Cardiovascular Collapse Associated With Irreversible Cardiomyopathy, Chronic Renal Failure, and Hypertension During Routine Dental Care

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Patients with multiple comorbid conditions visit the dental office every day, and although rare, complications from their conditions may occur during treatment. A case is presented of a 65-year-old African American woman with a history of severe cardiovascular disease, renal disease, and a reported local anesthetic allergy who experienced complete cardiovascular collapse during routine dental treatment from which she was successfully resuscitated. Treating clinicians should recognize the emerging symptoms and be proficient with a basic and advanced cardiac life support protocol to care for their patients safely and effectively until they can be transported to more advanced care facilities.

Key Words: Congestive heart failure; Cardiorenal syndrome; Cardiac arrest; Dental emergency.

Cardiovascular disease and stroke are very prevalent health ailments seen throughout the United States population. According to the American Heart Association's 2013 Update for Heart Disease and Stroke Statistics, in 2009 alone, cardiovascular disease-related deaths accounted for 32.3% of all deaths in the United States.¹ Patients with these diseases are regularly seen at the dental office, and although rare, complications from their illnesses may occur during treatment. Problems may present with a seemingly benign tachypnea and span across the severity spectrum to acute heart failure and death. Treating clinicians must be able to recognize, diagnose, and treat common emergent problems as well as respond effectively to less common or even rare, potentially life-threatening emergencies.² In this case report, a patient presented to the undergraduate dental school clinic for routine dental work and during a local anesthetic injection experienced severe chest pain, which progressed to complete cardiac and respiratory

failure in the dental chair, from which she was successfully resuscitated.

CASE DESCRIPTION

A 65-year old, 155-cm (5 foot, 1 inch), African American female weighing 70 kg presented to the University of Pittsburgh, School of Dental Medicine, for a single amalgam filling in the undergraduate dental clinic. The patient had a significant health history consisting of hypertension, angina, poorly controlled congestive heart failure, asthma, epilepsy, and end-stage renal disease for which she was hemodialyzed via a fistula in her left forearm 4 times per week. Other medical history included gastroesophageal reflux disease, hypercholesterolemia, anemia, latent tuberculosis, hypothyroidism, secondary hyperparathyroidism, insomnia, and glaucoma. The patient's current medication list is available in Table 1. Adverse drug reactions were reported to several medications during her initial screening appointment. These included "Novocain" resulting in dyspnea after

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Table 1. Patient Medication List*

<i>Medication</i>	<i>Pharmacologic Category</i>	<i>Patient's Intended Use</i>
Acetaminophen/hydrocodone	Analgesic combination	Postoperative pain (not currently taking)
Albuterol	Beta 2 agonist	Asthma (pm)
Amlodipine	Calcium channel blocker	Angina/hypertension
Aspirin 81 mg	Antiplatelet agent	Antithrombus formation
Calcium acetate	Phosphate-binding agent	Antihyperphosphatemia (ESRD)
Carvedilol	Beta-blocker	Congestive heart failure
Cinacalcet	Calcimimetic	Secondary hyperparathyroidism (ESRD)
Diphenhydramine	Antihistamine (H1 receptor antagonist)	Allergic rhinitis (pm)
Docusate	Stool softener	Stool softener
Epoetin alfa	Erythropoiesis-stimulating factor	Anemia (ESRD)
Famciclovir	Antiviral agent	Management of acute herpes zoster (not currently taking)
Lactulose	Laxative	Constipation
Lamotrigine	Anticonvulsant	Epilepsy
Levothyroxine	Synthetic thyroid hormone (T ₄)	Hypothyroidism
Nitroglycerine	Antianginal agent	Angina pectoris (pm)
Omeprazole	Proton pump inhibitor	Gastroesophageal reflux disease
Simvastatin	HMG-CoA reductase inhibitor	Antihyperlipidemia
Valsartan	Angiotensin II receptor blocker	Hypertension
Zolpidem	GABA/benzodiazepine receptor agonist	Sleep aid

* ESRD indicates end-stage renal disease; GABA, gamma-aminobutyric acid; HMG-CoA, 3-hydroxy-3-methylglutaryl-coenzyme A; pm, as needed.

injection, morphine with hallucinogenic symptoms, penicillin resulting in urticaria, and lisinopril precipitating a dry cough.

