Local Anesthetics
And Medically Complex Patients

by Alan W. Budenz, MS, DDS, MBA

Through steady advances in medical care, many patients who even a short time ago would not have survived systemic illnesses, or at best would have been confined to their beds or homes, are now active, mobile members of our society. As a result, patients with increasingly complex medical situations are in a position to seek dental treatment in private practice offices. The dentist must be prepared to deliver safe, efficient, and competent dental care by understanding the patient’s medical condition and medications. This information must be integrated into the dentist’s knowledge of the physiologic stresses of dental procedures and the pharmacology of the medications used in dentistry.

The injection of local anesthetic solutions to achieve anesthesia is one of the most commonly performed dental procedures. Prior to administering any medication, including local anesthetics, it is appropriate for the dentist to take a complete medical history and follow up any questions with the patient or through a consultation with the patient’s physician.

Though local anesthetics are remarkably safe at therapeutic doses, the practitioner treating medically complex patients must address two basic concerns pertinent to the use of local anesthetic agents: existing systemic diseases that may be exacerbated by the anesthetic agent and medications that may have adverse interactions with local anesthetic agents. This review will focus on a broad range of medical problems and considerations for the use of local anesthetics in these patient populations.

Cardiovascular Diseases

Local anesthetic agents themselves can affect the cardiovascular system, especially at higher doses. Cardiovascular manifestations are usually depressant and are characterized by bradycardia, hypotension, and cardiovascular collapse, potentially leading to cardiac arrest. The initial signs and symptoms of depressed cardiovascular function commonly result from vasovagal reactions (dizziness and fainting), particularly if the patient is in an upright position.\textsuperscript{1,2}
Cardiovascular diseases constituting contraindications to the use of local anesthetics in general, and to the use of vasoconstrictors in local anesthetics in particular, are often discussed in terms of absolute as opposed to relative contraindications.

Absolute contraindications for the use of local anesthetics with or without vasoconstrictors in patients with cardiovascular diseases exist only if the patient's condition is determined, by the dentist's review of the health history, to be medically unstable to the degree of posing undue risk to the patient's safety. Dental care should be deferred in these patients until their medical conditions have been stabilized under the care of their physicians. For patients with stabilized cardiovascular diseases, dental treatment may usually be delivered in near routine fashion, although, as the following sections will emphasize, the amount of vasoconstrictor-containing local anesthetic used may need to be limited and the patient carefully monitored.

Table 1. Summary of Local Anesthetic Use in Medically Complex Patients

<table>
<thead>
<tr>
<th>DISEASE</th>
<th>PRECAUTIONS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular disease</td>
<td>Use stress reduction protocol (Table 2).</td>
</tr>
<tr>
<td>Hypertension (controlled)</td>
<td>Minimize vasoconstrictor use.</td>
</tr>
<tr>
<td>• Nonselective betablockers (propranolol)</td>
<td>Avoid vasoconstrictors.</td>
</tr>
<tr>
<td>• Selective beta-blockers (Lopressor)</td>
<td>Minimize vasoconstrictor use.</td>
</tr>
<tr>
<td>• Other antihypertensive drugs (Clonidine, Aldomet, Reserpine)</td>
<td>Minimize vasoconstrictor use; monitor for injection site ischemia.</td>
</tr>
<tr>
<td>Angina and post-myocardial infarction</td>
<td>Minimize vasoconstrictor use.</td>
</tr>
<tr>
<td>Cardiac dysrhythmia (refractory)</td>
<td>Minimize vasoconstrictor use; avoid PDL &amp; intraosseous injections.</td>
</tr>
<tr>
<td>Congestive heart failure (controlled)</td>
<td>Minimize vasoconstrictor use.</td>
</tr>
<tr>
<td>• Digitalis glycosides (Digoxin)</td>
<td>Monitor for arrhythmias if using vasoconstrictor.</td>
</tr>
<tr>
<td>• Long-acting nitrates and vasodilators (Nitroglycerin, Isordil, Minipres)</td>
<td>Watch for decreased anesthetic duration.</td>
</tr>
<tr>
<td>Cerebrovascular accident (stroke)</td>
<td>No special precautions</td>
</tr>
<tr>
<td>Pulmonary Disease</td>
<td></td>
</tr>
<tr>
<td>Asthma</td>
<td>Stress reduction protocol (Table 2) ; minimize vasoconstrictor use.</td>
</tr>
<tr>
<td>Chronic obstructive pulmonary disease</td>
<td>No special precautions</td>
</tr>
<tr>
<td>Renal Disease (severe)</td>
<td>Reduced dosage; extend time between</td>
</tr>
<tr>
<td>Disease</td>
<td>Protocol/Precaution</td>
</tr>
<tr>
<td>-------------------------------</td>
<td>----------------------------------------------------------</td>
</tr>
<tr>
<td>Hepatic Disease (severe)</td>
<td>Reduced dosage; extend time between injections.</td>
</tr>
<tr>
<td>Pancreatic Disease</td>
<td></td>
</tr>
<tr>
<td>Diabetes</td>
<td>Stress-reduction protocol (Table 2).</td>
</tr>
<tr>
<td>Adrenal Disease</td>
<td></td>
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<tr>
<td>Adrenal insufficiency</td>
<td>Stress-reduction protocol (Table 2).</td>
</tr>
<tr>
<td>Pheochromocytoma</td>
<td>Avoid vasoconstrictors.</td>
</tr>
<tr>
<td>Thyroid Disease</td>
<td></td>
</tr>
<tr>
<td>Hyperthyroidism (controlled or euthyroid)</td>
<td>No special precautions.</td>
</tr>
<tr>
<td>Hypothyroidism (mild)</td>
<td>No special precautions.</td>
</tr>
<tr>
<td>Musculoskeletal Disease</td>
<td></td>
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</tbody>
</table>
Malignant hyperthermia  No special precautions.

Blood Dyscrasias

Sickle cell anemia  Stress reduction protocol (Table 2); minimize vasoconstrictor use.

Methemoglobinemia  Avoid prilocaine (Citanest).

DRUG INTERACTIONS  PRECAUTIONS

Antipsychotic drugs (Thorazine)  No special precautions.

Cocaine  Delay treatment for six to 72 hours.

Tricyclic antidepressants (Elavil) Minimize epinephrine; avoid levonordefrin.

Monoamine oxidase inhibitors  No special precautions.

Antianxiety drugs (benzodiazepines)  Minimize all anesthetics

Hypertension

It is estimated that more than 50 million people in the United States have high blood pressure or are taking antihypertensive medications.\textsuperscript{5,6} Because lack of compliance is a major problem in medical treatment of hypertensive patients, the dental practitioner is wise to measure blood pressure and evaluate the patient’s status at every visit.
The decision regarding whether a local anesthetic agent containing vasoconstrictor should be administered to a patient with hypertension or other cardiovascular disease is a common concern amongst dental practitioners. A rational approach to this question is to recall the effects and mechanism of the vasoconstrictors. One of the primary effects, and advantages, of vasoconstrictors in dental local anesthetics is to delay the absorption of the anesthetic into the systemic circulation. This increases the depth and the duration of anesthesia while decreasing the risk of toxic reaction. Additionally, the vasoconstrictor provides local hemostasis. Epinephrine and levonordefrin (neo-cobefrin) are the two vasoconstrictor agents commonly used in dental local anesthetic formulations. Although they do have slightly differing cardiac effects, they carry the same precautions for their use.

