Experimental data suggests that after contusion, the pulmonary blood flow to the contused lobe decreases significantly.[5] Furthermore, fibrinogen deposition occurs rapidly in the contused area of the lung.[6] These factors help decrease the amount of hemorrhage and the severity of the pulmonary shunt. Hypoxemia in our patient was dramatic in that it was severe, life-threatening, and very rapid. This degree of hypoxemia was indicative of a massive pulmonary shunt, which cannot be explained entirely by a left basal pulmonary contusion alone. We propose the following explanation.

The patient suffered basal pulmonary contusion during the treatment. Pulmonary contusion would immediately result in local disruption of pulmonary vasculature, with hemorrhage into adjacent alveoli and interstitial spaces.[5] However, since the patient was in a sitting position, the blood remained in the left lower lung field and interfered with gas exchange only in that region. The pulmonary shunt thus produced was not of sufficient magnitude to result in clinically significant hypoxemia. Once the patient was placed supine and transported to the recovery room, this blood trickled down to other dependent parts of the tracheobronchial tree, where air flow and the blood resulted in frothing, which in turn produced an "air-lock" mechanism in relatively large areas of both lungs and caused a massive pulmonary shunt and hypoxemia. This impression is further substantiated by the follow-up radiographs, which showed diffuse opacities in both lung fields, and by a significant improvement in the patient's oxygenation with each tracheobronchial suctioning after intubation.

In summary, we present a case of a life-threatening complication of lithotripsy. Lithotripsy usually is conducted at locations far from the operating room; the availability of help and personnel is limited. Since lithotripsy requires no operative surgery the seriousness of the procedure frequently is underestimated. It is in this setting that the anesthesiologist needs to maintain a high degree of suspicion for rare but serious complications, such as this one.

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Spontaneous Ventilation *via* Transtracheal Large-bore Intravenous Catheters Is Possible

LARRY T. DALLEN, M.D.* RODGER WINE, PH.D., M.D.,† JONATHAN L. BENUMOF, M.D.‡

Anesthesiologists sometimes are required to manage airways that are almost totally obstructed. It is widely agreed that transtracheal jet ventilation, with a high-pressure (3,420 cmH_2O [50 pounds per square inch]) oxygen source and through a suitably large-bore intravenous catheter placed below the level of obstruction, is a very quick, effective, life-saving treatment in the desperate situation in which both mask ventilation and intubation of the trachea are impossible.[1] However, it is also commonly believed that spontaneous ventilation (pressure gradient 7–35 cmH_2O [0.1–0.5 pounds per square inch]) through an intravenous catheter is impossible because of the high resistance of the intravenous catheter.[2–6] In this case report we show that spontaneous ventilation through a transtracheally placed large-bore intravenous catheter can be life-saving when the natural upper airway is chronically narrowed and complete airway obstruction is imminent or has occurred.

* Clinical Instructor of Anesthesia.
† Resident in Anesthesia.
‡ Professor of Anesthesia.

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Address reprint requests to Dr. Benumof: University of California at San Diego Medical Center, H-770A, 225 Dickinson Street, San Diego, California 92103.

Case Report

A 65-yr-old man presented with marked inspiratory and expiratory stridor due to a hard, rubbery neck tumor (a pharyngeal carcinoma that had metastasized to an old tracheostomy site). The tumor occupied all of the space anterior to the trachea from the larynx to the sternum, so that only a fingertip could be inserted between the tumor and the sternal notch. After observation in the hospital for several hours, it was obvious that the stridor was becoming more severe and that successful management of the impending complete airway obstruction was urgently necessary. We were very concerned that if any upper airway or tumor manipulation were attempted, the airway would become completely obstructed with edema or secretions or both. Tracheostomy under local anesthesia would be treacherous due to the location of the tumor.

