The Lower Jaw (Mandible) and Associated Structures: Part II

22

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22.1 Swellings and Infections of the Lower Jaw

Infective swellings in and around the lower jaw are usually due to a localised or spreading bacterial infection within the bone or fascial spaces. These can be considered as:

1. Acute infections. (a) Involving the soft tissues, (e.g. submandibular abscess), (b) involving bone (e.g. osteomyelitis) and (c) occurring in both.
2. Chronic infections of the mandible. (a) localised e.g. local necrosis; (b) diffuse, e.g. chronic osteomyelitis; (c) involving the temporomandibular joint.
3. Infections arising in a cyst
4. Infections occurring in pre-existing bone disease.
5. Specific infections e.g. actinomycosis.
6. Infections related to malignant disease, e.g. post-radiation necrosis.

In most cases the underlying cause is a dental infection usually involving the posterior teeth. However, it is important to exclude other possible sources. Less common causes include postoperative or untreated fractures, pre-existing bone disease (e.g. following radiotherapy), foreign bodies, salivary gland infections, suppurative lymphadenitis, skin cysts, and infections following local anaesthesia nerve blocks (ID block). Infections originating in the upper jaw can sometimes present as swelling apparently related to the lower jaw. Therefore both jaws usually require assessment. Tonsil and other throat infections can also spread to the submandibular region, but usually there is a prior history of a sore throat, which makes identification of the primary source easier. In many jaw infections patients usually have some

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predisposition—notably heavy smokers, but also diabetes and other causes of immunosuppression.

Diffuse infection which spreads throughout the loose connective tissue (but without suppuration)—"cellulitis", is common. In many cases this will resolve with antibiotics and treatment of the underlying cause (see also the chapter on the skin). However, untreated this can develop into a more extensive infection with abscess formation, systemic symptoms or worse. Darkening of the skin and increasing oedema are ominous signs. Necrotising fasciitis occurs when extensive necrosis of the tissues commences. This can rapidly progress and potentially become life-threatening. How aggressively and where an infection spreads to, depends on several factors, notably the local anatomy (e.g. which tooth the infection originates in), host resistance, effectiveness of treatment and virulence of the organism. The vascularity of the tissues is a key element in host resistance. This is one of the reasons why mandibular infections are more common than maxillary. The head and neck is, on the whole, a highly vascular region and this level of vascularity greatly assists in the defence against infection. Any conditions that impair the blood supply (smoking, radiotherapy or diabetes mellitus) will increase the risk of infection. Of note the blood supply to the fascia is more tenuous than that of muscle or healthy skin. This has important implications in some infections (necrotising fasciitis).

22.1.1 Fascial Spaces Related to the Mandible

Fascial spaces are actual, or potential cavities bounded by the deep fascia. Some of them are "potential spaces", that is, they do not exist in healthy individuals. They only become apparent in the presence of inflammation and spreading infection. Some spaces contain loose connective tissue, fatty tissue, salivary glands and lymph nodes, while others contain important neurovascular structures. Knowledge of the facial spaces and fascial planes is essential for understanding the spread, symptoms, management and complications of all head and neck infections. The mylohyoid muscle is a key landmark in this. It is often referred to as the 'diaphragm' of the mouth. This muscle separates the floor of the mouth from the upper neck (although the two are connected behind its posterior border). Above the muscle is the sublingual space. Below it lie the submental and submandibular spaces. Some of the spaces described are also relevant in anterior neck infections and are thus also described in that chapter.

22.1.1.1 Mental Space

This small space is located at the anterior region of the mandible, near the bone, beneath the mentalis muscle. Infection here is usually related to infected lower anterior teeth (incisors). Patients present with a firm, painful swelling over the chin. Incision and drainage may be performed intraorally.

22.1.1.2 Submental Space

This is found between the two anterior bellies of the digastric muscles. Above this space, is the mylohyoid muscle and below is the deep cervical fascia covered by

platysma and skin. The submental space communicates posteriorly with the submandibular space, allowing infection to spread backwards. In health it contains the anterior jugular vein and submental lymph nodes (level 1). Infection in this space usually originates from the lower anterior teeth (incisors), an overlaying skin abscess, or as a result of spread from other fascial spaces (mental, sublingual, submandibular). This presents as indurated and painful submental oedema and cellulitis, which may spread downwards, as far as the hyoid bone. Minor infections may be aspirated, but surgical access is usually required and is obtained behind the prominence of the chin in the upper neck.

22.1.1.3 Submandibular Space

This is a pyramidal-shaped space, bounded by the mylohyoid muscle medially, and laterally by the mandible above and the deep cervical fascia below. In health, the contents of the space are (1) the superficial lobe of the submandibular gland, which largely fills the space, (2) branches of the facial artery and (3) lymph nodes. The submandibular space communicates with (1) the sublingual space above (either through or behind the mylohyoid muscle), (2) the superficial facial space laterally and (3) the deep pterygoid space posteriorly. This latter space is important as infection here can rapidly spread throughout the neck. Infections may pass into the submandibular space from the mandibular molar teeth. This is because the attachment of mylohyoid becomes is higher towards the posterior aspect of the mandible. Thus the roots of the posterior teeth are more likely to be below mylohyoid than above. Surgical access to the space is obtained 2–3 cm below the lower border of the mandible (in order to avoid injury to the mandibular branch of the facial nerve).

22.1.1.4 Buccal Space

This is one of the most commonly affected spaces and often presents to the emergency department as a ‘fat face’. Infections can spread into it from both the mandible and maxilla. Infections arise primarily from mandibular or maxillary premolar or molar teeth, the apices of which lie outside of the buccinator muscle attachments. They are readily diagnosed because of marked cheek swelling although there may initially be minimal trismus. The space lays anteriorly, bounded by the buccinator muscle and superficial fascia and skin anteromedially. Posteriorly it passes between the masseter muscle and parotid fascia and the overlying platysma. Laterally is the deep fascia from the parotid capsule and the overlying platysma. The inferior boundary of this space is the insertion of the deep fascia into the mandible, the superior boundary is the zygomatic process of the maxilla. The buccal space contains the buccal fat pad, a syssarcosis (fat that enable intermuscular motion). Posteriorly it is continuous with the pterygoid space. Surgical access is usually obtained intraorally, low inside the cheek. Following a mucosal incision, a forcep is passed through the buccinator. A drain should always be placed. If identified, any offending tooth should be extracted. If the abscess points onto the skin, an incision can be made externally, but this will result in a scar.

22.1.1.5 Parotid Space

This space is located alongside the ramus of the mandible, between the layers of the parotid fascia. It communicates with the lateral pharyngeal and the submandibular spaces and contains the parotid gland, external carotid and superficial temporal arteries, retromandibular vein and the auriculotemporal and facial nerves. Infection of this space may arise from the teeth or the parotid gland itself. With parotid infections pus may be expressed from the papilla of the parotid duct when pressure is applied.

22.1.1.6 Masticator Space

The masticator spaces consist of the masseteric, pterygoid, and temporal space components. These spaces communicate with each other, as well as the buccal and deeper pharyngeal fascial spaces. Infections here often originate from the third molar tooth. The masticator space is complex. It is bounded laterally by the temporalis fascia, zygomatic arch and masseter muscle and medially by the medial and lateral pterygoid muscles. The temporalis muscle and mandibular ramus further divide the space into superficial and deep compartments. The superficial compartment contains the submasseteric space below and the superficial temporal space above. The deep compartment contains the superficial pterygoid space (or pterygomandibular space) below and the deep temporal space above. The superficial pterygoid space communicates with the deep pterygoid space. The superficial and deep temporal spaces together are also known as the infratemporal fossa space. The clinical hallmark of infection here is trismus with deep seated toothache. Swelling may not necessarily be prominent since the infection is deep to large muscle masses. When swelling is present, it tends to be brawny and indurated. Temporal space infections usually arise from posterior maxillary molar teeth and are noted in the chapter on the cheek. Swelling may be limited to the side of the face in the preauricular region and over the zygomatic arch and may be confused with a buccal space infection.

22.1.1.7 Submasseteric Space

This space is a cleft-shaped area between the masseter muscle and the lateral surface of the ramus of the mandible. Posteriorly it is bounded by the parotid gland, and anteriorly it is bounded by the mucosa of the retromolar area. Infection of this space usually arises in the lower third molars (pericoronitis). This is characterised by firm swelling over the masseter muscle. Severe trismus usually occurs.

22.1.1.8 Sublingual Space

Although this is anatomically within the mouth, it is mentioned here as infections from the mandible frequently extend into it. There are two connecting sublingual spaces above the mylohyoid muscle, either side of the midline. Together they make a freely connecting u-shaped space in the floor of the mouth, such that infection in one space can easily cross the midline and involve the opposite side. The roof of each sublingual space is the tongue and mucosa of floor of the mouth. The floor of each space is formed by the mylohyoid muscle. Laterally is the inner (lingual)

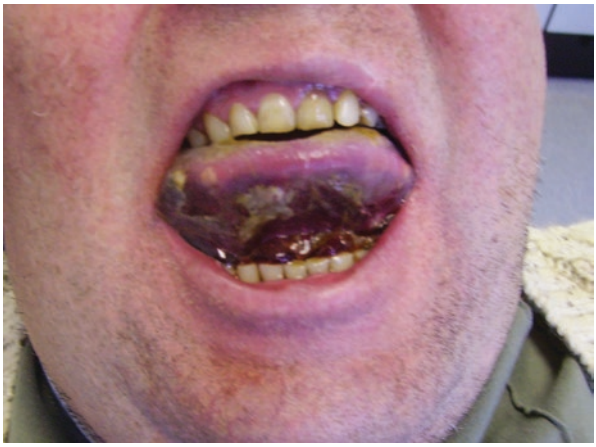

Fig. 22.1 Massive swelling into the sublingual spaces, resulting in tongue protrusion

surface of the mandible. Each space contains the submandibular duct (Wharton's duct), the sublingual gland, the lingual nerve, terminal branches of the lingual artery, and the deep lobe of the submandibular gland. Each space communicates posteriorly with the submandibular spaces. Thus, infections in the upper neck (submandibular space) can spread into the floor of the mouth, behind the mylohyoid muscle and vice versa. In very severe infections, both sublingual spaces and both submandibular and submental spaces can become involved. Posterior extension into the deep pterygoid space is also possible. Swelling in any of the spaces is therefore potentially very serious due to the ease of spread and the resulting threat to the airway. The tissues in the sublingual spaces are delicate and can easily distend, pushing the tongue up and backwards. These are almost always due to dental infections. When significant swelling is present, urgent decompression is required, even if there is no obvious pus—decompression also allows oedema to leak out (Fig. 22.1).

22.1.2 The Spread of Infection Beyond the Mandible

Spread of infections originating from the mandible occurs when pus perforates the bone at its weakest part. In the mandible, this is often the lingual bone in the molar region. From there infection can spread into the sublingual or submandibular spaces. As these spaces are only partially separated by a thin sheet of mylohyoid muscle, infection in either space easily spreads into the other. This can then cross the midline and involve the opposite spaces. As they progress further, infections within these fascial planes may spread downward along the cervical fascia, facilitated by gravity, breathing and negative intrathoracic pressure. In other patients the masticatory space is the initial site of spread of odontogenic infections. The masseter and medial pterygoid muscles are then frequently involved resulting in severe trismus. This makes intubation very difficult if surgery is required. Infection can also spread medially into the parapharyngeal space and posteriorly into the parotid space. The retropharyngeal space connects the base of the skull to the upper mediastinum and

normally contains loose fatty tissue. This is an important space because of its proximity to the airway and because infections here can quickly spread downwards resulting in mediastinitis, bronchial erosion and septicemia. The vertebral and vascular spaces may also be involved but this is rare. These are all devastating complications carrying a high morbidity and mortality. In less aggressive infections, or those partially treated with antibiotics, pus arising from a tooth may perforate the bone and accumulate between the bone and periosteum—a subperiosteal abscess. However the periosteum will eventually perforate and pus may either continue to spread throughout the soft tissues, or discharge through the skin, forming a sinus.

22.2 Life-Threatening Infections Related to the Lower Jaw

22.2.1 Ludwig's Angina

This is also discussed in the chapter on the front of the neck. Ludwig's Angina is a rapidly spreading, firm, brawny, indurated, cellulitis involving the submandibular, sublingual and submental spaces bilaterally. Patients are usually septic. The significance of this infection is reflected by its historical names of Morbus strangulatorius (so called, because of the choking effect), Angina maligna and Garrotillo (or Hangman's noose), although these are sometimes used in other severe oropharyngeal infections. The most common cause is an odontogenic infection, from one or more grossly decayed, infected teeth. Organisms are normally mixed aerobic-anaerobic flora. Once infection breaches the bone, it spreads into the adjacent space on the same side, progressing rapidly throughout the remaining spaces. The rapidity of spread is due to the presence of organisms (notably Streptococci) which produce significant amounts of hyaluronic acid and fibrinolysins. These break down the matrix of the connective tissues, opening up pathways for spread. Often there is no abscess, but rather firm induration within the floor of the mouth with surrounding oedema. This is clinically important because the muscular 'diaphragm' (mylohyoid muscle) attached to the mandible prevents tissue oedema from spreading downward. Instead, swelling progressively pushes the base of the tongue up and backwards, putting the airway at risk. This disruption to tongue function results in a characteristic speech pattern, often referred to as a 'hot potato' voice. When this is advanced, it is an obvious diagnosis. Patients present with gross swelling, involving the neck, lower jaw and the mouth. Elevation and displacement of the tongue, trismus, drooling of saliva, airway obstruction, sore throat, dysphagia and/or dyspnoea all represent significant infection. Early infections need to be treated aggressively and usually require admission. If neglected, Ludwig's angina quickly becomes an airway emergency. Rarely, infection can spread to the mediastinum, carotid sheath, or to the pterygopalatine fossa, where it can result in cavernous sinus thrombosis and subsequent meningitis. If not diagnosed and treated quickly it has a mortality rate of around 75% within the first 12–24 h. With aggressive surgical intervention, good airway control and antibiotics this has now dropped to 8% currently. Treatment involves urgent and extensive drainage plus antibiotics. Many patients require admission to the ICU.

22.2.2 Necrotising Fasciitis

Rarely, infections can become complicated with the development of necrotising fasciitis, a soft tissue infection characterised by progressive necrosis of fascia and adipose tissue. The overlying skin may initially appear uninvolved, making these infections deceptive. Because of this, necrotising fasciitis is often extensive by the time it is recognised. Patients are often immunocompromised secondary to associated conditions, such as diabetes and malnutrition. An initial "simple" infection quickly gets worse and the patient becomes unwell. If this is not recognised, the infection can extend further to involve the great vessels or mediastinum, resulting in systemic toxicity and sepsis. Imaging is therefore essential in most swollen, infected necks/faces. In the early stages it is often difficult to determine whether the swelling is secondary to cellulitis alone, or if there is an abscess requiring drainage. Plain films of the neck and the chest may be useful if they show subcutaneous emphysema or prevertebral swelling displacing the trachea. A chest X-Ray may also show a widened mediastinum or pleural effusion. However, CT is preferable and should be undertaken as soon as possible. CT will also determine the site of any abscess and help plan surgical access for drainage. Any patient with significant neck or lower jaw swelling should therefore have a CT of the neck and chest to assess the spread of infection. Treatment involves surgical debridement, drainage of the abscess, broad-spectrum intravenous antibiotics and removal of any underlying cause.

22.2.3 Descending Necrotising Mediastinitis (DNM)

Acute purulent mediastinitis may develop following cervical cellulitis, Ludwig's Angina, or as a complication of cervical trauma. This is a serious infection and is often fatal. Necrotising mediastinitis quickly spreads to involve the connective tissues surrounding the thoracic organs. As the infection spreads there is widespread cellulitis, necrosis and abscess formation. Sepsis quickly develops. Urgent CT and referral to a thoracic specialist is required. Even with aggressive drainage and high dose antibiotics, the mortality rate remains high. Surgical management includes cervical drainage and thoracotomy.

22.3 Determining the Severity of Infection

Symptoms and signs of any infection depend on its severity. This in turn is determined by a balance between the health of the patient, the virulence of the microorganism, treatment given so far and the fascial space, spaces or cavities involved. In the head and neck, patients usually present because of a localised or, more commonly, diffuse swelling involving the face or neck. There is usually pain, redness, and warmth over the swollen area—four of the five cardinal signs of inflammation. Pyrexia, tachycardia and generalised malaise are often present. With mandibular, throat and oral infections there may also be loss of the ability to swallow and speech

disturbance, loss of the ability to protrude the tongue and difficulty breathing and stridor. These latter signs (collectively loss of function, the fifth cardinal sign of inflammation) are indicative of a life-threatening airway emergency. Elsewhere in the head and neck, other functional restrictions will be related to the anatomy of the inflamed tissues (diplopia, visual impairment, loss of hearing, nasal congestion etc.). Intracranially severe and unaccustomed headaches, neck stiffness, photophobia and increasing drowsiness are extremely worrying features.

Untreated, infections (irrespective of their site of origin) will quickly result in localised tissue destruction, with thrombosis of local vessels and septic emboli, generalised bacteraemia, septicaemia, multi-organ failure and death. Infections of a more chronic nature may progress more slowly, resulting in a chronic abscess, scarring and osteomyelitis. Usually a full blood count will show an increased white cell count (leucocytosis). If there has been a chronic abscess, (for example secondary to tuberculosis) there may be an increased lymphocyte count with a normal neutrophil count. If the patient is dehydrated, the haematocrit will be raised. If significant dehydrated the urea and creatinine will be elevated indicating pre-renal failure. A raised ESR may indicate the presence of bacteraemia or septicaemia. Blood cultures should be taken. It is also important not to forget to take a random serum glucose sample. The patient may be an undiagnosed diabetic.

