

Management of Complications During Moderate and Deep Sedation: Respiratory and Cardiovascular Considerations

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The risk for complications while providing moderate and deep sedation is greatest when caring for patients already medically compromised. It is reassuring that significant untoward events can generally be prevented by careful preoperative assessment, along with attentive intraoperative monitoring and support. Nevertheless, we must be prepared to manage untoward events should they arise. This continuing education article will review critical aspects of patient management of respiratory and cardiovascular complications.

Key Words: Medical emergencies; Sedation; Anesthesia; Complications.

PRIMARY ASSESSMENT AND OXYGENATION

The management of any medical urgency or emergency should commence with a primary assessment, predicated on the emphasis of the airway, breathing, and circulation (ABCs) taught during all courses in basic life support. While it is common and appropriate for the office team to assess these parameters simultaneously, airway patency must be given initial priority. There is little purpose for assessing ventilation if any degree of obstruction is present. The head should be tilted back while the chin is lifted, and the mouth and throat examined for any foreign material. In the unconscious patient, it may be necessary to also thrust the jaw forward into a protrusive position in addition to the head-tilt, chin-lift procedure. Once the airway is in an optimal position for maximizing its patency, attention is directed to breathing or ventilation. Ask a conscious patient to take a slow, deep breath. If the patient is unconscious, you should “look, listen and feel” for ventilatory effort and airflow. A useful caveat is to place one hand on the diaphragm, since during quiet breathing the chest does not always rise noticeably, and to use the other hand to

feel for air movement. If breathing is still in question, use a stethoscope to auscultate the apices of both lungs for breath sounds. The carotid pulse can be palpated either during or following this assessment. While airway patency and breathing are being assessed by the doctor and chairside assistant, other portions of primary assessment should be performed by additional team members. One should record the pulse rate and hemoglobin saturation (SpO₂) by pulse oximeter, which confirms the presence of a radial pulse, and one should record the blood pressure at frequent intervals. One of the team members should be providing supplemental oxygenation. An enriched oxygen concentration is indicated for patients who are spontaneously breathing, regardless of their level of consciousness. This will improve oxygen content within the patient’s functional residual capacity and delay hypoxemia should apnea or obstruction develop. A summary of all components of a primary assessment is shown in Figure 1.

SUPPLEMENTAL OXYGENATION

The equipment required to provide supplemental oxygen includes a 100% oxygen source, a regulator, tubing, and either a nasal cannula or mask. Every office should be equipped with a portable E-cylinder of oxygen, re-

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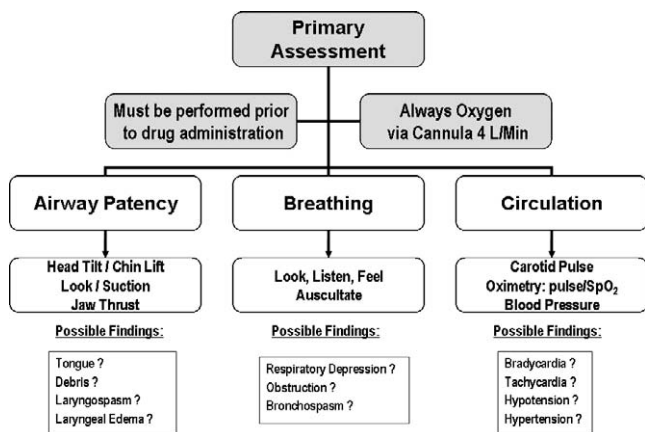


Figure 1. The primary assessment. Components of a primary assessment and conditions they reveal.

ardless of whether treatment rooms are plumbed from a central oxygen source, since emergencies can occur anywhere in the office. Generally speaking, E-cylinders should be replaced when their content falls below 1000 psi. Estimates of time remaining in oxygen cylinders can be approximated using the following formula, where $F = 0.3$ for E-cylinders and 3.0 for large H-cylinders that supply central plumbing:

$$(\text{psi} \times F)/\text{L}/\text{min} = \text{Time remaining in minutes}$$

For example if a flow of 10 L/min is required from an E-cylinder containing 1000 psi, the time remaining would be approximately 30 minutes.

The nasal cannula is ideal for administering supplemental oxygen to conscious patients who may be frightened by a mask. Each liter per minute oxygen provided via cannula increases the percent oxygen inspired (FIO_2) by $\sim 4\%$ above room air ($\text{FIO}_2 = 20 + [4 \times \text{L}/\text{min}]$).¹ This formula approximates oxygen concentration delivered by nasal cannula only. It is not applicable to masks. Flow rates above 4 L/min may become uncomfortable over time, but delivering 36% oxygen should be adequate supplementation for most situations in which the patient is breathing.

The nonrebreather mask with a reservoir may be appropriate to deliver high oxygen concentrations to unconscious, breathing patients. When using any mask, the flow rate should be at least 6 L/min to avoid feelings of suffocation. Provided the mask has a reservoir, this flow rate will deliver an oxygen concentration of $\sim 60\%$, and each additional L/min will increase FIO_2 by approximately 5%. As stated by Cairo and Pilbeam,² "Nonbreathing masks can theoretically deliver 100% oxygen, assuming that the mask fits snugly on the patient's face and the only source of gas being inhaled by the patient is derived from the oxygen flowing into the mask-reservoir system. In actual practice, disposable re-

breathing masks can deliver FIO_2 of 0.6 to 0.8." Previous concern regarding oxygen supplementation depressing hypoxic drive in patients with chronic obstructive pulmonary disease (COPD) is no longer considered valid. Current thinking is to provide whatever concentration is required to maintain oxygen saturation by pulse oximeter above 90%.³ Generally, this can be accomplished with the nasal cannula at flow rates described above.

