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The role of cardiopulmonary exercise testing and echocardiography prior to elective endovascular aneurysm repair

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ABSTRACT

INTRODUCTION Cardiopulmonary exercise testing (CPET) and transthoracic echocardiography (TTE) are common preparative investigations prior to elective endovascular aneurysm repair (EVAR). Whether these investigations can predict survival following EVAR and contribute to shared decision making is unknown.

METHODS Patients who underwent EVAR at a tertiary centre between June 2007 and December 2014 were identified from the National Vascular Registry. Variables obtained from preoperative investigations were assessed for their association with survival at three years. Regression analysis was used to determine variables that independently predicted survival at three years.

RESULTS A total of 199 patients underwent EVAR during the study period. Of these, 120 had preoperative CPET and 123 had TTE. Lower forced expiratory ventilation (FEV_1), ratio of FEV_1 to forced vital capacity, work at peak oxygen consumption and higher ventilatory equivalent for carbon dioxide were associated with increased mortality. Variables obtained from TTE were not associated with survival at three years although there was a low incidence of left ventricular systolic dysfunction and significant valvular disease in this cohort.

CONCLUSIONS CPET might be a useful adjunct to assist in shared decision making in patients undergoing elective EVAR and may influence anaesthetic technique. TTE does not appear to be able to discriminate between high and low risk individuals. However, a low rate of significant ventricular dysfunction and valvular disease in patients undergoing elective EVAR may account for these findings.

KEYWORDS

Preoperative testing – Abdominal aortic aneurysm – Endovascular – Cardiopulmonary exercise testing – Echocardiography

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Introduction

Endovascular aneurysm repair (EVAR) is an alternative to open surgical repair for abdominal aortic aneurysm (AAA) in patients who are suitable for either procedure.[1] Compared with open repair, in patients suitable for either, EVAR is associated with improved short-term survival and shorter hospital stay.[2] However, favourable immediate outcomes are not translated into any long-term survival advantage and EVAR is significantly more expensive than conventional open repair.[1,2] Among patients who are unfit for open AAA repair, those undergoing EVAR benefit from lower AAA related mortality but overall longer-term survival is not significantly different.[3]

In 2009, the National Institute for Health and Clinical Excellence (NICE) published a review on the use of EVAR for elective AAA repair, and recommended the development of assessment tools to stratify patients by procedure related mortality.[4] Preoperative testing must predict not only perioperative risk but also the perceived prognostic benefit of what is a prophylactic procedure.[4,5] Draft guidance on the diagnosis and management of AAA published by NICE in 2018 suggested that cardiopulmonary exercise testing (CPET) might be used to assist shared decision making but cited a lack of evidence to support its routine use.[6]

Much of the reduced survival following EVAR compared with age and sex matched controls is attributable to cardiovascular disease. Transthoracic echocardiography (TTE) might therefore also have a role in risk stratification and is recommended in European guidelines for selected patients but this has not been assessed in a systematic manner.[7,8] The aims of this study were to determine whether variables from preoperative CPET and TTE could

predict survival at three years in a cohort undergoing elective EVAR for AAA.

Methods

This was a retrospective observational cohort study of prospectively collected data. It was designed and reported according to the STROBE (Strengthening the Reporting of Observational Studies in Epidemiology) statement.[9] The study was undertaken at Leeds Teaching Hospitals NHS Trust, which is a tertiary referral centre for vascular surgery and interventional radiology, with access to CPET and TTE.

Patients aged ≥18 years who underwent elective EVAR for AAA between 1 June 2007 and 31 December 2014 at our institute were identified from the National Vascular Registry (NVR). Those with ruptured AAA, trauma related AAA and mycotic aneurysms were excluded from the study. The NVR data for each patient were confirmed with reference to the electronic healthcare record and written medical record.

Variables

Patients undergoing elective EVAR were referred for preoperative CPET and TTE as part of usual care. Any investigations performed within one year prior to EVAR were regarded as contributing to preoperative evaluation and were included in our analysis. Baseline characteristics recorded were comparable with those reported in the EVAR 1 and EVAR 2 trials,[2,3] including age, sex, AAA diameter, history of hypertension, chronic obstructive pulmonary disease, diabetes mellitus, ischaemic heart disease, peripheral vascular disease and chronic kidney disease. Smoking history, renal function by serum creatinine, and medications including antiplatelets and statin therapy were recorded. Where data were missing from the NVR, these variables were obtained from local electronic healthcare records.

