Preoperative fast heart rate: a harbinger of perioperative adverse cardiac events

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Recent studies have shown that approximately 10% of patients with, or at risk for, cardiovascular disease suffer a major adverse cardiac event (MACE) within 30 days of surgery.1–3 In such patients non-invasive and invasive preoperative investigations may reveal conditions needing preoperative treatment. In unselected populations such complications also occur, albeit less frequently, but investigations of the cardiovascular system are seldom, if ever, as extensive as in high risk patients. Could preoperative tachycardia be a valuable signal of increased risk?

For many years it has been known that tachycardia is an important causative factor for myocardial ischaemia and myocardial injury, including myocardial infarction, as a result of an imbalance between increased myocardial oxygen demand and reduced oxygen supply caused by the reduced duration of diastole.4–6 Tachycardia has been identified as a major cause of perioperative myocardial infarction for several decades both in daily life and the perioperative period.5–9 In prolonged non-cardiac surgery tachycardia and hypertension are independent predictors of adverse outcome.8 Similarly, in intensive care patients at risk for cardiac complications according to the revised cardiac risk index,10 prolonged periods of tachycardia are associated with adverse cardiac outcome including cardiac death.11 Myocardial damage, including myocardial infarction may result from the imbalance between oxygen demand and oxygen supply and also from the adverse effect of a fast heart rate on unstable plaques of atheroma resulting in their disruption and, possibly, coronary occlusion.12 Epidemiological data from the general population consistently demonstrate that a fast resting heart rate is associated with cardiovascular risk and mortality.5,13 The fast heart rate may be causative in itself, or simply be a marker of an underlying disease such as heart failure.14–16

Continuous monitoring of the ECG in the perioperative period has shown that silent myocardial ischaemia, often caused by tachycardia, is very frequent and is associated with adverse outcome in a variable proportion of patients.15–16 As only a weak correlation exists between silent ischaemia and major outcomes in some groups of patients,16 there has been increasing interest in monitoring troponins as markers of myocardial injury.17 The adverse prognostic role of elevated troponins was confirmed in a systematic review and meta-analysis of 14 studies with 3381 patients.18

The VISION study (Vascular Events in Non-cardiac Surgery Patients Cohort Evaluation) enrolled adult patients presenting for surgery (eligible patients were aged more than 45 yr and required at least an overnight hospital admission after non-cardiac surgery). In this prospective study serial ECGs and serial troponin T measurements were obtained before and for three days after surgery.19 The aim of the study was to document the risk of cardiovascular adverse events, represented by elevated troponin T, myocardial infarction according to its third universal definition,20 and mortality, in an essentially unselected population, as opposed to studies in high risk patients.1–3 Adverse outcome such as myocardial injury in non-cardiac surgery (MINS) occurred in 7.9% of patients (1197/15 087), myocardial infarction in 2.8% of patients (454/16 007) and 2% of patients died (315/16 037).

With extensive data on troponin T, the study showed that even modest increases in troponin T were associated with increased mortality within 30 days of surgery.19 This led to the development of the concept of MINS defined as prognostically
relevant myocardial injury as a result of ischaemia that occurs during or within 30 days after non-cardiac surgery. The importance of the study is that perioperative myocardial injury is primarily asymptomatic, such that in the absence of routine surveillance of troponin levels, four out of five patients are not identified.

The availability of data on outcome in a large number of patients in VISION, made it tempting to examine the possibility of a correlation between preoperative heart rate and adverse cardiovascular outcome including MINS. In their paper Abbott and colleagues report the result of a pre-defined secondary analysis of the effect of preoperative heart rate on MINS, myocardial infarction and mortality. MINS was defined as TnT≥0.03 ng ml⁻¹, adjudicated as because of an ischaemic pathology within 30 days after surgery after exclusion of non-ischaemic causes such as sepsis or pulmonary embolism.

Preoperative heart rate was defined as the last heart rate recorded before induction of anaesthesia. The authors found a clear association between fast preoperative heart rate and adverse outcome. Data analysis was carried out in two ways. Firstly data were classified in deciles of heart rate of approximately the same sizes. Multivariable logistic regression models were used to determine relationships between preoperative heart rate and MINS, myocardial infarction and death within 30 days of surgery. Secondly separate models were used to test the relationship between these outcomes and pre-defined binary heart rate thresholds, namely more than 70 bpm as in the general population, and more than 104 bpm as in surgical populations, these thresholds being taken from the relevant literature.

