PERIODONTAL DISEASE:
DIAGNOSIS AND TREATMENT

May 2002

Guidelines Department
These guidelines were produced using the method described in the guide “Clinical Practice Guidelines - Methodology to be used in France, 1999”, published by ANAES. The following learned societies were consulted during the drafting of these guidelines:

- Association Dentaire Française;
- Société Française de Parodontologie;
- Société Française de Biologie Clinique;
- Collège National des Enseignants en Parodontologie;
- Collège National des Généralistes Enseignants;
- Société Française de Radiologie et d’Imagerie Médicale;
- Société Française de Médecine Générale;
- Société Française de Stomatologie, Chirurgie Maxillo-Faciale et Chirurgie Plastique de la face;
- Société Française de Thérapeutique en Médecine Générale;
- Société Nationale Française de Médecine Interne;
- Société de Thérapeutique Odonto-Stomatologique;
- Société de Pathologie Infectieuse de Langue Française.

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GUIDELINES AND STANDARDS

These guidelines on the diagnosis and treatment of periodontal disease were produced at the request of the French national health insurance fund for salaried workers. They do not address the issues of screening or prevention of periodontal disease.

Guidelines are graded A, B or C according to the following system:

- a grade A guideline is based on scientific evidence established by trials of a high level of evidence, for example randomised controlled trials of high power and free of major bias, and/or meta-analyses of randomised controlled trials or decision analyses based on properly conducted studies;
- a grade B guideline is based on presumption of a scientific foundation derived from studies of an intermediate level of evidence, for example randomised controlled trials of low power, well-conducted non-randomised controlled trials or cohort studies;
- a grade C guideline is based on studies of a lower level of evidence, for example case-control studies or case series.

In the absence of scientific evidence, the proposed guidelines are based on agreement among professionals.

I. DEFINITIONS

The working group proposed the following definition:
Periodontal disease can be defined as multifactorial infectious disease. It is characterised by symptoms and clinical signs that may include inflammation (visible or invisible), varying degrees of spontaneous gingival bleeding or bleeding on probing, pocket formation related to loss of attachment and of alveolar bone, and tooth mobility, which may lead to tooth loss (agreement among professionals).

Specific clinical and/or epidemiological indices have been defined to evaluate degree of inflammation, presence of plaque, presence of calculus, clinical attachment level, and to measure pocket depth. The main indices are:

- **Oral hygiene indices**: the Greene and Vermillion oral hygiene index, the Silness and Löe plaque index (PI), the O’Leary plaque index, and the Marthaler calculus index (CI).
- **Indices of inflammation**: the Löe and Silness gingival index (GI), the sulcus bleeding index (SBI), and the Massler PMA index.
- **Indices of treatment needed**: the Periodontal Treatment Need System (PTNS) and the Community Periodontal Index of Treatment Needs (CPITN). The latter is currently used for epidemiological surveys, public health projects and promotion of periodontal health.

II. CLASSIFICATION OF PERIODONTAL DISEASE

The working group used the American Academy of Periodontology classification (Armitage 1999) (see Table 1). This classification is purely nosological (agreement among professionals).
### Table 1. Classification of periodontal disease (adapted from Armitage GC. Development of the classification system for periodontal and conditions. Ann Periodontol 1999:4:1-6.)

