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The Acquired Anterior Open Bite: Possible Aetiologies and Management Strategies

Abstract: An anterior open bite (AOB) is present when there is no vertical overlap between the upper and lower incisors when the buccal segment teeth are in occlusion, and most commonly develops during childhood. This article presents a series of cases in which an AOB has developed in adulthood, together with a discussion of potential aetiological factors and management strategies.

CPD/Clinical Relevance: A review of the potential causes, presentation and management of the anterior open bite, applicable to clinicians in primary and secondary care

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An anterior open bite (AOB) is a malocclusion that occurs when there is no vertical overlap between the upper and lower incisors when the buccal segment teeth are in occlusion.[1] The incidence of AOB in the population varies according to age and ethnic group. Development of an AOB is most commonly observed during childhood, with a reported prevalence of 17.7% of children in the mixed dentition.[2,3] In children and adolescents an AOB may develop due to the underlying skeletal pattern and mandibular growth patterns, dento-alveolar growth deficiency or behavioural habits such as digit sucking or tongue thrusting.[4] When associated with sucking habits, prevalence increases to 36.3%.[2] The development of an AOB is multifactorial and may be associated with conditions such as muscular dystrophy,[5] macroglossia and amelogenesis imperfecta.[6] The prevalence of an AOB in adults is 4%, with an increased incidence in African and Afro-Caribbean populations (5–10%).[4]

However, adults may present complaining of a newly developed AOB. This article aims to discuss the AOB acquired in adulthood with clinical examples. The authors have seen a selection of adult patients reporting apparent changes in their occlusion in whom an AOB appears to have developed. In some of these cases, no aetiological factor was immediately apparent.

We present a systematic approach in the taking of a history, examination, investigations and subsequent management with reference to clinical cases.

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Presentation

The development of an AOB in the adult patient may be first noticed by either the patient or clinician during a routine examination. The patient may describe aesthetic concerns with a newly forming 'gap' between the upper and lower teeth or functional difficulties associated with the inability to bring the incisor teeth into contact (Figure 1). This is sometimes attributed by the patient to a particular event, such as the extraction of a tooth or placement of dental restorations, although it seems more likely that these minor changes to the occlusal scheme draw the patient's awareness to the more gradual

Figure 1. A 50-year-old electrician, who reported that he was no longer able to strip wires with his anterior teeth. He had noticed this develop over several years. Wear facets were observed on the anterior teeth, providing convincing evidence that tooth-to-tooth contact was once possible.

changes that had been occurring. In other cases, the AOB appears to have occurred spontaneously with no 'initiating' event.

Aetiology

An anterior open bite can be broadly divided into two categories: skeletal and dental open bites. A skeletal open bite (that results at least in part from the vertical skeletal form) can be physiological or pathological. In dental open bites, the vertical skeletal pattern is not contributory.

Skeletal open bite: physiological causes

Craniofacial growth does not stop in adolescence[7] but continues into later life, although later changes are small. There is an increase in anterior face height that begins in the third decade,[8,9] with research suggesting that this increase continues well into the fourth decade.[10] In females, continued (facial) growth may accelerate during pregnancy.[11]

The mandible grows downwards and forwards in relation to the cranial base via growth in the ramus and the condyles.[7] Open bites that develop due to excessive vertical growth are termed 'skeletal open bites'.

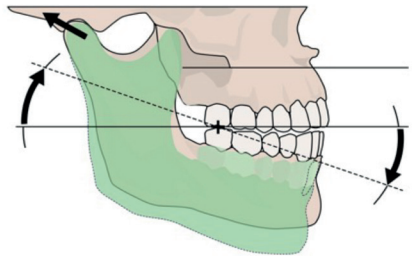

Figure 2 . A backwards rotational pattern of mandibular growth is associated with a centre of growth at the most distal molars and can result in an anterior open bite.

