

## Correspondence

# Significance of continuous blood gas monitoring in cardiac surgery with cardiopulmonary bypass

### EDITOR:

Standard non-invasive monitoring during anaesthesia usually includes pulse oximetry saturated pressure of oxygen ( $S_pO_2$ ) and end-tidal carbon dioxide (end-tidal  $CO_2$ ). They frequently fail to monitor blood gas changes during cardiac surgery due to reduction in perfusion, ambient light, hypothermia and the use of vasoconstrictors [1]. It is also impossible to monitor  $S_pO_2$  during cardiopulmonary bypass (CPB) because of the lack of pulsatile flow. Intermittent blood gas monitoring, the rule in cardiac surgery, often fails to detect the rapid changes that are frequent in the beginning and end of bypass. Continuous blood gas monitoring with the Paratrend 7 (Diametrics, Manchester, UK) could therefore be an interesting option. The aim of this prospective observational study was to compare conventional intermittent blood gas monitoring with the continuous Paratrend system in patients undergoing cardiac surgery with bypass.

The study was approved by the hospital Ethics Committee. Ten consecutive patients (age  $70 \pm 5$  yr) were included with written informed consent. Anaesthesia was induced with etomidate, midazolam and sufentanil with cisatracurium or rocuronium for muscle relaxation. Anaesthesia was maintained with isoflurane. The inspired oxygen fraction during anaesthesia was 50% and this increased if needed to keep  $S_pO_2 \geq 97\%$ . Monitoring included invasive blood pressure (BP), eight-lead ECG, central venous pressure (CVP),  $S_pO_2$  and end-tidal  $CO_2$ . Blood gases were assessed intermittently in the laboratory and continuously with the Paratrend 7 system.

Two anaesthetists took part in the study. One was responsible for the standard patient care according to the departmental guidelines. He had access to the conventional blood gas analysis,  $S_pO_2$  and end-tidal

$CO_2$ . However, he was blinded to the Paratrend data. A second anaesthetist monitored the continuous Paratrend blood gas data. Abnormal Paratrend data were classified as Level 1 (abnormal, but not life-threatening values) or Level 2 (dangerous). Level 1 and 2 limits are defined in Table 1. Level 2 abnormalities, with the exception of arterial pressure of oxygen ( $PaO_2 \geq 26$  kPa), were immediately communicated to the anaesthetist in charge of patient care. The number of therapeutic actions performed on the basis of the results of conventional blood gas analysis,  $S_pO_2$  and end-tidal  $CO_2$  were noted.

An average of 6.6 blood gas values per patient was outside the predefined limits. The mean duration of Level 1 and 2 abnormalities for arterial pressure of oxygen and carbon dioxide ( $PaO_2$  and  $PaCO_2$ , respectively), and pH in absolute time and as a percentage of operating time are shown in Table 1.

The main abnormalities found with the Paratrend system were hyperoxaemia and hypocapnia. These deviations were also discovered by intermittent blood sampling but with a significant delay. According to the Paratrend data, the patients were hyperoxaemic >31% of the operating time.  $PaO_2$  often exceeded 26 kPa (average duration 57 min). Hyperoxaemia has been implicated in microcirculatory deterioration [2] and exacerbation of the inflammatory process post-bypass [3].

The risk of hypoxaemia is substantial during cardiac surgery with CPB. It can be due to the patient's condition or to mechanical failure. However, episodes of intraoperative hypoxaemia found with the Paratrend were much less frequent than hyperoxaemia, representing only 8% of operating time. These episodes were revealed with a delay by traditional intermittent blood gas sampling, requiring the blind to be lifted on five occasions (Table 1). It seems that patients are voluntarily maintained hyperoxaemic, as anaesthetists fear hypoxaemia more than hyperoxaemia.

The information provided by pulse oximetry played only a minor part in the management of the patients. In 10% of operating time (bypass excluded), the  $S_pO_2$

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Table 1. Blood gas abnormalities recorded with Paratrend 7 system.

