Current Paediatrics (2004) 14, 459–464

ELSEVIER

Current
PAEDIATRICS

www.elsevierhealth.com/journals/cuoe

Understanding simple monitoring in paediatric intensive care

Jocelyn Hughes[a,*], Tariq Ali[b]

[a]*PICU, John Radcliffe Hospital Oxford, OX3 9DU, UK*
[b]*John Radcliffe Hospital, Oxford OX3 9DU, UK*

KEYWORDS
Intensive care;
Monitoring physiologic;
Oximetry;
Capnography;
Central venous pressure

Summary The development of reliable and affordable technology has made a significant impact in the care of the critically ill and it is now widely accepted that monitoring is a vital component in patient care. Arterial blood pressure can be measured by indirect and direct means although both can be subject to error in measurement. Arterial and central venous pressure provides numerical readings but the waveforms allow for further diagnostic interpretation. Pulse oximetry calculates oxygen saturation and encourages the clinician to think in terms of oxygen delivery, but can lead to errors with poor peripheral perfusion and with carboxyhaemoglobinaemia. Capnography gives an index of ventilation but further waveform analysis can be diagnostic of low cardiac output or bronchoconstriction. This review focuses on some of the practical uses and limitations of certain commonly used intensive care monitoring devices.

''It is what we think we know already that prevents us from learning'' (Claude Bernard).

Introduction

The Paediatric Intensive Care Unit (PICU) can be intimidating. Alarms ring, displays flash, leads and tubing snake around a small patient who almost disappears within the technology. The purpose of monitoring devices is to aid in patient assessment and to guide treatment. Often the array of available data can be bewildering and without some understanding of the potential pitfalls interpretation can be difficult and mistakes made. This review is to help improve understanding of some of the commonly used monitoring devices in Intensive Care by concentrating on their underlying principles and discussing some of the tricks and traps associated with their use.

Cardiac monitors

Non-invasive blood pressure

This is an indirect measurement. The manual method was first described by Nikolai Korotkoff in 1905. The more recent automated method is far more familiar on PICU.

Principles: the device for indirect non-invasive mean arterial pressure (Dinamap) is microprocessor controlled. The cuff inflates to above systolic pressure and then deflates incrementally whilst pulsations are detected by a transducer. Response time is about 30 s and systolic, diastolic and mean

*Corresponding author. Paediatric Intensive Care Unit, John Radcliffe Hospital, Headley Way, Headington, Oxford OX3 9DU, UK. Tel.: +44-1865-741166.
E-mail address: jossyhughes@aol.com (J. Hughes).

0957-5839/$ - see front matter © 2004 Elsevier Ltd. All rights reserved.
doi:10.1016/j.cupe.2004.05.009

pressures are usually displayed. The Dinamap has been validated in children.[1]

Traps: errors commonly arise from incorrect cuff size (narrow cuffs overestimate and vice versa). The American Heart Association recommends that the cuff be 40% of the midcircumference of the limb and then length must be twice the width.[2] Measurements are inaccurate with arrhythmias and episodes of low blood pressure. The thigh can be used in infants but may give higher readings.[3]

Invasive arterial pressure monitoring

Blood pressure can be measured directly via an intra-arterial catheter and electromechanical pressure transducer. This gives a beat-to-beat reading with great accuracy, even with arrhythmias and hypotension, and allows frequent non-traumatic blood gas analysis. Commonly the radial, femoral or dorsalis pedis arteries are cannulated, but the axillary, umbilical or brachial arteries can be used. The first direct measurement was by Reverend Stephen Hale in 1733; he inserted a brass tube into an artery of a horse and connected it to an upright glass tube. He noted that blood rose 8 feet above the level of the heart and rose and fell with each pulsation.[4]

Principles: the catheter tip is connected via a column of minimally compressible fluid (usually heparinized saline) to a remote pressure transducer. The column of saline compresses a diaphragm within the transducer and pulsations alter the diaphragm shape. Most pressure transducers are resistive bridge devices and produce changes in resistance and current, the signal is then amplified and processed digitally. The transducer must be zeroed to atmospheric pressure before use.

Tricks: apart from blood pressure and gas analysis the arterial waveform can be used to derive far more information about the patient's haemodynamic status. Myocardial contractility can be assessed from the gradient of the arterial upstroke (dP/dT), providing the aortic valve is normal; the steeper the upstroke the more contractile the myocardium and vice versa. The area beneath the curve up to the dicrotic notch gives an index of stroke volume.

