Basic sciences review

Preoperative Cardiopulmonary Risk Assessment by Cardiopulmonary Exercise Testing

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ABSTRACT

Objective: *To review the strategies for identification and subsequent management of high-risk patients that have been proposed to reduce perioperative morbidity and mortality.*

Data sources: *Articles and published reviews of studies on evaluation and management of patients to reduce perioperative morbidity and mortality.*

Summary of review: *Many strategies have been devised to evaluate the perioperative risk in elderly patients, particularly those who are about to undergo major intra-abdominal surgery. An assessment of cardiac and pulmonary responses to exercise-induced stress (i.e. cardiopulmonary exercise testing) using a bicycle ergometer, metabolic cart (to measure oxygen uptake and carbon dioxide excretion) and a 12 lead electrocardiograph has been found to be a non-invasive test that is quick, cheap, easy to perform and requires no special preparation. It has advantages over any other preoperative test by defining operative risk and allowing logical triage according to objective risk assessment. It is able to objectively evaluate the extent of any cardiac failure (by assessing the anaerobic threshold), myocardial ischaemia, provides insight into stroke index, the presence of pulmonary artery hypertension and defines obstructive and restrictive lung disease and ventilation perfusion inequality better than conventional preoperative respiratory function tests*

Conclusions: *In patients who are about to undergo major intra-abdominal surgery, preoperative cardiopulmonary exercise testing is an excellent predictor of risk in the postoperative period. No other test is able to offer such a comprehensive preoperative risk evaluation for an operative patient.* **(Critical Care and Resuscitation 2000; 2: 198-208)**

Key Words: Cardiopulmonary exercise testing, preoperative evaluation, perioperative management, anaerobic threshold

Preoperative assessment has traditionally concentrated on the detection of myocardial ischaemia and, to a lesser extent, congestive cardiac failure (CCF). However, a comprehensive evaluation should assess objectively the patient risk for cardiac and/or respiratory complications in the perioperative period. It must also include recommendations for treatment based on this assessment; in other words the evaluation becomes a tool for triage of the patient to varying levels of postoperative care.

The increased perioperative risk in patients with unstable coronary syndromes, decompensated CCF, severe valvular heart disease and symptomatic arrhythmias is well described.[1-3] Cardiac failure as a cause of postoperative mortality was also highlighted by Goldman[2] and Buck *et al.*[4] In the 1960's, the pioneering

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work of Clowes and Del Guercio[5] and the subsequent work of Shoemaker,[6] showed that in surgical patients the inability to increase cardiac output postoperatively was associated with increased mortality.

We contend that the major determinant of perioperative mortality is the inability of the heart to increase its output in response to surgical stress. This entity could be termed perioperative cardiac failure (PCF) and may only be apparent postoperatively when oxygen demand is increased. It may occur independently of both CCF and myocardial ischaemia though all three may coexist. We contend that the attention in preoperative evaluation should be focussed on the detection of 'forward' cardiac failure. This is frequently occult in the elderly although it may manifest itself as a reduced exercise tolerance. Normally, elderly patients adjust their level of activity when oxygen demand exceeds supply. The postoperative patient does not have this option.

In two studies published involving over 700 elderly patients we show that mortality risk is associated with PCF.[7,8] Myocardial ischaemia associated with PCF substantially increases the risk. However, in our studies only 35% of patients with PCF had demonstrable myocardial ischaemia and myocardial ischaemia not associated with PCF carried a low postoperative mortality. Myocardial infarction has not caused the death of any patient in our two published series. We have also documented that complications of surgery accounted for only 0.9% mortality overall. In other words, we found that postoperative mortality was a function of preoperative cardiopulmonary failure rather than myocardial ischaemia.

There has been a tendency in studies of perioperative cardiac events over the last decade to assume that a patient has coronary artery disease from the presence of risk factors such as age, diabetes mellitus or smoking. [9,10] Thus, the entry criteria in such studies may well include patients who have risk factors for coronary artery disease but in whom coronary disease has not been demonstrated. Determination of perioperative risk according to clinical predictors of cardiopulmonary disease may well exclude those patients who are asymptomatic and have no 'risk factors'. Other studies have grouped all types of surgery together as if the risk is the same regardless of the type of surgery.[9] Minor surgery does not carry the same risk as major surgery.[4] All such studies may be misleading.

CLINICAL RISK ASSESSMENT

Age as a single variable for risk assessment is not predictive of the cardiovascular reserve of an individual patient.[11] Although the anaerobic threshold (AT) diminishes with age, our study of 187 elderly patients showed no significant difference in average AT between age groups from 60 years of age to 85 years of age.[8] This was confirmed in a subsequent series involving 548 patients.[7]

More than 15 years ago it was found that the New York Heart Association (NYHA) classification of cardiac failure did not correlate well with aerobic capacity. A study by Lipkin *et al*,[12] showed a significant difference in maximum oxygen uptake between NYHA functional classes, but there was also considerable overlap. Itoh *et al*,[13] obtained similar results in 1990. Such a classification can obviously not be used for risk assessment of an individual patient. The 1994 revisions to the NYHA classification include a concept of functional capacity and objective assessment.[14] Objective assessment is based on measurements such as electrocardiograms, ECG stress tests, echocardiography and radiological imaging. These tests are discussed later but none are suitable as preoperative screening tests.

The American College of Cardiology/American Heart Association (ACC/AHA) guidelines

These guidelines are published as a framework for evaluation of cardiac risk for noncardiac surgery.[15] The low risk group is comprised of patients less than 60 years of age with no history of cardiopulmonary disease or non-specific ECG changes and may proceed to surgery with little evaluation. The high risk group includes those patients with acute coronary syndromes, decompensated cardiac failure, recent myocardial infarction and supraventricular arrhythmias. They are usually easily identified and will obviously need further assessment and management.

