ache underwent a posterior fossa craniotomy for excision of a large (maximum diameter 5 cm) right cerebellopontine angle tumor. Anesthesia was induced with sodium thiopental and maintained with fentanyl, droperidol, and 60% nitrous oxide. The patient was placed in a semilateral position (right shoulder elevated, head of table elevated 15 degrees, head rotated 20 degrees to the left, and the table tilted to the left). Retromastoid cranieotomy revealed a tense dura, and when the latter was incised the cerebellum bulged into the surgical field. In spite of hypocapnia (PaCO₂ 23), osmotic diuresis, and excision of the lateral aspect of the cerebellar hemisphere, access to the right cerebellopontine angle remained difficult. Accordingly, a twist drill hole was made in a right posterior occipital area, and a ventriculostomy needle was passed into the occipital horn of the right lateral ventricle. The CSF was allowed to drain to atmospheric pressure and posterior fossa access improved. During the ensuing surgery, table adjustments including rotation of the table away from the operative site and elevation of the head to approximately 30 degrees were made to facilitate microscope access. The ventricular cannula eventually was removed to facilitate placement of skull mounted retractor. After approximately 6 h of dissection, cerebellar swelling of reasonably sudden onset was noted. The retractors were removed, and when the ventricular needle was reinserted to again improve exposure, gas escaped under pressure. Nitrous oxide administration was discontinued. Because of persistent and extensive cerebellar swelling, the procedure was terminated shortly thereafter, and a postoperative CT scan was performed. The latter revealed enlargement of the right cerebellar hemisphere, without evidence of intracerebellar or intraventricular hemorrhage. There was air in the left lateral ventricle.

The significance of what is apparent in retrospect was overlooked as the procedure evolved. The pressure in the posterior horn of the lateral ventricle was reduced to atmospheric by CSF drainage and then reduced to subatmospheric levels by elevation and rotation of the head. Air therefore was entrained, and when the brain needle subsequently was removed, nitrous oxide inevitably entered the air containing, and now closed, space.

As the bard said, "there is nothing new under the sun," and that is certainly true of this case report. What it represents, however, is the confluence of a well-recognized physiologic phenomenon (the diffusion of nitrous oxide into a closed space) and what is a relatively uncommon peroperative occurrence (intermittent ventricular drainage). This case might be used as a spring board for a discussion of the role of nitrous oxide in neurosurgery, however, that discussion is not appropriate to this forum. The case is presented rather as a reminder that "in and out" drainage of ventricular CSF (which also may have been performed preoperatively in hydrocephalic children) may result in the creation of an air-containing closed space and that this situation represents a relative contraindication to the use of nitrous oxide.

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An Unusual Cause for Airway Obstruction in a Young Healthy Adult

To the Editor:—A 38-year-old male was admitted for open reduction and internal fixation of a discontinuity defect of right mandibular fracture. After induction of anesthesia, the surgeons then asked that we rotate the operating table sideways so that they may have more room to move freely. To allow that, we added extension tubing to the anesthesia circuit, using one copper connector to join two disposable corrugated tubes on each limb of the breathing circuit. The patient was breathing spontaneously with ease. Soon after the addition of the extension tubing to the circuit, while we were turning the table, the patient started to exhale against severe resistance. Then we disconnected the breathing circuit from the endotracheal tube. We checked the cuff and the placement of the endotracheal tube and suctioned some secretions from its lumen. The patient breathed more easily, and breath sounds were equal and clear bilaterally, but on reconnection of the anesthesia circuit to the endotracheal tube, we noticed that the same airway obstruction recurred. There were no auscultatory breath sounds on the expiratory tubing. We disconnected the extension tube on the expiratory side, and to our surprise, we discovered a coin (a penny) lodged inside the copper connector on the expiration limb and completely ob-

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of the diameter and color of the copper connector (fig. 1). A smaller coin would pass through; a larger coin would not enter. A copper coin lightly caught inside the tube might not be seen or heard. Airway obstruction might not occur at first but just might occur at a least suspected time. A large tidal volume, a cough, or a jerk on the breathing circuit might misdirect an anesthesiologist’s attention to secretions, bronchospasm, light anesthesia, or any other “usual” causes of airway obstruction, when, in fact, the cough and jerk probably just turned the coin sideways. Needless to say, a patent inspiratory limb with a totally obstructed expiratory limb in the breathing circuit may rupture the lungs unless corrected immediately. A clear lesson we learned is that one can not be too careful and that very unusual causes of airway obstruction do exist. An extension tube connector does not belong to the anesthetist’s pocket where coins might be kept nor does a coin belong in the anesthetist’s equipment box where the tube connectors might be stored.

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Pulmonary Edema Following Laryngospasm

To the Editor.—I read with interest the recent report by Lee and Downes, in which the authors state that no cases of pulmonary edema associated with laryngospasm have been reported in adults. I wish to report that I have recently encountered such a case.

Report of a Case

A 60-year-old woman with a history of carcinoma of the posterior pharynx, previously treated by a posterior pharyngectomy and radiotherapy, was admitted for endoscopy and vocal cord stripping under general anesthesia. The anesthetic was uneventful except for some difficulty with intubation. Because of distorted anatomy, the larynx was not visualized and the patient was intubated blind. Following extubation she developed intense laryngospasm, not relieved by 100% oxygen and positive end-expiratory pressure. The patient was reintubated, however, she still had obvious respiratory distress, despite relief of the upper airway obstruction, and intermittent positive-pressure ventilation (IPPV) was commenced. A chest x-ray taken shortly after reintubation showed bilateral pulmonary edema. A pulmonary artery catheter was inserted; the right atrial, pulmonary artery and pulmonary artery wedge pressures were within normal limits, as was the cardiac output. Ventilation was continued for 24 h, by which time repeat