Correspondence

Minimal invasive cardiac bypass circuits: safety issues and impact on inflammation

EDITOR:
We read with interest the correspondence about an initial clinical experience with the CorX™ and welcome any contribution to find new ways to diminish the inflammatory response during cardiopulmonary bypass (CPB) [1]. However, there are major drawbacks of the CorX™ system. First of all, the CorX™ system lacks a heat exchanger, which is imperative for rapidly cooling down a patient in emergencies and in case of CPB system failure. It is true that the heat exchanger does increase the total artificial surface of the CPB but basic security features should outweigh the need for the reduction of total surface area. In our unit we are working with the MECC system (Minimal Extracorporal Cardiopulmonary Circulation, Jostra Medizintechnik AG, Hirrlingen, Germany). The MECC has a greater total surface area (2.4 m²) than the CorX™ (1.4 m²) but it is still less than in conventional CPB systems. The crystalloid priming solution in the MECC is 700 mL compared to the CorX™ system which has a priming volume of 500 mL. However, there are still major security differences. In contrast to the CorX™ system a heat exchanger (surface area of 0.6 m²) is integrated into the MECC [2].

We would also like to stress the importance of normothermic CPB, which you achieve with a heating blanket (Bair Hugger 560 Cath Lab™; Augustine Medical, MN, USA), a warm operating theatre and the infusion of warmed fluids. The question remains if these efforts in order to keep a patient normothermic can equally be applied in large and obese patients who lose more heat because of their increased body surface area. Here again, rewarming is more difficult without a heat exchanger and priority should be given first to security.

During the few critical events with the MECC system, we learnt that it is essential to know how arterial gas bubbles could easily be removed. It has been described that the CorX™ system does have rescue features to remove gas bubbles and parts of the atroventricular loop are separable in order to connect it to a rescue system. In this respect, would the CorX™ also allow the speedy setup of a heat exchanger? In contrast to the MECC system the CorX™ also has no backup filter on the venous line to prevent venous embolism in case of accidental suctioning of huge amounts of air.

As was mentioned in the description of the CorX™, the absence of left ventricular venting could impede adequate drainage of the left ventricle. This, in contrast, is solved in the MECC system where the left ventricle can be vented with a negative pressure of −150 mmHg. Queries also remain whether the inner tubing surface of the CorX™ is coated with heparin which compared to non-coated tubing systems does change heparin requirements during CPB and consequently also affects inflammatory responses.

The location of cardioplegia in the CorX™ also remains unclear, because it does not figure on the description of the system. What is the exact exchange surface area of the CorX™ oxygenator and would it also provide sufficient gas exchange for obese patients? Reservoir bags replacing the missing hard-shell reservoir should also be included into the surface area because it forms part of the total surface area. The reservoir bag is part of a closed system without surface-to-air contact, which is also catered for in the MECC system.

The fact that the total surface area is diminished does not decrease events where an inflammatory response can get out of hand as we experienced in an obese patient who was entirely stable prior to elective coronary bypass surgery but progressively developed severe metabolic acidosis during MECC bypass. Maximal speed of pump flow is limited to 7 L min⁻¹ in both systems (CorX™ and MECC). This may not be enough for obese patients in order to deliver sufficient oxygen to the periphery and to compensate for the development of metabolic acidosis without increasing shear forces within the circuit and thereby increasing bio-incompatibility and inflammatory responses. The difference in priming volume and surface area does not justify sacrifices in security.

We admit that some security aspects of the MECC system currently are also unresolved. Therefore, we
only use this system for straightforward cases of coronary artery bypass surgery where neither intra-operative nor postoperative complications would be expected. We disagree that ‘the CorX™ system can be used safely’ and conclude that the CorX™ bypass system compared to the MECC system offers less security. We recommend awaiting further studies prior to fully accepting both of these low surface area systems as routine in clinical practice.

