

Sample Pages

Nina Pierpont, "Wind Turbine Syndrome: A Report on a Natural Experiment"
(K-Selected Books 2009)

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ONE

By way of explaining why on earth I wrote this book

I wrote this report because I saw a medical problem that few clinicians were paying attention to or, for that matter, seemed to understand. Dr. Amanda Harry in the United Kingdom led the way in recognizing the cluster of symptoms people experience around wind turbines.¹ I, myself, began encountering the problem from numerous emails and telephone calls I began receiving in 2004, shortly after wind developers turned up in my community and my husband and I started investigating industrial wind turbines.

The uniformity of the complaints quickly became apparent. It didn't take long to realize the potential for a relationship between these complaints, on the one hand, and *migraine, motion sickness, vertigo, noise and visual and gastrointestinal sensitivity, and anxiety*, which, taken together, form a coherent and interconnected neurologic complex in medical practice.

The breakthrough came in early 2006, when I interviewed a couple who were about to move out of their home because of their own and their children's symptoms. The interview supported the

¹ Harry, Amanda. 2007. Wind turbines, noise, and health. 32 pp. www.windturbinehealthhumanrights.com/wtnoise_health_2007_a_barry.pdf.

relationship between turbine-associated symptoms and migraine/motion sensitivity. Best of all, the interview introduced me to the curious phenomenon of vibration or pulsation felt in the chest. It was this element that caught the attention of the National Academy of Sciences in its 2007 report to Congress, *Environmental Impacts of Wind-Energy Projects*. The authors wanted to learn more about this effect of low frequency noise.²

This study is my answer to their question.

As I have worked to understand these complaints, I have benefited from new research allowing us to better understand neurologic phenomena like spatial memory loss and fear reactions in people with balance problems—symptoms that often “bored and baffled” clinicians, as one of my referees put it.³ Wind developers and acousticians have been even less charitable:

It’s . . . worth noting that studies have shown that a person’s attitude toward a sound—meaning whether it’s a “wanted” or “unwanted” sound—depends a great deal on what they think and how they feel about the source of the sound. In other words, if someone has a negative attitude to wind turbines, or is worried about them, this will affect how they feel about the sound. However, if someone has

² National Research Council. 2007. *Environmental Impacts of Wind-Energy Projects*. The National Academies Press, Washington, DC. 185 pp, p. 109 (Prepublication Copy). “Low-frequency vibration and its effects on humans are not well understood. Sensitivity to such vibration resulting from wind-turbine noise is highly variable among humans. Although there are opposing views on the subject, it has recently been stated (Pierpont 2006) that ‘some people feel disturbing amounts of vibration or pulsation from wind turbines, and can count in their bodies, especially their chests, the beats of the blades passing the towers, even when they can’t hear or see them.’ More needs to be understood regarding the effects of low-frequency noise on humans” (pp. 108–9).

³ I review and discuss this research in the Discussion section, p. 70.

a positive attitude toward wind energy, it's very unlikely that the sounds will bother them at all.⁴

Their patients [people living near wind turbines and reported on by Drs. Osborne and Harry] may well have been experiencing adverse symptoms, but we have to keep in mind that people who have failed, for whatever reason, in strong objections to a development, build up in themselves a level of unfulfilled expectations and consequent stress, which peaks after the failure and can overload their coping capabilities. This leads them to lay the blame on whatever straw they can clutch. This is especially so in group activities, where mutual support may turn to a mutual, interacting misery, which worsens the situation. . . . The very low levels of low frequency noise and infrasound which occur from wind turbines will not normally cause problems. If problems have occurred, it is possibly for some other stress-related reason.⁵

Brian Howe, a consulting engineer in acoustics for 20 years for HGC Engineering, said Ontario's guidelines for turbine noise are adequate and consistent with Health Canada studies. Most people near wind turbines aren't complaining about the noise, Howe said. In some cases, noise complaints could reflect higher anxiety levels from people who had unrealistic expectations of hearing virtually no sound, he said.⁶

⁴ Noble Environmental Power, LLC. Wind fact sheet #5: Are modern wind turbines noisy? p. 2. www.windturbinesyndrome.com/?p=698.

⁵ Leventhall, Geoff. 2004. Notes on low frequency noise from wind turbines with special reference to the Genesis Power Ltd. Proposal near Waiuku, NZ. Prepared for Genesis Power/Hegley Acoustic Consultants, June 4, p. 7.

⁶ Rennie, Gary. 2009. Wind farm noise limits urged. *The Windsor Star* (Ontario, Canada). February 24.

Responses like these are a pity. They're rubbish. There is nothing "psychosomatic" or malingering about it. The physiologic pathway flows from physical forces (air pressure changes, noise, vibration) to physical sensations (chest pulsations, internal vibration, tinnitus, headache, ear fullness) to brain integration of sensory signals to distortions of brain functioning (sleeplessness, concentration and memory deficits, physical symptoms of anxiety)—not the reverse. Research clearly shows there are precise and definable neurologic connections that explain how distorted sensory signals can derail normal psychological and cognitive function and, in fact, trigger physical symptoms. (It's worth pointing out that our understanding of brain function has progressed by leaps and bounds in the last 25 years, radically changing the landscape of psychology and psychiatry and, of course, neurology.⁷ Much of the research on vestibular function, whereon I draw, is even more recent, conducted within just the last 10–15 years.)

Leaving the pop psychology behind us, let's move on to evidence-based science. In the world of medicine my study is properly called a "case series," defined as *a descriptive account of a series of individuals with the same new medical problem*. Let me be clear: a case series is a standard and valid form of medical research. New illnesses are often introduced with case series whose role is to define an illness, suggest causation, and alert the medical and research profession to its existence. (This being one of the chief reasons for this report.) After an illness is defined and awareness raised, it becomes more feasible to do larger, more expensive studies to explore etiology (causation), pathophysiology, and epidemiologic characteristics.

⁷ See, for example, Schore, Allan N. 1994. *Affect Regulation and the Origin of the Self: The Neurobiology of Emotional Development*. Lawrence Erlbaum Associates, Hillsdale, NJ. 700 pp.

Case series don't typically have control groups. Nevertheless, I saw I needed a comparison group of similar, though unexposed, people to distinguish which symptoms were due to turbine exposure. The most similar unexposed people, of course, were my study subjects themselves prior to turbine exposure and after the end of exposure. I therefore set up a *before-during-after* study format, interviewing families who had already moved out of their homes due to symptoms or who were planning to move and had already spent periods away from home, during which turbine-associated symptoms abated.

This format served a three-fold purpose:

- 1) it ensured there was an "after" phase for each family,
- 2) it guaranteed that at least one member of each family was severely affected, enough to need to move, and
- 3) it provided validation for participant statements, since one can hardly discount the gravity of symptoms that force a family to vacate its home or perform expensive renovations aimed solely at noise exclusion.

Which brings us to what is known in science as a "natural experiment": *a circumstance wherein subjects are exposed to experimental conditions both inadvertently and ecologically (within their own homes and environments)*. Obviously, it would be unethical to expose people deliberately to potentially harmful interventions. Hence natural experiments, while less controlled, have an important role in clarifying the impacts of potentially toxic, man-made exposures.

The ecological dimension in the phrase *natural experiment* is worth emphasizing, since many elements of an exposure may not be reproducible in a laboratory, such as round-the-clock exposure,

exposure over months, or impacts on customary activities. For symptoms related to wind turbine sound there are also technical difficulties in reproducing in a laboratory the types of sound, air pressure variation, and vibration that my subjects' observations suggest are involved. Failure to provoke the same symptoms in a laboratory setting may tell us more about the limitations of the laboratory situation than about real-world effects.

To further create comparison groups, I collected information on all members in the ten families, not just the most affected. This widened the age range of subjects and gave me information on variably affected people who were all exposed to turbine noise capable of causing severe symptoms. I then used the natural variation within the study group to examine which elements of the *pre-exposure* medical history predicted which parts of the *during-exposure* symptom complex. By this method the study begins to answer the intriguing question of why some individuals are affected more than others by living near wind turbines, and which individuals in the general population are notably at risk for symptoms. It also suggests pathophysiologic mechanisms.

It would be difficult to do a conventional epidemiologic study of the health effects of wind turbines, at least in the United States, even if one were blessed with substantial funding and institutional backing, as I was not. By "epidemiologic," I mean studies in which random or regular sampling is used (as, for example, assessing everyone within three miles of a set of turbines, or every fourth name in an alphabetical listing of everyone within three miles), or case and control populations identified. The difficulty comes from the legal and financial stone wall of the *gag clause*.

In the course of this study I repeatedly encountered these clauses in leases between wind developers and landowners, in "good

did not allow me to determine whether in fact wind turbines played a role in these conditions during exposure. These conditions would require other kinds of study over and above the clinical interview and case series. (I have included them in a separate section of the *Results* in the REPORT FOR CLINICIANS because I think they may need attention from the medical research community.)

This study also does not tell us how many people are affected within a certain distance of wind turbines. But it does offer a framework for what to pursue in such a study (meaning, the next phase: epidemiologic studies), such as what symptoms to study and what aspects of the exposure to measure.

Shifting, now, to the format of the book. I wrote the REPORT FOR CLINICIANS as a (long) scientific article, beginning with an *Abstract* or brief summary, followed by an *Introduction* to the problem and background information, a description of the *Methods* used (including study sample selection), a presentation of the *Results* (which are the data secured during the study and its analysis), and finally *Discussion* of the results with interpretation of their meaning in the context of current medical knowledge. Data are compiled in *Tables* (numbered 1A, 1B, 1C, 2, and 3) included within the *Results* section.

REFERENCES are footnoted in the text and listed together towards the end of the book. I added a GLOSSARY of medical and technical terms to make the book more intelligible to non-medical readers, and a list of ABBREVIATIONS.