<table>
<thead>
<tr>
<th>I GINGIVAL DISEASE</th>
<th>IV PERIODONTITIS AS A MANIFESTATION OF SYSTEMIC DISEASES</th>
</tr>
</thead>
<tbody>
<tr>
<td>A- Dental plaque-induced gingival diseases</td>
<td>A- Associated with haematological disorders</td>
</tr>
<tr>
<td>1 Gingivitis associated with dental plaque only</td>
<td>Acquired neutropenia, leukaemias, other</td>
</tr>
<tr>
<td>a) without other local contributing factors</td>
<td>1) familial and cyclic neutropenia</td>
</tr>
<tr>
<td>b) with local contributing factors (see VIII A)</td>
<td>2) Down syndrome</td>
</tr>
<tr>
<td>2 Gingival disease modified by systemic factors</td>
<td>3) leukocyte adhesion deficiency syndrome</td>
</tr>
<tr>
<td>a) associated with the endocrine system</td>
<td>4) Papillon-Lefèvre syndrome</td>
</tr>
<tr>
<td>1) puberty-associated gingivitis</td>
<td>5) Chediak-Higashi syndrome</td>
</tr>
<tr>
<td>2) menstrual cycle-associated gingivitis</td>
<td>6) hypothyroidism syndrome</td>
</tr>
<tr>
<td>3) pregnancy-associated gingivitis, pregnancy-associated pyogen granuloma</td>
<td>7) glycogen storage disease</td>
</tr>
<tr>
<td>4) diabetes mellitus-associated gingivitis</td>
<td>8) infantile genetic agranulocytosis</td>
</tr>
<tr>
<td>b) associated with blood dyscrasias: leukaemia-associated gingivitis, other</td>
<td>9) Cohen syndrome</td>
</tr>
<tr>
<td>3 Gingival diseases modified by medications</td>
<td>10) Ehlers-Danlos syndrome (types IV and VIII)</td>
</tr>
<tr>
<td>1) drug-influenced gingival enlargements</td>
<td>11) hypophosphatasa</td>
</tr>
<tr>
<td>2) drug-aggravated gingivitis: oral contraceptive-associated gingivitis, other</td>
<td>12) other</td>
</tr>
<tr>
<td>4 Gingival diseases modified by malnutrition</td>
<td>C- Not Otherwise Specified</td>
</tr>
<tr>
<td>a) ascorbic acid-deficiency gingivitis</td>
<td>V NECROTIZING PERIODONTAL DISEASES</td>
</tr>
<tr>
<td>b) other</td>
<td>Necrotizing ulcerative gingivitis, necrotizing ulcerative periodontitis</td>
</tr>
<tr>
<td>B- Non-plaque-induced gingival lesions</td>
<td>VI ABSCESSES OF THE PERIODONTIUM</td>
</tr>
<tr>
<td>1 Gingival diseases of specific bacterial origin</td>
<td>Gingival abscess, periodontal abscess, pericoronal abscess</td>
</tr>
<tr>
<td>Neisseria gonorrhoea-associated lesions, Treponema pallidum-associated lesions, streptococcal species-associated lesions</td>
<td>VII PERIODONTITIS ASSOCIATED WITH ENDODONTIC LESIONS</td>
</tr>
<tr>
<td>2 Gingival diseases of viral origin</td>
<td>Combined periodontic-endodontic lesions</td>
</tr>
<tr>
<td>a) herpes virus infections</td>
<td>VIII DEVELOPMENTAL OR ACQUIRED DEFORMITIES AND CONDITIONS</td>
</tr>
<tr>
<td>primary herpetic gingivostomatitis, recurrent oral herpes, varicella-zoster infections</td>
<td>A- Localized tooth-related factors that modify or predispose to plaque-induced gingival diseases/periodontitis</td>
</tr>
<tr>
<td>b) other</td>
<td>Tooth anatomic factors, Dental restorations/appliances, Root fractures, Cervical root resorption and cemental tears</td>
</tr>
<tr>
<td>3 Gingival diseases of fungal origin</td>
<td>B- Mucogingival deformities and conditions around teeth</td>
</tr>
<tr>
<td>a) Candida-species infections: generalized gingival candidosis</td>
<td>1) Gingival/soft tissue recession: facial or lingual surfaces, interproximal (papillary)</td>
</tr>
<tr>
<td>b) linear gingival erythema</td>
<td>2) Lack of keratinized gingiva</td>
</tr>
<tr>
<td>c) histoplasmosis</td>
<td>3) Decreased vestibular depth</td>
</tr>
<tr>
<td>d) other</td>
<td>4) Aberrant frenum/muscle position</td>
</tr>
<tr>
<td>4 Gingival lesions of genetic origin</td>
<td>5) Gingival excess: pseudopocket, inconsistent gingival margin, excessive gingival display, gingival enlargement</td>
</tr>
<tr>
<td>a) hereditary gingival fibromatosis</td>
<td>6) Abnormal color</td>
</tr>
<tr>
<td>b) other</td>
<td>C- Mucogingival deformities and conditions on edentulous ridges</td>
</tr>
<tr>
<td>5 Gingival manifestations of systemic conditions</td>
<td>1) Vertical and/or horizontal ridge deficiency</td>
</tr>
<tr>
<td>a) mucocutaneous disorders</td>
<td>2) Lack of gingiva/keratinized tissue</td>
</tr>
<tr>
<td>1) lichen planus</td>
<td>3) Gingival/soft tissue enlargement</td>
</tr>
<tr>
<td>2) pemphigoid</td>
<td>4) Aberrant frenum/muscle position</td>
</tr>
<tr>
<td>3) pemphigus vulgaris</td>
<td>5) Decreased vestibular depth</td>
</tr>
<tr>
<td>4) erythema multiforme</td>
<td>6) Abnormal color</td>
</tr>
<tr>
<td>5) lupus erythematosus</td>
<td>D- Occlusal trauma: Primary occlusal trauma, Secondary occlusal trauma</td>
</tr>
<tr>
<td>6) drug-induced</td>
<td></td>
</tr>
<tr>
<td>7) other</td>
<td></td>
</tr>
<tr>
<td>b) allergic reactions</td>
<td></td>
</tr>
<tr>
<td>1) dental restorative materials: mercury, nickel, acrylic, other</td>
<td></td>
</tr>
<tr>
<td>2) reactions attributable to: toothpastes, mouthwash, chewing-gum additives, food additives</td>
<td></td>
</tr>
<tr>
<td>3) other</td>
<td></td>
</tr>
<tr>
<td>6 Traumatic lesions (factitious, iatrogenic, accidental)</td>
<td>chemical injury, physical injury, thermal injury</td>
</tr>
<tr>
<td>chemical injury, physical injury, thermal injury</td>
<td></td>
</tr>
<tr>
<td>7 Foreign body reactions</td>
<td></td>
</tr>
<tr>
<td>8 Not otherwise specified</td>
<td></td>
</tr>
<tr>
<td>II CHRONIC PERIODONTITIS</td>
<td></td>
</tr>
<tr>
<td>A localized, B generalized</td>
<td></td>
</tr>
<tr>
<td>III AGGRESSIVE PERIODONTITIS</td>
<td></td>
</tr>
<tr>
<td>A localized, B generalized</td>
<td></td>
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</tbody>
</table>
III. EPIDEMIOLOGY