Parameter	Abnormalities	Level 1 limits	Duration (min)	Duration (%)	Level 2 limits	Blind lifting (n)
PaO <sub>2</sub> (kPa)	Hyperoxaemia	>20	106 ± 91	33.7 ± 27.4	>26	27*
	Hypoxaemia	8–10	26 ± 25	8.3 ± 8.3	<8	5
PaCO <sub>2</sub> (kPa)	Hypercapnia	6–6.6	17.4 ± 22	5.5 ± 9.2	>6.6	2
	Hypocapnia	4–4.6	162 ± 93	51.5 ± 31.3	<4	24
pH	Acidosis	7.30–7.35	47.9 ± 85	15.2 ± 28.2	<7.30	8
	Alkalosis	7.45–7.50	153.6 ± 113	48.8 ± 37.2	>7.50	10

The duration of values outside Level 1 limits is given as absolute time and as percentage of the operative time. Values are mean ± standard deviation.

\*No blind lifting.

signal was not reliable. Anaesthetists tend to act only in cases of low SpO<sub>2</sub>. In our patients SpO<sub>2</sub> never fell below 95%. SpO<sub>2</sub> cannot discriminate fast changes and normal values can be associated with critical levels of PaO<sub>2</sub>, especially after weaning from CPB. Thus, continuous blood gas monitoring revealed episodes of hypoxaemia during anaesthesia that were not detected by pulse oximetry or intermittent blood gas analysis.

The Paratrend revealed that the patients were hypocapnic during more than 50% of the operation time. In 24 instances it was severe enough (PaCO<sub>2</sub> < 4 kPa) to qualify for blind lifting. Hypocapnia was also detected in 50% of the conventional blood gas samples but was followed by ventilatory modifications in only one-third of the cases. Low end-tidal CO<sub>2</sub> (<4 kPa) was recorded on average during 60% of operating time, but was not followed by any therapeutic action. This voluntary maintenance of hypocapnia may be explained by the fact that anaesthetists fear hypoventilation more than hyperventilation, which is considered less dangerous. The CO<sub>2</sub> level plays an important part in the adjustment of cerebral microcirculation, and extreme values have been held responsible for neurological disorders post-bypass [4].

Continuous blood gas monitoring is a useful tool to detect metabolic acidosis, which was present during 15% of operating time in our patients. We encountered eight serious episodes that required lifting of the blind. In the majority of the episodes, the acidosis was related to the haemodynamic state of the patient. Sometimes acidosis is the first sign of peripheral hypoperfusion, which can be masked by normal systolic BP.

In conclusion, we have shown that there may be long delays before blood gas abnormalities during

cardiac operations are discovered by conventional intermittent blood sampling, in combination with pulse oximetry and capnography, as compared to continuous Paratrend monitoring. In 10% of cases, by the time we had obtained the blood gas results, the acid–base status of the patient had changed outside the limits. Rapid changes in acid–base status occur at the beginning and at the end of bypass. It is important to be aware of the limitations of the traditional intermittent technique.

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## A new cardiopulmonary bypass circuit with reduced foreign surface (CorX™): initial clinical experience and implications for anaesthesia management

### EDITOR:

Cardiac surgery provokes a vigorous inflammatory response. The direct contact of the patient's blood with the foreign surface of the cardiopulmonary bypass (CPB) circuit plays an important role in triggering this response [1]. The heat exchanger and open hard-shell venous reservoir contribute to the total artificial surface and can initiate blood protein denaturation and damage to cellular elements like erythrocytes and platelets [2]. Beyond that, marked haemodilution by the prime solution of the CPB circuit can trigger significant fluid shifts from the intravascular to the extravascular space [3]. A growing body of evidence suggests that significant cardiodepression may be a consequence of myocardial oedema caused by decreased colloid osmotic pressure and increased capillary permeability as well as the effects of proinflammatory cytokines on the myocardium [1]. Therefore, CPB circuits with restricted foreign surface and smaller priming volume may reduce the inflammatory response and subsequently morbidity and mortality [3,4]. Recently, a new CPB circuit (CorX™; Cardiovention, Santa Clara, CA, USA) was developed which offers a significant reduction of total foreign surface. We present our initial experience with the device and some important issues related to the anaesthetic management.