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Reply

EDITOR:
We would like to thank Dr. Kiss and colleagues for their interest in our recent description of the CorX™ system (Cardiovention, Santa Clara, CA, USA) and their comments with special emphasis on safety issues in comparison to the MECC™ system (Minimal Extracorporal Cardiopulmonary Circulation, Jostra Medizintechnik AG, Hirrlingen, Germany) [1]. The authors express serious concerns regarding the lack of a heat exchanger in the CorX™ circuit in case of an emergency and if large and obese patients with an increased risk of hypothermia are operated. In case of any emergency due to CPB circuit failure, however, a substitute CPB circuit should be readily available within minutes and in our institution a complete replacement is accomplished within 5 min at most. Such a catastrophic event can occur on principle with any minimal invasive CPB circuit if huge amounts of air have entered the system. Under these circumstances, cooling with the CPB system on hand is impossible. Secondly, in contrast to the authors’ notion, large and obese patients are less prone to intra-operative hypothermia since the subcutaneous fat tissue is a perfect insulator in this respect. Furthermore, a small heat exchanger has been developed for use with the CorX™ system, which enables temperature homeostasis (but, on the other hand, adds to foreign surface) and is therefore only implemented if necessary.

Air entrainment is another important issue stressed by the authors. An integral part of the CorX™ circuit is the AirVac™, which is able to remove large quantities of air which have inadvertently entered the circuit. The effect on small bubbles and microbubbles has never been investigated in a systematic fashion so far. This holds true, however, also for the MECC™ system. In our experience, the ability of the backup filter on the venous line to prevent venous embolism is anything but impressive. In our institution, we use the MECC™ bypass with a special filter on the arterial line. Administration of cardioplegia can be easily accomplished with a separate roller pump, which is a part of our CorX™ console.

The authors then report a patient experiencing severe metabolic acidosis during MECC™ bypass and raise the question if the maximal pump flow of 7 L min⁻¹ is sufficient even for obese patients. This is somewhat contradictory to their emphasis on the presence of a heat exchanger, since oxygen consumption can effectively be reduced by cooling, which allows for a considerable reduction in pump flow. More important, a maximal pump flow of 7 L min⁻¹ as realized with the CorX™ and MECC™ circuit is also a standard feature of current conventional CPB circuits.

We agree that an excessive impact of total surface area on the degree and severity of inflammation certainly is not warranted. The inflammatory response to CPB is initiated immediately when the patient’s
blood is exposed to the CPB tubing and the oxygenator [2]. However, ischemia-reperfusion injury during CPB also contributes to the inflammatory response. A simple correlation between total surface area and the inflammatory host response therefore is overly simplistic, since surgical trauma, blood transfusion as well as gut translocation all may initiate inflammation [3], while on the other hand several perioperative interventions, for example volatile anaesthetics, modulate one or more of the pathways involved [4]. This is clearly emphasized by recent studies which could not demonstrate a beneficial effect of off pump cardiac surgery compared to conventional on pump procedures [5]. We are about to finish a large, randomized study comparing the MECC™ system with conventional CPB in our institution which will probably shed some light on this issue in the near future. Finally, we disagree that the MECC™ and CorX™ system differ regarding safety issues. Given the precautions mentioned above, both systems can be used safely in the suitable patient population.

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Postoperative airway management in head and neck oncoplastic surgery

EDITOR:
We read with interest Mishra and colleagues retrospective study describing the airway management in head and neck reconstructive surgery [1]. They conclude that oral cancer patients can be managed safely without the routine use of a tracheostomy. We believe that the airway management plan in head and neck oncoplastic surgery should not be limited to securing the airway for anaesthesia but include the postoperative period. After such major surgery, the resection or reconstruction or both of which involve the airway; it is in the postoperative period where any airway compromise may be difficult to manage [2]. Therefore, preoperative elective tracheostomies may be more frequently required for postoperative indications than for securing the difficult airway. We disagree with Mishra and co-workers, that an elective preoperative tracheostomy is technically difficult. On the contrary, we feel it is relatively easier when done electively. Our surgeons do not find neck dissections and/or flap reconstructions more difficult in the presence of a tracheostomy. We are able to provide a relatively unhindered operating field using a sterile flexible swivel mount to connect to the breathing circuit.

