Cardiopulmonary Exercise Testing

Key points:

- Cardiorespiratory function is an independent predictor of perioperative morbidity and mortality
- Cardiopulmonary exercise testing is a non-invasive, integrated assessment of cardiovascular and pulmonary function both at rest and under stress.
- Improved perioperative outcome is related to the ability to increase oxygen consumption in response to surgical stress
- CPET can be used to identify patients who are less able to meet the increased oxygen delivery demands of major surgery so informing perioperative planning including preoperative optimization and allocation of postoperative critical care resources
- A low preoperative VO2 max is associated with an increased risk of post-operative complications and mortality. The lower the VO2 max, the higher the risk

MCQs –

1  With regards to CPET results:
   a) VO2 max is a better predictor of operative risk than AT
   b) Ventilation should reach 80% of predicted during CPET in a normal patient
   c) At peak exercise the heart rate should be at least 80% of the predicted value
   d) Aerobic metabolism continues beyond the AT
   e) Beyond the AT CO2 levels start to decrease

2  When assessing patient suitability for CPET, absolute contraindications include:
   a) Left main stem coronary disease
   b) Unstable angina
   c) Desaturation to 90% at rest
   d) Pregnancy
   e) Hypertrophic cardiomyopathy
Indications for stopping a CPET test include:

- Ventricular ectopics
- ST segment depression of 1.5mm
- Desaturation to 90%
- Hypertension of >250mmHg
- Fall in systolic pressure of >20mmHg

The following results indicate a ‘deconditioned’ or unfit patient:

- VO2 max of >80% predicted
- AT <40% predicted VO2 max
- High heart rate reserve
- High ventilatory reserve
- Maximum oxygen pulse of <10ml/beat

With regards to preoperative CPET testing:

- Poor CPET results should mean that surgery is not offered to the patient
- The AT is the only result that indicates perioperative risk
- CPET is an objective method to determine physiological reserve under stress
- CPET results can be used to replace clinical judgment
- The results can be used to facilitate shared decision making discussions

Cardiopulmonary exercise testing (CPET) is a non-invasive technique that measures the simultaneous cardiovascular and respiratory response to exercise stress. In a cardiac exercise test, a patient with known or suspected cardiac disease runs on a treadmill whilst their electrocardiogram (ECG) is monitored for any signs of ischaemia. CPET goes one step further by attaching a mask or mouthpiece to perform analysis of the respiratory gases. This provides much more information, not only about the cardiac and respiratory systems but can give information about the brain, peripheral circulation and leg muscle function and how they function together under physiological stress (Figure 1).[1]
Increasingly preoperative CPET is being used for preoperative risk assessment and to enable and facilitate shared decision making conversations with patients.[2]

**What happens during exercise?**

As soon as exercise commences muscle cells will increase their oxygen demand. Exercise leads to physiological changes that increase oxygen uptake and remove waste products from active tissues.

Global oxygen uptake is the VO2 measured in mls/min. VO2 is directly proportional to the cardiac output. As the exercising muscles consume more oxygen there needs to be an increase in oxygen delivery and tissue extraction which results in an increase in minute volume ventilation and cardiac output.

\[
\text{VO2} = \frac{\text{cardiac output} \times \text{oxygen extraction}}{\text{arterial O2 content} - \text{venous O2 content}}
\]

During early exercise, oxygen delivery is usually able to keep up with the demand so adenine triphosphate (ATP), the body's energy supply, is produced by aerobic metabolism. As the length and intensity of exercise increase, oxygen supply will eventually no longer be able to keep up with demand. At this point, the continued production of ATP will be supplemented by anaerobic metabolism (Figure 2). This results
in the production of lactic acid which will result in the production of hydrogen ions. These hydrogen ions require buffering with bicarbonate:

\[
[H^+] + [HCO_3^-] \rightarrow [H_2CO_3] \rightarrow [CO_2] + [H_2O]
\]

The buffering process results in the production of carbon dioxide. The excess CO2 alters oxygen utilisation and CO2 production so that more CO2 is exhaled than O2 consumed. The point at which CO2 production exceeds O2 consumption is known as the anaerobic threshold. Anaerobic metabolism is not sustainable over long periods of time and as exercise continues the patient’s ability to increase their oxygen consumption any further is overwhelmed.
CPET to assess risk

