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In Reply:—Our case occurred 4 yr ago, when doses of intrathecal morphine (ITM) were considerably larger than in current practice.[1,2] Although it may be entirely correct that Abouleish's supposition that facial pruritus rather than ITM *per se* may be the etiologic factor in reactivating oral herpes simplex virus (HSV), we believed that we should report our observations in view of the findings of Crone *et al.*, who have described a 10–14% incidence of oral HSV following epidural morphine in parturients.[3,4] Why there should be such a significant difference between patients receiving intrathecal or epidural morphine is unclear. We are surprised by the lack of correlation between the administration of ITM and oral HSV in Abouleish's extensive experience, but submit that our observations do not conflict with his for two reasons.

First, with our high dose of ITM, the cerebrospinal fluid concentrations of morphine diffusing rostrally to the brainstem and particularly the region of the trigeminal nuclei would be anticipated to be greater and thus more likely to initiate pruritus and/or reactivate latent HSV in trigeminal ganglia.

Second, the addition of local anesthetic agents to spinal opioids (as is Abouleish's practice) may reduce the incidence of pruritus in trigeminal dermatomes,[5] although this has never been formally evaluated.

We believe it is premature to conclude that ITM as a cause for herpes simplex should be "scratched out," in light of the strong association, be it direct or indirect, of HSV and epidural morphine.[3,4] We would welcome further observations in this area and concur with Abouleish that until further information is available, ITM in low doses (0.2 mg) should continue to find a useful place in obstetric anesthetic practice.

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Value of the Activated Partial Thromboplastin Time for Preoperative Detection of Coagulation Disorders Not Revealed by a Specific Questionnaire

To the Editor:—The current trend in the preoperative evaluation of bleeding risk in surgical patients is toward a two-step assessment. The first step includes a detailed questionnaire on hemostatic history and an evaluation of the true surgical hemorrhagic risk. A positive history in turn indicates the need for a second step, which consists of directed laboratory tests.[1,2]

This approach relies on the hypothesis that history, at a lower cost than laboratory tests, has a sufficient sensitivity in detecting coagulation abnormalities and subsequent bleeding risk. To assess this hypothesis we carried out a prospective study on 4,141 adult surgical patients recruited over a 3-yr period (1985–1987). The study included all the patients admitted for general and vascular surgery without known acquired or congenital coagulopathies, who were not taking medication affecting hemostasis, had no conditions affecting coagulation, and were not hypovolemic, and who were able to reply to a structured questionnaire. Preoperative hemostatic evaluation included a specific questionnaire about hemostatic function, a clinical examination, and laboratory screening tests for bleeding time, platelet count, activated partial thromboplastin time (aPTT), prothrombin time (PT), and fibrinogen assay. For the chronometric tests (aPTT and PT), results outside the normal range were checked by a second test. Only the second test result was taken into account. Because a prolonged PT or aPTT cannot discriminate between a deficit in a coagulation factor and the presence of anti-factor antibodies, further tests were carried out to determine the nature of the coagulopathy.

Twenty patients were found to have an abnormal hemostatic screen; in this group, only 3 patients had a history of minor recurrent bleeding. One abnormality was detected by a prolonged PT, and 19 were detected by a prolonged aPTT. The other laboratory tests were normal. Among these 20 patients with abnormal coagulation tests, 8 presented a potential hemorrhagic risk: 3 patients had a deficit in factor XI; 1 had anti-factor VII antibodies; and 4 had von Willebrand's disease with VIII Rco < 30%.

In our series, only three patients with a coagulation disorder gave a positive history by questionnaire, and it could be argued that only these three patients presented a bleeding risk. However, it should be born in mind that not all patients may have been exposed to situations that would reveal bleeding. Surgery is a more critical situation, and it may upset an already precarious hemostatic balance in the patient.[3] On the other hand, aPTT led to the detection of 95% coagulation abnormalities, although the true bleeding risk of these abnormalities could not be assessed since patients at risk were receiving preventive

treatment which consisted of specific coagulation factors (treated with solvent detergent for inactivation of viruses). Although in the absence of a positive history, this approach may be controversial, nevertheless in the event of abnormal bleeding, early replacement therapy may be required during or after surgery. Clearly this is unlikely to be appropriate in the absence of a diagnosis characterizing the coagulopathy.[3] Our results show the value of aPTT in detecting these coagulopathies, and we think that this test should be part of the preoperative evaluation in surgical or anesthetic procedures where bleeding may represent a major problem.

