Severe myalgia associated with propofol sedation
doi: 10.1017/S0265021506001761

EDITOR:
Propofol is used when deep sedation, immobilization and quick emergence are required and is often used in children during magnetic resonance imaging (MRI) scans. Because the MRI scan is prolonged, a propofol infusion or repetitive small boluses are usually administered.

A 13-yr-old patient (weight 44 kg), underwent MRI scan of the head for evaluation of recurrent headaches. The patient was otherwise healthy and a neurological examination performed by a paediatric neurologist was unremarkable. The patient received during the MRI scan a total dose of 120 mg propofol and was fully awakened at the end of the scan. An hour later, the patient complained of progressive pain in both of her calves. A physical examination revealed severe disabling muscle pain in both legs. No other abnormalities were found. Full blood count, biochemical analysis, and creatine–phosphate kinase levels were within normal range. Urinalysis was normal. No fever or haemodynamic abnormalities were found. After 3 days the muscle pain began to decrease and the patient was discharged 2 days later with minor myalgia. The MRI scan of the head was normal. The patient’s guardians refused muscle biopsy.

Postoperative myalgia is a common complication which is usually associated with muscle relaxants and surgical stress. In the described case the patient was sedated solely with propofol for head MRI scan and developed severe myalgia.

There are two possible causes for this presentation: propofol infusion syndrome and toxic myopathy. Several reports described the ‘propofol infusion syndrome’, occurring in patients receiving prolonged high-dose infusions of propofol (>75 µg kg⁻¹ min⁻¹ for >24 h) [1–3]. Most of these cases occurred in critically ill patients. The syndrome is characterized by severe metabolic acidosis, myocardial dysfunction, rhabdomyolysis, myoglobinuria, renal failure, hypoxia and death. The mechanism of muscle toxicity is unknown. Muscle pathology reveals necrosis of skeletal and cardiac muscle. Discontinuation of propofol with supportive therapy for myoglobinuria, metabolic acidosis, hyperkalaemia and renal failure is the mainstay of treatment [4]. The pathogenesis of propofol infusion syndrome is not fully explained and potential mechanisms include lactic acidosis, metabolic acidosis and mitochondrial toxicity. Lactic acid production is caused by low cardiac output or a regional steal syndrome due to propofol influence as a cardiodepressant, cardiac failure or septic shock.

Metabolic acidosis may be caused by impaired hepatic lactate metabolism. Supportive evidence includes the development of a fatty or enlarged liver [5]. Intermediate dihydroxylated products of propofol are thought to have mitochondrial toxicity which could cause metabolic acidosis. Furthermore, there might be an occult mitochondrial dysfunction in the subjects who developed the syndrome [5,6].

Muscle tissue is highly sensitive to drugs and toxins because of its high metabolic activity and multiple potential sites for foreign substances to disrupt the energy-producing pathways. Children have reduced ability to metabolize or excrete drugs and thus have increased risk for toxic myopathy [7].

The exact incidence of toxic myopathy is unknown, but probably is often unrecognized. Early recognition of toxic myopathy is important since the likelihood of complete resolution is higher with early removal of the offending toxin. The diagnosis is based on lack of other known aetiology for the myopathy, lack of previous muscular symptoms and resolution of the symptoms after withdrawal of the suspected toxic agent. Drugs or toxins can produce mild symptoms of muscle pain and cramps, as in the patient we described, or cause a severe weakness, rhabdomyolysis, hyperkalaemia and renal failure [7]. In conclusion, this case of severe myalgia following propofol infusion might be caused by either...
a mild form of propofol infusion syndrome or toxic myopathy. Anaesthesia care providers should be aware of this complication and its causes.

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Paediatric renal transplantation: a single centre study
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EDITOR:

Renal transplantation is the definitive treatment of choice for children with end-stage renal failure. Clinical outcome indices in paediatric renal transplantation remain inferior to those in adults due to the higher incidence of acute tubular necrosis and graft loss from vascular thrombosis and primary non-function [1–3]. After Institutional Research Ethics Board approval, the anaesthetic and perioperative management of 34 consecutive patients who received a renal transplant at the Hospital for Sick Children, Toronto, Canada between January 2001 and August 2003 were reviewed. Results are expressed as mean ± SD.

