

Tolle Totum 

Reversing Stage 3 Kidney Disease

A Case Study

SHAWNA EISCHENS, ND

*Why is my urine frothy?
What can I do to help my kidneys from being damaged even further?
What is the cause of protein in my urine?*

“John” had asked previous physicians these questions but had received no helpful or hopeful answers. This patient walked into my office on 8 medications, frustrated with his medical team, and scared that death was imminent.

“Is there anything you can do to help my kidneys and reverse my diabetes?” he asked.

“Of course there is!” I said with excitement, knowing the incredible

power of the body to repair itself when provided with the proper conditions to do so. I was confident and hopeful for this particular patient because he had already shown a level of commitment by drastically changing his diet. When patients are invested in their health, I have more confidence and trust in the body’s ability to heal.

Background

This patient is one of the more than 30 million people in the United States diagnosed with type 2 diabetes mellitus (T2DM), which accounts for 90-95% of all types of diabetes.¹ In 2015, according to the CDC, an estimated 1.5 million new cases of diabetes were diagnosed among US adults

Continued on page 3



Student Scholarship - 1st Place Research Review 

Heartburn & Heartbeats

Exploring GERD & Atrial Fibrillation

**ANDREW HUBBARD, BSC (HONS)
ELLEN WONG, BSC (HONS), ND**

Atrial Fibrillation (AF) is the most commonly diagnosed heart arrhythmia in America.^{1,2} Most causes are of cardiovascular origin and include hypertension, coronary artery disease, and cardiac valve disorders; however, the list of non-cardiac causes, such as obstructive sleep apnea, chronic obstructive pulmonary disease (COPD) and electrolyte imbalances, appears to be increasing.² Most recently, gastroesophageal reflux disease (GERD) has emerged as a significant non-cardiac contributor to AF.^{3,4,5} Equally, AF and its subsequent treatment may also play a role in the development of GERD, further strengthening suspicions of a bi-directional phenomenon.^{6,7} Additionally, a large body of research has been conducted investigating the potential gastric-cardiac connection with respect to GERD and AF. Despite this,

the topic remains rather elusive in both conventional and alternative healthcare discussions. Thus, the aim of this paper is to review these conditions and shed light on their connection, discuss what mechanisms might be involved, and to evaluate effectiveness and suitability of the current medical treatments. We will also discuss how the naturopathic doctor can play a vital role in identifying and treating patients affected with these conditions using non-pharmacologic means.

Atrial Fibrillation

Atrial fibrillation is an irregular heart rhythm that stems from an uncoordinated contraction, or “quiver,” of the atrial chambers. This leads to ineffective blood pumping within the heart, which puts additional stress on the ventricles.² As clot formation often occurs in the setting of stagnant and inefficient blood pumping within the heart, patients with AF are at an increased

risk of thromboembolic events such as stroke, and are often thus relegated to life-long treatment with anticoagulant medications.⁸ AF is associated with heart failure, is an independent risk factor for early death, and is a significant detriment to quality of life.⁸


The prevalence of AF in the general population is estimated at 1-2% but increases in patients over the age of 60.² Risk factors for AF include but are not limited to: age, male gender, smoking, hypertension, obesity, obstructive sleep apnea (OSA), and cardiac valve defects.⁵

Patients with AF may be asymptomatic, or they may have brief (paroxysmal) episodes of fatigue and an irregular heartbeat. They may also present to the ER with more alarming features, such as syncope, tachycardia, and chest pain.² Patients are often treated with antiarrhythmic and anticoagulant drugs or undergo radiofrequency catheter ablation

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Friday July 13, 2018 (1:00-5:30 PM)

1:00-1:30 PM
1:30-2:15 PM
2:15-2:30PM

Carolyn Ledowsky, ND
Debby Hamilton, MD, MPH
Panel: Carolyn Ledowsky, ND & Debby Hamilton, MD, MPH

Why is methylation so important. What is it we do?
Environmental Toxins and infections in Autism
Questions

2:30-3:00 PM
3:00-3:45 PM

BREAK
Andrew Rostenberg, PhD

Genetic Roots of Stress and Anxiety. How the gut affects your mood.
Gut Case Studies
Endocrine Disrupting hormones – Who's at risk?
Questions
Close

3:45-4:15 PM
4:15-5:00 PM
5:00-5:15 PM
5:15 PM

Andrew Rostenberg, PhD
Nicole Bijlsma, ND, LAc
Panel: N. Biljsma, ND, LAc
Carolyn Ledowsky, ND

Saturday July 14, 2018

8:00-8:45 AM
8:45-9:30 AM

Carolyn Ledowsky, ND
Sara Wood, ND

Genetic SNP's that affect susceptibility to environmental toxins
Organophosphate and Endocrine Disrupters How to Test & Evaluate - Case Studies & Treatment
Questions

9:30-9:45 AM

Sara Wood, ND & Carolyn Ledowsky, ND
Carolyn Ledowsky, ND

Pathway planner & Case studies

9:45-10:45 AM

MORNING BREAK

Organic Acids Evaluation and methylation insights
Questions

10:45-11:15 AM

Matthew Pratt-Hyatt, PhD

Organic Acids Case Studies (RECORDING)
What is Awry in Essential Methionine and Folate Metabolism
Questions

11:15-12:30 PM

Matthew Pratt-Hyatt, PhD

When Hormones, Organic Acids and Genetics Collide: How to put the puzzle pieces of testing together.
Questions

12:30-12:45 PM

LUNCH

Close

12:45-1:45 PM

Andrew Rostenberg, DC

1:45-2:30 PM

David Quig, PhD

2:30-3:15 PM

David Quig, PhD

3:15-3:30 PM

AFTERNOON BREAK

3:30-4:00 PM

Carrie Jones, ND, MPH

4:00-4:45 PM

Carrie Jones, ND, MPH

4:45- 5:00 PM

Carrie Jones, ND, MPH

5.00pm

Sunday July 15, 2018

8:00-9:30 AM

Carolyn Ledowsky, ND & Nicole Bijlsma, ND, LAc
Questions: Carolyn Ledowsky, ND & Nicole Bijlsma, ND, LAc

Other environmental factors – heavy metals, viruses, lyme, mold, vaccines, diet. Who's susceptible

9:30-9:45 AM

Stephanie Seneff, PhD (VIA DIRECT LINK LIVE)

Glyphosate disruption of methylation

9:45-10:45 AM

Questions: Stephanie Seneff, PhD

10:45-11:00 AM

MORNING BREAK

GlyphosateGenetic SNP's and case studies

11:00 - 11:30 AM

Carolyn Ledowsky, ND

Putting it all together for patient assessment– where to start?

11:30-12:15 PM

Carolyn Ledowsky, ND

Close

12:15- 1:00 PM

1:00 PM

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Continued from top of page 1

18 years and older.¹ The total estimated annual cost of diagnosed diabetes in the United States in 2012 was \$245 billion, with medical expenditures being approximately 2.3 times higher than expenditures would be in the absence of diabetes.² Diabetes has been listed as the seventh leading cause of death, yet the following statistics show that diabetes is strongly correlated with heart disease, the #1 leading cause of death:

- At least 68% of diabetics age 65 or older die from some form of heart disease³
- Adults with diabetes are 2-4 times more likely to die from heart disease than adults without diabetes³
- Diabetes is listed as the only cause of death for 79 535 people, yet more than triple that number of people (252 806) have diabetes listed as an underlying or contributing cause on their death certificates¹

Regardless of these staggering statistics, we know that unregulated blood sugar levels have detrimental consequences for patients' health and national healthcare costs. Medications can help decrease hyperglycemia, which is essential for overall health, but they tend to come with harmful and comprehensive side effects. Medications are not curative, are not ideal for health restoration, and tend to instill a dependent, disempowering mentality based on a false sense of security. The road to curing or reversing T2DM requires more than medications alone. Reversal of any disease process requires an investment in personal health and a commitment to lifestyle changes. Foundations of health for

people with diabetes include the following:

- **Diet:** Small changes can have profound effects. Patients may do best with drastic changes to their current routine, yet many patients report feeling overwhelmed at the thought of an overnight diet overhaul. Educating and inspiring patients to make multiple, small changes – versus scaring them that they can never enjoy a dessert again – can have a more beneficial effect long-term. People are often shocked to learn that milk, beans, bread, potatoes, tortillas, and fruit can have a detrimental effect on blood sugar levels. Encouraging consumption of low-carb, nutrient-rich foods with plentiful antioxidants can help heal the entire person. These include green, leafy and cruciferous vegetables; wild fish such as salmon; organic, grass-fed/finished meat; green tea; and turmeric. Eggs, walnuts, almond butter, flax seeds, pumpkin seeds, and cheese may also be beneficial for someone with hyperglycemia.
- **Exercise:** Daily movement is key! A vigorous walk after dinner, bouncing on a rebounder while watching TV, or strength-training alternating with jumping jacks or dynamic planks for high-intensity interval training, are just a few ideas. Resistance exercise is ideal, as it burns 19 times more glucose compared to aerobic exercise.⁴
- **Sleep:** Less than 5 hours of sleep per night has been shown to increase a person's risk of obesity by 235%.⁴ Sleep deprivation leads to a decrease in both insulin sensitivity and fat

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Student Scholarship – 2nd Place

2nd Place Case Study – IBS with Obesity

Justin Wise, CA, B Comm

Peter K. Raisanen, NMD, BSc

Remarkably simple measures resolve a longstanding case of obesity and irritable bowel.

Tolle Causam

Autoimmune Disease: The Role of Gut Bacteria

Keegan Sheridan, ND

Gut bugs play critical roles in both the pathogenesis and prevention of autoimmunity.

cells' sensitivity to insulin.⁴ The body's natural response is to produce less leptin and more ghrelin, leading to increased hunger, especially for carbohydrates.⁴ Inadequate sleep also contributes to elevated cortisol,⁴ which can increase glucose and contribute to insulin resistance, all of which are counterproductive in patients with diabetes.

Case Study

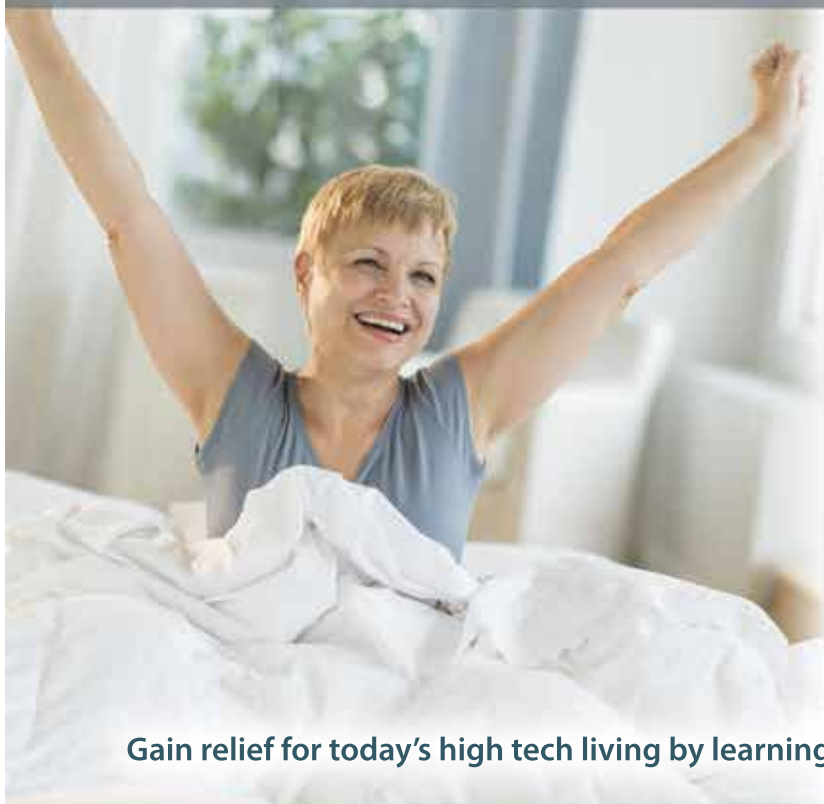
John, a 62-year-old Caucasian male, presented in my office after being given a diagnosis of T2DM within the previous 12 months. Diagnosed as pre-diabetic in 2010, he likely had uncontrolled hyperglycemia for many months, or even years. The

typical concurrent diagnoses with T2DM of hyperlipidemia and hypertension were also part of his medical history. Without an ideal comparison of urine microalbumin levels, there wasn't enough information for confirmation; however, according to KDIGO (Kidney Disease: Improving Global Outcomes) guidelines, this patient appeared to be in the "high risk" G3bA1 or G3bA2 kidney disease category. Regardless of his specific diagnosis, the sequela of kidney disease the previous summer came as a worrisome shock to him, and he implemented a drastic dietary change immediately upon hearing these findings.

John reported his previous health history to include excess weight since childhood, as well as rosacea, depression,

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anxiety, right-sided renal calculi, and acid reflux. His current health history included cataracts, bilateral hand tremors, leg cramps, insomnia, and some occasional mild, bilateral neuropathy that he described as “itching” or “like wearing socks” on his feet.

He claimed feeling like he was in good health until 2007, although he had some weight and reflux concerns before then. At his heaviest, he weighed 295 pounds (BMI of 43.6). He noticed his urine could appear foamy, and his bowel pattern tended to be soft or loose. His sleep was better than ever since improving his diet. He was sleeping 8-9 hours a night, and expressed a desire to continue using quetiapine, an antipsychotic, for the off-label use of insomnia.

His previous standard American diet, consisting of fast and fried food, candy,

chocolate, soda, and cookies, had recently and drastically changed to exclude these foods, in addition to potatoes and grains. He began eating more salads, cheese, fish, steak, and vegetables, while sticking to his routine of 2 meals a day. He had lost 25 pounds, with little to no exercise in just over 2 months. He was monitoring his fasting blood sugars in the morning and in the evening after dinner. With his recent dietary improvements, his blood sugars were better than previous months, but still high, ranging between 101-303 mg/dL in the weeks leading up to our appointment.

PCP-Prescribed Medications

1. **H D** Quetiapine fumarate 50 mg x 7 years for insomnia. Patient wanted to stay on this medication.
2. **R** Lisinopril 10 mg for hypertension
3. **R D** Ranitidine 150 mg x 2 at night x

- 31 years for acid reflux
4. **C** Metformin HCL 1000 mg twice daily for T2DM
5. **R** Glipizide XL 10 mg twice daily for T2DM
6. **R** Sitagliptin 50 mg for T2DM
7. **R D** Hydrochlorothiazide 25 mg for hypertension
8. **R** Na-bicarbonate 650 mg x 2 weeks after low CO2/bicarbonate lab finding

Key:

- R** = Caution with renal impairment
- D** = Caution with diabetes
- H** = Caution with hyperlipidemia
- C** = Contraindicated in people with eGFR <46

Note: One week prior to my patient’s first visit, he had self-prescribed acetaminophen for ear pain. Within 1 hour, he experienced vertigo, nausea, and gait disturbances.

Acetaminophen is not recommended for people with kidney or liver impairment. This patient’s poor kidney function likely put him at additional risk for such side effects.

Additionally, various statins had been prescribed by multiple physicians over the years for hypercholesterolemia. Statins come with specific precautions for people with renal impairment or diabetes. This patient reported experiencing flank pain as a side effect of these drugs, and elected to discontinue their use.

Self-Prescribed Supplements

1. Vitamin B1, B2, B6 (uncertain dosages)
2. B12, 1000 µg
3. Vitamin E, 1000 IU
4. Turmeric, 400 mg
5. Lutein, 20 mg
6. Probiotic

PE Findings

- Blood pressure: 112/84 mm Hg
- O₂ saturation: 98%
- Pulse: 84 bpm
- Weight: 260 lb
- Height: 5’9”
- Body mass index: 38.4

Although labs from various physicians had been ordered, only those done consistently and with comparable units are included in this article for fair comparisons.

Two weeks before our appointment, his labs show clear cause for concern: his kidneys were not properly functioning, resulting in electrolyte imbalance (Table 1, Figure 1).

Treatment

Prioritizing cost-effective supplementation to produce beneficial results is a goal with all patients; however, this patient’s extreme financial concerns were expressed and a modest treatment plan was presented. I commended and encouraged him on his commitment to his recent dietary modifications, and provided a dietary handout to further balance blood sugars.

Initial treatment recommendations included:

- 10-20 minutes of exercise each day
- Hot Epsom salt baths 3 times per week and/or sweating during exercise to balance elevated potassium levels. (Epsom salt baths were also indicated for his likely decreased magnesium levels, although we didn’t confirm this via lab testing until days later.)
- 5-10 drops per day of a liquid homeopathic combination product to promote healthy kidney, liver, and digestive function
- 6 caps per day of a proprietary product with kidney extract, to support and restore kidney function
- Supportive acupuncture and a blood draw for labs were done in office

John’s lab results arrived days later showing his blood type (O) and a detrimental progression of his kidney dysfunction during the weeks prior to our appointment. We discussed the fact that hydrochlorothiazide (HCTZ) and ranitidine are not recommended for people with kidney disease or diabetes. HCTZ promotes insulin resistance and diabetes, depletes magnesium, lowers HDL-C, and raises LDL-C, all of which are antagonizing and created an obstacle-to-cure for this patient.

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Joseph Burrascano Jr., MD

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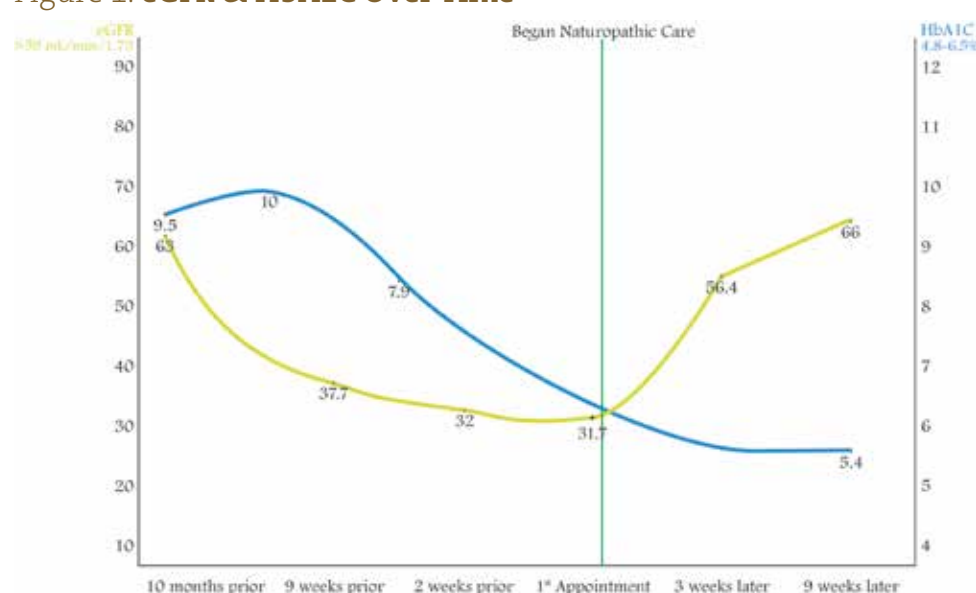
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Table 1. Lab Results Over Time

	Reference Interval	10 Months Prior	9 Weeks Prior	2 Weeks Prior	3 Weeks Later	9 Weeks Later
Fasting Glucose	65-99 mg/dL	240		145	91	110
BUN	8-27 mg/dL	21	40	56	19	21
Creatinine	0.57-1.27 mg/dL	1.23	1.87	2.2	1.34	1.17
eGFR	>59 mL/min/1.73	63	37.7	32	56.4	66
BUN/Creatinine	10.0-24.0	17.1	21.4	25.5	14.2	18
Sodium	134-144 mEq/L	139	137	132	140	141
Potassium	3.5-5.2 mEq/L	5.2	5	5.6	4.1	4.9
CO₂	18-29 mEq/L	26	18	19.5	18	24
TC/HDL-C Ratio	0-4.4	5.5		5.9		4.7
HbA1C	4.8-6.5% (AACE)	9.5	10	7.9		5.4
Magnesium, serum	1.6-2.3 mg/dL				1.6	2.1

(BUN=blood urea nitrogen; eGFR=estimated glomerular filtration rate; TC=total cholesterol; HbA1C=hemoglobin A1C)

Figure 1. eGFR & HbA1C Over Time



John was on the verge of stage 4 kidney disease. As a result, the following additional recommendations were made:

- Take 1 tsp 3x/day of an herbal combination tincture with nephroprotective/nephrorestorative benefits
- Increase kidney extract product to 9 caps per day
- Eat more wild/organic/free-range meat, fish, and/or eggs, and avoid gluten, in accordance with his blood type
- Stop HCTZ (with no previous or current edema, this was possible without weaning); instead begin amlodipine 5 mg

First Follow-up, 3 Weeks Later

John reported that his leg cramps and loose stool had ceased and that he had lost a few pounds. His daily blood sugars improved, with a range of 86-206 mg/dL. Since starting amlodipine, his hand tremors had worsened. He expressed a desire to get off of his medications and reported that his primary care physicians did not plan to run labs or have an appointment for another 6 months. He was aware that discontinuing medication was not realistic without making lifestyle changes and supporting his body to help balance blood sugars, cholesterol levels, and blood pressure while also helping his kidneys repair with supportive treatments. With a recent loss of his job and health insurance, John was unable to afford cash pricing for sitagliptin or any equivalent. Sitagliptin was his most recent medication prescribed for T2DM, is not recommended for patients with a GFR between 30 and 50 mL/min, and does not clinically lower HbA1c levels as well as other medications.

