Charles Dickens died of a stroke on June 9, 1870, aged 58 years. He was ill for the last 5 years of his life, although no diagnosis has yet accounted for his varied symptoms. Here, I propose that Dickens had a right parietal or parietal-temporal disorder.1

Dickens’s symptoms

My hypothesis originates from an unusual and highly specific symptom. In 1868, while going on foot to John Forster’s house at Palace Gate, Dickens noticed that “he could read only the halves of the letters of the shop doors that were on his right as he looked” (Life bk. 11, ch. 1). He had the same problem again on March 21, 1870, as described by Forster:

[H]e told us that as he came along, walking up the length of Oxford Street, the same incident had recurred as on the day of a former dinner with us, and he had not been able to read, all the way, more than the right-hand half of the names over the shops. (Life bk. 12, ch. 1)

Dickens’s friends were amazed at the peculiarity of the symptom and by Dickens’s “old fixed persuasion” that his symptoms originated with a medicine he had taken rather than from “any grave cause” (Life bk. 12, ch. 1). These accounts seem to describe a form of spatial neglect (Parkin 1996), probably neglect dyslexia (Ellis, Young, and Flude 1993), and if this diagnosis is correct, they would constitute the first published description of the syndrome; Forster’s three-volume Life of Charles Dickens (1872–74) pre-dates Hughlings Jackson’s brief and somewhat unclear account of spatial neglect in 1876.2

As in most people who are right-handed, Dickens’s disorder was left-sided, which suggests right parietal lobe damage, although data also implicate the superior temporal lobe (Karnath, Ferber, and Himmelbach 2001). Some of Dickens’s other symptoms might also have resulted from parietal disease, which often presents in varied and unusual ways (Critchley 1953). Dickens’s spatial neglect is unusual only because he recognized it as a problem, which does occur (Marshall 1988).
film director, Federico Fellini, for instance, had insight into his own spatial neglect (Cantagallo and Della Sala 1998). Most patients with spatial neglect are, however, aware of visual segmentation (Driver and Mattingley 1998) – i.e., they know the length of words and they are able to recognize that what they see is not the entire word (Ellis, et al. 1993, and McCarthy and Warrington 1990). Dickens noticed his problem most when reading proper names, perhaps because of the presence of contextual cues. To give modern examples, one could well realize something is wrong if a shop one passes regularly and which still has the same location, logos, and color schemes, is now seemingly called Bloomsbury’s rather than Sainsbury’s, or Piebald’s rather than McDonald’s. A prediction is that insight might be more frequent in neglect patients who are misreading proper names in context.

Onset of symptoms

Dickens had problems with his right cerebral hemisphere. This problem became evident on April 18, 1869, in the middle of a grueling – albeit lucrative – tour of public readings. After feeling ill he wrote to his doctor, Frank Beard, who rushed to Dickens, cancelled the tour, and brought him back to London, to be seen the next day by Sir Thomas Watson, physician-extraordinary to Queen Victoria, who wrote:

After unusual irritability, [Dickens] found himself, last Saturday or Sunday, giddy, with a tendency to go backwards, and to turn round … He had some odd feeling of insecurity about his left leg, as if there was something unnatural about his heel; but he could lift, and did not drag, his leg. Also he spoke of some strangeness of his left hand and arm; missed the spot on which he wished to lay that hand, unless he carefully looked at it; felt an unreadiness to lift his hands towards his head, especially his left hand – when for instance, he was brushing his hair.

Watson concluded that Dickens “had been on the brink of an attack of paralysis of his left side, and possibly of apoplexy” (Life bk.11; ch. 1). These fears were confirmed the next year when, at dinner, Dickens stood and collapsed to his left. Apoplexy was diagnosed, and Dickens died the next evening without having regained consciousness. No necropsy took place.
History of Dickens’s illness

Dickens had had signs of heart disease, perhaps precipitated by renal disease for several years (Bowen 1956), and in 1866 he was prescribed iron, quinine, and digitalis. Watson also noted signs of cardiac enlargement. The most confusing aspect of Dickens’s medical history was described by Forster as “that formidable illness in his [left] foot ... [which] baffled experienced physicians.” Dickens attributed his symptoms to frost-bite, which he first noticed in February, 1865.

I got frost-bitten by walking continually in the snow, and getting wet in the feet daily. My boots hardened and softened, hardened and softened, my left foot swelled, and I still forced the boot on; sat in it to write, half the day; walked in it through the snow, the other half; forced the boot on again the next morning; sat and walked again; and being accustomed to all sorts of changes in my feet, took no heed. At length, going out as usual, I fell lame on the walk, and had to limp home dead lame, through the snow, for the last three miles ...

A month later, Dickens wrote to Frank Beard: “Here this confounded foot as bad as ever again. I suffered tortures all last night, and never closed my eyes. We are now at work at the Poppy[-head] fomentations again” (Letters 11: 28).

