Non-invasive continuous haemodynamic and $PETCO_2$ monitoring during peroperative cardiac arrest

Pierre-Yves Gueugniaud MD,* Raoul Muchada MD,†
Marc Bertin-Maghit MD,* Nia Griffith MD,*
Paul Petit MD*

We describe a cardiac arrest which occurred during general anaesthesia in the prone position for surgical correction of lumbar kyphosis in a patient with Marfan's syndrome. Peroperative monitoring was routine with ECG, non-invasive arterial pressure, oximetry, $PETCO_2$ and central venous pressure, plus aortic blood flow and systolic time intervals via an oesophageal echo-Doppler device. Forty-five minutes after the start of surgery, a sudden decrease in aortic blood flow followed by a decrease in $PETCO_2$ suggested acute cardiac failure despite continuation of the ECG signal. Initial CPR in the prone position produced a slight increase in $PETCO_2$. When the patient was turned to the supine position and the legs elevated, chest compression was more efficient and spontaneous circulation was rapidly restored. Circulatory arrest could be explained by incompletely treated hypovolaemia, or by myocardial depression (decrease in aortic blood flow and lengthened pre-ejection period) combined with excessive hypotension in a patient with Marfan's syndrome, thus compromising coronary blood flow producing ST segment depression. Continuous non-invasive aortic blood flow and $PETCO_2$ monitoring proved valuable in the early detection and treatment of circulatory arrest and in the evaluation of the efficiency of peroperative CPR.

Nous rapportons un cas d'arrêt cardiaque per-anesthésique chez un jeune homme de 15 ans, porteur d'un syndrome de Marfan, survenant en décubitus ventral au cours de la chirurgie correctrice d'une cyphoscoliose lombaire. La surveillance hémodynamique per-opératoire comportait outre l'ECG, la pression artérielle non invasive, la pression veineuse centrale et la pression télé-expiratoire de CO_2 ($PETCO_2$), le monitorage en continu par écho-Doppler oesophagien du débit aortique et des intervalles de temps systolique. Quarante-cinq minutes après le début de l'intervention, la baisse progressive et rapide du DA, suivie par la chute de la $PETCO_2$ a mis en évidence précocement l'apparition d'une défaillance cardio-circulatoire, malgré la persistence d'une activité électrique cardiaque. La réanimation cardio-pulmonaire initiale, réalisée en position ventrale sur le cadre de Hall a donné comme résultat initial une très discrète augmentation de la $PETCO_2$. La mise en décubitus dorsal, la surélévation des jambes, et un massage cardiaque externe réalisé dans les conditions normales ont permis alors de récupérer rapidement de l'arrêt circulatoire. L'arrêt circulatoire peut être expliqué soit par une hypovolémie masquée, soit par l'association d'une dépression myocardique (débit aortique diminué, période de pré-éjection allongée) et d'une hypotension trop importante pour un patient porteur d'un syndrome de Marfan et pouvant induire une perfusion coronaire insuffisante (sous-décalage du segment ST). L'aide diagnostique apportée par la surveillance hémodynamique non invasive et par la capnographie en continu s'est avérée fondamentale pour diagnostiquer l'arrêt circulatoire, pour débuter rapidement les gestes de réanimation, et pour surveiller l'efficacité de la réanimation cardio-pulmonaire au bloc opératoire.

Key words
HEART: asystole;
MONITORING: End-tidal carbon dioxide, aortic blood flow, Doppler.

From the *Department of Anaesthesiology. Edouard Herriot Hospital, University of Lyon 1, 3 place d'Arsonval, 69437 Lyon, France and the †Department of Anaesthesiology and Critical Care, Clinique Eugène André, 107 rue Trarieux, 69003 Lyon, France.

Address correspondence to: Dr. P.Y. Gueugniaud, S.A.R. VII (Pavillon I), Hôpital Edouard Herriot, 3 place d'Arsonval, 69437 Lyon Cedex 03, France.

Accepted for publication 2nd June, 1995

Haemodynamic monitoring is essential during the management of critical or unstable patients. The monitoring devices usually provide only intermittent data unless invasive monitoring is used. We present a patient in whom continuous non-invasive monitoring with a combination of aortic blood flow (ABF) and $PETCO_2$ allowed the diagnosis and management of a peroperative cardiac arrest (CA).

