpatient Sample database of 5,184 patients between

2003 and 2008, showed average operative mortality of 21.6%[8]**Error! Bookmark not defined.**. However, in the advent of this decade there are multiple centres worldwide that are publishing in-hospital mortality rates for ATAD in the single digits[16171819].

Throughout the United States the model of aortic supercentres with high referral rates have existed for some time. It has been suggested that outcomes in thoracic aortic surgery could be improved nationwide in the United States if the acute care and emergency surgical treatment of most patients with ATAD were regionalized and restricted to institutions with high-volume multidisciplinary thoracic aortic surgery programs[20].

Andersen et al. from the Duke group published their results pertaining to the impact model of a multi-disciplinary team approach to ATAD[17]. They reported operative mortality before multi-disciplinary implementation was 33.9% and was statistically equivalent to the expected operative mortality rate of 26.0%. Operative mortality after multi-disciplinary implementation fell to 2.8% and was statistically better than the expected operative mortality rate of 18.2% using the International Registry of Acute Aortic Dissection pre-operative prediction model.

In the UK, centralization of expertise and service provision for type A aortic dissection does not exist. Operating on ATAD does not generally follow a selective referral protocol which in effect leads to a national mix and match between high and low volume surgeons. Compounding this is the lack of best practice evidence on structured referral from emergency room to operating room. This is due to multiple factors which unfortunately have not been quantified in the UK. Those factors are in essence related to lack of swift recognition of ATAD. There is also lack of substantial evidence on the actual time to referral once ATAD is actually identified. The aforementioned mandate a policy that will serve better patients' outcome and results across the UK.

Evidence of improved outcomes related to operative volume or surgeon expertise is often difficult to establish due to the infrequent nature of ATAD and consequent lack of statistical power that could potentially provide meaningful analysis[21].

The ideal definition of volume is inherently inconsistent; this makes diving into a discussion involving 'volume' highlight caveats that are not potentially attained or addressed between cardiac surgical units at large. The strong rationale of the volume-outcome relationship as reflected in literature springs from the catalyst for subspecialisation and centralization of aortic services. It is to provide centres with a large and reaching catchment areas the reciprocal improvement and effect on the subspecialized unit. It allows more robust referral to influx and therefore maintain an adequate voluminous exposure. Essentially, in the UK, thoracic aortic aneurysm service is in much need of such an approach and a national policy and mandate that would support such programs across the UK should be warranted. This should provide a sustained increase in volume to concentrated expertise that would allow the possibility to address dire surgical diseases and avert associated complications. It will then reciprocate this arrangement by ultimately reducing mortality and perhaps improving survival and aftercare post-surgery[22]. Beside this, the advent of technological superiority in diagnosis and surgical planning of aortic surgery and the understanding of the natural history is resulting in personalized and targeted therapies and surgical procedures to be done on a wider range of the affected population. This has allowed for such cost-effective diagnostic tests to be distributed to a smaller number of regional centres and for them to operate on this patient cohort. Hence, this has titrated the inexistence of specialist centres and diverted a large number of patients to be operated at local lower-volume institutions.

The development of standardization and subspecialisation of acute aortic services requires a comprehensive assessment of the current status in aortic surgery in the UK. As such, our analysis has demonstrated that there is significant variation of in-hospital mortality around the country with little relationship between volume and outcome at a hospital level. These results are contrary to those demonstrated by international groups including the Mount Sinai group utilizing the Nationwide Inpatient Sample of North America who reported that lower-volume surgeons and centres have approximately double the risk-adjusted mortality of patients undergoing repair by the highest volume care providers[23].

Our study does however demonstrate that higher individual surgeon volume was associated with lower in-hospital mortality. These relationships could be explained by a number of different factors such as; case mix per individual surgeon, selection bias and variations in turn down practice, concentration of expertise to a particular surgeon within a hospital and inadvertent subspecialisation of a surgeon with interests to aortic interventions. On the other hand, and as demonstrated in the context of the analysis; difference between low and high volume surgeons clearly point out the shorter aortic cross clamp and cardiopulmonary time but increased circulatory arrest time in the low-volume group which could be related to attempt of more frequent use or extended repair entailing arch segment replacement. Although this is not entirely understood, one way of scoping this further would be to look at this element within the cohort and run a thorough factor analysis

While this study demonstrates good overall mortality rates for ATAD in the UK, it is likely that further improvements could be achieved through the introduction of a quality improvement programme for ATAD surgery. It is vital that such a programme is implemented across the multidisciplinary thoracic aortic surgery team including anaesthesia, postoperative surgical intensive care and operative perfusion specialists. Such a programme should also involve standardized referral pathways and treatment protocols for ATAD repair. Another important contributing factor would be the development and implementation of a robust referral system and an initiative to hospital managers and commissioners for centralization of expertise in ATAD repair. This will reduce the waiting time and taxing of ATAD patients unnecessarily in acute services while diagnostics are being carried out.

Limitations

The main limitation of this study is its retrospective nature and the variable nature of data quality between institutions in the UK. There are also several confounding variables to consider that are not available in the NICOR dataset. The foremost of these factors is probably case selection: surgeons at tertiary referral centres are likely to operate on patients significantly longer after the acute event than local units; consequently their surgical outcomes may benefit from both temporal patient selection (more stable patients are more likely to survive transfer), and more aggressive individual patient selection informed by the additional complications (such as malperfusion syndromes) that manifest hours to days after initial presentation. Other possible confounding variables include delays between diagnosis and intervention, referral bias and clustering, and presence, severity, and duration of end-organ ischemia.

CONCLUSIONS

Concentration of expertise and volume to appropriate surgeons who perform increasingly more complex aortic cases would be required to change the current paradigm of ATAD outcomes in the UK. Whenever feasible, ATAD repair should be performed by a high volume surgeon in order to reduce operative mortality. It is reasonable to suggest a national standardization and quality improvement framework for ATAD treatment.

ACKNOWLEDGEMENTS

The authors acknowledge all members of the Society for Cardiothoracic Surgery in Great Britain and Ireland who contribute data to the National Adult Cardiac Surgery Audit registry. The National Institute for Cardiovascular Outcomes Research, UCL, London, provided the data for this study.

DATA SHARING

The United Kingdom National Adult Cardiac Surgery Audit registry is available to researchers upon application to the National Institute of Cardiovascular Outcomes Research (NICOR), University College London. Full details on the NICOR data sharing application process are available at https://www.ucl.ac.uk/nicor/access/application [last accessed: 22nd December 2016].

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Table 1: Patient characteristics, stratified by surgeon MAV group

	Mean annual volume < 4 (n=1319)	Mean annual volume ≥ 4 (n=231)	*P* value	Missing Data
Age at procedure (years)	63 (52, 72)	64 (53, 74)	0.10	0 (0)
Female gender	425 (32.2)	69 (29.9)	0.48	0 (0)
Body mass index (kg/m2)	26.4 (23.9, 29.7)	26.4 (24.2, 29.0)	0.54	48 (3.5)
Angina class IV	199 (15.1)	39 (16.9)	0.48	17 (1.1)
NYHA class ≥ III	354 (26.8)	68 (29.4)	0.41	23 (1.5)
Previous Q-wave MI	130 (9.9)	18 (7.8)	0.32	14 (0.9)
Recent MI (within 90 days)	83 (6.3)	4 (1.7)	0.006	10 (0.7)
Previous PCI	44 (3.3)	6 (2.6)	0.56	39 (2.5)
Previous cardiac surgery	75 (5.7)	20 (8.7)	0.08	127 (8.2)
Diabetes (diet or insulin controlled)	62 (4.7)	6 (2.6)	0.15	13 (0.8)
Current smoker	243 (18.4)	34 (14.7)	0.18	64 (4.1)
History of hypertension	905 (68.6)	167 (72.3)	0.26	27 (1.7)
Creatinine > 200 µmol/L	67 (5.1)	16 (6.9)	0.25	115 (7.4)
History of renal dysfunction	32 (2.4)	8 (3.5)	0.36	71 (4.6)
History of pulmonary disease	134 (10.2)	34 (14.7)	0.04	9 (0.6)
History of neurological disease	130 (9.9)	29 (12.6)	0.21	23 (1.5)
History of neurological dysfunction	93 (7.1)	20 (8.7)	0.39	15 (1.0)
Peripheral vascular disease	259 (19.6)	47 (20.4)	0.80	16 (1.0)
Non-sinus heart rhythm	120 (9.1)	18 (7.8)	0.52	99 (6.4)
Triple vessel disease	29 (2.2)	5 (2.2)	0.97	338 (21.8)
Left ventricular ejection fraction 30% - 50%	248 (18.8)	39 (16.9)	0.49	31 (2.0)
Left ventricular ejection fraction <30%	50 (3.8)	7 (3.0)	0.57	31 (2.0)
Intravenous nitrates or any heparin	200 (15.2)	29 (12.6)	0.30	6 (0.4)
Intravenous inotropes prior to anaesthesia	100 (7.6)	10 (4.3)	0.08	10 (0.7)
Pre-operative ventilation	77 (5.8)	12 (5.2)	0.70	15 (1.0)
Pre-operative cardiogenic shock	228 (17.3)	29 (12.6)	0.07	14 (0.9)

Continuous variables shown as median (25th percentile, 75th percentile); categorical variables shown as frequency (%)

NYHA = New York Heart Association, MI = Myocardial Infarction, PCI = Percutaneous Coronary Intervention

Table 2: Operative characteristics and in-hospital mortality, stratified by surgeon MAV group

	Mean annual volume < 4 (n=1319)	Mean annual volume ≥ 4 (n=231)	*P* value	Missing Data
Surgeon mean annual volume	2.0 (1.5, 2.7)	4.7 (4.3, 5.0)	<0.001	0 (0)
Hospital mean annual volume	8.7 (6.0, 13.2)	10.2 (8.7, 17.8)	<0.001	0 (0)
Elective procedure	21 (1.6)	3 (1.3)	>0.99	0 (0)
Urgent procedure	169 (12.8)	34 (14.7)	0.43	0 (0)
Emergency procedure	1037 (78.6)	181 (78.4)	0.93	0 (0)
Salvage procedure	92 (7.0)	13 (5.6)	0.45	0 (0)
Root segment	438 (33.2)	82 (35.5)	0.50	0 (0)
Ascending segment	1146 (86.9)	203 (87.9)	0.68	0 (0)
Arch segment	152 (11.5)	46 (19.9)	<0.001	0 (0)
Concomitant CABG procedure	171 (13.0)	30 (13.0)	0.99	30 (1.9)
Concomitant Valve procedure	521 (39.5)	99 (42.9)	0.34	29 (1.9)
Concomitant 'Other' cardiac procedure	395 (30.0)	76 (32.9)	0.37	34 (2.2)
Cardiopulmonary bypass time (mins)	196 (152, 259)	197 (154, 254)	0.86	44 (2.8)
Aortic cross clamp time (mins)	105 (74, 143)	109 (68, 147)	0.81	56 (3.6)
Circulatory arrest time (mins)	29 (20, 39)	20 (15, 31)	<0.001	402 (25.9)
In-hospital mortality	254 (19.3)	29 (12.6)	0.015	0 (0)

Continuous variables shown as median (25th percentile, 75th percentile); categorical variables shown as frequency (%)

CABG = Coronary Artery Bypass Grafting

Table 3: Logistic regression analysis for in-hospital mortality

Variable	Odds ratio	95% confidence interval	*P* value
Surgeon mean annual volume	0.853	0.733 - 0.992	0.039
Hospital mean annual volume	1.005	0.956 - 1.057	0.84
Age at procedure (years)	1.028	1.015 - 1.041	<0.001
Previous cardiac surgery	1.840	1.052 - 3.218	0.033
Peripheral vascular disease	1.505	1.051 - 2.156	0.026
Left ventricular ejection fraction <30%	2.896	1.374 - 6.104	0.005
Pre-operative cardiogenic shock	1.722	1.137 - 2.607	0.010
Salvage procedure	5.474	2.790 - 10.741	<0.001
Concomitant CABG procedure	2.135	1.412 - 3.229	<0.001
Cardiopulmonary bypass time (mins)	1.003	1.002 - 1.005	<0.001

Table 4: Cox proportional hazards analysis for survival from procedure and from 90-days post-procedure

Variable	Start time = procedure date			Start time = 90 days post-procedure		
	Hazard ratio	95% confidence interval	*P* value	Hazard ratio	95% confidence interval	*P* value
Surgeon mean annual volume	0.882	0.801 - 0.972	0.011	0.920	0.779 - 1.088	0.33
Hospital mean annual volume	1.029	1.000 - 1.059	0.050	1.020	0.972 - 1.072	0.42
Age at procedure (years)	1.028	1.019 - 1.037	<0.001	1.043	1.023 - 1.063	<0.001
Left ventricular ejection fraction <30%	2.495	1.586 - 3.926	<0.001	5.799	2.169 - 15.505	<0.001
Pre-operative cardiogenic shock	1.426	1.068 - 1.903	0.016	0.880	0.459 - 1.687	0.70
Salvage procedure	3.250	2.139 - 4.965	<0.001	2.138	0.791 - 5.778	0.13
Arch segment	1.414	1.047 - 1.909	0.024	1.200	0.653 - 2.207	0.56
Concomitant CABG procedure	1.629	1.235 - 2.150	<0.001	1.215	0.636 - 2.323	0.56
Cardiopulmonary bypass time (mins)	1.003	1.002 - 1.004	<0.001	1.001	0.998 - 1.003	0.53

Figure 1: Trend in mean annual volume (MAV) of ATAD activity and observed mortality. Each black dot corresponds to the mean mortality (vertical axis) for ATAD procedures performed by consultant surgeons with a specific MAV (horizontal axis). The size of the black dots is proportional to the total number of ATAD procedures performed by surgeons with the given MAV. Please note that although volume was modelled continuously, to improve legibility the number of surgeon procedures is grouped for every 0.25 of a year, therefore each dot can be comprised of multiple consultant surgeons. The blue line is a fitted smoothing curve to illustrate the trend, adjusted for pre-operative risk factors, and the grey-shaded area denotes approximate 95% confidence intervals. The red horizontal line represents the overall mean observed in-hospital mortality (18.3%) for the study cohort.