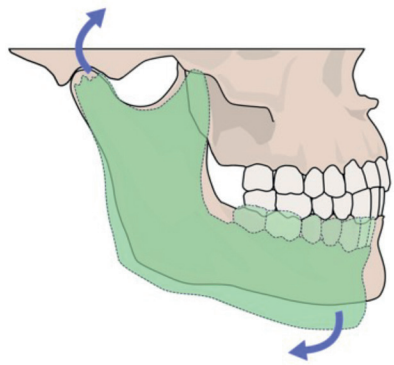

Figure 3. It is thought that as a consequence of condylar resorption and remodelling, the mandibular condyle can reposition over time into a more superior position within the glenoid fossa. This can create a rotational movement of the mandible resulting in a progressive AOB.

AOBs usually develop during growth in childhood or adolescence, in association with a backwards rotational pattern of mandibular growth (Figure 2). This backwards rotational growth is a result of a relative excess of growth of the anterior face height.[11] The centre of rotation for AOB development is typically at the most distal molars.[6] Rotational growth is usually complete by adolescence.

Skeletal AOB: pathological causes

A mandibular or condylar fracture may lead to derangement of the patient's occlusion. In these cases, a clear history of trauma would be expected, and a fracture would be apparent on radiographic examination. Other pathological aetiologies for AOB development are described below.

Condylar resorption

Throughout life, the condyles undergo remodelling to adapt the structure of the articulatory joint surfaces according to the functional demands on the joint.[12] This functional remodelling occurs in response to factors affecting how the joint is loaded, for example occlusal changes following orthognathic or other jaw surgery and trauma. This remodelling does not lead to any occlusal changes (indeed, it may occur as a response to these) or abnormal growth. In contrast, dysfunctional/pathological remodelling adversely affects joint function and occlusion.[13] Resorption and subsequent repositioning of the condyle within the glenoid fossa can lead to an AOB (Figure 3). Condylar resorption can result in altered shape or reduced volume of the condylar head, shortened ramus height, muscle or joint pain and reduced range of jaw movement, and may be caused by several factors (Table 1).[13–15]

Bilateral condylar resorption can cause an AOB, while unilateral resorption causes a lateral open bite on the contralateral side. Diagnosis is generally based upon patient history, examination and imaging (lateral cephalometry, CBCT or MRI). Serial imaging may demonstrate worsening of the condition. Imaging would demonstrate a reduction in size or height of the affected condyle(s), potentially

Causes of condylar resorption
Juvenile rheumatoid arthritis
Post-steroid therapy
Orthognathic surgery
Disc displacement disorders
Avascular necrosis
Idiopathic condylar resorption
Autoimmune and connective tissue disorders, eg scleroderma, systemic lupus erythematosus, Sjögren's syndrome, ankylosing spondylitis

Table 1. Causes of condylar resorption.[17]

Causes of condylar/ mandibular enlargement
Neoplasia, eg osteosarcoma, osteoblastoma, osteochondroma
Condylar hyperplasia
Cystic growth
Acromegaly

Table 2. Causes of condylar enlargement.