Upon arrival for an amalgam filling, the patient was seated in the dental chair and her medications and health history were reviewed by the student dentist. The patient reported there had not been any changes to her medications and that she had taken all prescribed medications as scheduled the night before and morning of her appointment. She did report a recent hospitalization 2 weeks prior for fluid overload. Her dialysis schedule was adjusted from 3 to 4 days per week after this event. She reported that her last dialysis was completed approximately 30 hours prior to her dental appointment. Baseline preoperative heart rate and blood pressure were taken and recorded at 84 beats per minute and 120/78 mm Hg, respectively. This was recorded in the electronic record. The dental student neglected to ask the patient if there had been any recent asthmatic or anginal episodes requiring use of her albuterol inhaler or nitroglycerine, respectively. Start checks were given by the supervising dentist.

One dental cartridge (1.7 mL) of 4% articaine (68 mg) 1:100,000 epinephrine (0.017 mg) was given to the patient as a supraperiosteal infiltration in the buccal vestibule near the right mandibular first premolar. After waiting several minutes for appropriate anesthesia, the student attempted to place the rubber dam clamp on the lower right mandibular first molar. The patient reacted to pain from placement of the clamp, so it was removed and an additional eighth of a cartridge of 4% articaine (9

mg) 1:100,000 epinephrine (0.004mg) was given in the interdental papilla. Soon after this second injection, the patient became anxious, complained of shortness of breath, and asked the student to sit her up in the dental chair. Before the chair was in its full upright position, the patient sat up, grabbed her purse, and self-administered 3 sprays of sublingual nitroglycerine, which she was prescribed as needed for anginal pain. She then informed the student that her heart was racing and he needed to call for help.

The Department of Dental Anesthesiology, the designated first responder for all medical emergencies occurring within the School of Dental Medicine, was called for emergency help of a patient who was having difficulty breathing and severe chest pain. The students were instructed to place the patient on full face-mask oxygen and attempt to keep her calm until the anesthesia department arrived on scene. Two anesthesia residents and 1 attending arrived to find the patient receiving full face-mask oxygen at a flow of 10 L/min. In obvious distress, she was sitting straight up in the dental chair complaining of difficulty breathing, severe chest pain, and impending doom. She repeatedly said, "Get me to the hospital, I'm not going to make it!" The team began applying monitors (a 3-lead electrocardiograph [EKG], automatic blood pressure cuff, and pulse oximeter) and questioned the patient. The treating dental student also informed anesthesia personnel that another blood pressure measurement was taken prior to their arrival and that her new pressure was 160/100 mm Hg. No new heart rate was reported by the student at that time.

The anesthesia team activated the school's emergency protocol in which emergency medical service was summoned to provide hospital transport.

The patient's vital signs were as follows after initial reading by the monitor:

- Blood pressure: 220/112 mm Hg
- Pulse: 115 beats per minute
- SpO₂: 100% on 10 L/min O₂ via full-face mask
- Respiratory rate: 22 breaths per minute

Attempts were made to reassure the patient and help reduce anxiety. The patient was encouraged to try and slow her breathing. She asked for an albuterol inhaler, which was offered. She self-administered 3 puffs. The patient's distress continued. No significant increase in heart rate was observed.

Intravenous access was established using a 22-gauge catheter in the dorsum of the right hand. A slow infusion of 0.9% normal saline (NS) via microdrip (60 gtt/mL) intravenous tubing allowing for a precise flow rate was started. Tachycardia was addressed with the cardioselective beta₁ receptor blocker esmolol (5 mg), without much effect. Diphenhydramine (50 mg) was administered in the unlikely event the patient was experiencing an allergic reaction to the local anesthetic. Reassessment of the patient's vital signs showed no response to therapy. Labetalol (10 mg), a mixed alpha/beta antagonist, was administered in an effort to further reduce heart rate and blood pressure.

A few minutes later, the patient's rapid respiratory rate slowed, and there was loss of consciousness. The patient was positioned supine in a reclined dental chair. Palpable radial and carotid pulses were present while the monitor showed a heart rate of 92 bpm in sinus rhythm. All respiratory effort eventually ceased. Positive-pressure ventilatory assistance with carbon dioxide sampling and 100% oxygen was administered via a bag-valve-mask device while supplies were gathered to secure an airway. The NPO status of the patient was unknown, and prevention of aspiration of stomach contents was of concern. Propofol (50 mg and 50 mg) and succinylcholine (100 mg) were administered intravenously to facilitate intubation. Cricoid pressure was held, and direct laryngoscopy with a Macintosh 3 blade was performed, resulting in a Cormack Grade I view of the patient's vocal cords. An 8.0-mm straight cuffed oral endotracheal tube was inserted into the patient's trachea, cuff inflated, and attached to the bag-valve mask. Bilateral breath sounds were confirmed with end-tidal CO₂, and the endotracheal tube was secured with tape at 22 cm in relation to the patient's maxillary dentition.

