The effects of periradicular inflamation and infection on a primary tooth and permanent successor

Mabel Mariela Rodriguez Cordeiro* / Maria Jose de Carvalho Rocha**

Primary teeth and the permanent successors must be understood as interdependent units, where each one of them interacts with and depends on each other. Pulpal inflammation/infection of a primary tooth and the spread of this condition over the periradicular tissues can lead to alterations in the dental germ of the permanent successor and to the surrounding structures if no therapy is done, i.e. endodontics or extraction. This work will present cases of permanent teeth that showed alteration in eruption and / or in development, as a consequence of inflammation / infection of the preceding primary teeth, such as: hypoplasia, morphological alteration on the dental crown or total arrest of radicular formation. The teeth analysed in this study belong to patients who attended the Universidade Federal de Santa Catarina Children's Dentistry Clinic. The earlier these lesions are diagnosed, the less were the destructive effects and the consequences on the primary tooth / permanent germ unit.
J Clin Pediatr Dent 29(3):193-200, 2005

INTRODUCTION

Primary teeth and the permanent successors must be understood as interdependent units, where each one of them interacts with and depends upon the other. This relationship can be altered by mechanical traumas or as a consequence of dental decay lesions. The homeostasis loss from this unit impacts on the tooth and the surrounding tissues, to different degrees of intensity.

The pulpal inflammation / infection of a primary tooth and the spread of this condition to the periradicular tissues can lead to alterations in the dental germ of the permanent successor and to the surrounding structures, if no therapy is done, i.e. endodontics or extraction. However, alterations that have already occurred in the germ cannot be recovered, regardless of the type of the treatment performed.[3, 7, 14]

Endodontic treatment is the last clinical resort in order to save a tooth with irreversibly damaged pulpal tissue or pulpal necrosis. The tooth, damaged through decay or through traumatic injury, is restored by this treatment to the original function and the health of periradicular tissue is maintained or restored. For this, some signs have to be observed, otherwise extraction and post-maintenance or recuperation of the dental arch space must be done.[14]

Extraction of a primary tooth, besides not curing the damage already caused, can unleash behavioral (conditioning), psychological, mastigatory, orthodontic and speech problems along with the possibility of developing harmful habits.[3]

The consequences of bacterial invasion in the pulpal cavity and the action of the products of the metabolism, along with pulp degradation, can alter the unit of the primary tooth/ permanent germ in an irreversible way. Tissue inflammation can occur, with the forming of abscesses, granulomas and cysts, internal and / or external inflammatory pathological resorptions of the primary tooth root and of the periradicular bone tissue, loss of the gubernacular channel and alterations in the germ formation of the permanent successor teeth.[17, 19]

Besides this, points of infection related to a primary tooth can have a significant effect on the general health of the patient, with the dissemination of the infection to further tissues through the blood (bacteraemia), such as brain abscesses, subacute bacterial endocarditis or even the formation of thrombosis that can lead to blood obstruction and brain ischemia.[15]

When apical periodontitis of primary teeth spreads to the permanent dental germ, it can interfere with formation, leading to injuries that can vary from hypocalcification or enamel hypoplasia, arresting

* Mabel Mariela Rodriguez Cordeiro, Av. Hercilio Luz, 1199- Apto. 102 - Centro, CEP: 88.020-00 1, Florianôpolis - SC - Brazil.

Voice: (48) 223-5533; (48) 9912-229 1

E-mail address: mabelmrcordeiro@hotmail.cOm

** Maria José de Carvalho Rocha, Rua Desembargador Urbano Salles, 111 - Apto. 504B - Centro CEP: 88.0 15-430, Florianôpolis - SC - Brazil.

Voice: (48) 224-2310

E-mail address: mjrocha@newsite.com.br

development of the permanent tooth, dentigerous cyst formation, germ expelling in a similar way to that seen in bone sequestrum in the osteomyelitis, alterations of the follicle with repercussions on the reduced enamel epithelium, in the gubernacular canal and, consequently, on the eruption itself.[5-7,9,11,17,19] The severity of the alteration in the development of a succedaneous permanent tooth germ depends on the stage of calcification (Nolla's stage) and the degree of infection in the primary tooth.[12]

When the development of the enamel is affected, this can manifest itself as hypoplasia, when the injury occurs during the period of deposition of the enamel matrix, or hypocalcification, when damage occurs during calcification and / or maturation of the matrix already deposited.[12]

Alterations in the eruption of a permanent tooth can appear at the time and in the route of the eruption, being related to the destruction of the gubernacular canal through the advance of the infection on the periradicular bone and to the premature loss of primary teeth.[8]

The gubernacular canal houses a cord by the same name, formed by remnants of surplus dental lamina, through which the follicle that includes the permanent teeth maintains connection with the lamina from the oral mucosa. As the permanent tooth makes the eruption movements, this canal is rapidly enlarged through local osteoclastic activity, thus marking, the eruptive path of the teeth.[16]

In order for an extraction of a primary tooth be considered premature, it must occur at least a year before the eruption of its permanent successor, or from radiographic evidence that the stage of formation is below Nolla's stage 6, i.e. complete formed crown.[10]

The presence of a resorbing primary precursor facilitates the eruption of permanent teeth, as it allows the passage through the alveolar bone. The late eruption can be explained through the formation of scar tissue, which forms a mechanical barrier for a permanent tooth, as well as, by the lack of the gubernacular canal. The acceleration occurs in the presence of extensive bone destruction, which is a result of a long period of inflammation caused by an infected deciduous tooth. This results in premature eruption of the permanent successor, often with immature radicular development and, occasionally with the migration of the tooth.[8]

This work presents cases of permanent teeth that showed alteration in the eruption and / or in development, as a consequence of inflammation / infection in the previous primary teeth. These patients attended the Universidade Federal de Santa Catarina Children's Dentistry Clinic.