There were 20 male and 14 female patients, with a mean age at transplant of 11.2 ± 5.3 yr (range: 2–18 yr) and weight of 36.1 ± 15.2 kg (range: 13–60 kg). There were five patients who weighed less than 15 kg. The aetiology of renal failure is listed in Table 1. Twenty-nine patients were receiving dialysis (17 haemodialysis, 12 peritoneal dialysis) at the time of transplantation. Five patients did not receive dialysis prior to renal transplantation as these patients were receiving donor organs from family members. Live-donor grafts were used in 56% of patients, 35% received a cadaveric adult kidney and 9% received a cadaveric paediatric kidney.

All patients were intubated and ventilated after induction of anaesthesia with intravenous agents: propofol (29 patients) or sodium thiopental (5 patients). Crystalloids, 5% albumin or red blood cell concentrate were given at 53 ± 24 mL kg\(^{-1}\) (range: 22–109 mL kg\(^{-1}\)) to maintain intravascular volume and achieve a central venous pressure (CVP) of 14 ± 2 mmHg (range: 11–19 mmHg) prior to release of renal vessel cross clamps. Ten patients (29%) received packed cells perioperatively to achieve a haemoglobin of 8–10 g dL\(^{-1}\). Mannitol (0.5–1.0 g kg\(^{-1}\)) and furosemide (1 mg kg\(^{-1}\)) were administered intravenously prior to

### Table 1. Aetiology of renal failure in transplant recipients.

<table>
<thead>
<tr>
<th>Cause of renal failure</th>
<th>Number</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obstructive uropathy</td>
<td>13</td>
<td>38</td>
</tr>
<tr>
<td>Glomerulonephritis</td>
<td>10</td>
<td>29</td>
</tr>
<tr>
<td>Renal dysplasia</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>Vesico-ureteric reflux disease</td>
<td>4</td>
<td>12</td>
</tr>
<tr>
<td>Congenital nephrotic syndrome</td>
<td>2</td>
<td>6</td>
</tr>
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<td>1</td>
<td>3</td>
</tr>
<tr>
<td>Total</td>
<td>34</td>
<td>100</td>
</tr>
</tbody>
</table>

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release of cross-clamps to ensure a brisk diuresis. Eleven patients (32%) required inotropic support with dopamine up to 10 µg kg⁻¹ min⁻¹ after release of cross clamps. Venous anastomosis sites were the iliac vessels (32 patients) and inferior vena cava/aorta (2 patients).

Graft survival was 94% at 1 yr. Renal vein thrombosis occurred in two living-related recipients each weighing less than 20 kg resulting in early graft loss. Venous thrombosis also occurred in one cadaveric adult kidney recipient (recipient weight <20 kg), which was successfully treated with anticoagulation therapy and an inferior vena cava filter. High-resolution Doppler ultrasound of the renal allograft was performed in the operating room after incision closure to identify all three cases of vascular thrombosis.

All patients were cared for postoperatively in a paediatric intensive care unit (ICU). Postoperative complications included pneumothorax (1 patient) and acute tubular necrosis (3 patients). One patient developed pulmonary oedema requiring tracheal reintubation and mechanical ventilation. Five patients remained intubated on admission to the paediatric ICU (three patients weighing <20 kg). One patient had Klippel–Feil syndrome, scoliosis and restrictive lung disease and required ventilation for 5 days. Three patients with focal segmental glomerulosclerosis required plasmapheresis postoperatively. Five patients required surgical exploration within 24 h postoperatively for venous thrombosis (3 cases) and bleeding (2 cases).

The rate of paediatric renal transplantation has been increasing in North America over the last 10 yr. With a concomitant increase in the incidence of end-stage renal disease, this trend is expected to continue. The benefits of renal transplantation in children are now well established and survival figures for older children are comparable to the adult population. A decade ago the 1-yr graft survival figures from the North American Paediatric Renal Transplant Co-operative Study was 79% [4]. More recent data demonstrates an improvement in graft survival to 92% [5]. In our study, the 1-yr graft survival rate was 94%.

Infants and small children weighing less than 25 kg are at highest risk for graft loss and mortality of any group undergoing renal transplantation [6]. Our experience was in keeping with this finding. All three episodes of renal vein thrombosis occurred in recipients less than 20 kg, two of which were organs from living-related donors and resulted in graft loss. The adult kidney can hold 300 mL of circulating blood volume and a significant portion of the infant’s cardiac output [7]. Despite the assumption that early graft loss may be more likely in this situation some studies have demonstrated that the increased mass of the adult-sized donor kidney may be protective for the infant recipient, provided there is no evidence of acute tubular necrosis [8]. Renal vein thrombosis was the single most important determining factor in graft failure in our study. High-resolution Doppler ultrasound of the renal allograft was used after incision closure in all cases to confirm forward diastolic flow and adequate perfusion, a technique that is well established in the literature [6].

