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"On the Causal Relationship of Gingivitis with Other Diseases". Original title in Polish: „O związku przyczynowym zapalenia dziąseł z innemi chorobami" by dr Teofil W. Kaczorowski (1830-1889)

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The oral cavity – by virtue of the long-standing custom of examining the tongue as a mirror of gastric conditions – has traditionally constituted an essential and almost preliminary subject of general medical inspection. However, during such examinations, the gums typically receive only cursory attention.

The neglect of careful evaluation of the gums in clinical cases stems primarily from the limited significance that medical science has hitherto ascribed to nutritional and structural deviations in this particular part of the body. Except for a few well-recognized abnormalities in gingival coloration – e.g., pallor in anemia, cyanosis in scurvy, a bluish-violet border in lead poisoning, petechiae in purpura, bleeding in haemorrhagic diathesis, oral thrush in poorly nourished infants and severely ill elderly patients, and specific inflammations such as those caused by syphilis or mercury – standard medical textbooks provide little to no further semiotic or pathogenic insight regarding gingival pathology, relegating the remainder of such concerns to the domain of surgery. Simple gingivitis, often associated with dental disease, has become almost exclusively the province of dentists, largely neglected by physicians.

Yet even ancient physicians noted, during the initial phase of dentition in children, a variety of functional and nutritional disturbances across multiple organ systems – including catarrhal inflammation of the digestive and respiratory tracts, central nervous system disorders, and diseases of the eyes and skin – which appeared to arise concurrently with, and to subside upon, tooth eruption. From these observations, they inferred a peculiar connection between the dentition process and such pathological states. Nevertheless, within the framework of "difficult teething," it was not the gums but the erupting teeth that were typically deemed the culprits. To facilitate tooth eruption, it was even customary to incise the gums with a knife.

Modern pathologists, however, reject the notion that a physiological developmental process such as teething could itself give rise to disease. They regard the aforementioned conditions as coincidental, unrelated to dentition per se, or – at most – as manifestations of the heightened reflex excitability typical of early childhood, particularly in relation to nervous system disturbances.

Nevertheless, despite authoritative scientific opposition, there remain numerous clinical observations – attested not only by many physicians but also by experienced mothers – that certain children exhibit symptoms such as febrile restlessness, vomiting, constipation or diarrhoea, persistent coughing, or recurrent convulsions with nearly every emerging tooth. These symptoms often resolve almost miraculously upon the appearance of the anticipated tooth. It is difficult to attribute such consistent correlations to mere chance.

In my opinion, both proponents and critics of the "difficult teething" hypothesis can be reconciled if

the basis of the causal relationship is sought not in the erupting teeth themselves, but in the condition of the gums. A closer examination of the gingiva during teething readily reveals that in children who teethe without complications, the gums – although slightly hypertrophic – do not change colour, and may even appear paler than usual. These children exhibit little more than increased salivation and mild gingival itching, which they express by pressing their lips together or placing hard objects in their mouths without showing any additional signs of irritation.

In contrast, children experiencing "difficult teething" exhibit markedly different gingival characteristics: the gums are reddened to varying degrees, dry, tense, glossy, swollen, and warmer to the touch – not only above the erupting tooth, but often across a broader area or even the entire maxillary arch. This presentation indicates an inflammatory irritation of the gingiva, which, although typically confined to hyperaemia, may – if prolonged – progress to superficial ulcerations (*erosiones*) or even to deeper abscess formation within the submucosal tissue.

Assuming such gingival inflammation may, under conducive circumstances, spread across contiguous mucosal surfaces – initially to the tongue and oral cavity, subsequently to the pharynx, and from there either downward to the stomach and intestines or to the larynx, trachea, and bronchi, or upward to the nasal cavity, through the nasolacrimal ducts to the eyes, through the ethmoid bone to the meninges, and via the Eustachian tubes to the inner ear, labyrinth, and brain – one may further hypothesize that irritation of trigeminal nerve endings within the inflamed gingiva could transmit stimuli to other branches of the same nerve or reflexively to vasomotor nerves in both adjacent and remote anatomical regions. Such mechanisms may provoke neuralgia, convulsive episodes and inflammation of the skin or internal organs. Within this framework, the spectrum of disorders associated with difficult dentition may be understood either as propagation *per continuum et contiguum* or as reflex complications arising from gingival inflammation.

To the process of teething itself, one need only attribute a facilitating role, to the extent that – during this developmental phase, as in others; such as ovulation – there is increased lymphatic flow surrounding the newly forming structure. This heightened flow promotes the migration of white blood cells, potentially initiating inflammation. However, the true proximate cause of inflammation should be sought in fungal infections, which find a favorable environment for colonization in necrotic organic matter and degenerated leukocytes.

Having long subscribed to this now increasingly evidence-based theory of parasitic causation – particularly in relation to inflammatory diseases – I have, for over a decade, sought to validate it through clinical outcomes based on targeted therapy, even before microscopic diagnostics had reached a level of precision sufficient to confirm its validity.[1]

Accordingly, in cases of gingivitis in children experiencing difficult teething, I have focused treatment almost exclusively on the continuous disinfection of the gums. I have repeatedly observed that the systemic diseases accompanying teething tend to subside – sometimes within mere hours (as in the case of nervous irritation) – and consistently resolve much more rapidly than with conventional treatment aimed at the specific symptomatic organs.

For disinfecting inflamed gums, I primarily employ iodine in a saline solution (e.g., sodium chloride 1.0 per 100.0 with tincture of iodine 0.25–0.05, adjusted for the child's age), which is generally well tolerated in this form. Since young children are unable to rinse the mouth effectively – and because contact of the solution with the oral and esophageal mucosa is in fact desirable – I administer it internally: one teaspoon every half hour to one hour. I have never observed any adverse effects; on the contrary, the treatment typically produces a beneficial impact on the stomach, which is frequently involved in the infectious process.

Within a few hours of initiating this regimen, gingival erythema and tension tend to subside, and the child's restlessness and fever diminish. If the gingival inflammation had been accompanied by vomiting, diarrhoea, severe coughing, or laryngeal rales, seizures, conjunctivitis, skin rashes, *et cetera*., all these various and seemingly unrelated pathological phenomena, which, when reduced to a common denominator, disappeared very quickly as the gums became paler, although the eruption of the tooth sometimes still lagged behind. It is self-evident that I am referring here to symptoms caused not solely by catarrhal inflammation, but also by conditions that, upon exacerbation during the eruption of nearly every tooth, often persist relentlessly for weeks.

If such a simple treatment – supplemented by the maintenance of regular bowel movements with the use of castor oil – can directly impact the resolution of gingival inflammation, and thereby alleviate distant sources of irritation, it is impossible to overlook the conclusion drawn from the therapeutic outcome (*ex juvantibus*), namely, that the point of origin of these varied disease phenomena is nothing other than gingival inflammation.

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1 „O terapii zastosowanej do teorii pasożytniczej" in: Pamiętnik z II Zjazdu Lekarzy polskich we Lwowie.

Justifying such a conclusion based on therapeutic effects is not an extraordinary occurrence in medical science. After all, this approach underlies diagnostic reasoning in certain ambiguous cases of infectious diseases, and it is the foundation upon which Lister built his famous theory of wound infection.

Further advancement along this therapeutic path led me to a discovery that the same causal connection between gingivitis and other ailments observed in teething children also exists in later life, *mutatis mutandis*, as one might expect *a priori*. Adults provide even more useful material to clarify this issue. While in children, gingivitis typically presents acutely, followed by quickly passing symptoms devoid of subjective signs, in older individuals, the chronic progression of certain gingival conditions, accompanied by health deviations, enables a more comprehensive study of both objective and subjective phenomena. This facilitates a clearer examination of the interrelationship between the supposed cause and its suspected consequences.

For, if one experiences those serious, prolonged diseases, which resist ordinary treatment and cease only after the gums have healed and reappear when gingival inflammation recurs, the argument – although based on a weak foundation of only therapeutic evidence – gains more weight. Furthermore, if, by intermittently suspending treatment of the gums, we are able to repeatedly induce the same symptoms as before, the case may even be considered a physical experiment.

By proceeding in this manner, I have reinforced my conclusions regarding the significance of a condition, hitherto underappreciated in pathological terms: inflammation of the gums. I now consider it my duty to draw the attention of my colleagues to this matter. Through collective investigation, the medical community may uncover this neglected etiological field, thus advancing one of the most important tasks of medical science, namely, the prevention of disease – a task that, in this domain, can be more effectively and efficiently addressed with the means at our disposal than in other pathological areas.

