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The acute apical abscess: Aetiology, microbiology, treatment and prognosis

Key words antibiotics, apical abscess, drainage, incision, microbiology

This review aims to summarise the current knowledge and recommendations regarding the diagnosis and treatment of the acute apical abscess in conjunction with the understanding of the microbiological background of this inflammation, thus providing an actual insight for the clinician.

Introduction

Dental clinicians can encounter situations in which proper and fast diagnosis and treatment are required. The acute apical abscess is such a common emergency in the dental office. Because of the rapid and often sudden progression of this inflammation, the patient will contact the dental office with pain and various symptoms relating to the acute disease. The patient requests the clinician’s immediate help to relieve him or her of the pain and the discomfort of associated symptoms and an emergency appointment has to be made. In this situation, it is of utmost importance that the clinician will make the correct diagnosis in a short time and start the treatment necessary to help the patient. A correct and timely treatment is also important to prevent the inflammation from spreading throughout the body and causing further severe infections. To make the correct diagnosis and to choose the correct treatment strategy, an up-to-date knowledge of the acute apical abscess is both necessary and advisable.

Antibiotic resistance is a natural phenomenon of which the public has become more aware in recent years and which is seen as a worldwide concern. The World Health Organization (WHO), the American Association of Endodontists (AAE) and the European Society of Endodontology (ESE) just recently expressed their concerns about this by organising awareness campaigns1-4.

Dental practitioners frequently use antibiotics as a therapeutic measure for treatment of acute abscesses4. The use of antibiotics should be kept to a minimum to prevent overuse and misuse. Acknowledging the risk of antibiotic resistance also means knowing when antibiotics should, could, or have to be used when an acute apical abscess has been diagnosed.

In summary, it is the aim of this review to describe the current knowledge and recommendations regarding the diagnosis and treatment strategies for the acute apical abscess, including lingering controversies on surgical or non-surgical drainage, closing or leaving the tooth open, and the need of systemic antibiotic medication.

Terminology and classification

The WHO has developed a diagnostic tool for epidemiology, health management and clinical purposes: the International Classification of Diseases (ICD). The ICD-10 is the most current version and in 2018 there will be a revision, the ICD-11. The periapical abscess, also referred to by the WHO as the dental abscess and the dentoalveolar abscess, are classified as an abscess with sinus tract – K04.6 – or without sinus tract – K04.7.
As for the terminology of the acute apical abscess, the American Dental Association (ADA)\(^5\) and the American Association for Endodontists (AAE)\(^6\) first define the word “abscess”. The word derives from the Latin word “ascendere” (to go away, to depart, to withdraw). The ADA currently defines an abscess as “acute or chronic localised inflammation, probably with a collection of pus, associated with tissue destruction and, frequently, swelling; usually secondary to infection”\(^5\). The AAE describes it as “a localised collection of pus within a tissue or a confined space”\(^6\).

Both agree on the fact that pus is formed; the difference in definition is that the ADA assumes that pus can be present, whereas the AAE states that pus is present. The ADA and AAE complete the definition by dividing the abscess into two categories, the acute and the chronic abscess.

The first category is the acute periradicular or acute apical abscess and both state: “An inflammatory reaction to pulpal infection and necrosis characterised by rapid onset, spontaneous pain, tenderness of the tooth to pressure, pus formation and eventual swelling of associated tissues”\(^5,6\). They list the following synonyms for this type of abscess: acute periapical abscess, acute alveolar abscess, dentoalveolar abscess, phoenix abscess, recrudescent abscess and secondary apical abscess.

The second category is the chronic periradicular or chronic periapical abscess and both societies agree on the terminology: “An inflammatory reaction to pulpal infection and necrosis characterised by gradual onset, little or no discomfort and the intermittent discharge of pus through an associated sinus tract”. The synonyms are chronic alveolar abscess, chronic dentoalveolar abscess, and suppurative apical periodontitis.

In 2008, a conference on Diagnostic Terminology was held by the AAE. The main objective was to find consent in the associated terminology regarding endodontic diagnosis. Conclusions were made at the conference and an online survey study was started by Glickman et al\(^7\) with 36 questions filled in by 64 conference attendees. Consensus was achieved if a concept was supported or not supported by 51%. The agreement regarding the terminology of the acute apical abscess was very strong; 83% agreed and 17% disagreed. The concept was: “Acute apical abscess is the preferred diagnostic term for an inflammatory reaction to pulpal infection and necrosis characterised by rapid onset, spontaneous pain, tenderness to pressure, pus formation, and swelling”\(^7\).

