FIGURE The modified Bullard blade.

pathology. Awake tracheal intubation using topical anaesthesia was used in four cases, and thiopentone-succinylcholine anaesthesia was used in three.

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Does propofol–nitrous oxide anaesthesia provide autonomic hyperreflexia (AH) prophylaxis in susceptible patients?

To the Editor:

I would like to report the case of an AH-susceptible patient who received a general anaesthetic with propofol and nitrous oxide. The patient was a 27-yr-old, 60 kg male scheduled for incision and drainage of a perirectal abscess. During the preoperative visit it was determined that he had an incomplete spinal cord lesion at the C₅₋₆ level. He had experienced episodes of AH on many occasions in the past, characterized by hypertension and headache and resulting from bladder overdistention. The patient received 10 mg of oral diazepam as a premedication. In the operating room, fentanyl 150 µg, and d-tubocurarine 3 mg, were administered, the patient's lungs were denitrogenated with oxygen, general anaesthesia was induced with propofol 150 mg (2.5 mg · kg⁻¹) and muscle paralysis for tracheal intubation was obtained with succinylcholine 120 mg. Anaesthesia was maintained with 66% nitrous oxide in oxygen and a continuous variable-rate infusion of propofol, commencing at 200 µg · kg⁻¹ · min⁻¹. The Figure provides the heart rate and blood pressure measurements recorded in the operating room (OR) and the recovery room (RR) with the elapsed time from induction of general anaesthesia provided along the x-axis. The details of the propofol infusion are also provided.

Initially the anaesthetic and operative procedure proceeded uneventfully. However, as the surgery was ending, the patient's BP was recorded at 190 mmHg systolic and over 200 mmHg shortly thereafter. His heart rate was below 50 bpm, he was diaphoretic and a diagnosis of AH was made. Two boluses of propofol 200 µg · kg⁻¹ were given and the infusion rate was increased. The systolic blood pressure decreased to 150 mmHg as the dressing was applied. The propofol infusion was stopped and the lungs were ventilated with 100% oxygen. The patient awoke and the trachea was extubated. En route to the recovery room the patient complained of severe

FIGURE

headache and said that he was "becoming hyperreflexic." In the recovery room the BPsys was recorded at 180 mmHg and then over 200 mmHg with a peak of 260 mmHg. The BP declined spontaneously over the next 10 minutes as his bladder was emptied with manual suprapubic pressure. There were no sequelae.

AH is a life-threatening complication of chronic spinal cord injury and it is usually seen in patients with cord injuries at or above the fifth thoracic level.¹ Following presentation of a noxious stimuli below the level of the lesion, a spinal reflex arc is established that results in sympathetic hyperactivity, systemic vasoconstriction and ultimately hypertension and bradycardia. Surgery is a potent stimulus for AH and may precipitate an episode even in a patient without a previous history of AH.² Both major regional block and general anaesthesia with potent volatile agents have been demonstrated to provide effective prophylaxis against AH during surgery.² A light plane of general anaesthesia has been cited as a factor in failed AH prophylaxis. Propofol (2,6-diisopropylphenol) is an intravenous general anaesthetic agent recently introduced into clinical practice in North America. Following termination of a continuous infusion there is rapid recovery of consciousness. Patient orientation occurs at serum levels slightly lower than those required for consciousness. Whereas this lighter depth of anaesthesia may be desirable to effect more rapid postoperative recovery and earlier patient mobilization it may be inadequate to provide prophylaxis against AH in susceptible patients. However, the rapid recovery from propofol may facilitate earlier detection and treatment of a postoperative episode of AH than would be likely with potent inhalational anaesthetic agents, thus making propofol an acceptable agent for induction and maintenance of GA in AH-susceptible patients. I would be

interested in the comments of Journal readers with respect to the role of propofol in the care of AH-susceptible patients.

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Hazardous modification of Bain breathing attachment

To the Editor:

Disconnection of the inner tube, at the machine end, is a well recognised hazard of the Bain co-axial breathing attachment, first reported in 1974.¹ This results in extensive rebreathing, and therefore it is essential that the integrity of the inner tube is checked prior to each use of the attachment.

Extended-length Bain attachments are available, in which the standard length co-axial tubing is replaced with tubing of approximately double the length. This allows greater mobility of the anaesthetic machine, without altering the performance of the breathing attachment.

Recently, during a routine check of equipment before an operating list, what appeared to be such an extended-length Bain attachment was found to be present on the anaesthetic machine. Visual inspection showed the inner tube to be connected to the fresh gas outlet of the machine mount. However, occlusion of the inner tube at the patient end of the attachment failed to produce a back-pressure effect. On closer examination, it was found that the "extended-length Bain attachment" consisted of a standard length attachment, onto which a further standard length of co-axial tubing had been connected. A casual inspection may have failed to reveal this dangerous modification, which would have caused extensive rebreathing, similar to that caused by disconnection of the inner tube at the fresh gas outlet of the machine mount.

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