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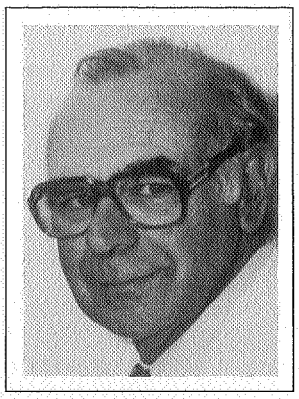

Tooth eruption : an emerging enigma

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Abstract: Tooth eruption is a genetically controlled but little understood event. Standard practice has been to divide it into three phases, (1) pre-eruptive. (2) prefunctional and (3) functional. More recently from an orthodontic point of view a six stage division, three stages prefunctional for individual tooth movement and three stages post functional for the whole dentition has been found more useful for clinical purposes.

The mechanisms of tooth eruption have been extensively investigated experimentally using the continuously growing and eruption incisors of rodents and lagomorphs both in the impeded and unimpeded state. Four principal theories (1) the alveolar bone growth theory, (2) the root growth and pulp cell proliferation theory, (3) the blood pressure and tissue fluid pressure theory, and (4) the periodontal ligament theory have been advanced to explain the mechanisms of tooth eruption but to date with little success. It has been difficult in these studies to identify the forces which move teeth or the controlling mechanisms of this chronologically precise phenomenon. Cause and effect have not been separated and tooth eruption remains an enigma.

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INTRODUCTION

Tooth eruption is a topic which emerges periodically with resurgence of interest by both clinicians and research workers who are concerned with and intrigued by the wide clinical implications and basic biological processes enshrined in this enigmatic phenomenon. Literature on the subject is voluminous particularly in the 1950's and 1970's (see reviews by Beyer 1957; Poole and Stack, 1976; and Moxham and Berkovitz, 1982) but despite much work only limited progress has been made in understanding the mechanisms of tooth eruption, how the process is initiated and what determines the precise chronology of the event. In general terms tooth eruption like other biological processes must be encoded in the genetic message contained in all cells of dentate animals but how this message is expressed and put into action is unclear. In certain genetic disorders in humans e.g. cleido-cranial dyostosis, the teeth develop but many or all fail to erupt. Future study of such disorders may help to identify some of the factors which are involved in tooth eruption.

Definition of Tooth Eruption

Teeth are said to erupt when they move from their point of origin and development in the jaws to emerge through the oral mucosa into the mouth and come into their final position of occlusion with their opposite member and correct position with respect to adjacent teeth. This movement is largely axial but minor movements in other than an axial direction also play a part in the final adjustment. Malplaced unerupted teeth often move in directions other than towards the oral cavity. e.g. an inverted supernumerary incisor (mesiodens) erupting into the nose (Stones 1962) and case reports of unerupted malplaced teeth migrating considerable distances through the jaws. (Sutton 1968). In all cases the movement has always been in the direction of the crown but whether the same forces and mechanisms are operating as in true tooth eruption is not clear.

Phases of Tooth Eruption

Standard convention has been to divide the eruptive process into three phases (Berkovitz 1975).

(1) Pre-eruptive phase: This is the period of development of the tooth in its bony crypt during which the enamel organ forms and mineralizes the tooth crown. During this phase there is peripheral growth and enlargment of the tooth germ and minor movements which readjust the position of permanent tooth germs with respect to their deciduous predecessors.
(2) Prefunctional phase: This phase commences with root formation and is completed when the tooth reaches the occlusal plane. During this phase the tooth moves axialy and occlusally from its place of development, penetrates the oral mucosa and emerges into the mouth.
(3) Functional phase: In this phase the tooth moves into occlusal contact with its opposite number to become completely functional. It is a phase that is seen as continuing throughout the life of the tooth with minor movements constantly occurring to adjust the tooth to occlusal wear.

The movement of a tooth from its place of development into the mouth and finally into occlusion has also been referred to as active eruption whilst the continual uncovering of the crown and later the root by the apical movement of the epithelial attachment has been called passive eruption (Gottlieb and Orban 1938). It is now believed (Steedle and Proffit 1985) that passive eruption only occurs in the human dentition in the presence of disease. In contrast to continually erupting teeth e.g. in rodents and continuously extruding teeth e.g. in sheep eruption in human teeth occurs by additions to the alveolar process and not by addition of tooth to the clinical crown.