When interpreting published data, it should be remembered that judgment criteria differ between studies. The increasingly systematic use of the CPITN index will make it possible to collect more uniform data in the future. Epidemiological data are limited to Europe.

Eighty percent (80%) of adults have gingivitis (grade C). Between 10 and 69% of the population studied have at least one loss of attachment $\geq 4$ mm. Between 1.6% (French data) and 40.1% (the former East Germany) of the population have a pocket depth $\geq 6$ mm. The critical age for tooth longevity in relation to periodontal destruction is currently about 60 years.

50% of adolescents aged 15 years have gingivitis and 50% of children have dental plaque; fewer than 30% of adolescents aged 15 have calculus. Depending upon the population, 1–9% of children aged 5–16 have loss of attachment and/or bone loss at one or more sites. This type of periodontal disease generally affects only a minority of the population, and in this case, in one or two sextants only. This prevalence has tended to remain stable or to improve as a result of improved general oral hygiene.

In view of the prevalence of periodontal disease and its potential seriousness, signs of periodontal disease should be routinely looked for during all dental check-ups.

IV. RISK FACTORS

It is difficult to interpret published data as the judgment criteria for periodontal disease vary between studies; they include loss of attachment, pocket probe depth, or CPITN. Studies are mainly case-control studies, which at best can report a significant association between a risk factor and periodontal disease. However, it appears to be possible to identify risk situations or factors which predispose to periodontal disease. These are:

— Bacterial flora

Bacterial flora have a specific role. The development of periodontal disease has been associated with the presence of various bacteria, and the formation of a biofilm by bacterial cooperation. The total number of bacteria is higher in periodontal disease, which suggests a causal relationship. The same bacteria may be found under different conditions in a healthy mouth and in adult periodontitis or aggressive periodontitis. The disease is characterised by an imbalance in the flora in favour of Gram- anaerobic strains, with a prevalence of certain organisms that is related to certain clinical characteristics of the disease. It is characterised by combinations of bacteria, and by possible transmission from mother to child or within a couple.

Interested readers may consult the AFSSAPS (French Agency for Health Product Safety) guidelines “Prescription of antibiotics in dentistry and stomatology”, which lists the bacteria found in periodontal disease.