Following this introduction, allow me to consider first the origin of what I regard as the highly significant condition of gingivitis – specifically, its fundamental and circumstantial causes. If contemporary pathological research increasingly identifies and, in some cases, experimentally confirms the pathogenic role of various microorganisms – chiefly fungi – in a growing number of diseases, one must indeed wonder how the oral cavity, the principal gateway through which all invisible enemies of our organism pass along with air and food, could remain unscathed. It is, after all, the vestibule to the body's inner chambers, where these agents must necessarily linger for some time.

Even in a state of good health, the mouth harbors fungi such as *Leptothrix* and *Oidium albicans*, which lie dormant for an opportunity and, once nourished by a suitable substrate, may proliferate into countless generations. Even the tooth, hard as a flint, cannot withstand their action: dental caries consists of the corrosion of the dentin (tooth bone) by *Leptothrix*, which Davain classified among the fission fungi, playing a vital role in initiating internal diseases.[2]

How much easier, then, must it be for these fungi to invade the soft gums – constantly exposed to trauma that breaks down the protective epithelium, and further offering favorable hiding places in the interdental spaces. Thus, the possibility of gingival infection by fission fungi cannot be reasonably doubted. However, strict proof based on microscopic analysis remains premature, given the current inability to exclude such fungi even from healthy mouths.

Let me briefly mention certain circumstantial harms which predispose the gums to inflammation, by lowering the resistance of their epithelium to the ever-present microbial threat. As with any organ, these harms can be categorized as mechanical, chemical, or functional in nature. Under the first category, one may include the trauma experienced by teething children when given hard foreign objects – such as bone rings or violet root chews – to alleviate the troublesome itching of the gums. Not only can these easily damage the mucous membrane, but through friction they may even more effectively implant infectious agents.

In adults, natural teeth offer some protection to the gums against trauma. However, one of the most significant stimuli for inflammation of both the gums and the palate is the use of artificial teeth, usually mounted on broad rubber plates. The pressure and tight suction exerted by these widely used prosthetic devices causes maceration of the mucous membrane – especially if they are not frequently removed and if the oral cavity is not thoroughly cleaned and disinfected after every meal. Between the artificial and natural jaws, a veritable breeding chamber for fungi arises, nourished by food remnants lodged therein and kept at an ideal warmth. The resulting fermentation – usually acidic – often acquires a putrid character, especially in cases where decayed teeth were not extracted prior to the fitting of the prosthesis, but merely shortened near the gum line, leaving rotting stumps that slowly infect the surrounding periosteum, and from there, the gums become constantly infected. In individuals who neglect proper oral hygiene, a repulsive odor can be detected from afar,

2 Willoughby-Miller observed *Leptothrix* only on the surfaces of decaying teeth; in the dentinal tubules, however, he found bacilli, and deeper still, micrococci.

and after removing artificial teeth, the gums and palate present a disgusting appearance resembling raw, red, and in places grey-spotted meat.

Among chemical irritants, the first to be mentioned must be inappropriate, spicy foods, artificially seasoned and cured, sticky, sweet pastries that adhere between the teeth, irritating the gums and at the same time providing nourishing substrate for fungi.

Acids, or substances prone to acidic fermentation, harm indirectly by predisposing teeth to decay, from which toxins later spread to the gums. The acidic reaction of oral fluids dissolves the calcareous parts of the tooth bone, thereby opening the interior to fungal invasion. Many medications, such as mineral acids, liquid iron preparations, mercury, lead, and phosphorus, act harmfully on teeth and gums.

Among functional irritants, we must add disturbances of the trophic nerves, excessive stimulation during periods of development, such as tooth eruption in children and puberty, menstruation, or pregnancy in adults. Most significantly, however, gingival vulnerability increases with a decline in nutritional innervation during general debilitation resulting from acute or chronic illnesses, inherited conditions, especially rickets and syphilis, and even psychological traumas, after which loss of appetite or stomatitis due to sudden fungal overgrowth in the oral cavity often occurs.

Lastly, the most important role in both the initiation and perpetuation of gingival inflammation must be assigned to the tooth itself, which, under certain circumstances, exerts all three kinds of harmful effects on the gums: mechanical (when it is loosened), chemical (when it decays), and functional to a degree (during eruption). A mobile tooth, even if healthy, irritates the gums not only through pressure but also by providing fungi with sheltered niches in the undermined gum tissue.

A decaying tooth, on the other hand, irritates the gums firstly through its sharp chemical secretions, and secondly by serving as a persistent breeding ground for fungi, which can infect the gums given any opportunity. Its involvement in gingivitis is most vividly demonstrated by the fact that individuals with completely healthy teeth, or without any teeth (such as children or the elderly), almost never suffer from mercurial, syphilitic, or phosphorus-induced gingivitis.

Now a few words about the individual forms of gingival inflammation. When examining the oral cavity, it is essential to ascertain whether the patient uses artificial teeth, and their removal must be insisted upon. This is somewhat delicate in the case of the fairer sex, but without this step, the most dreadful destruction of the gums can remain hidden from the physician's eye. In mature individuals, we usually find a few decayed teeth, particularly molars. If a tooth decays from the inside, it has little effect on the gums; but if it decays from the sides, the adjacent gum margins tend to be red, swollen, with a pronounced engorged border, sometimes slightly ulcerated. This is the most common form of localized gingivitis associated with a decaying tooth, which typically causes no clear discomfort, but when acutely exacerbated – whether by unsuitable foods or sudden temperature changes – easily provokes facial neuralgia along branches of the trigeminal nerve, or in worse cases, what we call "fluxion," i.e., inflammation of the connective tissue between the gums and periosteum, usually progressing to suppuration.

This form of gingivitis is regularly accompanied by a foul oral odor, noticeable in the morning or after prolonged fasting, essentially caused by periosteal decay – a well-known phenomenon. Another often overlooked consequence of this inflammation is a regular loss of appetite in the morning, originating from stomatitis, especially of the tongue, which at that time is thickly coated in grey. This catarrh arises from the spread of infection from the localized focus to the surrounding oral mucosa during sleep, when the usual flushing by drinks or abundant saliva is suspended, allowing pathogenic fungi to spread freely. The same mechanism explains the loss of appetite during the day after prolonged fasting, which we commonly call "being overly hungry."

The same limited form of irritative gingivitis can occur even with completely healthy teeth, spreading around several molars due to lingering irritating food debris, especially sticky sweets, after which calculus begins to form slowly along the tooth margins. This inflammation reveals itself by occasional neuralgias in the parotid or temporal areas, without necessarily causing appetite loss.

A second form of localized gingivitis is usually observed around teeth with healthy crowns, mostly the front ones, beginning with pronounced swelling of the gums, which take on a dark red or bluish hue and show signs of atony. The gums gradually recede from the tooth, roll back, and become covered on the inner surface with purulent mucus. The exposed part of the tooth becomes brittle, begin to flake, or become encrusted with calculus; in the first case, the tooth becomes increasingly loose and eventually falls out entirely, despite being healthy.

In this way, many people who neglect gum care lose all their teeth painlessly over time. Those affected by this degenerative gum condition – which might be termed atrophied (*athrophirend*) – experience, alongside the coated morning tongue and irregular appetite, frequent postprandial epigastric pressure, excessive belching of empty gases (indicative of abnormal fermentation in the stomach), accumulation of saliva in the mouth, and

at times cramping gastric neuralgias. Acute exacerbations of gingivitis may take on a putrid character, with febrile response and even cerebral irritation. After the remaining teeth fall out, these pathological symptoms disappear for good, lost appetite returns, and a phase of noticeably improved general health begins.

Finally, we must mention the diffuse gingival inflammation involving the entire jaw, which may occur regardless of the presence of diseased teeth. In acute febrile illnesses, this condition appears as a secondary phenomenon, an inseparable companion of prolonged fever, manifested by general hyperaemia of the gums and the entire oral cavity. The gums are intensely red, dry, shiny, and later begin to disintegrate, sometimes oozing in places where the gums and lower edges of the teeth are covered with a greyish, later dirty-brown, black coating resembling soot, which does not spread to the tongue. Involvement of the oral cavity becomes the main, if not the sole, reason for the patient's inability to ingest food due to overwhelming aversion.

