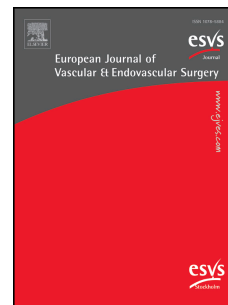


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External Validation of a Prognostic Model for Survival of Patients With Abdominal Aortic Aneurysms Treated With Endovascular Aneurysm Repair

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1 **External Validation of a Prognostic Model for Survival of Patients With**
2 **Abdominal Aortic Aneurysms Treated With Endovascular Aneurysm**
3 **Repair**

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16 **Short title:** External validation of a prognostic model for survival of patients with AAA treated
17 with EVAR

18 **Keywords:** Aortic aneurysm, Abdominal/surgery, Endovascular procedures/mortality,
19 Predictive model, Risk factors, Proportional hazards models, Survival analysis

20 **WHAT THIS PAPER ADDS**

21 The validated prognostic model identifies a high risk subgroup of patients with asymptomatic
22 abdominal aortic aneurysm (AAA) and a survival rate of only 16% at 10 years. The benefit of

23 endovascular aneurysm repair in these patients must be questioned as long as the AAA does
24 not carry a relevant risk of rupture.

25 **Objective:** Current guidelines recommend diameter monitoring of small and asymptomatic
26 abdominal aortic aneurysms (AAAs) due to the low risk of rupture. Elective AAA repair is
27 recommended for diameters ≥ 5.5 cm in men and ≥ 5.0 cm in women. However, data
28 supporting the efficacy of elective treatment for all patients above these thresholds are
29 diverging. For a subgroup of patients, life expectancy might be very short, and elective AAA
30 repair at the current threshold may not be justified. This study aimed to externally validate a
31 predictive model for survival of patients with asymptomatic AAA treated with endovascular
32 aneurysm repair (EVAR).

33 **Methods:** This was a multicentre international retrospective observational cohort study. Data
34 were collected from four European aortic centres treating patients between 2001 and 2021.
35 The initial model included age, estimated glomerular filtration rate (eGFR), and chronic
36 obstructive pulmonary disease (COPD) as independent predictors for survival. Model
37 performance was measured by discrimination and calibration.

38 **Results:** The validation cohort included 1 500 patients with a median follow up of 65 months,
39 during which 54.6% of the patients died. The external validation showed slightly decreased
40 discrimination ability and signs of overfitting in model calibration. However, a high risk
41 subgroup of patients with impaired survival rates was identified: octogenarians with eGFR
42 < 60 OR COPD, septuagenarians with eGFR < 30 , and septuagenarians with eGFR < 60 and
43 COPD having survival rates of only 55.2% and 15.5% at five and 10 years, respectively.

44 **Conclusion:** EVAR is a valuable treatment option for AAA, especially for patients unsuitable
45 for open repair. Nonetheless, not all these patients will benefit from EVAR, and an

46 individualised treatment recommendation should include considerations on life expectancy.
47 This study provides a risk stratification to identify patients who may not benefit from EVAR
48 under the present diameter threshold.

49 **INTRODUCTION**

50 Current treatment guidelines of the European Society for Vascular Surgery (ESVS) recommend
51 diameter monitoring of small and asymptomatic abdominal aortic aneurysms (AAAs) because
52 the risk of rupture is very low.¹ Elective AAA repair is recommended for asymptomatic AAA
53 with diameters ≥ 5.5 cm in men and ≥ 5.0 cm in women (class I, level A for men; class IIb, level
54 C for women).¹⁻³ However, robust data on the efficacy of elective treatment of all patients
55 with asymptomatic AAA with a diameter above this threshold are lacking, and the current level
56 of evidence classification in men has been questioned.⁴

57 As the burden of comorbidities increases, elective AAA treatment becomes less
58 effective or even futile in improving overall survival.⁴ Thus, personalised decision making in
59 patients with asymptomatic AAA could avoid unnecessary AAA treatments and reduce the
60 overall morbidity and costs associated with AAA. Nonetheless, this requires a reliable
61 assessment of the impending risk of aneurysm rupture, the risks related to elective repair, and
62 life expectancy. Only a complete picture of all competing risks will ultimately enable the
63 benefit of endovascular aneurysm repair (EVAR) to be assessed individually.⁸

64 The aim of the current study was to evaluate the overall survival of patients treated
65 with EVAR and thereby provide information on all cause mortality, the main adversary to the
66 efficiency of EVAR in comorbid patients. The survival of patients with asymptomatic AAA
67 varies greatly after elective aneurysm repair.⁵⁻⁷ Several patient characteristics and
68 comorbidities have been associated with patient survival after elective AAA repair. Still,

69 predicting survival after elective AAA surgery to support personalised decision making is not
70 yet established due to the lack of robust and validated tools.

