Purpose: The purpose of this review is to consider the current knowledge of the biology of dental caries and chronic periodontitis in the development of new diagnostic and prognostic tests. The use of these tests and their application to the diagnosis, prognosis, and management of these major disease entities are also presented in this review.

Materials and Methods: Literature review with author’s opinions.

Findings and Conclusions: Overall Clinical Objectives of Diagnostic Tests and Relevant Terminology: Diagnostic tests serve multiple clinical objectives such: a) identifying predisposing risk factors to modify risks and prevent disease; b) identifying early disease-associated biochemical or physical changes prior to clinical signs of disease; and c) to determine which specific type of disease is involved to guide selection of the most effective therapy. The term “diagnosis” is used in medicine to describe the determination of the nature of a disease. A “diagnostic” refers to tools, procedures, or technologies that are used in that determination. A diagnostic test or technology is used to identify elements of an existing disease process. Diagnostic tests differentiate whether a person has a specific disease at that time. The accuracy of a diagnostic test in detecting a specific disease is based on whether the disease element does or does not exist at the time of the diagnosis. Diagnostic test performance criteria are based on the following performance criteria: a) sensitivity; b) specificity; and c) predictive values. A “prognosis” is the prediction of the future course and/or outcome of a disease or a disease treatment, given current information. A prognosis is also described in terms of probabilities. Prognostic tools or technologies are used to make the prediction. The strength of a specific prognostic test is described in terms of the relative risk for a future event when the test parameter is present compared to when it is not present. “Risk markers” are biologic markers that either a) indicate disease or disease progression but are not causal or b) represent historic evidence of the disease. “Risk factors” are characteristics of the person or environment that, when present, directly result in an increased likelihood that a person will get a disease, and when absent, directly result in a decreased likelihood of disease. For oral diseases, modification of risk factors can both reduce the risk and decrease the disease.

Types of Diagnostic and Prognostic Information: Diagnoses are determined by analyzing the information obtained through the oral exam. The information collected during this exam includes demographic data such as age and gender, medical history, history of current and past disease, radiographic findings, and clinical features of observations. A diagnosis of periodontal disease can be made after analyzing all the information collected from a periodontal examination. This information includes: a) the presence or absence of clinical signs of inflammation such as bleeding upon probing; b) probing depths; c) extent and pattern of loss of clinical attachment and bone; d) patient’s medical and dental histories; and e) presence or absence of other signs or symptoms including pain, ulceration, and amount of observable plaque and calculus. Periodontal disease has been demonstrated to be the result of interplay between 3 factors: 1) bacterial challenge; 2) host response; and 3) other modifying factors. These modifying factors include the following: environmental and internal modifying factors. Environmental factors are defined as habits or behaviors that can be changed. Internal factors represent factors that are endogenous to the individual.

Bacterial Challenge: The presence of certain bacteria is known to cause periodontal disease. The failure to eliminate these bacteria is associated with disease progression and less favorable treatment responses. Common bacterial species associated with the majority of cases of chronic periodontitis are Porphyromonas gingivalis, Bacteroides forsythus, Actinobacillus actinomycetemcomitans and spirochetes. There is also a role for other bacteria such as Campylobacter rectus, Eubacterium nodatum, and Fusobacterium nucleatum to name a few. Microbial analysis may be conducted in order to obtain information useful for patient management. Results of the test may identify the nature of the patient’s periodontal infection and help guide
antimicrobial management. Microbial analysis may also be useful for compliant patients who have been treated for periodontal disease and continue to have evidence of infection.

**Host Response and Modifying Factors:**

- **Smoking:** Patients who smoke more than 10 cigarettes each day have an increased risk of more severe periodontal disease, a less predictable response to initial therapy, and more complicated therapeutic response. Progression of periodontal disease has also been shown to be 5 times greater in heavy smokers than in nonsmokers.

- **Irregular Dental Habits:** Patients who receive irregular dental care and do not comply with suggested oral hygiene instructions and maintenance appointments experience more disease, worse healing, and more tooth loss compared to individuals who are compliant and receive regular care.