The CASE HISTORIES (A1 through J4) present the raw narrative data—each individual subject's symptoms and statements—in table format, one person per table with separate columns for *before*, *during*, and *after* exposure, and separate rows for each organ or

functional system (sleep, headache, cognition, balance/equilibrium, ears/hearing, etc.).

The CASE HISTORIES are gathered together at the end of the clinical text. They are the backbone of my report. I deeply appreciate my subjects' willingness to be included herein.

The book is intended for physicians and other professionals and individuals who wish to better understand the wind turbine-associated symptom complex. This posed a dilemma: writing in the specialized language of clinical medicine and science is very different from the language one uses for laymen. Yet my goal is to reach both audiences. I solved the problem by adding (at my editor's insistence) a more conversational, parallel text, which I christened REPORT FOR NON-CLINICIANS.

The result is a book with two, tandem texts. They say the same thing. One says it in the language of the clinician (REPORT FOR CLINICIANS), the other in the everyday language of—well—my editor (REPORT FOR NON-CLINICIANS).

The goal of REPORT FOR CLINICIANS is scientific precision, including frequent expressions of my degree of certainty or uncertainty. Since the physics and the physiology I invoke are complex and not widely known among clinicians, I explain them in this text. Here, likewise, I quote and summarize numerous scientific articles, and I use numbers and statistics (albeit the simplest type known).

REPORT FOR NON-CLINICIANS says it all over again, this time in English my mother-in-law would understand. To accomplish this, I had to sacrifice a degree of scientific precision, since *plain English* and *scientific precision* don't always mix. I freely acknowledge that the REPORT FOR NON-CLINICIANS might set some clinicians' teeth on edge, and for this I beg their indulgence.

A second disclaimer. Readers should understand that Wind Turbine Syndrome is not the same as Vibroacoustic Disease.¹⁰ I say this because the two are often equated in the popular media. The proposed mechanisms are different, and the noise amplitudes are probably different as well.

Wind Turbine Syndrome, I propose, is mediated by the vestibular system—by disturbed sensory input to eyes, inner ears, and stretch and pressure receptors in a variety of body locations. These feed back neurologically onto a person's sense of position and motion in space, which is in turn connected in multiple ways to brain functions as disparate as spatial memory and anxiety. Several lines of evidence suggest that the amplitude (power or intensity) of low frequency noise and vibration needed to create these effects may be even lower than the auditory threshold at the same low frequencies. Re-stating this, it appears that even low frequency noise or vibration too weak to be heard can still stimulate the human vestibular system, opening the door for the symptoms I call Wind Turbine Syndrome. I am happy to report there is now direct experimental evidence of such vestibular sensitivity in normal humans.¹¹

Vibroacoustic Disease, on the other hand, is hypothesized to be caused by direct tissue damage to a variety of organs, creating thickening of supporting structures and other pathological changes.¹² The suspected agent is high amplitude (high power or intensity) low frequency noise. Given my research protocol, described above, my study is of course unable to demonstrate whether wind turbine exposure causes the types of pathologies

¹⁰ Castelo Branco NAA, Alves-Pereira M. 2004. Vibroacoustic disease. *Noise Health* 6(23): 3–20.

¹¹ Todd NPMc, Rosengren SM, Colebatch JG. 2008. Tuning and sensitivity of the human vestibular system to low-frequency vibration. *Neurosci Lett* 444: 36–41.

¹² Castelo Branco and Alves-Pereira 2004.

found in Vibroacoustic Disease, although there are similarities that may be worthy of further clinical investigation, especially with regard to asthma and lower respiratory infections.

Moving on, I have been asked if Wind Turbine Syndrome could be caused by magnetic or electric fields. I have no reason to think so. There has been extensive epidemiologic research since 1979 on magnetic fields and health, comparing people who live close to high power lines or work in electrical utilities or work in other industries where magnetic field exposure is likely to be high, to people who do not.¹³ This substantial body of research has produced no good evidence that magnetic field exposure causes cancer in children or adults, cardiac or psychiatric disease, dementia, or multiple sclerosis.^{14,15} After three decades of research, there is still no experimental evidence for a physiologic mechanism for any of the proposed effects of magnetic fields.¹⁶

This makes it difficult to do epidemiologic studies, since researchers don't know what exposure to measure, or what exposure period (e.g., last week or five years ago) might be relevant.¹⁷ An association has been shown between higher magnetic field exposure in utility workers and amyotrophic lateral sclerosis (ALS), a neurodegenerative disease, but this is most likely due to more frequent electric shocks in these settings, not to the magnetic

¹³ Ahlbom IC, Cardis E, Green A, Linet M, Savitz D, Swerdlow A; INCIRP (International Commission for Non-Ionizing Radiation Protection) Standing Committee on Epidemiology. 2001. Review of the epidemiologic literature on EMF and health. *Environ Health Perspect* 109 Suppl 6: 911–33.

¹⁴ Ahlbom et al. 2001.

¹⁵ Johansen C. 2004. Electromagnetic fields and health effects: epidemiologic studies of cancer, diseases of the central nervous system and arrhythmia-related heart disease. *Scand J Work Environ Health* 30 Suppl 1: 1–30.

¹⁶ Ahlbom et al. 2001.

¹⁷ Ahlbom et al. 2001.

fields.¹⁸ Claims that voltage and frequency irregularities in household alternating currents (what some refer to as “dirty electricity”) create a wide, non-specific swath of medical problems—from ADHD to rashes to diabetes to cancer—are completely unsubstantiated, and also have no plausible biologic mechanisms.¹⁹

A few words about peer review. Peer review is quite simple, contrary to the mystique it has acquired among wind developers (most of whom probably have a fanciful idea of what it is). Peer review *consists of sending a scholarly manuscript to experts in that particular field of knowledge, who are asked to judge whether it merits publication.* Simple as that. The identity of reviewers (also called “referees”) can be either known to the author (with book manuscripts, authors are routinely asked by editors to submit a list of recommended referees) or kept confidential.

If the referees (usually consisting of two or three) manage to convince the editor that the manuscript is not worthy of publication, the editor contacts the author and rejects the manuscript. If, on the other hand, the referees feel the manuscript merits publication subject to certain revisions and perhaps additions, the editor will forward their reports to the author and ask for a response. “Are you willing to make these changes? Do you agree with these criticisms? If not, give me compelling reasons why not.”

The author then revises the manuscript accordingly, except where she feels her referees are wrong—and manages to convince the

¹⁸ Johansen 2004.

¹⁹ I have asked Prof. Magda Havas, Environmental and Resource Studies, Trent University, Ontario, Canada, to remove references to Wind Turbine Syndrome from her PowerPoint presentation on hypothesized wind turbine health effects, because these references are inaccurate.

editor. Once the editor feels the author has addressed criticisms and suggestions adequately, he (she) proceeds with publication.

Lastly, referees do not have to agree with the author's arguments or conclusions. This is worth emphasizing. Their purpose is merely to certify that a) the manuscript conforms to conventional standards of scholarly or clinical research appropriate to the discipline, and, perhaps most important, b) the manuscript is a significant contribution to knowledge.

In the case of this book, a variety of scientists and physicians, all professors at medical schools or university departments of biology, read and commented on the manuscript and recommended it as an important contribution to knowledge and conforming to the canons of clinical and scientific research. Moreover, they did in fact suggest revisions, even substantial revisions and additions, all of which I made. Some gave me written reports to include in the book itself. See REFEREE REPORTS. Others offered to review the book after it was published.

That said, the litmus test of scientific validity is not peer review, which, after all, is not infallible, as the history of science amply demonstrates. Peer review is an important first step in judging scientific or scholarly merit. Still, the ultimate test is whether other scientists can follow the author's research protocol and get the same results, or if different lines of research point to the same conclusions.

That, of course, remains to be seen with this report.

I thank Dr. Joel Lehrer in particular for providing me with new information regarding vestibular function, contributions echoed by Drs. Owen Black and Abraham Shulman (all in otolaryngology/neurotology). I thank Professors Ralph Katz (epidemiology) and

My hope is that this report will balance the risk-benefit picture of wind turbines more realistically, and help those individuals, such as George Kamperman and Rick James, who are actively promoting noise control criteria that will stem the health and home abandonment problems documented here.

Kamperman and James have convinced me that a single, one-size-fits-all setback distance may not be both protective and fair in all environments with all types of turbines. Even so, it is clear from this study and others that minimum protective distances need to be:

- a) greater than 1–1.5 km (3280–4900 ft or 0.62–0.93 mi), at which there were severely affected subjects in this study;
- b) greater than 1.6 km (5250 ft or 1 mi), at which there were affected subjects in Dr. Harry's UK study;
- c) and, in mountainous terrain, greater than 2–3.5 km (1.24–2.2 mi), at which there were symptomatic subjects in Professor Robyn Phipps's New Zealand study.²⁶

Two kilometers, or 1.24 miles, remains the baseline, shortest setback from residences (and hospitals, schools, nursing homes, etc.) that communities should consider. In mountainous terrain, 2 miles (3.2 km) is probably a better guideline. Setbacks may well need to be longer than these minima, as guided by the noise criteria developed by Kamperman and James.

The shorter setbacks currently in use in the USA and elsewhere, 1000–1500 ft (305–457 m), are a convenience and financial advantage for wind developers and leaseholding landowners. They

²⁶ See pp. 31–32 for discussion and references.

have no basis in research on safety and health, and they make no clinical sense.

For those who read this report and recognize their own symptoms, the appropriate medical specialist to consult would be a neurotologist (or otoneurologist), who is an otolaryngologist (ear, nose, and throat doctor) who specializes in balance, the inner ear, and their neurological connections. When I sent this report out for critical review, these were the physicians who recognized a remarkably similar symptom complex from cases familiar to them—such as certain inner-ear pathologies.

To those of you living near turbines and recognizing your own symptoms within these pages: you are not crazy and not fabricating them. Your symptoms are clinically valid—and unnecessary. While wind developers rush headlong into yet more projects, you unfortunates will have to exercise patience as the medical profession catches up with what is ailing you. Meanwhile, my advice is, speak out. In *The Tyranny of Noise*, Robert Alex Baron calls for an end to “our passive acceptance of industry’s acoustic waste products.”²⁷

This will happen only when the suffering refuse to be silenced.