In chronic illnesses or even in individuals seemingly healthy and not recognizing themselves as ill, diffuse gingivitis arises in a markedly different form. Initially, it presents as a narrow red zigzagging edge along the margins of the teeth, which gradually widens. Along this band – extending slightly onto the crowns – a wider, dirty, sometimes greenish streak of sticky mucus may form. Microscopic examination reveals numerous fungal elements, matted epithelial cells, *Leptothrix* and *Micrococci*. As the inflammation progresses, this red band widens further, eventually encompassing the entire gum, which becomes more swollen, darkens, assumes a bluish hue, and tends to bleed and become painful even with light pressure.

This form of gingivitis is accompanied by a specific, acidic, unpleasant mouth odor, entirely different from the one caused by carious teeth; there is loss of appetite, aversion to meat dishes, nausea, bloating and pain in the gastric region, and a tendency toward constipation.

Sometimes it assumes an acute character, and when accompanied by fever, it is referred to as chronic gingivitis; it may progress or regress depending on dietary hygiene – yet the inflammatory process of the gums almost never entirely subsides, becoming a hidden source of general exhaustion, emerging slowly and gradually provoking an increasingly prolonged sequence of symptoms of nervous disorder, leading eventually to insomnia and, in some cases, even mental derangement.

Forms of gingival affliction arising from mercury, lead, syphilitic, or scorbutic intoxication shall be omitted herein, as they are sufficiently known and duly acknowledged in pathological literature.

Our main goal here is to draw attention to the most common, simple gingivitis, for which no person consults a doctor – because in its early stages, it causes little or no pain. By the time the patient presents to the physician, the consequences of gum disease – manifesting in distant organs – have usually become so dominant over the original disease focus that both the patient's and physician's attention is entirely concentrated on them. The initial site of the disease is either overlooked or dismissed as secondary. This very concurrence of circumstances likely explains why the causal link between gingivitis and other ailments has not yet received due recognition.

How many people walk the earth with unsightly mouths, yet who nonetheless appear to enjoy good health – seemingly, for they do not complain? If we consider, however, that even the most minor skin injuries are capable of causing serious illnesses, we cannot turn a blind eye to even slight irritations of the gums. We now know that the real threat of these minor injuries lies in putrid infection. Are we to believe that gums, once stripped of their epithelial covering, are protected by some "ironclad guarantee" from attacks by pathogens – those same pathogens we now guard so vigilantly against in every open wound? True, physiologists see the sulphocyanate in saliva as a disinfectant, protecting the mouth from decay of proteinaceous debris. Yet the limited efficacy of this defence is clearly evidenced by the foul odor of so many mouths we encounter, even when approaching them with a healthy nose.

Let us not forget that saliva is secreted primarily during chewing. Frequent intake of solid foods and rinsing with liquids may somewhat remove or neutralize the constant influx of contaminants from the outside world. But when these actions are suspended – even in otherwise healthy mouths, such as in cases of starvation – we can detect a putrid odor. This is even more the case if proteinaceous exudate from inflamed gums or teeth provides a constant nutrient supply for fungi. And one must admit, no self-respecting person would tolerate such filth on any part of their skin without seeking treatment – yet they endure it passively and indifferently in this part of their body, the very one they press close to their loved ones in the sign of deepest affection. Only ignorance can explain this strange tolerance, as mentioning someone's bad breath is considered socially improper. It is the duty of physicians to openly care for their patients in this regard.

Having now explained our view of the importance of gingivitis as a source of subsequent disorders, let us examine more closely the pathways it may take and takes in our body.

The most direct path, as already noted, leads into the oral cavity. Given the still-disordered state of science regarding the development and pathogenic role of fungi species found in the mouth, I do not aim to spec-

ulate on how specific species spread from their original site – the gums – to the oral cavity. For our strictly practical purpose, it suffices to say that two pathological fermentations can be distinguished in the mouth: acidic and putrid. It is known that these depend primarily on the nature of the nutrients provided to the fungi – the former arising from non-nitrogenous substances, the latter from nitrogenous ones. Both fermentations may be triggered by unhealthy, fungus-containing foods, especially in infants, without the presence of gingivitis.

However, when acidic fermentation suddenly appears in the mouth of a child fed healthy maternal milk, we will almost always find inflammatory signs on the gums. Putrid fermentation, apart from specific traumatic injuries in the mouth, always begins with the gums. Its results are most evident on the lush surface of the tongue, whose shedding epithelial cells are ideal for fungal colonization. Thus, in cases of acidic fermentation, the tongue becomes covered with an increasingly thick grey coating; in cases of putrid fermentation, a thin, dirty-yellow layer forms, through which red, swollen, later dry and shiny mucosa becomes visible.

From the tongue, the infection spreads along the tonsils to the pharynx. As these organs are richly supplied with taste nerves, their involvement inevitably leads to reduced appetite – still present to a degree in the first case (acidic), focused on sharp, irritating foods – but completely lost in the second (putrid), particularly for meat. One might consider this aversion an instinctive defence mechanism to cut off the fungi's food supply – were it not for the fact that prolonged abstinence ultimately harms the entire body.

The widespread belief that the tongue reflects the stomach, and that lack of appetite should be sought in the stomach's condition, particularly in its catarrh, must be eradicated from both physicians' and the public's minds. The state of the tongue determines appetite, not the reverse. As long as the tongue is clean, appetite remains – even in severe febrile illnesses.

Just recall tuberculosis patients who, due to constant coughing and rinsing their mouths, retain a huge appetite until death. The artificial method of feeding through a gastric tube, even in patients experiencing nausea or vomiting during voluntary intake, also proves that lack of appetite is not rooted in the stomach, but rather in the infected pharynx – which spasms upon contact with undesirable substances. If one can nourish such patients effectively while bypassing contact with irritated mouth and pharynx, it should logically be a physician's task to constantly disinfect those areas. Gone are the days of starving out disease.

More and more loudly, physicians now declare that most patients with prolonged fevers – especially in typhoid – die not of the disease itself but from starvation. Even when assuming that such patients lack appetite or thirst due to cerebral involvement, physicians rightly urge frequent intake of fluids and nourishment. Still, this advice is only partly accurate: these patients will eagerly drink cold water, but reject even liquid, cold foods – because their mouths and throats, dried and infested with parasites, have lost all appetite.

If we start regularly moistening the mouth with a disinfectant solution that is not offensive in taste, such patients soon drink milk or buttermilk by the litre. Following this principle, I have long used potassium chlorate in all febrile diseases. However, since larger doses of this drug were found to be toxic (though I have never personally seen such effects), I now use iodine in saline solution (Trae. Iodi 1% in NaCl solution), administering half to one tablespoon every 15–30 minutes. For the past five years, using this method on thousands of patients – many receiving up to 2 grams of iodine daily for weeks – I have observed no adverse effects. Even children take this medicine readily. Under its influence, the oral cavity retains moisture and cleanliness, avoiding the formation of those deadly black coatings that reappear quickly when treatment is suspended, e.g., overnight.

Iodine, even in minimal doses, is among the most effective anti-putrid agents, as shown by Binz. Its combination with sodium chloride is useful not only for masking iodine's taste and slowing decomposition, but also for replenishing essential blood components lost during fever – and NaCl itself has disinfecting properties. I consider my iodine-saline solution superior to the commonly used hydrochloric acid in febrile cases. Although hydrochloric acid initially appeals to patients, they soon grow weary of its taste; and despite its physiological benefit for digestion, like all mineral acids, it severely damages teeth – leading to the tooth loss often seen after severe fevers.

Only potassium permanganate might compete in oral disinfection, though it can only be used as a rinse – its taste and potential systemic toxicity preclude internal use.

In afebrile diseases, we also use the iodine-saline mixture to combat oral cavity infection, but in less frequent doses. Alternatively, we combine the iodine tincture with tincture of myrrh, valerian, eucalyptus, guaiac, oak galls, cinnamon, etc. (*Tra. Iodi p. 1: Tr. Myrrhae, or Valerian.* E*ucalypti, Guajaci, Gallarum, Cinamomi 10 p.*), 20 drops in a small glass of water. This mixture is recommended partly for thorough rinsing of the mouth and throat, and partly for slow swallowing – since few people can reach the lower pharynx effectively by rinsing alone. If someone were to swallow the entire mixture, it would not harm them – on the contrary, by disinfecting the stomach as well, it would aid digestion.

Such oral disinfection should be performed at least after every meal and, in severe cases of gingivitis, as often as every hour – or even more frequently – and always before bedtime. In the morning, before using a rinse, it is recommended to clean the teeth and gums of overnight residue and mucus using a brush coated with soap (the same kind used for washing the face). Rubber brushes are more suitable for this purpose than the usual bristle ones, which easily injure the gums – especially when ulcerated and prone to bleeding.

