Is there a higher risk of restless legs syndrome in peripheral neuropathy?

Erin Hattan, Colin Chalk and Ronald B. Postuma

Neurology 2009;72;955-960; originally published online Nov 26, 2008;
DOI: 10.1212/01.wnl.0000336341.72621.db

Is there a higher risk of restless legs syndrome in peripheral neuropathy?

Erin Hattan, MD
Colin Chalk, MD, CM
Ronald B. Postuma, MD, MSc

Address correspondence and reprint requests to Dr. Ronald B. Postuma, Division of Neurology, L7-305 Montreal General Hospital, 1650 Cedar Ave., Montreal, Quebec, Canada H3G 1A4
ronald.postuma@mcgill.ca

ABSTRACT

Objective: Associations between peripheral neuropathy and restless legs syndrome (RLS) have been described, but have not been consistently reproduced. If RLS prevalence is truly increased by neuropathy, this has important implications for RLS pathophysiology.

Methods: In a case-control design, 245 patients with peripheral neuropathy and 245 age- and sex-matched controls were screened for RLS using a standardized phone questionnaire based on international RLS diagnostic criteria. All persons who answered yes to three of four criteria were considered screen-positive. All screen-positive patients underwent a confirmatory diagnostic evaluation by a movement disorders specialist blinded to the neuropathy status of the patient. RLS prevalence was calculated and compared using Fisher exact test.

Results: A total of 65 (26.5%) patients with neuropathy screened positive compared to 25 (10.2%) controls ($p < 0.0001$). However, the diagnosis was confirmed in only 46% of screen-positive patients with neuropathy, vs 80% of controls ($p = 0.005$). Cramps and paresthesia without true diurnal variation or rest exacerbation were the commonest causes of false-positive screens. After diagnostic confirmation, the overall prevalence of RLS did not differ between neuropathy patients and controls (12.2% vs 8.2%, $p = 0.14$). However, when classified by etiology, RLS was found in 14/72 (19.4%) patients with hereditary neuropathy, a prevalence higher than found in controls ($p = 0.016$) and acquired neuropathy (9.2%, $p = 0.033$). Among patients with neuropathy, those with RLS more commonly had a family history of RLS (37% vs 15%, $p = 0.007$) and were younger (49.9 vs 61.4, $p = 0.0003$).

Conclusions: Restless legs syndrome is more prevalent among patients with hereditary neuropathy, but not in those with acquired neuropathies. *Neurology*® 2009;72:955–960

GLOSSARY

CIDP = chronic inflammatory demyelinating polyneuropathy; **GBS** = Guillain-Barré syndrome; **HMSN** = hereditary motor sensory neuropathy; **HSAN** = hereditary sensory and autonomic neuropathy; **IRLSSG** = International Restless Legs Study Group; **MGUS** = monoclonal gammopathy of uncertain significance; **NCS** = nerve conduction studies; **PPV** = positive predictive value; **RLS** = restless legs syndrome.

Restless legs syndrome (RLS) is characterized by an urge to move the legs at night (usually accompanied by unpleasant leg sensations), exacerbation of this urge with rest, relief with activity, and worsening of symptoms toward evening.[1] The prevalence of RLS in North American and European populations is approximately 10%.[2] Several lines of evidence point to abnormalities of the CNS in the pathophysiology of RLS. Pathologic studies have demonstrated loss of dopamine receptor staining in substantia nigra pars compacta in patients with RLS.[3] Additionally, there is evidence that RLS may be related to abnormal dopaminergic transmission in the A11 cell group located near the hypothalamus,[4] and to failure of CSF transport of iron into the CNS.[5]

Peripheral neuropathy is commonly listed as a secondary cause of RLS. However, prevalence estimates of RLS in neuropathy are extremely variable, ranging from 5.2 to 54%.[6-11] This

Supplemental data at www.neurology.org

Editorial, page 950

e-Pub ahead of print on November 26, 2008, at www.neurology.org.
From the Departments of Neurology & Neurosurgery and Medicine, McGill University, Montreal General Hospital, Montreal, Quebec, Canada.
Supported by the Weston Foundation and by the Fonds de la recherche en santé du Québec.
Disclosure: The authors report no disclosures.

variability between studies may be due to non-standardized definitions of neuropathy or RLS, diagnostic inaccuracy due to symptom overlap between neuropathy and RLS, and variations in etiology of neuropathy between cohorts. Most studies have lacked comparative control populations, and none have been blinded to neuropathy status. If there truly is an association between neuropathy and RLS, this has important implications for treatment and screening for RLS, and challenges our current concepts of RLS as a CNS disease.

