ABSTRACT

Because of technical and practical difficulties in relation to increased body size, haemodynamic monitoring of morbidly obese critically ill patients (i.e. body mass index \( \geq 40 \text{ kg/m}^2 \)) may be challenging. Obese and non-obese patients are not so different with respect to haemodynamic monitoring and goals. The critical care physician, however, should be aware of the basic principles of the monitoring tools used. The theoretical assumptions and calculations of these tools could be invalid because of the high body weight and fat distribution. Although the method of assessing haemodynamic data may be more complex in morbidly obese patients, its interpretation should not be different from that in non-obese patients. Indeed, when indexed for body surface area or (predicted) lean body mass, reliable haemodynamic data are comparable between obese and non-obese individuals.

KEYWORDS

Haemodynamic monitoring, critically ill patients, intensive care unit, obesity, cardiac output, vascular access, cardiovascular

INTRODUCTION

Obesity, in its epidemic presentation, is still increasing in the industrialised world. The prevalence of obesity in intensive care units (ICUs) has increased over recent years as well. Its prevalence has been estimated from 5 up to 25% and is associated with substantial morbidity and mortality. The degree of obesity is traditionally assessed by body mass index (BMI), which is the ratio of weight (in kilograms) to height (in meters squared). Current definitions and terms of obesity are: overweight: BMI 25.0-29.9 kg/m\(^2\); class I obesity: BMI 30.0-34.9 kg/m\(^2\); class II obesity: BMI 35.0-39.9 kg/m\(^2\); and class III obesity: BMI \( \geq 40 \) kg/m\(^2\). Class III is referred to as severe, extreme or morbid obesity. Reasons to initiate any form of haemodynamic monitoring in critically ill patients are multiple. The identification of a disease state and/or its complications is one major reason. Another reason is to understand the aetiology of the disease, usually shock-like pathology. Pending the cause of shock (hypovolaemic shock, cardiogenic shock, obstructive shock or distributive shock), treatment will be different and should be individualised. In line with this, another reason is to tailor treatment, according to the underlying disease, to improve supply, and meet the metabolic demands of the tissues. The ultimate goal is to improve the patient’s outcome. Notably, there is a tendency in time, mostly for safety reasons, to develop and use more non- and/or semi-invasive devices for continuous haemodynamic monitoring.

Indications for haemodynamic monitoring in critically ill patients are probably quite similar between obese and non-obese patients. The critical care physician, however, may be confronted with unique and challenging problems in morbidly obese patients related to changes of anatomy and fat distribution with higher body weights. Literature on haemodynamic monitoring in morbidly obese patients is scarce and sketchy. Haemodynamic monitoring protocols for the morbidly obese population differ between institutions and are frequently based on individual experiences. Moreover, most protocols are based on information from studies and reports derived...
Cardiovascular alterations in relation to obesity comprise an increase in total blood volume, cardiac output (CO) and stroke volume (SV), linearly with increasing body weight. The heart rate is unaffected. Left ventricular end-diastolic diameter is increased (i.e., dilatation) as a result of the increased left ventricular stroke volume. In obese individuals the left ventricular systolic function is depressed although left ventricular ejection fraction is increased. Right ventricular function seems to be unchanged. Systemic oxygen delivery is increased as a result of increased CO, serving the metabolic demands of excess fat.

The interpretation of haemodynamic data per se should not be complicated in case of morbid obesity. When indexed for body surface area (i.e., cardiac index (CI); CI = CO/body surface area in m²) the output is not different between obese and non-obese individuals, regardless the changes in CO. Other haemodynamic parameters (such as extravascular lung water measurement) may be indexed to predicted lean body mass, expressed as ml.kg⁻¹. In view of the cardiovascular effects of obesity, however, precautions in data interpretation between obese and non-obese patients must be taken into account, considering the technical and physiological principles and the algorithms of the applied haemodynamic monitoring devices.

Standard haemodynamic monitoring

Table 1 summarises standard methods of haemodynamic monitoring.

Physical examination

Central venous pressure (CVP) estimation by physical examination in non-obese patients is known to be difficult, resulting in a high inter- and intra-observer variability. In obese patients CVP measurement is even more difficult as CVP estimation may be obscured due to the high body weight. Jugular venous distension, hepatoglossal reflex, cannon waves (as a sign of atrioventricular dissociation), and tricuspid regurgitation may be diagnosed at the bedside. Low body (core) temperature and decreased diuresis in obese patients, similar to that in non-obese patients, may indicate insufficient circulation.

