Cardiopulmonary exercise testing and survival after elective abdominal aortic aneurysm repair†

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Editor’s key points
- There are few data on cardiopulmonary exercise testing (CPET) before aortic surgery and subsequent outcome.
- In this study, CPET variables were associated with reduced survival after aortic repair.
- However, CPET may be performed differently in different centres and confidence intervals in this study were wide.
- This study adds to the body of evidence on CPET as part of preoperative assessment, but its contribution remains uncertain and further data are required.

Background. Cardiopulmonary exercise testing (CPET) is increasingly used in the preoperative assessment of patients undergoing major surgery. The objective of this study was to investigate whether CPET can identify patients at risk of reduced survival after abdominal aortic aneurysm (AAA) repair.

Methods. Prospectively collected data from consecutive patients who underwent CPET before elective open or endovascular AAA repair (EVAR) at two tertiary vascular centres between January 2007 and October 2012 were analysed. A symptom-limited maximal CPET was performed on each patient. Multivariable Cox proportional hazards regression modelling was used to identify risk factors associated with reduced survival.

Results. The study included 506 patients with a mean age of 73.4 (range 44–90). The majority (82.6%) were men and most (64.6%) underwent EVAR. The in-hospital mortality was 2.6%. The median follow-up was 26 months. The 3-year survival for patients with zero or one sub-threshold CPET value (VO2 at AT<10.2 ml kg⁻¹ min⁻¹, peak VO2<15 ml kg⁻¹ min⁻¹ or VE/VO2 at AT>42) was 86.4% compared with 59.9% for patients with three sub-threshold CPET values. Risk factors independently associated with survival were female sex [hazard ratio (HR)=0.44, 95% confidence interval (CI) 0.22–0.85, P=0.015], diabetes (HR=1.95, 95% CI 1.04–3.69, P=0.039), preoperative statins (HR=0.58, 95% CI 0.38–0.90, P=0.016), haemoglobin g dl⁻¹ (HR=0.84, 95% CI 0.74–0.95, P=0.006), peak VO2<15 ml kg⁻¹ min⁻¹ (HR=1.63, 95% CI 1.01–2.63, P=0.046), and VE/VO2 at AT>42 (HR=1.68, 95% CI 1.00–2.80, P=0.049).

Conclusions. CPET variables are independent predictors of reduced survival after elective AAA repair and can identify a cohort of patients with reduced survival at 3 years post-procedure. CPET is a potentially useful adjunct for clinical decision-making in patients with AAA.

Keywords: abdominal aortic aneurysm; cardiopulmonary exercise test; cardiovascular surgical procedure; endovascular procedures

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Accurate assessment of perioperative risk and prediction of long-term clinical outcomes are essential in elective abdominal aortic aneurysm (AAA) repair as for most patients it is a prophylactic procedure. Several methods of assessing perioperative risk have been proposed in patients undergoing AAA repair, including risk prediction models, biomarkers, assessment of functional capacity, and genetic testing. Recent guidelines have emphasized that when indicated, a preoperative assessment of a patient’s functional capacity should be performed for patients undergoing major vascular surgery.†

Cardiopulmonary exercise testing (CPET) provides a ‘gold standard’ assessment of functional capacity. It has been used in elite sport performance and research for some time and is now increasingly utilized in the preoperative assessment of patients before major non-cardiac surgery. CPET has been
used to identify patients at increased risk of adverse perioperative outcomes in a variety of settings. The evidence for its role in risk stratifying patients undergoing AAA repair has so far been limited to a number of small single-centre studies. As a result of this there is uncertainty about its usefulness in the preoperative assessment of patients with AAA. A recent systematic review called for more research into its role in the preoperative assessment of patients undergoing vascular surgery.

A previous study by our group demonstrated that variables derived from CPET were independent predictors of 30- and 90-day mortality after elective AAA repair. While short-term outcomes are clearly important for both patients and clinicians, better understanding of the risks of mid-term adverse outcomes is important for clinical decision-making. The objective of this study was therefore to investigate whether preoperative CPET-derived variables are predictors of survival after elective open and endovascular AAA repair (EVAR).