As the tube was being secured, the patient's heart rate slowed to less than 40 beats per minute. The EKG rhythm showed a sinus bradycardia at 38 bpm with

normal QRS morphology. Atropine (0.8 mg) was administered with no resulting increase in heart rate. Blood pressure could not be obtained by the automatic cuff, nor was a radial or carotid pulse detected. The patient's shirt was removed, and chest compressions were initiated while automated external defibrillator pads were applied to the patient. Cardiopulmonary resuscitation (CPR) was interrupted while the automated external defibrillator analyzed the rhythm. No shock was advised, and asystole was noted on the EKG. Epinephrine (1 mg) was given intravenously as CPR was reinitiated and continued for 2 minutes to allow for circulation of the drug and again held for a rhythm check. The EKG revealed sinus tachycardia with a rate of 118 bpm, and blood pressure was measured at 238/130 mm Hg with palpable radial and carotid pulses. Midazolam (5 mg) was administered for sedative and amnestic purposes while the bag-valve mask was disconnected and the endotracheal tube suctioned to reveal pink, frothy fluid that had accumulated in the patient's airway. Furosemide (50 mg), although likely ineffective due to chronic renal disease, was given.

Over the next few minutes, the patient's vital signs normalized to a blood pressure of 128/75 mm Hg, a heart rate of 93 bpm in sinus rhythm, and an SpO₂ of 92%. Emergency medical service had arrived on scene and had begun to prepare the patient for transport. In their preparation, emergency medical service providers removed the tape securing the endotracheal tube and replaced it with a commercial tube stabilizer. Upon manipulation of the tube, the patient's heart rate again slowed to the point of asystole. CPR was restarted, and a second dose of epinephrine (1 mg) was given intravenously. CPR was continued for 2 minutes and was held for a rhythm check. The patient had returned to a sinus tachycardia with a rate of >110 bpm and palpable carotid and radial pulses. The patient was transferred from the dental chair to the EMS stretcher, placed on a transport monitor, and taken to the emergency room at the University of Pittsburgh Medical Center Presbyterian Hospital. The dental student and anesthesia resident accompanied the patient in the ambulance to the emergency room in order to provide a report for emergency room physicians.

In the emergency room, the patient had another episode of asystole and was again resuscitated with epinephrine (1 mg) and chest compressions. Calcium gluconate (1 g) was administered to stabilize the cardiac membrane and prevent further arrhythmias. Sedation was initiated with a propofol infusion. Upon stabilization, she was taken to the cardiovascular catheterization lab. Her catheterization results showed no evidence of atherosclerosis/blockage in the coronary system or any areas of infarcted tissue. The patient was admitted to the

cardiac intensive care unit for follow-up and monitoring. A bedside transthoracic echocardiogram was done that revealed an ejection fraction of 20% (prehospitalization recorded ejection fraction: 43%) immediately postarrest. Once stable, 3 days postarrest, sedation was discontinued and the patient was extubated. After returning to baseline, the patient was answering questions, following commands, and displayed intact anterograde memory. The patient required surgery to remove clots and revascularize the dialysis shunt in her left forearm due to stagnant blood flow during the cardiac episode. One week postarrest, her ejection fraction had rebounded to 46%, and she displayed no physical or neurological impairment. She was released from the hospital 8 days following the event. Her discharge diagnosis was respiratory failure and cardiac arrest secondary to stress-induced cardiomyopathy. The physicians speculated that a reaction to the local anesthetic could have been a precipitating factor.

DISCUSSION

Risk versus benefit must always be evaluated when it comes to patient care. The American Society of Anesthesiology Physical Classification System is commonly used for evaluation of medical risk associated with all surgical procedures.³ Perhaps the dental team did not appreciate the complexity of the patient's medical history. Certainly, a more in-depth interval health history should have been obtained by the treating student dentist/dentist supervisor. This error resulted in failure to obtain the needed medical consultation and necessary evaluations to ensure that the patient was as optimized as possible prior to initiating treatment. Caution must be used by clinicians when performing elective, outpatient procedures on patients with recent hospitalizations for life-threatening illness.