Cardiopulmonary exercise testing

CPET was performed on a cycle ergometer under the supervision of an appropriately trained anaesthetist and cardiac physiologist. In a controlled setting with resuscitation equipment available, patients are placed on a ramp of increasing resistance to achieve power increments of either 10W, 15W or 20W to maximal exertion. The cycle ergometer is linked to a software package (Breeze software, Welch Allyn, New York, US), with continuous electrocardiogram (ECG) monitoring and a metabolic cart (Medgraphics, King's Lynn, UK) containing a gas analyser allowing for continuous analysis of inspired and expired oxygen (O_2) and carbon dioxide (CO_2).

CPET was terminated at either patient perceived maximal exertion, at predetermined parameters, or owing to distressing cardiopulmonary or musculoskeletal symptoms. Predetermined parameters were ECG evidence of cardiac ischaemia (defined as ≥2mm ST depression or any ST elevation), other adverse ECG findings or a failure to maintain cadence. In keeping with previous studies,[10] threshold values for important CPET variables were defined as anaerobic threshold (AT) <10.2ml/kg/min, peak O_2 consumption (peak $\dot{V}O_2$) <15ml/kg/min and ventilatory equivalent for CO_2 ($\dot{V}E/\dot{V}CO_2$) >42ml/kg/min. The following variables were recorded for CPET: forced expiratory volume in one second (FEV_1), forced vital capacity (FVC), FEV_1:FVC ratio (FEV_1/FVC,) work at maximal O_2 consumption (work at peak $\dot{V}O_2$), O_2 consumption at anaerobic threshold (AT), maximal O_2 consumption (peak $\dot{V}O_2$), ST segment changes on ECG monitoring, resting heart rate (HR), HR at peak $\dot{V}O_2$, resting O_2 pulse, O_2 pulse at peak $\dot{V}O_2$ and ($\dot{V}E/\dot{V}CO_2$) at AT.

Transthoracic echocardiography

TTE was performed by a British Society of Echocardiography (BSE) accredited sonographer; all imaging acquisition and measurements were consistent with recommendations by the BSE. Left ventricular ejection fraction (LVEF) was measured by modified biplane Simpson's rule where endocardial definition allowed. LVEF was regarded as normal if 50–70%, as mildly impaired if 40–49%, as moderately impaired if 30–39% and as severely impaired if <30%.

TTE variables that were included in our analysis comprised: left atrial diameter; interventricular septum thickness; left ventricular internal diameter in diastole; fractional shortening; LVEF; left ventricular outflow tract diameter; diameter of the aorta at sinus of Valsalva, sinotubular junction, proximal ascending aorta, aortic arch and descending aorta; right ventricular diameter; pulmonary artery pressure; peak velocity of early diastolic mitral flow (E); peak velocity of late diastolic mitral flow (A); and E:A ratio. Valvular function was reported as either normal, trivial, mild, moderate or severe for either regurgitation or stenosis of aortic, mitral, tricuspid and pulmonary valves. For the purpose of analysis, valvular dysfunction was dichotomised and regarded as significant if there was either moderate or severe regurgitation or stenosis.

Outcomes

The primary outcome was all-cause mortality for up to three years. All-cause mortality for all patients was identified from our local electronic healthcare system. This is linked to the Office for National Statistics, which records all births and deaths in the UK.

Statistical analysis

Baseline variables for patients who did or did not undergo preoperative investigation, and for patients who were alive or dead after three years were compared using the chi-squared test and t-test for categorical and continuous variables, respectively. Normal distribution was tested using the Kolmogorov–Smirnov statistic. For variables recorded for CPET and TTE, survival analysis was performed using Cox regression, adjusted for age and sex. In all analyses, a p-value of <0.05 was considered statistically significant and listwise deletion was used for missing cases. All statistical analysis was performed using SPSS® version 23 (IBM, New York, US).

Ethical considerations

This study was regarded as service evaluation and did not therefore require specific ethical approval or patient consent, providing usual data protection was in place. Only members of the clinical team had access to routinely collected data, which were anonymised at the point of analysis. No specific funding was used for the completion of this study.