**Methods**

Data were collected prospectively as part of the standard multi-disciplinary assessment on all patients who underwent a symptom-limited maximal exercise CPET before elective AAA repair at Central Manchester Foundation Trust and University Hospital of South Manchester between January 24, 2007 and October 1, 2012. The cohort significantly over-laps with a previous study by our group on CPET and perioperative mortality after elective AAA repair. Both contributing hospitals are part of Vascular Governance North West which has both NRES Committee North West (09/H1010/2 + 5) and Section 251 approval. As stated in the terms of the VGNW ethical approval, because this project involved the analysis of pseudonymous, non-identifiable patient data, specific ethical approval was not required.

CPET was performed using a cycle ergometer and a ramped test (Wasserman) protocol, with the Ultima™ CardiO2MedGraphics equipment (Medical Graphics, St Paul, MN, USA) linked into the BreezeSuite™ software package (Medical Graphics, St Paul, MN, USA). CPET equipment was maintained under manufacturer maintenance contracts and calibrated before testing, in keeping with manufacturer recommendations. All CPET tests were performed and interpreted by appropriately trained consultant anaesthetists to a set of standardized clinical criteria across the two participating centres.

Baseline data were recorded and the patient then cycled for 3 min with no resistance at a rate of ~60 rpm. After these 3 min increasing resistance was applied at between 5 and 20 W min⁻¹. Each CPET was performed to achieve maximal patient effort. Criteria used to determine whether maximal effort was achieved were (i) heart rate >80% of predicted peak heart rate, (ii) respiratory exchange ratio >1.15, (iii) criteria for ventilatory limitation to exercise reached (breathing reserve <15%). The CPET was terminated if ST depression of >2 mm on the exercise ECG was observed, a cadence of >40 rpm could not be maintained, the patient experienced distressing cardiorespiratory or musculoskeletal symptoms or at the request of the patient. After the test patients were monitored until cardiorespiratory parameters returned to baseline levels. Data for the following CPET variables were collected: VO₂ at anaerobic threshold (AT) in millilitre per kilogram per minute, peak VO₂ in millilitre per kilogram per minute, and VE/VO₂. The following discriminatory thresholds for these CPET variables were selected a priori based on published studies shown to identify those at increased risk of morbidity and death among patients undergoing major non-cardiac surgery; VO₂ at AT <10.2 ml kg⁻¹ min⁻¹, peak VO₂ <15 ml kg⁻¹ min⁻¹, and VE/VO₂ at AT >42. Absolute patient weight in kilograms was used to calculate all variables. AT was determined using a combination of V-slope and ventilatory equivalent methods and recorded in millilitre per kilogram per minute. VE/VO₂ was recorded at AT, or when AT was unclear, taken to be the lowest recorded value during the incremental part of the exercise test.

Inducible cardiac ischaemia (ICI) was recorded when >1 mm of ST-segment depression in two or more adjacent ECG leads on the CPET exercise ECG, gas analysis changes, or both consistent with ischaemia were present. Reversible ischaemia present on either stress myoview or dobutamine stress echocardiogram within 5 years of surgery was also classified as ICI. Patients continued their usual medication up until CPET testing and heart rate limiting medications were not stopped. Patient co-morbidity data were collected either by the clinician responsible for the patient or by a clinical audit team. Preoperative laboratory investigations included haemoglobin (anaemia defined as <13.0 g dl⁻¹ for men and <11.0 g dl⁻¹ for women), urea (abnormal defined as >7.5 mmol l⁻¹), creatinine (abnormal defined as >120 μmol l⁻¹), and diagnosis of a juxta-supra renal AAA as defined by the operating surgeon. The primary outcome measure was survival after elective AAA repair. The follow-up data were collected using the NHS Demographic Batch Service on August 1, 2013.

**Statistical analysis**

All variables missing for more than 15% of subjects were excluded from analysis. For remaining variables, missing data were imputed with the median value for continuous variables other than CPET measurements and survival; the NHS Demographic Batch Service on August 1, 2013. Continuous variables were compared between open AAA repair and EVAR groups using an independent samples Student t-test for continuous variables and the χ² test for dichotomous variables. Categorical and dichotomous variables were examined graphically using Kaplan–Meier graphs, and compared using the log-rank test.

Continuous variables were assessed by fitting univariable Cox proportional hazards (PHs) regression models. The functional form of continuous variables other than CPET measurements was assessed by fitting smoothing curves to Martingale residual plots.
Multivariable Cox PH models were developed by including variables that were significant at the $P<0.20$ level at univariable analysis. The PH assumption was formally and graphically assessed using the Grambsch–Therneau test based on scaled Schoenfeld residuals. Variables found to significantly violate the PH assumption were used to stratify the baseline hazards function. All statistical analyses were performed using the R (version 3.0.1) statistical computing software. A $P$-value of $<0.05$ was considered statistically significant.

