Lyme Disease: Considerations for Dentistry

Gary M. Heir, DMD
Associate Clinical Professor
TMD and Orofacial Pain Center
University of Medicine and Dentistry
New Jersey Dental School
Newark, New Jersey
and Private Practice
Bayonne, New Jersey

Lesley A. Fein, MD, MPH
Currently, Private Practice
West Caldwell, New Jersey
Formerly, Member
New Jersey Task Force on Lyme Disease
Formerly, Member
Congressional National Health Care Reform Task Force

Correspondence to:
Dr Gary M. Heir
718 Boadway
Bayonne, New Jersey 07002

Although Lyme disease has spread rapidly and it is difficult to diagnose, a review of the dental literature does not reveal many references to this illness. Dental practitioners must be aware of the systemic effects of this often multiorgan disorder. Its clinical manifestations may include facial and dental pain, facial nerve palsy, headache, temporomandibular joint pain, and masticatory muscle pain. The effects precipitated when performing dental procedures on a patient with Lyme disease must also be considered. This study discusses the epidemiology and diagnosis of Lyme disease, its prevention, and factors to consider when making a differential diagnosis. Dental care of the patient with Lyme disease and currently available treatments also are considered. Three case reports are presented.

J OROFACIAL PAIN 1996;10:74–86.

key words: Lyme disease, orofacial pain, temporomandibular, facial nerve palsy, spriochetal disease, borreliosis

The rapid spread of Lyme disease in the United States has reached epidemic levels. Symptoms of Lyme disease can mimic hundreds of other disorders and create a diagnostic dilemma for dentists and physicians.[1] Despite this, a review of the dental literature finds a paucity of references to this illness.

Lyme disease was first reported in the medical literature in 1977 as "a previously unrecognized clinical entity, the epidemiology of which suggest transmission by an arthropod vector."[2] Although Lyme disease was only first described in the late 1970s, there is reason to believe that this condition has been a problem for a much longer time.[2] The emergence of Lyme disease in the United States in this century is thought to have occurred because of ecologic conditions favorable for deer. The reported national incidence of Lyme disease in 1993 was 3.3 per 100,000 population.[3] From 1982 through February 1995, a total of 69,626 cases occurring in 49 states were reported to the Centers for Disease Control (CDC) (information provided by the Lyme Disease Foundation, Hartford, CT, 1995).

Lyme disease is caused by a bacterial infection transmitted by the bite of certain very small infected ticks. The bacteria is a coiled spirochete, known as *Borrelia burgdorferi*. It is named for Dr Willy Burgdorfer of the US Public Health Services who first identified the spirochete in the bodies of the deer tick that carry it.[4]

Lyme disease, or Lyme borreliosis, which is caused by three groups of the spirochete *Borrelia burgdorferi*, is transmitted in North America, Europe, and Asia by ticks of the *Ixodes ricinus* complex. The most important Lyme infection–carrying tick in the

Northeast is the deer tick, or *Ixodes dammini*. This tick is very small; in the nymph stage it is comparable in size to a poppy seed (Fig 1). Ticks are active not only in the summer months as is commonly thought, but also at temperatures as low as 35°F.

Prevalence

Demographic profiles of those infected with *Borrelia burgdorferi*, or Lyme disease, indicate that individuals in suburban and rural areas are at high risk.[5] Victims of Lyme disease now number in the thousands. It has become the most prevalent tick-borne disease in the United States, and its geographic distribution appears to be spreading.[6] Although more than 60,000 cases have been reported in this country during the past decade, health officials acknowledge that this number is a gross underestimate.[7,8] The CDC reported that the number of Lyme disease cases rose 17% in 1991 from those reported the previous year. In 1990, there were 7,943 reported cases; in 1991, 9,344 cases were reported.[3] It is believed that these numbers reflect only 75% of those individuals actually infected. One county in New Jersey reported an increase of an amazing 322% in 1993.[9] In 1994, 11,835 cases were reported to the CDC (Fig 2).[6]

Although Lyme disease has been found in 49 states, it is concentrated in relatively few.[6] They are the east coast states of Massachusetts, Connecticut, Rhode Island, New York, New Jersey, Pennsylvania, Maryland, and Delaware. Other areas of the country in which this disease has also been reported are the north central and western states of Wisconsin, Minnesota, Michigan, and California. In these states, the bite of the western black-legged tick, *Ixodes pacificus*, is the source of infection[10] (Table 1).

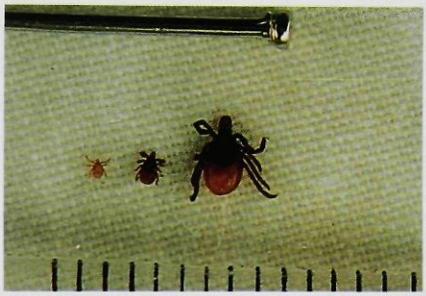


Fig 1 *Ixodes dammini* from nymph to adult stage, compared to the head of pin. The tick may vary in size from approximately 0.5 mm to 2.5 mm.

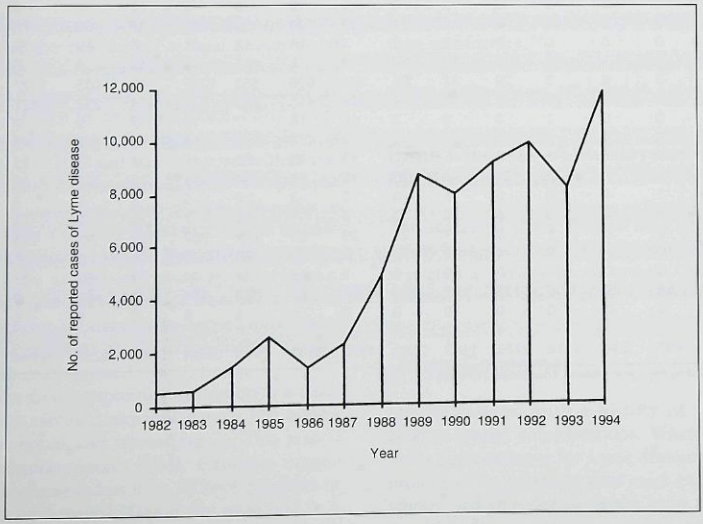


Fig 2 Increased prevalence of Lyme disease.