It is in the intermediate group where clinical predictors of cardiopulmonary disease are less reliable, or absent, that controversy exists as to appropriate preoperative evaluation. This group includes all patients over 60 years of age. A history and clinical examination will often not detect the presence of significant cardiopulmonary pathology. Investigation in this group has focussed on the detection of myocardial ischaemia and congestive cardiac failure. Myocardial ischaemia is not the only cause of CCF and it is unwise to assume that it is the only cause of PCF. For patients with intermediate clinical predictors of risk, including stable angina pectoris, previous myocardial infarction, compensated or prior CCF and diabetes mellitus, determination of functional capacity is recommended.

The ACC/AHA guidelines introduce the concept of 'surgery-specific risk'. Risk stratification is based on the degree of haemodynamic stress associated with specific procedures. Postoperative haemodynamic stress

is recognised as consequent on an increase in oxygen consumption. High-risk surgery includes major abdominal and thoracic procedures, particularly in the elderly and, specifically, major vascular surgery (i.e. prolonged surgery associated with major fluid shifts and/or blood loss). Intermediate risk surgery includes orthopaedic and prostatic surgery and low risk surgery includes peripheral procedures and laparoscopic procedures.

Whilst one may not agree completely with this classification, the recognition of surgery-specific risk represents a major advance in perioperative risk evaluation. We prefer to define surgery specific risk more objectively in terms of expected postoperative oxygen consumption values. The surgery is low risk when the expected postoperative $\dot{V}O_2$ is less than 120 mL/min/m^2; intermediate risk is equivalent to a $\dot{V}O_2$ 120 - 150 mL/min/m^2. The average postoperative $\dot{V}O_2$ following major intra-abdominal surgery including vascular surgery often exceeds 150 mL/m^2 and this equates to high risk surgery.[16] This represents about a 40% rise over basal oxygen consumption.

The same guidelines discuss functional capacity as an important determinant of perioperative risk. Functional capacity, they suggest, may be estimated in terms of metabolic equivalents (METS). One metabolic equivalent (MET) is defined as the average resting $\dot{V}O_2$ for a 70 kg, 40 year old male and is equal to 3.5 mL/min/kg. These guidelines suggest that patients unable to meet a 4-MET demand are at increased perioperative and long-term risk. The guidelines conclude, "non-cardiac surgery is safe for patients with a moderate or excellent functional capacity (4 METS or greater)".

The whole concept of an increase in METS is based on the need to increase cardiac output to meet exercise-induced stress. Without cardiopulmonary exercise testing, an estimation of METS is just that - an estimation. What is needed is an accurate and objective measurement of 'METS', or to be more precise, functional performance. Cardiopulmonary exercise (CPX) testing is designed to quantify functional capacity and is the best and most accurate way to measure cardiac performance. It is acknowledged by Kleber[17] as the 'gold standard' for evaluation of cardiac failure. Furthermore, the ACC/AHA guidelines for exercise testing states that data from CPX testing have proved to be reliable and important in evaluation of patients with heart failure.[18] Our studies suggest that patients unable to meet a 3.0 MET demand are at an increased risk.[11] This equates to a $\dot{V}O_2$ of 10.5 mL/min /kg.

The entire thrust of this approach is based on cardiac performance under conditions of stress, not rest, and not solely on the presence or absence of myocardial ischaemia. Preoperative risk evaluation performed at rest or based solely on evidence of ischaemia, not function, are therefore flawed as a screening test for surgical risk. In our studies, myocardial ischaemia predicted only 40 - 50% of patients at risk.[7] The ACC/AHA guidelines for exercise testing[18] state "One of the strongest and most consistent prognostic markers identified in exercise testing is maximum exercise capacity, which is at least partly influenced by the extent of resting left ventricular dysfunction and the amount of further left ventricular dysfunction induced by exercise".

The ideal screening test should provide an accurate assessment of myocardial function, detect myocardial ischaemia, be non-invasive and easily performed in elderly patients, reproducible and cost effective. What are the tests in common use for evaluation of perioperative risk that meet these criteria?

PREOPERATIVE SCREENING TESTS

Exercise ECG

The exercise ECG is widely used for the investigation of suspected coronary artery disease. The sensitivity of the test for detecting multivessel disease is reportedly 81%. In addition, it can provide an indirect assessment of functional capacity. Patients who achieve an estimated 7 METS or a heart rate of > 130 beats/min without ischaemia are identified as low risk.[19] In contrast, patients who are unable to increase their pulse rate to greater than 100 beats/min are deemed to be at high risk.[20] In two studies of elective surgical patients, the negative predictive value was reported at 93%.[21,22] In such studies, using a treadmill, many patients (30-70% of elderly vascular patients) have non-diagnostic tests due to inability to exercise adequately. Because of this, and its poor discrimination of functional capacity, the role of the exercise ECG as a screening test for elderly surgical patients is limited.

Radionuclide ventriculography

Radionuclide ventriculography (RNVG) determines ejection fraction, which is then used as an indicator of ventricular function. It is now accepted that ejection fraction correlates poorly with exercise capacity and peak oxygen uptake. Froelicher showed a poor correlation between ejection fraction and maximal oxygen uptake in patients with coronary artery disease not limited by angina.[23] In a study by Dunselman *et al*,[24] of NYHA class II and III patients with an ejection

fraction of less than 40%, only data derived from CPX testing were able to show differences between groups. The article states further that objective determination of exercise capacity is the only way to select patients for studies on heart failure. Given the inability of RNVG to provide an accurate, reproducible assessment of functional capacity its use as a perioperative screening test is not recommended.

Echocardiography

Transthoracic echocardiography (TTE) is non-invasive and easy to perform. It will not detect myocardial ischaemia but does provide assessment of both systolic and diastolic wall motion. A poor correlation exists between TTE findings and functional capacity; severe ventricular dysfunction on echo may also be associated with moderate to good functional capacity. A large cohort study of elective surgical patients performed by the perioperative ischaemia research group failed to support the use of TTE in the assessment of cardiac risk prior to non-cardiac surgery.[25] More recently, transoesophageal echo-cardiography (TOE) has become available but little data regarding the value of preoperative TOE for non-cardiac patients exists. In addition TOE is relatively invasive and requires the administration of a sedative.