CASE REPORTS

According to the clinical and radiographic characteristics of each case, a follow- up program was done, at predetermined intervals, with monitoring of the development of each case, through clinical and radiographic examinations, registered in files designed specifically for this purpose. Depending on each clinical situation, additional therapies were done: For primary teeth, endodontics and extraction, and for permanent successors, pulpotomies, aesthetic repairs, space recuperation, dental traction, splints, etc.)

CASE 1

A four years and four months old boy attended the Children's Clinic. He presented with teeth numbers 51, 61, 62, 75 and 85 having extensive decay lesions

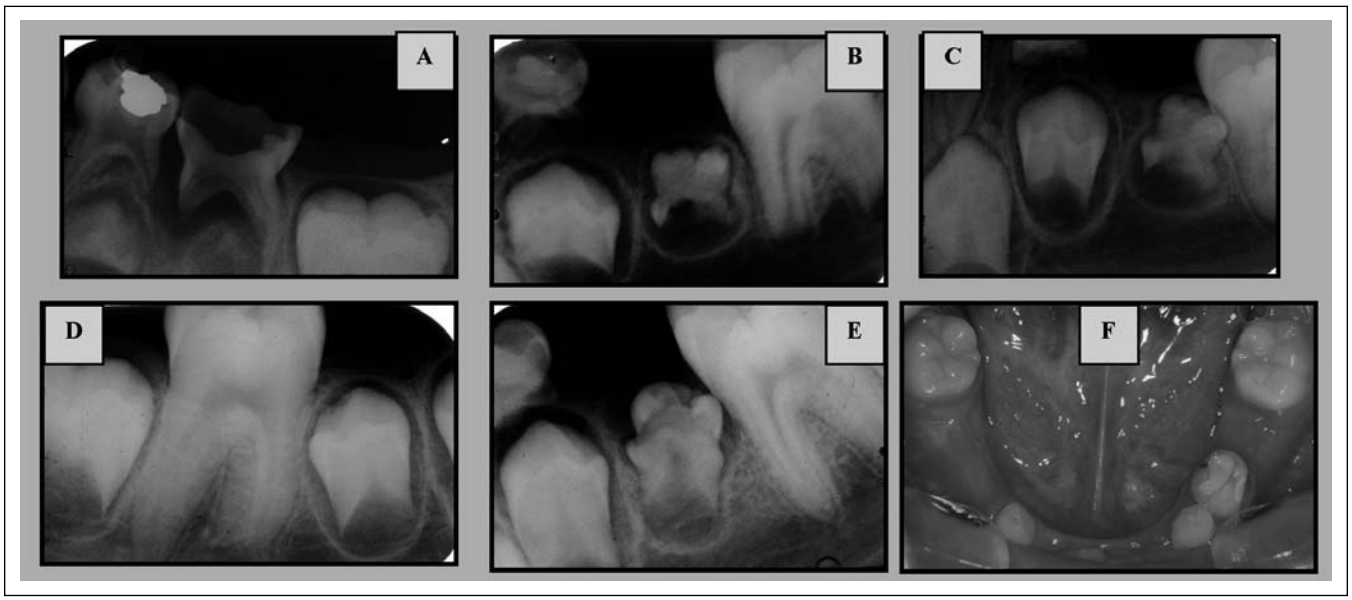

Figure 1. A. Diagnostic radiography of element 75. B. After 34 months of follow-up; C, after 51 months of follow-up. D and E. Latest radiographic aspects, after 71 months of follow-up; F. Latest clinical aspect.

and compromised pulp. All of these teeth received endodontic treatment, except tooth 75, which presented considerable decay lesion, perforation on the pulp chamber floor, making endodontic therapy non-indicated, and the presence of an extensive periradicular lesion (Figure 1A). It was also observed the small degree of development of the permanent tooth germ of number 35 (Nolla's stage 2), with the beginning of calcification of the cusp. (Figure 1A). Tooth 75 was extracted and after 34 months of follow-up (Figure 1B), a disorganized deposition of the calcified tissues in the crown of germ 35 was found, taking on an appearance similar to an odontoma.

In Figure 1C, after 51 months of follow-up, the radiographic image suggested an interruption in the dental development, and for a moment surgically removing number 35 was considered. However, it was instead decided to follow-up, and after 71 months (Figures 1D and E), radiography showed an image compatible with the beginning of radicular formation of the premolar, so it was decided to keep the germ.

