Evaluation and Management of the Cardiac Patient for Office Oral Surgery

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In the dental profession we are often asked to provide care for a variety of medically compromised patients. One of the more frequent and challenging is the patient afflicted with organic heart disease, atherosclerotic coronary artery disease (ASCAD), or congenital heart disease.

Throughout the course of dental surgical management, a great deal of physiological and psychological stress can be directed toward these patients. Physiologically, this stress is translated into a release of circulating catecholamines manifesting in many body functional changes, all designed to prepare the patient for the imminent traumatic experience — flight or fright syndrome.

One of the organ systems which responds markedly to this stimulation is the cardiovascular system (elevations in heart rate, cardiac output, etc.). Consequently, a thorough evaluation and understanding of the patient’s cardiac status is vitally important in insuring the patient’s well-being during the dental visit.

Cardiac disease can be generally classified into three basic categories for our purposes:
1. Congenital heart defects
2. Congestive heart failure (CHF)
3. Coronary artery disease (CAD)

Congenital Heart Defects

The incidence of congenital heart defects is approximately 0.8% of all live births which represents 8 cases per 1,000 babies.1 About 50% of all defects present without cyanosis, 25% with cyanosis, and 25% with clinical signs of obstruction to blood flow that may or may not result in hypoxia and cyanosis.

The patient with acyanotic congenital defects may be symptom-free until middle or late adulthood. More than one-half of these patients have ventricular septal defects.

Shunting of blood through the septal defect can lead to hypoxemia if the blood is directed from the right side of the heart to the left. This results in blood bypassing the lungs without becoming oxygenated. Small defects may go unnoticed for many years and are usually physically well tolerated until the right heart develops hypertrophy and congestive failure. The septal defect may also shunt blood from the left ventricle to the right ventricle resulting in left heart hypertrophy, pulmonary hypertension, and subsequently left or right heart failure.

Valvular defects, primarily aortic and mitral stenosis, can lead to elevated pressures in the left ventricle and left atrium, respectively. Often the only symptom is gradually decreasing exercise tolerance over several years. As the valvular disease progresses, harsh murmurs develop indicating abnormal blood currents (i.e., Eddy waves). If sudden death due to a rupture of a valve leaflet or papillary muscle does not occur, the progression of valvular defects usually leads to congestive heart failure.

Congestive Heart Failure

This entity is defined as a functional state in which the heart is unable to meet the needs of the other organ systems, even as compensatory mechanisms are at work. The causes of heart failure can be grouped into: 1) those resulting in the need for increased stroke volume, cardiac output, or impedance to ejection, such as pulmonary or systemic hypertension; 2) those causing abnormalities in contractile function of the myocardium, such as in cardiomyopathy; and 3) those causing restriction in filling such as valvular obstruction, or ventricular/pericardial stiffness following myocardial infarction.2 A patient with congestive heart failure often has a variety of symptoms ranging from dyspnea at rest, wheezing or congested lungs in severe cases, to dyspnea only on mild exertion and a “chronic cigarette cough.” The term cardiac insufficiency is the most accurate description of the milder form of failure.

Most patients with a history of cardiac disease requiring prior hospitalization will be on multiple drugs. These usually include a diuretic (e.g., lasix), a cardiac glycoside (e.g., digoxin), and additional med-
lications to control hypertension or arrhythmias. Clinically, these patients may present with any combination of the following physical findings: shortness of breath, orthopnea in the dental chair (inability to breathe in the supine position), various cardiac arrhythmias, pedal edema, cool and pale skin, and moist bronchial rales. Radiographically, on chest x-ray, there may be an enlargement of the cardiac silhouette (Kerley-B-lines, and cephalization of vasculature). A review of the patient's present medications is very helpful, and may lend important information as to the progress of the disease. Every drug that a patient is presently taking should be recorded by name and dosage and updated on a regular basis at subsequent visits. Cardiac glycoside administration usually indicates moderate to serious myocardial disease. Diuretics including lasix and spironolactone drugs are given for mild to moderate hypertension, but may be used with digoxin for congestive heart failure. Dental patients should continue these medications the day of treatment. The pulse rate and rhythm are important to monitor preoperatively and during the surgical procedure.

**Coronary Artery Disease**

This is a general term describing the inability of the coronary circulation to meet the required oxygen demands of the myocardium. The clinical manifestations of the disease are in the form of a variety of complaints including exertional or non-exertional chest pain (angina pectoris) and/or tightness of varying intensity, duration, location, radiation, and quality, as well as associated symptoms such as nausea, vomiting and diaphoresis.

There are four types of angina pectoris associated with myocardial ischemia — stable, unstable, pre-infarction, and Prinzmetal angina. Approximately one-third of the patients with Prinzmetal angina (pain at rest) have no atherosclerotic arterial changes seen on angiography. This entity is supposedly caused by a coronary artery spasm and can occur at any time under any conditions, including non-stressful situations. Prinzmetal angina usually develops in the second and third decades of life and can be associated with sudden cardiac arrest. The greatest problem in the pre-surgical evaluation of what appears to be angina pectoris (AP) is its differentiation from other medical problems (Table 1).