The downstroke of the curve gives information about resistance and compliance of the vascular bed. Low blood pressure with a low dicrotic notch and a steep downstroke is associated with peripheral vasodilatation (e.g. sepsis). Low blood pressure with a high dicrotic notch and a slowly decaying downstroke is characteristic of vasoconstriction, and is usually associated with hypovolaemia or vasoconstrictive drugs. There is a swinging variation in systolic pressure seen with intermittent positive pressure ventilation (IPPV), which is due to a reduction in right ventricular preload.[5] This 'pulsus paradoxus' has been shown to correlate well with hypovolaemia[6] and is a useful tool in PICU.

Traps: the transducer should be positioned at the level of the right atrium and must be changed when the patient's position is changed. Damping may occur and underdamping may lead to an overestimate of systolic pressure,[7] alternatively the system can be overdamped, usually as a result of air bubbles trapped in the circuit or because the tubing used is too compliant. High ventilatory pressures and air trapping can cause a 'swinging trace'[8] and should be considered before treating presumed hypovolaemia.

The contour of the arterial waveform changes as the monitoring site becomes more distal. When more distal sites are used the wave becomes steeper and the size can increase, giving overestimates of systolic pressure. Generally the mean arterial pressure stays true (Figs. 1–3).

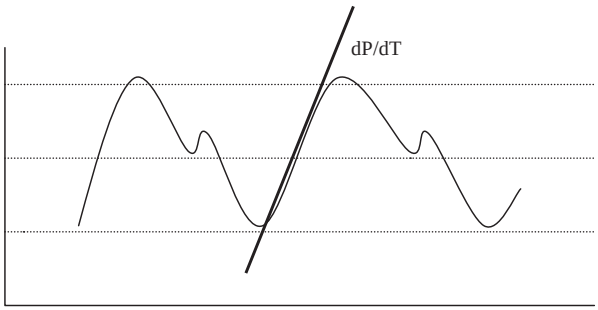

Figure 1 Normal arterial waveform. Myocardial contractility can be estimated from the gradient of the upstroke dP/dT.

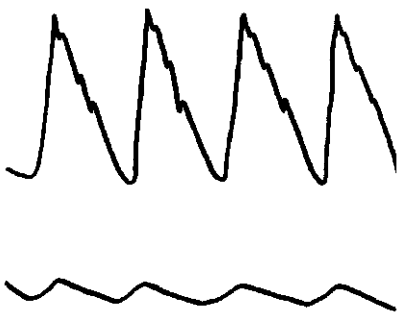

Figure 2 The upper trace shows an underdamped arterial wave; the lower trace is overdamped.

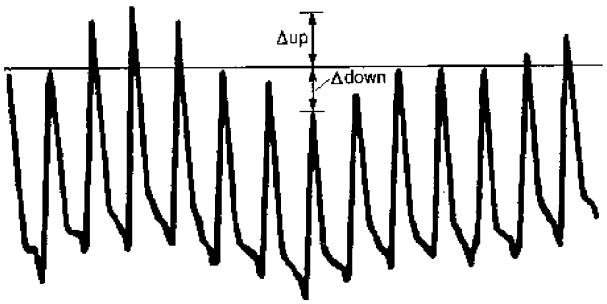

Figure 3 A swinging arterial trace-demonstrating the typical dip in systolic pressure with a positive pressure breath associated with hypovolaemia.

Central venous pressure (CVP)

Although CVP can be measured with a simple saline filled manometer it is more common in PICU to use the same electromechanical pressure transducers as with intra-arterial monitoring. Sites favoured are the internal jugular and femoral vein, although the subclavian vein can also be used. Generally the tip of the catheter lies near the right atrium or in the inferior vena cava. All three sites give generally reliable and consistent results.[9]

The aim of CVP monitoring is to give an estimate of right atrial pressure and thus right ventricular end-diastolic pressure (assuming a normal tricuspid valve), consequently the pressure transducer must be zeroed at the right atrium. CVP is mostly used as a marker for intravascular volume status (filling pressure), but it also varies with right heart function, venous tone, intrathoracic pressure and position of the patient (head-up decreases the CVP, whilst head-down increases it).

