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Delayed Passive Eruption - A predisposing factor to Vincent's Infection?

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SUMMARY

One thousand and twenty-five patients' records were examined for the incidences of Vincent's infection and delayed passive eruption (D.P.E.). D.P.E. was seen in 124 *patients* (12,1 *per cent*) *and occurred at a mean age of* 24,2 ± 6,2 *years. Vincent's infection was present in* 88 *patients* (8,6 *per cent, mean age* 25,5 ± 5,6 *years*) *of whom* 55 *also had D.P.E. Statistical analysis showed a very high correlation between the two conditions* ($P < 0{,}001$) *and no significant difference in their ages of occurrence. It is suggested that D.P.E. is a predisposing factor to Vincent's infection.*

OPSOMMING

Een duisend vyf-en-twintig rekords van pasiënte is vir die voorkoms van Vincent-tandvleisontsteking en vertraagde passiewe erupsie (V.P.E.) nagegaan. V.P.E. het in 124 *pasiënte* (12,1 *persent*) *met 'n gemiddelde ouderdom van* 24,2 ± 6,2 *jaar, voorgekom. Vincent-tandvleisontsteking het in* 88 *pasiënte* (8,6 *persent, gemiddelde ouderdom* 25,5 ± 5,6 *jaar*) *voorgekom, en onder hulle het* 55 *ook V.P.E. gehad. Statistiese ontleding het getoon 'n groot korrelasie tussen die twee toestande bestaan* ($P<0{,}001$) *maar dat daar geen betekenisvolle verskil tussen die twee ouderdomsgroepe is nie. Dit word voorgestel dat V.P.E. 'n predisponeerende faktor in Vincenttandvleisontsteking is.*

INTRODUCTION

Vincent's infection or acute necrotizing ulcerative gingivitis has been known for very many years. The predisposing factors to this condition have often been debated and are still in question. Stammers (1944) carried out an extensive study of the disease and reported on 1 017 cases. In his study he described local predisposing causes as gross neglect, food stagnation, calculus, overcrowding, mouth breathing, smoking and recent extractions and among systemic predisposing factors he included frequent colds, possible vitamin deficiency, recent illness, operations, pregnancy, overwork and lack of exercise.

Goldhaber and Giddon (1964) discussed concepts of the aetiology and treatment of acute necrotizing ulcerative gingivitis and stated that the most conspicuous predisposing factors included tobacco smoking, gingivitis, or local trauma, in association with acute psychological disturbance which apparently precipitated the disease in susceptible individuals.

An important factor described by many authors including Schluger (1949), Pindborg (1951), Giddon, Zachin and Goldhaber (1964) and Barnes, Bowles and Carter (1973) is the susceptibility of persons in the age group 19-26 years.

As Vincent's infection is said to be a fuso-spirochaetal infection with the association of *Bacteroides melaninogenicus* as another causative organism (MacDonald *et al* 1956), it is difficult to explain why these organisms are particularly pathogenic within a certain age range. Socransky and Manganiello (1971) felt that the proportions of predominant cultivatable organisms from the gingival crevice area of the preschool child appear generally to resemble that of the adult, with the exception that spirochaetes and *Bacteroides melaninogenicus* were not present in all children. They stated that the reason for the late establishment of these organisms was not clear; but conditions for their growth might not be provided by the gingival crevice in young children.

As both these organisms are anaerobic, anaerobic conditions must necessarily be present for their growth and in this respect an important anatomical consideration is the relationship of the gingiva to the crown. Manson (1963) stated that soon after eruption of a tooth the attachment of soft tissue was at the cemento-enamel junction so that there was a deep crevice which became shallower as the tooth erupted. Boyle, Via and McFall (1973) showed that from 15 years of age onwards, there is a regression of the cemento-enamel junction from the alveolar crest. With this change in the alveolar crest position there should also be change in the position of the gingival margin related to the cemento-enamel junction. In other words, there must be both active and passive eruption. These processes we define as follows:

Active eruption is the coronal movement of the teeth. Passive eruption is the apical migration of the gingival margin to approximate the cemento-enamel junction.

Should the margin remain high on the tooth crown in adulthood and not approximate the cemento-enamel junction, then one may talk of delayed passive eruption (DPE).

MATERIALS AND METHODS

One thousand and twenty-five patients' records cards, taken in sequential order, were examined. The patients'

age, sex, and the presence or absence of Vincent's infection and DPE were noted. Vincent's infection was diagnosed clinically without bacteriological confirmation. Clinical criteria included the characteristic ulceration on the crests of the papillae together with the typical foetor oris (Prichard 1966, Manson 1970 and Wade 1965).