POSITIVE PRESSURE VENTILATION

The patient with apnea is usually unconscious and will require positive pressure ventilation. Bag-valve-mask (BVM) devices with reservoirs can provide 90 to 95% oxygen concentrations, but their proper use requires considerable skill on the part of the operator. Proper head position, effective mask seal, and bag compression are skills that must be developed if they are to be used effectively. Such training is offered on manikins during healthcare provider basic life support courses. If ventilation remains difficult, airway adjuncts are indicated.

Oropharyngeal airways are adjuncts that improve airway patency by keeping the mouth open and preventing the base of the tongue from sagging against the posterior pharyngeal wall. The Guedel style of airway is hollow and facilitates insertion of a suction catheter to clear the pharynx of secretions. The Berman style lacks a lumen for this purpose. When attempting to ventilate a patient with apnea, a reasonable stepped approach is to attempt ventilation with a BVM alone, followed, if necessary, by insertion of an oropharyngeal airway. If this proves futile, one must consider advanced airway adjuncts such as tracheal intubation or insertion of a laryngeal mask airway (LMA).

Tracheal intubation is the "gold standard" of airway adjuncts. However, its use is limited to those having advanced anesthesia training such as oral and maxillofacial surgeons and dentist anesthesiologists. If intubation is unsuccessful or if the provider is not trained in this procedure, an LMA has gained status as the "second-best" airway adjunct because it is reasonably effective and technically less difficult to insert. However, training in the insertion of the LMA on simulation manikins or live patients is still necessary to be able to accomplish it successfully in an actual emergency.

The LMA is an airway adjunct that fits over the top of the larynx. The apex of the mask is inserted in the mouth, advanced towards the uvula, and continued through the natural bend of the oropharynx until it comes to rest over the pyriform fossa at the glottis. At this point the cuff around the mask is inflated with enough air to create a relatively airtight seal. The mask

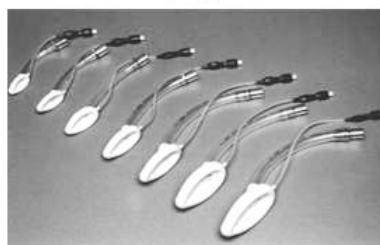
Nasal Cannula**Non-Rebreather Mask (NRB)****Bag Valve Mask (BVM)****Regulator****Oropharyngeal Airway (Guedel)****Laryngeal Mask Airway (LMA)**

Figure 2. Devices for oxygenation and ventilation. (Compilation from personal slides and slides modified from the American Heart Association.)

from a BVM is removed and the bag is directly attached to the tube's standard 15 mm connector of the LMA. Ventilation is confirmed by auscultation of breath sounds in the axillae and/or lung apices subsequent to squeezing the bag.

Skills required for advanced airway management are addressed during advanced cardiac life support training and include instruction in the proper use of oropharyngeal and laryngeal mask airways as well as endotracheal intubation. This training is also provided in human simulation courses sponsored by the American Dental Society of Anesthesiology. Devices for supplemental and positive pressure oxygenation are illustrated in Figure 2.

RESPIRATORY COMPLICATIONS

Management of Respiratory Depression

In general, the use of sedation has a positive influence on patients undergoing dental procedures. By reducing

fear and anxiety, there is less stress on the cardiovascular system, and vasovagal reactions are less likely to occur. When compared with local anesthesia alone, the two most significant negative variables introduced by moderate sedation, as well as deep sedation and general anesthesia, are the added risks for either respiratory depression, ie, hypoventilation, or airway obstruction in the deeply sedated or unconscious patient. Respiratory depression may present as a decrease in depth and/or rate of ventilation and is attributed to depression of respiratory control centers, which normally trigger breathing as carbon dioxide levels in the blood rise slightly above the normal threshold. All sedatives, opioids, and potent general anesthesia inhalation agents have the potential to depress central hypercapnic and/or peripheral hypoxemic drives, but this risk is minimal with moderate sedation, provided one uses conventional doses and monitors the patient appropriately. Nevertheless, one must be thoroughly skilled in managing respiratory depression in the event it should occur.

Like any complication, management of respiratory depression should commence with standard airway support as noted above. Pharmacologic reversal of the sedative agents is indicated whenever a dentist with training to a level of minimal or moderate sedation is faced with an unconscious patient, since airway complications such as laryngospasm, airway obstruction, aspiration, etc., may result in apnea or failure to respond adequately to oxygen supplementation and attempts at positive pressure ventilation. Pharmacologic intervention should be at least considered when respiratory depression occurs during treatment by a general anesthesia-trained dentist. Among the drug classes used for sedation and anesthesia, opioids are the most powerful respiratory depressants. If an opioid has been included in the regimen, naloxone (Narcan) should be the first reversal drug administered. Depending on the perceived urgency of the emergency treatment, it can be titrated intravenously in 0.1–0.4 mg increments every 3–5 minutes or 0.4 mg injected sublingually or intramuscularly every 5 minutes. Careful titration in no more than 0.1 mg increments is advised for any patient susceptible to cardiac irritability or hypertension. Generally, the maximum recommended dose is 0.8 mg, followed by a search for other causes if the response is inadequate. Naloxone should not be administered to a patient with a current history of opioid dependence, unless the event is life-threatening and other interventions have been futile.

