ORAL MEDICINE

INFECTIVE ENDOCARDITIS AND DENTISTRY

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ABSTRACT

Infective endocarditis is the infection of endothelium of heart and vessels. It is an uncommon condition associated with substantial mortality and morbidity. It is a common belief that it could possibly be prevented by appropriate use of antibiotic prophylaxis. The objective of this review is to provide the background knowledge of infective endocarditis in terms of etiology, pathogenesis, clinical features, diagnostic strategies and preventive aspects. The extensive search of Medline from 1986 to January 2007 was performed. There were almost 100 articles which were relevant to the subject and were chosen to extract the possible information.

There has been considerable controversy in the past as to what sort of dental procedures are to be blamed and which group of patients should receive prophylactic antibiotics. The guidelines have frequently been revised and two main guidelines namely of American Heart Association (AHA) and of British Society ofAntimicrobial Chemotherapy (BSAC) are currently being practiced. For a dentist these guidelines are often confusing and cumbersome. This article is aimed at relieving the undue anxiety of a dentist when he comes across with a patient predisposed to infective endocarditis and to create a better understanding between dentist, patient and cardiologist.

Key words: *Endocarditis, Dental Procedures, Prophylaxis*

INTRODUCTION

Infective Endocarditis is a microbial infection of endothelial surface of the heart. The characteristic lesion is vegetation, which is a mass of fibrin, platelets and microorganisms embedded inside. Heart valves are the most common sites of involvement beside septal defects, chordae tendinea and mural endocardium. Infection can also occur at arteriovenous Shunts whether congenital or acquired, arterio-arterial Shunts like Patent ductus arteriosus or co-arctation of aorta. Many species of bacteria, fungi, mycobacteria, mycoplasma, Rickettsia, and Chlamydia can cause infective endocarditis (IE) but majority of the cases are due to streptococci, staphylococci, enterococci, and gram negative coccobacilli 1,2,3.

Oftenly we use the terms acute and sub-acute infective endocatditis. Acute IE is a serious disease with marked toxicity, destruction of valves and meta static infection while sub-acute IE progresses over weeks and months. Acute infections are usually caused by Staphylococcus Aureus whereas sub-acute by Streptococcus viridians, Enterococcus and Gram-negative Coccobacillus. In recent years Staphylococcus has been shown to surpass Streptococcus viridians as the most common cause of infective endocarditis, the reason being the recent deterioration of anti-biotics susceptibility among the group of gram —positive cocci4-6.

The incidence of IE remains relatively stable from 1950 through 1987 to 1999, at about 4.2 to 5.9 per 100,000 patients year1. Higher incidence of 11.6 episode per 100,000 patients has been reported from Sweden'". Endocarditis usually occurs more frequently in men. Gender derived ratios range from 1.6 to 2.5 (women vs. men). The age specific incidence of endocarditis increases progressively over 30 years of age and exceeds 14.5 to 30 case per 100,000 persons years[3].

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The pre-disposing conditions like Congenital Heart Disease, Rheumatic Heart Disease, Mitral Valve Prolapse, Degenerative Heart Disease, Asymmetrical Septal Hypertrophy or Intravenous drug abuse is present in about 35-75 % of the cases[2]. Majority of the rest of the cases are due to involvement of prosthetic valves. Predisposing conditions remain unidentified in some patients[2-3]. The incidence of IE in children ranges from 1 in 4500 to 1 in 1280. These children usually have underlying Congenital Heart Disease.[1'2'3]

In adults, Mitral Valve Prolapse has emerged as a major predisposing structural cardiac abnormality accounting for 7 to 30 % of native valve endocarditis[12,13]. Rheumatic Heart Disease is the next common condition, accounting for 20-25 % of the cases reported in 1970 and now more recently reported from North America and Europe the RHD predisposed to IE in only 7 and 18% of the cases[2]. Usually the Mitral Valve is involved, more so in women followed by Aortic Valve, which is more commonly involved in men. In under developed part of the world, Rheumatic heart disease is still the most commonly found underlying cardiac abnormality and presents at early age[2]. In developed countries the incidence of Rheumatic heart disease has decreased however the incidence of endocarditis remains the same as the spectrum of underlying heart disease has changed. Congenital Heart Disease is responsible for 10-20 % of the cases. The most common lesions are Patent Ductus Arteriosus, Ventricular Septal Defect and Bicuspid Aortic Valve.

