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Winter 2016

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“The New Jersey Dental Association serves and supports its members and fosters the advancement of quality, ethical oral healthcare for the public.”

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From Your President

President's Message

Gregory LaMorte, DDS

“When you are president-elect of our New Jersey Dental Association, you visit each component dental society with the current president of NJDA. It is an interesting experience and rather enjoyable—different venues, different menus, different personalities and meeting new members. Now it is my turn as president to lead our “delegation” to visit your components.

We visited the Essex County Dental Society. I have been a member of ECDS for over three decades and miss very few meetings. It is my home component. ECDS hosts the fourth year dental students from Rutgers at its first meeting of the academic year. The meeting can be a little noisy, but the officers manage.

I am not writing to critique the food or the venues. (However, Hudson County Dental Society has the best view.) I am writing to stress how important the messages are. How important? Very important.

✓ The first message: Thank you for your membership in NJDA.
✓ The second message: We need you to give NJDA your email address, so we can save postage on “snail mail” and get you each the Monday morning emails. They contain the most important recent issues affecting dentistry. We want to keep you informed. (Contact AnnMarie in Membership: avarga@njda.org.)
✓ The next message: We encourage you to participate with your component and with NJDA. New ideas often come with new blood. We continue to move forward.
✓ The last, at least for now, is: We know we cannot please everyone all the time, but we do try our best.

They told me I could write seven hundred fifty words, but I think shorter is better. It sounds trite, but it is my honor to serve as your president.

As always, your comments are welcome. ☺

“I want to be thoroughly used up when I die… Life is no brief candle to me; it is a sort of splendid torch which I’ve got a hold of for the moment and I want to make it burn as brightly as possible before handing it on to future generations.”

— George Bernard Shaw

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On March 12, we mark the death (71 years ago) of Anne Frank, a young Jewish girl who grew up in Amsterdam during the Nazi occupation of the Netherlands in the Holocaust of World War II.

In July 1942, Anne's family was forced into hiding in the upper story of an Amsterdam warehouse, where they remained for 25 months. The rooms became more suffocating than any prison one could imagine. The Franks, who shared the space with another family and an elderly dentist, were unable to feel the sun's warmth, unable to breathe fresh air. While the warehouse was in operation during the day, there could be no noise of any kind — no speaking, no unnecessary movements, no running of water.

Then, in 1944, the hideout was discovered by the police. Of the eight who had been crowded into the sealed-off attic rooms, only Mr. Frank survived the ensuing horrors of the concentration camps. In March 1945, two months before the liberation of the Netherlands and three months before her 16th birthday, Anne Frank perished from typhus in the camp at Bergen-Belsen.

Anne may have been devoured by the concentration camps, but her voice was not stilled. From the pages of a small red-checkered, cloth-covered diary book, she speaks to us across the years. The diary was the favorite gift that Anne received for her 13th birthday. She named it Kitty and determined to express, to her new confidante, her innermost thoughts, concerns and desires. Between the covers of Kitty, the young girl recorded her moving commentary on war and its impact on human beings:

I see the eight of us with our “Secret Annexe” as if we were a little piece of blue heaven, surrounded by heavy black rain clouds. The round, clearly defined spot where we stand is still safe, but the clouds gather more closely about us and the circle which separates us from the approaching danger closes more and more tightly. Now we are so surrounded by danger and darkness that we bump against each other, as we search desperately for a means of escape.

We all look down below, where people are fighting each other, we look above, where it is quiet and beautiful, and meanwhile we are cut off by the great dark mass, which will not let us go upwards, but which stands before us as an impenetrable wall; it tries to crush us, but cannot do so yet. I can only cry and implore: “Oh, if only the black circle could recede and open the way for us!”

Finally the Franks were betrayed, and on August 4, 1944, the fury of the Gestapo burst upon them. The invaders confiscated the silverware and menorah, but they threw the family's papers to the floor, including Anne's diary, which was unexpectedly and miraculously recovered a year later by Otto Frank, Anne's father.

The Nazis had failed in their mission. Anne Frank: the Diary of a Young Girl was first published in 1947, has been translated into 65 languages and has sold more than 35 million copies. The Frank house draws an average of 4,000 visitors each day and more than 1 million each year.