The purpose of this study was to determine the age distribution for Vincent's infection in patients of a private practice and to correlate this with the incidence of DPE as a possible predisposing factor in the infection.

In this study DPE was deemed to be present when the gingival margin was coronal to the cemento-enamel junction at a level approximating the maximum convexity of the buccal or labial aspect of the tooth (Figs. 1 and 2) and associated with pseudo-pocketing which was assessed using a periodontal probe.

RESULTS

In the group studied more female patients were seen than males in the approximate ratio 3:2 (Table I).

More cases of DPE were seen than Vincent's infection and the absolute incidences as well as the ages of occurrence of each are shown in Table I. When the percentage incidences were calculated DPE cases were found to represent 12,1 per cent of the patients and cases of Vincent's infection 8,6 per cent (Table II). In 5,4 per cent of patients (55) both Vincent's infection and DPE were present.

Fig. 3 shows the age incidence in histogram form of DPE. The patients in this group who also have Vincent's infection are indicated. Fig. 4 shows the age distribution of patients with Vincent's infection as well as those who also had DPE.

The mean age for the occurrence of DPE was 24,2 ± 6,2 years while that for Vincent's infection was 25,5 ± 5,6 years. This is not a statistically significant difference.

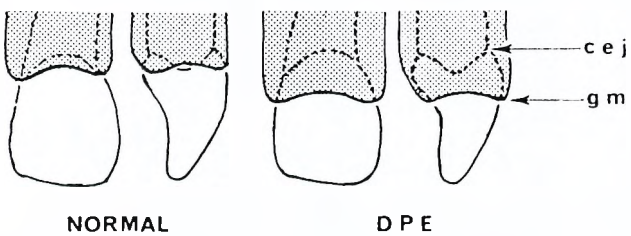


Fig. 1 Diagram of the position of the gingival margin (gm) in relation to the cement-enamel-junction (cej) in the normal adult and delayed passive eruption (D.P.E.).

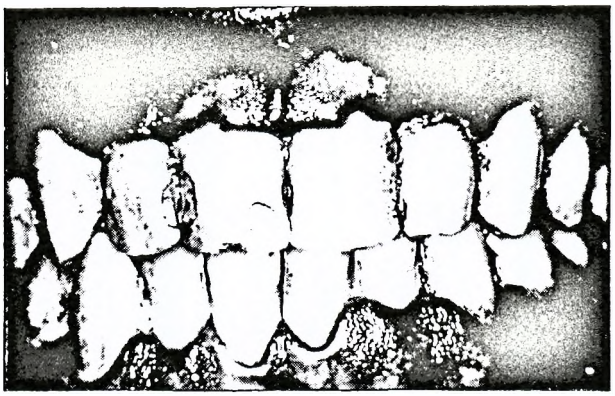


Fig. 2 Photograph of case showing Vincent's infection as well as D.P.E. The areas with D.P.E. and inflammatory hyperplasia are indicated in Fig. 2A which is a tracing of Fig. 2.

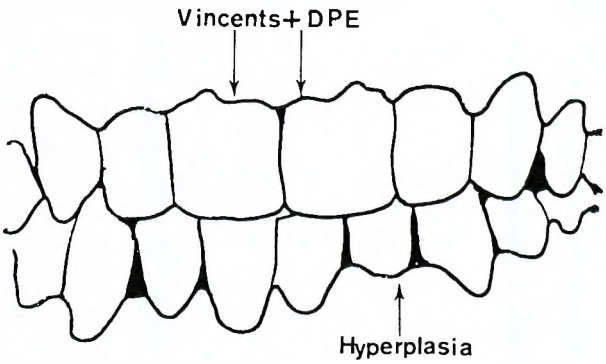


Fig. 2A.

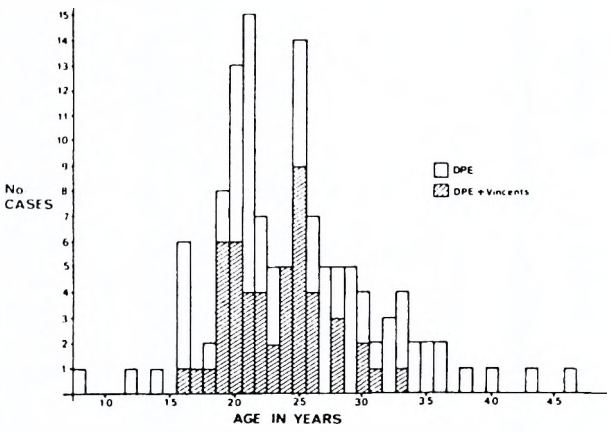


Fig. 3 Histogram showing the age incidence of D.P.E. and also those cases of D.P.E. having Vincent's infection as well.