Nowadays, planning includes maintaining the dental arch for the eruption of the tooth (Figure 1F), and when this happens, the need for functional and aesthetic recuperation of the dental element is evaluated.

CASE 2

A girl aged 5 years and one month attended the Children's Clinic with damaged pulp due to dental decay in teeth numbers 51, 61, 74, 75, 84 and 85 in which

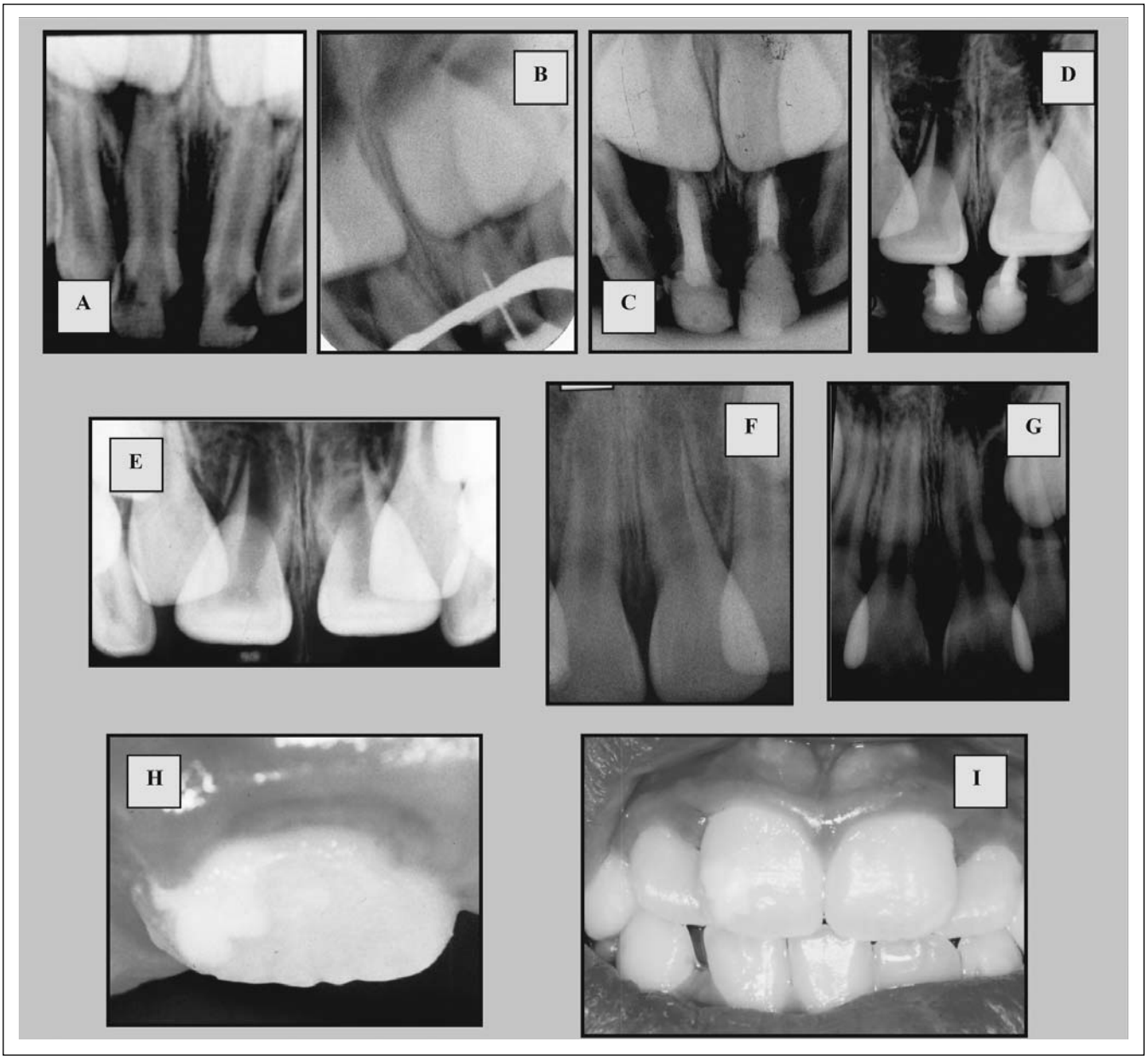

Figure 2. A, diagnostic radiography; B, radiography for endodontic treatment; C, 18 months follow-up radiography; D, 30 months follow-up radiography; E, follow-up radiography following extraction of the primary teeth after 34 months; G, radiography after 56 months of follow-up; H and I, clinical aspects of the permanent successors, showing enamel opacity on element 11.