The risk of IE in drug abuser is 2-5 % patient years. The most common cases are recognized in men, age ranges from 27-37 years,[7-10]. The most common site involved is the Tricuspid Valve (46-78%) followed by Mitral and Aortic Valve. In 75-93 % of the cases of IE in intravenous drug abusers valves were normal, before IE and the remaining patient had preexisting Mitral or Aortic disease. Fifty percent of the cases of IE in I.V. drug abuser are due to staphylococcus, but other organisms particularly unusual microorganisms like Corynebacterium, Lactobacillus and Nisseria species have been identified in about 3-5 % of the case[10]

Prosthetic valve endocarditis accounts for 7 — 25% of the cases of infective endocarditis. In most developed countries, during the first three months post replacement, the mechanical valves are at higher risk than bioprosthesis but later the risk of IE becomes same for both types[11,14,15,16]. Cases with onset within 2 months after surgery are called early onset prosthethtic valve endocarditis and are acquired in hospital. Cases occurring 12 months after surgery are called late prosthetic valve endocarditis and are community acquired. Cases between two and twelve months after surgery are mixed either hospital acquired or caused by less virulent microorganisms acquired from community. Late-onset prosthetic valve endocarditis is more frequent now because patients with prosthetic heart valves are surviving longer. Fortunately, these cases are caused by the same organisms as native valve endocarditis (NVE), and are usually easier to treat[14-16].

The HACEK group of fastidious gram-negative bacteria can cause endocarditis. HACEK is term used for endocarditis due to Haemophilus, Actinobacillus, Cardio bacterium, Eikenella, and Kingella species of bacteria. Clinically, these cases are characterized by a sub acute course, and often present with embolic lesions from large vegetations".

Most cases of fungal endocarditis occur in patients who are receiving prolonged antibiotics or parenteral nutrition through central vascular catheters. Such patients may also be immunocompromised. The most common species is Candida albicans, followed by Candida parapsilosis. Endocarditis due to Histoplasma capsulatum or Aspergillus species is rare[18,19].

Unusual cases of endocarditis should be considered when standard microbiologic techniques fail to provide a diagnosis. Q-fever endocarditis due to Coxiellaburnetii is an example. Patients may not have fever, but they frequently have underlying valvular heart disease and are on immuno-suppressive therapy. Bartonella henselae may also cause endocarditis, and diagnosis here is also difficult. Such infections are seen in the homeless and alcoholic population[20] Non bacterial thrombotic endocarditis may be caused by endothelial injury and hypercoagulable state. It is more common with advancing age and in patients with malignancy, chronic renal failure, SLE, intracardiac catheters and valvular heart disease. Bacteraemia associated with health care procedures converts nonbacterial thrombotic endocarditis (NBTE) to infective endocarditis (IE)21.

The interval between presumed bacteraemia and the onset of symptoms of IE is less than two weeks [22]. Fever may be absent in elderly patients. Heart murmurs are audible in 80 % of the patient. New murmurs or changing murmurs are usually found in acute 1E[23-28]. Spleen is enlarged in 15-50 % of the patients[22]. Certain peripheral manifestations of IE, Petechiae, Splinter or Sub-ungual Hemorrhage, Osler's node, Janeway lesion and Roth spots are not that common[29] The muscular symptoms like myalgias, arthralgias are quite common. Systemic embolisation is a serious complication and often found the cause of death at autopsy, Coronary, Cerebral, Renal and the Peripheral arteries maybe involved[29-32].

Von Reyn and associates[33] suggested the criteria for the diagnosis of endocarditis (Table 1). A positive valve culture or histology was the basis for the diagnosis of IE. Major criteria for probable endocarditis were either persistent bacteraemia with a new regurgitant heart murmur or valvular heart disease with vasculitis; or negative or intermittent bacteraemia with fever and a new regurgitant heart murmur with vasculitis.

