Obstructive Sleep Apnea/Fat Embolism Syndrome
From Survey of Current Issues in Surgical Anesthesia, presented by the Cleveland Clinic Anesthesiology Institute

Perioperative Management of Obstructive Sleep Apnea

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Overview: incidence of sleep apnea (SA) higher in patients with morbid obesity, but may occur in nonobese patients; SA increases risk for admissions to intensive care unit and infections following coronary artery bypass graft procedure; association exists between airway complications and general anesthesia; undiagnosed SA — majority of patients with SA not diagnosed; presume diagnosis during preoperative evaluation if symptoms suggestive of SA

Definition: cessation of breathing despite ongoing efforts to breathe; spontaneous obstruction of airway; hypopnea defined as decreased airflow for >10 sec; SA and hypopnea usually associated with decrease in oxygen saturation of >4%; sleep studies — laboratories grade SA (eg, mild, moderate, or severe) based on number of events per hour; criteria for grading probably comparable between sleep laboratories

Pathophysiology: usually occurs during rapid eye movement (REM) sleep; characterized by loss of upper airway tone; attempt to breathe exacerbates obstruction; contributing factors include obesity, circumference of neck, and abnormalities of upper airway (eg, giant tonsils); vicious cycle starts with obstruction during sleep followed by hypercarbia and hypoxia, arousal from sleep, hyperventilation to reduce hypercarbia, and return to sleep; daytime somnolence common, probably due to lack of nighttime sleep and interruption of REM sleep; long-term sequelae of SA — constant sympathetic activation, which leads to systemic hypertension, pulmonary hypertension, cor pulmonale, dysrhythmias, myocardial ischemia, and heart failure

Anesthetic considerations: sedatives, anxiolytics, and opioids reduce upper airway tone and worsen obstruction; use of these agents even as premedications can initiate SA cycle; medications remain in body after short procedures, and SA cycle may continue in recovery room; surgery affects sleep patterns (eg, sleep may be fragmented due to pain and anxiety); REM rebound may worsen SA for several days after surgery; risk higher in major surgery than in minor surgery; apnea-hypopnea index remains elevated during first 3 nights after surgery; responsibility of anesthesiologist does not end when patient leaves recovery room

Screening: patients who do not snore typically do not have SA; patient may be observed to stop breathing during sleep; severity of snoring or apnea unknown in patients living alone

STOP-BANG: STOP criteria — snoring, tired, observed to stop breathing, and high blood pressure; hypertension associated with SA, but speaker uncertain whether it can be considered risk factor; BANG criteria — high body mass index, age (SA more common in older individuals), neck circumference, and male sex; questionnaire valuable but utility in clinical setting uncertain

Sleep study: useful; does not replicate normal sleep at home, but probably transferable and seems to correlate well with SA

American Society of Anesthesiologists guidelines: risk for patients with SA graded according to invasiveness of anesthesia and surgery; ascending order of invasiveness — superficial surgery (eg, involving finger or toe); peripheral surgery with general anesthesia; airway surgery with moderate sedation; major surgery with general anesthesia; opioids — can cause somnolence, hypercapnia, and hypoxemia; perioperative risk — overall risk estimated by taking into consideration severity of SA, invasiveness of anesthesia and surgery, and need for opioids postoperatively

Society of Ambulatory Anesthesia (SAMBA) guidelines: questionnaire — determine whether patient has SA, whether patient optimized with continuous positive airway pressure (CPAP) device, and whether patient able to use CPAP device after surgery (if all affirmative, patient may proceed with ambulatory surgery); nonoptimized patients may not be suitable for ambulatory surgery and may benefit from consultation with pulmonologist or sleep specialist; patient not optimized if noncompliant or if sleep partner observes apnea; SAMBA recommendations — proceed with ambulatory anesthesia when treatment of comorbidities optimized and postoperative pain can be managed with nonopioids; ambulatory anesthesia not recommended for patients with presumptive diagnosis of SA who must be discharged on opioids

