A HISTORY OF TODD AND HIS PARALYSIS

OBJECTIVE: To describe the history of Robert Bentley Todd (1809–1860) and certain of his contributions to medicine, including his original and subsequent descriptions of “epileptic hemiplegia,” which came to be called “Todd’s paralysis.”

METHODS: The author conducted a comprehensive review of English-language literature, modern and historical, related to “Todd’s paralysis” and examined Todd’s original case histories and commentary by Todd, his contemporaries, and his successors.

RESULTS: Todd held that some patients “who recover from a severe fit, or from frequently repeated fits of epilepsy, are often found to labor under hemiplegia, or other modifications of palsy.” He believed that this resulted from “undue exaltation . . . [resulting in] a state of depression or exhaustion.” Interestingly, Todd was the first to present an electrical theory of epilepsy, supported by his own animal experimentation, well before his better-known successor John Hughlings Jackson (1835–1911) (famous for his investigations of partial epilepsies and the eponymous “Jacksonian march”).

CONCLUSION: Many neurologists and investigators followed Todd in acknowledging transient postictal paralysis as a distinct clinical entity. Yet whether the pathophysiology of “Todd’s paralysis” is related to “neuronal exhaustion” or excessive inhibition is still controversial.

KEY WORDS: Hemiplegia, Paralysis, Postictal, Seizure

Robert Bentley Todd (Fig. 1) was born in Dublin on April 9, 1809, the second son of 15 siblings in a large Anglican Irish family. His father, Charles Hawkes Todd, was Professor of Anatomy and Surgery at the Royal College of Surgeons in Ireland. His mother, Elizabeth Bentley, was the daughter of a colonel in the East India Company’s service (22). Robert entered Dublin University in 1825 to read for the bar, but his father died in 1826 at the age of 44 years (21, 23), leaving a large family with little means. Thereafter, Robert was persuaded to change his course of study to medicine. After obtaining a B.A. at Trinity College, Dublin, Todd entered the Royal College of Surgeons in Ireland and qualified as a Licentiate of the Royal College of Surgeons in Ireland in 1831. At Richmond Hospital in Dublin, he studied with the famous Robert J. Graves (of Graves’ disease). After moving to London, he spent two terms at Oxford in 1832, was granted its medical degree in 1833, and became a Licentiate of the Royal College of Physicians in London.

Between 1834 and 1836, he joined with several colleagues to establish a new private medical school, which would become the Westminster Hospital Medical School (26). In 1836, at the age of 27 years, he replaced Herbert Mayo as Chair of Physiology and Morbid Anatomy at King’s College (21).

TODD’S ACADEMIC WORK

Todd became a clinician, anatomist, pathologist, and physiologist. By 1835, he was already editing the first volume of what would become the massive five-volume, 6000-page Cyclopaedia of Anatomy and Physiology (30) (published in four parts between 1835 and 1859) (Fig. 2). In it, he collected the writings of expert authors with the goal of incorporating contemporary research in physiology and anatomy (expanding the method of clinicopathological correlation with microscopic findings). In Volume III, his article on “Nervous Centres” was the first to introduce the terms “afferent” and “efferent.” This volume also contains an early account of tabes dorsalis, the most common chronic disease of the spinal cord in the 19th century. Gowers (11) later credited Todd with the first really exact account of tabes dorsalis, which predated the descriptions by Romberg and Duchenne.

According to Lionel S. Beale (1), writing in the British Medical Journal in 1870, the Cyclopaedia did more “to encourage and advance the study of physiology and comparative and microscopic anatomy than any book ever published” (1, p 487). At King’s College, Todd was an excellent teacher as well: “his manner quiet and deliberate without ever being tedious. His
classes were always well-attended; and many of the old students used frequently to come to hear him”; indeed, “Dr. Todd was one of the most popular clinical teachers of his day” (1, p 487).

His distinguished pupil and colleague William Bowman came to King’s College in 1837. Bowman would become famous for his detailed descriptions of the histology of various organs, including the kidney (hence Bowman’s capsule in the kidney, Bowman’s membrane in the cornea, and Bowman’s olfactory glands). With Bowman, Todd published the two-volume monograph *Physiological Anatomy and Physiology of Man* in 1843. In this treatise, among other things, Todd described the sensory functions of the posterior columns of the spinal cord and differentiated tabes dorsalis and locomotor ataxia from pure motor paraplegias (22).