### Aetiology

Apical periodontitis is an infection at the apical part of a root, which can cause an inflammation of the adjacent tissues in that specific area\(^8-11\). The host will react to the infection with an inflammation that can develop into an acute apical abscess. The origin of this inflammation mainly is an infection inside the root\(^11-13\). The inflammatory response may also be caused by a chronic asymptomatic situation, which has now led to an acute reaction because of altered severity\(^14\). As a result of the inflammatory reaction, pus is formed and found inside the abscess\(^14,15\).

Treatment of the acute apical abscess at an early stage may help to prevent the spread of the inflammation to other regions of the head and neck. Not only can these odontogenic infections induce complications in narrow and distant tissues and spaces, in extreme cases the inflammation has even been reported to lead to death, caused by a sepsis or an airway obstruction\(^15-17\). Other sequelae reported as resulting from odontogenic infections are mediastinitis, fatal necrotising fasciitis and a brain abscess\(^18-21\).

It is therefore important to recognise the possibility of these critical situations and to perform treatment of the acute apical abscess as early and thoroughly as possible in order to prevent these sequelae.

### Microbiology

Understanding the microbiology of the inflammation is mandatory to develop a proper treatment plan. A frequently used method in studies for detecting bacteria has been the culture technique, whereas more recent studies use a molecular approach. Anaerobic bacteria from an acute abscess are not only a challenge to cultivate, but also need a specific environment in which to survive.

Nowadays, microbiologic research is performed using the molecular approach, as these techniques are so sensitive that they can detect certain species...
that are present only in very low quantities, but nevertheless may contribute to the microbiota and environmental circumstances of the acute apical abscess.

Associations between bacteria from infected root canals and the acute apical abscess have been demonstrated in a study by Nóbrega et al. It has already been recognised by earlier research that dento-alveolar abscesses contain a mixed flora of facultative and strictly anaerobic organisms. It is a commonly stated fact that the infection is a polymicrobial infection and that the cause cannot be related to a single microorganism. Furthermore, the apical abscess can be described as a process, which means that there is a change in the microbiota causing this acute reaction. In the oral samples, Streptococcus was the most abundant species; inside root canals it became Prevotella and Fusobacterium and in the abscess samples Fusobacterium was the most prevalent genus. The conclusion that can be made is that a shift has taken place in the microbiota, and that certain species are present in different sites of infections. This shift can then lead to the formation of an acute apical abscess. Montagner et al. were interested to see if associations could be found between bacterial communities in root canals and the acute apical abscess. They also used a molecular technique (open-ended) and took paired samples of root canals and the periapical abscesses. The analysis was done by terminal restriction fragment length polymorphism (t-RFLP) and showed that no single t-RF fragment was detected in all samples and that most of the t-RG fragments were detected in one sample only. Based on these findings, they concluded that the microbial communities found in root canals and in the acute apical abscess are different and that a shift takes place in the community.

Comparing the microbiota from the molecular studies (Table 1), it is obvious that most of the bacteria in the acute apical abscess are anaerobic and that the most abundant species differ from study to study.

Treponema species play an important role in the pathogenesis of endodontic infections and, in particular, for the acute apical abscess. The most prevalent species found in the acute apical abscess by culture and molecular approaches (Table 1) are Bacteroidetes, Fusobacteria, Firmicutes, Actinobacteria, Spirochaetes, Synergistetes and Proteobacteria. In abscesses, Firmicutes and Bacteroidetes are the most abundant phyla and combined they cover more than 70% of the total species.

### Clinics

The most common symptoms of an acute apical abscess are (spontaneous) pain, induced pain when pressure is applied on the tooth and swelling of the surrounding tissues. Trismus can also occur and other symptoms, such as fever, lymphadenopathy, malaise, headache and nausea, have also been reported.

### Diagnosis

As it is the result of an infection and inflammation, the acute apical abscess is associated with certain symptoms from the tetrad from Celsius: calor (warmth), dolor (pain), tumor (swelling), and rubor (redness and hyperaemia).

#### Visual inspection

Diagnosis starts with a thorough anamnesis, with a focus on the systemic health of the patient. This is followed by an examination of the face and neck area. Swelling of the face and/or the neck, as well as intraoral swelling, frequently occur as symptoms of an apical abscess. Surface temperature can be warm and the swelling can be localised or diffuse. Mouth opening may be limited, also known as a trismus due to the swelling, blocking the jaws from closing. It is important to observe if the patient’s airway is obstructed due to a swelling and if breathing is compromised.

