Emergency Drug Kits: Pharmacological and Technical Considerations

Daniel E. Becker, DDS

Associate Director of Education, General Dental Practice Residency, Miami Valley Hospital, Dayton, Ohio

The risk for complications while providing dental procedures is greatest when caring for patients having significant medical compromise. It is comforting that significant adverse events can generally be prevented by careful preoperative assessment, along with attentive intraoperative monitoring and support. Nevertheless, the office team must be prepared to manage untoward events should they arise. This continuing education article will address basic emergency drugs that should be available in all dental practices and additional agents that become essential for those practices providing various levels of procedural sedation or general anesthesia.

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

The most essential aspects of patient management when urgencies or emergencies arise consist of a thorough primary assessment of the patient and airway management. Once this is accomplished the need for drug administration and possible emergency medical service (EMS) assistance is determined. Virtually all dental offices are equipped with an emergency drug kit. In many cases this consists of a commercially prepared kit that is purchased and updated periodically by the manufacturer. These kits not only are expensive, but are almost always more cosmetic than actually functional. For an emergency drug kit to be serviceable, the doctor must be completely familiar with both the preparation of the drug and its administration. For this reason an emergency drug kit should be designed according to the background and training of the individual provider: a basic kit for those having undergraduate training or a more advanced kit for those with additional training in parenteral sedation or general anesthesia.

Received August 29, 2014; accepted for publication September 2, 2014.

Address correspondence to Dr Daniel E. Becker, Associate Director of Education, General Dental Practice Residency, Miami Valley Hospital, One Wyoming St, Dayton, OH 45409; debecker@mvh.org.

Anesth Prog 61:171–179 2014

ISSN 0003-3006/14
SSDI 0003-3006(14)

BASIC EMERGENCY DRUG KITS

Commercially prepared drug kits fail to address the needs of the typical general dentist. Upon completion of undergraduate training in dentistry, the graduate has rarely if ever prepared or administered an injectable medication other than local anesthetic formulations contained in anesthetic cartridges. There is absolutely no familiarity with preparing medications contained in ampules or vials, nor has there been any experience with intramuscular (IM) or intravenous (IV) drug administration. Should a crisis arise, there is no conceivable way most of the injectables contained in commercial kits can be used properly. For an emergency kit to be useful, the dentist must prepare a kit for personal use that is stocked with formulations that can be used comfortably. The kit should be stored in a readily accessible location and inspected periodically for expiration dates. When replaced, expired medications can be retained separately in a "simulation kit" and used periodically by the dentist and auxiliaries for practice in loading appropriate syringes. In recent years many drugs have temporarily become unavailable from manufacturers. It should be noted that medications retain their potency well beyond the labeled expiration dates, and *The Medical Letter on Drugs and Therapeutics* has addressed this issue:

There are no reports of toxicity from degradation products of currently available drugs. How much of their potency they retain varies with the drug, the lot and the storage conditions, especially humidity, but many drugs stored under reasonable conditions in their original unopened containers retain 90% of their potency for at least 5 years after the expiration date on the label, and sometimes much longer. Drugs in solution, particularly injectables that have become cloudy or discolored or show signs of precipitation, should not be used.[1]

When constructing an emergency drug kit, the route for drug administration must be considered. Although most emergency drugs are intended for IV administration, this route is not an option for most general dental practices. The sublingual injection is familiar to all dentists and should be considered as an alternative. Any drug indicated for IV administration is nonirritating enough to justify injection into submucosal tissues. Possible contentions include appropriate concentrations, doses, and rate of onset. Because vascularity is the principal determinant for absorption, it follows empirically that sublingual absorption would lie somewhere between the immediate availability following IV administration and that following IM administration. A limited number of published studies support this presumption.[2–4]

A basic emergency kit should contain drugs that are essential regardless of the level of training. Dentist anesthesiologists and oral maxillofacial surgeons have advanced training and employ more sophisticated techniques in procedural sedation and general anesthesia. They require additional medications that will be addressed later in this review. Table 1 summarizes the drugs that are essential in all offices and differ only in the route of administration for the injectables.

DRUGS FOR ALLERGY AND ASTHMA

All dental offices should have epinephrine available to manage major allergic reactions (anaphylaxis). The manufacturers' product information for all local anesthetic formulations includes the warning that epinephrine and equipment for positive pressure ventilation should be available to manage an allergic reaction should it occur. The alpha agonist action of epinephrine shrinks mucosa swelling and beta-2 agonist action relaxes bronchial smooth muscle. Beta-1 agonist action increases systolic blood pressure and heart rate. The patient should be reassured that a transient increase in heart rate will likely accompany improvement in allergic symptoms.