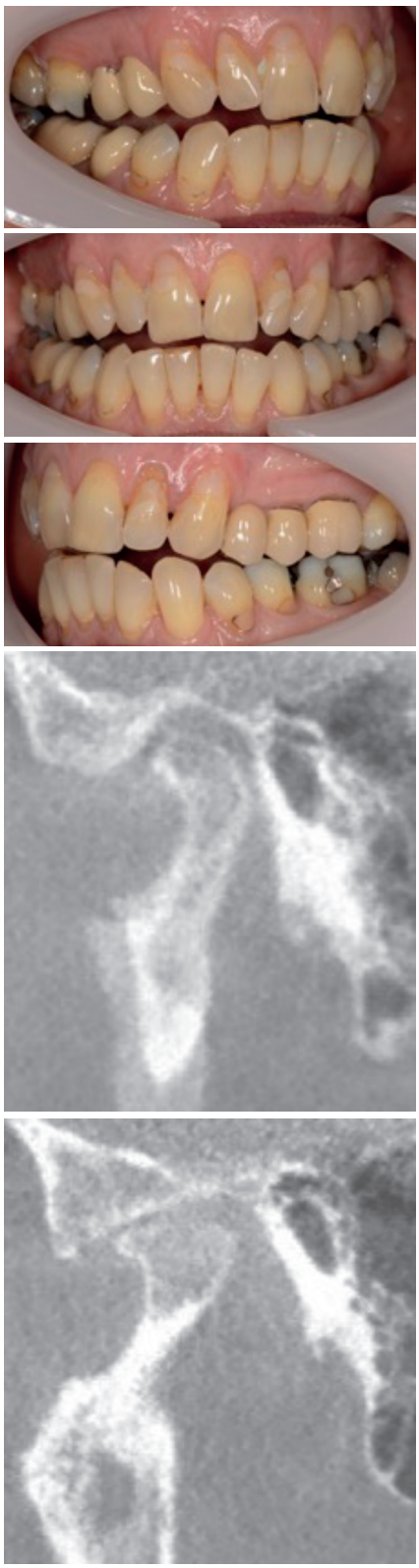

Figure 4. (a–c) Patient presenting with progressive anterior and right lateral open bite, with associated functional difficulties. There was no pain, but crepitus was evident in both joints. The patient had rheumatoid arthritis affecting multiple joints. **(d, e)** CBCT revealed degenerative changes in the left TMJ, with flattening and 'beaking' of the condyle, alteration in the glenoid fossa and articular eminence.

with loss of integrity of the cortical bone.[16] Proposed treatment options will depend on the severity of symptoms and include splint therapy to reduce joint loading, arthroscopy with joint lavage, condylar replacement surgery and orthognathic surgery to correct the occlusal deformity.[16]

Systemic disorders

Degenerative disorders (eg osteoarthrosis, osteoarthritis, rheumatoid arthritis, psoriatic arthritis) affecting the temporomandibular joints may lead to a reduction in condylar height and the development of an AOB (Figure 4a–c).[17,18] Imaging of the condyles in such cases reveals flattening of the condylar head and early osteophyte formation, known as 'beaking', whereby the medial aspect of the condyle appears more prominent and sharper (Figure 4d, e).

Autoimmune disorders such as scleroderma (an autoimmune connective tissue disorder also known as systemic sclerosis) may also lead to condylar resorption and AOB development.[19,20]

Idiopathic condylar resorption

Idiopathic condylar resorption (ICR) has been described and may also be known as idiopathic condylysis, condylar atrophy or progressive condylar resorption. This spontaneous condition[13] is not well understood, although may be hormonally mediated. ICR is most likely to affect females. ICR can affect subjects aged 10–40 years, but predominantly occurs during pubertal growth. Risk factors include a skeletal Class II relationship and high occlusal and mandibular plane angles.[16] Although there is generally anterior displacement of the discs, TMD symptoms are most often mild or absent.

Post-surgical changes

Condylar resorption has been reported to occur in 1 in 5000 patients following orthognathic surgery,[21] particularly in cases including those with a high Frankfort-mandibular plane angle, hypoplastic mandible or short posterior facial height.[22]

Disc displacement disorders

An association appears to exist between disc displacement and condylar resorption, although it is not known if resorption precedes disc displacement or vice versa. Chen reported a series of 13 cases with acquired AOB, who underwent MR imaging.[17] All but one of these patients reported a history of TMJ pain and 11 had a history of a click on opening and/or closing. Two patients had signs of arthritis in joints elsewhere in the body. All of these patients had morphological changes of one or both condyles. All diseased condyles were associated with anterior disc displacement. In this regard, the authors, therefore, concluded that the development of an AOB can be related to condylar degeneration, which is itself associated with anterior disc displacement.

Stewart presented a series of four cases of patients aged 14–53 years with recently developed AOB in whom a diagnosis of 'facial arthromyalgia' was made, and in whom plain radiographic examination revealed condylar resorption.[23]

Condylar enlargement

Open bite and malocclusions can be a clear consequence of benign and malignant lesions of the condyle. Benign and malignant tumours leading to hard tissue formation in the condyle include cyst formation, osteochondroma, osteosarcoma and osteoblastoma.

