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.

Edward Crosby BSc MD FRCPC
Department of Anaesthesia
Ottawa General Hospital, University of Ottawa
501 Smyth Road
Ottawa, Ontario, K1H 8L6

REFERENCES

- 1 Erickson RP. Autonomic hyperreflexia: pathophysiology and medical management. *Arch Phys Med Rehabil* 1980; 61: 431-40.
- 2 Schonwald G, Fish KJ, Perkasie I. Cardiovascular complications during anesthesia in chronic spinal cord injured patients. *Anesthesiology* 1981; 55: 550-8.

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.

D.N. Robinson
London, England

REFERENCE

- 1 Hannallah R, Rosales JK. A hazard connected with re-use of the Bain's circuit. A case report. *Can Anaesth Soc J* 1974; 21: 511-3.

Intubation and cervical spine injury

To the Editor:

I write concerning the Occasional Review by Suderman *et al.*¹ From their retrospective review of the neurologic outcome in 150 patients with unstable cervical spine injuries they concluded that "the admonition to avoid oral tracheal intubation in spine-injured patients remains unfounded," and that oral tracheal intubation performed after induction of general anesthesia "remains an excellent option for elective airway management" in these patients. I suspect that those conclusions will run contrary to the beliefs of many clinicians, and I think that those readers whose practices are challenged will be helped in evaluating those conclusions by some additional information about the patient population.

Many of us have been taught that laryngoscopy in an awake patient is inherently less hazardous than laryngoscopy performed in a patient who is anaesthetized and paralyzed. It has been my training that there are two potential (though unproven) benefits of awake techniques: access to the patient's subjective and objective neurologic response to the intubation manoeuvres, which provides the best possible warning of impending neurologic injury; and muscle tone, especially when there is pain/spasm, which serves as a physiologic splint. Before adjusting my practices on the basis of the report by Suderman *et al.*, I would like to know how the decision to intubate a patient awake or anaesthetized was made. It is my belief that the patients at greatest risk are those with an upper cervical, especially an atlanto-occipital, injury who are submitted to laryngoscopy and intubation in an anaesthetized state. The authors' latitudes with respect to drawing conclusions may be reduced considerably if those patients with high cervical column injuries and/or those patients perceived to have the most unstable injuries were "filtered off" and submitted to awake techniques. The readership might also be helped by a definition of "unstable." It is my understanding that there are radiologic patterns that are indicative of a potentially unstable injury but that in many instances confirmation of instability requires provocative manoeuvres.

Reduced to its simplest terms my question is: were there enough patients with *bona fide* high risk, unstable injuries who were intubated in the anaesthetized state to permit a firm conclusion that laryngoscopy and intubation under

anaesthesia do not entail a risk of cord injury? It may well be that it is only a small subset of patients with "unstable" cervical spine injuries who are at significant risk if direct laryngoscopy is performed under anaesthesia. However, quadriplegia is a devastating complication (no matter how low the incidence) and I am reluctant to see promulgated the idea that it is acceptable to put "all comers" with cervical spine injuries to sleep for their laryngoscopy unless the available data clearly excluded the presence of a subset whose risk of neurologic injury would be diminished by awake intubation.

John C. Drummond MD FRCPC
Associate Professor of Anesthesiology
University of California, San Diego
Staff Anesthesiologist
VA Medical Center, San Diego

REFERENCE

- 1 Suderman VS, Crosby ET, Lui A. Elective oral tracheal intubation in cervical spine-injured adults. *Can J Anaesth* 1991; 38: 785-9.

REPLY

Thank you for the opportunity to respond to Dr. Drummond's letter. He raises issues that are of concern to many clinicians. Traditional teaching has advanced and reinforced the tenet that awake laryngoscopy and intubation is less likely to result in secondary neurological injury in cervical spine-injured patients than the same procedure performed in an anaesthetized and paralyzed patient. However, given that no outcome data have been advanced in support of this doctrine, perhaps it is time to evaluate the principles on which it is based.

The belief that muscle pain and spasm serve as an effective spinal "splint" and prevent secondary neurological injury is not supported by clinical data documenting a high incidence of such injury in patients who are not recognized to have a cervical spinal injury at presentation and who do not have spinal immobilization performed at the outset. Failure to limit neck movement results in secondary injury in patients who are not subjected to laryngoscopy, intubation and surgery. Thus, there is no good evidence to suggest that the cervical muscles provide effective cord protection and this cannot be advanced as a justification for awake intubation.

We do not know what data Dr. Drummond would cite to support his belief that the level of injury is an independent predictor of instability following neck injury. We are not aware of data that suggest higher level injuries of the same magnitude are inherently more unstable than those in the lower cervical spine. In fact, cord injury appears to be more frequent in surviving trauma victims with cervical injuries below C₂ compared with those with injuries between C₁ and C₂ suggesting, perhaps, that the inverse is true.^{1,2} Anatomically, this may be explained by the greater luminal area in the upper cervical spine relative to cord size than in the lower cervical spine.

No patients were "filtered off" from our review. The care offered to the patients was not restricted by any protocol and was