Case report

A 15-yr-old boy ($55 \cdot kg^{-1}$, 1.93 m) with a diagnosis of Marfan's syndrome, was scheduled for surgical correction of a right dorsal and left lumbar scoliosis. Preoperative cardiovascular status revealed a blood pressure of 150/80 mmHg and a heart rate (HR) of 75 bpm. A grade I cardiac murmur compatible with aortic regurgitation was present. An echocardiogram showed a small aneurysmal dilatation of the ascending aorta and mitral valve prolapse without mitral regurgitation. Preoperative biochemistry was normal.

After premedication with hydroxyzine 25 mg and alprazolam 0,5 mg *po* general anaesthesia was induced with fentanyl 3 $\mu g \cdot kg^{-1}$ and propofol 1.5 $mg \cdot kg^{-1}$ *iv*, and maintained with isoflurane/N_2O ($FiO_2 = 0.4$) and fentanyl 2 $\mu g \cdot kg^{-1}$ every 20 min. Vecuronium, 0.07 $mg \cdot kg^{-1}$ was given to facilitate tracheal intubation, and to allow controlled ventilation ($f = 12 \cdot min^{-1}$; $V_T = 8$ $ml \cdot kg^{-1}$; I:E = 1:2). Central venous monitoring was via a right internal jugular line. Controlled arterial hypotension was achieved by progressive increments of isoflurane from 0.8% to 1.5% inspiratory concentration. The patient was then placed in the prone position on a Hall support.[1] Homologous autotransfusion and cell-saver blood collection were commenced. Non-invasive continuous monitoring including the following variables was recording: $PETCO_2$ by means of an infrared absorption capnograph (Normacap 200, Datex, Finland). Electrocardiogram (ECG) (ITS104, CGR, France); systolic, diastolic and mean arterial pressure (MAP) were monitored by digital plethysmography (Finapress 2300, Ohmeda, USA); continuous monitoring of arterial oxygen saturation (Satlite Trans, Datex, Finland).

Non-invasive haemodynamic monitoring was performed with an echo-Doppler aortic blood flow meter (prototype INSERM U 281 actually produced by Sometec - Dynemo®). This device allows measurement of the aortic diameter and blood velocity at the same anatomical level continuously and non-invasively. The measurement involves an oesophageal probe with a water-filled latex balloon surrounding the ultrasound transducer. The balloon maintains a constant angle of incidence of the ultrasound beams, allowing the captors to rotate freely against the oesophageal mucosal wall. It ensures the transmission of ultrasound waves without air interposition and dissipates the heat produced. The 10 mHz echo bidimensional scan and the 5 mHz pulsed Doppler system makes it possible to obtain a very clear image resolution. The aortic flow meter connected by a satellite device to visualise the haemodynamic profile is presented in table form and revised every eight seconds and recorded on soft magnetic support. This haemodynamic profile integrates ABF, MAP, HR, and calculates stroke volume (SV) and total systemic vascular resistance (TSVR). Moreover, systolic time intervals are measured from computerised analysis of the ECG signal (Q wave detection) and the acceleration signal which is derived from the Doppler velocity signal. Opening and closing of the aortic valve is detected by observation of the acceleration signal. Pre-ejection period (PEPi) and left ejection ventricular time (LVETi) are then measured automatically and continuously. PEPi and LVETi are indexed to HR. PEP/LVET ratio is also calculated automatically. The monitoring of the $PETCO_2$ together with the haemodynamic profile provides simultaneous analysis of the variation of ABF and $PETCO_2$ and allows early detection of alterations in the tissue or pulmonary perfusion.

After 30 min of controlled hypotension (MAP = 60 mmHg), the ECG showed ST segment depression >1 mm, followed by low ABF ($0.9\ L \cdot min^{-1}$) and a decrease in $PETCO_2$ (25 mmHg) without any change of HR ($110 \cdot min^{-1}$), CVP (12 cm H_2O) or oxygen saturation (99%). The N_2O and isoflurane were stopped immediately. A dobutamine infusion ($5\ \mu g \cdot kg^{-1} \cdot min^{-1}$) was started and 500 ml gelatin plasma substitute were given over the ten minutes preceding the cardiac arrest. Ringer lactate 1500 ml had already been given (blood loss measured through the cell-saver was 620 ml and autotransfusion, 220 ml). Nevertheless, the arrest occurred about 15 min after the recorded ST depression: pulseless electromechanical dissociation was immediately followed by asystole (Figure).