71 A single centre predictive model for survival after EVAR for AAA identified age, the
72 estimated glomerular filtration rate (eGFR), and chronic obstructive pulmonary disease
73 (COPD) as independent predictors for long term survival.⁹ The previously published temporal
74 validation demonstrated good discrimination ability for five year survival in four risk groups.⁷
75 The five year survival probabilities were 89% in “low risk” patients, 83% in “low to moderate
76 risk” patients, 68% in “moderate to high risk” patients, and only 40% in “high risk” patients.
77 The current study aimed to validate externally this predictive model on an international
78 multicentre clinical cohort.

79 **MATERIALS AND METHODS**

80 This retrospective observational cohort study includes all consecutive patients treated with
81 standard EVAR for asymptomatic AAA at four different European aortic referral centres: the
82 university hospital of Zurich, Switzerland (2003 – 2020), the university hospitals of Turku and
83 Helsinki, Finland (2010 – 2021 and 2002 – 2016, respectively), and the university hospital of
84 Leuven, Belgium (2001 – 2019). Patients with complex EVAR, including fenestrated, branched,
85 or parallel grafts, were excluded from this study. Further, all patients treated for symptomatic
86 or ruptured aneurysms and other indications like penetrating aortic ulcers were excluded.

87 This study was conducted according to the Principles of the Declaration of Helsinki and
88 reported in adherence to the TRIPOD statement (Transparent Reporting of a multivariable
89 prediction model for Individual Prognosis or Diagnosis).¹⁰ The local ethics committees in Bern
90 and Zurich Switzerland approved this study (BASEC-IDs: 2022-00489 and 2021-02311),
91 whereas the local committees in Turku, Helsinki, and Leuven waived approval of the study due
92 to its retrospective nature.

93 **Data collection and definitions**

94 Patient characteristics were obtained from local records and aggregated at an individual
95 patient level. Treatment indications were according to available ESVS guidelines from 2011
96 and 2018.^{1,11} Prior to that, a threshold of 55 mm (50 mm for females), rapid progress (≥ 5 mm
97 in six months), or saccular anatomy was used as indication criterion. Diameter measurements
98 were extracted from recordings without consulting the available images.

99 The baseline characteristics of the validation cohort were summarised and compared
100 to the original cohort. COPD was defined as any diagnosis of COPD at the time of operation or
101 any forced expiratory volume $< 80\%$ of the predicted capacity on pre-operative spirometry.
102 eGFR was calculated using the modification of diet in renal disease study formula using the
103 last pre-operative creatinine value within 30 days.

104 The primary outcome measure of this study was model performance measured by
105 discrimination and calibration for overall survival in the validation cohort. For patients treated
106 at the university hospitals of Leuven, Zurich, and Turku, survival information was obtained
107 from local hospital databases. All patients without a documented date of death by the
108 predefined study end date, 31 October 2022, were contacted during a cross-sectional
109 telephone survey between November 2022 and March 2023. For patients treated at the
110 University of Helsinki, survival information was provided by the Statistics Finland Cause of
111 Death registry. Completeness of follow up information was reported using the Follow-up
112 Index.¹² Survival information was trimmed at the study end date.

113 This overall dataset formed the “validation cohort”. The previously published patient
114 cohort treated at the university hospital of Bern formed the “original cohort” and was used
115 for comparison.

116 **Statistical analysis**

117 Predictor selection

118 The variable selection process for the predictive score based on the original cohort has been
119 described in detail.⁹ In summary, pre-selection of variables was conducted based on a
120 literature review to avoid a complete data driven variable selection. Thereafter, a machine
121 learning method (least absolute shrinkage and selection operator with 10 fold cross-validation
122 in a Cox model) was used for the variable selection for the predictive model. The predictive
123 model identified age, eGFR, and COPD (see Supplementary Table S1).

124 Predictive score

125 The beta-coefficients of the Cox model were used to create an easy to use risk score.⁷ Age was
126 grouped into quartiles and rounded to the next integer for practical reasons. eGFR was
127 grouped into quartiles according to the KDIGO classification, but G4 and G5 were merged.
128 COPD was available as a binary variable only and thus formed two groups.

129 The beta coefficients for each variable group were multiplied by 10 and rounded to the
130 nearest integer to create the score. This resulted in the following scoring for age: < 70 years =
131 0 points; 70 – 74.9 years = 9 points; 75 – 79.9 years = 10 points; ≥ 80 years = 17 points; for
132 eGFR: KDIGO G1 = 0 points; G2 = 1 point; G3a = 3 points; G3b = 6 points; G4/5 = 15 pts; for
133 COPD: if present = 7 points.