	Delayed Passive Eruption			Vincent's Infection		
	No.	Age Range	Mean age ± SD	No.	Age Range	Mean age ± SD
Male (n = 424)	47	16–46	25,7 ± 6,2	35	18–48	24,8 ± 5,3
Female (n = 601)	77	8–43	23,2 ± 6,0	53	16–49	24,6 ± 6,3
Total (n = 1025)	124	8–46	24,2 ± 6,2	88	16–49	25,5 ± 5,6

Table I. Absolute incidences of delayed passive eruption and Vincent's infection and the age in years at which they occurred.

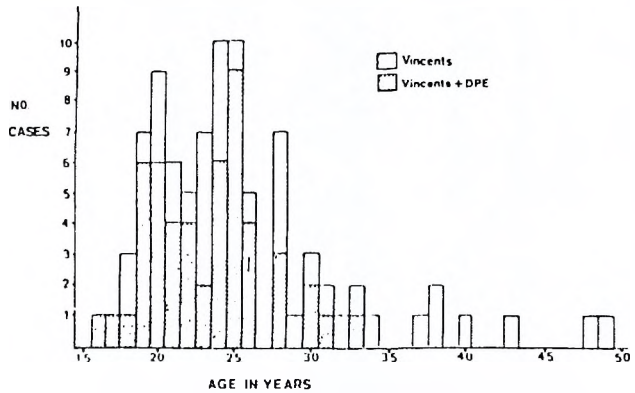


Fig. 4 Histogram showing the age incidence of Vincent's infection and those cases also having D.P.E.

Table II: Percentage incidences of D.P.E. and Vincent's infection.

	Delayed Passive Eruption	Vincent's Infection
Male	11,1	8,3
Female	12,8	8,8
Total	12,1	8,6

Table III: Association of Vincents' infection with DPE and gingival hyperplasia as determined from clinical photographs of 30 patients.

	Number of teeth	Percentage
Normal	143	39,2
Vincent's infection without DPE	85	23,2
Vincent's infection plus DPE	70	19,2
DPE without Vincent's infection	58	15,9
Vincent's infection without gingival hyperplasia	5	1,4
Vincent's infection with gingival hyperplasia	4	1,1

DISCUSSION

The group studied was not a normal population-group but one referred for consultations and possible treatment of periodontal and oral medical problems. Thus the incidence of Vincent's infection in this group could not be compared with the many studies carried out in groups such as army recruits and university students.

While it appeared from the absolute values that more females than males presented with Vincent's infection, when this was corrected for the numbers of patients seen, no significant difference was found between the two sexes. There was a similar finding with regard to DPE.

Both conditions occurred in statistically similar age groups (Table I, Figs. 3 and 4). The mean age of occurrence of DPE was however earlier in females ($P < 0,05$).

Statistical evaluation showed an extremely high correlation between the two conditions (χ^2 test, $P < 0,001$) so it is reasonable to suggest that DPE is a predisposing factor in Vincent's infection.

A question that now arose was whether the site distributions of the two conditions in fact coincided exactly. At the time of making the clinical observations this was not recorded so the colour transparencies taken at the initial visit were reviewed. Of the 88 patients with Vincent's infection, colour transparencies of 30 were available. The incidences of the various conditions are shown in Table III and once again a significant correlation was found ($P < 0,01$).

In this study we have shown a statistically significant association between Vincent's infection and delayed passive eruption. The reason for this is, we feel, that with the pseudopocketing present in DPE it is reasonable to expect an anaerobic gingival environment suitable for the bacteria responsible for Vincent's infection. This, together with the observations of Socransky and Manganiello (1971) on the oral flora, may explain the age group incidence of Vincent's infection.

Although it could be argued that DPE does not exist and is in fact merely an inflammatory hyperplasia, we feel that this is not so. DPE may be diagnosed in the absence of inflammation and therefore exists as a clinical entity on its own. It is obvious, as well, that if a severe Vincent's infection is present there will be a marginal inflammation and confusion could arise in determining a difference between inflammatory hyperplasia and DPE. A valuable distinguishing feature however, is that, while Vincent's infection may be localized to an individual papilla, DPE is more often than not visible at more than one tooth.

It is suggested then, that patients between 15 and 35 years of age having DPE be instructed in meticulous oral hygiene as the risk of their developing Vincent's infection is high.

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