Instead of soap, one can also use powdered calcined magnesia or powdered oyster shells. Artificial teeth should be removed and thoroughly rinsed during each cleaning of the oral cavity; in cases of severe gingivitis or any acute illness, they should be entirely set aside for a time. By continuously cleaning the mouth and throat in one of these ways, it is possible – regardless of the illness – to maintain at least a modest level of appetite.

Let us be clear, however: as long as decaying teeth remain in the oral cavity, constantly releasing putrid agents, proper disinfection is nearly impossible – hence the need for frequent rinsing. On this occasion, I should also note that many people's strong aversion to milk is mainly due to oral cavity infection from rotting teeth and gums. Such individuals resist drinking milk for breakfast especially, because their mouths are most contaminated in the morning and crave something sharp or stimulating instead. Additionally, milk that reaches the stomach tainted with fermentative agents from the mouth undergoes abnormal fermentation, causing digestive disturbances – and further strengthening the aversion to milk.

Combating oral infection with the methods described allows us to overcome even the most deep-rooted disgust toward milk products – especially if we assist the stomach further with alkaline preparations and bitters.

Infections of the oral cavity continue down the throat to the stomach, bringing with them both pathogenic fungi and their secretions. As long as the digestive organs are robust and resilient, they can probably handle this burden. However, when the system is weakened – whether by overly rich or heavy foods, or by general cachexia – the production and efficacy of digestive secretions decline, and foreign ferments gain the upper hand, severely disrupting digestion. This results in a range of ailments: pressure in the epigastrium, painful stomach cramps, bloating, belching of gas or sour liquids, nausea, and even vomiting. These local symptoms are often accompanied by more distant ones: headache, insomnia, and general nervous disorder.

These well-known cases, commonly labeled "dyspepsia," are usually attributed to gastric catarrh. Recognizing the inadequacy of attributing all digestive issues to the popular notion of stomach catarrh, researchers have introduced the concept of "nervous dyspepsia" to explain indigestion in chlorotic individuals (Leube).

Although it is not to be denied that innervation exerts a certain influence upon the process of digestion – as evidenced by digestive disturbances following strong emotional or physical stimuli, particularly of a depressing nature, or during certain developmental periods, especially those related to the sexual system and its disorders, and above all in cases of anemia, where changes in the composition of gastric juice and in the regularity of the movements of the digestive tract are observed. – I must nevertheless assert that, even in such instances of nervous dyspepsia, signs of gastric infection originating in the oral cavity can, in the majority of cases, be identified.

It has occurred to me all too frequently that young women afflicted with the most pronounced chlorosis, yet possessing a clean oral cavity, sound teeth, and intact – though pale – gums, exhibit no symptoms of digestive derangement, aside from a craving for acids or for substances known to neutralize them. Whereas, in the presence of disorders of the oral cavity, they are not only prone to all manner of dyspeptic complaints, but are also completely intolerant of iron preparations, which would otherwise be highly beneficial. Iron compounds – especially the acidic ones – appear to promote the development of certain fungi already present in the oral cavity, which then proliferate further into the stomach.

It is known that some physicians administer hydrochloric acid to such chlorotic individuals experiencing persistent dyspeptic symptoms before prescribing iron, with the aim of restoring proper digestive function. While I do not dispute the salutary action of this physiological agent of digestion, I must nevertheless remark that in such cases, I was able to attain the desired result far more expeditiously by thoroughly disinfecting both the oral cavity and the stomach – for instance, by administering, prior to meals, ten drops of *Tinctura Valerianae aetherata* and twenty drops of *Tinctura Jodi* in half a small glass of water, followed by an iron preparation in pill one hour after eating, which under such conditions produced no inconvenience whatsoever. By destroying the abnormal fermentative agents present in the accessible oral cavity, and by weakening their activity within the stomach, I have found it entirely feasible to dispense with the use of pepsin and peptones, without detriment to the patient – provided that rigorous attention is paid to maintaining oral hygiene.

It is thus impossible for me not to arrive at the conviction that even nervous dyspepsia is, for the most part, dependent upon gastric infection conveyed from the oral cavity, when it is itself infected through dis-

eased teeth and gums. In support of this view, I may incidentally recall the well-known fact that anemic women during menstruation and pregnancy are so frequently afflicted with toothaches, with the onset of caries invariably accompanied by gingivitis – disorders which consistently proceed in tandem with disturbances of digestion.

And yet, considering how the stomach is capable of executing its physiological functions even amid grave anatomical injuries – such as in carcinoma or perforating ulcer, provided they are not situated so as to obstruct its lumen – we must conclude that every case of dyspepsia rests upon abnormal fermentations of gastric contents. These fermentations are triggered by the proliferation of various fungi, the specific effects of which in several respects have already been recognized. These fungi may be introduced directly into the stomach through food and drink; yet even ingesta devoid of such contaminants may become tainted with them in the oral cavity of the individual who, being afflicted with gingivitis and dental disease, harbors within himself an inexhaustible breeding ground of such parasites.

A second locus for such cultivation is also to be found in the pharyngeal cavity when in a state of chronic inflammation. The stomach itself – excluding gross alterations of tissue, or irritations brought on by chemical agents such as alcohol – appears, in all probability, to contribute the least to the onset of dyspeptic symptoms. Indeed, what is commonly termed gastric catarrh is more likely a consequence, rather than a cause of impaired digestion.

It is commonly known that alkaline remedies are widely employed against indigestion. By rendering the nutritional environment of the upper digestive tract inhospitable to fungal growth, these agents help suppress abnormal fermentative process within the stomach. Diet acts in a similar direction – its benefit lying in the selection of foods unfavorable to the nourishment of minute parasites. It is common knowledge that even coarse foods, such as unsoaked herring, are well tolerated by habitual drinkers suffering *par excellence* from gastric catarrh. Less commonly acknowledged, yet observed almost daily, is the fact that patients afflicted with the gravest illness, whose stomachs cannot abide even the lightest of broths (as in the final stage of Bright's disease), nevertheless consume and digest salted herrings with relish. This tolerance may be attributed to the herring's content of salt and trimethylamine, both of which act as antidotes to the digestive disturbances caused by the lowest forms of microbial life.

Thus, in order to combat indigestion with lasting effect, the alimentary canal must be systematically disinfected, beginning with its most accessible station – the oral cavity. Among antiseptics, iodine remains preeminent: a substance as potent in action as it is, regrettably, unpalatable to the body.

In the mouth, at least, we possess the possibility of continual ventilation, the first condition of which, as mentioned above, is the removal of all inflammatory foci, foremost among them decayed teeth. These, if they cannot be adequately covered with fillings, must perforce be extracted.

Far more difficult is the case of the pharynx. It is frequently the site of chronic inflammation, often involving enlarged mucous follicles, and harbors battalions of fungi ceaselessly descending into the stomach. Though the spacious stomach cavity presents the greatest difficulty for complete ventilation – especially in persistent cases of indigestion resulting from gastric dilation– we are forced to resort to laborious lavages, these, however, remain comparatively rare.

In the majority of cases, indigestion takes root in the mouth and pharynx. It can be mitigated, to a considerable extent, by consistent disinfection, removal of hyperemias and inflammations of these organs, and finally by a diet tailored to their pathological condition. That inflammation of the gums and teeth stands in direct causal relation to indigestion, I could substantiate with a thousand anecdotal cases. I shall, however, limit myself to recounting but a few examples – those owing to the long-term and closely supervised nature of my observations, most clearly illustrate the impact of oral disease on appetite and digestive function.

1) A married woman, aged 50, despite aortic valve insufficiency acquired 30 years prior in the course of rheumatic fever, enjoyed for seven years the most impeccable appetite and digestion. After a grievous sorrow, she began to lose her appetite and, after each intake of solid food, to experience such tormenting cramp-like pain in the epigastrium, along with belching and heartburn, that she confined herself to liquid sustenance, mostly tea.

Carlsbad waters, though repeatedly tried, brought temporary relief during and shortly after their use, but the former suffering invariably returned, despite strict adherence to diet. A foul morning breath then led me to a more thorough inspection of the teeth, which – aside from a few excellently filled – revealed no notable defects. Yet the gingival margins – especially around the anterior teeth – exhibited marked swelling, slight ulceration and recession. The tongue was always coated, and the pharynx appeared reddened.

