

epiphyses, which had led to bilateral hip osteoarthritis. Preoperative examination revealed an excess of submandibular soft tissue, Mallampati grade 3 mouth opening and a thyromental distance of 6 cm.

She was adamant in wanting general anaesthesia for the procedure, but given her risk of oesophageal reflux, this would have mandated intubation, and difficult airway equipment (specifically fibre-optic bronchoscopy) was not available at the Independent Sector Treatment Centre to which she had presented. Fortunately, she gave competent consent to sedation, once the nature, purpose, risks and consequences of sedation had been explained to her.

After placement of standard monitoring equipment, a 20-G cannula was inserted into the dorsum of her left hand; 5 L min<sup>-1</sup> of supplemental oxygen was administered by face mask. End-tidal carbon dioxide monitoring was used to assess respiratory rate. In all, 50 µg fentanyl, followed by 50 mg boluses of propofol (200 mg in total) were administered to achieve sedation. The patient remained self-ventilating and rousable only to gentle physical stimuli. In all, 5 mL 0.5% bupivacaine mixed with 40 mg methylprednisolone were injected into each hip by a consultant orthopaedic surgeon (using a 24-G Whitacre spinal needle) prior to bilateral hip manipulation. Recovery was uneventful. She was discharged from hospital 3 h after the procedure.

WDS (congenital supra-bulbar paresis) is a form of cerebral palsy that was first described in 1956 [1], occurring as a result of congenital (heterogeneous, predominantly X-linked), bilateral perisylvian cortical dysfunction [2,3]. It is not an uncommon form of cerebral palsy, but it is underdiagnosed [4]. WDS displays variable expression, but is characterized by supra-bulbar paresis (100% prevalence, causing disordered oesophageal motility, speech difficulty, drooling, gastro-oesophageal reflux (41%) and aspiration), mild four limb pyramidal

tetraplegia (91%), cognitive (81%) and behavioural (41%) impairments, and epilepsy (28%).

Surgery may be required in childhood to insert grommets or gastrostomy tubes, or to correct congenital defects such as cleft palate or contractures. A multidisciplinary approach to perioperative care is advocated. Preoperative communication may be difficult, although adult patients with WDS should not be assumed to be incompetent to give consent for treatment [5]. Preoperative oral clonidine (5 µg kg<sup>-1</sup>) may be beneficial as a sedative and antisialogogue, and antacid therapy should be considered. Without additional sedation, regional anaesthesia may be compromised by behavioural difficulties. General anaesthesia necessitates tracheal intubation, due to the high risk of gastro-oesophageal reflux; this procedure may be complicated by palatal abnormalities (4%), dental problems (20%), jaw contractures (9%) or micrognathia (1.5–15%). Extubation should be performed with the patient awake, and in the left lateral or semi-recumbent positions.

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## Intralipid infusion in rabbit asphyxial pulseless electrical activity: a pilot study

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### EDITOR:

Augmenting conventional resuscitative efforts with infusion of lipid emulsions has resulted in successful

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resuscitation from intractable local anaesthetic-induced cardiac arrest in a number of recent case reports. These follow animal studies demonstrating efficacy for lipid infusion in local anaesthetic-induced cardio-toxicity [1] and other lipid-soluble drug toxidromes [2]. Two mechanisms of action have been forwarded as the basis for the observed beneficial effects of lipid infusion. Weinberg's 'lipid

sink' hypothesis [3] proposes reduced tissue binding by re-establishing equilibrium in a plasma-lipid phase. Additional investigators have proposed a metabolic stimulant effect with enhanced myocardial lipid utilization resulting in augmented cardiac performance. The premise of the latter garners potential benefit in non-lipophilic-toxin-associated cardiac arrest. Accordingly, we decided to explore the hypothesis that lipid infusion might result in improved resuscitation outcomes in an intact animal model of asphyxial cardiac arrest.

With the approval of the Ruakura Animal Ethics Committee (Ruakura Animal Research Centre, Hamilton, New Zealand), 16 sedated (ketamine  $50 \text{ mg kg}^{-1}$ , xylazine  $4 \text{ mg kg}^{-1}$  via intramuscular injection) adult (age 95–135 days) New Zealand White rabbits were studied. Animal care and husbandry was in accord with regulations set by the institutional Ethics Committee. All animals underwent arterial (connected in standard fashion to Hewlett-Packard 78834A neonatal monitor; Hewlett-Packard, Palo Alto, CA, USA) and venous cannulation, continuous electrocardiogram (ECG) monitoring, and formal tracheostomy with tracheal intubation under direct vision. After completion of invasive procedures during which time animals breathed room air, control animals received an intravenous (i.v.) infusion of  $3 \text{ mL kg}^{-1}$  0.9% saline solution over a 2-min period. Test animals received  $3 \text{ mL kg}^{-1}$  Intralipid 20% over an identical period. All fluids were pre-warmed to  $37^\circ\text{C}$  prior to delivery.