The major difference between classical AP and GI disorders is that the former is usually brought on by a fixed amount of exertion and subsides with rest, a pattern which seldom fits the latter. Musculoskeletal pain is usually slow in onset, of a longer duration, and duller in character. Pressure or bodily movement over the area intensifies this type of pain, but it must be kept in mind that AP has a wide variety of clinical presentations and is especially difficult to evaluate in the poor historian. Stable angina can be described as chronic, occurring predictably with exertion and alleviated quickly with rest and/or no more than three .4 mg nitroglycerine tablets sublingually on any one occasion. There is usually some event, such as exertion, overeating, or emotional upheaval that triggers the onset.

Unstable angina occurs while at rest, is not predictable in terms of precipitating factors, is triggered with minimal effort and lasts progressively longer than the stable type. If either the stable, unstable, or Prinzmetal types of angina seem to last longer, are more intense accompanied by hypotension and cardiac arrhythmias, a pre-infarction angina should be suspected.

**TABLE 1**

**Non-Cardiac Causes of Chest Pain**

| 1. Psychogenic | —hyperventilation, neurocirculatory asthenia (anxiety) |
| 2. Aortic | —aneurysm |
| 3. Pulmonary | —embolism, infarction, pleurisy, pneumothorax, pneumomediastinum |
| 4. Musculoskeletal —Tietze's syndrome, costochondrodynia, thoracic outlet syndrome, myositis, trauma |
| 5. Gastrointestinal —esophageal reflux, esophagitis or spasm; gallbladder, peptic ulcer and pancreatic disease |

**Management**

The treatment of the cardiac patient with dental pathology begins well before the actual surgery is contemplated, with the establishment of good rapport, reassurance, and good planning. Following the acquisition of the medical history, the patient should be classified according to the severity of the organic heart disease. Four classifications extending from the most benign to borderline complete heart failure have been proposed by the New York Heart Association. Class I patients that do not require special medications — some clinical evidence of coronary artery or valvular disease disclosed by tests as sophisticated as angiography to organic murmurs heard by chest auscultation are usually managed without any special precautions (except for antibiotic prophylaxis for patients with valvular heart disease). Class IV patients with severe impairment of normal functional activities such as walking, household duties and require frequent rest periods should be treated in a hospital setting for even minor elective procedures. Class II and III patients vary from those taking multiple medications to patients who have cardiorespiratory impairment during recreational activities. These patients generally require more specialized care such as cardiac monitoring, and good pain control and sedation (as indicated) to reduce myocardial oxygen requirements while not compromising the blood pressure and respiration. Care must
be exercised to adjust the dental chair in a semi-reclining rather than flat or full supine position that may impair breathing or increase the blood volume and cardiac work of the heart. Patients should continue their medication without interruption on the day of treatment and sublingual nitrates used prophylactically only if the patient is experiencing angina from anxiety of dental treatment. Exogenous vasoconstrictors are tolerated in low dosages with local anesthetics but even 3-4 ml of 1:100,000 dilution of epinephrine can increase serum catecholamine levels. For this reason, blood pressure and pulse rates and rhythm are recommended every 5 minutes for 20 minutes after injection. This may be done by the "floating" dental assistant or nurse who is free to take the vital signs.

Evaluation of Vital Signs

Blood pressure — a baseline pressure immediately when a patient arrives at the office is more important than after resting comfortably in the chair or waiting room. This evaluates the cardiovascular response to stressful activities leading up to the patient’s arrival which may even include the frustration of automobile traffic or just finding the office or a parking space. If an initial recording of greater than 140/90 decreases to below these values after resting, labile or unstable hypertension should be suspected. When compared to a patient who is consistently above 140/90 before and after stressful situations (stable hypertension), the labile patient will likely have extreme fluctuations in blood pressure with sympathetic stimulation from anxiety or catecholamine release.

Hypotension during a procedure should not exceed 20-30% of the baseline systolic or diastolic pressures. If a drop in blood pressure occurs, treatment should be conservative including changing the chair to a Trendelenburg position (head down), intravenous fluids and oxygen by mask. Care should be exercised to prevent the administration of excessive fluids (i.e., dextrose in water or lactated Ringer’s solution). A maximum of 5 ml of fluid per kilogram of body weight on a patient with heart disease can be given to help correct hypotension. More aggressive therapy should include ephedrine 12-25 mg/m² intravenously or intramuscularly. On the other hand, hypertension is best managed initially with cessation of the procedure. Administration of mask oxygen (not nasal cannula), and small amounts of intravenous analgesics titrated to control pain. Nitrous oxide, 35-50%, is very useful in the treatment of pain-induced hypertension. Several cases of hypertension (i.e., 220-260/110-140) have been managed initially, to relax the patient, by nitrous oxide analgesia with good success.