PE Findings

Blood pressure: 156/84 mm Hg
Pulse: 73 bpm
O₂ saturation: 98%
Weight: 258 lb

Remarkably, just a few weeks after beginning treatment, his labs showed a 78% increase in GFR, along with balanced electrolytes, BUN, and glucose.

Treatment

- Continue previous treatment, and add 1 scoop of a powder with glutamine and curcumin to encourage digestive healing. Experiment 1 week later with reducing ranitidine to every other day. If tolerated without reflux, take 1 ranitidine every third day the following week.
- Decrease daily amlodipine dose to 2.5 mg
- Begin a supplement that helps normalize blood pressure while supporting the cardiovascular system: 1 cap twice daily
- Monitor for any increase of blood sugars after discontinuing sitagliptin

Second Follow-up, 3 Weeks Later

John's daily blood sugars ranged from 89-190 mg/dL, even after stopping sitagliptin 2 weeks prior. He had lost 2 more pounds. Mild heartburn was observed by the patient nights 3 and 4 of his wean from ranitidine; otherwise, he had experienced no symptoms of heartburn. Exercise was still minimal. He had never experienced any irregular heart rhythms, and his physical exam was consistent with regular heart sounds and rhythms. Emotionally, he felt more balanced.

PE Findings

Blood pressure: 140/70 mm Hg
O₂ saturation: 97%
Pulse: 54 bpm
Weight: 256 lb

Treatment

- Continue with previous recommendations. Finish current digestive healing powder; don't replenish.
- Begin a supplement that helps balance blood sugars: 1 cap twice daily
- If blood pressure readings are within range, discontinue amlodipine 2 days before next appointment

Third Follow-up, 3 Weeks Later

Home-monitored blood sugars ranged from 77-179 mg/dL. The patient reported better readings, except after eating pasta or desserts, which clearly correlated with higher readings. A total of 3.5 more pounds were shed. He stopped amlodipine 3 days prior, and reported no concerns or symptoms. He reported feeling better overall.

PE Findings

Blood pressure: 145/80 mm Hg
O₂ saturation: 98%
Pulse: 57 bpm
Weight: 252.5 lb

John's labs continued to improve, including the best TC/HDL ratio and A1c % he had achieved in years.

Treatment

- Continue treatment plan, minus Epsom salt baths
- Discontinue Na-bicarbonate
- Finish and hold off on buying more self-prescribed supplements
- Increase exercise frequency and/or duration

Summary

Even with minor changes and a budget-conscious treatment plan, this patient's healing was profoundly rapid. Within weeks of beginning treatment, he decreased his medication use by 50% and was more balanced physically, emotionally, and physiologically. Proper diet, sleep, and exercise patterns create a foundation for health, while eliminating obstacles to cure and supporting the body with restorative treatments potentiates healing.

As Dr Henry Lindlahr stated, "Chronic supposed-to-be-incurable diseases yield to the natural treatments, provided there is enough vitality in the system to respond to treatment and the destruction of vital parts and organs has not too far advanced." I appreciate this patient's motivation to heal and commitment to his health before his diseases were too far progressed. I am filled with gratitude to our miraculous body, this patient, and naturopathic medicine for continuing to amaze me with the incredible, innate ability of the body to heal. ▀

References available online at ndnr.com



Shawna Eischens, ND, ("Dr E") is a 2012 graduate of SCNM who practices at Rockwood Natural Medicine Clinic in Scottsdale, AZ. As a primary-care physician who focuses on mental and digestive health, Dr E is passionate about helping people realize their greatness to achieve physical and emotional health. Outside of the office, she enjoys volunteering with Naturopathic Medicine Institute and Big Brothers Big Sisters, as well as exploring nature via foot, bike, car, or boat.

STAFF

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Publisher
Razi Ann Berry
publisher@ndnr.com

Founders
Razi Ann Berry
David A. Tallman, DC, ND
publisher@ndnr.com

Medical Editor
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Editorial Coordinator
Vanessa Wiley
editor@ndnr.com

Director of Sales and Marketing
Matthew Knapp
ads@ndnr.com

Circulation Manager
Tara Hall
circulation@ndnr.com

Creative Direction
Jaclyn Threadgill
jaclyn@ndnr.com
www.jthreadgill.com

Foreign Correspondent
S.A. Decker Weiss, NMD, FASA

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Scottsdale, AZ 85252-2968
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- Educational and grassroots programs that further the naturopathic cause
- Any other trend, event, or development you believe is pertinent to the profession

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Continued from bottom of page 1

in persistent cases where drug treatment has failed.²

The pathophysiology of AF is complex and uncertain; however, it is generally accepted that changes to cardiac muscle cells (myocytes) take on different electrical properties and begin disrupting the pace-making ability of the sino-atrial node (SA), which leads to uncoordinated atrial contractions.¹ As this progresses over time, a process of remodeling takes place in which irreversible changes to atrial tissue occur that lead to permanent AF. It is unclear what initiating factor causes myocytes to misfire and AF to initially develop, but it is suggested that local inflammation and mechanical stress are likely involved.¹

Gastroesophageal Reflux Disease

GERD is highly prevalent in North America and is one of the most common reasons for visits to primary care providers.³ The disease is characterized by the sensation of “heartburn” and regurgitation of acid from the stomach up into the esophagus. Reflux can present as erosive or non-erosive, the former being associated with esophagitis and dysplastic tissue changes, such as Barrett’s esophagus, which increases the risk of esophageal adenocarcinoma. Significant risk factors include smoking, obesity, OSA, and aging.⁷ Acid reflux symptoms are induced when sustained increases in intra-abdominal pressure (as seen in obesity) and consumption of large meals decrease lower esophageal sphincter (LES) tone. Increased visceral sensitivity and delayed gastric emptying may also be present.⁹ First-line treatment of GERD consists of an initial therapeutic trial of a proton-pump inhibitor, eg, omeprazole, and lifestyle modification.¹⁰

The Small Bridge In Between

Based on the close anatomical relationship between the esophagus and the left atrium, it would seem plausible that a disturbance in one tissue might affect the other. Indeed, only 2 mm separate the esophagus from the left atrium.¹¹ Interestingly, cardio-gastric interactions are not new. A rare and fatal complication of left atrial catheter ablation is the development of an atrio-esophageal fistula.¹¹ More commonly, however, patients will develop GERD or esophagitis after this procedure, demonstrating the potential impact of an inflammatory insult on proximal tissues.¹²

Reflux, Inflammation, & Vagal Stimulation

It is postulated that reflux symptoms leading to esophageal inflammation may disturb neighboring vagus nerve fibers, which then causes a cardiac reflex. This can in turn upset the heart rhythm, leading to episodes of AF.¹³ The heart receives input from both sympathetic and parasympathetic (vagal) signals, and this balance is crucial for maintaining normal conductivity. The following studies reveal that this balance is altered in patients with reflux symptoms and with AF. In many cases, lowering esophageal acid exposure via proton-pump inhibitors (PPIs) results in the resolution of AF symptoms.^{14,15} So, let’s examine this relationship a little closer.

Exploring the Evidence

A 2014 systematic review revealed a prevalence of AF ranging from 0.62%

to 14% in those patients with GERD as compared to those without GERD.³ The authors also found that patients experiencing more severe GERD symptoms had higher rates of AF.

Several large population-wide studies have examined prevalence rates of GERD with AF.^{4,5} In 2009, Kunz et al reported the relative risk (RR) of developing AF in those with pre-existing GERD to be 1.39. After adjusting for cardiovascular risk factors, this association fell to 1.19 but remained significant. Other risk factors reported in this study included hypertension (RR 1.75), smoking (RR 1.47), and diabetes (RR 1.21).⁵

Smaller studies, such as those conducted by Bunch et al,¹⁶ have shown no association between GERD and AF risk, although patients with a higher frequency of reflux symptoms reportedly had a slightly elevated risk of AF. Bunch et al also concluded that patients with esophagitis were at a significantly higher risk of developing AF (RR 1.94),¹⁶ supporting the local inflammatory hypothesis.

Hwang et al⁷ and Kubota et al⁶ have both reported a significantly increased

It is unclear what initiating factor causes myocytes to misfire and AF to initially develop, but it is suggested that local inflammation and mechanical stress are likely involved.

risk of developing GERD in patients with newly diagnosed AF. Those experiencing an increased severity of arrhythmia, which progressed from paroxysmal AF to permanent AF, reported higher reflux scores.⁶ This supports a potential bi-directional relationship.

To summarize, the research thus far has revealed a consistent upward trend for the prevalence of AF in patients with GERD and suggests that this association increases with a worsening of reflux symptoms and even more so in the presence of esophagitis. Conversely, AF appears to increase the risk of developing GERD, and this risk is further increased with worsening AF.

Does Suppressing Acid Suppress AF?

Two important studies have used unique approaches for evaluating the effects of GERD in AF and the subsequent effects of PPI therapy.

The first study used esophageal manometry (a technique for measuring acidity in the esophagus) and heart rate variability (a reliable and accurate way of measuring sympathetic and vagal activity) to correlate GERD and arrhythmic sequences.¹⁴ A significant number of patients demonstrated increased vagal responses corresponding with increased esophageal acid exposure. These patients then underwent PPI therapy, and both reflux and arrhythmic symptoms were significantly reduced.¹⁴

In the second study, 24-hour esophageal pH monitoring and simultaneous ambulatory ECG (Holter) monitoring were used to assess 3 cases of GERD and atrial arrhythmias.¹⁵ Reflux episodes were strongly correlated with episodes of AF in these patients, which resolved with acid-suppressive therapy. These studies, although small, provide the first real-time evidence regarding reflux events and atrial arrhythmias and their symptomatic resolution through PPI therapy.

While treatment of AF with PPIs in patients who have significant reflux symptoms has been shown to be effective at reducing AF events,^{14,15} these studies were of short duration and had small sample populations. Furthermore, the use of PPIs has been demonstrated to negatively impact nutrient and mineral absorption, including B12, calcium, vitamin D, and magnesium¹⁷; some studies suggest that low magnesium status could potentiate moderate-to-severe arrhythmias.¹⁸ Aside from patients with erosive esophagitis or Barrett’s esophagus, patients who are using PPIs to control

helpful.²² If LES function is compromised, or gastric emptying is delayed, clinicians may want to consider bitter and prokinetic herbs such as *Angelica*, *Zingiber*, *Gentiana*, and *Coleus*.²² As nicotine is a strong LES relaxant, smoking cessation should be strongly encouraged.

Structurally similar to PPIs, melatonin has been investigated for its potential benefits in the treatment of GERD. Studies have been promising thus far, suggesting that melatonin may be as effective as PPIs for controlling symptoms of GERD.²³ Melatonin might thus be an attractive, cost-effective option for patients who decline or do not respond to initial PPI therapy.

Lastly, acupuncture represents a promising strategy to treat the patient who presents with both AF and reflux symptoms. In fact, a recent meta-analysis concluded that acupuncture was at least as effective as drug therapy for treating arrhythmias and that a further benefit was observed when acupuncture was combined with drug therapy as compared with drug therapy alone.²⁴ The most commonly used acupuncture points reported in this study were Pericardium 6 (PC6), Heart 7 (HT7), and Urinary Bladder 15 (UB15). Acupuncture represents an ideal individualized treatment, as it can target both reflux symptoms²⁵ and the heart rhythm disturbances seen in these patients.

Closing Thoughts

Although still controversial, it appears that GERD and AF impact one another. While the prevalence of patients suffering from both GERD and AF ultimately remains low, there appears to be a significant clinical correlation between the two. The strength of this relationship increases when esophageal damage is present or when reflux symptoms increase, and is dependent on the type and severity of arrhythmia. Being aware of this clinical correlation may present clinicians with an opportunity to substantially improve patients’ quality of life and potentially prevent lifelong consequences of AF and side-effects of long-term drug use. Much can be gained by utilizing diet and lifestyle interventions such as healthy weight management, stress management, and fostering mindful eating behaviors. ■

[References available online at ndnr.com](#)



Andrew Hubbard, BSc (Hons), is currently studying in his 4th year at CCNM in Toronto. He has a passion for researching, writing, and speaking on topics that include gastrointestinal, neurological, and endocrine health. Andrew plans on opening an integrative clinic with a special interest in digestive wellness and is fascinated with designing future nutraceutical products. In between the clinic and the late-night PubMed searches, Andrew can usually be found sprinting out on the trails or at the gym re-inventing exercise to be more fun and enjoyable.



Ellen Wong, BSc (Hons), ND, is an associate professor at the Canadian College of Naturopathic Medicine (CCNM), where she graduated in 2009 and completed an accredited clinical residency in 2011. Currently, Dr Wong teaches an evidence-informed therapeutics class, as well as supervises interns at the Robert Schad Naturopathic Clinic (teaching clinic of CCNM). She practices in Ontario, Canada, with a focus on cardiovascular disease, its comorbidities, and geriatric care. In her spare time, Dr Wong explores the culinary scenes of Toronto, is an avid reader, and tries to squeeze in some time for exercise.

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Case-Based Naturopathic Curricula

Better in Concept Than Reality?

DAVID M. BRADY, ND, DC, CCN, DACBN

I read with great pleasure the article in *NDNR's* November 2017 issue, entitled "Rethinking Curriculum: Toward an Integrated Program in Naturopathic Medical Education," by Drs David Chandross and Fraser Smith. As a university vice-president overseeing a Health Sciences division within a comprehensive university that houses a naturopathic medical program alongside programs in nursing, physician assistant, nutrition, acupuncture/TCM, dental, clinical laboratory sciences, chiropractic, and doctoral health sciences education, I can assure you that I/we have confronted exactly these same issues across virtually all of these professions and healthcare paradigms. The issues of integrated learning, disaggregation of curricula content, development of novel methods of content delivery, and the addition of more "soft skills" to clinical education are not novel to the training of naturopathic physicians. However, it may be particularly challenging – and critical – for the naturopathic profession, since the very premise of our modern naturopathic medicine involves the integration of an amalgam of approaches and sub-disciplines within one very large tent (ie, botanical medicine, clinical nutrition, homeopathy, TCM, Ayurveda, physical medicine, counseling, etc).

At the University of Bridgeport, we are fortunate enough to have renowned Schools of Engineering and Education with well-developed STEM programs in various disciplines, including computer science, math, and biomedical engineering, where many of these same issues are being grappled with. Learning from one another across disciplines, and by virtue of our extensive online educational experience, has been key for us as we set off to modernize the way we educate newer generations of students in all of the STEM and health science fields. It is absolutely necessary that we train students to integrate information and attain competencies that bridge the traditional barriers of any one discipline or profession, as we understand that we are actually training students for jobs and fields that may not even exist yet.

That being said, I wanted to throw up several red flags based on hard-learned lessons and experiences in trying to migrate to the kind of new curriculum and program designs advocated in the extremely thoughtful and well-presented article by Drs Chandross and Smith. Firstly, in my 20-plus-year educational career, I have seen other institutions with professional programs – mainly in allopathic medicine and chiropractic – attempt such changes with somewhat mixed results, and, in a few cases,

disastrous ones. The disaggregated and cased-based curriculum design often seems better in concept, and looks better on paper, than it performs in reality. This is particularly true when institutions are dealing with faculty who are unfamiliar with this model and lack the appropriate training in this method of content delivery, with its heavy reliance on seamless integration and collaboration with faculty peers and program administrators. This design also places a great deal of responsibility back on the student regarding their individual outcomes. While some exceptionally motivated, intelligent, and confident students tend to thrive in this model, others can easily be left behind and left wondering why everything they needed to know was not clearly and systematically laid out and spoon-fed to them. These considerations must filter into the admissions process and the selection of applicants suitable for admission into such programs. With enrollment pressures facing the profession's institutions, it is a potentially very risky proposition for them to consider. This is also a very faculty-intensive model of educational delivery, and therefore very expensive to properly execute. Once again, with enrollment pressures come fiscal pressures for private institutions in healthcare fields such as naturopathic medicine, without large hospitals and healthcare networks supporting their academic programs.

The authors also rightly bring up concerns over how programmatic accreditors may view progressive changes in curriculum design and new and innovative forms of delivery. In my direct experience across professions, the programmatic accreditors have ranged from somewhat resistant to overtly oppositional to the kind of radical changes suggested in the Chandross and Smith article. This puts academic institutions at great risk in making such changes, even if they would like to, as they are clearly unwilling to jeopardize their program's accreditation status in the process. Remember, while the accreditation process is technically "voluntary," it is essentially a death sentence for a professional program to lose it.

Finally, from my direct experience in trying to affect positive legislative change regarding naturopathic medicine's scope of practice, including the granting of some level of prescriptive authority, one of the very first things that legislators and public health regulators ask is, "So, how many credits and courses in pharmacology do NDs take in their training?" Political opponents – mainly state medical associations – also try to cite perceived deficiencies in training or competency, strictly based on comparisons and assessments of professional curricula using these outdated measures, including; numbers of courses, clock hours, and credits in specific subjects. The task of explaining the virtues of the trend in clinical education away from discrete courses and credit loads in each "ology," and the integration of knowledge and skills acquisition within the context of clinical, case-based learning, systems-based courses, and clinical experiences adds yet another layer of difficulty in emerging successful in these legislative efforts regarding both initial licensure and/or scope of practice expansion for naturopathic doctors.

I wholeheartedly support the concepts that Drs Chandross and Smith put forth in their article, and I aspire to continue to affect such change in the training of the modern ND. I also believe it is critically important that we support the further development and funding of primary care and other specialty post-doctoral residency experiences as routine elements in ND training and licensure. However, I also wanted to acknowledge the significant challenges, as well as potential perils and pitfalls that will be in play as we chart this course. ▀

Sincerely,
Dr David M. Brady
University of Bridgeport

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WNF Update

Naturopathic Medicine in the Western Pacific Region



JON WARDLE, ND, PHD, MPH, LLM

While the World Health Organization (WHO) maintains a truly global focus, much of its practical work is conducted in its regional offices. One of those offices is the Western Pacific Regional Office (WPRO), headquartered in Manila, in the Philippines. The WPRO consists of 37 country members (see Figure 1). As occurs in other WHO regions, representation at a regional level is open to autonomous territories, whereas only full countries can maintain WHO membership. As such, membership of WPRO also includes the Chinese special autonomous regions (Hong Kong and Macau), the French Pacific island territories (French Polynesia, New Caledonia, and Wallis and Futuna), the US Pacific territories (American Samoa, Guam, and the Northern Marianas), and the British dependency of the Pitcairn Islands. The region is geographically disparate, including the Pacific Islands communities, Australasia, most of South-East Asia (except Indonesia, Myanmar, and Thailand) as well as China, Korea, the Philippines, and Japan. The absence of Indonesia (which is located in WHO's SEARO [South-East Asia Regional Office] region) physically separates the Southern and Northern countries of this region.