CorX™ is a disposable system consisting of an oxygenator with an integrated centrifugal pump at the bottom (Fig. 1). An air detector (AirVac™; Cardiovention, Santa Clara, CA, USA) at the top senses and evacuates air from the venous line. Following standard cannulation, the negative pressure generated by the centrifugal pump actively drains venous blood. The negative pressure in the venous line enables left ventricular (LV) venting and addition of prime, blood and other fluids. Excess blood in the system can be drained to a reservoir bag by the positive pressure in the arterial line. The reservoir bag is connected to the venous as well as the arterial line and when needed, the stored blood may be reinfused into the venous line. There is no cardiectomy suction, heat exchanger or hard-shell reservoir. The foreign surface, including the compact arterio-venous loop,

totals approximately 1.4 m<sup>2</sup>. Shed blood is retransfused via a cell saver. Temperature homeostasis is achieved with a heated blanket (Bair Hugger 560 Cath Lab™; Augustine Medical, MN, USA). All intravenous fluids are warmed and the operating room temperature is kept at 25°C.

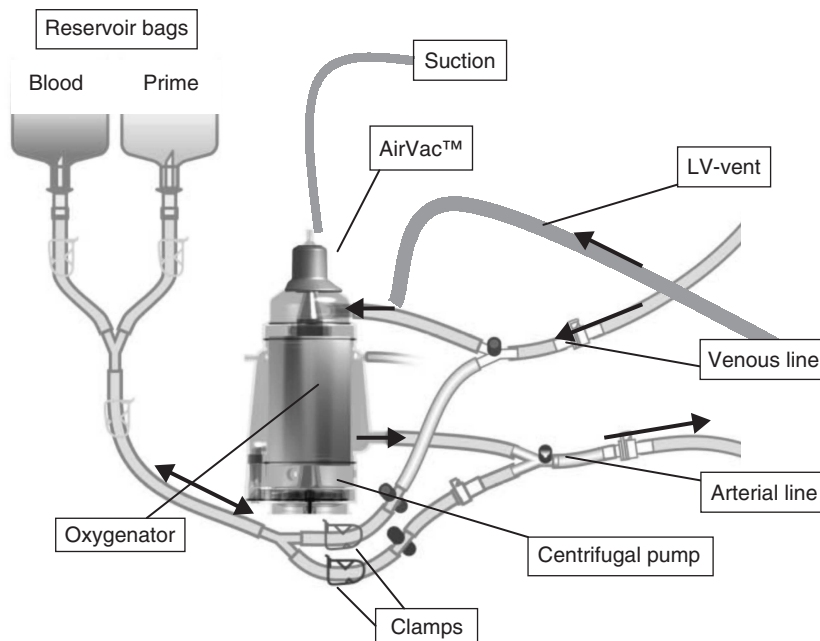
Following Institutional Review Board approval and written informed consent, nine ASA III patients (six males, three females) scheduled for elective coronary artery bypass surgery were enrolled in the study. Their mean age was  $64 \pm 13$  yr. After oral premedication with midazolam ( $0.1\text{--}0.2\text{ mg kg}^{-1}$ ) and clonidine ( $2\text{ }\mu\text{g kg}^{-1}$ ), anaesthesia was induced and maintained with propofol and sufentanil. The patients were ventilated with an air/oxygen mixture ( $F_{\text{I}}\text{O}_2$  0.5). Central venous and arterial catheters were inserted. The arterial line was connected to a cardiac output monitor (PICCO™; Pulsion Systems, Munich, Germany) for determination of haemodynamic variables (cardiac output, intrathoracic blood volume, systemic vascular resistance and extravascular lung water index). These measurements were performed in triplicate immediately before and 5 min after CPB. The operative and CPB techniques were strictly standardized. The crystalloid prime volume was 500 mL. Pump flow rate was  $2.5\text{ L min}^{-1}\text{ m}^{-2}$ . Normothermia was maintained. All patients were operated on by the same surgeon and anaesthetized by the same anaesthesiologist. A paired *t*-test was used for comparisons with a *P*-value  $<0.05$  considered to be statistically significant. Values are reported as mean  $\pm$  SD.

The duration of surgery was  $211 \pm 50$  min, CPB time was  $86 \pm 15$  min, and aortic cross-clamp time was  $49 \pm 15$  min. Total blood loss was  $2367 \pm 907$  mL and  $730 \pm 534$  mL was retransfused from the cell saver. Packed red cells were transfused in three cases (one patient received two units, and two patients, one unit each). There were no significant differences in cardiac output, intrathoracic blood volume, systemic vascular resistance or extravascular lung water index between the measurements before and after CPB (Table 1). In contrast, haematocrit was significantly lower after CPB ( $P < 0.01$ , Table 1). Nasopharyngeal temperature was  $36.2 \pm 0.5^\circ\text{C}$  before and  $36.2 \pm 0.6^\circ\text{C}$  after CPB ( $P > 0.05$ ). There were no adverse effects (air entrainment, circulatory arrest) related to the CorX™-use in any patient.

