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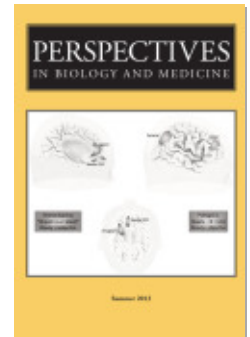
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## Was It Epilepsy?: Misdiagnosing Emily Dickinson (1830–1886)

Norbert Hirschhorn, Polly Longworth

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# WAS IT EPILEPSY?

*misdiagnosing Emily Dickinson*  
(1830–1886)

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NORBERT HIRSCHHORN\* AND POLLY LONGSWORTH†

**ABSTRACT** Lyndall Gordon's recent biography, *Lives Like Loaded Guns: Emily Dickinson and Her Family's Feuds* (2010), tells with high verve the story of generational infighting over poet Emily Dickinson's posthumous presentation to the world. Equally dramatic is Gordon's hypothesis that Dickinson suffered from epilepsy, which led Gordon to seemingly solve the ineffable mystery of Dickinson's reclusion, a conundrum in her own time and still so in ours. Gordon's startling diagnosis has been commended by book reviewers and on talk shows. Her hypothesis is based on two lines of inquiry. First, she avers that a compound called glycerine, which Dickinson took regularly in the early 1850s, was an anti-epileptic, basing this notion on its presence in a mixture containing the soporific chloral hydrate, a prescription first advised for epilepsy some two decades later. Second, Gordon proposes a genetic strain of epilepsy in the Dickinson family. In the process, Gordon recruits Dickinson's various illnesses to her hypothesis. This article refutes Gordon's claims on scientific, clinical, and biographical grounds. It reviews Dickinson's medical history to establish a differential diagnosis, in which epilepsy is considered and rejected.

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\*Public health physician, poet, and medical historian.

†Biographer whose work includes books on Emily Dickinson and her family.

Correspondence: Norbert Hirschhorn, 115 Greencroft Gardens, London, NW6 3PE, United Kingdom.

E-mail: bertzpoet@yahoo.com.

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In memory of Emily Dickinson scholar Dorothy Oberhaus. Like the poet, her Country was Truth.

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**FIGURE 1**

*Daguerrotype portrait of Emily Dickinson, c. 1847*

SOURCE: AMHERST COLLEGE ARCHIVES AND SPECIAL COLLECTIONS.

MANY BIOGRAPHERS OF Emily Dickinson (1830–1886) have attempted to explain her hermetic life and often enigmatic poetry by proposing one or another medical condition; included among these are strabismus, lupus erythematosus, agoraphobia, seasonal affective disorder, and schizophrenia. (Figure 1). It is unlikely that such a reductive approach can succeed, given the complex interactions of heritage, nurture, environment, and temperament (Hirschhorn and Longworth 1996). Lyndall Gordon (2010) joins earlier Dickinson biographers in advancing yet another single diagnosis, epilepsy, to try to solve the mysteries, by drawing principally on two lines of evidence: a medication Dickinson took, and a family history of seizure.

### THE MEDICATIONS

As detailed previously in an article on Emily Dickinson and tuberculosis, the poet indeed took multiple doses of glycerine between 1851 and 1854 as treatment for suspected tuberculosis (Hirschhorn 1999). In her teenage years, she had suffered from bouts of “severe cough . . . & general debility” (Dickinson

1958, Letter [L]13), causing her twice to drop out of school. As she entered her 20s, she was losing weight and feeling ill again. With a strong history of consumption on her mother's side, and the epidemic then raging in the Connecticut Valley where she lived, Dickinson's parents had cause to be alarmed. They consulted several physicians, one after another, finally appealing to Dr. James Jackson, the dean of physicians of Boston, co-founder of the Massachusetts General Hospital, and Harvard professor of medicine.