Spinal anesthesia vs general anesthesia: spinal anesthesia associated with fewer critical care admissions and shorter hospital stays among patients with SA; regional anesthesia recommended whenever possible

Guidelines for treatment: preoperative evaluation — review medical records; interview patient and family members (to obtain information about sleep history); review sleep studies; if presentation suggests SA, either delay surgery or treat as if patient has SA; characteristics — history of apparent obstruction during sleep; awakening from sleep with sensation of choking; in children, suspicion heightened if parents report trouble sleeping, restlessness, waking up with “night terrors,” sleeping in strange positions, and new onset of enuresis; teachers can relate whether students display aggression, are distracted, or difficult to rouse after nap; if SA suspected (ie, no sleep study performed), treat as if patient has moderate SA unless signs (eg, falling asleep during visit) suggest severe SA

Educational Objectives
The goal of this program is to improve the diagnosis and management of sleep apnea (SA) and fat embolism syndrome (FES). After hearing and assimilating this program, the clinician will be better able to:

1. Identify and screen patients who are at high risk for SA.
2. Choose the most appropriate type of anesthesia for a patient with severe SA.
3. Explain the pathophysiology of FES.
4. Recognize the clinical manifestations of FES.
5. Design a treatment strategy for a patient with FES.

Faculty Disclosure
In adherence to ACCME Standards for Commercial Support, Audio Digest requires all faculty and members of the planning committee to disclose relevant financial relationships within the past 12 months that might create any personal conflicts of interest. Any identified conflicts were resolved to ensure that this educational activity promotes quality in health care and not a proprietary business or commercial interest. For this program, members of the faculty and planning committee reported nothing to disclose.
Management: maintain positive airway pressure as much as possible using noninvasive techniques; positive pressure (when used correctly) helps patients sleep at night and reduces daytime sleepiness; may reverse hypertension and heart failure; anesthesiologists can play role in overall health care of patient by identifying and initiating treatment for SA; severe SA — patient not good candidate for ambulatory surgery if SA poorly controlled; consider type of surgery, contributing anatomic abnormalities, coexisting disease, type of anesthesia, need for postoperative opioids, and whether patient to be accompanied home by person who can observe him or her (patients with severe SA should not go home alone); free-standing ambulatory surgery center may not be appropriate for patients with severe SA; opioids should be avoided; avoid all continuous infusion except remifentanil (which has rapid excretion); maximize use of nonsteroidal anti-inflammatory drugs; provide supplemental oxygen; apply patient’s own CPAP machine as soon as possible; do not allow patients to lie in supine position (which worsens airway obstruction); supine position only useful when provider managing airway); patients discharged to floor should have continuous pulse oximetry with monitoring by staff

Discharge recommendations: patients safe to discharge when able to stay awake; when awake, patients safe to discharge if they did not receive opioids or benzodiazepines, although guidelines suggest waiting 3 to 4 hr

Fat Embolism Syndrome

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Epidemiology: in long-bone fractures, mobilization of some free fat from medullary bone into circulation always occurs; after fracture of femur or tibia, 100% of venous blood from lower extremities contains some fat; fat embolism syndrome (FES) — occurs in 23% of high-velocity fractures (eg, sports injuries, motor vehicle accidents); associated with complex pelvic fractures and long-bone fractures (occurs in 1% to 2% of femoral, humeral, and tibial fractures); also occurs in patients undergoing lower extremity joint replacement (incidence <1%)

Pathophysiology: FES fairly uncommon because lung usually capable of filtering relatively large volume of fat; sustained release or large bolus of fat exceeds filtering capacity of lungs and leads to end-organ damage; end-organ injury and hemodynamic and pulmonary side effects attributable to activation of lipase, which attacks lipid-embedded membranes (eg, in blood vessels); at some point, lung may become clogged and mechanical obstruction of small blood vessels may occur, leading to end-organ side effects; condition of patient with spiral fracture of femur (medullary bone) may become critical if lipase activated secondary to sustained release or large bolus of fat into venous system; incidence — FES occurs more often in young, healthy individuals because circulation active (good circulation out of bone and into venous system creates condition for sustained release of fat); FES also occurs more frequently in patients with right-to-left intracardiac shunt; less common in toddlers and elderly individuals

