

EVALUATION OF ULTRASTRUCTURAL ALTERATIONS IN THE DENTAL PULP IN PATIENTS WITH JUVENILE PERIODONTITIS - DISEASE COMMON IN RURAL POPULATION

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Abstract: The influence of juvenile periodontitis (JP) on ultrastructural alterations in dental pulp has been studied. The results indicate that ultrastructural alterations occur mainly in the odontoblasts, blood vessels and nerve fibres.

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INTRODUCTION

The condition of the oral cavity is closely related to general health state and remains in a direct and functional relationship with all the tissues of the organism. Because of the frequent occurrence of periodontium diseases and their noxiousness for the stomatognathic system, they pose a significant health problem, particularly in rural population [1, 25].

In the pathogenesis of periodontium diseases an important role is played by inflammation, fractional (injury) and systemic factors [8, 15, 20, 24]. The basic inflammation factor in periodontitis are bacteria localised on the dental plaque. The composition of the dental plaque depends on the diet, individual bacteria composition in the saliva, the age of the plaque and hygienic procedures applied to the oral cavity [6, 9, 14, 16, 18, 19].

One of the periodontium diseases is juvenile periodontitis which occurs in children and adolescents between 11 and 13 years of age, and later on [1, 2, 22].

The aim of the present work was to investigate the influence of juvenile periodontitis on ultrastructural

changes in the dental pulp which reveals vulnerability to the activity of various, both internal and external factors, and which also possesses high defence properties and is capable of acting against the infection by means of phagocytosis and immune response [13].

MATERIAL AND METHODS

The material used in the investigations was the dental pulp of teeth extracted from patients with juvenile periodontitis. The type of disease was established due to clinical and radiological examinations. In those patients it was necessary to extract the teeth because of the advanced inflammation, tooth looseness or for prosthetic reasons. In some patients periodontitis was not recognised, yet the teeth had to be extracted for orthodontic reasons or because of mechanic injuries of the jaw bones (control group).

The pulp was taken out with a preparation needle, after sectioning the tooth along its longitudinal axis. For the ultrastructural examination in the electron transmission microscope, the pulp was fixed in 4% solution of glutaraldehyde in the pH 7.4 phosphate buffer for

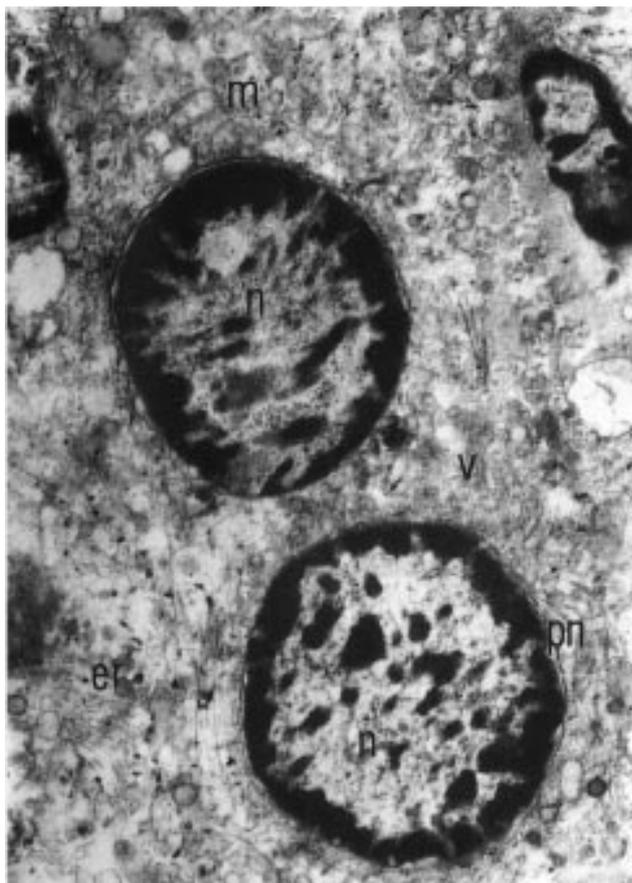


Figure 1. Control group. Odontoblasts. Nucleus (n), pores in the nuclear membrane (pn), granular endoplasmic reticulum (er), mitochondria (m), vacuoles (v). EM, $\times 4000$.