He followed this monograph with *The Descriptive and Physiological Anatomy of the Brain, Spinal Cord and Ganglia* in 1845. In this work, he reintroduced his concept of afferent and efferent nerves: “In the motor nerve the nervous force ordinarily travels from the center to the periphery, in the sensitive nerve it travels from the periphery to the center. The former is therefore *efferent*, with reference to the center . . . the latter is *afferent*” (31). This volume also contained some of Todd’s speculations on anatomy; for example, he doubted the existence of the spinal cord’s central canal as well as the existence of the “orifice to which Magendie has given the high-sounding title ‘Orifice des Cavités Encéphaliques’” (later, *foramen of Magendie*) (21, 31).

In his Lumleian Lectures delivered before the Royal College of Physicians in 1849 (23, 32), Todd addressed the pathology and treatment of convulsive disorders. “Convulsive disorders” included chorea, tetanus, and epilepsy, and Todd (32) defined convulsion as “all those irregular actions of sets of muscles that are wholly unrestrainable by the influence of the will, and which are excited and kept up by physical irritant.” Todd distinguished three types of convulsions: choreic, tonic, and clonic.

Todd saw epilepsy as a disease “characterized mainly by the occurrence at intervals sometimes remarkably uniform in duration, of attacks of loss of consciousness, frequently sudden, often preceded by some kind of warning. These attacks last for a longer or shorter interval, when the patient recovers, as if awaking from sleep, but continues in a drowsy state for a variable term” (32, p 667). Todd held that epilepsy was the outcome of “a state of abnormal nutrition of the brain,” resulting in “the unnatural development of the nervous force at particular times” (32, p 840). He drew an analogy between nerve fibers and nerve cells and zinc and copper in an electric battery:

Two dissimilar forms of nervous matter [nerve fibers and nerve cells], both bedewed by the same blood, represent the zinc and copper, with the intervening electrolyte of the battery: and as the activity of the
battery, and the energy of the galvanic force, depend on the amount of chemical action between the fluid, which is interposed between the metals and one of them, so the activity of the nutrient or chemical changes in the nervous centers determines the degree of development and vigour of the nervous force. And as in the galvanic battery we look to the nature of the metals as well as to that of the interposed fluid, to enable us to judge of the probable quantity and quality of the force generated; so, in the nervous battery, if I may so speak, we must look to the nature of the nervous matter of both kinds, and to the quantity and quality of the blood, to determine the amount of energy of the nervous force developed. (32, p 726).

To Todd, this “nervous battery” was concerned with the generation of epileptic phenomena. In these theories, as Edward H. Reynolds (25) points out, Todd was clearly influenced by his contemporary Michael Faraday (1791–1867). Todd himself states that “these periodic evolutions of the nervous force which give rise to the complete epileptic paroxysm may be compared to the electrical phenomenon described by Faraday under the name of disruptive discharge” (32, p 841).

Reynolds also makes the point that in addition to Faraday, Todd could have been influenced by his basic science colleagues at King’s College, who included Professor John Frederic Daniell, inventor of the first constant cell battery, and Professor Sir Charles Wheatstone, who developed the first electric telegraph; both of these men were associates of Faraday (EH Reynolds, personal communication). Thus, Todd was the first to formulate a true electrical theory of epilepsy, envisioning epileptic discharge as possessing “all the violence of the discharge from a highly charged Leyden jar” (32).

These concepts were strengthened by Todd’s own animal experiments. He carried out experiments on rabbits in which he applied galvanic stimulation to parts of the brain and claimed to localize epileptic paroxysms to the midbrain at the level of the corpora quadrigemina:

I then tried the corpora quadrigemina and the mesocephale. Having passed fine bradaws into the cranium in such a direction as I had previously satisfied myself would lead to this organ, I subjected it to the influence of the machine; general convulsions were produced, of a character essentially different from those which resulted from stimulating the spinal cord or the medulla oblongata. They were combined movements of alternate contraction and relaxation, flexion and extension, affecting the muscles of all the limbs, of the trunk, and of the eyes, which rolled about just as in epilepsy. On inserting the awls into the hemispheric lobes, still different effects were produced by the application of the machine. I could observe nothing like true convulsions; but slight convulsive twitchings of the muscles of the face took place, which were no more than what would be caused by the stimulus of galvanism acting upon the nerves of the face. (32, p 821)

The observation of “slight convulsive twitchings” on cortical stimulation did not lead Todd to investigate further the cortical or subcortical localization of stimulus-induced motor activity. Such evidence could have given him priority over Hitzig and Ferrier (neither of whom later mentioned Todd’s animal experiments). As Sir Geoffrey Jefferson remarked in 1935: “How modern all this is, though it has been eclipsed by much that was discovered later” (18).