Table 1 Incidence of Lyme Disease by State*

State	1980	1982	1983	1984	1985	1986	1987	1988	1989	1990	1991	1992	1993	1994	2/95	Total
AL	0	0	0	0	0	1	1	1	25	33	18	10	4	6	0	99
AK	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1
AZ	0	0	0	0	0	0	0	0	0	0	1	0	0	0	0	1
AR	0	0	1	4	0	0	0	16	10	22	29	21	8	8	0	119
CA	0	0	11	24	70	107	182	200	250	345	323	231	134	83	7	1967
CO	0	0	0	0	0	0	0	2	1	0	1	0	0	1	1	6
CT	52	135	73	483	699	0	215	362	774	704	1221	1760	1350	1762	0	9590
DE	0	1	4	1	0	0	6	4	25	54	72	218	143	78	1	607
DC	0	0	0	0	0	0	0	0	0	5	5	3	2	9	0	24
FL	0	0	0	0	2	0	1	0	6	7	23	24	30	27	0	120
GA	1	0	0	1	1	2	4	53	715	161	31	23	44	106	0	1142
HI	0	0	0	0	0	0	0	0	1	2	0	2	1	0	0	6
ID	0	0	0	0	0	0	0	1	42	1	2	2	2	3	0	53
IL	0	0	0	0	2	0	6	5	79	30	25	41	19	11	0	218
IN	0	0	0	1	0	1	3	0	8	15	13	21	32	14	0	108
IA	0	0	0	0	1	1	4	15	27	16	22	33	8	17	0	144
KS	0	0	0	0	0	0	1	0	15	14	22	18	54	14	3	141
KY	0	0	0	0	0	0	3	5	21	18	43	28	16	23	0	157
LA	0	0	0	0	0	0	0	2	2	3	6	7	3	2	0	25
ME	0	0	0	0	1	4	0	1	3	9	9	1	18	27	0	73
MD	1	7	4	11	20	15	27	66	138	238	274	183	180	379	30	1573
MA	11	15	13	33	69	163	95	80	129	117	290	212	148	256	9	1640
MI	0	0	1	0	1	0	4	21	165	134	110	35	23	33	0	527
MN	8	22	55	86	64	94	94	67	92	70	85	201	141	165	0	1244
MS	0	0	0	0	0	0	0	6	7	7	–	0	0	0	0	20
MO	0	0	0	2	1	1	4	5	108	205	193	150	108	87	0	864
MT	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0
NE	0	0	0	0	0	0	0	0	0	0	20	15	6	2	0	43
NV	1	0	0	0	0	0	0	0	7	2	7	1	5	1	1	25
NH	0	0	1	0	0	7	0	8	3	4	35	49	15	31	0	153
NJ	10	57	70	155	175	219	257	500	680	1074	852	681	786	1303	24	6843
NM	0	0	0	0	0	0	0	0	5	0	0	2	2	8	0	17
NY	7	170	267	466	1235	482	877	2637	3224	3244	3357	3370	2818	4417	18	26589
NC	0	0	1	16	14	6	2	19	61	87	81	65	86	77	3	518
ND	0	0	0	0	0	0	0	1	12	3	2	2	2	0	0	22
OH	0	0	0	3	2	2	14	39	99	36	173	32	30	78	6	514
OK	0	0	0	0	0	2	2	4	16	13	31	6	19	76	0	169
OR	0	0	1	10	5	10	19	4	5	11	5	13	8	0	0	91
PA	1	2	0	5	29	31	65	306	626	553	1022	1119	1085	1327	116	6287
RI	3	29	20	20	41	57	74	121	415	101	177	274	272	471	0	2075
SC	0	0	0	1	3	3	3	10	18	7	10	2	9	7	1	74
SD	0	0	0	0	0	0	2	2	3	2	1	1	0	0	0	11
TN	0	0	1	1	4	1	1	13	30	28	45	33	20	13	0	190
TX	0	0	1	18	172	8	33	18	82	44	15	73	48	45	0	557
UT	0	1	1	0	0	1	0	2	3	1	3	6	2	3	0	23
VT	0	0	0	0	0	0	0	1	1	11	7	9	12	13	0	54
VA	0	0	0	1	2	7	27	25	54	129	202	123	95	129	1	795
WA	0	0	0	0	0	0	8	9	33	30	3	14	9	0	0	106
WV	0	0	0	0	0	0	0	5	15	11	44	14	50	27	4	170
WI	25	58	69	176	135	162	358	246	762	337	424	525	401	0	0	3678
WY	0	0	0	0	0	0	0	0	6	5	9	5	9	5	0	39
†	106				4	2	2									114
Total	226	497	594	1518	2752	1389	2394	4882	8803	7943	9344	9658	8257	11144	225	69626

*Information provided by the Lyme Disease Foundation, Hartford, CT, 1995.
†Unknown.

In nature, the Lyme disease bacteria exists in a cycle involving ticks and small animals. Deer ticks prefer to live in the woods; dense, mature woods with a thick undergrowth of shrubs and small trees are their favorite habitat. They also are found, to a lesser degree, along the edge of the woods where the lawn meets the woods.[11] They are spread in the wild by animals such as birds, mice, raccoons, and deer, but domestic animals such as cats, dogs, horses, and cows can also carry infected ticks closer to and into the home.[4] When mice become infected, they remain so for long periods of time without any apparent ill effects; however, they spread the infection to immature ticks that feed on them. These infected ticks then spread the disease to other rodents and animals as well as humans. Adult ticks seem to prefer to feed on larger animals, especially deer. Deer are resistant to Lyme infection and do not directly participate in the life cycle of the Lyme bacteria except to provide blood meals for adult ticks and to potentially carry the ticks to regions not previously invaded. More than 20 species of birds are known to be infected and have been theorized to transport the ticks over great distances, resulting in spread to previously unaffected areas.[12] Fortunately, Lyme disease does not appear to be communicable between humans.[10]

Signs and Symptoms

The tick bite is usually painless and may go unnoticed because the tick injects a local anesthetic at the site of the bite. Symptoms of Lyme disease may vary and can appear days or even years after an individual is bitten by an infected tick. Early symptoms of Lyme disease may appear within days or weeks of a tick bite and can improve within several weeks, even without treatment.[13,14] Delayed symptoms of untreated Lyme disease can occur at any time of the year and frequently follow months of dormancy during which patients are asymptomatic. Late symptoms may appear weeks, months, or years later and may improve or come and go without treatment. In some cases, there may be no symptoms at all, which makes the disease difficult to diagnose.[15]