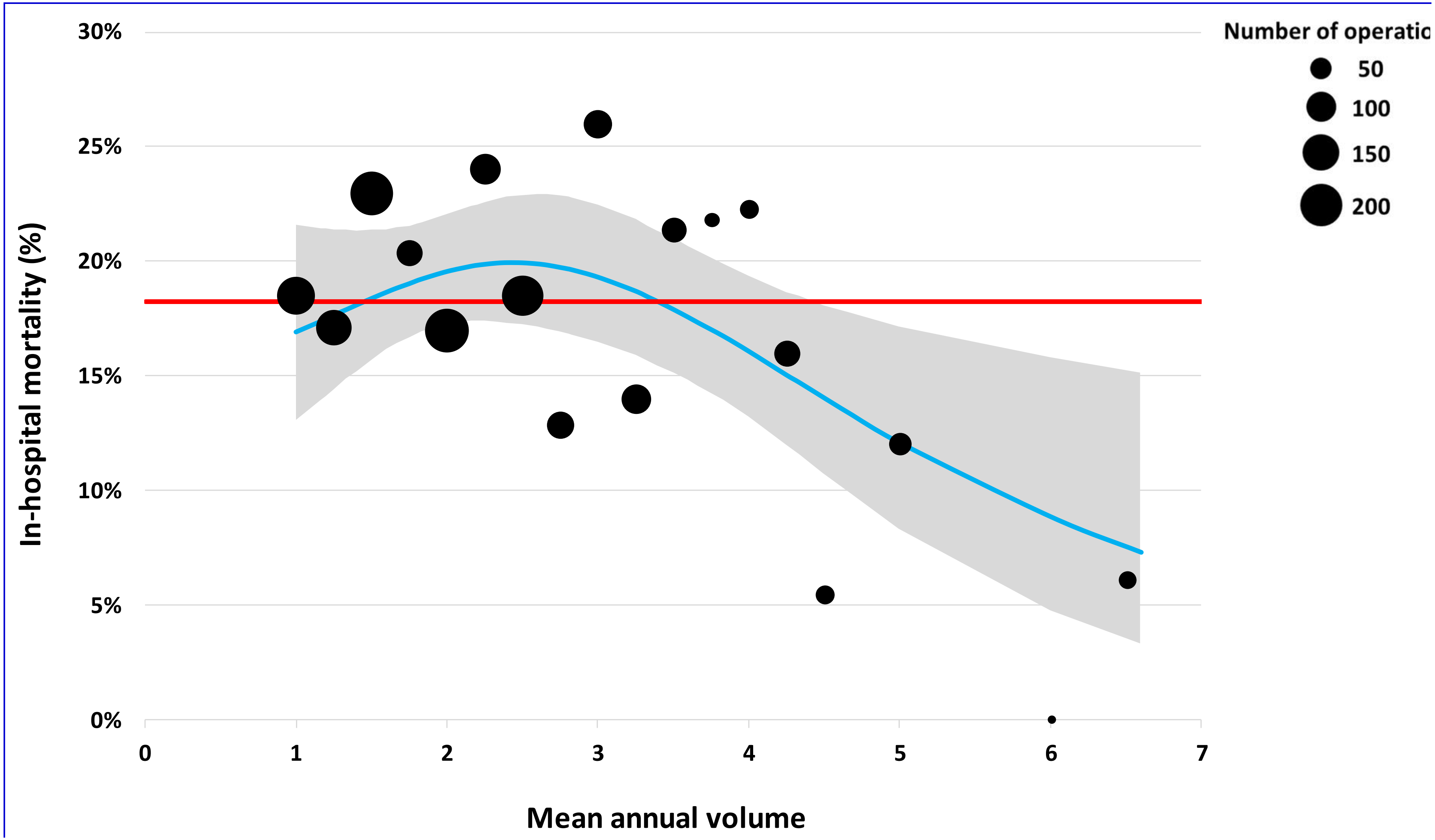


Figure 2a: Kaplan-Meier chart showing the cumulative probability of all-cause mortality for ATAD patients in the surgeon MAV groups. Landmark rebasing occurs at 90 days. Colored bands show approximate 95% confidence intervals.

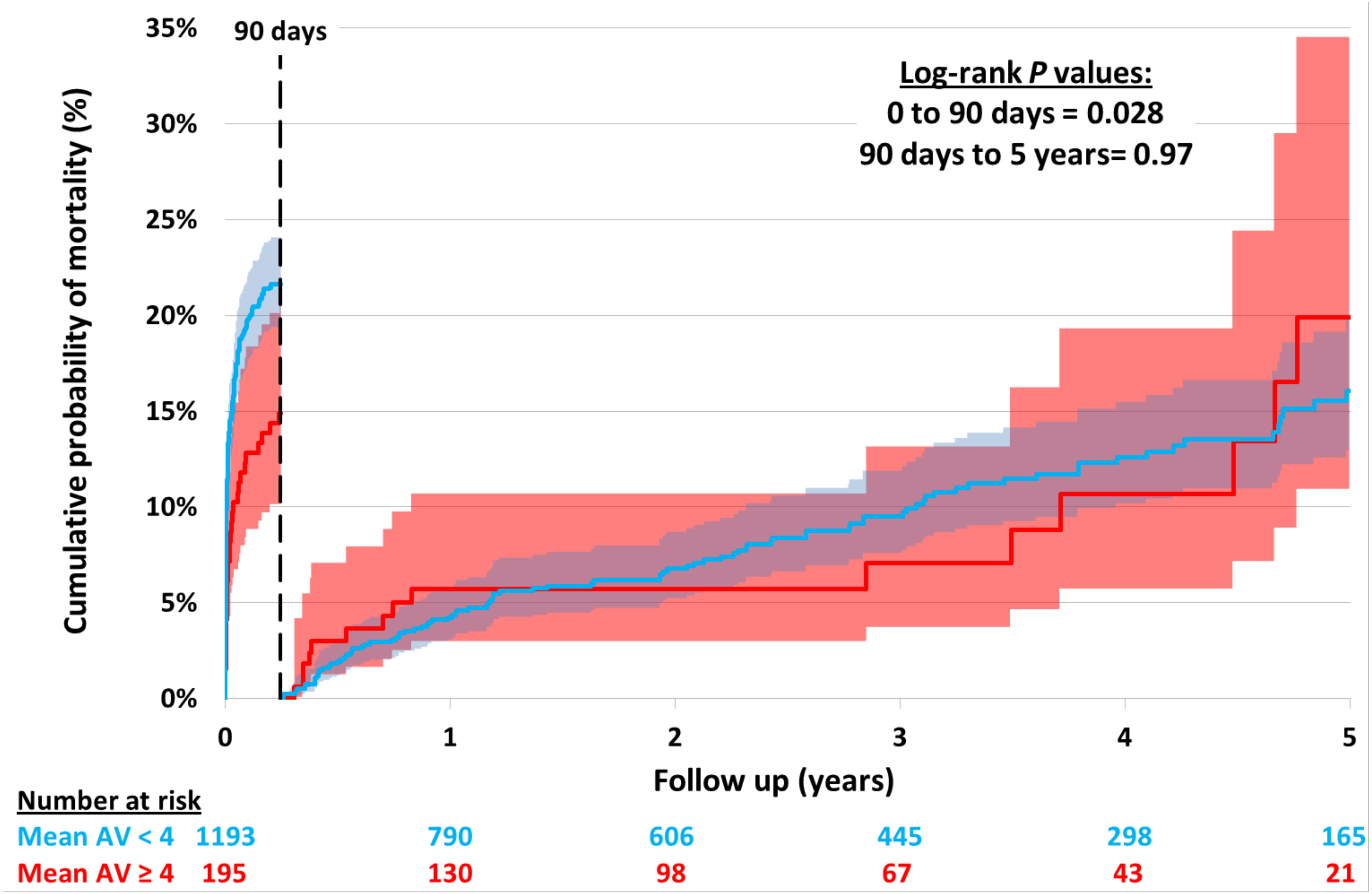


Figure 2b: Kaplan-Meier chart showing the cumulative probability of all-cause mortality for ATAD patients in the surgeon MAV groups. Colored bands show approximate 95% confidence intervals. (0-90 day detail from Figure 2a).