Acute tubular necrosis is a major determinant of graft failure in infants and children. It renders the kidney more immunogenic and is associated with a higher chance of acute rejection. Reversal of acute rejection in the first year is crucial for long-term survival [4,5]. Episodes of hypoperfusion and ischaemia in the perioperative period likely contribute to delayed graft function and, therefore, immunogenic activation to a greater extent than previously realised. Immunogenic activation makes the donor graft susceptible to early and delayed graft loss secondary to an increase vulnerability to host immunogenic attack [9]. It is for this reason that optimization of physiologic variables to ensure prompt graft perfusion at the time of unclamping and in the immediate reperfusion interval that follows is essential.

It is in this regard that the role of the anaesthesiologist is important in contributing to graft survival [7,9]. Crystalloids, 5% albumin or red cell concentrate were administered to maintain intravascular volume and achieve a normal to high CVP prior to release of renal vessel clamps. Infants receiving an adult kidney need adequate volume to avoid inadequate perfusion, acute tubular necrosis and renal artery thrombosis. Therefore aggressive volume loading prior to unclamping may be necessary bearing in mind the small but real risk of pulmonary oedema. In our study, one patient developed pulmonary oedema requiring tracheal re-intubation and mechanical ventilation for less than 24 h. The current study is consistent with others in that 1-yr graft survival in paediatric patients is now over 90%. Despite this significant morbidity may be associated with paediatric renal transplantation, as 13 patients in our series experienced complications within the first 24 h after transplantation.

Our data demonstrates 1-yr graft survival rates of over 90%, however infant recipients may be at higher risk for graft failure. The consequence of early renal graft loss is devastating for the patient and family, especially in living-related donor cases. Vigilance and attention to detail in the anaesthetic and perioperative management remains pivotal in the success of paediatric renal transplantation.

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We believe that there is no reason why we should necessarily reflect different levels of hypnosis and comfort. It is well known that hypnotics against perceiving discomfort when the noxious stimuli which should be decisive compared to prediction of lack of response to verbal command prove that the EEG readings provide any safeguard against recall conferred by the seizure to a yet not measured extent. Moreover, as the author points out, in this patient group there is (even) less knowledge about the reliability and informative content of BIS-values than during conventional surgery. The authors fail to explain why BIS readings under these circumstances are valid for ECT treatment.

Studies [2] have shown that EEG-based monitoring of anaesthesia depth does not reliably predict response to noxious stimuli which should be decisive compared to prediction of lack of response to verbal command which might be better predicted by BIS. Even without studies this appears obvious based on clinical experience. Studies [3,4] showing there is a close link between discomfort relieving measures and EEG readings fail to prove that the EEG readings provide any safeguard against perceiving discomfort when the noxious stimulus is actually applied. It is well known that hypnotics produce different levels of EEG readings without necessarily reflecting different levels of hypnosis [5].

We believe that there is no reason why we should assume that the author’s suggestion for monitoring this problem should have attracted attention. ECT treatment, in particular if successful in terms of seizure duration, does confer amnesia which according to all clinical experience reaches as far back as the application of the cranial shock current. So, as a corollary, if you increase your dose of hypnotics for induction prior to ECT, you will predictably decrease your protection against recall conferred by the seizure to a yet not measured extent. Moreover, as the author points out, in this patient group there is (even) less knowledge about the reliability and informative content of BIS-values than during conventional surgery. The authors fail to explain why BIS readings under these circumstances are valid for ECT treatment.

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We believe that there is no reason why we should assume that the author’s suggestion for monitoring
would to any extent improve quality of therapy and outcome in ECT. Little, if anything, suggests that BIS-values after induction for ECT are more significant than clinical experience. We were also surprised that the authors claimed that all 109 patients left the ECT suite basically with the same blood pressure as when they entered. This leaves us somewhat puzzled since there are blood pressure swings almost invariably associated with ECT treatment.