Although less likely to cause respiratory depression when used alone compared with narcotics, benzodiazepines can be reversed using the specific antagonist, flumazenil (Romazicon). Depending on the perceived urgency of the emergency treatment, it can be titrated in 0.2 to 1 mg increments intravenously every 2–3 minutes. Although minimal research is available on the speed and efficacy of intramuscular or sublingual injections (SLI) of flumazenil in patients with benzodiazepine overdose, it may be injected via those routes if intravenous access is not readily available. Flumazenil should not be administered to patients having a history of dependence on benzodiazepines, a seizure disorder managed by a benzodiazepine, or evidence of tricyclic antidepressant overdose.

Management of Airway Obstruction

Airway obstruction must be distinguished from respiratory depression. Although obstruction may result in hypoventilation, the patient's actual drive to ventilate (breathe) may or may not be obtunded.

Upper airway obstruction may be attributed to anatomical structures or foreign material, both of which are addressed during the initial "airway patency" portion of

the primary assessment. When these procedures fail to establish patency, pathological causes of obstruction must be considered, namely laryngospasm or laryngeal edema. These events can be distinguished visually by those trained in direct laryngoscopy, but otherwise the distinction is made empirically.

Laryngospasm is a reflex closure or spasm of the glottic muscles including the false and true vocal cords. In the conscious or moderately sedated patient, it is very transient and followed by a cough to clear the foreign material or secretions that irritated the tissues of the larynx and triggered the spasm. It also occurs during deep sedation or light planes of general anesthesia, but the obtunded patient may not be able to clear the irritating material, and therefore the laryngospasm can be dangerously prolonged. It occurs frequently in children and in adults who are smokers. Most often the patient is unconscious and the head, neck, and upper torso exhibit a "bucking" or "rocking" movement as the patient attempts to ventilate against the obstruction. Rather than the upper abdomen and the chest rising simultaneously during attempts to breathe, these movements will alternate due to laryngospasm or any other type of airway obstruction. In most cases the spasm will relax following sustained pressure using a BVM, but hypoxemia may result if the spasm does not resolve quickly, particularly if supplemental oxygen was not being used prior to the spasm. The airway should be suctioned followed by a forceful jaw thrust to open the airway, and the BVM should then be placed with enough force to establish a tight mask seal. Gentle continuous pressure from the bag should be applied until ventilations are successful. Pharmacologic reversal of sedative agents is indicated whenever a dentist with training to a level of minimal or moderate sedation is faced with an unconscious patient or when airway complications such as laryngospasm are diagnosed or even suspected. Pharmacologic reversal of sedative agents should also be considered when laryngospasm occurs during treatment from a general anesthesia-trained dentist. Once the patient has regained consciousness, the laryngeal spasm should resolve following vigorous coughing. In the event that the cords fail to relax or severe hypoxemia develops, additional pharmacologic intervention requires the use of neuromuscular blockers, such as intravenous succinylcholine. Generally, a very small dose (0.1–0.2 mg/kg) is all that is required, and should only supplement the continued application of positive pressure using a BVM. Succinylcholine is ideally administered by those with training in deep sedation and general anesthesia. A full intubating dose of succinylcholine (1 to 2 mg/kg) should be considered if direct laryngoscopy and/or tracheal intubation is anticipated.

Laryngeal edema is among the constellation of events

associated with major allergic (anaphylactoid) reactions. The swelling of laryngeal mucosa, as well as neighboring pharyngeal mucosa and tongue, may accompany anaphylactoid reactions and will generally present as stridor or high-pitched crowing sounds during ventilation. The conscious patient will grasp the throat and complain of throat tightness or tongue swelling. Management will require administration of epinephrine^{4,5} which “decongests” the mucosa via vasoconstriction. It may be administered by several routes and doses listed among the other emergency drugs summarized in the Table. The most conventional dose is 0.3 mg intramuscularly (IM), but in severe cases 0.5 mg may be indicated. Intravenous titration of 0.1 mg increments is reserved for the most severe or refractory episodes.

Bronchospasm is a lower airway obstruction due to contraction or spasm of bronchial smooth muscle. It may result from a Type I anaphylactic allergic reaction or an anaphylactoid reaction, independently or in combination with laryngeal edema, or as a consequence of the hyperreactive airway typical in patients with asthma. Regardless of the cause for bronchospasm, the patient will exhibit dyspnea and wheezing attributed to obstruction in the chest, not the throat or mouth. Bronchial smooth muscle is under autonomic nervous control and requires beta-2 sympathomimetics for relaxation. Following primary assessment, including oxygen supplementation, a selective beta-2 agonist such as albuterol should be administered by a metered inhaler. This is preferred over epinephrine because it is less likely to produce positive cardiotoxic side effects attributed to stimulation of cardiac beta-1 receptors. It should be mentioned that patients must cooperate if inhalants are to be administered effectively. Spacer chambers can be attached to inhalers, and minimize the need for a coordinated effort on the part of the patient. However, if a patient becomes hysterical, or for other reasons cannot be administered an inhalant, parenteral epinephrine can be administered (see Table).