CPET has been used in the objective measurement of healthy individuals (particularly athletes) and those with medical conditions such as heart failure\[^3, 4\], respiratory disease \[^5\] as well as patients being evaluated for heart-lung transplant \[^6\] for many years. The American College of Cardiology (ACC) and the American Heart Association (AHA) joint guidelines on risk stratification for non-cardiac surgery \[^7\] identify three broad areas for assessment:

1. Surgery-specific risk
2. Patient-specific risk
3. Exercise capacity

The majority of patients with cardiac or respiratory pathology develop symptoms which are exacerbated by exertion. Many diagnostic tests are done with the patient sitting in a chair or lying on a bed. To get a true picture of the functional capacity of the patient it makes more sense to exercise the patient and get measurements while they do so. An objective assessment of functional capacity must incorporate the direct measurement of oxygen consumption for which CPET is considered the gold standard. CPET puts the cardiac and respiratory systems under stress which allows the reserve capacity of the body to be assessed, particularly in terms of its ability to deliver oxygen to peripheral tissues. This is used as a model for the stresses the body undergoes during the perioperative period. Advanced analysis of a preoperative CPET may also uncover asymptomatic disease or specific functional reserve limitations that may allow intervention strategies to be suggested.

Reasons for doing CPET:

1. Investigating patients who are short of breath
2. Assessing the contribution of cardiac or respiratory pathologies to incapacity
3. Quantifying the extent of impairment
4. Assessing the risk to the patients of a surgical procedure
5. Measuring the response to an intervention
There are many different exercise protocols and measurement techniques that can be used for CPET. The majority of CPETs are performed on an exercise bike (known as a cycle ergometer). Treadmills can be used and in fact most people have a slightly higher exercise capacity on a treadmill. Many people are under the impressions that a treadmill would be better because people are more used to walking than riding a bike (Table 1). However, walking on a treadmill is very different. Unlike normal walking, on a treadmill the ground moves and the body does not. The patient has no control over the treadmill and if they cannot keep up the pace then they are at high risk of falling. Collecting the required data is also slightly more difficult as there is more movement artefact on a treadmill. Finally, things can get a little messy if a patient suddenly stops on a moving treadmill!

<table>
<thead>
<tr>
<th></th>
<th>Cycle Ergometer</th>
<th>Treadmill</th>
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<tbody>
<tr>
<td>Safer, less chance of falling off</td>
<td>Safer, less chance of falling off</td>
<td>Intimidating for many</td>
</tr>
<tr>
<td>Quieter</td>
<td>Quieter</td>
<td>Noisy</td>
</tr>
<tr>
<td>Less movement artefact (ECG &amp; BP)</td>
<td>Less movement artefact (ECG &amp; BP)</td>
<td>Movement artefact especially ECG/BP</td>
</tr>
<tr>
<td>Gradual increase in exercise level (Ramp)</td>
<td>Gradual increase in exercise level (Ramp)</td>
<td>Peak VO2 higher</td>
</tr>
<tr>
<td>Detects multiple conditions affecting heart, lungs and muscles</td>
<td>Detects multiple conditions affecting heart, lungs and muscles</td>
<td>Incremental exercise increase - patients may have difficulty in keeping up with large jumps in speed/incline</td>
</tr>
<tr>
<td>Greater exercise time so more information</td>
<td>Greater exercise time so more information</td>
<td>Shorter exercise time so less information</td>
</tr>
<tr>
<td>Exercise level easily adjusted so each patient exercises for approximately 8-12 minutes</td>
<td>Exercise level easily adjusted so each patient exercises for approximately 8-12 minutes</td>
<td>Improper use of hand supports affects results</td>
</tr>
</tbody>
</table>
Preparing the patient

Before carrying out diagnostic CPET, it is essential to take a detailed history and examine the patient clinically. Patients have often had other investigations such as ECG, full blood count, renal function etc. and it is useful to check these prior to carrying out CPET.

The test is considered safe with reported mortality rates of 2-4 per 100,000.[8] There are some absolute and relative contraindications to carrying out CPET (Table 2).[9] Patients awaiting major joint replacement are often able to complete the test so this should not be an excluding factor.