Non-invasive arterial blood pressure measurement

Arterial blood pressure measurement is an essential part of haemodynamic monitoring. Non-invasive blood pressure measurements (oscillometric or auscultatory by sphygmomanometry), however, are shown to be inaccurate in obese patients when compared with intra-arterial blood pressure readings. For accurate blood pressure measurement it is essential to use of a proper-sized blood pressure cuff in obese patients. The length of the bladder should be at least 75 to 80% of the circumference of the upper arm, and the width of the bladder should be more than 50% of the length of the upper arm and approximately 40% of the circumference of the upper arm. Systolic pressure may be overestimated if too small a cuff is used. Blood pressure and heart rate may alternatively be monitored non-invasively from the radial artery (e.g., with the Vasotrac system). The Vasotrac measurements were found to differ considerably compared with intra-arterial measurements in obese patients undergoing bariatric surgery although this method was judged to be more comfortable by patients when compared with oscillometric blood pressure methods.

Alternatively, blood pressures may be measured by ankle monitoring or by Doppler technique when blood pressure readings could not be obtained from the upper extremities. Ankle systolic and mean arterial pressures are significantly higher than brachial blood pressures but severe hypotension may interfere with ankle blood pressure monitoring. Systolic blood pressure may be assessed by the Doppler technique as well by use of an appropriate pressure cuff for the legs (calf). After reaching the systolic blood pressure Doppler tones will be audible at the foot arteries (i.e., arteria dorsalis pedis, arteria tibialis anterior) during de-sufflation of the cuff.

Invasive arterial blood pressure measurement (catheterisation)

Intra-arterial measurement of systemic blood pressure (direct measurement) is nowadays a routine procedure in the ICU. Besides blood pressure measurement, arterial cannulation facilitates repeated samplings of arterial blood gas analysis, which permits frequent assessment of oxygenation/gas exchange and ventilation, as well as acid-base monitoring. Arterial cannulation, however, may be difficult in obese patients because of the obscured anatomic landmarks and concomitant peripheral oedema.
In the early 1980s pulse contour analysis was introduced to calculate SV and CO by analysis of the arterial pressure pulse contour (waveform). Pulse contour analysis, as a technique, is based on the assumption that the contour of the arterial pressure waveform is proportional to SV, which can be estimated by the integral of the change in pressure from end-diastole to end-systole over time. This integral calculation comprises several assumptions with respect to individual patient characteristics. These assumptions, for example, implicate demographic variables (such as age, gender, height and weight) and vascular wall characteristics (depicted in skewness and kurtosis of the waveform, summarised in factor $X$), which may be quite different in obese compared with non-obese patients. It has been shown that uncalibrated pressure pulse contour analysis underestimates SV or CO in obese patients due to systematic errors by these assumptions. For these reasons, uncalibrated CO monitoring should be examined as a trend rather than a single value in obese patients.

**Electrocardiography**
One of the standard monitoring tools in the ICU is electrocardiography (ECG) or rhythm monitoring. Obesity is associated with a wide variety of ECG abnormalities. After substantial weight loss many of these ECG abnormalities are reversible. ECG alterations with obesity include changes in electrical axes, conduction times (including corrected QT interval) and P, QRS and T wave voltages. ECG abnormalities may serve as markers of risk for sudden cardiac death but not all ECG alterations are ominous. Some ECG changes, however, represent alterations in cardiac anatomy or morphology associated with obesity and/or its comorbidities. It has to be considered that

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**Table 1. Standard haemodynamic monitoring**