In the 19th century's medical theory, heritability was the predisposing soil for tuberculosis, and stress and under-nutrition its seeds. In Jackson's medical writings, he prescribed exercise, fresh air, nutrition, and also cod-liver oil as a supplemental restorative for suspected tuberculosis (Jackson 1855, 1861). Cod-liver oil, for those of us old enough to remember, is foul tasting, and it may have been the medicine with which her father unmercifully dosed Emily for a respiratory illness in 1848 (Dickinson 1958, L23). When sweet glycerine arrived, it was a welcome replacement. Jackson prescribed glycerine for Dickinson, and the prescription was filled at least nine times between September 1851 and December 1853, five of those times in double or triple quantity during the first seven months after Dickinson's single consultation. It is certain that the prescription was for Dickinson's suspected tuberculosis, not epilepsy, for she advised her brother Austin in March 1854, just three months after her last prescription was filled, "And if the cough troubles you follow my prescription, and it will soon get well" (Dickinson 1958, L156).

Glycerine is a sweet syrup derived from animal fat or vegetable oils, first prepared by a lead-based extraction process in 1845. It is one of the earliest medications derived chemically and was initially used as an emollient and antiseptic lotion. By 1850, given its properties, it began to be prescribed as a cough remedy and nutritional supplement for treating tuberculosis (see below). Glycerine also served for decades to disguise the taste of bitter medicines. It is still in wide use.

What Gordon found in her researches was a formula published in a popular self-help manual, published by William Witty Hall in 1874. Among dozens of uses for glycerine was its inclusion in a compound for epilepsy calling for "half an oz. of hydrate of chloral and twenty five drops of essence of peppermint in four oz. of pure glycerine" (Hall 1874, p. 739). Gordon (2010) comments: "Glycerine has many uses, but one of the medical uses in those days was for epilepsy. . . . This use of glycerine in the treatment of epilepsy (as distinct from its use for TB) has gone unnoticed" (p. 121.) The 1874 formula, however, bears no relation to what Dickinson was given 23 years earlier.

The claim for glycerine as an anti-epileptic at any time is unsupported by the evidence. Glycerine was never identified as an active principle against epilepsy in any official pharmacopoeia, dispensatory, or textbook on epilepsy written in the 19th century that we could locate. The active ingredient in Hall's formula for epilepsy was, in fact, chloral hydrate, first reported and used as a sleeping medication in 1869. It is acrid to the smell and taste and irritating to the stomach.

Thus it requires to be given with various syrups and sweeteners to disguise the taste; peppermint was included to soothe the stomach. Although mainly a hypnotic, chloral also had application against a range of convulsive disorders, which included tetanus, chorea, rabies, strychnine poisoning, toxemia of pregnancy, and alcoholic delirium tremens. It is still indicated for the latter, but much less commonly used (see SIN Foundation 2003–2012). The following provides detailed documentation on uses for both drugs in the mid- to late-1800s.

### GLYCERINE

The authoritative *Dispensatory of the United States of America* ran from 1833 to 1955 in many editions. Glycerine was first mentioned in the 1849 eighth edition as an external remedy for skin diseases. Its uses as an emollient and nutrient are mentioned in the 1858 edition:

Glycerin possesses extensive powers as a solvent and is an excellent excipient for many medicinal substances. . . . Employed internally as a therapeutic agent, it is deemed alterative, nutrient, and demulcent. . . . Dr. J.L. Crawcour, of New Orleans, has used it with supposed advantage in phthisis [tuberculosis], and prefers it to cod-liver oil. Dr. W. Lauder Lindsay made experiments with it, to determine its alterative and nutrient properties, and found it to increase the weight of the body. Some cases are cited by him, in which it appeared to act beneficially in tuberculosis and strumous affections [swellings often related to tuberculosis]. . . .

[It forms] a useful succedaneum for cod-liver oil, when the latter could not be borne by the stomach. (Wood and Bache 1858, pp. 1078–81)

Glycerine's use against tuberculosis fell out of favor toward the end of the century: "Although at various times much lauded in tuberculous diseases and in diabetes, [glycerine] has entirely failed to gain the confidence of the profession, and is now very rarely employed" (Wood, Remington, and Sadtler 1899, p. 660). Squire's 1864 edition of the *Companion to the Latest Edition of the British Pharmacopoeia* states: "Internally [glycerine] is nutrient and demulcent. It has been proposed as a substitute for Cod Liver Oil, but its nutrient properties are far inferior. . . . As an external remedy, however, it is highly valued, chiefly for its emollient and undrying properties. . . . It possesses great powers as a solvent, and is an excellent excipient for many substances" (pp. 92–93, 137).

