BJA

British Journal of Anaesthesia, 119 (1): 87–94 (2017)

doi: 10.1093/bja/aex164
Clinical Practice

Elevated preoperative heart rate is associated with cardiopulmonary and autonomic impairment in high-risk surgical patients

T. E. F. Abbott[1], G. Minto[2], A. M. Lee[1], R. M. Pearse[1] and G. L. Ackland[1,*], for the POM-HR, POMO-O and OPTIMISE study groups

[1]William Harvey Research Institute, Barts and the London School of Medicine and Dentistry, Queen Mary University of London, London EC1M 6BQ, UK and [2]Peninsula Medical School, Plymouth, UK

*Corresponding author. E-mail: g.ackland@qmul.ac.uk

Abstract

Background. Elevated preoperative heart rate (HR) is associated with perioperative myocardial injury and death. In apparently healthy individuals, high resting HR is associated with development of cardiac failure. Given that patients with overt cardiac failure have poor perioperative outcomes, we hypothesized that subclinical cardiac failure, identified by cardiopulmonary exercise testing, was associated with elevated preoperative HR > 87 beats min^{-1} (HR > 87).

Methods. This was a secondary analysis of an observational cohort study of surgical patients aged ≥45 yr. The exposure of interest was HR > 87, recorded at rest before preoperative cardiopulmonary exercise testing. The predefined outcome measures were the following established predictors of mortality in patients with overt cardiac failure in the general population: ventilatory equivalent for carbon dioxide ($\dot{V}_E/\dot{V}co_2$) ratio ≥34, heart rate recovery ≤6 and peak oxygen uptake ($\dot{V}o_2$) ≤14 ml kg^{-1} min^{-1}. We used logistic regression analysis to test for association between HR > 87 and markers of cardiac failure. We also examined the relationship between HR > 87 and preoperative left ventricular stroke volume in a separate cohort of patients.

Results. HR > 87 was present in 399/1250 (32%) patients, of whom 438/1250 (35%) had $\dot{V}_E/\dot{V}co_2$ ratio ≥34, 200/1250 (16%) had heart rate recovery ≤6, and 396/1250 (32%) had peak $\dot{V}o_2$ ≤14 ml kg^{-1} min^{-1}. HR > 87 was independently associated with peak $\dot{V}o_2$ ≤14 ml kg^{-1} min^{-1} {odds ratio (OR) 1.69 [1.12–3.55]; P=0.01} and heart rate recovery ≤6 (OR 2.02 [1.30–3.14]; P<0.01). However, HR > 87 was not associated with $\dot{V}_E/\dot{V}co_2$ ratio ≥34 (OR 1.31 [0.92–1.87]; P=0.14). In a separate cohort, HR > 87 (33/181; 18.5%) was associated with impaired preoperative stroke volume (OR 3.21 [1.26–8.20]; P=0.01).

Conclusions. Elevated preoperative heart rate is associated with impaired cardiopulmonary performance consistent with clinically unsuspected, subclinical cardiac failure.

Clinical trial registration. ISRCTN88456378.

Key words: cardiac failure; heart rate; surgery

More than 1.5 million major surgical procedures are carried out in the UK each year, and one in six patients will experience a complication after surgery.[1 2] One in 10 patients will sustain myocardial injury after non-cardiac surgery, which is strongly

Editorial decision: April 6, 2017; **Accepted:** May 2, 2017

Editor's key points

- An elevated resting heart rate can indicate some degree of cardiac or autonomic dysfunction in the general population.
- This study identified a relationship between elevated preoperative heart rate and impaired cardiopulmonary performance in the perioperative setting.
- A resting heart rate >87 beats min^{-1} in a patient booked for surgery should stimulate further cardiopulmonary evaluation.

associated with mortality.[3] However, the presence of coronary artery disease is a poor predictor of morbidity and mortality in these patients.[4] In contrast, elevated preoperative resting heart rate (HR >87 beats min^{-1}) is independently associated with myocardial injury and mortality.[5] Although tachycardia may be attributable to acute pathophysiology (e.g. sepsis, systemic inflammation), mechanisms to explain this association remain unclear.[6] Tachycardia may promote myocardial injury through oxygen supply–demand imbalance.[7] However, as treatment with β-blockers or clonidine fails to reduce the incidence of myocardial injury, other pathophysiological mechanisms are likely to be involved.[8 9]