## Results

### Patient characteristics and in-hospital mortality

During the study period 506 patients performed a preoperative CPET and went on to have elective AAA repair. The median time between CPET and surgery was 56 days (first quartile—third quartile 26–90). The mean age at operation was 73.4 (range 44–90) and the majority (82.6%) of patients were men. The majority (327, 64.6%) of patients underwent endovascular AAA repair (Table 1). Patients undergoing EVAR were more likely to be older, male, have a history of IHD and demonstrate limited functional capacity (as determined by CPET). They had lower haemoglobin levels and were less likely to have a juxta-supra renal AAA repair. AT could not be determined in 53 (10.5%) patients. The in-hospital mortality rate was 1.86% in the EVAR group, 4.00% in the open repair group and 2.61% overall.

### Survival analysis

The median follow-up time was 26 months with a maximum follow-up time of 67 months. There were 90 deaths overall in the study cohort. Inspection of the Kaplan–Meier graph stratified by open surgery and EVAR (Fig. 1) demonstrated that operation type failed to satisfy the PH assumption (Grambsch–Therneau test $P=0.007$) because of a crossing in the curves at $\approx 6$ months. Therefore, the model was stratified on this variable. After univariable analysis, the following variables were significant at the $P<0.20$ level: age ($P=0.013$), sex ($P=0.157$), diabetes ($P=0.035$), inducible cardiac ischaemia ($P=0.147$), statins ($P=0.019$), creatinine ($P<0.001$), elevated urea ($P=0.001$), haemoglobin ($P<0.001$), and the CPET variables, VE/VCO$_2$ at AT$<42$ ($P=0.005$), peak VO$_2<15$ ml kg$^{-1}$ min$^{-1}$ ($P<0.001$), and AT$<10.2$ ml kg$^{-1}$ min$^{-1}$ ($P=0.003$). The number of sub-threshold CPET variables was an important risk factor for reduced survival (Fig. 2, $P<0.001$). Patients with zero or one sub-threshold CPET variables had a 3-year survival of 86.4% compared with 59.9% in patients with three sub-threshold CPET variables (Table 2).

A strong linear relationship between peak VO$_2$ (ml kg$^{-1}$ min$^{-1}$) and AT (ml kg$^{-1}$ min$^{-1}$) for all pairwise-complete records was demonstrated (Pearson’s sample correlation coefficient $r=0.81$; regression slope $=0.508$, $P<0.001$) (Fig. 3). As there is significantly more missing data for AT, but the relationship between AT and peak VO$_2$ is strong, which would introduce collinearity into the regression modelling, AT was not included in the multivariable analyses. The final model is given in Table 3.

### Table 1 Differences in patient characteristics for patients undergoing open AAA repair and EVAR. Continuous data (with the exception of age which is shown as mean and range) are shown as mean (sd) and dichotomous data are shown as number (percentage). $P$-value calculated using $\chi^2$ test (dichotomous variables) and independent samples Student’s $t$-test (continuous variables): AAA, abdominal aortic aneurysm; EVAR, endovascular aneurysm repair; M, male; F, female; AT, anaerobic threshold; VE/VCO$_2$, ventilatory equivalents for carbon dioxide; peak VO$_2$, peak oxygen consumption; ICI, inducible cardiac ischaemia