In none of these texts is glycerine ever mentioned as a medication to treat or prevent epilepsy. We also examined textbooks and monographs of the time on epilepsy, searching for any mention of glycerine as a treatment for the disease. These included monographs by Edward Henry Sieveking (also cited by Gordon). In the first edition of *On Epilepsy and Epileptiform Seizures* (1858), 58 patients are presented; none received glycerine. In an appendix to Sieveking's later edition (1861) are listed 34 different formulae for treatment of epilepsy;

none contains glycerine. William Alexander (1889) reviewed treatments for epilepsy going back to 1831. None included glycerine. Henry Hartshorne, Professor of Theory and Practice of Medicine at Pennsylvania College, wrote a monograph in 1865 on glycerine and its uses, which makes no mention of epilepsy but does commend glycerine for a variety of skin conditions and notes its use in tuberculosis, principally as an expectorant. Walter Friedlander's (2001) history of epilepsy in the 19th and 20th centuries makes no mention of glycerine as a treatment, nor does Sir William Osler's (1892) magisterial textbook of medicine. The *Boston Medical and Surgical Journal*, forerunner to the *New England Journal of Medicine*, published in installments William M. Cornell's treatise, *Observations on Epilepsy* (1854). While eschewing bleeding and purges, he advised ancillary treatments with a variety of herbal sedatives and stimulants; glycerine was not on his list. The eminent neurologist of his time, C. E. Brown-Séquard, also makes no mention of glycerine in his chapter on epilepsy in a textbook of medicine owned by Emily Dickinson's physician, O. F. Bigelow (Quain 1883).

Finally, Dr. James Jackson's chapter on epilepsy in his *Letters to a Young Physician Just Entering Upon Practice* (also cited by Gordon) makes no mention of glycerine as a remedy for epilepsy. In fact, he writes: "I believe that, in a certain proportion of the cases of epilepsy, the disease is susceptible of relief. . . . This relief is not to be attained by any medicine with which I am acquainted, but by diet" (Jackson 1855, p. 29).

To bolster her case, Gordon would have it that Jackson counseled seclusion: "It could have been Dr Jackson who persuaded Emily Dickinson to accept the prospect of seclusion and singleness in the hope of doing something with the intellectual and creative gifts that this doctor had the capacity to discern" (Gordon 2010, p. 121). This would have required enormous prescience on the part of the good doctor, as by 1851 Dickinson had scarcely begun her apprenticeship as a poet.

### CHLORAL HYDRATE

Although known for decades, chloral was first discovered to be a soporific in 1869, coming into wide medical use shortly after. William Whitty Hall (1874) lauded the new finding as a "new anodyne" for promoting sleep (pp. 339–40). It was the combination of chloral hydrate with the sweetener glycerine listed among remedies for epilepsy in Hall's manual that caught Gordon's attention. *A Companion to the Latest Edition of the British Pharmacopoeia* also cited chloral's use as an "excellent hypnotic, producing sound and placid sleep," but also claimed it was effective against chorea, delirium tremens, and "nervous disturbances and restlessness" (Squire 1871, p. 85).

The 1880 edition of the *Dispensary of the United States of America* discusses chloral's role against convulsions due to various causes, although relegating it to secondary status against classic epilepsy: "It is simply as a soporific that chloral is

most efficient and most employed. . . . In spasms chloral is often useful, though generally inadequate to the cure of the more violent and obstinate of these affections." The "affections" include tetanus, hydrophobia, chorea and epilepsy, but, "in the latter it is at best but an occasional palliative" (Wood and Bache 1880, pp. 266, 270–71). One of us (NH) still made use of chloral hydrate in the 1960s to treat alcoholic delirium tremens at Boston City Hospital.