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Postoperative blindness – a rare but devastating complication
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EDITOR:
Postoperative visual loss (POVL) is one of the most unexpected complications after surgery with an estimated incidence varying from 0.01 to 1% depending on the type of surgery [1–4]. Although POVL is observed after almost any type of surgery 67% and 10% of all reported cases occur after prone spine procedures and cardiopulmonary bypass cases, respectively [2,4]. The most common cause associated with POVL (81%) is ischaemic optic neuropathy (ION) of the anterior (AION) or posterior (PION) part of the optic nerve [3,5]; only 6% of POVL cases were diagnosed as central retinal artery occlusion (CRAO). ION results from an ischaemic insult of the optic nerve characterized by oedema or pallor around the optic nerve and is often associated with a decrease in pupillary response [1,6]. Clinically ION is manifested by an unilateral or bilateral acute/subacute loss of vision or visual acuity hours to days after surgery. The descriptions of the visual deficits range from transient blurred vision to total blindness on one or both eyes [3]. Although various therapy strategies or interventions including high-dose steroids, mannitol, hyperbaric oxygen and furosemide were attempted no treatment has proven efficacy [5]. Prognosis for complete recovery from ION is poor and patients are frequently left with extensive visual impairment.

Because of the lack of a beneficial treatment for ION and the poor prognosis prevention of POVL is important. Suggested preoperative risk factors include hypertension, diabetes, polycythaemia, smoking, renal failure, narrow-angle glaucoma, atherosclerotic vascular disease, collagen vascular disorders and variations in ocular anatomy or physiology. Furthermore, numerous intraoperative risk factors include prolonged prone positioning, hypotension, large blood loss, anaemia, extensive fluid administration and vasoactive drugs [1,2,5,6]. However, there is no experimental or clinical evidence that the proposed
risk factors are causally linked to the occurrence of POVL. Thus, there exist currently no evidence based prevention strategies.

This treatment dilemma, the incomplete knowledge about the etiology of ION and an increased incidence of POVL has led the American Society of Anesthesiologists (ASA) Committee on Professional Liability to develop the ‘ASA Postoperative Visual Loss Registry’ in July 1999. Aim of the ASA registry is to collect detailed information about POVL cases, determine possible risk factors for POVL and improve our understanding about the etiology of POVL. Currently 129 (June 2005) cases of POVL have been submitted to the ASA Registry [3]. In a preliminary evaluation of 113 POVL cases reported to the ASA Registry 71 occurred in middle-aged patients (median 50 yr) undergoing spine surgery and were caused by ION [3]. In more than 90% anaesthetic duration was 
\[>6\] h and in 85% the estimated blood loss was \[>1L\]. 9 cases of CRAO had significantly lower mean anaesthetic duration and estimated blood loss compared to ION cases. Importantly 14 patients with ION had their heads placed in Mayfield pins with their eyes free from external pressure indicating that isolated ION is not caused by pressure on the globe [3]. Recently, the American Society of Anesthesiologists Task Force on Perioperative Blindness issued a practice advisory for perioperative visual loss associated with spine surgery [7].

In summary, POVL is a rare and disastrous complication particularly associated with spine and cardiac surgery. Several suggested but not proven risk factors include intraoperative hypotension, anaemia and large blood losses or prolonged prone position indicating a multifactorial etiology of POVL. Until now, there are no evidence-based treatment or prevention strategies available.

What can we do as an anesthesiologist about POVL? Apparently there is an urgent need for all anesthesiologists to closely follow literature and gain knowledge about POVL; the brief booklet about POVL for anesthesiologists available on the ASA closed claims webpage offers a good summary of this phenomenon [8]. We should be aware that patients undergoing prolonged prone spinal surgery and cardiopulmonary bypass surgery with hypotension and anaemia may have an increased risk of postoperative visual loss secondary to ischaemic optic neuropathy. Patients with POVL should be evaluated by an ophthalmologist as quickly as possible; a gross exam (e.g., reading letter or counting fingers) does certainly not rule out scotoma or delicate visual field deficits as it occurs in ION.

Finally, every anesthesiologist is highly encouraged to report any case of POVL following non-ophthalmic surgery to the ASA POVL Registry (www.asaclosedclaims.org).

Further research is needed to improve our understanding about the etiology of POVL, to determine risk factors and to clarify the mechanisms of ION. Recently developed animal models for ION may hopefully improve our knowledge about the mechanisms of ION and offer the possibility to develop appropriate treatment strategies [9].

