Double lumen endotracheal tubes do not accommodate the 6-mm Nd–YAG fiberendoscope and bleeding is usually too brisk to control with occlusion catheters. This problem may be resolved with intravenous staining of the tumor tissue with methylene blue for margin identification and with more experience resecting such lesions.

Significant airway edema may occur after laser resection and reocclude a major airway. This occurred following prolonged resections. If the bronchoscopist can ablate a small obstruction readily and easily, it is not necessary to watch these patients for long periods postoperatively. However, any patient who has had either a prolonged resection or removal of a large obstruction should be monitored carefully for 24 h following surgery.

Respiratory depression often follows prolonged procedures in which only iv anesthetics are used to provide general anesthesia. Since changing to inhaled anesthetics, we have not encountered this problem. To reduce support of possible combustion, we avoid nitrous oxide and keep the F\textsubscript{\text{Ig}}\text{O}_{2} below 0.5 during Nd–YAG laser resection as recommended by Vourch.\textsuperscript{1} Ignition of flammable endotracheal tubes during Nd–YAG resection is not a high risk because the laser beam is delivered distal to the end of the tube.

Controlled ventilation usually results in moderate hypercapnia (PaCO\textsubscript{2}, 45–60 mmHg) during these procedures. This problem is secondary to partial occlusion of the endotracheal tube by the bronchoscope in addition to the preexisting pathologic occlusion of a major lung field. Therefore, a large endotracheal tube is placed to provide maximum remaining luminal area for ventilation. Jet ventilation via a small metal endotracheal tube can be used to deliver oxygen and air for this procedure but not volatile anesthetics.\textsuperscript{1} We found the use of volatile anesthetics superior to iv anesthetics for prolonged resections. For this reason, we have not used jet ventilation but instead choose conventional controlled ventilation.

Based on our experience, general anesthesia using inhaled anesthetics with an F\textsubscript{\text{Ig}}\text{O}_{2} < 0.5 in N\textsubscript{2} during Nd–YAG laser resection of otherwise nonoperable and life-threatening major airway lesions is safe and not associated with either prolonged respiratory depression or risk of combustion. Mortality from massive hemorrhage is high. Because significant airway edema and occlusion may occur postoperatively and require immediate ventilatory support, we recommend that these patients be observed carefully for 24 h after surgery.

**REFERENCES**


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**Acute Sinus Arrhythmia during Surgery in the Fourth Ventricle: An Indicator of Brain-stem Irritation**

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Surgical procedures on or near the brain stem may involve risk of injury to lower cranial nerve nuclei and respiratory centers. Because of the proximity of these areas to the centers that govern cardiovascular function, the occurrence of abrupt cardiovascular changes has been used as an indicator of impending injury. Hypertension and tachycardia, bradycardia and hypotension, ventricular dysrhythmia, and, occasionally, bradycardia and hypertension are recognized responses to brainstem and/or cranial nerve irritation. Their occurrence should prompt an immediate warning to the surgeon.

We have observed an additional cardiovascular sign that may indicate brain stem injury. Two patients undergoing resection of posterior fossa tumors abruptly developed marked sinus arrhythmia during dissection on the floor of the fourth ventricle. This report details those
cases and speculates as to the mechanism and implication of an acute sinus arrhythmia.

REPORT OF TWO CASES

Patient 1. The patient was a 50-kg, 21-year-old woman with a 3-month history of nausea, vomiting, and dizziness. A CT scan demonstrated a fourth ventricular mass and hydrocephalus. Physical examination revealed a prominent neck and an atrial flutter. Premedication consisted of diazepam and chlorpromazine. Anesthesia was induced with thiopental and maintained with 60% N₂O and 4% fentanyl and diazepam in increments (total dose for the 12-hour procedure, 2,750 μg and 20 mg, respectively). Paralysis was achieved and maintained (presence of at least one twitch in response to train-of-four stimulation of the median nerve) with pancuronium and metocurine. A suboccipital craniectomy was performed with the patient in a lateral decubitus position. The tumor (a juvenile cerebellar astrocytoma) filled the fourth ventricle. A carbon dioxide laser was used for the dissection. The tumor came off the floor of the upper two-thirds of the fourth ventricle, however, in the lower third, the tumor capsule blended with the floor and no tissue plane was apparent. The initial attempt to define a plane resulted in an abrupt decrease in blood pressure (BP) from 130 to 155 mmHg systolic and decrease in heart rate (HR) from 105 to 85 beats/min. Continued dissection produced a series of transient decreases in both HR and arterial BP followed eventually by another increase in BP from 135 to 155 mmHg systolic. The latter was associated with a further decrease in HR and the simultaneous onset of a sinus arrhythmia (fig. 1). The heart rate varied by approximately 20 beats/cycle and the periodicity of that variation coincided exactly with the rate of controlled ventilation. As the dissection continued, there were numerous episodes of hyper tension accompanied occasionally by a slight reduction in HR. The sinus arrhythmia persisted for approximately 1 h. It terminated after a dissection-related episode of hypertension and bradycardia, which occurred in association with the abrupt onset of obvious respiratory effort as evidenced by epigastic movement (in spite of muscle relaxants and recurrent negative pressures on the central venous pressure trace. A sustained tachycardia (125 beats/min) ensued. The administration of intravenous fentanyl, diazepam, and thiopental did not reduce HR and proportion of 2 mg) was given iv. Postoperatively she was awake and obeying commands. Excitation of the trachea was performed 12 h later. The patient rapidly developed signs of upper-airway obstruction, and an endotracheal tube was reintroduced. Repeat extubation of the trachea was performed 5 days later. At that time, there was unilateral dysfunction of cranial nerves VI, VII, IX, X, and XII; however, upper-airway obstruction did not recur. The ensuing postoperative course was complicated by recurrent aspiration that ceased after gelfoam injection of the affected cord. She has undergone radiation and 1 year later has had partial recovery of cranial nerves VI, IX, X, and XII.