The British *National Dispensary* noted the unpleasant taste of chloral hydrate: "Its taste even in a watery solution is somewhat acrid and bitter, causing a sense of burning in the throat and sometimes in the stomach also" (Stille and Maisch 1879, p. 392). Thus many texts advised the use of sweeteners or flavorings to mix with chloral hydrate. One manual prescribed orange juice and oil of peppermint: "The taste of hydrate of chloral is quite unpleasant, but orange-juice completely covers it, and so does peppermint water or essence of peppermint" (Dick 1872, p. 436). Others recommended syrup of orange flowers, mint, orange, and vanilla (Wood, Remington and Sadtler 1899). Squire's *Companion to the Latest Edition of the British Pharmacopoeia* noted that glycerine "is sometimes employed as a sweetening agent in the place of syrup" (Squire 1864, p. 137). Hall advised that chloral hydrate "should never be taken except dissolved in some liquid in proportion of twenty parts to a hundred; it is best taken in beef-tea, or syrup of orange peel, or as an enema in gruel" (Hall 1874, p. 772). The proportion of chloral to "some liquid" of one to five is similar to Hall's chloral hydrate/glycerine proportion of one to eight. The use of glycerine as a solvent for chloral is also mentioned by other authorities of the time: "Chloral is freely soluble in water, alcohol, ether, chloroform, glycerine . . . fixed oils and volatile oils" (Oldberg and Wall 1887, p. 317).

To anyone's knowledge, Emily Dickinson never took chloral hydrate.

#### FAMILY HISTORY AND GENETICS OF EPILEPSY

Two individuals who were related to Dickinson and who suffered seizures were utilized by Gordon to support her hypothesis of a genetic strain of epilepsy in the Dickinson family. To what degree does heritability play a role? As discussed in a recent conference of the Epilepsy Research Foundation, some 14 genes are known to be associated with idiopathic epilepsy; these, however, occur mainly in large families with many members affected with the disease (Scheffer et al. 2007). The disease is shared with up to 80% concordance in identical twins. We consulted with Dr. Simon Shorvon, Professor of Clinical Neurology and Consultant Neurologist, Institute of Neurology, University College, London.<sup>1</sup> According to Shorvon, idiopathic epilepsy occurs in 5 to 15% of persons who have first-degree relatives (parents, siblings) with the condition; in less than 5% with

<sup>1</sup>Professor Shorvon has long been an international authority on epilepsy and its genetics. See <http://www.shorvon.eu/>.

second-degree relatives (nieces, nephews) with the condition; and spontaneously (no family history) in less than 1% of the general population.

“Ned” Dickinson, son of the poet’s brother Austin, had classic epilepsy, a record of which was kept by his father (Longworth 1984). The only other person in the extended families with a seizure was Zebina Montague, of whom a detailed record remains. He was Emily Dickinson’s first cousin once removed through her paternal grandfather, Samuel Fowler Dickinson, and also her first cousin twice removed through her paternal grandmother, Lucretia Gunn Dickinson, a doubling of kinship resulting from several interfamily marriages between the two prior generations of Dickinsons, Montagues, and Gunns.

A decade younger than Emily Dickinson’s father, Montague grew up a few dozen rods up the road from him in rural Amherst. Montague was handsome, energetic, and a competent student who enjoyed pranks. He attended the Amherst Academy and graduated from Amherst College in 1832.

Nothing in Montague’s self-described clinical history is compatible with classic, idiopathic epilepsy. In an autobiographical account written in 1852 for his 20th college reunion, Montague describes how, after graduation, he made his way south to Columbus, Georgia, to work, first in a dry goods store, then as bookkeeper of the city’s largest bank. He also served in the militia during a campaign of the Creek Indian Wars, contracting what was most likely malaria, the symptoms of which plagued him for several years. Upon gaining a prominent position in the bank at age 29, he suffered a paralytic stroke, which he blamed on overexertion in his duties. After six months of intense suffering from bleedings, blisterings, and cuppings, he was half-carried 1,500 miles home in rough conveyances, arriving in Amherst on November 20, 1839, a dramatic homecoming long remembered by his fellow townspeople.

Montague was partially paralyzed for the rest of his life, apparently on his left side (he could still speak and write). Three years after his stroke, as noted by Emily Dickinson in her first extant letter, April 1842, Montague had a fit and nearly bit his tongue in two (Dickinson 1958, L1). When presented with the case, Shorvon concluded that the sudden stroke Montague suffered at his young age was likely due to a subarachnoid hemorrhage, caused by the rupture of an intracranial aneurysm. Such persons who survive may suffer seizures for several years after the event, due to pressure injury to the brain (Gilmore et al. 2010). According to the National Institute of Neurological Disorders and Stroke (2011), a temporary paralysis, known as Todd’s paralysis, can occur after an epileptic seizure. However, permanent paralysis is not a feature of idiopathic epilepsy.