Odontogenic, tonsil, sinus and skin sources are by far the commonest causes of infections in and around the lower jaw. Dental infection arises within the necrotic pulp tissues following dental neglect or failed restorations. This usually spreads into the supporting jaw, presenting as a dental abscess. Other odontogenic causes include periodontal abscess related to the supporting structures of the tooth and pericorinitis related to partially erupted or impacted wisdom teeth. Thus dental infections are represented by the “three Ps” periapical, periodontal and pericorinitis. Dental related infections often present with localised pain, and tenderness around the causative tooth. There may also be a fluctuant swelling present, which if appropriate may be amenable to incision and drainage under local anaesthetic. Patients can also present with systemic signs of infection suggesting a septic host response. Most infections are usually due to a mixture of organisms, predominantly anaerobic bacteria which are commonly found as part of the oral, skin or nasal flora. If possible send samples for microbiology, as this may guide subsequent therapy if the patient doesn’t improve. Bacteroides species and anaerobic streptococci are the most commonly isolated organisms for many dental infections.

Assessment should focus on the following aspects:

1. Is there any suggestion of airway compromise?
2. Any change in the patient’s voice?
3. Is the patient complaining of any difficulty swallowing?
4. Over what time period has any swelling arisen (i.e. a large swelling which has arisen over several days can be managed differently to one that has arisen over a few hours).
5. Has there been any history of dental pain or treatment?

6. How far can the patient open their mouth and 'wiggle' their tongue? (This will need communicating to the anaesthetist as restriction suggests airway risk, and the possible need for an awake fibre-optic intubation). Document clearly the degree of mouth opening, and whether there is any suggestion the floor of mouth is raised.
7. HR/BP and temperature are basic assessment tools. Does the patient appear systemically unwell?
8. Is there any systemic disease such as diabetes or history of immunosuppression. Document a blood glucose recording as a base line.
9. Is there any history of foreign travel if there is no other obvious bacterial cause.
10. Could there be a foreign body?
11. Consider also the rapidity of onset of symptoms, and always bear in mind the potential for necrotising fasciitis which can rapidly become life threatening due to overwhelming sepsis.

Infections of the lower jaw can quickly extend into the subcutaneous tissue, resulting in a facial cellulitis, or more deeply into the fascial compartments. Palpation of the area can sometimes help differentiate between cellulitis and an abscess cavity, but is not entirely reliable. Imaging may be required in uncertain cases. If cellulitis is suspected, its border should be outlined with a suitable marker, and the patient reassessed regularly to determine if they are responding to treatment or whether surgical management is required. The main concern initially with these rapidly progressing infections is the potential effects on the airway. CT Scans if available, can help evaluate airway patency (or more importantly lack of), CT and US can delineate any collections amenable to drainage. All clinicians who are involved in the management of these patients should be familiar with the management of the difficult airway. Many guidelines exist, in the UK those from the 'Difficult airway Society' are very helpful.

22.4 Other Soft Tissue Infections Related to the Lower Jaw

22.4.1 Cellulitis

This is also discussed in the chapter on the skin. Cellulitis is a bacterial infection of the deep dermis and subcutaneous tissue. The most common infecting organism is group A streptococcus, followed by staphylococcus aureus. These gain access to the dermis and subcutaneous tissues through a break in the skin (e.g. during shaving). Cellulitis around the lower jaw and face can have many causes. These include:

1. Dental infections (tooth or cyst)
2. Skin cyst infections (e.g. sebaceous)
3. Infected lacerations with or without a retained foreign body
4. Infected lymph nodes (usually cellulitis is in the neck)
5. Submandibular infections
6. Untreated fractures

Infection arising from a tooth with cellulitis should warn the clinician to be possible early Ludwig's angina. Cellulitis is characterised by swelling, warmth, erythema and pain. The patient may present with minimal signs initially, or they may quickly become unwell with malaise, fatigue, chills and a fever. Regional lymphadenopathy may also be present. Initially, inflammation is confined to the deeper dermis and subcutaneous tissues resulting in an ill-defined redness to the skin. The skin may be finely dimpled giving it an "orange peel" appearance, due to tissue oedema. Management includes high dose antibiotics such as Flucloxacillin or Benzylpenicillin, which are effective against streptococci. Oral antibiotics are usually sufficient for most early or mild infections. However cellulitis should be treated with caution, it still has the potential to become life-threatening. In immunocompromised individuals and children a mixed infection may be present and a broader spectrum of antibiotic may be necessary. Neisseria meningitidis has also been reported to be an uncommon but notable cause of cellulitis in immunocompetent hosts even in the absence of bacterial meningitis. This is important as the usual empiric antibiotics in facial cellulitis do not treat this organism. When cellulitis involves the parapharyngeal and retropharyngeal spaces, the infection has easy access to the mediastinum, pericardium and thorax, significantly increasing mortality rates. Prompt recognition, immediate surgical intervention, broad-spectrum antibiotics and intensive medical support are then required. If the cellulitis is tense, it requires surgical drainage to release the inflammatory exudate. Therefore treat cellulitis in the head and neck with respect. It can quickly spread and result in abscess formation or necrotising fasciitis. Always consider the possibility of an underlying cause (often dental infections) and that the patient may be immunocompromised (diabetes and alcohol abuse).

22.5 Infections Within the Mandible

22.5.1 Pericoronitis

Although this is technically a soft tissue infection, from a practical viewpoint this should be considered as a potentially serious within the mandible, which if left untreated can rapidly progress. Pericoronitis is an infection within the gum over any partially erupted tooth, most commonly a lower third molar. Food trapping and stagnation creates an environment for recurrent infections by mixed microorganisms, notably anaerobes. Pericoronitis may present either acutely with suppuration or in a chronic form with recurrent pain and swelling. This is an important infection. From this anatomical site infection can quickly spread forwards into the sublingual or submandibular spaces, or backwards into the deep pterygoid space, from which it can quickly gain access to the mediastinum. Involvement of the masseter or medial pterygoid muscles can result in severe trismus. Wisdom teeth related infections are one of the commonest causes of severe infections in the head and neck.

22.5.2 Periapical Infections

Acute apical periodontitis occurs when purulent pulpitis spreads into the periapical space (the periodontal space around the tip of the root of a tooth). The tooth quickly becomes very painful and very tender to bite on. This is described in the chapter on the mouth. Untreated, periapical infection may perforate the mandibular cortex and enter into the adjacent fascial space. Which space is involved depends on the relationship between the tip of the root and the attachments of the adjacent muscles. Pus originating from apices above the mylohyoid muscle attachment usually spreads into the floor of the mouth (sublingual space), whereas infection from the apices below the mylohyoid attachment (second and third molar), spreads into the submandibular space.

22.5.3 Chronic Dentoalveolar Abscess

Chronic dentoalveolar (odontogenic) abscesses in both the upper and lower jaws can result in the development of a fistula, which periodically swells and then discharges pus. This can be seen either within the mouth or in the overlying skin. Infections are often asymptomatic or present with mild discomfort. The tooth itself may be sensitive to percussion. There may be puckering of the skin or mucosa and a chronic discharge which dries, resulting in the formation of a crust. Excision of the fistula alone will not treat this. The cause (i.e. the offending tooth) needs to be either extracted or undergo root canal therapy.

22.5.4 Infected Fractures

Mandibular fractures are reported to be associated with the highest rate of infections among maxillofacial fractures. Infected mandibular fractures are occasionally seen in most busy centres managing large numbers of fractures. It varies considerably. At one end of the spectrum are those who sustain injuries but delay seeking care until infection occurs. At the other end are the elderly and infirm in whom the injury goes unrecognised until infection develops. Patients may present with either a facial abscess or draining fistula. To be considered infected, there must be frank purulent drainage from the fracture site, either intraorally or through an extraoral fistula or facial cellulitis. Most infected fractures are open fractures and usually related to a tooth in the fracture line. Severe infections of mandibular fractures which progress to osteomyelitis are rare. However localised infection occurs relatively frequently, particularly in patients with poor oral hygiene, smokers and those that present late. Debilitated patients, diabetics and patients on steroids or chemotherapy are at high risk. Smoking is by far the most commonly associated risk factor. Infection often develops around fractures of the mandibular angle in association with a partially exposed third molars (wisdom teeth). The tooth forms an initial nidus of infection

which can then pass directly into the fracture. It is for this reason that some specialists routinely remove the wisdom tooth when repairing an angle fracture. However, any fracture (or the fixation plates used to repair it) can become infected if the patient is predisposed and their oral hygiene is poor. Comminuted fractures, which follow high energy impacts are also at risk of sequestration and infection. Not only are the bones disrupted but often the healing potential of the surrounding soft tissues is also impaired, contributing to an 'at risk' tissue environment. In some cases sequestra may develop and begin to extrude spontaneously into the mouth with minimal symptoms. In other patients an abscess may develop, requiring removal of the non-vital bone and any fixation hardware. More commonly, infected fractures present as chronically discharging wounds. Fixation (if the fracture has been previously treated) is often seen on the OPG to have failed, with loose screws and displaced plates. Sometimes these can be visible in the mouth, surrounded by granulation tissue and pus. Not surprisingly the fracture is usually mobile. These are difficult infections to treat, not helped in patients who are poorly compliant and who continue to smoke and sometimes fail to attend appointments. Management involves prolonged antibiotics, debridement and removal of any fixation hardware, associated teeth and dead bone, followed by stabilisation of the fracture (IMF, External fixation, reconstruction plates). Despite aggressive treatment infection can still persist, resulting in chronic osteomyelitis. Oral hygiene is a key factor to eliminate and prevent postoperative infection.

22.5.5 Osteomyelitis

Osteomyelitis may be defined as an infection in bone *with a tendency to progression.* This latter characteristic is what differentiates osteomyelitis from a dentoalveolar abscess, dry socket or the localised osteitis seen in infected fractures. It can involve both the cancellous bone and the cortical plates of the mandible, extending into the subperiosteal and surrounding tissues. Today, osteomyelitis is relatively uncommon due to the early use of antibiotics. However, antimicrobials have been reported to be less effective, as a result of increasing bacterial resistance. Even today, osteomyelitis can be a major source of morbidity, requiring multiple surgeries, with loss of the teeth and bone. Infection is most commonly associated with an odontogenic sources (infections of the teeth), but it can also occur following extractions and in some fractures. Patients who have had their jaws irradiated or those taking bisphosphonates (BRONJ) are particularly difficult to treat. Osteomyelitis is more common in the lower jaw, as the upper jaw has a relatively better blood supply, compared to the mandible's dense and less vascularised cortical plates. Before the antibiotic era, this was frequently fatal. Acute osteomyelitis is now less common than chronic osteomyelitis, with patients rarely presenting with obvious suppuration. A small amount of pus exuding from around a solitary tooth is more likely to be a periodontal abscess, but if multiple adjacent teeth are involved, mobile and the overlying soft tissue are inflamed, there is probably osteomyelitis present (Fig. 22.2).

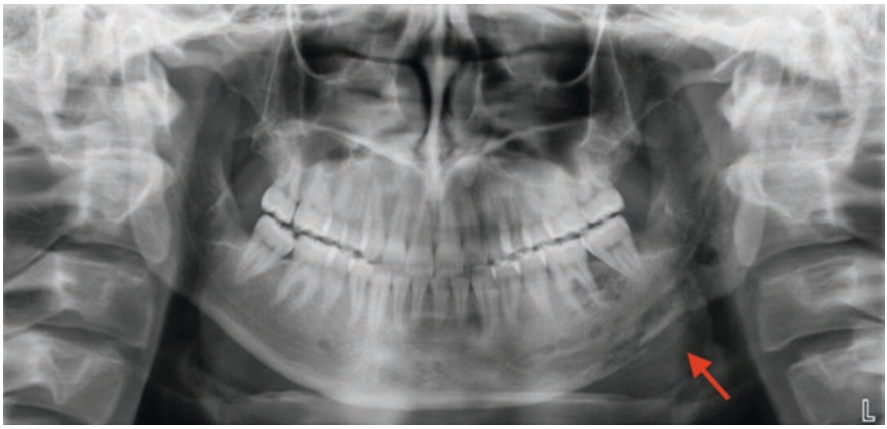

Fig. 22.2 Acute osteomyelitis. Orthopantomogram demonstrating diffuse left mandibular bony changes 6 weeks after the extraction of tooth # 17. Note extensive lytic changes and involvement of the lower cortex of bone (arrow)

Over the years a number of classifications have been devised—(1) suppurative or non-suppurative, (2) haematogenous or secondary to a focus of infection, (3) acute or chronic. A useful system divides osteomyelitis into acute or chronic forms based on the duration of the disease over 1 month or more.

1. Acute osteomyelitis
   (a) Focus of infection identified
   (b) Progressive
   (c) Haematogenous spread
2. Chronic osteomyelitis
   (a) Recurrent multifocal
   (b) Garré's
   (c) Suppurative or non-suppurative
   (d) Sclerosing (Fig. 22.3)

Clinical features depend on the type of osteomyelitis and the extent of infection. These overlap with many of the symptoms previous described for fascial space infections. Some symptoms and signs also overlap with advanced malignancy of the jaw, mandating the need for imaging and biopsy in many cases. Symptoms include:

1. Pyrexia, anorexia and malaise
2. Pain, swelling, erythema and tenderness.
3. Trismus
4. Bleeding or suppurating gums, with increased loosening of the teeth, sometimes with spontaneous loss
5. Numbness of the lower lip and jaw
6. Halitosis

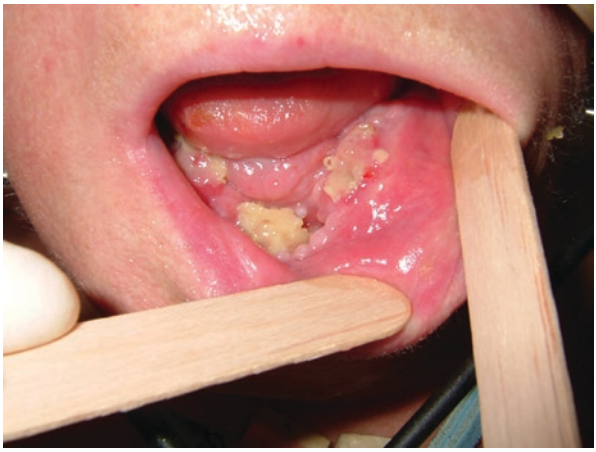

Fig. 22.3 Chronic osteomyelitis

7. Friable granulation tissue. Exposed necrotic bone and sequestrum formation is often seen in chronic infection.
8. Fistulae
9. Cervical lymphadenopathy

The pain in acute osteomyelitis is often described as a deep, boring pain, often out of proportion to the clinical picture. The pain in chronic infection is less intense but still deep seated and unremitting. In acute osteomyelitis it is very common to see swelling and erythema of the overlying tissues. This is initially soft but may later progress to a firm subperiosteal abscess. Fever often accompanies acute infection, whereas it is relatively rare in the chronic form. Paraesthesia of the inferior alveolar nerve is an important sign indicating pressure or ischaemia of the inferior alveolar nerve (Vincent's sign). Usually there is an obvious cause such as a decayed tooth. This may be tender and mobile. Most patients are either malnourished or immune deficient. Osteomyelitis is therefore commonly seen in smokers, diabetics and alcoholics as well as other high risk groups. Medications which predispose to infection include steroids, chemotherapeutic agents and bisphosphonates. Radiation therapy, sickle cell anaemia, osteopetrosis, fibrous dysplasia and other bone pathology can also diminish the blood supply to the area, increasing risk.

Investigations usually show a leukocytosis in the acute phase. However this is relatively uncommon in chronic osteomyelitis. There may also be a raised erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP). All patients require imaging. In some cases malignancy may present with similar features. A pathological fracture, secondary to tumour invading the bone, can also become infected, complicating the diagnosis. The OPG is a good choice for initial assessment of the lower jaw. This will often show bone changes suggestive of infection, plus any predisposing conditions such as dental disease, fractures or co-existing bone disease. However, a 'normal' Xray does not exclude infection. Radiographic changes lag behind the clinical features and it can take several weeks before bony changes become radiographically visible. Typical findings include a "moth-eaten" appearance to the bone,

or the presence of sequestra. These changes may also be seen in malignancies, which must therefore be kept in mind. Computerised tomography scan (CT) has now become the standard in evaluating osteomyelitis. Magnetic resonance imaging (MRI) can also help in early diagnosis by demonstrating loss of the marrow signal. These will also identify any soft tissue tumours invading the bone. In most cases biopsy is required, particularly if there is an associated pathological fracture (Fig. 22.4).

Infections are usually polymicrobial in nature. In most cases streptococci and anaerobic bacteria invade the bone from an infected tooth or untreated fracture. Primary haematogenous osteomyelitis is rare, generally occurring in the very young.