The conventional dose of epinephrine for anaphylactoid reactions is 0.3 mg in adults and 0.15 mg in children by IM injection. This is accomplished using a 1 : 1000 (1 mg/mL) concentration at volumes of 0.3 and 0.15 mL respectively. Several options are available for the dentist to use for this purpose. Most familiar is the EpiPen, but this option is expensive and the provider must be familiar with its use. This product is sold in packages of 2 syringes at a cost exceeding $300. This cost is compounded by the fact that both adult and child formulations must be on hand and typically expire in 2 years. (The extended time for retained potency described above in the *Medical Letter* does not apply to EpiPen.)

A more practical and economical approach is to purchase epinephrine in single-dose vials containing 1 mL (1 mg). Using a tuberculin syringe it is simple to withdraw either 0.15 or 0.3 mL. The syringe should have a 16–25 mm (5/8–1-inch) 25-gauge needle attached, and the medication is then injected into either the lateral thigh or deltoid muscle, whichever is most accessible. This is identical to an injection using an EpiPen. Sublingual injection cannot be recommended because the dose and rate of absorption have not been studied. Formerly epinephrine was recommended to be injected subcutaneously. However, absorption and onset were delayed considerably because subcutaneous vessels contain only alpha receptors. Epinephrine constricts these vessels and delays its own absorption. In contrast, muscle vasculature contains ample amounts of beta-2 receptors that dilate vessels and enhance absorption of epinephrine. Submucosal vessels resemble subcutaneous vessels and epinephrine absorption could actually be slower than that following IM injection.

The cost of a single-dose vial is ~$10. The identical amount is more readily available in glass ampules at a cost of ~$3, but considerable skill is required for safe and proper use. Using the simulation kit described above, the dentist should periodically prepare a syringe and simulate injection on a team member.

Minor allergic reactions include rash or urticaria with no evidence of airway obstruction (tongue or throat swelling). These reactions are not life threatening and do not require epinephrine. They can be permitted to run their course or administration of diphenhydramine can be useful. Diphenhydramine blocks histamine receptors that mediate cutaneous reactions but will not counter anaphylactoid events that are produced by additional mediators such as leukotrienes. Diphenhydramine can be administered as tablets, 25 mg for children and 50 mg for adults, or alternatively it can be injected IM in the deltoid muscle at the identical doses using a 50 mg/mL single-dose vial. Concentrations of 50 mg/mL are not recommended for IV use because of irritation and should therefore be avoided by sublingual injection as well.

An albuterol inhaler should be available to manage an episode of bronchospasm whether due to an asthmatic episode or as the sole manifestation of an allergic reaction. It has selective beta-2 agonist action to relax

Table 1. Summary of Suggested Drugs and Dosages for Basic Drug Kits*

Drug (Concentration)	*Basic Providers*	*Advanced Providers*
†Naloxone (0.4 mg/mL)	SLI: 0.4 mg (1 mL) q 4–5 min; maximum = 0.8 mg	IV: 0.2–0.4 mg (0.5–1 mL) q 2–3 min. Or dilute 0.4 mg to 10 mL and titrate 0.04 mg (1 mL) q 1 min; maximum = 0.8 mg.
†Flumazenil (0.1 mg/mL)	SLI: 0.2 mg (2 mL) q 4–5 min; maximum = 1 mg	IV: 0.2 mg q 2–3 min; maximum = 1 mg
Albuterol (metered inhaler)	2–3 inhalations q 1–2 min × 3 prn	2–3 inhalations q 1–2 min × 3 prn
Epinephrine (1 : 1000; 1 mg/mL)	IM: 0.3 mg (0.3 mL)	IM: 0.3 mg (0.3 mL)
Epinephrine (1 : 10,000; 0.1 mg/mL)	Not applicable	IV: Cardiac arrest: 1 mg (10 mL) q 5 min IV: Anaphylaxis: 0.1 mg (1 mL) q 3–5 min based on vital signs and ECG‡
Diphenhydramine (50 mg/mL)	IM: 25–50 mg (0.5–1 mL)	IV: 30–50 mg (diluted to 10 mg/mL) IM: 25–50 mg (0.5–1 mL)
Atropine (1.0 mg/mL)	SLI: 0.5 mg q 4–5 min (3 mg total)	IV: 0.5 mg q 2–3 min (3 mg total)
Ephedrine (50 mg/mL)	SLI: 25 mg (0.5 mL) q 5 min (50 mg total)	IV: Use 1 mL tuberculin syringe and titrate 0.1–0.2 mL (5–10 mg) q 2–3 min (50 mg total)
Nitroglycerin (0.4-mg tablets)	Sublingual: 1 tablet q 5 min prn; maximum 3 tablets	Sublingual: 1 tablet q 5 min prn; maximum 3 tablets
Aspirin (81-mg tablets)	4 tablets: chew and swallow	4 tablets: chew and swallow
Instaglucose gel (31-g tube)	Squirt ½ tube in buccal vestibule	Squirt ½ tube in buccal vestibule

* SLI indicates sublingual injection; IV, intravenous; prn, as needed IM = intramuscular; ECG, electrocardiogram.