Table 2: CPET contraindications

<table>
<thead>
<tr>
<th>Absolute Contraindications</th>
<th>Relative Contraindications</th>
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<tbody>
<tr>
<td>- Acute myocardial infarction (within last 3-5 days)</td>
<td>- Left main stem coronary disease or its equivalent</td>
</tr>
<tr>
<td>- Unstable angina</td>
<td>- Moderate stenotic valvular heart disease</td>
</tr>
<tr>
<td>- Uncontrolled arrhythmias causing symptoms or haemodynamic compromise</td>
<td>- Severe untreated hypertension (&gt;200mmHg systolic and &gt;120mmHg diastolic)</td>
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<tr>
<td>- Syncope</td>
<td>- Tachyarrhythmias or bradyarrhythmias</td>
</tr>
<tr>
<td>- Active endocarditis, myocarditis or pericarditis</td>
<td>- High-degree atrioventricular block</td>
</tr>
<tr>
<td>- Symptomatic severe aortic stenosis</td>
<td>- Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>- Uncontrolled heart failure or pulmonary oedema</td>
<td>- Significant pulmonary hypertension</td>
</tr>
<tr>
<td>- Acute pulmonary embolus</td>
<td>- Advanced or complicated pregnancy</td>
</tr>
<tr>
<td>- Lower limb thrombosis</td>
<td>- Electrolyte abnormalities</td>
</tr>
<tr>
<td>- Suspected dissecting aneurysm</td>
<td>- Orthopaedic impairment</td>
</tr>
<tr>
<td>- Uncontrolled asthma</td>
<td>- Dehydration at rest to &lt;85% on room air</td>
</tr>
<tr>
<td>- Desaturation at rest to &lt;85% on room air</td>
<td>- Respiratory failure</td>
</tr>
<tr>
<td>- Mental impairment leading to inability to co-operate</td>
<td>- Mental impairment leading to inability to co-operate</td>
</tr>
<tr>
<td>- Acute non cardiopulmonary disorder that may affect ability to exercise e.g. infection, renal failure, thyrotoxicosis</td>
<td>-</td>
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Usually a CPET consists of four phases:

1. **Resting baseline:** 2-3 minutes remaining stationary on the bike to determine resting gaseous exchange. Just putting a mask over the face can alter the breathing pattern therefore a minute or two is allowed for this to stabilise and give a stable resting baseline measurement.

2. **Unloaded cycling:** This is essentially the freewheeling stage. Once the patient starts to turn the ergometer pedals there will be another change in breathing pattern as they anticipate having to do additional muscular work. 2-3 minutes of cycling against no resistance allows stabilisation of baseline exercise measurements.

3. **Ramped exercise:** The resistance is slowly and continuously increased. For example a 15 watt ramp means that the work rate is increased by 15 watts every minute of the test. As the resistance gets harder the patient will at some stage be unable to turn the pedals at the required rate and will have to stop. Protocols are designed to produce a maximum predicted work for an individual in 10 minutes. The speed at which the resistance increases depends on the patient’s age, sex and co-morbidities.

4. **Recovery phase:** The workload is removed and the patient should continue with a short period of unloaded cycling as a ‘warm down’. This helps to prevent any sudden drop in blood pressure. All monitoring is continued in this phase as occasionally informative changes develop during recovery.

**Supervising a CPET**

In the early days of CPET, tests were often initially stopped above the anaerobic threshold (AT) but before symptom limitations because of safety concerns. However, a review of the safety of CPET has shown a mortality rate of approximately 2 to 5 per 100,000 in populations that include patients undergoing heart and lung transplants.[10] Now symptom limited tests are most commonly used where the patient is encouraged to exercise until they are unable to continue and have to stop. Reasons for stopping are recorded and this helps in determining the reason for a suboptimal exercise test.

There are a few occasions where it is necessary to stop the test early. Absolute indications to stop are largely cardiac (Table 3). It goes without saying that resuscitation equipment must be readily available during a CPET.
Dysrhythmias

Patients with heart disease may have some ventricular ectopics at rest, which will often disappear on exercise. It is more worrying if ventricular ectopics start to occur in pairs or triplets, or if there is a change in their morphology. It will usually be prudent to stop the test at this stage. Once short runs of ventricular tachycardia develop, the test should definitely be terminated. If atrial fibrillation develops during the test, then this is a good explanation for the patient’s symptoms and there is little point in continuing the test.