<table>
<thead>
<tr>
<th>Patient characteristic</th>
<th>Overall cohort, $n = 506$ (%)</th>
<th>Open repair, $n = 179$ (%)</th>
<th>EVAR, $n = 327$ (%)</th>
<th>$P$-value</th>
<th>Missing data, $n = 506$ (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>73.4 (44–90)</td>
<td>70.5 (48–86)</td>
<td>75.0 (44–90)</td>
<td>$&lt;0.001$</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Female</td>
<td>88 (17.4)</td>
<td>40 (22.3)</td>
<td>48 (14.7)</td>
<td>0.040</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Diabetes</td>
<td>48 (9.5)</td>
<td>14 (7.8)</td>
<td>34 (10.4)</td>
<td>0.431</td>
<td>25 (4.9)</td>
</tr>
<tr>
<td>Ischaemic heart disease</td>
<td>227 (44.9)</td>
<td>56 (31.3)</td>
<td>171 (52.3)</td>
<td>$&lt;0.001$</td>
<td>29 (5.7)</td>
</tr>
<tr>
<td>Treated hypertension</td>
<td>236 (46.6)</td>
<td>87 (48.6)</td>
<td>149 (45.6)</td>
<td>0.574</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Antplatelet medication</td>
<td>384 (75.9)</td>
<td>137 (76.5)</td>
<td>247 (75.5)</td>
<td>0.886</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Statin</td>
<td>383 (75.7)</td>
<td>133 (74.3)</td>
<td>250 (76.5)</td>
<td>0.667</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>Haemoglobin (g litre$^{-1}$)</td>
<td>13.3 ± 1.8</td>
<td>13.6 ± 1.9</td>
<td>13.2 ± 1.8</td>
<td>0.022</td>
<td>1 (0.2)</td>
</tr>
<tr>
<td>Urea (mmol litre$^{-1}$)</td>
<td>7.2 ± 2.7</td>
<td>7.1 ± 2.5</td>
<td>7.3 ± 2.9</td>
<td>0.407</td>
<td>1 (0.2)</td>
</tr>
<tr>
<td>Creatinine (μmol litre$^{-1}$)</td>
<td>103.7 ± 51.6</td>
<td>99.3 ± 31.8</td>
<td>106.0 ± 59.6</td>
<td>0.099</td>
<td>1 (0.2)</td>
</tr>
<tr>
<td>Supra/juxta renal AAA diameter (mm)</td>
<td>59 (11.7)</td>
<td>46 (25.7)</td>
<td>13 (4.0)</td>
<td>$&lt;0.001$</td>
<td>14 (2.8)</td>
</tr>
<tr>
<td>AT $&lt;10.2$ ml kg$^{-1}$ min$^{-1}$</td>
<td>63.1 ± 10.1</td>
<td>64.0 ± 11.0</td>
<td>62.5 ± 9.5</td>
<td>0.125</td>
<td>12 (2.4)</td>
</tr>
<tr>
<td>VE/VCO$_2$ at AT $&gt;42$</td>
<td>241 (47.6)</td>
<td>55 (30.7)</td>
<td>186 (56.9)</td>
<td>$&lt;0.001$</td>
<td>53 (10.5)</td>
</tr>
<tr>
<td>Peak VO$_2&lt;15$ ml kg$^{-1}$ min$^{-1}$</td>
<td>79 (15.6)</td>
<td>17 (9.5)</td>
<td>62 (19.0)</td>
<td>0.007</td>
<td>2 (0.4)</td>
</tr>
<tr>
<td>ICI</td>
<td>49 (9.7)</td>
<td>12 (6.7)</td>
<td>37 (11.3)</td>
<td>0.129</td>
<td>3 (0.6)</td>
</tr>
<tr>
<td>$\geq$2 sub-threshold CPET values</td>
<td>213 (42.1)</td>
<td>49 (27.4)</td>
<td>164 (50.2)</td>
<td>$&lt;0.001$</td>
<td>0 (0.0)</td>
</tr>
<tr>
<td>3 sub-threshold CPET values</td>
<td>54 (10.7)</td>
<td>10 (5.6)</td>
<td>44 (13.5)</td>
<td>0.010</td>
<td>0 (0.0)</td>
</tr>
</tbody>
</table>
Patient characteristics associated with reduced survival included male sex, diabetes not taking preoperative statins, low haemoglobin, VE/\dot{V}\text{CO}_2 > 42 at AT, and peak \dot{V}O_2 < 15 ml kg\(^{-1}\) min\(^{-1}\). The final model satisfied the assumption of proportional hazards (Grambsch–Therneau test \(P = 0.285\)).

### Table 2: Survival after elective AAA repair stratified by number of sub-threshold CPET variables

<table>
<thead>
<tr>
<th>Number of sub-threshold CPET variables</th>
<th>1-year survival</th>
<th>3-year survival</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–1</td>
<td>94.4%</td>
<td>86.4%</td>
</tr>
<tr>
<td>2</td>
<td>86.9%</td>
<td>78.0%</td>
</tr>
<tr>
<td>3</td>
<td>76.7%</td>
<td>59.9%</td>
</tr>
</tbody>
</table>

**Discussion**

This study demonstrates that variables derived from preoperative CPET testing are independent risk factors for reduced survival after elective AAA repair. Patients with multiple sub-threshold CPET values had significantly reduced survival compared with those with zero or one abnormal value. Peak \dot{V}O_2 (< 15 ml kg\(^{-1}\) min\(^{-1}\)) and VE/\dot{V}\text{CO}_2 (> 42) were independent predictors of reduced survival. These results are applicable to patients undergoing both open AAA repair and EVAR.