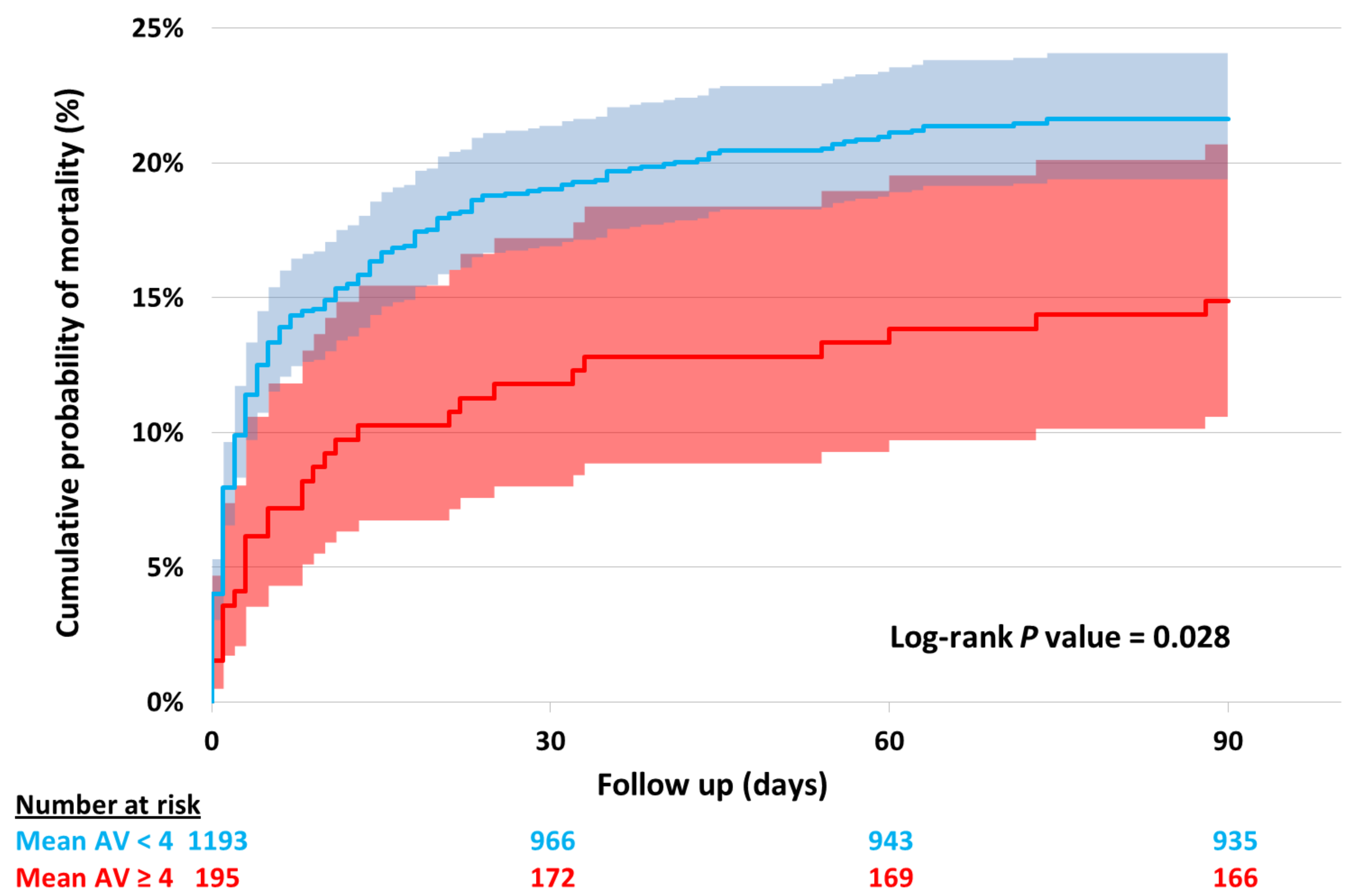
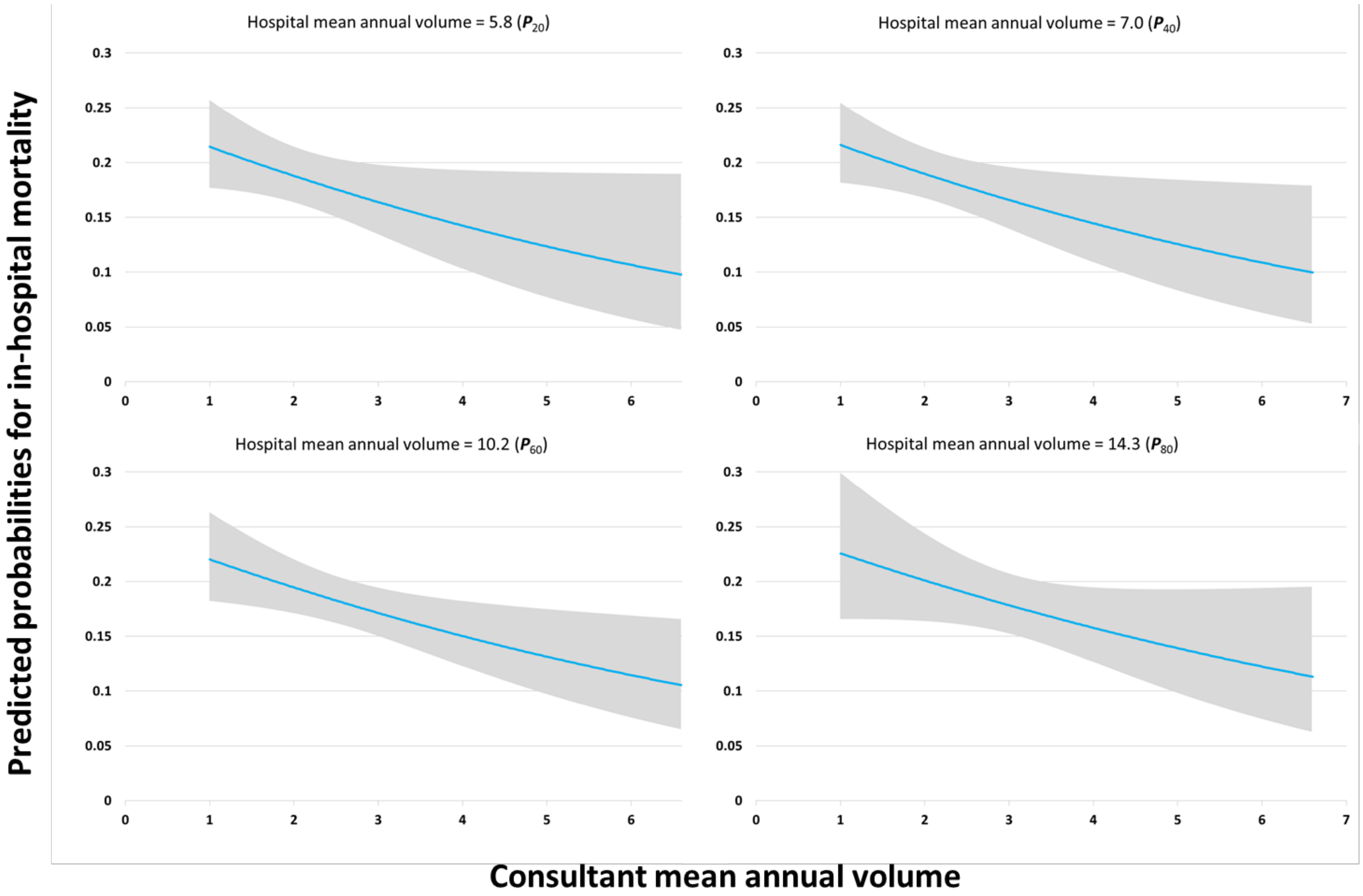
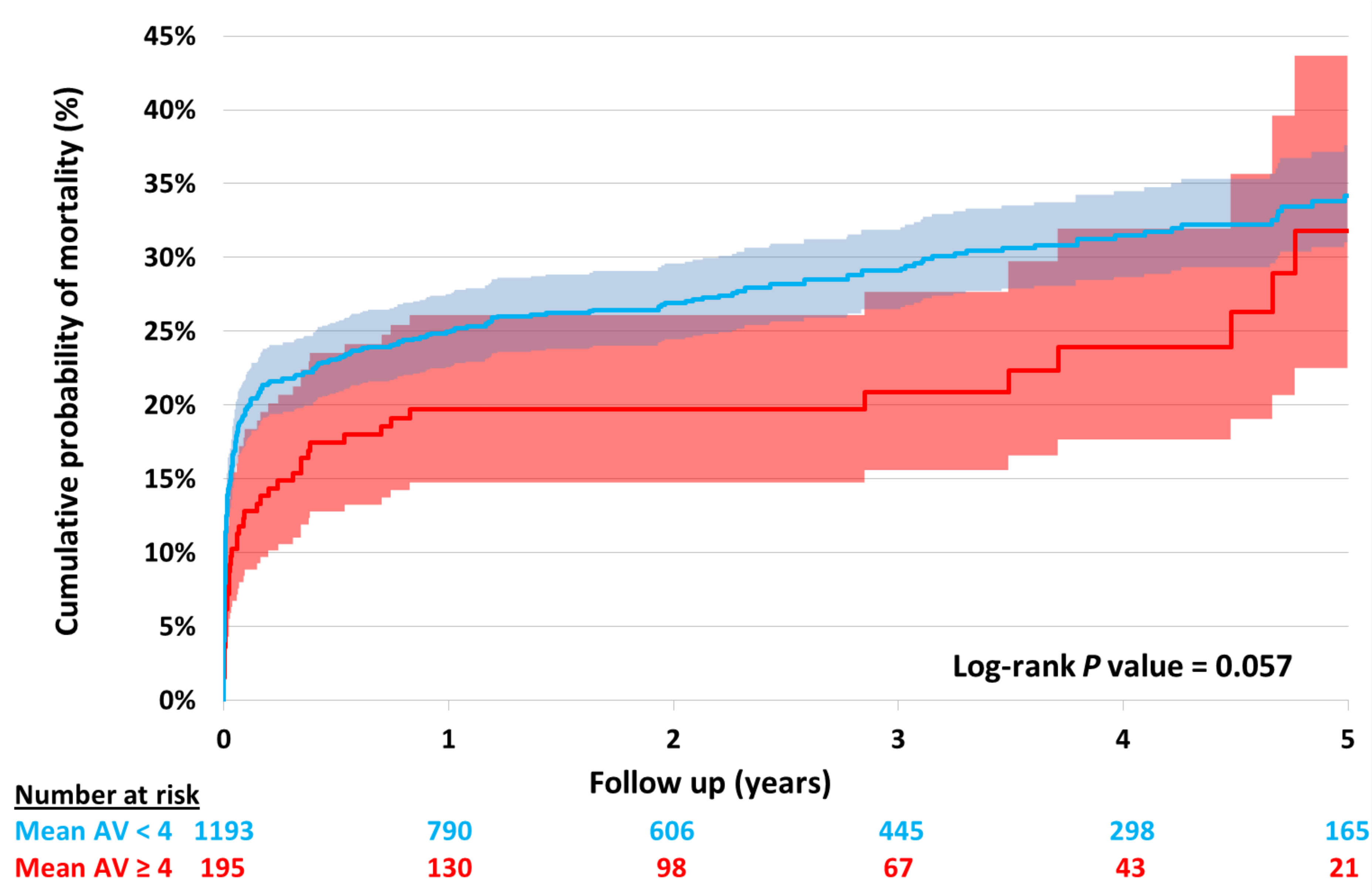


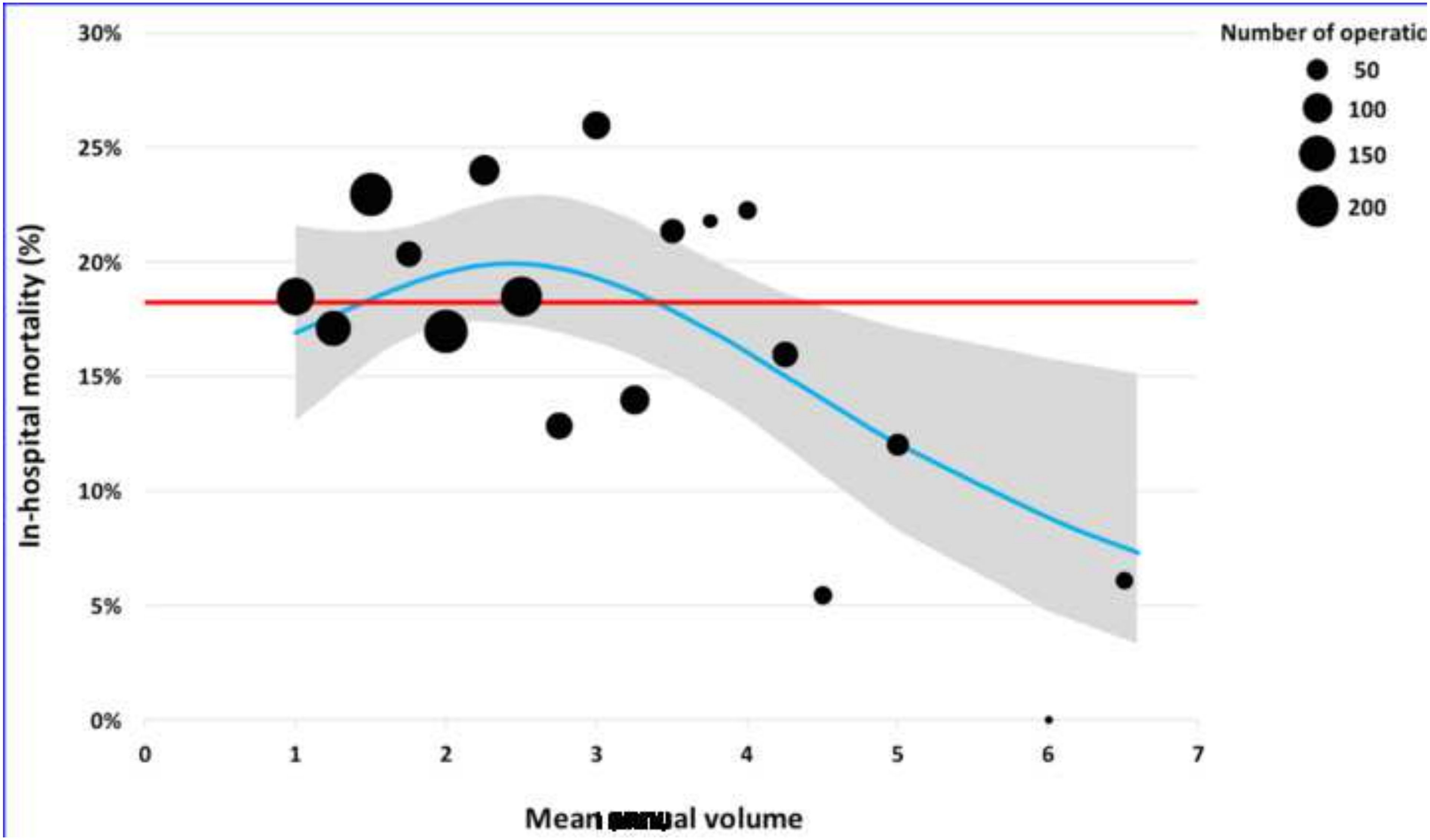
Figure 3: Panel chart showing the interaction between hospital and surgeon volume. The blue lines in each chart represent predicted probabilities of in-hospital mortality over the range of surgeon MAV for the 20th, 40th, 60th and 80th percentiles of hospital MAV, and the grey-shaded areas denote approximate 95% confidence intervals. Overall *P* value for interaction = 0.88

