Narcotrend-assisted propofol/remifentanil anaesthesia for prevention of awareness

Editor-I read with interest about the comparison of the use of Narcotrend and clinical assessment in judging the depth of anaesthesia while using total i.v. anaesthesia (TIVA).¹ I fully agree with the authors that the use of clinical assessment alone would lead to a greater deviation from a defined target while running TIVA. The Narcotrend, a computer-based EEG programme, is easy to use and has low running costs, besides in terms of prediction probability, the performance of the Narcotrend index and the bispectral index to predict propofol effect-site concentrations was comparable. I recently conducted a small study to evaluate the number of cases who developed awareness under anaesthesia, out of the 25 anaesthetists who replied, five had cases of awareness under anaesthesia, and out of these five, four were under TIVA. The most significant of these cases was a 52-yr-old lady having an abdominal hysterectomy, anaesthetized by myself using target-controlled infusion (TCI) with propofol and remifentanil. Despite using a $1 \ \mu g \ kg^{-1}$ bolus of remifentanil followed by an infusion of $0.2 \ \mu g \ kg^{-1} \ h^{-1}$ and running propofol at a target-controlled level of $6 \mu g m l^{-1}$, clinically evaluating that the patient is asleep such as loss of eyelash reflex, at induction, fall in arterial pressure and heart rate, during maintenance of anaesthesia, the patient complained about awareness under anaesthesia for a short while after induction. Since awareness under anaesthesia is indeed a serious issue, I would indeed welcome the use of Narcotrend if indeed it could decrease the incidence of awareness, and decrease incidence of nausea and vomiting as stated by the authors.

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Editor—I thank Dr D'Mello for his interesting comment. With respect to the case of awareness he mentioned, one may speculate that the patient experienced a period of wakefulness shortly after induction because propofol infusion was stopped due to the automatic lockout interval of the TCI device. This lockout mechanism is automatically activated, when the plasma target concentration is reduced. As it was mentioned, propofol anaesthesia was induced rapidly by achieving a plasma target of $6 \,\mu g \,m l^{-1}$. It is known from the literature that this results in adequate hypnosis after about 60 s. I guess a lower target plasma concentration was used after intubation; thereby, the automatic lockout interval was activated.

We agree with Dr D'Mello that most probably measuring the cerebral pharmacodynamics of propofol by the Narcotrend (or even any other device using the electroencephalogram) would have resulted in indicating a low level of hypnosis during this time. However, it is known that especially during the transition from consciousness to unconsciousness, the range of the Narcotrend level differs markedly inter-individually.² Therefore, it is difficult to define a threshold level differentiating the conscious and the unconscious state precisely in the individual patient. This holds true for all the other monitors of 'depth of anaesthesia', too.³ Therefore, it remains a challenge to clearly identify periods of wakefulness during general anaesthesia. However, I am sure using an EEG monitor, using TCI devices which include the effect-site concentration rather than the plasma concentration and careful clinical observation of the patient together will help to avoid periods of awareness.⁴

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Cardiac arrest during induction of anaesthesia in a child on long-term amphetamine therapy

Editor—The number of children on chronic amphetamine treatment for attention deficit hyperactivity disorder (ADHD) has dramatically increased during the last decade. Although several previous case reports¹ have described serious adverse reactions during general anaesthesia in adult patients on chronic amphetamine, very little is known about perioperative problems with paediatric patients. We report a case in which a 10-yr-old child on long-term methyphenidate (MPH) therapy presented a cardiac arrest during induction of general anaesthesia.

A 10-yr-old male child was undergoing an ambulatory laser therapy of a haemangioma on the face. The patient

had been taking MPH daily for 4 yr because of ADHD. Anaesthesia was induced by mask with sevoflurane. Arterial pressure and heart rate remained stable and oxygen saturation was 100%. After i.v. access was established, propofol 2 mg kg⁻¹ and alfentanil 10 μ g kg⁻¹ were administered. Immediately after, the child developed a severe bradycardia followed by asystole. I.V. atropine 0.5 mg was given twice and external chest massage was performed. Normal cardiac rhythm with correct haemo-dynamic measures was restored 30 s after the start of the cardiopulmonary resuscitation. The planned operation was continued. Operation and emergence were uneventful. The patient was taken to the post-anaesthesia care unit where his heart rate remained stable at 90 beats min⁻¹. He was discharged home on the same day.

Amphetamines are indirect sympathetic amines with powerful central nervous system stimulation activity and peripheral α and β actions. Chronic administration can result in depletion of norepinephrine and dopamine storage. This decreased reserve of endogenous catecholamine can contribute to a blunted sympathetic response which can lead to bradycardia and refractory hypotension during anaesthesia. In our case, the patient did not take his medication on the morning of surgery, but it has been shown that intraneuronal catecholamine levels may not return to normal for days to weeks after cessation of amphetamine use.²

Perioperative cardiac arrest in children has multiple causations.³ Propofol has been associated with bradycardia and asystole⁴ and the decrease in heart rate is more pronounced when propofol is combined with alfentanil.⁵

We found in the patient's medical files several previous uneventful general anaesthetics for the same procedure, before the patient was on amphetamine therapy. However, a severe bradycardia responding to atropine was noted during induction of a general anaesthesia several months earlier when the child was on amphetamine treatment. We believe that a blunted sympathetic response due to a chronic amphetamine exposition associated to the cardiac effects of propofol and alfentanil may have transformed a trivial bradycardia in a life-threatening asystole.

Therefore, the management of children on chronic amphetamine therapy should include avoidance or careful titration of cardiac depressor anaesthetic drugs. Direct acting vasopressors such as epinephrine or phenylephrine are preferable because of possible cross-tolerance to other indirect vasopressors such as ephedrine.⁶ Premedication or pre-treatment with atropine may also be useful.

In conclusion, we have observed a severe cardiovascular complication during induction of anaesthesia, possibly in relation to chronic amphetamine treatment. In view of the increasing number of children on such treatment, further studies on the anaesthetic implications of this are required to determine if a specialized anaesthetic approach is appropriate in this group. C. Perruchoud*

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What can you do in 12 weeks as a house officer in anaesthesia at a District General Hospital?

Editor—A house officer in anaesthetics and intensive care? It must be a holiday! Do you feel like a medical student again? Are you allowed to do anything? These are some of the comments I repeatedly heard throughout my 3 month placement from doctors in other specialities outside anaesthesia and intensive care (ITU). However, contrary to common belief, doing house jobs in anaesthetics and ITU is not just being an observer. It is a unique opportunity for junior doctors, who are less exposed to practical clinical skills, especially with the pressures of the European Working Time Directive, to acquire competences in performing essential and advanced clinical skills and to build their confidence in managing critically ill patients. Here, I explain how much I was able to do as a house officer in just 12 weeks.

The first thing someone learns at medical school is A B C. Although this is a simple principle known to every junior doctor, it is often not followed properly in practice. My hands-on experience in the anaesthetic room under direct supervision, mostly by a consultant, taught me how to approach a patient who suddenly stops breathing, how to use simple manoeuvres to maintain the airways, and more interestingly how to obtain definitive airways. I was able to perform 35 intubations and 42 LMAs during my time in anaesthetics, which enhanced my confidence in