— Hygiene

A significant relationship has been demonstrated between level of oral hygiene and periodontal status; the better the level of hygiene, the better the periodontal status. The
presence of deep pockets and loss of attachment is significantly related to the presence of
dental plaque (biofilm) and calculus. The need for more complex treatment is significantly
lower when hygiene is better (grade C).

Individuals who see their dental practitioner regularly have a significantly better level of
oral hygiene, less bleeding, fewer deep pockets and a lower level of need for complex
treatment (grade C).

— **Age**

Adults aged 16-24 have a significantly higher number of healthy sextants than adults aged
75 and over. Periodontal disease increases significantly with age (significant increase in
number of sextants affected, number of deep pockets, and loss of attachment and of bone).
Gingivitis during childhood is thought to predispose to the development of periodontal
disease (grade C).

— **Gender**

On average, men have significantly more plaque, gingivitis and periodontal pockets than
women do. In children and adolescents, boys have on average significantly more plaque,
bleeding and pockets than girls (grade C). This better periodontal status in girls is
significantly related to better oral hygiene.

— **Diabetes**

- Type 1 diabetics have significantly more gingivitis and significantly greater pocket depth,
  loss of attachment, and bone loss than non-diabetics (grade C). There are significantly
  more edentulous patients in this population (grade C).
- Type 2 diabetics have significantly more gingivitis and calculus, more periodontal pockets
  and more loss of attachment than non-diabetics (grade C).

— **HIV**

Male HIV-infected patients have been shown to have significantly more gingivitis and loss
of attachment and significantly deeper pocket depth than uninfected men (grade C). There
is an inverse relationship at the limit of significance (p<0.06) between CD4 level and
severity of attachment loss.

— **Pregnancy**

Although no studies of a sufficiently high level of evidence were found, acute episodes of
gingivitis and of periodontitis have been reported during pregnancy (agreement among
professionals).

— **Menopause**

At menopause, tooth loss is related to systemic bone loss (grade C). Women taking
hormone replacement therapy (HRT) have a lower risk of dental loss than women not
taking HRT (grade C). Before starting HRT at menopause in order to prevent tooth loss,
the anticipated benefit for the patient needs to be weighed against the risks relating to HRT
(agreement among professionals).

— **Lifestyle**

- *Smoking* is significantly associated with periodontitis (defined as loss of attachment)
  (grade C). The relative risk of periodontitis in a smoker increases with cigarette use and
length of time they have smoked compared with non-smokers (grade C). This risk reduces slightly when the subject gives up smoking (grade C).

- **Socio-economic level:** in northern European countries, where there are community education programmes to promote oral hygiene, studies have not found any significant difference in periodontal status related to socio-economic level (grade C). In France, it has been shown that treatment needs (CPITN) are significantly increased in lower socio-economic groups (grade C).

**Other factors predisposing to or aggravating periodontal disease**

There is insufficient scientific evidence to support a hypothesis of host influence on periodontal disease. However, the following local and general risk factors were assessed:

- **Local factors** are presence of caries, presence of calculus, dental morphology, and possible iatrogenic effect of dental treatment (restorations, dental appliances, orthodontic treatment). These local factors are likely to predispose to or aggravate periodontal disease, and should be corrected. To prevent iatrogenic effects, periodontal status should be assessed at the start of orthodontic treatment, and every three months during treatment. Scheduled assessment is recommended for the other local factors (agreement among professionals).

- **The general factors** identified are either inherent or acquired.
  - Inherent factors are age, gender, and genetic factors.
  - Acquired factors are immune deficiency, stress, nutritional factors such as vitamin C or calcium deficiency, alcohol consumption and drug use, and the use of certain medicines such as anti-cancer chemotherapy, calcium inhibitors, cyclosporin A, and phenytoin.

In practice (see guidelines “Patient records in dental practice” [French title: *Le dossier du patient en odontologie*], ANAES, 2000), it is recommended that when periodontal disease is discovered, a history should be taken to enquire into general disease, notably diabetes (type 1 or 2) or HIV seropositivity. A consultation with a doctor for diagnosis and/or treatment may then be advised. The history should record age, any family history of periodontitis, any current treatment, and lifestyle, i.e. smoking, oral hygiene level, socio-economic level, and in women, pregnancy or menopause.