<table>
<thead>
<tr>
<th>Monitoring device</th>
<th>Measurements / parameters</th>
<th>General remarks or problems</th>
<th>Remarks or problems in obese patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Physical examination: - Central venous pressure</td>
<td>Jugular venous distention, Hepatojugular reflux, Cannon waves</td>
<td>High intra- and inter-observer variability</td>
<td>Clinical judgement may be obscured by obesity</td>
</tr>
<tr>
<td>- Temperature</td>
<td>- Diuresis</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Arterial pressure by sphygmomanometry or oscillometric methods</td>
<td>Systolic and diastolic BP, Heart rate, Pulsus paradoxus</td>
<td>Reliable, if correct technique used</td>
<td>Reliable, if correct technique used</td>
</tr>
<tr>
<td>Arterial catheterisation</td>
<td>Parameters: Systolic BP – Diastolic BP – Mean arterial BP – Heart rate – Pulse pressure – Pulsus paradoxus</td>
<td>Standard monitoring tool in ICU</td>
<td>Due to alterations in arterial compliance characteristics, CO by uncalibrated arterial pressure waveform analysis is not reliable but may be used as trend in the obese</td>
</tr>
<tr>
<td>Arterial blood gas analysis</td>
<td>In case of arterial pressure waveform analysis: Stroke volume – Cardiac output – Pulse pressure variation – Stroke volume variation</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Electrocardiography</td>
<td>Heart rate, Dysrhythmias, Conduction disorders, Previous MI, Myocardial ischaemia</td>
<td>None</td>
<td>Changes related to obesity (see text)</td>
</tr>
<tr>
<td>Pulse oximetry</td>
<td>SpO₂, Heart rate</td>
<td>Inaccurate signals by motion artefacts, nail polish, hypotension, low cardiac output, vasoconstriction, hypothermia, haemoglobinopathy</td>
<td>None</td>
</tr>
<tr>
<td>Capnography</td>
<td>End-tidal CO₂</td>
<td>Related to minute ventilation and cardiac output</td>
<td>Higher difference in end-tidal CO₂ and arterial CO₂ compared with non-obese</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Check for correct endotracheal ventilation</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>Check for global circulation in case of resuscitation</td>
<td></td>
</tr>
</tbody>
</table>
Obesity, by itself, may invalidate commonly used ECG diagnostic criteria. Criteria for left ventricular hypertrophy, for example, may be invalidated by isolation effects of the body fat, yielding lower QRS-complex voltages.\(^3\) Obesity has been identified as an arrhythmogenic factor. Obesity is also associated with the occurrence of both supraventricular and ventricular arrhythmias and even sudden cardiac death.\(^4,5\) Atrial fibrillation is the most prevalent supraventricular arrhythmia associated with obesity. Atrial fibrillation in obese patients has been shown to be related to worse clinical outcome.\(^6,7\) Case reports and small studies reported a variety of cardiac arrhythmias and conduction disturbances in obese patients with left ventricular hypertrophy and sleep apnoea syndrome.\(^8\)

**Pulse oximetry**
Pulse oximetry is, alike ECG, one of the standard monitoring tools in routine critical care medicine.\(^9\) Pulse oximetry is based on spectrophotometric features of pulsatile arterial blood flow and absorption characteristics of oxyhaemoglobin and deoxyhaemoglobin with respect to two different wavelengths of light (660 nm (red light) and 940 nm (infrared), respectively).\(^10\) Pulse oximetry is probably the most valuable non-invasive method to continuously monitor oxygen saturation in patients at risk for hypoxaemia. There is still debate about the clinical benefit of early detection of hypoxaemia by oximetry in the perioperative period although the use of pulse oximetry in the ICU is considered to be a routine procedure.\(^11\) The pulse oximeter is less reliable in low perfusion states and hypothermia. In case of dyshaemoglobinemia (e.g. with carbon monoxide poisoning and methaemoglobinemia) pulse oximeters are not able to measure oxygenation adequately. It has been shown that pulse oximetry does not improve outcome in perioperative care.\(^12,13\) In this respect no differences seem to exist between critically ill non-obese and obese patients.

**Capnography**
During mechanical ventilation changes in patients’ cardiovascular and respiratory status can be assessed by capnography. Reduction in CO results in decreased end-tidal CO\(_2\) (EtCO\(_2\)) values and in increased end-tidal arterial CO\(_2\) differences. In case of effective cardiopulmonary resuscitation, increases in end-tidal CO\(_2\) signify an increase in CO (i.e. pulmonary capillary blood flow). In mechanically ventilated obese patients, capnography may be helpful to assess changes in the patient’s global haemodynamic status.\(^14\) In spontaneously breathing, unintubated patients sampling EtCO\(_2\) through a nasal cannula is potentially problematic when expired gas mixes with ambient air. The resulting inaccurate measurements produce artificially low values compared with a closed system with minimal dead space. Intermittent mouth breathing might also contribute to underestimated EtCO\(_2\) values. Since mouth breathing is common in obese patients, especially those with a history of obstructive sleep apnoea, exhaled flow distribution between the mouth and nose highly affects the accuracy of capnometry. Capnography in spontaneously ventilating obese patients, therefore, is not the ideal technique for adequate haemodynamic monitoring. Capnography in these patients may be used to assess the global (changes in) haemodynamic status.