Pulse

Preoperative pulse rate and rhythm should be recorded prior to local anesthesia. Slight intraoperative changes are expected, but particularly in patients taking beta-adrenergic blocking agents, excessive rises in heart rate can cause myocardial ischemia. A simple but practical formula of systolic blood pressure times heart rate (rate-pressure product) can be a useful guide to ischemia in cardiac disorders. If the product is greater than 12-14,000 (i.e., 140 rate and 100 systolic pressure), undesirable increases in myocardial oxygen consumption are likely to occur. Conversely, bradycardia in a cardiac patient usually indicates either drug toxicity, heart block, or hypoxia. The level of consciousness will be helpful in deciding whether treatment with fluids and vaso-pressors is required.

Arrhythmias which may develop, require rapid recognition with the aid of an electrocardiograph and treatment. Generally speaking, supraventricular arrhythmias are usually hemodynamically insignificant compared to the ventricular ones, unless the ventricular response to the supraventricular rhythm is such that left ventricular filling is decreased and oxygen demands are not met. This can be seen with atrial flutter (rate 300) with 2:1 block giving a ventricular rate of 150 which is too fast to allow proper filling. This type of arrhythmia requires early management with initial administration of droperidol chloride 5-15 mg/m² intravenously, a short-acting parasympathomimetic which increases vagal activity and slows the heart rate. Some patients may need digitalization. If the arrhythmia is refractory to these treatments, one may consider cardioversion at 40 watt-seconds, direct current shock with a cardiac defibrillator to correct the atrial arrhythmia.

Ventricular arrhythmias are almost always more life-threatening, and these include frequent premature ventricular contractions, ventricular tachycardia, and ventricular fibrillation. Occasional premature ventricular contractions (pvc’s) are usually benign unless occurring at a rate greater than 6 pvc’s per minute as documented on electrocardiogram. If ventricular tachycardia develops, it should be treated with lidocaine 1 mg/m² kg/m² intravenously followed by a continuous drip of 1-4 mg/m² minute and transportation of the patient to the nearest emergency room.

Cardiac monitoring with standard leads is the best way to continuously evaluate pulse rate and rhythm. But, with a minor adjustment in the placement of leads, the rhythm strip can also depict any changes in ST segments and T waves, which can reflect myocardial perfusion abnormalities such as myocardial or endocardial ischemia, or myocardial infarction.

Electrocardiographic monitoring is often advisable even for minor surgical procedures in the cardiac patient. The anterior, inferior, and lateral myocardium can be evaluated simply by shifting the classical standard three leads across the chest. These modifications significantly increase the amount of information obtained from cardiac monitoring, and improve our ability to properly manage the cardiac
patient. The normal placement of the chest leads is shown in the diagram (Fig. 1). By switching the leads in any of the three positions (Figs. 2, 3 and 4), the anterior, inferior, and the lateral myocardial areas can be monitored. The anterior myocardium can be seen by lead placement (Figs. 2 and 3) and the inferior-lateral areas (Fig. 4) with the ECG monitor switched to lead 2 position.

If the patient needs to have simultaneous monitoring of the anterior and lateral myocardium, lead placement in Figure 4 is most useful. With the monitor on lead 2 and the chest leads in Figure 4, electrical current from the inferior myocardium can be seen. By changing the switch on the cardiac monitor to lead 1, the anterior and lateral myocardium currents can be seen. The advantage of this lead arrangement is the chest leads do not need to be shifted around as in the previous monitoring pattern. Both myocardial ischemia and cardiac arrhythmias can be seen with either setting. Since ischemia is more commonly seen in the antero-lateral myocardium than in the inferior myocardium the modified V5 lead is most helpful. The following case illustrates the electrocardiographic pattern using the lead positioning in Figure 4.

![Figure 1](image1.png)

Conventional electrocardiographic lead position monitoring lead 2 (most commonly used to detect arrhythmias).

![Figure 2](image2.png)

Modified chest lead position to monitor anterior myocardium (MCL 1) monitor selector on lead 2.

![Figure 3](image3.png)

Modified chest lead position to monitor lateral myocardium (MCL 5) monitor selector on lead 2.

![Figure 4](image4.png)

Modified chest lead position to monitor anterior and lateral myocardium simultaneously. With the left arm lead in the V5 position (anterior axilla) and the left leg in the normal position on the chest wall, monitor selector is changed from lead 1 to lead 2, respectively.

![Figure 5](image5.png)

Classical lead 2 pattern recorded from the modified chest lead placement in Figure 4.

![Figure 6](image6.png)

Modified V5 lead from lead placement in Figure 4. Changes in ST segment and T wave are seen while recording the antero-lateral myocardium.
A 52-year-old female with a previous history of anterolateral myocardial infarction in October, 1981, presents for major oral surgery. She has a continual, unstable angina and is taking nitroglycerine as necessary. In the modified V₃ lead position the QRS height, p wave configuration, and the ST segments are slightly different from the conventional lead 2 position. With the history of anterolateral myocardial infarction, this lead pattern should be monitored continuously. Lead 2 can also be readily obtained should an inferior myocardial ischemia or a cardiac arrhythmia occur.

Summary
The cardiac patient represents a significant portion of the medically compromised patient population. Consultation with the patient’s cardiologist is advisable. The general dentist involved in their care should also be well versed in their proper evaluation and management in order to provide safe, effective treatment and avoid serious complications. An overview of our approach has been presented.

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