Thoracic complications associated with utilization of the air turbine dental drill

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Thoracic complications associated with the use of the high-speed air turbine dental drill have been reported sporadically in the anesthesia literature. This case report documents the potential sequelae associated with air entrainment from utilization of the high-speed air turbine dental drill in general dentistry.

Signs and symptoms of air entrainment include chest fullness, periorbital and facial edema, and crepitus. Prompt recognition of this phenomena is essential because of potential airway obstruction and additional complications, as the infused air dissects through the fascial planes. Cardiovascular collapse, associated with the development of venous air embolism, has also been reported as a life-threatening complication associated with the high-speed air turbine dental drill.

This is a case report of a patient who developed pneumomediastinum while receiving dental preparatory treatment for multiple crowns, veneers, and impressions.

Key words: Burr-tooth interface, crepitus, high-speed dental drill, pneumothorax, venous air emboli.

Case presentation
A 48-year-old Caucasian female presented for dental restorative treatment. Preparatory procedures for multiple crowns, veneers, and impressions were scheduled under intravenous sedation anesthesia in the dentist's office. The patient's medical history was significant for an ongoing anxiety disorder treated with alprazolam. The patient had no known drug allergies. Medications included ibuprofen and alprazolam. Medical history was also positive for smoking, one and one-half packs of cigarettes per day for 30 years. Physical examination revealed a 5 foot, 4 inch, 135-pound female in no apparent distress. Auscultation of the chest revealed heart sounds of regular rate and rhythm and lung fields were clear with no rhonchi or wheezing. The patient was classified as ASA physical status II.

The patient presented to the dental office 30 minutes prior to the scheduled procedure. She was escorted to the surgical suite, and an intravenous catheter was placed. Electrocardiograph monitor (lead II), automated blood pressure cuff (right upper extremity), pulse oximeter, and a precordial stethoscope were applied. Supplemental oxygen was administered at 2 L/min via nasal hood. The patient was given 3 mg intravenous midazolam and 50 µg of fentanyl at the beginning of the case. Propofol was administered in 10 mg increments throughout the procedure.

The course of the procedure and anesthetic commenced uneventfully. Approximately 65 minutes into the procedure, left periorbital edema was noted. Further examination of the patient's eye revealed periorbital edema and crepitus along the face, cheeks, and lateral aspects of the neck extending to the clavicle. The patient remained hemodynamically stable at this time. No additional sedative was administered, and the patient became oriented.

The patient stated that she felt fine and ques-
tioned the surgical team's obvious concern of her overall status. Upon further examination, the patient stated that she was having difficulty swallowing and a "brassy" voice quality was noted when the patient spoke. Due to potential airway impingement, the possibility of anaphylactic reaction or spontaneous pneumothorax, the rescue squad was called to transport the patient to the hospital. Careful assessment of the airway revealed no impingement of the soft tissue. Pulse oximetry at this time and throughout the procedure was at the 98% saturation level. The patient remained hemodynamically stable with systolic and diastolic blood pressures ranging between 120/80 and 110/70. Heart rate was 80-90 beats per minute. Bilateral breath sounds were equal prior to and during transport to the emergency room.

Upon admission to the emergency room, physical examination revealed the patient to be hemodynamically stable. Oxygenation was satisfactory, as demonstrated by a pulse oximeter reading of 99%. A chest x-ray was ordered by the emergency room physician and revealed pneumomediastinum. Bilateral breath sounds were equal. An ear, nose, and throat physician examined the patient and found her vocal cords and airway to be free of edema. The patient displayed no signs of airway impingement but described the feeling as a fullness in her neck and chest.

The initial diagnosis offered by the emergency room physician was spontaneous bleb rupture. This diagnosis arose by an exclusion process, which eliminated the most common causes of pneumothorax. The patient had not been subjected to penetrating trauma, which could result in rupture of the parietal pleura. She had not been subjected to subclavian vascular cannulation, which eliminated the possibility of visceral pleural rupture. The patient offered no history of chronic obstructive pulmonary disease and was not ventilated with positive pressure, thus excluding the possibility of alveolar rupture. Therefore, the diagnosis of spontaneous bleb rupture was made by the emergency room physician.