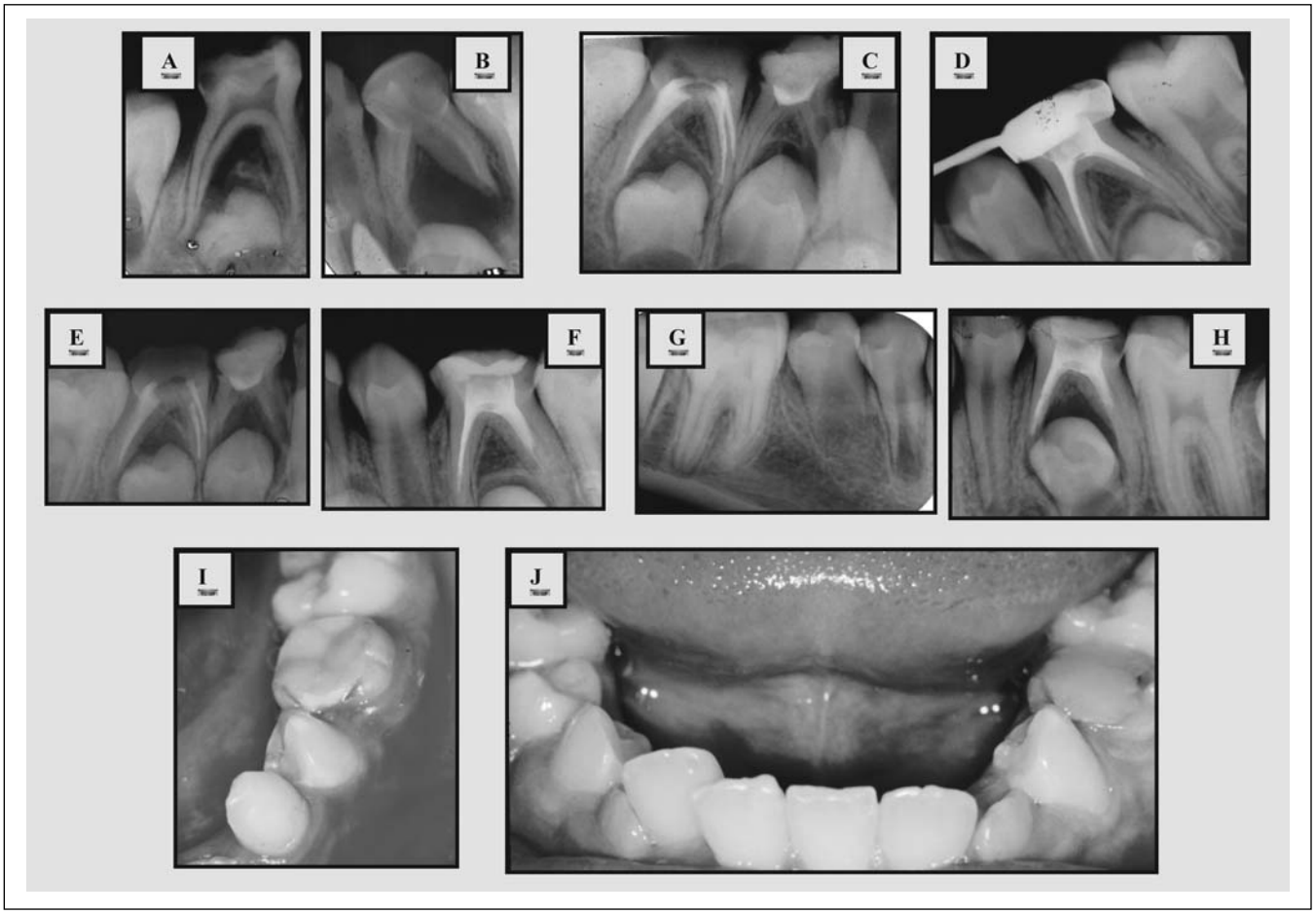

Figure 3. A and B: Diagnostic radiography of Case 2A. C and D, radiography after treatment. E and F, follow-up radiography, after 20 months. G and H, radiography after 55 months of follow-up. J, clinical aspects of element 34 and I

endodontic treatment was performed.

Radiographic diagnosis of the anterior teeth (Figure 2A) showed the presence of a periradicular lesion on element 51, close to the germ of permanent tooth 11(Nolla's stage 5), and element 61 showed a decay lesion with damaged pulp, but was free of lesions on the periradicular tissues.

Figure 2B shows stages of the endodontic treatment of the primary teeth (work length), and Figure 2C shows the follow-up radiography after 18 months of endodontic therapy.

After 34 months of follow-up, it was found to be necessary to extract the primary teeth, due to the stage of development of permanent teeth 11 and 21 (Nolla's stage 7), and the incomplete resorption of the canal filling material of zinc oxide and eugenol. (Figures 2D, E and F).

Figures 2H and I show the clinical aspects of element 11, which shows enamel opacity compatible with a hypocalcification. Element 21 shows no enamel alteration.

Radiography after 56 months of follow up show that the incisor edge has already reached the occlusal plan and there was radicular complementation (Figure 2G)

CASE 2A

This same patient had a clinically observed perforation on the pulpal chamber floor of element 84 and showed a large periradicular lesion on the diagnostic radiography (Figures 3A and B). The permanent successors (44 and 34) of teeth 84 and the contralateral 74, were at Nolla's stage 4 (Figures 3A and B). Tooth 84 was extracted and a space-maintaining device was placed (band-loop type) (Figure 3D). Figure 3C shows the contralateral at the same time when the space maintainer was fitted. The radiographic image in Figure 3D, after 20 months, shows 34 erupted, the space maintaining device was removed, however the contralateral 44, is unerupted and the predecessor is being endodontically treated (Figure 3C).

