

finished margins of vulcanite pressing on the interproximate spaces so as to make deep pockets and ultimate gum recession—these are very ordinary exciting factors in bringing about the first stage of pyorrhœa. The interdependence of the teeth and gums is imperative. The teeth will be retained in the socket, it may be for years, but it is never forever. The gums will tolerate an immense amount of mechanical and surgical abuse anywhere better than at the gingival line. Why is this? Because the soft glandular-like epithelium—though it is not a glandular structure—which lies at the neck of the tooth, is composed of more delicate cells than elsewhere in the gums; its fibres are more intimately connected with the cementum; the cementum is thinnest as it reaches the neck of the tooth, and the periodontal membrane depends for its tense hold on the roots upon the integrity of the fibres of the pericementum which unite with the cementum at the neck.

I believe there are constitutional predisposing causes of pyorrhœa alveolaris, but in searching and speculating in that direction, it is wise to search carefully for causes closer to view. We see mouths not only after or during illness, but in good health, which are human cesspools. The origin of a good deal of pyorrhœa alveolaris is no more obscure than the origin of a good deal of typhoid. Many people will tolerate more positive filth, and more pyogenic bacteria in their mouths than they would on their feet.

The etiology of caries is no longer a speculation; that of pyorrhœa alveolaris is entirely so. We know that it is a perversion of normal physiological action; that it is a deviation from the normal standard of health of the parts concerned, constituting a distinct disease that may end in the death and loss of the teeth. But we are not agreed as to whether or not it is infectious, local, constitutional, or both. It is a much named disease, whether it be called Rigg's Pyorrhœa Alveolaris, Suppurative Inflammation, Phagedonic Pericementitis. Is it due to salivary or sanguinary calculus, or both, or fungi, or bacteria? Is it wholly or in part due to traumatic inflammation? Is it a local expression of a systemic condition, and located in the mouth because of a predisposition of the gums, the pericementum and the alveoli? Is it a primary or a secondary lesion? Is it never present, excepting in a depraved state of the nervous system? Is it associated with gout, rheumatism, Bright's disease, locomotor ataxia, uterine troubles, and other constitutional complications; the excessive use of salt, or alcohol, which causes increased secretion of uric acid? Is the calculus uric acid? Is it connected with catarrh of the mucous membrane of the nose or pharynx, rachitis, tuberculosis, scorbutis, scrofula, chronic constipation, exanthematous diseases, malaria, diabetes, tabes dorsalis, dyspepsia, syphilis, anæmia, chlorosis, repeated pregnancy, bad air, bad food, hygienic neglect? Or will