ST segment changes

If clear ST segment depression of 2mm or more develops during the test, myocardial ischaemia is present. To some extent this will depend on the circumstances of the patient and the reason for the test. Myocardial ischaemia is a common diagnosis in the population of patients that attend for a CPET. They often report dyspnoea but have reasonable lung function. The oxygen pulse may fall towards the end of exercise if the myocardium becomes ischaemic and this can be used as a proxy for cardiac output.

Oxygen desaturation

Desaturation during CPET is uncommon, except when exercising patients with known lung disease. There are no absolute thresholds, but for a diagnostic CPET, if the saturations are normal at rest, then the test should be stopped if they start to fall below 80%. Falling saturations suggest significant pathology. However, if using a finger probe and the subject grips the handlebars tightly then this can cause apparent desaturation.

Blood pressure

Systolic blood pressure should rise during exercise. If it falls it usually signifies significant myocardial ischaemia.

Airflow limitation

CPET equipment that displays flow-volume plots can be used to detect expiratory airflow limitation. It should be looked for at intervals during the test, being indicated by a concave ‘scalloping’ of the expiratory loop.
If the test is terminated by the patient, then careful questioning is essential to determine the reason for stopping. When risk stratifying patients on a preoperative CPET reasons for stopping that include lower extremity muscle fatigue would be classed as lower risk than stopping due to angina or dyspnoea.[11]

**Measurements and variables collected during CPET**

The measurements made during CPET are summarised in table 4. (Modified from Levett et al. 2015 [12])

<table>
<thead>
<tr>
<th>Table 3: Indications for CPET termination</th>
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<tbody>
<tr>
<td>Chest pain indicative of ischaemia</td>
</tr>
<tr>
<td>Ischaemic ECG changes</td>
</tr>
<tr>
<td>Dysrhythmias – sustained or change in morphology</td>
</tr>
<tr>
<td>Development of heart block – second or third degree</td>
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<tr>
<td>Hypertension (&gt;250mmHg systolic or &gt;120mmHg diastolic)</td>
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<tr>
<td>Fall in systolic pressure &gt;20mmHg from the highest value during the test</td>
</tr>
<tr>
<td>Severe desaturation below 80% with signs and symptoms of severe hypoxaemia</td>
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<tr>
<td>Loss of coordination</td>
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<tr>
<td>Confusion</td>
</tr>
<tr>
<td>Dizziness or faintness</td>
</tr>
<tr>
<td>Signs of respiratory failure</td>
</tr>
<tr>
<td>Sudden pallor</td>
</tr>
</tbody>
</table>
The average CPET records approximately 5000 measurements. These measurements are used to produce multiple combinations graphs of measured haemodynamic and ventilator variables. The most common way the data is represented is on the 9-panel plot (Figure 3). When looking at perioperative risk the important test outcomes relate to markers of reduced functional reserve and measures that may indicate specific system dysfunction.
Respiratory exchange ratio (RER)

The RER is the CO2 elimination divided by the oxygen uptake. In a steady state conditions, the RER will equal the respiratory quotient (RQ). The RQ is determined by the fuel used for metabolic processes. An RQ of 1 would be seen if carbohydrates provided fuel, 0.7 for fats and 0.8 for protein. In reality the body uses a combination of fuels for metabolism and the RQ is just below 1. The RER is measured by gas exchange. In a true steady state RER = RQ as blood and gas transport mechanisms are keeping pace with tissue metabolism. At the start of the CPET the RER should be <1. During exercise as
the patient hyperventilates and lactic acid builds up the CO2 eliminated by gas exchange increases. This causes the RER to rise above 1. Measuring the RER is useful for several reasons. It can help to determine the AT and a rise to >1 shows that the patient has exercised to a good effort. It can be an indication of hyperventilation. At the start of the test the RER is often >1 as the patient hyperventilates due to anxiety and the anticipation of the exercise test. If this is the case it usually settles down and falls back below 1 as the patient starts exercising. Occasionally an erratic RER may be seen throughout the CPET. This may be an indicator of dysfunctional breathing where patients have abnormal ventilation that is unrelated to respiratory pathology. Some experts advocate the early use of CPET to diagnose dysfunctional breathing and identify these patients so that they are not exposed to further, potentially hazardous investigations and can begin to receive appropriate therapy.[14]

Markers of functional reserve

These markers include heart rate and ventilatory reserves, peak and maximal oxygen uptake and the anaerobic threshold. They have been found to relate to postoperative outcome in several surgical populations including abdominal, colorectal, vascular, hepatobiliary, oesophageal and liver transplantation.[15, 16, 17, 18]

Heart Rate Response

During the exercise phase of the CPET the heart rate usually increases steadily and is at the highest point just before the patient stops. The maximum recorded heart rate can then be compared to the predicted maximum heart rate. Normally 80% or more of the predicted heart rate will be reached at peak exercise.