- **Smokers** should always be advised to give up smoking; medical help may be offered.
- **Patients at risk of periodontal disease**, particularly diabetics (type 1 or 2), HIV-infected subjects, and menopausal women not taking HRT should have a routine dental and oral examination every six months (agreement among professionals). Checkup frequency depends on the individual patient and the level of periodontal disease.
- **Pregnant women** should undergo a routine clinical examination to look for signs of periodontal disease at the start of pregnancy, with a checkup six months later (agreement among professionals).
- **Patients in lower socio-economic groups** should be educated in oral hygiene during follow-up by practitioners. This may be reinforced if necessary by increasing the frequency of checkups (agreement among professionals).
- **As subjects get older**, monitoring should be stepped up, and children and adolescents with gingivitis or loss of attachment should be monitored at an increased intensity to prevent progression of periodontal disease (agreement among professionals).
V. **DIAGNOSIS**

V.1. **Clinical diagnosis**

A diagnosis of periodontal disease is initially suggested by the presence of clinical signs (redness, oedema or inflammation).

A periodontal clinical examination should assess the presence and quantity of bacterial plaque, look for bleeding on probing, measure pocket depth, clinical attachment level, assess tooth mobility and/or displacement, and any increase in local temperature. Bleeding on probing is regarded as an indicator of gingival inflammation. Absence of bleeding is a criterion of stabilisation in the course of the disease, except in smokers.

Gingivitis is diagnosed from clinical signs, e.g. redness, oedema, gingival hypertrophy or hyperplasia, and bleeding on probing without loss of attachment.

Pocket depth and attachment loss can be measured either with a graduated manual probe or with a pressure-controlled probe with visual control of probe depth, or with a pressure-controlled electronic probe with data recorded on computer.

Values measured have been found to be reproducible to the nearest mm in 85-98% of cases, depending on the study, and irrespective of type of probe (grade C). The same type of probe should be used for each series of measures, as values differ significantly depending on the type of probe used and the operator (grade C).

Periodontitis is diagnosed by the presence of attachment loss. This is a pathognomonic sign.

Tooth mobility should be looked for during the clinical examination. This may be measured by using subjective clinical indices, or by using the Periotest® measuring device (agreement among professionals). Measures obtained using a device differ significantly between examiners and between devices. It is therefore recommended that they should be performed by the same examiner, and using the same device (agreement among professionals).

During a periodontal examination, a periodontal chart should be added to the patient record to show loss of attachment and pocket depth. An index for gingival inflammation, bleeding on probing, mobility and plaque should be noted in the record (agreement among professionals).

V.2. **Radiological diagnosis**

Imaging will help to establish and confirm the diagnosis. In general, radiographic measurements underestimate the extent of bone loss. Film interpretation depends on the examiner's experience.

A full set of intraoral teleradiology radiographs is recommended for diagnosis and monitoring of periodontal disease when periodontal probing suggests bone loss (agreement among professionals).
− *A full intraoral teleradiology profile using periapical and bitewing views* is the gold standard method for visualising periodontal bone loss. The long cone paralleling method is preferable to the bissecting angle technique, which generally underestimates bone loss.

− *Digital radiography* has an efficacy similar to that of conventional radiography. Digital diagnostic aid systems do not appear to improve the quality of film interpretation significantly.

− *CT scanning* is more powerful than conventional radiography but is not recommended in current practice because it does not provide further significant information for deciding on treatment (agreement among professionals). If there is any doubt about the diagnosis, it may be offered as a second choice.

### V.3. Biological indicators

Determination of biological indicators of periodontal disease in the gingival fluid may be useful for diagnosis and prognosis, to identify those patients with periodontitis who will be resistant to treatment. However, no sufficiently sensitive and specific diagnostic tests have been developed yet for use in routine practice. They are still at the clinical research stage (agreement among professionals).

### V.4. Microbiological diagnosis

Microbiological diagnosis is based on three methods: bacteriology, immunology and molecular biology. These tests are not performed routinely to diagnose periodontal disease. Some may be used in aggressive periodontitis or periodontal disease which is resistant to treatment. Bacteriological testing, with sample culture and antibiotic sensitivity testing, is dependent on the availability of transport that will ensure the survival of anaerobic and capnophilic species, and a laboratory that can culture anaerobically (agreement among professionals).

### VI. TREATMENT

The aim of treatment is to prevent and control periodontal disease and to repair and/or regenerate the damaged periodontal tissue. The treatments available are non-surgical methods (supragingival calculus removal, scaling and root planing), drugs (antibiotics, antiseptics), and surgery.