One of the first symptoms of infection is a characteristic red, circular, expanding rash, the border of which is raised and warm (Fig 3). This rash is named erythema migrans (EM). Estimates suggest that it may present in less than 50% of tick bites or or may go undetected. Generally painless, it is sometimes associated with a mild stinging or itching. This rash can begin at least 4 days after the bite but might not appear for several weeks. The appearance of the rash is not always associated with other physical signs of illness. In less than 10% of reported cases, the rash could appear in several places as erythema chronicum migrans, in addition to the site of the bite.[16]

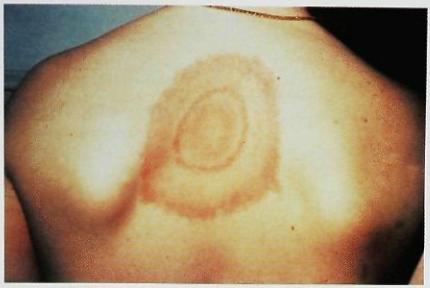


Fig 3 Erythema migrans rash. (Courtesy of Robert A. Schwartz, MD; reprinted with permission from Cutis 1991;47:229.[17])

As will be demonstrated in the following case reports, "An accurate and detailed medical history and careful physical examination form the basis required for arriving at a correct diagnosis of Lyme disease. Medical history and physical examination are of particular importance in this disease because currently available laboratory tests are less than satisfactory."[6]

Symptoms and clinical findings of the various stages of the disease are listed in Table 2.

Lyme Disease—A Multisystem, Multiorgan Disease

The stages of Lyme disease may be divided into early, disseminated, and chronic. Patients have reported a variety of symptoms that have been associated with these different stages (Table 3).

Dental Concerns

Dental patients with a history of Lyme disease require special considerations. When practicing in areas hyperendemic for Lyme disease, health status interviews or questionnaires must explore the possibility of Lyme disease in the same way a patient would be interrogated concerning any other perti-

Table 2 Manifestation of Lyme Disease by Stage[18]*

	Early Infection		Late Infection
System†	Localized stage 1	Disseminated stage 2	Persistent stage 3
Skin	Erthyema migrans (EM)	Secondary annular lesions	Acrodermatitis chronic atrophicans
		Diffuse erythema or urticaria	
		Evanescent lesions	
		Lymphocytoma	
Musculoskeletal		Migratory pain in joints, tendons, bursae, muscle, bone	Prolonged arthritis attacks
		Brief arthritis attacks	Chronic arthritis
		Myositis‡	Peripheral enthesopathy
		Osteomyelitis‡	Periostitis or joint subluxations below acrodermatitis
		Panniculitis‡	
Neurologic		Meningitis	Subtle mental disorders
		Cranial neuritis, Bell's palsy	Axonal polyneuropathy
		Motor or sensory radiculoneuritis	Leukoencephalitis
		Subtle encephalitis	Encephalomyelitis
		Mononeuritis multiplex	Spastic paraparesis
		Myelitis‡	Ataxic gait
		Chorea‡	Dementia‡
		Cerebellar ataxia‡	
Lymphatic	Regional lymphadenopathy	Regional or generalized lymphadenopathy	
		Splenomegaly	
Heart		AV node block	Cardiomyopathy
		Myopericarditis	
		Pancarditis	
Eyes		Conjunctivitis	Keratitis
		Iritis‡	
		Choroiditis‡	
		Retinal hemorrhage or detachment‡	
		Panopthalmitis‡	
Liver		Mild or recurrent hepatitis	
Respiratory		Nonexudative sore throat	
		Nonproductive cough	
		Adult respiratory distress syndrome‡	
Kidney		Microscopic hematuria or proteinuria	
Genitourinary		Orchitis‡	
Constitutional symptoms	Minor	Severe malaise and fatigue	Fatigue

*The staging system provides a guideline for the expected timing of the different manifestations of the illness, but this may vary in an individual patient.
†The systems are listed from the most to the least commonly affected.
‡Since the inclusion of this manifestation is based on one or a few cases, it should be considered possible but not proven manifestations of Lyme diseases.

Table 3 Patient Complaints [19]

Early	Disseminated		Chronic
	Early	Late	
EM rash	Headache	Severe headache, migraine headache	Migraine headache
	Joint pains	Crippling arthritis	Arthritis
	Body aches	Swollen joints	Loss of libido
	Night sweats	Heart problems	
	Sensitivity to light, sound, touch	Hypersensitivity to light, sound, touch	
	Migratory pains	Crippling migratory pains	Muscle weakness
	Fatigue	Severe fatigue	Fatigue
	Bell's palsy	Problems with vision and increased eye complications	Disequilibrium
	Heart palpitations	Seizures	
	Swollen glands	Nosebleeds	
	Stiff neck	Memory loss	
	Worsening of asthmatic symptoms	Confusion (Lyme fog)	
	Disorientation	Dyslexic reversals	Dyslexia
	Lyme fog	Sleep disturbances, nightmares	
	Conjunctivitis	Reports of abnormal magnetic resonance images, computerized axial tomography scans, electroencephalograms, cerebrospinal fluid	Hearing loss
	Sleep disturbances	Gastrointestinal symptoms	

nent medical history.[20] It may not be enough to merely ask the patient if he or she was ever diagnosed as having Lyme disease. The dental practitioner must be suspicious of the patient who provides a diverse medical history with multisystem complaints that seem to run in cycles; unexplained, recurrent illnesses require further investigation.[21]

Premedication

A percentage of dental patients require prophylactic antibiotic therapy for dental procedures. This may be because of a history of cardiac disorders, or other issues. However, when providing antibiotics for patients who have or have had Lyme disease, special considerations are necessary.

The infectious organism of Lyme disease, *B. burgdorferi*, can "hide" in tissue and seems to have the ability to remain dormant for long periods of time. The administration of antibiotics for any reason may result in an untoward effect on the patient's immune system, causing what is known as a *Jarisch-Herxheimer reaction.* Commonly known as *Herxheimer reaction,* the patient experiences an exacerbation of Lyme disease–related symptoms that may last from a few days to a few weeks. The Jarisch-Herxheimer reaction is a clinical syndrome occurring soon after the first adequate dose of an antimicrobial drug to treat infectious diseases such as Lyme disease, syphilis, and relapsing fever.[22] This reaction may be misconstrued as an allergic response by the patient or treating doctor, but a careful history may find otherwise.[23] In fact, the precipitation of Herxheimer reaction may assist in arriving at a diagnosis where none had been possible in the past.

