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Toward Optimizing Risk Adjustment in the Dutch Surgical Aneurysm Audit

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Background: To compare hospital outcomes of aortic aneurysm surgery, casemix correction for preoperative variables is essential. Most of these variables can be deduced from mortality risk prediction models. Our aim was to identify the optimal set of preoperative variables associated with mortality to establish a relevant and efficient casemix model.

Methods: All patients prospectively registered between 2013 and 2016 in the Dutch Surgical Aneurysm Audit (DSAA) were included for the analysis. After multiple imputation for missing variables, predictors for mortality following univariable logistic regression were analyzed in a manual backward multivariable logistic regression model and compared with three standard mortality risk prediction models: Glasgow Aneurysm Score (GAS, mainly clinical parameters), Vascular Biochemical and Haematological Outcome Model (VBHOM, mainly laboratory parameters), and Dutch Aneurysm Score (DAS, both clinical and laboratory parameters). Discrimination and calibration were tested and considered good with a C-statistic > 0.8 and Hosmer-Lemeshow (H-L) $P > 0.05$.

Results: There were 12,401 patients: 9,537 (76.9%) elective patients (EAAA), 913 (7.4%) acute symptomatic patients (SAAA), and 1,951 (15.7%) patients with acute rupture (RAAA). Overall postoperative mortality was 6.5%; 1.8% after EAAA surgery, 6.6% after SAAA, and 29.6% after RAAA surgery. The optimal set of independent variables associated with mortality was a mix of clinical and laboratory parameters: gender, age, pulmonary comorbidity, operative setting, creatinine, aneurysm size, hemoglobin, Glasgow coma scale, electrocardiography, and systolic blood pressure (C-statistic 0.871). External validation overall of VBHOM, DAS, and GAS revealed C-statistics of 0.836, 0.782, and 0.761, with an H-L of 0.028, 0.00, and 0.128, respectively.

Conclusions: The optimal set of variables for casemix correction in the DSAA comprises both clinical and laboratory parameters, which can be collected easily from electronic patient files and will lead to an efficient casemix model.

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INTRODUCTION

Background

Since 2013, it has been mandatory for all patients undergoing surgery for an abdominal aortic aneurysm (AAA) to be registered in the Dutch Surgical Aneurysm Audit (DSAA).[1] For a true interpretation of hospital outcomes, casemix risk adjustment has to be performed to level those differences in preoperative patient- and disease-related variables that influence outcome and which vary between hospitals.[2] Many of the variables present in casemix models are also represented in mortality risk prediction models, as was summarized in a recent systematic review.[3] Despite this multiplicity of models, no standard mortality risk prediction model in AAA surgery has been broadly implemented in clinical practice because every model has been developed for a certain population during a certain time period, which makes them less generalizable to other populations.[3]

Prediction models are based on physiological parameters, for example, the Glasgow Aneurysm Score (GAS),[4] on laboratory parameters, for example, the Vascular Biochemical and Haematological Outcome Model (VBHOM)[5] or mixed models such as the Dutch Aneurysm Score (DAS).[6] The physiology-only Vascular Physiological and Operative Severity Score for the enUmeration of Mortality and Morbidity [V(p)-POSSUM] contains one of the largest numbers of variables and has been extensively investigated.[7] All these variables were included in the original DSAA data set to calculate the V(p)-POSSUM score. However, preoperative surgical risk assessment has recently been shown to be possible using a maximum of eight, easily retrievable variables.[8] Because of the substantial registration burden imposed by the large number of variables, a validated optimal data set with a minimum number of risk adjustment parameters was needed, one in which the number of parameters is proportional to the number of events.[9]

Objectives

The aim of this study was to identify the optimal set for risk adjustment with a minimum number of casemix variables by means of an interhospital comparison of postoperative mortality for every patient with AAA and for patients with both elective and acute AAA and to internally validate this set. External validation was performed using previously developed preoperative risk prediction models for variable comparisons.

METHODS

Study Design and Setting

This study was set up in accordance with the STROBE statement for reporting of cohort studies.[10] It was designed to extract a minimum set of casemix variables and validate them internally. Subsequently, variables included in the casemix model were compared between hospitals and the casemix model and its variables were compared with existing mortality risk prediction models externally validated in the DSAA.