In *Lives Like Loaded Guns*, Lyndall Gordon cites Zebina Montague’s diary, but relies on Emily Dickinson’s comment (made at age 11) to suggest idiopathic epilepsy, ignoring Montague’s own testimony of the stroke that preceded the seizure by three years. Gordon proposes, comparing him by analogy to Dickinson, that Montague “became a permanent recluse. . . . From his late twenties until his seventies Zebina remained in seclusion” (Gordon 2010, pp. 132–33).



This is incorrect. Montague was an extrovert, and so he remained; of necessity he was housebound, but he welcomed visitors and enjoyed being carried to participate in all manner of public activities (Tyler 1881).

Gordon also proposes that epilepsy in the Dickinson line might have resulted from the doubled genetic heritage. The argument is unsupported on family history grounds because it lacks evidence of prior epilepsy in the Dickinson, Gunn, or Montague families. Similarly, Ned's maternal Gilbert family had no known cases of epilepsy; thus his case may be considered spontaneous. Montague lived to age 70, and by his own testimony suffered no more seizures, although the paralysis remained. His gravestone in Amherst's West Cemetery reads, "He was a sufferer from Paralysis for Forty-one years, which he endured with Christian resignation."

### EPILEPSY: A DIFFERENTIAL DIAGNOSIS

Epilepsy is defined as "a disorder of the brain characterized by an enduring predisposition to generate epileptic seizures and by the neurobiologic, cognitive, psychological, and social consequences of this condition . . . [and] requires the occurrence of at least one epileptic seizure" (Fisher et al. 2005, p. 470). The most important step in diagnosing epilepsy is to obtain a detailed medical history. While we don't have the patient before us, or her family, we do have her own acute self-observations and the testimonies of others to guide us to a reasonable conclusion. In this section we retrace the various medical conditions known to us that occurred in Emily Dickinson's life. These are the same conditions Gordon uses to conclude that Dickinson had epilepsy, to which we offer a rebuttal.

We have shown in detail that the respiratory symptoms and weight loss Dickinson suffered in her adolescence and early adulthood was suspected to be tuberculosis and treated as such (Hirschhorn and Longworth 1996). The next event of note occurred on January 15, 1854. In a letter to her future sister-in-law, Susan Gilbert, Dickinson describes in poetic detail what may have been a panic or severe anxiety attack induced by a church service that, unusually for her, she attended alone:

I'm just from meeting, Susie, and as I sorely feared, my "life" was made a "victim." I walked—I ran—I flew—I turned precarious corners— One moment I was not—then soared aloft like Phoenix, soon as the foe was by—and then anticipating an enemy again, my soiled and drooping plumage might have been seen emerging from just behind a fence, vainly endeavoring to fly once more from hence. I reached the steps, dear Susie. . . . How big and broad the aisle seemed, full huge enough before, as I quaked slowly up—and reached my usual seat! In vain I sought to hide behind your feathers—Susie—feathers and *Bird* had flown, and there I sat, and sighed, and wondered I was scared so, for surely

in the whole world was nothing I need to fear—Yet there the Phantom was, and though I kept resolving to be brave as Turks, and bold as Polar Bears, it did'nt [*sic*] help me any. (Dickinson 1958, L154)

Perhaps related, Dickinson in the spring of 1863 described to her cousins a serious bout of anxiety, again when left alone:

The nights turned hot, when Vinnie [her sister] had gone, and I must keep no window raised for fear of prowling “booger,” and I must shut my door for fear front door slide open on me at the “dead of night,” and I must keep “gas” burning to light the danger up. . . . these give me a snarl in the brain which don't unravel yet. (Dickinson 1958, L281)

Gordon adapted the above phrase, “Snarl in the Brain,” to title her main chapter diagnosing epilepsy. Panic and anxiety attacks may simulate forms of epilepsy. The topic is thoroughly considered in a multi-author collection of essays on “imitators of epilepsy” with over two dozen conditions listed (Kaplan and Fisher 2005). Although epilepsy may at times look like a panic attack, with Dickinson we have no documentation of recurrent attacks, which ought to be a feature of epilepsy.

