

# Dartmouth Gazette



HERE RANGE THE WORLD—EXPLORE THE DENSE AND RARE;  
AND VIEW ALL NATURE IN YOUR ELBOW CHAIR.

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# THAT BOURNE FROM WHENCE NO TRAVELLER RETURNS

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At the turn of the 19<sup>th</sup> century, Hanover, like much of the world, was vulnerable to devastating outbreaks of dysentery. Nathan Smith, the founder of the medical school, and others at Dartmouth were among those who tried to understand the causes of the disease, as revealed by student notes and theses from the time.

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n the fall of 1796, not long after proposing the creation of a medical school at Dartmouth, Nathan Smith left the small town of Hanover, N.H., and set sail for the British Isles. In Edinburgh, a center of medical education at the time, Smith intended to increase his medical knowledge, which would be useful preparation for teaching medicine at Dartmouth, and to add to his reputation as a scholar. By traveling at that particular time, Smith happened to be absent from Hanover in the summer of 1797, when an outbreak of dysentery swept through the town.

“The dysentery prevailed in this parish & was fatal to 24 persons in 12 weeks,” noted William Dewey in his records of deaths in Hanover that summer. “Twice within 3 weeks we carried into our Meetinghouse & from there to the Graveyard three deceased individuals at the same time. Again we carried one to the grave & escorted the mourners back to their dwelling lading along with us the Bier covered with the Gravecloth—& hastened from there to another house to perform the same duty to another deceased person.”

Three years later, another outbreak of dysentery struck Hanover. As recorded by the *Dartmouth Gazette*, “In the north and east parts of this town, a number of children have died of the Dysentery. This distemper seizes the youth with great violence, and hastens them to that bourne from whence no traveller returns” (the latter a paraphrase from Hamlet’s famous soliloquy in Act III, Scene I, of Shakespeare’s play).

Though Smith was away when the 1797 outbreak occurred, he was certainly no stranger to dysentery—nor were the students at the medical school he founded in Hanover. As early as 1791, writing in *The Massachusetts Magazine* on “The Causes and Effects of Spasms in Fevers,” Smith speculated on the causes of dysentery, and in his time at Dartmouth, he lectured on the subject to medical students. Notes taken from his lectures provide insight into how Smith understood and defined dysentery, and student theses from across the 19th century reflect the changing understanding of this deadly disease.

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King Henry V of England led his army to victory at the famous Battle of Agincourt in 1415, but not before the army was struck by an outbreak of dysentery. In 1422, Henry died of the disease.

“dysentery attacked the troops while still upon their march, and greatly thinned their ranks. Many died; others fell sick and were left behind in the different cities that lay upon the route.”

Hippocrates wrote that the disease was caused “by the overflow of phlegm and bile to the veins of the belly, producing ulceration and erosion of the intestine,” phlegm and bile being two of the ancient four humors that were thought to regulate health. Dysentery, he wrote, was “the phlegm descending downward from the brain.” He, like other Greek and Roman physicians, noted one of the cardinal—and sinister—features of dysentery that distinguished it from simple diarrhea, namely the bloody and “fleshy” stools. “If in a person ill of dysentery substances resembling flesh be discharged from the bowels, it is a mortal symptom,” Hippocrates wrote. (These “fleshy masses” (ξυσματα) were actually used in Roman times as a tonic to promote health, with the scrapings coming from the epidermis of slain gladiators!)

## ANCIENT EPIDEMICS

Long before Nathan Smith, medical practitioners and historians made note of outbreaks of dysentery. The English word for the disease appears to derive from two Greek words: *δυσ* and *ντερον*, which some have said are literally untranslatable into English but were described by Hippocrates as “dys-” (meaning bad, abnormal, or difficult) and “entera” (meaning intestines or bowels). Other words for the disease include *difficultas intestinorum* (Latin) and *atisara* (Sanskrit).