TABLE 1: MODIFIED FROM REFERENCE 33

Von Reyn Criteria for the Diagnosis of Infective Endocarditis
☐ Positive valve culture or histology or
☐ Persistent bacteraemia with new regurgitant heart murmur or valvular heart disease and vasculitis or
☐ Negative or intermittent bacteraemia and fever; plus new Regurgitant heart murmur and vasculitis

Diagnosis of IE nowadays is based on the presence of either major or minor clinical criteria according to modified Duke Criteria[34]. Major criteria in the Duke strategy included IE documented by data obtained at the time of open heart surgery or autopsy (pathologically definite) or by well-defined microbiogical criteria (high-grade bacteraemia or fungemia) plus echocardiographic data (clinically definite). Duke criteria gave diagnostic weight to bacteraemia with staphylococci or enterococci only when they were community acquired and without an apparent primary focus; these latter types of bacteraemia have the highest risk of being associated with IE.

The Duke criteria incorporated echocardiographic findings in the diagnostic strategy. Major diagnostic weight was given to only 3 typical echocardiographic findings: mobile, echo dense masses attached to valvular leaflets or mural endocardium; periannular abscesses; or new dehiscence of a valvular prosthesis.

Six common but less specific findings of IE also were included as minor criteria in the original Duke schema; intermittent bacteraemia or fungemia, fever, or major embolic events, nonembolic vascular phenomena, underlying disease or injection drug use, and echocardiographic abnormalities that fell short of typical valvular vegetations abscesses, or dehiscence. Clinically definite IE by the Duke criteria required the presence of 2 major criteria, 1 major criterion and 3 minor criteria, or 5 minor criteria.

Severe refinements have been made recently to both the major and minor Duke criteria[34-38]. As noted above, in the original Duke criteria, bacteraemiacriteria34-36rom *S aureus* was considered to fulfill a major criterion only if it was community acquired because ample literature has suggested that this parameter is an important surrogate marker for underlying IE. An increasing number of contemporary studies, however, have documented IE in patients experiencing nosocomial staphylococcal bacteraemia[37].

Therefore, the modified Duke Criteria (Tables 2A and 2B recommend the inclusion ofS *aureus* bacteraemia as a major criterion, regardless of whether the infection is nosocomially acquired (with or without a removable source of infection) or community acquired.

Lab tests show abnormal hematological parameters. Normochronic normocytic anemia, Low Serum Iron Level, Leukocytosis, elevated ESR is commonly found. Anemia worsens with increasing duration of illness and it may be absent in acute IE.White cell count may be normal in sub acute IE and presence of leukocytosis points towards acute **IE.** Circulating Immune Complexes, Rheumatoid factor, and C-reactive proteins are often positive and their presence correlates with disease activity. Proteinuria and Hematuria is most commonly encountered abnormality while performing urine analysis of such patients[38].

Echocardiography remains the gold standard diagnostic tool of the diagnosis of IE. Many patients can be

TABLE 2A: MODIFIED FROM REFERENCE 34

Definite Infective Endocarditis			
Pathological Criteria	**Clinical Criteria**	**Possible IE**	**Rejected**
Microorganisms demonstrated by culture or histological examination of a vegetation, a vegetation that has embolized, or an intracardiac abscess specimen	2 major criteria; or 1 major criterion and 3 minor criteria; or 5 minor criteria	***1 major criterion and 1 minor criterion; or 3 minor criteria***	Firm alternative diagnosis explaining evidence of IE; or Resolution of IE syndrome with antibiotic therapy for < 4 days; or No pathological evidence of IE at surgery or autopsy; with antibiotic therapy for <4 days; or Does not meet criteria for possible IE as above

TABLE 2B: MODIFIED FROM REFERENCE 34

Definition of Terms Used in the Modified Duke Criteria for the Diagnosis of Infective Endocarditis		
Major Criteria		**Minor Criteria**
Blood Culture for positive IE	**Evidence of endocardial involvement**	Predisposition, predisposing heart condition, or IDU Fever temperature > 38 ° C Vascular phenomena, major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, and Janeway's lesions Immunologic Phenomena: glomerulonephritis, Osler's nodes, roth's spots, and rheumatoid factor Microbiological evidence: positive blood culture does not meet a major criterion as above or serological evidence of active infection with organism consistent with IE
Typical microorganisms consistent with IE from 2 separate blood cultures; Viridians streptococci, *Streptococcus bovis*, HACEK group, ***Staphylococcus aureus; or*** community-acquired enterococci in the absence of a primary focus;	Echocardiogram positive for IE (**TEE recommended for patients with prosthetic valves, rated at least "possible IE" by clinical criteria, or complicated IE [paravalvular abscess]; TTE as first test in other patients**) defined as follows: oscillating intra cardiac mass on valve or supporting structures, in the path of regurgitant jets, or on Implanted material abscess; or new partial dehiscence of prosthetic valve; new valvular regurgitation (worsening or changing or preexisting murmur not sufficient	
or Microorganisms consistent with IE from persistently positive blood cultures defined as follows: At least 2 positive cultures of blood samples drawn > 12 h apart; or all 3 majority of > 4 separate cultures of blood (with first and last sample drawn at least 1 h apart)		
Single positive blood culture for *Coxiella burnetti* or anti-phase 1 lgG Antibody titer > 1:800		