By April 22, 1865, Dickens’s foot seemed to have healed, though in September, the symptoms returned; Dickens could not bear the foot to be touched, sat with it up, and had a special extra large boot made. Further painful episodes happened in January, 1866, after which the problem subsided until August 2, 1867, when the foot seems again to have swelled. Writing to Georgina Hogarth, he explained: “I cannot get a boot on – wear a slipper on my left foot – and consequently I am here [Liverpool] under difficulties.” Shortly afterwards, Forster received additional information: “I am laid up with another attack in my foot, and was on the sofa all last night in tortures. I cannot bear to have the fomentations taken off for a moment” (Letters 11: 406–07).

Dickens was still lame on September 11, 1867, and could not wear shoes. Recurrences continued throughout his life: in early 1868; in February and April, 1869; at Christmas, 1869; and finally in May, 1870, when he was so lame that he could not attend the Queen’s State Ball at Buckingham Palace (Letters 12: 519n.).

Apart from exquisite pain, the foot was also “very tender ... as though ... in hot water,” and showed “extreme sensitiveness [so that he was
unable] to put any covering upon it.” Although sometimes taking opiates (laudanum), Dickens’s problem was mostly treated with hot poultices and fomentations. “I have had the poultices constantly changed, hot and hot, day and night,” but the foot still remained “a mere bag of pain” despite having been “Viciously bubbled and blistered … in all directions” (Letters 12: 524; 528–9) Dickens’s pain was never properly diagnosed, and he died believing that his illness was localized to his foot, an idea backed up when Sir Henry Thompson, the country’s leading urologist, suggested that his pain “originates in the action of the shoe, in walking, on an enlargement in the nature of a bunion.” Gout was suggested as a diagnosis, but Dickens denied this possibility. While in Edinburgh, Dickens consulted the renowned surgeon and diagnostian, James Syme, who disputed Thompson’s opinion of gout. Instead, he said the pain originated from the cold, and affected the nerves and muscles. However, other authors, Forster, and Edmund Yates, thought Dickens’s pain was gout. “There is no one now, I suppose, who does not recognize that this pain in the foot and lameness were gouty expressions of internal disorder” (Recollections 2. 119). Gout, however, was a common Victorian diagnosis (Porter and Rousseau 1998). Dickens also reported other paroxysmal pains: in 1866 he reported severe pain in his left eye. Forster emphasized the side of the problems: “It was his left eye, it will be noted, as it was his left foot and hand” (Life bk. 11, ch. 3). In December, 1869, Dickens’s left hand was sporadically painful, by January 23, 1870, he had to wear a sling, and throughout February, 1870, his hand was constantly painful.

**Railway accident**

On June 9, 1865, Dickens was present at the Staplehurst railway disaster. Ten passengers were killed and 14 badly injured (Letters 11: 49n). Although some people have suggested Dickens’s health deteriorated after this incident, the painful left foot and other symptoms pre-date the accident. Nevertheless Dickens did have post-traumatic flashbacks as his daughter Mamie recalls in her book:

> My father suddenly clutched the arms of the railway carriage seat, while his face grew ashy pale, and great drops of perspiration stood upon his forehead, and though he tried hard to master the dread, it was so strong that we had to leave the train at the next station.” (1897, 112)

Dickens also acknowledged his fear to Forster after the crash:
I cannot bear railway travelling yet. A perfect conviction, against the senses, that the carriage is down on one side (and generally that is the left, and not the side on which the carriage in the accident really went over), comes upon me with anything like speed, and is inexpressibly distressing.” (Letters 11: 65)

**Dickens’s denial**

Despite an interest in medicine – Dickens was apparently a regular reader of *The Lancet* (Markel 1999), and his novels often described medical conditions – and despite repeated, unpleasant indications of serious disease, Dickens denied his symptoms. On September 2, 1867, he wrote to the *Times* denying rumors of illness, and the next day wrote to Charles Kent: “I never was better in my life – doubt if any body ever was or can be better – and have not had anything the matter with me but that squeezed foot, which was an affair of a few days [sic]” (Letters 11: 420).

Dickens’s friends, such as Yates, could not understand his attitude:

Never did [a] man wishing to deceive himself carry out his object so thoroughly as Dickens … What would he have thought, what would he have said, of any other man who could only read half the letters of the names over the shop-doors, who ‘found himself extremely giddy and extremely uncertain of the sense of touch, both in the left leg and the left hand and arm’, and who ascribed those symptoms ‘to the effect of medicine’? With what caustic touches would he have described a man who, suffering under all those symptoms, and under many others equally significant, harassed, worn out, yet travels and reads and works until he falls dead on the roadside!” (Recollections 2. 123–24)