A heart rate of >80% of the maximum predicted is referred to as a ‘low heart rate reserve’. This indicates that the possibility of increasing the heart rate further is limited. Patients with ‘normal’ cardiac function will reach >80% of their predicted heart rate and have a normal VO2 max. Patients who are unfit will reach their maximum predicted heart rate quickly but will have a low VO2 max. Patients who have impaired left ventricular function or those with pulmonary vascular disease with impaired right ventricular output rely on increasing their cardiac output by increasing their heart rate. They will also reach maximum heart rate quickly and have a low VO2 max.

A high heart rate reserve is when the recorded heart rate does not reach 80% of the predicted value. Chronotropic insufficiency may be due to submaximal effort but is also seen when exercise is limited by something other than cardiac function such as lung
Disease or peripheral vascular disease. It may also be due to beta-blockers or sinoatrial dysfunction.

Heart rate is often raised at the start of the CPET. Patients are frequently anxious and unsure what to expect leading to a tachycardia and hyperventilation. Usually both heart rate and ventilation settle once exercise commences and then a normal pattern is seen. A persistent tachycardia may be a warning sign of poor ventricular function. In this scenario, stroke volume can not increase so the only way to increase cardiac output is by a faster heart rate.

**Ventilatory Reserve**

Increasing ventilation during exercise is the primary way in which the arterial blood regulates gases and acid-base status with the increased metabolic demands of exercising muscles. The measure of ventilation used in CPET is ventilatory equivalents or VE. It is the sum of the volume of all the breaths in one minute and more commonly referred to in anaesthesia as the minute volume. It is affected by the frequency and depth of breathing. Younger patients are usually able to increase their tidal volumes approximately 3 to 5 fold to about 60% of their vital capacity. Older patients usually increase tidal volumes slightly less than this. Respiratory rate usually doubles at least during exercise. Patients often hyperventilate at the start of the case but then settle down as the load starts to kick in. This is not unusual and does not imply that their symptoms are due to hyperventilation, especially if the ventilation rises steadily once they settle down.

Maximum minute ventilation (VEmax) or maximum voluntary ventilation (MVV) is usually estimated from a subject’s FEV1.[19] MVV can be assessed by asking the patient to breathe as deeply and quickly as possible for 15 seconds and then measuring the amount of gas expired. However, this measurement is dependent on the motivation of the subject and will lead to hypocapnia, potentially to dizziness and syncope and can provoke bronchospasm.

The ventilatory reserve is based on the same concept as the heart rate reserve. If VEmax is >80% of the predicted value then there is little possibility of increasing ventilation any further. This is a low ventilatory reserve. Usually, even in patient with cardiac disease, cardiac output limits exercise. There is usually sufficient reserve in ventilation that it does not reach 80% of the predicted value. In lung disease the patient may stop due to ventilatory limitation. From a cardiac point of view, the heart rate is usually less than 80% predicted because it is as if the patient stopped before maximum
capacity. In clinical practice CPET rarely reveals significant lung disease that is not already known about from other diagnostic tests done before CPET is considered.

Peak and maximal oxygen uptake

When oxygen uptake is plotted against work, the VO2 increases linearly at 10ml/min/watt before plateauing (VO2 max) as one of the systems such as cardiac output or ventilation reaches its limit. In the perioperative test generally fatigue or symptoms are the limiting factor in the CPET and the plateau and VO2 max is rarely reached (Table 5). Consequently, VO2 peak (the last value of VO2 before stopping exercise) is used instead. VO2 peak represents the cardiorespiratory reserve. VO2 peak or max is subject to an individual's effort therefore motivation can affect the levels achieved.

Several studies have investigated the ability of VO2 max to predict adverse postoperative outcomes, particularly in those patients who are at high risk of complication or death. The three main findings from these studies are:[16, 20, 21]

1 A lower VO2 is associated with earlier death in the general population even if there is no concomitant diagnosed major illness.
2 VO2 max is closely related to the risk of death or major postoperative complication in cardiac and thoracic surgery and is associated to a varying degree with poorer outcomes in other major surgery.
3 A lower VO2 max is associated with a poorer outcome in chronic cardiopulmonary conditions.