By the time I finished interviewing and moved on to data analysis (February 2008), six of my ten families had moved out of their homes because of turbine-associated symptoms. Three months later (May 2008), when the first draft was complete and I contacted the families for their approval and permission to publish the information on them, two more had moved out because of their turbine-associated symptoms—bringing the total to eight of the ten. The ninth family could not afford to move, but had done extensive renovations in an

²⁷ Baron, Robert Alex. 1970. *The Tyranny of Noise: The World’s Most Prevalent Pollution, Who Causes It, How It’s Hurting You, and How to Fight It*. St. Martin’s Press, New York, p. 12.

effort to keep the noise out. (Renovations, ironically, that made the house worse to live in, since they could no longer heat it properly.) As of this writing, family number ten is struggling to remain in their home.

Behold ten families whose lives have been turned upside down because of the wind industry's acoustic waste products.

Finally, ask yourself why a country doctor practicing in the poorest county in New York State did this study, and not the Centers for Disease Control or some other relevant government agency. It's a fair question and a troubling one. I ask it myself.

It is well known that wind developers target impoverished communities for their wind farms. This explains the "poorest county" part of my question, and likewise why wind turbines quickly became a looming issue in my life four years ago. But it leaves unanswered the part about, "Why did I write this report, and not the government?"

To answer that would of necessity catapult this report (and me) into the treacherous territory of public policy. One would like to think science is not beholden (craven?) to public policy, but that would be naïve, would it not? Moreover, while the scientist in me would like to imagine that I can write this report and remain above the hurly-burly of public policy, I know this, too, is naïve. Wind Turbine Syndrome is an industrial plague. It is man-made and easily fixed. Proper setbacks are the best cure I know of; they do the job just fine. If I could scrawl this on a prescription pad and hand it to my subjects in this report, I would do so. No brilliant scientist needs to discover a new antibiotic or vaccine or sleeping pill to treat it.

Setbacks, however, are not considered matters of public health, but matters of public policy—what is called “politics.” And right there is the rub. In the global rush to wind energy there is almost no voice heard for public health repercussions. Where it is heard—at town meetings, on the Internet, in Letters to the Editor, in courtrooms—it is routinely ridiculed. I speak from experience.

Wind energy is being promoted by every state and national government I know of, under intense lobbying by wind development companies generally owned or otherwise capitalized by powerful investment banks which in turn take large tax write-offs and reap large government subsidies for their wind farm projects. These companies turn around and sell carbon credits (green credits). Perhaps this helps explain why no provision is made for clinical caution?

And perhaps this goes some way toward explaining why a pediatrician in rural New York State and a general practitioner in Cornwall, England—along with a handful of rank-and-file, community physicians elsewhere in the UK, USA, Australia, and who knows where else—are the ones funding this research and writing these reports.

Then so be it.

Three poems by Gail Atkinson-Mair, who has lived every page in this book:

The Moles

You call me to the window, not quite sure,
 “I really get the feeling we’ve got fewer moles
 —must be the cat.” An end to an unending war,
 you grin and raise your glass. You’re right. The holes
 that spotty-dicked the grass and made me think

of crazy golf have by some miracle grown rare. I
 frown and look away, then crash the dishes in the sink
 and fumble, ill at ease. Alarm bells ring—but why?
 There’s something not quite right today—
 a smooth expanse of light rich green and not one
 mole hill to be seen; a thousand velvet diggers gone.
 We look at one another and although
 our mud-filled brains urge us to stay
 our guts tell us—it’s time to go.

Home

She’s like the flies that buzz around inside
 the house, alight on window, table, chair
 and then take off. She stands, she sits, she looks
 around a moment, then she’s off. Eyes wide
 she searches, checks, then stops. Smooths hair
 from face, swipes dust from books.
 She’s pulled the plugs and fixtures out,
 switched off the mains, “Not there,” she said.
 She’s gone outside and come back in,
 It isn’t there. You know it’s not! I want to shout
 and make her stop. The buzzing in her head
 will drive her mad. She grabs the radio and plugs it in
 then plugs her ears. Her face is grey
 “Stop it now,” she screams at me, “and make it go away.”

My Back Yard

I had to come before I go insane.
 The plant you built has side effects: I vomit, weep,
 have dizzy spells and I’m depressed. The pain
 from pressure in my ears keeps me from sleep—
 I wake up drenched, have jitters, palpitations.
 Your “silent” noise impairs my concentration—
 I think you call that torture.

I no longer have a garden or a view, your
symphony of turbines has drowned the song of nature.

You say you've done what is required by law
but tell me where do people feature?

How old are you, Ms May? Aha, the menopause . . .

We call this problem, "NIMBY," I think you'll find . . .

Damn right, you are. It's not in your back yard—it's mine.

Sample Page

TWO

The REPORT, for clinicians

Abstract

This report documents a consistent and often debilitating complex of symptoms experienced by adults and children while living near large industrial wind turbines (1.5–3 MW). It examines patterns of individual susceptibility and proposes pathophysiologic mechanisms. Symptoms include sleep disturbance, headache, tinnitus, ear pressure, dizziness, vertigo, nausea, visual blurring, tachycardia, irritability, problems with concentration and memory, and panic episodes associated with sensations of internal pulsation or quivering that arise while awake or asleep.

The study is a case series of 10 affected families, with 38 members age <1 to 75, living 305 m to 1.5 km (1000 to 4900 ft) from wind turbines erected since 2004. All competent and available adults and older teens completed a detailed clinical interview about their own and their children's symptoms, sensations, and medical conditions a) before turbines were erected near their homes, b) while living near operating turbines, and c) after leaving their homes or spending a prolonged period away.

Statistically significant risk factors for symptoms during exposure include pre-existing migraine disorder, motion sensitivity, or inner-ear damage (pre-existing tinnitus, hearing loss, or industrial noise

exposure). Symptoms are not statistically associated with pre-existing anxiety or other mental health disorders. The symptom complex resembles syndromes caused by vestibular dysfunction. People without known risk factors are also affected.

The proposed pathophysiology posits disturbance to balance and position sense when low frequency noise or vibration stimulates receptors for the balance system (vestibular, somatosensory, or visceral sensory, as well as visual stimulation from moving shadows) in a discordant fashion. Vestibular neural signals are known to affect a variety of brain areas and functions, including spatial awareness, spatial memory, spatial problem-solving, fear, anxiety, autonomic functions, and aversive learning, providing a robust neural framework for the symptom associations in Wind Turbine Syndrome. Further research is needed to prove causes and physiologic mechanisms, establish prevalence, and explore effects in special populations, including children. This and other studies suggest that safe setbacks will be at least 2 km (1.24 mi), and will be longer for larger turbines and in more varied topography.

Introduction and Background

Policy initiatives in the United States and abroad currently encourage the construction of extremely large wind-powered electric generation plants (wind turbines) in rural areas. In its current format, wind electric generation is a variably regulated, multi-billion-dollar-a-year industry. Wind turbines are now commonly placed close to homes. Usual setbacks in New York State, for example, are 305–457 m (1000–1500 ft) from houses.¹ Developer statements and preconstruction modeling lead

¹Town of Ellenburg, NY, wind law: 1000 ft (305 m); Town of Clinton, NY, wind law: 1200 ft (366 m); Town of Martinsburg, NY, wind law: 1500 ft (457 m). For other examples in and outside NY State, see *Wind Energy Development: A Guide for Local Authorities in New York*, New York State Energy Research and Development Authority, October 2002, p. 27. <http://text.nysed.org/programs/pdfs/windguide.pdf>.

THREE

The CASE HISTORIES: The raw data

Sample Page

Case History A1 (page 1 of 2)

Person
Mr. A

Age
32

Pre-exposure health status
Good

Health history
No significant

Previous noise exposure
Diesel fishing boat
from childhood

Time to onset of symptoms
Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good but always easily awakened by noise.	“I didn’t really.” Hard to fall asleep. Frequent awakening due to child’s frequent awakening.	Good, at baseline. Child sleeping through night.
Headache	Rare, mild	Continuous headache at home which resolved after several hours away and resumed several hours after return, with onset 3 weeks into turbine start-up process. OTC and prescription analgesics, addition of glasses not helpful.	Resolved
Cognition	Normal. Runs own fishing business. Mild difficulty with memory, especially for names and faces.	Memory problems: “You’d think I was 99.” When arriving at a store or storage building, could not remember what he had come to get without a list.	Partial recovery: self-rated memory 80–85% at baseline, 2% during exposure, and 10% at 6 weeks after moving
Mood	Good. Usually does not show annoyance.	Loss of usual energy and enjoyment for spring fishing season. Mildly irritable.	Anger about home abandonment, otherwise resolved.
Balance/equilibrium	Normal, never carsick or seasick	“A little shaky on feet every now and then” at home.	Resolved
Ear/hearing	Mild subjective hearing loss attributed to diesel engine exposure, no tinnitus	Repetitive popping in ears for first 3 weeks. Tinnitus started several weeks after headache onset and worsened over time.	Resolved

Case History A1 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Eye/vision	Normal without glasses	Burning sensation in eyes. When headache and tinnitus were severe, eyes "felt like they were going to fall out on the table if you looked down." Had normal eye exam.	Resolved
Other neurological	Normal, mild concussion age 14	No change	No change
Cardiovascular	Normal including BP (110–120/80 in 2006)	Mild diastolic hypertension on one reading (128/94 on 4/4/07)	No further BP measurements obtained.
Gastrointestinal	Normal	Nausea when headache was severe. No vomiting or other gastrointestinal changes.	Resolved
Respiratory	Normal except smokes	No change	No change
Other		"You feel different up there, draggy, worn out before you even start anything." "It was a chore to walk across the yard." Symptoms were present in all wind directions, better during rain, and worse with wind from direction of turbines or from the 180-degree opposite direction.	When visiting family 100 km away, "I felt better all over, like you could do a cartwheel." Feels well at new house.