Given these uncertainties about the relationship between neuropathy and RLS, we conducted a systematic study of RLS prevalence in a large cohort of patients followed in a peripheral neuropathy clinic, using a standardized RLS diagnostic procedure with blinded assessment, compared to a matched control population.

METHODS

Patient selection and determination of neuropathy diagnosis. The study was conducted between July 2006 and August 2007. Approval was obtained from the Research Ethics Board of the Montreal General Hospital and participants gave informed consent according to the Declaration of Helsinki. All patients over 18 years in the clinical database of the McGill University Health Center Peripheral Neuropathy Clinic seen between 1998 and 2007 were eligible for inclusion. The diagnosis of neuropathy was based on clinical symptoms, signs of nerve dysfunction in the lower extremities, and standard nerve conduction studies (NCS). NCS were performed in all patients, but to allow inclusion of patients with isolated small fiber neuropathy, NCS abnormalities were not required if the diagnosis was supported by clinical signs or quantitative sensory testing. Exclusion criteria included dementia (Folstein Mini-Mental State Examination <24 with impairment of daily activities[12]) or inability to understand the consent process.

The most likely etiology of each patient's neuropathy was determined by clinical features, electrodiagnostic findings, laboratory data, and, in some patients, genetic testing. The etiologies were then divided into inherited or acquired, and the confidence with which the diagnosis of inherited was made was classified as certain, probable, or possible according to preset criteria (see appendix e-1 on the *Neurology*® Web site at www.neurology.org). Patients with inherited neuropathies were subdivided into hereditary motor sensory neuropathy (HMSN)1, HMSN 2, hereditary sensory and autonomic neuropathy (HSAN), and other inherited. Acquired neuropathies were classified as diabetic, toxin/medication-induced, inflammatory-demyelinating, monoclonal gammopathy of unknown significance-associated, vasculitic, autoimmune/inflammatory, other, and unknown. If multiple potential causes were found, the single likeliest cause was used for classification purposes.

Controls were selected from two sources: patient spouses or friends, and general neurology clinic patients. Spouse/friend controls (n = 151) were nominated by the patients themselves. To be eligible, the control had to be within 5 years of age and not genetically related to the patient. Neurology clinic controls (n = 94) were consecutively selected from general neurology outpatient clinics at the Montreal General Hospital. To prevent confounding by comorbidity, patients referred for pain syndromes other than headache, RLS, suspected neuropathy, or Parkinson disease were excluded as neurology clinic controls. Controls were frequency-matched to the patient group by sex and age (within 5-year intervals).

Screening. All eligible patients with neuropathy were sent an introductory letter notifying them of the study and advising them of an upcoming phone call. The letter included the opportunity to contact the research center if they did not wish to participate. Patients were then telephoned by a research assistant. At this time the project was described and verbal consent for participation in a telephone interview was sought; if granted, the interview proceeded directly. The interview consisted of systematic questionnaire based upon a validated telephone screening questionnaire assessing the diagnostic criteria for RLS, as determined by the International RLS Study Group[13,14] (appendix e-2). In addition to questions directly related to RLS symptoms, information was gathered about medical history, current and past use of medications, and family history of RLS. Medical records of each patient were reviewed to confirm medical history, use of medications, and results of laboratory testing where available.