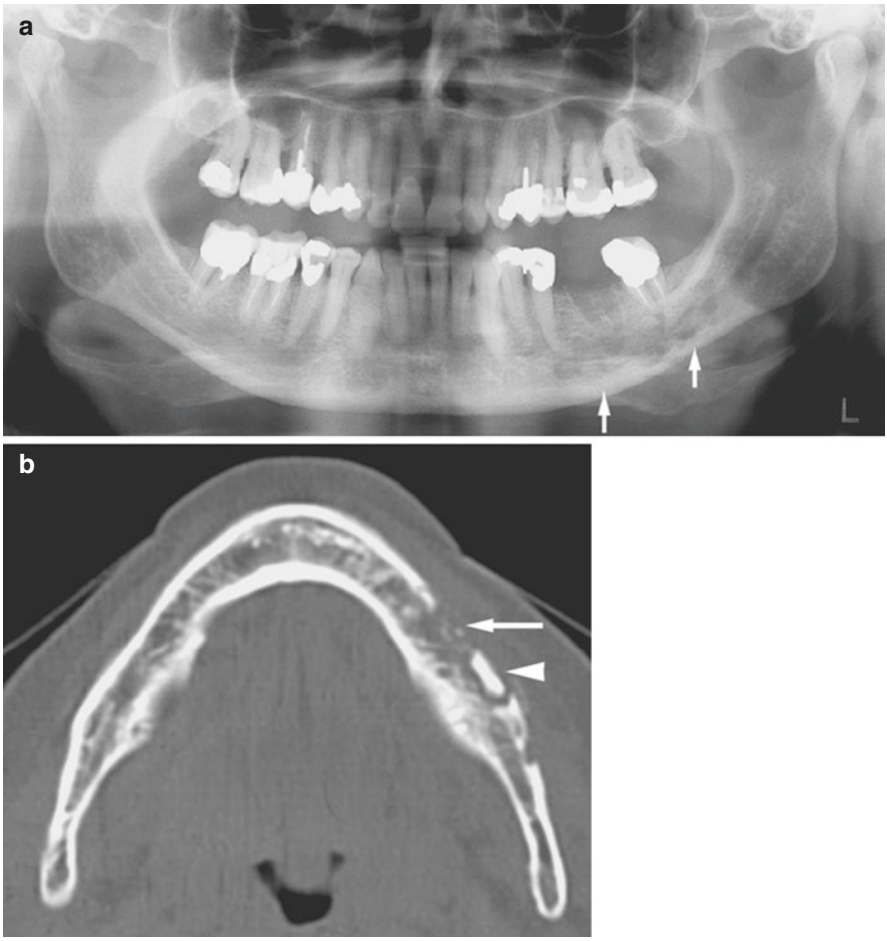

Fig. 22.4 Osteomyelitis, mandible; 46-year-old female with previous pain from molar that was extracted, but still pain and additionally, perimandibular swelling. (**a**) Panoramic view shows diffuse bone destruction (arrows). (**b**) Axial CT image shows diffuse destruction of buccal cortical bone (arrow) and sequestrum (arrowhead)

Actinomycosis is an unusual but important infection which has a propensity towards the mandible. This results in recurrent and chronic jaw abscesses, which discharge large amounts of pus, characteristically containing bright yellow ("sulphur") granules. This should be considered in any patient with chronic bone abscesses and discharging sinuses.

Acute osteomyelitis needs urgent admission, intravenous antibiotics and debridement with drainage of pus. In rare cases of infantile osteomyelitis, intravenous antibiotic therapy may successfully treat the disease, but antibiotic therapy alone is rarely curative in adults. Chronic osteomyelitis may be managed as an outpatient with appropriate long term antibiotics. If so, close followup is required. The decision to admit patients for intravenous antibiotics depends on a number of factors, including the severity of symptoms, signs of systemic involvement, extent of infection (seen on CT), coexisting medical problems and expected compliance. Empiric antibiotic treatment should be started based on initial Gram stain results, or suspected pathogens. Definitive culture and sensitivity reports will later guide the best choice of antibiotics. Surgical drainage, debridement and sequestrectomy, together with removal of any source of the infection is usually required. If necessary decortication of the mandible may also be undertaken. In severe chronic cases, resection of the affected bone with reconstruction may even be required. If acute osteomyelitis is not treated effectively, it can lead to chronic suppurative osteomyelitis. This is characterised by attempts at healing and a lesser degree of inflammation. Granulation tissue, new blood vessels form and sequestra occur.

22.5.6 Infections in Mandibular Osteoradionecrosis

Although radiotherapy is a major and important element in the treatment of many types of head and neck cancer, this is not without complications. Mandibular osteoradionecrosis (ORN) is a major debilitating disease which can result in chronic pain, bone loss, pathologic fractures and secondary infection. The pathogenesis of ORN is discussed later in this chapter. The mandible is commonly affected following high doses of curative intent radiation (>50–60 Gy). This results in a hypocellular, hypovascular and hypoxic tissue environment which is susceptible to trauma (such as tooth extraction), and the additive effects of poor oral hygiene, smoking and diabetes. Mucosal ulceration, another common complication, results in loss of the protective covering barrier, thereby allowing microorganisms in the mouth to enter areas of non-vital or immunocompetent tissues. Many bacterial species have been isolated in mandibular ORN, including oral, respiratory and skin flora, anaerobes and actinomyces. Candida osteomyelitis can also occur.

ORN is a common cause of secondary mandibular osteomyelitis which has been reported to occur in up to one fifth of irradiated mandibles. Usually infections occur late (>1 year) after the development of ORN, classically described as the development of denuded bone with fistulous tracts. Therefore in all head and neck cancer patients, previously treated with radiation, the development of new symptoms suggestive of infection or pain, requires carefully examined of the bone for signs of

ORN. Osteomyelitis commonly presents with a new or worsening pain, which may be non-specific or associated with worsening trismus, odynophagia, otalgia or numbness of the lower lip and teeth. Erythema, swelling or purulent drainage may also be present. Clinically and radiographically these finding can be difficult to differentiate from non infected ORN or recurrent tumour. Early radiographic features of ORN include cortical erosions and changes in the endosteum, periosteum and vascular channels. Cellulitis and abscess formation support the diagnosis of a superimposed infection.

Initial treatment may include of antibiotics, oral rinses, hyperbaric oxygen and local surgical procedures (incision and drainage of abscesses, sequestrectomy, and/or tooth extractions). If this fails some form of segmental mandibulectomy and reconstruction with bone free flaps may be necessary.

22.5.7 Primary Chronic Osteomyelitis (PCO)

This is a rare, non-suppurative inflammatory disease of the mandible of unknown aetiology. It can occur at any age. It has been subdivided into adult onset, early onset (depending upon age at presentation) and syndrome associated. PCO may occur in isolation of it may be associated with chronic recurrent multifocal osteomyelitis and SAPHO syndrome (synovitis, acne, pustulosis, hyperostosis and osteitis). Clinical features are varied and include recurring episodes of swelling in the lower jaw, trismus, cervical lymphadenopathy and numbness of the lower lip. However it is the absence of pus, fistula formation and sequestra formation that differentiates this from acute and chronic osteomyelitis. Investigation often show only a mild to moderate elevation of C-reactive protein, erythrocyte sedimentation rate and lymphocyte count. On imaging in the early stages of the disease there may be patches of mixed sclerosis and osteolysis, which later become markedly sclerotic. Biopsy is often required , which shows the presence of pagetoid bone formation, lymphocytes, plasma cells (suggestive of chronic inflammation) and varying amounts of medullary fibrosis. Surgical treatment (decortication) with antibiotics and non-steroid anti-inflammatory drugs is usually required (Fig. 22.5).

22.5.8 Actinomycosis

This is a specific chronic suppurative and granulomatous infection, which can present with chronic or recurrent swelling in the face and neck. It is normally a soft tissue infection but can occasionally involve bone. The causative microorganism is Actinomyces israelii, which is present in normal oral flora. Damage to the tissues (following dental extractions or fracture), creates a favourable environment for the organism to grow. Infection results in a firm, indurated swelling (usually in the submandibular region), which develops several weeks after the injury. Multiple abscesses with sinuses may subsequently develop. Discharge from the sinuses contains microscopic “yellow granules” (so-called sulphur granules). Radiographically

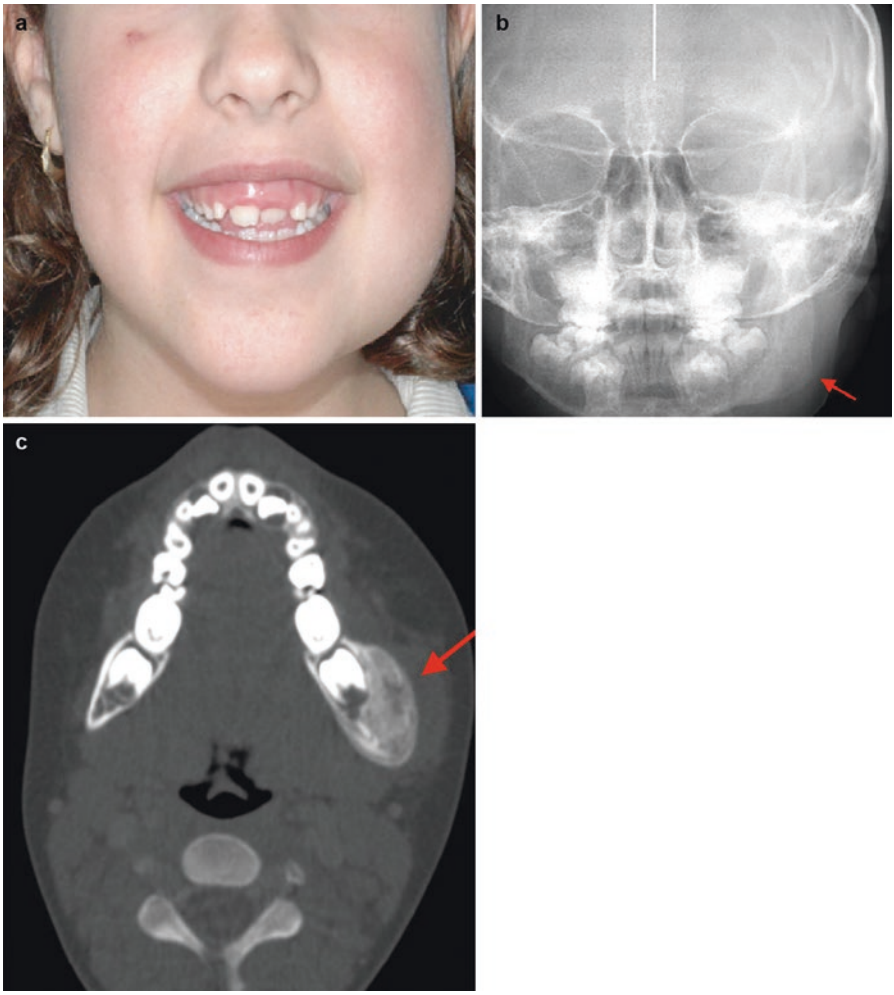

Fig. 22.5 (**a**–**c**) Primary chronic osteomyelitis (PCO). The child presented with pain, swelling, and tenderness of bony expansion and sclerosis (arrow) on plain film and (**c**) extensive subperiosteal bone formation on CT (arrow)

there may be bone destruction. Penicillin is the drug of choice, following incision and drainage. Antibiotic treatment must be continued for at least 6 weeks.

22.5.9 Syphilis

This is a chronic infectious disease, caused by the spirochete Treponema pallidum. Although rare today, primary (chancre), secondary (skin rashes, lymphadenopathy, mucous patches, and snail track ulcers), and tertiary (gumma or syphilitic

leukoplakia) may all occur in the oral cavity. Bony changes may occur during the tertiary stage, when the periosteum is infiltrated by gumma. This may be seen radiographically as a 'peeling away' of the periosteum, with the formation of sclerotic bony margins. Gumma may occasionally extend into the underlying bone resulting in syphilitic osteomyelitis. Long-term penicillin is the drug of choice, following debridement.

22.6 Infections Related to the Salivary Glands

Sialadenitis is the most common pathology affecting the salivary glands. This is inflammation of the salivary glands, which has several varied causes. Patients present with swelling of the gland, pain, foul taste in the mouth and sometimes xerostomia. The presentation may be acute, chronic or mixed. Causes of sialadenitis include acute or chronic infective, obstructive, immunoglobulin G4-related sialadenitis (IgG4-RS), lympho-epithelial, granulomatous and post-treatment sialadenitis. Infections of the salivary glands can be bacterial, viral, fungal, or even protozoal. Both the major and minor salivary glands can become infected and present as swellings around the lower jaw or the side of the face. 'Ascending' or retrograde infections, i.e. where bacteria in saliva pass along the ducts into the glands, commonly involve the parotid and submandibular glands. This type of infection is promoted by stasis in the normal flow of saliva, often secondary to stones, stricture or increased viscosity of its mucus component. Predisposing conditions are also commonly associated, (notably dehydration, diabetes, xerogenic drugs or immunosuppression). Fibrosis following radiotherapy and transitory bacteraemia, especially in the neonatal period, also predispose to infection.

Patients usually present with sudden onset of pain, localised to the gland and surrounding area. This may become red, swollen and tender and the patient may become pyrexial. Intraorally the mouth may appear dry, if the patient is dehydrated. Gentle massage of the gland may discharge pus from its duct. If the infection is not treated early, this may develop into an acute abscess or alternatively a chronic or recurrent infection. Repeated infections will result in progressive destruction of the gland. This aggravates the situation resulting eventually in a non-functional scarred gland. In the absence of an obvious abscess, management initially consists of antibiotics, rehydration, analgesia and correction of any systemic disorders. If obstruction from a stone is identified on imaging (usually a lower occlusal film), this should be removed to enable drainage. This can be done endoscopically in some specialist centres. However in many cases no stone is found and the most likely cause of obstruction is thick (inspissated) mucous. Gland massage, especially after meals and 'lemon drops' (to stimulate salivary flow), should be encouraged to help maintain a flushing effect, remove thick mucous and relieve stagnation of saliva. Abscesses need to be incised and drained on an urgent basis. If infection persists or continues to recur, excision of the gland ultimately may be necessary. This is best done when there is no active infection.

22.6.1 Viral Infections

Viral infection of the salivary glands most commonly occur as a result of haematogenous spread. A wide range of viral infections are known to occur, including Coxsackie, Parainfluenza, Influenza, ECHO virus, Epstein-Barr, HIV, enteroviruses, Cytomegalovirus and Lymphocytic choriomeningitis virus. Mumps is by far the most common viral infection of the parotid gland.

22.6.1.1 Mumps (Epidemic Parotitis)

Mumps is a highly contagious RNA Paramyxovirus that spreads rapidly among people living in close quarters. The virus is transmitted by respiratory droplets or direct contact, including the sharing of eating utensils and cups. Individuals are infectious for approximately 1 week before and after the onset of symptoms. Following resolution of infection patients are usually immune for life, although reinfection has been reported. Diagnosis is usually made on clinical grounds, but can be confirmed by isolating the virus from a swab of the parotid duct, using polymerase chain reaction (PCR). As with any inflammation of the salivary glands, the serum level of amylase is often elevated, but this is not routinely tested. Symptoms in adults are often more severe than in children. Initial prodromal symptoms include fever, myalgia, headache and lethargy. These are followed by painful swelling of one or both parotid salivary glands. This results in a non-suppurative acute sialadenitis, which lasts for about a week. Although in most cases there are no serious consequences of the infection, serious complications include meningitis, encephalitis, pancreatitis, sensorineural hearing loss and orchitis (inflammation of the testes or ovaries). In men this can result in infertility. Mumps is preventable by immunisation, given separately or as part of the MMR immunisation vaccine. Otherwise there is no specific treatment, other than bed rest, hydration and symptom control with analgesics.

22.6.2 Acute Submandibular Gland Sialadenitis

Submandibular sialadenitis accounts for about 10% of all cases of sialadenitis. Most are related to stones (calculi) within the gland or its duct. Submandibular secretions are mucinous and viscid, contributing to calculus formation. Other predisposing causes include surgical scarring or strictures secondary to radiation. Once the drainage of the gland is compromised, secretions can stagnate, thicken and then obstruct flow or calcify (a pathological chain of events that is seen elsewhere in the body—gall stones, renal calculi etc.). Patients may initially develop obstructive symptoms, but if infection occurs they then present with rapid onset of pain, swelling and induration of the gland. *Staphylococcus aureus* is the most common pathogen. However, gram-negative organisms may affects neonates and debilitated patients. The overlying skin can become cellulitic. Wharton's ducts may appear erythematous and gentle massage of the gland will often result in a discharge of pus. Occasionally a calculus may be palpable, but confirmation is necessary as some tumours may

present as hard masses in the floor of the mouth. Orthopantomogram or a lower occlusal view of the floor of the mouth may sometimes reveal a salivary soon as these are said to be radio-opaque in around 80% of cases. However sialography is contraindicated in the acute stage when active infection is present. Alternatively ultrasound can detect diffuse and focal lesions, some stones and dilated ducts. Sialography may be undertaken following the acute phase and can visualise the ductal system; however, it is an invasive procedure.

If symptoms of inflammation are present antibiotics need to be prescribed. If a stone is easily palpable in the mouth, it can sometimes be removed under local anaesthetic, often with a gush of thick fluid and immediate (but partial) relief of symptoms. Endoscopic removal may be possible with small stones, less than 5 mm or so. Lithotripsy, similar to that used in renal calculi can also be of help in selected cases. Stones need to removed whenever possible. If not and the infection progresses to a collection, incision and drainage of the submandibular fascial space must be carried out. The gland may then require removal later.