Our findings regarding cardiac output, intrathoracic blood volume, systemic vascular resistance and extravascular lung water index agree with data from

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**Figure 1.**  
CorX™ circuit. Arrows indicate direction of blood flow in the different lines.

**Table 1.** Haemodynamic parameters, haemoglobin and haematocrit before and after CPB (mean  $\pm$  SD,  $n = 9$ ).

	Pre-CPB	Post-CPB
CO (L min <sup>-1</sup> )	5.8 $\pm$ 0.9	6.7 $\pm$ 1.3
ITBV (mL)	1913 $\pm$ 317	1924 $\pm$ 501
ELWI (mL kg <sup>-1</sup> )	6.8 $\pm$ 1.9	7.6 $\pm$ 2.7
SVR (dyn s cm <sup>-5</sup> )	814 $\pm$ 173	778 $\pm$ 296
Haematocrit (%)	30.8 $\pm$ 3.2	25.4 $\pm$ 2.7*

CO: cardiac output; ITBV: intrathoracic blood volume; ELWI: extravascular lung water index; SVR: systemic vascular resistance.

\* $P < 0.01$  vs. pre-CPB.

studies using conventional CPB circuits [5]. In a preliminary animal study, Mueller and coworkers compared the CorX™ system with a conventional CPB circuit in a model of prolonged perfusion. They found an improved gas exchange, a stable haematocrit and a limited decrease in platelet count in the CorX™ group [6]. In our patients haematocrit was significantly reduced after CPB, similarly to conventional CPB [7]. The limited transfusion requirements (3/9 patients) despite the low pre-CPB haematocrit in our patients (30.8  $\pm$  3.2%) nevertheless suggests a positive impact of the reduced haemodilution of the CorX™ circuit. At the moment, sparing homologous blood transfusion seems to be an advantage of CorX™ compared to conventional CPB circuits.

We found that anaesthesiologist-directed temperature and intravascular volume control can compensate for the absent heat exchanger and reservoir without running the risk of haemodynamic instability or post-operative hypothermia. Temperature management is

crucial to avoid hypothermia and enable fast tracking. LV venting cannot be adjusted individually but depends on the negative pressure in the venous part of the circuit, which in turn depends on total pump flow. This may sometimes impede adequate drainage of the left ventricle. The CorX™ system may be used safely provided that the anaesthesiologist is familiar with this special CPB technique. Particular attention is necessary to avoid huge amounts of air entering the venous line of the CPB circuit (i.e. accidental venous decannulation or vent displacement), which could overwhelm the air suction capacity. An interdisciplinary approach including anaesthesiologist, surgeon and perfusionist is necessary for optimal management of cardiac surgery with the CorX™ system.

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## Continuous infusion of factor VIIIc during heart surgery in a patient with haemophilia A

### EDITOR:

Haemophilia A is an X-linked recessive genetic disorder due to a defective or deficient factor VIIIc protein, resulting in a tendency to bleed. It affects approximately 1/10 000 males. Continuous infusions of factor VIIIc concentrate have been used during surgical procedures in such patients [1,2]. However, the proposed infusion rates may not be adequate during open-heart surgery involving cardiopulmonary bypass (CPB) and associated haemodilution. We report our experience in a patient with haemophilia A, who underwent triple coronary artery bypass and mitral valve replacement.

This 53-yr-old male (body weight 80 kg) had a history of haemophilia A, and arterial hypertension, and stable angina pectoris. No incidence of significant bleeding was reported. His preoperative medications included bisoprolol and nitrates; aspirin was stopped 10 days before surgery. A coronary angiogram demonstrated two-vessel disease with occlusion of the right coronary artery (RCx) and a triple stenosis of left anterior descending coronary artery. Echocardiography revealed conserved left ventricular function and grade III mitral regurgitation. Relevant preoperative laboratory data were as follows (with reference values):

- haematocrit: 43% (40–54%)
- platelet count:  $143 \times 10^9 \text{ L}^{-1}$  ( $150\text{--}400 \times 10^9 \text{ L}^{-1}$ )
- prothrombin time: 76% (70–100%)
- activated partial thromboplastin time: 49 s (28–42 s)
- bleeding time: 4.5 min (<4 min)