Possible Effects From Dental Treatment

Exacerbation of Lyme disease–related symptoms may also arise from dental procedures that involve disturbance of connective tissue, such as periodontal surgery and endodontia. Spirochetes seem to have an affinity toward connective tissue. When surgical or invasive procedures are performed, the resultant healing and scar tissue formation may attract these organisms and result in increased symptoms.[24,25]

The introduction of an infectious process produces an immune response to its antigens. It has also been postulated that this response to *Borrelia burgdorferi* may trigger the onset of an autoimmune disease. "However, inflammatory diseases seem to continue, even when the organism is seemingly eliminated. In fact, many inflammatory diseases thought to be of an autoimmune nature may actually be caused by an immune response directed against non viable, but persistent microbial antigens present in the target tissues, or even against occult viable infectious organisms."[26] As with any similar disease process, when the patient is physically challenged by trauma, nonrelated illnesses, or dental procedures, a flare of the autoimmune/Lyme disease—related symptoms may be anticipated.

Temporomandibular Disorders

Lyme arthritis has commonly been associated with large peripheral joints such as the knee. However, the temporomandibular joint (TMJ) is the fourth most commonly affected joint in Lyme disease.[27] "The synovial membrane becomes hypertrophic with numerous folds producing villus like structures overlaid by hyperplastic synovial cells. Varying degrees of synovial edema are present. The presence of vasculopathy within the synovium and extensive fibrin deposits in the stroma are very suggestive of Lyme arthritis, even in the absence of clinical history."[28] Arthroscopy of the temporomandibular joint of a Lyme disease–infected patient, as in other joints so affected, reveals significant synovial inflammation and swelling (Hoffman D. TMD and Orofacial Pain Center, University of Medicine of New Jersey, unpublished data, 1995) (Figs 4a and 4b). Patients may present with ear pain, TMJ pain, or claudication of the masticatory musculature, which is secondary to Lyme disease rather than the result of primary temporomandibular disorders.[21] Temporomandibular disorders symptomology usually occurs early in the course of Lyme disease.[27,28] A meticulous clinical evaluation and history are vital in arriving at a differential diagnosis.

Neuralgia

Neuralgic facial pain, Bell's palsy, pain of the masticatory musculature, and TMJ pain are characteristic of Lyme disease and may cause a patient to seek a dental examination, as the following case reports illustrate.[29,30]

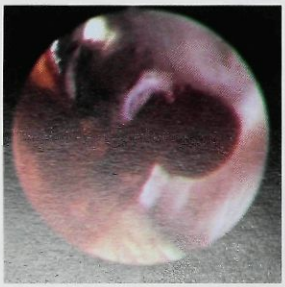
Case Reports

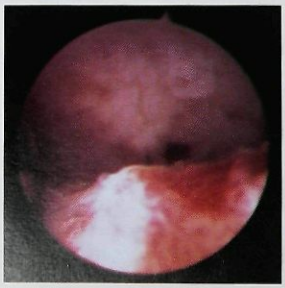
Case 1

JG is a 49-year-old woman with a 14-month history of neurologic symptomology. On close questioning, it was concluded that her symptoms probably date further back than that. Her chief complaints were:

1. Facial pain
2. Facial numbness
3. Pain in the left eye
4. Numbness of the extremities
5. Joint pain
6. Difficulty concentrating

The numbness was originally of the left side of the face but appeared to spread more into the left side of the forehead with time. It was occasionally associated with pain in the left eye and bilateral blurred vision. In retrospect, she recalled that she also had symptoms of extremity paresthesias and joint pain as well as cognitive dysfunction. However, she failed to mention these symptoms until much later.





Figs 4a and 4b Arthroscopic views of the temporomandibular joints of two patients with confirmed Lyme disease demonstrate significant synovial hypertrophy. Capillary invagination of the synovial plica *(left)*; vascularity of the synovial hyperplasia *(right)*. (Courtesy of David Hoffman, DDS).

Initial neurologic evaluation revealed hyperreflexia and extensor plantar responses. A magnetic resonance image (MRI) of the brain was negative, the cervical spine was positive for a small left C5–6 herniated disc, and the thoracic spine was positive for a central disc bulge at T6–7. It was thought that neither her symptoms nor signs were related to these findings.

By the time symptoms were present for a full year, she had undergone extensive dental treatment, which included restorations, endodontia, and an apicoectomy, with no resolution of her facial pain and numbness. Her neurologic examination was unchanged. A spinal tap was performed, revealing the presence of oligoclonal bands. Serologic testing revealed a Lyme disease titer that was positive, as was a Western Blot. Spinal fluid was analyzed and was borderline positive on enzyme-linked immunosorbent assay (ELISA) testing, although no Western Blot or Polymerase Chain Reaction test was performed. Following the spinal tap, the patient developed lumbosacral pain that has persisted, with a new onset of palpitations. A lumbar spine MRI revealed early degenerative joint disease without disc herniation.

The patient was finally treated for Lyme disease after approximately 2 years from the date of onset of her complaints. The treating physician immediately initiated therapy with oral amoxicillin. During the first week of therapy, the patient experienced severe exacerbation of her palpitations and had an abnormal electrocardiogram suggestive of myocarditis. This is consistent with a Herxheimer reaction, which is well known in Lyme disease, and strongly supports the diagnosis. This reaction also should be perceived as supportive evidence that in fact, she does have cardiac involvement, even in the absence of a positive echocardiogram.[32–35]

The patient was admitted to the hospital within a few days of initiation of treatment. Intravenous ceftriaxone sodium (Rocephin, Roche Laboratory, Nutley, NJ) was administered for presumptive Lyme carditis. The patient was continued on this medication for 8 weeks. She reported an initial flare of symptoms, followed by improvement lasting for several weeks, then another flareup several weeks later, which slowly resolved. This is fairly common in patients with Lyme disease because they enter cyclical phases during treatment. At an examination performed several months into treatment, the patient reported feeling considerably better but was still quite symptomatic. Of most concern was her significant cognitive dysfunction. Although she is a mathematician, she no longer teaches. She noticed that she would either forget what she was writing on the blackboard or would put something down other than what she had intended. She became disoriented and confused, and she developed short-term memory loss. She was also clearly depressed despite taking fluoxetine hydrochloride (Prozac, Dista Products, Indianapolis, IN).