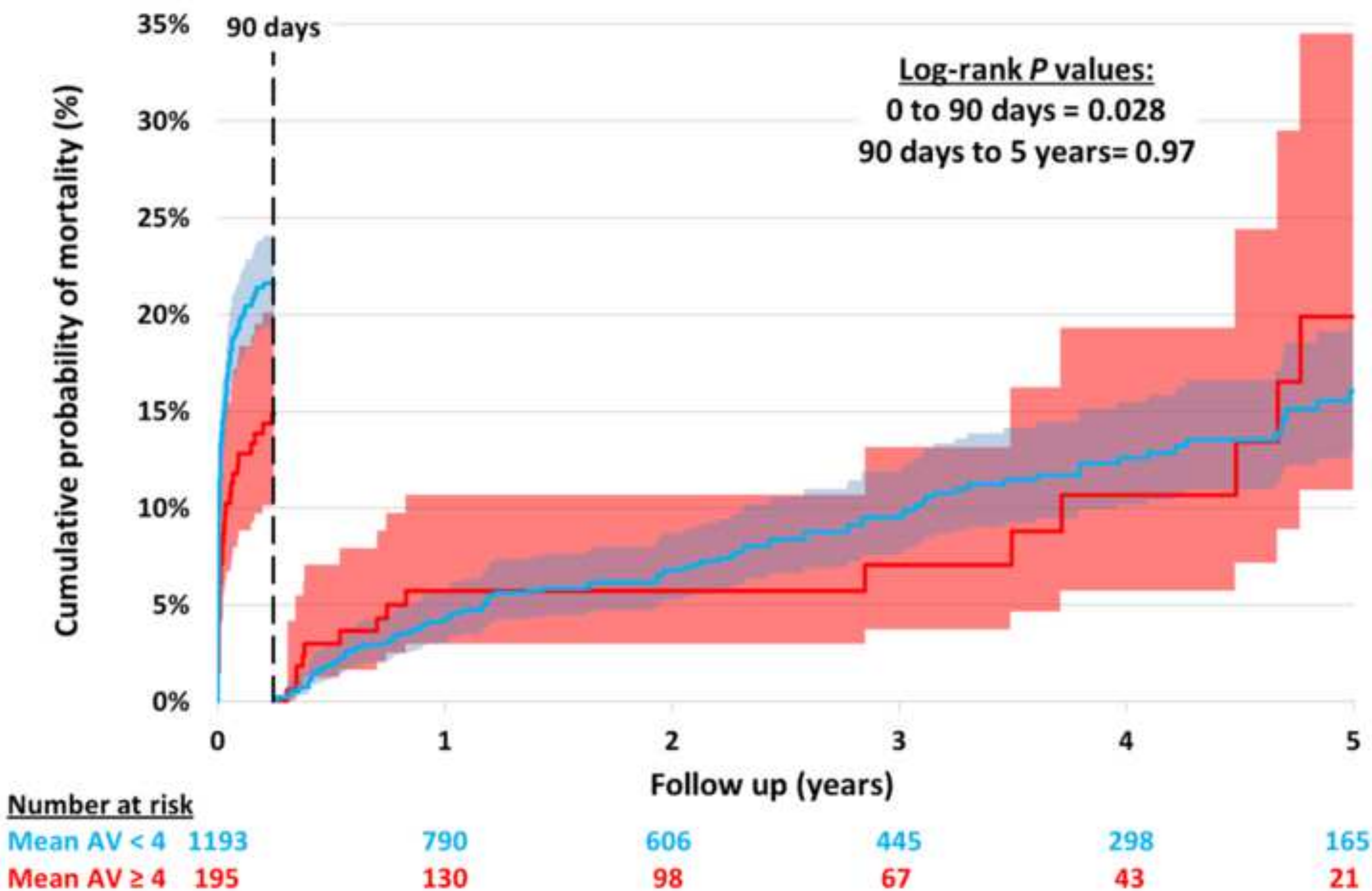


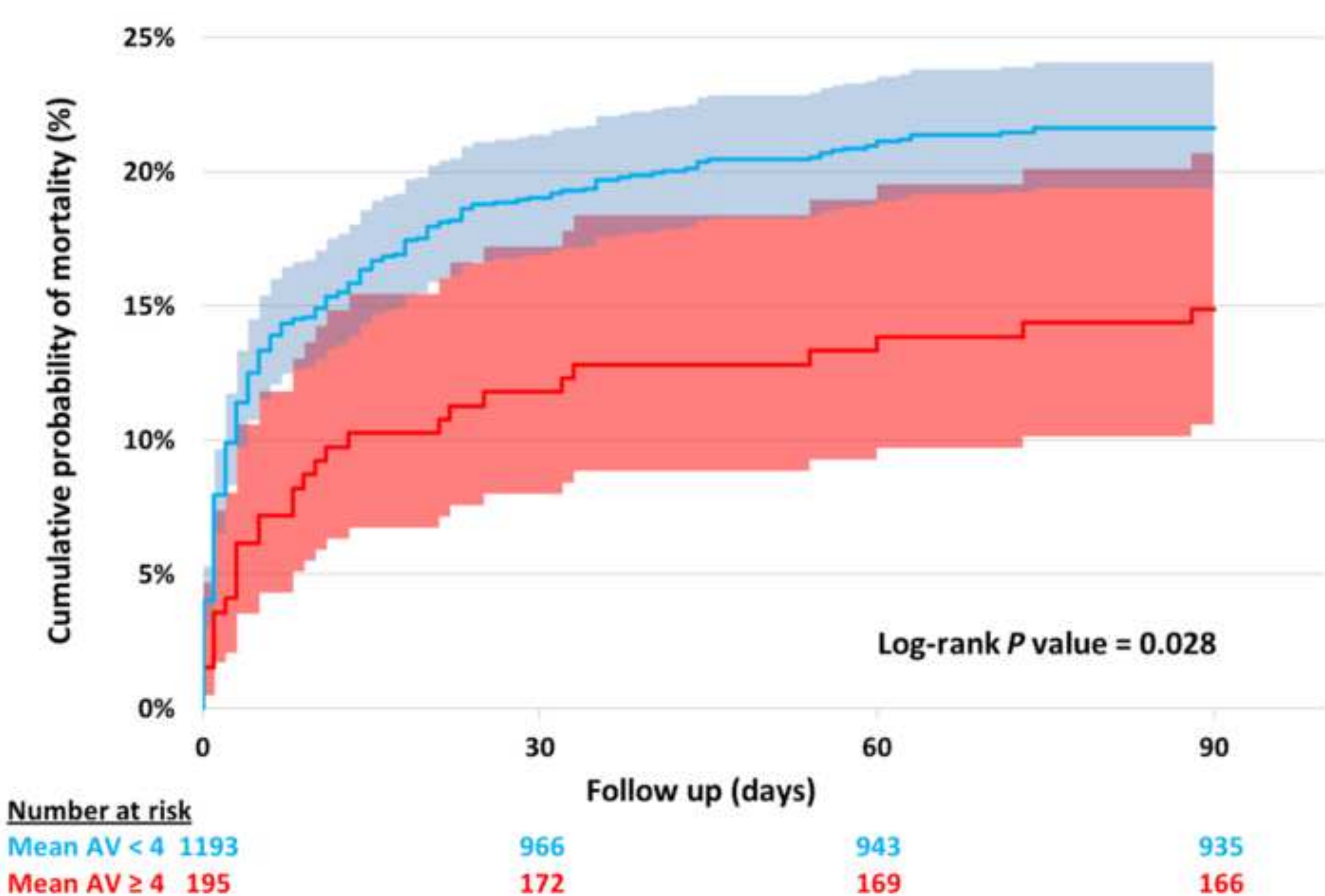
Supplemental Figure 1: Kaplan-Meier chart showing the cumulative probability of all-cause mortality for ATAD patients in the surgeon MAV groups. Colored bands show approximate 95% confidence intervals.

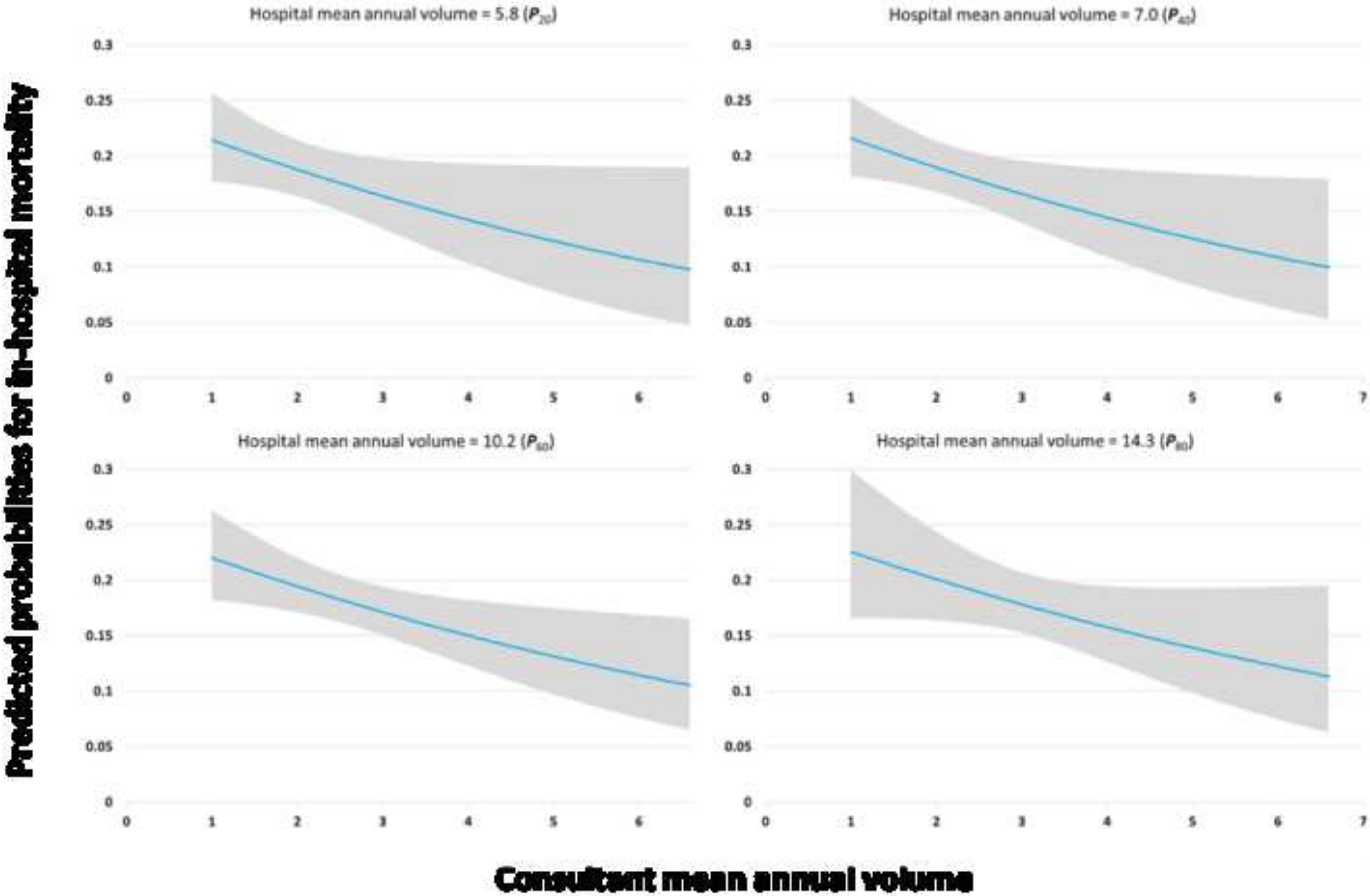


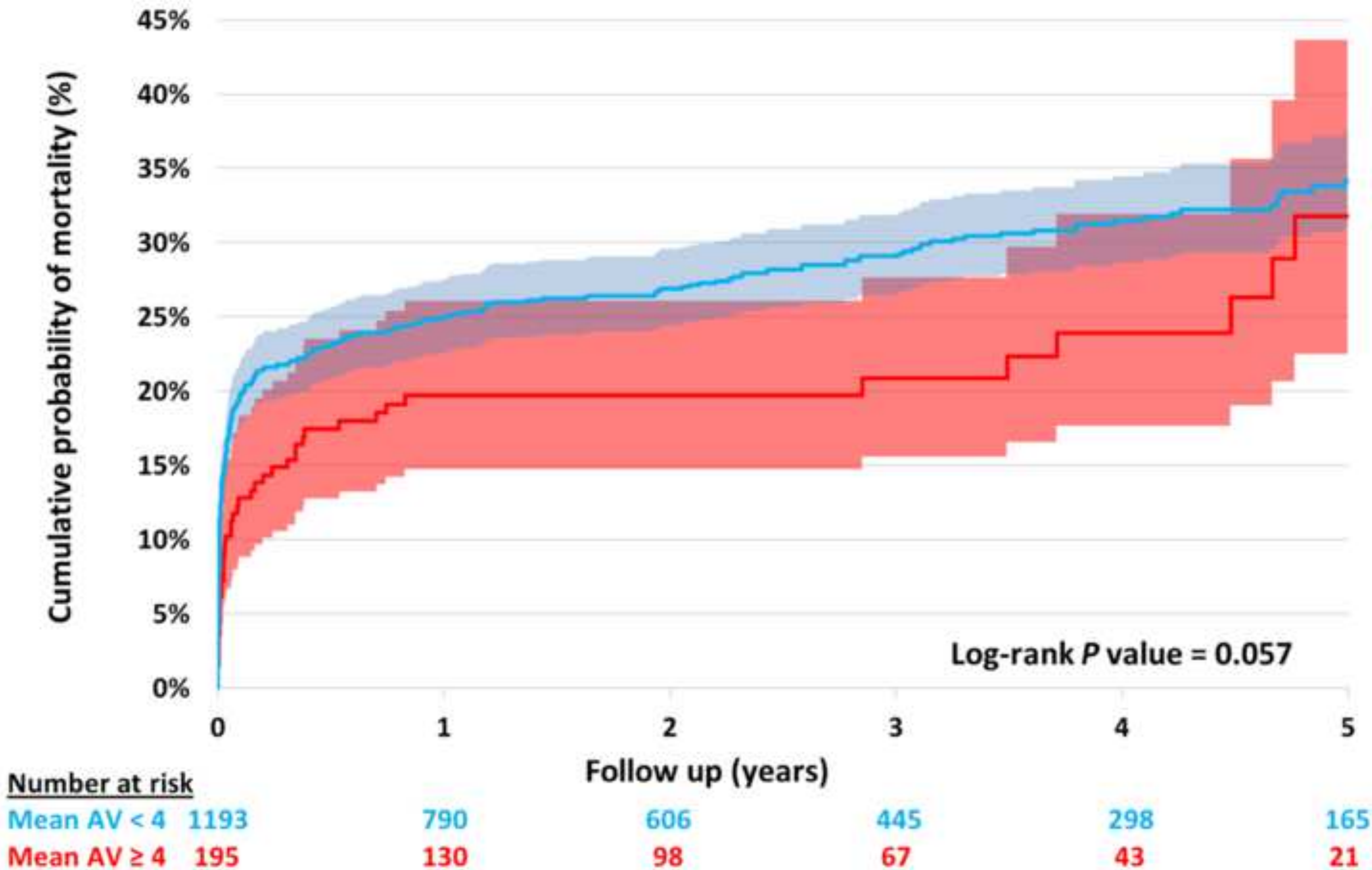
Figure(1) (see Info for Authors for details) Click here to download Figure(s) (see Info for Authors for details) ATAD figure1.tiff

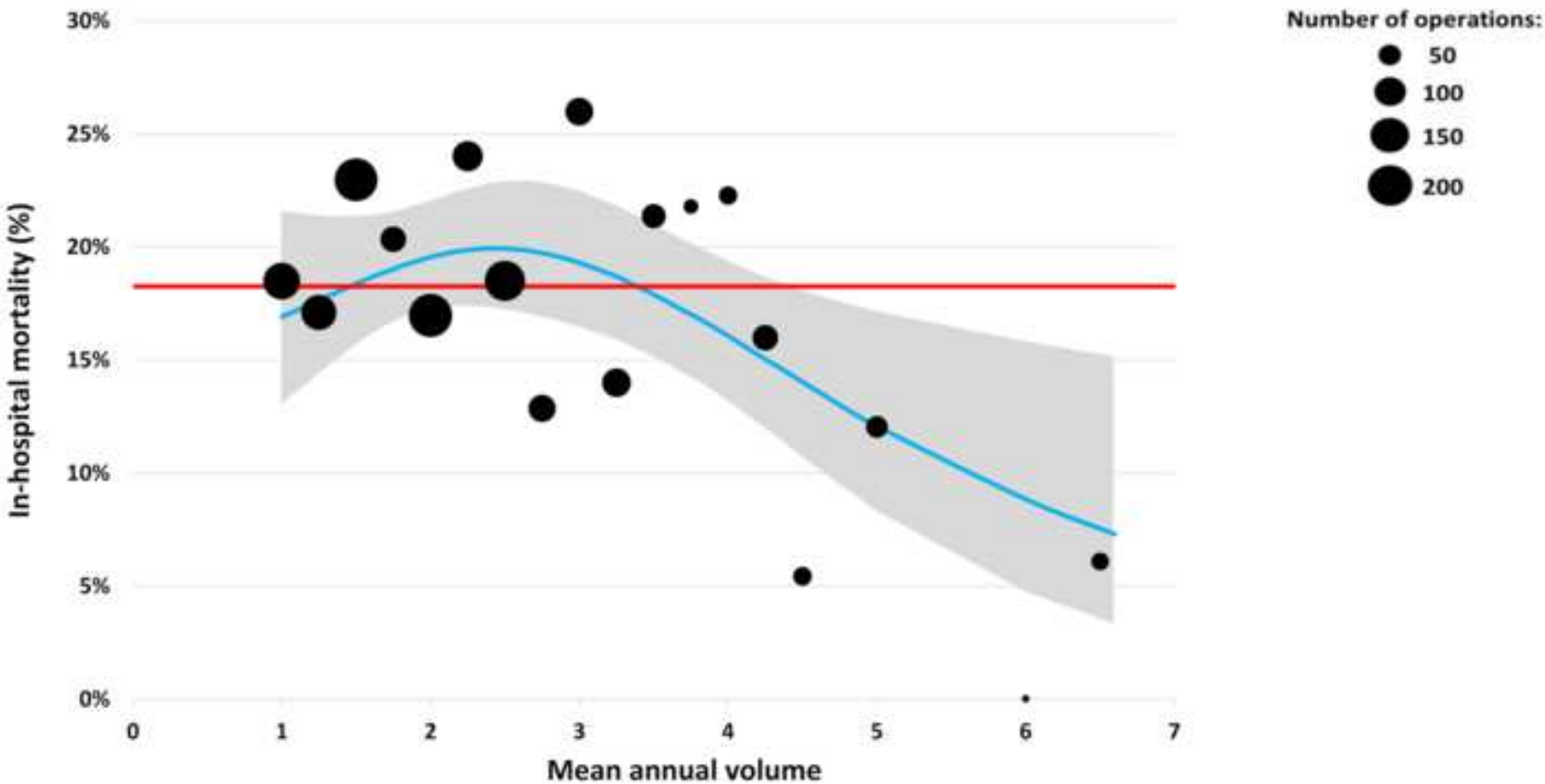



90 days
Log-rank P values:
0 to 90 days = 0.028
90 days to 5 years= 0.97
Cumulative probability of mortality (%)
35%
30%
25%
20%
15%
10%
5%
0%
0
1
2
3
4
5
Follow up (years)
Number at risk
Mean AV < 4 1193 790 606 445 298 165
Mean AV ≥ 4 195 130 98 67 43 21