Naturopathic Presence in the Region

The World Naturopathic Federation (WNF) has 3 full members representing practitioners in 3 WPRO countries – Australia, Hong Kong, and New Zealand. In general, naturopathic practitioners in the region follow a similar practice model (with a broad therapeutic scope) to that of naturopathic doctors in North America, which has been the primary contemporary influence on practice (though WPRO practitioners may have more educational emphasis on ingestible medicines and less on physical therapy compared to their North American counterparts). Australia and New Zealand are undoubtedly the powerhouses of naturopathic practitioner representation in the WPRO region – representing over 90% of the estimated 10 000 naturopathic practitioners in the region, as well as the majority of the profession's historical presence – with the profession only beginning to emerge in many of the other countries of the region. Small (often solo-member) naturopathic practitioner associations have been identified in a number of countries – Japan, Malaysia, and the Philippines – though have not regularly interacted with the broader (or even regional) naturopathic community. In some of these emerging nations (eg, Malaysia) the government has announced its intention to regulate and develop naturopathic education in that country,¹ even though the relatively small size of the profession in that country has not made it a priority.

The emerging nature of the profession in much of the region means that naturopathic graduates practicing in many WPRO countries have been trained

outside of those countries (Australia and North America seem to be the major source of practitioners). There have been some attempts to develop local training institutions (most notably at several institutions in the Philippines), and the first intake of students into the new naturopathic program (modeled on North American and Australasian curriculum) at Thailand's Surin Rajabhat University² in 2014 may change this intake (Thailand is in the WHO SEARO region, but is the closest university program to many countries within WPRO). In many of these countries strong indigenous traditional medicine cultures make it difficult for other medical systems to make traction. For example, professional associations are prohibited in China unless sanctioned by the government, but strong support for Traditional Chinese Medicine in that country has made it difficult for other medical professions such as chiropractic or naturopathic medicine to gain anything more than a tokenistic foothold.

In many of these countries strong indigenous traditional medicine cultures make it difficult for other medical systems to make traction.

In other WPRO countries a small naturopathic practitioner workforce exists but remains unorganized. For example, Singapore – like Hong Kong – has a nascent naturopathic practitioner community largely made up of North American and Australian naturopathic graduates due to its permissive immigration policies. Unlike Hong Kong, however, the Singaporean naturopathic community has not coalesced around a single professional association. In some countries, legal recognition of naturopathic medicine has come prior to the presence of an established local practitioner workforce. For example, in the Pacific Islands nation of Vanuatu, an Australian naturopathic graduate (Dr Alan Profke) has been granted recognition as a registered primary care provider in his Paradise Clinic, which provides care to the residents of Aore Island.³ While initiatives such as these are essential to expand the profession into new areas, they do not create a sustainable profession, and more efforts are needed to ensure that local naturopathic capacity is also developed so that countries are not reliant on overseas practitioners.

History of Naturopathic Medicine in WPRO

The precise history of the development of the naturopathic profession in the WPRO region is difficult to ascertain. From its outset, naturopathy was an international profession. The American Naturopathic Association had identified naturopathic doctors in 20 countries in 1918, with

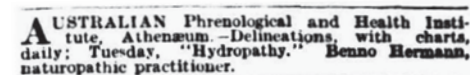
international practitioners listed (and some contributing to) Lust's *Universal Naturopathic Encyclopedia Directory*.⁴ In addition to the United States (the home country of the publication), this list included naturopathic practitioners in Australia, Fiji, Japan, New Zealand, and the Philippines. The preponderance of European names in that list of practitioners – both from the Continent and the British Isles – suggests that naturopathic practice entered the WPRO region from both North America and Europe simultaneously.

Then – as now – the Australian contingent of naturopathic practitioners was dominant (representing more than half of all listed practitioners in the region). There again the development of the profession appears to have been influenced early on by both European and North American developments. The first named naturopathic doctor in Australia was German immigrant (and student of Kneipp) Benno Hermann von Koenigswerder, who advertised his lectures

from 1903 and practiced in Melbourne and later in Brisbane (see Figure 2). However, many other newspaper articles referenced early Indian naturopathic developments (usually based on British training) as well as North American developments. For example, newspapers began quoting North American naturopathic journals as sources in health stories; an article by Dr Erieg from the *Naturopath and Herald of Health* was cited in a story on overeating and mental acuity in Toowoomba's *Darling Downs Gazette*.⁵

Australia is also the country where most historical records exist, and the naturopathic community appears to have been politically active early on. Lust, himself, spoke positively of legislative and professional developments in Australia, stating in the *Universal Encyclopedia* that “we look to Australia to inaugurate legislation to make poisoning the sick with drugs a penal offence.” However, the extent to which the history of naturopathy in the WPRO region (or even its most studied nation, Australia) has been studied is dwarfed by the scholarship that has been extended to the profession in North America and Europe. In Australia, for example, some academics have incorrectly assumed the profession was largely absent from early Australian history, and its entrenchment was really only spurred by the growth of the counter-cultural and natural healthcare movements of the 1960s and 1970s.⁶ Others have conflated the history of naturopathy with Western herbal medicine in Australia,

Figure 2. Newspaper Mention of Benno Hermann, 1903



The first specific naturopathic reference in Australian newspapers was published in *The Age* (Melbourne) on May 16, 1903.

and have viewed naturopathy as a subset of the herbalism tradition in Australia, or have confused the longstanding system of naturopathic medicine with the more recently emerged “natural medicine” movements.⁷ However, the presence of the profession in WPRO is longstanding.

The earliest Australian newspaper mentions of naturopathy as a specific form of medicine date back to 1903, and they highlight both the early entry of naturopathic medicine in Australia and the various international influences that helped to establish the medical system. Multiple newspaper entries of “naturopathy” occurred during 1903, and can lay claim to being the first specific and official mention of naturopathic medicine in Australia. However, it should be noted that references to core naturopathic modalities such as “nature cure,” “water cure,” and various elements of homeopathic, herbal, and dietetic medicine can be found much earlier – and the controversial question of the provenance of naturopathic medicine as an evolution of the Eclectic medical tradition may also mean that some earlier naturopaths may have categorized themselves as other therapists. The first mention of naturopathic medicine was a health column published in the Toowoomba-based *Darling Downs Gazette*, authored by an anonymous naturopath (signed off as “The Naturopath”) on the importance of sleep and exercise as a restorative agent for health.⁸ These were followed up that year by advertisements for individual practitioners in newspapers Brisbane (Queensland), Melbourne (Victoria), and Lismore and Armidale (New South Wales).

Despite often incurring the wrath of the medical profession, it appears that naturopaths did enjoy broad support and recognition from the public, albeit somewhat hampered by their small professional stature. Dr von Koenigswerder's position as a naturopathic physician in Brisbane was considered sufficient to draw on his expertise as an expert witness in several Court cases in the Queensland legal system.⁹ In 1905 the Victorian village of Mansfield advertised its attraction of naturopathic services as a sign of its progress.¹⁰ Maurice Blackmore – one of Australia's most well-known naturopaths and founder of what is now Australia's largest pharmaceutical company (which although classified as being part of the pharmaceutical sector, actually solely produces natural medicines) – was often the subject of attacks of the medical profession, which often sent investigators from the Medical Board of Queensland to charge him with practicing medicine without a license. After one such instance in 1951, the Queensland Parliament debated the veracity of Blackmore's practice and whether such actions were appropriate. In addition to noting the large number of testimonials to Blackmore's treatment from patients, Blackmore was also enthusiastically defended by 3 future Queensland Premiers.¹¹ This reflects the

contemporary scenario, whereby, despite lack of integration and recognition by government, naturopathic medicine remains enthusiastically supported by the public. In Australia, for instance, studies suggest that approximately 10% of the population regularly use a naturopath,¹² with one-third of patients using their naturopathic provider as their primary care provider.¹³ Naturopathic medicine is also the primary complementary medicine discipline in New Zealand.

Professional Naturopathic Development in the WPRO

Naturopathic development in the WPRO region occurred early. The Australian WNF member celebrates its centenary in 2019, when it will host the WNF Annual General Meeting. The first association for naturopaths in New Zealand occurred in 1940, when the New Zealand Association of Naturopaths and Osteopaths was formed, an association which still exists (though in a different iteration) to this day.¹⁴ In 1925 the South Australian association (the Botanic and Naturopathic Medical Association published the first Australian naturopathic journal, *Nature Cure and Medical Freedom*. This was followed by the *Ivaline Harbinger of Health* (1925-1927), *Nature's Path to Health* (1930-1950), *The Australian Naturopath* (1936-1964) in Australia, and *Health and Sunshine* (1930-1937) in New Zealand. However, beyond Australia and New Zealand, little is known about professional development of the naturopathic profession in other WPRO countries. This is in large part because, although early naturopaths have been identified, much of the profession outside of Australia and New Zealand was lost during the international post-war decline of the naturopathic profession and has only recently begun to develop again. For example, naturopathy in Hong Kong, the third WNF member, was only reintroduced to that territory 2 decades ago, when an Australian graduate began practicing in that territory.¹⁵

Although the naturopathic profession entered the WPRO region relatively early, the development of the profession has not been consistent. Despite early successes, the naturopathic profession in WPRO declined in the period following the Second World War. During the 1960s and 1970s, naturopathy was rejuvenated by the growth of the holistic health movement when, according to Evans,

the "new-style flower children" began to join the "old-style straight-backed nature cure adherents."¹⁶ While this undoubtedly rejuvenated the profession in terms of growth in practitioners and public utilization, it also created some dilemmas for the naturopathic profession in the region, as it began to be subsumed into a broader "natural medicine" movement. As naturopathic medicine was the only major complementary medicine discipline not regulated, one of the practical consequences of this was that its associations began to absorb other natural medicine professions that were also experiencing growth as part of the holistic health movement (such as acupuncture and homeopathy). This led to a situation where previously naturopathic associations became broader natural medicine associations (as was the case in Australia, where the National Association of Naturopaths slowly evolved into the Australian Natural Therapists Association),¹⁷ or naturopaths began to look towards other discipline associations that aligned with their professional interests better than multi-disciplinary associations (for example, in New Zealand where the New Zealand Association of Medical Herbalists became a natural home for many naturopaths). The lack of a distinct naturopathic voice in WPRO regions is only being remedied recently, in large part due to the catalyst of the formation of the WNF.

Naturopathic Education in WPRO

As in many other regions, early training of naturopathic practitioners focused on clinical apprenticeships and teaching clinics, with training and practice often taking place in institutes of naturopathy that also treated the public.¹⁸ This was often borne of necessity, as naturopathic training was not only unable to be accredited in formal education institutions (which were – and remain – largely public government institutions in the WPRO region), but naturopathic medicine itself was often discouraged or, in some cases, even illegal. In an article published in 1938 in *Nature's Path to Health*, the content of what such a course may have looked like was described by Frederick Roberts. (This description was followed by an advertisement for Robert's own college, the British and Australian Institute of Naturopathy):

The naturopath is a graduate of a naturopathic institution. His preparation for the professional careers as a naturopath consists of a four-year course of study, which includes what are known as the basic sciences. These sciences are: (1) Biology, (2) Chemistry, (3) Bacteriology, (4) Anatomy, (5) Physiology, (6) Embryology and Obstetrics, (7) Pathology, (8) Physical Diagnosis, (9) Psychology, (10) Nature Cure, (11) Hygiene and Sanitation, and other therapeutic subjects such as (1) Mechanotherapy or Physical Manipulation, (2) Hydrotherapy or Water Cure, (3) Electrotherapy, (4) Biochemistry [this was an early term for vitamin and mineral supplementation], (5) Dietetics, etc. The naturopath does not study materia medica or the prescribing of drugs, but he is taught phytotherapy or the use of herbs in the healing of the sick. In addition to the four years study of naturopathy or drugless healing, the naturopathic student spends one or two years at a naturopathic sanitarium or clinic. At the completion of his studies he obtains the degree of N.D. (Doctor of Naturopathy).¹⁸

Eventually, naturopathic education began to be brought into the university sector (where possible), and as private institutions began to be allowed to offer accredited degree programs, the standard of education in WPRO has been degree-level. There appears to have been some interaction between the Australian and international naturopathic education sectors. In 1971 it was announced in *The Health Leader*, for example, that after numerous years, Maurice Blackmore had managed to convince Dr Paul Hoffmeister, an eminent professor from the now defunct Sierra States University in Los Angeles, to teach naturopathic medicine at the Australian College of Naturopathy, Osteopathy and Chiropractic in Sydney (1 of the 3 constituent colleges – based in Brisbane, Sydney, and Melbourne – of the National Association of Naturopaths, Osteopaths and Chiropractors).¹⁹ These relations have continued over many years, with the WPRO naturopathic profession having a long history of collaboration with other regions.

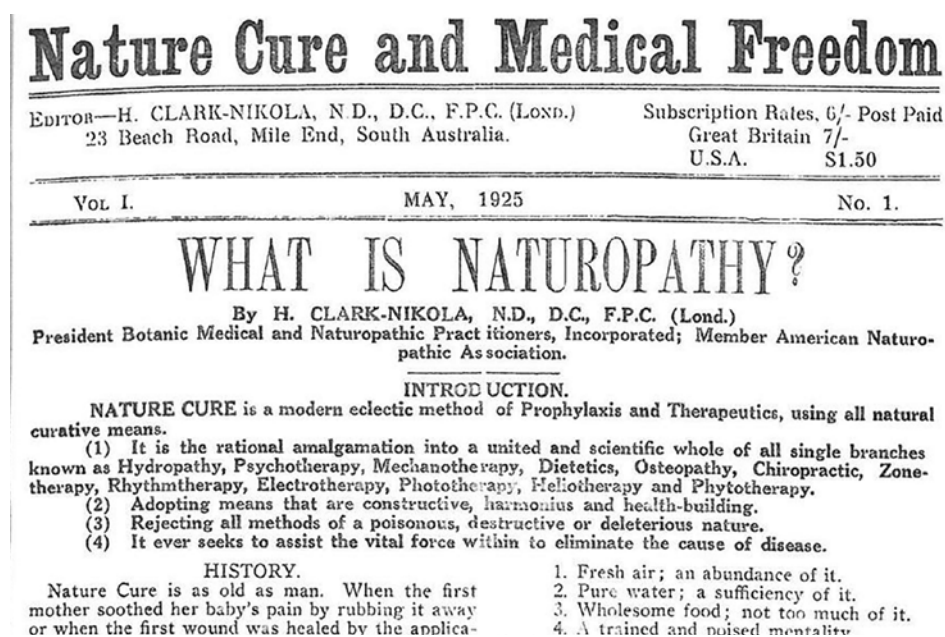
The first official foray of naturopathic education into the university sector was a partnership between Northern Territory University (now Charles Darwin University) and the Queensland Academy of Natural Therapies to develop a 4-year degree course through an institution called the Darwin Academy of Natural Therapies (DANT), which was announced and supported by the Northern Territory health minister of the time.²⁰ In the mid-1990s, reforms in Australia led to the ability of non-government universities and colleges to establish and offer degree courses, with the Australian College of Natural Medicine (Brisbane), Southern School of Natural Therapies (Melbourne), and the Australian Institute of Applied Sciences (Canberra) offering 4-year degree programs by 1995; Southern Cross University offered the first naturopathic program in a public university in 1996. Wellpark College and South Pacific College of Natural Therapies in New Zealand also have longstanding degree programs in naturopathic medicine, which are supported by government-funded placements.

However, as the profession remains largely unregulated or self-regulated in the region (although training exists that is within the highest quartile internationally), several unaccredited colleges still exist that do not meet WHO (or WNF) standards for naturopathic education. As such, the movement towards statutory regulation and recognition remains a priority area within the region. There is also a movement to enact regulations that allow growth in the naturopathic profession. For example, while the New Zealand naturopathic profession training institutions enjoy government funding and degree minimums, they are currently banned from adding an additional fourth year to their program by legislation. However, as in Australia, despite such barriers, the New Zealand profession remains committed to increased standards, including an active research agenda, and coordinates with international profession in research initiatives such as the International Research Consortium of Naturopathic Academic Clinics (IRCNAAC) – a North-American and Australasian collaboration leveraging clinical data from college training clinics. Research has more generally been one of the key accomplishments of the naturopathic profession in the WPRO region, with Australian (particularly) and New Zealand practitioners punching well above their weight in terms of research output both internationally with respect to the research output of the broader naturopathic community, and nationally when compared to other complementary therapy disciplines (such as chiropractic, osteopathy, or Chinese medicine) within their own nations.

While several challenges exist, the naturopathic profession in WPRO has numerous opportunities: relatively few practice restrictions, a developing education sector (including recent legislative changes that have banned non-degree education in naturopathic medicine), popular support among the general population, and a naturopathic profession that is holding its own in the international health research community. However, much of this success has occurred *despite*, rather than *because of*, government recognition and assistance, and the fragmented nature of the profession has often hampered efforts for greater recognition and integration. Developments such as the WNF have helped to serve as a catalyst for greater cooperation – both within and between countries in the region – and will hopefully provide the professional voice and leadership the profession requires to fully realize its potential in the region. ▀

Additional figure and references 7-20 available online at ndnr.com

Figure 3. *Nature Cure and Medical Freedom*; 1925



This was the first issue of the first local Australian (and WPRO) naturopathic journal, with "What is Naturopathy" as the lead article.

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Medical Resources for NDs

A review of current publications for the naturopathic industry



ALETHEA FLEMING, ND

Herbal Formularies For Health Professionals, Volume 1: Digestion and Elimination

After reading this first volume from Dr Jill Stansbury, my immediate reaction was that I can't wait for the remaining 4 in her series. Herbal textbooks are not widely available, to begin with, and this series promises to be a cornerstone not only for students, but also for working professionals who rely on clinical recommendations when treating patients.

Dr Stansbury's many years of teaching come through clearly in the structure and style of her writing. This book is designed to be useful to a wide audience, not only as a quick reference, but also as a teaching tool to help those who are less familiar with herbal medicine, helping them to understand the basic concepts of how and why to create a specific formula. Chapter 1 is about the art of herbal formulation, and it provides an excellent guide or refresher on the fundamentals.

The illustrative use of a triangle model allows for simplicity as well as a tidy

explanation of how to expand upon it. She recommends first selecting an appropriate base herb upon which to build a formula – an herb that is nourishing, tonifying, and safe for long-term use. Next, she suggests selecting an herb that is specific to the presentation of the patient and their illness, and then selecting a third herb that serves as a synergist for the formula and which may also address other conditions or energetic considerations, such as a tendency to cold/damp or dry/heat. Simple, but clear. One of the goals of this volume (and the series) is to make herbal formulation accessible to a wider audience of health professionals. Dr Stansbury's easy-to-follow structure gives this book an excellent chance of extending beyond an ND-only audience.

Patients of all ages are considered with care in her formulations; recommendations for infants, as well as for conditions commonly seen in our geriatric population, are included. Although this book is not an exhaustive exploration of all possible herbal formulae in the digestion and elimination systems, it serves as an excellent starting point with clear examples, and it provides the skill-set for going much further. If this were a recipe

book for cookies, the reader would learn how to make simple chocolate chip, sugar, and oatmeal cookies, but would also gain the confidence and inspiration to dig deeper and try a cocoa nib shortbread or a lacey almond wafer.

The formulae themselves are broad as well, encompassing different forms of herbal treatment. Dr Stansbury recommends imbibing teas, rather than pills or even tinctures, for more direct application in the case of some gastrointestinal and urinary issues or when cost is a big concern. Traditional tincture formulae abound, but with enough encouragement of variation that even a simply stocked dispensary lends itself to the creation of some of her suggested options. Pleasingly, she also includes suggestions for topical applications such as salves or lotions, as well as oral lozenges and medicinal vinegars. A wide variety of treatment options ensures that the practitioner can successfully meet the needs of the individual patient.

Early in their clinical training, all naturopathic medicine students learn the saying "When in doubt, treat the liver." This first volume focuses on the organs of digestion and elimination, supporting

the vital concept – found in most systems of medicine – that a patient's ability to detoxify well is a critical component of health. It is thus fitting that this is the focus of Dr Stansbury's first volume; subsequent volumes will build upon this base of knowledge and health. Particularly helpful is the chapter on skin issues, as herbal treatment of dermatological conditions is generally discussed much less in other books compared to liver, gastrointestinal, and urinary health issues.