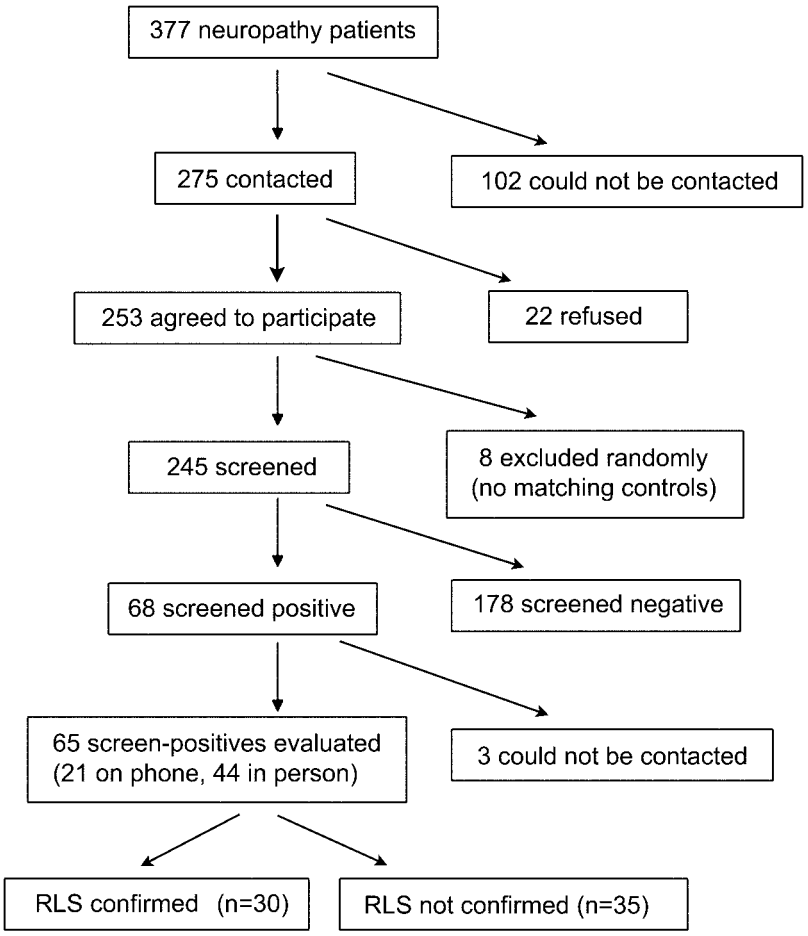
The controls were given the identical questionnaire, with the addition of a peripheral neuropathy screening interview (appendix e-3). The neuropathy interview was based on a validated five-item questionnaire developed for population surveys.[15] Controls with three or more positive responses on the neuropathy questionnaire were excluded from the study, and another control was found (14 potential controls were excluded on this basis).

The sensitivity of the RLS telephone screening questionnaire in a general population is reported to be 97% when positive responses to all four questions are obtained.[14] To allow for the possibility that the questionnaire might perform differently in patients with neuropathy, we elected to maximize the questionnaire's sensitivity and considered any subject who responded positively to three of the four questions to be screen-positive for RLS.

Diagnostic confirmation. Subjects (patients with neuropathy and controls) who were screen-positive on the RLS telephone questionnaire were invited to clinic to undergo a confirmatory diagnostic evaluation by a movement disorders specialist (R.P.) who was blinded to the subject's neuropathy diagnosis. During this consultation, the same RLS questionnaire was administered in person, clarifying whether each criterion was truly present. In the case of difficult differential diagnosis, full clarification of patient symptoms was allowed as part of the diagnostic process, even if this could disclose symptoms suggestive of neuropathy. If a patient was unable or unwilling to come to clinic for evaluation, a second telephone interview (conducted by R.P.) similar to the clinic evaluation was performed. All subjects ultimately determined to have RLS were offered standard investigation and treatment.

Outcome measures and analysis. The primary outcome measure was the prevalence of RLS in patients with neuropathy vs controls. Prespecified secondary outcomes were the prevalence of RLS in subtypes of neuropathy, the mean international RLS severity score[16] among patients with neuropathy with RLS vs controls, and the positive predictive value (PPV) and specificity of the screening questionnaire in patients with neuropathy vs

Figure Patient flow



RLS = restless legs syndrome.

controls. In addition, the prevalence of potential confounding factors or effect modifiers, such as gender, family history, anemia, renal failure, other comorbid conditions, pain or paresthesia as presenting symptom of neuropathy, and use of agents that potentially treat RLS symptoms, were compared between patients with neuropathy with and without RLS. For categorical variables (including the primary outcome), analysis was conducted using the Fisher exact test (two-tailed). For continuous variables, statistical analysis was conducted using Student *t* test (two-tailed).