Additional agents mentioned frequently in dental literature for managing asthma, and allergic or anaphylactoid reactions include aminophylline and corticosteroids. These are not recommended for initial acute treatment because of limited efficacy and significant toxicity (aminophylline) or delayed onset, eg, several hours (corticosteroids). Minor allergic reactions manifest cutaneously such as pruritus or rash and are not life-threatening. Unlike anaphylactoid reactions, which are mediated by powerful autotoxins such as leukotrienes, minor reactions are attributed to histamine release and can be managed with an antihistamine such as diphenhydramine (Benadryl).

CARDIOVASCULAR COMPLICATIONS

Moderate sedation and lighter levels of deep sedation usually have minimal influence on cardiovascular function. However, excessive drug dosages including local anesthetics alone or with vasoconstrictors and influences of dental treatment, such as inadequate anesthesia, may trigger cardiovascular changes.

Fainting, or vasovagal syncope, is the most common medical complication in dental practice. It is attributed to inadequate delivery of oxygen or glucose to brain tissues. In most instances, a decrease in perfusion is central in the pathogenesis and may be the result of primary cardiac disorders or, more commonly, vasovagal reactions triggered by fear or pain. In some cases vagal influences are severe enough to induce transient periods of asystole that persist for 30–40 seconds. Furthermore, it is common for patients to exhibit brief episodes of convulsive activity that can be confused with primary seizure.^{6,7} Regardless of the cause or severity, vasovagal events will generally subside during the time primary measures for assessment and airway support are instituted. Subsequently, attention must be directed toward abnormalities in blood pressure and heart rate that may or may not require pharmacologic intervention. If suspected vasovagal syncope does not spontaneously resolve quickly, other much more serious conditions such as complete heart block, stroke, myocardial infarction, and drug overdose should be considered and appropriate emergency procedures should commence.

Hypotension

The blood pressure required to perfuse tissues adequately varies from patient to patient, and is influenced by their medical status and posture at the time of assessment. Numerical values that change significantly from baseline should alert the clinician, but evaluation of tissue perfusion is the most significant component of cardiovascular assessment.

Color changes in the skin and mucosa and the rate of capillary refill subsequent to squeezing of the nail beds can be used as a guide for assessing perfusion of peripheral tissues prior to blood pressure measurement. The adequacy of blood perfusion within the central nervous system can be estimated by the conscious patient's response to verbal and painful stimuli, or by the pupillary reflex of the unconscious or heavily sedated patient. If perfusion is considered inadequate, the clinician may elect to increase blood pressure. To do this appropriately, several physiologic principles must be considered.

Systolic blood pressure is the result of force provided by ventricular systole. Cardiac output (minute volume output of the heart) sustains this pressure, and therefore

64 Management of Complications

Anesth Prog 54:59–69 2007

Relevant Data for Emergency Drugs

<i>Indication/Drugs</i>	<i>Action</i>	<i>Administration</i>
Respiratory depression		
Naloxone (0.4 mg/mL)	Opioid receptor antagonist	IV: 0.1–0.4 mg q 2–3 minutes IM/SLI: 0.4 mg q 4–5 minutes Maximum = 0.8 mg
Flumazenil (0.1 mg/mL)	Benzodiazepine receptor antagonist	IV: 0.2 mg q 3–4 minutes IM/SLI: 0.2 mg q 4–5 minutes Maximum = 1 mg
Laryngospasm		
*Succinylcholine (20 mg/mL)	Nicotinic receptor agonist; depolarization neuromuscular block	IV: 0.1–0.2 mg/kg (~5–20 mg)
Bronchospasm		
Albuterol (metered inhaler)	Selective beta-2 receptor agonist	2–3 inhalations q 1–2 minutes × 3 if needed
Epinephrine (1 : 1000/1 mg/mL) (1 : 10,000/0.1 mg/mL)	Alpha and beta receptor agonist	IM: 0.3–0.5 mg SLI: 0.2 mg IV: 0.1 mg q 3–5 minutes based on vital signs and ECG
Laryngeal edema/Anaphylaxis		
Epinephrine (1 : 1000/1 mg/mL) (1 : 10,000/0.1 mg/mL)	Alpha and beta receptor agonist	IM: 0.3–0.5 mg SLI: 0.2 mg IV: 0.1 mg q 3–5 minutes based on vital signs and ECG
Bradycardia/hypotension		
Atropine (0.4, 0.5, and 1.0 mg/mL)	Cholinergic (muscarinic) receptor antagonist	IV, IM/SLI: 0.5 mg q 4–5 minutes × 4 if needed
Ephedrine (50 mg/mL)	Releases norepinephrine; alpha/beta receptor agonist	IV: Dilute; 1 mL in 5 mL = 10 mg/mL; then 5–10 mg q 5 minutes up to 50 mg IM/SLI: (Undiluted) 25 mg q 5 minutes × 2 if needed
*Phenylephrine (10 mg/mL)	Selective alpha agonist	IV: (Double-dilute); 1 mL in 10 mL = 1 mg/mL Then discard 9 mL and dilute remaining 1 mL in 10 mL = 0.1 mg/mL Administer 0.1 mg increments q 3 minutes to 0.5 mg
Hypertension		
Nitroglycerin (0.4 mg tablet)	Venodilator	Topical sublingual: 1 tablet q 5 minutes × 3
*Labetalol (5 mg/mL)	Alpha and beta receptor antagonist	IV: 10 mg q 5 minutes—0.5 mg/kg bolus if needed for desired effect. Repeat as needed
Angina/Myocardial infarction		
Nitroglycerin (0.4 mg tablet)	Venodilator	Topical sublingual: 1 tablet q 5 minutes × 3 if needed
Morphine	Opioid receptor agonist	IV: 2.5 mg q 3–5 minutes to 10 mg Morphine 2.5 mg = Meperidine 25 mg = Fentanyl 25 mcg = Nalbuphine 2.5 mg
Aspirin	Antiplatelet agent	1 full strength (325 mg) or 4 baby-strength (81 mg) tablets: chew and swallow