End-organ consequences

Cutaneous: deposition of fat into cutaneous tissues and activation of lipase leads to inflammation and injury (signs include petechiae)

Central nervous system (CNS): entrance of fat into cerebral circulation and activation of lipase causes cellular injury leading to edema and hypoxemia; presence of right-to-left, left-to-right, or intrapulmonary shunt associated with increased bolus into CNS

Pulmonary: initially related to activation of lipase; subsequent mechanical effects cause pulmonary hypertension

Cardiovascular: related to mechanical obstruction, which leads to right ventricular failure

Diagnosis and clinical manifestations: diagnosis of exclusion; major criteria include respiratory symptoms with abnormal chest radiography and petechiae (relatively late findings); nonspecific signs include tachycardia, fever, retinal findings, presence of fat in urine, sudden drop in hemoglobin due to disseminated intravascular coagulation (DIC; relatively late finding), sudden drop in platelet count (relatively late finding), high erythrocyte sedimentation rate, and fat in sputum

Cutaneous and peripheral signs: flushing — seen in trunk, face, neck, and anterior chest; vasodilation occurs in response to histamine released when tissue injured by lipase; petechiae caused by injury to blood vessels due to activation of lipase

retina — cotton-wool sign caused by injury to blood vessels (late finding)

CNS signs: distinct change in level of consciousness (eg, anxiety, lethargy, disorientation, unconsciousness); deposition of fat in brain causes cerebral edema and increased intracranial pressure, which lead to focal neurologic signs, seizures, and persistent unconscious state

Pulmonary effects: fat emboli attacked by lipase, which also attacks alveoli and alveolar membrane; nonspecific signs include tachypnea, dyspnea, hypoxemia, and alveolar-arterial (A-a) gradient >100 mm Hg; signs that appear relatively late include wheezing, hemoptysis, pulmonary hypertension, and right heart failure; noncardiogenic pulmonary edema may occur; radiographic findings usually bilateral, but occur well after onset of symptoms

Cardiovascular effects: initial sympathetic discharge causes tachycardia and hypertension; electrocardiography shows right-sided changes; early in process, cardiovascular system hyperdynamic, and as disease progresses, cardiac output compromised (ie, hypotension develops); some patients may present with acute coronary syndrome due to coronary artery vasospasm caused by either fat or release of mediators of tissue injury (eg, histamine); end-stage manifestations include cyanosis, hypotension, and DIC

Laboratory studies: blood sample in red-top tube “taped to wall” for 15 min demonstrates third foamy white layer representing fat; fat may be seen in urine, but finding nonspecific; lipase level may be elevated, but this finding also nonspecific because trauma or surgery associated with elevated readings; observation of fat in tracheal mucus relatively specific finding

Conditions that raise index of suspicion for diagnosis: open reduction and internal fixation of long-bone fractures, especially multiple fractures; FES less common but still seen in endomedullary reaming procedures, particularly if tourniquet not used; because tourniquet used in total knee replacement, incidence of FES lower than that seen with total hip replacement; incidence high in fracture of pelvis with breaks in ring in ≥2 places; occasionally seen in association with pressurization of methylmethacrylate; incidence relatively high in major joint fusion (in, eg, knee); fulminate FES seen with bilateral total knee replacement in handful of cases; FES probably underdiagnosed in cemented total hip replacement

Establishing clinical diagnosis: high index of suspicion needed in high-risk cases; suspicious signs include hypoxemia, elevated A-a gradient, any acute change in mental status (particularly from alert to confused), unexplained seizure, petechial rash on anterior chest wall, visualization of fat load passing through heart on transesophageal echocardiography, and signs of right heart failure in patients at low risk for heart failure and with no other evident causes; acute cyanosis or cardiovascular collapse in young healthy patients with long-bone fracture should raise suspicion