Rather than subject the patient to a difficult airway manoeuvre only to perform a tracheostomy at the end of surgery or need it urgently, if we can predict its need (in the postoperative period) we would prefer to do the tracheostomy before the surgery [2]. A scoring system to identify patients requiring an elective preoperative tracheostomy has been recently reported [3]. We have already suggested the need for elective preoperative tracheostomies in patients with extensive malignancies and in those whom two-staged surgery is planned [4]. Therefore, in head and neck oncoplastic surgery, the postoperative period (and the need for Mishra and colleagues' criteria) may be a suitable opportunity to perform a preoperative elective tracheostomy to prevent any postoperative airway compromise and to provide a relatively unhindered operating field.

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for a tracheostomy therein) should be carefully considered in the initial airway management plan.

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Salivary gland swelling and lingual nerve injury with the ProSeal laryngeal mask airway

EDITOR:

Lingual nerve palsy, parotid and submandibular gland swelling have been reported in association with the classic laryngeal mask airway (LMA) [1]. We report all three occurring in the same patient with the ProSeal LMA.

A 64-yr-old female (height 158 cm, weight 76 kg, ASA I) was scheduled for day case hand surgery. She had no past medical history. Airway examination revealed Mallampati Grade 1 and good dentition. Induction was with midazolam 1.5 mg, alfentanil 0.5 mg and propofol 150 mg. Maintenance of anaesthesia was with sevoflurane 2–3% in oxygen 33% and air. A size 4 ProSeal LMA was easily inserted without any oropharyngeal resistance using the digital technique and the cuff inflated with 15 mL air. A water-based gel was used for lubricant. Most of the bite block was inside the mouth and the suprasternal notch tap test was positive, suggesting correct placement; however, ventilation was impossible despite a seal >50 cmH₂O. An algorithm for the management of airway obstruction was followed: both jaw thrust and cuff deflation resulted in complete relief of airway obstruction, suggesting the aetiology was mechanical compression of the vocal cords or infolding of the cuff [2]. Subsequent fibreoptic inspection confirmed that the aetiology was cuff infolding which became clinically significant when the cuff volume exceeded 5 mL. A size 12-F gastric tube was easily inserted and 5 mL clear fluid aspirated from the stomach. The procedure, which was uneventful and lasted 45 min, was completed with the cuff inflated with only 2 mL of air and pressure controlled ventilation set at 16 cmH₂O. Emergence was uneventful and the LMA was removed when the patient was able to open her mouth to command. There was no blood on the LMA or gastric tube. 2 h after the LMA was removed, the patient complained of difficulty speaking and swallowing, pain in the right side of her face, a lump in the floor of her mouth and a numb tongue. Examination revealed an enlarged non-tender right submandibular gland, an enlarged slightly tender right parotid gland and a right lingual nerve palsy. All other cranial nerves were intact and there was no evidence of tongue swelling or discolouration. There were no symptoms of sore throat or hoarseness. The patient was observed for 24 h, during which time all symptoms and signs disappeared, the parotid and submandibular gland swelling in 2–4 h and the lingual nerve palsy in 6–10 h.

The lingual nerve and the parotid and submandibular ducts are vulnerable to compression and distortion within the oral/pharyngeal cavities. Malposition, cuff overinflation and undersizing have been the probably

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aetiology in previous reports [1]. The probable aetiology in the current case was that the ProSeal LMA was too big even when fully deflated. Perhaps it would be wise to downsize if full deflation of the cuff is required to relieve airway obstruction. Another reason to downsize is that the fully deflated cuff has a less effective seal for protection against regurgitation and gastric insufflation [3].

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Pre-treatment of anaphylaxis, does it really work?

