Correspondence

Central antiemetic effect of atropine: our personal experience

To the Editor:

We want to report our original, effective and non-expensive method of preventing emesis during segmentary spinal analgesia. According to our method, atropine sulphate is administered together with opioids in the epidural or subdural space. Until now we have treated 30 patients (21 female and 10 male) suffering from cancer pain, 15 by the epidural and 15 by the subarachnoid route after approval from the Ethics Committee and with the patient's informed consent. All patients were previously treated with the usual oral antiemetics. When the emesis was intractable we added 20 µg atropine to 3 mg morphine in the case of epidural and 15 µg atropine to a single bolus of ≤0.6 mg morphine for subarachnoid injection. Total daily doses of atropine can range, according to the number of administrations, from 60 to 80 µg for the epidural and from 45 to 60 µg for the subarachnoid route. We have always obtained complete suppression of nausea and vomiting even if we doubled the daily opioid dose without any side effects.

Our idea arose from the observation that the chemical formula of atropine (MW 289) is similar to that of cocaine (MW 303) and meperidine (MW 247) and we hypothesised that atropine could occupy opioid receptors preventing the emetic effect (Figure).¹ Another explanation is possible: it is likely that atropine can act as a 5HT₃ receptors antagonist, like other new antiemetics: MDL72222, ICS 205.930 and granisetron, that derivate from tropine esterified respectively with benzoic and indolic acid, and whose molecule is closely linked with cocaine, that was one of the first reference molecules for the synthesis of new 5HT₃ antagonists.²

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FIGURE Plastic model of the opiate receptor simplified according to Thorpe (1984).

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Central anticholinergic syndrome complicating management of a difficult airway

To the Editor:

An 83-yr-old woman required urgent thyroidectomy, as her thyroid tumour was compromising her airway. Fiberoptic intubation (FOI) was planned and scopolamine, 0.4 mg *iv*, was administered preoperatively. Ten minutes later the larynx could not be visualized fiberoptically in the sedated patient and within five minutes she became apnoeic. Emergency tracheostomy followed unsuccessful direct laryngoscopy. Three hours after surgery, the patient, although rousable, remained agitated and uncooperative. Temperature was 37.9°C and the pupils were dilated. Neurological examination was normal. Physostigmine, 1 mg *iv* was administered and the patient immediately became calm and cooperative.

Airway management in the presence of airway obstruction remains an anaesthetic challenge¹ and even awake FOI may prove difficult.² The patient's airway "collapsed" after sedation with scopolamine; the *iv* route was chosen for rapid onset.³ Scopolamine-induced amnesia and sedation are accentuated in elderly patients where coma and respiratory depression with clinical doses have been described.⁵

Central Anticholinergic Syndrome (CACS) is a complication of antimuscarinic drugs and is implicated in many cases of postoperative agitation.⁴ Patients may exhibit motor incoordination, nausea and vomiting, hallucinations, shivering, fever, as well as dry mouth and skin, tachycardia and photophobia.⁵ Because of the multiplicity of factors, the diagnosis of CACS is often presumptive. Resolution of symptoms occurs within 2–20 min of physostigmine administration.

We conclude that management of difficult airways may be complicated by the side effects of scopolamine, manifesting as CACS with coma and respiratory depression.

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