The major findings of the study are that heart rates in the highest decile (>96 bpm) were significantly associated with MINS, myocardial infarction (MI) and mortality. A similar significant association was found for mortality for the ninth decile (88–96 bpm), while there was no association with MINS and MI. With the slower heart rate in the eight decile (83–87 bpm) the only significant adverse outcome was MINS. At slower heart rates, in the fifth decile (72–74 bpm), there was a statistically significant reduction in MINS but not MI or mortality. Following this general trend towards lesser adverse outcome with slower rates, heart rates in the lowest decile (<60 bpm) were associated with a lower incidence of mortality than in other deciles but not reduced MINS or MI.

As cardiovascular medication can influence both heart rate and outcome, the data were further analysed using data from patients on beta-blockers or rate controlling calcium channel blockers. While exclusion of patients who had received a rate controlling drug within 24 h of surgery did not alter the risk of adverse outcomes in the highest decile (>96 bpm), the beneficial effect of slower heart rate (<60 bpm) was no longer observed. Sensitivity analyses excluding patients with atrial fibrillation or emergency surgery had little effect on the relationship between heart rate and adverse outcome.

The predefined binary thresholds were also associated with myocardial injury, but less strongly than the highest heart rate decile. Heart rates >104 bpm were associated with MINS, MI and mortality, while heart rates >70 bpm were associated with MINS and mortality, but not MI.

The results of the multivariable fractional polynomial regression analysis showed a linear association between heart rate and the probability of myocardial injury, rather than a threshold above which postoperative myocardial injury increases.

As fast heart rate is clearly a harbinger of adverse cardiovascular outcome, the first question is why does it happen in preoperative patients? Regulation of heart rate results from the balance between sympathetic and parasympathetic activity. The effect of this balance is mediated by changes in the activity of the If channels (funny channels) that control the movement of Na⁺ and K⁺ ions across the cell membrane of the pacemaker cells, altering their rate of spontaneous depolarization. Anxiety, psychological stress, volume depletion and dehydration can all result in an increase in heart rate. All of these may occur during the preoperative period, especially in the era of day of surgery admission. Tachycardia may be indicative of heart failure, valvular heart disease, even of sympathetic overactivity in patients with phaeochromocytoma. It could also be a marker of an underlying infectious condition. It is therefore important to take notice of a fast heart rate and consider possible treatable causes.

If no obvious cause can be found, the second question is what can we do? Should we immediately initiate treatment with a rate-slowing agent, as maintaining a slow heart rate has been shown to reduce the duration of myocardial ischaemia. Many trials have shown that initiating treatment with beta-blockers in the immediate preoperative period reduces heart rate and reduces the risk of perioperative myocardial infarction. However, POISE, the largest RCT of perioperative beta-blockade, demonstrated that beta-blockade increased all-cause mortality and strokes. As a result, recent guidelines limit the introduction of beta-blockers shortly before non-cardiac surgery to patients at very high risk and insist on titration to effect and consider that starting the day of surgery is harmful. Do these guidelines preclude the goal-directed introduction of beta-blockers in the immediate perioperative period? No, POISE investigated the effect of a beta-blocker given for 30 days, starting the day of surgery, to patients at risk for or with coronary artery disease; it did not examine the effect of goal-directed administration of a beta-blocker to control fast heart rate immediately before surgery and for a limited period. Thus the 2014 guidelines do not apply to this situation. Furthermore, it may be detrimental to deprive patients of an important and effective treatment by throwing out the baby with the bath water. An alpha-adrenergic receptor antagonist such as clonidine could be introduced to reduce heart rate. Indeed low-dose clonidine was investigated in more than 10,000 patients in POISE 2. Clonidine reduced heart rate and resulted in significant bradycardia in 12% of patients, vs 8.1% in the placebo group. However, the slower heart rate was not associated with a reduction in adverse cardiac outcomes in patients with, or at risk for, cardiovascular disease.