Osteosarcomas

Osteosarcomas are malignant bone tumours.[24,25] Primary osteosarcomas arise predominantly before the age of 20 due to the highly active growth centres within bones at this time.[24,25] Secondary osteosarcomas generally occur in later life, for example Paget's disease of bone, and post-irradiation therapy.[24–26] Osteosarcoma of the jaw is rare, accounting for roughly 6–7% of all osteosarcomas.[25,26] Osteosarcomas present clinically as a sessile swelling of the jaw and may present with pain. When osteosarcoma occurs in the condyle, symptoms may mimic painful TMD. Treatment of osteosarcoma is usually resection of the lesion, +/- adjuvant therapies, depending on the extent and surgical margins achievable.[25,27]

Condylar hyperplasia

Excessive condylar growth (condylar hyperplasia) in adults has been described, although the causes and

course/limitations of this process are not well understood.[28] Endocrine factors (especially insulin-like growth factors), genetics, metabolic activity, trauma and artherosis are likely to play some part.[24,29,30] It is often difficult to differentiate osteosarcoma of the condyle and condylar hyperplasia, even histologically.[27,31,32] Condylar hyperplasia tends to occur at a slower rate of growth, and radiographic and intra-operative findings are relied upon for accurate diagnoses.[25,31] Single-photon emission tomography (SPECT) may be used to determine osteoblastic activity and thus whether the condyle is still actively growing.[29,30]

Condylar hyperplasia is characterized by excessive bone growth of the condyle, most commonly unilaterally, which stops without treatment.[29,30] Unilateral enlargement of one condyle presents as facial asymmetry, chin point midline shift and resulting crossbite malocclusion, and TMJ pain.[29,30]

Since condylar hyperplasia may be self-limiting, establishing whether the condition is inactive has been advised before commencing treatment. Where hyperplasia is active, treatment is usually by condylectomy. Where hyperplasia has arrested, treatment is via orthodontics and surgical mandibular repositioning.[29,30]

Acromegaly

Acromegaly is a disorder resulting from the production of excessive growth hormone after epiphyseal closure of bone plates.[33] Acromegaly has a prevalence of 40–70/1 million, with an annual incidence of 3–4/1 million.[34] Along with systemic manifestations, acromegaly results in progressive disfigurement of the face and extremities. Oral signs include mandibular overgrowth and prognathism, drifting of teeth and malocclusion, with development of a Class III malocclusion due to growth in the condyle and ramus.[35] Overeruption and hypercementosis of posterior teeth may occur, with the development of an AOB.

The GDP may be the first health professional to suspect acromegaly owing to the oral manifestations described. Where this is the case, an onward referral should be made for assessment via the patient's GMP.

Dental open bite

Continued adult growth and condylar remodelling are usually accompanied by physiological dento-alveolar compensation. In some patients, there is a failure of this process to occur, allowing the development of occlusal changes such as the development of AOB. A comparison could perhaps be drawn in a small proportion of patients undergoing restorative treatment for tooth surface loss in accordance with the Dahl principle. In these patients, the anterior teeth are built up, leading to initial posterior disclusion. While in almost all patients, the posterior occlusion is re-established, this does not occur in up to 6% of cases.[36] In other work, it was shown that 17% of unopposed teeth do not overerupt.[37] Therefore, there are some patients for whom compensatory tooth movement does not occur.

Figure 5. (a, b) This 44-year-old patient presented with an AOB following use of an anterior partial-coverage splint provided by her GDP for management of a TMD.

Figure 6. (a, b) This 57-year-old patient presented with an AOB that both the treating clinician and the patient reported some weeks after replacement of crowns UR7, UL6, LL6. The patient had a CBCT scan to investigate for any changes of the condyles, which showed normal anatomy of the condylar heads.