Todd’s diffuse theory of localization of function within the nervous system probably inhibited his investigations of epilepsy. He held that mentation was a function of the cerebral cortex, voluntary movement came from the corpora striata, and emotions from the upper midbrain (21, 29). For example, Todd held that “the center of volition . . . reaches from the corpora striata in the brain down the entire length of the anterior horns of the gray matter of the spinal cord, and includes the locus niger in the crus cerebri, and much of the vesicular matter of the mesocephale and of the medulla oblongata . . . disease of any part of this center is capable of producing paralysis” (33, p 19).

Reynolds (25) points out that Todd’s electrical theory of epilepsy predated Hughlings Jackson’s theory by at least 20 years. John Hughlings Jackson (1835–1911) (Fig. 3), often considered the father of British neurology, is credited with laying the foundations of modern understanding of epilepsy. His studies of unilateral convulsions are immortalized in the eponym “jacksonian epilepsy” (36), and in a famous 1873 article, he defined epilepsy as “occasional, sudden, excessive, rapid and local discharges of gray matter” (14). By the early 1870s, Frisch and Hitzig and Ferrier’s contemporary investigations of...
cortical localization lent experimental support to Jackson's clinical deductions (13, 17, 25).

**TODD'S PARALYSIS**

The 1849 Lumleian Lectures also contained Todd’s original description of postictal paralysis. He stated that:

A paralytic state remains sometimes after the epileptic convulsion. This is more particularly the case when the convulsion has affected only one side or one limb: that limb or limbs will remain paralytic for some hours, or even days, after the cessation of the paroxysm, but it will ultimately perfectly recover. (32, p 668)

Todd was not the first to describe this condition, which came to be called “Todd’s paralysis.” In 1827, Bravais described “l'épilepsie hémilégique” in a thesis submitted to the University of Paris (3). However, Todd was the first to describe this condition in detail.

Examples of this condition, which he called “epileptic hemiplegia,” were elaborated in his Clinical Lectures on Paralysis (33) (Fig. 4). These were lectures presented at King’s College Hospital in the early 1850s (collected and first published in 1854 in London and 1855 in Philadelphia). In this work, he separated “hysterical paralysis” from paralysis caused by actual lesions of the brain, including epileptic paralysis. He noted correctly that hemiplegia was “paralysis of one side of the body from disease of the opposite half of the brain” (33, p 32).

Todd’s original case reports of epileptic hemiplegia, each provided with varying amounts of clinical detail, are summarized in **Table 1**. From these and other patients, he concluded that “the distinction of a form of hemiplegia in connection with the epileptic paroxysm is well founded” (33, p 203).

What was the pathogenesis of this “epileptic hemiplegia”? Todd held that the “phenomena of the epileptic fit depend on a disturbed state of the nervous force, in certain parts of the brain—a morbidly excited polarity . . . This undue exaltation of the polar force induces, subsequently, a state of depression or exhaustion, not only in the parts primarily affected, but in parts of the brain connected with them” (33, pp 204–205). This, then, accounts for hemiparesis or hemiplegia in the territory of the brain affected by the convulsion. However, the clinical distinction between “apoplectic fits” (modern-day transient ischemic attacks or strokes) caused by vascular “congestion” and Todd’s “epileptic hemiplegia” was often unclear to Todd and his colleagues, especially in the absence of a witnessed convulsion.

**OTHER DESCRIPTIONS AND STUDIES OF TODD’S PARALYSIS**

Alexander Robertson, a Glasgow neurologist, reported in 1869 four more cases of paralysis after unilateral convulsions and concluded: “I am inclined to think that the late Dr. Todd was correct in supposing that severe and protracted convulsions may themselves, in some instances, be causative of palsy of a few hours’ or days’ duration through simply the exhausting influence exerted on the cells of the central ganglia, without much, if any, appreciable change of tissue” (27, p 519).

