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Reply:

We thank Dr. Hare for his thoughtful comments regarding our study. His main concern relates to the impact of the higher doses of noradrenaline and phenylephrine administered to patients in the intravenous nitroglycerin group, and a possible confounding influence on the relationship between intravenous nitroglycerin and the cerebral oxygen saturation. Acknowledging the physiological value of his hypothesis, we reanalyzed our data with two separate repeated analyses of covariance models including group, time and vasopressor (one model with noradrenaline and one with phenylephrine). In each model, the interactions between the vasopressor and the two other variables were non-significant. A significant effect was found only for noradrenaline on the right cerebral oxygen saturation, which had no influence over time on cerebral saturations related to intravenous nitroglycerin. The Table shows the P values of the differences in cerebral saturations between groups at different times, with or without noradrenaline. Even in the model including noradrenaline, patients in the intravenous nitroglycerin group better maintained their cerebral saturation over time compared to patients in the placebo group, whose cerebral saturation decreased over time. Therefore, we believe that the use of intravenous nitro-

TABLE Contrast analyses of a repeated analyses of covariance comparing P values with and without the effect of NA

	P-value – pairwise comparisons of times for each groups (adjusted by NA)	P-value – pairwise comparisons of times for each groups (unadjusted)
Placebo: pre nitro <i>vs</i> pre CPB	0.3520	0.4187
Placebo: pre nitro <i>vs</i> post CPB	< 0.0001	0.0004
Placebo: pre CPB <i>vs</i> post CPB	0.0003	0.0068
Nitro: pre nitro <i>vs</i> pre CPB	0.4871	0.1850
Nitro: pre nitro <i>vs</i> post CPB	0.5847	0.4934
Nitro: pre CPB <i>vs</i> post CPB	0.8814	0.7663

CPB = cardiopulmonary bypass, NA = noradrenaline.

glycerin itself was associated with better maintenance of cerebral oxygen saturation under the conditions of our study. Furthermore, only one patient had a circulatory arrest during his surgery, which should not have impacted significantly our results. Finally, we agree that potential negative impact of intravenous nitroglycerin should not be neglected. The higher postoperative CK-MB values and the increase postoperative bleeding observed in the experimental group are concerning. If these effects are shown to be related to the use of nitroglycerin in further studies, the potential benefits for improved brain oxygenation will have to be balanced against the risks of this intervention. This is why we concluded in our article that: “Further studies are warranted to determine if the potentially harmful effects detected in the present study are real and significant.”

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Anesthetic considerations for a steroid-dependent high-risk patient undergoing minimally invasive cardiac surgery

To the Editor:

A minimally invasive cardiac surgery (MICS) approach to mitral valve surgery and atrial septal defect (ASD) repair provides advantages of a smaller incision, less trauma, and more rapid recovery, in addition to a shorter hospital length of stay.¹ The anesthetic requirements for MICS procedures are unique and involve special considerations quite different from

conventional cardiac surgery. An MICS approach utilizes one-lung ventilation (OLV) for surgical exposure, transesophageal echocardiographic (TEE) guidance for placement of cannulae,² and regional techniques for postoperative pain management. Achieving these anesthetic goals may be challenging.³

A 54-yr-old female patient weighing 80 kg presented with shortness of breath. She was diagnosed with right-sided congestive heart failure from a large ostium secundum ASD and severe mitral regurgitation (MR). Her past history was notable for ulcerative colitis for which she underwent colectomy at nine years of age and required long-term steroid therapy. Preoperative examination revealed cushingoid facies, a buffalo hump, and bilateral pitting edema of the feet. Her blood pressure and heart rate were 104/78 mmHg and 94 beats·min⁻¹ respectively. The patient was considered a difficult intubation due to her marked cushingoid facies, short neck, and limited mouth opening. Investigations revealed pulmonary hypertension with a pulmonary artery pressure of 52/23 mmHg. Transthoracic echocardiography showed severe MR and a large ostium secundum defect (1.82 cm), with a dilated right atrium, dilated right ventricle, and dilated left atrium.