25%
20%
15%
10%
5%
0%
Cumulative probability of mortality (%)
0
30
60
90
Follow up (days)
Log-rank P value = 0.028
Number at risk
Mean AV < 4 1193 966 943 935
Mean AV ≥ 4 195 172 169 166


Hospital mean annual volume = 5.8 (P_{20})
Hospital mean annual volume = 7.0 (P_{40})
Hospital mean annual volume = 10.2 (P_{60})
Hospital mean annual volume = 14.3 (P_{80})
Predicted probabilities for in-hospital mortality
Consultant mean annual volume
0.3
0.25
0.2
0.15
0.1
0.05
0
0 1 2 3 4 5 6 7


Cumulative probability of mortality (%)
45%
40%
35%
30%
25%
20%
15%
10%
5%
0%
Log-rank *P* value = 0.057
0
1
2
3
4
5
Follow up (years)
Number at risk
Mean AV < 4 1193 790 606 445 298 165
Mean AV ≥ 4 195 130 98 67 43 21



Number of operations:
50
100
150
200
30%
25%
20%
15%
10%
5%
0%
In-hospital mortality (%)
0
1
2
3
4
5
6
7
Mean annual volume

Table 3: Logistic regression analysis for in-hospital mortality

Variable	Odds ratio	95% confidence interval	*P* value
Surgeon mean annual volume	0.853	0.733 - 0.992	0.039
Hospital mean annual volume	1.005	0.956 - 1.057	0.84
Age at procedure (years)	1.028	1.015 - 1.041	<0.001
Previous cardiac surgery	1.840	1.052 - 3.218	0.033
Peripheral vascular disease	1.505	1.051 - 2.156	0.026
Left ventricular ejection fraction <30%	2.896	1.374 - 6.104	0.005
Pre-operative cardiogenic shock	1.722	1.137 - 2.607	0.010
Salvage procedure	5.474	2.790 - 10.741	<0.001
Concomitant CABG procedure	2.135	1.412 - 3.229	<0.001
Cardiopulmonary bypass time (mins)	1.003	1.002 - 1.005	<0.001

Table 1 - Patient characteristics - stratified by surgeon MAV group

Table 1: Patient characteristics, stratified by surgeon MAV group

	Mean annual volume < 4 (n=1319)	Mean annual volume ≥ 4 (n=231)	*P* value	Missing Data
Age at procedure (years)	63 (52, 72)	64 (53, 74)	0.10	0 (0)
Female gender	425 (32.2)	69 (29.9)	0.48	0 (0)
Body mass index (kg/m2)	26.4 (23.9, 29.7)	26.4 (24.2, 29.0)	0.54	48 (3.5)
Angina class IV	199 (15.1)	39 (16.9)	0.48	17 (1.1)
NYHA class ≥ III	354 (26.8)	68 (29.4)	0.41	23 (1.5)
Previous Q-wave MI	130 (9.9)	18 (7.8)	0.32	14 (0.9)
Recent MI (within 90 days)	83 (6.3)	4 (1.7)	0.006	10 (0.7)
Previous PCI	44 (3.3)	6 (2.6)	0.56	39 (2.5)
Previous cardiac surgery	75 (5.7)	20 (8.7)	0.08	127 (8.2)
Diabetes (diet or insulin controlled)	62 (4.7)	6 (2.6)	0.15	13 (0.8)
Current smoker	243 (18.4)	34 (14.7)	0.18	64 (4.1)
History of hypertension	905 (68.6)	167 (72.3)	0.26	27 (1.7)
Creatinine > 200 µmol/L	67 (5.1)	16 (6.9)	0.25	115 (7.4)
History of renal dysfunction	32 (2.4)	8 (3.5)	0.36	71 (4.6)
History of pulmonary disease	134 (10.2)	34 (14.7)	0.04	9 (0.6)
History of neurological disease	130 (9.9)	29 (12.6)	0.21	23 (1.5)
History of neurological dysfunction	93 (7.1)	20 (8.7)	0.39	15 (1.0)
Peripheral vascular disease	259 (19.6)	47 (20.4)	0.80	16 (1.0)
Non-sinus heart rhythm	120 (9.1)	18 (7.8)	0.52	99 (6.4)
Triple vessel disease	29 (2.2)	5 (2.2)	0.97	338 (21.8)
Left ventricular ejection fraction 30% - 50%	248 (18.8)	39 (16.9)	0.49	31 (2.0)
Left ventricular ejection fraction <30%	50 (3.8)	7 (3.0)	0.57	31 (2.0)
Intravenous nitrates or any heparin	200 (15.2)	29 (12.6)	0.30	6 (0.4)
Intravenous inotropes prior to anaesthesia	100 (7.6)	10 (4.3)	0.08	10 (0.7)
Pre-operative ventilation	77 (5.8)	12 (5.2)	0.70	15 (1.0)
Pre-operative cardiogenic shock	228 (17.3)	29 (12.6)	0.07	14 (0.9)

Continuous variables shown as median (25th percentile, 75th percentile); categorical variables shown as frequency (%)

NYHA = New York Heart Association, MI = Myocardial Infarction, PCI = Percutaneous Coronary Intervention

Table 2: Operative characteristics and in-hospital mortality, stratified by surgeon MAV group

	Mean annual volume < 4 (n=1319)	Mean annual volume ≥ 4 (n=231)	*P* value	Missing Data
Surgeon mean annual volume	2.0 (1.5, 2.7)	4.7 (4.3, 5.0)	<0.001	0 (0)
Hospital mean annual volume	8.7 (6.0, 13.2)	10.2 (8.7, 17.8)	<0.001	0 (0)
Elective procedure	21 (1.6)	3 (1.3)	>0.99	0 (0)
Urgent procedure	169 (12.8)	34 (14.7)	0.43	0 (0)
Emergency procedure	1037 (78.6)	181 (78.4)	0.93	0 (0)
Salvage procedure	92 (7.0)	13 (5.6)	0.45	0 (0)
Root segment	438 (33.2)	82 (35.5)	0.50	0 (0)
Ascending segment	1146 (86.9)	203 (87.9)	0.68	0 (0)
Arch segment	152 (11.5)	46 (19.9)	<0.001	0 (0)
Concomitant CABG procedure	171 (13.0)	30 (13.0)	0.99	30 (1.9)
Concomitant Valve procedure	521 (39.5)	99 (42.9)	0.34	29 (1.9)
Concomitant 'Other' cardiac procedure	395 (30.0)	76 (32.9)	0.37	34 (2.2)
Cardiopulmonary bypass time (mins)	196 (152, 259)	197 (154, 254)	0.86	44 (2.8)
Aortic cross clamp time (mins)	105 (74, 143)	109 (68, 147)	0.81	56 (3.6)
Circulatory arrest time (mins)	29 (20, 39)	20 (15, 31)	<0.001	402 (25.9)
In-hospital mortality	254 (19.3)	29 (12.6)	0.015	0 (0)

Continuous variables shown as median (25th percentile, 75th percentile); categorical variables shown as frequency (%)

CABG = Coronary Artery Bypass Grafting

Table 4 - Cox proportional hazards analysis for survival from procedure and from 90-days post-procedure

Table 4: Cox proportional hazards analysis for survival from procedure and from 90-days post-procedure

Variable	Start time = procedure date			Start time = 90 days post-procedure		
	Hazard ratio	95% confidence interval	*P* value	Hazard ratio	95% confidence interval	*P* value
Surgeon mean annual volume	0.882	0.801 - 0.972	0.011	0.920	0.779 - 1.088	0.33
Hospital mean annual volume	1.029	1.000 - 1.059	0.050	1.020	0.972 - 1.072	0.42
Age at procedure (years)	1.028	1.019 - 1.037	<0.001	1.043	1.023 - 1.063	<0.001
Left ventricular ejection fraction <30%	2.495	1.586 - 3.926	<0.001	5.799	2.169 - 15.505	<0.001
Pre-operative cardiogenic shock	1.426	1.068 - 1.903	0.016	0.880	0.459 - 1.687	0.70
Salvage procedure	3.250	2.139 - 4.965	<0.001	2.138	0.791 - 5.778	0.13
Arch segment	1.414	1.047 - 1.909	0.024	1.200	0.653 - 2.207	0.56
Concomitant CABG procedure	1.629	1.235 - 2.150	<0.001	1.215	0.636 - 2.323	0.56
Cardiopulmonary bypass time (mins)	1.003	1.002 - 1.004	<0.001	1.001	0.998 - 1.003	0.53

Original Article

Acute Type A aortic dissection in the United Kingdom: Surgeons volume-outcome relation

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Disclosures: The authors have no conflicts of interest or any sources of funding to declare.

Word Count: 4,233

Glossary:

AAD: Acute Aortic Dissection

ATAD: Acute Type A aortic dissection

IRAD: International Registry for Acute Dissection

SCTS: Society for Cardiothoracic Surgery

NICOR: National Institute for Cardiovascular Outcomes Research

TAVI: Transcatheter aortic valve implantations

NACSA: National Adult Cardiac Surgery Audit

CCS: Canadian Cardiovascular Society

NYHA: New York Heart Association

ONS: Office for National Statistics

MAV: Mean Annual case Volume

IQR: Inter-Quartile Range

ABSTRACT

Objectives

Surgery for acute Type A aortic dissection carries a high risk of operative mortality. We examined the surgeon volume-outcome relation with respect to in-hospital mortality for patients presenting with this pathology in the United Kingdom.

Method:

Between April 2007 and March 2013, 1550 acute Type A aortic dissection procedures were identified from the National Institute for Cardiovascular Outcomes Research database. 249 responsible consultant cardiac surgeons from the UK recorded one or more of these procedures in their surgical activity over this time period. We describe the patient population and mortality rates, focusing on the relationship between surgeon volume and in-hospital mortality.

Results:

The mean annual volume of procedures per surgeon during the 6-year period ranged from 1 to 6.6. The overall in-hospital mortality rate was 18.3% (283/1550). A mortality improvement at the 95% level was observed with a risk adjusted mean annual volume >4.5. Surgeons with a mean annual volume over the study period ≥4 had significantly higher in-hospital mortality rates in comparison to surgeons with a mean annual volume <4 (19.3% vs. 12.6%; P = 0.015).

Conclusion:

Acute Type A aortic dissection patients who are operated on by lower volume surgeons experience higher levels of in-hospital mortality. Directing these patients to higher volume surgeons may be a strategy to reduce in-hospital mortality.

Keywords: aorta, dissection, aneurysm, surgeon volume-outcome, AAD

INTRODUCTION

Acute Type A aortic dissection (ATAD) is a lethal condition and a cardiac surgical emergency. The incidence of aortic dissection is 30-43 per million population per year and this is incrementally increasing[1,2,3]. Forty percent of patients with dissection are aged 60 to 74 but 27% are aged 17 to 59 years, thus all ages are affected[4].

In medically treated patients, mortality rates are 1-2% per hour after the initial event, with death due to coronary or other organ malperfusion, cardiac tamponade, acute heart failure due to aortic regurgitation or aortic rupture. Emergency surgery can convert a 90% mortality rate at 30-days to a 75-90% long-term survival rate[4]. Mortality rates may vary, the International Registry for Acute Dissection (IRAD)[5] and the UK Society for Cardiothoracic Surgery (SCTS) 'Blue Book'[6] have published operative mortality rates of 25.1% and 22.8% respectively. In contrast, the German registry GERAADA published their series with lower rates of 17%[7]. This variation in reported mortality might be due to the volume-outcome relationship that has been at the center of debate and discussion. The Mount Sinai group utilizing the Nationwide Inpatient Sample of North America reported that lower-volume surgeons and centers have approximately double the risk-adjusted mortality of patients undergoing repair by the highest volume care providers[8].

This study aims to report the national UK surgeon outcomes in the operated ATAD patient population and explore the relationship in this population between surgeon volume and adjusted in-hospital mortality.