"It's a wonder I haven't abandoned all my ideals; they seem so absurd and impractical. Yet I cling to them because I still believe, in spite of everything, that people are truly good at heart," Anne wrote in her diary. No one has described its impact more eloquently than Anne's biographer, Ernst Schnabel: “Her voice was preserved out of the millions that were silenced, this voice no louder than a child's whisper. It has outlasted the shouts of the murderers and soared above the voices of time.”
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Of course, your calls are always welcome and help us to determine issues that currently are affecting the profession. When you place a call to NJDA at 732-821-9400, about an EOB form or credentialing question, we’re able to research the matter and share the results with all our members. The call you place today may represent a problem that will, or soon will, affect them as well. We’ll get to the bottom of the problem for you and share what we’ve learned in an email alert or the quarterly Advocate. Additionally, when numerous calls come in about the same problem, NJDA will reach out to the agency, insurance carrier or the state board to resolve the situation.

When you contact us, you can be assured that we’ll track down an answer for you and when relevant, share it with your colleagues. Your input helps us identify trends that allow us to develop appropriate continuing education, legislative inquiries and programs. NJDA is your resource, but it is also your voice.

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What If?

What If My Patient Needs Emergency Treatment After Hours?

Joan Monaco, DMD,
Director of Dental Benefits, NJDA

On a Friday afternoon you see a patient, perform an extraction and close the office for the weekend. Later that evening or the next day, the patient still has a problem with excessive pain or bleeding. What should they do? What should you have done?

If your office is prepared, the patient will know whom to contact or whether an emergency room visit is warranted. What you should do is make prudent decisions now to save you and your patient anxiety and concern down the road.

Legally, currently in the state of New Jersey, there is no law stipulating that you must make yourself available to patients of record, nor do you need to have an on-call service or covering doctor. You may be obligated to provide an answering service, on-call doctor or other mechanism to remain in contact with a patient of record, if your insurance carrier requires you to do so. Certain commercial and CMS carriers in their contract language may require any or all of the following:

• Posting or notice of office hours
• Emergency hours and coverage
• A contingency plan to provide to your patient
• Stipulation that a patient of record be seen within 24 hours of notification by the patient of an emergency

For best practices after any invasive procedure, you may need to do the following, to remain in compliance with your insurance carriers and to provide optimal care to your patient:

• Provide verbal and written post-procedure instruction
• Document in the patient’s chart that instruction was given to the patient
• Provide the patient with 24/7 emergency contact information, which may include an after-hours call number, clinic or covering doctor’s phone number

In most instances, as part of the credentialing process, an insurance carrier will require that an emergency plan be in place for all patients of record.
Dr. Wayne Aldredge of Holmdel has been named president of the American Academy of Periodontology. Dr. Aldredge, who is the principal practitioner at Holmdel Periodontics and Implant Dentistry, was installed at the Academy’s 101st annual meeting in November, held in Orlando. Dr. Aldredge is a member of Monmouth-Ocean Dental Society.

Dr. Steven Rasner of Bridgeton was recently inducted into The American Society for Dental Aesthetics. A member of Southern Dental Society, Dr. Rasner also is a master in the Academy of General Dentistry. Dr. Rasner will be speaking at the Garden State Dental Conference & Expo in May, when his topic will be “The Thriving Fee-for-Service Practice.” Visit the NJDA website at www.njda.org for more information on his program and details on discounts for staff members.

Dr. Eric Sacks of Livingston is sponsoring a local branch of the Seattle Study Club. The group will meet at Lithos Greek Restaurant in Livingston. Dr. Sacks is an orthodontist and member of the Essex County Dental Society. For information on the Seattle Study Club, visit www.seattlestudyclub.com.

Rauchberg Dental Group welcomes its newest associate, Dr. Deepa Rupani, to its multi-specialty practice. Dr. Rupani has extensive experience in all areas of dentistry and is also an Invisalign® provider. The practice is located in Parsippany. Dr. Alan Rauchberg is a member of Tri-County Dental Society and Dr. Rupani is a member of Passaic County Dental Society.

Dr. Yasmi Crystal of Bound Brook was recently named one of the top 25 women in dentistry by Dental Products Reports. Dr. Crystal is a pediatric dentist and member of the Central Dental Society.