**Extended haemodynamic monitoring**

*Table 2* summarises further methods for haemodynamic monitoring.

**Central venous catheterisation**
CVP may be measured by single cannulation of the internal jugular, subclavian or axillary vein (or as part of) pulmonary artery catheterisation. Alternatively, peripherally inserted central catheters can be used in the obese to measure central venous pressure and to ensure reliable vascular access (infusion of fluids, intravenous medication, diagnostic blood draws).\(^16\) The efficacy of central venous pressure measurements to judge intravascular volume status or fluid responsiveness remains controversial, both in obese and non-obese patients.\(^17\)

**Pulmonary artery catheter**
The pulmonary artery catheter (PAC) has been used frequently in critically ill patients, after its introduction in clinical practice in the late 1970s.\(^18\) The PAC provides haemodynamic data (i.e., pulmonary artery pressure, pulmonary capillary wedge pressure, central venous pressure and CO by thermodilution) to judge cardiovascular status and to assist therapeutic decisions.\(^19\) An external pacemaker can be introduced via adjusted PAC systems in case of chronotropic incompetence or haemodynamically important bradyarrhythmias. Critically ill obese patients demonstrate elevated right atrial, mean pulmonary artery, and pulmonary artery wedge pressures. It should be noted that the validity of derived parameters indexed to body surface area has been questioned in morbidly obese patients. Several studies have demonstrated that these parameters are appropriate and valid when indexed to body surface area. The large body surface area in the obese patient, in fact, does not affect these measurements.\(^20,21\)

Inappropriate use as well as poor interpretation of the PAC data may increase morbidity and mortality.\(^22,23\) In a study involving patients including BMI >30 kg/m\(^2\), there
was no significant difference in outcome in high-risk surgical patients monitored with PAC versus central venous catheter. More importantly, a higher incidence of pulmonary embolism in the PAC group was reported. Not surprisingly, the use of PAC has decreased over the years, despite critical study results in favour of the PAC in selected patients treated by PAC-trained physicians.

### Table 2. Extended haemodynamic monitoring

<table>
<thead>
<tr>
<th>Monitoring device</th>
<th>Measurements / parameters</th>
<th>General remarks or problems</th>
<th>Remarks or problems in obese patients</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Central venous catheterisation</strong></td>
<td>Central venous pressure, waveform, respiratory variations Central venous blood gas analysis (pH, PcvO2, ScvO2, PcvCO2, Hb) Transpulmonary techniques (thermodilution or by indicator): Cardiac output – Stroke volume – Stroke volume variation – Intrathoracic blood volume – Extravascular lung water – Global end-diastolic volume – DO2</td>
<td>Locations for insertion: Internal jugular vein – Subclavian vein – Femoral vein Transpulmonary technique may be inaccurate in case of heart valve dysfunction (e.g. tricuspid and/or aortic valve regurgitation)</td>
<td>Femoral vein less favourable because of increased infection (intertrigo) and thrombosis risk in the obese</td>
</tr>
<tr>
<td><strong>Pulmonary artery catheter</strong></td>
<td>Central venous pressure Pulmonary artery pressure Pulmonary capillary wedge pressure Thermodilution CO SmvO2</td>
<td>CO estimation by PAC may be inaccurate in case of heart valve dysfunction (e.g. tricuspid and/or aortic valve regurgitation) PAC measurements less predictive with respect to global left ventricular function and preload conditions</td>
<td>Indexed haemodynamic measures are comparable for obese and non-obese</td>
</tr>
<tr>
<td><strong>Carbon dioxide rebreathing technique (NICO)</strong></td>
<td>CO (by Fick’s principle)</td>
<td>Only possible in mechanically ventilated patients</td>
<td>Accurate CO measurement depends on dead space ventilation and pulmonary shunts</td>
</tr>
<tr>
<td><strong>Echocardiography</strong></td>
<td>Cardiac output Flow parameters Anatomical data (dimensions, congenital defects and proximal great vessels) Ventricular systolic and diastolic function Heart valve anatomy and function Detection of myocardial ischaemia Pulmonary artery pressures Preload parameters Pericardial effusion Vegetation (Infective endocarditis) Neoplasm</td>
<td>Less suitable for continuous measurements TEE is advocated in case of (suspicion of) infective endocarditis TEE is not applicable in case of oesophageal / stomach diseases or severe coagulation disorders</td>
<td>Consider TEE in case of poor acoustic windows by TTE due to obesity</td>
</tr>
<tr>
<td><strong>Oesophageal Doppler</strong></td>
<td>Cardiac output Stroke volume Preload conditions Afterload conditions Cardiac contractility Stroke volume variation</td>
<td>CO measurement is highly depending on correct aortic diameter recording CO measurement is highly depending on proper positioning of Doppler probe</td>
<td>Indexed haemodynamic measures are comparable for obese and non-obese</td>
</tr>
</tbody>
</table>