Dobutamine stress echocardiography

Dobutamine stress echocardiography (DSE) is designed to detect myocardial ischaemia and has been advocated for patients who are unable to exercise adequately with treadmill testing. No objective measurement of functional capacity can be obtained, though wall motion abnormalities are detected. Its sensitivity and specificity for the detection of myocardial ischaemia are high and, as such, DSE is a useful adjunct in evaluating coronary artery disease. However, this test is expensive and the conduct and interpretation is operator dependent.[19] The use of DSE as a screening test for preoperative evaluation is not recommended.

Dipyridamole-thallium scintigraphy

In a study carried out by the perioperative ischaemia research group, dipyridamole-thallium scintigraphy has been shown not to be a valid screening test for detection of postoperative cardiac events, even in patients for vascular surgery.[26] Following these results, single photon emission computed tomography (SPECT) was considered. The combination of this with radionuclide angiography was used as a screening test in 457 patients scheduled for abdominal aortic reconstructive surgery.[27] The authors concluded that dipyridamole-thallium SPECT was not an accurate screening test of cardiac risk for abdominal aortic surgery.

Cardiopulmonary exercise testing

Cardiopulmonary exercise testing evaluates cardiac and pulmonary responses to exercise-induced stress. Cardiac response includes evaluation of ventricular function, circulatory function and evaluation of myocardial ischaemia. It is not possible to evaluate 'forward' cardiac failure clinically, other than by inference. As previously discussed, the NYHA functional classification of cardiac failure shows little or no correlation with objective measurements of aerobic capacity.[12,13,24] As an alternative objective measurement, Weber and Janicki have classified cardiac failure into four groups on the basis of CPX testing [28] (Table 1).

Table 1. Classification of cardiac failure by exercise testing

Functional class	*Cardiac failure definition*	$\dot{V}O_2$ *max (mL/min/kg)*	*AT (mL/min/kg)*
A	None	> 20.0	> 14.0
B	Mild	16.0 - 19.9	11.0 - 13.9
C	Moderate	10.0 - 15.9	8.0 - 10.9
D	Severe	< 10.0	< 8.0

We use a CPX test as the preoperative screening test to quantify the extent of cardiac failure, and to determine the presence or absence of myocardial ischaemia as well as its temporal relationship to changes in ventricular function. At the same time, we measure respiratory function in terms of obstructive or restrictive disease and any ventilation/perfusion mismatch. No other test, or combination of tests, is able to derive this information during conditions of exercise. It is non-invasive, simple to perform and costs less than the other tests described above.

The metabolic cart

CPX testing involves the use of a metabolic cart (Medgraphics Cardi-O_2, Medical Graphics Corp; St. Paul, MN), a bicycle ergometer (MGC Cardi-O_2 Cycle ergometer) and a 12 lead artefact free ECG (Mortara ELI-100XR). In physiological terms, the metabolic cart measures oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$) during continuously increasing exercise and simultaneously monitors a twelve lead ECG for detection of myocardial ischaemia and arrhythmias.

The metabolic cart measures $\dot{V}O_2$ and $\dot{V}CO_2$ on a breath-by-breath basis. The oxygen concentration in inspired and expired gas is measured by an oxygen analyser with a 90% response time of <90 msecs; the carbon dioxide concentrations are measured by an infra red analyser with a similar response time. The tidal volume is measured by a pressure differential pneumotachograph. The tidal volume is multiplied by the respiratory rate to give minute volume and thus $\dot{V}O_2$ and $\dot{V}CO_2$ may be calculated on a breath-by-breath basis and displayed as a 'per-minute' calculation.

The physiological basis of CPX testing

The relationship between oxygen consumption ($\dot{V}O_2$) and oxygen delivery ($\dot{D}O_2$) is described by the Fick equation where: Oxygen delivery ($\dot{D}O_2$) = cardiac output ($\dot{Q}$) x arterial oxygen content (CaO_2), and oxygen consumption ($\dot{V}O_2$) = cardiac output ($\dot{Q}$) x arterio-venous O_2 difference ($C(a-v)O_2$).

Cardiac output has virtually a linear relationship to oxygen consumption during the middle and later stages of exercise because the $C(a-v)O_2$ difference remains relatively constant in any individual over a large range of exercise.[29] In fact the $C(a-v)O_2$ is similar in cardiac failure as in health. A patient with heart failure has, therefore, the same mechanisms of exercise limitation as a normal individual, i.e. the limitation in aerobic capacity is due to an inability to increase cardiac output. Cardiac output is also a linear function of heart rate at higher levels of exercise because stroke index is maximally recruited early in exercise. [29]

In summary, limitation of oxygen consumption is a function of stroke index and pulse rate, under exercise conditions.

Conduct of the test

To conduct the test the patient is seated on the bicycle ergometer breathing via a pressure differential pneumotachograph and monitored by a 12 lead ECG. (Figure 1). The patient breathes at rest for about one minute and one is able to check the baseline values during this time. He is then asked to pedal the bicycle ergometer at about 55 - 65 revolutions per minute until the doctor asks him to stop.

The first three minutes of exercise are performed at 'zero' watts, i.e. there is no external load on the ergometer and the only work done is overcoming the mass of the legs being moved. With a zero watt bicycle the flywheel is assisted electrically to reduce the load further. This is termed 'unloaded' cycling. After three minutes the load is increased on the ergometer, in a continuous fashion, toward the maximum predicted work rate for that patient. This is termed a 'ramp protocol'. If the patient reaches maximum work rate or develops some form of distress or significant ECG change the test is ceased. This is termed a 'symptom-limited' test. The test is designed to last about six minutes after the unloaded cycling stage. This figure is calculated from the predicted maximum workload in watts that the subject should achieve based on age and height.