Our management plan was to place a 12-G intravenous catheter into the trachea between the caudad end of the tumor and the sternal notch so that jet ventilation could be used if airway obstruction occurred during subsequent upper airway manipulation. The entire procedure was performed in the operating room with routine anesthesia monitoring and with surgeons present and prepared for emergency tracheostomy. The patient was placed on an operating table that was flexed 30° head up. Preoxygenation was performed *via* mask, and oxygen was administered *via* nasal prongs at a 3-l/min flow throughout the procedure. Minimal sedation with midazolam (1 mg) was given before the skin overlying the trachea was anesthetized with 1% lidocaine.

We planned to enter the deformed trachea just above the sternal notch, in close proximity to the large vessels of the neck. Therefore, the tracheal lumen first was located by aspiration of air through a small (22-G) finder needle. After a single 12-G intravenous catheter (Angiocath 12-G, 3-inch, Deseret Medical, Sandy, UT) was successfully placed into the airway along the same trajectory as the finder needle, the patient immediately indicated that spontaneous breathing was easier. The trachea then was anesthetized with 4% lidocaine through the intravenous catheter. As expected, the local anesthetic bolus caused the patient to cough, but unexpectedly, high velocity gas and mucous was expelled through the intravenous catheter. At this point it appeared that jet ventilation would not be necessary.

A second 12-G intravenous catheter was carefully placed transtracheally alongside the first to improve spontaneous ventilation further. With the two transtracheal intravenous catheters, open to room air, and the residual native airway, the patient was able to maintain spontaneous ventilation throughout the procedure with minimal dyspnea and maintained a hemoglobin oxygen saturation of 100%.

The airway above the tumor then was topically anesthetized; a fiberoptic bronchoscope was passed sequentially through the nose, larynx, and tracheal stricture; and then a 7.0-mm cuffed, armored endotracheal tube was passed over the bronchoscope and into the trachea uneventfully. During the fiberoptic-endoscope–aided nasotracheal intubation, the trachea was completely obstructed by the fiberoptic bronchoscope for about 1 min. During this period, the patient was still able to ventilate spontaneously without increased distress solely through the two 12-G intravenous catheters, and a hemoglobin oxygen saturation of 100% was maintained. The intravenous catheters were removed once the airway was secured, and the patient was transferred to an intensive care unit.

Several days later, bronchoscopic laser vaporization of the intratracheal tumor and placement of a tracheal stent was performed. Regular follow-up with fiberoptic bronchoscopy by the pulmonologists reveals a continuing good outcome.

Discussion

The observation that placement of a 12-G intravenous catheter into the trachea can be life-saving even without high-pressure jet ventilation is important for several reasons. Anesthesiologists who perform transtracheal block already have this therapeutic maneuver in their repertoire of skills. In our case, the placement of the 12-G transtracheal intravenous catheters allowed time to secure the airway safely and definitively.

This case underscores the misconception that intravenous catheters are too small for spontaneous ventilation in adults. Many anesthesiologists think of a 14-G catheter when considering a *large-bore* catheter for transtracheal ventilation. This is probably because the 14-G catheter is the size most often mentioned in the literature on jet ventilation and because it is usually the largest catheter kept in an anesthesia cart. Although there is some evidence that the 14-G catheter is too small to allow spontaneous ventilation,[3–5] larger intravenous catheters have not been evaluated. The 12-G catheter has an internal diameter of 2.8 mm^2 and a cross-sectional area of 6.15 mm^2, which is slightly smaller than the cross-sectional area of a 3.0-mm endotracheal tube (7.06 mm^2). The two 12-G intravenous catheters have a total cross sectional area of 12.21 mm^2, which is just slightly smaller than that of a 4.0-mm endotracheal tube (12.56 mm^2). Of course, the likelihood that one or more intravenous catheters can provide an adequate airway for spontaneous ventilation is greatly increased if a small residual of the natural airway also contributes to the total cross-sectional area of the airway. Stridor becomes clinically evident in adults when the tracheal diameter is narrowed to 4 mm.[7] Using this value as the largest probable contribution of the natural airway, it corresponds to 60% of the total airway area when a single 12-G catheter is placed and 43% when two 12-G catheters are placed.