22.6.3 Acute Bacterial Parotid Sialadenitis

Mumps is the commonest cause of viral parotid swelling. Acute suppuration can also occur either as a secondary complication, or more commonly as a separate entity. Parotid saliva is primarily serous and provides less protection against retrograde (ascending) bacterial contamination than the mucoid saliva produced by the submandibular and sublingual glands. These glands also secrete antimicrobial agents, including lysozymes and IgA antibodies. Mucins also contain sialic acid, which agglutinate bacteria, preventing their adherence to the tissues. In acute suppurative parotitis ascending infection is the primary cause. The most common pathogens are Staph. aureus and anaerobic bacteria including Prevotella and Fusobacterium spp. Less frequent are the streptococci (including S. pneumoniae), gram-negative organisms, (including Escherichia coli, Klebsiella pneumoniae), and Pseudomonas aeruginosa. Actinomycosis of the parotid gland is rare but has been reported to present with features suggestive of a tumour. Similarly, facial nerve palsy has been reported to occur secondary to a parotid abscess. A few cases of involvement of the facial nerve in benign and inflammatory conditions of the parotid gland have been reported, but this should always raise suspicion of malignancy in the first instance.

Patients present with fever, pain, erythema and tender swelling over the gland. Pain is often very severe due to the relatively unyielding parotid fascia (effectively a compartment syndrome-type phenomenon) and fluctuance is generally not appreciated due to the dense fascia. Intraorally discharge of pus from the duct may be seen. If the infection is not treated early, this can develop into a chronic or recurrent infection. CT or US imaging of the gland is required to identify any abscess requiring drainage. Sialography is contraindicated in the acute stages as this can spread the existing infection. Progressive destruction of the gland aggravates the situation, resulting in a non-functional gland. Although the parotitis is very rare in neonates as

described it should be suspected in neonates with erythematous preauricular swelling. Treatment includes hydration and intravenous antibiotics. Since suppurative parotitis may invade the deep fascial spaces and is potentially life-threatening, patients should be admitted. If there is no abscess, management of acute parotid infections initially consists of intravenous antibiotics, rehydration, analgesia and screening and treatment of any predisposing conditions (diabetes). Pus swabs should be taken and sent for microbiology. Regular gentle massage of the gland, just before and after meals, and lemon drops or vinegar (anything acid) to stimulate salivary flow, helps to flush the gland and remove residual bacteria. If an obstruction is present, this should be removed if possible to allow drainage. Small, superficial abscesses may be aspirated under ultrasound guidance, but larger or deep abscesses will need to be incised and drained on an urgent basis. If infection continues to recur, progressive destruction of the gland and fibrosis will result in a chronically painful scarred and non-functional gland. Excision may then be necessary. This is best done when there is no active infection. Fistula formation is rare and should be regarded with suspicion for malignancy. With regards to any stones, sialendoscopy is increasingly used in both diagnosis and treatment of obstructive symptoms, in selected cases combined with endoscopic laser lithotripsy.

22.6.3.1 Juvenile Recurrent Parotitis (Recurrent Parotitis of Childhood)

This is a recurrent suppurative disease of the parotid glands. It is characterised by recurrent swellings in one or both parotid glands in children, clinically resembling mumps. First infection can develop as young as 3. Infections can then occur periodically several times a year, sometimes persisting into adulthood. Although the cause in unknown, it has been suggested the condition may be related to congenital malformation of the ductal system, genetic factors, allergies, autoimmune disease and IgA or IgG3 deficiency states. Virtually all patients become asymptomatic by the age of 10–15 years. Episodes last 3–7 days and can vary from mild and infrequent attacks, to severe and of such frequency that they prevent the child from attending school regularly. Acute episodes are managed conservatively with hydration, stimulation of salivation and analgesics. Patients with fever and purulent exudation require antibiotics. Severe disease may require parotidectomy.

22.6.4 Chronic Bacterial Sialadenitis

Both the parotid and submandibular glands can develop chronic infections—chronic sialadenitis. As with acute infections, underlying causes include salivary stagnation secondary to an underlying obstruction (duct stenosis, thick mucus or a stone). Other causes include Sjögren's syndrome and repeated episodes of acute suppurative infections. Both of these result in permanent damage to the gland, with sialectasis, ductal ectasia and progressive acinar destruction. The structure and function of the involved gland is thus gradually destroyed, resulting in a positive cycle of

decreased salivary secretion, thick mucus production and repeated infections. Patients present with recurrent swelling of the affected gland. Over time the gland atrophies and is replaced by fibrotic tissue, making it more palpable. Chronic sclerosing sialadenitis of the submandibular gland (Küttner's tumour—a misnomer) may be confused with a genuine tumour within the gland. This is now believed to be related to IgG4-related disease in some patients (see the chapter on the cheek and orbit) and can present with symptoms of chronic infection. Investigations include US, sialography, MRI and, if in doubt fine needle aspiration. Treatment of acute episodes is the same as for acute bacterial sialadenitis. Specific treatment may be required for any structural abnormality, such as a stricture or calculus. Interventional sialoendoscopy may be undertaken to remove any stones. Stents have also been placed. More aggressive treatment is required for patients with persistent problems. This includes removal of the entire gland.

22.6.5 Fungal and Parasite Infections

Fungal infections of the salivary glands are rare, but have been reported. These include infections with Candida, Apophysomyces and Rhizopus spp. Fungal infections usually occurs in debilitated and severely ill patients. Diagnosis is made following microscopy and culture of purulent discharge from duct or pus from an abscess. Treatment involves antifungal medication and occasionally removal of the involved gland. Parasite infections are very rare, but reported cases include infection of the parotid with toxoplasmosis and nematode larvae.

22.6.6 Granulomatous Infections

Granulomatous infections can be caused by mycobacterial diseases (tuberculous and atypical forms), actinomycosis, cat scratch disease, and tularemia. These usually present with asymptomatic enlargement of a nodule within a gland, sometimes raising suspicion of a tumour.

22.6.7 Pneumoparotitis

Although this is not an infection, it can predispose to one. This is an uncommon condition, most often seen in wind instrument players and glass blowers. Air is forcefully blown into the salivary ducts of one or both parotid glands. This can then become infected. Normally the duct orifice functions as a valve to prevent air from entering the gland. However, the repetitive high pressures and an incompetent valve can result in insufflation. Diagnosis is usually self evident based on the history of immediate painful swelling on vigorously blowing through the sealed mouth.

22.7 Non-infective Swellings of the Lower Jaw and Associated Lumps

22.7.1 Salivary Gland Pathology

Salivary gland pathology can either be obstructive, infective or neoplastic in origin. This is discussed elsewhere. Regardless of the type of the pathology, salivary gland disease can present with swelling and pain. This pain can sometimes mimic jaw pain because of the intimate relationship of the major salivary glands to the mandible.

22.7.2 Cystic Lesions and Tumour-Like Growths of the Mandible

These overlap with cysts and tumours arising within the upper jaw and so are also mentioned in that chapter. Embryologically both jaws are derived from the same structures which explains the peculiarity of some of these lesions which are only found in the jaws. Odontogenic cysts and tumours of the jaws develop from tooth-producing tissues or their remnants. These comprise a wide range of lesions, both histologically and in their behaviour. Both ectodermal and mesenchymal odontogenic tissues can undergo cystic or neoplastic change, giving rise to a bewildering variety of lesions. Cystic jaw lesions can be divided in many categories. These include (1) epithelial or non-epithelial, (2) odontogenic or non-odontogenic, or (3) developmental or inflammatory in origin. From a practical point of view, most cysts are radicular cysts (also known as inflammatory cysts). These are related to the root of a non-vital tooth. If there is no tooth present a residual cyst, keratocystic or odontogenic tumour may be present (Figs. 22.6 and 22.7).

Slow growing cysts often present as a painless firm swelling of the jaw, but they can present acutely following infection. This arises when oral bacteria gain access to its cavity. Larger cysts may present acutely following a pathological fracture. Importantly, some cystic lesions may be malignant or infiltrative in nature. These can present with paraesthesia of the inferior alveolar nerve—an important symptom

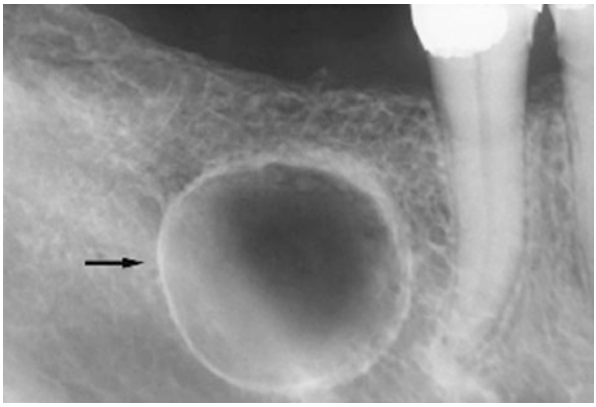

Fig. 22.6 Residual cyst, mandible; asymptomatic, teeth were extracted many years previously. Intraoral view shows round radiolucency with sclerotic border (arrow)

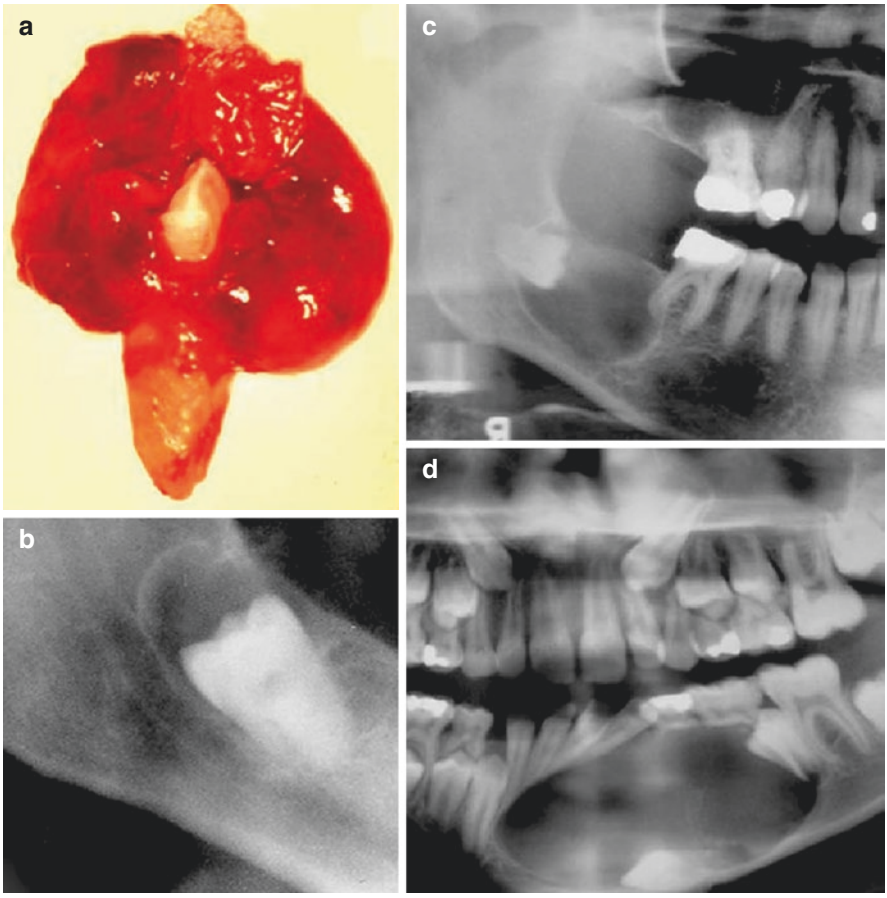

Fig. 22.7 The dentigerous cyst develops within the dental follicle space resulting in a soft tissue sac surrounding the crown of the affected tooth and attached at the enamel-cemential junction. (**a**) Photograph of extracted tooth with attached dentigerous cyst. (**b**) Small dentigerous cyst surrounding a distoangular third molar impaction. (**c**) Horizontally impacted third molar tooth with medium-sized dentigerous cyst. (**d**) Panoramic detail of large dentigerous cyst from an impacted left mandibular canine. The affected canine has been displaced to the lower border of the mandible. There is displacement of adjacent teeth and marked resorption of the roots of the primary molar teeth adjacent to this lesion

that should raise suspicion. Some apparent cysts on an Xray may also be AV malformations within the bone. These have major implications if an adjacent tooth needs to be extracted. In most cases an orthopantomographic (OPT) can visualise the extent of the cyst. With large cysts, a CT may be required. Key features to note are (1) the degree of lucency of the lesion, (2) its size and shape ('soap-bubble' appearance), (3) the surrounding border, (4) its relation to adjacent teeth and the inferior alveolar nerve, (5) displacement or resorption of teeth and (6) the presence of any opacity within the lesion. The differential diagnosis is long but commonly

includes keratocyst, central giant cell granuloma, ameloblastoma, calcifying fibroma, myxoma, AV malformation and multiple myeloma.

22.7.2.1 Odontogenic Keratocysts (OKC)

These are an important and common group of cysts. They are benign, unilocular or multicystic lesions, with a characteristic parakeratinised squamous epithelial lining and the potential for aggressive and infiltrative growth. They have high recurrence rates and are regarded by many specialists as keratocystic odontogenic tumours. OKCs are strongly related to genetic factors and can be a feature of the Nevoid Basal Cell Carcinoma Syndrome (NBCCS), also known as Gorlin-Goltz syndrome. These cysts are believed to originate from odontogenic epithelium or the basal cells of the oral epithelium. Remnants of the dental lamina (from which the tooth buds develop), are thought to be the source of the epithelial cells which eventually proliferate to form the cyst.

Odontogenic keratocysts represent about 10% of all jaw cysts. These may be classified according to their histological features (orthokeratinised and parakeratinised), which behave differently. Most cysts present in patients in their second and third decades, although in Gorlin-Goltz syndrome they can present in childhood (the youngest reported patient was one and a half year-olds). OKC can occur in any part of the jaws, but the vast majority of the lesions develop in the mandible, about half of which occur in the region of mandibular angle. Although OKCs are histologically benign, they are locally aggressive lesions and have a propensity to recur following treatment. They have a tendency to invade the surrounding structures. Once a cyst has breached the confines of the bone and entered the soft tissues, treatment becomes much more difficult. Most OKCs remain asymptomatic for many years until they reach a significant size. Patients may then complain of swelling, pain and a discharge of fluid into the mouth. Involvement of the inferior alveolar nerve may result in numbness of the lower lip. Secondary infection of the cyst usually presents as a ‘dental abscess’. Malignant transformation and development of squamous cell carcinoma has also been reported but is believed to be rare. These tumours are known as primary intraosseous odontogenic carcinomas (PIOC), where the squamous cell carcinoma has arisen within the jaws, presumably from the remnants of the odontogenic epithelium. Other types of odontogenic carcinomas have also been reported.

OKCs typically appear as round or ovoid radiolucencies with smooth, sometimes scalloped (‘soap-bubble’) margins. Several types are described—unilocular and multilocular. If an impacted tooth is seen within the cyst cavity this suggests the possibility of a dentigerous cyst. A useful feature to differentiate OKC from dentigerous cysts is whether the radiolucency is attached to the cementoenamel junction (dentigerous cysts) or envelops the entire tooth (OKC). Expansion of the cortical bone and resorption of dental roots may also occur. CT scan is required with large cysts. Ultimately however, the diagnosis of OKC is based on histological examination of specimens. Management is difficult, controversial and there remains a significant risk of occurrence. This is because of the difficulties in completely removing the thin fragile epithelium and the presence of ‘daughter’ or satellite microcysts.

Since these are benign lesions, radical surgery is not required in most cases. Treatment options include:

1. Enucleation
2. Enucleation with a very limited resection of bone (peripheral ostectomy).
3. Decompression—This may be undertaken with any large cystic lesion of the jaws. The aim is to encourage the cyst to shrink sufficiently to allow complete removal as a second-stage procedure. This reduces the risk of injury to important nearby structures, such as the inferior alveolar nerve. The term 'marsupialisation' is sometimes used, although technically this is a different procedure.
4. Application of Carnoy's solution—this is a chemical fixative which is initially applied to the cyst cavity, 'fixing' the tissues, killing the cells and making them more robust. This is then followed by removal of the cyst. Liquid nitrogen has also been used to treat these cysts.
5. Resection—Although very effective, this is associated with significant surgical morbidity and the need for reconstruction. As such, this is reserved for large or recurrent lesions, lesions involving the condyle and lesions associated with malignant transformation or pathological fracture of the jaw. Any soft tissue which is in direct contact with the cyst wall should also be excised.

22.7.2.2 Odontogenic Myxoma

Odontogenic myxomas are benign tumours derived from embryonic mesenchymal elements of developing teeth. They appear to originate from the dental papilla, follicle, or periodontal ligament. The evidence for this odontogenic origin arises from its almost exclusive location in the tooth bearing areas of the jaws, its occasional association with missing or unerupted teeth, and the presence of odontogenic epithelium histologically. Odontogenic myxomas are usually found in younger patients (10–35 years). Although intraosseous myxoma has been reported in various anatomical sites, the majority of these tumours occur in the mandible, followed by the maxilla. Tumours may appear multilocular or unilocular on OPT, sometimes quite large. Patients generally develop a painless, slowly enlarging expansion of the jaw. Since pain and hypoaesthesia are not common, the lesions may reach a considerable size before patient presents. Larger lesions may cause tooth displacement and bone expansion, later with loosening or displacement of the teeth. As the tumour expands, it frequently infiltrates adjacent structures notably the ramus. These require surgical resection, tumours are not radiosensitive.