134 The total score of the three variables was formed, and the cohort was divided into
135 quartiles compiling the four risk groups: ≤ 8 points, “low risk”; 9 – 13 points, “low to moderate
136 risk”; 15 – 18 points, “moderate to high risk”; ≥ 19 points, “high risk.” Of note, no combination
137 resulted in a sum score of 14 points. The risk score is provided in Supplementary Table S1 and
138 is available online.⁷

139 Model discrimination and calibration

140 The discrimination ability of the model was tested on the validation cohort using Harrell's
141 concordance statistics C with 95% confidence interval (95% CI) using DeLong's method.¹³
142 Calibration was visually inspected and quantified using the jack-knife pseudo-value method.
143 Discrimination and calibration of the predictive score were tested separately at five and 10
144 years. The same analysis was performed for the predictive model using age and eGFR as
145 continuous rather than categorical variables. Further, the observed survival for each of the
146 risk groups was compared between the original cohort and the validation cohort using the
147 likelihood ratio test.

148 **Further descriptive analysis**

149 Continuous variables (i.e., age, eGFR, creatinine, body mass index, AAA diameter) were
150 visually inspected for normality and summarised using the median and quartiles (Q1, Q3) since
151 they were skewed. Factor variables were compared by chi squared test and continuous
152 variables by the Kruskal–Wallis rank test. A Cox proportional hazard model was calculated for
153 the validation cohort, including all available variables previously identified to be associated
154 with survival to allow the inclusion of these data in future studies. For this analysis, multiple
155 imputations were performed for missing comorbidities using the “mice” package. Predictive
156 mean matching was used for continuous variables, multinomial logistic regressions were used
157 to impute factor variables. The number of imputed datasets was $m = 25$. The proportional
158 hazards assumption was tested and verified using scaled Schoenfeld residuals for each Cox
159 model.

160 All statistical analyses were performed using R Studio version 4.2.3 on MacOS version
161 12.5.1.

162 **RESULTS**

163 The four study sites treated a total of 1 616 consecutive patients with asymptomatic AAA using
164 EVAR during their respective inclusion periods. Information on COPD was unavailable in 87
165 patients and eGFR was missing in 29 patients, resulting in a final validation cohort of 1 500
166 patients. Baseline characteristics of the aggregated validation cohort prior to EVAR are
167 summarised in Table 1 and compared with the original cohort. Supplementary Table S2
168 summarises the baseline characteristics stratified by centre. The baseline characteristics of
169 the validation cohort were comparable with the original cohort. However, significant
170 differences were found for two variables of the predictive model. Patients in the validation
171 cohort had a diagnosis of COPD more often (31.1% vs. 23.7%, $p < .001$). Further, the eGFR was
172 significantly higher in the validation cohort than in the original cohort (77.8 vs. 66.1
173 ml/min/1.73², $p < .001$).

174 The median overall follow up of the validation cohort was 65 (Q1, Q3 37, 101) months
175 with almost complete follow up information (Follow-up Index 0.97). The overall survival times
176 were identical among the two cohorts: 71% (95% CI 67 – 75%) in the original and 71% (68 –
177 73%) in the validation cohort; 39% (32 – 48) in the original and 39% (36 – 42%) in the validation
178 cohort ($p = .70$), at five and 10 years, respectively. During the follow up, 54.6% ($n = 819$) of the
179 patients in the validation cohort died, whereas 35.7% ($n = 197$) of the patients died in the
180 original cohort. Further details on follow up and survival at one, five, and 10 years are
181 presented in Table 2.

182 ***External validation of the predictive model***

183 The external validation of the predictive model showed a slightly decreased discrimination
184 ability compared with the previously reported model performance: Harrell's C 0.62 (95% CI
185 0.60 – 0.65) compared with 0.70 (0.66 – 0.75) on the original cohort. However, calibration was

186 excellent: the predicted and observed overall survival was 69.5% and 70.3% after five years
187 and 37.0% and 38.3% after 10 years, respectively. The calibration curves at five and 10 years
188 (Fig. 1) are slightly S shaped but very close to the perfect calibration reflected by the diagonal
189 line.

190 Figure 2 and Table 2 show the survival at five and 10 years by risk group, comparing the original
191 and external validation cohorts. There was no statistically significant difference in the
192 observed survival between the original cohort and the validation cohort in the “low risk” group
193 ($p = .052$), the “low to moderate-risk” group ($p = .33$), and the “moderate to high risk”
194 ($p = .31$). However, the observed survival was significantly better in the validation cohort
195 compared to the original cohort for the “high risk” group ($p = .046$). The five year survival in
196 the original cohort for “high risk” patients was 40% (95% CI 32 – 50%) and 55% (49 – 62%) in
197 the validation cohort. This demonstrates a slight but significant overfitting of the model for
198 “high risk” patients with significantly better survival than predicted.

199 A multivariable Cox proportional hazard model for survival after EVAR, including all
200 available variables in the dataset, is available in Supplementary Table S3.

201 ***EVAR cohort over time***

202 Figure 3 and Supplementary Figure S1 show changes in the risk scores and age groups of
203 patients treated for EVAR in the validation cohort. A steady increase in the proportion of
204 octogenarians can be seen from 2001, where only 8.8% were 80+ years old, to 2019, where
205 39.7% of the treated patients were octogenarians. Of note, there was a change in this pattern
206 with a decrease in the proportion of octogenarians and a decrease in the proportion of < 70
207 years old in the years 2020 and 2021 (Supplementary Figure S1).