Following an horizontal orthopantomographic study of the eruption patterns of deciduous and permanent molars in patients ranging from 2 to 22 years Darling and Levers (1975, 1976) further divided the eruptive process into five stages. However longitudinal studies of tooth eruption (Carlson 1944, Burke 1967, Smith 1980) showed that in Stage 2 when the tooth was moving bodily toward the occlusal plane the eruption rate changed after emergence into the mouth. Steedle and Proffit (1985) therefore suggested that dividing the eruptive process into six stages was more useful from an orthodontic point of view. Thus six phases, three

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prefunctional for individual tooth movement and three post functional for the whole dentition are now proposed and classified as follows.
(1) follicular growth, (2) pre-emergent eruptive (3) post emergent eruptive (4) juvenile occlusal equilibrium (5) circumpubertal occlusal eruptive and (6) adult occlusal equilibrium. According to Steedle and Proffit (1985) this more detailed classification can help in the diagnosis and treatment planning of vertical growth problems.

THEORIES OF ERUPTION

Clearly some form of force is required to move teeth through the jaw tissues and oral mucosal to erupt into the mouth and finally to come into occlusion with their opposite numbers. Despite much research work particularly using the continuously growing rodent incisor little progress has been made in identifying the origin of this force or its nature. This force has been estimated as between 5 to 7gms. using elastic bands of varying tension over continuously erupting rodent incisors (Taylor and Butcher 1951, Miura and Ito, 1968) but the intermittant restraining forces may change the biological system and not truly represent the biological force. More recently intrusive forces of less than 1 gm. have been indicated as sufficient to move teeth (Burn, Murdock 1981, Steedle et, al 1983).

The main theories of the mechanisms of tooth eruption involve either pushing or tractive forces and are concerned with the role of (1) alveolar bone growth (2) root growth and pulp cell proliferation (3) blood pressure/tissue fluid pressure and (4) the periodontal ligament. Most attention is now directed to the periodontal ligament as the source of the eruptive force which may be due to tension within the fibres of the ligament or from the contractile or kinetic properties of the fibroblasts of the ligament. The other theories have largely been abandoned although there has been some renewed interest in the blood pressure thrust theory (Sutton and Graze 1985). However the hypothesis advanced by these workers is supported mainly by theoretical reasoning and until more experimental data is produced it is difficult to evaluate the role that blood vessel thrust may play in the movement of teeth. Certainly there is nothing to indicate how such a constant continuous force is controlled and modulated so that teeth erupt precisely at the correct time and on cue.

In the early movement of teeth from the position of the forming follicle to emergence through the oral mucosa the gubernaculum has been suggested as playing an important role (Hodson 1971). This fibrous tissue band which connects the tooth follicle to the oral mucosa, shortens, broadens and becomes less dense as the tooth erupts. It may present a pathway of lessened resistance to the tooth but clearly has no role once the tooth penetrates the oral mucosa and emerges into the mouth. Fibroblasts and reduced enamel epithelial cells may produce proteolytic enzymes which break down the connective tissues overlying the tooth and then absorb and destroy the waste products produced thus clearing a pathway for the passage of the tooth through the jaws. Such a mechanism could help to explain the timing of eruption. There is evidence (Engel 1951, Toto and Sicher 1966, Melcher 1967 and Ten Cate 1971) that enzymes capable of breaking down collagen, depolymerizing mucopoly-saccharides and disposing of waste products are present in the region between the cusp tip and the oral mucosa during tooth eruption.

Abnormal formation of the oral mucosa, the production of scar tissue etc. may prevent or delay tooth eruption and when these tissues are resected away the underlying tooth often erupts. During eruption teeth take the line of lease resistance and impedance to eruption may be offered by (1) retention of deciduous teeth and their remnants (2) adjacent malplaced permanent teeth and (3) the walls of the alveolar processes.