#### Palpation, percussion and tooth involvement

Palpation of the inflamed area is sensitive and the patient can find percussion of the infected tooth extremely painful. The patient usually reports severe pain during chewing or any contact with the antagonist teeth. Due to the fact that necrosis of the pulp has taken place, a sensibility
### Table 1  Microbiological findings for acute apical abscesses. Modified after Siqueira et al (2009)\(^22\).

<table>
<thead>
<tr>
<th>Publication</th>
<th>n</th>
<th>Technique</th>
<th>Most prevalent species (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>2016, Nobrega et al(^23)</td>
<td>20</td>
<td></td>
<td>Prevotella spp., Pseudoramibacter alactolyticus, Parvimonas micra, Dialister invisus, Filifactor alocis, and Peptostreptococcus stomatis</td>
</tr>
<tr>
<td>2016, George et al(^34)</td>
<td>18</td>
<td>Polymerase chain reaction Amplification using 16S rRNA gene-specific primers and Cy3-dCTP labelling</td>
<td>Fusobacterium nucleatum, Parvimonas micra, Megasphaera species clone CS025, Prevotella multisaccharivorax, Atopobium, Porphyromonas endodontalis</td>
</tr>
<tr>
<td>2012, Montagner et al(^33)</td>
<td>20</td>
<td>PCR</td>
<td>P. endodontalis, P. nigrescens, F. alocis, P. tannerae, and T. forsythia (F. nucleatum was not present in any AAA sample)</td>
</tr>
<tr>
<td>2012, Flynn et al(^21)</td>
<td>9</td>
<td>PCR cloning and sequencing</td>
<td>Fusobacterium spp, Parvimonas micra, Porphyromonas endodontalis, and Prevotella oris, Dialister pneumosintes and Eubacterium brachy.</td>
</tr>
<tr>
<td>2011, Santos et al(^13)</td>
<td>9</td>
<td>Pyrosequencing</td>
<td>Firmicutes (52%), Fusobacteria (17%) and Bacteroidetes (13%), Fusobacterium (17%), Parvimonas (11%) and Peptostreptococcus (13%)</td>
</tr>
<tr>
<td>2010, Montagner et al(^32)</td>
<td>20</td>
<td>Nested PCR</td>
<td>Special Treponema, T. socranskii (RC, 17/20; AAA, 15/20), T. denticala (RC, 8/20; AAA, 1/10); T. Medium (RC, 6/20; AAA, 9/20); and T. amylovorum (RC, 5/20; AAA, 9/20).</td>
</tr>
<tr>
<td>2010, Siqueira et al(^22)</td>
<td>42</td>
<td>Reverse capture checkerboard</td>
<td>Fusobacterium nucleatum (64), Parvimonas micra (52), Porphyromonas endodontalis (48), Olsenella uli (45), Streptococcus spp. (38), Eikenella corrodens (38), Bacteroides clone X083 (36), Prevotella baroniae (36), Treponema denticola (36)</td>
</tr>
<tr>
<td>2009, Sakamoto et al(^37)</td>
<td>7</td>
<td>T-RFLP; PCR, cloning, and sequencing, symptomatic</td>
<td>(57), Lachnospiraceae clone 55A-34 (57), Prevotella intermedia (43), Prevotella baroniae (43), Dialister pneumosintes (43), Eubacterium clone BPI-89 (43), Lachnospiraceae clone MCE7_60</td>
</tr>
<tr>
<td>2002, Khemaleelakul et al(^28)</td>
<td>17</td>
<td>Culture</td>
<td>Prevotella and Peptostreptococcus (57)</td>
</tr>
<tr>
<td>2001, Siqueira et al(^38)</td>
<td>27</td>
<td>Conventional checkerboard</td>
<td>Bacteroides forsythus (29.6% of the cases); Porphyromonas gingivalis (29.6%); Streptococcus constellatus (25.9%), Prevotella intermedia (22.2%), Prevotella nigrescens (22.2%), Fusobacterium periodonticum (18.5%), Fusobacterium nucleatum subspecies nucleatum (18.5%), and Eikenella corrodens (18.5%)</td>
</tr>
<tr>
<td>2010, Siqueira et al(^39)</td>
<td>22</td>
<td>Nested PCR</td>
<td>Treponema denticola (77), Porphyromonas endodontalis (68), Dialister pneumosintes (64), Tannerella forsythia (64), Porphyromonas gingivalis (59), Dialister invisus (53), Filifactor alocis (42), Fusobacterium nucleatum (41), Streptococcus spp. (41)</td>
</tr>
<tr>
<td>1998, Sakamoto et al(^40)</td>
<td>23</td>
<td>Culture</td>
<td>Peptostreptococcus spp. (52), Fusobacterium spp. (43), Prevotella oris (39), Streptococcus constellatus (35), Streptococcus intermedium (35)</td>
</tr>
<tr>
<td>1996, Kulekçi et al(^41)</td>
<td>13</td>
<td>Culture</td>
<td>Peptostreptococcus spp. (92), alpha-hemolytic streptococci (69), Prevotella intermedia/nigrescens (69), Fusobacterium nucleatum (38)</td>
</tr>
<tr>
<td>1991, Brook et al(^42)</td>
<td>32</td>
<td>Culture</td>
<td>Alpha-hemolytic streptococci (34), Porphyromonas gingivalis (22), Parvimonas micra (19), Fusobacterium nucleatum (16)</td>
</tr>
<tr>
<td>1989, Sundqvist et al(^24)</td>
<td>17</td>
<td>Culture</td>
<td>Fusobacterium nucleatum (71), Lactobacillus spp. (65), Prevotella intermedia/nigrescens (59), Parvimonas micra (53), Peptostreptococcus anaerobius (53), Eggerthella lenta (47)</td>
</tr>
<tr>
<td>1985, Heimdahl et al(^43)</td>
<td>58</td>
<td>Culture</td>
<td>Fusobacterium nucleatum (45), “Streptococcus milleri” (31), Parvimonas micra (29), Prevotella ruminicola (29), Prevotella melanogenica (26)</td>
</tr>
<tr>
<td>1983, Williams et al(^44)</td>
<td>58</td>
<td>Culture</td>
<td>Fusobacterium nucleatum (60), Bacteroides spp. (60), Parvimonas micra (50)</td>
</tr>
<tr>
<td>1976, Sabiston et al(^45)</td>
<td>58</td>
<td>Culture</td>
<td>Fusobacterium nucleatum (71), Streptococcus spp. (71), Parvimonas micra (22), Actinomyces spp. (17)</td>
</tr>
</tbody>
</table>
test – e.g. with CO₂ snow – will give a negative response. The tooth can also show increased mobility and elevation.