† Must be available if opioids or benzodiazepines are used for procedural sedation.

‡ Even if IV in place, IV administration should be considered only in the most severe cases or when IM administration fails to improve condition.[5]

bronchial smooth muscle with little or no action on cardiac beta-1 receptors. It should be mentioned, however, that patients must cooperate if inhalers are to be administered effectively. Patients may have difficulty firing the canister before inhalation and holding their breath for 10 seconds. Spacer chambers can be attached to inhalers, and minimize the need for a coordinated effort on the part of the patient. If a patient becomes hysterical, or for other reasons cannot be administered an inhalant, epinephrine can be injected IM as described above for allergic reactions.[6] If the primary assessment reveals both bronchospasm and laryngeal edema, the event is anaphylactoid, not asthmatic, and epinephrine is the drug of choice.

DRUGS FOR CHEST PAIN (ANGINA)

Nitroglycerin is indicated if a patient experiences an acute onset of chest pain. Episodes of chest pain can be caused by a variety of conditions such as acute anxiety or gastroesophageal reflux, but any acute onset should be presumed cardiac in origin. One cannot ascertain initially if the event is merely an episode of stable angina (fixed coronary stenosis and sudden myocardial oxygen demand) or an acute coronary syndrome (ACS; unstable angina due to plaque rupture or ensuing myocardial infarction). In either case nitroglycerin aids by dilating veins and reducing venous return (preload). This lessens strain-induced myocardial oxygen demand and generally relieves discomfort that is attributable to stable forms of angina. It is administered as a single tablet placed under the tongue and allowed to dissolve and absorb through mucosa. It should not be chewed or swallowed as it is completely metabolized during first-pass absorption.

A small bottle of nitroglycerin tablets (0.4 mg) is recommended for all emergency kits. It is also available as a spray formulation but at significant expense, ~$250–300. In contrast, a small bottle of tablets costs ~$15–25. The downside of tablets is that they have a shorter expiration date (1–2 years) that must be respected. If a bottle is opened, the tablets expire sooner and should be replaced within 3 months.

The actual need and timing for activation of EMS transport are not well established. The package inserts for nitroglycerin formulations instruct patients to access EMS when 3 doses of nitroglycerin over 15–20 minutes fail to relieve symptoms. Pollack and Braunwald[7] have suggested that EMS transport is indicated following administration of 3 doses of nitroglycerin over a 15–20-minute period for stable angina, but only 1 dose if angina is deemed unstable. Current American Heart

Association guidelines[8] address only ACS and encourage immediate EMS transport. They do not address stable angina. It may be impossible for the dentist to ascertain if the condition represents a stable or unstable event, and personal judgment must be used regarding subsequent action. For a patient with preexisting coronary disease, an episode of chest pain provoked by a particularly stressful intervention may well represent a typical episode of stable angina. In this case the patient will likely respond to a single dose of nitroglycerin and could very well be sent home after dental treatment is completed. In contrast, the patient who has no prior history of angina or experiences an episode that is not provoked should be transported to an emergency department for further evaluation.

If EMS transport is activated, the event is presumed a possible ACS and aspirin 300 mg should be administered. This is accomplished ideally by chewing and swallowing 3–4 baby aspirin tablets. Platelet aggregation is a key factor during ACS and the maximum antiplatelet influence of aspirin is achieved within 1 hour of administration.[9] Nitroglycerin can be continued every 5 minutes while awaiting EMS provided systolic pressure is at least 90 mm Hg and heart rate is within normal limits.

DRUGS FOR BRADYCARDIA/HYPOTENSION

Neurocardiogenic (vasovagal) syncope is not uncommon in the dental office. Pain, fear, and anxiety trigger a reflex leading to bradycardia and hypotension that reduces cerebral blood flow, leading to loss of consciousness. Primary assessment and reclining the patient with legs elevated generally results in recovery. However, in rare cases the episode persists, and drugs may be indicated to counter the event.

