understood why the 20G *iv* was functioning "great." If we had used the 20G for the dopamine and other drugs, serious problems could have arisen. We cannot ascertain how this happened as all concerned deny any involvement. However, somebody must have cut the *iv* line and made a knot in the tubing without telling us. The remote possibility that this happened by accident must also be considered.

We bring this case to your readership's attention for two reasons: first, a "hidden *iv*" should preferably not be used unless absolutely necessary and, second, a "hidden" *iv* working "great" may not be as good as you think.

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Misconception of tracheal intubation using a fiberoptic bronchoscope

To the Editor:

The role of the fiberoptic bronchoscope in the management of patients with a history of difficult intubation is well established. However, it has been suggested that this is an intubating technique with direct vision.^{1,2} This is incorrect. Tracheal intubation under direct vision refers to the placement of an endotracheal tube (ETT) under direct laryngoscopy. Fiberoptic intubation involves advancing the tip of the bronchoscope under "fiberoptic" vision into the glottic opening until the tip approaches the carina. The ETT is then advanced slowly and "blindly" over the bronchoscope into the trachea. The bronchoscope merely functions as an intubating guide, like a "gum elastic bougie," during this manoeuvre. At no time during the insertion of the ETT over the bronchoscope can the tip of the ETT be directly visualized. As a result, tracheal intubation using a fiberoptic bronchoscope should be used with great caution in patients with retropharyngeal abscess, pedunculated polyps or tumours at the vocal cords, and foreign body in the upper airway.

Although tracheal intubation using the fiberoptic bronchoscope is a safe and effective intubating technique for many patients, its limitations cannot be overlooked.

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Physostigmine, propofol and the GABA_A receptor

To the Editor:

Fassoulaki *et al.*, are to be congratulated for their observation that physostigmine increases the dose of propofol required to induce anaesthesia.¹ However, their comments regarding the effects of propofol on the GABA_A receptor require further clarification.

Biochemical, electrophysiological and molecular studies indicate that propofol increases GABA_A receptor activity by binding to a site that is distinct from the benzodiazepine recognition site.² Propofol increases receptor function by at least three mechanisms. However, it is not certain which of these mechanisms account for propofol's clinical effects.³ Furthermore, the *in vitro* effects of propofol on the GABA_A receptor are highly dependent on experimental procedure (e.g., the concentration of GABA) or biological factors (e.g., strain of mice). Therefore, reports that correlate the behavioural properties and *in vitro* actions of anaesthetics must be interpreted with caution.⁴ Nevertheless, increasing evidence suggests that the GABA_A receptor is a primary site for propofol's neurodepressive effects. The observation that physostigmine (a drug which increases general arousal) alters the potency of propofol does not detract from this suggestion. Physostigmine also antagonizes the sedative properties of midazolam and a clear relationship has been established between midazolam's sedative-hypnotic effects and the flumazenil-sensitive site on the GABA_A receptor.^{2,5} The report by Fassoulaki *et al.* serves to remind us that clinically relevant concentrations of anaesthetics influence multiple types of receptors (to a greater or lesser degree). These receptors act in concert to "fine tune" consciousness or create the complex behavioural state we refer to as general anaesthesia.

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