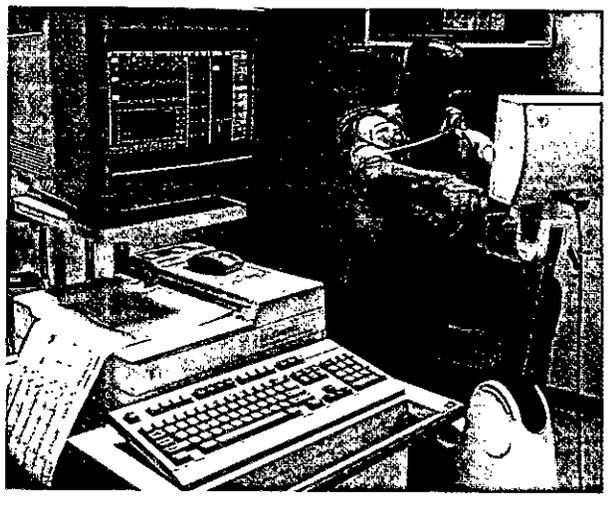

Figure 1. A patient seated on the bicycle ergometer breathing via the pneumotachograph. The gas sampling tube and the pressure lines from the pneumotachograph mouthpiece are attached to the metabolic cart. A 12-lead ECG is monitored throughout the test, the ECG obtained at rest is printed and can be seen at the left hand side. The screen displays the ECG and gas data in real time.

Interpretation of the test

The appearance of a completed test is shown in Figure 2. The data viewed in this fashion, as a temporal plot, gives very little useable information. Further analysis is necessary in order to derive the information required and this is achieved using mainly bivariate analysis.

Anaerobic threshold

The most important variables relative to cardiac function are the anaerobic threshold (AT) and the peak $\dot{V}O_2$. The latter is difficult to obtain in the elderly and is dependent on patient motivation. The AT is easier to detect and is independent of patient motivation. The AT is the point at which anaerobic metabolism is necessary to supplement the existing aerobic metabolism, i.e. oxygen supply to the exercising muscles is not adequate. This will result in release of lactate into the circulation and a metabolic acidosis will ensue.

It is possible, therefore, to determine the AT by constant measurement of serum lactate levels. The AT

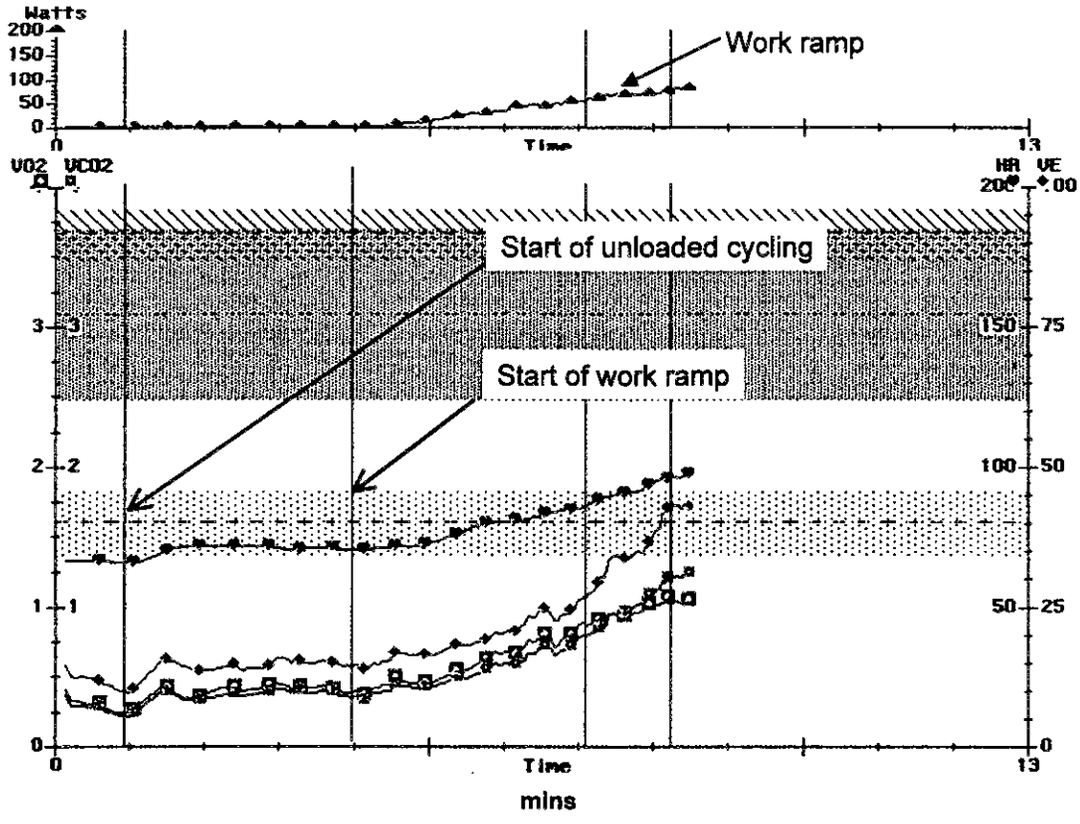

Figure 2. Data collection graph for completed cardiopulmonary exercise testing. The upper panel shows work rate in Watts. The lower panel shows (from top to bottom) heart rate (HR red), minute ventilation (VE, green), oxygen consumption (VO_2, pink) and carbon dioxide production (VCO_2, blue). All data plotted with respect to time. Note that the values for VCO_2 are initially lower than VO_2 but later approach then exceed those for VO_2 as work rate increases.