Results

During the study period, 199 patients were recorded in the NVR as having undergone elective EVAR at our institution. Of these, 149 (75%) underwent some form of preoperative investigation. A total of 120 patients had CPET while 123 had TTE within a year prior to EVAR (Table 1). Overall, 172 patients (86%) were male. The mean age was 76.0 years (standard deviation [SD]: 7.7 years) and the mean preoperative AAA diameter was 63.7mm (SD: 10.0mm). The mean follow-up duration was 4.9 years (SD: 2.6 years), during which time 98 patients (49.2%) died. The 30-day mortality rate was 1.5% (n=3). Complete follow-up data were available for all patients at 3 years and for 143 patients (72%) at 5 years.

A complete dataset was available for most comorbidities, with the exception of smoking history, which was recorded for 169 cases (84.9%). A total of 39 patients (19.5%) were hypertensive, 61 (30.5%) had ischaemic heart disease and 67 (38.8%) had a smoking history. The majority were receiving cardioprotective medications: 141 (70.9%) were prescribed a statin and 129 (64.8%) antiplatelet medication. Age, sex, AAA diameter and major comorbidities were not significantly different between those who underwent preoperative investigation and those who did not (Table 2).

Survival

The all-cause mortality rate at 30 days, 1, 2, 3 and 5 years was 1.5% (n=3), 6.5% (n=13), 14.1% (n=28), 23.1% (n=46) and 37.1% (n=53) respectively. In unadjusted survival

Table 1 Number of patients undergoing preoperative investigation prior to endovascular aneurysm repair

Investigation	*n*
Total	199
TTE	123
CPET	120
TTE only	29
CPET only	26
TTE and CPET	94
No investigations	50

CPET = cardiopulmonary exercise testing; TTE = transthoracic echocardiography

Table 2 Clinical characteristics for patients undergoing cardiopulmonary exercise testing and/or transthoracic echocardiography and for patients who did not have any investigations

Variable	All (n=199)	Any investigation (n=149)	No investigations (n=50)	*p*-value
Mean age in years	76.0 (SD: 7.7)	76.0 (SD: 7.8)	76.2 (SD: 7.4)	0.865
Male sex	172 (86.4%)	129 (86.6%)	43 (86.0%)	0.918
Mean AAA ø in mm	63.7 (SD: 10.0)	63.2 (SD: 9.1)	65.2 (SD: 11.9)	0.248
Hypertension	39 (19.6%)	27 (18.1%)	12 (24.0%)	0.365
COPD	17 (8.5%)	12 (8.0%)	5 (10.0%)	0.670
Diabetes	16 (8.0%)	14 (9.4%)	2 (4.0%)	0.225
Smoking history	65 (32.7%)	42 (28.2%)	23 (47.9%)	**0.010**
IHD	61 (30.7%)	45 (30.2%)	16 (32.0%)	0.811
CHF	11 (5.5%)	9 (6.0%)	2 (4.0%)	0.585
CVD	4 (2.0%)	2 (1.3%)	2 (4.0%)	0.247
CKD	8 (4.0%)	7 (4.7%)	1 (2.0%)	0.401
Mean creatinine in μmol/l	101.1 (SD: 51.0)	104.4 (SD: 56.5)	92.0 (SD: 29.2)	0.152
Statin	141 (70.9%)	109 (73.2%)	32 (64.0%)	0.218
Antiplatelet	129 (64.8%)	101 (67.8%)	29 (58.0%)	0.243

AAA = abdominal aortic aneurysm; CHF = chronic heart failure; CKD = chronic kidney disease; COPD = chronic obstructive pulmonary disease; CVD = cerebrovascular disease; IHD = ischaemic heart disease; SD = standard deviation

analysis, there was no significant difference in outcomes between those who underwent preoperative assessment and those who did not (Fig 1). Baseline variables that were associated with reduced survival were age and a clinical diagnosis of chronic heart failure (Table 3).