Figures 3E and F shows a radiographic image of 34 erupted (Nolla 7) and the contralateral at Nolla's stage 6, but unerupted. Clinically, 34 showed enamel opacity in the buccal cusp point (Figures 3I and J). After 55 months of follow-up, 34 are at Nolla's stage 9 (Figure 3H), whilst the contralateral is at stage 8 (Figure 3G), both in the mouth cavity.

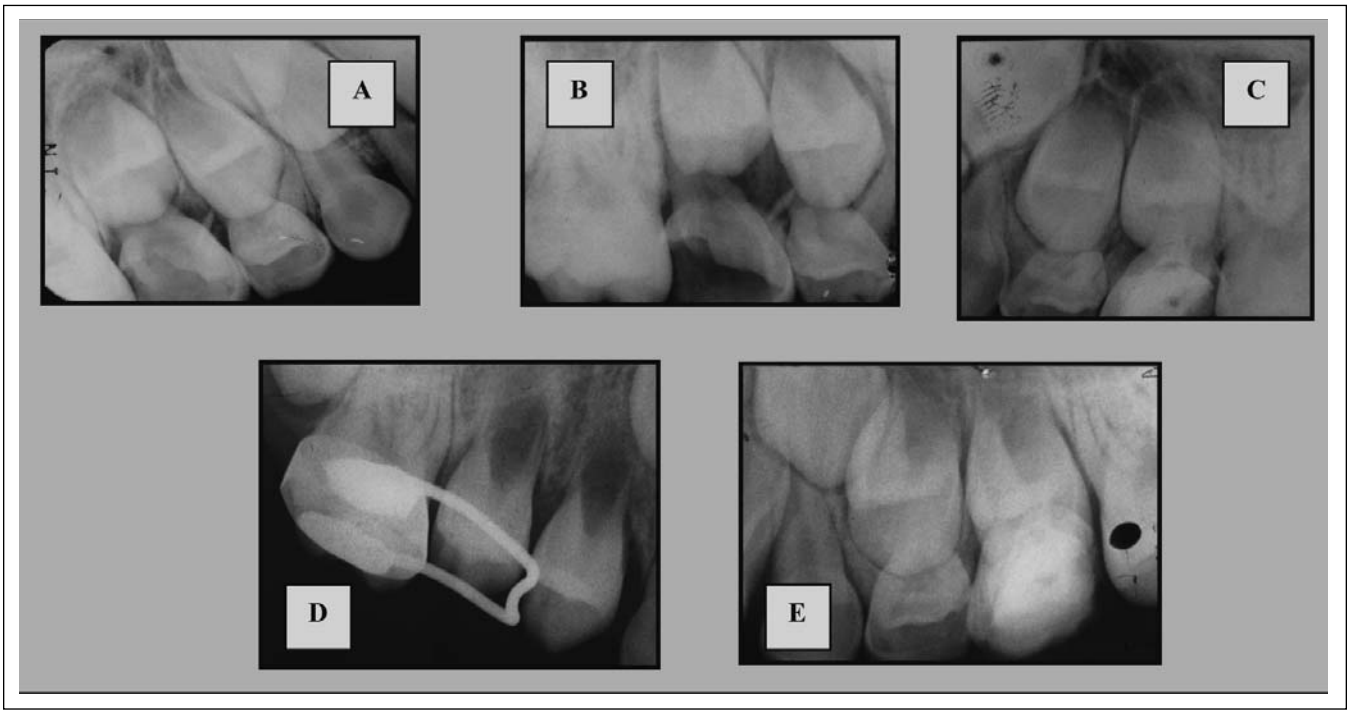

Figure 4. A and B, initial diagnostic radiography; C, contralateral tooth with endodontic treatment; B, Element 15's eruption, after 8 months follow-up, with a band-loop type space maintaining device; E, contralateral tooth, after 8 months follow-up of endodontic therapy, with its permanent successor still unerupted.

CASE 3

A boy aged 3 years and two months attended the Children's Clinic, with damaged pulp in teeth 55 and 65.

Tooth 55 was extracted for being unsuitable for endodontic treatment, as there was radicular resorption with perforation on the pulp chamber floor. (Figures 4A and B). The permanent successor (15) was at Nolla's stage 4. A space-maintaining device of the band loop type was used in the region (Figure 4D). After a period of 8 months follow-up, tooth 15 began erupting, and for this reason the space maintainer was removed, even though this tooth presented extremely immature radicular development (Nolla's stage 5) (Figure 4D). The contralateral successor is at Nolla's stage 5, unerupted and the primary tooth is being endodontically treated (Figure 4E)

CASE 4

A boy aged 6 years and 4 months, attended the Children's Clinic to have surgery on a supernumerary tooth (mesiodens). A panoramic radiograph was requested to evaluate the case before the surgery (Figure 5). In this radiograph and in periapical and interproximal incidences (Figures 5A, B, C and D), it was verified that the second primary molars were missing along with the premature eruption of teeth 35 and 45.