In all cases, education in oral hygiene is an essential part of treatment. The practitioner should give patients information and teach them how to brush their teeth, encouraging them to brush their teeth regularly, ideally after every meal, and a minimum of twice a day (morning and evening). The use of dental floss and interdental brushes should be explained if necessary. Every follow-up visit or checkup should be used as an opportunity to reinforce oral hygiene education and to reinforce patient motivation. Smokers should always be encouraged to give up smoking.

### VI.1. Non-surgical treatment

− *Mechanical treatment*

Studies to evaluate the efficacy of scaling and root planing do not provide any level of evidence. The following guidelines are based on agreement among professionals.
Scaling followed by polishing is recommended to treat gingivitis.
Scaling and polishing and root planing are recommended to treat periodontal disease.
Scaling and root planing for interradicular lesions appear to work together to reduce pocket depth but seem to have no effect on level of attachment. Study results do not agree.

**Topical antiseptics**

Antiseptics for which data are available from randomised studies are sanguinarine and chlorhexidine.

- **Sanguinarine** administered on a biodegradable vector does not significantly improve clinical criteria (probing depth, improvement in attachment), compared with scaling and root planing (grade C).
- **Chlorhexidine** administered as a varnish seems to have no effect on probing depth, level of attachment, bleeding on probing and bacterial flora (grade C). Chlorhexidine administered on a biodegradable vector (not yet available in France) may be proposed in combination with scaling and root planing to treat deep pockets (> 5 mm) (grade B). Subgingival irrigation with antiseptic may be proposed in combination with scaling and root planing, although its efficacy has not been confirmed by any studies (agreement among professionals).

**Antibiotics**

- **Topical antibiotic therapy**: the benefit of controlled-release topical antibiotic therapy alone has not been confirmed in treatment for periodontitis (agreement among professionals). It may be proposed as an adjuvant to mechanical treatment (agreement among professionals). **Subgingival irrigation** with antibiotics is not recommended (agreement among professionals).
- **Systemic antibiotic therapy**: according to AFSSAPS, the indications for antibiotic therapy in curative treatment for periodontitis should depend on the subject's risk of infection. They have defined two groups of subjects (see Annex):
  1. subjects with no known risk of infection (subject regarded as healthy);
  2. subjects at risk of infection subdivided as follows:
     - group A with locally-identified risk of infection and/or systemic infection (septicaemia)
     - group B with a risk of infection related to a secondary localisation of bacteria, i.e. a new focus of infection remote from the primary focus (infective endocarditis, infection of a joint prosthesis).
Antibiotic therapy is recommended for group A and B subjects during treatment for periodontitis. Antibiotic therapy is not justified in healthy subjects with chronic gingivitis or a periodontal abscess; its benefit in chronic periodontitis has not been established. Antibiotics may be combined with mechanical treatment as a second-line therapy. The choice of antibiotic should also be guided by the clinical context and severity of infection, or adjusted if necessary according to antibiotic sensitivity test results.

VI.2. **Surgery**

Three forms of surgery are used: flap debridement, guided tissue regeneration, and bone grafting. These guidelines describe their efficacy in relation to the type of lesion to be treated, i.e. infrabony defects or surgical interradicular lesions.
Treatment of infrabony defects

- For lesions ≤ 6 mm, there are insufficient published data to conclude that there is any difference in efficacy between flap debridement and guided tissue regeneration methods, irrespective of the type of membrane. The guided tissue regeneration method uses a resorbable or non-resorbable membrane. The published data are insufficient to decide whether one type of membrane is safer than another. The practitioner should inform the patient that a second procedure will be required approximately 6 weeks after the first procedure to remove a non-resorbable membrane.

- For lesions ≥ 6 mm, bone grafts seem to be more effective than flap debridement methods alone in improving bone-related criteria (increased bone mass, level of alveolar bone). Conflicting results have been reported for pocket depth and improvement in attachment (grade C). Filling methods may be proposed (agreement among professionals); autogenous bone is the first choice for filling material (agreement among professionals).

Treatment of interradicular lesions

In the surgical treatment of class II interradicular lesions, flap debridement methods, guided tissue regeneration methods (with or without resorbable membrane) and filling methods significantly improve probing depth (grade C) and level of attachment (grade C). One of these methods may be proposed to treat class II interradicular lesions (agreement among professionals).

