International Neurosurgery Rotation in New Zealand: Analysis of Operative Experience

Sir,

The duration of neurosurgical training in the United States is 7 years and includes 30 months of electives, which allows time for trainees to obtain research or additional clinical experience. The University of Virginia (Charlottesville, Virginia, USA) is one of only a handful of neurosurgical training programs in the United States which still integrates an international rotation. Every year, two residents from the University of Virginia are sent to Auckland City Hospital, and one resident is sent to Christchurch Hospital for a minimum of 12 months. During this yearlong rotation, the residents serve as the equivalent of senior neurosurgical registrars in the Surgical Education and Training program of the Royal Australasian College of Surgeons. The case volumes from the New Zealand rotation have previously been summarized.[1] However, detailed operative logs regarding the type of cases and lesions encountered during this rotation are absent. Therefore, the aim of this report is to critically analyze the operative experience of a 6th year University of Virginia neurosurgery resident who spent 12 months as a senior neurosurgical registrar at Auckland City Hospital.

A prospectively collected list of operating theater cases performed by a senior neurosurgical registrar at Auckland City Hospital from June 8, 2015, to June 3, 2016, was retrospectively evaluated. Lesions were classified by pathology, location, and presentation. Cases were categorized by the type of procedure performed. A total of 334 cases were performed by the registrar, and they are summarized in Table 1. The cases included 35 pediatric (age <18 years) patients, ranging in age from 1 month to 16 years. More than 70% of procedures performed by neurosurgeons in the United States are spinal operations, primarily for the treatment of spondylosis. By comparison, <10% of cases in the present analysis were performed for a spinal disorder, which indicates the predominantly cranial bias of the procedures during this rotation. Significant advances in neuroendovascular technology and widespread adoption of endovascular approaches by the cerebrovascular community has substantially decreased the number of intracranial aneurysm patients undergoing surgical treatment in the United States.[2-5] The ramifications of this paradigm shift in aneurysm treatment include a more limited exposure of trainees to the microsurgical techniques necessary to perform safe and effective clipping of an aneurysm.

In this analysis, a total of 27 intracranial aneurysms were surgically clipped in 24 patients. Of the 24 aneurysm patients, 18 were treated in the setting of acute subarachnoid hemorrhage (75%). Of the 27 aneurysms, 12 were located on the middle cerebral artery (44%), nine were located at the anterior communicating artery (33%), three were located at the internal carotid artery terminus (11%), and one each were located at the anterior choroidal, distal anterior cerebral, and superior cerebellar arteries (4%). Of the five surgically resected brain arteriovenous malformations, four were ruptured (80%), and one was located in the posterior fossa (20%). The two remaining intracranial vascular lesions included a petrosal dural arteriovenous fistula and a premotor cavernous malformation. An additional 26
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| Procedure Classification                                      | Number |
|---------------------------------------------------------------|--------|
| Craniotomy for intracranial vascular lesion                   | 31     |
| Aneurysm                                                      | 24     |
| Arteriovenous malformation                                    | 5      |
| Cavernous malformation                                        | 1      |
| Dural arteriovenous fistula                                    | 1      |
| Craniotomy for tumor                                          | 78     |
| Intra-axial                                                   | 51     |
| Extra-axial                                                   | 27     |
| Craniotomy for trauma or stroke                               | 30     |
| Decompressive craniectomy only                                | 4      |
| Epidural hematoma evacuation                                  | 5      |
| Subdural hematoma evacuation                                  | 13     |
| Intracerebral hematoma evacuation                              | 8      |
| Pain procedures                                               | 12     |
| Microvascular decompression for trigeminal neuralgia          | 6      |
| Microvascular decompression for hemifacial spasm              | 2      |
| Posterior fossa decompression for Chiari I malformation       | 2      |
| Balloon rhizotomy for trigeminal neuralgia                    | 2      |
| Functional procedures                                         | 5      |
| Deep brain stimulator electrode implantation or replacement   | 1      |
| Implantable pulse generator implantation or replacement       | 4      |
| Craniotomy for epilepsy                                       | 3      |
| Biopsy procedures                                             | 11     |
| Craniotomy                                                    | 6      |
| Burr hole                                                     | 5      |
| Spine (vascular)                                              | 3      |
| Spine (tumor)                                                 | 9      |
| Extradural                                                    | 1      |
| Intradural extramedullary                                     | 6      |
| Intramedullary                                                | 2      |
| Spine (degenerative)                                          | 10     |
| Fusion (with instrumentation)                                 | 3      |
| Decompression (without instrumentation)                       | 7      |
| CSF diversion                                                 | 45     |
| Primary shunt implantation                                    | 26     |
| Shunt revision                                                | 14     |
| Shunt explantation or externalization                         | 4      |
| Endoscopic third ventriculostomy                              | 1      |
| Miscellaneous procedures                                      | 61     |
| Burr drainage of chronic subdural hematoma                    | 25     |
| Washout of postoperative hematoma or wound infection          | 17     |
| Cranioplasty                                                  | 4      |
| Peripheral nerve                                              | 3      |
| Osteoma                                                       | 3      |
| CSF leak repair                                               | 3      |
| Other                                                         | 6      |
| Endovascular procedures                                       | 7      |
| Aneurysm coil embolization                                    | 3      |
| Aneurysm flow diversion                                       | 2      |
| Diagnostic cerebral angiography                                | 2      |
| Minor procedures                                              | 29     |