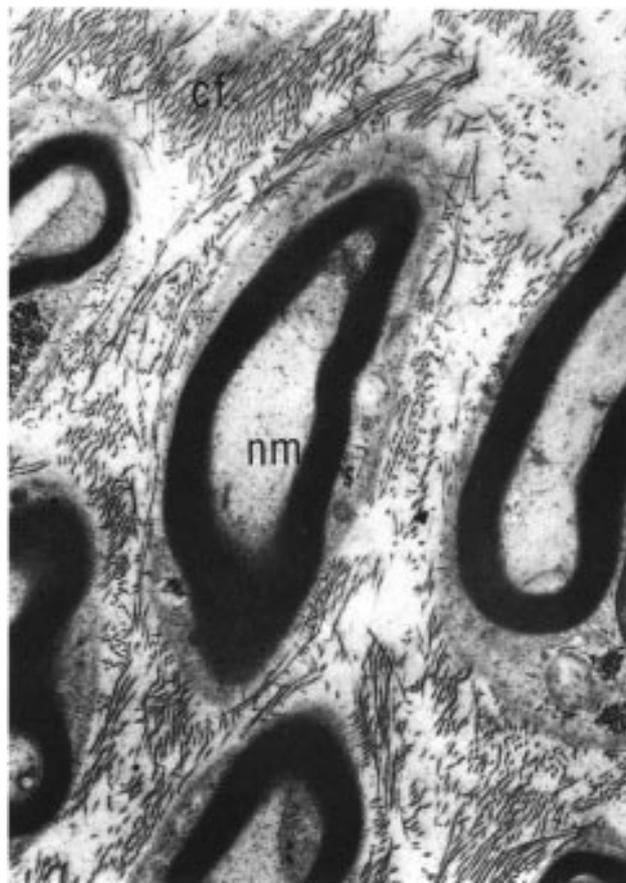


Figure 2. Control group. Myelin nerve fibres (nm) and collagen fibres (cf) in the dental pulp. EM, $\times 4000$.

3-5 hours, and then in 1% solution of osmium tetroxide (OsO_4) at 4°C for 1 hour. After dehydration in graded series of alcohols, and then in propylene oxide, the material was embedded in gelatine capsules of Epon 812. The blocks were polymerised at temperature of 45°C for 12 hours. The ultrathin sections were made with the OM-U3 ultramicrotome. The sections were counterstained with uranyl acetate and lead citrate. Photographs were taken through a Tesla BS-500 electron transmission microscope.

The results obtained were subject to statistical analysis. The degree of alterations in the dental pulp of the examined groups was expressed for every histogram in percent (%). The numbers and percentage of specimens of dental pulp were compared, according to the degree of alterations, in the Figures. The significance of the differences was assessed by χ^2 test.

RESULTS

Control group (C). In the control picture of the dental pulp special attention should be paid to the odontoblasts with large oval nuclei and proper distribution of the chromatin near the nuclear areola (Fig. 1). Regularly

arranged pores can be seen in the nuclear areola. In the cytoplasm, abundant polyribosomes, endoplasmic reticulum and mitochondria of normal structure occur.

The subodontoblastic layer contained a large number of nerve fibres with dark myelin sheath (Fig. 2). The pulp core possessed numerous star- or spindle-shaped fibroblasts. Their nuclei were oval or elongated, and contained agglomerations of the chromatin under the nuclear sheath (Fig. 3). In their cytoplasm streaks of collagen fibres, and blood vessels were observed.

Experimental group - juvenile periodontitis (JP). In the dental pulp with juvenile periodontitis (JP) some alterations in the odontoblast structure were observed. In their cytoplasm vacuoles appeared, there was less endoplasmic reticulum, the mitochondria were swollen and deprived of their crests. The nuclei of the odontoblasts did not contain chromatin nodules, they were homogenous (Fig. 4).