The patient remained hemodynamically stable and was admitted for overnight observation and intravenous antibiotics.

Discussion

Thoracic complications associated with the utilization of the air-driven dental drill have been reported in medical literature since 1900; however, reports in the anesthesia literature have been limited. Pneumomediastinum and subcutaneous emphysema associated with dental procedures have been reported in the literature. Each year, the American Dental Association reports that more than 167,000,000 dental procedures are performed utilizing the high-speed dental drill. With this significant number of procedures being performed on an annual basis, timely recognition and treatment of thoracic complications associated with dental procedures is imperative.

Patients presenting for root canal, extractions, and general dentistry are at risk for air entrainment and the cardiovascular compromise that may result. This potential air entrainment starts with the utilization of the air turbine drill. There are currently two types of dental drills utilized in dentistry. The high-speed (restorative type) dental drill, operated by compressed air at 30-40 psi drives the burr-tip at approximately 400,000 rpm, is utilized in general dentistry for restorative work and fillings. Although most of the air (80%) driving the turbine is vented outside the mouth through the dental handle, a stream of pressurized air (20%) is exhausted at the burr-tooth interface to prevent overheating and cellular destruction of the tooth. (Figure 1). Air exhausted at the burr-tooth interface presents the opportunity for air to be driven into the underlying soft tissues and fascial planes.

The second drill (surgical type) rotates at approximately 200,000 rpm. The advantage of this instrument is that it provides the oral surgeon high speed capability with relatively low torque. The driving air utilized by these surgical drills is vented away from the surgical field. There is a separate water drip for cooling purposes. The surgical dental drill avoids the risk of air entrainment by utilization of this separate water drip for cooling purposes, as opposed to relying on the forced air coolant at the burr-tooth interface of the high-speed dental drill outlined earlier. Surgical dental drills are widely utilized by oral/maxillofacial surgeons and in hospital facilities but are not widely used in general dentistry. Unfortunately, the high-speed drill is utilized for all dental purposes, which increases the patient's risk of air entrainment.

The etiology of air emboli and entry into the body during a dental surgical procedure must be examined. Mandibular extractions, dental implants, crown, veneer preparation work, and similar procedures can lead to total submersion of the burr-tip with the resultant exhausted air being forced into the surgical wound. For subcutaneous air to develop there must be increased oropharyngeal pressure and disruption of the oropharyngeal tissue. "Air may enter the soft tissues either directly by being forced down the gingival crevice or indirectly by being forced down a root canal and..."
leaving by way of a perforation in the covering alveolar bone.16

To understand this etiology, knowledge of the anatomy of the cervicomediastinal fascial spaces and planes is necessary.17-21 “The deep cervical fascia consists of a superficial layer, a visceral layer, and a parietal layer.” These layers partition the neck into three potential spaces, which can all serve as portals of entry into the mediastinum (Figure 2).22 Once the pressurized air has gained access by way of traumatized tissue in a confined space, it dissects subperiosteally and through loose connective tissue to the fascial planes of the face and neck.23-25 Air enters the open venous drainage vessels, as well as funneling nonvascular air dissections into surrounding superficial loose soft tissues.26 The air then gains access to the retropharynx and subsequently to the superior and anterior mediastinum and pericardium.27,28

Signs and symptoms of air entrainment range from mild to life threatening (Table I).29 Small amounts of air forced into the local tissue can cause mild discomfort, swelling, and crepitus when palpated. When large amounts of air are absorbed into tissues, discomfort and swelling are significantly increased. Additional signs and symptoms of air entrainment include sudden onset of dyspnea, chest pain, and cervical swelling during the procedure and can lead to chest fullness 2-6 hours postoperatively.

Thoracic infections associated with contaminated air utilized by the high-speed handpiece are potentially fatal.14 When large amounts of air are absorbed, the likelihood of infection increases as the absorbed air forces bacteria from the surgical site into the underlying tissues.30 Descending necrotizing mediastinitis may result from direct extension of contaminated nitrogen or compressed air utilized to power the air turbine dental handpiece. Descending necrotizing mediastinitis is an aggressive form of mediastinitis, with a mortality rate of 40%. Complete mediastinal drainage coupled with broad spectrum aerobic and anaerobic coverage must be initiated immediately to decrease the morbidity and mortality associated with descending necrotizing mediastinitis.22

Deaths reported in the literature are secondary to venous air embolism. The pressurized air from the dental handpiece enters the mandible, moving to the pterygoid venous plexus, the superior vena cava, and the right atrium. As Ely et al report, this accumulation in the right ventricle

Figure 1
High-speed air driven dental handpiece

The commonly used dental drill or air turbine dental handpiece is an important predisposition to pneumatic or septic complications. A close-up of the dental drill head shows the drilling burr and exhaust jets for air and water.