Anamnesis was then conducted in order to obtain information about the two-second inferior bicuspids. The findings were: the mother stated that the child had never been to a dentist before and that the teeth "fell out on their own, they were dry, hollow". When asked if the teeth were decayed, she answered "yes"; the mother also reported that [she] never noticed fistulae or pus drainage in the area, however the gums were always red, inflamed. As for when the primary teeth "fell out", the answer was when the child was 5 years old.

Tooth 35 was in the mouth, "held" by the gums with excessive mobility (Figures 5H and I), and in the radiographic image it was observed that it was at Nolla's stage 4 (Figure 5A).

The occlusal surface of tooth 45 presented signs of decalcification; therefore it was covered with glass ionomer cement, as a measure to protect the immature enamel (Figures 5G and H). This same dental element was at Nolla's stage 7, however, radiographically, the radicular walls were few radiopaque (thin), suggesting a deficiency in dentine production and in dentine and cement calcification (Figure 5B).

After 6 months, the clinical and radiographic aspects showed no significant changes. However, although radicular extension in tooth 45 increased (Figure 5F), the walls were still thin, showing a production deficiency in the calcified tissue, i.e. dentine and cementum.

As for tooth 35, there was a decrease in the radiolucid space under the tooth (Figure 5E). It appears that the bone tissue got closer to the tooth, whose formation is still at Nolla's stage 4. In the cervical region, by the distal, there seems to be an inductional attempt to coronal complementation.

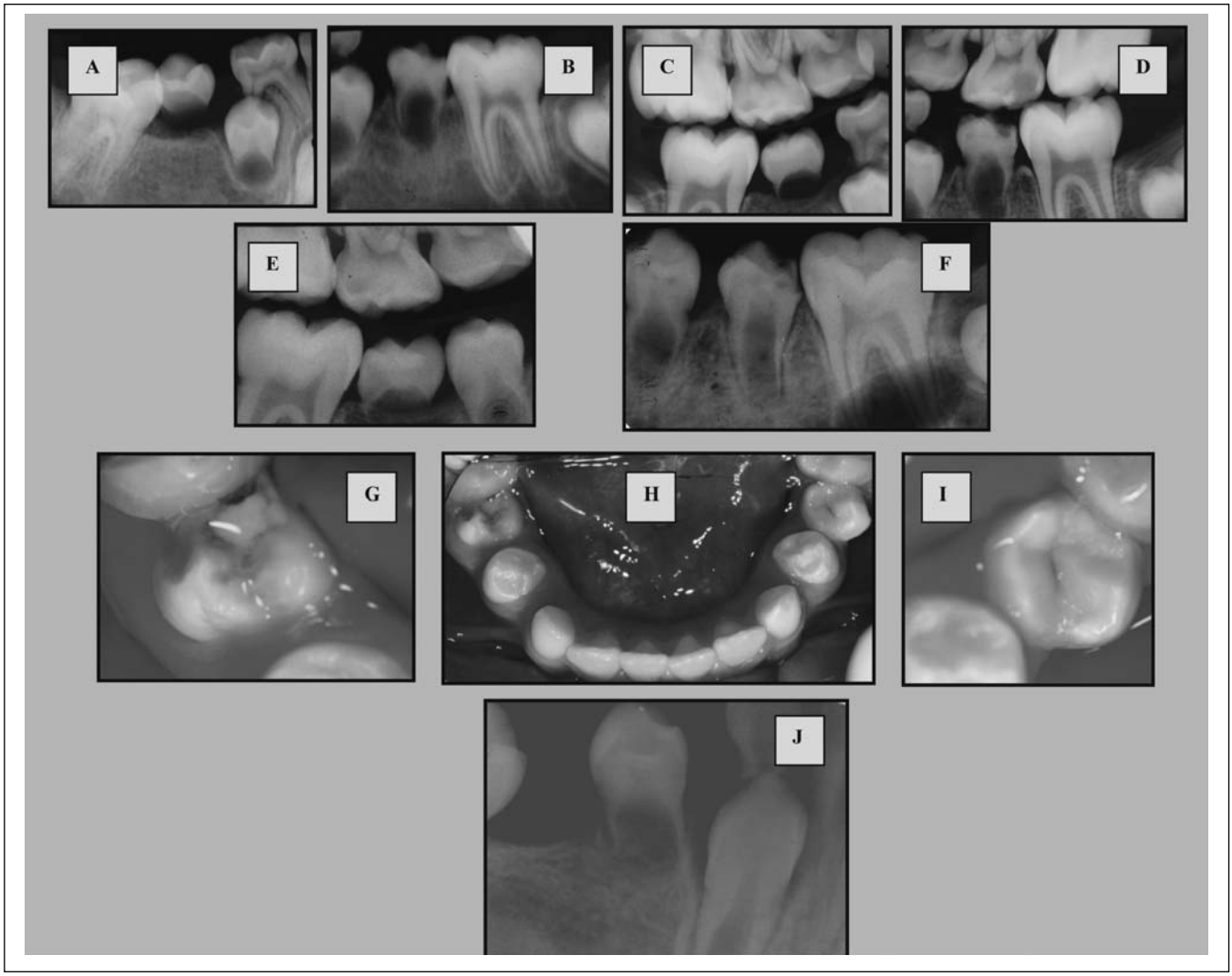

Figure 5. A, B, C and D, initial diagnostic radiography; E and F, 7 months follow-up radiography; G, H, J, recent clinical aspects, after 11 months follow-up and I.