A few months after the latter episode, Dickinson began to suffer an eye condition lasting two years, which we have postulated was acute and chronic iritis/uveitis (Hirschhorn and Longworth 1996). All circumstantial evidence points to the diagnosis, including aching in the eyes, aversion to light—“the snow light offends them, and the house is bright,” she wrote her cousins (Dickinson 1958, L302)—and the specific treatments offered by Boston's pioneer ophthalmologist, Henry Willard Williams: eye drops, dimmed light, no close work, and perhaps a paracentesis to relieve the pressure. Dickinson described a treatment as “painful” (Dickinson 1958, L302). An ophthalmologist has recently accepted the diagnosis (Blanchard 2012). Assessing this episode, Gordon implies that Dickinson suffered photic epilepsy: “For a person who was predisposed, repeated flashes of light would set off convulsions,” and that Williams's treatment was meant to stop the attacks by curing the photosensitivity (Gordon 2010, p. 127). There is, however, nothing in Dickinson's own descriptions to suggest “repeated flashes of light.”

Gordon also proposes that Dickinson may have suffered absence seizures, otherwise known as petit mal, which are brief periods of lapses in consciousness, most often seen in children. She cites an essay by Dickinson's former schoolmate, Emily Fowler Ford, sent by mail in the mid-1890s to Mabel Loomis Todd, Dickinson's first editor and compiler of her poems and letters. This record, Gordon narrates, told how Dickinson would suddenly drop crockery, and then in embarrassment hide the fragments behind the fireplace screen. Gordon gives no source for the document, but the 11-page original exists at

Amherst College (Amherst College Library, Archives and Special Collections, Todd Ms. 328). There is no mention at all of dropping plates.<sup>2</sup> With no further evidence of “absences” in the biographic record, we may safely conclude that Dickinson did not have petit mal seizures.

By the close of the Civil War, with nearly two-thirds of both her life and poetic output behind her, Dickinson entered the full-time seclusion at home that is too often taken as her defining character. Nonetheless, her correspondence with a great number of friends and relations continued unabated, and she continued to see certain privileged persons. No one knows the precipitating cause of the seclusion, if one cause exists, although Gordon suggests it was the stigma of epilepsy. To Emily’s sister Lavinia (Vinnie), it “was only a happen,” a gradual withdrawal over time, a preference for seclusion at home (Sewall 1974, p. 153).

In April 1881, when Dickinson was 50, came an early indication of physical frailty. A nighttime fire, whipped by high winds, destroyed a main business section in Amherst, threatening Austin’s office and sending burning fragments toward the Dickinson property (Sewall 1974). Up all night, Dickinson was terrified and exhausted, and took to bed for a week. According to Gordon (2010), “she’d had a blackout, perhaps a seizure” (p. 167). In fact, in the letter describing the incident written to her cousins after she recovered, there is no mention of a blackout (Dickinson 1958, L691). On June 14, 1884, however, Dickinson did black out, from noon to late at night. As she recounted two months later to her cousins, “I saw a great darkness coming. . . . I had fainted and *lain unconscious for the first time in my life*” (Dickinson 1958, L907, emphasis added).

We believe these episodes heralded Dickinson’s decline to death that we have diagnosed as accelerated hypertension. In our paper (Hirschhorn and Longworth 1996) we offered the following clinical clues (with references): morning headache (1882); severe pain in the back of the head upon her nephew’s death (1883); a sudden spell of unconsciousness lasting from noon to late at night (June 1884); a second hours-long episode four months later, leaving her confused for days (October 1884); mainly confined to bed in the last six months of her life (1885–86); probable shortness of breath, but not appearing ill otherwise; prescription of digitalis; two days’ unconsciousness with stertorous breathing, then death (May 15, 1886), with a May 13 prescription for chloroform in olive oil (i.e., by mouth), perhaps for convulsions; a death certificate diagnosis of “Bright’s disease,” duration said since 1883.