* indicates medications the authors suggest be used only by those with advanced formal training in deep sedation and general anesthesia; IV, intravenous; IM, intramuscular; SLI, sublingual injection; ECG, electrocardiogram.

it can be influenced by heart rate and stroke volume. Of these two factors, stroke volume is most significant in adults because it provides the “surge” that creates the systolic pressure. Except for small children and infants, the heart rate acts merely as a “compensator” for changes in stroke volume. For example, slow rates are common in well-trained athletes, but rapid rates are required to sustain adequate cardiac output for patients having low stroke volumes due to heart failure.

Stroke volume is influenced directly by myocardial contractility, which is augmented by sympathetic stimulation of beta-1 receptors, and by venous return to the heart (preload). According to the Frank-Starling law, preload is directly related to stroke volume, but there is a limit to this relationship. If a critical preload volume is exceeded, congestion occurs. This volume is lower for patients having compromised cardiac function, and should be considered when positioning a patient. Although the Trendelenburg position is cited most often as the preferred position for patients experiencing medical emergencies, it may allow excessive venous return (increase preload) and compromise patients with cardiac or respiratory disease. In fact, it appears this position offers few advantages.^{8,9} A semi-reclined, ie, semi-Fowler, position is more appropriate when managing most medical complications.

At the completion of systole, the ventricles enter a period of rest (diastole) and their pressure decreases to zero. However, blood (aortic) pressure does not decline this far because resistance within the arterial system sustains a diastolic pressure. The factor most responsible for diastolic pressure is variably called aortic resistance, systemic vascular resistance, or peripheral resistance. Although blood volume and viscosity are contributory, arterial diameter is the principal determinant of this resistance. Therefore, drugs that constrict arteries will increase diastolic pressure, and those that produce arterial dilation will decrease diastolic pressure.

For the heart to eject a stroke volume, ventricular systole must generate a pressure that exceeds peripheral resistance. In other words, ventricular pressure must exceed diastolic pressure. This resistance to ventricular ejection is described as “afterload” and, for a patient with heart disease, elevated diastolic pressure may hinder ejection of an adequate stroke volume. For this reason, administration of vasopressors to elevate diastolic pressure could result in a negative influence on cardiac function, particularly in patients with congestive heart failure. On the other hand, coronary artery blood flow occurs during diastole, so a reasonable aortic diastolic blood pressure must be present for the heart to nourish itself before the next systolic contraction occurs.

In general, a systolic pressure of 90 mm Hg should sustain mean arterial pressure sufficiently to perfuse tis-

ues in the recumbent patient. (Systolic pressure is used as a reference point because diastolic pressure may be difficult to ascertain in the hypotensive patient.) However, if systolic and/or diastolic pressures drop 15–20 mm Hg below baseline, tissue perfusion could be compromised and therefore should be assessed. Stroke volume and systolic pressure can be elevated in 2 manners:

- (a) Improve venous return by positioning the patient, administering intravenous (IV) fluid, or administering drugs that provide venoconstriction to increase venous pressure and preload;
- (b) Increase myocardial contractility (inotropy) using drugs that activate beta-1 receptors on myocardial cells, providing a positive inotropic influence.

If a hypotensive patient exhibits syncopal signs and symptoms, dental treatment should be delayed until a full primary assessment is completed. If an intravenous line is in place, or can be established readily, 250–500 mL of physiologic solution, such as saline or Ringer’s lactate, should be administered rapidly unless congestive heart failure with pulmonary edema is suspected. Generally, this will increase preload sufficiently to improve stroke volume and raise systolic pressure. When this maneuver cannot be accomplished, or proves unsuccessful, the patient’s heart rate should guide further treatment. If bradycardia is present, ie, heart rate < 60 beats per minute, administer atropine until the rate is within normal limits. If the heart rate is >60 beats per minute and pressure remains low, increasing the rate further may do little to improve systolic pressure. Tachycardia merely reduces the time allocated for diastolic filling and each subsequent stroke volume will decline. Reduction of the duration of diastole also reduces the time for coronary artery blood flow to the myocardial cells, which may result in myocardial ischemia.