The most recent Western Blot performed was positive for IgG proteins consistent with Lyme disease. Her most recent electrocardiogram was unremarkable and her examination revealed a blank facial expression that could be mild paresis or simply a reflection of her depressed affect. She does not have complete facial palsies; however, incomplete facial palsy, which has been seen clinically in patients with Lyme disease, improves with treatment.

Case 2

CC is a 28-year-old woman who was referred for a facial pain evaluation. Her chief complaints were:

1. Stabbing pain in the left TMJ
2. Bilateral facial pain during chewing, which was described as jaw muscle fatigue that increases with continued function
3. Migraine headache (diagnosed by the physician)
4. Neck pain
5. Upper body numbness
6. Visual problems—persistent blurred vision

The patient also stated that she felt "depressed and handicapped." While her history was reviewed, she stated that she had been healthy until approximately 1990, when she had several episodes of "big," swollen knees, which were treated by nonsteroidal anti-inflammatories with apparent success. The knee problem resolved but left her with what she described as a "mild arthritis." Since that time, she has experienced what was diagnosed as episodes of chronic sinusitis and sinus infection. There was occasional shortness of breath. She was given antibiotic therapy in June 1993, for approximately 1 month. In July 1993, she experienced an acute migraine attack with "strokelike" symptoms, which included a left-side facial nerve palsy; numbness in her face; slurred speech; a tingling sensation in her face, arms and hands; and blurred vision. The blurred vision persisted for 11 weeks. She also described possible claudication of leg muscles. She experienced additional episodes of headache, which were described as bilateral and arising from the shoulders and neck with radiating pain into the forehead. Neck stiffness accompanied these headaches. The headaches were described as

debilitating and were preceded by the blurred vision. The headaches would last several days or weeks. She also described episodes of stabbing pain in her face, which resulted in "facial droopiness" that for lasted several hours.

During the time prior to the episodes of swollen knees, she was quite active and enjoyed hiking along the Appalachian Trail. During the past few years, however, she has felt more and more disabled. She stated that she no longer has felt confident about driving. She added, "I feel as though I've lost my independence due to the illness and constant pain." She does not recall a tick bite.

This patient has been evaluated by many physicians, including neurologists and otolaryngology specialists. She had been given a variety of medications with little effect. They include nonsteroidal anti-inflammatory drugs, subclinical doses of propranolol hydrochloride (Inderal, Wyeth-Ayerst Laboratories, St David, PA), and tricyclic antidepressants. A brain MRI showed areas of plaque or enhancing lesions, but this was not deemed clinically significant at the time.

An evaluation of the patient's TMJs and masticatory musculature found that she had a normal mandibular range of motion, with slight crepitation of the left joint. Both joints were moderately tender to palpation, and the masticatory musculature was also moderately to severely tender. Although the clinical presentation might have accounted for some of the facial pain and headache complaints, it was believed to be responsible for only a small portion of her problems.

A palpatory evaluation of the cervical and upper back muscles was remarkable for multiple tender areas. A cranial nerve screening evaluation was positive for asymmetry of peripheral vision, which was less on the right (cranial nerve II), hyperalgesia of the right cheek and forehead (cranial nerve V, divisions 1 and 2), ie, pain is produced by light touch, and a slight weakness of the left lip during grimace or smiling (cranial nerve VII).

The differential diagnoses were:

1. Capsulitis/synovitis of the TMJs, bilaterally
2. Associated myalgia of the masticatory musculature
3. Ruling out of Lyme disease prior to accepting the diagnosis of migraine
4. Ruling out of fibromyalgia (characterized by multiple, nonreferring tender points in muscles)
5. Consideration of a demyelinating process such as multiple sclerosis

Treatment options were discussed with the patient regarding potential temporomandibular disorders. She was also advised that local therapies might not be productive if other systemic conditions were present. She was referred for serologic testing for Lyme disease, and the results were positive. The patient is currently receiving antibiotic therapy and is improving. Since treatment began, she has not experienced any additional episodes of TMJ pain, facial pain, or Bell's palsy.

Case 3

JN, a 34-year-old woman, presented for a facial pain evaluation with chief complaints of:

1. Pain of the right cheek
2. Pain of the forehead and chin
3. Paralysis of the lower half of the right side of her face

Coincident with her pregnancy in 1993, the patient developed a dental infection of the maxillary right first premolar. This tooth had prior endodontic therapy in 1991, and the dentist elected to perform an apicoectomy, which was done in July 1993. The patient described this procedure as difficult and recalled that the wound required resuturing on four occasions. Eventually it was allowed to granulate in. She recalled that soon after this procedure, she began to experience a sense of numbness associated with the surgical site as well as a dull aching sensation on the right side of her face. She also became aware of "electrical jabs deep in the upper jaw." She attributed this discomfort to the "healing process" and anticipated recovery. The discomfort seemed to persist, and there was point tenderness over the surgical location.

On the morning of January 28, 1994, she awoke with a partial palsy of the right side of her face. She was aware of an inability to purse her lips. During the course of the day, the condition worsened until she was not able to close her eye, contain liquids in her mouth, or smile. She called her dentist who referred her to an oral surgeon. On examination, according to the patient, the surgeon offered only two treatment options. They were either to repeat the apicoectomy or to remove the involved tooth. The tooth was subsequently extracted, but as might have been anticipated, no relief of the Bell's palsy ensued.

The patient next contacted her obstetrician/gynecologist, who referred her to a neurologist. Facial nerve palsy was diagnosed. Diagnostic testing confirmed a lower motor neuron disorder. Because the patient was pregnant, medications were not considered.

Since onset, facial muscle function has returned to approximately 80% of normal, but the patient continues to experience a dull, aching, tight, or tugging pain in her face extending from above the right eye, through her cheek and into her maxillary and mandibular right jaw.

A clinical examination found no dental etiology for any facial pain complaint. An evaluation of the TMJs and jaw function was unremarkable. However, a palpatory examination of the masticatory musculature did disclose multiple tender areas of these structures, particularly on the right.

A cranial nerve evaluation was positive for weakness of the facial nerve, particularly of the muscles of the lower half of facial expression. The patient was able to fully close both eyes. The right eye could be forced open, but not as easily as reported earlier by the patient's neurologist.