208 The same observations were made for the burden of risk score. Patients were
209 healthier, in terms of the risk score, with only 3.9% high risk patients in 2001, whereas 32%

210 high risk patients in 2020. A steep drop in the proportion of high risk patients was documented
211 for 2021, when only 11.1% were in the high risk group (Fig. 3).

212 **DISCUSSION**

213 This international, multicenter, external validation of a predictive model for the survival of
214 patients with AAA treated with EVAR showed a modest reduction in the discriminatory ability,
215 but excellent model calibration. The prognostic model confirmed a high risk subgroup of
216 patients with a survival rate of only 55% at five and 16% at 10 years, respectively:
217 octogenarians with eGFR < 60 *or* COPD, septuagenarians with eGFR < 30, and septuagenarians
218 with eGFR < 60 *and* COPD. This contrasts with the excellent median life expectancy of the
219 general population aged 80 in Switzerland, which in 2022 was 8.8 years for men and 10.4 years
220 for women.¹⁴ The benefit of EVAR in high risk patients must therefore be questioned as long
221 as the AAA does not carry a relevant risk of rupture.

222 Nonetheless, a complete picture of the risk–benefit balance of EVAR in this patient
223 cohort should consider the following three points: (1) the median AAA diameter was 58 mm,
224 about 1 cm smaller than in the patients who had participated in the EVAR-2 study.⁴ The
225 diameter of high risk patients was significantly larger ($p < .001$, Supplementary Figure S2).
226 However, 50% of all patients in the high risk group had AAA diameters < 60 mm, and
227 approximately 25% had AAA diameters < 55 mm. The risk for AAA rupture might be lower than
228 historical data suggested, but understanding the natural progression and rupture rates
229 remains limited.^{15–17} (2) The 30 day mortality rates after EVAR decreased but for these
230 electively treated asymptomatic patients were still 1.1%.⁵ (3) Like previous studies, this study
231 shows that patients treated electively for an AAA have a relatively poor long term survival of
232 only about 40% after 10 years.^{5,18–20} Given this context, a substantial proportion of patients
233 treated in this validation cohort may not have lived sufficiently long to realise the advantages

234 of this preventative treatment. Strict adherence to the current diameter threshold in high risk
235 patients or even expanding the treatment criteria for AAA patients beyond the binary
236 threshold of 55 mm diameter would enhance the quality of patient care. Of note, an
237 association between initial AAA diameter and survival after elective EVAR has been previously
238 described.²¹ However, AAA was eliminated in the variable selection process as the magnitude
239 of this association was not strong enough.^{7,9} Still, this association was confirmed in the
240 multivariable analysis of this cohort, HR 1.01 per millimetre AAA diameter increase (95% CI
241 1.00 – 1.02, $p < .001$); see Supplementary Table S3.

242 The decision regarding preventive treatment for asymptomatic AAA in elderly and/or
243 severely comorbid patients is challenging. The long term results of the EVAR-2 trial
244 demonstrated no increase in overall life expectancy for the EVAR group vs. the non-treated
245 group.⁶ Of the originally included 404 patients in the EVAR-2 trial, only 17% (69/404) survived
246 more than eight years, and these patients were younger, with higher body mass index, higher
247 eGFR, and better forced expiratory volume in 1 second at the time of enrolment.⁶ The 10 year
248 survival rate of the high risk cohort in the current study is comparable to the overall survival
249 rate of patients from the EVAR-2 study. In contrast improvements in the peri-operative
250 mortality rates have been achieved: a recent analysis from the American College of Surgeons
251 National Surgical Quality Improvement Program of almost 25 000 patients undergoing EVAR
252 between 2005 and 2013 showed a substantially lower 30 day mortality rate of 1.9% for high
253 risk patients compared with the 7.3% reported by the EVAR-2 trial.^{6,22} The peri-operative
254 mortality rate in the high risk cohort was 2.2% and was comparable to these US data. Adkar
255 *et al.* identified the presence of at least one criterion of impairment (respiratory, cardiac, or
256 renal) or their combination as risk factors for 30 day mortality.²² These risk factors for
257 increased peri-operative mortality were comparable to the risk factors for long term survival

258 identified in this study. Hence, individuals with elevated peri-operative risk have a reduced life
259 expectancy, prompting a need to scrutinise the advantages of elective AAA treatment based
260 on the existing diameter threshold. Clinicians automatically and intuitively weigh different
261 risks based on their experience and thus are likely to withhold EVAR in some patients.
262 Diameters were already significantly larger in high-risk patients compared to the other risk
263 groups ($p < .001$; Supplementary Figure S2). This study provides a risk stratification to support
264 and improve such decisions in the future.