Cardiopulmonary exercise testing

CPET was performed in 120 cases (60%), with a mean exercise time of 5.4 minutes (SD: 3.8 minutes). AT was achieved in 98 patients (81.7%). Patients had a mean FEV_1 of 2.2l (SD: 0.7l), a mean FVC of 3.7l (SD: 2.9l) and a mean FEV_1/FVC ratio of 0.6 (SD: 0.1). The mean work at peak $\dot{V}O_2$ was 83.5W (SD: 32.2W), the mean $\dot{V}O_2$ at AT was 10.9ml/kg/min (SD: 2.8ml/kg/min), the mean peak $\dot{V}O_2$ was 14.7ml/kg/min (SD: 3.6ml/kg/min) and the mean $\dot{V}E/\dot{V}CO_2$ at AT was 36.8ml/kg/min (SD: 8.5ml/kg/min). AT was <10.2ml/kg/min in 43 patients, peak $\dot{V}O_2$ was <15ml/kg/min in 65 patients and $\dot{V}E/\dot{V}CO_2$ was >42ml/kg/min in 38 patients. In 4 cases (3.3%), there were ischaemic ECG changes, the average resting HR was 74.7bpm (SD: 15.0bpm), increasing to 112.4bpm (SD: 21.4bpm) at peak $\dot{V}O_2$. The mean resting O_2 pulse was 4.35ml/beat (SD: 1.6ml/beat) and the mean O_2 pulse at peak $\dot{V}O_2$ was 10.7ml/beat (SD: 2.78ml/beat).

At three years, unadjusted survival was significantly associated with a lower FEV_1, FEV_1/FVC, work at peak $\dot{V}O_2$, peak $\dot{V}O_2$, O_2 pulse at peak $\dot{V}O_2$ and subthreshold $\dot{V}O_2$, and a higher $\dot{V}E/\dot{V}CO_2$ at AT and $\dot{V}E/\dot{V}CO_2$ above threshold (Table 4). However, when adjusted for age and sex, only FEV_1, FEV_1/FVC, work at peak $\dot{V}O_2$, $\dot{V}E/\dot{V}CO_2$ at AT and $\dot{V}E/\dot{V}CO_2$ above threshold remained significant (Table 5).

Transthoracic echocardiography

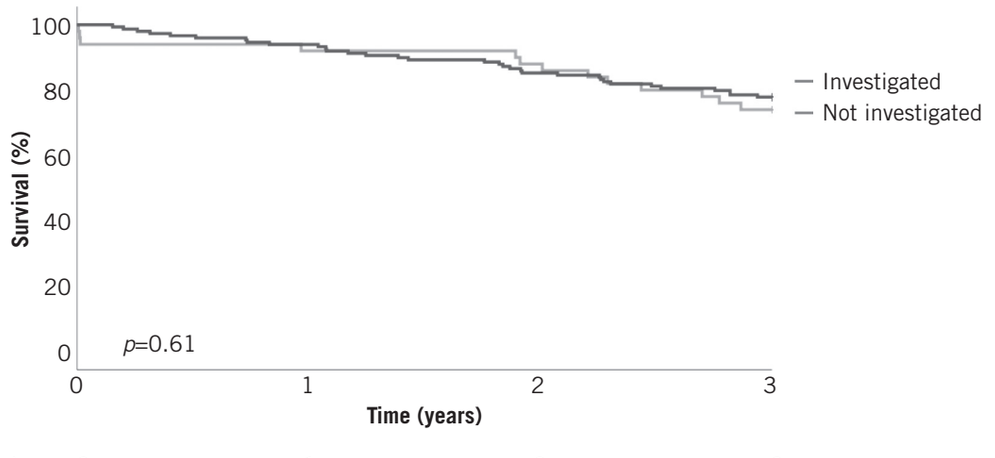
A total of 123 patients (62%) underwent TTE and imaging quality allowed for measurement of LVEF for 121 (98%) of these. The mean LVEF was 51.1% (SD: 9.2pp). LVEF was in the normal range in 86 cases (70.0%), mildly impaired in 24 (19.5%), moderately impaired in 6 (4.9%) and severely impaired in 5 (4.1%).

Image quality was sufficient to provide an assessment of aortic regurgitation (AR) in 121 cases, aortic stenosis (AS) in 121 cases, mitral regurgitation (MR) in 122 cases, mitral stenosis (MS) in 120 cases, tricuspid regurgitation (TR) in 111 cases, tricuspid stenosis (TS) in 112 cases, pulmonary regurgitation (PR) in 99 cases and pulmonary stenosis (PS) in 101 cases. AR was reported as moderate in four patients (3.3%) while AS was moderate in two cases (1.7%) and severe in one case (0.8%). This individual was asymptomatic at the time of EVAR and underwent transcatheter aortic valve implantation some years following EVAR. MR was reported as moderate for six patients (4.9%), severe for two (1.6%) and TR was reported as moderate for six patients (5.4%). There were no cases of significant MS, TS or PS but one patient (1.0%) had moderate PR.