Patients and Data Source

Patients who had undergone surgery between 2013 and 2016 for primary juxtarenal or infrarenal AAA, both elective and acute, were prospectively registered in the DSAA and included for analysis. Details of the DSAA have been published previously.[11,12] The DSAA data of 2015 were verified over a randomly selected group of hospitals.[13] Where data regarding date of surgery, date of birth, operative setting/urgency (elective or acute and symptomatic or ruptured aneurysm), type of procedure (endovascular aneurysm repair [EVAR] or open surgical repair [OSR]), or mortality were missing, patients were not included for further analysis. In the Netherlands, the minimum volume per hospital was set at 20 operations per year and hospitals where fewer than 60 patients had been registered over a four-year period were excluded from the analysis.

Primary Outcome

The primary outcome was in-hospital or 30-day mortality. Subgroups of elective (EAAA) and acute operations (AAAA), based on symptomatic (SAAA) or ruptured AAA (RAAA) patients, were analyzed separately.

Statistical Analysis

Patients in whom EVAR had been converted to OSR were analyzed following intention-to-treat concept and included with EVAR. First, baseline characteristics were analyzed for the overall group (AAA) for EAAA and AAAA surgery. Continuous variables were tested for normality and linearity. Subsequently, if not normal or linear, variables were analyzed in categories. Missing or unknown values for categorical variables were estimated using multiple imputations. Multiple imputations were performed by an iteration of 10 data sets using the automatic imputation method in SPSS (version

Table I. Arithmetic formulas of mortality risk scores according to VBHOM, GAS, and DAS

Model	Model formula
VBHOM[a]	−2·257 + (0·1511 * male) + (0·9940 * mode of admission) + (0·05923 * age [continuous in years]) + (0·001401 * serum urea [continuous mmol/l]) − (0·01303 * sodium [continuous mmol/l]) − (0·03585 * potassium [continuous mmol/l]) − (0·2278 * hemoglobin [continuous g/dl]) + (0·02059 * white cell count [continuous * 10^9/l])
GAS	GAS: age (years) + (17 for shock) + (7 for myocardial disease) + (10 for cerebrovascular disease) + (14 for renal disease).
DAS[a]	−4.73 + (age * 0.074) + (systolic blood pressure [mm Hg]/10 * −0.12) + (1 for cardiopulmonary resuscitation) + ([hemoglobin {g/dL}/10^3] * −1.27).

[a]To calculate mortality risk, use exp(model)/1 + exp(model).

23.0) for the following variables: cardiac status, pulmonary status, malignant comorbidity, Glasgow Coma Scale (GCS), electrocardiography (ECG), sodium, potassium, creatinine, hemoglobin, white blood count (WBC), pulse, aneurysm size, age, gender, blood pressure, and three indicator variables; year of surgery, hospital, and setting.

Univariable analysis was performed to identify variables associated with mortality ($P < 0.05$). Casemix variables were analyzed in a multivariable logistic regression enter model with backward manual selection to reduce the chance of overfitting.[14] A selection P value of $P < 0.1$ was used to reduce the set of variables to as few as possible.[9]

Hospital Variation

Those casemix variables selected for multivariable analysis were also studied for between-hospital variation as if no variation is present, casemix correction would not be of great importance. By means of calculating continuous variables into dichotomous variables by the mean and dichotomizing categorical data into the presence or absence of a certain patient characteristic, the percentages were analyzed by hospital. Significant variation was reached if hospital percentages extended beyond the 95% confidence intervals.

Model Validation

Internal validation was performed by 100% apparent validation in which the population used for the development of the model is also used for internal model validation.[9]

External validation of the overall AAA group and the EAAA and AAAA subgroups was performed with three standard mortality risk prediction models (VBHOM, GAS, and DAS[4–6]; Table I). Two models were selected from an earlier systematic review, one based on laboratory values (VBHOM) and one based on clinical parameters (GAS). The third model was a newly validated Dutch model for RAAA surgery (DAS). Model performance was analyzed using the C-statistic and Hosmer-Lemeshow (H-L) tests for both the discrimination and calibration of these models. An area under the curve (C-statistic) of $\geq$0.7 described a moderate discriminative ability and $\geq$0.8 a good discriminative ability. P values $\geq$0.05 for the H-L showed sufficient calibration; the expected outcome did not significantly differ from the observed outcome.