Dr. John Beckwith of Hillsborough recently was named a diplomate of the American Board of Oral Implantology. Diplomates must pass the ABOI Part I and II examinations. Dr. Beckwith was one of 18 dentists so designated in 2015. He is a general dentist, and member of the Central Dental Society.

Eric Nelson of British Columbia, recently contacted the Journal of NJDA in the hope of connecting with the daughter of a friend he made while traveling in Korea. Mr. Nelson believes the woman is a dentist practicing in New Jersey. His friend, a Mr. Chung, was a broom maker in Seoul. Mr. Nelson lost touch with him about ten years ago. If you are Mr. Chung’s daughter, or know who she might be, please contact Lorraine Sedor, managing editor, at lsedor@njda.org.

NJDA welcomes Samantha Bove to the staff, as marketing specialist administrator. Sam is a recent graduate of SUNY New Paltz and will be working on meetings and events, and social media.

To include items in Members in the News, please contact Dr. Harvey S. Nisselson, editor, at hn3@cumc.columbia.edu or Lorraine Sedor, managing editor, at lsedor@njda.org or 732-821-9400.
For Your Dental Practice
Reverse the Medicare Part D Prescriber Requirement. The ADA is continuing to pursue a two-pronged approach to the issue of enrollment in or opting out of Medicare as required by the Medicare Part D regulation. While the ADA fights for legislation to repeal this provision, it is providing the best and most useful information for members to comply.

Two-Year Delay of Cadillac Tax. There is a two-year delay of enforcement of the “Cadillac Tax,” which would impose a 40 percent excise tax on high-cost employer sponsored health plans. The delay provides additional time to address the unintended consequences associated with the tax, such as the adverse effects on FSAs that are used by many patients to pay for dental care.

Reform ERISA. The ADA advanced legislation that would help consumers receive the full value of their dental coverage. H.R.1677 requires all self-insured health plans that offer dental benefits to provide uniform coordination and assignment of benefits.

For the Dental Profession
Non-Covered Services Legislation. ADA, along with the American Optometric Association, helped craft the “Dental and Optometric Care Access Act” (DOC Access Act), H.R.3523, which prohibits federally regulated dental or vision benefit plans from dictating what a doctor may charge a plan enrollee for items or services not covered by the plan.

Increased Support for the “Action for Dental Health” Bill. H.R. 539 will allow state organizations to qualify for oral health grants to support activities that improve oral health education and dental disease prevention. The ADA has secured 80 bipartisan cosponsors.

Improving Medicaid. The ADA, along with the American Academy of Pediatric Dentistry, submitted comments to the Centers for Medicare and Medicaid Services on a proposed rule that aims to align Medicaid managed care plans more closely with the plans offered in the commercial market. The proposed rule would require Medicaid managed care plans to adhere to a minimum loss ratio, provide actuarially sound rates and ensure network adequacy among other requirements. Similar requirements are already in place for any plans seeking certification as a qualified health plan to be offered in the marketplaces.

For Your Patients & the Public
Fluoridation. The ADA obtained a statement from the Surgeon General endorsing community water fluoridation, and also advanced a House resolution commemorating the 70th anniversary of community water fluoridation. The ADA also secured a $2 million increase in funding for the CDC’s Division of Oral Health for FY 2016.

Preventing Tobacco Use. The ADA successfully petitioned the FDA to deny a tobacco company’s modified risk tobacco product application to label ten snus tobacco products as less harmful to human health than smoking.

Expanding Flexible Spending Accounts. H.R. 1185 would increase the annual maximum contribution to flexible spending accounts to $5,000 and permit rollover of all unused monies in the account.

For more information, visit ADA.org/engage or ADA.org/advocacy. Or contact NJDA Director of Governmental Affairs, Jim Schulz, at jschulz@njda.org.
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NJDADC
Oral Pathology Quiz #90

Presented by Rutgers School of Dental Medicine Biopsy Service

The RSDM oral pathology faculty are showing the clinical presentation of some relatively common lesions for readers to self-evaluate their skills in clinical differential diagnosis. You are expected to choose the most likely clinical diagnosis on the basis of history and clinical or radiographic appearance with the appreciation that definitive diagnosis requires microscopic examination of the specimen.