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References

Anaesthetists inability to assess depth of anaesthesia: why not use the IFT?

doi: 10.1017/S0265021506001815

EDITOR:
The article on subjective assessment of anaesthesia by Hadzidiakos and colleagues [1] is a fascinating glimpse into the ‘art’ of assessing ‘depth of anaesthesia’. As the authors [1] point out, ‘hypnosis’ (i.e. unconsciousness) is the major aim of general anaesthesia but we anaesthetists continue to try and quantify this state by using surrogate markers such as the clinical signs of anaesthesia or, more recently, some numerical surrogate provided by an electronic ‘Anaesthesia Brain Monitor’. Such monitors provide an output which, because of very wide inter-individual variability, can only be understood in terms of a patient’s probability of being conscious. Whatever number one chooses to aim for will represent either too much anaesthetic drug for some patients or not enough for others. I doubt that the average patient would relish the idea that they are about to be paralysed and undergo major surgery with even a 1% probability of being conscious, let alone a 10–20% probability!

While I agree that there is no ‘gold standard’ for defining depth of anaesthesia (we are not even clear if such an entity as ‘depth’ of anaesthesia exists) but there is a ‘gold standard’ for detecting the onset of consciousness when muscle relaxants are being used – the Isolated Forearm Technique (IFT) [2]. It is thus extremely disappointing to note that the authors continue to perpetuate the myth that the IFT can only be used for 30 min [1]. The IFT was first described by Tunstall [3] as a method of directly assessing the presence of consciousness during Caesarean section, up until delivery of the baby. After this point the tourniquet was deflated and anaesthesia was deepened. Since Dr Tunstall used a suxamethonium infusion, when the tourniquet was released the arm became paralysed. I had the privilege of training under Dr Tunstall in the 1970s and with his encouragement I investigated the IFT during non-obstetric surgery using non-depolarizing relaxants and reported in 1979 [4] that the IFT could be used very effectively during prolonged surgery (in one of these early cases I used the IFT for over 4 h in a patient having surgery for oesophageal varices). Since then many publications have described the technique in detail [5–8]. I would urge the authors, and readers in general, to read further about the technique. If any wish to try it for themselves I would add the caveat that the IFT does not work well with pancuronium and doses of rocuronium still need to be investigated.

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References

The effect of dexamethasone on recovery from cleft palate surgery

do: 10.1017/S026502150600175X

EDITOR:
Cleft palate repair is most frequently performed in children between 6 months and 10 yr. Cleft palate surgery may cause swelling, vomiting, pain and poor oral intake. Dexamethasone has combined antiemetic and anti-inflammatory effects, may decrease postoperative oedema and subsequently may improve oral intake [1–3]. Dexamethasone has been shown to reduce the incidence of vomiting by children after tonsillectomy [1,4,5]. We undertook this prospective double-blind randomized study to examine the effect of dexamethasone on recovery in children after cleft palate surgery.

After we obtained hospital Ethics Committee approval and written, informed consent of the parents, 86 children aged 6 months to 10-yr-old ASA physical status I or II, undergoing cleft palate repair, using a standardized anaesthetic technique, were studied. Immediately after the induction of anaesthesia, the patients were randomly allocated to one of two groups, dexamethasone group (n = 43) receiving dexamethasone 0.5 mg kg$^{-1}$ i.v. (intravenous), and the placebo group (n = 43) receiving normal saline. In addition to patient characteristics data, time to post anaesthesia care unit (PACU) discharge, first oral intake (delay between the arrival into PACU and the first oral intake), quality of oral intake (evaluated by the following scale 1: excellent, child requests food; 2: good, child accepts it when offered; 3: fair, child accepts it when coaxed; 4: poor, child refuses it) and vomiting were evaluated. There were no significant differences between the two groups with respect to age, weight, ASA physical status, duration of surgery or anaesthesia and PACU stay duration. The time to first oral intake was significantly shorter, and the quality of oral intake was significantly better, in the dexamethasone group. The incidence of postoperative vomiting was similar in the two groups. We conclude that dexamethasone 0.5 mg kg$^{-1}$ i.v. improves the quality of oral intake and shortens the time to first oral intake in children undergoing cleft palate surgery although further studies including a larger patient population should determine whether dexamethasone is effective in controlling postoperative vomiting for this type of surgery.