RESULTS

Patients

A total of 13,417 patients with an AAA were registered in the DSAA, of which 12,524 (93.3%) had a primary AAA for which either OSR or EVAR was performed. In total, 99.1% ($n = 12,416$) of these patients were analyzed. Two hospitals that had only performed 12 and 3 operations, respectively, over a 4-year period were excluded. Of the remaining 12,401 patients, there were 9,537 EAAA patients (76.9%), 913 (7.4%) SAAA patients, and 1,951 (15.7%) patients with a RAAA (Fig. 1).

Overall, 8,614 patients (69.5%) underwent EVAR, compared with 3,787 (30.5%) undergoing OSR. The percentage of EVAR in EAAA patients was 77.1%; in SAAA patients 60.2%, and in RAAA patients, this was 36.4%. The mean age was 73.2 (7.9 SD) years and the majority were male (85.5%, $n = 10,596$). Detailed information about baseline characteristics, disease specifics, and interventions can be found in Table II (both the original and the imputed data set).

Outcome

Overall mortality was 6.5% ($n = 809$): EAAA surgery 1.8% ($n = 172$), 6.6% ($n = 60$) for SAAA surgery, and 29.6% ($n = 577$) for RAAA surgery. Mortality for AAAA surgery (combined SAAA and

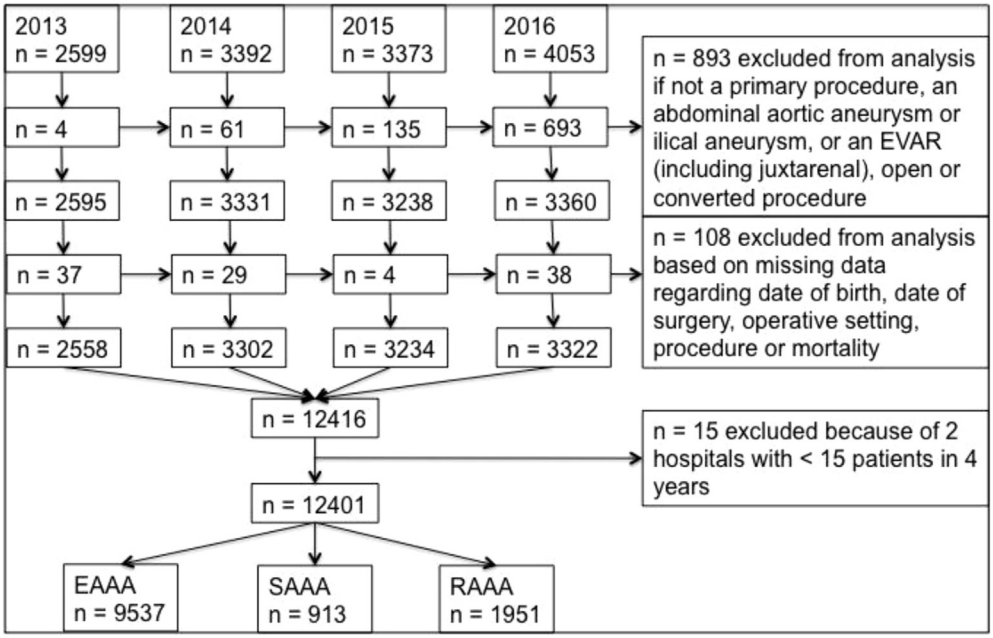

Fig. 1. Flowchart of selected data.

RAAA) was 22.2%. By procedure, elective procedures had the lowest mortality (0.7% EVAR, 5.4% OSR) compared with mortality after symptomatic procedures (4.5% EVAR and 9.6% OSR) and acute ruptures (22.2% and 33.8%).