The likelihood that a patient will tolerate spontaneous ventilation through one or more intravenous catheters is increased if the preexisting obstruction has been chronic and slowly progressive, as it was in this patient, rather than sudden in onset. In the chronic case, presumably the patient has become used to breathing through a narrow orifice so that the intravenous catheter represents an increase in the size of the airway as opposed to an acute decrease, as would occur with sudden complete airway obstruction. In a search of the literature, No information on tolerance or adaptation to upper airways obstruction was found. Furthermore, a survey of anesthesiologists, pulmonologists, and thoracic surgeons familiar with upper airway obstruction revealed that none was aware of research on this subject. We found no study that evaluated the flow characteristics or ventilation parameters for adults breathing spontaneously through small (3–5-mm) endotracheal tubes or intravenous catheters larger than 13-G. Much larger-diameter intravenous catheters (7-Fr) are frequently available in operating suites today and could be used for the purpose described here.

In the assessment of velocity of flow through a tube, the diameter of the tube is the primary determinant, but the length must also be considered.[8] Halving the length of a catheter halves the resistance to flow, so in the selection of a catheter to use for transtracheal ventilation, it is reasonable to pick the shortest one available.

Transtracheal appliances for emergency access to the airway are commercially available. Most of these are designed to assist placement of a tube through the cricothyroid membrane, which was unavailable in this case because of its involvement in the tumor. Although percutaneous dilational tracheostomy devices may be useful in experienced hands, they may have devastating complications. In general, problems are associated with injury to the tracheal walls and the use of positive-pressure ventilation, and the resulting widespread emphysema.[9] As anesthesiologists, we are most experienced with the use of intravenous catheters. If familiar equipment and techniques are used, the likelihood of traumatizing the airway in emergency situations is reduced.

In summary, this case demonstrates that a chronically progressively obstructing airway may be enlarged to a cross-sectional area that is adequate for spontaneous ventilation, at least for a short period of time, by placing one or more 12-G transtracheal intravenous catheters. This case also demonstrates that the decision threshold to use this therapeutic option, which has not been reported previously, should be reasonably low for anesthesiologists faced with the patient who needs just a slight enlargement of his or her natural airway to sustain life.

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Therapeutic Suppression of a Permanent Ventricular Pacemaker Using a Peripheral Nerve Stimulator

JOSEPH P. DUCEY, M.D.,* CECIL W. FINCHER, D.O.,† CURTIS L. BAYSINGER, M.D.‡

Patients with permanent cardiac pacemakers present a potential problem for the anesthesiologist. The operating room represents an electrical environment that may interfere with normal pacemaker function.[1–3] Specifically, the ability of the pacemaker to sense electromagnetic potentials other than intrinsic myocardial potentials may lead to inhibition. Even inadvertent pacemaker reprogramming has been described.[4,5] This environment is especially dangerous when electrocautery is used, and numerous reports have delineated adverse effects, which include pacemaker inhibition as well as the precipitation of various dysrhythmias, including ventricular fibrillation and asystole.[6–8]

Optimal cardiac output may be difficult to maintain in patients with ventricular pacemakers. Patients with pacemakers also may have intrinsic cardiac disease and poor ventricular function. Furthermore, ventricular pacemakers eliminate the augmentation of ventricular filling by atrial contraction, and wall motion is asynchronous and dysfunctional during ventricular pacing.[9,10] An im-

* Major, United States Army, Medical Corps.
† Captain, United States Army, Medical Corps.
‡ Major, United States Army, Medical Corps Reserve.

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Address reprint requests to Dr. Ducey: Department of Surgery, Anesthesiology Service, Brooke Army Medical Center, Fort Sam Houston, TX 78234-6200.

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