Aetiology of malocclusion of the teeth

Malocclusion of the teeth is not itself a disease, although it can be caused by pathological processes or injury, and can predispose to pathological changes such as caries and periodontal disease. It is not peculiar to the human race, having been observed in a wide variety of mammalian species. Some malocclusions involve no more than misplacement of individual teeth, but others, particularly among the primates, affect groups of teeth or even the whole dental arch.[1] Minor degrees of malocclusion have been found in primitive man, and there is evidence that modern man, living under primitive conditions, is not entirely free of malocclusion or irregularity of the teeth, albeit less frequently and less severely than those living in civilised communities.[2–4]

Hybridisation has been shown by Stockard to produce quite severe malocclusion in dogs.[5] It has been suggested by Hunt that one of the effects of civilisation as we know it has been to provide greatly increased opportunities for hybridisation, or intermarriage, and he speculates how much this contributes to malocclusion in modern man.[6]

Normal occlusion of the teeth

Any discussion on the causes of malocclusion should be held against a background understanding of normal occlusion as malocclusion is, by definition, a departure from normal occlusion. As each tooth erupts it is guided by the interdigitation with the opposing teeth into a correct relationship based on the morphology of the teeth. Thus the upper incisors should overlap the lower incisors by about one third of their length, and each upper premolar and molar should occlude a little outside and behind its lower homologue. Thus a tooth by tooth description defines the relationship of each tooth within and between the two arches.[7] In the course of this, the mandible is growing forward relative to the maxilla, a process which should continue well into adulthood. It should be reflected in the way the teeth fit together, but this is likely to be inhibited by interlocking of the tooth cusps when they are not worn down during development.

Local causes of malocclusion

Abnormalities of tooth number, form, or position may cause local irregularities and consequent malocclusion. Extra teeth are found in about 1% of children, and may either resemble teeth of the normal series or have conical or invaginated crowns. Apart from wisdom teeth, congenital absence of teeth occurs in about 4% of children. The condition is often familial. Occasionally missing and extra teeth have been found in the same mouth, suggesting that the two might have a common origin, namely a defect of tooth budding. Extra teeth are usually found in the incisor region, but the upper lateral incisor and lower second premolar are the teeth most frequently missing. Absence of an upper lateral incisor on one side may be accompanied by a diminutive lateral on the other. One might speculate whether they are two stages in an evolutionary reduction of the number of our teeth. A further complication of these cases might be the ectopic eruption of the upper canine when it is deprived of the guidance it normally has from a full size lateral incisor.

Loss of a tooth by extraction or by traumatic injury allows migration of adjacent teeth into the vacated space. If the tooth is a deciduous molar, the successional tooth may become excluded from the arch of teeth. Traumatic injury of a deciduous incisor frequently causes death of its pulp. Subsequent failure of the root to be absorbed will guide the successional incisor to an abnormal lingual position. Anchylosis of a tooth, usually a deciduous molar, prevents it from moving occlusally with the other teeth, and often delays its being shed. As a result the successional tooth erups ectopically. Biederman found this condition to affect homologous teeth of each side much more frequently than opposing teeth.[8] He concluded that the cause is more likely to be disturbance of the process of root absorption and compensating apposition of alveolar bone than to be caused by excessive force on the occlusal surface.[8]

Growth of the facial bones

Any disturbance of jaw growth is likely to be reflected in the occlusion of the two sets of teeth. Factors which determine the way bones grow are therefore relevant to the aetiology of malocclusion. In studying the resemblance of the skull in identical twins, it was observed that the similarity was much stronger for individual bones than for the skull as a whole.[9] It is suggested that there is some lattitude in the way bones fit together in the skull. This is of some importance as the upper and lower teeth are mounted in different bones.

It has been shown in a series of animal experiments that the head can be considered as being made up of mutually dependent functional cranial components.[10] Each component comprises a skeletal unit which has a biomechanical role, and a functional matrix whose activity determines the form of any skeletal elements within it. Thus vigorous use of temporalis muscle will enhance the strength and size of the coronoid process. Indirectly it will also influence the alveolar process by virtue of reactive forces from the teeth when they are brought into occlusion. Because the teeth are part of the skeletal unit they also become subject to some modification. This is achieved by attrition and wear of the occlusal and interproximal surfaces of each tooth.