Tricks: normal values for CVP are hard to define. Healthy babies have a CVP of -2 to $+4$ mmHg, whereas ventilated infants are usually higher at 4–8 mmHg. Isolated readings are of little value in PICU and the response of the CVP to a fluid challenge reveals far more: a young hypovolaemic patient may have a normal CVP due to compensatory venoconstriction (not so common in babies and infants with less developed sympathetic responses). A fluid challenge will allow the cardiac output to rise and venodilatation to occur, leading to a low CVP more in keeping with hypovolaemia. In contrast an elevated and sustained CVP in response to fluid primarily reflects poor heart function.

The normal CVP trace has 3 waves: a, c and v. The a waves represent atrial contraction, the c wave is caused by ventricular contraction against a closed tricuspid valve and the v wave represents atrial filling (Table 1).

Table 1 Characteristics of abnormal CVP waves.

Cannon a waves	Complete heart block, pulmonary hypertension, pulmonary stenosis
Absent a wave	Atrial fibrillation
Giant v wave	Tricuspid regurgitation

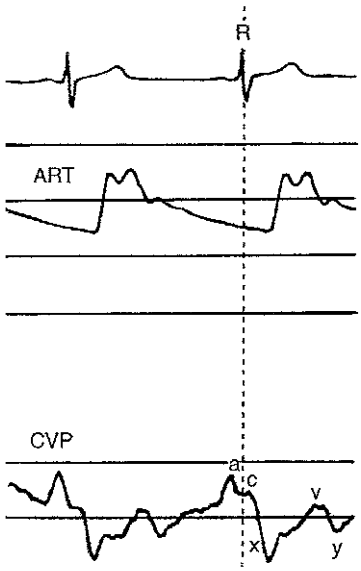

Figure 4 Simultaneous ECG, arterial and CVP waveforms.

Traps: changes in CVP occur with ventilation, as this leads to changes in intra thoracic pressure. Normal inspiration produces a fall in CVP, and IPPV generally produces a rise in mean CVP. Positive end-expiratory pressure (PEEP) may also increase the CVP.[10] It is usual to measure CVP at the end of expiration. CVP does not give a reliable guide to left atrial pressure when there is disparity between the function of the right and left heart, such as in heart or lung disease when pulmonary hypertension is present (Fig. 4).

Respiratory monitors

Pulse oximetry

The poor reliability of cyanosis in the recognition of arterial hypoxaemia was first described in 1947.[11] The advent of pulse oximetry in the 1980s was a major medical advance and has widespread use in modern hospitals. In 1851 Auguste Lambert

described the Beer–Lambert law that states that the concentration of an unknown solute in a solvent can be determined by the amount of light it absorbs. If this is applied to oxygenated and deoxygenated haemoglobin then the relative amounts of each can be calculated.

Principles: two wavelengths of light (660 and 940 nm) which have different absorption spectra for oxygenated and deoxygenated haemoglobin are transmitted from light emitting diodes through an arterial bed, such as a finger, toe or ear lobe. A microprocessor compares the absorption of the two wavelengths and the pulsatile and background absorption, giving a ratio of oxygenated to deoxygenated haemoglobin. A voltage proportional to transmitted light passes through a microprocessor and is converted to a waveform, which is displayed with data for pulse rate and oxygen saturation.

Tricks: pulse oximeters are simple to use and non-invasive, giving a continuous reading (with some delay). Oxygen saturation (SaO_2) is far more useful in estimating oxygen delivery than partial pressure of oxygen (pO_2). It is useful to think in terms of oxygen delivery, as maintenance of organ oxygenation is the cornerstone of so much intensive care treatment

$$\text{Oxygen delivery} = \text{cardiac output} \times \text{oxygen content of blood},$$

$$\text{Oxygen content} = SaO_2(\%/100) \times \text{haemoglobin} \times 1.31 + \text{dissolved oxygen}.$$

(The figure 1.31 is the amount of oxygen in millilitres that combines with 1 g of haemoglobin.)

Dissolved oxygen is generally negligible, thus[12]

$$\text{Oxygen delivery} = \text{cardiac output} \times SaO_2/100 \times Hb \times 1.31.$$

As you can see oxygen delivery can be estimated easily, and if cardiac output and haemoglobin concentration are adequate SaO_2 can be reduced to 90% without compromising oxygen delivery too much. This allows one to confidently reduce aggressive and potentially damaging ventilatory parameters or levels of FiO_2 whilst maintaining oxygen delivery, without repetitive blood gas sampling.