265 EVAR was initially invented as a less invasive alternative for patients unfit for open
266 repair.²³ Figures 2 and 3 depict the initial application of EVAR in relatively healthier patients,
267 while its extensive utilisation among elderly and more critically afflicted patients became
268 predominant only during the last decade. This might be caused by a more restrictive use of
269 EVAR in younger and healthier patients following long term results showing advantages for
270 open repair.^{5,24} A sharp decline in the proportion of high risk patients was documented for
271 2021, where only 11.1% were in the high risk group. This sharp decline falls within the COVID
272 19 pandemic and could be caused by reduced elective surgery capacity, especially for elderly
273 and comorbid patients who are likely to require intermediate care or intensive care after
274 treatment. It will be interesting to see if this trend continues in the years after COVID or if
275 there is even a catch up effect.

276 The use of EVAR in low risk patients (< 70 years with eGFR \geq 60, independent of COPD)
277 who are expected to live longer is a separate topic of discussion. More than 60% of these
278 patients will still be alive after 10 years and beyond, thus at risk for late complications.⁵
279 Primary open repair may still be the preferred treatment option for these patients. The
280 upcoming years will show if there will be a trend towards an open first strategy for young low
281 risk patients.

282 Limitations

283 The model performance was validated and confirmed robust discrimination ability and
284 excellent calibration to successfully identify a subset of high risk patients for impaired long
285 term survival. The main limitations of this international multicentre external validation study
286 is the retrospective extraction of routinely collected data which inherently carries a risk of
287 bias: no routine pre-operative measurement of forced expiratory volume in 1 second was
288 performed and COPD diagnosis was partly subjectively coded; furthermore, some
289 heterogeneity and inconsistencies in AAA diameter measurement must be assumed over the
290 two decades of the study period.

291 The calibration curve (Supplementary Figure S1a,b) and the survival plots show an S shaped
292 model performance with better accuracy for the moderate risk groups. In contrast, the model
293 slightly overestimates mortality in the high risk group and slightly underestimates mortality in
294 the low risk group. This can be indicative for some degree of model overfitting or underfitting.
295 Further validation of the model in cohorts with different case mix (i.e., higher or lower degree
296 of comorbidities) is needed to better understand calibration in the extremes of the calibration
297 curve.

298 In general, the clinical applicability of any predictive model in daily routine needs to be
299 carefully assessed. The idea behind risk stratification with predictive tools is to support clinical
300 decisions rather than drive them.

301 Conclusion

302 The role of EVAR as a valuable treatment option remains undisputed, especially for patients
303 unsuitable for open repair. Nonetheless, not all these patients will benefit from EVAR, and an
304 individualised treatment recommendation should include considerations on life expectancy.

305 This study provides a risk stratification to identify patients who may not benefit from EVAR
306 under the present diameter threshold.

307 **CONFLICTS OF INTEREST**

308 None.

309 **FUNDING**

310 None.

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Table 1. Baseline characteristics of the aggregated validation cohort prior to endovascular aneurysm repair compared with original cohort.

Variable	Original cohort N = 552	Validation cohort N = 1 500	p value
Male sex	503 (91.1)	1 370 (91.3)	.88
Age – y	76.0 (69.4, 80.6)	75.2 (69.3, 80.0)	.44
Arterial hypertension	458 (84.3)	1 194 (80.6)	.055
Missing	9	19	
Diabetes mellitus	108 (19.6)	252 (17.2)	.23
Missing	0	39	
Dyslipidaemia	467 (84.6)	1 061 (72.6)	<.001
Missing	0	38	
BMI – kg/m ²	27.0 (24.0, 30.0)	26.1 (24.0, 29.2)	.004
Missing	29	447	
Smoking	390 (73.3)	855 (60.6)	<.001
Missing	20	90	

<i>COPD</i>	131 (23.7)	470 (31.3)	<.001
<i>eGFR – mL/min/1.73 m²</i>	66.1 (51.0, 80.9)	77.8 (63.0, 87.5)	<.001
<i>Creatinine – mmol/L</i>	91 (77, 111)	93 (80, 112)	.036
<i>PAD, Fontaine class</i>			<.001
No PAD	436 (79.0)	496 (48.5)	
Fontaine I	49 (8.9)	486 (47.6)	
Fontaine II	46 (8.3)	38 (3.7)	
Fontaine III	21 (3.8)	0 (0.0)	
Fontaine IV	0 (0.0)	2 (0.2)	
Missing	0	478	
<i>Coronary artery disease</i>	308 (56.0)	746 (50.1)	.019
Missing	2	12	
<i>Myocardial infarction</i>	113 (20.6)	353 (31.7)	<.001
Missing	4	387	
<i>Aneurysm diameter – mm</i>	58.0 (54.0, 62.5)	58.0 (55.0, 65.0)	.014
<i>Missing</i>	25	7	

385 Continuous variables are presented by median (quartiles 1, 3). Counts are presented as *n* (%). Data were
386 complete if not stated explicitly. Factor variables were compared by chi squared test, continuous variables by
387 the Kruskal–Wallis rank test, respectively. BMI = body mass index; COPD = chronic obstructive pulmonary
388 disease; eGFR = estimated glomerular filtration rate according to the “Modification of Diet in Renal Disease
389 Study” (MDRD) in mL/min/1.73m²; PAD = peripheral arterial disease as clinical stage according to the Fontaine
390 classification.