Although it was unlikely that any dental pathology or any of the dental procedures performed for this patient were in any way involved in the onset of Bell's palsy, dental pathology was considered to be the primary etiology by her treating dentists.

Although the Bell's palsy seems to have evolved coincidentally with various dental surgeries, there is no support for any connection between the condition and procedures. The onset of the paralysis was remote from the dental surgeries. Initially, the medical history was unremarkable except for a report of asthma and minor allergies. However, after a series of serologic tests, the patient tested positive for Lyme disease.

Testing, Treatment, and Other Concerns

Laboratory Testing for Lyme Disease

The issue of laboratory testing for Lyme disease is one fraught with controversy.[36,37] The standard test performed by most commercial laboratories is the ELISA. Although this test has been studied extensively, there are numerous test kits, many of which are unreliable. Several studies have demonstrated that the same tube of blood sent to different laboratories yield markedly disparate results. Certain autoimmune diseases are thought to cause a false positive result, a factor further confusing the issue. Very few laboratories in the country are consistently accurate.

The Western Blot is a highly sophisticated method of testing for Lyme disease. The patient's blood is cross-reacted with a series of antigens that are derivatives of the proteins of *Borrelia burgdorferi*. This method is highly specific, since certain fragments of these proteins are unique to this organism. As with other tests, the commercial kits are variable in their sensitivity. Reagents need to be maintained and purified under strictly controlled circumstances. Again, there are only a handful of specialized laboratories with the advanced technology necessary to provide consistent results. Inconsistencies have been observed between and within commercial laboratories. Overall, combining all the studies, the sensitivity and specificity of both tests are equal. When interpreting these tests, referral to the recommendations of the CDC for standardization and interpretation of Lyme disease Western Blot testing is suggested.

The Western Blot should be performed on all positive or equivocal ELISA specimens. In addition to the CDC recommendations, it must be pointed out that other proteins not included in the CDC recommendations are now recognized as very species specific for *Borrelia burgdorferi* and must be included in any study of any patient suspected of having Lyme disease. An analysis of confirmed Lyme disease in patients concluded that tests that included these species-specific proteins not included in the CDC recommendations were 27.7% more specific than those that did not include them (Tilton RC, North American Analytic Laboratory Group, New Britain, CT, 1994).

Many immunologists disagree with the strain restrictions and patient restriction. Based on 5,034 positive specimens, it has been found that proteins not included in the CDC recommendations are significant and must be included in the criteria.[38] By including these bands, the sensitivity of the results is increased from 74% to 87.2% (Tilton RC, North American Analytic Laboratory Group, New Britain, CT, 1994). Therefore, these proteins must be included despite the CDC recommendations.

In addition to the aforementioned comments regarding CDC criteria, many clinicians disagree with the CDC recommendations about performing Western Blots only to confirm equivocal or positive ELISA tests. An ongoing study that has analyzed 20,000 cases reveals that a significant percentage of patients have positive Western Blots in the absence of any findings on ELISA testing (Tilton RC, unpublished study, North American Analytic Laboratory Group, New Britain, CT, 1995).

Both the IgM and the IgG Western Blots should be used in the serodiagnosis of early Lyme disease. The specificity of the positive IgM blot criteria decreases after this time, and positive IgM results in patients with late symptoms must be viewed cautiously. Prior antibiotic therapy may invalidate the results.[39] In any case, these are only recommenda-

tions, and the diagnosis remains one that is primarily clinical. The laboratory should be used as a secondary source for support for or against a diagnosis.

Two laboratories currently perform another serologic test called the enzyme capture assay, which is very specific for recent infection. In preliminary studies, this test has a 60% probability of detecting early cases.

Several laboratories are now doing the Polymerase Chain Reaction, which is a test in which specific genetic fragments of the bacteria are detected in the blood, urine, spinal fluid, and synovial fluid. The yield on this test is yet to be determined. A urine test of great value is the Lyme Urine Antigen test. This test is also a method of detecting the breakdown products of the Lyme bacteria.

An additional blood test performed in only one laboratory in the country is the Gunderson test. This test is for the detection of "killer" antibodies in a patient's serum; ie, the ability of the antibody to kill live bacteria in a laboratory setting. If positive, this is a definite indication of infection.

Treatment

"Because infection rates in ticks are high, primary care physicians are faced with the dilemma of whether to treat patients bitten by these ticks immediately or to wait and treat them only if the symptoms of Lyme disease develop."[40] The Lyme Disease Foundation currently advocates immediate preventive antibiotic therapy at the first sign of a tick bite in endemic areas.[41] It is generally agreed that early treatment is important.[42] Currently accepted treatment includes antibiotic therapy. In some cases, oral antibiotics are the treatment of choice. In more chronic cases, intravenous antibiotic therapy is required.[16,43,44]

Prevention

The control of Lyme disease is a difficult problem. Investigations continue to lead to a better understanding of the complicated ecology of Lyme disease, identifying areas where ticks commonly carry the Lyme bacterium. Recent research has studied methods to make such endemic areas safer, including application of insecticides and the use of deer fences. Future methods may include host-targeted insecticides, environmental alteration, and biologic control.[45]

According to Fish,[45] a medical entomologist from New York Medical College, "The big problem is the suburbs, or the woodburbs. You don't have these ticks where you don't have deer." He and other experts have made the following suggestions[45,46] to avoid Lyme disease for people who spend time in areas infested with *Ixodes dammini*, the tick that carries *Borrelia burgdorferi*:

1. When in the woods, brush, or tall grass, socks and shoes should be worn, pants should have bottoms tucked into shoes, and shirts should be tucked in with a snug collar, long sleeves, and cuffs. Brush and leaf litter should be avoided.
2. An insect repellent containing diethyltoluamide (DEET) should be applied and reapplied as necessary to exposed skin, socks, lower pant legs, sleeve cuffs, and collars. For children, repellent should be applied to clothing, not to skin.
3. After an outing, a shower should be taken to wash away unattached ticks. A daily full-body exam should be performed on people and pets, keeping in mind that nymphs are the size of a pin head before feeding.
4. All ticks should be removed promptly with fine-pointed tweezers placed on the mouth parts, not the head or body, as close to the skin as possible. The area the tick came from should be washed and swabbed with alcohol. The tick should not be handled.
5. Individuals can make their yards inhospitable to ticks by removing leaf litter and keeping soil relatively dry. Borders should be cleaned up, and an insecticide should be used to treat the yard edge and a little way into adjacent wooded areas.
6. Individuals should watch for the bull's-eye lesion: red with a clearing in the center that may follow infection. If an infection is suspected, medical help should be sought promptly.