Clinical and radiographic aspects, after 10 months of follow up (Figure J); show that there was no alteration in tooth 45, which presents as in previous images. As for tooth 45, there was no evolution in development, but an increase in the radiolucency in the space under the tooth can be clinically observed, with bone resorption and / or the presence of inflammation / infection.

It is important to stress that the first contiguous primary molars also suffered the effects of the infection, which affected the second molars (Figures 5A, B, C and D), and because of that, presented premature eruption, with rotation and immature radicular development (Nolla's stage 5)(Figures 5 E, F and H).

DISCUSSION

The premature loss of a primary tooth can unleash eruption problems in the permanent teeth, even if treatments are done to recover and maintain the space, as there is no way to accelerate the radicular formation or stop the eruption of a permanent tooth with immature root development (Cases 2A, 3 and 4). This agrees with Fanning's affirmation[8] that the degree of permanent tooth development does not alter after the extraction of the primary predecessor, and that an eruption impulse occurs after the extraction of the primary element, independent of the development stage or biological age of the child.

Among cases of permanent teeth alteration due to inflammation / infection of the primary predecessor, the majority are related to eruption problems, be they due to acceleration or delay or even due to space loss in the dental arch. In second place in cases of permanent teeth alteration, come enamel alterations.

Although there was a high incidence of primary teeth showing periradicular inflammation / infection, few permanent successors presented structural alterations, instead eruption alteration was the most common (Cases 1, 2A, 3 and 4). Matsumiya[9] stated that this can be the result of factors such as the presence of a defense phenomenon in the permanent tooth germ

follicle, usually more efficient at preventing the entrance of infection in the permanent enamel organ. Another factor would be the reduced vital force of the enamel epithelium, once there is regression of inflammation, the viable ameloblasts will begin functioning.

Another factor is the quick formation of gum fistulae, restricting the inflammation to the proximities of the permanent tooth germ. In this situation the germ is rarely affected by the periapical inflammation, even in a relatively serious case. Even if these factors are accepted as true, we cannot admit that they always are sufficient to protect the permanent germ from the chemical mediators of inflammation, or from the products of bacterial metabolism or from the proteolysis of the products of pulp tissue, especially when these are found at very early development stages. (Cases 1, 2, 2A and 4)

What is observed, however, is that when the onset of periradicular tissue inflammation coincides with the precocious stages of the formation of the permanent successor, the damaged inflicted on the germ will be greater, as the alteration happens in the follicular tissue, which is working towards the development of the tooth. The length of contact, which the periradicular lesions maintain with the permanent successor, also has an influence on the gravity of the sequel.[1] In the cap, bell or secretory stage of the hard tissue matrix, alteration of the permanent tooth will be more serious, affecting the coronal morphology (Case 1, Figs. 1B, 1C and 1E) or radicular morphology (Case 4, Fig. 5A, 5C and 5E).

Yet, if the damage occurs in the calcification or maturation stage, the defect will occur in the microstructure of the tissue, presenting enamel opacities, although the morphology will be maintained (Cases 2 and 2A). In case 4, due to the premature eruption, there was no post-calcification maturation in the enamel matrix, a function accomplished by the reduced enamel epithelium, showing the fragility of the tooth in an aggressive oral environment (Case 4, Figures 5G and 5H).

In radicular structural alterations, the cervical line for root development is only formed after the total crown outline. This happens when the inner and the outer epithelium get together in the still unformed enamel-cement junction, forming the Hertwig Epithelial Sheath.[16] In Case 4, the permanent tooth achieved eruption with coronal development still incomplete (Nolla 4), which besides exposing the immature enamel to the oral environment, also endangers radicular development, as the crown was not completely outlined and the stages of forming the Hertwig Epithelial Sheath were interrupted (Case 4, Figures 5A, 5C and 5E).

The alterations involving the dental crown are aesthetic, and usually do not involve immediate cosmetic intervention, especially in very young patients[2] (Cases 2 and 2A). When this need exists, they can be conducted after the eruption, of the tooth through restorative processes or prosthesis (Case 1). However, alterations that involve radicular structures not only compromise the permanence of the tooth in the arch, but also unleash both aesthetical and functional problems (Case 4).

Our findings agree with Weinlander,[18] who stated that when injury occurs precociously, at the initial Nolla's stage, even the removal of the primary tooth may not correct the damage to the permanent tooth, but the extent of aggression can be confined if normal periradicular conditions are restored.