Three-year survival was not associated with left ventricular dysfunction or significant valvular dysfunction in unadjusted analysis (Table 6). Conversely, survival was associated with right ventricular diameter although this was not significant when adjusted for age and sex (hazard ratio: 1.049, 95% confidence interval: 0.992–1.109, p=0.095).

Discussion

In our analysis of outcomes following elective EVAR, variables obtained from preoperative CPET were related to reduced



Time (years)	0	1	2	3
Investigated	149	140	127	116
Not investigated	50	46	44	37

Figure 1 Kaplan–Meier plot of survival following endovascular aneurysm repair for patients stratified by preoperative investigation

medium-term survival. A lower FEV_1, FEV_1/FVC and work at peak $\dot{V}O_2$, a higher $\dot{V}E/\dot{V}CO_2$ at AT and $\dot{V}E/\dot{V}CO_2$ above threshold were associated with reduced survival when adjusted for age and sex, as was a reduced peak $\dot{V}O_2$ but this was not statistically significant when adjusted for age and sex. TTE did not provide useful prognostic information in patients who had undergone EVAR. However, a low rate of significant left ventricular dysfunction and valvular disease was observed, which may have been due to inclusion bias. It is possible that where preoperative investigations revealed prognostically important cardiovascular disease these patients were managed conservatively.

There was low mortality at 30 days, which might suggest appropriate case selection. Nevertheless, it is notable that patients who underwent preoperative investigation did not significantly differ from those who did not (Table 2). CPET and TTE were commonly used in the preoperative assessment of patients at our institution, with 75% undergoing some form of investigation within the year prior to EVAR. A large proportion of patients in this cohort did not achieve threshold values for important CPET variables: AT, peak $\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$. If patients who are unfit for conventional open repair are offered EVAR then this would be contrary to the recommendations of the EVAR 2 trial, which found no survival advantage compared to conservative management.[11] However, CPET is just one component of preoperative assessment, and the mortality rate of this cohort does not suggest inappropriate patient selection.

CPET is a recognised preoperative assessment tool prior to major intra-abdominal surgery. It provides a reliable and objective assessment of an individual's physiological capacity, and therefore 'fitness' for a given procedure.[12] CPET has been used in patients with AAA without serious adverse events.[13] While CPET has been shown to predict mortality in various types of open surgery, there is a lack of evidence for endovascular intervention, with studies being limited by small numbers and a low event rate at 30 days.[12,14–18]

Goodyear *et al* investigated the role of CPET prior to AAA repair.[19] However, their study only included 25 patients who underwent EVAR. In a pooled analysis, Hartley *et al* found that low CPET values (AT <10.2ml/kg/min and peak $\dot{V}O_2$ <15ml/kg/min) prior to EVAR and open repair were associated with reduced survival.[20] Grant *et al* also performed a pooled analysis of open and EVAR cases, with a total of 327 patients undergoing EVAR, and found that survival was associated with peak $\dot{V}O_2$ <15ml/kg/min and $\dot{V}E/\dot{V}CO_2$ >42ml/kg/min.[10] Our study replicates these findings in a cohort only undergoing EVAR, with a significant reduction in survival for subthreshold values of $\dot{V}E/\dot{V}CO_2$ and a non-significant association with subthreshold peak $\dot{V}O_2$.

Peak $\dot{V}O_2$ provides an objective measure of fitness and is the maximal O_2 consumption a patient can achieve despite increasing work. It is effort dependent, requiring a motivated individual. Interestingly, in our cohort, although LVEF was not associated with outcomes, a clinical diagnosis of heart failure was. This is not entirely surprising, as

Table 3 Relationship between clinical characteristics and survivorship at three years