Table 1 Results of diagnostic evaluation

	Neuropathy patients	Controls	*p* Value
Men/women	107/138	107/138	N/A
Age (mean ± standard deviation)	61.0 ± 14.7	62.0 ± 14.0	N/A
Screened positive (≥3 criteria)	68 (27.7%)	26 (10.6%)	<0.0001
Screened positive, all four criteria only	45 (18.4%)	15 (6.1%)	<0.0001
Diagnosed with RLS	30 (12.2%)	20 (8.2%)	0.14
Proportion screen positive diagnosed (≥3 criteria)	30/65 (46.2%)	20/25 (80%)	0.005
Proportion screen positive diagnosed (all four criteria only)	25/43 (58.1%)	13/15 (86.7%)	0.065
Mean IRLSSG score (mean ± standard error)	17.3 ± 1.5	12.7 ± 1.0	0.020

RLS = restless legs syndrome; IRLSSG = International Restless Legs Study Group.

RESULTS **Patient recruitment and participation.** Patient recruitment and flow are presented in the figure. A total of 377 patients were found in the Neuropathy Clinic database. Of these, 245 (65%) participated in the study. A total of 102 patients could not be contacted, 22 refused to participate, and 8 men were randomly excluded after diagnostic evaluation because of a lack of matched controls. Therefore, the total number of subjects participating in the study was 490 (245 patients with neuropathy and 245 controls). Within each group, there were 138 women, and the average age of all subjects was 61 ± 15 years (table 1).

Prevalence of RLS and predictive value of screen. The results of the RLS prevalence evaluation are presented in table 1. Of the patients with neuropathy, 68 (27%) were screen-positive by the initial RLS telephone questionnaire. Of these 68 patients, 44 were further evaluated in clinic and 21 by telephone interview (3 patients could not be contacted for the confirmatory evaluation). The diagnosis of RLS was confirmed in 30 of these patients, yielding a PPV for the RLS telephone screening questionnaire of 46%. On post hoc analysis, if the more demanding criterion of positive responses to all four questions on the screening questionnaire was used, 43 patients with neuropathy would have been screen-positive, 25 of whom were confirmed to have RLS, yielding a PPV of 58%. Using a cutoff of three positive responses captured 22 additional patients, of whom 5 (23%) ultimately proved to have RLS. In our neuropathy population, the specificity of the screening questionnaire was 82% using a 3/4 criteria cutoff, and 91% using a 4/4 cutoff.

Of the 245 controls, 26 (10.6%) were screen-positive with the initial RLS telephone questionnaire. This screen-positive rate was substantially lower than that in patients with neuropathy ($p < 0.0001$). Of these 26 controls, 14 were further evaluated in clinic, and 11 by telephone interview (one control could no longer be reached). The diagnosis of RLS was confirmed in 20 (80%) of the evaluated controls, yielding a PPV of 80%, higher than that in patients with neuropathy ($p < 0.005$). If the stricter cutoff of 4/4 criteria was used post hoc for the screening telephone questionnaire, 15 controls would have been considered screen-positive, of whom the diagnosis was confirmed in 13 (PPV = 87%). Using a cutoff of three positive responses captured 10 additional subjects, of whom 7 ultimately proved to have RLS. The specificity of the RLS telephone screening questionnaire in the control population was 98% us-

ing 3/4 criteria and 99% using 4/4 criteria, values higher than in the neuropathy population ($p < 0.0001$ for both). There was no difference in RLS prevalence between spouse/friend controls (7.9%) and clinic controls (8.5%).

Overall, after the comprehensive evaluation, we found the true prevalence of RLS to be slightly higher in patients with neuropathy than in our controls (12.2% vs 8.2%), but this difference was not significant ($p = 0.14$). The mean IRLS severity score was higher among patients with neuropathy than controls (17.3 vs 12.7, $p = 0.02$).

Causes of false-positive screens. In the neuropathy group, 35 patients who were screen-positive proved not to have RLS. The symptoms of 11 of these patients lacked diurnal variation—most stated that their symptoms seemed more noticeable at night because of less distraction but that they were of equal intensity throughout the day, during periods of similar lack of distraction. An additional 10 patients denied relief of their symptoms with movement or augmentation with rest. Six patients had absence of both diurnal variation and relief with movement. In 8 patients exploration of the quality of the symptoms revealed that they were experiencing cramps (i.e., painful muscle spasms with evidence of muscle rigidity on palpation) rather than restlessness. In the 5 control subjects who were screen-positive but did not have RLS, two lacked diurnal variation, one had uncomfortable feelings but no urge to move the legs, one had transient symptoms that had resolved and could not be clarified further, and one had cramps.