**Effect on Dickens’s work**

Dickens’s last complete novel was published in 1865. The first monthly installment of *Our Mutual Friend*, which Dickens started writing in 1863, was published in May, 1864. Dickens had part of the manuscript with him in the railway accident, and had to go back to the carriage for it. Inevitably, the effects of the accident took a toll and hindered progress. At work on the novel’s fourth book, he found composition more difficult. “Fancy!” he exclaimed to Forster, “fancy my having under-written number sixteen by two and a half pages – a thing I have not done since *Pickwick!*” (Letters 11: 67). Drawing closer to the
conclusion, to another friend he wrote in mid-August 1865, he was “now finishing with great pains” (Letters 11: 83). Contemporary critical acclaim for the book was muted, and Dickens started to concentrate more on his public readings than on writing. Moreover, arranging the contractual details for his last, incomplete novel, he endorsed an arrangement by which *The Mystery of Edwin Drood* was to have been completed within twelve monthly installments rather than the customary twenty numbers, a provision which carries hints of the author’s intimations of his mortality.

**Analysis of symptoms**

Dickens had a confusing mixture of symptoms, which mostly affected his left side, and which coincided with a decline in his literary productivity. Most striking of symptoms was his inability to see the left half of words, a symptom almost pathognomonic of right parietal damage, and which had not then been described in neurology. Dickens's unwillingness to move his left hand might also suggest motor neglect (Triggs et al. 1994). Could Dickens's other symptoms also be due to right parietal disorder?

The foot pain could well have been central in origin. Although usually regarded as thalamic (Dejerine-Roussy syndrome⁵), about half of patients with central post-stroke pain have a non-thalamic lesion, which is often supratentorial (Boivie and Leijon 1991). Cortical pain usually arises after parietal lobe lesions (Marshall 1951; Michel et al. 1990), so-called pseudothalamic syndrome, and most patients have right hemispheric lesions and left-sided pain (Michel et al. 1990).

Pain perception is associated with the right hemisphere (Pauli et al. 1999), and thalamic pain arises more often with right-sided damage (Nasreddine and Saver 1997). Central pain can also be associated with changes in skin temperature, trophic skin changes, and oedema in the affected limb (Boivie and Leijon 1991). Parietal lobe pain is often restricted to the face, hand, lower leg, or foot (Michel et al. 1990), sometimes of a paroxysmal quality, with a Jacksonian-like march (Fields and Adams 1974), named after the neurologist Hughlings Jackson, who first described the phenomenon in 1863, in which an epileptic discharge slowly spreads across the motor or sensory cortex, causing movement or sensation in the associated body areas. In Dickens’s case, there is a suggestion of hypalgesia (the hot poultices causing tissue damage) and of allodynia or hyperpathia (light touch being unbearable), which are both symptoms that arise in parietal damage (Marshall 1951; Fields and Adams 1974). The pain has been described by others as burning, gnawing, and electric, rather like Dickens’s description of a pain like
hot water. Dickens’s apparent indifference to his problems might be due to anosognosia, a frequent occurrence in right hemisphere damage (Hécaen 1962; Ghika et al. 1999), particularly parietal lesions (Starkstein et al. 1992). Anosognosia arises as a result of temporary cortical dysfunction, as in Wada testing, when it affects the left side of the body in particular (Carpenter et al. 1995).

Dickens’s feeling of railway carriages falling to the left could be due to vestibular symptoms of cortical origin. Patients with spatial neglect and anosognosia respond favorably to cold caloric stimulation (Rode et al. 1998); right parietal damage results in absence of normal feelings of self motion (Rode et al. 1998); parietal lobe afferents and efferents go to the vestibular system (Büttner and Lang 1979; Frederickson et al. 1974); vestibular stimulation activates a part of the cortex adjacent to the parietal cortex damaged in spatial neglect; vertiginous seizures occur in temporoparietal epilepsy (Williamson et al. 1997); and stimulation of parietal cortex can make patients feel as though they are rolling off the operating table.

**Conclusion**

For the last 5 years of his life, Charles Dickens had a range of symptoms, mostly affecting his left side, including hemispatial neglect for words, paroxysmal pains, hyperpathia or allodynia and hypoalgesia in the left foot, and a feeling of the world falling to the left. He also seemed to be unaware of the seriousness of his symptoms, which suggests some form of anosognosia. Dickens was known to have had a definite attack of right-sided cerebral insufficiency, and he died of apoplexy affecting the right side of the brain. Dickens’s left-sided symptoms thus could suggest damage to the right temporoparietal area.

**NOTES**

1 I thank Professors John Marshall, Ian Robertson, and Lauren Harris for their careful comments on a draft manuscript, and Diane Cheung for provision of an obscure reference. A version of this paper previously appeared in *Lancet* and is republished with permission by


4 See, for example, Marshall (1998), Garcia-Ruiz and Gulliksen (1999), Jacoby
A rare neurological disorder in which the body becomes hypersensitive to pain as a result of damage to the thalamus.

WORKS CITED