Abnormalities of dental arch relationship

Although heredity is undoubtedly of importance in determining the relationship between upper and lower dentitions, there is no single gene responsible. Hereditary influences are more likely to be polygenic and may also have indirect influences. One example of this is achondroplasia, in which impaired growth in the cranial base inhibits forward translation of the maxillary complex, causing it to appear retrognathic.[11] Studies on twins have suggested that genetic control may be stronger for facial height than for facial depth.[12 13] Thus cases with extremes of incisor overbite associated with extremes of lower face height appear to have a strong familial tendency. Mandibular prognathism due to excessive mandibular growth is also an inherited character.[14]

Mouth breathing has been shown to cause the mandible to grow in a more downward direction at the expense of some forward growth. At the same time upper and lower incisors become inclined more lingually with a tendency to an open bite and a crossbite.[15] Correction of the mouth breathing as a result of treatment has been shown to allow spontaneous reversal of these effects.

Many congenital malformations involve malocclusion of the teeth. Of these the most frequently seen are clefts of the lip and palate. Evidence of ethnic, familial, and twin studies suggest that heredity plays an important part in the aetiology of clefts of the lip and primary palate, but not in

that of an isolated palatal cleft. Research has shown that unilateral clefts in both animals and man occur twice as frequently on the left side as on the right. At the time the primary palate is being formed, the artery supplying the right side provides the shortest and most direct blood supply from the heart.[16] This suggests that impairment of the blood supply may be a factor in localising the side of the cleft.

Hemifacial microsomia includes some rare congenital craniofacial malformations which occur unilaterally. They are believed to arise from an expanding focal haematoma which forms in the region of the stapedial artery at a critical stage of morphogenesis. This causes non-selective destruction of differentiating tissue in the vicinity of the ear and jaw.[16] On the other hand, mandibulofacial dysostosis includes deformities which occur bilaterally and are thought to be inherited as an autosomal dominant trait. They affect the malar bones, ears, and mandible symmetrically, but there is wide variation in their expressivity, even in the same family. Such massive disturbances of growth have a profound effect on the occlusion of the teeth.

Crowding of the teeth

The most prevalent cause of malocclusion in modern man is crowding of the teeth, which appears to arise from a disproportion of size between the teeth themselves and their supporting bone. Moderate and severe crowding affects about one fifth of British subjects.[17] Comparison of crowded and uncrowded cases has shown that the arch size is smaller in crowded cases, and to a less extent the teeth are larger.[18] Growth of the jaws, in common with that of the body generally may, under adverse nutritional conditions, fail to achieve its full genetic potential.[19] The crown size of teeth is largely determined genetically[20], and is established well before growth of bones is complete. Like other epithelial tissues, such as skin and finger nails, tooth enamel should be subject to progressive loss of substance in the course of function. The amount depends on the physical properties of the diet, and the vigour with which the jaws are used for this and other purposes. There is consistent evidence of appreciable attrition on both the occlusal and interproximal surfaces of the teeth in all specimens of primitive man, and in surviving populations living under stone age conditions.[21] Study of the development of their dentitions has shown that the interproximal wear occurs as the teeth erupt, and as much as 14 mm of tooth substance can be lost from the sum of tooth widths.[2–4]

The interdental space created by the loss of tooth substance closes up as the wear occurs. Experimental evidence suggests that approximal contact between the teeth is normally maintained by continual contraction of the transeptal fibres of the periodontal membrane.[22] This is essential to protect the interdental papillae from damage. The wear causes flattening of the interproximal surfaces in such a way that it enhances the stability of the dental arch.[2–4] Studies of people living under stone age conditions have shown that the reduction of tooth height by occlusal wear is compensated by continual eruption of upper and lower teeth up to the age of 40, thus maintaining the lower face height.[23] The two processes are complementary but are independent of each other. In other studies it has been found that the continued eruption of teeth occurs also in modern man living under civilised conditions, even though attrition has not occurred.[24] There is therefore slow but measurable increase in lower face height, and this is accompanied by some downward and backward rotation of the mandible. The absence of attrition means that the teeth retain their original size, but the jaws are no bigger than those of our ancestors. This is undoubtedly one factor in the aetiology of crowding. Preservation of point contact denies to the modern dentition the stability which should be afforded by flattened interproximal surfaces. As a result a mild irregularity may become worsened as the transeptal fibres of the periodontal membrane contract.

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