RLS prevalence and etiology of neuropathy. Seventy-two of our 245 (29.3%) patients with neuropathy had hereditary neuropathy (table 2). Diagnoses included 20 with HMSN-1 (14 certain, 5 probable, 1 possible), 31 with HMSN-2 (17 probable, 14 possible), 11 with HSAN (6 probable, 5 possible), and 10 with other inherited neuropathies (3 X-linked HMSN, 5 hereditary neuropathy with liability to pressure palsies, 2 other/unknown). In patients with hereditary neuropathy, the diagnosis of RLS was confirmed in 19.4%, a prevalence higher than in patients with acquired neuropathy (9.2%, $p = 0.033$) and controls (8.2%,$p = 0.016$). Findings were similar if patients with diagnosis of possible inherited neuropathy were excluded (table e-1). In contrast, the RLS prevalence in patients with acquired neuropathy was similar to controls (9.2% vs 8.2%, $p = 0.73$). No subtype of acquired neuropathy had an RLS prevalence higher than the control prevalence.

Characteristics of patients with neuropathy with and without RLS. Patients with neuropathy with RLS were on average younger than those without (49.9 vs 61.0 years, $p < 0.001$) (table e-2). Of patients with neuropathy with RLS, 37% had a family history of RLS, compared to 15% of those without RLS ($p = 0.007$). A total of 53% of patients with neuropathy with RLS were men, compared to 43% of patients with neuropathy without RLS ($p = 0.43$), suggesting that gender was not a risk factor for RLS in patients with neuropathy. There were no differences in any other demographic and medical variables between patients with neuropathy with and without RLS. In particular, hemoglobin and ferritin levels, and prevalence of anemia, renal failure, or diabetes did not differ, suggesting that confounding by these comorbid conditions was not present. There was no difference in the use of potential RLS suppressant agents or in the predominant neuropathy symptom (pain, paraesthesia, or weakness) between patients with neuropathy with and without RLS.

DISCUSSION We have found that the overall prevalence of RLS in patients with neuropathy was not different from the prevalence in age- and sex-matched controls. However, in patients with inherited neuropathies, the prevalence of RLS was significantly increased compared to controls and patients with acquired neuropathies.

Previous investigations of the relationship between RLS and neuropathy have produced conflicting results. The first systematic evaluation found RLS in only 5.2% of patients with neuropathy—this study lacked controls and blinding.[10] An unblinded study in patients with Charcot-Marie-Tooth disease reported the prevalence of RLS to be 0/17 in CMT type 1 patients, compared to 10/27 (37%) in CMT2 patients.[6] Subsequent unblinded surveys by the same investigators found a 29% prevalence of RLS in patients with neuropathy (37% in acquired neuropathies, 9% in inherited neuropathies), compared to 9% in controls,[8] and a prevalence of RLS in 33% of patients with small-fiber neuropathy associated with diabetes.[7] Finally, a review of selected patients with neuropathy with symptoms of pain or paresthesias found a prevalence of RLS of 54%.[11] Estimates of the prevalence of neuropathy in patients with RLS have ranged from 2.7 to 37%.[2,17,18] Again, many of these studies lacked controls, and in none was the evaluation conducted in a blinded fashion. One of these studies found evidence of neuropathy on biopsy in 37% of patients with RLS, suggesting that neuropathy can still be an important trigger of RLS.[18] In general, symptoms typical of neuropathy were absent, electrophysiologic changes were variable, and results of quantitative sensory testing were not provided. Our study selected patients with a clinical diagnosis of neuropathy, and therefore persons with a

Table 2 RLS prevalence by neuropathy subtype

	No.	Final diagnosis of RLS, n (%)	*p* Value
Controls, total	245	20 (8.2)	
Neuropathy group, total	245	30 (12.2)	0.14 vs control
Hereditary	72	14 (19.4)	0.016 vs control
			0.033 vs acquired
HMSN 1	20	2 (10.0)	0.68 vs control
			1.0 vs acquired
HMSN 2	31	5 (16.1)	0.18 vs control
			0.33 vs acquired
HSAN	11	4 (36.4)	0.013 vs control
			0.021 vs acquired
Other inherited	10	3 (30.0)	0.051 vs control
			0.073 vs acquired
Non-hereditary (acquired)	173	16 (9.2)	0.73 vs control
			0.033 vs hereditary
Diabetic	35	5 (14.3)	0.22 (vs control)
Toxic	6	2 (33.3)	0.09 (vs control)
GBS/CIDP	26	2 (7.7)	1.0 (vs control)
MGUS	4	0	1.0 (vs control)
Vasculitis	6	1 (16.7)	0.41 (vs control)
Other inflammatory	11	0	1.0 (vs control)
Other	27	1 (3.7)	0.70 (vs control)
Unknown	58	5 (8.6)	1.0 (vs control)