22.8 Tumours and Other Growths of the Mandible

Tumours that are derived specifically from bone are generally classified according to the matrix that their cells produce. Tumours that produce a cartilaginous matrix are classified as chondrosarcomas, those that produce bone (osteoid) are classified as osteosarcomas. Other tumours that lack this distinct matrix are classified as fibrosarcomas. A further group of tumours whose cells are of dental origin can also occur

in the upper and lower jaws. Many different types exist, but ameloblastoma is perhaps one of the more important examples. Chondrosarcomas and osteosarcomas are the most common sarcomas involving the facial skeleton. Other rarer types include Ewing's sarcoma and peripheral primitive neuroectodermal tumour (pPNET), malignant fibrous histiocytoma (MFH) and angiosarcoma. The pathology of all these tumours is complicated and interpretation of biopsy samples is a highly specialist area of expertise.

In general terms, tumours of the mandible can be considered as either (1) tumours invading from the surrounding oral mucosa (squamous cell carcinomas), (2) primary bone tumours or (3) metastatic tumours. Primary intraosseous malignancies are much less common than locally invasive squamous cell carcinomas. Their presentation can vary although primary tumours tend to be aggressive. On imaging, any lesion which has irregular ill-defined borders, erosion of the cortical bone and appears to be resorbing dental roots, should be regarded with suspicion. Numbness of the inferior alveolar nerve is a very important sign. With all tumours, patients may present with swelling of the lower jaw and associated cervical lymphadenopathy. Pain is not usually an early feature but this will increase as the tumour grows. In all bony swellings of the mandible it is important to specifically look for (1) numbness of the lower lip (this may indicate infiltration of the inferior alveolar nerve) (2) pathological fracture, (3) limitation in mouth opening (infiltration of tumour into the masseter or medial pterygoid muscles can result in trismus) (4) mobile teeth and bleeding gums. Large tumours may also invade into the overlying skin and present with fistulae.

These tumours require thorough oncological staging imaging and biopsy. If suspected, red flag referral is required. Once the diagnosis is confirmed treatment is then planned based on the tumour type, its stage and the patient's general condition. This is usually done in a multidisciplinary head and neck oncology setting. Tumours that are relatively benign (such as ameloblastic fibromas, ameloblastic fibro-odontomas and cementoblastomas) may be treated by enucleation with or without a limited resection. More aggressive tumours (such as ameloblastoma, calcifying epithelial odontogenic tumour and ameloblastic odontoma) require wider margins of excision. Malignant tumours require radical surgery, reconstruction and adjuvant treatment.

22.8.1 Ameloblastoma

Ameloblastoma is the most common odontogenic neoplasm affecting the jaws, although it accounts for only 1% of all tumours involving the maxilla and mandible. It often develops in young adults. It is a locally aggressive but benign tumour of epithelial origin that is believed to arise either from the remnants of dental lamina, the lining of an odontogenic (dentigerous) cyst, or the basal epithelial cells of the oral mucosa. This tumour tends to invade locally and therefore has a high tendency to recur. Tumours usually present as a slow-growing, painless, bony swelling which may perforate the bone and infiltrate the soft tissues. Ameloblastomas can be classified as

solid or cystic with various subtypes. This is useful in that it has some bearing on their behaviour. Solid tumours tend to be locally aggressive and recur if inadequately excised. Cystic ameloblastoma is much less aggressive. On imaging (OPT) about half of ameloblastomas appear as radiolucent multilocular lesions. Any radiolucency with a 'soap-bubble' appearance should therefore be regarded with suspicion. Only very rarely are these benign cysts. Treatment is primarily surgical, although the most appropriate treatment is controversial. Surgery ranges from conservative treatment (enucleation, curettage, and cryosurgery), to radical resection (Figs. 22.8 and 22.9).

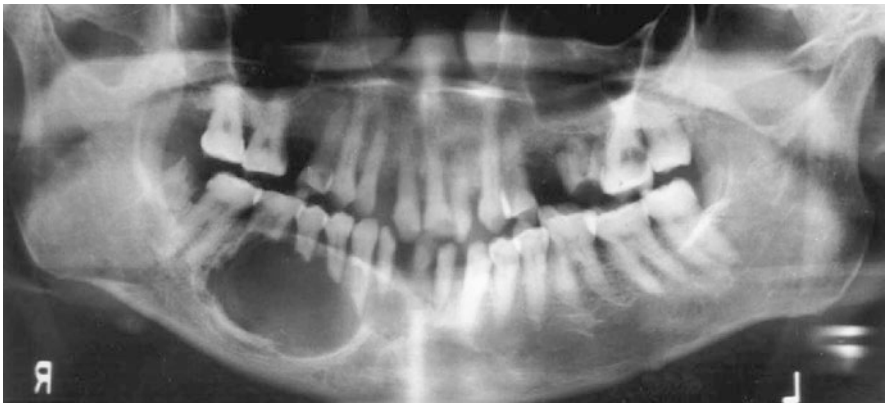

Fig. 22.8 Ameloblastoma in the right mandibular body. The lesion resulted in resorption of the apices of the superjacent teeth, but in downward displacement of the intact subjacent mandibular canal

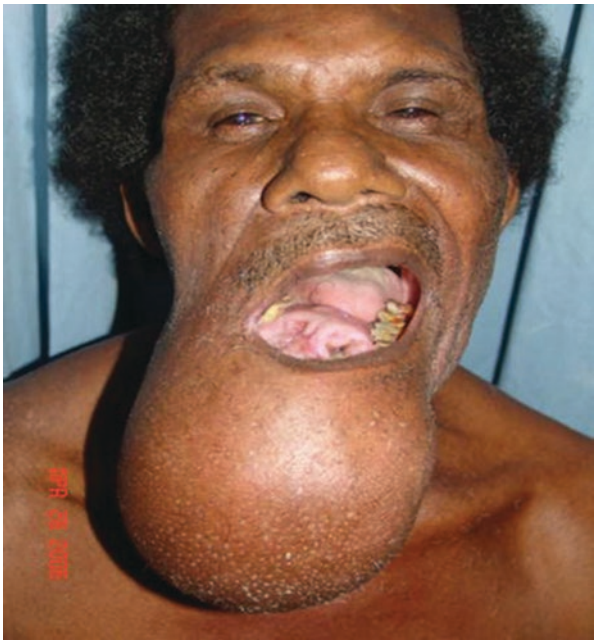

Fig. 22.9 Huge mandibular ameloblastoma involving both halves of the mandible requires hemimandibulectomy

22.8.2 Chondrosarcoma

This is a diverse group of malignant tumours of cartilaginous origin. Most patients present with a painless swelling. These vary from well-differentiated benign cartilaginous tumours to high-grade aggressive malignancies. Surgical excision is the preferred treatment, with adjuvant radiotherapy if necessary. Distant metastases are unusual. The most common cause of death is local recurrence that invades the skull base.

22.8.3 Osteosarcoma

This is a malignant tumour comprised of cells that produce osteoid (primitive bone). Tumours involving the jaws occur in older patients and tend to metastasise late. The mandible is more frequently affected than the maxilla. Most tumours are high-grade but some may be more slow growing. Pain is a common symptom. Surgical resection remains the main treatment of choice, with adjuvant chemotherapy and/or radiation. Recurrences are usually local, but distant metastases, commonly pulmonary, can occur in up to half of patients (Fig. 22.10).

22.8.4 Ewing's Sarcoma/Peripheral Primitive Neuroectodermal Tumour

These are thought to represent a spectrum of tumours arising from the primitive neuroectoderm. They are characterised by both their histological features and by a specific genetic defect. Ewing's sarcoma of the jaw more commonly involves the

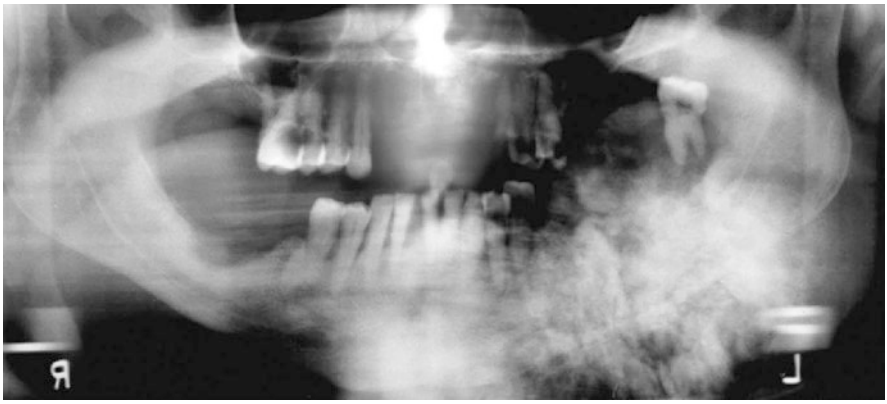

Fig. 22.10 Osteogenic sarcoma of the left mandible. The lytic phase of the lesion has destroyed the outline of the mandibular canal. Note the "sunburst" appearance of new bone formation that is considered a classic, but not invariable, feature of the condition

mandible and presents as an osteolytic lesion with pain and swelling. It tends to affect teenagers mostly. It is considered to be a systemic disease. Treatment includes chemotherapy, surgery and radiotherapy. The 5-year survival of patients is about 75%.

22.8.5 Malignant Odontogenic Tumours

These are rare tumours and may occur as (1) malignant transformation within an ameloblastoma—ameloblastic carcinoma and (2) primary intraosseous carcinoma (thought to develop from residual odontogenic epithelium within the jaws). Malignant ameloblastoma and ameloblastic carcinoma almost always involve the mandible and have been reported to metastasise to the lungs and lymph nodes. Primary intraosseous carcinoma is an aggressive disease that is best treated like squamous carcinoma of the oral cavity that has invaded bone. Surgical resection in all these tumours is usually required and is often combined with radiotherapy and chemotherapy.

22.8.6 Metastatic Tumours

These usually occur in patients between 50 and 70 years of age. The most common primary sites are the breast, prostate, adrenal, colon and kidney. The mandible is commonly involved and about a third of patients present with an oral lesion as the first sign of their malignancy.

22.9 Osteoblastoma and Osteoid Osteoma

Osteoblastoma and osteoid osteoma are generally felt to be variants of the same lesion. Giant osteoid osteoma is a larger version of osteoid osteoma. These are benign but true neoplasms. Osteoblastoma occurs more commonly in the vertebrae and long bones, but has been described in the jaws. Within the head and neck, the mandible is the most common site. This can grow rapidly and present with localised pain. Most cases occur in the second decade of life, rarely after the age of 30 years. Osteoid osteoma is a smaller version of the osteoblastoma. Radiographic features are variable, usually consisting of a combination of a well circumscribed radiolucency and radiopacity. A “sunray” pattern of new bone formation, similar to that described in malignant bone tumours may be seen. Treatment is generally conservative surgical excision. Recurrences are rare (Fig. 22.11).

22.9.1 Mandibular Tori

Tori are painless bilateral discrete bony growths which occur on the inner aspect of the mandible. Prevalence varies substantially between ethnic groups, with lower

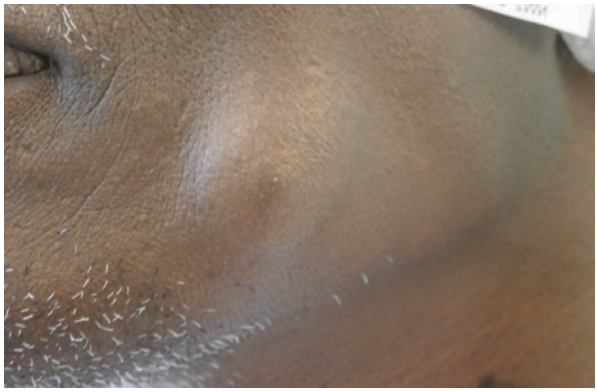

Fig. 22.11 Mandibular osteoma

prevalence in whites (about 8%) and blacks (about 16%) and higher prevalence in Asian and Inuit populations. Torus mandibularis is thought to be caused mainly by environmental factors, such as bruxism, vitamin deficiencies and calcium-rich supplements, although genetic background may plays a role. They are slightly more common in males than in females. Clinically, tori have normal overlying mucosa and are usually an incidental finding. On occasion tori may cause confusion in the edentulous patient. The absence of teeth and alveolar bone can make these appear to be more prominent. They are often felt under the mucosa and can sometimes be confused with salivary calculi, or induration from a tumour. Usually no treatment is required.

22.9.2 Chondroma

These are benign tumours of mature cartilage. They are extremely rare in the jaws. Most occur in the mandibular condyle, suggesting that these lesions may arise from the cartilage of the TMJ. Tumours present as a slowly progressive painless swelling. Treatment usually requires conservative surgical excision.

22.9.3 Synovial Chondromatosis and Osteochondroma

These occur in the temporomandibular joints and may be considered variants of chondroma and osteoma. In synovial chondromatosis there is proliferation of multiple small particulate-sized chondromas within the joint capsule. These present with pain and swelling and also with displacement of the mandible towards the unaffected side. Treatment is excision. Osteochondroma is a benign lesion which may present with a malocclusion or mandibular asymmetry. Treatment is symptomatic but may include surgical excision.

22.9.4 Aggressive Mesenchymal Tumours of Childhood

Children and young adults can occasionally develop aggressive and rapidly growing tumours of bone which, although appearing benign histologically, behave aggressively. The exact nature of these lesions remains unknown. Genetic, endocrine, and traumatic factors have been suggested. Any bone can be affected including the jaws. Although the lesions are benign histologically, aggressive surgery may be necessary. Radiation and/or chemotherapy may also be required.

22.9.5 Central Giant Cell Granuloma

Central giant cell granuloma is a lesion that occurs almost exclusively in the jaws. Although not normally considered an odontogenic lesion, the fact that it only occurs in the jaws would suggest there is some relationship to the teeth or related structures. This usually occurs in the anterior parts of the jaws in the second and third decades. Most lesions follow a benign course, but more aggressive lesions have been reported. Radiographically, appearances can vary (well-defined/ill-defined/multilocular radiolucency, with or without displacement of the teeth). Treatment is usually surgical (curettage). However, there is a 15–20% recurrence rate. Calcitonin and Interferon have also been used with some success. Histologically, the central giant cell granuloma and the brown tumour of hyperparathyroidism are indistinguishable and therefore hyperparathyroidism must be excluded. This is achieved by checking serum calcium, phosphate, and parathormone levels.

22.9.6 Giant Cell Tumour

The giant cell tumour is extremely rare in the jaws. This lesion is aggressive and is considered by some to be a variant of a low-grade osteosarcoma. Treatment is controversial but mainly involves resection (Fig. 22.12).

22.10 Conditions Causing Progressive Asymmetry of the Lower Face

These overlap somewhat with the cysts, tumours and tumour-like conditions described above, but can be considered as a separate group from a clinical perspective. They comprise a large number of conditions that can result in asymmetry of the lower face and mandible and odd appearances on an Xray (usually an OPT). Some include congenital and chromosomal conditions, many of which are very complex and beyond the scope of this book. Other conditions may cause progressive facial asymmetry later in life and may occasionally result in the patient attending urgently.

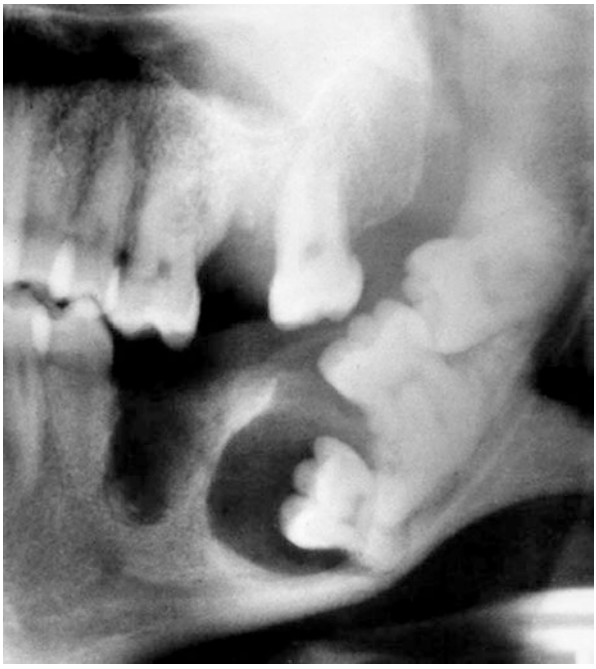

Fig. 22.12 Calcifying epithelial odontogenic tumor causing downward displacement of the mandibular first permanent molar which shows envelopment of the crown by a radiolucency resembling a dentigerous cyst. The mandibular canal is displaced toward the lower border of the mandible

The term "benign non-odontogenic lesion" refers to a mixed group of growths and tumours, which in many cases are difficult to accurately classify. Some of them only seem to occur in the jaws, suggesting that despite the absence of odontogenic structures, they are in some way 'dental' in origin. The list is long and includes fibro-osseous disease, osteoblastoma and osteoid osteoma, benign tumours of bone-forming cells, synovial chondromatosis and osteochondroma, lesions containing giant cells, vascular malformations, Langerhans cell histiocytosis, nonodontogenic cysts of the jaws, neurogenic tumours, Paget's disease, massive osteolysis and tori. Some of these have already been discussed.