John Hughlings Jackson acknowledged Todd’s primacy in defining “epileptic hemiplegia”: “After a convulsion beginning unilaterally, there is very often hemiplegia. Hemiplegia so occurring was called Epileptic Hemiplegia by Dr. Todd” (15). Acknowledging both Todd and Robertson, Jackson attributed the paralysis to exhaustion of nervous centers: “I have found that the hypothesis advanced is in all essential respects the hypothesis of Todd and Alexander Robertson . . . The local paralysis remaining when the convulsion is over . . . is supposed to be the external negative condition answering to the internal negative condition of exhaustion of some fibers over-worked from the excessive discharge of cells connected with those fibers. The nerve-fibers being only exhausted, not destroyed, as in some other kinds of paralysis, post-epileptiform paralysis is temporary” (16, p 434). Jackson believed that the more severe the attack, the longer the ensuing paralysis. Jackson went on to describe lesions responsible for postepileptiform paralysis: “We speak of cases in which a small tumor or other ‘foreign body’ in the midcortical region of the brain leads to instability of cells near it—leads to what I have called a ‘discharging lesion’; the cells rendered unstable occasionally ‘explode’ or liberate much energy, or, in other words, discharge excessively, and all of them much more nearly simultaneously than the comparatively stable cells do in health. After their excessive discharge has ceased, as signified by cessation of spasm, there often is paralysis, and that paralysis
TABLE 1. Todd’s original case reports of “epileptic hemiplegia”

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Seizure presentation</th>
<th>Postictal state</th>
</tr>
</thead>
<tbody>
<tr>
<td>Jonathan Woolley</td>
<td>10</td>
<td>Fits confined to R side “not lasting above four or five minutes”</td>
<td>R hemiparesis with paralysis “of motion only, and was not complete, a slight amount of power remaining”</td>
</tr>
<tr>
<td>Thomas Orton</td>
<td>34</td>
<td>“Electric shock traversing the whole of his left side, and he fell down insensible, foamed at the mouth, bit his tongue, and was convulsed”</td>
<td>L hemiplegia including facial paralysis</td>
</tr>
<tr>
<td>H. Pitt</td>
<td>26</td>
<td>“In each fit he became paralyzed on the right with relaxed muscles”</td>
<td>R hemiparesis, “became worse after each fit and recovered to a certain point before the next”</td>
</tr>
<tr>
<td>Mary A. Godfrey</td>
<td>29</td>
<td>Described only as “fits”</td>
<td>L hemiparesis for “only half an hour. . . . The paralyzed parts had their sentient power very much diminished, and there was ptosis of the left upper lid”</td>
</tr>
<tr>
<td>Ellen Biddlecomb</td>
<td>24</td>
<td>“She suddenly fell, and was convulsed and insensible for half an hour”</td>
<td>L arm monoparesis which “always subsided in one or two days”</td>
</tr>
<tr>
<td>Arnold Young</td>
<td>58</td>
<td>Each fit was “ushered in by a numbness and twitching of the right great toe, extending gradually up the leg and side to the arm” followed by “convulsive twitchings”</td>
<td>R arm and leg paralysis also involving R face and R tongue</td>
</tr>
<tr>
<td>Sarah Bone</td>
<td>63</td>
<td>“She foamed at the mouth slightly, and . . . her left hand was convulsively clenched”</td>
<td>“decided paralysis of the left side, inability to protrude her tongue straight, and her face drawn to the right side”</td>
</tr>
<tr>
<td>“A gentleman, a</td>
<td>Not stated</td>
<td>Aura of “giddiness in the head and sinking in the stomach” followed by falling “unconscious, the muscles of the mouth, left arm and leg, working spasmodically”</td>
<td>“temporary paralysis of the left side, particularly of the leg, which disappears in from fifteen to twenty minutes”</td>
</tr>
<tr>
<td>patient of my</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>friend Mr. Lynch,</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>of Sudbury</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>“A gentleman”</td>
<td>25</td>
<td>“In the course of the night he evidently had a fit, as indicated by the disturbed bed-clothes”</td>
<td>Left hemiplegia and left facial palsy “of only a few days’ duration”</td>
</tr>
</tbody>
</table>

* R, right; L, left. From, Todd RB: Clinical Lectures on Paralysis, Disease of the Brain, and Other Ailments of the Nervous System. Philadelphia, Lindsay & Blakiston, 1855 (33).
that cortical stimulation produced temporary “exhaustion” (5, 9).