Although several adrenergic drugs may be acceptable to manage hypotension, ephedrine is often an ideal choice for several reasons. Hypotension encountered during dental practice is usually attributed to either vasovagal episodes or the use of sedatives and anesthetics that depress sympathetic outflow to the cardiovascular system. In either case, ephedrine specifically counters these influences indirectly by stimulating norepinephrine release from sympathetic nerve endings. Also, ephedrine acts directly on alpha- and beta-adrenergic receptors, leading to vasoconstriction and increased rate and contractility of the myocardium. Ephedrine constricts veins to a greater extent than arteries, which enables it to increase preload more than afterload.¹⁰ This results in less of an increase in myocardial oxygen demand compared with other vasopressors. Finally, unlike epinephrine and other catecholamines having brief durations of action, ie, 5–10 minutes, the cardiovascular ef-

fects of ephedrine continue for 60–90 minutes. Ephedrine can be administered intravenously in 5–10 mg increments every 3–5 minutes, or 25 mg of ephedrine can be administered by sublingual or intramuscular injection. Exceeding a total dose of 50 mg is not recommended.

Rarely, hypotension may be accompanied by tachycardia, so the cardiostimulant effects of ephedrine may be undesirable. This situation occurs most often when hypotension is the result of spinal anesthesia, hypovolemia, or dehydration and is unlikely in the dental setting where hypotension is generally either vagus-induced or attributed to depressant drugs of the central nervous system (CNS). Phenylephrine is an alpha-adrenergic agonist that is useful for treating hypotension when tachycardia is present or when any increase in heart rate should be avoided, such as for a patient with significant coronary artery disease. Phenylephrine produces vasoconstriction, improving preload and systolic pressure, and produces arterial constriction, which increases diastolic pressure. The elevation in mean arterial pressure may trigger a baroreceptor-mediated reduction in heart rate.¹⁰ Phenylephrine is typically administered intravenously in 0.1 mg increments or by continuous IV infusion. The use of phenylephrine is best reserved for those with training in deep sedation and general anesthesia.

Hypertension

Sudden elevations in blood pressure are not that uncommon in dental practice, regardless of whether sedation is being provided. What establishes a significant elevation has not been defined, but “hypertensive crisis” is the conventional term for sudden elevations in diastolic pressure ≥ 120 mmHg.^{11,12} Unfortunately, this term is alarming and does not take into account the patient’s baseline pressure. In patients with chronic hypertension, autoregulation of cerebral blood flow is reset to a higher level, and abruptly lowering pressure can lead to cerebral ischemia. This is particularly true for geriatric patients.¹¹

By convention, a hypertensive crisis is regarded an “urgency” if the patient remains asymptomatic and an “emergency” if signs or symptoms are present, such as chest pain, headache, or visual disturbances. Hypertensive urgencies rarely require treatment other than a “time-out” to calm down. They are most likely attributed to waning local anesthesia, a need to use the restroom or restlessness during lengthy procedures. Gallagher summarized this issue vividly in an article for physicians in the emergency department (ED)¹²:

“The most sensible approach to the patient in the ED found to have very high blood pressure, with-

out evidence of acute end organ damage, is referral for outpatient management of serious disease that needs to be treated, not urgently, but for life. Focusing on the height of the column of mercury in the sphygmomanometer confers no demonstrable benefit on the patient and risks doing harm.”

Symptomatic hypertensive crisis is a medical emergency and requires emergency medical services (EMS) transport to the nearest emergency department. While awaiting transport, it is appropriate to consider drug therapy, provided there are no signs of stroke, eg, aphasia, paresthesia, or paralysis. When stroke is suspected blood pressure should not be lowered. Nitroglycerin administered as a 0.4 mg sublingual tablet is the safest agent for this purpose and will not decrease pressure excessively, provided one does not exceed a dosage of 1 tablet every 5 minutes up to a maximum of 3 doses if needed.

Alternatively, those with advanced training in deep sedation and general anesthesia may consider 2 other drugs to reduce blood pressure. One is labetalol (Normodyne, Trandate), which is available for intravenous use. Unlike other intravenous vasodilators such as hydralazine, labetalol also produces beta-blocking activity that prevents reflex tachycardia. It should be carefully titrated in 10 mg increments every 5 minutes, and supine blood pressure should be recorded before adding each additional increment,¹⁰ taking care not to overshoot the desired blood pressure endpoint. It should not be used in patients with asthma because its antagonist action on bronchial beta-2 receptors may induce bronchospasm. In these cases, esmolol (Brevibloc) is a safer option, although it lacks alpha-adrenergic blocking activity. It is a selective beta-1 receptor antagonist and can be administered intravenously in increments of 10 mg up to 0.5 mg/kg. Because of its very short duration of action, additional doses every 10 minutes may be needed to maintain the effect. Neither labetalol nor esmolol should be administered without continuous electrocardiographic (ECG) monitoring and continual blood pressure assessment.

Sinus Tachycardias

Transient episodes of tachycardia are triggered most often by pain, stress, and vasopressors included in local anesthetic solutions. However, tachycardia can also be a reflex response to hypoxia or hypotension, and these should be considered during patient assessment before treatment. Once these possibilities have been attended, persistent tachycardia may cause the patient to complain of palpitations. In this case, IV fluids should be administered to support blood pressure in the event the rapid heart rate is attempting to sustain the blood pres-

sure. If the episode continues, a selective beta-1 receptor antagonist such as esmolol (Brevibloc) can be titrated intravenously in the above dose range to gradually decrease sympathetic stimulation to the heart. Esmolol has a short duration so that in case the heart rate drops too precipitously, it should recover within a few minutes.