CO = cardiac output; PAC = pulmonary artery catheter; TEE = transoesophageal echocardiography; TTE = transthoracic echocardiography.

**CO₂ rebreathing techniques**

CO₂ rebreathing techniques can be used as a non-invasive method for calculating CO. CO₂ rebreathing as a technique is based on the differential Fick equation and is applicable in mechanically ventilated patients. The precision of CO measurements in critically ill patients may vary by lack of haemodynamic stability, intrapulmonary shunting and other factors.
and dead space ventilation. Morbidly obese patients with pre-existing lung disease or postoperative atelectasis showed poor agreement of haemodynamic parameters derived from CO\textsubscript{2} rebreathing techniques when compared with the gold standard. Newly developed equipment, algorithms and software have improved the performance of these techniques. However, further developments and validations are needed.

**Echocardiography**

Echocardiography has proved to be of value in the management of patients with haemodynamic instability in the ICU as it may provide both anatomical and functional cardiovascular information. Transthoracic echocardiography (TTE) is often severely limited in obese patients because of poor acoustic windows. Dramatic improvements in ultrasound image quality have been achieved with the development of harmonic imaging. Harmonic imaging exploits the formation of ultrasound signals that return to the transducer at a multiple of the transmitted (fundamental) frequency, referred to as the harmonic frequency. Other enhancements include the use of contrast agents capable of producing left ventricle opacity from a venous injection to delineate the endocardial border. Several contrast agents are currently available that contain albumin microspheres filled with perfluorocarbon gas, allowing for passage of contrast through the lungs with appearance of contrast in the left ventricle.

Transoesophageal echocardiography (TEE) can provide detailed information in most obese patients in contrast to transthoracic echocardiography. TEE is particularly useful in critically ill patients with unexplained haemodynamic instability to rule out left and/or right ventricular failure, tamponade, hypovolaemia, and valvular dysfunction. Moreover, the presence of multiple indwelling catheters, the need for parenteral nutrition, and prolonged mechanical ventilation increase the likelihood of bacteraemia and subsequent endocarditis. Because the classical clinical findings associated with endocarditis are uncommon or difficult to assess in obese patients, echocardiography may facilitate the diagnosis of endocarditis, as part of the diagnostic process (Duke’s criteria).

**Oesophageal Doppler**

Pulsed Doppler techniques can be used to assess blood flow velocities. The oesophageal Doppler technique measures blood flow velocity in the descending aorta by means of a Doppler transducer (4 MHz continuous-wave, or 5 MHz pulsed-wave, according to manufacturers) placed at the tip of a flexible probe. Oesophageal Doppler, in essence, provides almost continuous haemodynamic information. Oesophageal Doppler measurements, however, are very sensitive to probe movement. This implies that this method can only be used in patients who are not moving much (i.e. in operating room settings or in well-sedated ICU patients).

The algorithms used to calculate CO and SV vary slightly according to manufacturers and are highly influenced by correct aortic diameter estimation, as is calculated by R square. The estimation of stroke volume using oesophageal Doppler relies on the measurement of stroke distance in the descending aorta (= velocity-time integral), which is then converted into systemic stroke volume. Oesophageal Doppler has the possibility to estimate SV variations in time (i.e. stroke volume variation) which may be indicative of fluid responsiveness and may guide fluid balance in sepsis and trauma patients.