The last item is of particular importance. Her attending physician, O. F. Bigelow, was never allowed by Dickinson, when conscious, to do a physical examination. On the two occasions of unconsciousness, a different doctor attended

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<sup>2</sup>Among the rough notes of a conversation between Mabel Loomis Todd and her daughter, Millicent Todd Bingham, made in 1931, occurs this brief comment: “Vinnie said Emily broke dishes by the wholesale” (Yale University, Millicent Todd Bingham Papers 1865–1968, MS 496D). We consider such testimony a slender reed on which to hang a diagnosis of petit mal.

her. Thus Bigelow could only go on medical history and what he observed when she was in coma. Bright's disease, as defined in the medical textbook he owned, was an assemblage of kidney pathologies, which in advanced or chronic stages included signs of heart enlargement, heart failure, a "tense and sustained pulse," headache, convulsions and "hemorrhagic apoplexy" (Quain 1883, pp. 178–79, 182). Primary hypertension as a separate entity would not be described and measured until several years later. No uremic signs of kidney disease were noted with Dickinson's last illness, as her niece recalled: "When she was able to see us at all, I never saw her look or act ill" (Bianchi 1932, p. 67). Thus "Bright's disease," in a 55-year-old in coma, with onset after her nephew's death two years earlier, was the most logical diagnosis to make at the time.

Both Gordon and we have taken the diagnostic trail further by analyzing prescriptions that may have been intended for Dickinson in the last years of her life. The Adams Pharmacy was one of at least four serving Amherst at the time, and Bigelow one of a half dozen practitioners writing prescriptions for that pharmacy, which are found in a scrapbook in the Amherst College Archives. The chits related to Dickinson's illness date from October 9, 1883, through May 13, 1886 (the latter has gone missing). It is important to stress that none of the prescriptions are written specifically for "Emily Dickinson," perhaps to protect her known desire for privacy. We have examined any prescription for any "Dickinson" around the dates of Emily's lapses in unconsciousness, looking for medications that seem relevant. For clarity, Gordon's and our choices are given in Table 1, along with their details.

Gordon uses these data to propose that Dickinson's collapses and coma were due to epilepsy. In her pursuit of the glycerine theory, Gordon has mistaken two prescriptions containing glycerine as medications to be taken internally. In fact, both are preparations for external use: #17561 with carbolic acid (phenol), an antiseptic lotion, and #17900 with boracic acid, used to heal ulcers in the mouth.

The medications Gordon identifies include preparations with various stimulant, tonic, soporific, and circulatory effects: arsenic, strychnine, hyosyamus (henbane), ustilago (from corn smut, analogous to ergot), digitalis, and chloroform. In 19th-century pharmacotherapy, many medicines served multiple conditions, there was great overlap, and none here were specific to epilepsy (*Merck's Manual* 1899). Gordon also mentions finding "3–6 grains of ergotine" advised in the chapter on epilepsy in Bigelow's textbook (p. 216). However, she has misread: this recommendation is in the chapter on Bright's disease (3–5 grains, subcutaneously, for hemorrhages; Qain 1883).

Our differential diagnosis rules out the diagnosis of epilepsy with as much certainty as possible, given the fragmentary biographic data.