391

Table 2. Survival Information.

Variable	Original N = 552	Validation N = 1 500	p value
Follow up – mo (Q1, Q3)	56 (23, 77)	65 (37, 101)	<.001
Follow up index	0.95	0.97	<.001
Number of deaths	197 (35.7)	819 (54.6)	n.a.
<i>Overall survival</i>			
30 days	98.7 (97.8–99.7)	98.9 (98.3–99.4)	.70
5 years	70.8 (66.7–75.2)	70.8 (68.4–73.2)	.70
10 years	39.2 (32.3–47.6)	38.7 (35.7–42.0)	.70
<i>1 year survival by risk group</i>			
Low risk	98.6 (96.6–100)	95.7 (93.8–97.7)	.052
Low to moderate	98.6 (96.8–100)	94.6 (92.5–96.8)	.33
Moderate to high	91.8 (87.0–96.8)	92.7 (90.1–95.3)	.31
High risk	81.6 (75.3–88.6)	90.6 (87.2–94.1)	.046
<i>5 year survival by risk group</i>			
Low risk	89.1 (83.7–94.9)	86.2 (82.7–89.8)	.052
Low to moderate	83.7 (77.4–90.6)	74.0 (69.8–78.5)	.33
Moderate to high	68.4 (59.5–78.6)	61.8 (56.9–67.2)	.31
High risk	39.9 (31.7–50.2)	55.2 (49.3–61.8)	.046
<i>10 year survival by risk group</i>			
Low risk	74.8 (63.5–88.1)	61.2 (55.6–67.5)	.052
Low to moderate	43.2 (30.1–62.0)	43.2 (37.6–49.6)	.33
Moderate to high	23.4 (12.3–44.8)	24.6 (19.6–30.8)	.31

High risk	9.6 (3.0–31.2)	15.5 (10.3–23.1)	.046
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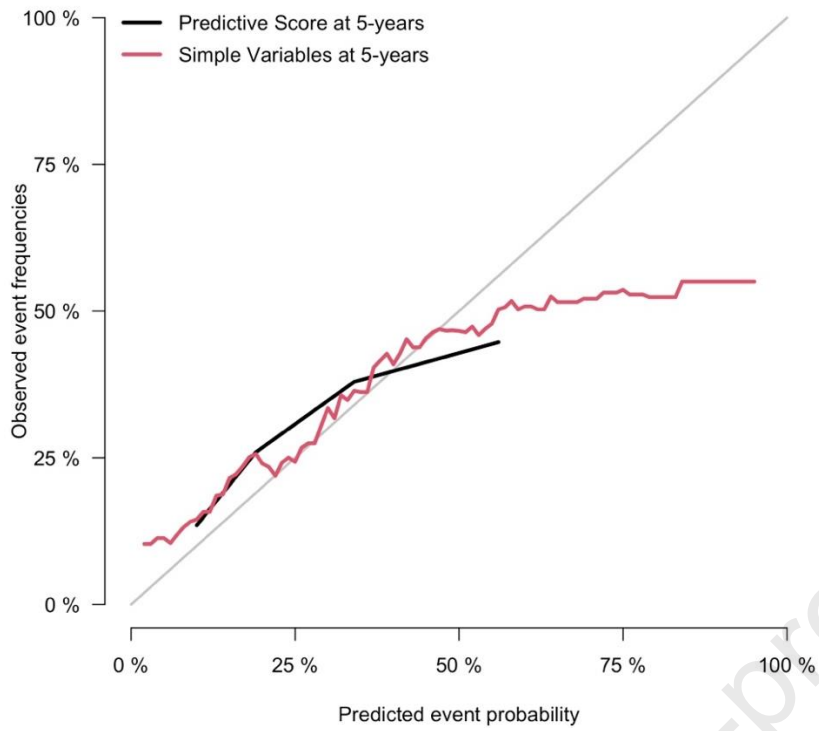
392 Data are presented as median (interquartile range), *n* (%), or % (95% CI), Median follow up time is presented with
 393 months and quartiles (Q1, Q3). Survival is presented overall and by risk group with percentages and 95% CI given
 394 by the Kaplan–Meier estimators. CI = confidence interval; n.a. = not available.

395 **Figure 1.** (A) Model calibration for survival at five years after endovascular aneurysm repair
 396 (EVAR). (B) Model calibration for survival at 10 years after endovascular aneurysm repair.
 397 Calibration curves of the Cox models at five years (Supplementary Figure S1a) and at 10 years
 398 (Supplementary Figure S1b). The black line reflects the performance of the risk score; the red
 399 line reflects the performance of a model using age and estimated glomerular filtration rate as
 400 continuous variables and chronic obstructive pulmonary disease as a binary variable.