imaged adequately by Trans Thoracic Echo. However, TEE (Trans Esophageal Echo) has invariably been required in patients who have strong clinical suspicion of 1E[36,39]. The establishment of microbial cause is of utmost importance. Infection is recovered from blood or from the surgically removed vegetation. Three separate sets of blood Cultures are required within 24 hours, obtained via separate venepunctures. Laboratory should be instructed to look for atypical microorganisms also.

The clinical features of infective endocarditis result from the local destructive effects of intracardiac lesions, septic embolisation, hematogenous spread leading to persistent bacteraemia and deposition of immune complexes.

Dental Procedures as risk factor

Bacteraemia commonly occurs during activities of daily living such as tooth brushing or food chewing. Significant bacteraemias caused by organisms commonly associated with IE are attributable to specific procedures[46-46]. Genesis of bacteraemia generally is considered when a dental procedure leads to bleeding[44'45]. Complete denture prostheses for edentulous mouth do not pose risk of IE. Decreased risk was suggested by use of dental floss once a day[47].

There is overwhelming evidence that bacteraemia result from oral/dental surgical and non-surgical treatment. However, the risk of **IE** due to this bacteraemia is still debated.[48-51] Risk of IE increases in individual with poor periodontal health or other oral infections (52-56). Rinsing with Chlorhexidine Gluconate or Povidone-Iodine is recommended prior to manipulation of Dental tissue. To date, there is no conclusive evidence that confirms the reduction of bacteraemia by oral bio load[57-65].

Any bacteraemia occurring during dental treatment does not significantly increase the risk of Endocarditis [66]. A recent Cochrane review [67] concluded that there was no evidence to support the use of prophylactic penicillin to prevent endocarditis in invasive dental procedures. The working party however, agreed that ideally a prospective double blind trial to evaluate the risk/benefit of prophylactic antibiotics should be carried out. This is unlikely to take place, as the number of the patients required will be too much and the recent guidelines have already recommended the prophylaxis for the patients considered to be at high risk[68].

Good oral hygiene is probably the most important factor in reducing risk of IE in susceptible individuals and access to high quality dental care should be facilitated once a patient is found to have cardiac anomaly putting him at higher risk ofEndocarditis. He should be referred to a dentist to optimize the dental hygiene, Patients undergoing valve replacement, placement of aortic grafts or conduit should also be referred for dental assessment. The dental treatment preferably should be completed fifteen days ahead ofplanned cardiac surgical procedures in order to allow dental mucosal healing, All elective dental procedures should be delayed for at least three months after cardiac surgery[68].

The degree of oral inflammation and infection is proportional to the incidence and magnitude of bacteraemia[69'70] Dental procedures which can lead to significant bacteraemia and those creating Medium and low risk of bacteraemia are shown in Table 3[71-76].

TABLE 3: MODIFIED FROM THERAPEUTIC GUIDELINES JUNE, 2006.