**TABLE 1** PRESCRIPTIONS IN EMILY DICKINSON'S LAST YEARS OF LIFE\*

Date	Prescription number	Prescribing physician	Patient	Medications and commentary
<i>October 5, 1883, the day that her treasured nephew Gilbert died, Emily was put to bed and experienced severe pain in the back of the head.</i>				
9 Oct. 1883	17143	E. Hitchcock, Jr.	Dickinson	"Quinia Sulph, Fowler's arsenite solution." Fowler's solution contained a salt compound of arsenious acid and was considered an "alterative," one that restores health generally, with many uses; mentioned in particular for severe headache ("hemicranias," "periodical headache"). Quinine was also used for severe headache ( <i>D-USA</i> , pp. 24-25, 1236, 1322).
12 Oct. 1883	17150	C. W. Cooper	Miss L. Dickinson	"Tinct. nucis vom." Nux vomica, a source of strychnine, was used as a tonic and also specified against "palsy" (paralysis) ( <i>D-USA</i> , p. 561).
<i>In 1884 Dickinson suffered two episodes of unconsciousness: 14 June and 12 October.</i>				
15 June 1884	17533	D. B. N. Fish	Miss V. Dickinson	"Elix. Gentia with Tinct. Chloride Iron." Gentian and iron were used as tonics ( <i>D-USA</i> , pp. 411, 1396).
28 June 1884	17561	O. F. Bigelow	Dickinson	"Glycerine, acid Carbolic [phenol] lotion." Gordon misidentifies this antiseptic lotion as medicinal glycerine. See <i>D-USA</i> , p. 1486, for dilution of carbolic acid with glycerine.
15 Oct. 1884	17735	D. B. N. Fish	Dickinson	"Tinct. gelsemium, tinct. hyoscyami, ext. ustilago." Gelsemium from Carolina jasmimine was used as a relaxant ("nervous and arterial sedative"); hyoscyamus (henbane) is an anodyne and soporific ( <i>D-USA</i> , pp. 409-10, 461). Ustilago is derived from corn smut, a parasitic fungus thought analogous to ergot, one of many medications listed for headache. See <i>Merek's Manual</i> 1899, pp. 131-32. "It serves a useful purpose in <i>impaired cerebral circulation</i> , with dizziness, unsteadiness of motion, or lack of command over the intellectual faculties, dull headache in top of head, disordered vision, etc." (Felter and Lloyd 1898, original italics).
<i>Records show Dickinson in decline from 1885 until her demise in May 1886, with signs suggesting heart failure.</i>				
1 Jan. 1885	17900	D. B. N. Fisher	Dickinson	"Glyceride [a chemical derivative of glycerine] of boracic acid apply twice daily." Gordon misidentifies this medication against mouth infections (aphthous ulcers, thrush, "cracked tongue") as medicinal glycerine instead of an emollient ( <i>D-USA</i> , pp. 569, 787).

**TABLE 1 (CONTINUED)**

30 Jan. 1885	17918	O. F. Bigelow	Dickinson	“Tr. Ferri chloride, Tr. Digitalis.” Iron tonic, and medication for congestive heart failure.
30 Jan. 1885	17919	O. F. Bigelow	Dickinson	“Emplastrum Belladonna.” Plaster applied to the skin for “myalgia,” here perhaps for chest pain.
17 Aug. 1885	18350	D. B. N. Fisher	Dickinson	“Quinia Sulph., Strychnine sulph., Acid phosp., Calcis lactophos;” Both quinine and strychnine are listed for headache and migraine-like “hemisrania” ( <i>Merk’s Manual</i> 1899, pp. 131–33). The phosphate and calcium preparations were used as nutritional tonics ( <i>D-USA</i> , p. 1032).
13 May 1886		O. F. Bigelow		“Chloroform, Olive oil.” The prescription slip has been lost from the Adams Pharmacy archive; the information is drawn from Leyda 1960, 2:471. Note that this is a preparation to be taken by mouth, not inhalation; a “sedative narcotic” ( <i>D-USA</i> , p. 962) used for convulsions (Alexander 1889, p. 146; Quain 1883, p. 303).

\*For each prescription we give the date, the prescription number, the prescribing physician, the patient’s name, and the medications prescribed. Note that no prescription was written specifically for “Emily Dickinson”; prescriptions written for “Dickinson” on dates around key moments in her life are shown here. “L. Dickinson” and “V. Dickinson” refer to the poet’s sister Lavinia (Vinnie), who was Emily’s housemate and care-giver. Information about contemporary prescribing practices is drawn from Wood and Bache 1869, abbreviated as *D-USA*.

Source: Adams Pharmacy Prescription Volume, Emily Dickinson Collection, Box OSB7, Amherst College Archives and Special Collections.

CONCLUSION

The posthumous diagnosis of epilepsy in notable people is often incorrect (Hughes 2005). We have gone to lengths to counter yet another startling diagnosis, this time in Emily Dickinson, not as a literary exercise but in an effort to correct a fallacy about an iconic American poet, whose poetry and persona resist such a reductive explanation. The misdiagnosis, coming in an otherwise influential book by a well-regarded biographer, is likely to stay in the public's mind for some time to come. Reviewers for the most part have accepted Gordon's premise, and interviewers on public radio stations have been enthusiastic (Gross 2010; Lopate 2010). Just this year an eminent poet and critic found the epilepsy hypothesis persuasive (Chiasson 2013). We believe it incumbent on biographers to be accurate in handling factual information, to investigate thoroughly their original sources, and to secure expert advice where theirs is lacking.

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