EDITOR:
The incidence of anaphylactic reactions during anaesthesia has been reported from 1 in 10,000 to 1 in 20,000 [1]. But the exact frequency of anaphylaxis during hydatid cyst operations is not known [2]. Anaphylaxis has also not been reported during percutaneous hydatid cyst treatment [2]. Pre-treatment with histamine receptor antagonists and/or corticosteroids to prevent anaphylaxis is still controversial [3,4]. Treatment of anaphylaxis is based on stopping the administration of the offending agent and inhibiting the effects of allergic mediators immediately. We would like to describe the treatment of an anaphylactic reaction in a patient undergoing percutaneous hydatid cyst aspiration focusing on the ineffectiveness of pre-treatment of anaphylaxis.

A 9-y-old, 25 kg, male was admitted for ultrasonographic-guided percutaneous drainage of a hydatid cyst in the liver in the interventional radiology unit. The same procedure had been performed 2 months previously. Anaphylaxis had not occurred during the previous attempt at percutaneous drainage. The patient was given 10 mg kg\(^{-1}\) per day albendazole for 1 week before the procedure. In addition pheniramine maleate 50 mg and methylprednisolone 25 mg were given intravenously (i.v.), 30 min before starting the procedure for prophylaxis against anaphylaxis. Sedation and analgesia during the procedure was performed with i.v. 0.03 mg kg\(^{-1}\) midazolam and 2 µg kg\(^{-1}\) fentanyl. Haemodynamic parameters and oxygen saturation were in the physiological limits according to his age. As soon as cyst puncture was performed with a biopsy needle, generalized erythema was noticed. The patient developed respiratory distress, periorbital oedema and became bradycardic then pulseless. Orotracheal intubation was performed immediately and epinephrine 1:1000, 0.3 mg was given i.v. After cardiopulmonary resuscitation (CPR) was started, a second dose of pheniramine maleate 50 mg and methylprednisolone 25 mg were administered i.v. Subsequently, 5 min later, peripheral pulses became palpable and systemic arterial pressure was 40/18 mmHg. CPR was discontinued and 2 min later the heart rate was 100 min\(^{-1}\) and systemic arterial pressure 140/88 mmHg. Serum trypase values were 24 µg L\(^{-1}\) (normal baseline < 14.1 µg L\(^{-1}\)) 1 h after the procedure and 21 µg L\(^{-1}\) 6 h later. The patient was admitted to the intensive care unit, had an uneventful postoperative period and subsequently was discharged 2 days later without any neurological sequelae.

Hydatid disease caused by *Echinococcus granulosus* is an endemic disease in South and Central America, Western Europe, the Middle East, Russia and China.

References

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As surgical treatment is associated with a high rate of morbidity and a long period of hospital stay, there has been a trend towards using percutaneous treatment of liver hydatid cysts [5]. Yaghan and colleagues [2] pointed out that, the most important problem during percutaneous cyst aspiration procedure was the risk of anaphylactic shock due to dissemination of fluid into the abdomen. Men and colleagues [6] treated 168 hydatid cysts in 111 patients percutaneously and they reported one case of death due to anaphylactic shock. Akhan and Ozmen [5] reported that the death rate due to anaphylactic shock might be stated to be in between 0.1% and 0.2% in these cases according to the literature.

Anaphylactic reactions during anaesthesia have been reported with increasing frequency. Mertes and Laxenaire [1] suggested that life-threatening allergic reactions (as percentages) during anaesthesia were due to neuromuscular blocking agents (69.2%), latex (12.1%), antibiotics (8%), hypnotics (3.7%), colloids (2.7%), opioids (1.4%) and other agents (2.9%). The anaphylactic reaction in our case might have occurred due to intrahepatic or intraperitoneal hydatid cyst fluid contact following by iatrogenic cyst membrane perforation.

Anaphylactic reactions are life-threatening situations which may happen suddenly during anaesthetic management. Anaphylactic hypersensitization starts after exposure of an antigen that stimulates the production of immunoglobulin E (IgE). If a new contact with antigen occurs in the host, mediators such as histamine, leukotrienes, tryptase and prostaglandins are released [1]. Anaphylactoid reactions have the same clinical symptoms, but neither sensitizing IgE antibodies nor previous exposure to the antigen is involved in its mechanism [1]. Although there was neither adverse event nor anaphylactic reaction in our patient during his previous operation, the anaphylaxis might occur due to the high antigenicity of proteinaceous hydatid cyst fluid.