Two medications are useful to manage hypotensive events. When bradycardia is present atropine should be administered. Its anticholinergic action counters vagal influences on the heart and allows heart rate to increase. This is generally accompanied by an increase in blood pressure, and consciousness returns. It is ideally purchased in 1-mL single-dose vials (avoid ampules) containing 1 mg/mL. Atropine can be injected sublingually or IM in doses of 0.5 mg (0.5 mL) using a tuberculin syringe as described for epinephrine above. The dose can be repeated every 5 minutes up to 3 mg total based on heart rate and blood pressure readings. Atropine is also available in concentrations of 0.3 and 0.4 mg/mL, but these cannot be recommended. Doses lower than 0.5 mg may be associated with paradoxical vagotonic effects that result in further slowing of heart rate.[10,11]

If hypotension is accompanied by a normal heart rate, any further increase in rate is unlikely to improve blood pressure, so atropine is ineffective. In this case ephedrine is ideal. This drug not only stimulates endogenous norepinephrine release but also activates alpha receptors on veins to improve venous return and beta-1 receptors to improve cardiac contractility, both of which increase systolic blood pressure. It resembles epinephrine in action but is much longer acting and far less powerful. Ephedrine also increases heart rate, and for the dentist wishing to further simplify the emergency kit, atropine could be eliminated and ephedrine used for all cases of persistent hypotension regardless of heart rate. Ephedrine is available in 1-mL single-dose vials (avoid ampules). It can be injected sublingually or IM in doses of 25 mg (0.5 mL). The dose can be repeated once in 5 minutes if necessary based on blood pressure readings.

DRUGS FOR HYPOGLYCEMIA

Hypoglycemia can occur in any patient and is the most common acute event in those with diabetes. As serum glucose dips below 60 mg/dL compensatory hormones are released, including epinephrine, and this accounts for the early warning of tachycardia, shakiness, and diaphoresis. As the concentration drops further, generally $<$50 mg/dL, cognitive and additional central nervous system functions become impaired. Hypoglycemic reactions must be taken seriously because brain tissue relies on glucose exclusively as an energy substrate. Provided they retain consciousness, patients can be permitted to drink a sweetened beverage, but a viscous glucose concentrate should be available for more severe episodes. The concentrate should be placed in the buccal vestibule, where it dissolves and seeps down the esophagus and into the stomach. Contrary to popular belief, glucose cannot diffuse through oral mucosa.[12]

REVERSAL DRUGS

For offices providing sedation using regimens other than nitrous oxide, reversal drugs must be immediately available. Benzodiazepines are the principal agents used for this purpose, and their effects can be effectively reversed by flumazenil. This is also true for the so-called Z compounds such as zolpidem (Ambien) because these agents also act as agonists at benzodiazepine receptors.

Flumazenil is an antagonist for benzodiazepine receptors and counters the effects of any drug that acts as an

agonist at these sites. It is formulated as 0.1 mg/mL in 5- and 10-mL multidose vials. Its elimination half-life is 40–80 minutes, but its distribution half-life is only 4–11 minutes. This renders its duration of action shorter than that of most benzodiazepines, and resedation is possible. This is typically a concern when a large single dose or cumulative doses of a benzodiazepine (eg, midazolam 10 mg IV or its equivalent) have been administered along with a neuromuscular blocking agent and multiple anesthetic agents.[13] Resedation is probably not an issue following conventional oral doses for procedural sedation, eg, triazolam $\leq$0.75 mg, diazepam $\leq$20 mg, or lorazepam $\leq$4 mg. Nevertheless, it is wise to observe the patient for 30 minutes following reversal of these doses and 60 minutes if greater doses have been administered.

The conventional dose of flumazenil is 0.2 mg (2 mL) administered IV every 1–2 minutes to a maximum of 1 mg. However, it is effective when administered by sublingual injection in identical increments every 3–5 minutes.[3,4] It should not be used in patients who are taking benzodiazepines chronically for anxiety or seizure disorders. Drug withdrawal or seizures may be triggered in these patients. The anterograde amnestic effect of benzodiazepines may not be reversed by flumazenil, and it can provoke panic attacks in patients with a history of panic disorder. Nausea and vomiting may occur following its use, especially when administered rapidly by IV infusion.

The use of opioids is not encouraged for oral sedation, but if they are included in any regimen naloxone must be available for reversal. Naloxone acts as an opioid receptor antagonist and is formulated as 0.4 mg/mL in single- and multiple-dose vials. Its elimination half-life is 30–90 minutes, making its duration of action shorter than that of most opioids with the exception of fentanyl and its derivatives.[13] It can be administered by virtually any route including sublingual injection. The conventional dose is 0.4 mg (1 mL) and may be repeated in 3–5 minutes. When administered IV, it can be diluted and titrated in 0.1-mg increments to help avoid nausea. Reports of sudden release of sympathetic activity leading to cardiac excitability and pulmonary congestion are not a concern in the dental setting, where local anesthesia prevents sudden unmasking of pain.