### Radiographic diagnosis

Dental radiographs are mandatory for the final diagnosis. In the presence of an acute apical abscess radiographically, a radiolucency can be seen around the apical part of the involved tooth (Fig 1). This lesion may vary considerably in size, and can be very small or even completely missing (Fig 2). If a radiolucency can be detected, the acute apical abscess derives from exacerbation of a previous chronic asymptomatic situation. What can also be observed radiographically is that the involved tooth usually shows a deep carious lesion, a previous restorative or endodontic treatment or a fracture (Figs 3 and 4).

Radiographically, the acute form can initially present without visible periradicular bone loss at the apical part of the infected tooth. This is due to the fact that the inflammation process develops very quickly and will be visible only when cortical bone is involved.
Whether CBCT can also be used as a helpful tool in diagnosing the acute apical abscess has been investigated by Abella et al.\textsuperscript{51} on 161 teeth with 340 roots. Sixteen teeth (42 roots) were diagnosed as having an acute apical abscess. Teeth with an acute apical abscess were significantly more frequently observed in the CBCT than in periapical radiographs. In radiographs, 15.1% of periapical lesions could be seen at the roots with an acute apical abscess, compared with 48.2% lesions in the CBCT. Nevertheless, this does not justify routine CBCT diagnosis in cases of acute apical abscesses.

**Treatment of the acute apical abscess**

Following final diagnosis a treatment plan can be made. Therapy should start by removing the necrotic pulp tissue and achieving drainage for evacuation of the pus. Pus is defined by the AEE\textsuperscript{5} as “an inflammatory exudate fluid product of inflammation containing leukocytes and the debris of dead cells and tissue elements liquefied by enzymes elaborated by polymorphonuclear leukocytes”. Because neutrophils are part of our innate immune system, their presence is abundant and their capability to phagocyte cells plays an essential role in our host defence mechanism\textsuperscript{52}.

Ricucci et al.\textsuperscript{53} investigated 50 extracted teeth and found inflammatory cells in all lesions classified as abscesses. They described a dense accumulation of polymorphonuclear leukocytes (PMNs) with lymphocytes, macrophages and plasma cells.