*Denotes number of patients treated; a total of 27 aneurysms were treated. CSF: Cerebrospinal fluid*

nonvascular skull base cases were performed, including 17 tumor resections, eight microvascular decompressions, and one repair of tegmen tympani defect.

In summary, the international neurosurgery rotation in New Zealand exposes residents at the University of Virginia to a wide range of intracranial pathology and affords invaluable operative experience in complex cranial surgery, particularly with regard to vascular and skull base procedures. The rotation sites in New Zealand are not accredited by the Accreditation Council for Graduate Medical Education, and therefore, these cases are not accounted for in the resident’s official procedural log. However, the operative experience obtained during the New Zealand rotation considerably improves the resident’s overall competency in both cranial neurosurgery and microsurgery.

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Friedreich Ataxia: Clinical Feature and Electrophysiological Symptoms

Friedreich ataxia is inherited as an autosomal recessive disorder involving the spinocerebellar tracts, dorsal columns in the spinal cord, the pyramidal tracts, and the cerebellum and medulla. The majority of patients bear a recessive GAA triplet-repeat expansion on intron 1 of both alleles while a minority carry an expansion on one allele and a point mutation or deletion on the other. [1,2] The disease-causing genotype leads to decreased production of frataxin. [1,2] Mutations cause oxidative injury associated with excessive iron deposits in mitochondria; frataxin deficiency leads to mitochondrial iron accumulation, deficient production of adenosine triphosphate, and a potential rise in free radical generation. [1,2]

These events lead to onset of a variety of symptoms, such as gait disturbance, loss of sensation, areflexia, and dyscoordination, beginning usually between ages 5 and 15 years although later onset is not uncommon. [3] Approximately two-thirds of individuals with Friedreich ataxia have cardiomyopathy and up to 30% have diabetes mellitus. [4] The loss of frataxin function in mitochondria accounts for these pathogenic processes in Friedreich ataxia. Mitochondria are essential for the sensing of nutrients by the β cell and for the generation of signals that trigger and amplify insulin secretion, known as stimulus-secretion coupling. Moreover, in the intrinsic pathway of apoptosis, pro-apoptotic signals converge on mitochondria, resulting in mitochondrial Bax translocation, membrane permeabilization, cytochrome c release and caspase cleavage. [5]

The ataxia is slowly progressive and involves the lower extremities to a greater degree than the upper extremities. In general, results of electrophysiologic studies including visual, auditory brainstem, and somatosensory-evoked potentials are often abnormal. [6] In the article “Diabetes Mellitus as the Presenting Feature of Friedreich Ataxia,” [7] the authors report a case of an 8-year-old girl who initially presented with diabetic ketoacidosis and was treated as case of insulin-dependent diabetes mellitus. Furthermore, they report an axonal type of generalized sensory neuropathy and lower MCV in tibial nerve. The authors assumed the diagnosis of Friedreich ataxia for the patient. However, neuroimaging and FXN gene analysis were not conducted. The co-morbidity of diabetes mellitus and peripheral neuropathy can also result from mitochondrial disorders, and could represent a complication of hereditary motor and sensory neuropathy [8] in a diabetic patient by chance. In addition, atypical Friedreich ataxia due to compound heterozygosity for FXN GAA expansion and a point mutation may present a greater diagnostic dilemma.

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