Patient 2. The patient was a 12-year-old, 50-kg girl with a history of headache and diplopia. Physical examination revealed papilledema and nystagmus, and a CT scan showed hydrocephalus with an obliterated fourth ventricle. Dexamethasone and furosemide were administered. The patient was brought to the operating room unanesthetized. Anesthesia was induced with thiopental, fentanyl, and lidocaine iv and maintained with 60% N₂O and increments of fentanyl (1,950 μg for the 7-hour procedure). Paralysis was achieved by iv administration of metocurine and pancuronium. A suboccipital craniectomy was performed in the prone position. The tumor, a juvenile pilocytic astrocytoma, originated in the vermis and filled the fourth ventricle. During laser dissection of the vermis, the BP was 110/60 mmHg and the HR was 80 beats/min. During early attempts to dissect the tumor off the floor of the fourth ventricle, an irregular, low amplitude sinus arrhythmia developed. As dissection proceeded, there were several transient increases and decreases in BP, and the sinus arrhythmia became more pronounced. Continued dissection resulted in a sudden increase in systolic BP from 135 to 155 mmHg, and simultaneously the sinus arrhythmia became more regular and of higher amplitude with respiration-related changes in rate of up to 20 beats/min. The surgeon stated that he was working in the vicinity of the tenth nerve nuclei. The sinus arrhythmia persisted for approximately 90 min, at which time further dissection resulted in the abrupt onset of respiratory effort (epigastic movement, negative pressures on CVP) and the appearance of a sinus tachycardia (140 beats/min). As the heart rate subsided over the ensuing 20 min, the sinus arrhythmia recurred and persisted to the end of the case. The patient was taken to the ICU, where she was awake and obeying commands. The sinus arrhythmia was no longer apparent. When the trachea was extubated 12 h later, there was no evidence of cranial nerve dysfunction or of abnormal respiratory rate or pattern.

DISCUSSION

Cardiovascular centers, respiratory control areas, and the nuclei of the lower cranial nerves lie in close proximity in the brain stem. The latter are in the floor of the fourth
VENTRICLE

PRESSOR AREA

DEPRESSOR AREA

Fig. 2A. Dorsal view of the brain stem with the cerebellum removed. Left half: cranial nerve nuclei (modified from Crosby¹). Numerals correspond to cranial nerves: m = motor, s = sensory. Right half: cardiovascular centers (pressor and depressor areas modified from Alexander⁴ and respiratory centers (modified from Berger⁵). VRG = Ventral respiratory group. DRG/CI = Dorsal respiratory group/cardioinhibitory center. B. Coronal section through upper medulla at the level of the arrow. Anatomical information modified from Gatz.¹²

Note that the VRG coincides anatomically in part with IX, X (also known as the nucleus ambiguous) and the DRG/CI coincides largely with IX, X (also known as the nucleus tractus solitarius).

ventricle¹ at the level of the pons and upper medulla, and the respiratory² and cardiovascular centers³⁵ lie in immediate ventral, ventrolateral, and ventrocaudal re-

lation to them. (Fig. 2) Accordingly, abrupt cardiovascular alteration during posterior fossa manipulations may indicate impending injury to important adjacent structures. Several patterns of cardiovascular response are observed. Bradycardia and hypotension may indicate stimulation of cardioinhibitory parasympathetic efferents in the nucleus ambiguous.⁵⁶ Reduction of heart rate and blood pressure of lesser magnitude may represent stimulation of the depressor area with a consequent reduction in the vaso-constrictor and chronotropic tone.⁵⁷⁸ Hypertension and tachycardia may represent direct stimulation of the pressor area.⁵⁹ Stimulation of the trigeminal sensory nucleus also has been implicated as a cause of hypertension and tachycardia and, occasionally, ventricular ectopy (Marshall BM, personal communication). This nucleus contains pain fibers from cranial nerves, VII, IX, and X, in addition to V,¹⁰ and ventricular ectopy may reflect the autonomic consequences of stimulating pain fibers from a richly innervated and reflex-defended area (face, eyes, glottis).