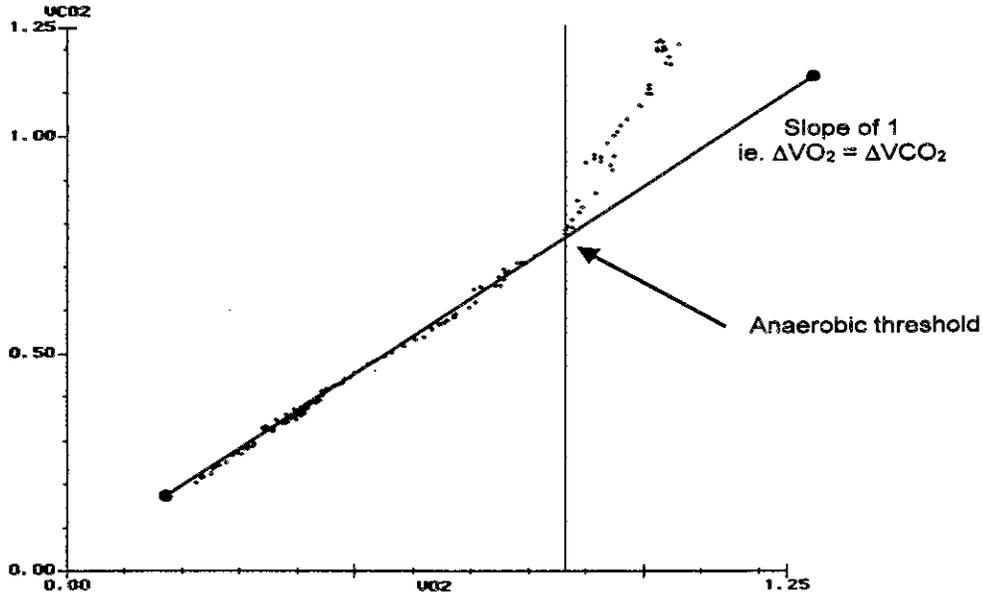

Figure 3. 'V-slope' method for determination of the anaerobic threshold (AT). The plot is bivariate, independent of time. Note the vertical line to the x-axis, the AT is expressed in terms of VO_2.

is represented by the $\dot{V}O_2$ at the point where the serum lactate starts to rise. Such a method is invasive and cumbersome and has been improved by a method described by Beaver *et al.*[30] At the AT, lactate is being released into the circulation. For each one mmol rise in plasma lactate the plasma bicarbonate will fall by one mmol. This causes a metabolic acidosis. In order to compensate for this there will be an increase in the elimination of carbon dioxide. Thus the rate of rise of $\dot{V}CO_2$ ($\Delta\dot{V}CO_2$) will increase - not because of excessive CO_2 production - but excess CO_2 elimination. If one now plots the $\dot{V}O_2$ on the X-axis and the $\dot{V}CO_2$ on the Y-axis of a graph the slope will be '1'. It will remain at 1 until the point where the rise in $\Delta\dot{V}CO_2$ is greater than the rise of $\Delta\dot{V}O_2$. At that point the slope will exceed '1'; this is the AT and this method of determination is termed the '$\dot{V}$-slope' method (Figure 3).

The AT may be confirmed by other means. At the AT there will be a change in the relationship of the expired minute volume ($\dot{V}E$) to the $\dot{V}O_2$ and $\dot{V}CO_2$. These relationships are termed the ventilatory equivalents for oxygen and carbon dioxide, the $\dot{V}E/\dot{V}O_2$ and $\dot{V}E/\dot{V}CO_2$. They are dimensionless numbers. The nadir for these equivalents occurs at the AT, after that point both of these numbers increase. Thus on a bivariate plot of ventilatory equivalents against $\dot{V}O_2$, the point where the $\dot{V}E/\dot{V}O_2$ rises from its nadir is the AT. These points may also be seen in Figure 4.

At the AT the minute ventilation increases in order to increase elimination of CO_2 to correct the metabolic acidosis. This is not associated with an increase in $\Delta\dot{V}O_2$; thus if the minute volume increases and $\Delta\dot{V}O_2$ remains the same then the end tidal oxygen concentration will rise. This may also be seen in Figure 4.

If the $\Delta\dot{V}CO_2$ rises and $\Delta\dot{V}O_2$ remains the same then the respiratory exchange ratio (RER) will increase. Some authorities use this point where the RER exceeds 1 as representative of the AT. This method will clearly overestimate the true AT as determined by 'V-slope'.

The 'V-slope' method is the most reliable and reproducible for determination of AT. The other three points are confirmatory only. Sometimes, however, where the AT is difficult to obtain the use of all four methods of identification may be useful.

Other physiological variables

Stroke index may also be evaluated by CPX testing. As stated before $C(a\text{-}v)O_2$ is maximised early in exercise and any further increase in oxygen consumption must be achieved by increase in cardiac output. The latter is a function of stroke index and pulse rate. Stroke index is also fully recruited early in exercise; thus cardiac output becomes linearly related to pulse rate. The increase in $\dot{V}O_2$ on a bicycle ergometer is linear at 10 mL/min/watt for aerobic exercise; it is thus possible to relate the increase in $\dot{V}O_2$ to the pulse rate. There is a defined normal relationship between these two parameters. If the relationship between pulse rate and $\dot{V}O_2$ is above normal this is interpreted as a reduction in stroke index. This signifies poor contractility. Beta adrenergic blocking agents may interfere with this interpretation by reducing the pulse rate response during exercise.

This concept may be carried further. If the stroke index remains constant the $HR/\dot{V}O_2$ relationship should be linear. If the stroke index decreases for any reason during exercise, this relationship is lost, and there will be an increase in the slope of $HR/\dot{V}O_2$. Myocardial ischaemia reduces ventricular compliance and stroke index; this will result in a rise in the $HR/\dot{V}O_2$ slope. Figure 5 shows a normal $HR/\dot{V}O_2$ response. Other causes of reduction in stroke index include exercise-induced asthma severe enough to increase pulmonary vascular resistance resulting in inadequate filling of the left heart.

As a further extension of this concept, primary pulmonary artery hypertension may also be suspected from CPX testing. This condition results in a poor left ventricular function resulting in a low AT. Left heart filling will be compromised due to flow resistance. This may worsen at moderate to high exercise rates producing a change in slope of the graph as described above. If the increase in pulmonary artery resistance has been brought about by long standing pulmonary disease this will result in a poor ventilatory equivalent for oxygen. Thus a patient with a triad of: 1) a low AT, 2) an elevated $HR/\dot{V}O_2$ slope, and 3) a significantly elevated $\dot{V}E/\dot{V}O_2$, almost certainly has pulmonary artery hypertension. This should be confirmed and further evaluated preoperatively by a pulmonary artery catheter.