Removal of the pus can be achieved in different ways\textsuperscript{14,48,54}. If a swelling is present, pus can be removed by an incision for drainage (Fig 5). In some cases the incision should be kept open using a small plastic drain (Fig 6). Another way to drain pus is through the root canal of the involved tooth, or a combination of both techniques. The clinician should first investigate if drainage through the root canal can be achieved or if it is obstructed by either a previous root canal filling or other restorative materials thus preventing drainage. Slight over-instrumentation to achieve apical patency may be helpful in such cases to achieve drainage through the root canal (Fig 7), which in many cases will result in spontaneous relief of pain.

Cleaning of the infected root canal and the removal of intracanal microorganisms are essential for healing. Starting conventional root canal treatment does not only remove the necrotic tissue and bacteria from inside the root canal, but also creates space for the drainage of the pus. Exact determination of endodontic working length using an electronic device does not seem to be affected by pus inside
the root canal. Preparation with larger instrument sizes seems to be recommended, although no clinical evidence exists. This also holds true for the disinfection protocol. After cleaning, an intracanal antimicrobial dressing, such as calcium hydroxide, should be placed. These initial steps are commonly accepted for emergency treatment of the acute apical abscess, as described in the literature.4,15,55,6,5,57,58,59.

There has been, and still is, some controversy as to whether or not to leave the tooth open for drainage.60 Leaving the tooth open presents an opportunity for microorganisms from the oral cavity to re-infect or colonise the root canal. A study from Tjäderhane et al.61 demonstrated that leaving the tooth open will result in a higher number of intracanal bacteria. Siren et al.62 also detected a higher number of enteric bacteria when root canals were unsealed at some point during treatment. A reduction in exacerbations could be seen when drainage, cleaning, placing an intracanal antimicrobial dressing, and a temporary seal were performed on the first visit.63-67. By contrast, Tjäderhane et al. 61 (1995) reported that leaving the tooth open for drainage had no effect on the incidence of flare-ups. Leaving the tooth open requires more apical surgery65 and more appointments.66 On very rare occasions the draining of pus or exudate from the root canal cannot be controlled. In this situation leaving the tooth open may be an option and if so, it is recommend seeing the patient within 24 h for further treatment and re-evaluation.30,68.

In conclusion, leaving the tooth open for drainage is not recommended and temporary coronal sealing the tooth seems to be favourable.62,64,65,66,67,68.

Antibiotics

The global concern about the use of antibiotics in general is expressed in numerous fact sheets published by the WHO (2016), warning that the overuse and misuse of antibiotics will lead to antibiotic resistance. Therefore, a clinician must make a careful decision whether to prescribe antibiotics or not, and not to overprescribe.4,48,69,70,71. In 1990, a survey among diplomats of the American Board of Endodontics demonstrated an increase in the use of antibiotics. Antibiotics were prescribed in 3.5% to 13.7%72 of vital pulp cases. The behaviour of prescribing changed when the pulp was non-vital and without swelling, an increase was seen up to 60.5%. The highest numbers were found when the pulp was non-vital, with the presence of a swelling: 60.5% to 88.2% of the responding endodontic specialists in these cases would prescribe antibiotics. Studies have shown that bacteria that are present within the acute apical abscess are susceptible for antibiotics like penicillin.28,73,74,75. Fouad et al. investigated if penicillin supplementation had an effect on reducing the symptoms and the course of recovery of the localised acute apical abscess after emergency treatment.57 The authors stated that penicillin had no significant effect as supplementation for treatment of the acute apical abscess, when using the described endodontic protocol. Henry et al. also looked at the effect of penicillin and observed the symptoms, swelling and postoperative pain of 41 patients (41 teeth).58 In their prospective, randomised, double-blind and placebo-controlled study, no significant difference in pain reduction and swelling was detected whether or not penicillin was given. Their results corresponded with the results from Fouad et al.57.

In a recent review, Cope et al. looked at the effect of systemic antibiotics for treatment of acute apical abscesses.51 In their Cochrane-based review they came to the conclusion that the available literature to date is of very low quality, with no evidence concerning the efficacy of antibiotics for this purpose. The question remains, however, why no effect took place, especially because it is known that the bacteria involved are susceptible to penicillin. Fouad et al. explained that an antibiotic can only work if it can infiltrate the inflamed tissue and reach the infected area.57. Secondly, even if it is able to arrive at the inflammation site, there must be a sufficiently high concentration of the antibiotic to be effective. Fouad et al. theorise that because of the presence of necrotic tissue within the apical abscess, the blood supply will be limited and as a result the efficacy of the antibiotic is reduced. The limited blood supply making antibiotics less effective was also mentioned by König et al.76. It was demonstrated that an antibi-otic could only be effective if it had been administered in advance, so that the antibiotic is present while bacteria are infiltrating the tissues. This means that when the periapical abscess has already been
established, the antibiotics have lost their efficacy against the microbiota\textsuperscript{58,77,78,79}.