Table 2. Stress Reduction Protocol

- Morning appointments are usually best.
- Keep appointments as short as possible.
- Freely discuss any questions, concerns, or fears that the patient has.
- Establish an honest, supportive relationship with the patient.
- Maintain a calm, quiet, professional environment.
- Provide clear explanations of what the patient should expect and feel.
- Premedicate with benzodiazepines if needed.
- Ensure good pain control through judicious selection of local anesthetic agents appropriate for maintenance of patient comfort throughout the procedure.
- Use nitrous oxide as needed (avoid hypoxia).
- Use gradual position changes to avoid postural hypotension.
- End the appointment if the patient appears overstressed.
There are no absolute contraindications to the use of vasoconstrictors in dental local anesthetics, since epinephrine is an endogenously produced neurotransmitter. In 1964, the American Heart Association and the American Dental Association concluded a joint conference by stating that “the typical concentrations of vasoconstrictors contained in local anesthetics are not contraindicated with cardiovascular disease so long as preliminary aspiration is practiced, the agent is injected slowly, and the smallest effective dose is administered.”

It has long been recommended that the total dosage of epinephrine be limited to 0.04 mg in cardiac risk patients. This equates to approximately two cartridges of 1:100,000 epinephrine-containing local anesthetic. Levonordefrin is considered to be roughly one-fifth as effective a vasoconstrictor as epinephrine and is therefore used in a 1:20,000 concentration. In this concentration, levonordefrin is considered to carry the same clinical risks as 1:100,000 epinephrine. The results of a number of studies indicate that the use of one to two 1.8 ml cartridges of local anesthetic containing a vasoconstrictor is of little clinical significance for most patients with hypertension or other cardiovascular diseases, and that the benefits of maintaining adequate anesthesia for the duration of the procedure far outweighs the risks.

However, the use of more than two cartridges of local anesthetic with a vasoconstrictor should be considered a relative rather than an absolute contraindication. If, after administering one to two cartridges of vasoconstrictor-containing local anesthetic with careful preliminary aspiration and slow injection, the patient exhibits no signs or symptoms of cardiac alteration, additional vasoconstrictor-containing local anesthetic may be used, if necessary, or local anesthetic without epinephrine can be used. Some practitioners prefer to achieve initial anesthesia with a nonvasoconstrictor-containing anesthetic agent such as 3 percent mepivacaine or 4 percent prilocaine plain and then use a small amount of local anesthetic with vasoconstrictor to supplement cases of inadequate anesthesia. While this is a viable protocol, a safer choice is to use a minimal amount of vasoconstrictor-containing local anesthetic first and then supplement as necessary with nonvasoconstrictor-containing agents.

The advantage of using the epinephrine-containing anesthetic first is that it will minimize blood flow in the injection site, thereby holding the local anesthetic in place, optimizing the anesthetic effect while minimizing the rate of plasma uptake and potential toxicity. Since nonvasoconstrictor containing local anesthetics produce localized vasodilatations, addition of a vasoconstrictor-containing agent after first
injecting with a nonvasoconstrictor containing local anesthetic can be expected to produce increased cardiovascular alterations. The goal should always be to minimize the dosage of local anesthetic with or without vasoconstrictor; but if additional vasoconstrictor will provide improved pain control for the dental procedure, it is not contraindicated.

If a patient has severe uncontrolled hypertension, elective dental treatment should be delayed until his or her physician can get the blood pressure under control. But if emergency dental treatment is needed, the clinician may elect to sedate the patient with valium and use one to two cartridges of local anesthetic with a vasoconstrictor. This dose will have minimal physiologic effect and will provide prolonged anesthesia. The greater risk in such a scenario is that without the epinephrine the anesthesia will wear off too soon; and the endogenous epinephrine produced by the patient, because of pain from the dental procedure, will be much greater and more detrimental than the small amount of epinephrine in the dental anesthetic cartridge.15, 18

Another concern for the dental practitioner is the possibility of an adverse interaction between the local anesthetic agent and a patient’s antihypertensive medication, particularly the adrenergic blocking agents. The nonselective beta-adrenergic drugs, such as propranolol (Inderal), pose the greatest risk of adverse interaction.19 In these patients, an injection of vasoconstrictor-containing local anesthetic may produce a marked peripheral vasoconstriction, which could potentially result in a dangerous increase in blood pressure due to the pre-existing medication-induced inhibition of the compensatory skeletal muscle vasodilatation. This compensatory skeletal muscle vasodilatation normally acts to balance the peripheral vasoconstriction effects in nonmedicated patients. The cardioselective beta blockers (Lopressor, Tenormin) carry less risk of adverse reactions. Both classes of beta blockers may increase serum levels of anesthetic solutions due to competitive reduction of hepatic clearance.20 Though these considerations are theoretically important, there is still little risk of a problem if the total dose of anesthetic, with 1:100,000 epinephrine or its equivalent, is limited to one to two 1.8 ml cartridges.

Other antihypertensive medications, such as the central sympatholytic drugs, for example Clonidine and Methyldopa (Aldomet), and the peripheral adrenergic antagonists such as Reserpine as well as the direct vasodilators, may potentiate adrenergic receptor sensitivity to sympathomimetics, resulting in a magnified systemic response to vasoconstrictor-containing anesthetics.19 However, once again, these medications pose no significant risk as long as the vasocon-
Angina Pectoris and Post-Myocardial Infarction

Patients with stable angina without a history of infarction generally have a significantly lower risk of adverse reactions to dental anesthetics than do patients with unstable angina or a history of recent (less than six months prior) myocardial infarction. Stress and anxiety reduction play a crucial role in the management of these patients, and excellent pain control throughout the dental procedure is essential. The use of local anesthetics containing a vasoconstrictor is recommended as part of the stress reduction protocol for these patients (Table 1). The dosage of the vasoconstrictor should be limited to that contained in one to two 1.8 ml cartridges of vasoconstrictor-containing anesthetic. For patients with unstable angina, recent myocardial infarction (less than six months), or recent coronary artery bypass graft surgery (less than three months), elective dental treatment should be postponed. If emergency treatment is required, stress-reduction protocols with antianxiety agents are appropriate, and the above limitation of one to two cartridges of vasoconstrictor-containing anesthetic must be strictly observed.

Cardiac Dysrhythmia

Proper identification of patients with an existing cardiac dysrhythmia, commonly called arrhythmias, or those patients who may be prone to developing dysrhythmia, is essential and requires a physician consult to determine the current status. Patients with coronary atherosclerotic heart disease, ischemic heart disease, or congestive heart failure are susceptible to stress-induced cardiac dysrhythmias. Stress-and anxiety-reduction protocols are again of paramount importance. Local anesthetic agents containing vasoconstrictors are appropriate for maintenance of adequate pain control during dental procedures. Elective dentistry should be avoided in patients with severe or refractory dysrhythmias until their physicians can get the problem under control. Once again, it is reasonable and safe to limit the total dose of local anesthetic to no more than two 1.8 ml cartridges per appointment. The use of periodontal ligament or intraosseous injections using a
vasoconstrictor-containing local anesthetic is not recommended in these patients.\textsuperscript{22}

Congestive Heart Failure

Patients who are under physician care and well-controlled with no complications can be treated relatively routinely. Limitation of vasoconstrictor dosage to two 1.8 ml cartridges of vasoconstrictor-containing anesthetic is advised. Patients taking digitalis glycosides, such as digoxin, should be carefully monitored if vasoconstrictors are used since interaction of the two drugs may precipitate dysrhythmias. Additionally, patients taking long-acting nitrate medications, such as nitroglycerin, Isordil, or Isorbid, or taking a vasodilator medication such as Minipres may show decreased effectiveness of the vasoconstrictor in local anesthetics, and therefore shorter anesthesia duration.\textsuperscript{21}

Cerebrovascular Accident

Atherosclerosis, hypertensive vascular disease, and cardiac pathoses such as myocardial infarction and atrial fibrillation are commonly associated with the occurrence of strokes. A patient who has suffered a stroke is at greater risk for having another one than is a patient who has never had one. It is recommended that dental treatment be deferred for six months following a stroke because of the increased risk of recurrent strokes during this period. After six months, dental procedures may be provided with the use of vasoconstrictor-containing local anesthetics where required for adequate pain control. If the stroke patient has associated cardiovascular problems, the dosage of local anesthetic with vasoconstrictor should be minimized in accordance with the guidelines for their specific cardiovascular disease.\textsuperscript{21}

- Pulmonary Disease

The most common pulmonary diseases encountered in the dental office are asthma, tuberculosis, and chronic obstructive pulmonary disease,
which includes chronic bronchitis and emphysema. While the status of tuberculosis infection in a patient is of the utmost concern to dental practitioners, and the patient's infection must be under control before elective dentistry is done, it poses no implications with regard to the use of dental local anesthetics.