Although grouped under different disease conditions, Dr Stansbury's formulae are specific to individual presentations, and she emphasizes the importance of treating the individual, not just the diagnosis. It's delightful to see a clear embodiment of the tenets of naturopathic medicine throughout this book. ▀

Just the **FACTS**

Title: Herbal Formularies for Health Professionals, Volume 1: Digestion and Elimination, Including the Gastrointestinal System, Liver and Gallbladder, Urinary System, and the Skin

Author: Jill Stansbury, ND

Publisher: Chelsea Green Publishing

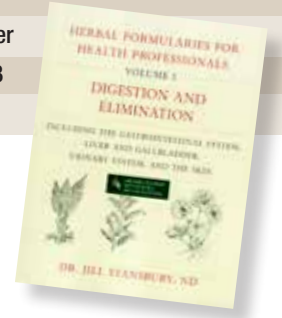
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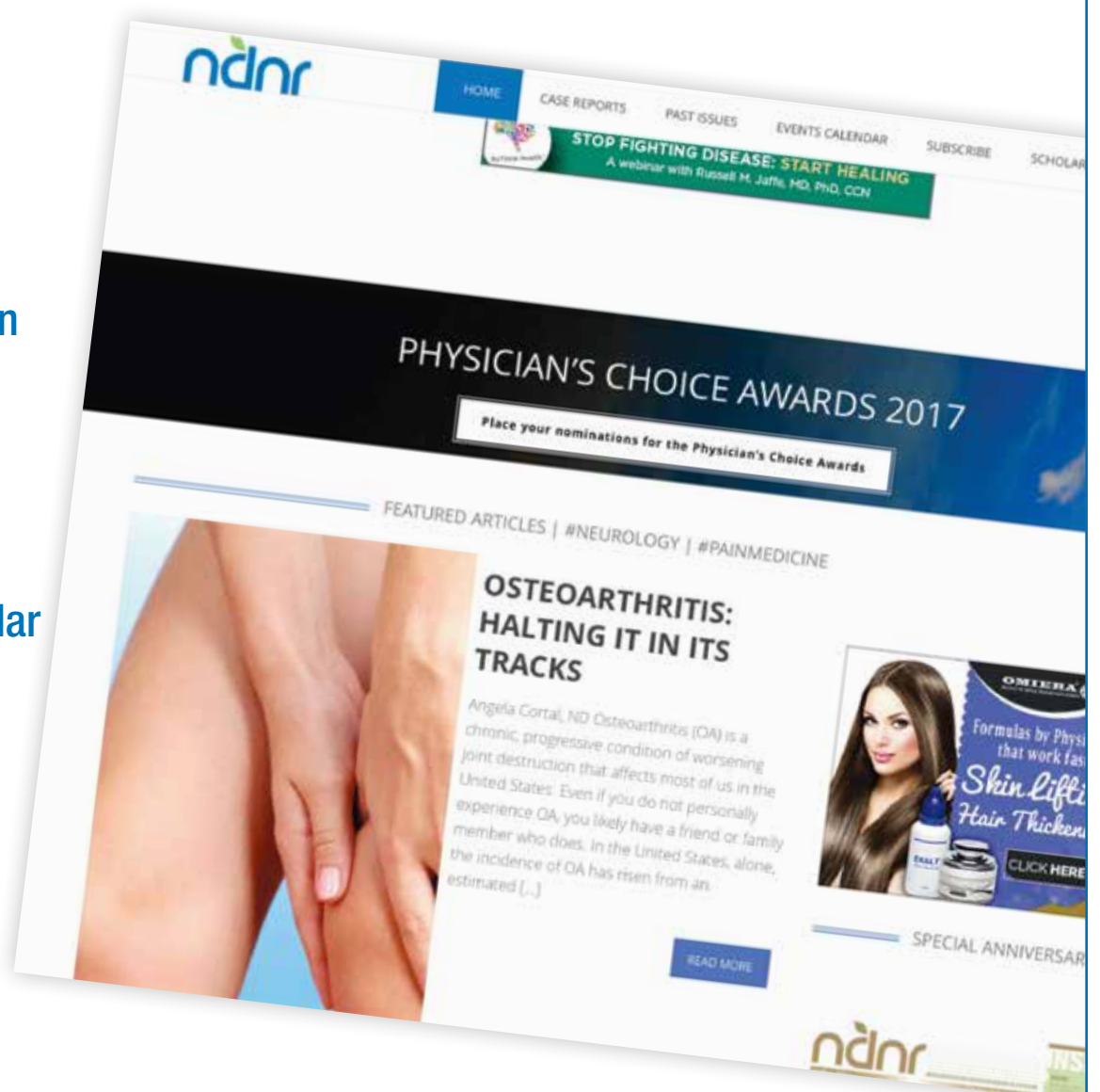


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Diverticulitis

Naturopathic Care

MONA MORSTEIN, ND, DHANP

Diverticular disease is the fifth most important gastrointestinal condition in Western countries.¹

Diverticulosis is a condition featuring small herniated pouches, generally around 5-10 mm, in the colonic mucosal layer. They can occur deep within the mucosa and submucosal tissue where blood vessels lie. Diverticulosis usually occurs in the sigmoid colon, but diverticula can develop anywhere in the large intestine.

Diverticulosis occurs in around 5% of people under 40 years of age, but this number climbs to 65% by age 85.¹ However common diverticulosis may be, 80-85% of diverticulosis cases remain asymptomatic; only 5% of patients tend to develop diverticulitis (inflammation of diverticula), and around 10-15% of these patients will suffer complications requiring surgical intervention.¹

Etiology

There are several etiological factors related to the development of diverticular disease (DD):

1. Abnormal slowing of colonic motility, excessive colonic contractibility, and either normal or increased resting intracolonic pressure
2. Decreased colonic tensile wall strength, ie, reduced strength of collagen and muscle fibers due to cross-linking of abnormal collagen fibrils; this weakens the colon wall, allowing the pouches to develop more easily
3. Chronic mucosal low-grade inflammation
4. Imbalance in the colonic microbiome
5. Visceral hypersensitivity, ie, excessive perception of physiological stimuli, such as colonic distention

DD is seen less in vegetarians, and more in those who eat a low-fiber diet and higher meat/fat diet. It is seen more in those who struggle with chronic constipation, and in patients with Ehlers-Danlos syndrome or Marfan syndrome. Breaking from a long-held mistaken dietary view, nuts and seeds do not cause DD and do not need to be avoided by those who have had an episode of diverticulitis.²

Diverticulitis is inflammation of 1 or more diverticula. Diverticulitis may result from fecal matter or undigested food particles becoming stuck in the diverticula, causing swelling, vascular compromise, and possible perforation. It may also be caused by intraluminal pressure or thickened food particles that cause erosion of the diverticular wall, leading to inflammation, focal necrosis, and possible perforation.

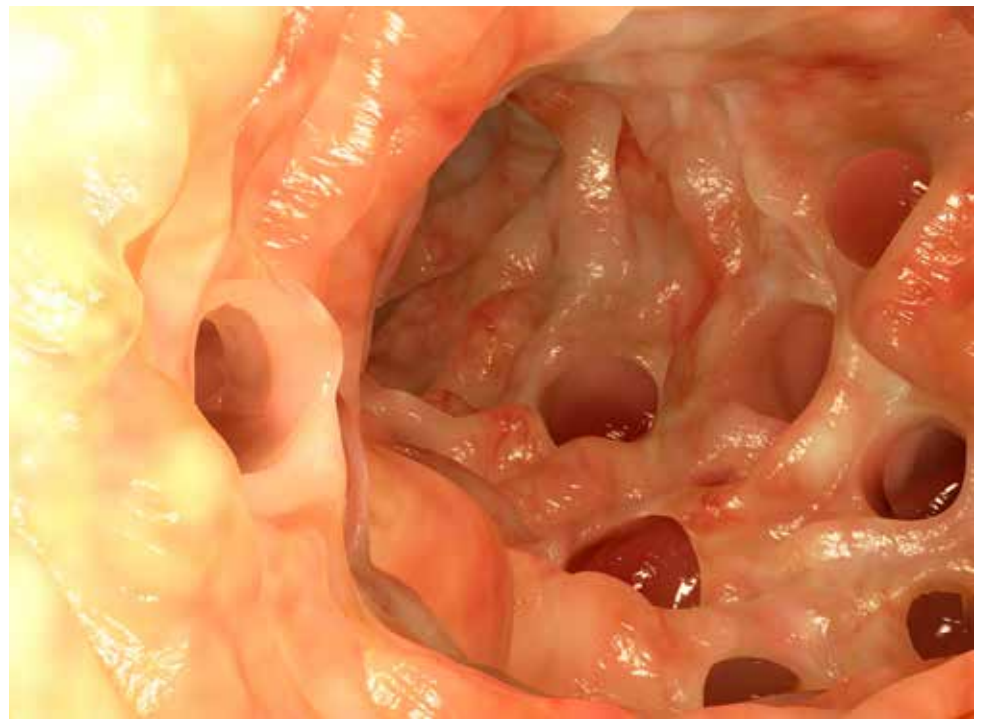
Diverticulitis (DS) is more prevalent in Western countries, particularly in individuals over 60 years old; obese individuals are also at higher risk. A younger patient (less than 45 years old) who develops DS is more likely to have complications and require surgery.¹

In Caucasians, left diverticulitis with fistulation is most common. Asians experience more right-sided DS, and a greater tendency to bleed.¹

Clinical Presentation

There are several categories of diverticular disease:

- Diverticulosis (asymptomatic)
- Diverticulitis (symptomatic):
 - Simple DS features inflammation without complications, and is readily controlled through conservative measures, such as pain relief, antibiotics, and bowel rest. A naturopathic physician can feel comfortable treating a patient with simple diverticulitis.
 - Complicated DS can feature perforation, peritonitis, fistula,



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obstruction, bleeding, abscess, and phlegmon. Fistulas can develop between the colon and the vagina, urinary tract, or the skin. A naturopathic physician should refer this patient to the ER.

Diverticulitis can become recurrent, either as a low-grade manifestation or as actual repetitive and overt clinical appearances. After the first occurrence of acute DS, managed without surgery, the 5-year recurrence rate is 20-50%. Of those that do recur, 25% will develop complications, 1-2% will require hospitalization, and 0.5% will require surgery.¹

Seventy percent of patients with acute diverticulitis will present with left lower-quadrant (LLQ) pain.¹ If the patient is Asian or actually has appendicitis, it may be RLQ pain. The pain is generally worse from eating and may be better with defecation or flatulence. There may be nausea and vomiting, though in my experience this is not common. DS patients can have constipation or diarrhea, and the pain often spreads out over the entire transverse area of the colon.

As with many gastrointestinal conditions, the "red flag" is fever. If a patient presents with fever, a referral to the ER is required, as a colonic perforation has likely occurred. If no fever, a naturopathic physician can feel safe and responsible treating a DS patient.

Workup

The differential diagnosis in the gut is extensive: Acute appendicitis,

pyelonephritis, OB/GYN conditions (eg, pelvic inflammatory disease, ovarian cysts, ectopic pregnancy), colorectal carcinoma, inflammatory bowel disease (IBD), colitis, pancreatitis, urinary tract infection, viral gastroenteritis or food poisoning, and irritable bowel syndrome.

In the office visit, conduct a thorough intake, take vitals, and do a physical exam. A CBC (perhaps STAT) is indicated; it's also good to check liver enzymes, and pancreatic amylase and lipase. You should do an in-office pregnancy check in female patients (if sensible), and a urinalysis. During the physical exam, do a good in-depth abdominal exam; complications requiring referral to the ER may present as abdominal distention, decreased/absent bowel sounds, rebound pain and guarding, or abdominal mass.

The main imaging lab for diagnosis is a CT scan. A double-contrast barium enema is also a useful tool, but is used less frequently than CT scans. If this was a patient's first DS episode, it is helpful after treatment to get a colonoscopy in order to ascertain the number and size of existing diverticular pouches.

As noted, fever is an instant ER referral. Also consider a referral when the patient cannot handle oral hydration, when the patient does not significantly improve with naturopathic care within 2 days, if the patient is immunocompromised or has significant comorbidities, or when the patient has severe enough pain that narcotic analgesia seems necessary (or, think homeopathy!).

Treatment

Conventional Approach

Conventional care of uncomplicated DS consists of a clear liquid diet with antibiotics; however, antibiotics may also be selected on a case-by-case basis.¹ Commonly used antibiotics include: ciprofloxacin with metronidazole; trimethoprim-sulfamethoxazole with metronidazole; moxifloxacin; or amoxicillin with clavulanic acid. Food can be reintroduced in 2-3 days as clinical improvement occurs. Mesalazine has been shown to improve symptoms and prevent recurrence of diverticulitis.³

Naturopathic Approach

Naturopathic medicine can be enormously effective in treating uncomplicated diverticulitis. Treating the acute condition is fairly easily done in most patients, and then long-term prevention can also be comprehensively established.

Naturopathic physicians do not have to use antibiotics to treat acute DS cases. At least 2 studies^{4,5} shows that antibiotic treatment can be avoided in simple, non-complicated diverticulitis and that outpatient management is safe.

Naturopathic medical testing for DS should include a 7-day diet diary, IgG Subsets 1-4 food sensitivity testing, salivary cortisol testing, and a comprehensive stool and digestive analysis (CSDA). Food sensitivities can promote inflammation in the gut lining, leading to recurrent DS. High or low cortisol can cause intestinal inflammation; high cortisol may be associated with gastrointestinal

perforation,⁶ and promote inflammation in IBD,⁶ and low cortisol does not trigger the brain to reduce the overall body inflammatory response.⁷ A CSDA can reveal challenges to the microbiome, dysbiosis, and uncover any short-chain fatty acid deficiencies that compromise the health of colonic cells.

The most dangerous medication for DS patients is non-steroidal anti-inflammatory drugs (NSAIDs).⁸ These drugs are devastatingly damaging to the entire gastrointestinal tract. I easily found a key study stating that NSAIDs are associated with a higher risk of DD. NSAIDs are more strongly associated with causing complicated DS than uncomplicated DS.⁸ NSAIDs cause a greater occurrence of bleeding with DS. The use of aspirin for 4-6 days a week significantly increases the risk of DS; even daily low-dose aspirin will increase DS risk.⁸ NSAIDs have been found to cause intestinal permeability within 1 hour of ingestion and to cause inflammatory changes throughout the intestinal tract, although aspirin may be less problematic than other NSAIDs.⁹

Alcohol should also be avoided. Alcohol, in and of itself, can significantly increase the risk of diverticulosis.¹⁰ I have had an adult male patient, an oenophile, whose recurrent diverticulitis episodes (4-6 per year) ceased when he stopped drinking his beloved wine.

For an acute episode, have the patient rest at home and only drink water, tea or broths. Consider using a non-tincture, comprehensive antimicrobial product. Dose it aggressively – hourly for the



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first 1-2 days – and then reduce the dose to 5-6/day until the patient has entirely recovered. If such a product is not used, antimicrobials and immune stimulators include high-dose vitamin A drops, liposomal or regular vitamin C, vitamin D3 (in thousands of IUs per body weight), *Echinacea/Hydrastis canadensis* (goldenseal) capsules, and anti-inflammatory products such as *Curcuma longa* (curcumin), *Boswellia*, and bromelain. Do a homeopathic case; look closely at Bryonia, which comes up often in my practice.

Add in demulcents to sooth and help heal the tissues:

1. Slippery elm gruel: 1.5 tsp to 1-2 cups of water: Take 3 times daily
2. Mixed gut-soothing formulas (eg, L-glutamine, N-acetyl-glucosamine, citrus pectin, deglycyrrhizinated licorice [DGL], and other soothing herbs): Take 1 tbs 2 times daily
3. N-acetyl-glucosamine (NAG), 700 mg: Take 2 capsules 3 times daily (This is an under-used nutraceutical for gut healing)
4. Fish oils: 2000-4000 mg EPA per day
5. Probiotics: \geq 100 billion CFU/day

Recommend that the patient apply castor oil packs over the lower inflamed colonic tissue – 45 minutes in the morning and evening.

I've never had these therapies fail in acute and preventive diverticular disease.

Case Study 1

A 62-year-old woman presented with acute LLQ pain. She had a past medical history of diverticulitis 2 years prior.

She experienced acute pain in response to touch or movement; however, all vitals were WNL, and she had no fever or other signs of complications. PE revealed abdominal pain in the LLQ and across the transverse colon. Blood was drawn for a CBC, which was shown the next day to be WNL.

The patient was sensitive to gluten but was eating it regularly. She was also eating a lot of refined sugar. She also had a stressful work schedule, seeing students 10 hours per day for 5 hours Monday through Friday, and for 5 hours on Saturday for piano lessons.

I told her to stop the piano lessons, not eat, drink only water, tea, and broths, and to rest.

Using the above protocol, she responded within a day, and by 4 days later the diverticulitis was gone. A cortisol test had shown elevated cortisol at 2 points during the day, so we discussed getting her back to swimming, which she loves and does well, working less, and doing some meditation.

It took her another year to commit to avoiding gluten and refined sugar, and she had some recurrent diverticulitis due to her problematic eating, but it was all controlled with naturopathic care.

Case Study 2

A 67-year-old man presented with recurrent diverticulitis – 2-3 times per year for the last 2 years. It presented as LLQ pain. He had been on recurrent antibiotics. He also took ibuprofen or naproxen daily for osteoarthritis. I had him do a diet diary, and I ordered food sensitivity testing, a cortisol test, and a CSDA.

He was sensitive to dairy and ate a very pro-inflammatory diet, including

high dairy intake, 1-2 glasses of alcohol a day, low vegetable intake, daily sugar in desserts, and rare omega-3 food intake. The stool analysis showed low beneficial bacteria but was otherwise WNL. His cortisol was low at 1 point during the day. We discussed many significant dietary changes.

I prescribed a probiotic (100 billion CFU per day); a gut-soothing formula containing L-glutamine, NAG, citrus pectin, DGL, and other herbs (1 tbs twice daily); slippery elm gruel; NAG (700 mg); a multiple vitamin/mineral; and fish oils. He was also told to stop the NSAIDs.

Around 2.5 months later, the patient had no more colonic or arthritic pain. He noticed that drinking alcohol while socializing would cause a small flare-up of pain, as did the NSAIDs when he took them.

After a few months, we reduced his supplements to a multiple vitamin/mineral; fish oils; slippery elm once a day; and the mixed formula to once a day. He has done well since. ▀

References 8-10 available online at ndnr.com



Mona Morstein, ND, DHANP, is considered an expert in both gastroenterology and hormonal conditions, especially diabetes. Dr Morstein taught gastroenterology for 11 years at a naturopathic medical school. She is a frequent lecturer on webinars and at conferences, including the first and second SIBO SOS™. She has numerous lectures archived at MedicineTalkPro.org. Her book, *Master Your Diabetes: A Comprehensive, Integrative Approach for Both Type 1 and Type 2 Diabetes* is a highly regarded publication for both the diabetic patient and medical practitioners. A senior vitalist in the Naturopathic Medicine Institute, she practices in Tempe, AZ, at Arizona Integrative Medical Solutions: www.drmmorstein.com.

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Traumatic Brain Injury

Clinical Applications & Plausible Interventions

CORA STOVER, ND
DIANA ZITSERMAN, ND
RADLEY M. RAMDHAN

Traumatic Brain Injury, or TBI, has been noted in history, literature, and medical societies for quite some time. Boericke noted it in one of his “*Never well since...*” rubrics, along with other rubrics. In 1924, Cassasa was recognized as the first to describe the phenomenon and what repeated traumatic injuries might do to a person, and coined the term *dementia pugillistica* (or DP). Cassasa observed that boxers who were being used for training purposes would eventually display a particular pattern of symptoms.¹

These symptoms first appeared in the extremities, as a slight flopping of the foot or leg, only noticeable upon walking or at intervals, or as a slight unsteadiness in gait or equilibrium. A peculiarity was the fact that these fighters could fight exceptionally well, but as soon as they stopped or walked back to their corner, the abnormality became apparent. Further symptomology included confusion and muscular slowing. Other terms historically associated with this phenomenon, which were generally applied to an intoxicated person, included: Punch Drunk, Cuck-coo, Goofy, cutting paper dolls, and slug nutty.¹ Cartoons often served to depict the syndrome; some famous sayings and characters were based on this association.