Chest Pain: Angina/Myocardial Infarction

Ischemic heart disease is a condition whereby coronary perfusion is inadequate for myocardial oxygen requirements. If atherosclerosis is significant, the dental clinician can do little to improve coronary blood flow. In the outpatient setting, one should devote complete attention to reducing myocardial oxygen requirements by maintaining the pre-existing heart rate and blood pressure so that the compromised coronary perfusion remains adequate.

When a patient with a previous history of angina pectoris experiences chest pain, the dentist should perform a complete primary assessment and direct attention to reducing myocardial oxygen demand. Comforting the patient may reduce stress-induced increases in heart rate and blood pressure. At low blood concentrations, such as those following sublingual administration, nitroglycerin dilates systemic veins and reduces venous return, which reduces preload. The resulting reduction in diastolic wall tension in the heart may also allow for improved coronary perfusion, especially in the subendocardial regions. Nitroglycerin can be repeated every 5 minutes, up to 3 times if needed, until symptoms improve or side effects such as hypotension or reflex tachycardia occur. A systolic pressure of less than 90 mmHg would contraindicate further use of nitroglycerin. Other vasodilators the patient may be taking could also be problematic. Specifically, nitroglycerin is contraindicated if the patient has taken the erectile dysfunction (ED) agents sildenafil or vardenafil within 24 hours or taken the ED agent tadalafil within 48 hours.¹³ Hypotension is particularly troublesome because a very low diastolic pressure reduces coronary blood flow which could further compromise myocardial perfusion. The hypotension also may cause reflex tachycardia which increases myocardial oxygen demand. Although reclined patients are less likely to experience these problems, blood pressure and pulse should be assessed before administering each dose of nitroglycerin. When symptoms subside, the clinician must use personal judgment regarding subsequent action. For example, a patient who responds nicely to a single dose or two of nitroglycerin could very well be sent home after his dental treatment is completed. In contrast, the patient who requires more than the usual dose of nitroglycerin to relieve symptoms should be considered for transport to an emergency de-

partment for further evaluation of a possible acute coronary syndrome. If possible, the dentist may decide to consult with the physician who has been medically managing the patient's angina pectoris to help make this decision.

If three doses of nitroglycerin over a 15–20 minute period fail to relieve symptoms in the patient previously diagnosed with angina pectoris, the clinician should assume that an infarction is evolving, and EMS transportation must be summoned.⁵ Additionally, if the patient has no history of angina pectoris, the EMS must be summoned immediately, as a myocardial infarction must be considered the most likely cause. While awaiting the arrival of the EMS, aspirin should be administered. Prior to swallowing, it is advantageous to have the patient chew one standard 325 mg tablet or 4 baby strength (81 mg) tablets to enhance the rate of absorption. Unless chewed, the enteric-coated aspirin will have a delayed onset. Platelet aggregation is a key factor during coronary thrombosis, and the maximum antiplatelet influence of aspirin is achieved within 1 hour of administration.¹⁴ Nitroglycerin can be continued every 5 minutes if needed, provided systolic pressure is at least 90 mm Hg and heart rate is within normal limits. Alternatively, if pain and anxiety are persistent, an opioid such as morphine can be administered. Opioids not only relieve pain and anxiety, but also reduce peripheral resistance (decreased afterload) and enhance venous capacitance (decrease preload). This reduces myocardial oxygen demand, ie, a nitroglycerin-like effect. Because opioids are more likely to produce hypotension if nitroglycerin has been administered, the clinician should monitor blood pressure carefully during intravenous titration.

Cardiac Arrest

The most feared sequel to myocardial infarction is a lethal cardiac arrhythmia, eg, ventricular tachycardia or fibrillation. One should be familiar with EMS response times for one's particular locale. This will aid decisions regarding any need to deliver more advanced care. If cardiac arrest occurs, the office team should administer cardiopulmonary resuscitation (CPR) as instructed in all healthcare provider basic life support (BLS) courses and apply an automated external defibrillator (AED). For those trained in advanced cardiac life support (ACLS), consideration should be given to providing the initial portion of the cardiac arrest algorithm. Provided EMS is summoned immediately, it is unlikely there will be a need to follow the standard ACLS algorithm very far. The 2005 American Heart Association (AHA) Guidelines emphasize a renewed emphasis on CPR and defibrillation. The time required to initiate these early in-

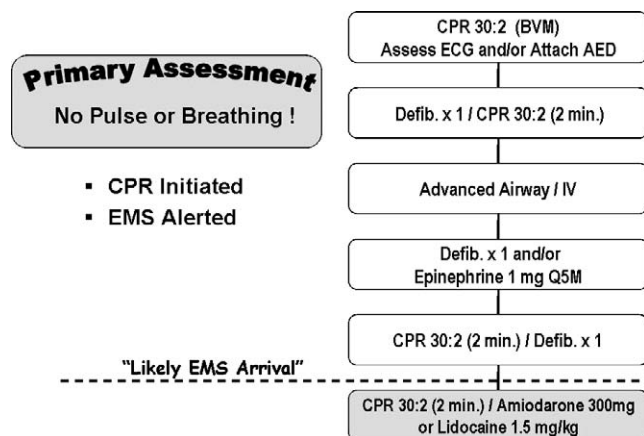


Figure 3. Abridged version of advanced cardiac life support (ACLS) cardiac arrest algorithm. Once primary assessment confirms cardiac arrest and emergency medical services (EMS) with ACLS capability is alerted immediately, the office team following the 2005 American Heart Association (AHA) algorithm may not reach the administration of antiarrhythmic drugs before help arrives.

terventions will likely obviate any need for further treatment other than advanced airway and administration of intravenous epinephrine before the EMS arrives. An abridged cardiac arrest algorithm is presented in Figure 3.