Research and Prognosis

With an increase in public awareness of Lyme disease, research in treatment and prevention of Lyme disease has intensified. This research has taken three distinctive avenues of investigation.

Currently there are very few reliable tests for the diagnosis of Lyme disease. At this time, the clinical expertise of the physician performing the examination remains the key. Investigators are seeking new and reliable tests to confirm the presence of a *Borrelia burgdorferi* infection.

Although antibiotic therapy is the best treatment available for Lyme infection, the medications used are not always 100% effective in all

cases. The majority of patients do respond favorably; however, more specific antibiotics are being developed, which will hopefully result in more predictable results. Ancillary treatment modalities include analgesia, antidepressants, acetazolamide (Diamox, Lederle Laboratories, St David, PA), nutritional supplements, and physical therapy.

A vaccine against Lyme disease is already available for veterinary use. However, it is not yet available for use in humans, although experimental versions are now being tested.[47,48] The efficacy has not yet been determined. Unfortunately, there are substantial flaws in the study design that may interfere with the interpretation of results.[49–51]

Summary

Lyme disease, a debilitating infection that may cause a variety of mild to crippling symptoms, has been confounding patients and physicians for many years. Through diagnostic failure, mistreatment, and overtreatment, the cost to the public and the physical and psychologic toll taken on those suffering with this disease has been exorbitant.[52] It is to be hoped that the spread of this disease can be controlled with heightened public and professional awareness; dedicated physicians, dentists, and health care providers; and advances in research. The public and health care professions must remain diligent in their pursuit of a solution.

Acknowledgment

The authors would like to thank Lori Heir for her assistance with the research for this study.

References

1. Lukasik S, Wojtowicz A, Betkowski A. Oddzialu Otolaryngologicznego Szpitala Wojewodzkiego Nr 1, Rzeszowie. Wiad Lek 1993;46:506–510.
2. Steere AC, Malawista SE, Snydman DH, Shope RE, Andiman WA, Ross MR, Steele FM. Lyme arthritis: An epidemic of oligoarticular arthritis in children and adults in three Connecticut communities. Arthritis Rheum 1977;20:7–17.
3. Orloski K, Bailey R, Campbell G, Dennis D, Herrington J. National Surveillance for Lyme Disease—USA [pamphlet]. Fort Collins, CO: Centers for Disease Control and Prevention, National Center for Infectious Diseases, Division of Vector-Borne Infectious Diseases, 1995.
4. Craft JE, Schoen RT. Lyme Disease. Croton, CT: Pfizer Central Research, 1993.
5. Clinical Briefs. Am Fam Physician 1993;48:155.
6. Diagnosis and treatment of Lyme disease, National Institute State of the Art Conference. In: Clinical Courier [newsletter of the National Institute of Arthritis and Musculoskeletal and Skin Diseases and National Institute of Allergy and Infectious Disease]. 1991;9(5).
7. Taylor GC. Lyme disease: An overview of its public health significance. J Environ Health 1991;54:24–27.
8. Thacker SB, Keewhan C, Brachman PS. The surveillance of infectious diseases. JAMA 1983;249(9):1181–1185.
9. Brandes J. Lyme disease in Hunterdon increases 322%. Star-Ledger 1993 Nov 4:50.
10. Centers for Disease Control and Prevention (CDC). Information Service, Atlanta, 1994.
11. Smith-Fiola, Porcellini. Am Nurseryman 1991. Cited in: Prevent Tick Bites: Prevent Lyme Disease. Rutgers Cooperative Extension of Ocean County, 1992.
12. Masters EJ, Donnel H, Fobbs M. Missouri Lyme Disease: 1989–1992. J Spirochetal Tick-Borne Disease Dis 1994;1(1).
13. Steere AC. Lyme disease—1993. Bull Rheum Dis 1993; 42(6):4–7.
14. Steere AC. Lyme disease. Proc Natl Acad Sci USA 1994; 91(7):2378–2383.
15. Barbour AG, Fish D. The biological and social phenomenon of Lyme disease. Science 1993;260(5114):1610–1616.
16. Burrascano JJ Jr. Managing Lyme Disease: Diagnostic Hints and Treatment Guidelines for Lyme Borreliosis, ed 10. East Hamptom, NY, 1995.
17. Schutzer SE, Schwartz RA. Diagnosing Lyme disease: Often simple, often difficult. Cutis 1991;47:229–232.
18. Steere AC. Lyme disease. N Engl J Med 1989;321: 586–596.
19. Land D, DeSilva DM. Coping With Lyme Disease. New York: Henry Holt, 1993.
20. Steere AC. Lyme disease: A growing threat to urban populations. Proc Natl Acad Sci USA 1994;91:2378–2383.
21. Goldfarb D, Sataloff RT. Lyme disease: A review for the otolaryngologist. Ear Nose Throat J 1994;73:824–829.
22. Negussie Y, Remick DG, DeForge LE, Kunkel SL, Eynon A, Griffin GE. Detection of plasma tumor necrosis factor, interleukins 6, and 8 during the Jarisch-Herxheimer Reaction of relapsing fever. J Exp Med 1992;175:1207–1212.
23. Hurley JC. Antibiotic-induced release of endotoxin: A reappraisal. Clin Infect Dis 1992;15:840–854.
24. Georgilis K, Peacocke M, Klempner MS. Fibroblasts protect the Lyme disease spirochete, *Borrelia burgdorferi*, from ceftriaxone in vitro. J Infect Dis 1992;166:440–444.
25. Klempner MS, Noring R, Roger RA. Invasion of human skin fibroblasts by the Lyme disease spirochete, *Borrelia burgdorferi*. J Infect Dis 1993;167:1074–1081.
26. Behar S, Porcelli SA. Mechanisms of autoimmune disease induction. Arthritis Rheum 1995;38:458–476.
27. Steere AC, Dwyer E, Winchester RJ. Association of chronic Lyme arthritis with HLA-DR4 and HLA-DR2 alleles. N Engl J Med 1990;323:219–223.
28. Duray PH. Histopathology of human borreliosis. In: Coyle PK (ed). Lyme Disease. Chicago: Mosby Yearbook, 1992:49–58.
29. Kelsey JH. Lyme disease: An important consideration in the differential diagnosis of TMD. J Mich Dent Assoc 1990;72:209–210.
30. Fallon BA, Nields J. Lyme disease: A neuropsychiatric illness. Am J Psychiatry 1994;151:1571–1583.
31. Eppes SC, Klein JD, Caputo GM, Rose CD. Physician beliefs, attitudes, and approaches toward Lyme disease in an endemic area. Clin Pediatr (Phila) 1994;33:130–134.