Chronic Traumatic Encephalopathy

Dr Bennet Omalu is world-renowned physician and neuroscientist who has been investigating the effects of repetitive brain injuries in professional sports players. It was a Professor Henry Miller who in 1966 coined the term Chronic Traumatic Encephalopathy (CTE) for the progressive neurodegenerative syndrome due to repetitive concussions and/or traumatic brain injuries.² Omalu was the first to publish evidence of the syndrome in 2005.³ CTE is 1.5 times more likely to be seen in males than females.⁴ First discovered in the brain of NFL Hall-of-Famer, Mike Webster, in 2002, CTE was subsequently identified in NFL players, wrestlers, hockey players, deceased military veterans, domestic abuse victims, motor vehicle accident sufferers, and others.⁵ Bennet Omalu’s story was portrayed in the 2015 cinematic hit, *Concussion*, and his growing foundation and published work has continued to spread throughout the scientific and medical community.

CTE is a distinctive disease, and often a sequela to TBI. Clinically, CTE presents as a combined syndrome featuring mood disorders, behavioral and/or cognitive impairment, and occasional sensorimotor involvement. CTE is caused by “single, episodic, or repetitive blunt force trauma to the head, with repeated acceleration-deceleration forces to the brain.”⁵ A CTE patient’s brain appears largely unremarkable; however, microscopic examination of post-mortem brains reveals primary and secondary proteinopathies. Dr Omalu found that the primary proteinopathy of CTE is tau protein-related, whereas secondary proteinopathies include amyloid plaque and other abnormalities. Dr Omalu and his team have pioneered the study of CTE-related proteinopathies by developing an in-vivo PET scan named FDDNP-PET, which measures tau tangles and amyloid plaque deposition in living tissue.⁵

TBI is often associated with a breached blood-brain barrier.

Gross Morphology & Pathophysiology

Areas within the brain that are thought to be damaged in CTE include: the corpora striata (part of the basal ganglia comprising the caudate nucleus [which contains endorphins] and the lentiform nucleus, which when probed produce sensations of thirst; its function is essentially motor); the corona radiata (a sheet of white matter that runs ventrally as the internal capsule and dorsally as the semioval center – the corona radiata is responsible for most of the neural traffic to and from the cerebral cortex); the cerebral cortex (gray matter, which plays an important role in consciousness, thought and action; it includes 4 lobes: frontal, parietal, occipital, and temporal) or below the tentorium cerebelli (extension of the dura mater that separates the cerebellum from the occipital lobes).⁶

The pathology of DP and CTE are thought to involve hemorrhages that are later replaced by gliosis, a degenerative progressive lesion. In 1924, Cassasa described these areas like this:

At autopsy, sections of the brain showed multiple usually punctate hemorrhages scattered over various parts of the parenchyma of the brain.

Lacerations of the scalp, fractures of the skull, cortical lacerations or hemorrhages – except for occasional pia-arachnoid hemorrhages – were not found. Microscopic examination showed these punctate hemorrhages to be located around the blood vessels in the perivascular spaces of Virchow-Robin [an immunological space around an artery and vein – not capillary, and pia mater that may be expanded by leukocytes].¹

Clinical Manifestation, Diagnosis & Testing

Diagnosing brain injury can be extensive and expensive. Two scales which should be used in any case of brain injury include the Glasgow Coma Scale and the Ranchos Los Amigos Scale. Imaging, including angiography, computed tomography (CT), magnetic resonance imaging (MRI), and X-rays, should be utilized to observe patterns of brain injury due to external and internal forces

or other possible traumas. Functional imaging, such as electroencephalography (EEG) and transcranial doppler, may be utilized to observe communication and neural patterns. Because biofeedback can pick up changes in autonomic nervous system function that can occur during inflammation and encephalopathy, it might assist in our understanding and treatment of TBI/CTE.^{7,8}

Clinical manifestation of TBI sequelae may include the signs and symptoms shown in Table 1.

In addition, signs and symptoms in young children may include persistent crying, inability to be consoled, listlessness, refusal to nurse/eat, irritability, and behavior changes.⁹

Suggested biomarkers include a CSF:serum albumin ratio (which can reveal blood-brain barrier dysfunction); neuro-inflammatory cytokines such as interleukin (IL)-6, IL-8 and IL-10 (typically increased after severe TBI); and other acute-phase response proteins.¹⁰ Acute axonal injury biomarkers include total tau and neurofilament light polypeptide (NFL), which tend to increase 4-10 days after injury; these 2 tests are considered most accurate and to correlate with injury.

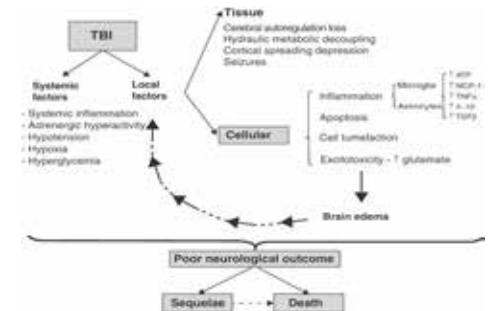
Myelin basic protein (measured in blood) is also associated with TBI. Another promising marker for acute neuronal damage is CSF measurement of γ -enolase, also known as neuron-specific enolase (NSE); this is a glycolytic enzyme in neuronal cell bodies and is also present in erythrocytes and endocrine cells. Calpain and caspase (breakdown products of neuronal spectrin α chain [α II spectrin]) tend to increase in the CSF during acute-phase brain injuries.¹⁰ Serum S-100 β may be a useful marker of astrocyte injury or death and correlates with the Glasgow Coma Scale; however, it is not useful in children under 2 years of age.¹¹

Pathogenesis of Neuroinflammation

In order to understand CTE, it is helpful to first explore the pathogenesis of TBI-related neuroinflammation. TBI is generalized into 4 categories, as follows: “1) primary injury that disrupts brain tissues; 2) secondary injury that causes pathophysiology in the brain; 3) inflammatory response that adds to neurodegeneration; and 4) repair-regeneration [of neurons].”¹² A primary injury to the brain initiates the secondary injury, or the cascade of multiple molecular events that lead to neurodegeneration.

In the first hours after TBI, in cases of focal injury, damaged cells are found in clusters. In cases of diffuse injury, these changes are more diffuse in the brain. Sites of injury contain both necrotized neurons and non-neuronal cells, and hemorrhagic areas can become intraparenchymal hematomas in cases of more extensive injury.¹³ High ventricular intracranial pressure after TBI can indicate a poor outcome.¹² Varying degrees of edema and cellular swelling can also be present, and a broad, asymmetrical distribution of axon swelling can indicate diffuse axonal damage.¹³ TBI and cellular responses are

Figure 1. TBI & Cellular Effects on Brain Tissue¹³



(Image courtesy of Rovegno et al, 2012)

outlined in Figure 1.

Secondary mediators are known to be tissue-destructive, involving inflammation and apoptosis.¹⁴ TBI is often associated with a breached blood-brain barrier (BBB), which can be accompanied by endothelial activation and recruitment of white blood cells, as well as microglial activation and migration toward the injury. This can result from a number of mechanisms, such as chemoattractant protein-1 (MCP-1) and cytokines, eg, transforming growth factor-beta (TGF β). Release of tumor necrosis factor-alpha (TNF α) and IL-1 β in turn promote increased expression of an intermediate filament called glial fibrillary acidic protein (GFAP). Leukocytes and activated cells release proinflammatory cytokines, nitric oxide, and free oxygen radicals, which introduces phenotypic changes called “neuroplastic remodeling,” in which increased branching of dendrites occur to

Table 1. Possible Clinical Sequelae of TBI^{6,7,8}

Unconsciousness (left side injury lasts longer than right side)	Behavioral/mood changes (display of pathological jealousy/paranoia)	Difficulty with memory, concentration, attention, thinking
Dilation of one or both pupils (anisocoria)	Difficulty with processing speed and executive functioning	Cushing’s Triad: Depressed HR, irregular respirations, and increased BP
Alexithymia (inability to identify, understand, process, and describe emotions)	Lack of motor coordination/balance	Nausea/vomiting
Blurred vision/double vision	ringing in ears	Confusion
Bad taste in mouth	Fatigue/lethargy	Headache
Convulsions	Dizziness	Sleep pattern changes
Slurred speech	Aphasia, dysarthria	Weakness/numbness in limbs
Social judgment deficits	Concussion, post-concussion syndrome	

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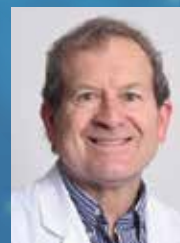
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form new synaptic connections.¹³

Other common features include intracellular increases in free calcium, disturbances in ion concentration (eg, increased potassium or decreased magnesium), acetylcholine deficiency (particularly in chronic cases), dynorphin (an endogenous opioid receptor correlated with regional neurodegeneration), nitric oxide (induced by ischemic damage), and calcium-dependent cysteine proteases including calpain and caspase-3 (which are contributors to cell death following TBI).^{12,15} In more severe TBI, astrocytosis can lead to tissue scarring, impairing structure and function of the brain areas involved in the trauma.¹³

Another key player is the excitotoxicity and spread of apoptosis endured by the traumatized tissues. NMDA receptor activation is responsible for excitatory signals. In fact, it has been documented that TBI-induced glutamate excitotoxicity is the initiating signal for apoptosis of damaged cell membranes. Increased extracellular glutamate levels activate glutamate receptors, leading to intracellular calcium overload, which stimulates mitochondrial oxidative stress and activation of the caspase-dependent and independent apoptosis, ie, delayed cell death.¹⁵ Other studies also suggest that a heterodimer formation of pro-apoptotic transcription factors, such as c-Fos and c-Jun, can regulate the expression of genes coding for opioid peptides, amyloid beta protein, and nerve growth factor, which have been shown to be overexpressed in TBI.¹²

TBI and CTE appear to represent a

progressive neurodegenerative state in which tau and amyloid beta proteins accumulate within the tissues over time. These molecular mechanisms have been shown to be initiated by the extrasynaptic NMDA activation that produces soluble amyloid-beta, and caspase-3 activation that leads to formation of tau neurofibrillary tangles.¹⁵ As we know, amyloid proteinopathy is associated with Alzheimer's disease (AD), and tau-related neurofibrillary tangles are associated with Parkinson's disease (PD). Therefore, CTE post-TBI can be a concomitant condition with dementia, AD, and PD.

Treatment of TBI

Treatment of TBI varies, based on its severity. These range from simple measures such as rest in mild TBI cases, to surgery in more severe cases. Additional acute and chronic treatments exist, as well as long-term rehabilitation options. Initial acute treatments for TBI may include clearing the lungs to aid breathing, maintaining healthy circulation to support and protect the brain and other vital organs, monitoring heart rate, medicating the patient to reduce risk of secondary damage, and removing any blood clots that may result in the patient's life being compromised. Chronic treatment and long-term rehabilitation measures can include assistive technology, counseling and/or therapy, medication, physical therapy, or speech therapy.⁷

Allopathic Approach

Management of a sports-related concussion often includes cognitive rest, physical

rest, medication, and a gradual transition back into playing.⁷ Cognitive rest involves avoidance of activities that require extra attention, such as text messaging, video games, television, computer use, or school work. Physical rest is encouraged to avoid exacerbation of symptoms. NSAIDs, acetaminophen, or amitriptyline may be used for persistent headaches; sleep medications, anxiolytics, and SSRIs may be utilized for depressive symptoms. Transition back to play is also an important part of the process, where it is a step-by-step process from non-impact to full contact over a period of days, with the patient being in a symptom-free state.⁷

Naturopathic Approach

Our focus as naturopathic doctors should be on treating with natural anti-inflammatories, adaptogens, neuroprotective and neuro-regenerative agents, and analgesics. This can be accomplished through herbs, diet, nutraceuticals, homeopathy, and other modalities. Treatments can also be approached with different strategies in mind. These may include reducing the short- and long-term impacts of inflammation on brain tissues; supporting regrowth of brain tissues and optimal neuronal function; managing peripheral symptoms such as tremor, palsy, or neuropathy; optimizing circulation to the brain to reduce inflammation and promote regeneration (especially in cases involving hemorrhage), and managing any associated neuropsychiatric symptoms.

The short- and long-term impacts of inflammation on the brain can be reduced by plant flavonoids such as anthocyanidins from blueberry (*Vaccinium macrocarpon*), glycosylated flavonoids such as ginkgo (*Ginkgo biloba*) flavone glycosides, curcumin (*Curcuma longa*), boswellia (*Boswellia serata*), and omega-3 essential fatty acids. Oats (*Avena sativa*), rich in calmodulin and phosphatidylinositol, can be used to improve nerve function and promote regrowth of the brain tissues. Mullein (*Verbascum thapsus*) contains piscicide, which has analgesic effects. Valerian (*Valeriana officinalis*) has anticonvulsant effects, and *Huperzia serrata* has nootropic and neuroprotective effects and has been seen to potentiate the effects of acetylcholine. These are all herbs that can be used internally to help manage any peripheral symptoms.¹⁶

External application of St John's wort (*Hypericum perforatum*) or chili pepper (*Piper frutescens*) have both demonstrated effectiveness in treating neuropathy. Herbs such as hawthorn (*Crataegus* spp), rosemary (*Rosmarinus officinalis*), linden (*Tilia* spp) and ginger (*Zingiber officinale*) can be utilized to optimize circulation, helping to reduce inflammation and promote regeneration. Rhodiola (*R. rosea*) and ashwagandha (*Withania somnifera*) may be utilized for their adaptogenic properties as well as their ability to support the brain in the case of associated neuropsychiatric symptoms. St John's wort can also be used for its antidepressant effects. Skullcap (*Scutellaria lateriflora*), lemon balm (*Melissa officinalis*), kava kava (*Piper methysticum*), and *Bacopa monnieri* are all herbs that can be utilized as nervines and to manage any associated neuropsychiatric symptoms.¹⁶

Homeopathic remedies that can be useful for TBI include Arnica, Belladonna, Hypericum, Cicuta, Natrum sulphuricum,

and Helleborus. Arnica is useful in those who deny any problems following the trauma. Belladonna is useful when there is significant heat, redness, throbbing, and fullness in the head after an injury. Hypericum can be effective in patients with sharp and shooting pains, spasms, or seizures after a head injury. Cicuta is useful in cases where seizures occur following the head injury, or the head injury is severe enough to have caused mental retardation. Nat Sulph is useful for long-term symptoms following a head injury that includes personality changes. Helleborus is useful in a patient that develops severe mental dullness and may talk very slowly after the head injury.¹⁷

Hyperbaric Oxygen Therapy (HBOT) is also a useful treatment for TBI. HBOT involves the medical therapeutic use of oxygen at a high atmospheric pressure. It can be used for both acute and chronic TBI patients; however, some studies have revealed that it has been more effective in the chronic TBI patient groups.^{18,19}

Historically, the Roman Army utilized herbs as part of their treatment plan for soldiers with TBI. Tea made from *Glaucium corniculatum* was sometimes used because it acts as an acetylcholinesterase inhibitor.²⁰ A decoction of the roots of *Asparagus officinalis* was used to reduce edema. A mixture of acetum (vinegar) and the juice of the plant *Glaucium flavum* was used to clean the wound area; this was followed by a dressing containing a mixture of honey, lint, and *Aloe vera*. Diet and hydrotherapy also played roles in the treatment plans. In more severe cases of TBI, *Papaver somniferum* or *Mandragora officinarum* was used for analgesia. In patients with weaker constitutions, *Hyoscyamus niger* combined with *Papaver somniferum* was sometimes used as an anesthetic.²⁰

In addition to support with herbs, nutrition, homeopathy and hyperbaric oxygen therapy, it is important to support the natural healing process by ensuring that patients get ample rest and have a gradual phase of return to the sports that they were playing prior to their TBI. And, of course, it is vital to initially take a proper case history and perform a comprehensive physical exam in order to gather the information that will help determine the best treatment plan. ▀

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Cora Stover, ND, is a doctor of Naturopathic Medicine who provides services to the New Haven and Fairfield counties and is bringing medicine back into the home via home-health. She enjoys working with a variety of patients and conditions. Her passions include laughing, gardening, learning, and being mystified by nature and our experiences.

Diana Zitserman, ND, is a licensed naturopathic physician and acupuncturist, specializing in Whole Person Wellness at Collaborative Natural Health Partners in Manchester, CT. After completing her studies at Pennsylvania State University, Dr Zitserman pursued a 7-year research career at Fox Chase Cancer Center, which included 8 journal publications. Furthering her medical studies in naturopathy and Traditional Chinese Medicine at the University of Bridgeport, she combined her love of science with an innate passion for understanding human interaction, nature, and alternative healing. Dr Z uses natural therapies to create balance and health restoration for people of all ages.



Radley M. Ramdhan is currently working toward a Doctorate of Naturopathic Medicine and a Master of Science in Acupuncture at the University of Bridgeport; he anticipates graduation in May 2018. Serving in the New York Army National Guard since 2012, Radley recently returned from a deployment to Kuwait/Iraq for Operation Inherent Resolve 2016/2017. Radley has a passion for and desire to work with veteran and pediatric populations. Writing, traveling, and playing sports are some of Radley's pleasures.



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Sleep-Disordered Breathing

An Under-recognized Cause of Chronic Disease – Part 2

DAVID NORTMAN, ND

Part 1 of this article covered the pathophysiology and clinical presentation of *obstructive sleep-apnea syndrome* (OSAS). Below, we continue with a description of the sleep study and an overview of treatment methods.

Laboratory Diagnosis

The primary tool for studying all sleep disorders is the *Level 1 sleep study*, or *polysomnography* (PSG), during which cerebral, respiratory, cardiovascular, positional, and other measures of sleep quality and behavior are recorded and interpreted during the subsequent scoring of the raw data. PSG reliably detects apneas, hypopneas, *respiratory effort-related arousals* (RERAs), and a wealth of other information relevant to OSAS and other sleep disorders. Like all tests, PSG is subject to inter-test variability, so a single night of testing can occasionally miss OSAS and may need to be repeated.^{1*} If a *continuous positive airway pressure* (CPAP) machine is prescribed, a follow-up titration PSG is often performed in order to establish the baseline flow-pressure that eliminates arousals; however, since the advent of automated CPAP machines, this step is sometimes omitted.

The severity of OSAS is commonly graded by the *apnea-hypopnea index* (AHI) – a score that reflects the night's average hourly number of apneas and hypopneas; <5 is considered normal, 5-15 mild, 15-30 moderate, and >30 severe. The *respiratory disturbance index* (RDI), introduced once *upper-airway resistance syndrome* (UARS) was recognized, incorporates (on the same severity scale) the impact of not only apneas and hypopneas, but also RERAs (ie, RDI = AHI + RERA). Although both AHI and RDI are in current use, the more standardized AHI remains popular with physicians (for guiding treatment) and insurance providers (for determining eligibility for treatment). Yet, just because detecting RERAs with sufficient sensitivity is technologically challenging, their impact should not be ignored, lest “non-OSA” OSAS be left untreated.**

The arousal index (AI) is a measure of sleep fragmentation as reflected in arousals from any cause, so it is up to the clinician to interpret the meaning of spontaneous arousals that are not secondary to respiratory or other detectable reasons. Some degree of sleep fragmentation is normal and is likely aggravated by the polysomnography setup, but it may also be reflective of chronic pain or other neurological or psychological causes. The AI is known to increase with age in normal subjects, but definitive normal values have not been established. In general, sleep data should be interpreted holistically, as the severity of sleep disturbance depends also on the clinical presentation and other factors such as severity of hypoxemia, length of arousal events, time spent in slow-wave sleep (SWS) and rapid-eye movement (REM) sleep, and frequency of arousals during these deeper stages of sleep.^{1***}

A home-based (typically Level 3)

sleep study is sometimes ordered in place of PSG for the sake of cost-savings and convenience. It can detect obvious apneas and hypopneas but may miss subtler airflow restrictions such as RERAs. Its main drawback is the lack of brainwave monitoring, which means that none of the findings can be correlated with the sleep stages, and sleep length (critical for calculating both AHI and RDI) can only be estimated. Thus, it is a suitable screening tool for those likely to have moderate-to-severe apnea, but it cannot be used to rule out OSAS, due to its limited accuracy and sensitivity.