From that point, rinsing of the oral and pharyngeal cavities with a solution of iodine and myrrh, a portion of which was to be swallowed, removed the gastric pain within days and restored her former appetite. Whenever, however, the patient – imbued with an almost pathological aversion to any "Latin" culinary remedy – ne-

glected, even for a day, her oral cleansing, particularly during menstruation, the gums, persistently somewhat congested, would swell anew, the foul breath would return, and with it the whole train of former dyspeptic symptoms. Repeating itself with some regularity over the past five years, this interplay between the diseased gums and the attendant symptoms of appetite loss and indigestion leaves not the slightest doubt as to the causal connection I propose.

2) Gymnasium professor P., 40 years of age, had suffered from poor digestion since his student days, manifesting in postprandial discomfort, headaches, and persistent insomnia – thereby compelled to maintain the most cautious diet. Four years ago, his long-standing ailment intensified dramatically, culminating in an unshakeable aversion to food, gastric pain even after liquid nourishment, and the aforementioned nervous disturbances that confined him to bed. Emaciated and despairing, he appeared to be at death's door.

Summoned to him two months into his illness, I was immediately struck by the characteristic foul odor from his mouth, and upon examining his oral cavity, I found the gums around all otherwise sound teeth to be deeply reddened, swollen, bleeding in places, with lightly ulcerated edges. The patient, preoccupied only with his stomach, confessed then that he had been spitting blood from the mouth for many years, but had never addressed the matter, nor had any physicians paid it heed, as he experienced no oral pain.

After 24 hours of mouth rinses – iodine with kitchen salt every 15 minutes – his appetite reawakened; a week later, on a milk diet, digestion returned; and within two weeks, he exhibited a pronounced appetite for meat, which he then tolerated well. Complete recovery followed after healing of the gums, for which – given the marked vascular dilations – he was treated thrice daily with a tincture of oak bark. The patient initially used the salted solution internally, but later limited himself to mouth rinsing alone. Each time he – at first disbelieving the connection between diseased gums and gastric distress – abandoned the prescribed oral hygiene, he relapsed into dyspepsia. Eventually, having been convinced by experience, he began tending his gums more diligently, washing them each morning with soap, then rinsing with a weak potassium permanganate solution, and has since enjoyed robust health. One summer, returning from vacation where he had neglected his gum care, he suffered a mild relapse of his condition, which, however, was entirely resolved shortly after he resumed vigorous local treatment.

3) Anna S., a 70-year-old widow, bedridden for nearly a year due to general debility and exhaustion, emaciated to the utmost degree, anemic, regarded as incurable she was admitted to the hospital. Apart from coffee, she takes no nourishment, owing to her aversion to other foods and the gastric pains she experiences after ingestion. In both jaws, to the fore, protrude six teeth, already much loosened, surrounded by markedly swollen and at their margins ulcerated gums. The tongue is ever red and dry; likewise the act of swallowing; the epigastric region is tender to pressure; the abdomen distended; a tendency to constipation prevails. Other functions present nothing abnormal, although the patient, believing herself to be paralyzed in the lower limbs, had despaired of recovery.

Upon the extraction of all teeth, the gums, with the aid of iodine and myrrh, healed within the space of a week; the tongue became moist and properly coloured; a most lively appetite from that moment began rapidly to restore the waning strength of the old woman, who, after six weeks, steady again on her feet, left the hospital in full health.

As a standard example of the causal connection between gingivitis and acute affections of the alimentary tract, I must mention the entire series of patients who present themselves immediately after every Christmas season, during which the consumption of sticky confections such as marzipan and gingerbread is the order of the day. These are cases of gastric fever, with aversion to food, severe stomach pains, occasional vomiting and constipation, often accompanied by intercostal neuralgia. The characteristic fetor of the breath and the red border of the gums at once direct me to the primary cause of the ailment, which indeed, under vigorous disinfection of the gums, subsides far more rapidly than upon the administration of those usual remedies aimed at the stomach and intestines alone.

To this class also must be assigned the cases of gastric distress dating from the use of artificial teeth insufficiently cleansed.

4) Mr. Z., aged 50, of exemplary constitution and nourishment, having never suffered any serious illness in his life, had for several years experienced, after each dinner, a distressing distension in the upper part of the abdomen, which subsided only after several hours – thus, once digestion was complete. When consulted, and as always beginning the examination with the teeth, I found the upper jaw covered by artificial teeth, and upon their removal, the gums and the entire hard palate appeared markedly reddened and swollen. Furthermore, the left lobe of the liver was found to be significantly enlarged and slightly tender upon pressure.

Since the patient, who for two years had not once removed his artificial teeth, began to attend most diligently to the hygiene of his oral cavity – removing the teeth after every meal and brushing the gums and palate with an infusion of iodine and oak bark – his former abdominal discomforts disappeared, and upon seeing

him again after four weeks, I found no trace of liver enlargement. It thus seems that fungi descending from the oral cavity into the stomach may not only cause profuse gas formation during digestion but may even, by penetrating through the stomach wall into the adjacent hepatic lobe, induce the aforementioned swelling of the left hepatic lobe.

The result of treatment limited solely to the inflamed gums and palate, and the prevention of further fungal growth in the oral cavity, should convince even the staunchest sceptic that only the fungal infection was the cause of the aforementioned abnormalities, which, as I have had the opportunity to observe over several years, have never recurred.

Even more frequently than in adults, I observe the harmful influence of even the slightest gingival irritation upon the digestive tract in infants, though they are fed uniformly at the mother's breast – making improper nourishment a less likely cause. Even limited reddening of the gums provokes in infants digestive disturbances accompanied by great restlessness and fever, which, after a preliminary purgative and frequent administration of a saline iodine mixture, are usually allayed within a day.

In the further course of diseases arising from gingivitis – through contact transmission along mucous membranes – I must mention inflammation of the larynx. It occasionally occurs in persons wearing artificial teeth who neglect proper cleansing of the oral cavity. This laryngeal involvement reveals itself through extremely persistent coughing in violent and prolonged fits, later joined by hoarseness and copious, watery, stringy, foamy expectoration. Closer examination reveals a dark-red coloration of the entire pharynx and laryngeal entrance – that is, the epiglottis and aryepiglottic folds (less often the true vocal cords) – with a smooth and glistening appearance.

1. In the year 1875, I reported in this very place the case of Miss P., who for a year had suffered from an exceedingly troublesome cough, hoarseness, and abundant expectoration of mucous, occasionally blood-tinged sputum, wasting away and being considered by several physicians to be tubercular. Examination revealed extensive ulceration of the entire upper jaw, covered by a broad rubber plate with artificial teeth, which the patient had not removed for a year; the palate was spongy, dark red, and mottled with grey patches; the entire pharynx was inflamed and glistening up to the entrance of the larynx, where a small grey erosion the size of a large pinhead was observed on the right true vocal cord, upon a pink background. All treatments had been fruitless until the patient, hitherto prevented by vanity from parting with her artificial teeth, finally agreed to refrain from wearing them for two weeks. Rinses and inhalations of iodine and oak bark tincture quickly resolved the inflammation of the oral cavity, pharynx, and larynx, along with the cough, leaving only a slight roughness of the voice due to minor loss of substance in the affected vocal cord. Having since been instructed to maintain rigorous care of her dental hygiene, she remains healthy to this day.

The grey spots on the palate were identified as Oidium albicans, and the ulcer on the vocal cord was likely caused by the same parasite.

2. Mrs. C., married, aged 42, had suffered for two years from attacks of the most violent coughing, especially when lying flat, depriving her of sleep and exhausting her strength to the point of near-fainting. She had been treated by various, even specialist, physicians who had regarded the case as hysterical, with only fleeting relief – chloral being the most effective.

Objectively, nothing could be found in this well-nourished, even corpulent woman, except for a diffuse redness of the entire pharynx and laryngeal entrance, more pronounced in several streaks. Only after the removal of artificial teeth, which she had used for two years, did slight ulcerations appear around the gums and a marked swelling and redness of the entire upper palate, directly continuing into those dark-red streaks on the posterior wall of the pharynx. The patient also confessed that, on the advice of a dentist, she rarely removed the teeth so as not to disrupt the close adherence of the rubber plate to the palate. After a week without the teeth and careful disinfection of the oral cavity, the coughing fits ceased permanently.

I shall not elaborate further on the propagation of gingival inflammation from the pharynx through the Eustachian tube to the inner ear; I shall only mention that the American specialist Saxton (American Journal of the Medicine) in one hundred cases of inner ear involvement, found the cause six hundred times in diseased teeth, which amounts nearly to the same thing as if he had attributed it to the gums, since one usually accompanies the other.