Adjuvant treatment with surgery

- Biological glue. Published data are inadequate to conclude that there is any benefit in using biological glue combined with guided tissue regeneration methods. Its use was not recommended (agreement among professionals).

- Growth factors. Their benefit in treating periodontal disease is still under clinical investigation. In a phase I/II study, they did not seem to lead to antibody formation. Further studies are needed to establish their safety and efficacy.

- Embryonic enamel matrix derivative. Published data do not make it possible to conclude on any benefit from the use of embryonic enamel matrix derivative of animal origin in combination with surgical methods. Further studies are needed.

VII. PERIODONTAL DISEASE AS A RISK FACTOR FOR OTHER DISEASES OR SITUATIONS

The epidemiological studies identified found a relationship between periodontal disease and infective endocarditis, coronary disease, stroke, premature labour, lung infection and sinus infection.

VII.1. Periodontal disease and infective endocarditis

No specific studies have been carried out on the prevalence and incidence of infective endocarditis in subjects with periodontal disease. However, in view of the potentially lethal nature of infective endocarditis and the infectious nature of periodontal disease, prophylaxis for infective endocarditis needs to be recommended in subjects with periodontal disease who are known to have a heart condition at risk of endocarditis (see
Annex). Such prophylaxis consists of treating the periodontal disease in order to eradicate foci of infection, and giving antibiotic prophylaxis before certain treatment procedures. Treatment is based on agreement among professionals:

- **Heart conditions at high risk:**
  Loose teeth associated with periodontitis and teeth with interradicular lesions should be extracted. Periodontal surgery is not recommended.

- **Heart conditions at lesser risk:**
  Conservative treatment may be proposed for mild to moderate periodontal disease. For treatment of more serious periodontal disease, if the risk of endocarditis inherent in the treatment procedure is known, the approach adopted to date has been to perform dental extractions and avoid periodontal surgery if possible. The working group felt that this approach could be modified slightly, and that treatment should be decided in close consultation with the cardiologist treating the patient, and according to the patient’s response to treatment.

On the subject of antibiotic prophylaxis, interested readers may consult the AFSSAPS guidelines “Prescription des antibiotiques en odontologie et stomatologie” (in French only) and the revised guidelines “Prophylaxis of infective endocarditis” (“Prophylaxie de l’endocardite infectieuse”), published in June 2002.

**VII.2. Other situations**

There are insufficient data to reach any definite conclusion on the existence of a causal relationship between periodontal disease and coronary disease, fatal or non-fatal stroke, premature labour, diabetes, certain lung diseases and sinus infection. Nevertheless, the working group recommended special clinical monitoring for pregnant women (agreement among professionals). If periodontitis is discovered it should be treated, and obstetric monitoring should be increased as periodontal disease seems to be significantly associated with a risk of prematurity and low birthweight (grade C). In patients with stroke, diabetes, coronary disease, lung disease or sinusitis, the oral cavity should be examined by the clinical practitioner to look for signs of periodontitis and if there is any doubt at all, a specialist opinion should be sought (agreement among professionals).

**VIII. TREATMENT STRATEGY**

The treatment strategy is summarised in the form of a decision tree shown in Fig. 1.

**IX. PROPOSALS FOR FUTURE ACTION**

The working group encountered major problems in producing these guidelines because of the lack of any level of evidence in the literature in this area.

- The relative efficacy of treatments could not be assessed because of the lack of similar and comparable judgment criteria. The working group proposed that a chart be produced describing the judgment criteria to be used. The most important criterion is attachment level.
- The working group felt that more research is needed to allow a better assessment of the risk of infective endocarditis following oral or dental procedures. A knowledge of this risk in relation to the procedure in question (tooth extraction, scaling, periodontal
surgery) would make it possible to adjust prophylaxis and possibly to avoid systematic multiple tooth extractions in at-risk patients.