The patient was scheduled for mitral valve replacement and ASD repair using a pericardial patch through a right mini-thoracotomy. The patient had marked osteoporosis and a thin sternum, and we therefore favoured a minimally invasive technique so as to avoid sternotomy and possible sternal infection and wound dehiscence. Deflation of the right lung was required to facilitate right heart exposure and achieve optimum surgical access. While we usually insert a double lumen tube (DLT) to provide OLV for MICS procedures, bronchial blockers are considered when DLT insertion is relatively contraindicated.⁴ As laryngoscopy revealed an oropharyngeal class III airway which was mildly edematous, we chose the latter approach. The patient's trachea was intubated with a 7.0-mm endotracheal tube. Attempts to place a bronchial blocker through the single lumen tube failed. Since the right lung could not be isolated, we elected to initiate cardiopulmonary bypass (CPB) early to allow surgical access and facilitate exposure of the right heart. Cardiopulmonary bypass was initiated through femoro-femoral bypass. A single venous cannula was placed through the femoral vein into the right atrium using a Seldinger-based technique under TEE guidance. The total pump time was 248 min and ventricular fibrillation time was 181 min. Following surgery, the patient was weaned from CPB while receiving dobutamine and epinephrine infusions and her trachea was

extubated six hours postoperatively. In this case, both intrathecal opioids and thoracic paravertebral blocks were avoided due to a long standing buffalo hump with spinal deformity, so intravenous patient-controlled analgesia was used. Unfortunately, our patient suffered fractured ribs as a result of the thoracotomy. However, this did not delay the weaning process and wound healing. Her recovery was uneventful.

Although a small thoracotomy for MICS may seem less painful than full sternotomy, Walther *et al.*⁵ reported that a lateral thoracotomy is more painful than conventional sternotomy in the first 48 hr postoperatively following MICS procedures. Various techniques for pain management following MICS are described in the literature. Due to the potential for epidural hematoma formation with epidural blocks, we generally use intrathecal opioids, thoracic paravertebral blocks, or patient controlled analgesia for postoperative pain management. The latter option provided the safest approach in this case, as a result of the patient's pre-existing buffalo hump and spinal deformity.

Minimally invasive cardiac surgery is gaining popularity for cardiac surgery. The anesthetic plan must take into consideration the possibility of conversion to full sternotomy if OLV fails as a result of poor oxygenation or hemodynamic instability. Further studies are warranted to compare outcomes of conventional and MICS techniques involving high risk cases. Meanwhile, we continue to tailor our anesthetic practice to procedural and patient needs, as anesthetic management plays a key role in the success of MICS procedures.

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Ondansetron and carpopedal spasm

To the Editor:

Ondansetron, a serotonin (5-HT₃) antagonist, is claimed to lack direct blocking action on the dopaminergic system; therefore, it was considered to be free of extrapyramidal symptoms (EPS). However, ondansetron-associated EPS have been reported on several occasions.^{1,2} We describe an unusual presentation of ondansetron-induced EPS characterized by carpopedal spasm.

A 44-yr-old woman with a history of acoustic neuroma underwent removal of a craniotomy plate. The anesthetic induction (propofol, midazolam, succinylcholine, fentanyl) and the course of her anesthesia (isoflurane, hydromorphone) were uneventful. As prophylaxis for nausea she received ondansetron 8 mg *iv* in divided doses, at the beginning and end of surgery. During emergence she was hyperventilating. Following extubation her hands were in a flexed and fisted position and her legs were in plantar flexion (carpopedal spasm) (Figure). She was noted to be sweating profusely and a dyskinetic tremor of her entire body was observed. In light of the patient's hyperventilation this was interpreted as hypocalcemia, and calcium chloride 200 mg *iv* was given without effect. Shortly thereafter, her eyes were noted to be rolling up (oculogyric crises). At that point a dystonic reaction to ondansetron was suspected and 25 mg of diphenhydramine was given without effect. The patient was responsive, cooperative, and following commands. In the recovery room her room air oxyhemoglobin saturation was > 95% and body temperature was 36.6°C. Postoperatively, carpopedal spasm dominated the clinical presentation. She was awake, alert and oriented. On examination she was unable to relax her hands and feet; however, she had intact strength in all four extremities, and Babinski signs were absent. Deep tendon reflexes were intact and symmetric. Her sensory exam was intact to light touch, vibratory, and pinprick sensation and her tongue was in the midline. Finger-to-nose and heel-to-shin examinations were bilaterally intact. Serum

FIGURE Carpopedal spasm induced by ondansetron.

electrolytes were within normal limits. Because the patient had pronounced twitching of the corner of the mouth (right hemi-facial spasm/oromandibular dystonia), a blink reflex testing by electromyography was ordered to rule out facial nerve abnormalities. Both facial and trigeminal nerve conduction studies were normal. A magnetic resonance imaging scan obtained before surgery was normal, and was not repeated postoperatively. Dystonic posture of both wrists and fingers resolved by the following morning at which point she was discharged.

We describe a rare EPS reaction to ondansetron with the clinical picture dominated by protracted carpopedal spasm. Despite the fact that ondansetron has no clear-cut ability to block dopamine receptors (an effect necessary to induce EPS),³ anti-dopaminergic adverse effects (EPS) have been described, especially