The endothelium and basilemma of the blood vessels were damaged, at their wall some pericytes were visible (Fig. 5). In the pulp core a major accumulation of neutrophilic granulocytes (neutrophils) was observed (Fig. 6) which possessed a multilobar nucleus and

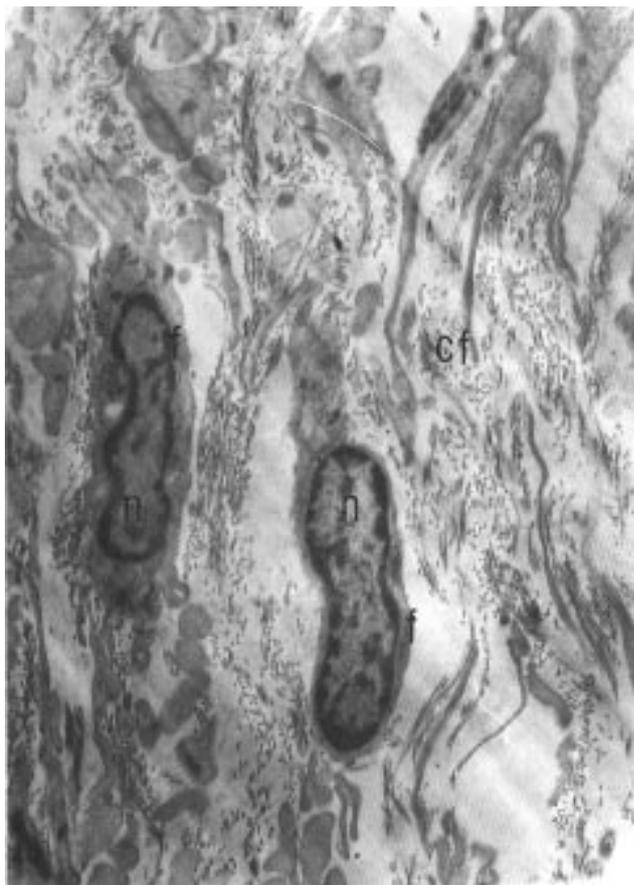


Figure 3. Control group. The pulp core with fibroblasts. Fibroblasts (f) with oval nuclei (n) and collagen fibres (cf). EM, × 4000.

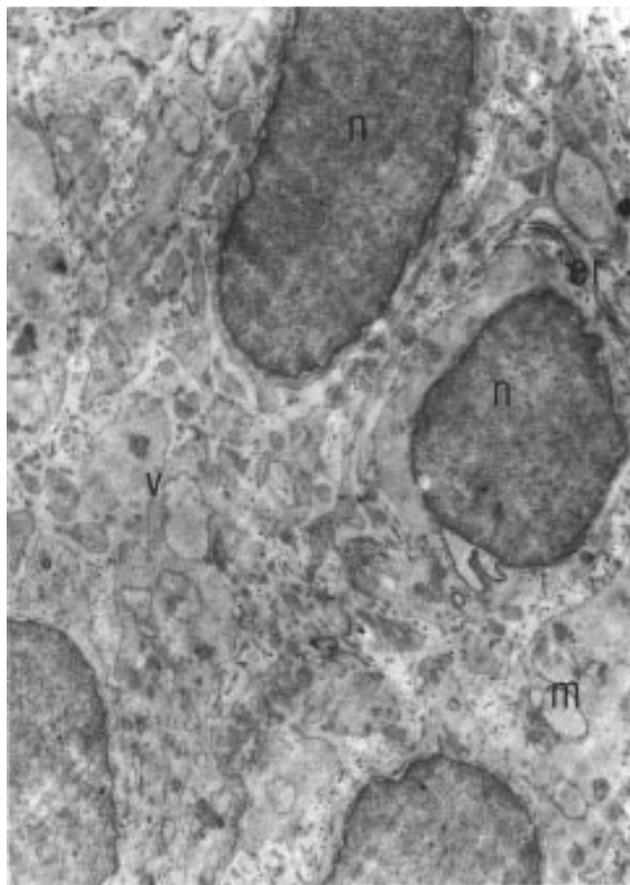


Figure 4. Experimental group. Odontoblasts containing: homogenous nuclei (n), granular endoplasmic reticulum (er), mitochondria (m), vacuoles (v). EM, × 4000.

numerous granules (Fig. 7). The fibroblasts revealed cytoplasm vacuolisation.

Statistical analysis. The summarized results of the studies are illustrated in three Figures. Figure 8 presents the degeneration of the odontoblasts in the dental pulp in juvenile periodontitis (JP) in comparison to the control group (C). In JP there were 29.4% of major alterations in the odontoblasts (++) , 58.8% alterations of medium degree (+), and 11.8% revealed no changes. None of the control group subjects showed the presence of changes.