As the relationship between $\dot{V}O_2$ and work rate is normally 10 mL/min/watt[29] then a graph of $\dot{V}O_2$ vs. watts should confirm such a slope if the patient has normal aerobic response to exercise. This is useful when interpreting difficult tests and is a useful confirmation that the aerobic capacity is normal.

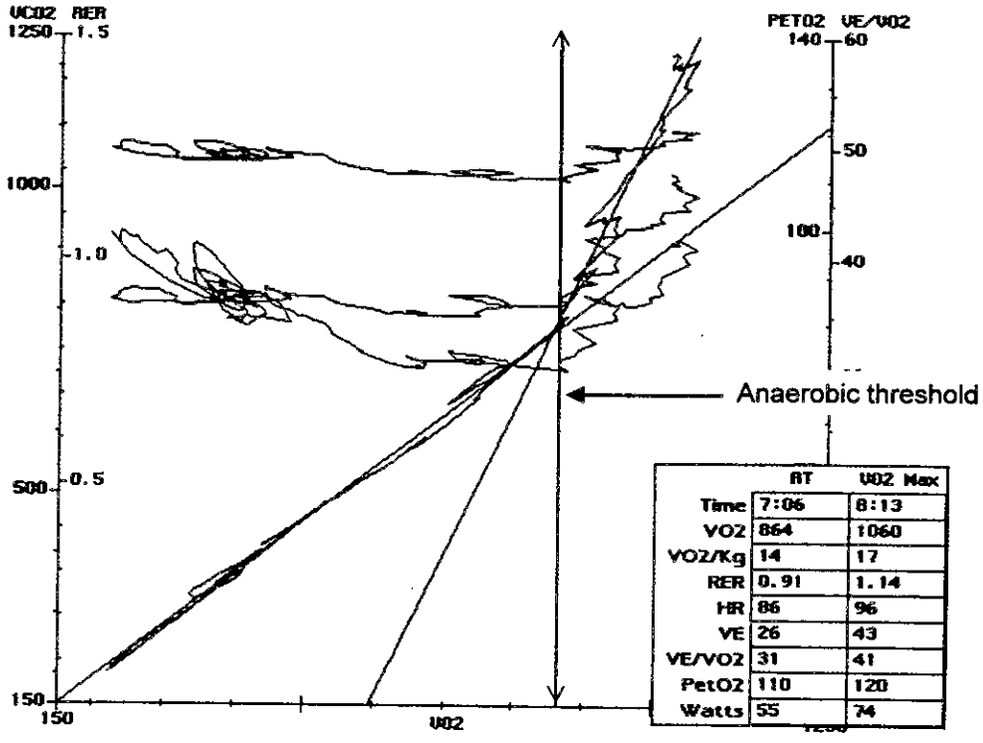

Figure 4. Bivariate graph showing ventilatory equivalents as confirmatory means for anaerobic threshold (AT) determination. The main panel shows plots for end tidal oxygen tension ($PETO_2$, red), ventilatory equivalent for carbon dioxide (VE/VCO_2, green) and the 'V-slope' (blue). Note the increase in ventilatory equivalents and end tidal oxygen which occur after the AT. The grey lines are 'best fit' lines for the 'V-slope', their intersection is the AT. These lines are generated by the software of the metabolic cart and show a slope of '1' for the VO_2/VCO_2 relationship below AT with a slope above '1' after the AT; important data at the AT and at maximum VO_2 are displayed in the panel at lower right.

Respiratory Evaluation

During exercise there is a continuing increase in minute volume. This may be achieved by an increase in tidal volume, respiratory rate or a mixture of both. The computer is able to display on a continuous basis, flow volume loops, capnography and oxygrams. This allows accurate diagnosis of restrictive and obstructive lung disease without the artefact normally associated with resting spirometry. The $\dot{V}E/\dot{V}O_2$ alluded to previously is able to quantify ventilation/perfusion imbalance. If this variable is abnormally high it implies that the work of breathing for any given $\dot{V}O_2$ will be elevated and that there is a $\dot{V}/Q$ mismatch.

Myocardial Ischaemia

The temporal relationship between the onset of myocardial ischaemia and the anaerobic threshold is important. We have shown that exercise ischaemia may be broadly grouped into two subsets.[31] The first subset comprises patients in whom myocardial ischaemia develops early in exercise and is associated with a low AT. The average AT in this subset was 10.4 mL/min/kg (SD 1.74) - Weber & Janicki Class C (Table 1). The second subset comprises patients in whom the ischaemia develops at or above the AT. The average AT in this subset was 13.9 mL/min/kg (SD 2.28) - Weber & Janicki Class B . In our studies the latter subset of myocardial ischaemia did not appear to be a major risk factor and was not evident postoperatively. Conceptually this would suggest that the increase in oxygen consumption postoperatively did not exceed a critical level, i.e. over the surgical AT, and thus myocardial ischaemia was not manifest.