Orthodontic relapse

Retention of an orthodontically corrected AOB can be challenging. A study examining the long-term outcomes of combined orthodontic and orthognathic surgery treatment demonstrated relapse in around 40% of cases.[38] This may be attributed to vertical growth and eruption of posterior teeth into late teens and early adulthood.[39] Relapse may also occur because of tongue size/posture, unfavourable growth pattern, orofacial musculature, respiratory problems, dental movements, and condylar resorption following orthognathic surgery.[39]

Differential overeruption

The wear of anterior partial-coverage splints may lead to differential overeruption of the uncovered posterior teeth and the development of an anterior open bite (Figure 5).[40] AOB development has been described in 2% of cases with partial-coverage Essix retainers.[41] A patient presenting with a new AOB should be questioned as to whether they have worn any occlusal appliance. With the increasing availability of over-the-counter splints, the patient may not necessarily have been provided with one by a dentist.[42] Partial-coverage splint wear should be discontinued, and a period of monitoring before active treatment is initiated as re-establishment of the previous occlusal contacts may occur.

It may not be excessive wear of a segmental appliance, however, that leads to the development of the anterior open bite. Lifestyle habits can have a significant impact. The clinician should be mindful to consider all possibilities, including those such as thumb-sucking in adulthood.

It has been shown that 83% of unopposed teeth overerupt and around 50% go on to cause a deflective contact or interference.[37] However, it seems somewhat unlikely that this would occur following extraction of an opposing tooth to the extent that an AOB develops spontaneously.

Periodontal disease

Periodontal disease includes the progressive loss of alveolar bone supporting the dentition. When this occurs there can be drifting and spacing