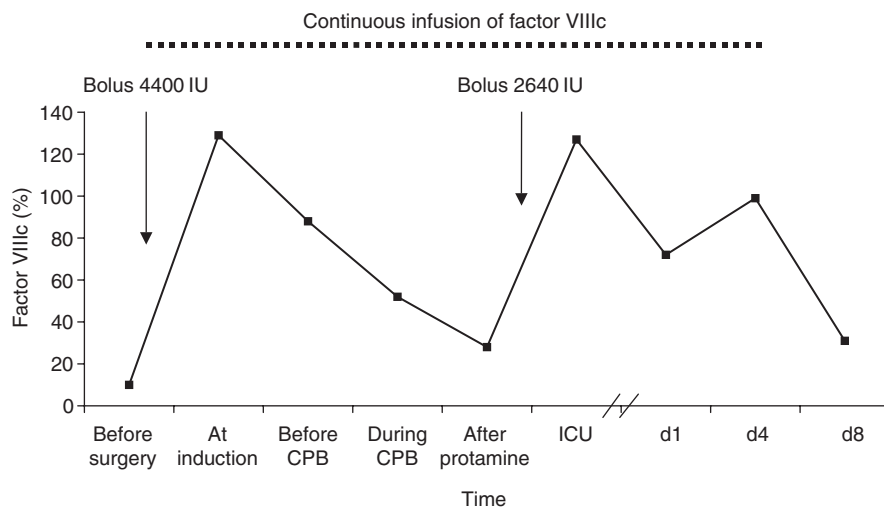
- factor VIIIc: 10% of normal
- factor VIIIvW: 116% of normal
- factor IX: 134% of normal.

Two hours prior to surgery, a bolus of  $50 \text{ IU kg}^{-1}$  of factor VIIIc solvent/detergent (Central Fractioning Department, Belgian Red Cross) was slowly injected, thus obtaining a factor VIIIc concentration of 129% of normal (Fig. 1). This was followed by a continuous infusion of  $3 \text{ IU kg}^{-1} \text{ h}^{-1}$ . Tranexamic acid 1 g was given before incision. Heparin  $300 \text{ IU kg}^{-1}$  before CPB resulted in an activated clotting time  $>999 \text{ s}$  (ACT II, Medtronic Hemotec Inc., Englewood, CO, USA). The bypass system was primed with Hartman's solution to obtain a haematocrit of 22%. Triple bypass was performed with the left internal mammary artery sequentially to the left anterior descending artery and the first diagonal, and the gastroepiploic artery to the right posterior descending artery. The mitral valve was replaced with a Carbo-medics mechanical prosthesis. Mild hypothermia to  $31.4^\circ\text{C}$  was achieved during valve replacement. Total bypass time was 175 min. Heparin was reversed with 380 mg of protamine, which gave a protamine/heparin ratio of 1.52. The post-protamine activated clotting time was 190 s.

Factor VIIIc values were difficult to obtain when the patient was heparinized. Despite the continuous infusion, the factor VIIIc level gradually fell to 28% after protamine (Fig. 1). A second bolus of  $33 \text{ IU kg}^{-1}$  of factor VIIIc was administered, as well as four units of fresh frozen plasma to control severe bleeding before wound closure (prothrombin time 27%). Intraoperatively, 916 mL of blood from a cell-saver device and four units of packed red blood cells were transfused to maintain the haematocrit greater than 20%. At the end of the procedure the platelet count

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**Figure 1.** Perioperative time course of factor VIIIc levels. Arrows indicate the bolus injections of factor VIIIc concentrate. The continuous infusion is indicated by a dotted line. d1, d4 and d8, respectively, are postoperative days 1, 4 and 8.

was  $65 \times 10^9 \text{ L}^{-1}$  and the haematocrit was 19%. Therefore, six units of pooled platelets and two units of packed red cells were given on arrival in the intensive care unit (ICU) where the patient remained stable. Catheters and chest drains (total drainage volume 1590 mL) were removed during continued factor VIIIc infusion. The patient was transferred to the ward on the 2nd postoperative day. The course was uneventful and the infusion of factor VIIIc was stopped on day 3. The patient was discharged on the 9th postoperative day with a residual factor VIIIc level of 27%. Coumarin therapy was instituted to prevent mitral valve clotting. At 6 weeks follow-up the patient was doing well and had no bleeding complications.