With the patient still in the prone position, cardiopulmonary resuscitation (CPR) was attempted. Chest compressions were started underneath the Hall support while the surgeons closed the operative field. During CPR in the prone position, the carotid artery pulse was not palpable and the $PETCO_2$ only increased to 10 mmHg. Adrenaline 2 mg, calcium chloride 0.5 g, and sodium bicarbonate 50 mEq, were given *iv* before the patient was turned to the supine position and the legs elevated. Spontaneous circulation diagnosed by increasing ABF and $PETCO_2$ was restored a few minutes after the patient was returned to the supine position (ten minutes after the arrest) (Figure).

Surgery was postponed and the patient transferred to the ICU where the haemodynamic status remained stable without vasopressors. He made a full recovery without neurological sequelae and the trachea was extubated 18 hr later. Twenty-four hours later, echocardiography demonstrated inferoapical akinesia, probably linked with temporary myocardial ischaemia. Repeated measurement one week later was similar to the preoperative data.

Discussion

This ASA 3 patient was scheduled for a complicated sur-

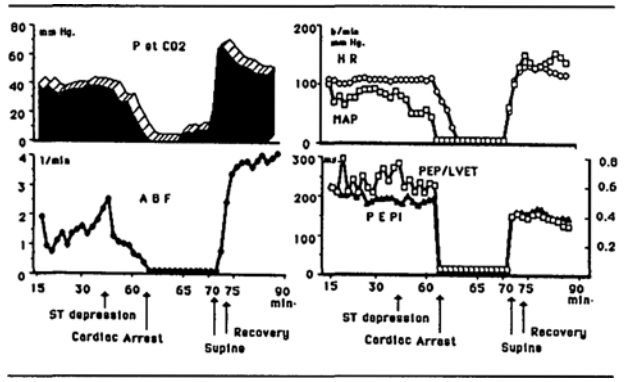

FIGURE Evolution of $PETCO_2$, aortic blood flow (ABF), systolic time intervals (STI) through pre-ejective period index (PEPi) and left ventricular ejection time index (LVETi), heart rate (HR), and mean arterial pressure (MAP) before cardiac arrest (CA) and during cardiopulmonary resuscitation (CPR).

gical procedure, which in our institution is generally performed using controlled arterial hypotension, using halogenated anaesthetic agents: we have had no morbidity until this present case. The possibility of severe blood loss and major fluid replacement means that careful haemodynamic monitoring is essential. Despite strict vigilance, the patient suffered acute myocardial damage which was displayed by ST change and followed by cardiovascular collapse and cardiac arrest. This cardiovascular failure could be explained by various mechanisms. Firstly, hypovolaemia is one of the most important causes of electromechanical dissociation and worsens mitral valve prolapse: incompletely treated hypovolaemia could not be excluded in this case. Secondly, the contribution of Marfan's syndrome should be considered. The patient sustained myocardial ischaemia as demonstrated by ST segment depression and immediate postoperative echocardiography. In this case, arterial hypotension produced by isoflurane may have reduced coronary perfusion. Indeed, myocardial failure was suspected with the prolonged PEPi and initially low ABF. While Marfan's syndrome is not associated with coronary anomalies, the mitral valve prolapse may have been involved in the myocardial disturbance. The other common complications and potential causes of cardiac arrest associated with Marfan's syndrome, such as aortic dissection or rupture, or spontaneous pneumothorax from lung cysts were also subsequently excluded. Thirdly the aortocaval compression whilst lying prone on the Hall support might have played a major role in the initiation of arrest. Restoration of spontaneous circulation occurred only when the patient was turned supine suggesting that the positioning and the severe kyphoscoliosis might also have made this hypotensive patient susceptible to aortocaval compression. Knowing the previous variations in ABF and PEPi, sudden indirect circulatory causes as venous air embolism could be rejected.

The usefulness of the transoesophageal Doppler-echographic technique for continuous measurement of ABF in the descending aorta in various clinical situations has been reported.[3-5] It correlates well with cardiac output measurement (thermodilution and electromagnetic flow).[3-4] The $PETCO_2$ may reflect changes in ventilation, metabolic and tissue perfusion ("wash out" of CO_2).[6] This variable correlates with cardiac output,[7] and acute changes in $PETCO_2$ under stable ventilatory and metabolic conditions must be attributed to changes in tissue perfusion.[6,8] Variation in $PETCO_2$ is a good marker of tissue perfusion.[9] In this patient, the $PETCO_2$ and ABF curves showed an early, marked and consistent diminution (whereas HR remained stable), as previously described.[8,10,11] The absence of expired CO_2 during cardiac arrest, even when the patient's lungs were being ventilated, suggests that there was no tissue or pulmonary perfusion.