Another test sometimes used in evaluating OSAS is the *multiple sleep latency test* (MSLT), which quantifies the degree of daytime sleepiness.

Treatment

OSAS is readily treatable through a variety of methods; however, there is no universal solution for all patients, and the most common therapies involve an adjustment period that limits compliance. CPAP therapy is the most popular approach and is usually the first and only one offered to patients. But it need not be the last one, nor does it or any other treatment need to be used on its own. A successful approach to OSAS may require multiple solutions, and, as naturopathic physicians, we are perfectly positioned to encourage patients to seek further treatment when the prescribed one has proven inadequate.

CPAP Therapy

CPAP was introduced in the early 1980s, revolutionizing the treatment of OSAS. Yet despite its unrivaled efficacy among non-surgical solutions, CPAP is poorly tolerated by many patients. Concerns include claustrophobia, airway dryness or congestion, increased risk of respiratory infection, mask leakage or pressure points, restriction of movement during sleep, tooth movement from mask pressure, noise, aerophagia, and, last but not least, interruption to the sleeping partner. The required equipment maintenance further decreases long-term compliance. Ultimately, most patients require weeks of acclimatization, and many never learn to tolerate the treatment.

CPAP comfort has improved over the years, thanks to better mask design, automatic pressure settings, built-in humidification and heating, and virtually silent operation. In recent years, automatic-pressure (Auto-CPAP/APAP) machines and bi-level (BiPAP/VPAP) machines, with independent exhalation and inhalation pressures, have become available. These have further improved patient comfort, but access is still limited by knowledge on the part of sleep specialists and by insurance considerations. Given its high efficacy when tolerated, CPAP users should be encouraged to receive proper training and follow-up, proactively seeking optimal settings, masks, and machine types until compliance is achieved.

Oral Appliance Therapy

Oral appliance therapy, typically in the form of a *mandibular advancement device*

(MAD), can be used to reposition the mandible anteriorly in order to expand the airway. Compact and convenient, it is compatible with drinking and speaking, is unobtrusive and much simpler to use than a CPAP machine, and can be implemented even without a sleep study. It is most effective in milder forms of OSAS and in patients with low body mass index (BMI). Although typically less effective than CPAP in reducing arousals, the higher compliance rate makes it a serious contender to CPAP. Oral appliance therapy can be a standalone treatment or a useful adjunct to CPAP therapy, as expanding the airway allows for lower CPAP pressure settings and a reduction in pressure-related side-effects such as mask leakage and airway dryness.

A MAD is custom-fitted by a dentist trained in sleep medicine. The adjustment period, during which the jaw is gradually protruded to the treatment position, may involve temporary temporomandibular joint (TMJ) discomfort. Tooth movement resulting in bite alteration commonly occurs in the long term, but this can usually be managed successfully, and in some cases may even be a desirable outcome. Do-it-yourself devices marketed for snoring or sleep apnea should be avoided, as they are usually ineffective and may cause harm in the long term.

Surgical

Surgical options for OSAS may be resorted to when other approaches have failed or not been complied with. [For a detailed overview of surgical options, you might check the book by Fairbanks et al.²] A popular soft-tissue surgery is *uvulopalatopharyngoplasty* (UPPP), although it has a high recurrence rate and is ultimately not very effective. *Maxillomandibular advancement* (MMA) is a radical but highly effective surgery that can be considered when all other options have been exhausted. A growing array of surgeries is available depending on the precise location of airway restriction, including septoplasty (deviated-septum correction), tonsillectomy, superficial implants to stabilize the soft palate, radiofrequency ablation of the base of the tongue, and bariatric surgery to address morbid obesity.

Positional


The simplest thing a patient can do to improve OSAS symptoms is to manage one's sleep position. Moderate neck hyperextension in the supine position, using a “contour” or “anti-snore” pillow or a suitable neck brace may offer relief. Encouraging the patient to side-sleep may be a partly effective solution to mitigate the effects of gravity on the airway, and various worn devices that discourage sleep



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in the supine position are available for this purpose. The effectiveness of side-sleeping should ideally be evaluated through PSG, especially if it is relied on exclusively. As an adjunct to CPAP treatment, positional therapy often allows for lower pressure settings and greater comfort.

Behavioral

Various behavioral therapies may be effective for addressing OSAS. First and foremost is weight loss. In overweight patients this is a reliable method of improving or resolving OSAS, although in some cases addressing OSAS first might be necessary.

Myofunctional therapy (retraining of oral and facial muscles), sometimes combined with a tongue-tie release, is a promising and still-evolving approach that is highly compatible with naturopathic principles.³ On the do-it-yourself front, nocturnal mouth breathing in adults and older children can be both diagnosed and addressed by taping the mouth with medical tape, ensuring first that the nasal passages are clear.

Pediatric Considerations

No age is immune from *sleep-disordered breathing* (SDB). OSAS is more common than supposed in children, and along with snoring and mouth breathing, should always be addressed promptly because of their considerable long-term impact on development and behavior.⁴ In infants, sudden infant death syndrome (SIDS) may be a consequence of undiagnosed SDB. Suffice it to say that infants with unreasonable sleep dysregulation or perplexing ailments should undergo a sleep study as part of their overall evaluation.

The following may be indicative of OSAS in children:

- Unusually restless sleep
- Behavioral issues (including ADD/ADHD and autism spectrum disorders)
- Decline in school performance
- Bedwetting
- Cardiovascular problems

Mouth breathing in childhood may affect craniofacial development and alter the shape of the airway at any point up to early adulthood, such that even a previously well-formed jaw may fail to complete its proper development if it goes unchecked for a long enough time. When enlarged tonsils or adenoids are to blame, their removal should be considered unless the issue can promptly be addressed by other means, as the negative immunological consequences of removal are far less significant than those of mouth breathing. Allergies should likewise be promptly addressed for the same reason. Habitual mouth breathing that arises after a cold, or persists after addressing some other underlying cause, can be addressed through behavioral retraining or specialized dental appliances.

There is evidence that, much like dental caries, dental malocclusion and palatal underdevelopment are diseases of civilization.⁵ The widespread substitution of breastfeeding with bottle feeding (whether with mother's milk or formula), and the subsequent consumption of pureed and soft foods that do not require chewing, deprive the jaw of the mechanical forces necessary for its proper development. Although these trends are reversing

somewhat, it remains important to inform parents of the benefits of maintaining a degree of continuity with ancestral child-rearing habits.

The growing skull of children offers further treatment options beyond those available to adults, namely orthodontic expansion of the palate and dental arches, with the aim of enlarging the upper airway. Orthotropics, an emerging subfield of pediatric orthodontics, aims to prevent both dental and airway issues by optimizing facial growth. On the other hand, outdated orthodontic methods that involve removing teeth in order to reduce crowding – a symptom of an underdeveloped arch – can further exacerbate an already restricted airway. Certain surgeries available to adults are not appropriate in children because of the likelihood of spontaneous resolution over time, and mandibular advancement is not usually applicable in a developing jaw.

Navigating the System

Polysomnography is a reliable and comprehensive diagnostic tool for OSAS, but due to its cost, access to PSG is tightly regulated. Family physicians and dentists continue to be poorly informed about the prevalence of OSAS and its atypical presentations, and dentists are generally not trained in detecting its telltale signs in the oral cavity. As naturopathic physicians, we should consider that untreated OSAS, even when mild, may be an unrelenting obstacle to the optimal health that we seek for our patients.

In this vein, sleep dentist Mark Burhenne has proposed that *sleep ability* be considered among the chief determinants of health, alongside such measures as blood pressure and BMI.⁶ [Burhenne's compact book is a useful initial resource about OSAS, both as a patient guide and as a basic resource for family physicians.] Until such a cultural shift takes place, we can play a valuable role in informing our patients about OSAS and helping them to navigate the system. It is also worth developing a proficiency in interpreting sleep reports rather than relying on the interpretation of a specialist who may be less informed about the patient's overall clinical condition.

Common Obstacles

The following obstacles are commonly encountered:

1. The patient refuses to accept the possibility of having OSAS: Mobile apps that record snoring or restless sleep sounds are available and provide more persuasive evidence than the testimony of a spouse, although not all cases leave an audible trace.
2. Being refused a referral to a sleep study: The patient may need to find out the relevant local regulations and educate the family physician about OSAS.[†]
3. Being offered a home-based sleep study: This is an acceptable starting point, but PSG should still be pursued in case of a negative result. In addition, despite advances in CPAP machine automation, PSG is still considered the gold standard for calibrating treatment and ruling out more complex SDB.
4. Being told that the PSG result is negative for OSAS: This is a common occurrence and should not be taken at face value, for reasons discussed above. Moreover, an AHI or RDI of <

- 5 is considered normal and treatment will normally not be offered; however, in the presence of symptoms related to sleep fragmentation, it may be beneficial to reduce the index to <1 through conservative treatment such as oral appliance therapy or positional therapy. Finally, when diagnostic results are inconclusive, CPAP, oral appliance, or positional therapy can be attempted on an empirical basis.
5. Being offered only a limited range of CPAP machines and masks: The patient should insist on access to the most appropriate setup in order to achieve compliance.
6. Not being offered combination therapy when indicated: CPAP is commonly combined with an oral appliance, and there is a growing array of minimally invasive surgical procedures and other potentially useful innovations.⁶
7. Not having proper follow-up, and stopping effective therapy: The patient may need to be reminded on an ongoing basis of the importance of persisting with treatment. Also, patients considered to have only mild OSAS are less likely to be supported by the medical system.

Successful treatment of OSAS may precipitate its own psychological challenges, including depression due to the permanent lifestyle change, regret about not having begun therapy sooner, and even overwhelm from the newly found ability to interact with the world once sleep quality has been restored. These will require appropriate counseling support.

Discussion

Vis Medicatrix Naturae

As naturopathic physicians, we tend to believe that the body possesses a deep intelligence that promotes optimal health under optimal conditions. While this is true when these conditions are met during early development, when they are not it becomes apparent that the *vis medicatrix naturae* operates within constraints set by evolution. Recognizing that nature's intelligence is not omnipotent may help us to become more attuned to undiagnosed OSAS in our patient population, as well as motivate us to prevent airway abnormalities in the next generation through proper education and pediatric care.

Tolle Causam

From the preceding discussion, it should be apparent that the etiology of OSAS is complex. The patency of the upper airway can be compromised by inflammation, tissue enlargement or laxity, and inadequate development of the surrounding bony structures. When we consider that each of these is in turn caused by multiple factors, opening the airway through naturopathic means alone is often impractical. While it may be tempting to improve sleep quality through the great variety of tools available to us, the treatment of OSAS should be focused on the airway, even when its compromised condition might be construed as a symptom of some other underlying cause. Anatomical causes are generally not correctible in adults except surgically, and inflammation, excessive weight, and other soft-tissue concerns are exacerbated by the sleep disorder itself and may not be correctible unless sleep is addressed first.

Mind, Body, and Spirit

In reflecting on the deeper meaning of sleep apnea and breathing difficulties, broadly speaking we may say that it reflects a throat-chakra issue; however, the fascinating book *Messages From The Body*⁷ offers more specific clues. Here is a sampling of rubrics:

- Sleep apnea: "*Maternal deprivation.*" *They are intensely sensitive, fearful and longing for mother love... they dare not express or even acknowledge these feelings out of fear of total rejection and abandonment... they are so self-suppressing that they are suffocating themselves.* [Note that inadequate breastfeeding is a potential contributor to OSAS.]
- Inflammation of the palate: "*Life sucks!*" *They have intense resentment of their lot in life. They do not find what has been dealt them at all to their taste...*
- Suffocation: "*Self-revulsion.*" *They are "choking to death" on their own guilt and shame. They feel that they should be thoroughly punished or even destroyed for their "sins"...*

Such patterns are vital determinants of psychic health and well-being. As we hold space for them, let us make room for sleep quality and airway integrity as crucial factors in the optimal health of the entire human organism. ▀

[References available online at ndnr.com](#)

* Factors such as insufficient REM sleep due to sleep disturbance associated with the test setting, insufficient sleep in the supine position, variations in neck tilt and head position, avoidance of medications or alcohol normally taken at home, and variability of the condition from night to night, may all conspire to produce a falsely normal score.

** In fact, in cases where arousals are detected with no corresponding respiratory disruptions (such that not only AHI, but even RDI, is normal), it may be worth retesting using more sensitive means. Intraesophageal-pressure monitoring is the gold standard, as it measures respiratory effort directly; however, because it requires a specialized esophageal catheter, it is not performed routinely. Most commonly, airflow limitation is measured using a pressure-sensitive nasal cannula, where increased respiratory effort is implied by a "flattening" of the roughly sinusoidal normal curve of the nasal-pressure channel. An older technology based on a temperature-based flow sensor is still in use, but it is not sensitive enough for detecting subtle airflow limitations. A "normal" AHI or RDI may at times mask an abnormally high frequency of arousals during REM sleep; this information is available from a full sleep report.

*** In order to perform an independent assessment of the sleep data, it is worthwhile obtaining access not only to the sleep report but also to the graphs that summarize the arousal patterns and related metrics. For example, a normal AHI or RDI may mask an abnormally high frequency of arousals during REM sleep, or – conversely – the absence of SWS or REM sleep during the unfavorable sleep-study conditions may mask respiratory events that would otherwise have occurred during these stages.

† Along with Mark Burhenne's aforementioned book, an excellent introduction for both patients and physicians is: Park S. *Sleep, Interrupted*. New York, NY: Jodev Press; 2008.



David Nortman, ND, studied at the University of Toronto before graduating from CCNM in 2004. More recently he completed a master's degree in Philosophy of Science from Tel-Aviv University. Dr Nortman has completed extensive training in classical homeopathy, utilizing it to address the full spectrum of chronic conditions. He practices locally in Toronto and worldwide on a long-distance basis, and serves as a homeopathic consultant and mentor to naturopathic colleagues. Dr Nortman has published numerous articles in homeopathic journals and has lectured to both lay and academic audiences. Currently, he is writing a book on homeopathy and its interaction with modern science and philosophy. Website: www.homeopathyzone.com.

Lyme Disease

A Whole-Person Approach

DARIN INGELS, ND, FAAEM

Lyme disease has become the fastest-growing insect-borne infectious disease in the United States, Europe, and Asia. In 2018, we are already hearing about the rising tick population and the expectation that Lyme disease will only become more epidemic than it already is. More than 300 000 Americans and 65 000 Europeans are affected annually by this epidemic – and these numbers seem to climb every year.^{1,2}

The conventional medical understanding of Lyme disease is somewhat limited to those who have acute Lyme disease. In contrast, chronic Lyme disease, or post-Lyme syndrome, has become controversial and has poor consensus among physicians regarding the best way to diagnose and treat it.

An acute case of Lyme disease consists of symptoms that develop within days or weeks of a tick bite and which may be treated relatively easily at the outset. A chronic case occurs when Lyme makes its way deep into the system and keeps recurring, possibly with periods of remission. However, both types of Lyme are misunderstood.

Acute Lyme Disease

The early acute phase of Lyme disease generally occurs 3-30 days after a tick bite. Symptoms may include³:

- Fever
- Chills
- Pounding, throbbing headache
- Persistent, noticeable fatigue
- Numbness and tingling
- Muscle and joint aches
- Swollen lymph nodes
- Facial or Bell's palsy
- Erythema migrans (EM) rash ("bull's-eye rash")

The most well-known and characteristic symptom of the acute phase is the EM, or bull's-eye rash. It begins at the site of a tick bite, and expands until it reaches a diameter of 12+ inches. Rarely itchy or painful, this rash sometimes clears as it enlarges, such that it resembles a target or "bull's eye." The presence of the EM rash is pathognomonic for Lyme disease.

Although a bull's-eye rash and flu-like symptoms are classic early signs of Lyme, many people never experience either of these. Some people get vague symptoms (or none at all) and may harbor the infection for years without knowing it. And hundreds of thousands of people have the typical symptoms of Lyme without even knowing that they have the disease, until long after it has deeply entrenched itself into their bodies. Lyme disease is called "The Great Mimic" because it resembles numerous other illnesses. As a result, it is not uncommon for physicians to overlook Lyme disease as they attribute symptoms to something else. Lyme disease has been misdiagnosed as chronic fatigue syndrome, fibromyalgia, multiple sclerosis, mononucleosis, Parkinson's disease, Alzheimer's disease, and many other disorders.

Chronic Lyme Disease

People suffering from chronic Lyme disease can look clinically different, and may present with symptoms such as³:

- Fatigue
- Abdominal pain and bowel changes
- Memory loss or cognitive impairment
- Numbness or tingling of extremities
- Sensory distortion of skin (burning sensations), especially in hands or feet
- "Wandering" joint or muscle pain
- Light or sound sensitivity
- Dizziness or vertigo
- Sleep disturbances

- Cardiac problems: mitral valve prolapse, heart block, heart palpitations, chest pain
- Balance or coordination problems
- Endocrine disruption: hypothyroidism, irregular menses, etc

The Centers for Disease Control and Prevention (CDC) states that up to 10% of people with acute Lyme disease will go on to develop chronic Lyme disease or Post-Lyme Syndrome⁴; however, many Lyme-literate physicians feel this number is grossly underreported.

Lyme Testing: Pitfalls of the 2-Tiered System

Adding to the challenge of Lyme disease is making a correct diagnosis. According to the CDC, Lyme disease is a clinical diagnosis, which means it is based on symptoms and other factors rather than solely on a lab test. Lab testing for Lyme disease has been woefully inadequate for decades, due to the poor sensitivity and specificity of the testing itself. Currently, the CDC recommends a 2-tier testing system, starting with a Lyme screen test.⁵

One study found that in 55 people with known Lyme disease, <46% of them had either IgG or IgM antibodies on their Lyme screen. A good screening test should pick up at least 95% of the people being tested for their condition, according to the College of American Pathologists.⁶ The Lyme screening test does not meet this criterion.

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In fact, the Lyme screen test may only pick up about 56% of people who have Lyme disease.⁷ To make matters worse, the CDC guidelines do not recommend doing a Western Blot when the Lyme screen is negative. Research shows that the Western Blot is more accurate in diagnosing Lyme disease because it looks for very specific antibodies often seen in Lyme disease patients but not found in healthy controls.⁸

The Western Blot is the second stage of the CDC's recommended 2-stage testing. This test is also an antibody test, but measures multiple antibodies associated with *Borrelia* infection. In 40 years of research, we have learned that some antibodies are specific to Lyme, while others are not. The presence of 23, 34, 39 and 93 kd antibodies are strongly associated with exposure to Lyme and may suggest infection.⁹ But since antibodies can hang around for years, and even decades, the presence of antibodies does not necessarily indicate a current infection.

The Western blot is also highly prone to false negatives, ie, negative for antibodies despite active infection. This occurs because it can take weeks for the body to develop antibodies against Lyme, which is why the CDC does not recommend doing a Western Blot test on anyone whose illness occurred within the previous month. Other labs tests, including PCR testing and direct staining, have poor sensitivity and poor reliability in screening for Lyme disease.

So, the bottom line is that a negative test does *not* exclude the possibility of having Lyme disease, but a positive test confirms exposure to the organism causing Lyme disease. It is important to evaluate lab testing in conjunction with a patient's clinical symptoms. An individual could have been exposed to *Borrelia* and never developed Lyme disease but may still have antibodies in their bloodstream. This is why it is necessary to always consider your patient's symptoms before starting any treatment.