Asthma

Dental management of asthmatic patients is primarily aimed at prevention of an acute asthma attack. Knowing that stress may be a precipitating factor in asthma attacks, adherence to stress-reduction protocols is again essential and implies the judicious use of local anesthetics containing vasoconstrictors when the planned procedure requires extended depth and duration of anesthesia. However, caution has been recommended based upon Food and Drug Administration warnings that drugs containing sulfites can be a cause of allergic reactions in susceptible individuals. Studies suggest that sodium metabisulfite, which is used as an antioxidant agent in dental local anesthetic solutions containing vasoconstrictors to prevent the breakdown of the vasoconstrictor, may induce allergic, or extrinsic, asthma attacks.

Data on the incidence of this problem occurring is limited, and suspicion is that it is probably not a common reaction even in sulfite-sensitive patients since the amount of metabisulfite in dental anesthetics is quite small. Indications are that more than 96 percent of asthmatics are not sensitive to sulfites at all; and those who are sensitive are usually severe, steroid-dependent asthmatics. As Perusse and colleagues conclude, “We believe local anesthetic with vasoconstrictor can be used safely for nonsteroid-dependent asthma patients. However, until we know more about the sulfite sensitivity threshold, we recommend avoiding local anesthetic with vasoconstrictors in corticosteroid-dependent asthma patients on account of a higher risk of sulfite allergy and the possibility that an accidental intravascular injection might cause a severe and immediate asthmatic reaction in the sensitive patient.”

Chronic Obstructive Pulmonary Disease
The two most common forms of chronic obstructive pulmonary disease, characterized by chronic irreversible obstruction of ventilation of the lungs, are chronic bronchitis and emphysema. Patients with chronic obstructive pulmonary disease already have decreased respiratory function, making it mandatory that the dental practitioner take every precaution to avoid further respiratory depression. There are no contraindications to the use of therapeutic doses of local anesthetics in these patients. However, any patient with chronic obstructive pulmonary disease who also suffers from coronary heart disease and/or hypertension must be managed in accordance with the guidelines provided for those diseases.

Renal Disease

In general, drugs excreted by the kidney, such as dental local anesthetics, may not be metabolized and cleared from the bloodstream as quickly as normal in the presence of renal disease. Total anesthetic dosage may need to be reduced and the interval of time between subsequent injections may need to be extended. Though this is a consideration, it is not a factor in most dental procedures provided that the total local anesthetic dosage is kept to a safe minimum.\textsuperscript{10}

Hepatic Disease
For patients with known liver function impairment, drugs metabolized by the liver should be avoided if possible, or the dosage at least decreased. Since all of the amide local anesthetics are primarily metabolized in the liver, the presence of liver disease and the status of liver function are important to the dentist. A history of hepatitis infection is not uncommon in most dental office patient pools. In completely recovered patients, local anesthetics may be administered routinely. However, patients with chronic active hepatitis or with carrier status of the hepatitis antigen must be medically evaluated for impaired liver function. Local anesthetics may be used in these patients, but it is recommended that the dose be kept to a minimum.

In patients with more advanced cirrhotic disease, metabolism of local anesthetics may be significantly slowed, leading to increased plasma levels and greater risk of toxicity reactions. Total anesthetic dosage may need to be reduced and the interval of time between subsequent injections may need to be extended. In these cases, initial injection with rapid-onset anesthetics such as lidocaine or mepivacaine followed by injection with a long-acting anesthetic like etidocaine or bupivacaine may be the best protocol for limiting total anesthetic dosage while achieving adequate pain control duration.

Cimetidine (Tagamet) has been shown to significantly reduce the metabolic clearance of amide local anesthetics through the liver. However, the probability of cimetidine and therapeutic doses of local anesthetic interacting to produce a toxic level of local anesthetic in the bloodstream is unlikely and unreported. Other histamine H2 receptor antagonist drugs such as ranitidine (Zantac) or famotidine (Pepcid) do not share cimetidine's metabolic inhibition of liver enzymes.

❖ Pancreatic Disease

Diabetes
Patients with either Type I insulin-dependent diabetes mellitus or Type II noninsulin-dependent diabetes mellitus, can generally receive local anesthetics without special precautions if control of their disease is well-managed. Consultation with a patient’s physician, as well as frank discussion with the patient, can determine the current status and what, if any, precautions are needed. Stress-reduction protocols, including excellent pain control, are of paramount importance and use of local anesthetics with vasoconstrictors is recommended when appropriate as long as the dosage is kept to the minimum needed.

Special caution should be used for patients with Type I diabetes who are being treated with large doses of insulin. Some of these patients, so-called brittle diabetics, experience dramatic swings between hyperglycemia and hypoglycemia; and the use of vasoconstrictors should be minimized due to the potential for vasoconstrictor-enhanced hypoglycemia.

❖ Adrenal Disease

Adrenal Insufficiency

No alteration of local anesthetic use is required for patients with adrenal insufficiency. Of greatest concern for treatment of these patients is the maintenance of good anesthesia during the dental procedure and good postoperative pain control to reduce stress.

Pheochromocytoma

The cardinal symptom of this tumor of the adrenal medulla or of the sympathetic para-vertebral ganglia is hypertension due to the increased secretion of endogenous epinephrine from these tissues. These patients are also prone to cardiac dysrhythmias. Due to the risk of potentiating cardiovascular problems, the use of vasoconstrictor-containing local anesthetics is contraindicated in these patients. No elective dental treatment should be rendered until the disease is medically corrected.
Thyroid Disease

Hyperthyroidism

The use of epinephrine or other vasoconstrictors in local anesthetics should be avoided, or at least minimized to one to two cartridges, in the untreated or poorly controlled hyperthyroid patient. Hypertension and cardiac abnormalities, especially dysrhythmias, are common in the presence of excessive thyroid hormones. However, the well-managed or euthyroid patient presents no problem and may be given normal concentrations of vasoconstrictors.

Hypothyroidism

In general, the patient with mild symptoms of untreated hypothyroidism is not in danger when receiving dental treatment. However, patients with mild to severe hypothyroidism may have exaggerated responses to local anesthetics due to the central nervous system depressant effects. Dosage should be kept to a minimum in mild hypothyroid patients, and dental treatment is best deferred in severe hypothyroidism until the patient’s condition can be corrected by his or her physician.

Musculoskeletal Diseases

Malignant Hyperthermia
This rare, but potentially fatal, muscle disease was at one time believed to be induced by administration of amide local anesthetics. However, leading authorities, including the Malignant Hyperthermia Association of the United States, do not advise any special precautions for the use of amide anesthetics in patients susceptible to malignant hyperthermia.