Case Number 1

Figures 1 and 2: Courtesy Dr. Daniel Barabas, Ridgewood

A 79-year-old white male presented with asymptomatic mixed red-white areas posterior to his full denture on both sides of the midline at the hard palate-soft palate border. The area on the left side was slightly larger, measuring about one cm in maximum dimension. The irregularly shaped red base was partially covered by white curd-like plaque material. Some of the white component sloughed away when gentle pressure was applied. No other areas of the mouth were involved. The photograph was taken after a course of antifungal rinses had not reduced the size of the affected mucosa. The patient was unaware of any potential habits that could have been predisposing factors and his denture was clean and well-fitting. Which of the following is the most likely diagnosis?

A. White sponge nevus
B. Oral hairy leukoplakia
C. Herpangina
D. Candidiasis

Case Number 2

Figure 3: Courtesy Dr. Daniel Barabas, Ridgewood

A 74-year-old white male presented with an irregularly shaped, indurated enlargement fixed to the mucosa in the floor of his mouth. The asymptomatic lesion measured approximately 2 cm in maximum dimension. Small adherent white plaques partially coated the surface of the lesion that was otherwise covered by mucosa of normal color. Regional lymph nodes were within normal limits. Which of the following is the most likely diagnosis?

A. White sponge nevus
B. Oral hairy leukoplakia
C. Herpangina
D. Candidiasis

Case Number 3

Figure 4: Courtesy Dr. David Dugan, Oneonta, NY

A 74-year-old white male presented with an irregularly shaped, indurated enlargement fixed to the mucosa in the floor of his mouth. The asymptomatic lesion measured approximately 2 cm in maximum dimension. Small adherent white plaques partially coated the surface of the lesion that was otherwise covered by mucosa of normal color. Regional lymph nodes were within normal limits. Which of the following is the most likely diagnosis?

A. Benign hyperkeratosis
B. Squamous cell carcinoma
C. Hodgkin lymphoma
D. Canalicular adenoma
A 49-year-old healthy white female presented with an asymptomatic, red, white and pink, sessile enlargement on the interdental papilla between her right maxillary premolars. It measured approximately 8 mm in maximum dimension, was moderately firm in consistency, and did not blanch on pressure. There was no significant pocket formation and teeth were vital. The free gingivae throughout her mouth exhibited mild enlargement. Radiographs were within normal limits. Which of the following is the most likely diagnosis?

A. Parulis  
B. “Strawberry” hemangioma  
C. Epulis granulomatous  
D. Pyogenic granuloma

Answers on page 16
1. **Sleep bruxism is considered a common sleep disorder**

Sleep bruxism (SB) is a common sleep disorder defined as ‘a repetitive jaw-muscle activity characterized by clenching or grinding of the teeth and/or by bracing or thrusting of the mandible during sleep.’\(^1\) SB is commonly associated with tooth grinding sounds, the pathognomonic sign, as reported by bed partners, parents, or siblings.\(^2\) SB can lead to tooth wear and fracture, muscle fatigue, orofacial pain, and headache.\(^3, 4\)

2. **Sleep bruxism etiology appears to be centrally regulated and multifactorial**

As seen in multiple clinical prospective studies, the pathophysiology of SB is still unknown but appears to be centrally regulated and multifactorial, such as sleep arousal, autonomic sympathetic cardiac activation, genetic predisposition, neurochemicals, psychosocial components, and other sleep disorders.\(^2, 3\)

3. **Sleep bruxism has a higher prevalence throughout childhood and no gender differences**

Patient awareness of SB is reported in eight percent of the general adult population, with a higher prevalence throughout childhood (with 14% to 38% in children less than eleven years old) and decreases with aging (3% after 60 years of age).\(^2\) No gender differences in SB have been reported.

4. **Sleep bruxism may be associated with facial pain but not with temporomandibular dysfunction**

A prospective clinical study suggested that SB patients with low frequency masticatory muscle activity are at greater risk of reporting transient morning orofacial pain than those with moderate to high frequency activity.\(^5\) Another study using sleep recordings showed the absence of a causal or exacerbating relationship between frequency of SB (repetitive jaw-muscle activity) and myofascial temporomandibular dysfunction.\(^6\)

5. **Sleep bruxism can be secondary to some exogenous factors or medical diseases**

Iatrogenic or secondary SB is associated with alcohol, nicotine, caffeine, medication (i.e., amphetamines, antidepressives, antiparkinsonian, etc.) and drug use (i.e., cocaine and ecstasy).\(^2\) It is also associated with some neurological, sleep-related, and movement disorders.\(^2\)

**References**


**About the Authors**

Kelvin I. Afrashtehfar DDS, MSc, is a resident in the Department of Prosthodontics and Restorative Dentistry, McGill University, Montreal.