Conventional Treatment

The conventional medical treatment for Lyme disease is a course of antibiotics for up to 3 weeks after diagnosis. This has been the recommendation of most government, medical, and public health organizations in the United States for almost 40 years. While mainstream infectious-disease doctors do not generally deviate from the CDC recommendations, others feel that the standard 21 days of antibiotic therapy is not enough to defeat Lyme disease. Many doctors who are members of the International Lyme and Associated Diseases Society (ILADS) will use longer courses of antibiotics, both oral and intravenous – for weeks, months, or even years.¹⁰ Concurrent use of 3-4 antibiotics is now common.

Most bacteria replicate every 20 minutes, but *Borrelia* species replicate much more slowly, every 1 to 16 days.¹¹ Since many antibiotics commonly used to treat Lyme disease only work when the organism is replicating (bacteriostatic), they are not effective when the organism is in a dormant state, and therefore would take longer to eradicate the infection. Current guidelines from the CDC only address acute Lyme disease and do not address post-Lyme syndrome at all.

Long-term antibiotic use kills off a large portion of the friendly gut bacteria, which are necessary for maintaining the health of the microbiome and immune system and

fighting infection. Long-term antibiotics can also lead to kidney and liver damage, as well as many other harmful side effects.

Although there is evidence that antibiotics can be effective in acute Lyme disease, studies on long-term antibiotic use either show no effectiveness at all or limited benefits that stop when antibiotics are discontinued.

Naturopathic Treatment

In line with the tenets of naturopathic medicine, the treatment of Lyme disease should encompass the whole person, including diet, lifestyle habits, comorbid conditions, immune status, and environmental exposure, in addition to treating active infection. A whole-body approach to Lyme disease addresses many of the underlying factors that keep people with Lyme disease ill.

Diet

Although many diets have been reported to help people with Lyme disease, I have personally found an alkaline diet to help most Lyme patients. This involves eating foods that promote better cellular alkalinity. There are no specific studies on an alkaline diet and Lyme disease, but the few studies published on alkaline diets,

in general, have observed health benefits, including reducing inflammation.¹² Note that following an alkaline diet does not change blood pH, as has been purported by some individuals.

I recommend eliminating all processed foods, sugar, and coffee, and primarily eating organic fresh vegetables, legumes, and nuts. It is also necessary to limit animal protein to less than 20% of total dietary intake for the week, as animal protein breaks down into acid-forming byproducts. For patients who have difficulty making dietary changes, it may be helpful to have them work with a nutritionist for meal planning and guidance.

Intestinal Health

Research shows the intestinal tract accounts for 70-80% of immune function; thus, maintaining healthy intestinal permeability, balanced microflora, and optimal digestion is essential for overcoming any chronic infection. It is important to ensure proper intestinal motility to prevent dysbiosis and mucosal inflammation, which in turn can disrupt normal intestinal function. Comprehensive stool analysis and/or organic acid testing can help provide insight into specific areas of intestinal function that are imbalanced. The treatment in this area should be specific to your patient, but I often consider herbs and nutrients that help promote better motility, reduce inflammation, rebalance gut microflora, and treat overgrowth of pathogenic microbes. Some specific considerations include magnesium salts, probiotics, glutamine, curcumin, and fish oils.

Treat Active Lyme Infection

Several herbal protocols have been used to treat both acute and chronic Lyme disease, many of which I have used successfully in

my clinical practice. I have also found that herbs are generally better tolerated than antibiotics, and come with fewer adverse side effects. The 2 herbal protocols I have used the most are Dr Zhang's Modern Chinese Medicine protocol and a modified form of Dr Lee Cowden's protocol.

Dr Zhang is a Chinese medical doctor and licensed acupuncturist in New York City who developed a series of Chinese herbal formulas to help eradicate Lyme, boost immune function, improve circulation, and modulate autoimmunity. When I myself had Lyme disease, his protocol helped me to get off antibiotics and dramatically improve my health. Dr Lee Cowden, a cardiologist in Dallas, TX, prior to retirement, developed a series of liquid tinctures made from plants from the Amazon rainforest in South America. His protocol has been studied at the University of New Haven by Dr Eva Sapi and has been found to be more effective than antibiotics.¹³ His protocol also helps eradicate infection, reduce inflammation, and improve detoxification.

I have also used protocols from Stephen Buhner, Byron White, and a specialty herbal formula company with unique products. I prefer the other protocols, as I've observed greater clinical effectiveness

with Dr Zhang's and Dr Cowden's protocols; I've also seen fewer die-off reactions using these protocols, which are common with any Lyme treatment.

Lifestyle & Environmental Control

In addition to taking antibiotics or herbs to treat the active infection, it is also important to manage lifestyle habits that can undermine your patient's immune system, making it harder to overcome an infection. Getting good-quality sleep, getting plenty of exercise (as tolerated), and managing stress all play significant roles in immune system function. I encourage anyone with Lyme disease to eliminate from their home and work environment all toxins and toxicants that are known immune disruptors, in order to optimize their immune function.

One of the biggest external factors influencing Lyme disease is mold. Mycotoxicity and mold allergy can cause symptoms that are almost identical to Lyme disease, and many people with mold problems are unaware of their exposure. I highly recommend Lyme patients have their home tested for mold using spore-trapping and ERMI (Environmental Relative Moldiness Index) to help identify hidden sources of mold, so that it can be remediated if present. Additionally, urine mycotoxin testing or mold allergy testing can help establish whether mold is contributing to one's health issues.

Immune Modulation & Detoxification

Research shows that Lyme disease can trigger an autoimmune reaction against certain neurological proteins, which may account for many of the symptoms of post-Lyme syndrome. Therefore, therapies

that help modulate the immune system are ideal for correcting immune dysfunction. I have used low-dose immunotherapy (LDI) successfully with hundreds of my Lyme patients. Developed by Ty Vincent, MD, LDI uses homeopathic dilutions of a Lyme nosode to downregulate Th2 responses to Lyme, essentially "turning off" the autoimmune reaction to *Borrelia* spp. This isopathic approach has helped numerous Lyme patients overcome their symptoms. To learn more about how to do LDI, please visit progressivemedicaleducation.com.

Other forms of immunotherapy, such as sublingual immunotherapy (SLIT) can help modulate regulatory T cells (Tregs) against mold, pollen, dust, or foods, and lower the total body immune burden.¹⁴ I have found this to be a useful adjunctive therapy in treating Lyme disease, especially in those with a history of allergies prior to being infected with Lyme.

Detoxification is a limiting obstacle for many chronic Lyme patients. Although some patients are genetically predisposed to being slow metabolizers, many naturopathic therapies can still help promote better detoxification and elimination. Research shows infrared sauna can help eliminate toxic metals and chemicals stored in the tissues and organs.¹⁵ The temperature and length of treatment should be modified for each person according to what they can tolerate, but regular sauna therapy can improve many Lyme symptoms.

I also highly recommend colon hydrotherapy, for many of the same reasons that I recommend sauna therapy. It is an effective way to promote better bowel function and improve motility and elimination. I have had some Lyme patients that failed every therapy they tried except colon hydrotherapy, which helped them improve. I recommend 1-2 sessions per week for the first 4 weeks and to then adjust the schedule based on clinical response.

Summary

Early detection and treatment of Lyme disease gives your patient the best chance of recovery without future complications. Many naturopathic therapies can be as or more effective than conventional antibiotic therapy, as antibiotic therapy only addresses eradication of the organism and fails to address underlying immune, autoimmune, and detoxification dysfunction. Lyme disease is a complex illness with more than 100 associated symptoms, and its prevalence continues to rise. Most naturopathic physicians in North America are likely to see Lyme patients in their practice at some point, and they are well suited, in general, to treat Lyme. Following basic naturopathic principles, along with some understanding of Lyme-specific protocols, can greatly improve the health of Lyme patients and help them to overcome their illness. ▀

[References available online at ndnr.com](http://ndnr.com)



Darin Ingels, ND, FAAEM, is a respected leader in natural medicine, with more than 26 years of experience in the healthcare field. He is board-certified in Integrated Pediatrics and is a Fellow of the American Academy of Environmental Medicine. Dr Ingels has been published extensively and is the author of *The Lyme Solution: A 5-Part Plan to Fight the Inflammatory Autoimmune Response and Beat Lyme Disease* – a comprehensive natural approach to treating the disorder. He specializes in Lyme disease, autism, and chronic immune dysfunction. He uses diet, nutrients, herbs, homeopathy, and immunotherapy to help his patients achieve better health.

A Century After the Spanish Flu

SUSSANNA CZERANKO, ND, BBE

The very old and the very young showed themselves, on the whole, less susceptible.

Dr Bernard Fantus, 1918, p.635

None are so blind as those who cannot see that the average mortality of influenza patients treated by homeopathic physicians was actually only about one-thirtieth of the average mortality reported by all physicians.

Dr William A. Pearson, 1919, p.11

The German aspirin has killed more people than the German bullets have.

Dr C. J. Loizeaux, Des Moines, ID, 1919, p.589

increase “visible/tangible examples of the seriousness of the illness (e.g., pictures of children, families of those affected coming forward) and people getting vaccinated (the first to motivate, the latter to reinforce).” (Nowak, 2015) The language used in the media is designed to evoke fear and terror. An example: “Ten more children have died of influenza in the United States this week, bringing the total to 63, federal health officials with the [CDC] said today in their weekly flu update. This flu season is shaping up to be a historically severe one with no end in sight.” (Brooks, 2018) Brooks, an MD who appears to be contributing to this fear generation, has posted many similar statements fueling

the motivation needed to fill the queues at flu vaccination clinics and pharmacies. The increased number of billboards and signs this year in my own city, Portland, OR, suggests that we might be faced with a repeat epidemic such as the one Americans faced a century ago.

Scared to Death

Fear as a precipitating factor in the 1918 flu epidemic was noted by many who lived during its reign of terror. At the same time, there was a global war in full throttle, tearing apart families and ravaging Europe. Within this turmoil the broad spread of the Spanish Flu took an equally shocking toll. Shortly after this dreadful period,

Crawford Green, MD, noted, “We saw many sad cases of influenza-phobia among adults, but the fear reached the children, too. In many cases when they were stricken with the disease, they were literally almost scared to death.” (Green, 1920, p.1103) Eli G. Jones, MD, commented that those who harbored fear were most likely to contract the flu. (Jones, 1918, p.553) A. H. Waterman, MD, noted, “As in all panics the emotion of fear seemed to kill judgment.” (Waterman, 1919, p.379)

This year marks the 100-year anniversary for the Spanish Flu that effectively terminated WWI and left, by some estimates, between 50 and 100 million dead. WWI was responsible for 9 million

A century ago, the “Spanish Flu” decimated populations globally. Flu season 2018 is reminiscent of that event, already breaking records, so the claims go, with more North Americans than ever affected, according to media and announcements by the Centers for Disease Control and Prevention (CDC). The H3N2 virus strain has been reported by the CDC to cause severe symptoms that are potentially deadly, especially for vulnerable populations such as children and the elderly. What’s more, the reports add, the flu shot developed is showing signs of being less effective this year. Messages like these dominate the media, undermining health by spreading fear and worry among the public. However, is this any surprise? This pattern repeats every year, sounding the alarm that every man, woman, and child needs a flu shot.

CDC Fear Mongering

Fear of harm is the driving thrust implemented by the CDC and other health system authorities to ensure compliance with the annual flu shot ritual. The flu shot is the colloquial term; however, the CDC refers to it as “immunization,” which is a sanitization of its actual character, a “vaccination.” In this regard, Robert Kennedy reports, “Every year, the Centers for Disease Control and Prevention (CDC) and pharmaceutical companies mount an aggressive campaign in the mainstream media to persuade Americans to get their flu shots.” (Kennedy, 2018) Not surprisingly, “flu shots are big business: industry analysts estimate that within the next five years, the U.S. flu vaccine market will be worth almost \$3 billion annually.” (Kennedy, 2018) Kennedy continues, “As pharmaceutical companies bombard American consumers with ubiquitous billboards, drugstore enticements and radio announcements to ‘get your flu shot now,’ the CDC has advised the industry to hike demand through the use of a ‘recipe’ of scare-mongering messaging.” (Kennedy, 2018)

The CDC’s strategy for fostering public interest and high vaccine demand was implemented by Glen Nowak, PhD, Associate Director for Communications at the CDC. In a PowerPoint presentation, he formulates the necessary steps required of medical experts and public health authorities to stress the danger of the flu and to urge the American population to get their flu shot. He cites the need to



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casualties. Concomitantly, the Spanish Flu left behind in its wake devastating and unequalled colossal collateral damage never before experienced in the world.

The symptoms accompanying a flu are malaise and fever. Both of these symptoms cause concern and often initiate a rapid response by a patient to treat these symptoms. Fear of fever is the compelling motivation. Infection or other pathologies accompanied by fever are often the most visible and the target for treatment. Dr André Saine outlines the historical controversies regarding the fever that still persist after centuries of discourse and debate. Naturopaths and early doctors responded to the presentation of fever in the spirit of the Hippocrates school, where fever was “often encouraged and celebrated, as it was seen as the most important of the body’s natural defenses.” (Saine, 2017, p.1) Saine

chronicles the players who promoted “fever phobia” and who paved the way to view a fever “as injurious to health, and treatment with antipyretic medication came to be considered a necessity.” (Saine, 2017, p.2) The diverging views held by Homeopaths, Naturopaths, and Allopaths regarding the fever and its treatment were present on the world stage during the Spanish Flu of 1918. Different theories, approaches, and statistics reveal much when we look at the Spanish Flu, most particularly when we compare how this pandemic was respectively handled by these different groups.

Opium, Ipecac, and Aspirin

The *standard of care* established by the “regulars” included excessively large doses of aspirin, Dover’s powder, consisting of opium and ipecac, and a strengthening diet composed of large quantities of animal

protein. No two treatments could be more diverging and opposite than the allopathic treatment protocols and those used by the Naturopaths and Homeopaths.

When we review the letters and testaments from Naturopaths reporting on their flu cases in Benedict Lust’s publications, we get a glimpse of what some of their methods entailed, and are soon reassured that flu epidemics are not destined to resolve in gravesites. Benedict Lust published many accounts from his colleagues in 1918 and the following year. There is a resounding message that Naturopaths had no trouble treating the Spanish Flu patient. In fact, their patients usually recovered. The typical death toll of the regular doctors was colossal; those documented by naturopathic doctors was significantly less. What stands out in naturopathic patient care outcomes

during the Spanish Flu was the lack of drug intervention. Using hydrotherapies and fasting and hydrating patients – a reflection of respect for the fever’s role in the resolution of illness – was so effective that only about 1% of patients died under the care of a Naturopath.

Naturopathic Protocols

Lust published letters from practicing Naturopaths who treated influenza in 1918. One such letter from E. P. Kondis, ND, a practicing Naturopath from Waterbury, CT, who reported that he saw “58 patients [with the flu], of whom five developed pneumonia and no deaths.” (Kondis, 1919, p.44) Kondis outlined his treatment protocol, which relied upon hydrotherapy, dietary recommendations, and herbal remedies. He writes, “For fever, cold application to the head. Hot or cold applications to the body. Cold or hot drinks as lemonade, elder flowers or chamomile tea, etc. Enemas. Sponge bath. Gargle with common salt.” (Kondis, 1919, p.44) His treatment for pneumonia also used cold compresses to the head and chest, and hot applications applied to the feet. (Kondis, 1919, p.44) These treatments used by Kondis supported the patient’s own healing mechanisms, exhibited by the acute symptoms of the flu.

William Havard, a Naturopath, writes, “We cannot state too often nor too emphatically that *acute diseases* are forms of the reaction which the body makes to poisonous or irritating substances and are in themselves healing efforts of Nature.” (Havard, 1918, p.866) He continues,

All acute diseases are for this identical purpose, and will only occur in a body containing foreign substance. To endeavor in any manner to suppress the reaction would be working counter to Nature’s method of destroying and eliminating the trouble maker, and will either cause death or at the least serious impairment of organic function and lay the foundation for chronic disease. (Havard, 1918, p.866)

Havard strongly warns against some of the recommendations of the health boards who were so eager to suppress fever with gram-doses of aspirin. (Havard, 1918, p.865)

James C. Thomson, ND, DO, a Naturopath and Osteopath from Edinburgh, writes that he saw 87 influenza cases. His treatment protocol included...

the application of cold compresses to the kidneys; the compress was changed hourly. In individual cases, compresses were also applied to the throat, chest, etc., with occasional osteopathic attention. As to the diet, this was reduced while any symptoms of discomfort were present—usually two to three days complete fast followed by fruit and gradual return to normal. All recovered quite rapidly, usually in from five to seven days. (Thomson, 1919, p.100)

Thomson reports that he lost one patient whose “wife and family were very hostile to the Nature Cure treatment.” (Thomson, 1919, p.100) The man was given suppressive drug treatment and hospitalized, and died.

Homeopathic Successes

Homeopathic doctors practicing in 1918 offered patients a hope to survive



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the ravaging claws of the 1918 flu. Homeopaths saved hundreds of lives by following and trusting their homeopathic teachings. Dr Green writes, "The homeopathic physicians throughout the country were not following some empty fad or fancy that so frequently leads even homeopaths astray, but by the nature of things, were constrained to fall back upon the faith of their fathers, *similia similibus currentur*, and it served them well." (Green, 1920, p.1102)

Dr William Pearson reports on a survey of Homeopaths who treated influenza, conducted by the Bureau of Homeopathy of the American Institute of Homeopathy in June 1919. Two-thousand Homeopaths were sent the questionnaire and all of the reports received were tabulated. The results indicated that 26 795 patients were treated by the 88 Homeopaths who replied to the survey, and the number of deaths totaled 273 – a 1.06% average mortality. (Pearson, 1919, p.11) The homeopathic success compared with the average mortality rate of 30% demonstrated that those fortunate enough to be treated by a Homeopath had a higher chance of survival.

Dr Edward Davis provided a list of hygienic and preventative measures of personal cleanliness. He writes, "Urge the importance of fresh air and the avoidance of chill and overheat. In fighting the epidemic give no medicine and use no treatment which may depress the vital forces, especially the heart of the patient." (Davis, 1918, p.552)

Pearson reported on the nurses attending to the flu patients that were treated at the Hahnemann Hospital. All the nurses "were working day and night without any consideration for their own health, a total of 57 of them at different times had to finally go to bed, and a large proportion of these had influenza, but not a single one of them developed pneumonia." (Pearson, 1919, p.13)

Dr T. McCann of Dayton, OH, treated 1000 cases of influenza. He writes, "I have had no losses. I want no credit given me for these results." (McCann, 1919, p.845) He continues, "Given an individual in a fair degree of health when stricken with this malady, there is no reasonable excuse for a homeopathic physician losing a single case." (McCann, 1919, p.845)

Dr E. Jones, a Homeopath who practiced in Buffalo, NY, writes, "There is an epidemic of this disease [influenza] in Buffalo; from 75 to 100 deaths are reported within 24 hours." (Jones, 1918, p.553) Like many of his homeopathic colleagues, Jones had considerable experience treating influenza epidemics and had never lost a case of influenza in nearly 50 years of practice. He writes, "[Influenza] is one of the *easiest* diseases to cure if a doctor knows his *Materia Medica*." (Jones, 1918, p.553)

In his paper, which was published by 3 principle newspapers, he provides a few suggestions for treatment:

If you should have chilly spells, and your nose feels stuffed up, great thirst, very restless and feverish, put five drops Tincture Aconite in half a glass of water, take one teaspoonful every hour. Put your feet in hot water, as hot as you can bear it, keep them in the water for 15 minutes. Drink plenty of hot lemonade. (Jones, 1918, p.553)

Dr James W. Ward reports 100% recoveries in 182 patients and reveals the 3

most frequently used remedies:

Gelsemium 3x: Controlled the fatigue cases having chilliness, moderate fever, sneezing, watery nasal discharge. No thirst. Yawning. Drowsy but often sleepless. Deep-seated, dull aching in the limbs and joints. Banded feeling around head or occiput fullness.

Eupatorium 3x: Marked chill followed by fever 103° to 105° F/39° to 41° C. Pulsating frontal or occipital headache. Intense aching in back. Deep bone aching. Nausea. Vomiting. Larynx rough, scraping sensation producing cough. Oppression of chest with soreness.