Elevated serum tryptase levels can be observed in anaphylactic and anaphylactoid reactions, but Mertes and Laxenaire [1] pointed out that, the positive predictive value of tryptase for the diagnosis of anaphylaxis was 92.6%. In addition they suggested that a serum tryptase concentration >25 µg L⁻¹ was strongly in favour of an anaphylactic mechanism. As serum tryptase values were 24 µg L⁻¹ (normal baseline < 14.1 µg L⁻¹) 1 h after the procedure and 21 µg L⁻¹ 6 h later, we can say that the reaction in our case contains an anaphylactic mechanism.

There have been some controversies about corticosteroid and antihistaminic pretreatment for pharmacological prophylaxis of anaphylaxis. Fisher and Doig [4] suggested that pretreatment with antihistamines and corticosteroids might block or ameliorate the severity of reactions. Lorenz and colleagues [7], in their prospective randomized study, also found that combined use of H1 and H2 antihistamines were effective for the prophylaxis of anaphylactoid reactions provoked by gelatine solutions used as volume expander. However, Mertes and Laxenaire [1] pointed out that the beneficial effects of steroid and antihistamine pretreatment may be due to their uses in only histamine-mediated anaphylactoid reactions. In the same manner, prophylactic approach to severe anaphylaxis has been found to be ineffective by Serlock and colleagues [3]. Severe anaphylactic reaction occurred in our case, despite antihistamine and corticosteroid pretreatment. The ineffectiveness of pretreatment of anaphylaxis in our patient may be due to the severity of the anaphylactic reaction. This result was comparable with the proposals of Mertes and colleagues and Serlock and colleagues. We suggest that pretreatment for anaphylaxis is not always effective especially in severe anaphylactic reactions.

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Severe hypoxaemia following intravenous nitroglycerine administration in an obese patient: case report

EDITOR:
The hypoxic pulmonary vasoconstriction (HPV) response is an important physiological event for preserving the ventilation/perfusion ratio [1]. It is a self-modulatory mechanism diverting pulmonary blood flow from poorly ventilated and hypoxic to better-ventilated and normoxic lung regions [2,3]. Anaesthesia accentuates decreases in functional residual capacity (FRC) in patients who are obese compared with individuals who are not obese. The decrease in FRC impairs the ability of obese patients to tolerate even brief periods of apnoea [4]. Besides these common respiratory problems in patients with obesity, any adverse condition disturbing their respiratory physiology may alter oxygen balance. Drugs that interfere with the protective mechanism of HPV may adversely affect gas exchange leading to hypoxaemia [5]. This case report describes severe desaturation episodes in a patient with obesity, hypertension and diabetes mellitus after intravenous (i.v.) nitroglycerine administration.

Case report

A 43-yr-old, morbidly obese (105 kg) man presented to the emergency department with recent-onset abdominal pain, fever (37.3°C), nausea and vomiting. His medical history included poorly controlled hypertension and diabetes mellitus of 5 yr duration. His preoperative blood pressure (BP) was 166/118 mmHg, and an electrocardiogram showed a normal sinus rhythm of 96 min⁻¹. Results of his physical exam were normal except for being morbidly obese and having rebound on the right lower quadrant of his abdomen. His white blood cell count was 13 400 mm⁻³, and all other haematological and biochemical blood results were unremarkable. His chest radiograph had no pathological signs. After performing an abdominal ultrasonography, he was transferred to the operating theatre for an appendicectomy.