Observations of dysentery antedate Hippocrates. In the fifth century B.C., for example, Herodotus recounted the effects of dysentery on the army of Xerxes:

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Other Greek physicians and philosophers wrote extensively about dysentery as well. Celsus referred to it as *tormina* (the gripping of the bowels), while Aretaeus pointed to the prominence of ulcerations. The ancient Roman physician Galen noted four varieties of the disease, only one of which was accompanied by ulceration, and he made the first recorded use of the word *scybala* (σχυβάλα, or feces) to characterize the fleshy masses.

Then, for centuries, physicians contributed little to the description of the symptoms and signs of dysentery. By the 17th and 18th centuries, many of the writings on dysentery focused on other aspects of the disease, including its etiology and treatment. Several commentators noted an apparent relationship to the seasons, an observation Hippocrates made as well:

*For when the suffocating heat sets in all of a sudden while the earth is moistened by the vernal showers and by the south wind, the heat is necessarily doubled from the earth, which is thus soaked by the rain and heated by a burning sun, while at the same time men's bellies are not in an orderly state, nor is the brain properly dried.*

Writers debated the cause of dysentery. Most thought it was unconnected to food and drink, but some did think the disease was contagious, which, before the discovery of microorganisms, simply referred to contact with some external agent. John Pringle, an 18th-century Scottish physician who published a classic treatise on military hygiene and dysentery, claimed that the disease was acquired by soldiers through contact with foul straw as they slept. But he also proposed that it might be transmitted in the privies “after they had received the dysenteric excrements of those first stricken.”

Many medical practitioners noted that the disease sometimes became an epidemic, often in the context of military activity. The English army nearly lost the 1415 Battle of Agincourt because of

*“Dysentery is one of the four great epidemic diseases of the world. . . . It has been more fatal to armies than powder and shot.”*

epidemic dysentery. Indeed, a few years later, Henry V, who had led his men to victory at Agincourt, succumbed to the disease. Dysentery was omnipresent in the European wars of the 17th and 18th centuries, including Napoleon's campaigns and the Crimean Wars. It accounted

Dysentery was the subject of extensive debate among physicians in the 19th century. These notes were taken by a medical student during an 1816 lecture by Nathan Smith, the founder of the medical school at Dartmouth, and they reflect the understanding of the disease at the time. “That dysentery is not contagious is now past a doubt in my mind,” Smith said in a lecture to medical students.

for 8 percent of the American deaths in the Mexican War of 1846 to 1848, and for 25 percent of deaths in the U.S. Civil War. And it was the major cause of nontraumatic morbidity and mortality in the German Army in World War I. As the eminent physician William Osler wrote in 1892, “Dysentery is one of the four great epidemic diseases of the world. . . . It has been more fatal to armies than powder and shot.”

In the 18th- and 19th-century U.S., dysentery was common among civilian populations as well, including the outbreak in Hanover in the summer of 1797. Some of the leading families in Hanover were affected by the epidemic, with the greatest impact falling on their young children, many of whom are buried in the Dartmouth College cemetery. The infant son of Jedediah Baldwin, one of New England's most prominent clockmakers and silversmiths, died on the same day as a 21-year-old Dartmouth College student named John Merrill. These are two of the three alluded to in William Dewey's account of multiple burials. The infant son of Benjamin Gilbert, a local attorney, died on September 1. Josiah Green, whose Main Street dry goods and grocery stores was patronized by many, including Nathan Smith, lost two children: Ira, age 11, and Josiah Jr., age 2. Abel Holden, a friend and fellow Masonic Lodge member of Eleazar Wheelock, lost two children on August 28. Eighteen-month-old Samuel Curtis, the son of town selectman Colonel David Curtis, died on September 11.

No Hanover family was more affected than that of Isaac and Amelia Leavitt Bissell. Isaac was the son of Israel Bissell, a patriot post rider who, according to legend, may have ridden from Watertown, Mass., to Connecticut on April 19, 1775—the day of Paul Revere's famous ride—to bring news of the British attack. The Bissells had 13 children between 1777 and 1796. Four of them died within two weeks of each other in the 1797 epidemic, and all were buried in the Dartmouth cemetery. Five-year-old Asaph was spared. He went on to attend Nathan Smith's medical school before leaving Dartmouth with Smith in 1813 to finish his medical education at Yale, becoming one of Smith's first students there. Another surviving son, 18-year-old Isaac Jr., was the father of George Bissell, considered the father of the American oil industry and one of Dartmouth's most prominent alumni and benefactors in the 19th century. (The now-raised Bissell Hall was given by George as one of the first university gymnasiums in the country. The Bissell name is now remembered by a prominent grave in the Dartmouth Cemetery and by a dormitory in the Choates cluster.)