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References
Mitral valve surgery and acute renal failure

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EDITOR:

Acute renal failure (ARF) is a serious complication following cardiac operations performed with cardiopulmonary bypass (CPB) and carries a high mortality rate [1]. Prior studies have attempted to identify predictors of ARF or to develop risk stratification algorithms. Valve operations are an independent predictor of ARF [2,3]. To our knowledge, no study has attempted yet to find an independent association between ARF and a specific type of valve operation (i.e., repair or replacement).

The aim of this study is to define the incidence, predictors and mortality related to ARF after different types of mitral valve (MV) surgery.

From January 1998 to January 2003, we studied 1276 consecutive adult patients who underwent isolated MV surgery with CPB. We excluded from our population patients with dialysis dependence and combined procedures. MV repair was mostly performed with the edge-to-edge technique with the positioning of a rigid ring while patients who had mitral replacement mostly had a mechanical valve implanted.

All patients received a standard premedication and monitoring. Anaesthesia was induced in all patients with fentanyl–propofol–pancuronium, maintained with propofol, isoflurane, and additional doses of fentanyl. CPB was conducted with an institutional custom pack including a coated membrane oxygenator, with mild hypothermia (32–33°C). Non-pulsatile perfusion was used throughout the study, with perfusion maintained between 2 and 2.8 L min\(^{-1}\) m\(^{-2}\). The pumps were primed with crystalloid solution, mannitol 18% 0.5 g kg\(^{-1}\) formulated to achieve a hematocrit of 18% or more during CPB. Packed red blood cells were added to achieve the desired hematocrit of 18% or more during CPB. Packed red blood cells were added to achieve the desired hematocrit and as required by the clinical circumstance. Intermittent cold (4°C) blood cardioplegia was infused by means of heat exchanger and two roller pumps.

Perioperative ventricular dysfunction occurring after cardiac surgery and CPB was managed with heart rate and rhythm control, preload and afterload optimization and, when these manoeuvres were ineffective, with inotropic drugs. Dopamine was the first sympathomimetic drug used and when it was unable to resolve the low-output syndrome it was coupled with epinephrine and/or enoximone and/or an intra-aortic balloon pump. Prophylactic use of a balloon pump was performed in patients with functional MV disease and with ejection fraction <30%.

Prophylactic strategies such as hydration, dopamine, fenoldopam and mannitol were used according to anaesthesiologist’s preference. Loop diuretics have been administered early in the course of ARF to convert an oliguric to a nonoliguric state. ARF was defined as a postoperative 100% increase in serum creatinine (doubling from baseline values). Renal replacement therapy was initiated by the attending nephrologist and intensivist based on the clinical situation. Renal support was provided in all cases by continuous veno-venous haemofiltration (CVVH, Prisma CFM, Hospal Lyon, France) using high flux AN69 membranes with a membrane surface of 0.60 m\(^2\).

Statistical analysis. Data were analysed using the SAS statistics package. Dichotomous variables were compared using \(\chi^2\)-test with Yates correction. Continuous measures are expressed as mean ± SD unless otherwise indicated and were compared with a \(t\)-test for paired or unpaired data, as appropriate. A multivariate stepwise logistic regression was used to assess the independent correlates of ARF.

Of 1276 patients included in the study, 32 (2.5%) developed postoperative ARF. The incidence of ARF for MV replacement and MV repair was 8% (25/312) and 0.7% (7/964) respectively \((P < 0.001)\). The overall population was 57 ± 12.8-yr-old, 41% (528) female, 6.3% (81) with ejection fraction <40%. All perioperative clinical and patient characteristics are depicted in Table 1 together with a univariate analysis to study their association to ARF: patients who developed ARF had a higher incidence of preoperative comorbidities and perioperative complications.