Blood Dyscrasias

Sickle Cell Anemia

Profound anesthesia as part of a proper stress reduction protocol is essential in management of these patients. The use of vasoconstrictor-containing local anesthetics is considered safe as long as the dosage is limited to one to two cartridges.30

Methemoglobinemia

Methemoglobin is hemoglobin that has been oxidized and can no longer bind and transport oxygen. While present in everyone, it normally makes up less than 1 percent of the circulating red blood cells. Increases in methemoglobin levels can be induced by administration of local anesthetic solutions, particularly prilocaine (Citanest), usually when in combination with other medications that also increase the methemoglobin level.20,31 Examples of common medications that may produce this interaction are Cipro, Bactrim, Septra, Dapsone, Macrodantin, Macrobid, Isordil, Nardil, and nitroglycerin.32 Patients with methemoglobinemia or taking medications associated with this disease may be safely treated with local anesthetic injections, with or without vasoconstrictors; however, the dosage should be minimized and the use of prilocaine avoided.
Drug Interactions

Antipsychotic Drugs (Phenothiazines)

There are no contraindications for use of any local anesthetics, with or without vasoconstrictors, in patients taking lithium for bipolar disease. For bipolar patients taking a phenothiazine type of drug such as chlorpromazine (Thorazine) or risperidone (Risperdal), fluctuations in blood pressure are common. Local anesthetics with vasoconstrictors used in normal amounts will not usually produce adverse effects. However, consultation with the patient's physician is recommended before dental treatment, and the patient should be carefully monitored for possible hypotensive episodes during the appointment.

Cocaine

The main concern in patients abusing cocaine is the significant danger of myocardial ischemia, cardiac dysrhythmias, and hypertension. Patients high on cocaine should not be treated in the dental office for a minimum of six hours following the last administration of cocaine, although the longer the time since the last use of the drug the better, with some researchers recommending deferral of dental treatment for 24 to 72 hours.

Tricyclic Antidepressants

Although use of tricyclic antidepressant drugs such as imipramine (Tofranil) and amitriptyline (Elavil) is decreasing, they are still prescribed for significant numbers of patients. One or two cartridges of epinephrine-containing local anesthetic can be safely used in patients taking these drugs; however, these patients should be carefully observed at all times for signs of hypertension due to enhanced sympathomimetic effects. Levonordefrin-containing local anesthetics are not recommended due to a greater tendency toward hypertension producing receptor potentiation than is seen with epinephrine.
Monoamine Oxidase Inhibitors

Dentists have long been cautioned about potential interactions of drugs of this class, for example the antidepressant phenelzine (Nardil), the Parkinson's disease drug selegiline (Eldapryl), and the antimicrobial furazolidone (Furoxone), relative to vasoconstrictor-containing local anesthetics. These cautions were based upon a fear of induction of severe hypertension due to interaction of vasoconstrictor-containing anesthetics with the MAO inhibitors. However, both animal and human studies have failed to yield evidence of such an interaction. Vasoconstrictor-containing local anesthetics may be used without special precautions in patients taking MAO inhibitor drugs.

Antianxiety Drugs

Diazepam (Valium), one of the most widely prescribed drugs in the United States, is a potent central nervous system depressant. Dosage of all local anesthetic agents should be kept to the minimum necessary for good pain control in patients taking benzodiazepine antianxiety drugs due to their additive depressive effects.
Summary

Local anesthetics, with or without vasoconstrictors, may be safely used in most medically complex patients. Observance of the following simple safety guidelines should be universal for administration of local anesthetics to all patients:

- Aspirate carefully before injecting to reduce the risk of unintentional intravascular injection;

- Inject slowly—a maximum rate of one minute per carpule is widely recommended [10,29]—and monitor the patient both during and after the injection for unusual reactions;

- Select the anesthetic agent and whether to use it with or without a vasoconstrictor based upon the duration of anesthesia appropriate for the planned procedure; and

- Use the minimum amount of anesthetic solution that is needed to achieve an adequate level of anesthesia to keep the patient comfortable throughout the dental procedure.

Adherence to these simple guidelines will reduce the risk of adverse reactions to the local anesthetic agents themselves or to the vasoconstrictors contained in local anesthetics. A further safety guideline useful for the majority of medically complex patients is to reduce the amount of local anesthetic containing a vasoconstrictor to no more than two 1.8 ml cartridges. If additional anesthetic volume is needed to maintain adequate pain control for the procedure, nonvasoconstrictor anesthetics can be used for subsequent injections. However, the use of additional cartridges of vasoconstrictor-containing local anesthetics is not an absolute
contraindication in patients who show no sensitivity to vasoconstrictor agents in local anesthetics. Table 1 summarizes the use of local anesthetic agents for many diseases and drug situations encountered in medically complex dental patients.

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References


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Chapter 2.
Managing the Patient with Severe Respiratory Problems

by Mitchell B. Day, DDS

Abstract: The dental management of patients with severe respiratory problems continues to be a significant challenge to the dental health care practitioners. Chronic obstructive pulmonary diseases, such as chronic bronchitis and emphysema, are the fourth leading cause of death in the United States. Asthma has increased in prevalence during the past 20 years, and the rate of death from this chronic inflammatory disease of the airways has also risen despite recent advances in medical treatments. This article will review the pathophysiology and medical treatment modalities for these chronic pulmonary diseases, as well as discuss the recognition and management of dental patients with these diseases and provide an understanding on how to avoid precipitating factors that could initiate an acute episode in the dental care setting.

The dentist in practice today must be prepared to provide care for patients with complex medical conditions. As people live longer, and with advances in medical care, dentists will be treating more medically compromised individuals. Consequently, dentists now find themselves increasingly committed to understanding dental patients’ overall medical diagnosis and therapy. Because dentists operate at the origin of the upper airway, and many dental procedures are deemed stressful, patients with chronic respiratory diseases are at special risk. Routine dental care can be provided in the dental
office when the dentist is knowledgeable about pulmonary diseases and pays specific attention to risk assessment and those precautions that are necessary to prevent acute exacerbations of a respiratory disease state. The entire dental team should be familiar with the signs, symptoms, and management of an emergent episode associated with asthma or chronic obstructive pulmonary diseases.

Asthma is a chronic inflammatory disease of the airways characterized by nonspecific hypersensitivity to a variety of stimuli that can precipitate acute episodes of bronchospasm and mucous secretion that result in airway obstruction. It is estimated that more than 14 million people in the United States have asthma, with as many as 4.8 million children being affected. The prevalence of asthma has increased in the United States since 1960, and the mortality rate has risen significantly throughout the 1980s and 1990s. In California from 1983 to 1996, there was a 30 percent increase in the number of hospital admissions for patients with a diagnosis of asthma.

Individuals with asthma experience acute episodes of tracheobronchial irritation that present with coughing, wheezing, and dyspnea, the classic clinical triad of the disease. Although asthma was once thought to be isolated acute exacerbations of bronchospasm, medical research has now clearly defined the role of inflammation in its pathophysiology. Contemporary medical management now emphasizes patient education and compliance in the use of long-term control medications that have anti-inflammatory effects on the airways, such as corticosteroids, Nedocromil, Cromolyn sodium, and the
leukotriene inhibiting agents. During acute exacerbations or "attacks," fast-acting or "quick-relief" medications, inhaled beta_2 agonists, and anticholinergics are used to reverse the airway obstruction resulting from bronchial smooth muscle contraction, epithelial edema, and mucous secretion.

The etiology of asthma is not clearly defined. Given the varied expression of the disease, it appears to be multifactorial in origin. The traditional classification of asthma describes two basic types: extrinsic and intrinsic.