We have been able to partially reconstruct a map of Hanover in 1797 and have found that the Bissell, Gilbert, and Baldwin families all lived within one block of each other on the current Main and Lebanon Streets. Jedediah Baldwin's silversmith store was next to the Gilbert residence. All of these were located close to the only water well in Hanover at the time, which was on the site of the present Reed Hall. Josiah Green's grocery and dry goods store was also located on Main Street, though we could not determine its exact location. It seems reasonable to speculate that a common source of

This record of deaths in Hanover by William Dewey noted that dysentery "was fatal to 24 persons in 12 weeks" during a 1797 outbreak.

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**Dysentery.**  
That dysentery is not contagious is now past a doubt in my mind. It arises from certain circumstances of place that cannot be accounted for. It generally proves most fatal to children of any disease they are subject to. The attendant fever is of the typhoid, though sometimes of the inflammatory kind. Costiveness usually precedes the attack. While the principal seat of the disorder seems to be in the lower & large intestines, there is an increased action of the smaller ones as is evident by vomiting. This disordered action of the large guts produces a secretion of mucus, which constitutes a principal part of the stools, which is the grand criterion to distinguish dysentery from diarrhoea.

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In D<sup>r</sup> 1797 The Dysentery prevailed in this parish & was fatal to 24 persons in 12 weeks - I thought to be remarked however that several persons were sick with other complaints & this set in with the former one & destroyed them - It was said that only three died of Dysentery alone - This is probably correct - Twice within 3 weeks we carried into our Meeting house 4 from thence to the graveyard three deceased individuals at the same time - Again we carried one to the grave & escorted the Mourner back to their dwelling lodging along with us the Beer covered with the grave cloth & hastened from thence to another house to perform the same duty to another deceased person - Previous to these - Infantile Fits & Internal Ulcers with not less than 4 or more cases of Consumption added very largely in this scene of desolation & death -

contamination of this well or perhaps of shared sanitary facilities might have been the source of this 1797 epidemic.

### DEBATES OVER DYSENTERY

After spending nine months abroad, Nathan Smith returned to Hanover in time to begin teaching classes in the fall of 1797. Like other physicians, Smith would certainly have had dysentery on his mind, as shown by his lectures. In notes written during a lecture by Smith in 1812, medical student Jesse Goodwin wrote that dysentery is "another disease of the intestines which is produced by the climate or season. . . . It seems to be a disease initiated in the lower portions of the large intestines, the whole are sometimes affected." Goodwin went on to note that the disease is "preceded by costiveness [a synonym for constipation] and frequently attended with puking. It is a disease of the inner coat of the intestines. In some cases the intestines have been so corroded that pieces of them are entirely gone. In this disease no feces appear in the stool but a liquid Mucus." Notes from a lecture by Smith in 1816 report that dysentery "generally proves most fatal to children of any disease they are subject to." (Smith left Dartmouth in 1813 and moved on to found Yale's medical school, but he returned in 1816 and spent a term teaching at Dartmouth.)

In his 1822 thesis, student Stephen Eaton described an epidemic of dysentery that appeared in Hanover the previous year. According to him, the symptoms of the "great distemper" are "excessive vomiting,

violent pain in the bowels, and particularly at the navel and stomach, constant necessity of going to stool and painful tenesmus & gripes; accompanied by mucus and bloody discharge, small indeed but so frequent that the rectum hardly ceases to evacuate; fever very seldom entirely absent."

Both at Dartmouth and beyond, dysentery was a source of contention and debate among physicians. The details of the disease seemingly evaded even the best minds of the time, as noted by medical student Joseph Pemberton in his 1828 thesis. "The pathology of dysentery . . . has been a fruitful source of dissension among medical writers," Pemberton wrote, going on to say that the disease "not infrequently triumphs over the skill and sagacity of the most discerning minds."