METHODS

NICOR database

Prospectively collected data were extracted from the National Institute for Cardiovascular Outcomes Research (NICOR) National Adult Cardiac Surgery Audit (NACSA) registry (version 4.1.2) on 20th November 2014 for all adult cardiac surgery procedures performed in the UK. As described elsewhere, reproducible cleaning algorithms were applied to the database[9]. Briefly, duplicate records and non-adult cardiac surgery entries [including transcatheter aortic valve implantations (TAVIs)] were removed, transcriptional discrepancies harmonized and clinical and temporal conflicts and extreme values corrected or removed. Data summaries are returned regularly to each unit for local validation as part of the NACSA in the UK[10].

For this study, records were included that corresponded to the following criteria: procedure on one or more of the root, ascending or arch aortic segments with a recorded pathology of "Acute Dissection" that were performed in England and Wales between 1st April 2007 and 31st March 2013. Records missing responsible consultant cardiac surgeon data (recorded in the form of General Medical Council registration number) were excluded.

Baseline and operative variables

For each procedure, data are recorded on patient characteristics, comorbidities, surgical team, intraoperative factors and postoperative outcomes. For this study, we extracted data on patient age at the time of procedure (years), gender, body mass index [BMI, defined as weight (kg) / height^2 (m^2)], Canadian Cardiovascular Society (CCS) angina class, dyspnoea (dichotomized as New York Heart Association (NYHA) grade < III and NYHA grade ≥ III), recent myocardial infarction (defined as within 90 days of surgery), history of major cardiac surgery, diabetes (diet or insulin controlled), smoking status, history of hypertension, serum creatinine >200 μmol/l, history of renal dysfunction, history of pulmonary disease, history of neurological dysfunction, extracardiac arteriopathy, preoperative heart rhythm, left ventricular ejection fraction (classified as <30, 30–50 and >50%), IV inotropes prior to anaesthesia, preoperative ventilation, preoperative cardiogenic shock, operative urgency, concomitant CABG and valve procedures, cardiopulmonary bypass time, aortic cross-clamp time, and circulatory arrest time.

Administrative data were also extracted including: patient admission, procedure and discharge dates, responsible consultant cardiac surgeon and anonymized hospital identifier. Further details of variable definitions are available at: http://www.ucl.ac.uk/nicor/audits/adultcardiac/datasets.

Outcomes

The primary outcome for this study was in-hospital mortality, defined as death due to any cause during admission to the base hospital for cardiac surgery. The secondary outcome was mid-term mortality followed up to 5 years. Follow-up data up until the point of discharge was collected by the NACSA clinical registry system and post-discharge survival data was collected by linking the records via patient NHS numbers to the Office for National Statistics (ONS) death registry, which records all deaths in England and Wales. The final date of follow-up was 30th July 2013. Data on cause of death was unavailable. An attempt to back-fill missing in-hospital mortality data was made by record linkage to the ONS registry prior to applying the extraction criteria.

Case volumes

For analytical purposes, case volumes are presented both continuously and categorically. In the continuous analysis, the mean annual case volume (MAV) was calculated. This was achieved by taking the total number of procedures for each surgeon and dividing this by the number of years in which they contributed data to the registry. In the categorical analysis, the case volume was stratified into two groups (surgeon MAV of ATAD procedures <4 and ≥ 4 over the study period). The cut point was selected as being clinically meaningful after the introductory analysis showed it to be the approximate inflection point for improved mortality.

Statistical analysis

Categorical and dichotomous variables are summarized as absolute number and percentage. Non-normally distributed continuous data are summarized as median and inter-quartile range (IQR). The prevalence of missing data in the registry for baseline and operative measurements, as well as in-hospital mortality, are reported. Due to the relatively low number of missing data items for the majority of the variables, categorical variables with missing data were imputed with the baseline category and continuous variables were imputed with the mean value before calculations were performed.

Where categorical comparisons are made between groups the chi-squared test was used, for similar comparisons between continuous variables the Wilcoxon rank-sum test was used.

To quantify the relationship between MAV and in-hospital mortality we performed three separate multivariable regression analyses. Firstly, an initial balancing score was fitted for each patient using a mixed-effects linear regression model. The dependent continuous variable used was a log transformation of the surgeon MAV, with random intercepts for each hospital, and the following patient variables were entered as independent variables: age, gender, body mass index, smoking, renal failure, hypertension, pulmonary disease, neurological disease, neurological dysfunction, peripheral vascular disease, recent myocardial infarction, unstable angina, arrhythmia, New York Heart Association class, previous cardiac surgery, diabetes, ejection fraction, cardiogenic shock, pre-operative ventilation, operative urgency, cardiopulmonary bypass time, circulatory arrest time, surgery on the aortic arch and concomitant procedures. The volume-outcome relationship was then assessed by plotting estimated restricted cubic spline regression functions with three knots between the surgeon MAV and in-hospital mortality; these were then adjusted for patient case-mix by entering the balancing score into the regression model. The spline estimates were based on a standard, fixed effects logistic regression model. The balancing score therefore acts in a similar way to a propensity score[11,12], but is generalized beyond a dichotomous treatment assignment.

Secondly, in order to examine the contribution of hospital volume to outcome, a logistic regression model for in-hospital mortality was fitted including random intercepts for each hospital, with surgeon and hospital MAV entered into the model as continuous variables, along with the independent demographical and procedural variables listed above, interactional terms between hospital and surgeon MAV were also explored.

Thirdly, a similar approach was taken to assessing the contribution of MAV to mid-term mortality. Cox proportional hazards models were fitted, again with random intercepts for each hospital and with surgeon and hospital MAV entered into the model as continuous variables, along with the independent demographical and procedural variables listed above. In order to demonstrate any non-proportional effects of early mortality two models were fitted, one with a start time of the procedure date and the second with a start time of 90-days post-procedure.

To evaluate the categorical difference in volume, Kaplan-Meier charts were used to plot the actuarial 5-year survival, incorporating a landmark cutoff of 90 days where the groupwise mortality rates were rebased to zero. The log-rank test was used to assess the equivalence of death rates between groups in both phases of the analysis.

Statistical analyses were performed with SAS version 9.3 (SAS Institute, Cary, NC). In all cases a P value <0.05 was considered statistically significant.

RESULTS

Characteristics of the study population

1632 patients were identified from the NACSA database as having had surgery for ATAD during the time period. Patients who had surgery on the descending and/or thoracoabdominal segments of the aorta (n=63; 3.9%), and 19 (1.2%) records that lacked responsible clinician data were excluded from the analysis. The 1550 that remained were included in the study analysis dataset, however 162 (10.5%) records lacked follow-up mortality data and are excluded from that element of the analysis.

The 1550 patients were admitted to 41 different hospitals throughout England and Wales and were under the care of 249 different consultant cardiac surgeons. The mean surgeon MAV was 2.6 (SD = 1.2; median (IQR) = 2.3 (1.6, 3.3), with 199 of 249 surgeons (79.9%) performing fewer than 10 procedures overall. A total of 41 surgeons performed a single ATAD procedure. The highest number of procedures performed by a single surgeon during the study period was 33. The mean hospital MAV was 9.6 (SD = 4.6; median (IQR) = 8.7 (6.0, 13.2). The highest number of procedures performed by a single hospital during the study period was 107.

Pre-operative and operative differences between the two categorical groups are shown in Tables 1 and 2. Surgeons in the lower MAV group were more likely to operate on patients who had a recent MI, whilst being less likely to operate on patients with a history of pulmonary disease or patients who required surgery on the aortic arch. Surgeons in the lower MAV group also reported significantly longer circulatory arrest times.

In-hospital mortality and case volume

The overall in-hospital mortality rate for all ATAD patients was 18.3% (283 patients). Figure 1 plots the observed in-hospital mortality against the adjusted surgeon MAV. Somewhat counter-intuitively, the curve begins below the national mean rate at the lowest volumes then rises and peaks between 2 and 3 procedures per year, before decreasing in an approximate linear trend in higher volume surgeons. Significant in-hospital mortality improvements can be observed beyond a surgeon MAV of 4 to 4.5.

The unadjusted in-hospital mortality rate decreased from 19.3% in the group of surgeons who had a MAV <4 during the study period to 12.6% in the group who had a MAV ≥4; *P* = 0.015 (Table 2). Figure 2a illustrates the groupwise trends in 5-year follow up mortality rates, including a landmark rebasing at 90 days. The early difference in mortality rates is significant at the 0.05 level (log-rank test *P* value = 0.028), however this difference is not sustained in the second era, from 90 days to 5 years (P = 0.97). (Figure 2b is a detail from Figure 2a which charts the 90 day mortality only, included for clarity).

The logistic regression model shown in Table 3 demonstrates a similar in-hospital mortality advantage for surgeons who perform a greater number of operations, after adjustment for casemix and hospital volume, increasing surgeon MAV (assessed as a continuous variable) was associated with a significant reduction to in-hospital mortality (adjusted OR=0.853 (95% CI 0.733 to 0.992) P = 0.039). Other associated variables were: increasing age, previous cardiac surgery, peripheral vascular disease, left ventricular ejection fraction <30%, cardiogenic shock, salvage priority, concomitant CABG procedure, and increasing cardiopulmonary bypass time. Hospital MAV was not associated with a difference in in-hospital mortality (adjusted OR=1.005 (95% CI 0.956 to 1.057) P = 0.84).

Figure 3 includes four charts which show the interaction between surgeon and hospital MAV with regards to in-hospital mortality. The predicted probabilities of in-hospital mortality over the range of surgeon MAV are shown for the 20^{th}, 40^{th}, 60^{th} and 80^{th} percentiles of hospital MAV (which are, respectively, 5.8, 7.0, 10.2 and 14.3 cases per year). Visual inspection of these allows us to infer that there are no substantial differences in the relationship between surgeon MAV and in-hospital mortality as hospital MAV increases. The associated interaction test P value = 0.88.

Follow-up mortality and case volume

The results of the two Cox proportional hazard models are shown in Table 4. In the 'Start time = procedure date' model, higher surgeon MAV (assessed as a continuous variable) was again associated with a significant reduction in death (adjusted HR=0.882 (95% CI 0.801 to 0.972) *P* = 0.011). Other variables that were associated with a greater hazard of death were: increasing age, left ventricular ejection fraction <30%, cardiogenic shock, salvage priority, surgery on the aortic arch, concomitant CABG procedure and increasing cardiopulmonary bypass time. Hospital MAV was not significantly associated with a difference in death (adjusted HR=1.029 (95% CI 1.000 to 1.059) *P* = 0.050).

In the 'Start time = 90 days post-procedure' model, higher surgeon MAV was not associated with a significant reduction in death (adjusted HR=0.920 (95% CI 0.779 to 1.088) *P* = 0.33). Other variables that were associated with a greater hazard of death were: increasing age and left ventricular ejection fraction <30%. Hospital MAV was not significantly associated with a difference in death (adjusted HR=1.020 (95% CI 0.972 to 1.072) *P* = 0.42), suggesting that the significant effect observed in the 'Start time = procedure date' model is both non-proportional and also greatly reliant on the large early mortality burden.

DISCUSSION

It has been shown that out of every 1,000 emergency department patients presenting with acute back, chest, or abdominal pain, three patients with ATAD are diagnosed[13]. This is a fatal condition with a dire prognosis unless the patient receives immediate surgical intervention. The IRAD has published outcomes from multiple centres worldwide with an average in-hospital mortality of 25.1% in 2005[14]. European registries in the United Kingdom and Germany have published operative mortalities of 23.1% and 17% respectively[15]. A recent publication from Mount Sinai Medical Centre, using the Nationwide Inpatient Sample database of 5,184 patients between

2003 and 2008, showed average operative mortality of 21.6%[8Error! Bookmark not defined.]. However, in the advent of this decade there are multiple centres worldwide that are publishing in-hospital mortality rates for ATAD in the single digits[16171819].

Throughout the United States the model of aortic supercentres with high referral rates have existed for some time. It has been suggested that outcomes in thoracic aortic surgery could be improved nationwide in the United States if the acute care and emergency surgical treatment of most patients with ATAD were regionalized and restricted to institutions with high-volume multidisciplinary thoracic aortic surgery programs[20].

Andersen et al. from the Duke group published their results pertaining to the impact model of a multi-disciplinary team approach to ATAD[17]. They reported operative mortality before multi-disciplinary implementation was 33.9% and was statistically equivalent to the expected operative mortality rate of 26.0%. Operative mortality after multi-disciplinary implementation fell to 2.8% and was statistically better than the expected operative mortality rate of 18.2% using the International Registry of Acute Aortic Dissection pre-operative prediction model.

In the UK, centralization of expertise and service provision for type A aortic dissection does not exist. Operating on ATAD does not generally follow a selective referral protocol which in effect leads to a national mix and match between high and low volume surgeons. Compounding this is the lack of best practice evidence on structured referral from emergency room to operating room. This is due to multiple factors which unfortunately have not been quantified in the UK. Those factors are in essence related to lack of swift recognition of ATAD. There is also lack of substantial evidence on the actual time to referral once ATAD is actually identified. The aforementioned mandate a policy that will serve better patients' outcome and results across the UK.

Evidence of improved outcomes related to operative volume or surgeon expertise is often difficult to establish due to the infrequent nature of ATAD and consequent lack of statistical power that could potentially provide meaningful analysis[21].

The ideal definition of volume is inherently inconsistent; this makes diving into a discussion involving 'volume' highlight caveats that are not potentially attained or addressed between cardiac surgical units at large. The strong rationale of the volume-outcome relationship as reflected in literature springs from the catalyst for subspecialisation and centralization of aortic services. It is to provide centres with a large and reaching catchment areas the reciprocal improvement and effect on the subspecialized unit. It allows more robust referral to influx and therefore maintain an adequate voluminous exposure. Essentially, in the UK, thoracic aortic aneurysm service is in much need of such an approach and a national policy and mandate that would support such programs across the UK should be warranted. This should provide a sustained increase in volume to concentrated expertise that would allow the possibility to address dire surgical diseases and avert associated complications. It will then reciprocate this arrangement by ultimately reducing mortality and perhaps improving survival and aftercare post-surgery[22]. Beside this, the advent of technological superiority in diagnosis and surgical planning of aortic surgery and the understanding of the natural history is resulting in personalized and targeted therapies and surgical procedures to be done on a wider range of the affected population. This has allowed for such cost-effective diagnostic tests to be distributed to a smaller number of regional centres and for them to operate on this patient cohort. Hence, this has titrated the inexistence of specialist centres and diverted a large number of patients to be operated at local lower-volume institutions.