Figure 9 presents the hyperaemia and damage to vessels in the dental pulp of the JP group in comparison to the control. Changes of major degree (++) occurred in 52.9%, of lower degree (+) in 41.2%, and lack of alterations in only 5.9%.

Figure 10 shows the frequency of inflammatory infiltrations in the pulp of the examined patients with JP in comparison to the control group. Major alterations (++) occurred in 52.9%, minor changes (+) in 47.1%, and cases with no changes were not found.

In all cases (Figs 8-10), the frequency of alterations in the patients with juvenile periodontitis was much greater compared to the control group and the differences were highly significant ($\chi^2 > 24, p < 0.001$).

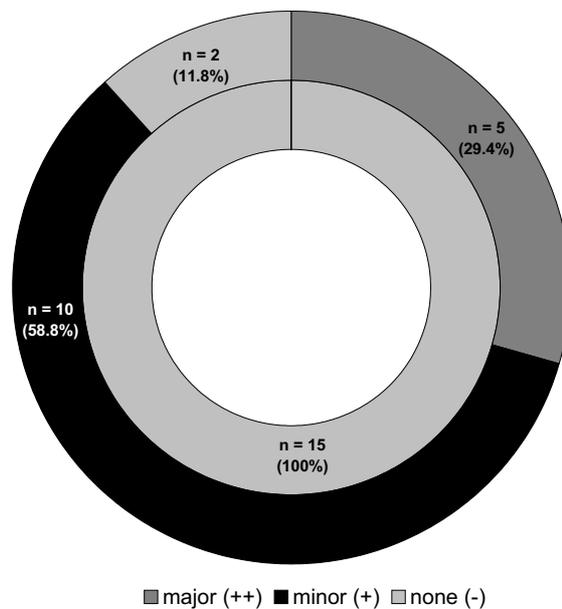


Figure 8. Degeneration of odontoblasts in the dental pulp of the juvenile periodontitis (JP) group (n=17) compared to the control (C) group (n=15).

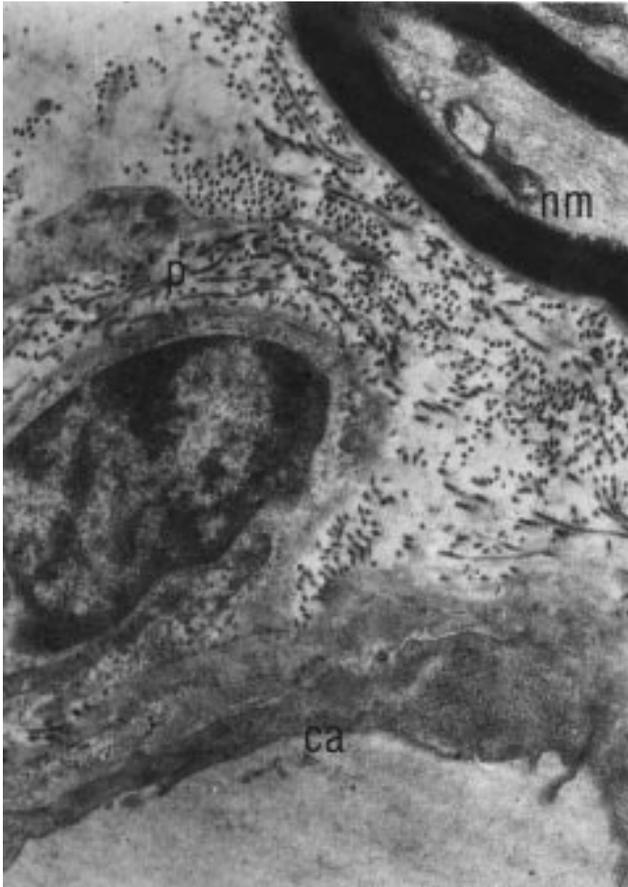


Figure 5. Experimental group. Nerve fibres (nm), capillary vessels (ca) and pericytes (p) in the dental pulp. EM, × 4000.