22.10.1 Parry-Rhomberg Syndrome

This is considered to be a neurocutaneous disorder in which there is progressive hemifacial atrophy, involving both the soft tissues and bones. The condition usually arises in the first or second decade of life and is slightly more prevalent in females. In the lower face the most common site of initial change is in the buccinator region. It then continues with atrophy of skin, followed by fat, fascia and bone. About half of patients develop trigeminal neuralgia. If the skin and soft tissues are of good quality, management involves corrective jaw surgery and onlay grafts. Where there has been significant atrophy of the soft tissues, free tissue transfer may be required.

22.10.2 Condylar Hyperplasia

This condition affects the mandibular condyles. It has been suggested to be a genetic condition, as nearly one third of patients have a family history for the condition. Symptoms usually commence in the teenage years and progresses slowly into the mid-twenties. The condition is divided into two types—(1) hemimandibular elongation (increased growth in one condyle, resulting in progressive asymmetry and the mandible being displaced away from the affected side) and (2) hemimandibular hyperplasia (in which there is unilateral excessive growth in the condyle and the body of the mandible. This results in an enlarging mandible, with bowing of the lower border on the jaw). Bone scans are often indicated to measure the metabolic activity within the bone. Management is controversial. Some specialists advise a high condylar shave once the condition has been confirmed on bone scan. The rationale is to remove the growth centre and halt progression. However others state that the morbidity of a high condylar shave is too great and that the condition should be allowed to 'burn out' naturally. Once it has been stable for 6 months corrective surgery can be undertaken.

22.11 Benign Fibro-Osseous Diseases

This refers to a number of conditions in which there is replacement of normal bone with tissue composed of collagen and fibroblasts, with varying amounts of mineralised tissue. The commonest conditions include fibrous dysplasia, cemento-osseous dysplasia, and fibro-osseous neoplasms. Not all result in enlargement of the jaws.

22.11.1 Fibrous Dysplasia

This is an uncommon and benign developmental disease of bone. It is caused by a gene mutation which results in abnormal activity of the bone forming mesenchyme. Usually there is slowing of bone maturation during the woven bone stage, with the development of irregularly shaped bony trabecullae, containing mixed fibrous elements. Fibrous dysplasia is classified as either monostotic (where the condition involves one bone) or polyostotic (more than one bone). The monostotic subtype often involves the mandible, although the midface, sphenoid and frontal bones are may also be involved. Albright's syndrome is a specific variant of the condition in which multiple foci of fibrous dysplasia are associated with skin hyper-pigmentation, endocrine disturbances, precocious puberty and/or hyperthyroidism. Craniofacial fibrous dysplasia is defined as fibrous dysplasia confined to bones of the craniofacial complex. In this form the maxilla, zygoma, sphenoid, frontal bones, nasal bones, and base of the skull can all be involved.

The jaws are commonly involved in all types of fibrous dysplasia, especially the maxilla. Onset is usually during the first and second decades. Typically lesions undergo periods of activity and periods of quiescence. When active, they

are often symptomatic resulting in throbbing or discomfort. When the mandible is involved patients may present with pain, progressive asymmetry, a changing bite and paraesthesia of the lower lip. Teeth can become displaced. Radiologically (depending on the stage of the disease) the bone can look sclerotic, pagetoid or cystic in nature, resulting in a range of appearances. The most common appearance is often described as 'ground glass'. Although not normally considered as a premalignant lesion, sarcomatous change has been reported to occur. Some cases may represent a low-grade osteosarcoma from the outset. Otherwise, classical fibrous dysplasia appears to be a lesion that "burns out" when the patient is in the late teens or early twenties. However, cases of active fibrous dysplasia have been reported much later than this.

Treatment is generally symptomatic. If the lesion is asymptomatic, a biopsy may be required to confirm the diagnosis. Treatment in the mandible can then be conservative. Medical treatment involves using bisphosphonates to prevent loss of bone. Surgery may be required to recontour the jaw, or in severe cases resect the diseased portion, which is then reconstructed with free tissue transfer. Surgical treatment is best reserved for quiescent periods because lesions are vascular and can bleed quite profusely. Regrowth can be expected in 25–50% of cases. In rare cases a more aggressive procedure may be necessary including mandibular and maxillary resection.

22.11.2 Cemento-Osseous Dysplasia

Cemento-osseous dysplasia involves the tooth-bearing part of the jaws and is probably the commonest manifestation of fibro-osseous disease. It is usually asymptomatic and requires no treatment. The aetiology of this condition remains unknown, but local trauma may play some part. It is more commonly seen in females and in African Americans.

22.11.3 Periapical Cemento-Osseous Dysplasia

This presents as circumscribed lesions in the periapical areas of vital teeth, commonly in the anterior mandible.

22.11.4 Focal Cemento-Osseous Dysplasia

This presents as a non-expansile radiolucency with associated opacities, often in edentulous areas of the mandible. They often occur in sites of previous dental extractions and may represent some type of abnormal healing.

22.11.5 Florid Cemento-Osseous Dysplasia

This condition has a predilection for middle-aged African American females and presents as a painless non-expansile lesion, often involving two or more jaw quadrants. Many patients are partially or totally edentulous. Radiographically it appears as multiple confluent lobular radiopaque masses in tooth-bearing areas. Lesions may be associated with superimposed infection and osteomyelitis.

22.11.6 Familial Gigantiform Cementoma

Familial gigantiform cementoma is a form of dysplasia which usually involves multiple sites, often the anterior mandible. Variably expansile lesions develop during childhood and can grow rapidly. Treatment is usually surgical being limited to cosmetic recontouring.

22.11.7 Ossifying Fibroma

Ossifying fibroma (cemento-ossifying fibroma) usually presents as a well-demarcated mixed radiolucency and radiopacity with smooth sclerotic borders. These are usually solitary and occur most commonly in the mandible. This is believed to be a true neoplasm that occurs in the third and fourth decades. It appears to be confined to the jaws and other craniofacial bones. Treatment is surgical.

22.11.8 Juvenile Aggressive Ossifying Fibroma

This is a variant of ossifying fibroma, with lesions occurring in younger children and adolescents and with a more aggressive behaviour. Conservative excision is still the recommended treatment, although lesions involving the craniofacial region may require more extensive surgery. Recurrence rates of between 20 and 50% have been reported.

22.11.9 Cherubism

Cherubism is a familial condition in which there are multiple lesions affecting the facial bones. Because of the involvement of the mandible, maxilla and orbital floor, the face has a rounded appearance and the eyes tend to look upward. This results in the patient having a cherubic appearance. Teeth are often displaced. During active periods the lesions are very vascular. Radiographically the lesions appear "honeycombed" and can be very extensive. Treatment of cherubism is usually conservative. The lesions normally stabilise and become less vascular toward the end of the second decade. It is then that cosmetic recontouring can be carried out (Figs. 22.13 and 22.14).

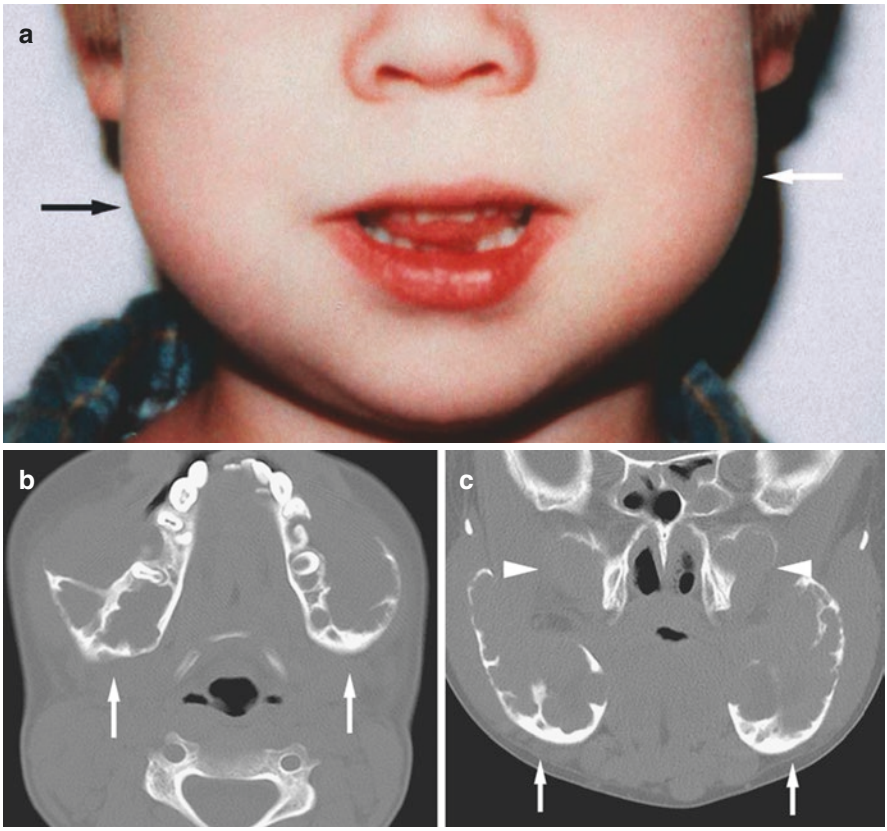

Fig. 22.13 Cherubism; 6-year-old male with gradually increasing swelling of face and mandible bilaterally. (**a**) Clinical photograph shows bifacial swelling (arrows). (**b**) Axial CT image shows expanded jaw radiolucencies bilaterally (arrows). (**c**) Coronal CT image confirms bilateral jaw expansion (arrows). Note also bilateral expansion of maxilla (arrowheads)

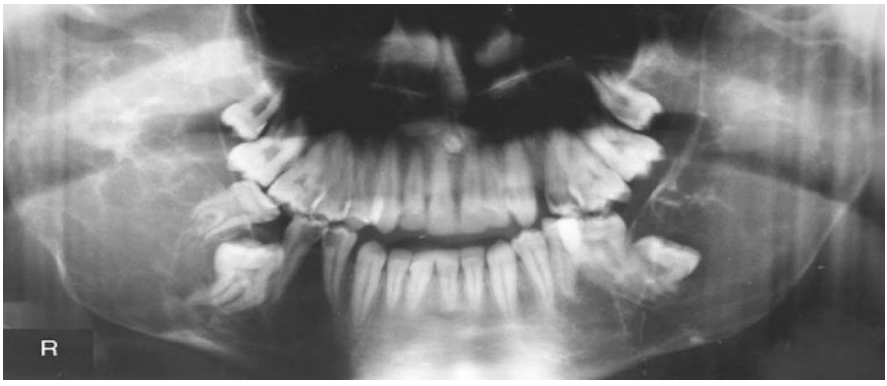

Fig. 22.14 Cherubism. The trabecular patterns within the bilateral lesions of the mandible obscure the outlines of the mandibular canals in the affected areas

22.11.10 Paget's Disease (Osteitis Deformans)

Paget's disease is a chronic progressive disease of bone remodelling, that is characterised by abnormal bone resorption and deposition affecting either a single bone (monostotic) or many bones (polyostotic) including some in the head and neck. The most commonly involved bones are the pelvic girdle, spine, lumber region, thoracic region and cervical and skull bones. Involvement of maxilla and mandible is relatively uncommon but has been estimated to occur in around 15% of cases with slight predilection for the maxilla. Its cause is still unknown, but genetic and environmental factors may play a role. Viruses, including paramyxovirus, canine distember virus, and respiratory syncytial virus have all been reported to be possible causative agents.

The disease usually affects the elderly and starts with excessive bone resorption, but is later followed by an increase in bone formation. Typically the involved bone is mechanically weaker, larger, less compact, more vascular and is thus more susceptible to fracture than normal adult lamellar bone. The overlying skin of the affected bones can become warm on palpation due to this increased vascularity. In extensive cases patients may go on to develop cardiac problems with high cardiac output, cardiomegaly, cardiac failure and rarely death. Although it is rare for the mandible to be involved, clinical symptoms include pain, deformity, and unexpected fracture, although the initial course of the disease is usually asymptomatic. Acute presentation is therefore unlikely unless a fracture occurs. Clinical examination may reveal excessive warmth, due to hypervascularity and paraesthesia of the inferior alveolar nerve due to bony compression. These symptoms would also suggest infection or malignancy. Diagnosis is usually confirmed on imaging and following bone biopsy. Radiographs show a characteristic cotton wool appearance and other radiographic findings such as well-circumscribed radiolucencies, loss of the lamina dura, pulpal radio-opacities, root resorption, and hypercementosis. Elevated serum alkaline phosphatase levels and hydroxyproline in the urine are also found. Paget's disease cannot be cured, but treatment may be indicated to relieve symptoms. This includes calcitonin, bisphosphonates (help regulate bone growth and reduce pain), analgesics, calcium and vitamin D supplements and rarely, surgery. Mithramycin, a potent cytotoxic antibiotic, can also be used for the resistant cases. Rare complications include osteosarcoma, fibrosarcoma, chondrosarcoma, malignant fibrous histiocytoma, malignant giant cell tumour and lymphosarcoma.

22.12 Myositis Ossificans (Ossifying Pseudotumour)

Myositis ossificans (MO) is a benign condition often seen in the extremities of young patients. It is characterised by non-neoplastic, heterotopic bone formation within a muscle. In some patients it is a reactive and self-limiting process, but in its early stages may be confused with osteogenic sarcoma. The disorder is divided broadly into progressive and traumatic forms. The progressive form is an autosomal dominant disease in which multiple heterotopic ossifications develop in muscles,

fascia, tendons and ligaments, sometimes within families. Traumatic MO is a disease in which muscles become ossified following trauma, burns, surgical manipulation or repeated injury. Haematoma, or damage to the muscle induces an exaggerated reparative process which progresses to ossification. In the head and neck MO is seen more commonly in the masseter, followed by the medial pterygoid, lateral pterygoid, and temporalis muscle. Most cases give a history of trauma resulting in a painful swelling, trismus and dysphagia. Over time this becomes progressively harder, limiting the function of the muscle (with restricted mouth opening). Differentiating myositis ossificans from a malignant bone lesion such as osteosarcoma can be difficult and usually requires imaging and biopsy. Ultrasound is an effective and non-invasive method for detecting foci of ossification. Plain films, CT and MRI and biopsy will also be required. Management depends on the size and effects of the calcified mass. If symptoms are minimal, gentle muscle stretching exercises may prevent further restriction of muscle action. Nonsteroidal anti-inflammatory (ibuprofen or naproxen) can help reduce pain and swelling. Ultrasound and extracorporeal shock wave therapy (ESWT) have been reported to prevent progression of calcification, relieve pain and accelerate healing. Surgical excision of the bony mass is occasionally required if it severely restricts mouth opening or is very disfiguring.

22.12.1 Aneurysmal Bone Cyst

Aneurysmal bone Cysts are non-neoplastic lesions that most commonly involve the mandible, maxilla, cervical spine, and skull. They have also been reported very rarely in the other facial bones, sinuses, larynx, and hyoid bone. They are made up of many blood-filled spaces that do not have an endothelial lining. Radiographically cysts appears as a well- circumscribed “soap-bubble” type lesions. Numerous theories with regard to their aetiology have been suggested. The most popular of these is a relationship to a precursor benign tumour, or some sort of response to trauma. Rapid enlargement is likely to be related to bleeding with the cyst. Treatment involves curettage, although haemorrhage can be a problem.

22.12.2 Vascular Malformations

Intra-osseous mandibular arteriovenous malformations (AVM) are developmental lesions, but in later life they can undergo rapid expansion following trauma, infection or hormonal influences, such as during pregnancy. Although they are rare (5% of all vascular malformations) they are of great clinical importance, particularly in the case of high-flow AVMs. When the jaws are involved dental extractions can result in massive blood loss. Lesions are divided into (1) Simple low (2) Simple high flow lesions and (3) Combined lesions. High flow arteriovenous malformations can present after the second decade as a result of new vessel recruitment. Vascular malformation should always be kept in mind when reviewing radiolucencies on

imaging—this will reduce the likelihood of unwittingly performing a biopsy of an undiagnosed high flow vascular lesion. Radiographically, the differential diagnosis includes central giant cell granuloma, aneurysmal bone cyst, ameloblastoma, odontogenic keratocyst, and odontogenic myxoma. Low flow malformations contain calcifications or phleboliths within them.

If suspected, these lesions will require CT angiograms and then if problematic, interventional radiology for embolisation followed by resection. Treatment has been substantially improved by embolisation, which can considerably reduce the blood flow to the malformation. However, embolisation alone is not sufficient to definitively treat these malformations. This is because any residual blood flow will often lead to progressive revascularisation and alteration of the vascular anatomy, making further embolisation much more difficult. For this reason resection is commonly undertaken soon after embolisation. In an attempt to avoid loss of bone and teeth, some specialists prefer to undertake curettage of the bone lesion and packing with oxidated cellulose, thereby preserving mandibular continuity. This must be undertaken no more than 24–48 h after initial embolisation.

22.12.2.1 Haemangiomas

Haemangiomas within the bone of the mandible are extremely rare. These arise from a proliferation of the vessels within the medullary cavity of the bone. Most remain relatively contained and therefore usually present as a painless, firm swelling. Occasionally the pulsatile nature of a medium to high flow lesion may be present with the sensation of throbbing (which may clinically suggest an infection) or with tinnitus. Some haemangiomas can be more locally destructive secondary to their pressure effects and can erode bone and result in numbness of the lower lip or mobility of the surrounding teeth. Bleeding from the gums, either spontaneously, or following minor brushing is a worrying sign and ends careful evaluation. The main issue here is determining the extent of a lesion and it vascularity. This may have major implications should adjacent teeth need extracting or the patient sustains a fracture. True arteriovenous malformations are rare, but these can result in torrential and life-threatening haemorrhage following dental extractions.