RLS = restless legs syndrome; HMSN = hereditary motor sensory neuropathy; HSAN = hereditary sensory autonomic neuropathy; GBS = Guillain-Barré syndrome; CIDP = chronic inflammatory demyelinating polyneuropathy; MGUS = monoclonal gammopathy of uncertain significance.

neuropathy which presented with isolated RLS symptoms would not have been included in the neuropathy group of our study. Therefore we cannot rule out the fact that there may be a specific subtype of subclinical neuropathy that can increase prevalence of RLS.

We have found specificity and PPV of screening procedures for RLS to be significantly lower in neuropathy cohorts than in control populations. This is not surprising, as the two diagnoses have significant clinical overlap. RLS and neuropathy are both associated with positive symptoms such as pain, paresthesiae, and cramps, all symptoms that may become more noticeable during lack of distraction (which tends to occur at night). This suggests that evaluation of RLS in patients with neuropathy must be performed with caution.

Why RLS appears to be increased in inherited but not in acquired neuropathy is unclear. The increased prevalence of RLS seemed to be most prominent in HMSN 2 and HSAN, with no clear increase in HMSN 1 (although statistical power is insufficient to confirm this). HMSN 2 and HSAN are genetically heterogenous. It is conceivable that some of the genes responsible for these types of neuropathy could have CNS expression and alter CNS dopamine or iron concentrations. Alternatively, there may be pathologic processes in genetically determined axonopathies or neuronopathies, but not in Schwann cell disorders which, in some fashion, unmask symptoms of RLS. The high prevalence in HSAN raises the possibility that certain types of neuropathic sensory symptoms could contribute to RLS, but this is difficult to reconcile with our finding that symptom subtype was not different in patients with and without RLS.

Some limitations of this study should be noted. A number of patients with RLS declined a complete diagnostic evaluation (generally because they felt symptoms were too mild), and so we cannot rule out subclinical iron deficiency in these cases. Although evaluators were blinded to neuropathy diagnosis, several patients inadvertently unblinded themselves during clinical interview, as the process of clarification of symptoms would sometimes disclose symptoms suggestive of peripheral neuropathy. Patients were recruited from a subspecialty neuropathy clinic, which would have an enriched population of certain subtypes (for example, hereditary neuropathies) at the expense of other subtypes (for example, diabetic neuropathy)—it is possible that RLS prevalence could differ in a population-based neuropathy patient group. Genetic confirmation studies of HMSN 2 and HSAN are generally not available, limiting the certainty of these diagnoses. However, analysis with restriction to definite and probable cases of inherited neuropathy did not alter our results. Although the study was large, we cannot rule out the possibility of a small increase in RLS prevalence in acquired neuropathy—post hoc analysis suggests that this study would have 50% power to find a RLS prevalence of 1.7× control values, and 80% power to find a 2× increased prevalence. However, given the fact that point prevalence estimates were almost identical in patients with acquired neuropathy and controls (9.2% vs 8.2%), we feel that a substantial increase in RLS prevalence in patients with acquired neuropathy is unlikely.

Our study also has numerous advantages. The diagnostic evaluation was performed according to a systematic and standardized two-staged screening and diagnostic confirmation protocol. Controls were selected in a systematic manner from two different sources, and were evaluated with an identical protocol as patients with neuropathy. The evaluation of RLS status was performed blinded to neuropathy status, which removes an important source of bias. The study is relatively large, with a sample size at least

twice those of previous studies, and had reasonably good participation rates (65%). Finally, the study includes a systematic analysis of potential confounds and effect modifiers.

AUTHOR CONTRIBUTION

Statistical analysis was conducted by R.B. Postuma.

Received May 12, 2008. Accepted in final form August 29, 2008.