When reversing sedation with combination drug regimens, the opioid component should be addressed first, as its potentiation of sedation or respiratory depression is the likely culprit. Naloxone will likely trigger withdrawal in patients who are opioid dependent and should be avoided unless the situation is life threatening. In these patients reversal of the benzodiazepine may be preferable.

EMERGENCY KITS FOR THE ADVANCED PROVIDER

Dentist anesthesiologists and oral maxillofacial surgeons who provide deep sedation and general anesthesia will require additional emergency drugs beyond those in basic kits, including additional agents for managing hypoglycemia and hypotension. These are summarized in Table 2.

Hypoglycemia can be managed most rapidly by infusing 25–50 mL of a 50% IV dextrose solution. It should be infused slowly, keeping in mind that its osmolarity leads to venous irritation. Glucagon is a less attractive alternative and is expensive; a 1-mg prefilled syringe cost $\sim$$150–200. Glucagon elevates blood glucose primarily by stimulating glycogenolysis and can be ineffective in patients having limited stores of glycogen, such as the poorly nourished or those who have been dieting. Following IM administration of 1 mg, glucose levels peak in 20–30 minutes. IV administration provides a faster onset (10–20 minutes) but is associated with a high incidence of nausea and vomiting.[13] IV doses should be reduced to 0.5 mg and repeated if necessary.

Phenylephrine is an additional drug that may be useful for managing hypotension. When secondary to hypovolemia, hypotension is often accompanied by tachycardia where the cardiotonic 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 vagally induced or attributed to central nervous system depressant drugs. However, patients who have had nothing by mouth (NPO) for extended periods prior to general anesthesia induction may present with a relative hypovolemia secondary to dehydration.

Phenylephrine is an alpha adrenergic agonist. It produces venoconstriction, improving venous return and systolic pressure, and increases diastolic pressure by providing arterial constriction. The subsequent elevation in mean arterial pressure may trigger a baroreceptor-mediated reduction in heart rate.[14] Phenylephrine is formulated as 10 mg/mL in single-dose vials and should be diluted to 0.1 mg/mL to facilitate IV titration of 0.1–0.2-mg increments every 2–3 minutes. (See Table 2.) Its duration is $\sim$15–20 minutes, and adequate fluid administration should be included to counter hypovolemia that may be present.

Epinephrine can be considered in the event a severe episode of bradycardia with hypotension develops. One option is to carefully titrate 0.05–0.1-mg (0.5–1-mL) increments using a prefilled 10-mL syringe of 1 : 10,000 that is available in all emergency kits for cardiac arrest. Avoid any temptation to administer the

Table 2. Summary of Suggested Drugs for Advanced Providers*

Drug	*Indication*	*Administration*
50% dextrose (0.5 g/mL)	Hypoglycemia	IV: administer 5–10 mL increments into an open-running IV infusion q 1–2 min. Generally 25–50 mL total is sufficient.
Phenylephrine (10 mg/mL)	Hypotension with tachycardia	IV: Use tuberculin syringe. Draw 0.1 mL (1 mg) and dilute to 1 mL (0.1 mg/mL) Administer 0.1 mL (0.1 mg) increments q 2–3 min.
Epinephrine 1 : 1000 (1 mg/mL)	Anaphylaxis	IM: 0.3 mg (0.3 mL) deltoid or vastus lateralis
	High degree AV block, severe bradycardia/hypotension	IV infusion: Dilute 1 mg (1 mL) in 500 mL = 2 μg/mL. Start infusion at 4 μg (2 mL)/min and titrate up to 10 μg/min.
Epinephrine 1 : 10,000 (0.1 mg/mL)	Cardiac arrest	IV: 1 mg (10 mL) q 5 min
	Anaphylaxis	IV: 0.05–0.1 mg (0.5–1 mL) q 2–3 min†
Labetalol (5 mg/mL)	Acute hypertension	IV: 10–20 mg q 5 min (300 mg total)
Esmolol (10 mg/mL)	Narrow complex tachycardias	IV: 20 mg q 1–2 min (0.5 mg/kg bolus if severe). Repeat as needed.
Lidocaine 2% (20 mg/mL)	Wide complex arrhythmias	IV: 1.0–1.5 mg/kg q 3–5 min to 3 mg/kg total.
	Cardiac arrest	IV: 1.5 mg/mL then 0.75 mg/mL if required.
Amiodarone (50 mg/mL)	Cardiac arrest	IV: 300 mg (6 mL) then 150 mg (3 mL) if required
Succinylcholine (20 mg/mL)	Laryngospasm	IV: 0.1– 0.2 mg/kg (~5–20 mg)
	Intubation	IV: 1–2 mg/kg

* The following summarizes emergency drugs that should be added to those previously addressed for basic providers. IV indicates intravenous; AV, atrioventricular; and IM, intramuscular.