As for the causal link between gingivitis and ocular diseases, I also leave the judgment to specialists due to the lack of sufficient observations. Nonetheless, I cannot omit a case I personally experienced, which greatly contributed to my focusing more attention on the etiological significance of inflammation of the teeth and gums. In the year 1877, an interstitial keratitis appeared in my left eye for unknown reasons, progressing slowly in a perpendicular line from the outer edge, about which leading ophthalmological authorities unanimously stated that, if unchecked, it would cover the entire cornea. When more than two-thirds of the membrane was already clouded with infiltration and severe ciliary neuralgias began to occur, as well as pain in points above

and below the orbit, I noticed that the evening outbreak of these pains was always preceded by burning in the gums of the two upper left molars, beneath the eye. The second of these had long been decayed and was filled a year earlier, with a drilled hole left open for pulp ventilation. Instinctively sensing a connection between this tooth and the neuralgias, I had the filled tooth extracted – against the advice of my physician, who did not share my view – after which I spent the first pain-free night in four weeks. Two days later, however, the suborbital pain returned, heralded once more by burning in the gums around the first molar, otherwise entirely healthy. Only after the extraction of this tooth – indeed completely intact, but surrounded by inflamed gums – did all pain permanently disappear; the previously entirely insensitive cornea regained its sensitivity, and the inflammatory condition, which had resisted even the most heroic treatments, halted at the stage it had reached and began to subside, later allowing for the appearance of only superficial, localized infiltrates in the vascularizing outer layer.

To explain the causal link between the keratitis and the teeth, I must add that shortly before the onset of the eye condition, while maintaining the patency of the drilled hole in the filled tooth, I noticed – by the putrid odor of the needles used for this purpose – that the dental pulp had undergone necrosis. It is therefore possible that the purulent discharge, having infected the gums of the diseased and adjacent healthy tooth, via fungal propagation through the lymphatic channels of the connective tissue sheath of the relevant trigeminal branch, induced inflammation of that branch (*neuritis ascendens*), which, having spread further to the branch supplying the eye's upper surface and having desensitized the cornea, opened the way for infection to seed in the very substance of the cornea. At the same time, constant irritation of the trigeminal nerve endings in the inflamed gums of both teeth may have, via retrograde action on the vasomotor nerves of the cornea, on the one hand promoted the advance of inflammation in the eye, and on the other triggered the periodic neuralgic outbreaks radiating to the infraorbital and supraorbital foramina. With the resolution of gingivitis after both teeth were extracted, the entire pathological condition came to a halt.

Speaking of the connection between diseased teeth and gums and the eye, I allow myself finally to parenthetically mention one circumstance: that in several cases of glaucoma I observed, there was always extensive destruction of the teeth and pronounced gingivitis. It may perhaps be a reflex transferred to the vasomotor nerves of the eyeball from irritation of the trigeminal nerve in the jaws, serving as a stimulus for that still insufficiently explained pathological process.

That irritation of the trigeminal nerve in diseased teeth, by radiating to other branches of that nerve, causes neuralgias of the head, is a generally known fact. I would, however, like to clarify this fact by the observation that it is not so much the tooth itself, but rather the inflammation of the surrounding gums that matters here. Anyone can easily convince themselves that teeth decaying from the inner crown, the so-called *caries sicca*, as long as the pulp is not exposed, break down slowly without any pain, and never cause pain in more distant points along the trigeminal nerve. It is a different matter when the tooth decays from the sides, for in such a case it regularly infects the gums and often the periosteum as well; among these, the former typically causes radiating pains around the eye, in the forehead, temples, and ear, while the latter provokes only a localized, constant pain. Although a decaying tooth is often the primary cause of gingivitis – frequently persistent until the tooth is extracted – there are nevertheless too many cases in which mere inflammation of the gums around otherwise entirely healthy teeth triggers violent, somewhat periodic neuralgias. This can especially be observed after acute gingivitis caused by unsuitable sugary foods affecting the posterior teeth; and in cases of chronic gingivitis of the anterior teeth, especially in the elderly, one sometimes sees radiating pains above and below the orbit, which are most rapidly resolved by removing the already loosened teeth.

In general, all facial neuralgias – aside from the exceedingly rare cases of tumors located near the branches of the trigeminal nerve – can be attributed to inflammation of the teeth and gums, and by targeting the inflammation in treatment, neuralgias can be most easily resolved. If, despite healing of the gums – usually requiring removal of the relevant teeth – the neuralgia persists stubbornly, one must suppose that due to prolonged nerve irritation, changes have developed in the nerve tissue requiring intervention of a surgical knife.

There would not be enough space here to present the relevant case studies confirming the above statement.

Cases also occur where, during severe gingivitis, neuralgias appear in trunks emerging from the spinal cord, e.g., intercostal nerves.

Thus, I observed in the Sisters of Mercy hospital a woman, 56 years old, suffering from painful sensations in her left side and severe pain in the left sciatic nerve. Unable to find other abnormalities except for severe gingivitis and decayed teeth on the right side of the jaw, I began by treating the oral cavity, after which all pain gradually subsided. Three months later, the woman returned, complaining of the same ailments. Finding a new flare-up of gingivitis, I extracted the remaining decaying teeth aggravating the inflammation, and within a week, the patient regained full health, which,

as I verified two years later, remained permanent. I do not venture to explain this coincidence, but the fact remains a fact.

The skin is also a field upon which gum irritation can send waves of reflex vasomotor nerve activity. It is known that in teething children rashes recurring periodically – which earlier physicians attributed to the process of teething – are often observed. It would be difficult to provide indisputable evidence for this claim; however, the recurrence of the same rash on the face during each more difficult eruption of a tooth speaks in its favor. In support of a similar supposition, let the following case concerning an adult serve as an example.

Franciszka W., 34 years old, was admitted to the municipal hospital on November 2, 1880, with a corrosive lichen that had affected both eyelids of her right eye for over three months. At first, suspecting a syphilitic basis due to the pronounced mucopurulent discharge from the uterus, specific treatment was undertaken, both internal and external, but entirely without success. I then cauterized the affected areas with a thermocautery and treated the eyelids with a carbolic acid solution, initially with very favorable results: by December 9, the upper eyelid had completely healed, and the lower one had healed except for a small ulcer near the inner corner of the eye.

However, after a few days, the scar began to break down, nodules formed on the edges of the ulcerated surface, which quickly disintegrated into deep ulcers, and soon the entire lower eyelid presented a semicircular ulcer more than one centimetre wide, resistant to all further treatment efforts. In this condition, I presented the patient at the end of December to my colleague Dr. Wicherkiewicz, who advised the use of a curette.

Before applying it, however, I noticed a strong redness of the gums above the upper right molars, some of which were decayed, and began administering frequent rinses of the oral cavity with a saline-iodine mixture. Already on the second day, the edges of the ulcer paled, and from then on, under the usual carbolic dressings, the healing was so evident that within a week the entire surface was covered with a scar, except for a small spot the size of a peppercorn at the inner corner of the eye.

On March 6, 1881, a new nodule appeared on the lower edge of the ulcer, and only then did the patient mention that the last upper right molar was causing her considerable pain. After the extraction of that tooth, the ulcer already looked better the very next day, and by the fourth day, with continued rinsing of the mouth with the iodine mixture, the remainder of the ulcer had completely healed.

Although corrosive lichen is a disease *sui generis*, which cannot be directly attributed to irritation of the gums or teeth, it is nonetheless only too evident that the persistence of this disease was likely due to renewed hyperemic intensifications, triggered reflexively through the irritated branches of the trigeminal nerve of the upper jaw – while after removing the source of irritation, the ulcer quickly healed.

It remains for me to demonstrate the causal connection between gingivitis and irritation of the nervous centers. As for convulsions, which I have already mentioned above and which frequently afflict teething children, I recall from my own practice numerous cases in which children who were previously healthy – and who later developed quite normally – shortly before the eruption of each new tooth, fell into general eclamptic seizures without any apparent cause. These seizures usually subsided after administration of chloral hydrate per rectum and subsequent purgation.

Since experience taught me to also consider gum irritation in these nervous disturbances – and indeed, in every case I found the gums at the site of the anticipated tooth swollen and reddened – I intentionally refrained from using narcotic medications. Instead, after the convulsions had passed, I recommended that the child be given every half an hour rinses of the mouth with a saline-iodine mixture, followed later by castor oil, and from then on, the convulsions did not recur.