Treatment: decrease mobilization of fat by discontinuing manipulation of long bone; if possible, place pneumatic tourniquet
to temporarily stop bolus effect; support cardiovascular system (if patient in hyperdynamic state, do not lower blood pressure with vasodilators because ensuing right-sided abnormalities may interfere with left-sided function); pulmonary system and CNS — sometimes have opposing needs; for pulmonary support, positive end-expiratory pulmonary pressure, administration of nitrous oxide, and specific pulmonary vasodilators may be required; however, increase in pressure transmitted to intracranial system should be avoided; role of extracorporeal devices — as FES typically occurs in young healthy individuals, chances of recovery good if patients survive initial trauma; extracorporeal membrane oxygenation and left ventricular assist devices can be used to ensure ongoing oxygenation in patients with limited lung function; intracranial pressure — progressive increase eventually results in herniation and permanent damage; management critical

Acknowledgments

Dr. Jacobs and Dr. Tetzlaff were recorded at Survey of Current Issues in Surgical Anesthesia, held December 1-5, 2014, in Naples, FL, and presented by the Cleveland Clinic Anesthesiology Institute. For information about upcoming CME activities sponsored by the Cleveland Clinic Anesthesiology Institute, please visit ccfcme.org/gosurganes. The Audio Digest Foundation thanks the speakers and the Cleveland Clinic Anesthesiology Institute for their cooperation in the production of this program.

Suggested Reading

OBSTRUCTIVE SLEEP APNEA/FAT EMBOLISM SYNDROME

To test online, go to www.audiodigest.org and sign in to online services.
To submit a test form by mail or fax, complete Pretest section before listening and Posttest section after listening.

1. Hypopnea is defined as a decrease in airflow for:
   (A) ≥8 sec   (B) ≥10 sec   (C) ≥12 sec   (D) ≥14 sec

2. Sleep apnea (SA) usually occurs during _______ sleep.
   (A) Stage 1   (B) Stage 2   (C) Stage 3   (D) Rapid eye movement

3. After anesthesia, the apnea-hypopnea index remains elevated for _______ in patients with SA.
   (A) 1 day   (B) 2 days   (C) 3 days   (D) 4 days

4. Which of the following is not a risk factor for SA according to STOP-BANG criteria?
   (A) Fatigue   (B) High blood pressure   (C) Older age   (D) Female sex

5. Which of the following statements about management of patients with severe SA is true?
   (A) Ambulatory surgery center is appropriate
   (B) Continuous infusion of remifentanil is acceptable
   (C) Administration of opioids is recommended
   (D) Supine is preferred position in recovery room

6. The rate of fat embolism syndrome (FES) in high-velocity fractures is _______, whereas the rate of FES in lower extremity joint replacement is _______.
   (A) 3%; <1%   (B) 23%; 2%   (C) 3%; 2%   (D) 23%; <1%

7. All the following statements about FES are true, except:
   (A) Common in young healthy individuals
   (B) Associated with long-bone fractures
   (C) End-organ damage attributable to activation of histamine
   (D) Mechanical obstruction of small blood vessels may occur

8. In FES, the initial sympathetic discharge causes _______, whereas _______ develops in the later stages of the disease.
   (A) Hypertension; hypotension
   (B) Hypotension; hypertension

9. Which of the following findings is most specific in a diagnostic workup for FES?
   (A) Fat in blood
   (B) Fat in tracheal mucus
   (C) Fat in urine
   (D) Elevated lipase level

10. Which of the following surgical procedures is associated with a relatively low incidence of FES?
    (A) Open reduction and internal fixation of long-bone fracture
    (B) Bilateral total knee replacement
    (C) Major joint fusion
    (D) Total hip replacement

Answers to Audio Digest Anesthesiology Volume 57, Issue 09: 1-B, 2-B, 3-A, 4-C, 5-D, 6-B, 7-A, 8-B, 9-A, 10-D