- More generally, there is a need for properly designed studies of risk factors and of the impact of periodontal disease on general health.
- A new working group should study the issue of prevention.
Periodontal disease: diagnosis and treatment

**Aggressive periodontal disease**

- Systemic antibiotic therapy
- Mechanical treatment: pocket < 4 mm: scaling, polishing / pocket > 4 mm: scaling, root planing, polishing
- Reassessment after 6-8 weeks

**Chronic periodontitis**

- General deterioration
- Local deterioration
- Improvement, with persistence of bleeding and localised pocket

**Improvement, with persistence of bleeding and localised pocket**

- Root planing
- Reassessment after 6-8 weeks

**No improvement + no further bone loss visible on radiograph**

- Flap debridement
- Maintenance every 3 weeks

**No improvement + bone loss visible on radiograph > 6 mm**

- Surgery: Flap debridement / flap + filling / guided tissue regeneration
- Maintenance every 3 weeks

**Improvement (pocket < 4 mm, no bleeding)**

- Maintenance every 6 months

**General deterioration**

- Local deterioration
- Improvement, with persistence of bleeding and localised pocket

**Improvement (pocket < 4 mm, no bleeding)**

- Maintenance every 3 months

**Local deterioration**

- Improvement, with persistence of bleeding and localised pocket

**Medical opinion**

- No improvement + no further bone loss visible on radiograph
- Flap debridement
- Maintenance every 3 weeks

**No improvement + bone loss visible on radiograph > 6 mm**

- Surgery: Flap debridement / flap + filling / guided tissue regeneration
- Maintenance every 3 weeks

**Improvement (pocket < 4 mm, no bleeding)**

- Maintenance every 6 months

**Stabilisation or improvement: maintenance every 3 months, tailored to individual patient**

**Deterioration: consider tooth extraction(s) or palliative maintenance**

Fig. 1: Strategy for treating periodontitis
ANNEX

- **Subjects with no known risk of infection (subjects classed as healthy)**
  These are subjects presumed to be healthy, with no particular risk factors or risk situations, subjects with heart conditions not at risk of infective endocarditis (atrial septal defect, mitral valve with thin valves and no murmur, functional murmur, patients with pacemakers, patients with implantable defibrillators, patients who have undergone coronary bypass surgery, or left-to-right shunt with no residual shunt (more than 6 months previously), mitral annular calcifications, ischaemic heart disease, hypertensive heart disease, dilated cardiomyopathy, Kawasaki disease without valvular dysfunction, rheumatoid arthritis without valvular dysfunction, interventional cardiology (percutaneous valvuloplasty, coronary stent placement, etc.), peripheral vascular disorders).

- **Subjects at risk of infection.** These are divided into two types of subjects, with an A or B risk:
  - **Type A risk** corresponds to risk of locally-identified infection and/or of systemic infection (septicaemia). Risk factors are: transplant or graft under immunosuppressant drug cover, except for patients on cyclosporin alone who have a lower risk of infection; immune deficiency: congenital, induced by long-term drug use (corticosteroids, chemotherapy, etc.), infection-related (HIV, etc.), immunological (lupus erythematosus, systemic disease, etc.); chronic uncontrolled disease: diabetes, impaired renal and/all hepatic function, subjects with chronic bacterial or fungal infection, etc; malnutrition: bedridden subjects, dehydrated subjects, socially deprived subjects, drug addicts, alcohol-dependent subjects.
  - **Type B risk** corresponds to a risk of infection by bacteria in a secondary location, i.e. a new focus of infection some way away from the primary focus (infective endocarditis, infection of a replacement joint). These are the subjects at risk of infective endocarditis. **AFSSAPS** and the Consensus Conference held in 2002 proposed a classification of heart conditions related to risk of endocarditis (see Table 2) and certain subjects with a risk of infection of joint prostheses.

**Table 2. Heart conditions at risk of infective endocarditis**

<table>
<thead>
<tr>
<th>High-risk heart conditions</th>
<th>Low-risk heart conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Artificial valves (mechanical, homograft, bioprosthesis)</td>
<td>Other congenital heart disease (non-cyanotic heart disease other than atrial septal defect)</td>
</tr>
<tr>
<td>History of infective endocarditis</td>
<td>Valvular disease: insufficiency, thickening, and aortic bicuspid valve, mitral insufficiency</td>
</tr>
<tr>
<td>Cyanotic congenital heart disease not treated surgically</td>
<td>Acquired valvular dysfunction</td>
</tr>
<tr>
<td>Surgical shunt (pulmonary-systemic)</td>
<td>Mitral valve prolapse with mitral insufficiency and/or valve thickening</td>
</tr>
</tbody>
</table>

Obstructive hypertrophic cardiomyopathy