Grading of perioperative risk

As discussed above, the ACC/AHA guidelines accept that there are two components to perioperative risk assessment. The first relates to the patient and the second to the proposed surgery. We have published a classification system that includes both parameters[31] (Table 2). An alphanumeric system is used to describe the extent of cardiac failure based on the Weber & Janicki classification (A-D); the surgery specific risk in terms of $\dot{V}O_2$ (1-3) and modifiers of risk including

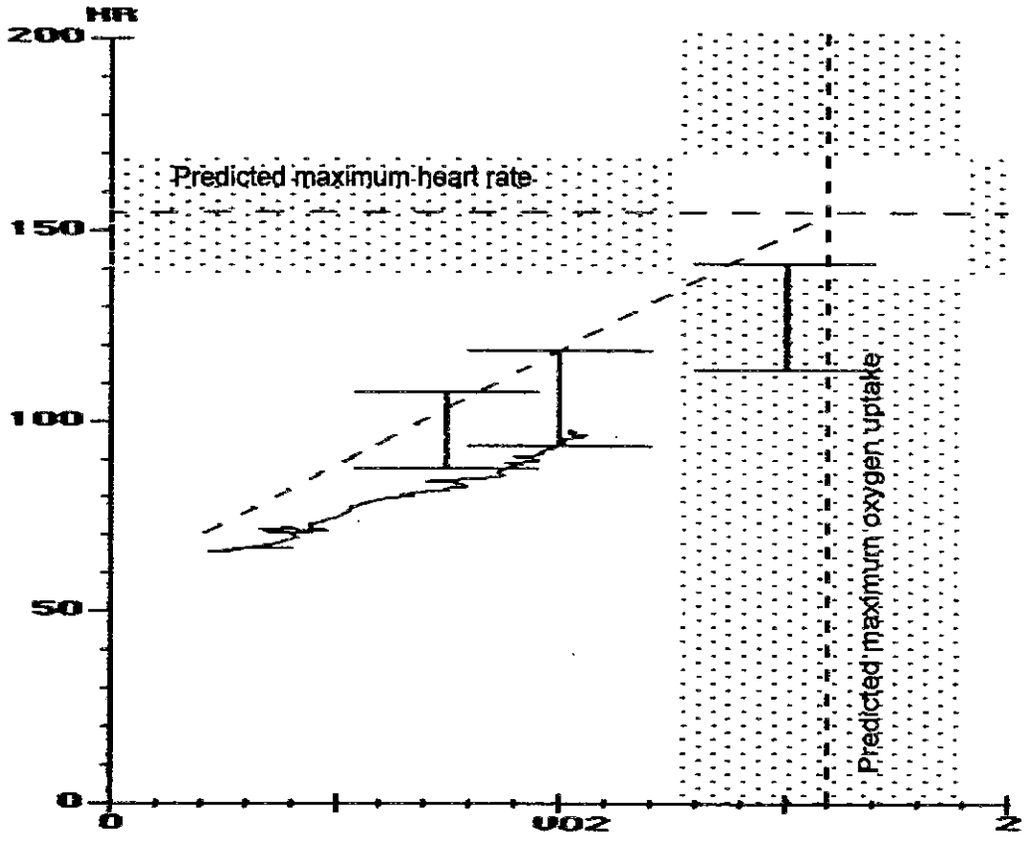

Figure 5. Heart rate/VO2 response graph. This relationship gives an indication of stroke index. The grey dashed 'line of identity' runs from a heart rate of 70 at resting VO2 to the predicted maxima for heart rate and VO2. The I-bars indicate submaximal predicted ranges for heart rate with respect to VO2 (mean ± 4SEM).

Table 2. Code and definitions for grading of perioperative risk

Degree of cardiac failure as $\dot{V}$ *O2 at AT mL/min/Kg*	*Expected postoperative* $\dot{V}$ *O2, mL/min/kg*	*Organ system dysfunction*
A: > 14	1: < 120	P: $\dot{V}$ E/ $\dot{V}$ O_2 > 35
B: 11.0 - 13.9	2: > 120 and < 150	p: $\dot{V}$ E/ $\dot{V}$ O_2 > 28 and < 35 A: Supraventricular tachycardia
		a: Other arrhythmia
C: 8.0 - 10.9	3: > 150	I: Ischaemia before AT I: Ischaemia after AT
D: < 8.0		

AT = Anaerobic threshold, P or p denotes degree of pulmonary dysfunction, , A or a denotes type of cardiac arrhythmia, I or I denotes onset of myocardial ischaemia

myocardial ischaemia (I) or arrhythmias (A) and pulmonary function (P). The latter data are taken from the CPX study, not the history or clinical risk factors. Thus a B3I classification describes a patient with mild cardiac failure (B), significant myocardial ischaemia (I) scheduled for major surgery (3)

Triage based on CPX testing

Patients who are classified as high surgery specific risk are admitted electively to the intensive care unit (ICU) preoperatively regardless of the AT. This group comprises surgery involving large fluid shifts or prolonged operating time (e.g. abdominal aneurysm surgery, Whipple's procedure, etc.) Patients who are classified as Weber & Janicki class C or D scheduled for major abdominal or thoracic surgery are also admitted to the ICU. They are invasively monitored and oxygen derived parameters are optimised. Renal function is measured by creatinine clearance and pulmonary function assessed by measurement of intrapulmonary shunt. This provides our anaesthetists and intensivists with comprehensive baseline physiological data, reduces anaesthetic induction time and

guarantees a bed postoperatively in ICU.

Patients for major abdominal or thoracic surgery who have significant ischaemia or pulmonary dysfunction are admitted to the high dependency unit postoperatively for ECG and respiratory monitoring, even if they are class A or B. All other patients are managed on the ward regardless of the surgery or history of clinical risk factors. We have demonstrated that patients who have neither cardiac failure nor myocardial ischaemia do not need to be in a special care area postoperatively.