The evolution of the debate over dysentery can be seen in these records. In the early 19th century, many physicians seemed to believe that the disease had two distinct forms. "There are two degrees of this disease," read the notes from one lecture by Nathan Smith. "The one mild, the other very malignant." Other physicians distinguished between varieties of the disease by referring to one as "simplex" and the other as "pyretica." Student Jesse Little described these two forms in 1826: "The first [simplex] being when unaccompanied by fever, the second, pyretica, accompanied with fever, gripes, loss of strength and depression of spirits." Yet this binary categorization seems to have fallen out of favor by later in the century, when George Seely Thompson, in his thesis on dysentery, described dysentery as a single disease, despite differences in presentation: "The disease varies much

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in its form and character. It may be epidemic, adynamic, bilious, intermittent, remittent, or typhus.”

Much of the debate and uncertainty associated with dysentery revolved around the question of whether it was contagious. As noted, contagious during this period simply meant a disease caused by an external agent. In 1791, Smith wrote that “that it is often communicated from one person to another is very probable, but that it is never propagated by other means is doubtful. I am of the opinion that it is. I have known children attacked with a dysentery . . . who lived several miles from any other family and have never been abroad or approached by any person affected with the disease.” Smith was convinced that dysentery is “often produced by a contagion arising from the putrefaction of vegetable matters,” which would have accounted for its frequent appearance in the late summer months, when the year’s crop began to decay.

In the following years, however, it appears that Smith changed his mind. Notes from a lecture delivered in 1816 quote Smith stating: “that dysentery [sic] is not contagious is now past a doubt in my mind. It arises from certain circumstances of place that cannot be accounted for.” Reflecting further changes in the thinking at the time, George Seely Thompson’s 1878 student thesis, written about the time the germ theory of disease was being corroborated, presented a mixed view:

*The question has been much agitated whether dysentery is a contagious disease or not but it has been conceded by nearly all authors that it is not so in its ordinary form but circumstances have arisen in which individuals have been successively affected in epidemics and has led to the opinion with some that in this character it is propagated by contagions.*

A closely related issue was determining the underlying cause of dysentery. This, too, was a matter of great speculation in the early 19th century, and an opportunity for early epidemiological sleuthing. Many observers cited changes in the weather as a cause, noting as Eaton did that “among the remote causes no one is more essentially necessary to produce epidemic dysenteries than moisture, for in low and marshy places where all other circumstances were favorable it has not appeared until the rain fell.” Smith’s student Jesse Little wrote in his notes that “It appears singular to me that in one part of a town it proves fatal when in another part contagious to it the people are only vaguely affected if at all.” Cooper also seemed to recognize that outbreaks of dysentery were clustered together. Echoing his teaching from Smith, he concludes that dysentery was caused by “gases of decomposing vegetable and animal matters rising from the soil. Primarily originating in this manner, it may afterward be propagated by individuals coming into contact and

spread through specific poison among themselves, and especially, may it be communicated by the drinking water becoming contaminated by the stools, or as some believe, by means of the excretion and exhalation from the body of one sick with the disease.” While these explanations do not cite specific outbreaks, one wonders if the proximity of affected families (like the Bissels, Baldwins, and Gilberts of the 1797 epidemic) informed this epidemiological speculation as to the causes of dysentery.

The conclusions and observations by Nathan Smith and other medical scholars add depth and context to our current knowledge of dysentery. Looking back on the evolution of understanding of dysentery within the Dartmouth community reveals the insight of our predecessors and calls us to appreciate the progress they were able to make without the technological advances we have access to today.

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A micrograph of the bacterium *Shigella*, a cause of as many as 120 million cases of dysentery each year.