Extrinsic or allergic asthma is associated with an allergic stimulus that results in the activation of airway epithelial mast cells. The immunoglobin E (IgE)-dependent process is initiated when the individual is exposed to an environmental allergen such as dust, pollen, tobacco, wood smoke, molds, house mites, or animal dander. The mast cells release the inflammatory mediators (i.e., histamine), which promote an immediate bronchospasm often referred to as the "early phase" reaction. With the continued action of these mediators, eosinophils and neutrophils migrate into the airways and a "late phase" reaction results in tissue injury leading to airway obstruction through bronchial smooth muscle contraction, epithelial cell shedding, mucous secretion, plasma extravasation, and airway edema.8,9

The extrinsic asthmatic patient presents with a known allergic history that has its onset in childhood or early teens. Skin tests to allergies are positive, and blood tests often reveal elevated IgE levels. Extrinsic asthmatic attacks tend to be intermittent and exhibit seasonal variation.

In contrast, intrinsic or nonallergic asthma is different in that no allergic stimulus is identified. Skin testing is negative and elevated IgE levels are not seen in this form. The onset of intrinsic asthma is usually seen in adults, and the acute exacerbations tend to be continuous. Endogenous factors, including emotional stress, or other idiopathic stimuli are thought to initiate the attacks.10

It should be noted that very few patients who are diagnosed with asthma exhibit the features of a purely extrinsic or intrinsic form
and most will have complex or varied presentation. Furthermore, other forms of asthma have been described, and include drug-induced asthma associated with the intake of aspirin, NSAIDs, angiotension-converting enzyme inhibitor, and metabisulfite preservatives. Exercise-induced asthma is another form that is seen in adolescents and young adults and attributed to vigorous physical activity.

The National Asthma Education and Prevention Program has developed a revised classification for asthma. Individuals with chronic asthma are classified based on the severity of their symptoms, when the symptoms occur, and how often they occur. In addition, the assessment of lung function is vital to the classification. Patients are assigned as mild intermittent, mild persistent, moderate persistent, or severe persistent. This newer classification has resulted in a stepped approach to diagnosis and medical management that addresses the causal inflammatory processes with the goal of preventing or limiting the acute symptoms of the disease.

At initial presentation, patients with asthma give a history of recurrent coughing, wheezing, difficulty breathing, and chest tightness. Based on the severity and frequency of these symptoms, pulmonary function testing further delineates the degree of airway obstruction. Spirometry is used to quantify the degree of disease with forced expiratory volume in one second (FEV₁). In addition, peak expiratory flow (PEF) can be followed daily by patient-administered spirometry in a moderate to severe diagnosis as a method of monitoring the disease and evaluating the response to medication therapies.

Individuals with mild intermittent asthma (experience symptoms twice a week or less) exhibit relatively normal PEF values between attacks and have nocturnal symptoms less than two times per month. These patients rarely require daily medications for long-term control. Behavior modification to avoid factors that stimulate acute exacerbations and use of fast-acting beta-adrenergic bronchodilators define the first step approach in these patients.
If symptoms occur more than two times per week, nocturnal episodes occur more than twice a month, and pulmonary function values show a decreased ratio of FEV₁ to forced vital capacity (FVC) with variability in PEF of 20 percent to 30 percent, then these patients are classified as having mild persistent asthma. Initial therapy and additional stepwise therapies may be more complex for these individuals. Long-acting anti-inflammatory drugs are used daily and may include low-dose inhaled corticosteroids, the mast cell stabilizers Cromolyn sodium or Nedocromil, and the newer leukotriene antagonist drugs such as Zafirlukast and Zileuton. 12-14

Patients with a diagnosis of moderate persistent asthma exhibit symptoms daily, use fast-acting beta-adrenergic bronchodilators daily, and experience nocturnal symptoms more than once a week. These patients have FEV₁ to FVC ratios that are less than 80 percent and the variability in PEF can be greater than 30 percent. Treatment with multiple therapeutic agents may be required to establish long-term control of their symptoms. In addition, medium- to high-dose inhaled corticosteroids are often indicated. Many of these patients may be placed on the long-acting beta-selective agonist bronchodilator, Salmeterol, for prolonged maintenance. 15-16 In addition, albuterol’s beta agonist properties can be used as a long-acting agent when given orally as an extended release tablet and are useful in long-term control of nocturnal symptoms. The diagnosis of moderate persistent asthma requires that the patient also take an active role in the treatment and monitoring of his or her disease state. Long-term control of this disease can be greatly enhanced by educating the patient on the complex medication regimens, the value of self-monitoring with spirometry, and the proper technique in using inhalers and nebulizers, as well as avoiding exposure to potential stimulating factors. 2, 17

Patients with severe persistent asthma can experience daily symptoms and acute exacerbations, which can, in turn, limit their physical activities. Nocturnal symptoms are common. Lung function is highly variable with the FEV₁ to FVC ratio at 60 percent or lower and variability in PEF at more than 30 percent. The multiple medication regimens used to treat moderate persistent asthma may prove to be inadequate. A step-up in therapy is now indicated for severe persistent asthma with higher dose inhaled corticosteroids and long-acting beta agonists serving as the preferred pharmacotherapy. When severe asthma exacerbations continue, systemic corticosteroids
are used as a short-term therapy to help alleviate the severity of the exacerbations despite the potential significant systemic side effects of oral administration. In addition, sustained-release theophylline is considered in the management of the severe persistent asthmatic not responding to the other drug modalities. Once a mainstay in asthma therapy, theophylline is now a secondary or tertiary agent mainly used to treat the nocturnal symptoms because of concerns for the drug’s narrow therapeutic range requiring frequent serum level monitoring, potential drug interactions and adverse effects on multiple organ systems.

Given the varied expression of asthma, long-term successful management of the disease is achieved through very individualized stepwise drug therapy and an emphasis on patient education and compliance. All patients with asthma can experience mild, moderate, or severe exacerbations, which can evolve to a life-threatening episode. The stepwise approach to diagnosis and therapy has greatly decreased the severity and frequency of asthma symptoms in many patients. While quick-relief, fast-acting inhaled bronchodilator drugs treat the severe symptoms of bronchial airway obstruction in the acute asthmatic attack, long-term control medications offer the greatest potential to alleviate asthma symptoms, improve pulmonary function, and diminish the disease’s overall morbidity. Table 1 provides both a classification and treatment protocol for the medical management of asthma. This format can assist the dentist in the development of a patient’s risk assessment prior to initiating dental treatment.

The primary objective for the dentist in the management of a patient with a medical condition is to prevent any complications related to that condition as a result of dental treatment. The asthmatic patient can be treated for his or her dental needs when the dental health practitioner has developed a risk assessment that is individualized for that patient. This assessment begins with an appropriate understanding of the patient’s medical history. The health history questionnaire and a comprehensive interview by the dentist is the foundation of the risk assessment process.
For the asthmatic patient, the dentist should determine the following aspects of that patient’s disease history:

- Classification or type of asthma;
- Current medication regimens;
- Patient’s understanding and compliance with medical treatment;
- Factors that precipitate acute exacerbations;
- Frequency and timing of episodes;
- How often the fast-acting or quick-relief bronchodilators are used; and
- History of emergency room visits or hospitalizations.

This information is then used to determine the stability and severity of the disease, provide an indication for a medical consultation prior to dental treatment, and guide the practitioner in the development of an appropriate management protocol. The patient’s understanding of his or her disease and compliance with the prescribed medical therapies is of vital importance. The asthmatic patients who are most likely to experience frequent acute exacerbations and present a higher risk of having a complication associated with dental treatment are those who are not compliant with their complex drug regimens and have a poor perception of the diagnosis of asthma. These patients and patients with a diagnosis of severe asthma should have a medical consultation prior to any extensive or stressful dental treatments.