Main Results

Univariable analysis. Table II shows the association of baseline characteristics with the outcome mortality for the imputed data set. Overall, age, gender, cardiac comorbidity, pulmonary comorbidity, GCS, aneurysm size, setting, ECG, creatinine, sodium, potassium, WBC, systolic blood pressure (SBP), pulse rate, and hemoglobin were associated with mortality. Subgroups of special interest and of influence on mortality were a decreased GCS 12−14 (OR, 9.81; 95% CI, 2.81−34.26) and <9 (OR, 15.26; 95% CI, 1.87−124.47) and urgent setting RAAA (OR, 22.87; 95% CI, 19.11−27.36).

Multivariable analysis. Overall, independent variables associated with mortality were age, gender, pulmonary comorbidity, operative setting, GCS, SBP, ECG, hemoglobin, and creatinine (Table III). The strongest overall predictors of mortality were increased pulmonary comorbidity, GCS, and setting (RAAA). Tables IIIb and IIIc show subgroup analyses for EAAA and AAAA patients. Potassium, aneurysm size, and malignancy were additional independent factors associated with mortality.

Hospital Variation

Significant interhospital variation was observed for all variables included in the model, with the exception of gender.

Model Validation

Internal validation, for the overall casemix model, showed a good pooled calibration (an H-L of 0.198) and good C-statistic of 0.871. For the elective submodel, the pooled C-statistic was 0.703 with an H-L of 0.476. For the acute submodel, the pooled C-statistic was 0.785 with an H-L of 0.109 (Table III and Fig. 2A−C).

External validation revealed that overall the VBHOM had the highest discriminative performance with a C-statistic of 0.836 and an H-L of 0.028, followed by the DAS with a C-statistic of 0.782 and an H-L of 0.000. Validation of the GAS resulted in a pooled C-statistic of 0.761 and an H-L of 0.128. For the subgroup of EAAA, pooled performance of GAS was 0.608 and H-L 0.645, of VBHOM 0.612, and H-L 0.614, and of DAS 0.622 with an H-L of 0.456. Performance of these models for AAAA surgery was 0.711 and an H-L of 0.218 and 0.687; and H-L 0.448 and 0.716; and an H-L of 0.004,

Table II. Baseline variables in the original data set, in the imputed data set, and the odds ratio of variables in the imputed data set that are associated with mortality