Respiratory arrest was induced via i.v. administration of vecuronium  $0.1 \text{ mg kg}^{-1}$ . Ensuing pulseless electrical activity (PEA) was defined by the development of mean arterial pressure (MAP)  $\leq 20 \text{ mmHg}$  in the presence of potentially perfusing cardiac rhythm on ECG. Two minutes after the onset of PEA, we began resuscitative measures with manual external chest compressions at 160–180 compressions per minute, and pressure-controlled mechanical ventilation (100% oxygen at  $10 \text{ cmH}_2\text{O}$  pressure-controlled ventilation at 12 breaths per minute) via a Nuffield series 200 paediatric ventilator (Penlon Ltd, Abington, England). Five minutes after the commencement of resuscitative efforts, epinephrine  $1 \text{ mL kg}^{-1}$  1:10 000 was administered, and repeated 1 min later if spontaneous circulation had not been restored. External chest compressions were continued to 15 min in animals failing to achieve return of spontaneous circulation (ROSC).

ROSC was defined by maintenance of MAP  $> 35 \text{ mmHg}$  (equivalent to 50% referenced MAP) in the absence of external chest compressions for a period of greater than 1 min. On achieving ROSC, animals were monitored to 15 min with acquisition

of haemodynamic parameters (pulse rate, MAP) at 2.5-min intervals. External chest compressions were not re-instituted in animals achieving ROSC but later exhibiting a second period of cardiac arrest. At the termination of the monitoring interval, all animals were killed via i.v. administration of  $3 \text{ mL}$  ( $300 \text{ mg mL}^{-1}$ ) pentobarbitone.

Time to PEA was  $244 \pm 74 \text{ s}$  in the saline group and  $231 \pm 76 \text{ s}$  in the Intralipid-treated group. Cardiac rhythm at PEA in the saline group was: sinus tachycardia in two animals, 2:1 atrio-ventricular block in three animals, sinus bradycardia in two animals and junctional bradycardia in one animal. Cardiac rhythm at PEA in the Intralipid-treated group was: sinus tachycardia in four animals, 2:1 atrio-ventricular block in two animals and sinus bradycardia in two animals. Four animals in the saline group ( $n = 8$ ), and seven animals in the Intralipid group ( $n = 8$ ) exhibited ROSC and survived to protocol termination ( $P = 0.106$ ). No difference in time to ROSC in surviving animals was observed. Of the four animals achieving ROSC in the saline group, one animal did so prior to epinephrine administration. Similarly, one animal in the Intralipid-treated group achieved ROSC prior to epinephrine administration.

These data demonstrate no statistically significant difference in ROSC for Intralipid-pre-treated rabbits undergoing resuscitation from asphyxial PEA. The study hypothesis is however not convincingly rejected given the limitations of the experiment. Primarily, due to the preliminary nature of the study and limited data on which to base sample size estimation, the study is insufficiently powered to detect lesser differences in resuscitation outcome. Furthermore, administration of Intralipid as pre-treatment rather than intra-arrest limits generalization of study findings to any clinically encountered arrest situations. These data may however be gainfully employed to power future studies investigating the potential utility of lipid infusion in non-lipophilic-toxin-induced cardiac arrest models.

The prospect of improved outcome in PEA secondary to hypoxic cardiac stunning via augmenting myocardial energetics through administration of preferential myocardial substrates is intriguing. Support for lipid administration in animal models of cardiac arrest exists. Augmented contractile performance associated with increased myocardial high-energy phosphate content has been demonstrated in isolated rabbit hearts following myocardial stunning when Intralipid was administered during reperfusion [4]. Furthermore, Intralipid infusion both into bypass perfusate, and i.v. following return of coronary flow, has been shown to improve haemodynamic parameters in dogs undergoing

cardiopulmonary bypass following ischaemic cardiac arrest [5]. More recently, Stehr and colleagues [6] have demonstrated an independent positive inotropic effect for lipid emulsion in bupivacaine-induced cardiac depression in rat hearts. Given that myocardial substrate preference under conditions of increased lactate concentration is known to shift toward oxidation of free fatty acids [7], improved outcomes with lipid infusion in cardiac arrest secondary to generalized myocardial ischaemia may not be unexpected. Further studies addressing the beneficial mechanism of action of lipid emulsions in local anaesthetic and non-lipophilic toxin-induced arrest models are warranted.

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## Propofol for the management of glycine-mediated excitatory symptoms of TURP syndrome

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#### EDITOR:

Fluid overload and hyponatraemia are central to the development of the TransUrethral Resection of Prostate (TURP) syndrome after transurethral prostate surgery [1]. Hyperammonaemia and hyperglycinaemia can also be part of this syndrome [2,3]. Hyperammonaemia or hyperglycinaemia usually presents with central nervous system (CNS) depression although hyperglycinaemia exhibits a CNS excitatory action through its positive action on *N*-methyl *D*-aspartate (NMDA) receptors [4]. We describe a patient who developed CNS excitation following

TURP surgery possibly because of hyperglycinaemia and responded to propofol sedation.

A 72-yr-old male weighing 65 kg and with a history of controlled hypertension for 15 yr underwent TURP surgery under a subarachnoid block. His medication (diltiazem 30 mg and metoprolol 50 mg each once daily) was continued till the day of surgery. He fasted overnight and was premedicated with alprazolam 0.25 mg on the night before and the morning of the day of surgery. On examination, his heart rate was 64 beats min<sup>-1</sup> and blood pressure 140/70 mmHg. He had a normal electrocardiogram (ECG) and haematology, biochemistry and coagulation profiles were within the normal limits. Transthoracic echocardiogram revealed an ejection fraction of 65% with normal pulmonary arterial pressure and elevated left ventricular filling pressure.

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