It is known that such convulsions usually occur in the second year of life, particularly when the eye teeth begin to erupt, which are greatly feared by mothers for this very reason. That cerebral congestion, caused by gingival inflammation via reflex pathways, ultimately leads in cachectic children to tuberculous infection of the soft meninges is in no way an objection to the reality of the stimulus we assert. It is, rather, a matter worth reflecting on: why children clearly marked by hereditary predisposition to tuberculosis mostly succumb only during teething to tuberculous meningitis, even though they had developed quite normally until that period.

Gingival inflammation, through reflex action on the vasomotor nerves of the meninges, initially causes ischemia and subsequently venous hyperemia in their vessels, thereby rendering them, as a circumstantial factor, susceptible to tuberculous seeding – something previously lacking an appropriate substrate.

It is also worth recalling at this point a fact not invented, but often recounted by mothers of children who teethed with difficulty and died amidst symptoms of severe cerebral involvement: that the teeth only became visible after the death of the poor child, having only then emerged as the swollen gums, inflamed during life, subsided.

From these considerations follows a therapeutic guideline: how harmful one must regard, in such cases, the still occasionally practiced cutting of the

gums—a procedure once common. Likewise, the favored use of calomel in paediatric practice, though in this age less likely to cause stomatitis, should be avoided and replaced with castor oil.

Among adults, only once (in the practice of my colleague Wicherkiewicz) did I have the opportunity to observe a similar case of inflammation of the meninges in connection with the jaw, which developed slowly not long after a very difficult extraction of a molar tooth – an event that affected the antrum of Highmore – and although the condition ultimately ended in recovery, it was followed by two repeated cerebral hemorrhages caused by arterial degeneration, which eventually led to death.

On the other hand, I am acquainted with not infrequent cases of severe neuroses which, though remote echoes from the true source of the disorder, could ultimately be traced to the teeth and gums, for they ceased only after the jaws were finally healed. I have in mind hysteria, epilepsy, and mental illnesses. I could cite a large number of cases to support the causal relationship between inflammation of the gums and teeth and that still enigmatic condition, hysteria, which is so mistakenly attributed solely to disorders of the reproductive system. I will limit myself to a brief outline of several cases that have been under my observation for many years.

1) A.D., aged 30, a maidservant, who had suffered for many years from persistent headaches, insomnia, spasms accompanied by crying, finally fell into such general exhaustion that for two years she became completely unable to work and had to seek help in a convent of the Sisters of Charity. The tearing pains in both temples, depriving her of rest day and night; complete loss of appetite; spasmodic pains after each meal with occasional vomiting; persistent constipation; heavy menstruation; heightened sexual drive; general nervous sensitivity that led to crying, spasms, and trembling of limbs; joint pains; and general motor weakness – all comprised a disease picture in which the condition of nearly all her molar teeth being decayed and her gums extremely inflamed and hyperemic left no room to identify any other objective cause. Gradual removal of the worst teeth and continuous disinfection of the oral cavity using an iodine solution mixed with myrrh and valerian – which was also prescribed for partial ingestion – alongside mild laxatives, restored appetite within a week and improved digestion. After relief of the headaches, which were caused by irritation of the trigeminal nerve in the jaws, natural sleep returned, and gradually the other symptoms of nervous disorder abated. Under the invigorating influence of cold baths and showers, her strength improved so much that within a few months she was able to return to her work. In the following years, the previous ailments would occasionally reappear, particularly shortly before menstruation, such as loss of appetite and headaches, but each time I could confirm a fresh flare-up of gum inflammation. This occurred because the woman, unable to bring herself to have all the decayed teeth extracted, often neglected proper oral hygiene.

2) M. Pr., a schoolteacher, about 30 years old, presented herself to me two years ago in a state of general nervous collapse, which had forced her to abandon her profession. Complete loss of appetite, spasmodic stomach pains after eating, persistent constipation, headaches, intercostal and spinal pains, palpitations, insomnia, crying fits with spasms, irregular menstruation accompanied by leukorrhea, and deep despair about regaining health were her complaints. Her appearance – emaciated like a skeleton, frail, unsteady, and very prematurely aged – reflected them. A characteristic foul odor from her mouth, noticeable even from a distance, led me to discover, under the deceptive appearance of artificial teeth, the most revolting putrid focus spread across the entire palate and front part of the upper gums, which bulged like ramparts over the rotting stumps of teeth left behind by a careless dentist. Further observation of the patient revealed chronic colitis and heightened sexual excitability.

After all the stumps were extracted and the mouth healed, followed by castor oil purgation, all the disease symptoms that had persisted for over a dozen years subsided within six weeks. River baths used during the summer so strengthened her health that she truly appeared as if reborn.

I could enumerate many such examples of utter physical and moral collapse, especially in unmarried women engaged in intellectual work, in whom – even though this statement may seem trivial to some – the most prominent manifestations of hysteria, across a long chain of disorders affecting various organs, can be traced to decaying gums and teeth as the first link. The initial scene begins here with loss of appetite and indigestion, followed by persistent spasmodic constipation and, as a result, chronic colitis. Impaired general nutrition, along with frequent toothaches and headaches, lead to insomnia that torments a brain already wearied by daily work. Congestion in the abdominal organs creates disorders in the reproductive system, stimulating their excitability and driving these poor victims of unbridled nerves – despite their most religious principles – toward masturbation, which ultimately ruins their already shattered health. Such is the genesis of many cases of hysteria and hysteroepilepsy.

I dare further believe that inflammation of the teeth and gums, on the background of hereditary or acquired frailty, is capable even of producing mental disorders.

This belief should not appear overly frivolous now that psychiatry admits not only emotions directly affecting the central nervous system, but also irritation of peripheral organs as stimuli for mental disturbances. Why should persistent irritation of the terminal branches of the trigeminal nerve in the teeth and gums not also serve as occasional stimuli for mental derangement, especially since this nerve's periphery lies so close to the brain, and even mild stimulation of this periphery can cause visible reflexive congestion in certain sensory organs, such as the eye? Why should impressions constantly transmitted from this source to the sensory nerves not, in a pathologically predisposed brain, become focal points radiating into psychological perception, eventually producing illusions and hallucinations—repeated episodes of which could only derail the balance of the mind?

The following cases incline me greatly toward such a supposition, as in two of them, relapses of mental illness occurred each time following pronounced gum inflammation; once that condition was resolved, the signs of mental exaltation ceased, and the principal hallucinations, though still faintly lingering for a while, eventually dissipated with nutritional improvement.

1) Mrs. K., a widow of a landed gentleman, was transferred at the beginning of February 1880 from the Garczyński Institution to the Sisters of Mercy asylum with symptoms of extreme madness, accompanied by paralysis of the lower limbs.

The patient, aged 52, hereditarily predisposed, in addition to the loss of her estate, was suddenly struck by the death of her daughter. Having fallen for a time into a state of brooding, she erupted into the most violent exaltation, remaining in a state of uninterrupted agitation, shouting, and delirium day and night, from which it was evident that she was constantly tormented by visual hallucinations.

She refused to take nourishment, which had to be administered by force, and only with the use of chloral hydrate could she be subdued for a few hours in her ceaseless restlessness.

She remained in this condition for two months, until at last, prompted by the repulsive stench emanating from the patient's mouth – where, in the lower jaw, five final teeth still protruded, already loosened and surrounded by putrid gums – I proceeded to extract them and began administering, every half an hour, ½ spoonful of a saline-iodine mixture. After three days, the tongue, hitherto covered with a thick layer of grey coating, began to cleanse and the patient – who had previously considered all food offered to her to be poisoned – began to eat on her own with evident appetite. Since the hallucinations, although still occasionally troubling her, ceased to frighten or provoke her into violent actions, she now behaved calmly. After two weeks, she began to get out of bed, and after four weeks – during which, due to pronounced anemia, strengthening remedies were administered – she left the institution completely cured.

2) A. R., the wife of a cab driver, aged 39, hereditarily predisposed, fell into madness three weeks after her seventh childbirth. After three months of periodic exacerbations, she was admitted on January 21, 1881, to the mental ward of the city hospital. Constantly agitated, physically restless and rambling, she showed signs of persecutory delusions, suspected poison in food, and clearly suffered from auditory and gustatory hallucinations. Aside from a mild uterine catarrh, examination revealed extensive ulceration of the gums around several decayed teeth, emitting a foul odor from the mouth.