† For anaphylaxis IM 0.3 mg (1 : 1000) always preferred even if IV in place. IV administration should be considered only in the most severe cases or when IM administration fails to improve condition.[5]

0.3-mg dose recommended IM for allergic reactions. The cardiovascular stimulation from this amount administered IV can be catastrophic.

The more preferable option is to titrate diluted epinephrine as a continuous drip at a rate of 2–10 μg/min.[13,15] This can be kept available in the emergency kit by taping a 1-mg ampule or vial of epinephrine (1 : 1000) to a 500-mL bag of solution along with an infusion line that ideally delivers 15 drops/mL. Add 1 mg (1 mL) epinephrine to the IV bag, providing a concentration of 2 μg/mL. Commence the infusion adjusting the drip rate to 1 drop every 2–4 seconds to deliver 1–2 mL (2–4 μg) per minute and adjust the rate according to the response in heart rate and repeated blood pressure recordings. A rate of 1 drop per second will deliver 4 mL or 8 μg/min. Of course the use of an infusion pump is preferable if available.

Laryngospasm will likely be encountered at some point when providing deep sedation or general anesthesia, so succinylcholine must be available. Laryngospasm is rarely if ever encountered by basic providers who offer moderate (conscious) sedation. If it occurs, it is very transient and followed by a cough to clear the foreign material or secretions that irritated the larynx. During deep sedation or light planes of general anesthesia laryngospasm is more likely, and the obtunded patient may not be able to clear the irritating material. In most cases the spasm will relax following a mild, sustained pressure using a bag valve mask or anesthesia mask, but hypoxemia may result if the spasm does not resolve quickly, particularly if supplemental oxygen was not provided prior to the spasm. The airway should be suctioned and followed by a forceful jaw thrust to open the upper airway, the so-called Larsen maneuver, and a bag valve mask or anesthesia mask should then be placed with enough force to establish a tight mask seal.[16] Gently apply continuous pressure from the bag until ventilations are successful; muscles will generally relax as their oxygen supply declines. Succinylcholine should be administered in the event the laryngeal adductor muscles fail to relax and severe hypoxemia develops. Generally, a very small subparalyzing dose (0.1–0.2 mg/kg) is all that is required, and should only supplement the continued application of positive pressure using a bag valve mask

or anesthesia mask. It should be avoided in patients susceptible to hyperkalemia, such as those with muscular dystrophy or other myopathies. A full intubating dose of succinylcholine (1–2 mg/kg) should be considered if direct laryngoscopy and/or tracheal intubation is anticipated.

Cardiovascular complications are not particularly common, but the emergency kit for advanced providers should include medications for managing tachyarrhythmias and hypertension should they occur. Narrow complex tachycardias are atrial in origin and can be managed with a beta blocker. Esmolol is ideal for this purpose. It is a selective (beta-1) antagonist that is metabolized rapidly, providing a relatively brief duration of action (10–30 minutes). It is available as 10 mg/mL in 10-mL vials, and its suggested dose for gradual control is 0.5 mg/kg over 1 minute.[13] More conventionally, it can be titrated in 20-mg increments every 1–2 minutes. Continuous electrocardiographic monitoring and repeated blood pressure assessments should accompany its use.

Like all narrow complex tachycardias, supraventricular tachycardia can also be managed with esmolol.[13] Nevertheless, current advanced cardiac life support guidelines suggest carotid massage and adenosine as first-line management,[15] so the advanced provider will likely consider adding adenosine to the emergency kit. Adenosine is expensive to maintain, however, and its transient effects can be disconcerting for those who have not used it previously. A single-dose 2-mL syringe or vial containing 6 mg costs ~$50 and a second 12-mg dose may be required if the initial dose fails.

Adenosine acts by depressing the atrioventricular node to eliminate reentry impulses implicated in supraventricular tachycardia. It acts immediately and is rapidly eliminated within ~30 seconds. For this reason it must be administered as a bolus into an open running infusion or followed with a 20-mL saline flush and will likely prove ineffective if administered into a vein more distal than the antecubital area of the arm.[13] The initial dose is 6 mg, and if conversion does not occur in 1–2 minutes, a second dose of 12 mg should be administered. These doses must be halved in patients who have a transplanted heart or are medicated with carbamazepine or dipyridamole. Adenosine can produce bronchoconstriction and should be avoided in asthmatics or those with significant chronic obstructive pulmonary disease. The action of adenosine is countered by xanthines such as caffeine or theophylline and may be ineffective in their presence. Following administration, patients will likely experience transient flushing, dyspnea, and chest tightness. The electrocardiogram tracing will likely show a 10–20-second period of asystole before normal sinus rhythm begins. The sum of this information brings into question the actual need or cost-effectiveness of maintaining adenosine in the office emergency kit.