In the first half of the 20th century, there were no further significant advances in the study of postictal paralysis per se, although its presence could be of clinical value in localizing lesions for surgical treatment (9, 20). In the past half-century as well, perhaps surprisingly, there have been only a few formal studies of Todd’s paralysis. In 1959, Meyer and Portnoy (6) studied postictal paralysis in 9 patients and in experimental monkeys and cats. In their experimental animals, they recorded cerebral blood flow (and examined pial arterioles via a pial window), pH, electroencephalograms, and oxygen tension during the postepileptic period. Despite increased local cortical blood flow after seizures, cortical oxygen tension fell in the affected cortex. They held that the phenomenon of Todd’s paralysis results from temporary neuronal anoxia (24). However, just two years later (1961), Efron (6) refuted Meyer and Portnoy’s evidence and adduced evidence in support of the idea that Todd’s paralysis is a result of increased inhibition rather than exhaustion, Gowers’ original explanation.

Two more recent studies demonstrate postictal hyperperfusion in patients with Todd’s paralysis. In 1975, Yarnell (35) reported the case of a patient with right-sided focal seizures associated with angiographic and radioisotopic evidence of left brain hyperperfusion and early venous drainage. He theorized that postictal paralysis was not caused by “exhaustion” but rather by local vasomotor and/or metabolic changes that led to functional local cortical ischemia and AV shunting. More recently, in 1998, Kimura et al. (19) studied postictal single-photon emission computed tomography in 2 patients with Todd’s paralysis. Measuring technetium uptake 18 hours and 31 hours after the seizures, respectively, demonstrated cerebral hyperperfusion in each case.

The most complete modern study of the epidemiology of Todd’s paralysis was by Rolak et al. (28). These authors prospectively evaluated 229 patients with secondarily generalized tonicoclonic seizures and identified 14 with transient focal weakness. In this group, postepileptic paralysis persisted from half an hour to 36 hours, with a mean of 15 hours (28). Interestingly, a recent report of two patients illustrates that bilateral Todd’s paralysis may develop after seizure onset in the supplementary motor area (2).

Even today, the precise physiological explanation for Todd’s paralysis is obscure. Whether the exact cause of Todd’s paralysis is depressed neuronal metabolism (which would be a modern term for “exhaustion of the nervous elements”) or active inhibition of motor centers, as hypothesized by Gowers, still awaits further elucidation. As Gowers admitted, postictal focal exhaustion and inhibition may coexist: “A center may be in a state of partial inhibition and partial discharge, and either condition may preponderate and ultimately obtain exclusively” (12, p 123). Many mechanisms may account for the postictal state in general, including neurotransmitter depletion, neuronal desensitization, altered local cerebral blood flow (34), and various forms of active inhibition (7).

**TODD’S PANACEA AND DEMISE**

In addition to his many academic pursuits, Todd developed a large private practice, accompanied by a widespread reputation for overprescribing alcohol for fever and other ailments. Todd and his Dublin teacher Robert J. Graves reacted against the contemporary “antiphlogistic” treatments of bleeding and purging and advocated support of nourishment and stimulation. This included prescription of large quantities of alcohol for stimulation (“he often ordered his patients a pint and a half of brandy a day in addition to wine and port” [4, p 893]). On the occasion of Todd’s resignation from King’s College in 1853, in his farewell lecture, he paid tribute to Graves: “From him I imbibed a taste for physiological enquiry; and, under his guidance and direction, my first studies upon that subject were pursued” (22, p 12). He also must have imbied a great deal of his own panacea, because, like his father Charles Hawkes Todd, Robert Bentley died by hematemesis secondary to hepatic cirrhosis in his own consulting room in January 1860.

**CONCLUSIONS**

Robert Bentley Todd’s main contributions to neurology and medicine were his account of postictal hemiplegia, the introduction of the terms afferent and efferent, the Cyclopaedia, his description of tabes dorsalis, his founding of King’s College Hospital, and his multiple innovations in medical and nursing.