Only a few cases of complex cardiac surgery procedures in patients with haemophilia A have been reported in the literature. In most of them bolus injections of factor VIIIc were given. However, MacKinlay and colleagues reported the use of continuous infusions of factor VIIIc in three cases [3]. Although the biological half-life of factor VIIIc is about 8–12 h, a continuous infusion compensates for factor VIIIc consumption and optimizes safety [4].

Other therapeutic options include tranexamic acid, desmopressin and aprotinin, alone or in combinations. Desmopressin administration in haemophilia A patients undergoing coronary artery bypass grafting (CABG) is controversial as it seems to increase the von Willebrand factor but not factor VIIIc [5]. Tranexamic acid has been used in haemophilia patients and was administered to reduce intra- and postoperative bleeding.

It has been reported that a factor VIIIc bolus of  $50 \text{ IU kg}^{-1}$  usually achieves a blood level of 100%. We combined this standard bolus with a continuous infusion at  $3 \text{ IU kg}^{-1} \text{ h}^{-1}$  aiming at a factor VIIIc level above 50% during surgery and for the first 4 postoperative days [6]. This proved not to be sufficient

and a second bolus of  $33 \text{ IU kg}^{-1}$  was needed at the end of bypass. This can be explained by haemodilution during bypass and possible consumptive coagulopathy. Moreover, cell-saver blood has a very low concentration of coagulation factors [7]; we have previously found a concentration of 3% of factor VIIIc in blood reinfused after cell-saver processing in a non-haemophilic patient.

In conclusion, complex heart surgery is feasible in patients with haemophilia A. A bolus dose of  $50 \text{ IU kg}^{-1}$  of factor VIIIc, followed by a continuous infusion of  $3 \text{ IU kg}^{-1} \text{ h}^{-1}$  was not sufficient during CPB combined with a cell-saver device. A second, half-dose bolus compensated for the defect and stopped the bleeding. Continuous infusions can be stopped after chest drain removal. More experience is needed in order to standardize factor VIIIc infusion protocols during complex cardiac surgery in haemophilic patients.

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## Heart failure after Caesarean section for twin delivery

### EDITOR:

Pulmonary oedema after delivery represents a rare but life-threatening situation. We present the management and outcome of a patient with heart failure following Caesarean section. Amniotic fluid embolism, eclampsia, peripartum cardiomyopathy and idiopathic cardiomyopathy represented the differential diagnoses.

A 34-yr-old primipara was admitted to our hospital with threat of a premature delivery of twins at 25-week gestational age. A premature birth protocol was initiated with tocolytic therapy. The patient had no history of cardiovascular disease and was on propylthiouracil therapy for hyperthyroidism. Laboratory results showed a haemoglobin value of  $9.7 \text{ g dL}^{-1}$ . Leucocytes, platelets, electrolytes, renal, liver and thyroid function, vitamin B<sub>12</sub> and folic acid were all normal. An autoimmune screening (anti-cardiolipin antibody, anti-beta 2-glycoprotein I antibody, Waaler Rose reaction, lupus like anticoagulants, anti-Thyroid stimulating hormone (TSH) receptor antibody, anti-thyroglobulin antibody, anti-thyroid microsomal antibody) proved negative. Only anti-nuclear factor tested positive.

The patient was scheduled for Caesarean section under spinal anaesthesia due to intra-uterine growth retardation of one twin, peripheral oedema and proteinuria ( $0.4 \text{ g } 24 \text{ h}^{-1}$ ). After Caesarean section, the patient presented signs of acute pulmonary oedema with rapid onset of dyspnoea, tachypnoea, hypertension (220/130 mmHg) and fatigue. She was admitted to the intensive care unit (ICU) with suspected diagnosis of amniotic fluid embolism and pre-eclampsia. She required tracheal intubation and ventilatory support.

A thoracic-abdominal computed tomography excluded embolism. A first bedside echocardiographic examination revealed left ventricular failure with an ejection fraction of 40%. Subsequent echocardiography showed worsening of cardiac function with an ejection fraction  $<20\%$ . A pulmonary artery catheter and an intra-aortic balloon pump (IABP) were inserted.