The classic Cushing (or brain-stem ischemia) response consisting of hypertension and bradycardia is less common. Brain-stem ischemia is thought to result in an increased output from the pressor center, while the bradycardia may be mediated vagally via the baroreceptor reflex arc.⁵

We believe that an abrupt and pronounced sinus arrhythmia should be added to this warning list of cardiovascular alterations. The pathophysiology of the phenomenon must remain speculative. However, an alteration in the normal heart rate response to lung inflation and an enhanced sensitivity of the normal baroreceptor reflex are two possible explanations for a variation of heart rate that occurs in synchrony with ventilation and/or with ventilation-related fluctuation in arterial pressure. Vagal afferents from pulmonary stretch receptors are known to cause reflex cardiovascular changes⁴,⁹,¹⁰,¹¹ (inflation causes vasodilation and cardiac slowing), and the sinus arrhythmia may have been the result of some alteration in the processing of these afferent stimuli due to interference with the vagal nuclei or their connections. Brainstem injury can produce a related phenomenon in cats, Alexander⁴ observed that the decrease in activity in sympathetic nerves to the feline heart that normally occurs during lung inflation was enhanced by brain-stem section at the level of the lower third of the fourth ventricle.

With respect to the baroreceptor reflex, the nucleus tractus solitarius (which contains the sensory nucleus of cranial nerves IX, X) and the nucleus ambiguus (which contains the motor nuclei of cranial nerves IX, X) are the brain-stem relays involved in rapid baroreceptor reflex responses (Korner,¹¹ p. 927), and irritation of these two nuclei or their interconnections may have occurred. The location of stimulation during the onset of the sinus arrhythmia in the first case was uncertain, while in the second instance, the surgeon stated that he was in the
vicinity of the “tenth nerve nucleus.” In further support of the participation of one or more of these nuclei in this response is the observation of sudden respiratory effort during further manipulation of their vicinity during the second case (in the first case, the site of stimulation at the time of respiratory effort was unclear). Neurons of the dorsal and ventral respiratory groups lie within the nucleus tractus solitarius and the nucleus ambiguus, respectively, and stimulation of these areas might be expected to elicit respiratory activity. Accordingly, the appearance of the sinus arrhythmia may reflect irritation of nuclei, which are critical to the control of respiration as well to the motor and sensory function of the larynx and pharynx. As a relevant aside, the occurrence of respiratory movements during both of these cases serves as a reminder of the importance of the appropriate administration (and monitoring) of muscle relaxants. Brainstem stimulation may produce respiratory activity, hiccupping, and emesis, and any of these motor patterns occurring suddenly can result in accidental injury by instruments in the field or as a result of herniation of brain through the craniotomy. Our practice during dissection in the posterior fossa is to administer relaxants in small increments until the first twitch in response to train-of-four stimulus (usually of median or ulnar nerve) is absent or of very low amplitude. This, of course, does not provide paralysis of the diaphragm, but motor responses that we have observed have not been of hazardous magnitude.

These cases permit speculation as to the prognostic implication of an abrupt sinus arrhythmia. In both instances, it was an early response to dissection on the floor of the fourth ventricle. In the first, it is impossible to determine whether the event that incited the sinus arrhythmia contributed to the postoperative deficits because numerous episodes of tachycardia and hypertension and occasionally bradycardia and hypotension ensued as dissection proceeded under the duress of surgical constraints. In the second instance, the surgeon was able to pursue a more conservative dissection that frequently was interrupted and/or modified by the appearance of cardiovascular alterations. In this patient, there were no recognized deficits at the time of extubation on the following day. We suspect, therefore, that the sinus arrhythmia may constitute an “early warning” of injury on the floor of the fourth ventricle and accordingly should be sought as a harbinger of more dramatic and potentially morbid events. It might not have been recognized in these instances were it not for on-line polygraph (trend) recording of heart rate as determined by beat-to-beat measurement of the R-R interval, and an acute sinus arrhythmia may have been overlooked many times.

The occurrence of an acute sinus arrhythmia or other abrupt cardiovascular change during brain stem retraction or manipulation may indicate impending injury to cranial nerve nuclei and/or respiratory centers and should prompt immediate communication with the surgeon. In addition, in any patient in which this phenomenon is observed, respiratory rate and pattern as well as function of cranial nerves IX and X (gag reflex, palate movement) should be evaluated prior to extubation of the trachea. Our approach to extubation has been conservative. In the face of extensive fourth-ventricle dissection accompanied by recurrent cardiovascular responses or equivocal cranial nerve examination, we often defer extubation of the trachea in favor of 24 h of observation. Our rationale is that edema is only likely to worsen in the immediate postoperative period and that a situation of marginal function may deteriorate unexpectedly.

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