32. Lesniak OM, Lirman AV, Antiufev VF. Heart disorders in Lyme disease. Klin Med (Mosk) 1994;72:45–47.
33. Joksimovic Z, Saranovic-Kuljic M, Lazarevic-Milanovic N, Jankovic-Nikolic A, Pavlovic-Nikolic S. Lyme carditis—Case report. Glas Srp Akad Nauka [Med] 1993;43:237–239.
34. Woolf PK. A 12 year old with fever and rash. Pediatr Care 1994;10:249–250.
35. Steere AC, Batsford P, Weinberg M, Alexander J, Berger HJ, Wolfson S, Malawista SE. Lyme carditis: Cardinal abnormalities of Lyme disease. Ann Intern Med 1980; 93:8–16.
36. Fung BP, McHugh GL, Leong JM, Steere AC. Humoral immune response to outer surface protein C of *Borrelia burgdorferi* in Lyme disease: Role of the immunoglobulin M response in the serodiagnosis of early infection. Infect Immunol 1994;62:3213–3221.
37. Satz N. Immunology and diagnostic test results in Lyme borreliosis. Schweiz Med Wochenschr 1992;122:1779–1791.
38. Dressler F, Whalen JA, Reinhardt BN, Steere AC. Western blotting in the serodiagnosis of Lyme disease. J Infect Dis 1993;167:392–400.
39. Golightly MG. Laborite for the Diagnosis of Lyme Disease. Stony Brook, NY: Stony Brook University Hospital, Dec 1994.
40. Costello CM, Steere AC, Pinkerton RE, Feder HM. A prospective study of tick bites in an endemic area for Lyme disease. J Infec Dis 1989;159:136–139.
41. Rationale for antibiotic prophylaxis upon tick bite. Lyme Disease Foundation Newsletter. Hartford, CT: Lyme Disease Foundation, 1994.
42. Massoraotti EM, Luger SW. Treatment of early Lyme disease. Am J Med 1992;92:396–403.
43. Rahn DW, Malawista SE. Lyme disease: Recommendations for diagnosis and treatment. Ann Intern Med 1991;114:472–481.
44. Wolf SR, Heininger U, Schneider W, Wenzel D. Current aspects in diagnosis and therapy of pediatric facial paralysis. HNO 1994;42:624–628.
45. Fish D. Environmental risk and prevention of Lyme disease. Am J Med 1995;24:98:2S–8S.
46. Rutz DA. Recommendations for Control of Ticks in New York State. Milbrook, NY: Cornell Cooperative Extension, Farm and Home Center [newsletter]. July 1990.
47. Chang YF, Appel MJ, Jacobson RH, Shin SJ, Harpending P, Straubinger R, et al. Recombinant OspA protects dogs against infection and disease caused by *Borrelia burgdorferi*. Infect Immun 1995;63:3543–3549.
48. Telford SR III, Kantor FS, Lobet Y, Barthold SW, Spielman A, Flavell RA, Fikrig E. Efficacy of human Lyme disease vaccine formulations in a mouse model. J Infect Dis 1995;171:1368–1370.
49. Fikrig E, Telford SR III, Wallich R, Chen M, Lobet Y, Matuschka FR, et al. Vaccination against Lyme disease caused by diverse *Borrelia burgdorferi*. J Exp Med 1995; 181:215–221.
50. Marconi RT, Konkel ME, Garon CF. Variability of osp genes and gene products among species of Lyme disease spirochetes. Infect Immun 1993;61:2611–2617.
51. Wilske B, Preac-Mursic V, Jauris S, Hofmann A, Pradel I, Soutschek E, et al. Immunological and molecular polymorphisms of OspC, an immunodominant major outer surface protein of *Borrelia burgdorferi*. Infect Immun 1993;61:2182–2191.
52. Barbour AG. Biological and social determinants of the Lyme disease problem. Infect Agents Dis 1992;1:50–61.

Resumen

Enfermedad de Lyme: Consideraciones Odontológicas

Aunque la enfermedad de Lyme se ha expandido rápidamente y es difícil de diagnosticar, la revisión de la literatura odontológica no revela muchas referencias sobre esta enfermedad. Los practicantes de odontología deben estar al tanto de los efectos sistémicos de este desorden que es a menudo multiorgánico. Sus manifestaciones clínicas pueden incluir dolor dental y facial, parálisis del nervio facial, cefalea, dolor de la articulación temporomandibular, y dolor muscular masticatorio. Los efectos que son precipitados cuando se realizan procedimientos dentales en un paciente con enfermedad de Lyme, también deben ser considerados. Este estudio discute la epidemiología y diagnóstico de la enfermedad de Lyme, su prevención, y factores para considerar cuando se hace un diagnóstico diferencial. También se considerarán el cuidado dental del paciente con enfermedad de Lyme y los tratamientos disponibles actualmente. Se presentan tres casos.

Zusammenfassung

Lyme-Arthritis: Überlegungen für die Zahnmedizin

Obwohl sich die Lyme-Arthritis schnell verbreitet hat und schwierig zu diagnostizieren ist, sind in der zahnärztlichen Literatur nur wenige Hinweise auf diese Erkrankung zu finden. Zahnärzte sollten die möglichen klinischen Manifestationen kennen: Gesichtsschmerzen, Zahnschmerzen, Fazialisparese, Kopfschmerzen, Kiefergelenk- und Kaumuskelschmerzen. Es müssen auch mögliche Auswirkungen von zahnärztlicher Tätigkeit bei solchen Patienten erwogen werden. Diese Studie diskutiert die Epidemiologie und Diagnose der Lyme-Arthritis, ihre Prävention und Faktoren, welche bei der Differentialdiagnose zu berücksichtigen sind. Die zahnärztliche Betreuung dieser Patienten, sowie momentan mögliche Behandlungen werden auch besprochen. Es werden drei Fallbeispiele geschildert.