Anaerobic Threshold (AT)

This marker is well known and many healthcare professionals are aware that a low AT is associated with a higher perioperative risk. The AT is the point at which oxygen demand exceeds supply and aerobic metabolism is overwhelmed. Beyond the AT,
Anaerobic processes supplement aerobic metabolism with the production of lactic acid. Lactic acid is buffered by bicarbonate to produce more CO2. As anaerobic metabolism takes over the CO2 production increases and exceeds oxygen consumption. The point that this occurs is visible on several different graphs produced during the CPET. It is usually most easily visualised on the modified V-slope graph (Figure 4). This graph plots oxygen consumption (mls/min) against CO2 production (mls/min). The graph produces a linear correlation. As the AT is reached and the CO2 production increased the gradient of the linear relationship changes. This generally produces a distinct point on the graph. Changes in other measured CPET variables such as VE/VCO2, VE/VO2 and end-tidal oxygen levels can be used to confirm the AT.

AT is an objective end-point that is unrelated to effort. It has been shown to be an independent predictor of post-operative complications [22, 23] A normal AT should be greater than 40% of the predicted VO2 max. It can be a lot higher in certain population groups as detailed in table 6.
Anaerobic metabolism occurs when the circulation is not able to deliver enough oxygen to meet the metabolic needs of the tissues. This may occur at a lower exercise intensity than normal for several reasons:

- Low cardiac output
- Peripheral circulation to the leg muscles is obstructed
- Peripheral oxygen saturation of arterial blood is low

By keeping the above list in mind the AT then gives us additional information about how the body is performing during exercise.

Markers of specific system dysfunction

Ventilatory equivalents

Ventilatory equivalents are important markers of a poor ventilation/perfusion (V/Q) ratio. Ventilatory equivalents is the minute ventilation (VE) divided by with oxygen (VE/VO2) or carbon dioxide (VE/VCO2). Using VE/VO2 it looks at the millilitres of air that need to move in and out of the lungs to get a millilitre of oxygen uptake. It is an index of how well the lungs work. The more air that needs to move in and out of the lungs to get the oxygen uptake then the worse the lung function. The VE/VCO2 indicates how many litres of air are being breathed to eliminate 1 litre of CO2.

During exercise both the VE/VO2 and VE/VCO2 will initially fall as cardiac output increases and the V/Q matching becomes more equal. The lowest point for VE/VO2 indicates when the lungs are working at their most efficient. After the AT is reached the VE/VO2 will start to climb steadily. This is not because the lungs have become less efficient but because the blood flow out of the lungs is fully saturated with oxygen and no more is able to be taken up. After the AT the VE/VCO2 will initially remain relatively
steady. It will then start to climb as anaerobic metabolism causes the CO2 levels in the blood rise and ventilation increases to buffer the acidaemia. One of the methods of determining the AT is looking for the low point of VE/VO2 where the VE/VCO2 is still at a steady state and has not yet started to climb (Figure 5).

The slope of the relation between VE and VCO2 (commonly referred to as the VE/VCO2 slope) is used as a measure of ventilatory efficiency. The normal VE/VCO2 slope is usually less than 30. Up to 34 is considered normal. An increased VE/VCO2 may be seen in lung conditions, heart failure and pulmonary hypertension.[24, 25, 26]

**Oxygen pulse**

The oxygen pulse is simply oxygen uptake (VO2) divided by heart rate. It represents the amount of oxygen taken up by the lungs into the blood with each heart beat. The more blood flowing through the lungs, the more oxygen that is taken up. The oxygen pulse can be used as an indirect indicator of cardiac stroke volume. A normal patient should achieve an O2 pulse of at least 10ml/beat at peak exercise and often 15ml/beat or more. The oxygen pulse is plotted on the same graph as the heart rate. In a normal patient the oxygen pulse will increase during the early part of the test as stroke volume increases, then it tails off and any further increase in cardiac output is related to the
heart rate. A plateau in the O2 pulse at a low value implies limited cardiac output either secondary to cardiac disease or disorders of the pulmonary circulation. If cardiac ischaemia suddenly develops during the test, then stroke volume may become impaired and the O2 pulse may suddenly plateau or tail off.