Laboratory investigations revealed a haemoglobin value of  $10.8 \text{ g dL}^{-1}$ , leucocyte count of  $13.4 \times 10^9 \text{ L}^{-1}$  and normal platelet count. Electrolytes, renal and liver functions were normal. Chest X-ray showed an enlarged heart and pulmonary oedema.

Central venous pressure (CVP), pulmonary artery pressure and pulmonary artery wedge pressure were 15, 27 and 16 mmHg, respectively; cardiac output was  $9.0 \text{ L min}^{-1}$  (cardiac index  $5.4 \text{ L min}^{-1} \text{ m}^{-2}$ ). A diagnosis of postpartum cardiomyopathy was made and an appropriate treatment instituted. The patient was treated with a continuous infusion of dobutamine  $5 \mu\text{g kg}^{-1} \text{ min}^{-1}$ , sodium nitroprusside  $0.5 \mu\text{g kg}^{-1} \text{ min}^{-1}$ , nitrates, high dose furosemide, corticosteroids, hydralazine, digoxin, angiotensin-converting enzyme (ACE) inhibitors, low molecular weight heparin and continuous positive airway pressure. Her condition gradually improved, and the IABP was removed after 3 days. She was weaned from mechanical ventilation after 4 days, and transferred to the coronary ICU after 8 days. An echocardiograph showed an ejection fraction of 42%, a left ventricular end diastolic volume of 77 mL and a left ventricular end systolic volume of 45 mL. After 14 days, she was discharged on oral therapy with furosemide, carvedilol, spironolactone, an ACEinhibitor and a decreasing prednisone dose. The discharge echocardiography showed a left ventricular ejection fraction of 45–47%.

Since then the patient has been symptom free, with residual slight fatigue only. Echocardiography after 5 months showed a left ventricular ejection fraction of 47% and her two babies were well after a follow-up of 9 months.

Peripartum cardiomyopathy is a rare and life-threatening disease of unknown aetiology that affects previously healthy women. Incidence ranges from 1 in 1300 to 1 in 15 000 pregnancies and it constitutes  $<1\%$  of all cardiovascular events related to pregnancy. Although the aetiology of the disease remains unclear [1], many causes have been suggested, including myocarditis, abnormal immune response to the haemodynamic stress of pregnancy and prolonged tocolysis. Risk factors include advanced age ( $>30 \text{ yr}$ ), multiparity, black racial origin, twin pregnancy, obesity, chronic hypertension, pre-eclampsia or eclampsia, alcohol and cocaine abuse,

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malnutrition, familiarity, puerperal infections and postpartum anaemia [2]. Today, no prophylaxis is known apart from recognising and treating the above-mentioned predictive factors.

Heart failure during the peripartum period was first described in 1849. The first reports, in the last century, are attributed to Ritchie, Virchow and Porak [2]. In 1971, Demakis and colleagues described criteria for its diagnosis, namely the development of heart failure during the last month of pregnancy or within 5 months after delivery, lack of an aetiology for the cardiac failure and absence of heart disease prior to the last month of pregnancy. More recently, echocardiographic evidence of diminished left ventricular systolic function was suggested as a further criterion. Some authors use very strict echocardiographic criteria; for instance, ejection fraction  $<45\%$ , or M-mode fractional shortening  $<30\%$ , or both and end-diastolic dimension  $>2.7 \text{ cm m}^{-2}$  [3].

Most patients (78%) present with symptoms during the first 4 months postpartum. Only 9% present in the last month before delivery, and 13% either  $>1$  month antepartum or  $>4$  months postpartum. Clinical signs are those of heart failure, i.e. dyspnoea, cough and orthopnoea. Other common symptoms are haemoptysis, chest pain and abdominal pain. Cardiomegaly, tachycardia and a third heart sound (85%) can be found on physical examination. Frequently, an elevated blood pressure (BP), increased jugular venous pressure, hepato-splenomegaly, peripheral oedema, crackles on lung auscultation, a mitral regurgitation murmur and dysrhythmia are also present [4]. Rarely, a lower extremity arterial thromboembolism may be the initial manifestation of peripartum cardiomyopathy. More frequently this is a later complication. The tendency towards formation of thrombi is probably caused by the hypercoagulation state of late pregnancy, in combination with stasis and turbulent flow in the dilated heart.