Nelly Huynh, PhD, is a member of the Faculty of Dentistry, Oral Health Department, Université de Montréal and the CHU Sainte-Justine Research Center, Montréal, Canada.
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Case Number 1  
**Answer: A. Frictional keratosis**

Frictional keratosis (A) is a common benign condition caused by chronic mechanical trauma. It presents as white plaques that may have a roughened surface. It is reversible. In some cases, there are eroded foci within the white plaques and they may exhibit surface keratin shredding. Causes include: chewing the tongue (“morsicatio linguarum”), chewing the buccal mucosa (“morsicatio buccarum”), toothbrush gingival abrasion, and irritation due to dentures or broken teeth. Clinical judgement is often necessary to distinguish frictional keratosis from leukoplakia (B), which is a precancerous white patch. It is defined as an adherent white plaque that cannot be identified as benign on the basis of clinical criteria. It is, therefore, a diagnosis of exclusion. Among the conditions that must be excluded are lichen planus, white sponge nevus, nicotine stomatitis, leukoedema, and frictional keratosis. In this case, the intermittent reduction in size of the plaques suggested that it was more likely to be frictional keratosis than leukoplakia. Biopsy was appropriately performed to confirm that clinical impression.

Leukoedema (C) is excluded because the grey-white mucosal changes (filmy white to thick and corrugated) affect the buccal mucosa and other areas bilaterally and symmetrically. Dyskeratosis congenita (D) is a rare, inherited life-threatening disease characterized by hyperpigmentation of the skin, nail abnormalities, and bone marrow disorders as well as oral leukoplakia and cancer.

In addition to the conditions mentioned above, the differential diagnosis of oral white plaques includes chemical burns, oral hairy leukoplakia, candidiasis, tobacco pouch keratosis and other rare inherited diseases.

Case Number 2  
**Answer: D. Candidiasis**

Candidiasis (D) is a superficial fungal infection that presents on the oral mucosa as white removable or adherent plaques (as well as lesions with other appearances). A red base covered by removable, white, soft creamy or blue-white material is typical of pseudomembranous candidiasis or “thrush”. The chronic white adherent plaque form of candidiasis may exhibit shredding of surface keratin. Microscopically, this correlates with hyperplastic candidiasis. Candida albicans is a component of the normal oral flora and clinically apparent lesions appear when there are local or systemic predisposing factors such as xerostomia, use of broad spectrum antibiotics or corticosteroids, or debilitating diseases. The patient in this case would be referred for a medical work-up when no local predisposing factors were identified and the lesions did not resolve with topical antifungal therapy.

White sponge nevus (A) is an inherited condition in which white velvety or thick corrugated changes affect the buccal mucosa, other oral sites and other mucous membranes. Oral hairy leukoplakia (B) is excluded because in almost all cases the white adherent lesions occur in immunologically-compromised individuals, primarily on the lateral borders of the tongue. Herpangina (C) is excluded because it is an acute mild viral infection presenting as vesiculo-ulcers on the soft palate and pharynx.

The differential diagnosis of oral white plaques also includes leukoplakia, frictional keratosis, nicotine stomatitis, tobacco pouch keratosis, lichen planus, leukoedema and other rare inherited diseases.

Case Number 3  
**Answer: B. Squamous cell carcinoma**

Squamous cell carcinoma (B) is by far the most common malignancy affecting the oral mucosa. It may present as a white plaque, red patch, ulceration, or an indurated, often ulcerated enlargement. In this case, the indurated swelling was partially coated by white plaques. The floor of the mouth is the second most common intraoral site for oral cancer. The tongue, especially its lateral borders, is the most common site. Prognosis correlates with clinical staging that is based on tumor size, regional lymph node metastases, and distant metastases. This case was an early stage lesion, the five-year survival rate for which ranges from 58% to 72%.