At a multivariate analysis that included all factors in Table 1 with entry and exit values \(P < 0.05\), MV replacement was an independent risk factor for the development of postoperative ARF (odd ratio (OR): 4.0, 95% confidence interval (CI): 1.49–10.59, \(P < 0.01\)) together with low-output syndrome (OR: 13.7, 5.4–34.9, \(P < 0.01\)), emergency surgery (OR: 8.5, 1.4–52.2, \(P = 0.02\), creatinine > 124 \(\mu\)mol L\(^{-1}\)) (OR: 7.9, 2.8–22, \(P < 0.01\)), reopening for bleeding (OR: 4.5, 1.4–14.3, \(P = 0.01\)), diabetes (OR: 4.4, 1.08–18.2, \(P = 0.04\)) and age.
Hospital death occurred in 23/1276 (1.8%) patients: those who developed ARF had a 46.9% (15/32) incidence of death vs. 0.6% (8/1244) in the patients who did not develop ARF. ARF requiring renal replacement therapy occurred in 20 patients (1.6%): 3/964 (0.3%) in the MV repair group and 17/312 (5.4%) in the MV replacement group (P = 0.001). Patients who developed ARF requiring renal replacement therapy had a 65% incidence of death. Death occurred in 19/312 (6.1%) MV replacement and in 4/964 (0.4%) MV repair patients (P = 0.0001).

The main result of our study is that finding the MV replacement is an independent risk factor for the development of ARF after MV surgery (OR: 4.0; 95% CI: 1.5–10.6). The other risk factors for this complication in our study population were perioperative low-output syndrome, emergency operation, preoperative renal impairment, reoperation for bleeding, diabetes and age. The aetiology of ARF after cardiac surgery is multifactorial. Factors include occult renal ischaemia, renal injury from endo- and exotoxins, and decreased renal reserve; MV surgery can be associated with an ischaemic injury to the kidneys due to a low-output syndrome. An alternative mechanism of renal ischaemia during MV surgery is embolic.

In conclusion, our study identifies risk factors for the development of ARF in MV surgery and, for the first time, shows that MV replacement is an independent risk factor for this complication.

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References

Table 1. Perioperative variables and their association to the development of ARF following mitral valve surgery.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Overall population (n = 1276)</th>
<th>ARF (n = 32)</th>
<th>No ARF (n = 1244)</th>
<th>P</th>
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<tbody>
<tr>
<td>Age (mean ± SD)</td>
<td>57 ± 12.9</td>
<td>67 ± 8.5</td>
<td>57 ± 12.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Female gender</td>
<td>528 (41%)</td>
<td>10 (31%)</td>
<td>518 (41%)</td>
<td>0.3</td>
</tr>
<tr>
<td>Creatinine &gt; 124 µmol L⁻¹</td>
<td>64 (5.0%)</td>
<td>12 (37%)</td>
<td>52 (4.2%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Low ejection fraction</td>
<td>81 (6.3%)</td>
<td>9 (28%)</td>
<td>72 (5.8%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Diabetes</td>
<td>37 (2.9%)</td>
<td>4 (12%)</td>
<td>33 (2.7%)</td>
<td>0.01</td>
</tr>
<tr>
<td>COPD</td>
<td>121 (9.5%)</td>
<td>5 (16%)</td>
<td>116 (9.3%)</td>
<td>0.2</td>
</tr>
<tr>
<td>Stroke</td>
<td>69 (5.4%)</td>
<td>1 (3.1%)</td>
<td>68 (5.5%)</td>
<td>0.47</td>
</tr>
<tr>
<td>Prior cardiac surgery</td>
<td>131 (10%)</td>
<td>14 (44%)</td>
<td>117 (9.4%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Emergency surgery</td>
<td>10 (0.8%)</td>
<td>5 (16%)</td>
<td>5 (0.4%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>IABP device</td>
<td>50 (3.9%)</td>
<td>8 (25%)</td>
<td>42 (3.4%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Duration of CPB (mean ± SD)</td>
<td>73 ± 26.3</td>
<td>109 ± 34.9</td>
<td>72 ± 24.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mitral valve repair</td>
<td>964 (75%)</td>
<td>7 (22%)</td>
<td>957 (77%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Mitral valve replacement</td>
<td>312 (24%)</td>
<td>25 (78%)</td>
<td>287 (23%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Blood losses &gt; 1000 mL</td>
<td>80 (6.3%)</td>
<td>10 (31%)</td>
<td>70 (5.6%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Blood transfusion</td>
<td>382 (30%)</td>
<td>27 (84%)</td>
<td>355 (28%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Re-operation for bleeding</td>
<td>41 (3.2%)</td>
<td>9 (28%)</td>
<td>32 (2.6%)</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Low-output syndrome</td>
<td>147 (11%)</td>
<td>24 (75%)</td>
<td>123 (9.8%)</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

ARF: acute renal failure; COPD: chronic obstructive pulmonary disease; IABP: intraaortic ballon pump; CPB: cardiopulmonary bypass.