Enamel defects observed sustain Coll and Sandrian's statement,[6] that these defects are related to the presence of periradicular infection in the predecessor primary tooth, before endodontic treatment, and not as a consequence of radicular canal therapy (Case 2, Figures 2A, 2H and 2I). Well conducted endodontic manoeuvres cannot be blamed for alteration in the permanent tooth germ, as the objective is to restore the balance of the tissue. Usually problems occur due to infections that existed before the treatment or those remaining from endodontic therapy. In Case 2, element 11's hypocalcification was probably due to the presence of a periapical lesion on the primary tooth. This lesion was probably present during calcification and / or enamel maturation, and was observed afterwards, during the initial diagnostic radiography and anamnesis.

Endodontic or extraction treatment does not correct alterations that have already been caused in the permanent tooth germ, but avoid it worsening.[3,7,14] As observed in Case 3 (Figure 4D), extraction did not stop the premature eruption of the permanent tooth, and even increased the risk of space loss in the dental arch, which is an additional concern regarding space maintenance or recuperation and in the follow-up of this treatment. A successful endodontic treatment (Cases 2A and 3, Figures 3H and 4E, contralateral teeth, avoided premature eruption, as it restored balance to the tissue, allowing the primary tooth to complete the biological cycle and performs all of the main functions, i.e. mastication, space maintenance, eruption guide, etc.

The temporary nature of primary teeth does not mean that they are disposable, that is, as though they could be taken away from the stomatognathic system at any moment, without causing alteration to the homeostasis of the system. The maintenance or the rescue of primary teeth is vital to the local balance in the mouth and no less important to the general health of the young patient.

We conclude that decayed primary teeth must be followed-up by both radiographic and clinical examinations, in order to verify the extension of the lesion, as they develop in the primary dentition, usually without presenting relevant clinical symptoms. This characterises "clinical silence", which is not synonymous with health or repair. The earlier these lesions are detected,

in theory, the less the destructive effects and the sequelae on the permanent germ / primary tooth unity will be. In risk-benefit evaluation, the primary tooth can only be kept in the mouth if it does not cause damage to the permanent successor. When endodontic therapy is not indicated, extraction should be the chosen treatment. Despite the fact that none of these alternatives give back what was destroyed, they limit damage and rescue children's health.

REFERENCES

1. Andreasen JO, Andreasen FM. Texto e atlas colorido de traumatismo dental. 3 ed. Porto Alegre, Artmed, 2001.
2. Baratieri LN, et al. Odontología restauradora. fundamentos e posibilidades. São Paulo, Santos, 2001.
3. Binns JR. WH, Escobar A. Defects in permanent teeth following pulp exposure of primary teeth. J Dent Child 34: 4-14, 1967.
4. Brook AH, Winter GB. Developmental arrest of permanent tooth germs following pulpal infection of deciduous teeth. Br Dent J 139: 9-11, 1975.
5. Brook AH, Winter GB, Osborne JA. The occurrence of benign cystic lesions of the jaws in children. Proc Br Paedod Soc 1: 16, 1971.
6. Coll JA, Sadrian R. Predicting pulpectomy success and its relationship to exfoliation and its succedaneous dentition. Pediatr Dent 18: 57-63, 1996.
7. Cordeiro MMR. Tratamento endodôntico em dentes decíduos: conseqüências nos dentes permanentes sucessores. Monografia. Universidade Federal de Santa Catarina. Curso de Especialização em Odontopediatria. 1999.
8. Fanning E. A. Effect of extraction of deciduous molars on the formation and eruption of their successors. Angle Orthodont 32: 44-53, 1962.
9. Matsumiya S. Experimental pathological study on the effect of treatment of infected root canals in the deciduous tooth on growth of permanent tooth germ. Int Dent J 18: 546-559, 1968.
10. Müller de aAraújo MG. Ortodontia para clínicos: programa pré-ortodôntico. 4 ed. São Paulo, Santos, 209-231, 1988.
11. Myers DR, et al. Histopatology of furcation lesions associated with pulp degeneration in primary molars. Pediatr Den 9: 279-282, 1987.
12. Niswander JD, Sujaru C. Relationship of enamel defects of permanent teeth to retention of deciduous tooth fragments. J Dent Res 41: 808-814, 1962.
13. Nolla CM. The development of the permanent teeth. J Dent Child 27: 254-266, 1960.
14. Rocha MJC. Tratamento endodôntico de dentes decíduos comprometidos por cárie. 10o Livro Anual do Grupo Brasileiro de Professores de Ortodontia e Odontopediatria, Belo Horizonte 1: 28-30, 2001.
15. Slots J. Systemic manifestations of oral infections. In: Slots J, Taubman M. Contemporary oral microbiology and immunology. St. Louis, Mosby, pp. 500-510, 1992.
16. Ten Cate AR. Histologia bucal. Desenvolvimento, estrutura e função. 2 ed. Rio de Janeiro, Guanabara, 1985.
17. Valderhaug J. Periapical inflammation in primary teeth and its effect on the permanent successors. Int J Oral Surg 3: 171-182, 1974.
18. Weinlander GH. Clinical effect of formaldehyde preparations in pulpotomy for primary molars. J Can Dent Assoc 37: 154-158, 1971.
19. Winter GB, Kramer IRH. Changes in periodontal membrane, bone and permanent teeth following experimental pulpal injury in deciduous molar teeth of monkeys. Arch Oral Biol 17: 1771-1779, 1972.