Patterns that may be seen in specific disease states

**Cardiac disease**

Patients with cardiac disease often have a low cardiac output. Exercise capacity is limited because these patients are unable to transport sufficient oxygen from the lungs to the muscles. Anaerobic metabolism will therefore be needed sooner to supplement aerobic metabolism and provide ATP to power muscle function. The following results may be seen:[27]

- Low VO2 max
- Rapid and early rise in heart rate with a low heart rate reserve
- A plateau in the O2 pulse with a peak value of <10ml/beat
- Low AT
- High VE/VCO2 slope

**Lung disease**

Patients with lung disease are often unfit so have a low AT. They frequently stop early due to breathlessness and have problems with getting rid of CO2. The following results may be seen:[9, 28]

- Low VO2 max
- High heart rate reserve
- Low ventilatory reserve, a high VE/VCO2 slope and desaturation

**Pulmonary vascular disease**

Usually patients with pulmonary vascular disease have relatively normal mechanics of breathing so are able to increase their ventilation sufficiently. But they tend to desaturate and have a low cardiac output due to obstruction of the pulmonary vascular system. The following results may be seen:[29]
• Low VO2 max
• Low heart rate reserve
• Low oxygen pulse
• Low AT
• High VE/VCO2 slope and desaturation

Peripheral vascular disease

The subset of patients that are referred for a CPET test in the work up for major surgery often have a degree of peripheral vascular disease (PVD). PVD prevents oxygen being delivered to the leg muscles therefore anaerobic metabolism is needed far earlier than usual. As exercise continues the patient develops leg pain (the classic ‘cramps’ associated with PVD) and stop. The following results may be seen:[30]

• Low VO2 max
• High heart rate and ventilatory reserve
• Low AT

Sub-maximal effort test

For a variety of reasons, a patient may perform a sub-maximal effort test. Motivational factors can contribute as can factors such as arthritic joint pain. Some muscles diseases can cause patterns that resemble sub-maximal effort, except that the AT is low. Careful questioning of the patient about why they stop exercising is the key to determine this. Sub-maximal effort on a CPET is seen as:[9, 31]

• Low VO2 max
• High heart rate and ventilatory reserve
• AT not reached, indeterminate or normal

Deconditioning

Many patients are unfit or ‘deconditioned’ but do not have any formal cardiac or respiratory pathology. Due to the symptoms they may describe on questioning and the difficulty in determining whether these symptoms are due to lack of fitness or disease (breathlessness on exertion, reduced exercise tolerance etc.) they are often referred for CPET prior to major surgery. Deconditioned patients have a lower stroke volume
therefore the heart rate will increase rapidly to achieve the same cardiac output as a fitter individual. Deconditioning can produce similar results to those seen with mild cardiac disease and mild myopathies. Often the patient’s symptoms at the time of stopping the test (chest pain/muscle cramps) can help to guide the diagnosis but occasionally other investigations may be required. CPET results in deconditioning may show:[32]

- Low VO2 max
- Low heart rate reserve and a high ventilatory reserve
- Maximum oxygen pulse of <15ml/beat
- Low AT - usually <40% of predicted VO2 max

Surgery is associated with a substantial burden of perioperative morbidity and mortality. As life expectancy continues to rise and an increasing proportion of patients present for surgery at a later age with more co-morbidities this represents a challenge for both anaesthetists and surgeons. Having the ability to assess the functional capacity adds an important perspective. CPET is a powerful tool to reliably and objectively assess the functional reserve and look at how patients physiologically react to being put under physical stress. CPET is not intended to replace clinical examination or clinical judgement but is designed to supplement the information already gathered. It provides an individualised estimate of patient risk that can be used to inform collaborative decision making and patient consent. It can assist in identifying areas that need addressing and how the patient could be ‘fitter’ before surgery. It is rarely a ‘pass/fail’ test in the work up to major surgery i.e. a test where good performance = able to proceed with surgery and vice versa but a test that can give a wealth of information to enable the appropriate perioperative management plan to be considered. Having CPET information can massively benefit and facilitate the shared decision making discussion between the patient, anaesthetist, surgeon and perioperative team.

MCQ answers

1 TFTTF
2 FTFFF
3 FFFTT
4 FTFTF
5 FFTFT
References


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