*Exposure period 5 months.

**Interviewed 6 weeks after move.

Case History A2 (page 1 of 2)

Person
Mrs. A

Age
33

Pre-exposure health status

Good. Pregnant during exposure and delivered at term 4 days before moving.

Health history

Polycystic ovarian syndrome and metabolic syndrome. Caesarian section for first delivery.

Previous noise exposure

Worked at biomedical chemical plant for 5 years with 1–2 hours/week exposure to noisy areas.

Time to onset of symptoms

Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Normal. Sleeps through noises other than children.	Frequent awakening	Normal, resolved
Headache	Rare, mild	Occasional headache	At baseline
Cognition	Concentration “great,” works as accountant	Noticed concentration problem at work when training someone; working to focus; trainee had to help	Resolved
Mood	Good, including during and after first pregnancy	Irritable	Resolved
Balance/equilibrium	Gets seasick but not carsick	Slight unsteadiness	Resolved
Ear/hearing	Normal hearing. Persistent middle ear fluid in late 20s, resolved. Tinnitus in past when emerging from noisy plant.	Repetitive popping in ears and decreased hearing for first 3 weeks, then tinnitus began. Tinnitus varied with exposure and worsened over time.	Tinnitus resolved, but has new difficulty understanding conversation in a noisy room. Has to watch speaker’s face.
Eye/vision	Wears glasses. Eyes water if strained.	No change	No change
Other neurological	Normal, no concussion	No change	No change
Cardiovascular	Normal except h/o temporary stress-related hypertension at age 22.	Normal	Normal
Gastrointestinal	Nausea and GER during pregnancy	No change	Resolved after delivery

Case History A2 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Respiratory	Normal, no asthma or smoking.	Lower respiratory infection for 6 weeks not treated until after delivery and move.	Resolved
Other		“Not noisy like a chainsaw, more like pulsating annoyance. To another person it wouldn’t sound loud.”	
Animals		Dog barks at windmills and up more at night.	Improved dog behavior

*Exposure period 5 months.

**Interviewed 6 weeks after move.

Case History A3

Person

Son A

Age

2½

Pre-exposure health status

Good

Health history

Term birth, normal growth and development

Previous noise exposure

No significant

Time to onset of symptoms

Immediate

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Slept through night 12 hours without awakening. Always a good sleeper.	Night terrors 2–5 times each night, 30 minutes to calm down and return to quiet sleep.	At baseline. Night terrors resolved. Awakes once briefly for drink and goes back to sleep.
Headache	None	No apparent headaches.	None
Cognition	Good speech development with lots of words and no sound confusion.	Began to confuse <i>t</i> with <i>k</i> sounds and <i>w</i> with <i>l</i> sounds.	Vocabulary, sentences, and conversational skills are good but still confusing sounds.
Mood	Good-natured, sensitive, bright, listened well for age.	Oppositional, cranky, “a completely different kid for a few months.”	“Instantaneous” resolution when moved, resumed former behavior.
Balance/equilibrium	Normal for age	No change	No change
Ear/hearing	Normal hearing test at birth. One episode of otitis media.	Pulled ears and got cranky synchronously with adult episodes of headache and tinnitus.	Resolved
Eye/vision	Normal	No change	No change
Other neurological	Normal	No change	No change
Cardiovascular	Normal	No change	No change
Gastrointestinal	Normal	No change	No change
Respiratory	Normal, no asthma	No change	No change

*Exposure period 5 months, age 27–32 months.

**Information provided by parents 6 weeks after move.

Case History A4

Person
Infant daughter A

Age
7 weeks

Pre-exposure health status
N/A: born 4 days before end of exposure period

Health history
Healthy newborn, 38-week gestation, birth weight 2.95 kg

Previous noise exposure
N/A

Time to onset of symptoms
N/A

	Pre-exposure	During exposure*	Post-exposure**
Sleep	In utero, 1st and 2nd trimester	In utero, 2nd and 3rd trimester	Sleeps well
Headache			N/A
Cognition			Normal alertness
Mood			Good, calms easily
Balance/ equilibrium			N/A
Ear/hearing			Normal hearing test at birth
Eye/vision			Normal eye exam at birth
Other neurological	Normal fetal movement	No change	Nurses well
Cardiovascular	Normal fetal heart tones and sonogram	No change	Normal
Gastrointestinal			Normal
Respiratory			Normal

*Exposure period 5 months, all in utero.

**Information provided by parents 6 weeks after move, 7 weeks after birth.

Case History B1 (page 1 of 2)

Person
Mr. B

Age
55

Pre-exposure health status
Good

Health history

Surgery 4 times for benign prostatic hypertrophy, once for hand injury

Previous noise exposure

Diesel fishing boat from childhood

Time to onset of symptoms

Immediate with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good	Delayed onset and repeated awakenings; prescribed sleep aid.	Resolved
Headache	Rare, mild	Continuous, head and ears “sizzling.” “It got in your head and would dang well stay there.” Started “at back of head, then down sides, then affected right eye.” Prescription and non-prescription analgesics minimally helpful.	At baseline
Cognition	Normal	“Trouble remembering”; “a little problem concentrating” blamed on sleep deprivation	“Pretty good, a little problem still.”
Mood	Good	Stress, “lots, pretty near near’n I could take, it just burnt me, the noise and run-around”; prescribed anxiolytic.	Improved, still takes some anxiolytic.
Balance/equilibrium	Normal, never seasick or carsick, no vertigo.	Wobbly, staggering, off-balance “like had drunk.” No falls. Occasionally felt dizzy.	Resolved, on roof shingling without problems.
Ear/hearing	Normal hearing on left and mild sensorineural loss at 4 kHz on right in 2006. Intermittent left tinnitus since 2005.	Tinnitus continuous and bothersome, “ringing and sizzling,” and interfering with conversation comprehension. Ears popped “like an airplane.” Ear wax increased.	Resolved

Case History B1 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Eye/vision	Normal with reading glasses	Intermittent right eye pain “like a force on it, like pressure on the eye, the inside part, in the head.” No change in vision. Eye pain/pressure synchronous with headache.	Resolved
Other neurological	Normal, no concussion	No change	No change
Cardiovascular	Normal with BP 126/82, 126/88, 112/70 in 2006	Mild BP elevation 140/80, 132/90, 152/92. After started anxiolytic, BP 128/84.	Resolved, BP 110/68
Gastrointestinal	Normal, no GER, not prone to nausea	Frequent nausea	Resolved
Respiratory	Slight asthma as child. Never smoked.	Two episodes of feeling of weight on chest while lying on couch, which resolved when he stood up. Lower respiratory infection in 5th month of exposure.	Normal
Rheumatologic	Osteoarthritis	No change	No change
Other	Little road traffic or other noise	“That stuff [turbine noise] doesn’t get out of your head, it gets in there and just sits there—it’s horrible.”	Not bothered by “all kinds of traffic” at new location; “after a while you don’t hear it.”
		Felt pulsation in ears and chest while outside when there was fog in the valley between the turbines and the house.	Resolved
		Spent more time at shore at boat, away from house and property, for symptom relief.	
		Hum heard and felt in double glazed picture window when turbines running.	

*Exposure period 5 months.

**Interviewed 6 weeks after move.

Case History B2 (page 1 of 2)

Person
Mrs. B

Age
53

Pre-exposure health status
Good

Health history

Hysterectomy and
cholecystectomy, 4 births

Previous noise exposure

Diesel fishing boat
intermittently for
decades

Time to onset of symptoms

Several weeks, with
progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good	Delayed onset, repeated awakening, difficulty going back to sleep, nocturia. Ear plugs somewhat helpful.	Resolved
Headache	Rare, mild	Continuous except when left property or wind in favorable direction.	Resolved
Cognition	Normal	Concentration disturbed; confused if went on errands without list, had to return home.	Partly resolved at 6 weeks, up to remembering three things without a list.
Mood	Good, hard worker, not moody.	Anxiety, guarding against irritability, upset and “in a turmoil” when symptoms worse.	Resolved
Balance/equilibrium	Normal, never carsick or seasick.	Some unsteadiness and gait change.	Resolved
Ear/hearing	Normal hearing test in 2005, no tinnitus.	Tinnitus and ear pain continuous except when left property or wind in favorable direction. Ear irrigation at clinic worsened tinnitus.	Resolved
Eye/vision	Normal with glasses	Eyes irritated, burning, runny. Ebb and flow of eye symptoms synchronous with headache and tinnitus.	Burning resolved but visual blurring noted when chemotherapy started.
Other neurological	Normal, no concussion	No change	No change
Cardiovascular	Normal including BP	Mild BP elevations 132–140/80–90	Unknown

Case History B2 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Gastrointestinal	GER and post-tussive vomiting.	No change	Worsened with chemotherapy
Respiratory	Chronic cough secondary to GER and smoking.	Breath "short every once in a while, like [while] falling asleep, breathing wanted to catch up with something, hard to explain."	Resolved, normal breathing pattern.
Oncologic	Felt well though had undiagnosed breast cancer.	Breast cancer diagnosed. Mastectomy 4 weeks before end of exposure.	Chemotherapy started.
Other		Left house repeatedly to get relief of symptoms, interrupting work and tasks.	Resolved
Machines	Refrigerator quiet	Refrigerator became loud and was replaced, but new one was also loud.	New refrigerator was moved to new house and is quiet.
	Furnace quiet	Furnace became loud. Circulator was replaced and the furnace was still loud.	

*Exposure period 5 months.