Bryonia 1x: Much chilliness, frequently with heat in head. Flushed face and much thirst. Heat more internal. Transient sweats of single parts. Dry cough which yields to blood specked expectoration. Constriction of chest and pressure on sternum. The symptoms all aggravated by motion... Desire to but unable to take deep breath. (Ward, 1919, p.856)

Dr Dudley Williams reports that fellow Homeopaths practicing in Rhode Island "found very little variation in the remedial treatment of influenza. Arsenic, bryonia, eupatorium perfoliatum, baptisia, and gelsemium seem to be in pretty general use, and the opinion was that if the case was seen early enough it seldom ran into pneumonia." (Williams, 1919, p.1003) Dr Harry Baker, of Richmond, VA, dubbed "gelsemium as the *genus epidemicus* and covered 95% of the cases." (Baker, 1919, p.682) McCann cites his 4 remedies that covered uncomplicated cases as "gelsemium, bryonia, eupatorium and arsenicum." (McCann, 1919, p.845)

The complications arising from allopathic care often prolonged recovery or else, sadly, compounded the virulence of the bacterial infection and escalated high levels of fatalities. The indiscriminate use and reliance upon "salicylates, including aspirin and quinine were almost [Allopathy's] sole stand-bys." (Williams, 1919, p.1003) We now know that the aspirin dosages during the Spanish Flu exceeded safe levels. "On 5 October 1918, *The Journal of the American Medical Association* recommended aspirin: 'The acetylsalicylic acid may be given in a dosage of 1 gm. (15 grains) every three hours...or a smaller dose combined with 0.1 gm. (2 grains) acetophenetidin, until symptomatic relief is secured' (p 1137). These recommended doses (1000–1300 mg), with frequencies ranging from hourly to every 3 hours, resulting in daily doses of 8–31.2 grams, are above the maximum safe dose." (Starko, 2009, p.1407) Dr W. A. Dewey of Ann Arbor, MI, witnessed cases of "pulmonary edema suddenly brought on by aspirin." (Dewey, 1919, p.1005) "Cases overdosed with aspirin have most all required sulph. in high potency." (Gier, 1919, p.1100)

Dr Charleton Harkness of Chicago, IL, was one of the medical doctors stationed at Camp Lee with 60 000 recruits. He writes, "About 15,000 cases passed through the Base Hospital and ... there were between 700 and 800 deaths." (Harkness, 1919, p.587) Harkness did not use aspirin and did not have access to his homeopathic remedies. He relied upon a few tinctures in his possession, such as gelsemium that he diluted. "I had 13 deaths in the six weeks, and was complimented by my

chief medical officer as having the lowest death rate in the hospital." (Harkness, 1919, p.587) The effects of aspirin were noted and discontinued in this particular hospital, causing a huge decline in the death rate. (Harkness, 1919, p.588)

Liquid Diet

Homeopaths also made dietary recommendations consisting principally of fasting or liquids. Green comments, "The dietetic treatment of influenza is of great importance. I know of no disease, not even typhoid, in which a strictly liquid diet is of more importance." (Green, 1920, p.1102) McCann prescribed an egg white with orange juice every 3 hours for the first 2 days, adding a vegetarian diet of fruits, vegetables, and some cereals. Dr J. Johnson of Michigan put patients on "a strict diet [keeping] the patient in bed and not allowed to sit up until about two days after the fever was gone." (Johnson, 1919, p.1097)

Lessons Lost

With the remarkable success that Homeopaths had during the 1918 Spanish Flu epidemic, it is puzzling and even incomprehensible that during the ensuing century, Homeopathy – with all of its journals, schools, hospitals and MD-trained Homeopaths – would vanish from the healthcare terrain. In the April 1919 issue of the *Journal of the American Institute of Homeopathy*, a notice posted by the Bureau of Census appeared, announcing, "A Joint Influenza Committee has been created to study the epidemic and to make comparable, so far as possible, the influenza data gathered by the Government departments."

(1919, p.1190) This census, comparing the treatments and outcomes of Homeopaths and Allopaths, was never published.

Today, we have the luxury of hindsight to see that aspirin was prescribed in toxic doses, resulting in hiking the mortality rate of pulmonary edema and pneumonia. The reliance upon pharmaceuticals has not lessened but rather has increased over the past century. Will we one day find ourselves scratching our heads and mumbling, "What were the scientific doctors thinking?" Dr Andrews of West Virginia saw the correlation between a common allopathic vaccine, antipyrim, and the increased incidence of pneumonia and fatalities in the 1889 flu epidemic. Thirty years later, the same pattern repeats with aspirin use and pneumonia and fatalities. (Andrews, 1918, p.720) There is a French expression which captures this sad outcome: "Plus cela change, plus c'est toujours le même chose." ▀

References available online at ndnr.com



Sussanna Czeranko, ND, BBE, a graduate of CCNM, is a licensed ND in Oregon. Sussanna is a frequent presenter, nationally and internationally. As Curator of the Rare Books Collection at NUNM, she is the author of *The Hevert Collection*, a 12-book series about naturopathic medicine, the ninth volume of which is now complete, *Mental Culture in Naturopathic Medicine*. Sussanna founded The Breathing Academy, a training institute for naturopathic doctors to incorporate a scientific model of breathing therapy called Buteyko into their practice. Her next large project is to complete the development of her new medical spa in Manitou Beach, Saskatchewan – *Manitou Waters Centre*. She is the co-founder of the International Congress on Naturopathic Medicine and the inaugural conference chair of the Healing Skies Naturopathic Medicine Conference, in Saskatchewan.

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Collaborative Teaching & Learning

What Mary Goggins Taught

DAVID J. SCHLEICH, PHD

Like all professional education preparatory programs, medical education has 3 tiers, elucidated well by Donald Schon in his seminal 1986 book, *Educating the Reflective Practitioner*. The tiers include the basic sciences, applied sciences, and a practicum of some sort. In our field, this translates into discipline-specific classes in tier 1, such as anatomy, physiology, histology, pharmacology, and so on; and applied sciences in tier 2, like botanical medicine, homeopathy as a medical system, physical medicine, etc. In the final tier or stage of medical education, we conjugate our senior interns through a challenging clinical regimen. The least common denominator throughout this process, though, was a framework designed for individual learners. Translation: “sage on the stage” teaching, passive individualized delivery and receipt of information and content, and clinical training choreographed by autonomous supervising physicians. Clinic competencies were tested individual by individual. We have, though, learned much in the ensuing years about team learning, teaming up teachers, and teaming up program design and delivery. A half-century back, my Grade 6 teacher, Mary Goggin, knew that.

Miss Goggin was a Commonwealth Exchange Teacher from Ireland. She had many tales and related counsel gleaned from characters who worked the farms and little shops in and around Cahera, Drimoleague and Meenies, tiny villages back then in County Cork, where she was from. Our teacher would always draw on such wisdom from those whom she called “my life team,” and she wanted us to get in the “team habit” as soon as we could. Smack dab in the first week of that school year, in fact, she was introducing us to the importance of collaborating with each other. As part of her pitch, she referenced Mr Teagan O’Neill from her own personal “life team.” We knew that he and his wife Nola were real because their picture was on our class bulletin board. They were local farmers from Cahera who had unerring advice on most matters. In this instance the O’Neills’ particular wisdom was related to Miss Goggin’s comments about how we would be working in teams all year. She further explained that that specific advice about teams, when she first heard it back in Ireland, had been directed by “mother and father O’Neill” toward their son, Quinn. Quinn listened and learned because his parents were very much part of his own “life team.”

Apparently, Quinn did not want to farm like his parents and his grandparents before them. Rather, he and his new bride, Kate, wanted to open a pub to make their living. Miss Goggin reported that Mr and Mrs O’Neill were disappointed. Even so, they proffered counsel, as any good team member might. Teagen is reported to have said, “Shearin’ fleece, servin’ beer; s’all the same, lad. Don’t matter which, ‘cause you and Kate’ll be needing a new team who know what you’re about. They will shorten the road.” Miss Goggin said that Nola

O’Neill weighed in at that same family meeting too. She added, “May the roof above you never fall in, and may your team never fall out.” Miss Goggin was teaching us that day those around us make the best – and sometimes only – teams we have.

The Team Habit

It went like that all year long. Mary Goggin frequently called on the legendary wisdom of the O’Neills and others who seemed to have wisdom available to apply to any problem. She admired them so much that she had affixed a picture of a group from Ireland to the class bulletin board. There were 7 in that group, including the O’Neills and stolid, unsmiling County Cork farming people standing in front of a mossy rock fence. In the center, surrounded by her “life team,” was Mary Goggin holding her newly minted teaching diploma, all smiles and light. We didn’t understand the words on the diploma she was proudly holding, but we knew it was official and that the entire group was celebrating: *Bilingual*

We have learned much in the ensuing years about team learning, teaming up teachers, and teaming up program design and delivery. A half-century back, my Grade 6 teacher, Mary Goggin, knew that.

Certificate, National Schools, AN ROINN OIDEACHAIS 1954. She gave us a copy of that picture at the end of the year. What is useful to note is that Miss Goggin came out of a very repressive and controlling educational system at the time. She became a certified teacher, but found her voice and her style as a teacher with the help of many mentors. She had written “Life Team” above that picture. She also had affixed a map of County Cork to our class bulletin board, above which map were the words, “The Centre of The Known Universe.” What?

From the get-go, Miss Goggin organized our class of 35 into 7 rotating “teams for life,” as she called them. She routinely assigned tasks of all kinds to those constantly shifting teams. Of course, we snubbed the Grade 5s and the Grade 7s and 8s who weren’t doing teams. Because our teacher was very cool, we also wondered how long she might last, given the ultra-conservative place our school was. We recognized that we were lucky to have such a different teacher, lucky to be doing things differently than others in the same school. And, as we began that year of teams and groups, at first we squabbled about which team was on top, which team was on bottom, and who got whom for the team of the day. Gary Enright could do fractions and decimals like nobody’s business. Wilfred O’Donnell knew history like crazy. Eileen Demarco could spell big-time. Philip

Bonicci could sing “Dark Moon” like Pat Boone. Diane Belaysis liked boys with brains, but the only boy in the class as tall as she was, was Philip. My point here: after all these years I remember my Grade 6 classmates vividly and we changed, the whole lot of us. I attribute it to those hard-working teams. We were challenged for 40+ weeks and the bonds were strong. Early rivalries shifted into very enduring friendships. Mary Goggin went back to Ireland at the end of that school year and was a pen-pal with many of us for years and years. I even went to visit her as a young man with long hair on a long trek through Europe.

I remember the very first team task. We were all poised, thinking we were going to be competing with the other teams on some task or challenge. Not! Miss Goggin announced, on that first memorable day when the “team talent” got launched, “the best way to learn is to teach.” She explained that if there was something one person didn’t get on any

trying to understand “kinetic energy” and “latent heat.” I could not get the concepts, for some reason, and was horrified when she told me to teach “latent heat” back to my own team group the very next day. Good grief.

Let me tell you, though, that in doing what she advised, I got it. Because I had to get ready for the questions and quizzical looks of team classmates, I drilled down into the topic and was as prepared as I could be for explaining the notion of how melting (or freezing, if one thought about it backwards) worked. Miss Goggin had told us that it was possible to have water in solid state, and water in liquid state, at exactly the same temperature. That it took extra energy to convert water as a solid into water as a liquid, but that same energy did not necessarily increase the temperature of the resulting water, baffled me and, as it turned out, baffled most everybody else in the class too, except Wilfred O’Donnell, the resident brainiac who invariably tormented me with questions. By the time I had “taught” this to my team, I had experienced an epiphany. The epiphany was in getting it that the heat which broke the force of attraction between particles was not siphoned off to increase the kinetic energy of the particles themselves, thus no rise in temperature in the melted water. Easy. I recall this phenomenon to this very day.

It went like that all year. One time we were debating the notion of “might is right” in the history of nations. Different teams had different approaches to the affirmative and negative of that particular conundrum. Miss Goggin often asked one of us to “teach back” to our team that week confounding topics like that. Clever pedagogy. We all got used to responding to our classmates’ questions and gradually kinder teasing. We got so used to not learning alone that year, that when Grade 7 rolled around and Mr Whitney Humphrey sidled into his “sage on the stage” routine, we responded by creating learning teams of our own. He didn’t like it too much.

What that Grade 6 cohort internalized was that we were best together, helping each other. We had to get good at figuring out our differences, navigating conflicts, talking clearly, avoiding any kind of bullying. We got pretty flexible, and definitely got good at shifting gears. We did have our wobbles, but because we rotated through many team configurations that school year, we came out the other side as confident individuals who knew how to get together on a task. This attitude and those skills stayed with me. For example, the very next year I made the high school basketball team, even though I was short and couldn’t dribble a ball up or down the court very well; what I had was great accuracy in sinking the ball from almost anywhere. I convinced the coach that the team could count on me to deliver hoops from anywhere. I asked him, though, if I could count on them to dribble the ball and fake out the other team. The tutelage of that 5-foot Irish woman from Cork and all those “sticking together to get the work done” stories from farmer O’Neill are with me yet.

Team-Based Education

At university I chose a business degree program and brought Mary Goggin's magic with me. I soon discovered that team training and team work were hallmarks of curriculum delivery in that business school. Over the years I became aware of manifestations of team-based education and training in all kinds of corners of society, such as the military, emergency responders, community policing teams, fire and rescue squads, event coordinators, sales teams, and even political parties. It was, though, when I found my way into the medical-education field a quarter-century back, that I realized there might be some catching up to do.

Tuckman's (1965) early work about how teams take shape is as valuable now as it was over a half-century ago as a guide to how to incorporate Goggin Magic into medical education, in the sense of encouraging such team design, delivery, and continuous improvement. Whether our intention is to move toward the notion of "patient-centeredness" as the norm by eschewing individual clinicians motivated by individual achievement, or whether we need principal investigators to collaborate with others to get research questions answered, team skills can help. What lies before us, notwithstanding the bruising being applied to the Affordable Care Act's notions of coordinated care and medical education (and research) where common commitment coupled with shared skills, shared information, and shared goals can be hugely effective. As Baker et al (2005) put it, there is a place for collective

performance where mutual, rather than individual, accountability in the landscape of medical education obtains. I wondered more than once about how to take the best of Mary Goggin's ideas, learned from Teagan and Nola O'Neill and the other farmers near Drimoleague, and apply them to what we do in colleges and universities with teams of academic choir soloists all over America?

Four Stages in Team Development

Tuckman taught us that the 4 stages in the development of a team (forming, storming, norming, and performing) apply as much to students as to their teachers. Students in their study groups can assimilate all that information in digestible chunks more handily, and into the bargain, retain more of it. Furthermore, if we build into the process of our delivery opportunities for our learners to reconcile in their own heads what they weren't sure about, we'd be optimizing learning. The teacher becomes the student and the student becomes the teacher, as Miss Goggin used to put it. Gagne (1970) and Weber and Karman (2005) confirm with their research that such collaborative learning stimulates more active engagement and better retention than passive learning anytime. What Teagan O'Neill would say, as reported by Miss Goggin, "If you'll be wanting to get more wool from that sheep, ask around and go deep; there's farmers here what know and will be showing you if you ask." In-depth learning, in other words, can be beautifully reinforced by teamwork, collaborative knowledge-sharing, and

reinforcing of what is learned.

The lessons in our health system's delivery habits are very tough to grapple with. Solutions include team-training, of both teachers and students, all along the path of their learning and credentialing. For example, multidisciplinary and team-based groups need to be encouraged more broadly. A splendid example of this is reported by Morrison et al (2010) who remind us about such a process from the aviation industry, called "crew resource management" (CRM). CRM has been very successful in reducing severe aviation errors and enhancing industry safety – great goals for medicine too. Clancy and Tomberg (2007) remind us that this kind of approach is very present in American health systems in something called the "TeamSTEPS™" program (Team Strategies and Tools to Enhance Performance and Patient Safety). Morrison et al, though, point out that because students in medical schools are not officially on hospital teams, they don't benefit from early team-training and can't contribute easily to the units and departments (such as ORs, labor and delivery, ICUs, medical center clinics). Duh.

What are those team skills, again, that are so important to coordinated care? Dunnington and Williams (2003) summarize them beautifully: "effective communication with patients and families, patient counseling and education, cooperative work-sharing with health care professionals, and the ability to instruct students and other health care professionals" (p.257). The ACA contemplates doctors (allopathic and

naturopathic), pharmacists, nurses, social workers, and others sharing responsibility for patient care. Team approaches are more valuable than ever.

You'd swear that some of those who have been thinking and writing about the power of teamwork in medical education, in fact in education generally, had been in Mary Goggin's Grade 6 classroom in southwestern Ontario back in 1957.



David J. Schleich, PhD, is president and CEO of the National University of Natural Medicine (NUNM), former president of TruStar Health, and former CEO and president of CCNM, where he served from 1996 to 2003. Previous posts have included appointments as vice president academic of Niagara College, and administrative and teaching positions at St. Lawrence College, Swinburne University (Australia) and the University of Alberta. His academic credentials have been earned from the University of Western Ontario (BA), the University of Alberta (MA), Queen's University (BEd), and the University of Toronto (PhD).

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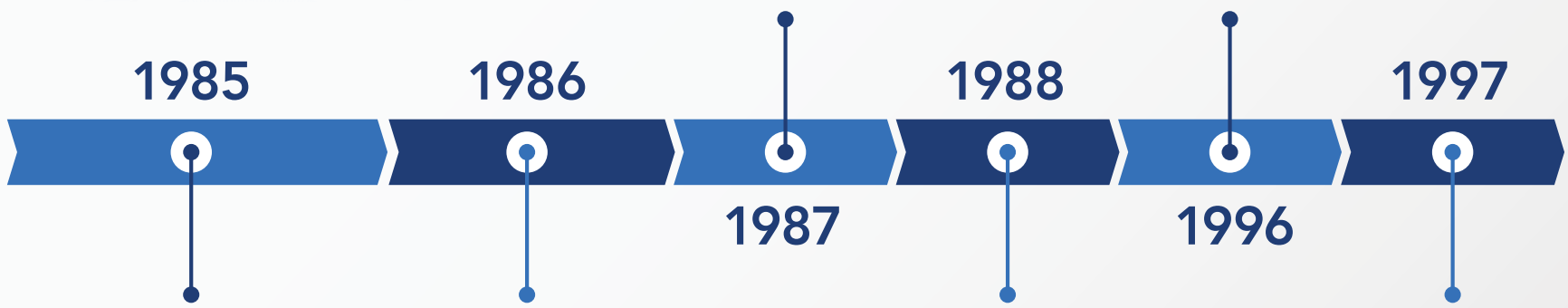
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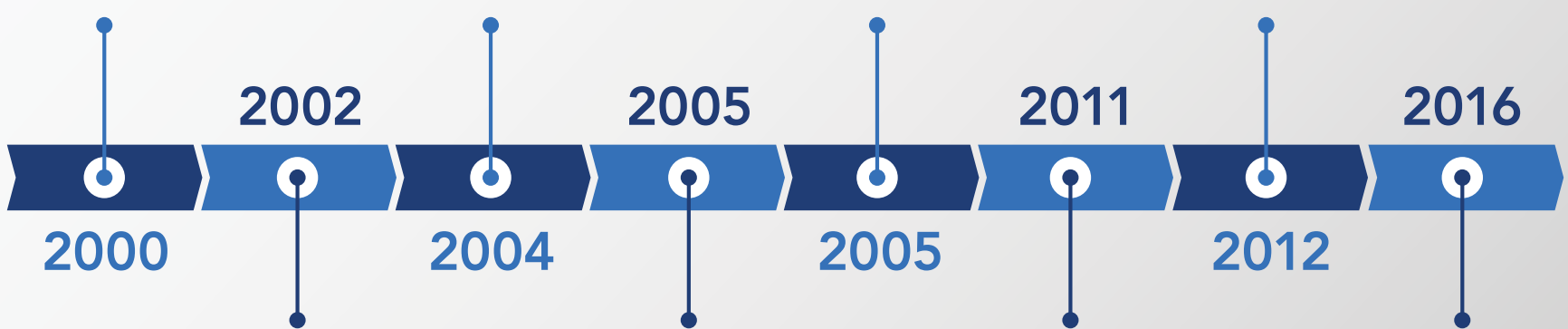
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