Much of the research into theories of tooth eruption has used the continuously growing and erupting incisor teeth of rodents and lagomorphs (see reviews by Moxham and Berkovitz,1982). It has been assumed on the grounds that these teeth were a later evolutionary development than non-continuously erupting teeth, that the mechanisms of eruption were the same in both cases. Although a reasonable assumption it is open to question. Of more concern however in these studies is the interpretation and evaluation of data from the impeded and non-impeded tooth respectively and the difficulty of distinguishing between cause and effect.

(1) *Alveolar bone growth theory*

Experimental work using bone, cell and tissue markers has demonstrated little relationship between alveolar bone growth and tooth development and eruption. (See review by Berkowitz, 1975). It has been impossible to distinguish between bone deposition associated with jaw growth and that associated with tooth eruption. In separate experiments using tritiated proline to make new collagen in primate tooth eruption (Kenney and Ramjford, 1969), tetracycline bone markers in rat molar development, and the inferior dental canal as a fixed point in human tooth eruption (Thomas 1965) it has been shown that the amount of axial movement of teeth equalled tooth length plus amount of fundic bone deposited. This suggested that bone deposition plus tooth growth might provide the eruptive force. Deposition of bone on the side walls of the alveolar crypt also might assist in the relocation of the crypt in the developing jaws whilst bone deposition on the alveolar crest during the prefunctional phase of tooth eruption might put tension on the fibres of the dental follicle thereby creating an eruptive force. Similarly bone deposition in the furcation of molar teeth might produce pressure in an axial direction.

There are conflicting results regarding the relationship of fundic bone deposition to tooth eruption, some of which may reflect species differences, and it is difficult to distinguish between bone activity related to tooth eruption and that associated with jaw growth i.e. cause and effect cannot be distinguished. There is no conclusive evidence to support this theory.

(2) *Root growth and pulp cell proliferation theory*

The active stage of tooth eruption takes place during root formation, growth and proliferation of the pulpal tissues suggesting that these events may be the source of the eruptive force (Scott and Symons, 1982). The cell proliferation theory gains some support from the fact that the Sigmoid curve can be applied to both the eruption rates of human maxillary incisors and to simple growth patterns (Burke 1967). Also it has support from the apparent relationship between the circadian pattern of mitotic activity of odontogenic tissues and the circaden pattern of eruption and attrition of rodent incisors (Michaeli and Weinrebb, 1968; Mantell 1973).

A cushion hammock ligament around the base of the rodent incisor was described by Sicher (1942) to protect the underlying bone from resorption by the axial forces generated by cell proliferation. However this structure was not observed in every species and is unnecessary in order to validate the cell proliferation theory. Bone is very resistant to resorption and intermittant forces stimulate bone formation rather than resorption. Fundic bone at the base of upper rodent incisors was not resorbed when eruption was impeded although the basal root tissues buckled. Similar phenomena were rarely observed in lower incisors. The folding was attributed to masticatory forces and was absent in the incisors of lathyritic rats when the teeth were unimpeded. (Berkowitz 1975).

The pulp proliferation theory was supported by increased eruption rates and concomitant increase in basal cell proliferation in unimpeded rodent incisors (Ness and Smale, 1959; Zaficek et al. 1972). On the other

hand experimental destruction of the basal tissues (Berkowitz and Thomas,1969) or transection of the continuously growing rodent incisor failed to prevent eruption of the distal segment which in the unimpeded state reached rates of eruption similar to controls. (Berkowitz 1972).

In the human many upper canines erupt over distances greater than their total length similarly unerupted and/or impacted incisors and canines with fully formed roots often erupt once they are surgically exposed and the obstruction to eruption removed. It has been reported also (Sutton 1968) that human mandibular second premolars have migrated considerable distances through the jaws over a period of years. These findings indicate clearly that root growth alone cannot account for the forces of eruption.

Antimitogenic drugs injected into experimental animals have retarded eruption rates (Berkowitz 1972) but the results are dose dependent and difficult to interpret although they appear to support the cell proliferation theory. Colchicine increases the viscosity of hyaluronic acid and thus the increased resistance of the connective tissues may explain some of the retardation of eruption. The action of these drugs, as is also the case with irradiation, may not be confined to the basal proliferative zone as a similar retardation in unimpeded eruption rates is found in root resected teeth where the basal proliferative zone has been removed and the pulp destroyed (Berkowitz 1972).