High-risk dental procedures	Medium-risk dental procedures	Low-risk dental procedures
Extraction Periodontal procedures including surgery and root planning Replanting avulsed teeth Other surgical procedures (e.g.) (implant placement, apicoectomy)	Periodontal probing Intraligamentary and intra-osseous local anaesthetic injection Supragingival calculus Removal/cleaning Rubber dam placement with clamps (where risk of damaging gingival) Restorative matrix/band strip placement Endodontics beyond the apical foramen Placement of orthodontic bands Placement of interdental wedges Subgingical placement of retraction cords, antibiotic fibers or antibiotic strips	Oral-examination Infiltration and block local anaesthetic injection Restorative dentistry Supragingival rubber dam Clamping and placement of rubber dam Intracanal endodontic procedures Removal of sutures Impressions and construction of dentures Orthodontic bracket placement and adjustment of fixed appliances Application of gels Intraoral radiographs supragingival plaque removal

Antibiotics prophylaxis is indicated to prevent endocarditis when a patient with known cardiac condition undergoes dental treatment. Dental procedures induce bacteraemia with bacterial species that often are typical causative microorganisms leading to endocarditis.Endocarditis carries high morbidity and mortality; therefore, antibiotics should be administered to susceptible patients before dental procedures to prevent endocarditis [51]. In experimental models antibiotics have been proven as a definite treatment to prevent endocarditis[5177,78], On these grounds, prophylaxis against endocarditis is widely recommended[45,51].

The need for antibiotics for endocarditis prevention has been questioned repeatedly8°-86. The less the evidence there is, the more antibiotic we give. Three major studies[82,86,87] and several outcomes analyses and commentaries8°, [81, 84, 85] have presented substantial grounds to challenge the value of the invalidated practice of administering antibiotics before dental procedures to prevent endocarditis.

A Dutch study[82] assessed 427 patients with endocarditis and found that 64% of the patients would have been candidates for prophylaxis based on previous cardiac conditions. This study further concluded that even if antibiotic prophylaxis was 100% effective and was provided to all patients who were at risk, small fraction (5.3%) would be potentially prevented.

TABLE 4 MODIFIED FROM CONCISE GUIDELINES OF BRITISH CARDIAC SOCIETY[79]

Dental Procedures and Antibiotic prophylaxis for high-risk and low risk cases	
Prophylaxis Required	**Prophylaxis not required**
Periodontal probing	Dental examination
Sialography	Intra – oral radiographs
Polishing teeth with rubber cup	Extra – oral radiographs
Oral irrigitation with water jet	Fissure sealants
Light scaling	Fluoride treatments
Deep scaling	Air polishing
Scaling teeth with hand instrument	Infiltration local anaesthetic injections
Scaling with ultrasonic instrument	Nerve block local anaesthesia
Intraligamental local anaeshetic injections	Oral airway for GA
Comprehensive dental treatment under	Nasal airway for GA
general anaesthesia and extraction and fillings	Laryngeal mask airway for GA
Rubber dam placement	Slow and fast drilling of teeth (without rubber dam)
Matrix band and wedge placement	Root canal instrumentation with the root canal
Gingival retraction cord placement	Vital pulpotomy of primary molar
Root planning (similar to scaling)	Pulpotomy of permanent tooth
Antibiotic fibers or strips placed subgingivally	Alginate impressions
Gingivectomy	Placement of removable appliances
Periodontal surgery	Band placement and cementation
Root canal instrumentation beyond the apex	Band removal
Avulsed tooth reimplantation	Adjustment of fixed appliances
Non – vital puloptomy of primary molar	Incision and drainage of an abscess without extraction
Tooth separation	Dental implants – transmucosal fixture
Expose or expose and bond tooth/teeth	Suture removal
Extraction of single tooth	Removal of surgical packs
Extraction of multiple teeth	Exfoliation of primary teeth
Incision and drainage of an abscess with extraction	Tooth brushing
Mucoperiosteal flap to gain access to tooth or lesion	Flossing
Dental implants (as mucoperiosteal flap)	Use of interdental wooden points

A more recent study[87] assessed 287 cases from Philadelphia area and concluded that there was no significant relationship between development of IE and dental procedures even in patients with valve disease. The statistical risk for endocarditis did not change regardless of whether antibiotics were used in dental treatment. Although this study confirmed that underlying heart disease is the major risk factor of IE. Only few cases of IE were prevented with the use of chemoprophylaxis even if antibiotics were 100% effective and given to 100% of the patients when indicated.

David Durack in a review article[88] said that time has come to scale back on prophylaxis against endocarditis before dental treatment. In the matrix of procedures related to predisposing conditions, prophylaxis should be downgraded to "not recommend" for most dental procedures except extractions and gingival surgery (including implant placement) and for most underlying cardiac conditions except prosthetic valves and previous endocarditis. When any one or more of these four high-risk factors is present, prophylaxis should follow the present American Heart Association (AHA) guidelines [45].