Patients with a confirmed diagnosis of cardiac failure syndrome are at very high risk of perioperative mortality.[10 11] Cardiopulmonary exercise testing (CPET) can identify cardiopulmonary dysfuntion, autonomic dysfunction, or both, and has been used for prognostication in patients with confirmed cardiac failure and for risk assessment before surgery.[12–14] In the general population, elevated resting HR is an independent risk factor for the development of heart failure.[4 15 16] Therefore, elevated preoperative HR may indicate underlying subclinical cardiac impairment; thus generating several plausible, and potentially novel, pathophysiological mechanisms that may contribute to perioperative myocardial injury, morbidity, and mortality.[5 7 17 18]

We hypothesized that elevated preoperative resting HR (>87 beats min^{-1})[5] was associated with impaired cardiovascular function, autonomic function, or both, consistent with subclinical cardiac failure. We tested this hypothesis by evaluating cardiovascular and autonomic factors derived from preoperative cardiopulmonary exercise testing that are known to be associated with clinical outcome in patients with heart failure.[3]

Methods

Study design

This was a secondary analysis of data obtained prospectively from the Perioperative Morbidity – Heart Rate (POM-HR) study, a multicentre observational cohort study of high-risk patients undergoing non-cardiac surgery. The methods have been published previously.[19] The study was approved by Research Ethics Committee (Camden and Islington; MREC:12/LO/0453) and registered (ISRCTN88456378).

Patient population

Patients were eligible for inclusion if they were aged ≥45 yr, were undergoing major surgery predicted to last for >2 h, and were referred for CPET as part of their routine preoperative assessment. Patients provided written informed consent before taking part in the study (before exercise testing). The exclusion criteria were refusal of consent or contraindications to CPET. These criteria are very similar to the eligibility criteria for the VISION study.[3]

Data collection

In POM-HR before surgery, patient age, gender, operative time, established measures of preoperative risk (including diabetes mellitus, cardiac, and cerebrovascular disease) and haemoglobin were recorded. All participants underwent CPET.

Outcome measures

The primary outcomes measures were the following three CPET-derived variables that are established and independent predictors of mortality in patients with cardiac failure: ventilatory equivalent for carbon dioxide ($\dot{V}_E/\dot{V}co_2$) ≥34; HR recovery ≤6; and peak oxygen consumption ($\dot{V}o_2$) ≤14 ml kg^{-1} min^{-1}.[12] Secondary outcome measures were other CPET-derived cardiopulmonary and autonomic variables known to be associated with postoperative clinical outcomes or cardiovascular morbidity in the general population, as follows: preoperative pulse pressure, oxygen consumption at the anaerobic threshold, peak oxygen pulse, peak HR, and HR reserve. Full details for the original papers detailing the prognostic value of these variables are provided in Supplementary Table S1.

Cardiopulmonary exercise testing

Cardiopulmonary exercise testing was carried out at each participating hospital in designated CPET laboratories. Before CPET, participants were instructed to continue their normal medications up to, and including, the day of the test. The CPET was conducted using a standard incremental ramp protocol to maximal exercise tolerance using an electromagnetically braked cycle ergometer. Equipment was calibrated before each test, including calibration of the gas analyser using standard reference gases. During CPET, continuous 12-lead electrocardiography (HR), intermittent sphygmomanometry (arterial blood pressure), and breath-by-breath measurement of gas exchange were performed. Before each test, arterial blood pressure and heart rate were measured in the sitting position after at least 30 s rest. Participants were instructed to continue cycling as the ramp (in watts) increased, until they were unable to continue because of symptom-limited fatigue. After reaching peak exercise tolerance, continued physiological measurements were recorded during the period of recovery from exercise.

The anaerobic threshold was determined by two independent assessors according to published guidelines using the modified V-slope method and confirmed by ventilatory equivalents for oxygen ($\dot{V}_E/\dot{V}o_2$) and carbon dioxide ($\dot{V}_E/\dot{V}co_2$). Oxygen consumption (in millilitres per kilogram per minute) was measured at the anaerobic threshold and at peak exertion. The $\dot{V}_E/\dot{V}co_2$ was measured at the anaerobic threshold and presented as a ratio. Resting HR was defined as the HR measured before each test after 30 s in the sitting position. Peak HR, which reflects sympathetic activation, was defined as maximal HR achieved during exercise. Heart rate recovery, a measure of parasympathetic activity, was calculated as the difference between peak HR and HR 1 min after the end of peak exercise, thus representing the change in HR during the 1 min after exercise (in beats per minute). Peak oxygen pulse (in millilitres per beat), a surrogate marker for cardiac stroke volume,[20] was calculated as peak oxygen consumption (in millilitres per minute) divided by peak HR (in beats per minute).