Wide complex tachycardias and ectopies are generally ventricular in origin. Premature ventricular contractions are normally harmless unless they occur frequently enough to lower blood pressure, are multifocal in origin, or occur in couplets. In these cases myocardial ischemia is likely present and EMS transport is required. This is obviously true for runs of ventricular tachycardia as well. While waiting for EMS to arrive, an antiarrhythmic drug may be administered. Advanced cardiac life support guidelines address various agents, including procainamide, amiodarone, and sotalol, but lidocaine is still included as an option. IV lidocaine is more familiar, safer, and far less expensive to maintain in the emergency kit. It is available in 5-mL prefilled syringes as a 2% (20 mg/mL) concentration and administered initially as 1–1.5 mg/kg. Half this amount can be repeated in 5–10 minutes up to 3 mg/kg total while awaiting EMS transport.[13,15]

Transient episodes of hypertension are not uncommon, but they rarely require drug intervention. By convention, an acute hypertensive event is regarded as an urgency if the patient remains asymptomatic and an emergency if symptoms present—normally 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:

> The most sensible approach to the patient in the ED [emergency department] found to have very high blood pressure, without 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.[17]

A symptomatic hypertensive event is a medical emergency and requires 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 there should be no attempt to lower blood pressure.) Labetalol (Normodyne, Trandate) is the most common agent suggested for managing acute hypertensive events. It is a nonselective beta blocker that also imparts mild alpha-blocking action to provide vasodilation. It is formulated as 5 mg/mL in several sizes of vials, but the 20-mL vial is most appropriate for the office emergency kit. It has a fairly long duration of action (several hours or more) and should be titrated

Table 3. Optional Drugs for Advanced Providers

Drug	*Conventional Indication*
Adenosine	Supraventricular tachycardia
Aminophylline	Anaphylaxis, asthma
Glucagon	Hypoglycemia
Cortisol/methylprednisolone	Anaphylaxis
Procainamide	Wide complex tachycardia
Sotalol	Wide complex tachycardia
Vasopressin	Cardiac arrest

carefully in 10–20-mg increments every 5 minutes after reassessing the blood pressure and the electrocardiogram.[13,18] It should not be used in asthmatic patients, because its antagonist action on bronchial beta-2 receptors may induce bronchospasm. Caution is advised if a significant amount of epinephrine-containing local anesthetic has been administered within the previous 10 minutes, eg, $\geq$50 μg. Nonselective beta blockers can interact with epinephrine to produce a sudden elevation in blood pressure and further complicate matters.[19] Esmolol (Brevibloc) is a safer but somewhat less effective option when labetalol must be avoided.

Most of the so-called code drugs addressed in advanced cardiac life support courses for cardiac arrest have questionable merit. Prompt EMS response, effective chest compressions, airway management, and early defibrillation are the key aspects of cardiac arrest resuscitation. Approximately 90% of successful resuscitations occur following the initial defibrillation.[15] When the first defibrillation attempt fails, epinephrine 1 mg is indicated, but any small benefit is a slight increase in the rate of return of spontaneous circulation. No benefit is found in improving subsequent discharge within 30 days.[20] Furthermore, vasopressin offers no advantage over epinephrine.[20] The use of antiarrhythmic drugs following subsequent defibrillations likewise carries dubious benefit. There is no evidence to either support or refute the use of lidocaine, and limited evidence credits amiodarone with increased return of spontaneous circulation but no improvement in the rate of discharge.[21]

It is sensible to include 2 prefilled syringes of epinephrine in the emergency kit for administration while awaiting EMS. The actual benefit of maintaining amiodarone is dubious. Lidocaine is already included in the emergency kit, as addressed above for tachyarrhythmias, and is still recognized as an alternative to amiodarone. Nevertheless, amiodarone is inexpensive, and it is reasonable to include 3 single-dose 150-mg vials in the emergency kit. The initial dose is 300 mg and any subsequent dose is 150 mg. When following the current American Heart Association guideline for ventricular fibrillation/pulseless ventricular tachycardia, the actual timing for administration of an antiarrhythmic drug will likely be preceded by EMS arrival.

Additional drugs are sometimes included in guidelines published by professional organizations, but their actual benefit is doubtful. These are not addressed to any extent in this article because of their expense, complexity, and questionable usefulness in the dental office. Nevertheless, advanced providers may wish to become familiar with them as additional options at their discretion. These are summarized in Table 3.