Medically compromised patients, including all designated American Society of Anesthesiology IV and many American Society of Anesthesiology III, should have a thorough preoperative history and physical examination performed by their primary care physician with any other testing (12-lead electrocardiogram, echocardiography, stress testing, etc) deemed necessary to assess the patient's functional capacity and likelihood of poor outcome during dental treatment. This will help ensure that the patient is optimized and capable of undergoing the stress that these procedures precipitate. If the treating clinician or primary care physician suspects a high probability of poor outcome, alternatives to treatment in the office, such as referral to more advanced care providers or treatment at a local hospital, may be

necessary to ensure patient safety and reduce morbidity and mortality.

For the first time in 2010, after years of successful dental treatment under local anesthesia, the patient reported that she had an allergic reaction to what she called "Novocain" causing shortness of breath during a dental appointment. As practicing dentists know, patients often refer to the entire drug class of local anesthetics as "Novocain" and repeatedly report having an allergy to the class. However, investigation shows that most reported "allergies" to local anesthetics are psychogenic in origin or related to unwanted epinephrine side effects and that true allergies are extremely rare.^{4–6} Although this patient was tachypneic, most of the clinical signs were the exact opposite of true allergy/anaphylaxis. The patient exhibited extremely high blood pressure, no visible skin involvement, and no apparent wheezing. The clinical manifestations were more congruent with signs and symptoms of the patient's diagnosed congestive heart failure during a stressful situation rather than a local anesthetic allergy. In the case described above, 1 dental cartridge (1.7 mL) of 4% articaine, an amide local anesthetic, with 1:100,000 epinephrine was injected in the buccal vestibule near the mandibular right first premolar, and approximately one eighth of a carpule was injected into the papilla around the mandibular right first molar for placement of the rubber dam clamp. In retrospect, the patient may have been better served by receiving an inferior alveolar nerve block with a plain local anesthetic, such as 3% mepivacaine. Although the amount of epinephrine given, approximately 0.021 mg, is well below the maximum recommended dose of 0.04 mg in cardiovascular compromised patients, perhaps her cardiovascular and renal disease should have precluded the use of a any vasoconstrictor.⁵ It is unlikely, yet possible, that the small amount of epinephrine given could have precipitated such a dramatic response.

The most important independent risk factor for poor outcome and all-cause mortality in patients with heart failure is renal dysfunction. Blood volume, vascular tone, and hemodynamic stability depend on a delicate balance between the heart and kidney.^{7,8} Each dysfunctional organ can initiate or worsen pathology in the other organ. Severe dysfunction rarely occurs in isolation. The disease process is perpetuated through common hemodynamic, neurohormonal, and immunologic/biochemical feedback pathways. Baseline glomerular filtration rate appears to have a stronger association with poor prognosis compared with left ventricular ejection fraction, or New York Heart Association functional class.^{9,10} Therefore, reversing or treating renal dysfunction may improve heart failure.¹¹ Because renal dysfunction in patients with congestive heart failure has such a poor

Table 2. Types of Cardiorenal Syndrome

Type	Definition	Primary Event	Secondary Event
Acute cardiorenal (type 1)	Acute worsening of heart function leading to kidney injury and/or dysfunction	Acute heart failure, acute coronary syndrome, cardiogenic shock	Acute kidney injury
Chronic cardiorenal (type 2)	Chronic abnormalities in heart function leading to kidney injury or dysfunction	Chronic abnormalities in cardiac function	Progressive chronic kidney disease
Acute renocardiac (type 3)	Acute worsening of kidney function leading to heart injury and/or dysfunction	Abrupt worsening of renal function	Acute cardiac disorder (eg, heart failure, arrhythmia, or pulmonary edema)
Chronic renocardiac (type 4)	Chronic kidney disease leading to heart injury, disease, and/or dysfunction	Chronic kidney disease	Decreased cardiac function, cardiac hypertrophy, and/or increased risk of adverse events
Secondary cardiorenal syndrome (type 5)	Systemic conditions leading to simultaneous injury and/or dysfunction of heart and kidney	Systemic disease	Both cardiac and renal dysfunction

prognosis, treating clinicians must understand the pathophysiological connection. This connection is often referred to as cardiorenal syndrome.