---

**FIGURE 5. Photograph of Sir William Richard Gowers (1845–1915).**

Copyright © Congress of Neurological Surgeons. Unauthorized reproduction of this article is prohibited.
education (4, 26). For these alone, he is certainly worthy of his statue outside the current King’s College Hospital. However, the fact that Todd was also the first to formulate an electrical theory of epilepsy is little known. This distinction is usually credited to John Hughlings Jackson, who described his epilepsy theory in his own brilliant Lumleian Lectures in 1890, a full 41 years after Todd’s Lumleian Lectures “On the pathology and treatment of convulsive diseases.” That postictal hemiplegia should have come to bear the name “Todd’s paralysis” is well deserved. Todd himself, however, might not have desired such an eponym: “I must say that I cannot regard it as any compliment to the great names of our profession, to attach them to any of the numerous ills which flesh is heir to” (10, p 334).

REFERENCES


COMMENTS

I thoroughly enjoyed reading this eloquent account of the history of Todd and his paralysis. I certainly learned a great deal, and Todd seems a remarkable man who has not received appropriate recognition until this article. I suspect that very few of us knew that he was the first person to introduce the terms “afferent” and “afferent” and to stress the electrical nature of brain activity. He also went on to found the Westminister Hospital Medical School before migrating down to King’s College Hospital in South London. He was indeed a truly remarkable man, and I welcome this beautifully researched and written article on him.

Christopher B.T. Adams
Oxford, England

Robert Bentley Todd is a name well known to the modern neurosurgical audience. Yet his eponym, “Todd’s paralysis,” is known to every student of medicine, so common is its usage. After reading this article, one gains a real understanding of how Todd developed his anatomic and physiological concepts on epilepsy. In addition, the reader is also
made aware of Todd’s considerable contributions to the field of medicine. Also woven into this story is a nice historical perspective on some of the cast of characters working at the same time on trying to understand how the brain works. By the end of the article, we also gain an appreciation that despite modern imaging and diagnostic tests, we still do not fully understand what happens to one’s brain in the development of Todd’s paralysis. But this is only one shortcoming, because when you finish reading this piece, Robert Bentley Todd has become a real person, never to be looked at the same. This is an excellent historical contribution, nicely outlined and researched here, and offers a look at some of the other giants of the day in addition to Todd: Jackson, Gowers, Hitzig, and others.

James T. Goodrich
Bronx, New York

This is a delightful account of an individual who is important in the history of neurology, and the article gives us a wonderful historical perspective on the man, his work, and the time during which this original description and Todd’s concepts developed. Surely there must have been intensive and pervasive excitement in the scientific community about the discoveries by Faraday and others involving electricity. Todd’s electrical theories of brain function reflect this excitement and the revolutionary nature of these discoveries. It is a shame that Todd was not able to incorporate his experimental work and its results into a theory of cerebral localization. As the author, Dr. Binder, points out, Todd’s concept of brain function simply did not allow for what we believe is the correct interpretation of his observations. This is a wonderful insight into the medical history of our field and gives the reader a new sense of respect for the discoveries and observations made by Dr. Todd.

Edward R. Laws, Jr.
Charlottesville, Virginia

It is enjoyable to learn more about both the substantial contributions and the person of Robert Bentley Todd, known to most of us otherwise only as an eponym attached to seizure-related hemiplegia. It is also interesting to note that in addition to his appreciation of seizures as an electrical event (adapting to his field the paradigm of a most popular physical phenomenon of the day), Todd so presciently viewed the whole condition of epilepsy as one of “a state of abnormal nutrition of the brain.” Recent investigations into the energy metabolism of the epilepsy substrate, generally perceived as provocative and novel, may be rediscovering a perspective first appreciated more than 150 years ago.

David W. Roberts
Lebanon, New Hampshire

In-training Liaison

The Congress of Neurological Surgeons exists for the purpose of promoting public welfare through the advancement of neurosurgery by a commitment to excellence in education and by a dedication to research and scientific knowledge.

—Mission Statement, Congress of Neurological Surgeons

Inherent in this commitment is a critical charge to serve the needs of the in-training individual. Considering the importance of this vital group within the neurosurgical community, the Journal has established a position within its board structure termed In-training Liaison. The individual holding this position will act as a spokesperson especially addressing the needs and concerns of individuals in in-training positions globally, as they relate to journal content and perspective.

The current individual holding this position is:

Alfredo Quiñones-Hinojosa, M.D.

Issues attendant to in-training matters should be conveyed to Dr. Quiñones-Hinojosa at the Department of Neurosurgery, University of California, San Francisco, 505 Parnassus Avenue, Room M-779, San Francisco, CA 94143-0112. Tel: 415/502-2965; Fax: 415/753-1772; email: quinones@neurosurg.ucsf.edu.