The development of standardization and subspecialisation of acute aortic services requires a comprehensive assessment of the current status in aortic surgery in the UK. As such, our analysis has demonstrated that there is significant variation of in-hospital mortality around the country with little relationship between volume and outcome at a hospital level. These results are contrary to those demonstrated by international groups including the Mount Sinai group utilizing the Nationwide Inpatient Sample of North America who reported that lower-volume surgeons and centres have approximately double the risk-adjusted mortality of patients undergoing repair by the highest volume care providers[23].

Our study does however demonstrate that higher individual surgeon volume was associated with lower in-hospital mortality. These relationships could be explained by a number of different factors such as; case mix per individual surgeon, selection bias and variations in turn down practice, concentration of expertise to a particular surgeon within a hospital and inadvertent subspecialisation of a surgeon with interests to aortic interventions. On the other hand, and as demonstrated in the context of the analysis; difference between low and high volume surgeons clearly point out the shorter aortic cross clamp and cardiopulmonary time but increased circulatory arrest time in the low-volume group which could be related to attempt of more frequent use or extended repair entailing arch segment replacement. Although this is not entirely understood, one way of scoping this further would be to look at this element within the cohort and run a thorough factor analysis

While this study demonstrates good overall mortality rates for ATAD in the UK, it is likely that further improvements could be achieved through the introduction of a quality improvement programme for ATAD surgery. It is vital that such a programme is implemented across the multidisciplinary thoracic aortic surgery team including anaesthesia, postoperative surgical intensive care and operative perfusion specialists. Such a programme should also involve standardized referral pathways and treatment protocols for ATAD repair. Another important contributing factor would be the development and implementation of a robust referral system and an initiative to hospital managers and commissioners for centralization of expertise in ATAD repair. This will reduce the waiting time and taxing of ATAD patients unnecessarily in acute services while diagnostics are being carried out.

Limitations

The main limitation of this study is its retrospective nature and the variable nature of data quality between institutions in the UK. There are also several confounding variables to consider that are not available in the NICOR dataset. The foremost of these factors is probably case selection: surgeons at tertiary referral centres are likely to operate on patients significantly longer after the acute event than local units; consequently their surgical outcomes may benefit from both temporal patient selection (more stable patients are more likely to survive transfer), and more aggressive individual patient selection informed by the additional complications (such as malperfusion syndromes) that manifest hours to days after initial presentation. Other possible confounding variables include delays between diagnosis and intervention, referral bias and clustering, and presence, severity, and duration of end-organ ischemia.

CONCLUSIONS

Concentration of expertise and volume to appropriate surgeons who perform increasingly more complex aortic cases would be required to change the current paradigm of ATAD outcomes in the UK. Whenever feasible, ATAD repair should be performed by a high volume surgeon in order to reduce operative mortality. It is reasonable to suggest a national standardization and quality improvement framework for ATAD treatment.

ACKNOWLEDGEMENTS

The authors acknowledge all members of the Society for Cardiothoracic Surgery in Great Britain and Ireland who contribute data to the National Adult Cardiac Surgery Audit registry. The National Institute for Cardiovascular Outcomes Research, UCL, London, provided the data for this study.

DATA SHARING

The United Kingdom National Adult Cardiac Surgery Audit registry is available to researchers upon application to the National Institute of Cardiovascular Outcomes Research (NICOR), University College London. Full details on the NICOR data sharing application process are available at https://www.ucl.ac.uk/nicor/access/application [last accessed: 22nd December 2016].

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Table 1: Patient characteristics, stratified by surgeon MAV group

	Mean annual volume < 4 (n=1319)	Mean annual volume ≥ 4 (n=231)	*P* value	Missing Data
Age at procedure (years)	63 (52, 72)	64 (53, 74)	0.10	0 (0)
Female gender	425 (32.2)	69 (29.9)	0.48	0 (0)
Body mass index (kg/m2)	26.4 (23.9, 29.7)	26.4 (24.2, 29.0)	0.54	48 (3.5)
Angina class IV	199 (15.1)	39 (16.9)	0.48	17 (1.1)
NYHA class ≥ III	354 (26.8)	68 (29.4)	0.41	23 (1.5)
Previous Q-wave MI	130 (9.9)	18 (7.8)	0.32	14 (0.9)
Recent MI (within 90 days)	83 (6.3)	4 (1.7)	0.006	10 (0.7)
Previous PCI	44 (3.3)	6 (2.6)	0.56	39 (2.5)
Previous cardiac surgery	75 (5.7)	20 (8.7)	0.08	127 (8.2)
Diabetes (diet or insulin controlled)	62 (4.7)	6 (2.6)	0.15	13 (0.8)
Current smoker	243 (18.4)	34 (14.7)	0.18	64 (4.1)
History of hypertension	905 (68.6)	167 (72.3)	0.26	27 (1.7)
Creatinine > 200 µmol/L	67 (5.1)	16 (6.9)	0.25	115 (7.4)
History of renal dysfunction	32 (2.4)	8 (3.5)	0.36	71 (4.6)
History of pulmonary disease	134 (10.2)	34 (14.7)	0.04	9 (0.6)
History of neurological disease	130 (9.9)	29 (12.6)	0.21	23 (1.5)
History of neurological dysfunction	93 (7.1)	20 (8.7)	0.39	15 (1.0)
Peripheral vascular disease	259 (19.6)	47 (20.4)	0.80	16 (1.0)
Non-sinus heart rhythm	120 (9.1)	18 (7.8)	0.52	99 (6.4)
Triple vessel disease	29 (2.2)	5 (2.2)	0.97	338 (21.8)
Left ventricular ejection fraction 30% - 50%	248 (18.8)	39 (16.9)	0.49	31 (2.0)
Left ventricular ejection fraction <30%	50 (3.8)	7 (3.0)	0.57	31 (2.0)
Intravenous nitrates or any heparin	200 (15.2)	29 (12.6)	0.30	6 (0.4)
Intravenous inotropes prior to anaesthesia	100 (7.6)	10 (4.3)	0.08	10 (0.7)
Pre-operative ventilation	77 (5.8)	12 (5.2)	0.70	15 (1.0)
Pre-operative cardiogenic shock	228 (17.3)	29 (12.6)	0.07	14 (0.9)

Continuous variables shown as median (25th percentile, 75th percentile); categorical variables shown as frequency (%)

NYHA = New York Heart Association, MI = Myocardial Infarction, PCI = Percutaneous Coronary Intervention

Table 2: Operative characteristics and in-hospital mortality, stratified by surgeon MAV group

	Mean annual volume < 4 (n=1319)	Mean annual volume ≥ 4 (n=231)	*P* value	Missing Data
Surgeon mean annual volume	2.0 (1.5, 2.7)	4.7 (4.3, 5.0)	<0.001	0 (0)
Hospital mean annual volume	8.7 (6.0, 13.2)	10.2 (8.7, 17.8)	<0.001	0 (0)
Elective procedure	21 (1.6)	3 (1.3)	>0.99	0 (0)
Urgent procedure	169 (12.8)	34 (14.7)	0.43	0 (0)
Emergency procedure	1037 (78.6)	181 (78.4)	0.93	0 (0)
Salvage procedure	92 (7.0)	13 (5.6)	0.45	0 (0)
Root segment	438 (33.2)	82 (35.5)	0.50	0 (0)
Ascending segment	1146 (86.9)	203 (87.9)	0.68	0 (0)
Arch segment	152 (11.5)	46 (19.9)	<0.001	0 (0)
Concomitant CABG procedure	171 (13.0)	30 (13.0)	0.99	30 (1.9)
Concomitant Valve procedure	521 (39.5)	99 (42.9)	0.34	29 (1.9)
Concomitant 'Other' cardiac procedure	395 (30.0)	76 (32.9)	0.37	34 (2.2)
Cardiopulmonary bypass time (mins)	196 (152, 259)	197 (154, 254)	0.86	44 (2.8)
Aortic cross clamp time (mins)	105 (74, 143)	109 (68, 147)	0.81	56 (3.6)
Circulatory arrest time (mins)	29 (20, 39)	20 (15, 31)	<0.001	402 (25.9)
In-hospital mortality	254 (19.3)	29 (12.6)	0.015	0 (0)

Continuous variables shown as median (25th percentile, 75th percentile); categorical variables shown as frequency (%)

CABG = Coronary Artery Bypass Grafting

Table 3: Logistic regression analysis for in-hospital mortality

Variable	Odds ratio	95% confidence interval	*P* value
Surgeon mean annual volume	0.853	0.733 - 0.992	0.039
Hospital mean annual volume	1.005	0.956 - 1.057	0.84
Age at procedure (years)	1.028	1.015 - 1.041	<0.001
Previous cardiac surgery	1.840	1.052 - 3.218	0.033
Peripheral vascular disease	1.505	1.051 - 2.156	0.026
Left ventricular ejection fraction <30%	2.896	1.374 - 6.104	0.005
Pre-operative cardiogenic shock	1.722	1.137 - 2.607	0.010
Salvage procedure	5.474	2.790 - 10.741	<0.001
Concomitant CABG procedure	2.135	1.412 - 3.229	<0.001
Cardiopulmonary bypass time (mins)	1.003	1.002 - 1.005	<0.001

Table 4: Cox proportional hazards analysis for survival from procedure and from 90-days post-procedure

Variable	Start time = procedure date			Start time = 90 days post-procedure		
	Hazard ratio	95% confidence interval	*P* value	Hazard ratio	95% confidence interval	*P* value
Surgeon mean annual volume	0.882	0.801 - 0.972	0.011	0.920	0.779 - 1.088	0.33
Hospital mean annual volume	1.029	1.000 - 1.059	0.050	1.020	0.972 - 1.072	0.42
Age at procedure (years)	1.028	1.019 - 1.037	<0.001	1.043	1.023 - 1.063	<0.001
Left ventricular ejection fraction <30%	2.495	1.586 - 3.926	<0.001	5.799	2.169 - 15.505	<0.001
Pre-operative cardiogenic shock	1.426	1.068 - 1.903	0.016	0.880	0.459 - 1.687	0.70
Salvage procedure	3.250	2.139 - 4.965	<0.001	2.138	0.791 - 5.778	0.13
Arch segment	1.414	1.047 - 1.909	0.024	1.200	0.653 - 2.207	0.56
Concomitant CABG procedure	1.629	1.235 - 2.150	<0.001	1.215	0.636 - 2.323	0.56
Cardiopulmonary bypass time (mins)	1.003	1.002 - 1.004	<0.001	1.001	0.998 - 1.003	0.53

Figure 1: Trend in mean annual volume (MAV) of ATAD activity and observed mortality. Each black dot corresponds to the mean mortality (vertical axis) for ATAD procedures performed by consultant surgeons with a specific MAV (horizontal axis). The size of the black dots is proportional to the total number of ATAD procedures performed by surgeons with the given MAV. Please note that although volume was modelled continuously, to improve legibility the number of surgeon procedures is grouped for every 0.25 of a year, therefore each dot can be comprised of multiple consultant surgeons. The blue line is a fitted smoothing curve to illustrate the trend, adjusted for pre-operative risk factors, and the grey-shaded area denotes approximate 95% confidence intervals. The red horizontal line represents the overall mean observed in-hospital mortality (18.3%) for the study cohort.