(Reprinted with permission from Ely EW, Stump TE, Hudspeth AS, Haponik EF. Page 459.14)
Figure 2
The different fascial planes in the neck by which infections may spread inferiorly into the mediastinum

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Table I
Signs and symptoms of subcutaneous emphysema
- Brassy voice
- Periorbital edema
- Dysphagia
- Chest fullness
- Chest pain
- Crepitus:
  - Periorbital
  - Face and neck

Table II
Prevention of subcutaneous emphysema
- Minimal tissue retraction
  - Particularly in the lingual region
- Avoid submersion of dental handpiece
- Do not exceed tissue incision to the lingual aspect of the anterior border of the ramus of the mandible
- Utilization of surgical handpiece for:
  - Third molar extractions
  - Oral and maxillofacial surgery

outflow tract obstructs blood outflow and produces major hemodynamic compromise of the patient. Although presenting symptomology can mask other pathophysiological states (i.e., anaphylaxis and allergic reaction) when utilizing the air turbine dental drill, the presence of crepitus and or periorbital edema offers a confirmation of air embolism.

Although anaphylaxis and spontaneous pneumothorax are often offered as an initial diagnosis, the presence of crepitus during dental procedures is a reliable indicator of air entrainment. The sudden onset of pulmonary edema should raise the suspicion of venous air embolism. There are two postulated mechanisms for the development of induced venous air embolism pulmonary edema. The first mechanism postulates an increase in hydrostatic pulmonary vascular pressure due to mechanical blockage and to vasoconstriction. The second mechanism suggests that pulmonary edema occurs because of increased capillary permeability secondary to endothelial cell damage. The anesthetist must be prepared to handle airway obstruction, dysphagia, and anxiety in the awake patient due to collapse of soft tissue spaces associated with the infused pressurized air. Cardiovascular collapse must be dealt with in a timely manner. An accurate diagnosis avoids unnecessary invasive testing that may be ordered by the attending physician unfamiliar with this phenomenon. Fortunately, most patients presenting for treatment of air entrainment and pneumomediastinum secondary to the dental drill recover uneventfully in 5 to 7 days.

In order to decrease the incidence of thoracic complications associated with the dental drill, it is important to avoid overly aggressive or poor surgical technique (Table II). Delay of dental restorative work or oral surgery for patients with preex-
isting infection or localized oral pathology is advocated, as it renders the tissue more vulnerable to injury. Selection of the proper surgical hand-piece with a separate water coolant drip for oral surgical procedure is imperative. Additional care must be taken to avoid submersion of the hand-piece during deep mandibular surgery.

Conclusion

Although a rare and oftentimes benign complication, pneumomediastinum and air embolism associated with the use of the high-speed dental drill can be life threatening (Table III). The anesthesiologist must be familiar with the onset and presentation of symptoms and treat them effectively. With proper airway support and cardiovascular management, patients subjected to air emphysema generally recover uneventfully.

Table III

Complications associated with subcutaneous emphysema

- Infection
- Descending necrotizing fasciitis
- Venous air emboli:
  - Hemodynamic compromise
- Emergent airway compromise
  - Impingement and collapse
  - Pneumomediastinum
  - Pneumopericardium
  - Pneumothorax

REFERENCES


AUTHOR

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Antagonism may be delayed in the presence of debilitation, carcinomatosis, and concomitant use of certain other drugs, such as quinidine, verapamil, and certain calcium channel blockers. In patients with hepatic or renal disease, the rate of recovery from neuromuscular blockade may be delayed. In these cases, the duration of neuromuscular blockade may be prolonged, and the use of a neuromuscular stimulator to monitor drug response may be necessary to assure adequate muscle relaxation.

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