Given the complex expression of asthma, management protocols for asthmatic patients should be tailored to their individual needs based on the dentist’s risk assessment. With stress being a primary precipitating factor for the stimulation of an acute asthmatic attack, a stress-free environment is essential in treating all asthmatic dental patients. The anxious patient may require sedation for dental procedures. The use of nitrous oxide-oxygen inhalation sedation should be considered and
can be combined with short-acting oral benzodiazepines. Nitrous oxide is not irritating to the airway, does not cause a depression of respiration, and may have an analgesic effect to supplement the use of local anesthesia for pain control. The time of day and the length of dental treatment visits should be adjusted to prevent a stress-inducing situation. For those patients with moderate to severe persistent asthma, it is appropriate to have them prophylactically administer their own fast-acting bronchodilator medication preoperatively to their treatment appointment and ensure they have used their inhaled corticosteroid medications as scheduled.

The preoperative management of the asthmatic patient centers on avoiding factors that can stimulate an exacerbation of acute symptoms leading to bronchospasm. In dental treatment, the potential of drug interactions is of primary consideration. Aspirin and nonsteroidal antiinflammatory drugs should be avoided in the management for postoperative pain, as they are known to stimulate asthmatic attacks. Relative contradictions for both narcotics and barbiturates have been identified for asthmatic patients. Drugs from both groups can increase the risk of bronchospasm and should not be used.

Furthermore, the use of certain antibiotics to treat orofacial infection is contraindicated in moderate to severe asthmatics who are taking theophylline. Ciprofloxacin and the macrolides (i.e., erythromycin, clarithromycin and azithromycin) alter the metabolism of theophylline, which can result in toxic serum levels of this drug.

The selection of a local anesthetic is important when treating asthmatics. Many local anesthetic solutions contain sulfites as a preservative. Sulfites are known to precipitate acute asthmatic attacks and allergic reactions. These compounds are found in local anesthetic preparations containing epinephrine and levonordefin, and these preparations should not be used in patients known to be sensitive to sulfites.

1) Mild Intermittent Asthma

Fast-acting bronchodilators –for quick-relief acute episodes
A. Non-selective beta agonist
- Epinephrine (Primatene Mist)
- Ephedrine (Eted II)

B. Selective beta 2 agonist
- Albuterol (Ventolin, Proventil)
- Terbutaline (Brethaire)
- Tirbuterol (Maxair)
- Bitolterol (Tornalate)

2) Mild Persistent Asthma

Fast-acting bronchodilators – for quick-relief acute episodes

Long-term control medication – anti-inflammatory

Usually one medication daily

A. Low-dose inhaled corticosteroids
- Triamcinolone (Azmacort)
- Fluticasone (Flonase)
- Flunisolide (Aerobid)
- Budesonide (Plumicort)
- Beclomethasone (Beclovent)

B. Inhaled nonsteroidal anti-inflammatory
- Cromolyn sodium (Intal)
- Nedocromil (Tilade)

C. Leukotriene-inhibiting drugs
- Zafirlukast (Accolate)
- Zileuton (Zyflo)
3) Moderate Persistent Asthma

Fast-acting bronchodilator – for quick-relief acute episodes

Long-term control medication – anti-inflammatory

One to two medications daily

A. Medium-dose inhaled corticosteroids
B. Inhaled nonsteroidal anti-inflammatory
C. Leukotriene-inhibiting drugs
D. Long-acting bronchodilators
   • Isoproterenol (Isuprel)
   • Metaproterenol (Alupent)
   • Salmeterol (Serevent)
   • Albuterol (Oral Tablets)

4) Severe Persistent Asthma

Fast-acting bronchodilator – for quick relief acute episodes

Long-term control medications – anti-inflammatory

Multiple medications daily

A. High-dose inhaled corticosteroids
B. Long-acting bronchodilator
C. Methylxanthines
   • Sustained-release theophylline
D. Oral Corticosteroids
   • Prednisone (Deltasone)
- Prenisolone (Delta-Cortef)
- Methylprednisolone (Soll-Medrol)

The onset of symptoms associated with an acute asthmatic attack can vary considerably in asthmatic patients. With stress being a major factor in stimulating asthmatic bronchospasm, the practitioner should be alert to increasing anxiety or apprehension in patients. Most asthmatic episodes are accompanied with the onset of a cough, the patient complaining of a feeling of chest tightness, dyspnea, and wheezing. Once an acute episode is diagnosed, the following maneuvers are indicated to reverse the underlying bronchospasm:

- Discontinue dental treatment;
- Place the patient in an upright sitting position;
- Assist the patient in the administration of fast-acting bronchodilator, or administer albuterol (Ventolin/Proventil) from an emergency kit metered-dose inhaler;
- Administer oxygen using a nasal cannula, nasal hood or full-face mask at 2-3 L/minute; and
- Reassure the patient and act calmly.

If the episode resolves, consider the need for a medical consultation and reevaluate the office stress-reduction protocol, the length and time of the dental appointments, and the need for sedation with further appointments.

If the attack continues after initial maneuvers, a serious medical emergency is indicated:

- Call 911 or contact the community emergency medical response number;
Administer epinephrine by injection sublingual, intramuscular, or subcutaneous. A 1:1000 concentration is used, give 0.3 to 0.5 ml; repeat doses can be given at 20 minute intervals.

- Assist airway, breathing, and circulation as needed;
- Continue oxygen; and
- Monitor vital signs.

The patient will require transfer to a medical facility by trained emergency medical personnel for treatment with intravenous corticosteroids, airway management, and evaluation of the medical treatment for the asthma.

The American Thoracic Society has defined chronic obstructive pulmonary disease as a disease state characterized by the presence of airflow obstruction due to chronic bronchitis or emphysema; the airflow obstruction is generally progressive, may be accompanied by airway hyperactivity, and may be partially reversible. It is estimated that 12 to 14 million people in the United States suffer from chronic obstructive pulmonary disease, and the disease is currently the fourth leading cause of death. While deaths due to cardiovascular disease are on the decline, death from chronic obstructive pulmonary disease has increased by more than 40 percent since the early 1980s.
Two very different conditions are included in the definition of chronic obstructive pulmonary disease: chronic bronchitis and emphysema. These two conditions have their own distinctive set of symptoms and underlying pathophysiology. Chronic bronchitis is described in clinical terms as the presence of a cough with the production of sputum for at least three months for a minimum of two years in succession. Emphysema is defined in pathological terms as the abnormal, nonreversible enlargement of the air spaces distal to the terminal bronchioles associated with alveolar wall destruction. It should be noted, however, that very few patients with chronic obstructive pulmonary disease exhibit symptoms exclusive of chronic bronchitis or emphysema. In fact, many patients share symptoms attributed to both conditions. 24

Both conditions share a common etiology, with exposure to tobacco smoke being the primary cause of chronic obstructive pulmonary disease. However, for reasons that are unclear, only about 15 percent of cigarette smokers actually develop clinically significant chronic obstructive pulmonary disease, while tobacco smoking is known to account for 80 percent of the risk of acquiring the disease. 28,29 The incidence of chronic bronchitis in cigarette smokers increases with age and the amount of cigarettes consumed, indicating that the risk is dose- and duration-dependent. Passive smoking or "second-hand smoking" may also contribute to the development of the disease. Children of smokers are known to have a higher incidence of respiratory symptoms, and respiratory function is decreased when measured through pulmonary function testing. 24

Other identifiable risk factors include environmental pollutants such as ambient air pollution, indoor irritants, and occupational airborne hazards. Based on epidemiological evidence, a nonspecific hyperresponsive airway condition has been identified as another possible risk factor leading to chronic obstructive airflow disease; but the specific causes of this condition have not been well-defined.