REFERENCES

1. Abramowicz M, ed. Drugs past their expiration date. *Med Lett Drugs Ther*. 2009;51:101–102.

2. Rajpal S, Ali R, Bhatnagar A, Bhandari SK, Mittal G. Clinical and bioavailability studies of sublingually administered atropine sulfate. *Am J Emerg Med*. 2010;28:143–150.

3. Heniff MS, Moore GP, Trout A, Cordell WH, Nelson DR. Comparison of routes of flumazenil administration to reverse midazolam-induced respiratory depression in a canine model. *Acad Emerg Med*. 1997;4:1115–1118.

4. Hosaka K, Jackson D, Pickrell JE, Heima M, Milgrom P. Flumazenil reversal of sublingual triazolam: a randomized controlled clinical trial. *J Am Dent Assoc*. 2009;140:559–566.

5. Tran TP, Muelleman RL. Allergy, hypersensitivity and anaphylaxis. In: Marx JA, Hockberger RS, Walls RM, et al, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. Philadelphia, Pa: Mosby Elsevier; 2009.

6. Nowak RM, Tokarski GF. Asthma. In: Marx JA, Hockberger RS, Walls RM, et al, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. Philadelphia, Pa: Mosby Elsevier; 2009.

7. Pollack CV Jr, Braunwald E. 2007 update to the ACC/AHA guidelines for the management of patients with unstable angina and non-ST-segment elevation myocardial infarction: implications for emergency department practice. *Ann Emerg Med*. 2008;51:591–606.

8. O'Connor RE, Brady W, Brooks SC, et al. Part 10: acute coronary syndromes: 2010 American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2010;122(suppl 3):S787–S817.

9. Peto R, Collins R, Sackett D, et al. Drug therapy: aspirin, heparin, and fibrinolytic therapy in suspected acute myocardial infarction. *N Engl J Med*. 1997;336:847–860.

10. Brown JH, Laiken N. Muscarinic receptor agonists and antagonists. In: Brunton LL, Chabner BA, Knollmann BC, eds. *Goodman and Gilman's The Pharmacological Basis of Therapeutics*. 12th ed. New York, NY: McGraw-Hill Companies Inc; 2011.

11. Glick DB. The autonomic nervous system. In: Miller RD, Eriksson LI, Fleisher LA, et al, eds. *Miller's Anesthesia*. 7th ed. Philadelphia, Pa: Elsevier Churchill Livingstone; 2009.

12. Gunning RR, Garber AJ. Bioactivity of instant glucose. Failure of absorption through oral mucosa. *JAMA*. 1978;240: 1611–1612.

13. *Drug Facts and Comparisons in Clin-eguide*. New York, NY: Wolters Kluwer Health; 2012.

14. 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.

15. Neumar TW, Otto CW, Link MS, et al. Part 8: adult advanced cardiovascular life support: 2010 American Heart Association Guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2010;122:S729–S767.

16. Larson CP Jr. Laryngospasm—the best treatment. *Anesthesiology*. 1998;89:1293–1294.

17. Gallagher EJ. Hypertensive urgencies: treating the mercury? *Ann Emerg Med*. 2003;41:530–531.

18. Gray RO. Hypertension. In: Marx JA, Hockberger RS, Walls RM, et al, eds. *Rosen's Emergency Medicine: Concepts and Clinical Practice*. 7th ed. Philadelphia, Pa: Mosby Elsevier; 2009.

19. Becker DE. Adverse drug interactions. *Anesth Prog*. 2011;58:31–41.

20. Lin S, Callaway CW, Shah PS, et al. Adrenaline for out-of-hospital cardiac arrest resuscitation: a systematic review and meta-analysis of randomized controlled trials. *Resuscitation*. 2014;85(6):732–740.

21. Boyd T, Brady W. The "code drugs in cardiac arrest"—the use of cardioactive medications in cardiac arrest resuscitation. *Am J Emerg Med*. 2012;30:811–818.

CONTINUING EDUCATION QUESTIONS

1. According to *The Medical Letter on Drugs and Therapeutics*, drugs generally retain at least 90% of their potency for _____ past their expiration dates.

   A. 6 months
   B. 1 year
   C. 3 years
   D. 5 years

2. When managing bradycardia in adults, atropine may produce paradoxical slowing of heart rate if administered in doses lower than:

   A. 0.5 mg
   B. 1 mg
   C. 2 mg
   D. 3 mg

3. Succinylcholine must be available in offices that provide which of the following?

   (1) moderate sedation (2) deep sedation (3) general anesthesia

   A. 1 and 2
   B. 1 and 3
   C. 2 and 3
   D. 1, 2, and 3

4. For providers with advanced training, which of the following drugs can be used to manage all forms of narrow complex tachycardia?

   A. adenosine
   B. amiodarone
   C. esmolol
   D. lidocaine