In this patient, the induration of the enlargement, fixation, irregularity in shape, and location in a high risk site made benign hyperkeratosis (A) much less likely than carcinoma. Hodgkin lymphoma (C) is a lymphoproliferative malignancy that is extremely rare in the oral cavity. Canalicular adenoma (D) is excluded because it is a benign neoplasm of minor salivary glands. It is freely movable within the mucosa and most cases occur in the upper lip.

In addition to squamous cell carcinoma, the differential diagnosis of a chronic, irregular, indurated and fixed enlargement in the floor of the mouth includes desmoplastic fibroma, mesenchymal sarcomas, and deep scars.
Case Number 4  

**Answer: D. Pyogenic granuloma**

Pyogenic granuloma (D) is a very common reactive lesion presenting as a well-circumscribed, usually red nodule that may be pedunculated or sessile. The lesion often grows very rapidly. It is usually painless. The surface is typically smooth, but may be lobulated or even warty. It is frequently ulcerated with a fibrinopurulent “pseudomembrane” on the surface. Hemorrhage may be spontaneous due to its vascularity and lack of epithelial covering. The generalized gingival enlargement in this patient represented chronic hyperplastic gingivitis. Pyogenic granulomas can occur anywhere in the mouth. They may become irritation fibromas as they mature.

Parulis (A) is excluded because it is a fluctuant red or yellow abscess in the gingiva derived from acute infection at the apex of a non-vital tooth or from an occluded periodontal pocket. It drains through alveolar cortical plate, follows the path of least resistance, creates a sinus tract, and causes ballooning of overlying mucosa. The swelling tends to develop at the mucogingival junction. “Strawberry” hemangiomas (B) are bright red, bosselated, cutaneous lesions that appear in newborn infants. Epulis granulomatousum (C) is a reactive proliferation of inflamed granulation tissue growing out of a recent extraction socket.

The clinical differential diagnosis of discolored circumscribed gingival nodules in adults also includes other reactive enlargements (peripheral ossifying fibroma and peripheral giant cell lesion), hematoma, adult gingival cyst, benign melanocytic nevus, vascular leiomyoma, melanoma, and Kaposi’s sarcoma.

The Oral Pathology Quiz is presented by faculty of the Rutgers University—Rutgers School of Dental Medicine, Division of Oral Pathology, Drs. Joseph Rinaggio, and Lawrence C. Schneider. Clinicians who have clinical pictures and/or radiographs of cases suitable for future quizzes should call Dr. Schneider at (973) 972-4375. E-mail: Lawcschneider@aol.com.

Biopsy kits may be obtained without charge by calling (973) 972-1646. Faculty are available to answer questions Monday through Friday, from 8:00 AM to 4:00 PM.
When most people have a common “cold” (sniffle/coughing episodes) they call the MD and ask for an antibiotic prescription. However, antibiotics will not kill the “germs” that have attacked them. Why? Because the invader is a virus, not a bacteria. Bacteria are organisms that can survive on their own without using a host’s protein machinery to reproduce. Antibiotics target those processes relating to the cell’s survival and reproduction. Viruses do not have the cellular processes to produce their own proteins for survival and reproduction and must use a host’s cells (often ours) to accomplish these tasks. So antibiotics will not “kill” the virus. Interestingly, the word “antibiotic” means “against life” and viruses can be classified as non-living, thus the antibiotic is not effective. So we might view a virus as “not being alive, but not quite dead.”

Background
Although dentists do not become involved in managing upper respiratory infections that are virally induced, their daily practice can involve patients with medical histories of viral infection (hepatitis B, hepatitis C, human immunodeficiency virus [HIV], herpes simplex oral lesions [“cold sores”], human papilloma virus [HPV]).