22.12.3 Traumatic Bone Cyst

Traumatic bone cyst (also known as idiopathic bone cyst and simple bone cyst), is usually asymptomatic and a chance finding on radiographs. It occurs most commonly in the mandible, particularly in the posterior region. This appears as a well-defined radiolucency, usually with scalloped margins beneath the roots of the overlying teeth. The cause is unknown but has been suggested to result from intramedullary haemorrhage following trauma, whereupon the blood clot liquefies and is then resorbed, leaving an empty space. On surgical exploration these lesions typically contain straw-coloured fluid and have no lining.

22.12.4 Stafne's Bone Defect

This is also known as a static bone cyst and is often found by chance on a radiograph as a well-defined radiolucency along the lower border of the mandible, below the inferior alveolar nerve. This is caused by an indentation of submandibular salivary gland into the lingual mandible. This can be confirmed by sialography or CT.

22.13 Sinuses and Fistulae Arising from the Lower Jaw

A sinus is an abnormal, blind-ending tract, which opens onto an epithelial surface. This includes any epithelial surface, not just the skin. A sinus can involve mucosa (mouth, pharynx, anus, rectum, vagina), intestinal epithelium, bronchial epithelium, bladder epithelium and so on. A fistula is an abnormal communication between two epithelial surfaces. When a dental abscess is neglected, it can slowly erode through the bone and overlying mucosa to drain intraorally. Alternatively it can drain externally and form a sinus into the skin. Multiple sinuses are uncommon and if present these indicate extensive chronic infection (notably osteomyelitis) or possibly malignancy. Preceding or precipitating events prior to the appearance of the fistula/sinus help make the diagnosis, for example injury, swelling and previous dental/facial surgery, including a history of malignancy or radiotherapy. Any untreated abscess, tumour or cause of non-healing can result in a discharging sinus. Therefore a sinus tract may be the result of:

1. Dental abscess
2. Chronically infected dental root
3. Chronic osteomyelitis
4. A foreign body in the skin
5. An infected osteosynthesis plate
6. An untreated fracture
7. A necrotic lymph node
8. An underlying tumour
9. A carbuncle
10. Congenital cysts

A sinus in the skin usually appears as a small punctum, occasionally surrounded by a small area of induration. If gently squeezed there may be discharge of pus or debris. When the underlying cause (commonly a dental infection) is quiescent, the sinus often appears to heals over, only to recur later. Simply excising this without treating the underlying cause will only result in recurrence at a later date. Management of a sinus is therefore primarily the elimination of the underlying condition. Fistulae may occur if pathology drains both intraorally and onto the skin. These are uncommon and tend to occur in chronic infective, inflammatory or neoplastic conditions. In all cases a microbiological swab should be taken from any discharge. If there is no obvious dental pathology, consider osteomyelitis, MRONJ,

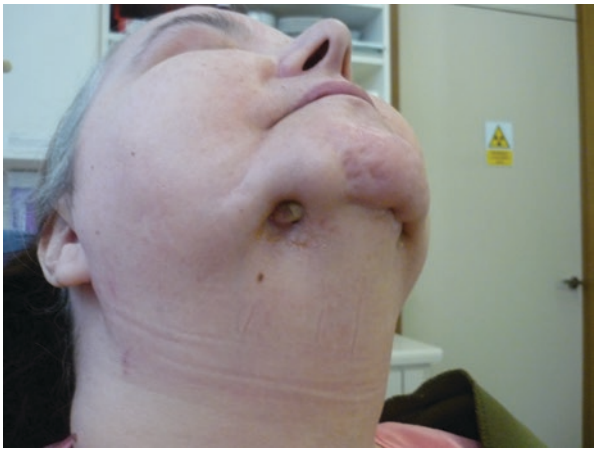

Fig 22.15 Chronic discharging sinus as a result of untreated dental infection

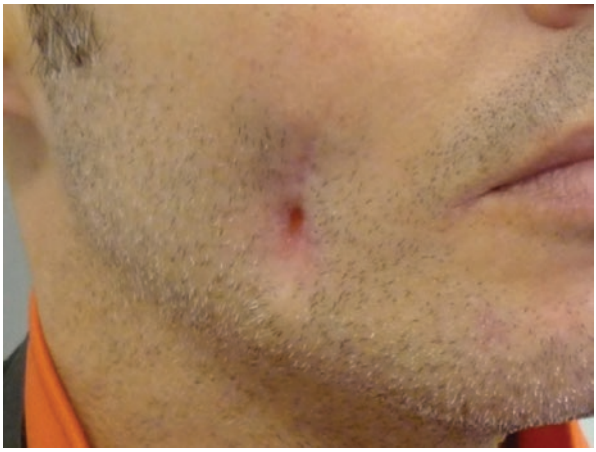

Fig 22.16 Chronic fistula arising from infected tooth

malignancy and actinomycosis. Some of these conditions have already been discussed in this chapter. If the cause is not an obvious a biopsy should be done before any excision of the sinus. Once malignancy is excluded and infection has resolved the sinus or fistula can then be excised and closed if necessary (Figs. 22.15 and 22.16).

22.13.1 Osteoradionecrosis (ORN)

ORN of the mandible is a late, but serious complication of radiotherapy. Patients can present with pain, ulceration, fistulae, fractures and infection. It is characterised by bone necrosis and a failure of the tissues to heal. The lower jaw is particularly susceptible, due to its relatively low vascularity and greater bone density. Radiation reduces the vascularity of the irradiated tissues by stimulating a pathological

process known as endarteritis obliterans. This results in a progressive, hypovascular, hypoxic and hypocellular environment, with diminished cellular activity and poor wound healing. This makes the bone and surrounding soft tissues vulnerable to trauma (including tooth extraction) and infection. If secondary infection develops, it can spread through the non-vital bone. Clinical severity of ORN is wide. This depends in part on the amount of irradiation received and other factors, notably smoking and poor dental health. ORN is a progressive disorder and therefore all patients remain at risk for the remainder of their lives. Patients may remain symptom free for many years with chronic non-progressive ORN. This remains stable with no need for treatment. However complications can develop any time following irradiation, often within the first few years after completion of treatment. Patients may develop a painful non-healing ulcer with exposure of the underlying bone, or sequestrum formation. They may also develop severe limitation of mouth opening as a result of muscle fibrosis. In severe cases patients can present with an orocutaneous fistula, pathological fracture and numbness of the lower lip. Diagnosis is mainly based on these clinical findings occurring in previously irradiated tissues, but biopsy should always be performed to exclude tumour recurrence. Radiological features include a 'moth-eaten' appearance of osteoporotic bone.

Management is based on controlling infection and providing sufficient analgesia and nutritional support. Good oral hygiene, saline irrigations, antibiotics and minimal debridement (removal of visibly loose bone) is often all that is required. Hyperbaric oxygen (HBO) has been reported to be of benefit, but is controversial. This has been reported to increase tissue oxygenation, angiogenesis and thus improved healing. Recently, newer treatments with pentoxyphilline (PTX), tocopherol (Vit-E) and clodonate have shown promising results. Surgery is reserved for persistent or severe cases. This includes resection of non-vital bone (sequestrectomy, hemimandibulectomy etc.), with or without free tissue transfer. Surgery may be indicated in cases of extensive ORN with intractable pain, multiple discharging fistulae, large areas of exposed necrotic bone, or a non-healing fracture. Preventive measures and meticulous dental assessment before and after irradiation, has decreased the incidence of ORN. Dental extractions should be delayed if possible until at least 9–12 months after completion of radiotherapy (Figs. 22.17 and 22.18).

22.13.2 Medicine Related Osteonecrosis of the Jaws

Medicine related osteonecrosis of the jaws (MRONJ), previously referred to as bisphosphonate related osteonecrosis of the jaws (BRONJ), is a complication of treatment with a range of medications but most commonly with bisphosphonate drugs. This commonly affects both jaws, especially the mandible. Bisphosphonates bind to the minerals in bone and inhibit osteoclastic function. Osteoclastic mediated bone resorption and bone turnover are therefore severely disrupted. Because the drug is not metabolised, high concentrations will remain in the bone for a long

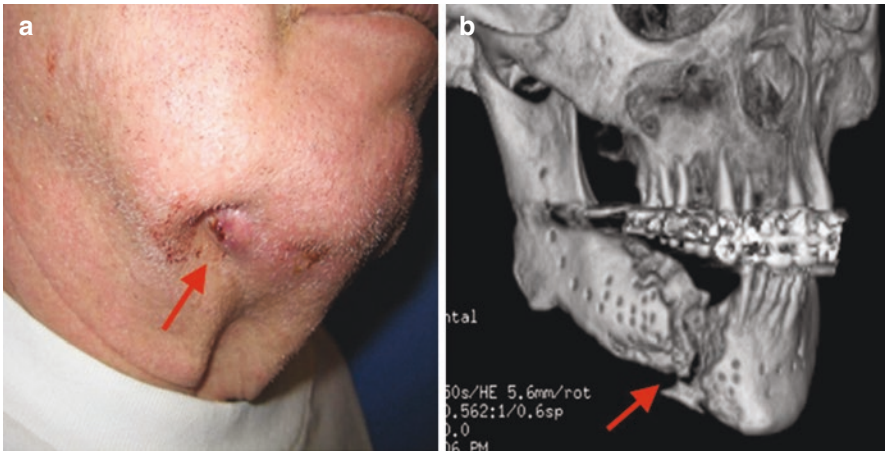

Fig. 22.17 Chronic infectious osteomyelitis superimposed in osteoradionecrosis, (**a**) Oroculaneous fistula (arrow) developing in the setting of chronic infectious osteomyelitis with (**b**) associated pathologic fracture (arrow) of the mandible

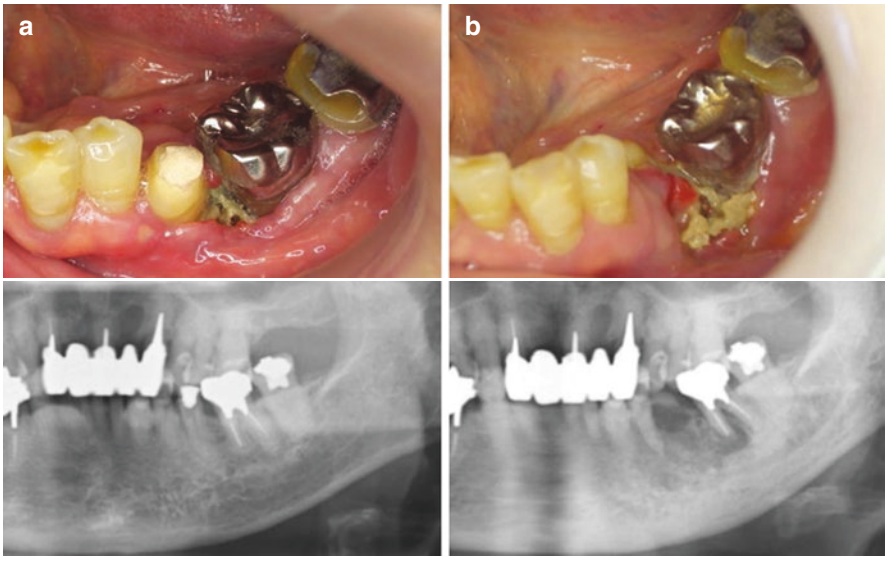

Fig. 22.18 Osteoradionecrosis following radiation therapy. (**a**) The mandible had been in the field of radiation for the floor of the mouth squamous cell carcinoma. One year after completion of radiation, the necrotic bone had developed. (**b**) At 3 years, the osteoradionecrosis continued to increase with frequent bouts of swelling, pain and suppuration. The second premolar exfoliated. Antibiotics were ineffective and a sequestrectomy was performed

period of time. Bisphosphonates are most often used in patients with osteoporosis but are also used to treat bone pain, hypercalcaemia and bone metastases in advanced breast and prostate cancer and multiple myeloma. It may be given intravenously or orally. Commonly used drugs include Pamidronate, Zolendronic acid, alendronate, Clodronate and risedronate. Patients with MRONJ usually present with a non-healing extraction socket or exposed bone with sequestrum formation, localised swelling, loosening of teeth and purulent discharge. They may also present with a pathological fracture. Radiographs usually show regions of 'mottled' bone and sequestrum formation. Depending on the clinical features, the differential diagnosis includes other common inflammatory conditions (such as alveolar osteitis or periodontitis), ORN, chronic osteomyelitis and malignancy. Usually the history of bisphosphonate prescription and clinical features is a enough to establish a diagnosis, but biopsies should be taken to exclude malignancy. Once the diagnosis has been confirmed treatment involves meticulous oral hygiene, antibiotics and removal of loose sequestra. Long term antibiotic treatment may be required. Cessation of the drug should be encouraged if possible—a "drug-free holiday", but this will need to be for several months due to the half life of the drug. Ozone therapy has been reported to offer some benefit, but is controversial (Figs. 22.19, 22.20, and 22.21).

22.14 Pain and Numbness in and Around the Mandible

Pain in and around the mandible may occur as a result of local injury or disease, or it can be referred. Infections and traumatic injuries have been discussed in their relevant sections. The conditions discussed here are local causes of pain that may present acutely. In all of these conditions, the predominant symptom is typically pain, although there may be other symptoms. Some of these conditions are discussed in great detail in the chapter on the mouth.

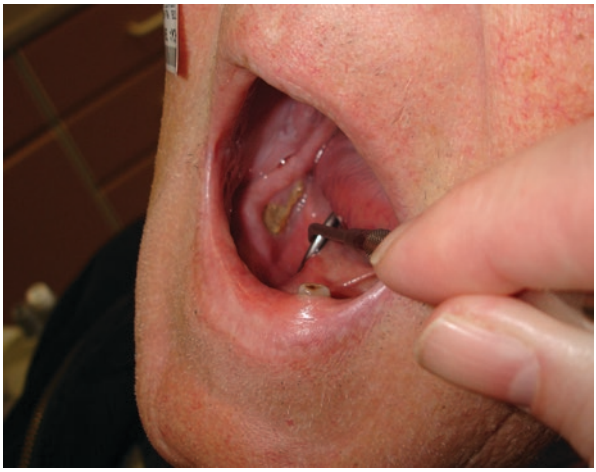

Fig. 22.19 MRONJ (BRONJ) Non vital exposed bone in the right mandible

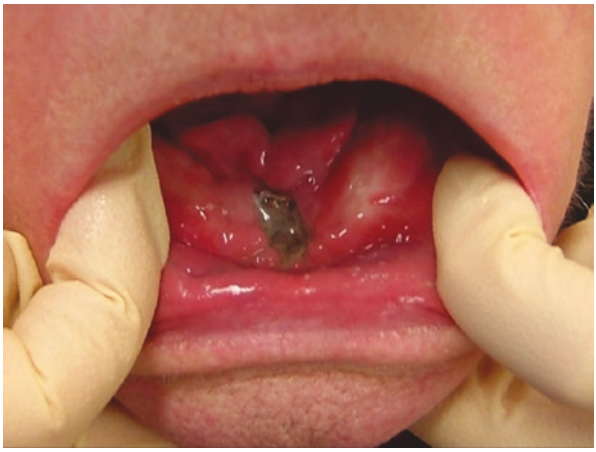

Fig. 22.20 MRONJ (BRONJ) exposed non vital in the anterior mandible

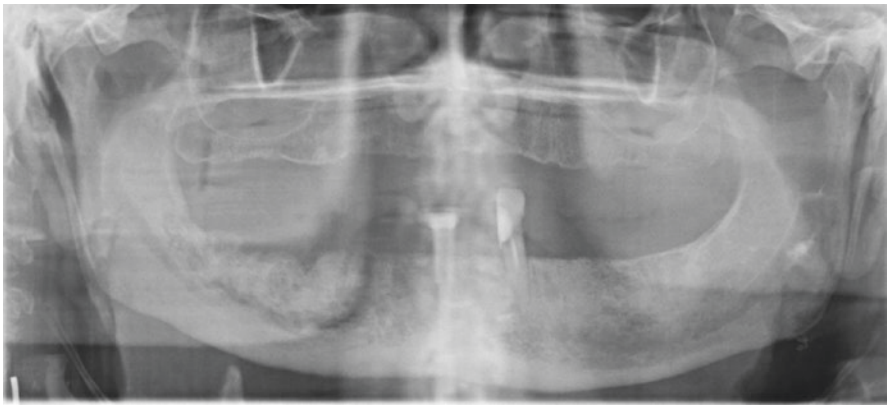

Fig. 22.21 MRONJ (BRONJ) with moth eaten bone in the right body of the mandible

22.14.1 Toothache: See the Chapter on the Mouth

Odontalgia This is a short-lasting diffuse pain, often due to exposed dentine. It is bought on by local stimuli (hot and cold drinks, tooth brushing etc.). The pain can be sharp or dull and is usually mild to moderate in intensity, lasting less than a second to a few minutes. Treatment is usually with a dressing or restoration.

Pulpitis This is pain due to inflammation of the dental pulp. This is also provoked by local stimuli. It can vary in character, and can be perceived as sharp, poorly localised, dull ache, or a throbbing pain. Intensity can vary from moderate to severe, lasting minutes or hours, with episodes that may continue for days. Spontaneous resolution of pain suggested that the tooth pulp has died. This may then change to a periapical-type pain (periapical periodontitis). Treatment requires removal of the

pulp or extraction of the tooth, and analgesia (NSAIDs or paracetamol). Antibiotics are not always required.