Variable	All (*n*=199)	Survivors (*n*=153)	Dead (*n*=46)	*p*-value
Preoperative investigation	149 (74.9%)	116 (75.8%)	33 (71.7%)	0.576
Mean age in years	76.0 (SD: 7.7)	78.9 (SD: 7.2)	75.2 (SD: 7.6)	**0.003**
Male sex	172 (86.4%)	132 (86.3%)	40 (87.0%)	0.906
Mean AAA Ø in mm	63.7 (SD: 10.0)	63.7 (SD: 9.4)	63.6 (SD: 11.7)	0.954
Hypertension	39 (19.6%)	32 (20.9%)	7 (15.2%)	0.393
COPD	17 (8.5%)	12 (7.8%)	5 (10.9%)	0.520
Diabetes	16 (8.0%)	10 (6.5%)	6 (13.0%)	0.155
Smoking history	65 (32.7%)	52 (40.0%)	13 (28.3%)	0.754
IHD	61 (30.7%)	50 (32.7%)	11 (23.9%)	0.258
CHF	11 (5.5%)	6 (3.9%)	5 (10.9%)	**0.011**
CVD	4 (2.0%)	3 (2.0%)	1 (2.8%)	0.928
CKD	8 (4.0%)	4 (2.6%	4 (8.7%)	0.066
Mean creatinine in µmol/l	101.1 (SD: 51.0)	100.8 (SD: 55.4)	102.3 (SD: 32.5)	0.865
Statin	141 (70.9%)	109 (71.2%)	32 (7.0%)	0.826
Antiplatelet	129 (64.8%)	100 (65.4%)	29 (19.6%)	0.773

AAA = abdominal aortic aneurysm; CHF = chronic heart failure; CKD = chronic kidney disease; COPD = chronic obstructive pulmonary disease; CVD = cerebrovascular disease; IHD = ischaemic heart disease; SD = standard deviation

heart failure is a complex syndrome, caused by a variety of pathologies, with LVEF being only one measure which correlates poorly with symptoms.

An important finding in our study was that $\dot{V}E/\dot{V}CO_2$ was linked to outcomes at three years. Failing to meet the threshold of <42ml/kg/min was associated with a near twofold increase in risk of mortality at our primary endpoint. $\dot{V}E/\dot{V}CO_2$ is a measure of the relationship between minute ventilation and CO_2 production (which is driven by muscle stimulation and therefore perfusion), and has been shown to predict mortality following open AAA repair.[14] It is consequently not surprising that this measurement had greater fidelity than peak $\dot{V}O_2$ in these multimorbid patients where the ventilation signal and autonomic activation might be particularly enhanced. $\dot{V}E/\dot{V}CO_2$ has been linked to reduced heart rate variability, diminished cardiac output and pulmonary hypertension; it is higher in those with chronic heart failure compared

Table 4 Relationship between mean cardiopulmonary exercise testing variables and survivorship at three years

Variable	All (*n*=120)	Survivors (*n*=93)	Dead (*n*=27)	*p*-value
FEV_1in l	2.2 (SD: 0.7)	2.4 (SD: 0.6)	1.8 (SD: 0.6)	**0.001**
FVC in l	3.7 (SD: 2.9)	3.9 (SD: 3.2)	3.0 (SD: 0.8)	0.224
FEV_1/FVC	65.0 (SD: 11.1)	66.7 (SD: 10.2)	58.9 (SD: 12.2)	**0.003**
Work at peak $\dot{V}O_2$ in W	83.5 (SD: 32.3)	90.3 (SD: 31.3)	60.4 (SD: 24.6)	**<0.001**
$\dot{V}O_2$ at AT in ml/kg/min	10.9 (SD: 2.8)	11.1 (SD: 2.7)	10.1 (SD: 3.1)	0.160
Peak $\dot{V}O_2$ in ml/kg/min	14.7 (SD: 3.6)	15.2 (SD: 3.6)	12.9 (SD: 3.0)	**0.004**
ST depression	4 (3.3%)	4 (4.3%)	0 (0%)	0.458
HR at rest in bpm	74.7 (SD: 15.0)	73.9 (SD: 15.6)	77.3 (SD: 12.5)	0.316
HR at peak $\dot{V}O_2$ in bpm	112.5 (SD: 21.4)	114.5 (SD: 21.5)	105.5 (SD: 19.9)	0.057
Resting O_2 pulse in ml/beat	4.3 (SD: 1.6)	4.4 (SD: 1.6)	4.0 (SD: 1.4)	0.316
O_2 pulse at peak $\dot{V}O_2$ in ml/beat	10.7 (SD: 2.8)	11.1 (SD: 2.8)	9.4 (SD: 2.5)	**0.006**
$\dot{V}E/\dot{V}CO_2$ at AT in ml/kg/min	36.8 (SD: 8.5)	35.9 (SD: 6.5)	40.5 (SD: 13.7)	**0.030**
AT <10.2ml/kg/min	42 (35.0%)	35 (37.6%)	8 (29.6%)	0.445
Peak $\dot{V}O_2$ <15ml/kg/min	65 (54.2%)	45 (48.4%)	20 (74.1%)	**0.018**
$\dot{V}E/\dot{V}CO_2$ >42ml/kg/min	38 (31.7%)	25 (26.9%)	13 (48.1%)	**0.037**

