the guideline for interventional cardiologists is 75 percutaneous coronary interventions per year per operator.[8]

5. Experience from cardiology showed that mandated interventional cardiology training programs' accreditation by ACGME and mandated board certification of the trainees reduced the number of graduates by 50%.[8]

An immediate call to action should consider the above recommendations not only to address manpower but also to meet our societal responsibility for future, high-quality neurointerventionalists.

1. Zaidat OO, Lazzaro M, McGinley E, et al. Demand-supply of neurointerventionalists for endovascular ischemic stroke therapy. Neurology 2012;79:S35–S41.
2. Fiorella D, Hirsch JA, Woo HH, et al. Should neurointerventional fellowship training be suspended indefinitely? J Neurointerventional Surg 2012;4:315–318.
3. Suzuki S, Saver JL, Scott P, et al. Access to intra-arterial therapies for acute ischemic stroke: an analysis of the US population. AJNR Am J Neuroradiol 2004;25:1802–1806.
4. Cross DT, 3rd, Tirschwell DL, Clark MA, et al. Mortality rates after subarachnoid hemorrhage: variations according to hospital case volume in 18 states. J Neurosurg 2003;99:810–817.
5. Gupta R, Horev A, Nguyen T, et al. Higher volume endovascular stroke centers have faster times to treatment, higher reperfusion rates and higher rates of good clinical outcomes. J Neurointerv Surg Epub 2012 Jul 25.
6. Grigoryan M, Chaudhry SA, Hassan AE, Suri FK, Qureshi AI. Neurointerventional procedural volume per hospital in United States: implications for comprehensive stroke center designation. Stroke 2012;43:1309–1314.
7. Freed GL, Nahra TA, Wheeler JR; Research Advisory Committee of American Board of Pediatrics. Counting physicians: inconsistencies in a commonly used source for workforce analysis. Acad Med 2006;81:847–852.
8. Feldman T. Interventional cardiology manpower needs: how many of us are there? How many should there be? How many will we need in the future? Catheter Cardiovasc Interv 2003;58:137–138.

VARICELLA-ZOSTER VIRUS ENCEPHALITIS AND VASCULOPATHY IN A PATIENT TREATED WITH FINGOLIMOD

Patricia H. McNamara, Janice M.T. Redmond, Colin P. Doherty, Dublin: Ratchford et al.[1] reported a patient with fingolimod-related varicella encephalitis and vasculopathy. The patient's baseline mobility was wheelchair-bound, which means that his Expanded Disability Status Scale (EDSS) score[2] was at least 7.0. This suggests that he was no longer in the inflammatory stage of multiple sclerosis (MS) and had secondary progressive MS (SPMS). It is unclear whether the patient's condition met criteria for natalizumab but—at a minimum—the condition must have progressed while on natalizumab. There is neither licensing support nor data from pivotal trials for efficacy of this drug in patients with SPMS.[3] The evidence supporting the extension of natalizumab beyond the standard 24 months is also lacking. Fingolimod is licensed for patients with active relapsing-remitting MS but Ratchford et al. stated that this patient was clinically stable. This case highlights an interesting and serious consequence of starting therapy with fingolimod, but the evidence for starting and continuing treatment with this medication—and indeed natalizumab—is lacking in this patient group. This case report also emphasizes the need for evidence-based care, which is safer and more cost-effective.

Author Response: **John N. Ratchford, Kathleen Costello, Daniel S. Reich, Peter A. Calabresi, Baltimore:** We thank McNamara et al. for their interest in our case report. We agree that natalizumab and fingolimod should only be prescribed for patients with relapsing forms of MS or in the context of a clinical trial, unless definitive trial data could prove their efficacy in progressive MS. An EDSS score of 7.0 would suggest that our patient had SPMS, but that was not the case; our patient accrued disability in a stepwise fashion. Furthermore, the lack of any progression in the absence of relapses while on natalizumab or at any other stage of this disease is inconsistent with the diagnosis of SPMS. It would be unfortunate if patients who accrue disability through severe relapses were deprived of an effective medication by misinterpreting "progression of disease" for "progressive disease."

1. Ratchford JN, Costello K, Reich DS, et al. Varicella-zoster virus encephalitis and vasculopathy in a patient treated with fingolimod. Neurology 2012;79:2002–2004.
2. Kurtzke JF. Rating neurologic impairment in multiple sclerosis: an expanded disability status scale (EDSS). Neurology 1983;33:1444–1452.
3. Polman CH, O'Connor PW, Havrdova E, et al. A randomized, placebo-controlled trial of natalizumab for relapsing multiple sclerosis. N Engl J Med 2006; 354:899–910.

Author disclosures are available upon request (journal@neurology.org).

Varicella-zoster virus encephalitis and vasculopathy in a patient treated with fingolimod

Patricia H. McNamara, John N. Ratchford, Janice M.T. Redmond, et al.

Neurology 2013;81;306
DOI 10.1212/01.wnl.0000432547.27815.74