**Interviewed 6 weeks after move.

Case History B3

Person

Daughter B

Age

19

Pre-exposure health status

Good

Health history

ACL tear and knee surgery

Previous noise exposure

Music

Time to onset of symptoms

Immediate

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good	No change	No change
Headache	Rare, mild	No change	No change
Cognition	Good, university student	No change (between terms and not studying)	No change
Mood	“Always irritable at home”	If home more than 2 days, “heavy” feeling, lost motivation and energy, slept more	Normal energy and mood
Balance/equilibrium	Normal, never carsick or seasick	No change	No change
Ear/hearing	Ears often dry, itchy, and painful	No change	No change
Eye/vision	Normal	No change	No change
Other neurological	Normal, no concussion	No change	No change
Cardiovascular	Normal	No change	No change
Gastrointestinal	Normal	No change	No change
Respiratory	Normal, never smoked	No change	No change
Other		“Hard, heavy feeling behind ear, like someone sitting on it.”	Resolved

*Due to college and activities, exposure limited to 10 hours on weeknights over 2 months.

**Interviewed 7 weeks after family moved.

Case History C1
(page 1 of 2)

Person
Mr. C

Age
45

Pre-exposure health status
Good

Health history
Back injury with neuropathic pain

Previous noise exposure
Diesel fishing boat for decades

Time to onset of symptoms
Immediate when all turbines running

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good, sound sleeper	Delayed onset with repeated awakening. Wakes up tired. Feeling of pulsation keeps him awake night and day.	Improved, but not resolved because of depression.
Headache	Rare, mild	No change	No change
Cognition	Normal	Pulsations interrupt concentration; cannot read when pulsations present.	Persistent forgetfulness noted 2 years after moving, with ongoing depression.
Mood	Good	Tired, "cannot recuperate."	Persistent stress of not having his own home and loss of assets. Irritable. Enjoyed going to his abandoned home, but mood worsened with stay of several hours or more. Depression increased in winter 2 years after move.
Balance/equilibrium	Normal, seldom seasick	No change	No change
Ear/hearing	Normal hearing, no tinnitus	Infrequent tinnitus. Hard to hear conversations outside when turbines noisy.	Resolved
Eye/vision	Normal with glasses	No change	No change
Other neurological	Normal, no concussion	No change	No change

Case History C1 (page 2 of 2)

	Pre-exposure	During exposure*	Post-exposure**
Cardiovascular	Normal including BP	No change	No change
Gastrointestinal	Normal	No change	No change
Respiratory	Normal, no smoking for 10 years	Feels pulsations in chest, holds breath, fights sensation in chest, not breathing naturally.	Resolved
Rheumatologic	Back pain from injury	No change	No change
Other		Unable to rest, relax, recuperate in house, "always in a state of defense," drives away in car to rest.	Resolved
		Feels like "energy coming within me," "like being cooked alive in a microwave."	Resolved
		Sensation of pulsation is very disturbing and interrupts concentration and sleep.	Resolved
		Infrequent sensation of throat swelling and obstruction to breathing.	Resolved
		Fog (150 days/year) amplifies noise.	
Animals		Lobster fishery moving further offshore since wind turbines present and increased death in lobster pounds.	

*Exposure period 15 months to all turbines, 21 months to at least 2 operating turbines. Interviewed 2 weeks before move and 8, 12, 18, 21, and 25 months after move.

**Ongoing partial exposure for house maintenance, increased to many hours per week during winter 2 years after moving.

Case History C2 (page 1 of 2)

Person
Mrs. C

Age
42

Pre-exposure health status
Good

Health history

Migraine disorder, 6 healthy term pregnancies without hypertension

Previous noise exposure
No significant

Time to onset of symptoms
Immediate when first turbines operational, with progression

	Pre-exposure	During exposure*	Post-exposure**
Sleep	Good	Delayed onset, frequent awakening, hyperalert when awakened, nocturia; "no good rest in 10 months."	Resolved including nocturia
Headache	Migraine frequency varied, never awoke her at night; headache onset in childhood.	Headache onset day or night, 5–6 nights/week at maximum.	Resolved, no migraines
Cognition	Normal, very organized mother of 6 children, "ready a month in advance for birthday parties."	Disorganized; could not handle as many things at once; difficult to plan and track cooking; "I thought I was half losing my mind."	Resolved including ability to multitask
Mood	Good, lots of energy	Tired, anxious, irritable.	Improved, but still sadness and stress related to loss of home and living with parents
Balance/equilibrium	Lifelong motion sensitivity in cars, boats, swings, standing on wharf seeing boats go up and down. No vertigo.	Frequent dizziness, vertigo, and nausea preceding headaches.	At baseline
Ear/hearing	Normal hearing, no tinnitus	Tinnitus began when first 2 turbines operational; no change in hearing.	Hyperacusis
Eye/vision	Normal, no glasses	Nystagmus, subjective blurring	Persistent subjective blurring
Other neurological	Normal, no concussion	No change	No change

FOUR

The REPORT all over again, in plain English for non-clinicians



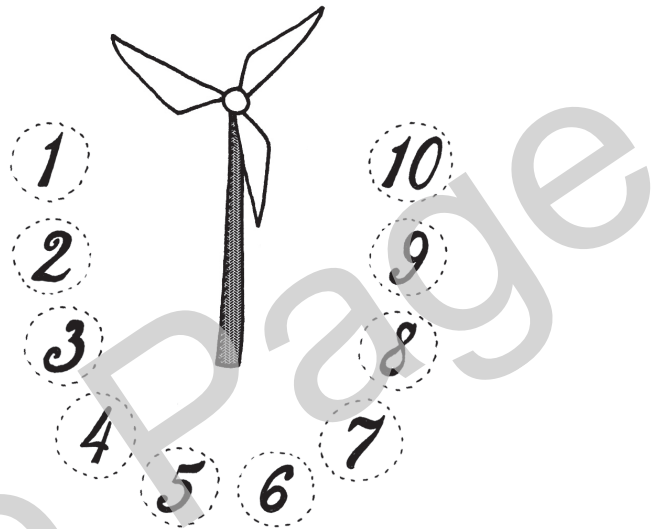
Abstract and Background

I interviewed 10 families living near large (1.5 to 3 MW) wind turbines, all of which were built since 2004. This gave me 38 people, from infants to age 75. Their symptoms formed a cluster. (See GLOSSARY for clinical terms.)

- 1) sleep disturbance
- 2) headache
- 3) tinnitus (pronounced “tin-uh-tus”: ringing or buzzing in the ears)
- 4) ear pressure
- 5) dizziness (a general term that includes vertigo, light-headedness, sensation of almost fainting, etc.)
- 6) vertigo (clinically, vertigo refers to the sensation of spinning, or the room moving)
- 7) nausea
- 8) visual blurring
- 9) tachycardia (rapid heart rate)
- 10) irritability

- 11) problems with concentration and memory
- 12) panic episodes associated with sensations of internal pulsation or quivering, which arise while awake or asleep

People in these families noticed that they developed these symptoms after the turbines started running near their homes. They noticed that when they went away, the symptoms went away. When they came back, the symptoms returned. Eight of the 10 families eventually moved away from their homes because they were so troubled by the symptoms, in some cases abandoning their homes.



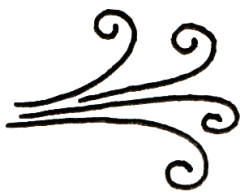
Hence the definitive result of my report is that wind turbines cause the symptoms of Wind Turbine Syndrome (WTS). I show this in the common-sense way described above.

Let's clarify something immediately. Not everyone living near turbines gets these symptoms. As a solo, unfunded researcher I could not get the samples needed to figure out what percentages of people at what distances get the symptoms. This needs to be done next. But I could (and did) look at the question of why some people are susceptible and others not, plus who is susceptible, and I used these patterns to explore the *pathophysiology of Wind Turbine Syndrome* (what's going on inside people to cause these specific symptoms).

I would like readers to look at this study—including the detailed accounts I provide of people's experiences around turbines and

their health backgrounds—and be able to make their own decisions about whether they should be exposed to these machines.

That said, I was able to prove mathematically that people with pre-existing migraines, motion sensitivity (such as car-sickness or seasickness), or inner-ear damage are especially vulnerable to these symptoms. Equally as interesting, I was able to demonstrate that people with anxiety or other pre-existing mental health problems are not especially susceptible to these symptoms.



This contradicts wind industry literature, which argues that people who worry about or otherwise dislike the turbines around their homes are the ones getting ill. I show this to be complete nonsense.

Here is what's going on, as I piece together the evidence. *Low frequency noise or vibration tricks the body's balance system into thinking it's moving.* Like seasickness. (It's vital to understand that the human balance system is a complex brain system receiving nerve signals from the inner ears, the eyes, muscles and joints, and inside the chest and abdomen. Because the eyes are involved, visual disturbance from the blades' shadow flicker adds to the balance disturbance.)

Let me repeat this, because its significance is huge. *Low frequency noise or vibration from turbines deceives the body into thinking it's moving.* So what, you say? Not so fast! Research within the last 10 years has demonstrated conclusively that *the way our bodies register balance and motion directly affects an astonishing array of brain functions.*

How? By direct neurologic linkages connecting the organs of balance to various, seemingly unrelated brain functions.

Glossary

A-weighting network: an electronic filter that reduces the contribution of low frequencies to a sound measurement; see pp. 36–38, 214–15.

Acute gastrointestinal infection: nausea, vomiting, abdominal pain, and diarrhea, generally self-limited and caused by a viral infection of the gastrointestinal tract.

Agoraphobia: an abnormal fear of leaving the house.

Air-conducted sound: sound that travels through the air and reaches the inner ear by way of the external auditory canal, tympanic membrane (eardrum) and the three ossicles of the middle ear. See *bone-conducted sound*.

Airways: trachea, bronchi, and bronchioles, the tubular structures through which air passes to reach the air sacs or alveoli of the lungs.

Amaurosis fugax: temporary loss of vision in one eye.

Analgesic: pain medication.

Anticoagulation: use of medications such as heparin or warfarin to decrease the tendency of the blood to clot. Higher INR (international normalized ratio of prothrombin time) values, used in the monitoring of warfarin administration, indicate slower or less effective clotting.