**FUTURE TECHNIQUES**

**Impedance techniques**

Cardiac function may be non-invasively assessed by bio-impedance techniques. The theoretical basis for impedance cardiography is founded on Ohm’s law, in which the thorax is considered a cylinder with two major components: a poorly conductive cylindrical static tissue impedance surrounding a high conductive cylindrical blood resistance. After applying a constant, low-amplitude (0.5-4.0 mA), high-frequency (50-100 kHz), alternating electrical current to the thorax a voltage and time dependent induced voltage change can be measured. The voltage changes are used to calculate stroke volume, as well as CO, systemic vascular resistance and thoracic fluid content.

The accuracy of transthoracic bio-impedance technique depends on tissue, organ and fluid physics. These physical characteristics are summarised in the calculations by (assumed) constants. These assumptions are mostly derived from empirical geometric constructs in humans with average characteristics, including length and body weight. The pathophysiological assumptions, however, may not hold true in the obese patient, thereby invalidating SV measurements (e.g. due to differences in aortic compliance and differences in intrathoracic volume and content). The routine use of impedance cardiography for the measurement of CO in obese patients is still being questioned, despite attempts to improve the calculation algorithms. Alternative forms of bio-impedance techniques are being developed to improve accuracy (e.g., endotracheal CO monitoring). Future studies are needed to prove clinical application and benefit of these techniques in the critical care setting, including the care of the morbidly obese patient.

**Microcirculatory investigation**

Apart from systemic haemodynamic consequences, the microcirculation may be impaired in many disease or

Lagrand et al. Haemodynamic monitoring of obese ICU patients.
The microcirculation comprises the blood flow and perfusion of vessels smaller than 100 microns. Evaluation and modification of the microcirculation by variable means (e.g. near infrared spectroscopy; Sidestream darkfield imaging; orthogonal polarisation spectral imaging; gastric tonometry, sublingual capnometry) is the subject of future investigation. Whether this is relevant and/or beneficial in obese critically ill patients is still to be answered.

**Vascular Access**

Central venous access may be challenging in obese patients once the traditional anatomical landmarks are not that clear or even lacking. The increased skin-blood distance and the short, stubby neck in morbidly obese patients may make internal jugular and subclavian venous cannulation even more difficult. In line with this, more complications with central venous catheter placement are observed in obese patients (e.g. increased incidence of catheter malpositions and local puncture complications). These complications are associated with lower experience of operators and higher numbers of needle passes. In obese patients the number of skin punctures with catheter insertion attempts and delayed catheter changes have been shown to be related to more catheter-related infections and thrombosis. With respect to infection prevention, femoral venous access may be less desirable or even impossible because of severe intertrigo in the obese. Peripherally inserted central catheters can be used in the obese to measure central venous pressure and to ensure reliable vascular access. Peripheral vascular access in morbidly obese patients, however, may be problematic and, for example, may necessitate placement of a central venous line for administration of medication intravenously or to facilitate regular blood draws for diagnostic purposes.

Two-dimensional ultrasound can be used to localise and define the anatomy of central veins. Colour Doppler may be of help to differentiate between veins and arteries by variable means (e.g. near infrared spectroscopy; Sidestream darkfield imaging; orthogonal polarisation spectral imaging; gastric tonometry, sublingual capnometry) is the subject of future investigation. Whether this is relevant and/or beneficial in obese critically ill patients is still to be answered.

**Conclusions**

Haemodynamic monitoring of morbidly obese patients in the ICU may be technically difficult. Because of the increased body weight, the ICU physician may be confronted with unique, challenging problems in morbidly obese patients. With respect to haemodynamic monitoring most aspects are quite equal between obese and non-obese patients. The clinician, however, should be aware of the basic pathophysiological principles of the applied monitoring tools. Its theoretical assumptions and calculations may be invalidated because of the high body weight in obese patients. When indexed for body surface area (e.g., CO) or predicted lean body weight (e.g., extravascular lung water) reliable haemodynamic data are comparable between obese and non-obese individuals. Morbid obesity, therefore, should not complicate the interpretation of haemodynamic data.

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