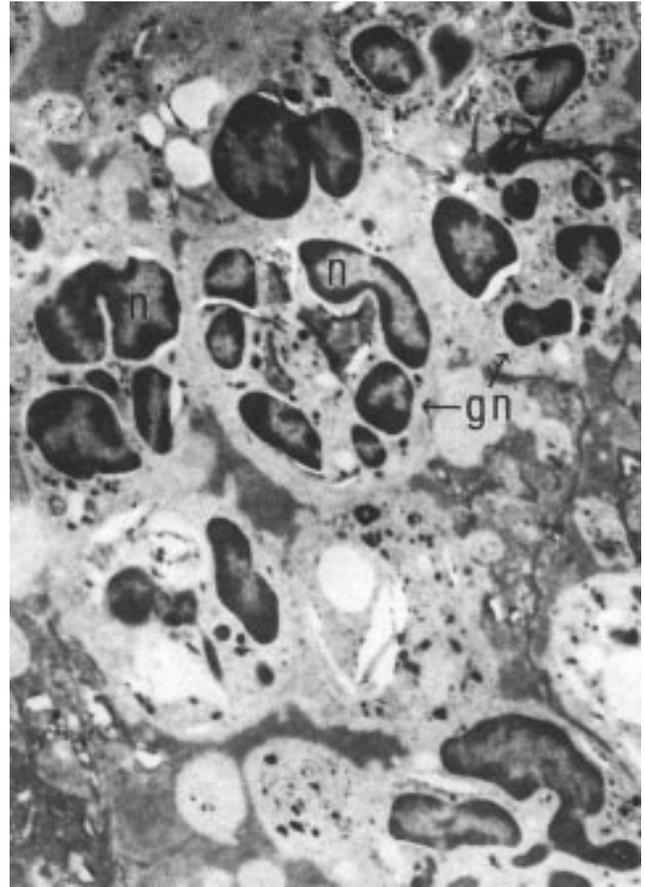


Figure 6. Experimental group. Neutrophilic granulocytes (gn) with polymorphous nuclei (n) in the dental pulp. EM, × 3000.

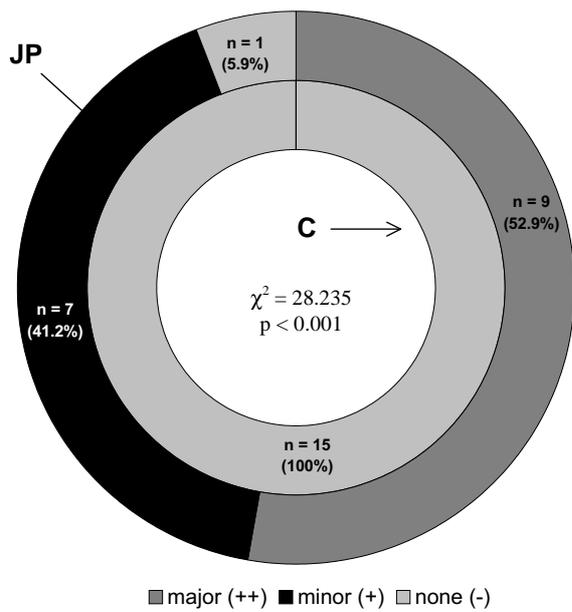


Figure 9. Hyperaemia and damage to vessels in the dental pulp of the juvenile periodontitis (JP) group (n=17) compared to the control (C) group (n=15).

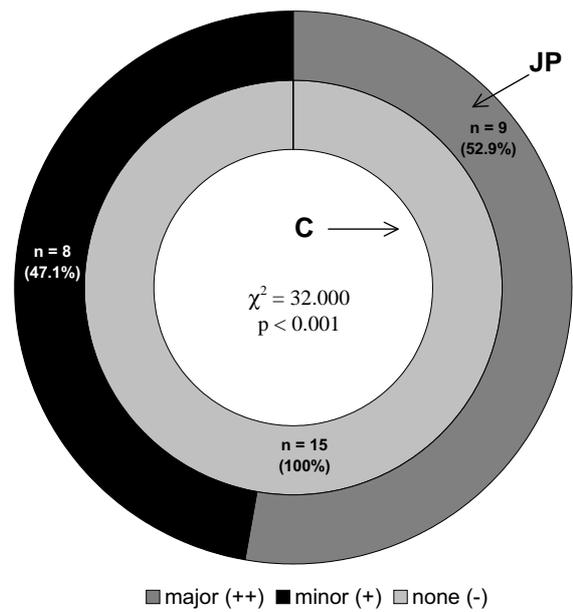


Figure 10. Inflammatory infiltrations in the dental pulp of the juvenile periodontitis (JP) group (n=17) compared to the control (C) group (n=15).