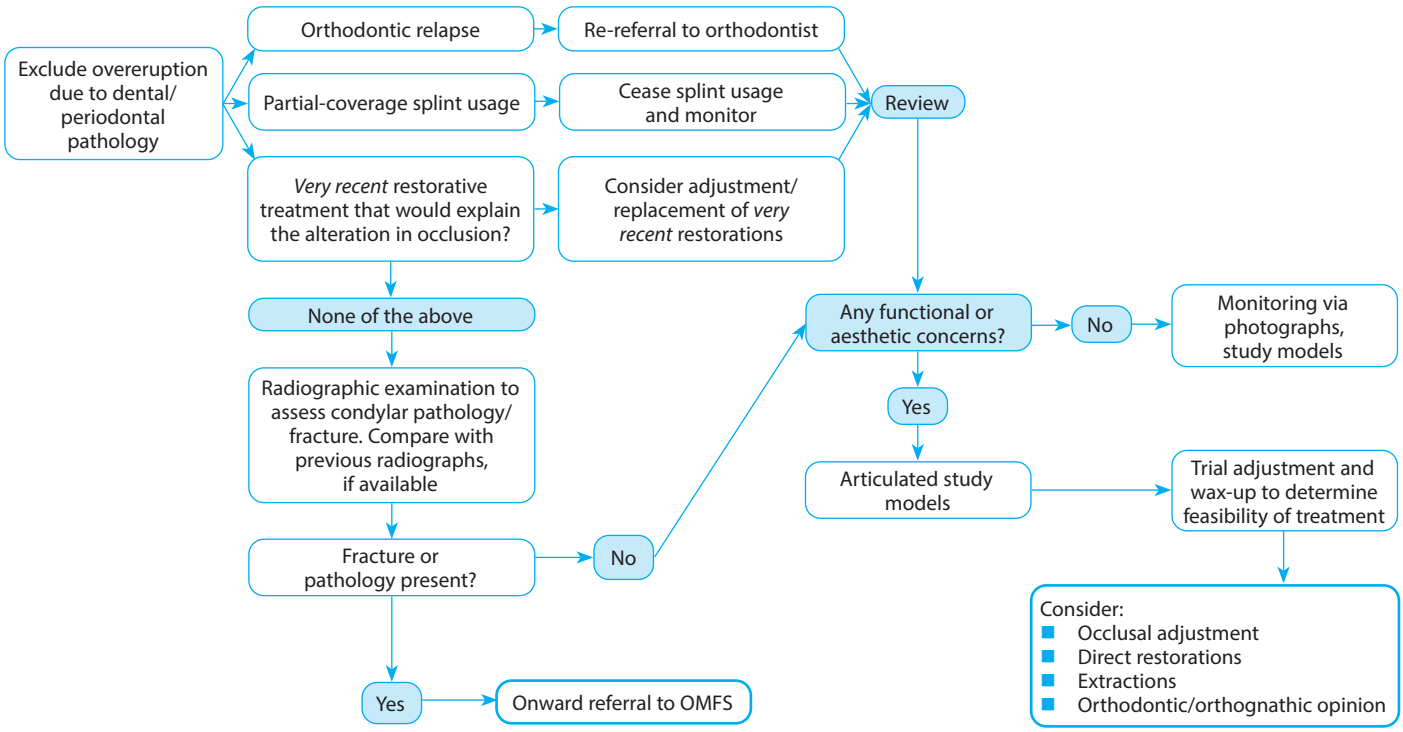

Figure 7. Flow-chart to assist in assessment and planning.

of teeth, which when affecting the anterior dentition may result in the development of an anterior open bite. Treatment should be focused on stabilization of the periodontal disease. Teeth with bone loss sufficient to result in significant tooth movement may not be amenable to orthodontic repositioning, even once the disease is stabilized, and as such, the likely options for management are acceptance or camouflage with selective addition of composite resin.

Extrusion due to peri-apical infection

In the event of an acute peri-apical abscess, inflammatory exudate may lead to extrusion of the affected tooth.[43] This will usually be very tender to bite on and may be associated with sudden AOB development if a posterior tooth is affected.

Occlusal instability

Significant dental work or tooth loss involving teeth that were initial or deflective contacts may lead to occlusal changes (Figure 6). This would therefore change the way the condyle seats in the fossa (albeit that this may not be by much). This has been described as a potential aetiological factor for condylar resorption or remodelling as described above.[14]

Abnormal tongue function and size (macroglossia)

An enlarged tongue (macroglossia) can cause dentomusculoskeletal deformities, such as an AOB, and instability of orthodontic treatment. It may also create masticatory, speech and airway management problems. True macroglossia may be developmental, such as haemangioma, lymphangioma or most commonly muscular hypertrophy. Macroglossia occurs commonly with conditions such as Down's and Beckwith–Wiedemann syndromes. Acquired factors include acromegaly, amyloidosis, myxoedema or a tumour involving the tongue. Acquired factors often require treatment for the underlying cause primarily. The tongue will reach its approximate adult size at the age of 8 years, and later changes may indicate an underlying cause of macroglossia. Most AOBs are not related to macroglossia, and following orthodontic treatment the tongue is very adaptable to changes in the oral volume; however, in the event of true macroglossia, the AOB is likely to relapse following orthodontic and orthognathic surgery. In these cases, a reduction glossectomy may be planned alongside the orthodontic treatment to reduce its volume.[44]

Pseudomacroglossia is a condition where the tongue is of normal size but appears large due to displacement of the tongue by habitual posturing, hypertrophied tonsillar tissue, cysts or tumours, or appearance of the tongue relative to low palatal vault or deficiency in the mandibular or maxillary arches. Pseudomacroglossia must be distinguished from true macroglossia as the primary cause often requires treatment, ie orthognathic surgery to treat a mandibular deficiency.

Once an AOB has developed, the patient may develop an adaptive tongue-thrusting habit to maintain an anterior oral seal when swallowing/speaking. Tongue thrusting was previously thought to be an aetiological factor in the development of AOB, but it is now considered an adaptive behaviour.[11] However, tongue thrusting may inhibit dento-alveolar compensation and perpetuate the AOB.