Antihypertensive: blood pressure medication.

Anxiolytic: anti-anxiety medication.

Arthralgia: joint pain without objective signs of inflammation (see *arthritis*).

Arthritis: pain and/or stiffness in joints with accompanying objective signs of inflammation, such as redness or swelling.

Asthma: intermittent and reversible respiratory difficulty caused by partial obstruction of small airways by inflammation/swelling and constriction of smooth muscle around the airways. Asthma

attacks may be provoked by any kind of respiratory infection, allergic exposures, or irritant exposures.

Ataxia, ataxic: in reference to gait, unsteady on feet, difficulty with balance or coordination in walking, or difficulty maintaining posture, for neurologic reasons.

Atrial fibrillation: an abnormal heart rhythm in which the small chambers do not pump rhythmically, but instead vibrate erratically, placing patients at risk for stroke from blood clots that can form inside the heart.

Autonomic nervous system: the involuntary part of the nervous system that regulates automatic body functions such as heart rate, blood pressure, gastrointestinal function, sweating, glandular output, pupillary reflexes, airway smooth muscle tone, and others. The autonomic system includes sensory receptors (for afferent signals or input to the central nervous system) and effector neurons (for efferent signals or output to organs). It consists of opposing sympathetic and parasympathetic networks. Sympathetic stimulation speeds the heart and readies the body for optimal “fight or flight” activity. Parasympathetic stimulation slows the heart, lowers blood pressure, and facilitates digestion.

Baroreceptors: pressure detectors, as in blood vessels or lungs.

Basilar migraine: migraine with auras representing brainstem effects, including vertigo, tinnitus, fluctuations in level of consciousness, and temporary motor deficits.

Bilateral: on both sides of the body.

Binaural processing: brain integration of hearing signals from both ears.

Bone-conducted sound: sound or vibratory stimulus reaching the inner ear via direct solid-to-solid and solid-to-fluid transmission, without passing through or utilizing the tympanic membrane or middle ear ossicles. It is created by placing a vibrating object against the skin over a skull bone,

typically the mastoid process immediately behind the ear. See *air-conducted sound*.

Bronchodilator: medication used to relax airway smooth muscle in the treatment of asthma, usually inhaled.

C-weighting network: an electronic filter that reduces the contribution of very low frequencies to a sound measurement, but less so than an A-weighting network; see pp. 36–38, 214–215.

Caloric test: a test of semicircular canal function and the vestibulo-ocular response. In the caloric response to ice water in the external auditory canal, thermal convection induces fluid movement within the horizontal semicircular canal, creating an illusion of head movement that is reflected in eye movement via the vestibulo-ocular reflex.

Cardiac arrhythmia or dysrhythmia: specific types of irregular heartbeat, often occurring episodically.

Catecholamine: a class of biochemicals that function as neurotransmitters in the brain and as hormones produced by the sympathetic part of the autonomic nervous system, such as epinephrine (adrenaline), norepinephrine, and dopamine.

Central: occurring in the brain (central nervous system), as opposed to a peripheral neural receptor, effector, or organ. For example, central processing, central origin, central dysfunction.

Cerebellum, cerebellar: a posterior/inferior portion of the brain with important functions in coordination and integration of movement.

Cerebrospinal fluid: clear fluid that circulates from fluid spaces (lateral ventricles) in the brain, where it is produced, through the other ventricles and around the brain and spinal cord.

Chemotherapy: in this report, refers specifically to medications given for cancer treatment.

Cilium, cilia: actively motile, hair-like projections from epithelial cell surfaces in the airways and Eustachian tubes that beat in

synchrony to move mucus out of these moist, air-filled spaces, towards the pharynx.

Circadian rhythm: a daily physiologic cycle, such as sleep and wakefulness or the peaks and troughs of cortisol secretion.

Cochlea: spiral-shaped sensory organ of hearing, part of the inner-ear membranous labyrinth. See pp. 200–201.

Collagen: a protein which is the chief substance of connective tissue, cartilage, tendons, etc.

Concussion: mild brain injury produced by impact to the head resulting in brief unconsciousness, disorientation, or memory problem.

Conductive hearing loss: hearing loss due to problems in the outer ear, tympanic membrane, or middle ear.

Coronary artery disease: partial obstruction or narrowing of the small arteries that supply the heart muscle.

Cortex, cortical: the outer cellular layers of the two cerebral hemispheres of the brain.

Cortisol: a major natural glucocorticoid hormone produced by the adrenal cortex in a regular daily rhythm and in response to stress, which exerts diverse effects on tissues and metabolic processes throughout the body.

Cranial vault: the space in the skull that contains the brain.

Diaphragm: the dome-shaped sheet of skeletal muscle that separates the thoracic (chest) and abdominal cavities and enables breathing.

Dysfunction: malfunction or poor functioning.

Elastin: an elastic connective tissue protein, which gives elasticity to certain structures, such as arterial walls.

Electroencephalogram (EEG): a recording of brain waves monitored in a specific fashion, used in studies of seizure disorder and sleep.

Endolymphatic hydrops (EH): a condition of distorted fluid and pressure relationships between the endolymph and perilymph, which are the two fluid compartments in the inner ear. This

causes erratic and distorted balance and, often, hearing signals to be sent to the brain. Meniere's disease and perilymphatic fistula are examples of conditions with endolymphatic hydrops.

Epithelial basement membrane: a thin layer of extracellular proteins and mucopolysaccharides that lies at the base of and supports the layers of cells comprising an epithelium, such as the linings of airways, mouth, esophagus, intestine, pleura, etc.

Eustachian tube: a tube that connects the middle ear with the nasopharynx, or upper part of the throat behind the nose. It allows equalization of air pressure on either side of the tympanic membrane.

Fibromyalgia: a condition of chronic pain of unclear origin, in muscles, ligaments, and tendons, without inflammation.

Gastritis: inflammation of the lining of the stomach causing pain and nausea.

Gastroesophageal reflux (GER): reflux or intrusion of acidic stomach contents into the esophagus; heartburn.

Gastrointestinal (GI) tract: stomach, small intestine, and colon or large intestine.

Glucose instability: in diabetes, fluctuating blood sugar levels that go too high or too low.

Glucosuria: glucose in urine, a sign of poor diabetic control.

Graviceptors: neural detectors of gravity and acceleration; see pp. 73–74, 234–35.

Great vessels: the large arteries and veins immediately around the heart, including the aorta, pulmonary artery, pulmonary veins, and superior and inferior vena cavae.

Hair cells: mechanoreceptive cells in the inner-ear labyrinthine organs (cochlea, semicircular canals, utricle, and saccule). These cells send neural signals when mechanically perturbed or bent. Local properties of parts of the membranous labyrinth control how the hair cells are perturbed.

Hippocampus: a brain region in the medial temporal lobe critical to spatial navigation and formation of new episodic memories.

Hyperacusis: oversensitivity to sound, with normal sounds seeming painfully loud.

Hypertension: high blood pressure.

Hypopharynx: the lower part of the throat, just above the larynx (vocal cords).

Immissions: in acoustics, sound from the point of view of the person or location receiving the sound. *Emissions* in this context refers to the sound as it leaves the source.

In utero: in the uterus during pregnancy.

Infrasonic: sound frequency below hearing range, generally considered to be 20 Hz or less.

Irritable bowel syndrome: recurrent episodes of abdominal pain and diarrhea, often with alternating periods of constipation, without any pathologic or inflammatory changes in the gastrointestinal tract.

Labyrinthine organs, membranous labyrinth: the inner-ear organs, including the cochlea, utricle, saccule, and semicircular canals. See *otolith organs* and *semicircular canals*, and pp. 200–201.

Lower respiratory infection: bronchitis, pneumonia, or pneumonia with pleural effusion (pleurisy).

Lupus: systemic lupus erythematosus, a systemic inflammatory or autoimmune disease affecting the skin, joints, gastrointestinal tract, kidney, blood, and brain.

Macula: in the otolith organs (utricle and saccule), the patch of sensory hair cells plus superimposed mass of otoconia in a protein matrix (sometimes called *macule*). See p. 200.

Magnetic resonance angiography (MRA): a noninvasive imaging method for examining the patency of blood vessels.

Magnetic resonance imaging (MRI): soft-tissue imaging using magnetic fields, providing the most detailed images of living brain structure available. Functional magnetic resonance imaging (fMRI) quantifies blood flow to different brain structures during specific activities.

Malaise: a vague sense of not feeling well.

Mastoid: a bony structure immediately behind the ear that contains air-filled cells connected to the middle ear.

Mediastinum: the central portion of the chest or thorax between the lungs, containing the heart, great vessels, trachea, esophagus, lymph nodes, and other structures.

Mesentery: a fold of membranous tissue encasing and attaching the small intestine and other abdominal organs to the inside of the peritoneal (abdominal) cavity, also supporting blood vessels and nerves to the organs.

Microvilli: hair-like projections from epithelial cell surfaces that increase absorptive surface area, for example, in the small intestine.

Migraine: a hereditary, episodic, neurologic condition generally involving severe headaches that may be preceded by visual or other sensory phenomena such as tingling or numbness (aura), with symptoms of nausea and sensitivity to light and sound commonly accompanying headaches. A headache may be one-sided or pounding. Aura and accompanying symptoms may include vertigo, tinnitus, temporary focal weakness or paralysis, temporary loss of vision, vomiting, or loss of consciousness. Sensory sensitivities and triggers include motion, odors, a wide variety of foods (especially products of fermentation or aging, caffeine, chocolate, and varieties of plants), hormonal state, and sleep deprivation.

Migraineur: a person who gets migraines.

Myocardial infarction (MI): heart attack, or obstructed coronary blood flow leading to death of cardiac muscle.

