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Endotracheal Tube Replacement in Patients with Cervical Spine Injury

To the Editor:—Several devices have been described for use when an endotracheal tube is exchanged in a patient with a compromised airway. These include the jet stylet and the fiberoptic bronchoscope. However, each of these methods is not always reliable when used alone. We describe a case in which the two techniques were combined for the exchange of an endotracheal tube in a patient with cervical spine instability.

A 73-yr-old man with metastatic prostate carcinoma presented with acute quadraparesis secondary to fracture of the odontoid process and C1–C2 instability. The endotracheal tube developed a cuff leak and required replacement. A medium-size tracheal tube exchanger (Sheridan, Argyle, NY) was placed through the original endotracheal tube. The exchanger was connected to a jet ventilation source *via* a 14-G intravenous catheter, and positive pressure ventilation was stopped briefly. Jet ventilation was tested to evaluate that it would provide adequate gas exchange. A fiberoptic bronchoscope (Olympus FL Tracheal Intubation Fiberscope-4 mm deflectable insertion tube, Lake Success, NY) was guided orally into the trachea around the deflated cuff of the original 8.0 endotracheal tube. When the carina was visualized with the fiberscope, the original endotracheal tube was removed. A new 7.5 endotracheal tube was threaded into the trachea over the fiberscope.

Many techniques of airway management have been used in cervical spine instability. Mask ventilation has been shown to move the C-spine more than any other technique.[1] Direct laryngoscopy remains the fastest and most reliable method of tracheal intubation, but this is known to cause movement of the C-spine.[2] Axial traction for the purpose of stabilizing the C-spine during laryngoscopy has not been proved to be protective. Cricothyroidotomy may be accomplished without C-spine movement, but no studies prove this. Benumof described the ideal method of extubation for endotracheal tube exchange as "one that permits withdrawal from the airway that is controlled, gradual, step-by-step, and reversible at any time."[3] The advantages of the jet stylets include guidance into the laryngeal inlet in the presence of distorted anatomy as well as attachment to jet ventilation. The adequacy of minute ventilation with jet stylets has been documented over a full range of sizes of the endotracheal tube exchanger and values for lung compliance.[4] Direct visualization is the most significant benefit of fiberoptic bronchoscopy, and provides the best success with the difficult airway. Many models allow application of topical anesthesia, suction of secretions, and insufflation of oxygen during exchange. Use of the fiberscope as a jet stylet has been described but not widely studied in humans.[5] Watson recommended that endotracheal tube exchange with the fiberscope "should be attempted with the backup of proven alternatives. . . ."[6] Combining the two techniques would provide backup in case of difficulty. We found three major advantages to using the fiberscope in conjunction with a jet stylet. First, it allows for examination of the laryngeal inlet for edema, which may predict further difficulties with instrumentation. Second, it is important to locate the tip of the endotracheal tube exchanger to be inside the original endotracheal tube to minimize the chances of developing barotrauma to the trachea from the jet ventilation source. Most important, however, is the security afforded by two instruments in the trachea for endotracheal tube placement guidance, because this allows for greater airway control.

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Voltage-sensitive Calcium Channels and Ischemia

To the Editor:—The L-type voltage-sensitive calcium channel (VSCC) mediates changes in excitable cell function in many tissues, including the heart, skeletal, and smooth muscles and the central nervous system. The processes of ischemia and reperfusion have been shown, in the heart and the central nervous system, to be associated with an increase in intracellular Ca^{2+}. One possible mechanism for increased intracellular calcium, discussed in the paper by Drenger *et al.*, is that of influx of calcium from the extracellular space through new VSCC.[1] Drenger *et al.* demonstrate in the heart that the number of VSCCs, as measured by radioligand binding, is increased immediately after a brief period of ischemia.[1] This phenomenon has been observed in the CNS and reported by several laboratories, including our own.[2–4] Our previous studies, using a dog model of global cerebral ischemia, showed a 350% increase in the B_{max} of L-type Ca^{2+} channels without a change in K_d after 10 min of ischemia. The increase persisted for several hours and still differed from control after 24 h of reperfusion.

Drenger *et al.* state that ischemia causes "a growth in the number of available VSCC in the sarcolemma" and suggest that "the increase in available VSCC is explained by a mechanism of differential unmasking of latent channels in the cell membrane and was related to a methylation process of the membrane phospholipids." No data are presented to support this statement. Another possibility not considered by the authors is that the membrane population isolated after ischemia differs from that isolated from control tissue.

Our own unpublished data using a similar binding technique in a regionally ischemic model demonstrated wide variability from animal to animal, preventing us from drawing conclusions with regard to a change in B_{max}. However, we found that 15 min of *global* cardiac ischemia results in a marked increase in the B_{max} for isradipine binding to porcine cardiac sarcolemma and also found an equivalent increase in the activity of the enzyme 5′-nucleotidase, widely used as a marker for sarcolemmal membranes. Our unpublished data suggest that the increase observed in [^{3}H]-isradipine binding might be due to an artifact of the purification process that occurs in ischemic tissue. Because the time from the initiation of ischemia to the assay for VSCC is too short for *de novo* synthesis of VSCC, the appearance of new binding sites with the same affinity as native channels suggests that the cell contains an excess of previously sequestered channels that may be functional but are revealed from a hidden membrane pool along with other sarcolemmal components, *e.g.*, 5′-nucleotidase.

The important observation by Drenger *et al.* that halothane decreases [^{3}H]-isradipine binding in ischemic membranes *in vitro* suggests a therapeutic possibility for the use of halothane during ischemia but requires *in vivo* corroboration and careful consideration of the synergistic negative inotropic effects of ischemia and volatile anesthetics.

The experiments described above were performed by William Curtiss, M.D., Satoshi Yasukohchi, M.D., and Mary Quigg, M.S., at the Johns Hopkins University, Baltimore, Maryland.

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