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REFERENCES

1. Goldman L. Cardiac risks and complications of noncardiac surgery. Ann Intern Med 1983; 98:504-513.
2. Goldman L, Caldera DL, Nussbaum SR, et al. Multifactorial index of cardiac risk in noncardiac surgical procedures. N Engl J Med 1977;297:845-850.
3. Detsky AS, Abrams HB, Forbath N. Scott JG, Hilliard JR. Cardiac assessment for patients undergoing noncardiac surgery. A multifactorial clinical risk index. Arch Intern Med 1986;146:2131-2134.
4. Buck N. Delvin HB, Lunn JN. Council of enquirty into perioperative deaths. 1997; The Nuffield Trust.
5. Clowes GHA Jr, Del Guercio LRM. Circulatory response to trauma of surgical operations. Metabolism 1960; 9:67-81.
6. Shoemaker WC. Cardiorespiratory patterns of surviving and nonsurviving postoperative patients. Surg Gynecol Obstet 1972; 134:810-814.
7. Older P. Hall A, Hader R. Cardiopulmonary exercise testing as a screening test for perioperative management of major surgery in the elderly. Chest 1999; 116:355-362.
8. Older P. Smith R. Courtney P. Hone R. Preoperative evaluation of cardiac failure and ischemia in elderly patients by cardiopulmonary exercise testing. Chest 1993; 104:701-704.
9. Mangano DT, Layug EL, Wallace A, Tateo I. Effect of atenolol on mortality and cardiovascular morbidity after noncardiac surgery. Multicenter Study of Perioperative Ischemia Research Group. N Engl J Med 1996; 335:1713-1720.
10. Poldermans D, Boersma E, Bax JJ, et al. The effect of bisoprolol on perioperative mortality and myocardial infarction in high-risk patients undergoing vascular surgery. Dutch Echocardiographic Cardiac Risk Evaluation Applying Stress Echocardiography Study Group. N Engl J Med 1999; 341:1789-1794.
11. Wasserman K. Preoperative evaluation of cardiovascular reserve in the elderly. Chest 1993; 104:663-664.
12. Lipkin DP, Perrins J. Poole-Wilson PA. Respiratory gas exchange in the assessment of patients with impaired ventricular function. Br Heart J 1985;54:321-328.
13. Itoh H. Taniguchi K, Koike A, Doi M. Evaluation of severity of heart failure using ventilatory gas analysis. Circulation 1990; 81 :II31-II37.
14. AHA Medical/ Scientific Statement. 1994 revisions to Classification of Functional Capacity and Objecive Assessment of Patients with Diseases of the Heart. Circulation 1994; 90:644-655.
15. Eagle KA, Brundage BH, Chaitman BR, Ewy GA, Fleisher LA, Hertzer NR, et al. Guidelines for perioperative cardiovascular evaluation for noncardiac surgery. Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. Committee on Perioperative Cardiovascular Evaluation for Noncardiac Surgery. Circulation 1996; 93:1278-1317.
16. Older P. Smith R. Experience with the preoperative invasive measurement of haemodynamic, respiratory and renal function in 100 elderly patients scheduled for major abdominal surgery. Anaesth Intensive Care 1988; 16:389-395.
17. Kleber FX, Sabin GV, Winter UJ, Reindl I, Beil S. Wenzel M, et al. Angiotensin-converting enzyme inhibitors in preventing remodeling and development of heart failure after acute myocardial infarction: results of the German multicenter study of the effects of captopril on cardiopulmonary exercise parameters (ECCE). Am J Cardiol 1997; 80:162A167A.
18. Gibbons RJ, Balady GJ, Beasley JW, Bricker JT, Duvernoy WF, Froelicher, et al. ACC/AHA guidelines for exercise testing: executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Exercise Testing). Circulation 1997; 96:345-354.
19. Hollenberg SM. Preoperative cardiac risk assessment. Chest 1999; 115:51S-57S.
20. Gerson MC, Hurst JM, Hertzberg VS, Doogan PA, Cochran, MB, et al. Cardiac prognosis in noncardiac geriatric surgery. Ann Inter Med 1985; 103:832-837.
21. Carliner NH, Fisher ML, Plotnick GD, Garbart H. Rapoport, Kelemen MH, et al. Routine preoperative exercise testing in patients undergoing major noncardiac surgery. Am J Cardiol 1985; 56:51-58.
22. McPhail N, Calvin JE, Shariatmadar A, Barber GG, Scobie, TK. The use of preoperative exercise testing to predict cardiac complications after arterial reconstruction. J Vasc Surg 1988;7:60-68.
23. Froelicher VF. Interpretation of specific exercise test responses. In: Froelicher VF, editor. Exercise and the heart. 2 ed. Chicago IL: Year Book Medical Publishers, 1987:83-145.
24. Dunselman PH, Kuntze CE, van Bruggen A, Beekhuis H. Piers B. Scaf AH, et al. Value of New York Heart Association classification, radionuclide ventriculography, and cardiopulmonary exercise tests for selection of patients for congestive heart failure studies. Am Heart J 1988; 116: 14751482.
25. Halm EA, Browner WS, Tubau JF, Tateo IM, Mangano DT. Echocardiography for assessing cardiac risk in

patients having noncardiac surgery. Study of Perioperative Ischemia Research Group. Ann Intern Med 1996; 125:433-441.

26. Mangano DT, Hollenberg M, Fegert G. Meyer ML, London MJ, Tubau JF, et al. Perioperative myocardial ischemia in patients undergoing noncardiac surgery--I: Incidence and severity during the 4 day perioperative period. The S1ndy of Perioperative Ischemia (SPI) Research Group. J Am Coll Cardiol 1991;17:843-850.
27. Baron JF, Mundler O. Bertrand M, Vicaut E, Barre E, Godet G. et al. Dipyridamole-thallium scintigraphy and gated radionuclide angiography to assess cardiac risk before abdominal aortic surgery. N Engl J Med 1994; 330:663-669.
28. Weber KT, Janicki JS. Cardiopulmonary exercise testing for evaluation of chronic cardiac failure. Am J Cardiol 1985;55:22A-31A.
29. Anonymous. Measurements during integrative cardiopulmonary exercise testing. In: Wasserman K, Hansen J.E, Sue D.Y, Whipp BJ, Casaburi R. editors. Principles of exercise testing and interpretation. 2 ed. Malvern, PA.: Lea & Febiger, 1994:52-79.
30. Beaver WL, Wasserman K, Whipp BJ. A new method for detecting anaerobic threshold by gas exchange. J Appl Physiol 1986; 60:2020-2027.
31. Older P. Hall A. The role of cardiopulmonary exercise testing for preoperative evaluation of the elderly. In: Wasserman K, editor. Exercise gas exchange in heart disease. Armonk, NY.: Futura Publishing Company, 1996:287-297.