- **Diabetes:** Axelsson (2002) demonstrated in one cross-sectional study that diabetes was the only systemic disease positively associated with attachment loss. Diabetes has also been shown to influence periodontal destruction by causing alterations in collagen metabolism and phagocytic cell function.

- **Genetic Factors:** Twin studies have demonstrated a variance in clinical expression of chronic periodontal disease among these patients. Fc-gamma receptor and interleukin-1 have been demonstrated through studies to increase the risk of progression and severity of chronic periodontitis. Approximately 30-43 percent of the Caucasian population tests positive for the specific combination of IL-1 gene variations associated with disease progression and more severe periodontal disease expression.

- **Biochemical Parameters:** A number of enzymes, tissue breakdown products, and inflammatory mediators are released from host cells and tissues during the development and progression of periodontal infections. These include the following: 1) Host-derived enzymes such as matrix metalloproteinase-1 produced by fibroblasts and epithelial cells; 2) Host-derived inflammatory mediators such as prostaglandin E2, interleukin-1 and 1a, acute phase proteins, and immunoglobins which are found in the GCF; and 3) Tissue breakdown products such as hydroxyproline which is a marker found in connective tissue breakdown.

**Diagnosis and Prognosis of Dental Caries:** Dental caries is a bacterial disease that results in the breakdown of tooth structure in the presence of sugar by acid forming bacteria such as Streptococcus mutans, Streptococcus sobrinus, and several other lactobacillus species. Current technologies demonstrate diagnostic thresholds that inherently limit practical clinical detection of many lesions until late stage cavitation. The risk factors for prediction of caries activity include: 1) the levels of acidogenic bacteria; 2) the frequency of fermentable carbohydrate ingestion; and 3) the level of salivary flow. The risk for future cariogenic activity involves the following considerations: 1) the frequency of ingestion of fermentable carbohydrates; and 2) irregular dental care present in minority children who are economically underprivileged, elderly, chronically ill, and institutionalized individuals.

**Stages of Disease and Stage Specific Diagnostic Information:** Oral hygiene practices and risk factors contribute to the movement of patients from one disease stage to another. By managing or controlling environmental risk factors, it is possible to slow or stop disease progression.

- **Health Stage:** The goal of oral health is to maintain a disease-free condition by practicing good oral hygiene and managing the risk factors that contribute to disease. Risk algorithms can be used to systematize evaluations to improve predictability of care. At the health stage, patients should be managed with a routine oral examination and a periodontal examination, as well, to explore risk factors such as smoking, systemic disease, and intake of fermentable carbohydrates.

- **Subclinical Disease:** The goal of treatment for this phase is to identify pathological processes before clinical disease develops. The detection of subclinical disease requires some type of assessment technology that identifies biological or structural changes in critical tissues before signs and symptoms are evident clinically. No strong technology candidates are available at this time for detection of subclinical disease in periodontal disease. Assays of biochemical markers of periodontal disease progression are in development. These markers include: 1) Enzymes such as aspartate aminotransferase, matrix metalloproteinases and alkaline phosphatase to name a few; 2) Inflammatory mediators such as prostaglandin E2, acute phase proteins, and several proinflammatory cytokines; and 3) Tissue breakdown products such as glycosaminoglycans, hydroxyproline,
and several bone-associated proteins.

**Early to moderate Clinical Disease:** The goals of this phase of disease for both caries and periodontal disease are to identify and manage disease progression factors and to repair or reverse early tissue destruction. Assessments of caries and periodontal disease patients in the early to moderate disease state involve the standard clinical evaluations for past disease experience. Also included should be assessments of risk factors that contribute to disease activity and future progression. For caries this includes assessment of dietary intake of fermentable carbohydrates, cariogenic bacteria levels, and salivary flow. For periodontal disease, risk assessment includes assessment of total bacteria levels, the presence of diabetes, smoking, and genetic predisposition.

**Severe Generalized Disease:** The goal of this phase is to stop the progression of current disease by eliminating bacterial reservoirs, repair structural damage, restore function, and establish a disease prevention regimen based on the patient’s risk factors.