This is the largest study to date exploring the association between preoperative CPET variables and survival after elective AAA repair. It is also the first study to report that CPET variables are associated with survival in a cohort of patients that includes patients undergoing EVAR. Coupled with the previous analysis on short-term outcomes after elective AAA repair conducted...
by our group, each CPET variable studied has been shown to be potentially useful for predicting outcomes after elective AAA repair. For 30-day mortality VO₂ at AT<10.2 ml kg⁻¹ min⁻¹ is an independent predictor of outcome with a peak VO₂<15 ml kg⁻¹ min⁻¹ being an independent predictor of 90-day mortality. For survival a peak VO₂<15 ml kg⁻¹ min⁻¹ and a VE/VCO₂ at AT of ≥42 were independent predictors. An AT of <10.2 ml kg⁻¹ min⁻¹ may have been an independent predictor of reduced survival but it was not examined in the multivariable analysis because of collinearity.

AT and peak VO₂ are both measures of aerobic or functional capacity and it is therefore not surprising that a strong linear relationship between the two was demonstrated. The association between AT and early mortality has been demonstrated in series of patients undergoing major non-cardiac surgery which have included some open AAA repairs. AT is not reliant on patient motivation and has been shown to be a reproducible measure of aerobic capacity in preoperative patients. Although its estimation can be subjective it has been shown to be reliably interpreted between different clinicians. However, the AT may not be apparent in all patients. The approach adopted for imputing missing AT data in this study was selected by the research team as it was felt that for patients in whom AT could not be determined it was more likely to be sub-threshold. This approach is a potential limitation and as a result peak VO₂ rather than AT was included in the multivariable analysis.

Peak VO₂ is simply the highest VO₂ achieved by an individual during an exercise test. The peak VO₂ achieved during a test is therefore effort dependent. VO₂ max represents the limit of functional capacity for an individual and is reached when there is a plateauing of the VO₂ response to exercise despite an increasing work rate. VO₂ max is rarely achieved in clinical practice but when a maximal effort CPET is performed by an individual, the peak VO₂ achieved should provide a reasonable reflection of their VO₂ max. In this study, all CPET was performed with the intention of achieving maximal patient effort. This is not the case in other centres in the UK where sub-maximal testing may be performed. Given these results, where safe and feasible, CPET should be performed to a maximal effort to facilitate risk stratification. Peak VO₂ is also associated with increased perioperative complications in patients undergoing lung resection surgery. An elevated VE/VCO₂ (≥42) has previously been shown to be an important predictor of mid-term mortality in AAA repair. An elevated VE/VCO₂ is likely to be multi-factorial in nature and represent systemic disease severity. In patients with heart failure abnormal VE/VCO₂ has been significantly correlated with increased ventilation perfusion mismatch, decreased cardiac output, elevated pulmonary pressures, decreased alveolar-capillary membrane conductance, and diminished heart rate variability. VE/VCO₂ as a predictor has the advantage of high test reliability and does not depend on the mode of exercise or testing protocol used. Other risk factors that were associated with an increased risk of reduced survival in this study included low preoperative haemoglobin, not taking preoperative statins, diabetes, and male sex. Anaemia has been found to correlate with unfavourable outcomes in both surgical and non-surgical populations, and has been found to be associated with reduced long-term survival after EVAR. This study adds further evidence to the existing literature that statin usage is associated with improved outcomes after AAA repair. Diabetes has previously been found to be associated with reduced survival in patients undergoing AAA repair. The improved survival in women in this study is unusual and may be a reflection of patient selection practices at the two centres.

This study represents contemporary practice at two tertiary vascular centres with good in-hospital mortality rates. CPET was performed as part of routine multi-disciplinary preoperative assessment and was utilized in clinical decision-making. As expected because of the observational nature of the study, there were significant differences in the patient characteristics between the open AAA repair and EVAR groups. Patients with limited functional capacity were more likely to undergo EVAR and those undergoing open repair were more likely to have unfavourable anatomy for EVAR however the objective of the study was not to compare treatment groups. Although this is the largest study to date of CPET in AAA repair, the sample size remains relatively small for a modelling study.