After administration of an iodine-valerian tincture, the unpleasant mouth odor subsided, the patient began to calm down, and a week later, as if awakened from a deep sleep, she became aware of her previous insanity. On February 15, shortly before her menstruation, she experienced a mild recurrence of hallucinations and agitation, which, however, passed within a few days, and on April 16, in good nutritional condition and complete health, she was discharged.[3]

3) A. K., a turner, aged 60, previously treated seven years earlier in the same mental ward for acute confusion lasting a year, was readmitted at the end of February 1881 with the same mental affliction, poorly nourished and anemic. He spoke incessantly of the strangest things, sang, recited like an actor, and remained constantly in an elevated but not violent mood.

In his mouth, only two lower central front teeth remained, surrounded by ulcerated and foul-smelling gums.

After the gums healed through the use of an iodine and myrrh tincture, the patient – previously extremely averse to food – developed a strong appetite and calmed down day by day. In early May, he was discharged completely cured after only two months of treatment, having recovered from the same mental illness that had previously required a six-month stay.

3 After three years of uninterrupted mental health, the same person was again admitted to the asylum in a similar state, albeit of lesser intensity. This time as well, there was ulceration of the gums combined with decay of the alveolus of a diseased canine tooth. After the extraction of this tooth and the healing of the gums, the exaltation quickly subsided, and only occasional auditory hallucinations – acknowledged by the patient herself – reappeared during the approach of menstruation, though without any accompanying disturbance. These too ceased after significant improvement of her greatly deteriorated nutrition.

4) L. A., a traveling seller of religious images, aged 30, bearing the hereditary mark of mental illness, poorly nourished due to constant travel, fell into brooding with religious delusions and was admitted at the beginning of March 1881 to the city hospital with evident anemia. He exhibited general dejection, from which he was only occasionally roused by religious visions that compelled him to rise from bed and grasp at phantoms in the air, later describing them as saints.

He refused to eat, barely drank, had fairly healthy teeth but severely reddened gums, and emitted a horrible stench from his mouth.

As the gums healed, appetite increased already on the fifth day of administering iodine with valerian, visual hallucinations ceased, iron preparations completed the treatment, and the patient left the institution at the end of April completely cured.[4]

5) M. S., aged 39, wife of a laborer, childless, anemic, with irregular menstruation, fell into madness after being terrified by a fire that broke out in her home. Admitted on February 28, 1881, to the city hospital, she raved, sang, and screamed incessantly, saw people and various insects persecuting her, rejected food claiming it was poisoned, soiled herself, resisted any approach, and exhibited a persistently elevated mood, at times verging on delusions of grandeur. Only on the third day could she be examined, revealing diffuse ulceration of the gums around severely decayed front teeth.

With the use of an iodine-valerian mixture, after three days the patient began to calm down and eat; by March 7, she regained some awareness, asked how she had come to be there, slept better though still incontinent. On March 9, she was neat in appearance, asked for meat, and was completely calm. On March 16, at the insistence of relatives, she was discharged in a thoroughly favorable condition, with a warning to maintain oral hygiene. On April 16, she returned to the institution in the same state of madness as before, also presenting with renewed ulceration of the gums.

Following the same treatment, within a week she regained her appetite, calmed down, and after two weeks returned to lucidity as if awakened from sleep. Three days later, for unknown reasons, she again experienced mental irritation and insomnia, though she continued eating heartily.[5]

6) Mrs. A., aged 53, the wife of a rural landowner, amidst profuse rectal bleeding during the course of colitis and the resulting severe anemia, fell into madness with sensory hallucinations and persecutory delusions. After an unavailing stay in two specialized institutions, she was admitted in 1883 to the Sisters of Charity asylum in anticipation of imminent death. In addition to bloody-purulent discharges from the large intestine, she exhibited very extensive ulcerations of the gums. With treatment of these physical ailments using castor oil and a saline-iodine mixture, and following the revival of her previously poor appetite, both the physical and mental state of the patient – previously completely unapproachable even to her family – improved so significantly within two months that, aside from the delusion that enemies were burning her at night on crosses, she became entirely calm and reconciled with her husband. However, after being released from the institution too hastily, she again experienced such marked agitation within a few months that it became necessary to admit her once more to a specialized asylum.

Thus we have a series of cases of mental disturbance in which, after the removal of gingival inflammation and ulceration and the extraction of decaying teeth, aversion to and refusal of food – at first the most prominent symptoms – subsided quickly, and with them the mental state, particularly violent excitations, diminished increasingly noticeably.

Although, under strict critical scrutiny, it cannot be maintained that in each of these cases irritation of the oral cavity was exclusively and causally related to the mental disturbance – since it is well known that in mental illnesses *foetor ex ore*, or oral cavity infection, may be a very common, secondary phenomenon due to patients refusing regular nourishment and constant loud struggles, as a result of which the mouth dries out. Nevertheless, it is difficult to resist the temptation to suppose that among the peripheral foci capable of triggering outbreaks of insanity in a nervous system predisposed to mental disorders – foci among which the sexual organs are foremost – the oral cavity may also hold a significant place, particularly the gums, which are rich in connective tissue and therefore prone to inflammation. It may be that in certain cases of mental illness – such as the second case cited here – the stimulating current from several peripheral elements of the body is transferred together or successively to the strained brain. After one element subsides, another continues to send an ever-weakening current, until, with the strengthening of general nervous resistance, equilibrium of mental function returns.

4 After two years, a relapse occurred, accompanied by kidney inflammation, which led to the patient's death within two months.

5 This patient, remaining thereafter for two years in the asylum, where the gradually subsiding excitement seemed to transition into a state of mental debility, eventually returned to a normal mental condition, as good nutrition was consistently maintained. Released from the asylum, she has, up to the present, enjoyed good health.

In any case, I consider it my duty to draw attention to an aspect of the aetiology of mental illnesses that has hitherto been completely neglected – an aspect which, once legitimized, could considerably broaden the therapeutic horizon and, more importantly, our preventive measures.

Finally, I must also mention that gum infections may even become the origin of general cachexia. A well-known example is scurvy, which begins with inflammation and ulceration of the gums and leads to general blood infection with haemorrhages indicating that the toxin introduced into the gums spreads throughout the body along the blood vessels.

A case similar to scurvy – which is nearly unheard of in our region – I happened to observe in 1874 in an unmarried woman around 50 years old, whom I was called to by colleague Dr. Freudenreich. This woman, whose nutritional conditions were not conducive to developing scurvy, fell into extreme anemia, as a result of which her lower limbs became heavily swollen and covered with numerous petechiae. A significant decline in strength had kept the patient bedridden for two months, and all available therapeutic measures had proven ineffective. In the patient's oral cavity, I was struck by the massive deposits of tartar covering the only three remaining teeth – located in the middle of the lower jaw – with a ridge two centimetres thick. The upper jaw, though toothless, was entirely healthy, while the gums of the lower jaw were swollen, bluish, and with slightly suppurating edges.

Seeing in the condition of the gums the entire cause of the cachexia, I advised, for the purpose of healing the gums, the removal of both the enormous tartar deposits and the teeth themselves. Following this, and with the use of iron supplements, the patient regained her health within two weeks.

Over the years, I have observed many cases of premature senile decay accompanied by extensive gum and tooth putrefaction, and I cannot resist the conviction that the starting point of premature aging lies in the continuous infections of the oral cavity, which spread throughout the organism the seeds of chronic inflammation and arteriosclerotic degeneration of the arteries and endocardium, causing the gradual withering of the most important organs and leading to premature death. Our old proverb about hearty old men having "strong teeth" is therefore full of truth, for translated into our terms, it means that people with healthy teeth and gums are not so easily susceptible to the invisible enemies of health that surround us.

I have also noticed that many people, since they began wearing artificial teeth, and neglecting proper oral hygiene, suddenly began to suffer from gallstones and urinary stones. These are first formed by fungi cultivated beneath artificial teeth, which then enter the bloodstream.

Even if our views on the harmful importance of inflammatory conditions in the oral cavity – particularly of the gums and teeth – may appear exaggerated or overly one-sided, in the face of growing recognition of the pathogenic role of microbial life, no one can deny that continuous infection of the oral cavity due to rotting teeth and gums cannot be harmless to our health. Nor can one deny that the now widespread medical emphasis on disinfection pays far too little attention to the organ that most offends our senses – the mouth.

In our view, oral hygiene should become one of the most important hygienic concerns – not, as is too often the case, in the hands of vendors of cosmetic products or dentists, but in the hands of physicians, both in times of health and especially during any illness. In such cases, the most diligent disinfection – if only to preserve the patient's waning appetite – should constitute one of the most cardinal therapeutic indications. The necessity of such a directive becomes all the more urgent today, when the majority of the population wears artificial teeth, which, due to improper maintenance, contribute even more to the pollution of the gateway to our body.