Medical treatment of peripartum cardiomyopathy does not differ from that for other forms of congestive heart failure. During pregnancy, it includes digoxin, loop diuretics, sodium restriction, afterload reducing agents, hydralazine and nitrates. After delivery, ACE inhibitors are used. High-dose ACE inhibitor treatment has been associated with a significant decrease in interleukin-6 levels, one of the pro-inflammatory cytokines that may play a role in the genesis of peripartum cardiomyopathy. Due to a high risk for venous and arterial thrombosis, anticoagulation with heparin should be instituted [1]. The role of immunosuppressive therapy remains ambiguous [2].

Patients who fail to recover may require intensive care, pulmonary artery catheterization, intra-aortic balloon counter-pulsation, and, as an extreme measure,

a left ventricular assistance device. Cardiac transplantation should be considered for patients who fail therapy.

Strict bed rest that was advised in early studies may have contributed to the development of deep venous thrombosis and subsequent pulmonary embolism and may in part be an explanation for the high mortality rates in older studies. Since normal pregnancy can cause symptoms similar to heart failure, the New York Heart Association classification may not adequately estimate the severity of the heart failure. Mortality from peripartum cardiomyopathy remains high, about 25–50% [3]. However, compared with patients with idiopathic cardiomyopathy, patients with peripartum cardiomyopathy have a better survival [5]. About half of the patients recover without any complication. Persistence of the disease after 6 months indicates irreversible cardiomyopathy and a worse survival [4]. Causes of death include chronic progressive congestive heart failure, fatal dysrhythmias and thromboembolic complications [2].

In case of persisting left ventricular dysfunction, subsequent pregnancy can be very dangerous and should be discouraged although successful pregnancies, or changes in left ventricular diameter of fractional shortening have been reported [6]. However, recurrence of peripartum cardiomyopathy despite a rapid normalization of heart size and heart function after a previous pregnancy is possible [7]. Women should be informed of that the risk is high.

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## Checking anaesthetic equipment

### EDITOR:

In 1995, a system for checking anaesthetic machines was developed and given the name 'COVERS' [1]. That system was reported in this journal and we considered it to be superior to that generally recommended at the time [2].

COVERS has two components. First there is, COVERS itself, a pre-use checklist the completion of which is noted in the diary for the machine wherein any problems must also be recorded. Secondly, there is a comprehensive checklist to eliminate non-recurring faults which is completed by an anaesthetist and operating department practitioner or equivalent member of staff together when a machine is returned from service or breakdown before it resumes front-line duty.

*The Checklist for Anaesthetic Equipment 2004* [3] recently issued by the Association of Anaesthetists of Great Britain and Ireland includes changes that are pertinent with respect not only to the anaesthetic machine and associated equipment but also to address breathing system incident concerns [4].

In this publication (Section 2: procedures) we note the sensible change that, whilst implementation of the checks remains the responsibility of the anaesthetist who must be satisfied that they have been carried out correctly, the task itself may be undertaken by a suitably trained member of the team. New inclusions are first, the requirement for the oxygen failure alarm to be checked on a weekly basis and, secondly, the need to have immediately available an alternative means to ventilate the patient including an oxygen cylinder. Also, the new guidance that confirmation of pre-use checking should be recorded in the logbook/diary provided with the anaesthetic machine, as we currently do, is valuable.

The advice that 'the "first user" check after servicing is especially important and must be recorded as such' is restated in the guidelines but still no advice is given as to the form that this should take. Our current practice in this regard has two components:

- (a) The "A" check which is the pre-use checklist carried out by the responsible operating department

practitioner alone without any time pressure first, after return of the anaesthetic machine from simple repair when no disassembly has taken place and, secondly, twice between services as a back up to the daily checks.

- (b) The "B" check which is a comprehensive checklist [1] carried out by anaesthetist and operating department practitioner together following the six monthly service or any major repair requiring reassembly.

We particularly commend this practice to all readers any of whom can be supplied with a copy of the comprehensive checklist on request.

Our department considers that the 2004 Association of Anaesthetists recommendations are now appropriate to adopt as the standard pre-use checklist and COVERS will be exchanged for these which will introduce a conformity of additional benefit to our rotating trainees.

We continue to believe, however, that use of the comprehensive checklist in addition and as described is very necessary and should be a part of routine practice and teaching.

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