Mitral valve prolapse without mitral regurgitation is not included among the underlying conditions requiring routine prophylaxis because although a signifi cant number of cases of endocarditis do occur in patients with mitral valve prolapse, the denominator of susceptible persons is large and the risk encountered by an individual patient with mitral valve prolapse is lower than that for patients with prosthetic valves, previous endocarditis, or both[51]. Furthermore, the prognosis for cure of viridians streptococcal endocarditis in a patient with mitral valve prolapse is good[12-16].

Although failure of Antibiotic Prophylaxis is not rare but failure to give prophylaxis is considered to be a malpractice. Despite all the challenges recommendations for IE prophylaxis are existing and should be incorporated into day to day dental practice.

Before recommending the prophylactic antibiotic, one must remember the common cardiac conditions which can pose risk of Infective Endocarditis.They have been divided into three sub-groups, High, moderate and low risk group according to Therapeutic guidelines revised in July 2006 (Table 5) Mitral valve prolapse with mitral regurgitation has been placed in high risk group by these guidelines which differ from the AHA[45] guidelines and British society of antimicrobial chemotherapy (BSAC) guidelines",[89] in which mitral valve prolapse associated with mitral regurgitation is placed in medium risk group,

TABLE 5: MODIFIED FROM THERAPEUTIC GUIDELINES JUNE, 2006

High-risk conditions	Medium-risk conditions	Low-risk conditions
Prosthetic cardiac valves Bioprosthetic Homograft Previous infective endocarditis Complex cyanotic Congenital Heart Disease (transposition, tetra logy of Fallot) Surgically constructed Systemic-pulmonary shunts or conduits Mitral valve prolapse with clinically significant regurgitation Acquired valvular dysfunction (e.g. rheumatic heart disease) in indigenous patients	Acquired valvular dysfunction (e.g. rheumatic heart disease) in non-Indigenous patients Congenital cardiac malformations other than those defined as high-or-low-risk hypertrophic cardiomyopathy Significant valvular/haemodynamic dysfunction associated with septal defects	Isolated secondum Atrial septal defects Surgical repair of septal defects Previous coronary artery bypass grafts or stents Mitral valve prolapse without regurgitation Physiological, functional or innocent murmur Previous Kawasaki disease without valvular dysfunction Cardiac pacemakers Pulmonary stenosis Heart-lung transplants

The mortality in patients with Infective Endocarditis remains very high. It depends upon many factors, including the nature of causative microorganisms being highest for pseudomonas Aeuriginosa (50%), followed by Staphylococcus Auerus (26 — 47%), streptococcus bovis and streptococcus viridians (4 — 16%) and also the host response. Immunosuppressed patient and infected with HIV have worse prognosis. Similarly, patients who get complications like Perivalvular and Myocardial abscess have worse outcome. The overall mortality rate of both native valve and prosthetic valve endocarditis remains around 20 — 25%. Relapse of the endocarditis can occur within 6 months. Relapse rate is usually around 2%. The treatment failure is another important factor, leading to high mortality particularly in patients with Staphylococcus Endocarditis [90].

Over the past few decades, substantial improvements have been made in the diagnosis and management of Infective Endocarditis, Treatment requires multi disciplinary approach. Successful and safe patients' management is accomplished appropriately by obtaining a detailed medical history and physical examination. The examination should be aimed at identifying the physical signs. Medical consultation is recommended if a dentist comes across with a patient who has the history suggestive of cardiac abnormality or who has physical signs suggestive of heart diseases.

In general, the trend has been to more specifically describe those conditions that pose significant risk for patients and to delineate low or negligible risk situations, As a result, antibiotic prophylaxis is now recommended for fewer conditions. These changes also reflect improvements in the understanding of these disease processes and changing the attitude toward the use of antibiotic. The most notable among these changes include reducing the oral dose of amoxicillin from 3 grams to 2 grams, recommending that the follow-up dose of antibiotic be discontinued, and replacing erythromycin with other antibiotics as alternatives to the penicillin. Dajani and colleagues[45] have repor-

TABLE 6: MODIFIED FROM; DAJANI AD, TAUBERT KA, WILSON W, ET AL, PREVENTION OF BACTERIAL ENDOCARDITIS, RECOMMENDATION BY THE AMERICAN HEART ASSOCIATION JADA 1997, 128; 1142-1151, THERAPEUTIC GUIDELINES JUNE, 2006.