A genetic disorder of α1-antitrypsin deficiency is known to cause chronic obstructive pulmonary disease and accounts for
less than 1 percent of those individuals diagnosed with chronic obstructive pulmonary disease.

α₁-antitrypsin is produced in the liver and acts in the lungs to inhibit the action of neutrophil elastase, which leads to the enzymatic breakdown of the lungs’ elastin connective tissue. 31 Multiple autosomal co-dominant alleles at a single locus contribute to the inheritance of α₁-antitrypsin, which in turn results in a varied expression of this genetic abnormality. When a severe deficiency of α₁-antitrypsin is present, nonsmokers can develop bronchiectasis, chronic bronchitis, and basilar emphysema. The onset of airflow obstruction can develop before the age 50, and individuals with this condition are at extreme risk of acquiring clinically significant chronic obstructive pulmonary disease.

The anatomical and physiological changes that result in the clinically significant chronic airflow obstruction condition occur over a long period of time with exposure to the predisposing causative agent. Expiratory flow values are used to diagnose and establish the severity of the disease. Cigarette smokers with obstructive disease will show a progressive decline in the FEV₁ at a rate two to three times faster than nonsmokers. With smoking cessation in individuals with mild to moderate airflow obstruction, the rate of deterioration can return to that seen in nonsmokers, and their symptoms may decrease. 33

In patients with chronic bronchitis, the airflow obstruction is the result of sustained inflammation that proceeds with mucus cell metaplasia and increased mucus production, loss of respiratory epithelium, mucous, and fibrosis of the associated bronchiolar and alveolar walls. Early on in the disease process, there is a marked increase in the airflow resistance in the peripheral airways. Over time with progressive deterioration, the obstruction extends to the bronchiolar walls. Acute exacerbations of this condition have been associated with respiratory tract infections. *Streptococcus pneumoniae*, *Haemophilus influenzae*, and *Moraxella catarrhalis* have been isolated in the lower bronchi of patients with chronic bronchitis. The exact role of infection in the long-term progression of the disease process is poorly understood. 34
Emphysema develops when there is an irreversible enlargement of the bronchioles and the alveoli. Distal to the terminal bronchiole, there is a concomitant breakdown in the alveolar ducts and the alveolar sacs, which will extend to the collapse of the terminal bronchiole. This leads to an airflow obstruction that hinders expiration. The breakdown of the distal air spaces occurs as a result of a protease-antiprotease imbalance, which leads to a degradation in the elastin in the alveolar walls. It is proposed that cigarette smoke can activate macrophages in the lungs to release factors that directly stimulate neutrophils to secrete elastases and combine with oxidants and free radicals, which in turn oxidize α1-antitrypsin. The combined effect on the lung parenchyma is a destructive process that is characterized by the degradation of elastin and an inhibition of new elastin synthesis.

Several different types of emphysema have been described based on the anatomical areas within the lung that are affected. Centriacinar (centrilobular) emphysema is most often seen in the chronic cigarette smoker and is associated with the respiratory bronchioles, first with airway enlargement and destruction and then advancing distally. Panacinar (panlobular) emphysema is a uniform enlargement and breakdown of the air spaces throughout the acinus and is observed in patients with severe α1-antitrypsin deficiency.

The airflow obstruction in both chronic bronchitis and emphysema develops over a long period with exposure to cigarette smoke. The anatomical changes are irreversible once a moderate amount of disease progression is established.

Individuals with chronic obstructive pulmonary disease usually present after the fifth decade of life with increasing complaints of chronic cough with sputum production or acute chest illness, which includes dyspnea, or shortness of breath on exercise. The individual with advanced chronic bronchitis is often described as a "blue bloater," presenting with a chronic productive cough. The patient's lung sounds are wet, characterized by rates and rhonchi. The patient reports a history of frequent chest infections. Findings of wheezing and dyspnea in this patient could be erroneously diagnosed as asthma. A
chest radiograph appears normal. Arterial blood gas studies reveal a marked reduction in PaO\(_2\) (hypoxia) and increase PaCO\(_2\) (cyanosis). The patient also shows signs of peripheral edema associated with right heart failure, (cor pulmonale) and may have marked pulmonary hypertension.

The patient with emphysema is described as a "pink puffer," presenting with dyspnea and minimal or no cough. Typically, the patient is thin with a "barrel chest" appearance and quiet chest sounds on auscultation. Hyperinflated lung fields and an increase in the anterior to posterior dimensions of the patient's chest are noted. The chest radiograph shows hyperinflation, a "small" heart and a flattened diaphragm. Arterial blood gas studies reveal normal to slightly reduced PaO\(_2\) and PaCO\(_2\).

Both chronic bronchitis and emphysema show marked decrease in the FEV\(_1\)/FVC ratio. When lung volumes are measured, there is an increase in the residual volume (RV), and a normal or increased functional residual capacity (FRC) is observed. Pulmonary function tests are essential in determining the severity of the disease state, with FEV\(_1\) serving to quantify the degree of airflow impairment. When correlated with factors such as age and the assessment of the levels of hypoxia and hypercapnia through arterial blood gas analysis, this information is used to individualize the diagnosis, prognosis, and treatment regimen appropriate for the patient with chronic obstructive pulmonary disease.

Because chronic obstructive pulmonary disease cannot be cured, medical treatment is directed at reducing the degenerative effects of the disease and managing the acute and chronic symptoms of chronic bronchitis or emphysema. Smoking cessation is the single most important therapy for patients with obstructive airway disease and proves to be the greatest challenge for the patient and the physician in managing the disease. 36

The reasons that people smoke cigarettes are multifactorial and include issues of nicotine addiction, education, income, conditioned psychological responses, and mental health. Clinician intervention, community support, and pharmacological treatments combined offer the best hope in achieving smoking abstinence in patients, and even still long-term success rates are often less than one-third of the patients that enroll in multifaceted programs. 37 Exposure to other potential inhaled
irritants such as air pollution, home and personal care aerosolized products, and industrial or workplace irritants can cause acute exacerbations in patients and should be avoided.

Treatment of chronic bronchitis is directed at reversing or preventing airflow obstructions caused by inflammation, infection, and mucus secretion. Immunization with pneumococcal and influenza vaccines is an important component in the long-term management of the disease. The use of antibiotics is reserved for acute exacerbations and directed at known pathogens that can cause a superimposed acute bacterial tracheobronchitis. As with the treatment of asthma, the pharmacological treatment of chronic bronchitis and emphysema is managed in an individualized stepwise manner correlated to the severity of the disease. To ensure the patient’s participation in successful long-term management of the disease, it is essential to educate the patient about the diagnosis, disease severity, and indications for the medications used in treating the disease.

Bronchodilators are used in the treatment of both chronic bronchitis and emphysema. The beta₂ agonists can contribute to the relief of symptoms but do not contribute the same type of sustained bronchodilation with improved pulmonary function that can be seen in asthmatics.

In older chronic obstructive pulmonary disease patients, these drugs may also have sympathomimetic side effects that complicate concomitant cardiovascular diseases. Anticholinergic inhaled agents offer a slower onset, longer action, and fewer side effects. The atropine derivative, ipratropium bromide, is available as a metered-dose aerosol and has shown to be effective in relieving symptoms resulting from chronic airway obstruction.