As the patient was still on the Hall support in the prone position, CPR was initially attempted under unfavourable conditions. Despite that, there was a small increase in $PETCO_2$, reaching a plateau of about 10 mmHg. This suggests that, in spite of technically unmeasurable ABF during chest compression and lack of plethysmographic recording of arterial pressure, CPR was effective. Monitoring $PETCO_2$ during cardiac arrest has been described previously.[12-14] Improvement in the ABF and the $PETCO_2$ curves were observed coinciding with the restoration of spontaneous circulation. The high levels of $PETCO_2$ reached could correspond with the wash out of accumulated CO_2 in non-perfused tissues.[7,10-14] They could also have been due to bicarbonate metabolism. The $PETCO_2$ changes observed may be taken as a good prognostic sign.[10,12]

In conclusion, this case illustrates the importance of ABF and $PETCO_2$ monitoring in evaluating the efficiency of peroperative CPR. Moreover, continuous ABF monitoring is likely to be helpful in the peroperative management of critically ill patients during anaesthesia and may allow early diagnosis of myocardial depression based on STI changes and early detection of circulatory collapse when ABF decreases simultaneously with $PETCO_2$. This report also outlines that resuscitation manoeuvres should be instituted promptly even under difficult patient positioning when the recommended international basic CPR guidelines could not be immediately applied.

References

1 *Relton JES, Hall LE.* An operation frame for spinal fusion. A new apparatus designed to reduce haemorrage during operation. J Bone Joint Surg Br 1967; 49B: 327–32.

2 *Muchada R, Vernier F, Fady J-F, Haro D, Lavandier B, Cathignol D.* A new automatic measurement of systolic time interval (STI). J Cardiothorac Vasc Anesth 1992; 6 (suppl.1): 22.
3 *Lavandier B, Muchada R, Chignier E, Fady JF, Birer A, Cathignol D.* Assessment of a potentially noninvasive method for monitoring aortic blood flow in children. Ultrasound Med Biol 1991; 17: 107–16.
4 *Muchada R, Rinaldi A, Cathignol D.* Monitorizzazione non invasiva del profilo emodinamico durante l'anestesia generale. Minerva Anestesiol 1990; 56: 199–205.
5 *Gueugniaud PY, Caritat R, Barreiro G, Muchada R, Bertin-Maghit M, Petit P.* Surveillance hémodynamique non invasive chez les brûlés: faisabilité et intérêt de la débitmétrie aortique. Réanimation Urgences 1993; 2: 533–8.
6 *Brunel D, Muchada R.* Evaluation de la perfusion tissulaire par surveillance simultanée du débit aortique et de la capnographie. Presse Med 1991; 20: 1665–6.
7 *von Planta M.* Noninvasive monitoring of blood flow during cardiac arrest and cardiopulmonary resuscitation with $PETCO_2$. *In*: Mocavero G, Gullo A, Palandini VA, Romano E (Eds.). Recent Advances in Anaesthesia, Pain, Intensive Care and Emergency. Trieste: A.P.I.C.E. 1989; 359–63.
8 *Bourgain JL, Benlabed M, Tandonnet F.* Etude critique de l'intérêt de la capnigraphie en anesthésie. Ann Fr Anesth Reanim 1987; 6: 100–6.
9 *Brunel D, Muchada R, Vernier F, Fady JF, Cathignol D.* Variation du CO_2 téléexpiratoire ($PetCO_2$) et des paramètres hémodynamiques. Nouvelles approches de la perfusion tissulaire. Ann Fr Anesth Reanim 1991; 10: R176.
10 *Krivosic-Horber D.* Place de la capnographie dans le monitorage anesthésique. Ann Fr Anesth Reanim 1989; 8: 175–81.
11 *Weil MH, Bisera J, Trevino RP, Rackow EC.* Cardiac output and end-tidal carbon dioxide. Crit Care Med 1985; 13: 907–9.
12 *Falk JL, Rackow EC, Weil MH.* End-tidal carbon dioxide concentration during cardiopulmonary resuscitation. N Engl J Med 1988; 318: 607–11.
13 *Gudipati CV, Weil MH, Bisera J, Deshmukh HG, Rackow EC.* Expired carbon dioxide: a noninvasive monitor of cardiopulmonary resuscitation. Circulation 1988; 77: 234–9.
14 *Rozenberg A, Carli PA, Bousquet M, Lamour O, Barrier G.* End tidal CO_2 monitoring during prehospital cardiopulmonary resuscitation. Anesthesiology 1990; 73: A531.