Near-field sound: sound at distances significantly less than one wavelength, especially applicable to hearing under water (e.g., in fish), where wavelengths of sound are much longer than in air (by a factor of 5 at the same frequency), and for lower sound frequencies (which have longer wavelengths in any medium). Near-field sound detection involves detection of particle movement or bulk flow of the medium, rather than a repetitive

pressure fluctuation as for *far-field sound* detection in air, for which the mammalian ear and cochlea are specialized.

Neuroanatomic: referring to the anatomy of neural linkages in the brain.

Neuroendocrine: relating to cells or tissues that release hormones into the blood in response to a neural stimulus.

Night terror: a parasomnia, or sleep disturbance occurring during disordered arousal from the deeper stages of sleep, in which a person (usually a child) may scream, act afraid, say nonsensical things, or get up to do irrational or fearful things, all without memory in the morning.

Nocturia: awakening and getting up repeatedly in the night to urinate.

Nocturnal enuresis: bed-wetting while asleep.

Norepinephrine: a central catecholamine neurotransmitter, sympathetic nervous system neurotransmitter, and vasoactive adrenal medullary hormone.

Nystagmus: a pattern of eye movement indicating a disordered vestibulo-ocular reflex that is often due to disordered vestibular signaling or processing, as in the caloric test.

Orbit: the eye socket or hollow space in the skull that contains the eyeball and its associated structures.

Otitis media: middle-ear infection.

Otoconia: microscopic calcium carbonate stones positioned in a protein matrix over the mechanically sensing hair cells of the mammalian utricle and saccule.

Otolith organs: the utricle and saccule, labyrinthine organs of the inner ear that detect linear acceleration, including gravity, by virtue of microscopic calcium carbonate stones or *otoconia* positioned in a protein matrix over the mechanically sensing hair cells. See pp. 200–201.

Palpitations: irregular or pounding heart at times not expected from activity or exertion.

Panic attack: an episode of sudden intense fear out of proportion to circumstances, which may be accompanied by symptoms of dizziness, sweating, trembling, chest pain, palpitations, and the feeling of not being able to get enough breath.

Parabrachial nucleus: brain center involved in extended vestibular system influence, located in the pons.

Parasomnia: a sleep disturbance occurring during disordered arousal from the deeper stages of sleep, such as sleep walking, sleep talking, and night terrors.

Paresthesia: tingling or “pins and needles” sensation, as when a numb extremity is waking up.

Parkinson’s disease: a neurologic degenerative disease involving dopamine-producing neural tracts in the brain and affecting movement and psychiatric status.

Pericardium: the two-layered membranous sac that encloses the heart and the roots of the great vessels, in which the heart beats.

Perilymphatic fistula syndrome: see *endolymphatic hydrops* and pp. 93, 227.

Pharynx: the throat.

Pleura: the outer epithelial surface of the lung and the lining of the thoracic cavity, providing low friction surfaces for lung movement.

Pleurisy: inflammation or infection of pleura, which can accompany pneumonia.

Polyuria: excessive daily volume of urine, a typical sign of high glucose levels in diabetics.

Positron emission tomography (PET): a method of functional imaging that quantifies glucose uptake by different brain regions as a measure of activity.

Posturography: a form of balance testing that is sensitive to the vestibulo-spinal reflexes, including the influence of inner-ear, visual, somatosensory, and central processing on the movements by which a subject remains balanced and upright.

Pressure equalization (PE) tube: a tube inserted through a small, surgically placed hole in the tympanic membrane after removal of middle-ear fluid, to provide aeration.

Proton pump inhibitor: medication used to limit stomach acid production in the treatment of gastroesophageal reflux, gastritis, or ulcer.

Resonance: a property of sound; see pp. 36, 211–14.

Retina, retinal: the light-sensing neural structure at the back of the eye.

Sacculae: one of the two otolith organs of the vestibular (balance) organs of the inner ear (also called sacculus).

Scotoma: temporary loss of vision in one part of the visual field.

Semicircular canals: bilateral labyrinthine organs of the inner ear that detect angular acceleration of the head by virtue of fluid shifts deflecting mechanically sensing hair cells. See pp. 200–201 and *caloric test*.

Sensorineural hearing loss: hearing loss due to problems in the inner ear/cochlea, vestibulocochlear nerve (cranial nerve VIII), or brain centers that process sound.

Sequela, sequelae: a pathologic condition that develops from another pathologic condition, such as chronic middle-ear fluid and hearing loss being sequelae of repeated acute ear infections.

Serotonin: a brain and gastrointestinal neurotransmitter.

Serous otitis media: viscous fluid in the middle ear (middle-ear effusion) that may obstruct sound transmission, usually occurring after a series of acute ear infections.

Somatic nervous system: the sensory and motor nervous system from and to the skin, skeletal muscles, and associated tendons and ligaments, whose signals may be consciously perceived and voluntarily modified.

Somatosensory: sensory input from the skin, skeletal muscles, tendons, and ligaments.

Sonic: sound frequency in the range of human hearing.

Syncope, syncopal: fainting caused by low blood flow to brain.

Tachycardia: rapid heartbeat.

Taxon, taxa: a group or groups in the scientific categorization (Linnaean taxonomy) of living things.

Temporal bone: solid bone at the base of the skull, in which the labyrinthine organs lie.

Thalamus: a part of the brain involved in part in relaying sensory information to the cerebral cortex.

Tinnitus: “ringing in the ears,” which may be a tonal sound, buzzing, white noise, or other types of sound heard in one or both ears. The sound itself is not present in the outside environment.

Trachea: the large central airway between the larynx (voice box) and the split or bifurcation of the right and left bronchi.

Tympanic membrane: eardrum; the layer of taut, thin tissue that separates the external auditory canal from the middle ear.

Ultrasonic: sound frequency above hearing range, generally considered to be 20,000 Hz or more.

Upper gastrointestinal symptoms: gastroesophageal reflux, gastritis, and/or ulcer.

Utricle: one of the two otolith organs of the vestibular (balance) organs of the inner ear (also called utriculus).

Vasculitis: inflammation of blood vessels, which can cause restriction of blood flow.

Vasoconstriction: constriction of a blood vessel.

Vertigo: the spinning form of dizziness, in which the visual surround seems to move.

Vestibular: pertaining to the balance organs in the inner ear (utricle, saccule, and semicircular canals) or to the integrated balance system in general, as in “vestibular areas of the brain.”

Vestibular evoked myogenic potential (VEMP): a vestibular reflex neural response, used clinically and in research to test specifically for otolith function or stimulation. Ocular vestibular evoked myogenic potential (OVEMP) is similar. See pp. 85–86, 203.

Vestibulo-colic reflex: a fast or “short-latency” neural response across a short, three-neuron brain arc from the otolith organs to brainstem vestibular nuclei to brain nuclei controlling the muscles of the neck to neck muscles, whose purpose is immediate, automatic stabilization of the head in response to detected motion.

Vestibulo-ocular reflex: a fast or “short-latency” neural response across a short, three-neuron brain arc from the semicircular canals and otolith organs to brainstem vestibular nuclei to brain nuclei controlling extraocular eye muscles to eye muscles, whose purpose is immediate, automatic compensatory movements of the eyes in response to detected head motion, to stabilize the visual field during movement.

Vestibulo-spinal reflex: like the vestibulo-colic reflex but involving muscles below the neck (along the spinal column and in the legs) to stabilize posture during movement and rapidly correct potential falls.

Vibroacoustic disease (VAD): a type of noise-related illness. See pp. 109–11.

Visceral Vibratory Vestibular Disturbance (VVVD): a sensation of internal quivering, vibration, or pulsation accompanied by agitation, anxiety, alarm, irritability, rapid heartbeat, nausea, and sleep disturbance. See pp. 55–60, 76–79, 224, and 235–36.

Whiplash injury: an injury to the neck (cervical vertebrae) caused by abrupt acceleration or deceleration, as in an automobile accident.

References

Académie nationale de médecine de France. 2006. “Le retentissement du fonctionnement des éoliennes sur la santé de l’homme, le Rapport, ses Annexes et les Recommandations de l’Académie nationale de médecine.” 17 pp. www.academie-medecine.fr/sites_thematiques/EOLIENNES/chouard_rapp_14mars_2006.htm.

Ahlbom IC, Cardis E, Green A, Linet M, Savitz D, Swerdlow A; INCIRP (International Commission for Non-Ionizing Radiation Protection) Standing Committee on Epidemiology. 2001. Review of the epidemiologic literature on EMF and health. *Environ Health Perspect* 109 Suppl 6: 911–33.

Babisch W. 2003. Stress hormones in the research on cardiovascular effects of noise. *Noise Health* 5(18): 1–11.

Babisch W. 2005. Guest editorial: Noise and health. *Environ Health Perspect* 113(1): A14–15.

Babisch W, Beule B, Schust M, Kersten N, Ising H. 2005. Traffic noise and risk of myocardial infarction. *Epidemiology* 16(1): 33–40.

Baerwald EF, d’Amours GH, Klug BJ, Barclay RM. 2008. Barotrauma is a significant cause of bat fatalities at wind turbines. *Curr Biol* 18(16): R695–96.

Balaban CD. 2002. Neural substrates linking balance control and anxiety. *Physiol Behav* 77: 469–75.

Balaban CD. 2004. Projections from the parabrachial nucleus to the vestibular nuclei: potential substrates for autonomic and limbic influences on vestibular responses. *Brain Res* 996: 126–37.

Balaban CD, Thayer JF. 2001. Neurological bases for balance-anxiety links. *J Anxiety Disord* 15: 53–79.

Balaban CD, Yates BJ. 2004. The vestibuloautonomic interactions: a teleologic perspective. Chapter 7 in *The Vestibular System*, ed. Highstein SM, Fay RR, Popper AN, pp. 286–342. Springer-Verlag, New York.

Baron, Robert Alex. 1970. *The Tyranny of Noise: The World's Most Prevalent Pollution, Who Causes It, How It's Hurting You, and How to Fight It*. St. Martin's Press, New York.