## DYSENTERY IN THE 21ST CENTURY

Since that 1797 outbreak, the medical community has made great progress in understanding dysentery. We know today that the disease is most often caused by species of the bacterium *Shigella* (bacillary dysentery) or by the amoeba *Entamoeba histolytica* (amoebic dysentery). *Shigella* alone is responsible for as many as 120 million cases of severe dysentery each year, with the majority of those cases occurring in developing countries and in children under the age of five. Usually ingested from contaminated food or water, the bacteria are invasive, and

the shiga toxin produced by the bacteria acts on the vascular endothelium and on small blood vessels, breaking down the lining and leading to hemorrhage.

This level of knowledge is quite a change from the 19th century, when medical student Herman Cooper wrote in his 1882 thesis that “under microscopical examination,” the stool of patients with dysentery presents “epithelium cells, blood, exudation and pus cells, remnants of the mucous membrane and other cells and bodies which have not been fully described.” Yet, while modern science may have the ability to fully describe these “bodies,” even today no etiologic agent for dysentery is identified in more than half of cases.



- ▶ Although dysentery is rare in the U.S.
- ▶ today, it remains a public health
- ▶ challenge in parts of the developing world, particularly in areas that lack adequate access to clean drinking water.

Treatments for dysentery have also progressed. In the 19th century, there were no effective medications. In Eaton's words, "there are few acute diseases less beholden to nature for a cure, or attended with more deceitful indications than dysentery. The hemorrhage seems to require repeated bleedings, the discharges from the bowels strong astringents, the pain constant opiates, yet unless these remedies are used with caution they tend more to complain than remove the disease." In his thesis, Joseph Pemberton discussed the variation in opinions about how best to treat the disease. He noted "while some depend almost entirely upon the lancet, others place equal confidence in the powers of mercury and a third class rely principally upon diaphoretics." As Thompson noted in his thesis, often nothing could be done other than to make the patient as comfortable as possible: "when nothing else can be done it is well to forget sometimes the disease in order to consider . . . the patient, therefore support life with good old brandy and beef blood."

Patients suffering from dysentery today will most likely be treated to a course of antimicrobial agents, such as ampicillin or ciprofloxacin, two of the treatments recommended by the World Health Organization. But even as treatments have improved, challenges remain in addressing the problem of dysentery around the world. In the U.S., outbreaks are most common in high-density settings, such as nursery schools, mental institutions, and military barracks. It is also common among travelers from industrialized nations to the developing world. With the availability of antibiotics and easy access to hygienic conditions, these outbreaks rarely prove fatal. But diarrheal diseases still rank fourth globally as a cause of death, and second as a cause of years of productive life lost due to premature death and disability. *Shigella* in particular has been responsible for numerous fatal epidemics over the past century. Outbreaks in Central America from 1969 to 1973 were responsible for more than 20,000 deaths. Over the past two decades, epidemic dysentery has devastated refugee communities in central and southern Africa. During the Rwandan Genocide in 1994, dysentery ravaged refugee camps in Zaire, leading to approximately 20,000 deaths in the first month.

The best hope today for improving outcomes and reducing dysentery epidemics is to improve sanitation, increase access to antibiotics, and develop vaccinations against the most common bacterial causes of dysentery. According to a 2012 Progress Report on Drinking Water and Sanitation, in the world's least developed countries only 11 percent of people have access to piped water and open defecation is practiced by nearly a quarter of the population. In a meta-analysis of studies conducted between 1990 and 2009, it was estimated that over 99 percent of diarrheal deaths could be prevented by timely treatment with ciprofloxacin or ceftriaxone. Vaccine development against the causes of dysentery, particularly *Shigella*, is a challenge due to the large number of disease-causing variants. Currently, candidate shigellosis vaccines are in advanced development, and include both killed and live vaccine approaches. The World Health Organization estimates that a vaccine with even 70 percent efficacy could prevent up to 91 million infections with *Shigella* and 605,000 deaths each year.

The devastation described by William Dewey in 1797 would be rare in Hanover today. But no matter where we live, we are all connected to the burden of diarrheal diseases. Progress in reducing death from dysentery has been uneven, and much work remains to be done. From Nathan Smith to the World Health Organization, from the students of Dartmouth Medical School in 1816 to the medical students of today, we are all faced with the challenge of ensuring that no person in the 21<sup>st</sup> century travels prematurely to "that bourne from whence no traveller returns."

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