When the symptoms associated with chronic obstructive pulmonary disease are continuous or more severe and their management with the bronchodilators is suboptimal, the use of sustained-release theophylline is considered. Theophylline has been shown to improve respiratory muscle function, stimulate the respiratory center, and improve mucociliary clearance. Patients with co-existing cardiac disease, cor pulmonale, and pulmonary hypertension may benefit from the improved cardiac output, reduced pulmonary vascular resistance, and improved myocardium perfusion that theophylline can produce.
The use of corticosteroids has limited applications in the treatment of chronic obstructive pulmonary disease given that the disease process is primarily one of tissue degeneration and destruction with little or no reversible component.

For patients with chronic bronchitis, the inhaled corticosteroids may have an application in treating airway inflammation. Because of the potential long-term effects of high-dose oral corticosteroids, their use is limited to selected patients that show improvement on treatment of acute exacerbations that respond to short-course therapy.
I. Mild to Moderate Disease

A. Smoking cessation—physician intervention
   • Behavior modification program
   • Nicotine replacement therapy
   • Transdermal patch and oral medications
   • Support groups

B. Eliminate environmental exposure/causes
   • Air pollution
   • Industrial or workplace irritants
   • Household sources

C. Physical conditioning (improving functional capacity)
   • Exercise training to improve respiratory function and exercise tolerance
   • Nutrition and hydration

D. Immunizations: influenza and pneumococcal

E. Antibiotic therapy for acute infections/ exacerbations of chronic bronchitis attributed to known pulmonary pathogens

F. Treat symptoms of bronchospasm
   • Anticholinergic aerosol agents—ipratropium bromide (Atrovent)
   • Sympathomimetic agents—beta₂ agonist meter-dosed inhalers
II. Moderate Disease

A through F

When symptoms continue and improved pulmonary function is suboptimal

G. Sustained-release theophylline

H. Systemic or inhaled corticosteroids

• High-dose prednisone short course

• Lowest dose prednisone in long-term management

• Prolonged systemic steroid therapy is avoided

III. Severe Disease

A through H

With evidence of progressive hypoxia and hypercarbia

I. Supplemental oxygen therapy, minimum 18 hours daily with $\text{PaO}_2 \leq 55\text{mm Hg}$ or $\text{SaO}_2 \leq 89$

J. Note therapy for congestive heart failure, cor pulmonale and polycythemia

Patients with co-existing heart disease such as cor pulmonale and right heart failure will often be placed on diuretics or arterial vasodilators to treat pulmonary hypertension that develops with severe chronic obstructive disease. Their overall medical management is complex, and these patients will require constant observation by the treating physician. As the effects of the disease become more severe, monitoring and treating progressive hypoxemia and pulmonary hypertension will require long-term supplemental oxygen administration. The use of oxygen therapy has been shown to enhance survival rates and greatly improve the quality of life for individuals with severe chronic obstructive pulmonary disease. The supplemental administration of oxygen for a minimum of 18 hours/day at a rate of 2L/minute via nasal cannula maintains oxygen saturation levels of greater than 90 percent. When patients present with a
PaO$_2$ of less than or equal to 55 mm Hg or a SaO$_2$ of less than 88 percent, this is considered an absolute indication for oxygen therapy. Long-term oxygen administration can improve cardiac function, reverse polycythemia, and reduce the symptoms associated with cor pulmonale.

Pulmonary rehabilitation is a multifaceted effort directed at educating the patient, improving physical conditioning and nutrition, and supporting the psychological needs of patients with chronic obstructive pulmonary disease. Table 2 summarizes the stepwise treatment protocol for the medical management of chronic obstructive pulmonary disease patients.

Patients with moderate to severe chronic obstructive pulmonary disease seeking dental care require a risk assessment that identifies the type of disease, establishes the severity of the condition, and documents the success or compliance of the patient’s medical treatment. In addition, other medical conditions, which may affect the chronic airway obstructive disease, should be identified and evaluated. While the majority of patients with significant symptoms of productive cough or shortness of breath will have sought medical evaluation and treatment, the dental health practitioner must be alert to patients with untreated or undiagnosed symptoms of chronic bronchitis or emphysema. The health history questionnaire and the dentist-patient dialogue can alert the practitioner to possible concerns for respiratory disease. The physical exam, through observation and auscultation of the patient, can reveal signs of chronic obstructive pulmonary disease.

When a concern for a patient’s respiratory status is noted and an understanding of the functional airway reserve is desired, a simple "breath holding" test can be performed. For example, with the patient sitting upright, have him or her take as deep an inspiratory breath as possible while holding the nostrils closed. Ask him or her to hold his breath as long as possible. Note the length of time before he or she must exhale and desire another breath. People that can only hold their breath for 10 to 20 seconds may have a moderate degree of pulmonary compromise, while those that cannot go longer than 10 seconds may have severe obstructive airway disease. Patients with
moderate to severe airflow obstruction should have a medical consultation prior to any extensive or stressful dental treatment.

The reduction of stress and avoidance of any procedures that may depress a patient’s respiratory function are essential in the management of patients with moderate to severe chronic obstructive pulmonary disease.

Patients should be offered a professional and reassuring environment with short, focused dental treatments early in the day. If appropriate, sedation can be considered, but potent sedatives, barbiturates, or narcotics should be avoided as they can depress the respiratory drive. Nitrous oxide and high flow rates of oxygen are contraindicated because their use can result in respiratory depression in patients with severe disease exhibiting CO₂ retention. Placing a patient in a reclined position or the use of a rubber dam can contribute to a sense of respiratory compromise. Low-flow supplemental oxygen administration via nasal cannula at rates of 2 to 4 L/minute is appropriate even in patients with severe disease.

The use of certain drugs in the dental-related treatment of chronic obstructive pulmonary disease patients should be avoided. Anticholinergic or antihistamines can alter tracheobronchial secretion, which may promote an acute respiratory infection or stimulate inflammation or irritation to the airways leading to further airflow disturbance. When antibiotic therapy is indicated for an odontogenic infection or prophylaxis, patients taking theophylline should not be given macrolides, ciprofloxin, or clindamycin, which can lead to methylxanthine toxicity.

A primary concern for patients with moderate to severe chronic obstructive airway disease is avoiding situations in dental treatment that will promote an acute episode of respiratory distress. When concomitant cardiovascular disease is present, these patients will also have a risk of myocardial infarct, heart failure, morbid arrhythmias, and acute pulmonary edema precipitated by respiratory depression. The early recognition
and treatment by the dental team of a patient with chronic obstructive pulmonary disease developing respiratory failure is paramount. Shortness of breath, wet airway sounds, expiratory wheezing, elevated blood pressure and heart rate, and increasing apprehension or agitation may all accompany a patient in respiratory distress. Once an acute episode is diagnosed and treatment has stopped, the following maneuvers are indicated:

- Place patient in an upright sitting position;
- Administer oxygen at a low-flow rate of 2-4 L/minute via nasal cannula or hood;
- Assess and assist airway, breathing and circulation;
- Monitor vital signs; and
- Summon emergency medical assistance.

The severity of the disease and the patient's medical management must be reassessed and warrants the transfer of the patient to a medical facility for further evaluation.

Dental care for patients with respiratory diseases continues to be an important aspect of the practice of contemporary dentistry. Routine dental care can be provided for patients with severe respiratory problems when the dentist is knowledgeable about pulmonary diseases and is familiar with the signs, symptoms, and management of an emergent episode associated with asthma or chronic obstructive pulmonary disease. Patients with severe respiratory problems can receive safe and appropriate care when the dental team has conducted a proper risk assessment and tailored the necessary dental treatment to each individual patient's needs and tolerance. Preparation is vital to the prevention of a medical emergency arising from dental treatment in patients that are compromised by serious health conditions.
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