Antibiotic Prophylactic Regimens for Certain Dental Procedures		
Situation	**Antibiotic**	**Regimen**
Standard prophylaxis	Amoxicillin	Adults: 2.0 grams; Children: 50 mg/kg orally one hr before procedure
	Ampicillin (oral treatment Not tolerated)	Adults: 2.0 g IM§ or IV§; Children: 50 mg/kg IM or IV within 30 min. before procedure
Allergic to Penicillin	Clindamycin	Adults: 600 mg; Children: 20 mg/kg orally 1 hr. before procedure
	Oral therapy not tolerated	Adults: 600 mg; Children 15 mg/kg IV 1 hr. before procedure
	Cephalaxin or cefadroxil	Adults: 2.0 g; Children 50 mg/kg orally 1 hr. before procedure
	Oral therapy not tolerated	Adults: 1.0 g; Children: 25 mg/kg IM or IV within 30 min. before procedure
	Azithromycin or clarithromycin	Adults: 500 mg; Children: 15 mg/kg orally 1 hr. before procedure

ted that 2 g of amoxicillin provides several hours of antibiotic coverage, Table 6 shows the new recommendations for prophylactic coverage for certain dental procedure

In conclusion, the patient must be instructed to report back if he suffers from any unexplained illness after dental procedure, Patients with physiological heart murmurs do not require prophylaxis. Self reported history of heart valve disease should not be the sole criterion, In cases of corrective surgery for a patient with patent ductus arteriosis, antibiotic coverage is only required for the initial 6 months after surgery. Sufficient data to support prophylactic antibiotic in cardiac transplant recipients is lacking. It is recommended to consult cardiologist if such patients attends dental clinic. It is worth noting that such patients have reduce immune response and will require meticulous care jointly by physician and dental care provider. Fixed acrylic orthodontic appliances often harbor high level of streptococcus viridians. These appliances should not be used in high risk patients. Removable appliances should be cleaned regularly, When several appointments are needed for invasive dental treatment, interval of 9 —14 days should be left to reduce the risk of overgrowth of organisms. The use of antibiotic prophylaxis during placement of dental implant is controversial. More knowledge is required to clarify the controversies.

Despite the appropriate Antibiotic Prophylaxis, Infective Endocarditis may still occur. The frequently updated guidelines for Chemoprophylaxis against IE are confusing and cumbersome, so the dentist is ultimately responsible to make decision on his or her clinical judgment to select patient who will benefit from prophylaxis. Considering that the incidence of endocarditis following most procedure is low; a reasonable approach should consider the degree to which the patient's underlying condition carries risk of endocarditis, apparent risk of bacteraemia with procedure, potential adverse effects of antibiotics used and cost effectiveness of recommended prophylactic regime. The AHA has removed Erythromycin from the current recommendations because of its complex pharmacokinetics and its side effects particularly the gastrointestinal tract disturbances. However the physicians and dentists who have successfully used this drug may elect to continue with same practice.

Prevention of IE does not mean that the emphasis should be on antibiotic prophylaxis only but more vigilance should be on the patient's dental care and systemic illnesses as a whole. Adequate and prompt treatment of infection responsible for bacteraemia or fungemia, removal of colonized intravascular devices, effective management of recurrent infections responsible for chronic bacteraemia is more important.

High risk patients, who had past history of Infective Endocarditis, history of valve replacement, or have undergone surgically constructed systemic or pulmonary shunts require special attention and supervised care jointly by physician and dentist. The most recent guidelines by British society of antimicrobial chemotherapy (BSAC) have recommended limiting the prophylaxis to only this high risk group. The debate over the relationship between dentistry and Infective Endocarditis will continue. More research particularly randomized clinical trials are required to clear the controversies, however the evidence is emerging that dental treatment is less likely to be the cause of IE than thought previously. Despite the fact that there is lack of evidence favoring antibiotics prophylaxis the existing recommendations remain unchanged and need of safe and wise incorporation of these guidelines in the dental practice is of utmost importance,

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