REFERENCES

1. Allen RP, Picchietti D, Hening WA, Trenkwalder C, Walters AS, Montplaisi J. Restless legs syndrome: diagnostic criteria, special considerations, and epidemiology: a report from the restless legs syndrome diagnosis and epidemiology workshop at the National Institutes of Health. Sleep Med 2003;4:101–119.
2. Hogl B, Kiechl S, Willeit J, et al. Restless legs syndrome: a community-based study of prevalence, severity, and risk factors. Neurology 2005;64:1920–1924.
3. Connor JR, Boyer PJ, Menzies SL, et al. Neuropathological examination suggests impaired brain iron acquisition in restless legs syndrome. Neurology 2003;61:304–309.
4. Paulus W, Dowling P, Rijsman R, Stiasny-Kolster K, Trenkwalder C, de Weerd A. Pathophysiological concepts of restless legs syndrome. Mov Disord 2007;22:1451–1456.
5. Allen R. Dopamine and iron in the pathophysiology of restless legs syndrome (RLS). Sleep Med 2004;5:385–391.
6. Gemignani F, Marbini A, Di GG, Salih S, Terzano MG. Charcot-Marie-Tooth disease type 2 with restless legs syndrome. Neurology 1999;52:1064–1066.
7. Gemignani F, Brindani F, Vitetta F, Marbini A, Calzetti S. Restless legs syndrome in diabetic neuropathy: a frequent manifestation of small fiber neuropathy. J Periph Nerv Syst 2007;12:50–53.
8. Gemignani F, Brindani F, Negrotti A, Vitetta F, Alfieri S, Marbini A. Restless legs syndrome and polyneuropathy. Mov Disord 2006;21:1254–1257.
9. Iannaccone S, Zucconi M, Marchettini P, et al. Evidence of peripheral axonal neuropathy in primary restless legs syndrome. Mov Disord 1995;10:2–9.
10. Rutkove SB, Matheson JK, Logigian EL. Restless legs syndrome in patients with polyneuropathy. Muscle Nerve 1996;19:670–672.
11. Nineb A, Rosso C, Dumurgier J, Nordine T, Lefaucheur JP, Creange A. Restless legs syndrome is frequently overlooked in patients being evaluated for polyneuropathies. Eur J Neurol 2007;14:788–792.
12. Folstein MF, Folstein SE, McHugh PR. "Mini-mental state": a practical method for grading the cognitive state of patients for the clinician. J Psychiatr Res 1975;12:189–198.
13. Hening WA, Allen RP, Thanner S, et al. The Johns Hopkins telephone diagnostic interview for the restless legs syndrome: preliminary investigation for validation in a multi-center patient and control population. Sleep Med 2003;4:137–141.
14. Hening WA, Allen RP, Washburn M, Lesage S, Earley CJ. Validation of the Hopkins telephone diagnostic interview for restless legs syndrome. Sleep Med 2008;9:283–289.
15. Meijer JW, Smit AJ, Sonderen EV, Groothoff JW, Eisma WH, Links TP. Symptom scoring systems to diagnose distal polyneuropathy in diabetes: the Diabetic Neuropathy Symptom score. Diabet Med 2002;19:962–965.
16. Allen RP, Kushida CA, Atkinson MJ. Factor analysis of the International Restless Legs Syndrome Study Group's scale for restless legs severity. Sleep Med 2003;4:133–135.
17. Ondo W, Jankovic J. Restless legs syndrome: clinicoetiologic correlates. Neurology 1996;47:1435–1441.
18. Polydefkis M, Allen RP, Hauer P, Earley CJ, Griffin JW, McArthur JC. Subclinical sensory neuropathy in late-onset restless legs syndrome. Neurology 2000;55:1115–1121.

Announcement of Winner

2009 Resident and Fellow Section Writing Award

Neurology® is delighted to announce that **Megan Alcauskas, MD**, a resident at Mount Sinai Hospital in New York City, is the recipient of the first Annual Resident and Fellow Section Writing Award. The Resident and Fellow Section editorial team gave the award for the article "Right Brain: Reading, writing, and reflecting: Making a case for narrative medicine in neurology" (*Neurology*® 2008;70:891–894), which was co-authored by Rita Charon, MD, PhD.

The Resident and Fellow Section Writing Award is intended to recognize the extraordinary writing abilities of those currently training in neurology. The 2010 award will be announced early next year, and eligible articles will include any submission published during 2009 in the Resident and Fellow Section. No formal submission process is required. For questions, contact Kathy Pieper, Editorial Office, *kpieper@neurology.org*.