All CPET data were collected prospectively in a standardized way across the two centres, however, a potential limitation of the study is that tests were not independently reviewed before analysis to ensure standardization. This was not felt to be necessary by the research team attributable to the standardization of methods across the two centres. A limitation of the study is that the recording of ICI differed between the two centres, with one deriving ICI exclusively from the CPET test and the other recording ICI as present when demonstrated on non-invasive stress testing. Therefore, since ICI was not exclusively defined by CPET testing it has not been included as a CPET variable for this study.

A potential limitation of the analysis approach is that although CPET variables are recorded as continuous variables

### Table 3 Cox proportional hazards multivariable analysis for survival after elective AAA repair. The baseline hazard function is stratified on operation type (open surgery or EVAR). HR, hazard ratio; CI, confidence interval; AT, anaerobic threshold; VE/VCO₂, ventilatory equivalents for carbon dioxide; peak VO₂, peak oxygen consumption

<table>
<thead>
<tr>
<th>Variable</th>
<th>Hazard ratio (95% CI)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>1.008 (0.977–1.041)</td>
<td>0.603</td>
</tr>
<tr>
<td>Female</td>
<td>0.436 (0.224–0.849)</td>
<td>0.015</td>
</tr>
<tr>
<td>Diabetes</td>
<td>1.954 (1.035–3.687)</td>
<td>0.039</td>
</tr>
<tr>
<td>Inducible cardiac ischaemia</td>
<td>1.640 (0.901–2.986)</td>
<td>0.106</td>
</tr>
<tr>
<td>Statin</td>
<td>0.583 (0.376–0.905)</td>
<td>0.016</td>
</tr>
<tr>
<td>Creatinine (μmol litre⁻¹)</td>
<td>1.002 (0.998–1.006)</td>
<td>0.278</td>
</tr>
<tr>
<td>Urea (mmol litre⁻¹)</td>
<td>1.066 (0.988–1.151)</td>
<td>0.101</td>
</tr>
<tr>
<td>Haemoglobin (g dl⁻¹)</td>
<td>0.842 (0.744–0.953)</td>
<td>0.006</td>
</tr>
<tr>
<td>VE/VCO₂ ≥42 at AT</td>
<td>1.628 (1.009–2.627)</td>
<td>0.046</td>
</tr>
<tr>
<td>Peak VO₂&lt;15 (ml kg⁻¹ min⁻¹)</td>
<td>1.676 (1.002–2.803)</td>
<td>0.049</td>
</tr>
</tbody>
</table>
they were dichotomized for this analysis. This was a pragmatic choice based on clinical judgement and previously published studies, to balance model fit and model complexity given the relatively small number of outcomes. Although the median follow-up time is relatively short, 3-year survival is clearly an important outcome after elective AAA repair as data from randomized controlled trials suggests that from ~2 years onwards survival is the same for patients who undergo open AAA repair or EVAR.

This study demonstrates that preoperative CPET to maximal effort can identify patients with reduced survival after elective AAA repair independent of the type of repair. These risks can be weighed against the risk of AAA rupture which is frequently expressed in terms of rupture risk per year to facilitate clinical decision-making. The costs of CPET are relatively low at approximately £200 per patient at our centres. CPET is also safe with only a minimal risk of adverse events. However, the exact value of its contribution to preoperative assessment along with its cost-effectiveness is still uncertain. Further studies assessing the utility of CPET alongside clinical prediction models are required.

Authors’ contributions
All authors were involved in the conception and design of the study. N.A.W., A.C.P., D.A. performed the CPET tests. E.D.C. and R.A.H. collected and collated the data. E.D.C. and S.W.G. cleaned the data. G.L.H. and E.D.C. performed the analysis with input from S.W.G. S.W.G. drafted the manuscript which was revised by G.L.H., N.A.W., D.A., and C.N.M. All authors approved the final version for submission. C.N.M. will act as guarantor for the study.

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Declaration of interest
None declared.

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References

435
22 Grambsch PM, Therneau TM. Proportional hazards tests and diagnostics based on weighted residuals. Biometrika 1994; 81: 515–26

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