Viral Structure and Basics of Viral “Attack” on Human Cells:
As stated above, viruses must use the host cellular processes to reproduce—in essence using these functions to generate new viral proteins. The basic viral structure consists of an outer protein “envelope” or overcoat that contains the proteins allowing for binding to the host cell. (Fig.1)

Within the viral envelope is another structure, a protein coat or capsule (“capsid”) which encloses the viral genetic material necessary for reproduction, RNA or DNA. (Fig. 2) The virus attaches to the organism’s cell wall and the capsid with its genetic material is passed into the cell. The capsid will then release the genetic material so as to initiate the steps in protein reproduction. (Fig.3)

Fig. 1

Fig. 2

Fig. 3

Basic human protein production at the genetic level involves using our DNA which is transcribed to RNA and eventually translated to form new proteins essential for human structure and function. Our DNA resides in our cell’s nucleus and RNA is formed inside the nucleus and transported outside the nucleus for future protein production. Viruses release their RNA or DNA into human cells and these genetic molecules use the host’s cellular processes to make new proteins leading to the formation of new viruses (also called virions) at a rate of millions or more per day. (Fig. 3)

Symptoms of viral infections (our “common cold”) are mostly due to the body’s immune system reacting to the foreign invader. This also holds true for symptoms and organ damage associated with hepatitis B and hepatitis C infections. Our immune system...
may clear a viral attack or it may become chronic. Further system damage from chronic infection may not be due to the immune response but be secondary responses to the ongoing primary infection.

Viruses and our Immune System

If you have a small cut on your hand or a dental infection, the bacterial infection is cleared by your innate immune system (cells, tissues, molecules), that part of our immune defense capability that has existed since ancient times. It is called the "innate" system because we are born with it. However, viruses have developed the ability to evade detection by the innate system and, over eons, our bodies developed the adaptive immune system (T-Cells, B-Cells) which is able to defend against a viral invader. So the last time you had a "cold" the symptoms of sore throat and sniffles were due to your body's innate immune inflammatory response to the virus. Most "colds" last about ten days because it takes that long for the adaptive immune system to awaken and remember the invader and kill it. However, at times, these smart viral invaders have multiple processes that they use to escape our immune system. Briefly, as viruses replicate, they are constantly undergoing genetic mutation and the adaptive immune system cannot keep up with recognizing the new offspring.

Viruses and Cancer:

It is estimated that about 10% of all cancers worldwide are caused by viral infection. In essence, a viral infection may lead to a genetic change which induces cellular proliferation and/or inhibits those processes that control cellular growth. A benign wart is a harmless version of the processes that may lead to cancer.

The chronic inflammation caused by viral invasion is also considered to be another key factor leading to carcinogenesis. The chronic cell turnover in the inflammatory process, the ongoing tumor generation of inflammatory chemicals (cytokines) and the fact that the adaptive immune system may become exhausted in fighting the inflammatory process may all be factors leading to tumor generation.

Today, the current increase in the diagnosis of HPV-related oropharyngeal cancer is a prime example of viral attack. HPV is a small DNA virus that is capable of infecting human keratinocytes of the skin and mucous membranes. Although there are more than 100 subtypes of HPV, HPV 16 accounts for 90% of all HPV-related head and neck cancers. The HPV virus is smart enough to commonly localize in the lingual tonsils; in this area the tissue produces a protein (CD-1 ligand) that inactivates immune system T-Lymphocytes thus preventing an attack on the tumor. The result is that further growth of a tumor is not hindered.

Viruses—At Times Our Friends

- Vaccines—We are all familiar with the medical procedures known as "vaccinations."—A vaccine is a weakened, killed form of a virus or bacteria, or a part of the structure (protein, polysaccharide or one of its surface proteins) or a weakened toxin of the organism. It is most often injected or can be a nasal spray, but it does not cause clinical disease; instead the vaccine
“educates” your adaptive immune system to remember the attacker and provide the essential defense mechanisms to kill the invader if it enters your body again.

- Viral Vectors—Fighting Disease
  - Fighting cancer—cytotoxic medications are attached to viruses—since viruses know how to attach to our cells or deliver their payloads into a cell, there are now therapies whereby a cytotoxic chemical is brought to, or delivered into, a cancer cell using the virus as the medicine carrier. The viral vector can also deliver a new gene or shut down an unwanted one to control disease.² Additionally, an immune system component (e.g. dendritic cell) can be part of a viral vector to aid in fighting cancers.

Summary
This overview is intended as a baseline understanding of viral biology, pathology and developing medical therapeutic regimens using viral vectors. The dental clinician is encouraged to pursue further information as patients present with viral related illness or are undergoing emerging therapies.

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Figure 3—Original by author

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About the Author
Harold V. Cohen, DDS, is a professor at Rutgers School of Dental Medicine.

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