Calcium channel blockers such as verapamil and diltiazem are effective in slowing heart rate. However they have relatively potent myocardial depressant effects. This may be undesirable, especially where volatile anaesthetics are going to be used as the main anaesthetic agents. Moreover there is little data on their prophylactic use shortly before surgery and available studies show them to be ineffective in preventing myocardial ischaemia and adverse outcome.

Ivabradine is a blocker of the If channels that regulate the spontaneous depolarization of pacemaker cells. It benefits patients with cardiac failure and those with coronary artery disease and reduced systolic ventricular function. There have been small studies of the efficacy of ivabradine given perioperatively but the data is, as yet, insufficient for firm conclusions to be drawn because there were too few outcomes. As there is only limited scope for slowing heart rate safely, and apart from beta-blockers little evidence for their efficacy, we should consider, in light of the paper, by Abbott and colleagues that preoperatively elevated heart rate is important as an indicator of increased perioperative risk.
looking systematically, and not only in high-risk patients, for myocardial injury by serial troponin assay and repeated ECGs in patients with tachycardia, bearing in mind that, most of the time, myocardial ischaemia and myocardial infarction are silent in the perioperative period and remain undetected unless positive steps are taken for their identification. There is a logic to this approach as myocardial injury can be treated by reinforcing conventional treatment, essentially aspirin, platelet antagonists, statins, goal-directed introduction of beta-blockers, and where appropriate referring the patient to an interventional cardiologist for emergency coronary revascularization. Observational evidence suggests that cardiovascular drugs used for secondary prevention are also beneficial in patients who develop myocardial injury postoperatively. Patients on statins experience beneficial evidence suggests that cardiovascular drugs used for secondary prevention are also beneficial in patients who develop myocardial injury postoperatively. Patients on statins experience secondary prevention are also beneficial in patients who develop myocardial injury postoperatively. Patients on statins experience.

A fast preoperative heart rate may further indicate the need for more extensive postoperative monitoring including ‘track and trigger’ and the use of remote automated systems for continuous non-invasive monitoring of oxygen saturation, bp and ST-segment analysis. This may seem science fiction but it is already actively developed and utilized. In these patients prevention of hypotension and hypoxaemia, often severe and unrecognized on the ward23 are important as both are closely associated or causative of myocardial infarction, but may not be detected in the absence of adequate monitoring.

Fast preoperative heart rate: beware!

References

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Valid consent – A pathway to improved care

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By improving our practice in how we obtain a patient’s consent for surgery and anaesthesia we may discover a pathway to better perioperative care and end-of-life decision-making.

Obtaining a patient’s informed consent to undergo surgery may involve the consideration of multiple aspects of their care: the relative merits of an operative vs non-operative approach, the risks and benefits of regional or general anaesthesia, the impact of their co-morbidities on immediate and long-term perioperative risks, the psychological and social factors that may play a part in their recovery, and their likely outcome not just in terms of survival but also their quality of life. The role of the anaesthetist in helping the patient and surgeon towards truly informed consent is variable. Increasingly patients are brought in to hospital on the day of their surgery and there may be scant documentation regarding the subtleties of the conversations that have taken place concerning the decision to proceed to surgery. We have learnt from the Supreme Court judgement of Montgomery vs Lanarkshire Health Board¹ that we need to be more fastidious in our explanations and in the documentation of relative risks.

Our role as anaesthetists varies. Sometimes it will be appropriate for us to limit our discussions specifically to the anaesthetic aspects of the patient’s care. Sometimes it will be appropriate for us to lead or coordinate more comprehensive discussions ourselves, or to call for a multidisciplinary approach, but as the General Medical Council makes clear in its guidance document² it will always be part of our duty of care to patients to ensure that perioperative considerations include every aspect of care, not just survival, and that those considerations are properly documented. Towards the end of life these discussions can be particularly delicate, and for those patients especially we must ensure that the drive to shortened hospital admission times does not remove the opportunity to establish a real rapport between the patient, their family and all the staff involved in their care. Well-intentioned targets for hospital discharge and survival may distort clinical judgement regarding what is really best for

¹ Montgomery vs Lanarkshire Health Board [2015] UKSC 12
² General Medical Council: Good Medical Practice (2013)