Future of robotic anaesthesia

Editor—I enjoyed reading the study by Hemmerling and colleagues.1 A robotic technique would fill a gadget loving, coffee drinking anaesthetist with enthusiasm, but I would like to understand the fundamental point of what are we trying to achieve by using this technique of tracheal intubation. I wonder whether the development of this technique is driven by increasing patient safety, or reducing airway complications, challenging the time from induction of anaesthesia to start of surgery, and decreasing morbidity and mortality. I believe that this line of research does not carry much importance without a clinically important hypothesis. I do appreciate the effort involved in this project; it is an interesting academic article, and that is where it stands, without any hypothesized clinical use in the future.

Declaration of interest

None declared.

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Reply from the authors

Editor—We thank Dr Kanagarathnam for his questions related to our study.1 Indeed, I am reminded of a similar response many years ago when the DaVinci robotic system was presented: what for, will it replace doctors, and what is its benefit? We presented a new concept of robot-assisted tracheal intubation in a pilot study. Where do we go from here? Many more technical developments will be required which will focus on exactly the questions raised: can this lead to reduced morbidity or reduced workload for the anaesthetist? Can it lead to higher precision of human gestures or can it even lead to a replacement of human gestures as aesthetist? Can it lead to higher precision of human gestures, lead to reduced morbidity or reduced workload for the an-

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Declaration of interest

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Hallucinations associated with cerebrospinal fluid leakage after a lumbar puncture

Editor—We read with interest the article by Loures and colleagues1 describing a patient who developed a psychiatric-like illness after cerebrospinal fluid (CSF) leakage after a dural puncture. We experienced a similar case with a 10-yr-old boy who was suspected of having mitochondria disease and underwent a lumbar puncture as a part of his work-up. Just after the procedure, he developed a headache, and CSF leakage was confirmed. After the leakage was treated, he had a continuing headache and, notably, developed hallucinations. In addition to his altered mental status, he had neck stiffness. We checked possible causes of the psychiatric change including blood sugar, ammonia, lactate, electrolytes, calcium, liver enzymes, and markers of kidney function and infection, all of which were normal. Brain computed tomography did not show any change that could explain his mental change. We concluded that his hallucinations were the result of intracranial hypotension due to CSF leakage after the lumbar puncture. Treatment with acetaminophen, rehydration, and bed rest were initiated, and his mental status improved progressively within hours. Our case supports the idea suggested by Loures and colleagues1 that psychiatric symptoms can occur as a result of CSF leakage after a lumbar puncture. However, there are differences between our case and theirs. First, the main psychiatric symptom was hallucinations in our patient and behavioural dysfunction, disinhibition, and impulsivity in theirs. Secondly, the onset and period to remission of the psychiatric
symptoms: onset 4 days after lumbar puncture and resolution within hours after treatment in our patient vs 4 weeks until onset and several days until remission with treatment in theirs. The imaging findings were unremarkable in our patient, whereas several findings, including downward displacement of the brainstem, were identified in their patient. Based on the findings in these cases, we conclude that (i) psychiatric symptoms can be associated with CSF leakage and (ii) the clinical and radiological aspects of the cases can vary.

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Reply from the authors
Editor—We read with great interest the report by Dr Y. Kakisaka and colleagues of a paediatric case similar to ours.1 Their report highlights the fact that post-dural puncture headache is not always characterized by orthostatic headache associated with neck stiffness, tinnitus, hypoacusia, photophobia, or nausea.2 Clinical presentation can sometimes be atypical and patients can present with neuropsychiatric symptoms. The diagnosis may be particularly difficult to make. Computed tomography scan is of limited value for the diagnosis of the perforation as illustrated in their report. Sometimes, images of a subdural fluid collection, obliteration of subdural cistern, and ventricular collapse can be seen and suggest an intracranial hypotension, but this is not always the case.3 Cranial magnetic resonance imaging (MRI) with gadolinium injection is the radiological test of choice. Images will show a diffuse dural pachymeningeal enhancement, reflecting the presence of small thin-walled dilated blood vessels in the subdural space.4 We would therefore recommend prompt neurological evaluation (cranial MRI with contrast injection) for patients who present with neuropsychiatric symptoms after dural perforation.

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None declared.

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Failure of esmolol to control tachycardia associated with thyroid storm after subtotal thyroidectomy

Editor—Thyroid storm is a potentially life-threatening disorder marked by fever, sweating, and tachycardia. It usually requires treatment with β-blocker. Esmolol, a cardioselective β-blocker, has been shown effectively to control tachycardia in patients with thyroid storm.5,6 Here, we describe a case in which esmolol therapy did not control sinus tachycardia in a postoperative patient with thyroid storm.

A 33-yr-old, 56 kg Chinese female had progressive swelling in the neck for 7 yr. She had been diagnosed with hyperthyroidism 4 yr previously. Because of medical treatment failure, subtotal thyroidectomy was performed under general anaesthesia using propofol and sufentanil infusions. Anaesthesia and surgery were uneventful, and the patient was transferred to the recovery room. After 30 min, she regained consciousness and adequate respiratory strength. However, she was febrile (38.6°C), restless, and had profuse sweating despite adequate analgesia. Arterial pressure was 156/107 mm Hg and an ECG showed a sinus tachycardia (152 beats min⁻¹). As thyroid storm was suspected, esmolol 30 mg was given i.v. over 1 min, followed by midazolam 5 mg. Ice packs and alcohol sponging were used to lower body temperature, methimazole 20 mg was given by the nasogastric tube, and hydrocortisone 100 mg and nicardipine 0.25 mg were administered i.v. After 20 min, the temperature had decreased to 37.4°C, but tachycardia and hypertension persisted. A further dose of esmolol 30 mg was given, followed by an infusion at an initial dose of 50 µg kg⁻¹ min⁻¹, titrated up to 300 µg kg⁻¹ min⁻¹ (the maximum recommended dosage) over a 50 min period. However, the patient remained tachycardic and hypertensive. At that time, no signs and symptoms of congestive