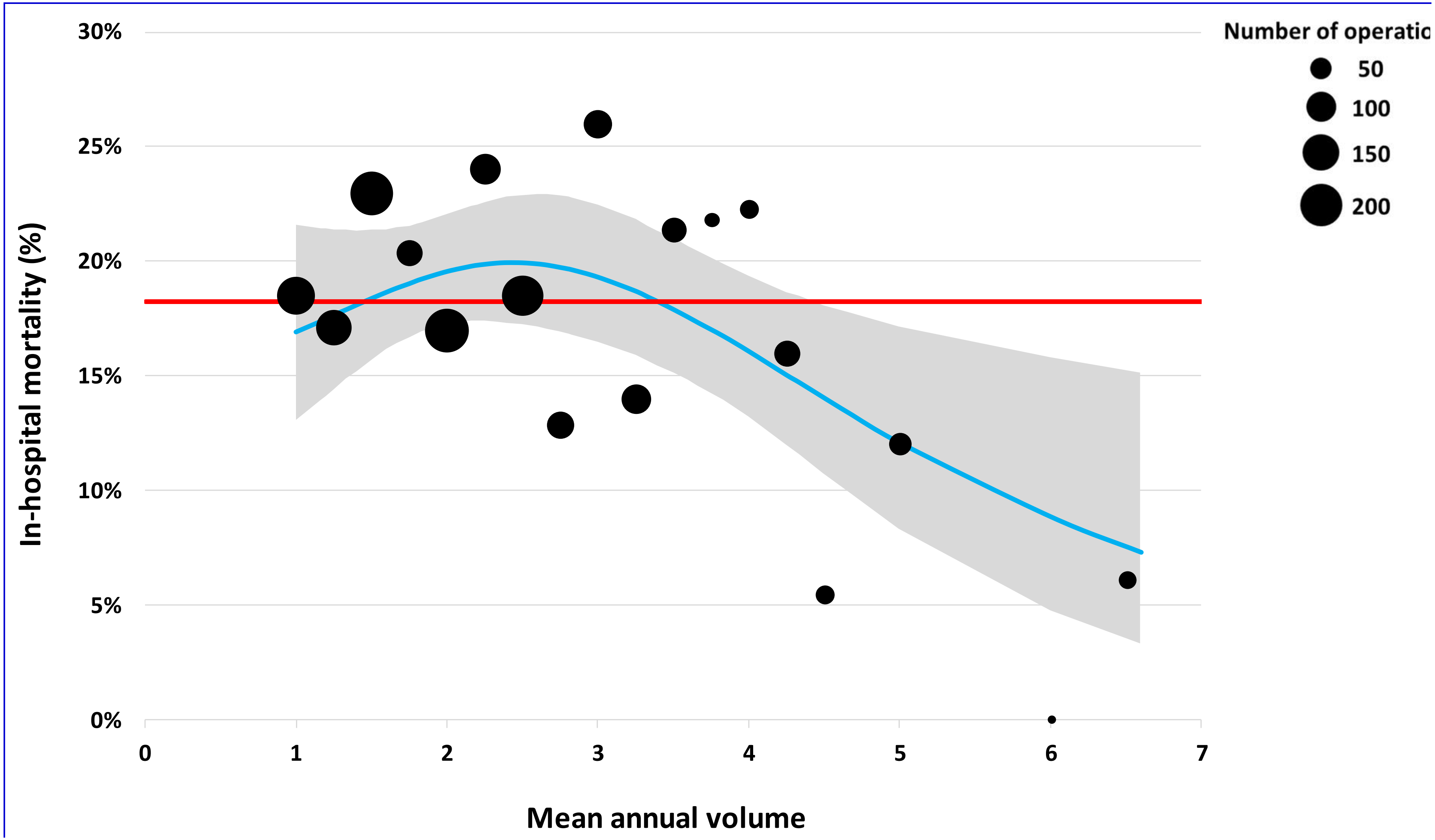
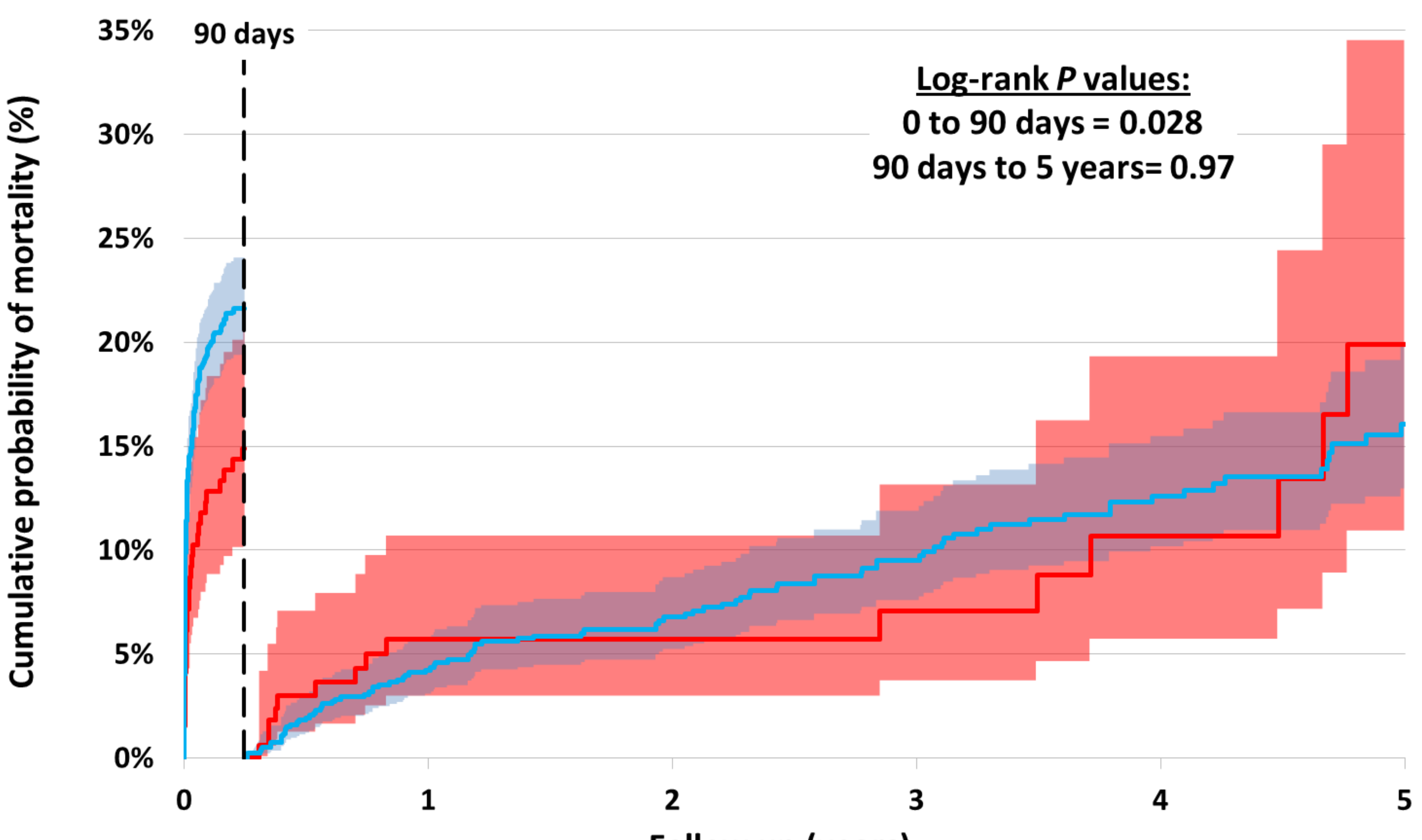


Figure 2a: Kaplan-Meier chart showing the cumulative probability of all-cause mortality for ATAD patients in the surgeon MAV groups. Landmark rebasing occurs at 90 days. Colored bands show approximate 95% confidence intervals.



Number at risk						
Mean AV < 4	1193	790	606	445	298	165
Mean AV ≥ 4	195	130	98	67	43	21

Figure 2b: Kaplan-Meier chart showing the cumulative probability of all-cause mortality for ATAD patients in the surgeon MAV groups. Colored bands show approximate 95% confidence intervals. (0-90 day detail from Figure 2a).

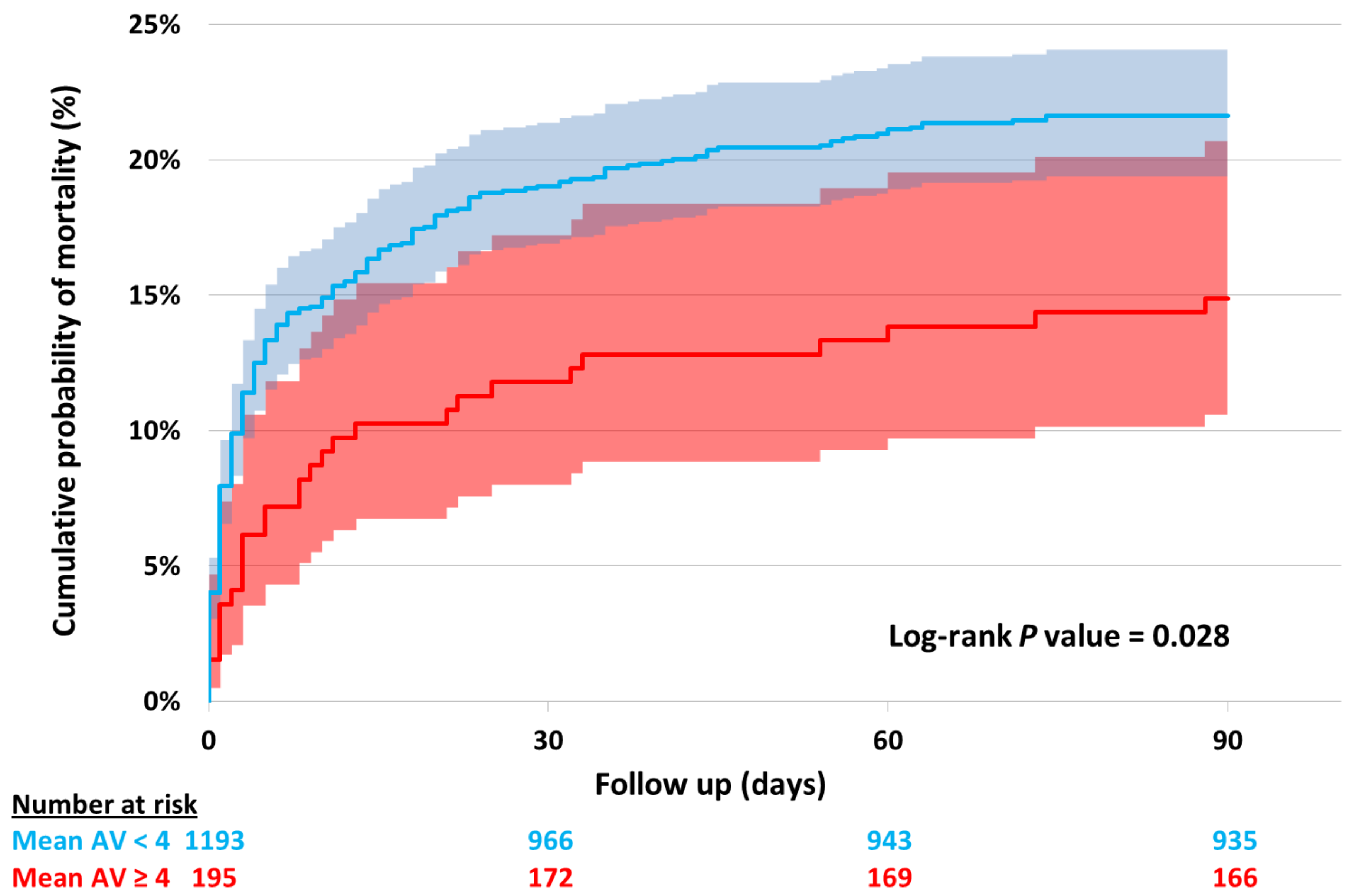
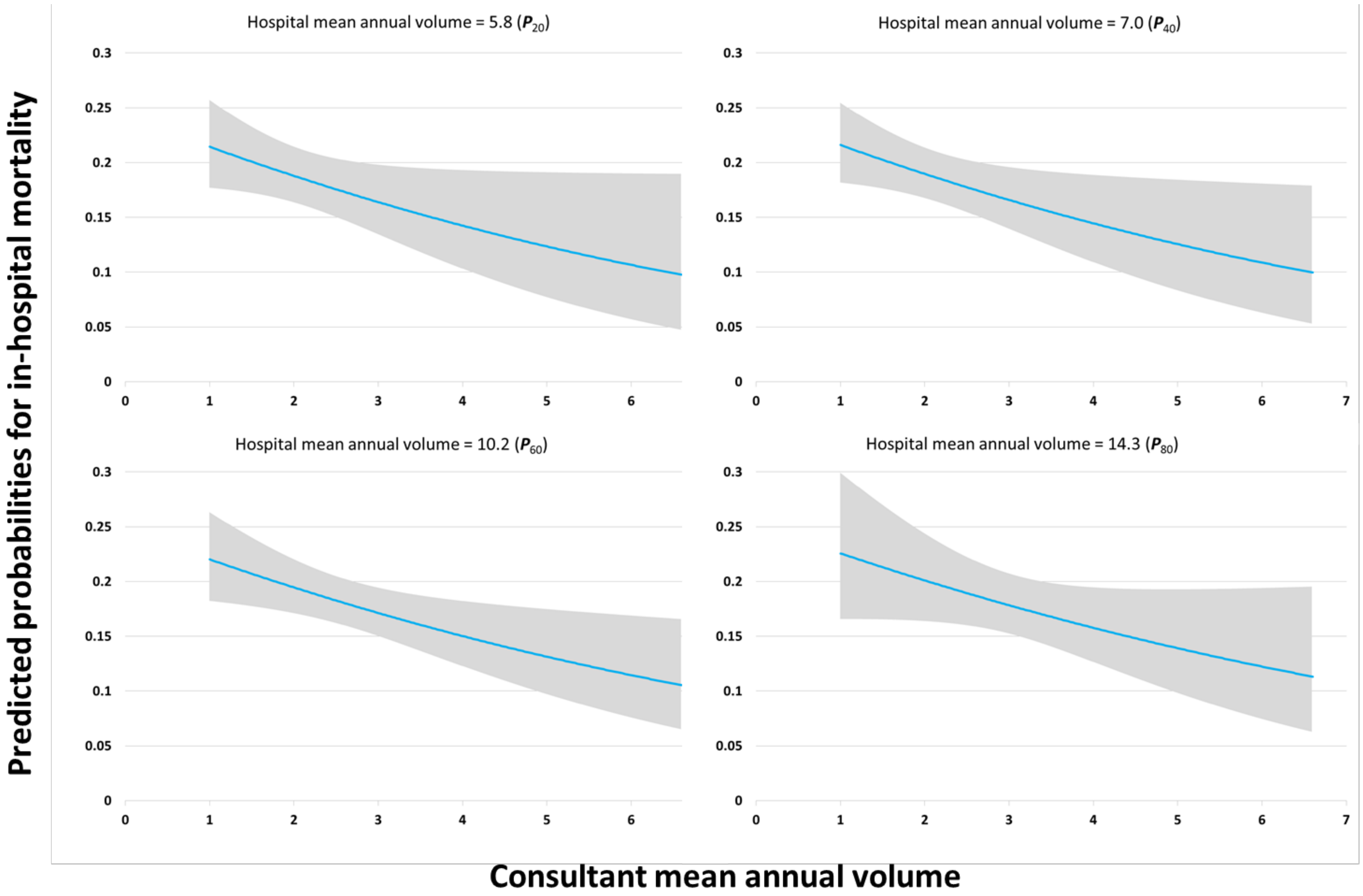


Figure 3: Panel chart showing the interaction between hospital and surgeon volume. The blue lines in each chart represent predicted probabilities of in-hospital mortality over the range of surgeon MAV for the 20th, 40th, 60th and 80th percentiles of hospital MAV, and the grey-shaded areas denote approximate 95% confidence intervals. Overall *P* value for interaction = 0.88



Supplemental Figure 1: Kaplan-Meier chart showing the cumulative probability of all-cause mortality for ATAD patients in the surgeon MAV groups. Colored bands show approximate 95% confidence intervals.