Cardiorenal syndrome is defined by a physiologically normal kidney becoming pathological over time due to a diseased heart or a normal heart becoming pathological over time due to impaired renal function. Cardiorenal syndrome includes a variety of acute or chronic conditions whereby the primary failing organ can be either the heart or the kidneys.¹²

There are currently 5 classification subtypes of cardiorenal syndrome that reflect its pathophysiology, time frame, and the development of concurrent cardiac and renal disease. The different subtypes can be seen in Table 2.¹³ There is great complexity involved with this condition and a lack of clarity with regard to definitive diagnosis and management.¹⁴ Chronic hypervolemia, hyperkalemia, and untreated uremias can contribute to pulmonary edema, cardiac arrhythmias, and myocardial depression, respectively. A patient with cardiorenal syndrome may be prone to acute decompensated heart failure due to diastolic dysfunction, increases in blood pressure from excessive activation of the renin-angiotensin-aldosterone system, and eventual ischemia from increased myocardial oxygen demand related to profound peripheral vasoconstriction.¹⁵

Renal failure is common in patients with heart failure and translates to a dramatic increase in mortality. A systematic review performed by Smith et al¹⁶ identified studies characterizing the association between heart failure and renal impairment. It consisted of 80,098 hospitalized and nonhospitalized heart failure patients who were then divided into classes of those with any impairment (creatinine >1 mg/dL; estimated glomerular filtration rate <90 mL/min) and moderate to severe

renal impairment (creatinine ≥ 1.5 mg/dL; estimated glomerular filtration rate <53 mL/min) groups. Sixty-three percent of all patients had some renal impairment, while 29% were determined to have moderate to severe renal impairment based on these parameters. After 1-year follow-up with all patients, 38% with any renal impairment and 51% with moderate to severe impairment had died versus only 24% with no baseline renal dysfunction. It was determined that mortality worsened incrementally across the range of renal function, with a 15% increased risk of death for every 0.5-mg/dL increase in creatinine and 7% increased risk for every 10-mL/min decrease in estimated glomerular filtration rate.¹⁶

The dominant pathophysiology of heart disease in patients who are on dialysis is that of diastolic dysfunction and left ventricular hypertrophy. Patients with left ventricular hypertrophy have a resulting reduction in systolic function as well, predisposing them to a risk of sudden cardiovascular collapse and death.¹⁷ These individuals have a significantly compromised ability to handle stress and the accompanying elevations in heart rate and blood pressure that come with stress-induced catecholamine release. Diligent management with beta-blockers, angiotensin receptor blockers, and angiotensin-converting enzyme inhibitors as well as routine dialysis treatments give this patient population the best possible prognosis for cardiovascular and renal stability.

The dentist who chooses to treat patients with complex medical problems must also know how well the patient adheres to his or her prescribed medication regimen and take a careful interval functional assessment. In this case, although the dental student did ask the patient if she had taken her medications the day of the

appointment, he did not ask whether she had been taking all prescribed medications in the extended preoperative period. Her vitals, with a preoperative heart rate of 84 bpm and a heart rate of 115 bpm after injections, would indicate that she may not have been compliant with her medications, as one would expect optimal beta blockade to inhibit tachycardia and attenuate blood pressure during a crisis.

Prior to cardiovascular collapse, the patient was able to articulate some medical history and current details on her status. The quick medical history review revealed the patient's complicated problem list, which included but was not limited to congestive heart failure, end-stage renal disease, asthma, and anxiety. It was quickly apparent that the patient's status was declining and that intravenous access was necessary to deliver life-saving medications. Access was quickly established in the dorsum of the patient's right hand, and an infusion of normal saline was started. Normal saline is the carrier of choice in end-stage renal disease patients because of its lack of potassium in solution.¹⁸ Lactated Ringers, a balanced salt solution, contains 4 mEq/L of potassium, which can cause hyperkalemia if given in large quantities to this patient population who are unable to excrete the excess potassium effectively. Because of the patient's end-stage renal disease and congestive heart failure, saline was used sparingly and only as a carrier for medications, as fluid overload was a concern. It was necessary to avoid placing the intravenous catheter in her left arm to prevent possible damage to the arteriovenous shunt used for dialysis treatments.