Management strategies

Radiographic examination is prudent to exclude fracture or pathology of the condyles such as hyperplasia, neoplasia or significant condylar resorption. If there is doubt as to this, referral would be indicated to an oral and maxillofacial

Figure 8. (a–d) This 50-year-old patient presented with an AOB that the patient reported occurring over several months. The patient had a CBCT scan to investigate for any changes of the condyles, which showed normal anatomy of the condylar heads. **(e, f)** Two sets of articulated models with a facebow record were constructed. One set had individual pin dies of the posterior teeth and die spacer was painted on articulated study models to assess whether tooth reduction and selective extractions would be feasible. The order of adjustments on the models is noted to allow this to be replicated clinically. The articulated study models must be verified clinically to ensure these are accurately mounted. **(g–j)** The final result following extraction of the UL8, onlay LL6, selective tooth adjustments of posterior teeth and direct composite restorations on teeth LR6, LR5, LL6 at 3 months follow up.

surgery department. Where there is any concern as to the presence of an underlying systemic condition such as acromegaly, referral to the patient's GMP is warranted, and should occur before undertaking any dental operative management. The GDP faced with cases such as these may well wish to refer the patient for specialist assessment.

If a patient presents with a newly developed AOB, it would be useful to attempt to obtain any previous radiographs to enable comparison of the bony anatomy and assess whether any underlying changes could explain the AOB.

A flowchart to assist in assessment and planning is included in Figure 7. In the absence of functional or aesthetic concerns, active treatment may not be required. In this instance, it would be prudent to record study models and photographs to enable ongoing monitoring.

Occlusal adjustment

Before any occlusal intervention, duplicate study models should be articulated, and trial adjustments performed on models (Figure 8). This will determine the feasibility of this treatment strategy.

Depending on the severity of the AOB and the patient's concerns, selective occlusal adjustment may be the only treatment required (Figure 8 and 9). Occlusal adjustment is not advised in the presence of any painful muscular TMD: in this instance, any adjustment should only be considered following successful stabilization splint therapy (the exception being an obvious interference created by a newly placed restoration).[45]

It is worth noting that occlusal adjustment is significantly easier on study casts than intra-orally – the clinician can readily manipulate the casts and articulator and is not impeded by soft tissues or the patient's tolerance of treatment. The clinician should be aware that any proposed adjustments are demanding for both the patient and operator alike

and, where significant, should be carried out over a series of short appointments to reduce the risk of pulpal sequalae. Further, the extent of adjustment must be weighed against the proposed benefits. Where anticipated, the patient should be aware that reduction of existing restorations, including extracoronal restorations, may necessitate their replacement if excessive thinning, perforation or fracture should occur.

Restoration of teeth

Restorative intervention in these cases is best preceded by diagnostic wax up on study models to enable the height and form of restorations to be planned. It may not be feasible to build up all of the teeth into occlusal contact but the number of occlusal contacts can usually be increased. Teeth may be restored directly using composite resin (Figure 10), or indirectly through minimal preparation onlays or replacement of existing crowns. If the aetiology is unknown or there is a suspected risk of progression, then direct composite restorations are the treatment of choice as further addition may be made should further occlusal change occur.

Extraction of teeth

Where there are only one or two contacting teeth, consideration may be given to extraction. Again, this should be trialled on study models to determine the level of improvement gained and assist in the consent process.

Unless the extracted tooth was causing an interference due to localized apical pathology, extraction may not return the occlusion to its previous state. Additional adjustments or restorations may be required to further improve the situation.

Orthodontic strategies

A joint orthodontic-restorative approach may need to be considered, particularly where a significant AOB is present. Orthodontic treatment strategies would likely aim to intrude the contacting molar teeth, although extrusion of the anterior teeth is also likely to occur. Obtaining sufficient anchorage is an issue in these cases, and temporary anchorage implant-devices may be used. In severe cases, consideration may be given to orthognathic surgery.[46]

Conclusions

The development of an AOB in adulthood may present challenges to the dental practitioner in terms of diagnosis and management. The presence of underlying systemic disorders or neoplasia should be excluded, with consideration given to onward referral for appropriate assessment.