401 **Figure 2.** (A) Survival at five years after endovascular aneurysm repair (EVAR). (B) Survival at
 402 10 years after EVAR. Survival at five and 10 years after EVAR for the original and validation
 403 cohorts stratified by risk score groups and presented with a 95% confidence interval. A total
 404 of 819 deaths were observed in the validation cohort, which included 1 500 patients; a total
 405 of 197 deaths were observed in the original cohort, which included 552 patients.

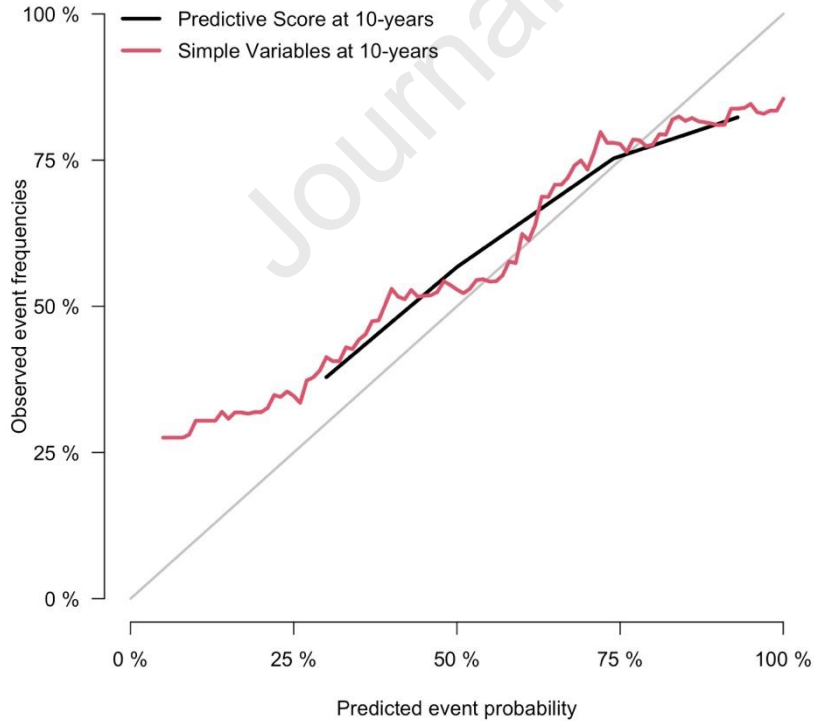
406 **Figure 3.** Change in risk group over the study period. This figure shows the proportion of
 407 patients treated in four risk groups for the validation cohort.

408 **Figure 1.**



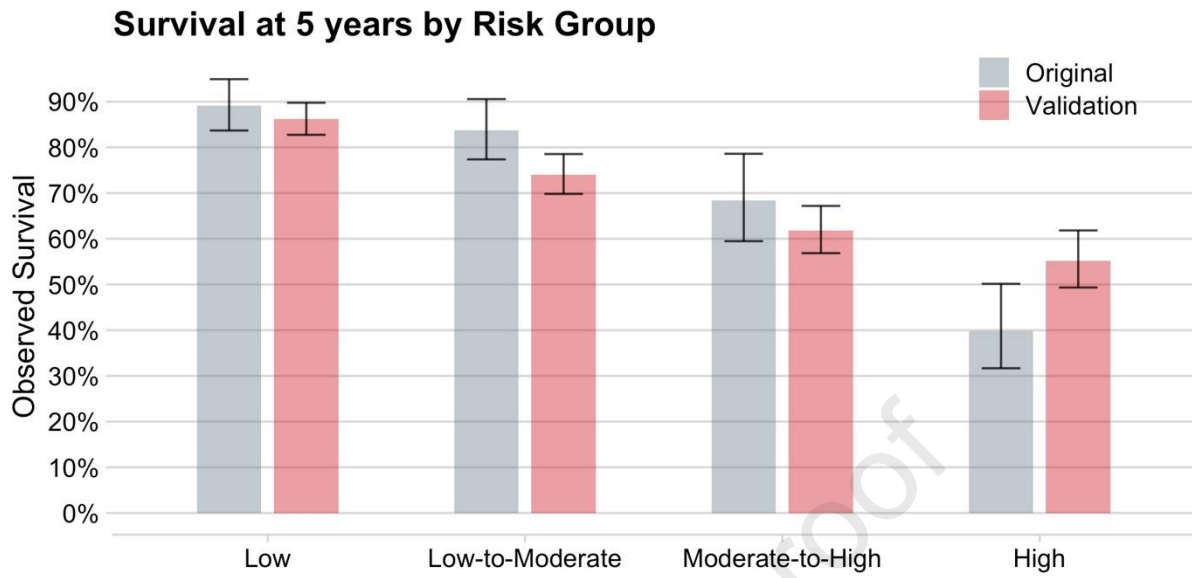
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411 **Figure 1b**