Traps: pulse oximeters have been calibrated against healthy volunteers and have a high potential for error at low saturations[13]—obviously the manufacturers could not induce severe hypoxia in volunteers. This should be borne in mind in regard to children with low SaO_2 from congenital heart disease. If in doubt an accurate fractional saturation can be obtained from most modern gas machines with built in co-oximeters.

Both carboxyhaemoglobin (from smoke inhalation or fumes) and methaemoglobin (from nitric oxide use) can falsely elevate SaO_2 and thus mask hypoxaemia. Again, co-oximetry can exclude these dyshaemoglobinaemias. Poor peripheral perfusion,[14] nail varnish, high ambient light and motion artefact can also be sources of error in pulse oximetry.

Capnography

This is a continuous non-invasive measurement of expired carbon dioxide tension. Although initially developed in the 1950s capnography was mainly a research tool. Technical improvements have led to the widespread use of capnography in anaesthesia and its increasing use in intensive care. End-tidal carbon dioxide ($E'CO_2$) is defined as the peak CO_2 value during the expiratory phase of ventilation. $E'CO_2$ approximates to arterial pCO_2 in patients

Table 2 Conditions that alter $E'CO_2$.

Increases in $E'CO_2$	Increased production ie fever, catabolism, malignant hyperthermia Hypoventilation
Decreases in $E'CO_2$	Decreased production ie hypothermia Decrease in cardiac output Pulmonary embolism Air embolism Decrease in pulmonary perfusion ie right to left shunt
Absent $E'CO_2$	Oesophageal intubation Disconnection from ventilator

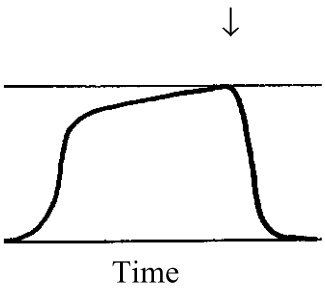

Figure 5 A normal capnograph trace, the arrow indicates the end tidal value.

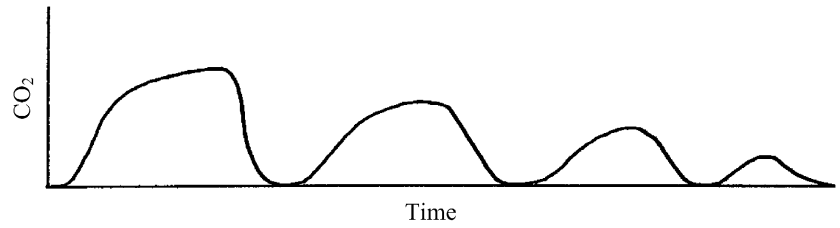

Figure 6 A decrease in the size of successive capnograms is seen with a rapid decrease in pulmonary perfusion secondary to sudden hypotension or cardiac arrest.

with normal heart and lungs, thus providing a useful non-invasive index of ventilation. Again, examination of the phasic changes in capnography reveal considerably more information. Capnography gives the only sure guarantee that the trachea has been intubated successfully. The capnograph is a mandatory requirement in all operating theatres and there are compelling reasons why this should also apply to all intensive care environments.

Principles: generally capnography relies on the principle of infrared spectroscopy—that every gas has unique absorption characteristics to infrared light, which can be used to quantify the amount (partial pressure) of a particular gas. Thus a capnograph consists of a sample chamber through which infrared light is shone, and a photodetector on the opposite side which creates a voltage proportional to the partial pressure of CO_2 in the chamber. The capnograph is usually placed between the endotracheal tube and the expiratory limb of the ventilator tubing; the sample chamber can be in-line or a sidestream sampler can be used. Inspired and expired CO_2 form a square wave capnogram that has been described as a snake that has swallowed an elephant. Generally, respiratory rate and $E'CO_2$ are displayed (Table 2).