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Additional Safety Measures When Changing Endotracheal Tubes

To the Editor:—I was recently asked by the primary physicians of a 26-yr-old post–motor vehicle accident patient with a cervical spine fracture and multilobar pneumonia to change a 7.0-mm-ID nasotracheal tube that had been *in situ* for 2 weeks (since admission to the hospital) to an 8.0-mm-ID orotracheal tube because they were experiencing technical difficulties with tracheobronchial tree toilet and were concerned about the development of maxillary sinusitis. Halo and chest cast fixation caused the patient's head and neck to be rigidly fixed in 20° flexion. The epiglottis could not be visualized with direct laryngoscopy even when the patient was fully anesthetized and paralyzed. The method of changing the nasotracheal tube to an orotracheal tube in this case used two safety procedures that had been reported individually before, but to my knowledge had never been used together. The two procedures involved passage of a fiberoptic bronchoscope into the trachea alongside the existing tube,[1,2] and the passage of a jet stylet catheter (tube exchanger) through the old nasotracheal tube to be used for jet ventilation backup if the new orotracheal tube did not pass over the fiberoptic bronchoscope.[3,*]

The patient's lungs were ventilated with 100% oxygen; intravenous glycopyrrolate 0.2 mg was administered; and complete neuromuscular blockade was confirmed with a neuromuscular blockade monitor. A Williams Airway Intubator was passed into the oropharynx, and an Olympus LF-1 fiberoptic bronchoscope was passed through the airway intubator.[4,5] The existing nasotracheal tube was identified and with moderate difficulty the fiberoptic bronchoscope (which was jacketed on its proximal end with a endotracheal tube) was passed into the trachea through the anterior triangle bounded by the anterior commissure and the anterior convexity of the nasotracheal tube. A medium-sized Sheridan Tube Exchanger was then passed through a self-sealing diaphragm in the elbow connector to the nasotracheal tube and down the existing nasotracheal tube, until the tip of the tube exchanger could be visualized by the fiberoptic bronchoscope to be just above the tracheal carina. Passage of the tube exchanger through the nasotracheal tube caused the peak inspiratory pressure to increase from 26 to 29 cmH_2O, and the capnogram waveform was unchanged. The nasotracheal tube was then withdrawn over the tube exchanger and the orotracheal tube passed over the fiberoptic bronchoscope into the trachea. The fiberoptic bronchoscope was withdrawn, and during withdrawal the placement of the new orotracheal tube was confirmed to be three tracheal rings above the carina. After additional capnographic confirmation of tracheal placement of the endotracheal tube, the tube exchanger was withdrawn.

The value of this technique of exchanging endotracheal tubes lies in the staged and controlled withdrawal of an old endotracheal tube and reentry into the trachea with a new endotracheal tube. The use of the tube exchanger allows for jet ventilation if the new endotracheal tube does not enter the trachea.[3,*] Using wall pressure (50 psi), the jet ventilation tidal volume and minute ventilation through a medium-sized tube exchanger can totally support ventilation in adult patients.[6] Of course, the appropriate connection of the tube exchanger to the jet ventilator must be immediately available.[7] The presence of the fiberoptic bronchoscope allows for repeated attempts, if required, to pass the orotracheal tube over the fiberoptic bronchoscope after appropriate rotation of the orotracheal tube.[8–10] The suction port of the fiberoptic bronchoscope can also be used to insufflate oxygen and for jet ventilation as well.† Finally, cricothyrotomy or tracheostomy may be performed semielectively as long as oxygenation and ventilation are adequate by one or both means of jet ventilation (tube exchanger or fiberoptic bronchoscope).

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