Figure 9. (a, b) This 25-year-old patient presented with a Class II division 2 incisor relationship with a reduced and incomplete incisal overbite. The patient reported that the space between the upper and lower incisors had increased over a short period, complaining of difficulty in eating. **(c, d)** Following selective tooth adjustment the overbite was increased, and more posterior contacts were achieved. The patient was monitored for further changes and was satisfied with the occlusal relationship. After a period of monitoring and preventive advice regarding the non-carious tooth surface loss, direct composite restorations were planned to restore selective posterior teeth, which will, in turn, achieve further posterior occluding units.

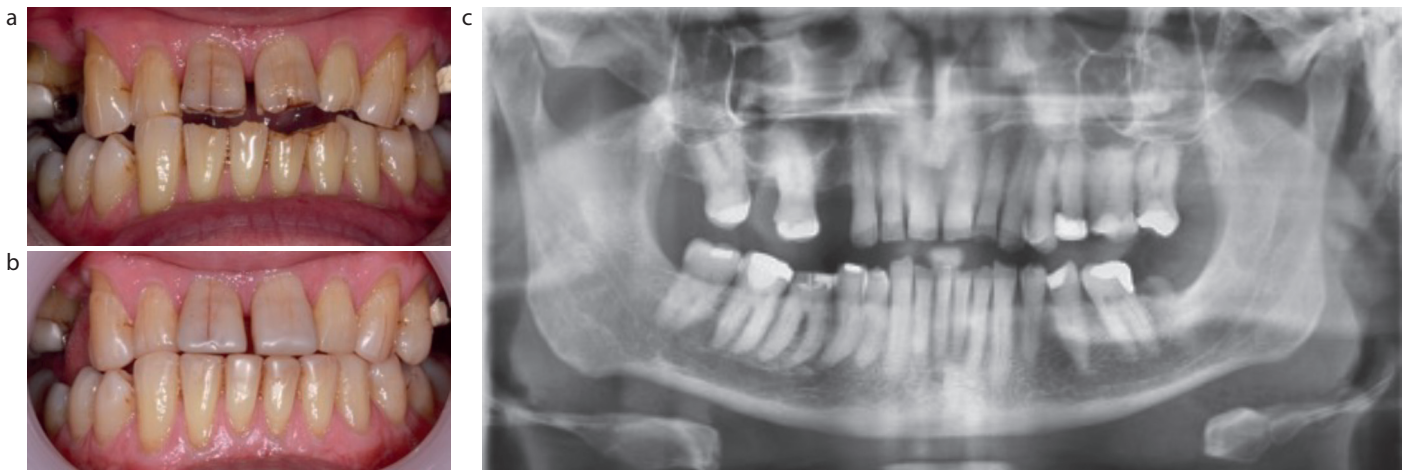

Figure 10. (a) A 63-year-old patient presented with a new AOB. Wear facets were present on the anterior teeth that were no longer in contact. No abnormalities of the condyles were observed, and acromegaly was excluded following testing arranged through the patient's general medical practitioner. Selective adjustments had already been made to achieve contacts on the UR2–LR3 and UL3–LL3 following trial adjustment on articulated study models to assess what was achievable. **(b)** Direct composite restorations were placed based on a diagnostic wax-up to provide even occlusal contacts LR4–LL5. **(c)** The OPT demonstrated an asymmetrical appearance of the condyles (right side larger); however, as there was a clear rotation of the patient (larger molars on the right side) this was not investigated further.

The aetiology for the AOB might not be obvious, but following the exclusion of underlying pathologies, bony changes in the condyles or mandible are the most likely causative factor. Management strategies have been described, which include acceptance, occlusal adjustment, restoration or extraction of teeth. Multidisciplinary care may be warranted in conjunction with orthodontics or oral and maxillofacial surgery. Given many of the changes are idiopathic, or of uncertain aetiology, patients must be informed that the outcome of any proposed intervention is also uncertain.

While this article aims to provide a comprehensive overview as to the possible aetiologies and management strategies of an anterior open bite, the GDP should consider referral of such cases for assessment and/or management by relevant specialities.

Compliance with Ethical Standards

Conflict of Interest: The authors declare that they have no conflict of interest.

Informed Consent: Informed consent was obtained from all individual participants included in the article.

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