Periapical Periodontitis and Abscess These cause a severe throbbing pain arising from the periodontal tissues. It is continuous and well-localised discomfort, typically aggravated by pressing on the tooth.

Atypical Odontalgia This is also known phantom tooth pain, or neuropathic orofacial pain, and is characterised by chronic pain in a tooth or teeth, or in a site where teeth have been extracted or following root treatment, but without an identifiable cause. The pain is often clearly localised by the patient but over time, it may move to another tooth or spread further. It is often described as a severe, continual throbbing pain, which can vary from mild to intense, especially with hot or cold stimuli. Atypical odontalgia may be a neuropathic-type pain or it may signify psychological issues. Some studies have found an association between atypical odontalgia and depression and anxiety. However, the significance of this is unclear. Counselling, avoidance of unnecessary dental treatments and antidepressants form the basis of treatment.

Dry Socket Dry socket is amongst the most commonly encountered complications following extraction of teeth, especially from the mandible. It can have several causes, most commonly, loss of the clot from a socket. Patients who have required surgical removal of the tooth, particularly a mandibular wisdom tooth are most likely to have a fair amount of post-operative pain. The incidence of dry socket following removal of mandibular third molars is around 10–20% and is believed to occur as a result of breakdown of the initial blood clot, possibly as a result of a low-grade infection. The underlying bone then becomes exposed and very painful. This is more likely to occur in patients who smoke and in female patients who take oral contraceptives.

Dry socket typically presents at 24–72 h following dental extraction. Thus patients present with worsening symptoms after 1–3 days. In such cases dry socket, retained roots, or local infection should be considered. An x-ray should be taken to check for residual root fragments and rule out any pathological fracture of the mandible. It is also important to exclude an abscess, cellulitis, fever, lymphadenopathy and trismus, as these may require admission for intravenous antibiotics. The pain is usually intense, throbbing and a constant ache. Exposed bone may be seen and there may be halitosis. Management involves irrigation of the socket, gentle débridement, placement of an sedative dressing and antibiotics. Alvogyl is the most common dressing. This contains butamben (anaesthetic), eugenol (analgesic) and iodoform (antimicrobial). In the majority of cases these local measures are satisfactory and the socket heals in 2–4 weeks.

22.14.1.1 Herpes Zoster (Shingles)

Shingles is an acute herpetic infection which can affect any sensory dermatome, but which commonly affects the Vth cranial nerve. It is a secondary manifestation of a

previous Varicella Zoster (VZ) virus infection. This usually affects older age groups and the immunosuppressed. On the face shingles can erupt in any of the branches of the trigeminal nerve, or cervical plexus. If this affects the ophthalmic branch it is very painful and debilitating and is often associated with severe ocular complications. Pain may precede or follow eruptions and this may last from one to several weeks. Post-herpetic neuralgia is chronic pain and skin changes following herpes zoster. There may be burning, or itchy, 'crawling' dysaesthesias in skin. In the acute phase local anaesthetic injections such as bupivacaine, may help relieve severe pain. Transcutaneous Nerve Stimulation (TENS), capsaicin cream, and tricyclic antidepressants are also useful.

22.14.1.2 Trigeminal Neuralgia ('Tic Douloureux') (See Also the Chapter on the Upper Jaw)

Trigeminal neuralgia is a disease most commonly seen in middle aged and elderly patients. It is defined by The International Association for the Study of Pain (IASP), as "sudden usually unilateral severe, brief, stabbing, recurrent episodes of pain in the distribution of one or more branches of the trigeminal nerve". The International Headache Society (IHS) divides Trigeminal Neuralgia into two distinct categories (1) Classical and (2) Symptomatic (or secondary). Classical TN includes patients in which no identifiable cause can be found for their TN, other than microvascular compression of the trigeminal nerve. Symptomatic TN describes those patients in which an identifiable cause can be found, such as a tumour, arteriovenous malformation or multiple sclerosis (MS). Not every patient with TN fulfils the IHS diagnostic criteria and therefore the diagnoses of "atypical" or "type II" Trigeminal Neuralgia has been suggested.

Trigeminal neuralgia is more common in women, with a peak incidence between 50 and 60 years of age. In young patients caution is required before making this diagnosis—it can be an early feature of multiple sclerosis, HIV disease, or a lesion somewhere along the distribution of the trigeminal nerve (centrally or peripherally). Therefore do not make this diagnosis in young patients until they have been fully investigated. The cause of trigeminal neuralgia is probably multi-factorial with current evidence suggesting local nerve compression within the skull base, altered neuronal processing and possibly a demyelinating process. Neurovascular compression syndrome (NVCS) is defined as the direct contact and mechanical irritation of the Trigeminal nerve (TN) by a nearby blood vessel. Other similar compression syndromes have been described and include hemifacial spasm (CN VII), vestibulocochlear neuralgia (CN VIII), and glossopharyngeal neuralgia (CN IX). The transition zone between central myelin and peripheral myelin has been suggested to be an anatomical area susceptible to mechanical irritation.

Patients usually complain of a sharp, intense, lancinating pain, often bought on my touching a specific trigger point. The pain quickly spreads across the distribution of a branch of the trigeminal nerve. This is almost always unilateral, with over 30–40% of patients showing a distribution affecting both the maxillary and mandibular divisions. In about 20% of patients, the pain is confined to the mandibular division. Episodes may last up to several hours. It is not unusual for attacks to

become more frequent and increasing intensity. Management is usually medical, typically using anticonvulsant agents. Trigeminal neuralgia usually responds well to carbamazepine and/or amitriptyline, and a muscle relaxant such as baclofen. Carbamazepine remains the drug of choice with an initial regime of 100 mg three times daily being gradually increased to a maximum of 1200 mg daily, titrated against its effect. However some patients may develop side-effects such as tremor, dizziness, double vision and vomiting. Patients should also undergo regular monitoring of liver function and full blood count. Long-term treatment has been reported to cause folic acid deficiency (with megaloblastic anaemia) and hyponatraemia in the elderly. Alternative agents include phenytoin, sodium valproate, lamotrigine, baclofen. If effective, treatment can then be slowly withdrawn after 3–6 months. Surgical management is occasionally required. This includes cryotherapy to the nerve, alcohol/glycerol injections and neurosurgical nerve decompression. Alternatively Gamma knife (stereotactic radiosurgery) may be undertaken. High resolution imaging is used to provide precise definition the anatomy which enables a focused beam of ionising radiation to irradiate the proximal trigeminal nerve at its entry point near the pons. Results so far have been very promising.

22.14.1.3 Acute Sickle Cell Crisis

An important pathological event of sickle cell anaemia is vascular occlusion. This may involve both the microcirculation and larger vessels. Occasionally patients can present with severe jaw ischaemia and pain that is out of proportion to other clinical findings. Management is that of the crisis and strong analgesia.

22.14.2 Referred Pain

The physiological basis of pain is discussed in the chapter on the upper jaw. In any acutely presenting patient it is important to always be aware of the more common causes of referred pain in the lower jaw. These include cardiac and oesophageal disease and neoplasms of the pharynx, nasopharynx and base of the tongue. Lesions of the ear and eustachian tube and those of the major salivary glands or intracranial lesions may also present with ‘jaw pain’ or ‘toothache’.

22.14.3 Numbness of the Lower Lip

Patients who present acutely with spontaneous altered sensation of the lower lip need careful assessment. Tumours, cystic lesions, osteoradionecrosis, pathological fractures, Pagets disease, central haemangiomas and medicine related osteonecrosis of the jaw can all present with numbness. Such “entrapment neuropathy” is caused by pressure or mechanical irritation from nearby anatomic structures. Iatrogenic injury is an important cause and a common source of litigation. An initial OPT and PA mandible should be taken to exclude a pathological fracture. Further imaging may be indicated, directed by clinical suspicion. Rarely a ‘numb chin’ can be a

presenting feature of a paraneoplastic neurological disorder, associated with breast, ovarian and lung cancer.

22.15 Temporomandibular Dysfunction (Facial Arthromyalgia)

The temporomandibular joint (TMJ) is a synovial ball and socket type joint, the 'ball' being the mandibular condyle, and the 'socket', the glenoid fossa of the temporal bone. A fibrous capsule surrounds and supports the joint. Between the condyle and the fossa is a fibro-cartilaginous disc—the meniscus. This is attached peripherally to the inner surface of the joint capsule and normally moves in harmony with the condylar head. Movement of the joint is bought about by the four muscles of mastication (masseter, temporalis and medial and lateral pterygoids) and to a lesser extent by gravity, all of which act across the joint to effect movements. Of these, the lateral pterygoid muscle is particularly important. This is inserted into both the condylar neck and the anterior aspect of the meniscus and is important in mouth opening. Movement across the joint is complex. On opening the mouth from the closed position, a hinge-type movement occurs for the first 1 cm. This changes to a forward translation movement, in which the condyle rotates and bodily slides forwards and downwards, along the sloping surface of the eminence. This is accompanied by the meniscus. Very little movement of the joint is possible laterally, although some rotation through a vertical axis is possible, to enable lateral excursions of the jaw.

Temporomandibular dysfunction syndrome (TMJDS) is a general term used to describe a variety of problems that can affect these main structures—the masticatory muscles, the temporomandibular joint (TMJ) and adjacent structures. It is probably the most common cause non-dental pain in the maxillofacial region. Temporomandibular dysfunction is not always progressive or destructive. Three types of common dysfunction are described (1) myofascial pain and dysfunction, (2) internal derangement and (3) osteoarthrosis. Tumours of the joint can also occur, but these are rare:

1. Myofascial pain and dysfunction is by far the commonest disorder. It is mostly a muscular-related problem that arise secondary to 'parafunctional' habits, such as clenching or grinding of the teeth (bruxism). Stress, anxiety and depression are well known to be commonly associated. Myofascial pain is characterised by pain and tightness in the muscles of mastication.
2. Internal derangement describes a condition in which the articular disc (the meniscus) becomes sited in an abnormal position or moves in an abnormal way during opening and closing. This results in mechanical interference of the joint with symptoms, such as restriction in movements. This is the most common non-painful type of TMD dysfunction, although it can result in localised discomfort. Disc disorders can result in limitation of movement, clicking and crepitus on opening and closing. Loose bodies within the joint can also result in similar symptoms. These require surgical removal.

3. Osteoarthrosis is a localised degenerative disorder that affects mainly the articular surfaced of the temporomandibular joint. It is usually seen in older age groups. Other types of arthritis can also involve the joint.

The aetiology of TMJDS is a controversial topic, partly because TMJDS is a collective term used to describe a variety of conditions rather than a single one. Temporomandibular disorders involving the muscles of mastication (myofascial pain) are more common in young to middle-aged adults, while disorders resulting in arthritic changes are more common in the elderly. Various aetiological factors have been suggested, including stress, anxiety and sometimes depression, and it is most likely that the condition is multi-factorial, with one cause exacerbating the effects of another. The most prevalent age group is between the ages of 20 and 40 years. Suggested factors in the aetiology of TMJ disorders include:

1. Parafunction—this includes activities such as tooth clenching or grinding—'bruxism' (often subconsciously or during sleep), or abnormal movements of the jaw during function (e.g. exaggerated movement from side to side—'chewing the cud', nail biting etc.). These movements may result in unbalanced forces across the joints and painful muscle spasm.
2. Occlusal anomalies—these are a common feature of patients with TMJ dysfunction. However, there are also many patients who have abnormal bites yet have no TMJ symptoms. Poorly made dentures can also contribute to TMJ dysfunction, especially in patients with a reduced vertical facial height.
3. Trauma, either directly from an impact, or indirectly from wide stretching (e.g. following anaesthesia or dental treatment) has been suggested to result in tears and the development of adhesions in and around the disc. Traumatic inflammation (synovitis) can also occur, resulting in pain and altered function.
4. Stressful life events in patients with poor coping mechanisms are also commonly noted in patients with TMJ dysfunction, compared to non-affected patients. Anxiety neuroses and affective disorders (particularly depression) are common. Psychogenic factors are thought to exacerbate the symptoms perceived by the patient, rather than be a primary cause of temporomandibular disorders.
5. Osteochondral loose bodies are an uncommon finding in the TMJ. Loose bodies within the joint can be divided in three groups: (1) synovial chondromatosis; (2) osteochondral fracture fragments and (3) degenerative arthritis or avascular necrosis. The most common symptoms of loose bodies are localised pain, joint noise and locking. These patients require CT or MR to confirm the presence of the loose body.
6. Developmental defects in the joint (hypoplasia, etc.)
7. Degenerative joint diseases such as osteoarthritis, arthroses
8. Autoimmune diseases including rheumatoid arthritis, psoriasis and lupus

22.15.1 Pathophysiology

Since TMJDS comprises a group of disorders, the pathophysiology depends upon the specific type of disorder.

1. Inflammatory or degenerative joint diseases. Inflammation within the joint leads to progressive joint destruction and remodelling of the bone. Causes for this may be systemic (such as rheumatoid arthritis and other polyarthritic conditions), or localised (following chronic and repetitive "microtrauma" from occlusal imbalance, or excessive loading of the TMJ during clenching. The pathophysiological mechanisms of autoimmune-based arthritis involving the TMJ the same as that found in other joints.
2. The pathophysiology of painful muscle dysfunction is unclear but is thought to involve fatigue in the muscles, with spasm and inflammation. Tightening in these muscles results in pain and an inability to open the jaw fully. However no chemical mediators have yet been discovered.
3. Internal derangement (disc displacement). Direct trauma can result in mechanical displacement of the meniscus. However, many patients with internal derangements report no history of trauma. It has therefore been suggested that chronic inflammation within the joint (secondary to bruxism, or other causes) results in a 'sticky' disc, which no longer moves in harmony with the condyle. Irregular movements are sometimes perceived as 'clicking' of the joint. Untreated, this may progress to the point where the disc itself becomes wedged between the condyle and the glenoid fossa, acting in the same way as a doorstop, resulting in locking.

22.15.2 Assessment and Management

TMJDS is a "diagnosis of exclusion", that is, all other conditions affecting the joint or surrounding tissues (trauma, tumours, infections, degenerative disease etc.) must be confidently ruled out before the diagnosis can be made. In many cases this is possible following a detailed history, careful examination and if necessary with imaging. In some hospitals, a standardised questionnaire is an efficient way to assess patients. Symptoms are common and it has been estimated that up to three-quarters of the general population may develop at least one or more of these clinical features, but only about 5% will actually seek treatment. Key questions to ask include:

1. Site of the pain—localised to the joint or more in the muscles
2. Onset—sudden or gradual
3. Known precipitants (grinding, stress etc.)

4. Clicking, grating noises or locking
5. Change in the bite
6. Any systemic arthritis, rheumatoid arthritis, or other disorders
7. Recent trauma

Examination focuses on the range of movement of the jaw, noting any asymmetry in joint movement, tenderness, crepitus or clicking and swelling or lumps. In some patients the masticatory muscles may be hypertrophic and very obvious clenching of the teeth. There may also be signs of dental (occlusal) wear. These are indicative of bruxism or grinding. In most patients an initial panoramic film (OPT) will provide adequate information to determine whether significant joint changes have occurred. However if there are significant occlusal changes, localised pain, clicking or locking in the joint, or the joint appears abnormal on the OPT further imaging is required, usually CT or MRI. CT is particularly good at looking at the bones themselves, whilst MRI is required if internal derangement is suspected. MRI is now considered the gold standard for detection of derangements of the disc. Abnormal positions and morphology may be classified as:

1. Anterior dislocation with or without recapture upon opening
2. Medial or lateral displacement (seen in coronal images)
3. A “stuck” disc that fails to translate normally while the condylar head moves toward the articular tubercle
4. Abnormal morphology of the fibrous portion of the disk and/or the bilaminar zone
5. Abnormal signal of the fibrous portion of the disk and/or the bilaminar zone
6. Joint space effusions
7. Other fluid collections or unusual findings

Morphologic abnormalities are much better judged by arthroscopy. If autoimmune disease is suspected a full blood cell count (FBC) with differential white cell count, erythrocyte sedimentation rate (ESR) and autoantibodies should be taken. If any of these blood tests are positive, the patient should be referred to a rheumatologist.

Management is complicated and depends on the type of dysfunction. In many patients several problems may coexist. For example, patients with internal derangement may also have myofascial pain. These may require treatments separately or together. Soft or hard splint therapy has been the mainstay of treatment for many years and has been shown to be of benefit in both myofascial pain and disc problems. The aim of the splint is to reduce parafunctional activities. Splints may also be designed to reposition the mandible into a favourable posture to reduce pressure on the displaced disc. Other commonly used treatments include nonsteroidal

anti-inflammatory drugs, soft diet, jaw exercises, use of ice or heat packs and gentle massage. Any clenching or bruxism must be addressed. Teaching the patient to keep their teeth apart and jaws relaxed often results in a significant improvement within a short time. A short course of muscle relaxants may help. In severe cases botulinum toxin "botox" may be injected directly into the muscles of mastication, particularly those that are hypertrophic. However this should be used with caution. These muscles can waste significantly resulting in unsightly cosmetic defects. Patients therefore need to be counselled carefully before giving this. Chronic discomfort has been shown to respond to antidepressants, including amitriptyline, nortriptyline and others, but this is probably best prescribed by a specialist. Treatment of a clicking or locking joint (internal derangement) depends on the status and movement of the disc during opening and closing. In addition to the above measures joint arthrocentesis or disc surgery may be required.