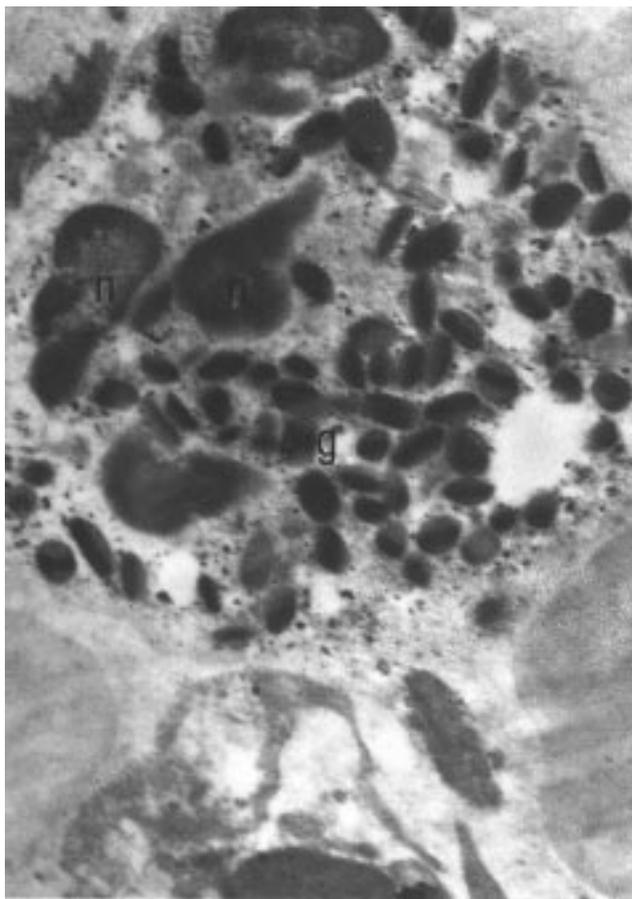


Figure 7. Experimental group. Neutrophil of the dental pulp with trilobate nucleus (n) and numerous granules (g) in the cytoplasm. EM, $\times 7000$.

DISCUSSION AND CONCLUSIONS

It is assumed that there exist some individual predispositions for the development of early periodontium diseases to which juvenile periodontitis (JP) belongs [17]. The frequency of juvenile periodontitis cases is related mainly to the presence of two major pathogenic factors: *Actinobacillus actinomycetemcomitans* and *Porphyromonas gingivalis* [10]. It is suggested that there is a possibility of genetic transmission of the periodontium diseases [21]. Children whose parents suffered from periodontitis are more susceptible to develop these diseases as well.

The localisation of the dental pulp and its direct contact with the periodontium through the apical foramen suggest that the pathological processes occurring in the periodontium may have influence on the structure of the pulp [11, 12, 23]. This is proved, among others, by the investigations done by Badillo *et al.* [3], Barańska-Gachowska *et al.* [4] and Bernick-Nedelman [5] who examined the pulp in the ageing process and in periodontal diseases.

While analysing the problem of periodontal diseases, and juvenile periodontitis (JP) in particular, attention was paid to the ultrastructural changes in the dental pulp of the patients examined. The changes referred to the degeneration

of the odontoblast epithelium, the widening of the blood vessels and the occurrence of vast inflammatory infiltrations. The latter consisted mainly of neutrophilic granulocytes (neutrophils). Structural alterations were also observed in fibroblasts and nerve fibres. As pointed out by some authors [4, 7], the inflammatory infiltrations may also occur in teeth with improper filling.

It may be concluded on the basis of statistical analysis that the most significant changes in juvenile periodontitis in comparison to the control group refer to the blood vessels and numerous inflammatory infiltrations, which makes them different from the pictures of the pulp in adult periodontitis [11]. Major alterations were also found in the odontoblasts which were degenerated in many cases.

The problem of periodontium diseases and their influence on the tooth issues is complicated and difficult to assess. The present study explains this issue to a certain extent, and indicates that in juvenile periodontitis there will always be changes in the structure of the dental pulp. The character of these alterations may be to some degree an indicator of disease changes connected with juvenile periodontitis, yet they often depend on individual features and general health state of the patient.

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