AT = anaerobic threshold; FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity; HR = heart rate; SD = standard deviation; $\dot{V}E/\dot{V}CO_2$ = ventilatory equivalent for carbon dioxide; $\dot{V}O_2$ = oxygen consumption

Table 5 Regression analysis of cardiopulmonary exercise testing variables associated with survival, adjusted for age and sex

Variable	Hazard ratio	95% CI	*p*-value
FEV_1	0.593	0.353–0.996	**0.048**
FEV_1/FVC	0.967	0.942–0.994	**0.015**
Work at peak $\dot{V}O_2$	0.980	0.970–0.991	**<0.001**
Peak $\dot{V}O_2$	0.925	0.851–1.006	0.069
O_2 pulse at peak $\dot{V}O_2$	0.893	0.787–1.013	0.078
$\dot{V}E/\dot{V}CO_2$ at AT	1.042	1.017–1.067	**0.001**
Peak $\dot{V}O_2$ <15ml/kg/min	1.339	0.786–2.280	0.283
$\dot{V}E/\dot{V}CO_2$ >42ml/kg/min	1.885	1.128–3.151	**0.016**

AT = anaerobic threshold; CI = confidence interval; FEV_1 = forced expiratory volume in one second; FVC = forced vital capacity; $\dot{V}E/\dot{V}CO_2$ = ventilatory equivalent for carbon dioxide; $\dot{V}O_2$ = oxygen consumption

with controls and in those with heart failure, it predicts adverse outcomes.[21]

TTE was a common preoperative investigation in our study, and has been proposed as a method of stratifying patients owing to an appreciation that much of the reduced survival following AAA repair compared with age and sex matched controls is due to cardiovascular disease.[7] European guidelines advise screening for cardiovascular risk factors by detailed history taking, resting ECG and clinical examination, recommending TTE in those with cardiac risk factors or positive cardiac history.[8] Despite this, the role of TTE prior to EVAR has been little evaluated.

In a prospective analysis of 273 patients undergoing elective EVAR, O'Driscoll *et al* found that variables that determined survival over a mean follow-up period of 3.2 years were lower LVEF, greater diameter of tubular ascending aorta and presence of MR.[22] The strongest predictor of survival was the presence of MR, with a stepwise increase in hazard for moderate and severe regurgitation. The authors suggested risk stratification based on the severity of MR but our study was unable to replicate these results. In our cohort, significant MR (moderate or severe) was reported in only 8 patients compared with 36 in the study by O'Driscoll *et al*.[22]

The finding that a greater tubular ascending aorta diameter was highly predictive of survival was also not replicated in our analysis and may have represented a type 1 error. However, it was not possible to account for those patients declined for EVAR on the basis of these investigations. Furthermore, a low rate of left ventricular dysfunction in this cohort might be due to inclusion bias. In those without signs, symptoms or personal history of

Table 6 Relationship between mean transthoracic echocardiography variables and survivorship at three years