Baseline variables	Original data set *N* (%)	Imputed data set *N* (%)	Imputed OR for mortality (95% CI)
Age (mean [SD])	73.2 (7.9)	73.2	**1.07 (1.06−1.08)**
Sex			
Male	10,596 (85.4)	10,604 (85.5)	Ref.
Female	1,795 (14.5)	1,797 (14.5)	**1.50 (1.26−1.80)**
Missing	10 (0.1)	-	-
Card.			
No cardiac problems	5,699 (46.0)	6,112.1 (49.3)	Ref.
Peripheral edema	958 (7.7)	1,024.9 (8.3)	1.12 (0.95−1.31)
Elevated central venous pressure	190 (1.5)	206.4 (1.7)	**1.56 (1.18−2.05)**
Medication[a]	4,769 (38.5)	5,057.6 (40.8)	**1.83 (1.05−3.20)**
Unknown	785 (6.3)	-	-
Pulm.			
No dyspnea	8,882 (71.6)	9,333.2 (75.3)	Ref.
Dyspnea exercise	2,432 (19.6)	2,544.7 (20.5)	**1.45 (1.20−1.76)**
Invalidating dyspnea	357 (2.9)	375.7 (3.0)	**1.85 (1.29−2.66)**
Dyspnea rest	136 (1.1)	147.4 (1.2)	**3.84 (2.44−6.04)**
Unknown	594 (4.8)	-	-
Mal			
No malignancy	10,127 (81.7)	10,236.3 (82.5)	Ref.
Malignancy	2,143 (17.3)	2,164.7 (17.5)	0.86 (0.71−1.05)
Unknown	131 (1.1)	-	-
GCS			
15	11,076 (89.3)	11,375.8 (91.7)	Ref.
12−14	290 (2.3)	442.8 (3.6)	**9.81 (2.81−34.26)**
9−11	65 (0.5)	330.3 (2.7)	7.90 (0.48−130.8)
<9	119 (1.0)	252.1 (2.0)	**15.26 (1.87−124.47)**
Unknown/missing	851 (6.9)	-	-
Aneurysm size (mean [SD] [mm])	63.14 (14.2)	63.30	**1.04 (1.04−1.05)**
Setting			
EAAA	9,537 (76.9)	-	Ref.
SAAA	913 (7.4)	-	**3.83 (2.83−5.18)**
RAAA	1,951 (15.7)	-	**22.87 (19.11−27.36)**
ECG			
No abnormalities	6,260 (50.5)	7,329.9 (59.1)	Ref.
Atrial fibrillation	802 (6.5)	965.6 (7.8)	**2.23 (1.70−2.93)**
MI or other	3,432 (27.7)	4,105.5 (33.1)	**2.01 (1.63−2.48)**
Unknown	1,907 (15.4)	-	-
Creatinine			
normal, 45−100	7,579 (61.1)	7,707.4 (62.2)	Ref.
Not normal, <45 or >100	4,455 (35.9)	4,693.6 (37.8)	**3.18 (2.73−3.70)**
Unknown	367 (3.0)	-	-
Sodium			
normal, 135−145	10,348 (83.4)	11,227.6 (90.5)	Ref.
Not normal, <135 or >145	1,035 (8.3)	1,173.4 (9.5)	**2.77 (2.29−3.35)**
Unknown	1,018 (8.2)	-	-
Potassium			
normal, 3.5−5.0	10,593 (85.4)	11,279.1 (91.0)	Ref.
Not normal, <3.5 or >5.0	1,025 (8.3)	1,121.9 (9.0)	**2.39 (1.97−2.91)**
Unknown	783 (6.3)	-	-
WBC (*10^9) (mean [SD])	9.21 (3.24)	9.06	**1.23 (1.20−1.25)**
Hemoglobin (mmol/l) (mean [SD])	8.43 (1.20)	8.43	**0.50 (0.48−0.53)**
SBP (mm Hg)	135.95 (26.5)	135.77	**0.97 (0.97−0.97)**

(*Continued*)

Table II. Continued

Baseline variables	Original data set *N* (%)	Imputed data set *N* (%)	Imputed OR for mortality (95% CI)
Pulse			
normal, 60−100 bpm	9,206 (74.2)	9,905 (79.9)	Ref.
Not normal, <60 or >100 bpm	2,279 (18.4)	2,496 (20.1)	**1.81 (1.54−2.13)**
Unknown	916 (7.4)	-	-

Bold values are statistically significant.
$n = 12{,}401$ patients.
[a]Hypertension, angina pectoris, diuretics, or digoxin.

Table III. Final multivariable analysis model with the odds ratio for the entire AAA group and elective and acute AAA separately

	3a. AAA overall	3b. EAAA subgroup	3c. AAAA subgroup
Mortality risk prediction variables	Beta-coefficients	Beta-coefficients	Beta-coefficients
Age (years)	0.045		0.055
Gender (male)	−0.398	−0.677	−0.323
Pulmonary comorbidity (dyspnea during exercise)	0.509	0.707	0.364
Pulmonary comorbidity (invalidating dyspnea)	0.587	0.338	0.681
Pulmonary comorbidity (dyspnea in rest)	0.970	1.420	0.702
Ruptured AAA	2.519		
Symptomatic AAA	1.279		−1.285
Glasgow Coma Scale (12−14)	0.589		0.583
Glasgow Coma Scale (9−11)	1.123		1.239
Glasgow Coma Scale (<9)	1.182		1.296
Hemoglobin	−0.113	−0.289	
ECG (atrial fibrillation)	0.387	0.393	0.397
ECG (ischemia or other)	0.322	0.391	0.294
Creatinine	0.398	0.304	0.469
Systolic blood pressure	−0.005		−0.007
Potassium		0.518	
Aneurysm size		0.019	
Malignancy			0.340
Intercept	−5.910	−2.748	−4.909
Area under the curve	**0.871**	**0.703**	**0.785**
H-L	**0.198**	**0.476**	**0.109**

Bold values are statistically significant.

respectively. See Figure 3A−C for the area under the curves by setting.