SUMMARY

Preoperative and intraoperative assessment of cardiovascular and respiratory status is essential for patient care and for effective management of untoward events. Instructions for managing each specific event can be prepared in algorithm format and placed in a plastic bag along with specific medications and syringes. During the chaos of medical urgencies and emergencies, the clinician cannot be expected to precisely recall the algorithms and drug dosages explained throughout this chapter. However, fundamental principles of physiology and patient assessment should be familiar if the clinician is to properly assess the status of the patient and select the appropriate treatment protocol. It is this familiarity that distinguishes cognitive from technical ability, and assures optimal care for the patient.

REFERENCES

1. American Thoracic Society. Standards for the diagnosis and care of patients with chronic obstructive pulmonary disease (COPD). *Am J Respir Crit Care Med.* 1995;152:s77–s120.
2. Cairo JM, Pilbeam SP. *Mosby's Respiratory Care Equipment.* 7th ed. Philadelphia, Pa: Mosby Inc; 2004:62–88.
3. Reilly JJ, Silverman EK, Shapiro SD. Chronic obstructive pulmonary disease. In: Kasper DL, Braunwald E, Fauci AS, et al, eds. *Harrison's Principles of Internal Medicine.* 16th ed. New York, NY: McGraw Hill; 2005:1547–1554.
4. Sampson HA, Munoz-Furlong A, Campbell RL, et al. Second symposium on the definition and management of anaphylaxis: summary report—second National Institute of Allergy and Infectious Disease/Food Allergy and Anaphylaxis Network symposium. *Ann Emerg Med.* 2006 Apr;47(4):373–380.
5. Hazinski MF, Chameides L, Hemphill R, eds. 2005 American Heart Association Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care. *Circulation.* 2005;112(24 suppl):IV1–IV196.
6. Deihl RR, Linden D. Images in clinical medicine. Neurocardiogenic syncope. *N Engl J Med.* 1998;339:312.
7. Thrush DN, Downs JB. Vagotonia and cardiac arrest during spinal anesthesia. *Anesthesiology.* 1999;91:1171–1173.
8. Sibbald WJ, Paterson NA, et al. The Trendelenburg position: hemodynamic effects in hypotensive and normotensive patients. *Crit Care Med.* 1979;7:218–224.
9. Coonan TJ, Hope CE. Cardio-respiratory effects of change of body position. *Can Anaesth Soc J.* 1983;30:424–437.
10. Lawson NW, Johnson JO. Autonomic nervous system: physiology and pharmacology. In: Barash PG, Cullen BF, Stoelting RK, eds. *Clinical Anesthesia.* 5th ed. Philadelphia, Pa: Lippincott-Raven Publishers; 2006:275–333.
11. Varon J, Marik PE. The diagnosis and management of hypertensive crisis. *Chest.* 2000;118:214–227.
12. Gallagher EJ. Hypertensive urgencies: treating the mercury? *Ann Emerg Med.* 2003;41(4):530–531.
13. Kloner RA. Cardiovascular effects of the 3 phosphodiesterase-5 inhibitors approved for the treatment of erectile dysfunction. *Circulation.* 2004;110:3149–3155.
14. Collins R, Peto R, Baigent C, Sleight P. Aspirin, heparin, and fibrinolytic therapy in suspected acute myocardial infarction. *N Engl J Med.* 1997;336:847–860.

CONTINUING EDUCATION QUESTIONS

1. Which of the following is the approximate FIO_2 provided by a nonrebreather mask with a reservoir delivering 8 L/min oxygen?
 - A. 60%
 - B. 70%
 - C. 80%
 - D. 100%
2. Which of the following is correct regarding administration of epinephrine?
 - A. 0.3 mg is contained in 3 mL of a 1 : 1000 concentration
 - B. 0.5 mg is indicated for a patient in cardiac arrest
 - C. 0.5 mg is contained in 5 mL of a 1 : 10,000 concentration
 - D. 0.1 mg increments using a 1 : 1000 concentration should be used for intravenous injections when managing severe anaphylactoid reactions
3. During your assessment you find that Mrs. Jones is unconscious and apneic. If your first attempt to ventilate with a BVM is unsuccessful, your next intervention before attempting further ventilation may include any of the following EXCEPT:
 - A. retilting the head, repositioning the mask
 - B. inserting an oropharyngeal airway
 - C. performing a jaw thrust
 - D. inserting an LMA
4. Your patient loses consciousness prior to intravenous sedation, but is breathing and has a pulse. The blood pressure is 80/50 mm Hg and the pulse rate is 84 beats per minute. Each of the following is appropriate for managing this situation EXCEPT:
 - A. administer supplemental oxygen
 - B. administer atropine, 0.5 mg IV, IM, or SLI
 - C. administer ephedrine, 10 mg IV or 25 mg SLI or IM
 - D. administer 250–500 mL IV fluid