Alternatively to the pulp as the organ primarily implicated in the cell proliferation theory, the dental follicle has been proposed as the force generating agent (Ness and Smale, 1959) though little mitotic activity has been reported in the follicle at the base of rodent and lagomorph incisors.

Overall in any of the above work it is difficult to relate cause to effect and there is no substantial evidence that cell proliferation is the eruptive force.

(3) ***Blood vascular and tissue fluid pressure theory***

Blood pressure (capillary rather than arterial) acting at the base of the tooth has been advanced as the eruptive force (Beyer 1957). The tooth forms a closed system which prevents dissipation of blood pressure and the pressure concerned (about 25mm. Hg) is sufficient to move teeth.

Experimental inferference to the nerve supply of dental blood vessels e.g. sympathectomy or resection of the inferior dental nerve which affects the calibre of the vessels and therefore the pressures within them have provided contradictory data (Berkovitz 1975). Some workers have produced increased eruption rates in unimpeded and impeded rat and rabbit incisors whilst in others no effect has been recorded. In some cases the impeded adjacent control incisor showed a decreased eruption rate which could explain the apparent but false increase in the experimental tooth.

Attempts to alter the vascularity of the dental tissues (artery ligation, nutritional disturbances and surgical interference) (Beyer 1957) were equivocal but lateral blood flow did correlate with eruption rates. However tissue fluid pressures were not measured and the significance of the findings cannot be assessed.

A pressure gradient does exist between the pulp (25 mm. Hg) and the periodontal ligament (9mm. Hg) and between the pulp and the tissue fluid of the dental sac (10±5mm. Hg). Forces of up to 15g. could be generated by these pressure differences to move teeth (Van Hassel and McMinn, 1972). However root resection and transection argue against the blood vascular and tissue fluid pressure theory (Berkovitz and Thomas, 1969; and Berkovitz 1971).

Once a tooth has penetrated through the oral mucosa the pressure gradient between the pulp and the periodontal ligament could provide the eruptive force. To date the matter is unresolved.

(4) ***Periodontal Ligament Theory***

The periodontal ligament has been suggested as the source of the eruptive force by virtue of either :
(a) the contraction of its collagen fibres (Thomas 1976)
(b) the intermediate plexus (Sichen 1942)
(c) the kinetic and contractile properties of fibroblasts (Ness 1967, Beersten 1976).

In this theory the tooth is pulled into position rather than pushed. In the case of (a) the forces of eruption are postulated as being generated by contraction of the oblique fibres brought about by cross linking and aggregation of fibres during collagen maturation in the periodontal ligament. This would require a high turnover of collagen in the periodontal ligament and such has been demonstrated by many workers (see review by Sloan, 1982) but this turnover could be in response to tooth movement produced by other forces and not necessarily related to the rate of eruption.

To provide a mechanism for the ready remodelling of the periodontal ligament an intermediate plexus was postulated (Sicher 1942) as a zone existing between fibres of the periodontal ligament attached to the cementum and fibres attached to the alveolar bone. It was described as a zone of less mature precollaginous fibres which allowed the continuous re-arrangement and reattachment of fibres as the tooth advanced during eruption without disturbing or breaking the cemental or bony attachment. Unfortunately there is little histological support for the existence of the intermediate plexus and it may well be an artifact due to fibre orientation and angle of section. There is no evidence of a higher collagen turnover in the region of the intermediate plexus which would be necessary for it to function in the way stated.

Although fibroblast cell migration in the different zones of the periodontal ligament during rodent incisor and molar tooth eruption has been adequately demonstrated and quantitated (Zajicek 1974, Perera and Jonge,1981) and the contractile properties of fibroblasts have also been shown there is little evidence to link these events with the generation of forces necessary for the eruption of teeth. (Shore and Berkovitz 1978, 1979).

In summary then it may be said that to the present whilst mechanisms can be identified which are capable of generating the forces necessary for tooth eruption experimentally none of these mechanisms can be unequivocally and directly associated with tooth eruption. It has been impossible in most cases to distinguish between cause and effect. It is possible that tooth eruption is brought about by multiple events but again evidence is lacking to support this. Even more difficult has been the identification of the controlling mechanisms which regulate these forces to determine the precise chronology of tooth development and eruption.

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