The available literature supports the use of antibiotics when the patient shows systemic complications, such as fever, cellulitis, lymphadenopathy, dysphagia, closure of the eye, facial swelling and/or trismus and for abscesses in patients that are immunocompromised\textsuperscript{14,54,80}.

The main reason to prescribe antibiotics for these groups is because there is a higher risk of a secondary infection, which can lead to spreading bacteria into the blood circulation. In 2017, Segura-Egea et al additionally suggested that antibiotics should also be considered in certain patients with a localised congenital or acquired altered defence capacity; this includes patients with infective endocarditis, prosthetic cardiac valves, or with recent prosthetic joint replacement, but at the same time Segura-Egea et al recognised that there is little evidence to use antibiotics for these situations in general\textsuperscript{4}. This recommendation can also be found in the previous literature\textsuperscript{14,51,54,80}.

The position statement of the AAE is very similar to that of the ESE\textsuperscript{2,81}. No statements were given by the AAE regarding osteomyelitis, prosthetic joint replacement, trismus and the predisposing conditions regarding endocarditis. The ESE also presents a more detailed and elaborated definition of the predisposing condition (risk of developing infective endocarditis and patients with complex congenital heart effects, prosthetic cardiac valve or a history of infective endocarditis) and the impaired immunologic function (leukaemia, HIV/AIDS, end-stage renal disease, dialysis, uncontrolled diabetes, chemotherapy, steroids or immunosuppressive post-transplant medications or inherited genetic defects). In conclusion, a consensus can be seen between the ESE and the AAE regarding the use of antibiotics as an adjunctive to endodontic treatment\textsuperscript{2,81}.

\section*{Discussion}

\subsection*{Microbiology}

As commonly stated, the acute apical abscess is regarded as a polymicrobial infection and its development cannot be related to a single specific microorganism\textsuperscript{23,28,30,31,32,33,86}. The introduction of new culture techniques has shed light upon the microbiota of the acute apical abscess. The purpose of finding new species with molecular techniques that could be related to the acute apical abscess should be advised, but one must be careful not to immediately correlate the presence of a new species automatically with a pathogenic role in the microbiota of the acute apical abscess. It is therefore advisable that new discovered species should be monitored over a longer period of time to define their role in the microbiota of apical abscesses\textsuperscript{15,22,46}.

\subsection*{Radiographic diagnosis}

Whether a CBCT is an additional tool, or even a replacement of the conventional radiograph when diagnosing an acute apical abscess, remains questionable. Although the first findings\textsuperscript{51} showed that a lesion could be detected with CBCT in 48.2\% compared with 15.1\% with conventional radiographs; this is currently the only study specifically diagnosing the acute apical abscess with CBCT. Therefore, more studies have to be performed to give more power to the overall findings. Furthermore, a position statement of the ESE regarding the use of CBCT in endodontics states that the use of CBCT with a limited field of view (FOV) for the diagnosis of radiographic signs of periapical pathosis is only justified when
there are contradictory (nonspecific) signs and/or symptoms.

### Treatment of the acute apical abscess

Removal of the necrotic pulp tissue and pus are essential factors to promote healing. In the presence of a swelling, the removal of pus can be achieved by incision or drainage through the tooth. Knowing that besides the pus the necrotic pulp tissue must also be removed, it could be advisable to start root canal treatment immediately. In this way, both factors are addressed and no incision may be required. Concerning the use of antibiotics respective studies should be evaluated with care. The studies from Fouad et al.\(^5\) and Henry et al.\(^6\) did not show significant differences between treatment with or without penicillin as an adjunct. In a systematic review, Cope et al. evaluated the literature on effects of systemic antibiotics.\(^1\) Only two studies out of 36 articles met the selection criteria; these where randomised controlled trials (RCTs) with a parallel group design. Cope et al. concluded that there is a very low level of evidence, therefore not allowing determining the effects of systemic antibiotics on adults with symptomatic apical periodontitis or an acute apical abscess.\(^5\) Currently the AAE and the ESE seem to have found an agreement regarding the use of antibiotics as an adjunctive to endodontic treatment (Table 2).\(^2,8\)

### References


31. Oei and Hülsmann. The acute apical abscess