DISCUSSION

Key Results

The current DSAA data set was based on V-POSSUM, of which the V(p)-POSSUM was regarded as being the casemix adjustment model for outcome comparison between hospitals. After thorough investigation, a limited number of casemix variables to decrease the registration burden were arrived at. A mix of easily collectible variables was identified including patient identifiers (age and sex), physiological variables (cardiac comorbidity represented by ECG, pulmonary comorbidity, GCS [in RAAA patients], and SBP), setting (EAAA, SAAA, and RAAA), anatomical findings (AAA diameter in EAAA patients), and laboratory results (creatinine and hemoglobin). Calibration for all three models varied widely for the population of the DSAA. However, DAS calibrated well for EAAA patients only compared with VBHOM that did not calibrate well overall and GAS that had a good calibration for all groups of patients.

Overall, many variables had a significant association with mortality after both univariable and multivariable logistic regression. However, previous studies have shown that casemix adjustment has a

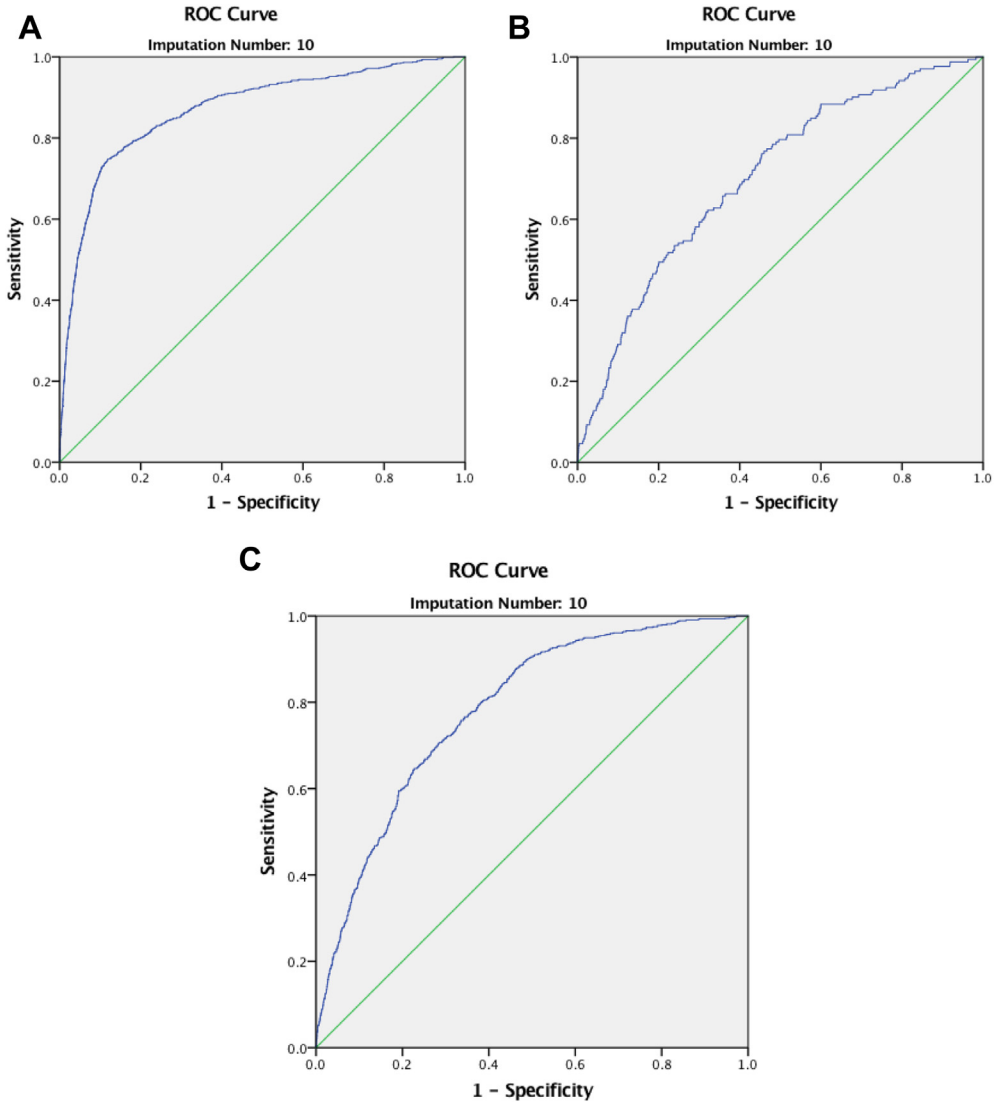

Fig. 2. Tenth iteration of ROC curves after internal validation of the DSAA casemix model. **(A)** Overall AAA; **(B)** EAAA; and **(C)** AAAA.

limited effect on the observed difference between hospitals.[11,15] Although patient casemix seemed to influence outcome, it did not explain—or only in part—the observed differences between hospitals.[15,16] These interhospital differences could also be related to differences in structural and process factors, by patient selection or by the proficiency of the surgical team.[15–17] Therefore, risk adjustment by casemix seems to have little impact on outcome differences between hospitals when compared with no risk adjustment.[17] A more complex casemix, which includes older patients with more comorbidity, may be counterbalanced by the continuous improvement in quality of health care.[18] However, maintaining risk adjustment by means of a limited set of patient casemix variables will remain necessary to moderate potential discussion among hospital stakeholders regarding differences in outcomes.

Some variables were more predictive for mortality by operative setting. For example, hemodynamic parameters and GCS were particularly important factors in acute AAA surgery, whereas comorbidity and AAA morphology were more associated with mortality in elective AAA surgery. Consequently, the observed differences between the mortality risk prediction models seemed to be partially related to the population analyzed and the period of