Variable	All (*n*=123)	Survivors (*n*=97)	Dead (*n*=26)	*p*-value
LA ø in mm	40.2 (SD: 8.5)	39.4 (SD: 6.6)	43.0 (SD: 13.2)	0.060
IVS thickness in mm	13.0 (SD: 2.7)	12.9 (SD: 2.9)	13.0 (SD: 2.0)	0.922
LVIDd in mm	47.2 (SD: 9.1)	47.1 (SD: 8.2)	47.6 (SD: 12.1)	0.822
FS in %	25.6 (SD: 10.3)	26.6 (SD: 10.5)	23.4 (SD: 7.9)	0.233
LVEF in %	51.0 (SD: 9.2)	51.4 (SD: 8.2)	49.4 (SD: 12.2)	0.334
LVOT ø in mm	20.8 (SD: 2.1)	20.6 (SD: 2.1)	21.6 (SD: 2.0)	0.074
Ao ø at SOV in mm	33.9 (SD: 3.6)	34.0 (SD: 3.6)	33.7 (SD: 3.5)	0.710
Ao ø at STJ in mm	27.4 (SD: 4.0)	25.9 (SD: 4.0)	27.9 (SD: 4.0)	0.060
Proximal ascending Ao ø in mm	31.5 (SD: 3.7)	31.8 (SD: 3.8)	30.6 (SD: 3.1)	0.239
Ao ø at arch in mm	26.6 (SD: 4.2)	26.3 (SD: 4.1)	29.0 (SD: 4.9)	0.225
Descending Ao ø in mm	25.8 (SD: 11.8)	25.8 (SD: 11.8)	–	–
RV ø in mm	38.5 (SD: 6.3)	37.5 (SD: 5.5)	42.3 (SD: 8.0)	**0.009**
Aortic regurgitation	4 (3.3)%	3 (3.1%)	1 (3.8%)	0.143
Aortic stenosis	3 (2.4%)	3 (3.1%)	0 (0%)	0.364
Mitral regurgitation	8 (6.5%)	4 (4.1%)	4 (15.4%)	0.106
Mitral stenosis	0 (0%)	0 (0%)	0 (0%)	–
Tricuspid regurgitation	6 (4.9%)	3 (3.1%)	3 (11.5%)	0.200
Tricuspid stenosis	0 (0%)	0 (0%)	0 (0%)	–
Pulmonary regurgitation	1 (0.8%)	1 (1.0%)	0 (0%)	0.773
Pulmonary stenosis	0 (0%)	0 (0%)	0 (0%)	–
PA pressure in mmHg	32.2 (SD: 11.5)	32.7 (SD: 12.0)	31.0 (SD: 10.5)	0.621
E in m/s	0.7 (SD: 0.3)	0.7 (SD: 0.3)	0.8 (SD: 0.3)	0.387
A in m/s	0.8 (SD: 0.2)	0.8 (SD: 0.3)	0.9 (SD: 0.2)	0.816
E/A	0.9 (SD: 0.4)	0.4 (SD: 0.0)	0.4 (SD: 0.1)	0.957

A = peak velocity of late diastolic mitral flow; Ao = aorta; E = peak velocity of early diastolic mitral flow; FS = fractional shortening; IVS = interventricular septum; LA = left atrium; LVEF = left ventricular ejection fraction; LVIDd = left ventricular internal diameter in diastole; LVOT = left ventricular outflow tract; PA = pulmonary artery; RV = right ventricle; SoV = sinus of Valsalva; SD = standard deviation; STJ = sinotubular junction

cardiovascular disease, TTE is probably of limited value as a screening investigation.

Study limitations

The main limitations of this study are the small sample size and the single centre setting. The exclusion of acute cases and those not recorded in the NVR in years where the case ascertainment rates were low, as well as changes in practice over the study period with an increasing caseload, may have limited the study size. It is part of usual care at our institution that all individuals referred for consideration of EVAR undergo TTE and CPET prior to EVAR. That no form of preoperative investigation was undertaken in 50 cases might have introduced ascertainment bias. However, patients who did or did not undergo preoperative investigations were similar at baseline.

This study assessed outcomes and the association with preoperative investigation following EVAR; it did not include cases in which EVAR was considered but not undertaken. It remains possible that some patients were excluded on the basis of these investigations. Furthermore, this study did not involve a direct comparison of risk stratification of CPET and TTE. In our cohort, 120 patients underwent CPET and 123 TTE but only 94 underwent both. This study population therefore comprises a heterogeneous mix of patients although all individuals included in the study underwent preoperative assessment (clinical assessment, blood tests, chest radiography and electrocardiography).

Conclusions

Our study reiterates that CPET might be an important tool for patient stratification prior to elective EVAR but did not demonstrate any value in preoperative TTE. CPET variables provide the most objective assessment of a patient's cardiovascular fitness, predict survival, help to exclude patients who are at a high perioperative risk and may help guide anaesthetic technique.

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