Routine anaesthesia monitors were applied, and thiopental (5 mg kg⁻¹), fentanyl (2 µg kg⁻¹) and atracurium (0.3 mg kg⁻¹) were administered for the induction of general anaesthesia. The trachea was intubated with an 8.5 mm endotracheal tube with no problem. His heart rate, BP and oxygen saturation (SpO₂) measurements after induction were 72 min⁻¹, 180/100 mmHg, and 99%, respectively. Anaesthesia was maintained with 1% isoflurane in 40% O₂ : 60% N₂O. Soon after beginning the operation, the patient’s systolic BP suddenly increased to 200 mmHg. Nifedipine 10 mg was administered sublingually. Because there was no change in his BP, 0.5 mg nitroglycerine was administered i.v. The patient’s BP gradually decreased to about 140/90 mmHg; however, his SpO₂ measurement also decreased simultaneously to 90% and remained at that level for the next 5 min. After increasing the inspiratory O₂ fraction (FiO₂) to 1.0, we suctioned the endotracheal tube and auscultated his chest to be sure that both lungs were ventilated equally. His SpO₂ improved to a maximum of 97%, and his respiratory effort began. However, 10 min later his BP again increased to 190/110 mmHg, and another 0.5 mg nitroglycerine was administered. This time, even though he was breathing effectively, the patient’s SpO₂ decreased to 79% at which point-controlled ventilation with 100% O₂ was begun. Arterial blood gas analysis revealed a partial pressure of arterial oxygen (PaO₂) of 70 mmHg, partial pressure of carbon dioxide (PaCO₂) of 41 mmHg, base deficit of 4.7 mEq L⁻¹ and pH value of 7.31. As the operation was completed and the patient was awake he was extubated and given 100% O₂.

His desaturation continued however. The radiograph of his chest was completely normal. We therefore decided to transfer the patient to our surgical intensive care unit (ICU). His first arterial blood gases on arrival to the ICU with oxygen (8 L min⁻¹) by nasal cannula were PaO₂ of 53 mmHg (SpO₂ 84%), PaCO₂ of 30 mmHg, base deficit of 4.2 mEq L⁻¹ and pH value of 7.33. His electrolyte and blood glucose levels were within normal ranges. Within 2.5 h after the initial event, the patient was haemodynamically stable, and his SpO₂ increased gradually to about 98% while supplemental oxygen therapy was gradually decreased to 2 L min⁻¹. He was transferred to our general surgery ward later that day and discharged from the hospital 2 days later.

Discussion

Our patient was obese with uncontrolled hypertension and diabetes mellitus. To our knowledge, this is the first case report of a severe desaturation episode in a patient with obesity, hypertension and diabetes mellitus after i.v. nitroglycerine administration due to inhibition of HPV. We initially suggested no correlation between the hypoxia and nitroglycerine in the first
hypoxaemia attack and repeated the same dose. After the second hypoxaemia attack, however, we assumed that the nitroglycerine which had been administered twice, was to blame. Even though his desaturation persisted, to prevent his hypertension from becoming more severe, we extubated him and referred him for monitoring in our ICU.

HPV is inhibited by several mediators present in the blood or released from lung parenchyma such as substance P, calcitonin, and atrial natriuretic peptides, and by endothelium-derived vasodilators such as prostacyclin and nitric oxide. It also may be inhibited by vasodilating drugs including calcium channel blockers, nitroglycerine and halogenated anaesthetics [5]. Nitroglycerine-induced hypotension may cause significant impairment in pulmonary gas exchange in patients with normal lung function. In our case, we think that nitroglycerine administration was the primary cause of the hypoxia as the patient became desaturated with the administration of nitroglycerine.

It is known that the vasoconstrictor response of the pulmonary circulation to hypoxaemia and acidosis is different from that of the systemic vasculature and appears to be suited to matching of lung perfusion and ventilation [3]. Hypoxic areas of the lung have reduced blood flow by vasoconstriction [3]. When hypotension is induced in patients with normal pulmonary function, pulmonary artery pressure decreases and the effects of gravity put more blood through dependent areas where most of the shunt units are [6]. Until our administration of nitroglycerine in this patient, his hypertension had, in effect, protected his saturation.

Obesity has been shown to restrict ventilation, especially if the patient is in the supine position. Obesity augments the size of individual fat cells without increasing blood flow and fat tissue mass is thus hypoperfused [4]. In this case, these factors would also have affected our patient’s $S_{p}O_2$.

In conclusion, administration of drugs interfering with the HPV response, such as nitroglycerine, must be closely monitored, especially in patients with obesity and hypertension.

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