Activation of the pituitary-adrenal axis is a prominent response to stress, which may be attributed to pain and anxiety. Small amounts of endogenous catecholamines, including epinephrine, and larger amounts of cortisol and vasopressin slowly upset the patient's delicate homeostatic balance.¹⁸ Upon arrival, sedation was considered in an effort to control anxiety but was not immediately administered based on lack of NPO status, extensive medical history, and the patient's current vital signs and symptoms. Three puffs of albuterol were initially administered, as the patient indicated that she was having trouble breathing, like during a typical asthma attack. This proved ineffective, provided no relief, and the patient's condition continued to decline. Of note, beta₂ adrenergic agents, such as albuterol sulfate, lower serum potassium levels and have been used to treat hyperkalemia in patients with chronic renal failure.¹⁹ With a heart rate >100 bpm and a blood pressure of 220/112 mm Hg, compensated heart failure quickly became decompensated. Beta-blockers were administered to reduce cardiac output and stress on an already compromised heart. Esmolol, a beta₁ selective blocker, was given first because of its immediate peak effect and

short duration of action. Rate control was of primary concern; the goal was to reduce cardiac output (cardiac output = heart rate × stroke volume). After the first small 5-mg esmolol dose, no rate control was achieved. Perhaps before moving to a longer-acting mixed agent, such as labetalol, escalating doses of esmolol could have been administered every minute or two until the heart rate responded accordingly. Because the patient was experiencing tachycardia and hypertension, the decision was made to switch to labetalol with both alpha and beta receptor-blocking properties to more effectively bring down the blood pressure.²⁰ It is commonly administered in hypertensive emergencies as an initial slow injection of 20 mg followed by 40–80 mg every 10 minutes (to a maximum dose of 300 mg) until desired pressure is reached. It has an onset of action of 1–2 minutes with peak effect in 10–15 minutes.²¹ Labetalol (10 mg) was administered, which also had no immediate effect on the heart rate or blood pressure. The tachypnea slowed to respiratory depression, which ultimately led to respiratory arrest, with loss of consciousness. A pulse and pressure were still present. Some may contribute the collapse to what may be perceived as an aggressive therapy. Managing patients with a delicate homeostatic balance can prove to be a unique challenge for practitioners.

Propofol and succinylcholine were used to facilitate tracheal intubation. With the very limited health history, the patient's persistent high blood pressure, and a modified rapid sequence intubation necessary to prevent aspiration of stomach contents, propofol/succinylcholine seemed to be appropriate. After revisiting the event, etomidate may have been a better choice than propofol as it provides cardiovascular stability and could have prevented a significant reduction in blood pressure. Perhaps tracheal intubation without drugs should have been considered.

After successful resuscitation from the first asystolic event, midazolam (5 mg) was administered. Since the entire event may have been precipitated by a catecholamine surge secondary to stress and anxiety, the perceived benefit in the now-intubated patient was felt to be worth the risk.

During the patient's hospitalization, laboratory results showed a slight rise in troponin I levels on the day following the event. Troponin I is a key blood marker seen when muscle tissue dies and is an indicator for acute myocardial infarction.²² The patient was taken to the cardiac catheterization lab, and no areas of true infarcted tissue were visualized. We speculated, with corroborating literature, that the rise in troponin I levels was secondary to the chest compressions done during CPR.²³ The patient's 12-lead EKG postarrest showed a sinus tachycardia with occasional premature ventricular com-

plexes and a left bundle branch block. Lab values taken immediately postarrest indicated that the patient's electrolytes were within normal limits, ruling out hyperkalemia-induced arrhythmias as the cause of the episode.

The patient had to undergo surgery to revascularize the dialysis shunt placed in her left forearm; however, she made a full physical and neurological recovery after being discharged from the hospital. Although it has not been decided how she will undergo dental treatment in the future, the safest and most likely option is to have the patient admitted to the hospital and have all remaining dental work completed using moderate/deep sedation or general anesthesia. This ensures that the patient will be monitored with intravenous access so that the catecholamine response to the local anesthetic injections will be attenuated and, in the event that another emergency does occur, hospital resources are immediately available to manage this patient with such an extensive medical and dental history.

CONCLUSION

As with any type of surgery, dental treatment, no matter how routine, can bring about stresses that can precipitate serious, life-threatening events. With effective implementation of stress-reduction techniques, all but about 10% of life-threatening situations in the dental office can be prevented. The remaining 10% are classified as sudden and unexpected and are unpreventable.²⁴

General practitioners treat patients with multiple medical conditions every day. Clinicians, when performing elective, outpatient procedures on patients with multiple severe and/or complex comorbidities, including recent hospitalizations for life-threatening illness, must use caution. Precautionary measures include the dentist taking a thorough history and physical examination including complete vital signs. Consultation with the patient's primary care physician and referral to more advanced care providers are frequently necessary. Medical optimization reduces risk. Stress reduction protocols with sedation and judicious use of vasoconstrictors should be considered. It is also advisable that dentists who manage patients with significant medical compromise stay current in basic life support training and develop an emergency response plan to ensure that patient morbidity and mortality may be minimized or avoided.

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