Tricks: as a non-invasive measure of pCO_2 capnography can monitor the adequacy of alveolar ventilation during IPPV and when breathing spontaneously. Ventilation can be monitored continuously and the ventilator adjusted as necessary. This is particularly useful when weaning off a ventilator, since the efficiency of ventilation can be estimated, and patient–ventilator synchrony can be improved according to the capnogram. Generally the need for frequent blood gas sampling can be avoided. $E'CO_2$ is particularly valuable as an indicator of ventilator disconnection (the capnogram trace disappears). A drop in $E'CO_2$ can also signal an accidental bronchial intubation. A fall in pulmonary perfusion leads to a fall in $E'CO_2$ (and an increase in the difference between measured pCO_2 and $E'CO_2$), and may signify low cardiac output or the occurrence of a pulmonary or air emboli. There

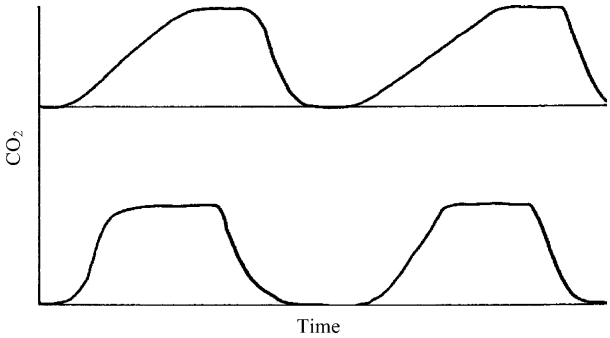

Figure 7 The slow rise of the capnogram in the upper panel is seen with bronchospasm. The improved capnogram in the lower panel is after bronchodilator therapy.

is also a distinctive capnogram seen in obstructive airways disease as different lung units empty at different rates.

Traps: right to left intracardiac shunts produces an 'apparent dead space' and lead to a discrepancy between pCO_2 and $E'CO_2$—the pCO_2 rises above the $E'CO_2$ (Figs. 5–7).[15]

Conclusion

Monitoring in PICU is intended as an aid to patient assessment and to guide treatment. Without an understanding of the principles involved it is easy to misinterpret some of the available data. Additionally, with scrutiny and insight there is often more information available than initially meets the eye.

References

1. Park M, Menard S. Accuracy of blood pressure measurements by the Dinamap monitor in infants and children. *Pediatrics* 1987;**79**:907–14.
2. Kirkendall W, Feinleib M, Freis E, Washington D, Allyn L. Recommendations for human blood pressure determination by sphygmomamometer. *Circulation* 1980;**62**:1146A–55A.

3. Pascarelli E, Bertrand C. Comparisons of blood pressures in the arms and legs. *N Engl J Med* 1964;**270**:693.
4. Pickering G. Systemic arterial hypertension. In: Fishman A, Richards D, editors. *Circulation of the blood—men and ideas*. New York: Oxford University Press; 1964. p. 487–541.
5. Morgan BC, Martin WE, Hornbein TF, Crawford EW, Guntheroth WG. Haemodynamic effects of intermittent positive pressure ventilation. *Anesthesiology* 1966;**27**:584–90.
6. Coyle CP, Teplick RS, Long MC, Davison JK. Respiratory variations in systemic arterial as an indicator of volume status. *Anesthesiology* 1983;**59**:A53.
7. Rothe CF, Kim KC. Measuring systolic arterial blood pressure. *Crit Care Med* 1980;**8**:683–9.
8. Perel A, Segal E. Systolic pressure variation—a way to recognize dynamic hyperinflation. *Br J Anaesth* 1996;**76**: 168–9.
9. Chait HI, Kuhn MA, Baum VC. Inferior vena caval pressures predict right atrial pressure in pediatric cardiac surgical patients. *Crit Care Med*. 1999;**27**:219–24.
10. Pinsky M, Vincent J-L, De Smet J-M. Estimating left ventricular filling pressure during positive end-expiratory pressure in humans. *Am Rev Respir Dis* 1991;**143**:25–31.
11. Comroe JH, Botelho S. The unreliability of cyanosis in the recognition of arterial anoxaemia. *Am J Med Sci* 1947; **214**:1–6.
12. Lumb AB. Quantification of oxygen delivery. In: *Nunn's applied respiratory physiology*. Butterworth-Heinemann; 2000. p. 284.
13. Severinghaus JW, Naifeh KH, Koh SO. Errors in 14 pulse oximeters during profound hypoxia. *J Clin Monit* 1989;**5**: 72–81.
14. Clayton DG, Webb RK, Ralston AC, Duthie D, Runciman WB. A comparison of the performance of twenty oximeters under conditions of poor perfusion. *Anaesthesia* 1991;**46**:3–10.
15. Fletcher R. The relationship between the arterial to end-tidal CO_2 difference and haemoglobin saturation in patients with congenital heart disease. *Anesthesiology* 1991;**75**: 210–6.