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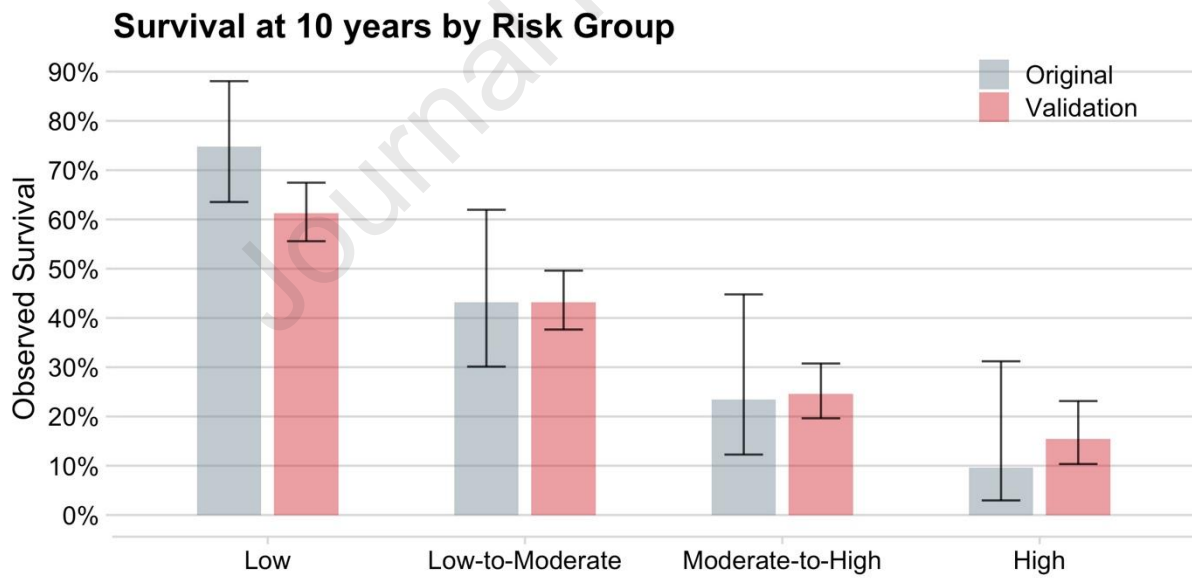
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414 **Figure 2. A.**

415

416 **B**

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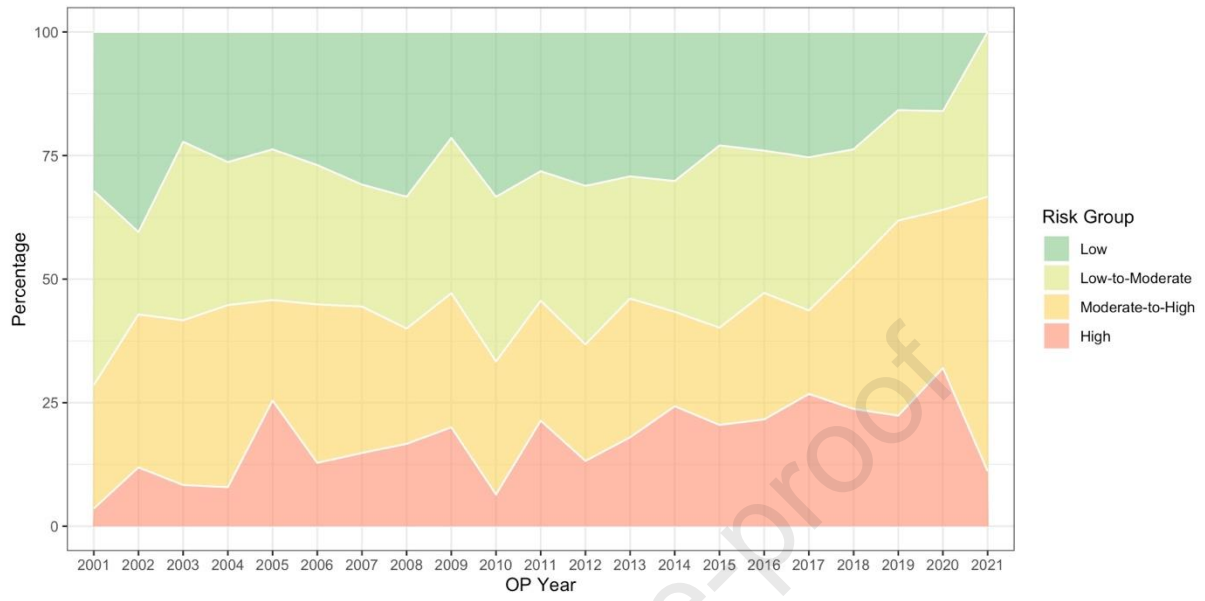


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420 **Figure 3.**

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