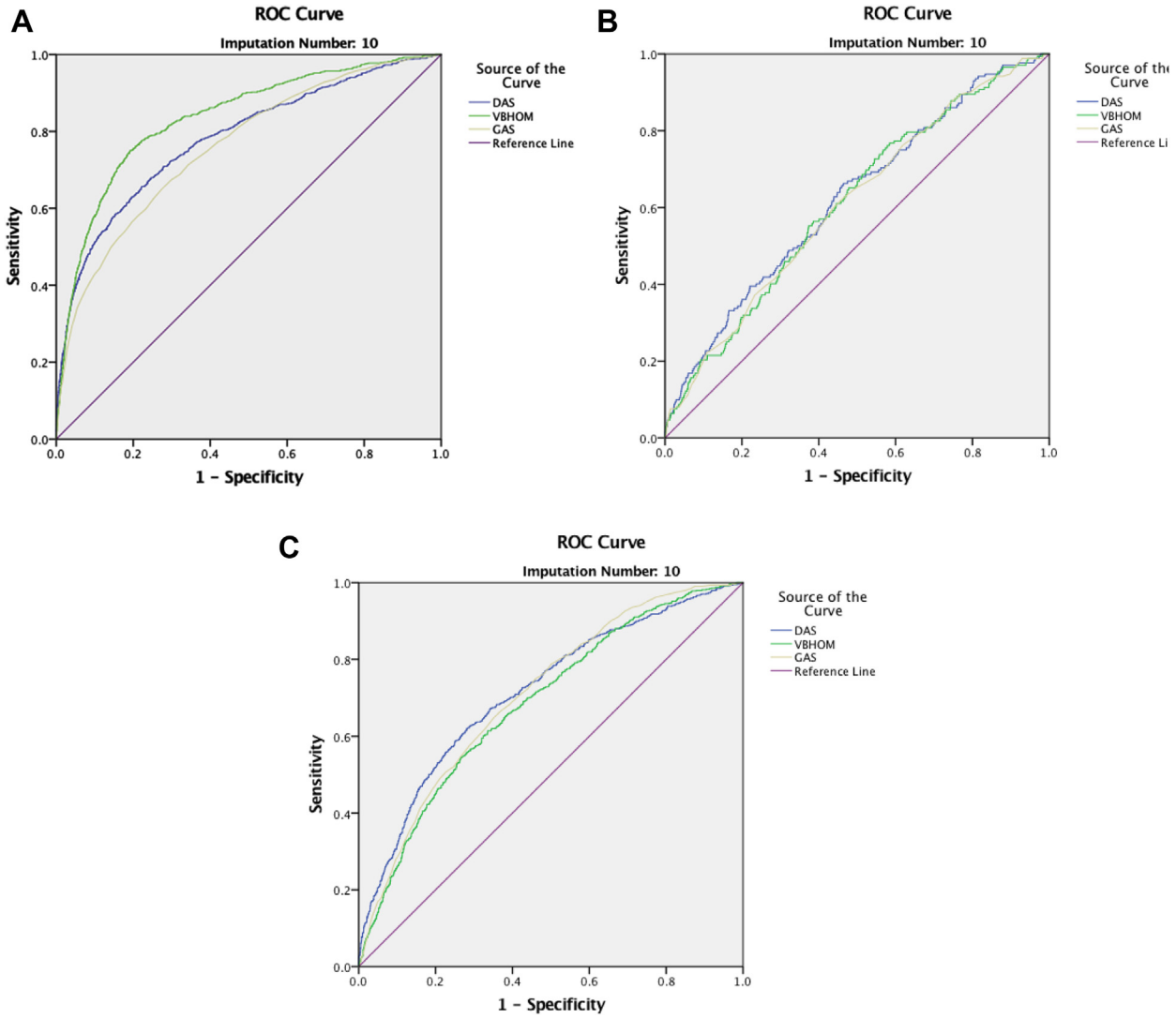

Fig. 3. Tenth iteration of ROC curves after external validation of VBHOM, GAS, and DAS. **(A)** Overall AAA; **(B)** EAAA; and **(C)** AAAA.

development. For example, DAS was built recently on a Dutch RAAA population, whereas GAS and VBHOM were developed over 10 years ago in an overall AAA population, when EVAR had just started to become common practice.[3,19] Consequently, DAS had the best discriminative performance in AAAA surgery on comparison with VBHOM and GAS. However, calibration by H-L of DAS was very significant indicating a low generalizability of the population analyzed. This could be due to the fact that SAAA patients were also included in the AAAA cohort of the DSAA, resulting in a lower mortality than that in the RAAA population in which DAS was developed. Moreover, mortality risk prediction only explains the association of the variables with mortality and need to be included in the model, whereas casemix variables that are associated with mortality and which do not differ between hospitals not necessarily need to be adjusted for.

Missing data in our study were resolved by multiple imputations. Another option to handle missing data would be to allocate missing values toward the ''normal'' category under the assumption ''if not registered then it may not be present at all'' as in V-POSSUM, for example.[7,20] Proper handling of missing values is important in prediction models. Automatic transfer (IT links to the electronic patient file) of hospital data and of expense claims from other specialists treating comorbidities, to the

web-based vascular registry (DSAA) will improve registry compliance and the validity of the data.

Limitations

In the Netherlands, the DSAA is mandatory and in 2015 was validated in 15 randomly selected hospitals.[13] There were no significant registration flaws. Only some minor complications were not registered. Important factors for casemix correction and mortality were not missed. However, there could have been under-registration of patients or other data could have been missed per individual patient. Another limitation of this study is that the DSAA contains a limited set of casemix variables, based on V-POSSUM, and therefore, it is possible that other relevant variables were disregarded. However, risk adjustment will always be limited to a fixed set of variables, leaving immeasurable confounders unadjusted for.[21]

CONCLUSIONS

It was possible to establish a compact set of 10 variables, that is, age, sex, cardiac comorbidity, pulmonary comorbidity, GCS, SBP, setting, aneurysm size, creatinine, and hemoglobin for casemix correction in AAA surgery in the DSAA. Preoperative casemix variables associated with mortality can be found in existing mortality risk prediction models, such as GAS and VBHOM, but when performing casemix correction, they should be extracted from the data set under analysis and ideally differ between hospitals.

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