Beasley R, Clayton T, Crane J, von Mutius E, Lai CK, Montefort S, Stewart A; ISAAC Phase Three Study Group. 2008. Association between paracetamol use in infancy and childhood, and the risk of asthma, rhinoconjunctivitis, and eczema in children aged 6–7 years: analysis from Phase Three of the ISAAC programme. *Lancet* 372(9643): 1039–48.

Beranek LL. 2006. Basic acoustical quantities: levels and decibels. Chapter 1 in *Noise and Vibration Control and Engineering: Principles and Applications*, ed. Ver IL, Beranek LL, pp. 1–24. John Wiley & Sons, Hoboken, NJ.

Berglund B, Hassmen P, Job RFS. 1996. Sources and effects of low frequency noise. *J Acoust Soc Am* 99(5): 2985–3002.

Brandt T, Bartenstein P, Janek A, Dieterich M. 1998. Reciprocal inhibitory visual-vestibular interaction. Visual motion stimulation deactivates the parieto-insular vestibular cortex. *Brain* 121(Pt. 9): 1749–58.

Brandt T, Dieterich M. 1999. The vestibular cortex: its locations, functions, and disorders. *Ann NY Acad Sci* 871: 293–312.

Brandt T, Schautzer F, Hamilton DA, Bruning R, Markowitsch HJ, Kalla R, Darlington C, Smith P, Strupp M. 2005. Vestibular loss causes hippocampal atrophy and impaired spatial memory in humans. *Brain* 128: 2732–41.

Cappa S, Sterzi R, Vallar G, Bisiach E. 1987. Remission of hemineglect and anosognosia during vestibular stimulation. *Neuropsychologia* 25: 775–82.

Castelo Branco NAA. 1999. A unique case of vibroacoustic disease: a tribute to an extraordinary patient. *Aviat Space Environ Med* 70(3): A27–31.

Castelo Branco NAA, Aguas AP, Pereira AS, Monteiro E, Fragata JIG, Tavares F, Grande NR. 1999. The human pericardium in vibroacoustic disease. *Aviat Space Environ Med* 70(3): A54–62.

Castelo Branco NAA, Alves-Pereira M. 2004. Vibroacoustic disease. *Noise Health* 6(23): 3–20.

Castelo Branco NAA, Monteiro M, Ferreira JR, Monteiro E, Alves-Pereira M. 2007. Bronchoscopy in vibroacoustic disease III: electron microscopy. *Inter-Noise 2007*, August 28–31, Istanbul, Turkey.

Clark C, Martin R, van Kempen E, Alfred T, Head J, Davies HW, Haines MM, Barrio IL, Matheson M, Stansfeld SA. 2005. Exposure-effect relations between aircraft and road traffic noise exposure at school and reading comprehension: the RANCH project. *Am J Epidemiol* 163: 27–37.

Claussen CF, Claussen E. 1995. Neurootological contributions to the diagnostic follow-up after whiplash injuries. *Acta Otolaryngol Suppl* 520, Pt. 1: 53–56.

Coermann RR, Ziegenruecker GH, Wittwer AL, von Gierke HE. 1960. The passive dynamic mechanical properties of the human thorax-abdominal system and of the whole body system. *Aerosp Med* 31(6): 443–55.

Cohen S, Glass DC, Singer JE. 1973. Apartment noise, auditory discrimination, and reading ability in children. *J Exp Soc Psychol* 9: 407–22.

Colebatch JG, Day BL, Bronstein AM, Davies RA, Gresty MA, Luxon LM, Rothwell JC. 1998. Vestibular hypersensitivity to clicks is characteristic of the Tullio phenomenon. *J Neurol Neurosurg Psychiatry* 65: 670–78.

Colebatch JG, Halmagyi GM, Skuse NF. 1994. Myogenic potentials generated by a click-evoked vestibulocollic reflex. *J Neurol Neurosurg Psychiatry* 57(2): 190–97.

Curthoys IS, Kim J, McPhedran SK, Camp AJ. 2006. Bone conducted vibration selectively activates irregular primary otolithic vestibular neurons in the guinea pig. *Exp Brain Res* 175(2): 256–67.

Dieterich M, Brandt T. 2008. Functional brain imaging of peripheral and central vestibular disorders. *Brain* 131(10): 2538–52.

Eckhardt-Henn A, Breuer P, Thomalske C, Hoffmann SO, Hopf HC. 2003. Anxiety disorders and other psychiatric subgroups in patients complaining of dizziness. *J Anxiety Disord* 17(4): 369–88.

Edge PM, Mayes WH. 1966. Description of Langley low-frequency noise facility and study of human response to noise frequencies below 50 cps. NASA Technical Note, NASA TN D-3204, 11 pp.

Eriksson C, Rosenlund M, Pershagen G, Hilding A, Ostenson C-G, Bluhm G. 2007. Aircraft noise and incidence of hypertension. *Epidemiology* 18(6): 716–21.

Ernst A, Basta D, Seidl RO, Todt I, Scherer H, Clarke A. 2005. Management of posttraumatic vertigo. *Otolaryngol Head Neck Surg* 132(4): 554–58.

Evans GW, Maxwell L. 1997. Chronic noise exposure and reading deficits: the mediating effects of language acquisition. *Environ Behav* 29(5): 638–56.

Evans GW. 2006. Child development and the physical environment. *Annu Rev Psychol* 57: 423–51.

Fay RR, Simmons AM. 1999. The sense of hearing in fishes and amphibians. In *Comparative Hearing: Fish and Amphibians*, ed. Fay RR, Popper AN, pp. 269–317. Springer-Verlag, New York.

Feldmann J, Pitten FA. 2004. Effects of low-frequency noise on man: a case study. *Noise Health* 7(25): 23–28.

Findeis H, Peters E. 2004. Disturbing effects of low-frequency sound immissions and vibrations in residential buildings. *Noise Health* 6(23): 29–35.

Foudriat BA, Di Fabio RP, Anderson JH. 1993. Sensory organization of balance responses in children 3–6 years of age: a normative study with diagnostic implications. *Int J Pediatr Otorhinolaryngol* 27(3): 255–71.

Frey, Barbara J, and Hadden, Peter J. 2007. Noise radiation from wind turbines installed near homes: effects on health. 137 pp. www.windturbinenoisehealthhumanrights.com/wtnhhr_june2007.pdf.

Furman JM, Balaban CD, Jacob RG. 2001. Interface between vestibular dysfunction and anxiety: more than just psychogenicity. *Otol Neurotol* 22(3): 426–27.

Furman JM, Balaban CD, Jacob RG, Marcus DA. 2005. Migraine-anxiety related dizziness (MARD): a new disorder? *J Neurol Neurosurg Psychiatry* 76: 1–8.

Furman JM, Redfern MS, Jacob RG. 2006. Vestibulo-ocular function in anxiety disorders. *J Vestib Res* 16: 209–15.

Garcia J, Ervin FR. 1968. Gustatory-visceral and telereceptor-cutaneous conditioning: adaptation in internal and external milieus. *Commun Behav Biol* 1: 389–415.

Geminiani G, Bottini G. 1992. Mental representation and temporary recovery from unilateral neglect after vestibular stimulation. *J Neurol Neurosurg Psychiatry* 55(4): 332–33.

About the author

I am a New Englander by many generations, growing up in a family of teachers and writers. My grandfather, like me, was a physician and ecologist. After being blessed by a fine elementary school (New Canaan Country School, 1970) and high school (Milton Academy, 1973), I attended Yale on a National Merit Scholarship, graduating in 1977 with a BA in biology. I earned a PhD (1985) in behavioral ecology at Princeton (training that I use substantially in my work in behavioral pediatrics), did a post-doctoral fellowship in ornithology at the American Museum of Natural History (NYC), and, as an over-the-hill woman of thirty-two, went to the Johns Hopkins University School of Medicine, where I earned the MD degree (1991).

I wanted to give my ecology training a human face. I chose the face of a child, becoming a pediatrician by completing internship at the Children's National Medical Center, Washington, DC, and residency at the Dartmouth-Hitchcock Medical Center, Lebanon, NH (because my husband, a country lad, detested Washington).

Despite his feelings toward Washington, and his improbable name (Calvin Luther Martin), my husband is a respectable man (retired Rutgers University professor and author of well-known scholarly books). Our two children (my stepchildren) are grown and have made us grandparents.

I am 54 years old.

I am an unabashed lover of wildness. I did my PhD research living in a tent in the Amazon jungle for several years, studying bird behavior. In pursuit of wildness and native cultures, my husband and I lived for another several years with Yup'ik Eskimos on the

Alaska tundra, near the Bering Sea, where I became chief of pediatrics at a native-run hospital. Likewise, we spent a summer living on the Navajo reservation, as I did a sub-internship in medical school.

For three years I ran a general pediatrics practice in Malone, Franklin County, NY (poorest county in the state), where I was, as well, the pediatrician for the St. Regis Mohawk Nation (Hogansburg, NY). For the next three years (2000–03) I was Senior Attending in Pediatrics at Bassett Healthcare, Cooperstown, NY (and, must confess, never darkened the door of the Baseball Hall of Fame). Bassett is a teaching hospital of Columbia University, and I was Assistant Clinical Professor of Pediatrics at Columbia's College of Physicians & Surgeons.

I am a board-certified pediatrician licensed in the State of New York and Fellow of the American Academy of Pediatrics. These days I limit my practice to behavioral medicine, seeing both adults and (chiefly) children, drawing my patients from an extensive area of rural upstate New York. I have had considerable post-graduate training in behavioral medicine, which I have been able to integrate with my doctoral training in behavioral ecology.

My research on Wind Turbine Syndrome is the offspring of behavioral medicine married to behavioral ecology.

Most of all, I love what I do. I believe in compassion and grace and get tremendous pleasure and joy out of my patients. (To children's delight, I carefully count their toes.) I run my practice out of my home as an old-fashioned doctor's office. Cheerful, light, airy, perhaps the faint smell of my husband's burnt toast wafting through the house. Norman Rockwell's America.