Elevated amounts of antibodies specific for acetylcholine receptors were detected in 87 percent of sera from 71 patients with myasthenia gravis but not in 175 sera from individuals without myasthenia gravis, including those with other neurologic or autoimmune diseases. Antireceptor antibodies were not directed at the acetylcholine binding site of the receptor. Presence or titer of antibody did not appear to correlate with age, sex, steroid therapy, or duration of symptoms. Myasthenia gravis patients with only ocular symptoms had lower antibody titers, while the majority of titers in myasthenia gravis patients with thymoma exceeded the median titer of the myasthenia gravis group as a whole. Assay of antireceptor antibody should prove a useful test in the diagnosis of myasthenia gravis.

Antibody to acetylcholine receptor in myasthenia gravis

Prevalence, clinical correlates, and diagnostic value

JON M. LINDSTROM, Ph.D., MARJORIE E. SEYBOLD, M.D., VANDA A. LENNON, M.D., Ph.D., SENG A WHITTINGHAM, M.D., and DRAKE D. DUANE, M.D.

Animals immunized with acetylcholine receptor purified from electric organs of *Electrophorus electricus* or *Torpedo Californica* demonstrate striking similarities to patients with myasthenia gravis.¹ These similarities include easy fatigability,^{2,3} decrementing muscle action potential responses to repetitive nerve stimulation,^{2,3} improvement with anticholinesterase drugs,^{2,3} increased sensitivity to curare,^{2,3} small miniature end-plate potentials,⁴ and simplification of the postsynaptic membrane of the neuromuscular junction.^{5,6} Animals with experimental autoimmune myasthenia gravis develop antibodies to electric organ acetylcholine receptor, a small fraction of which also recognizes muscle

acetylcholine receptor.^{2,10} The paucity of acetylcholine binding sites demonstrable in nerve-muscle junctions of patients with myasthenia gravis suggested that antibody to acetylcholine receptor might be present.⁷ Almon, Andrew, and Appel⁸ reported that 30 percent of myasthenia gravis sera tested contained a globulin that blocked the binding of iodine ¹²⁵I α -bungarotoxin to acetylcholine receptor prepared from denervated rat muscle. Employing acetylcholine receptor prepared from human muscle as antigen, we found that in myasthenia gravis,^{9,10} as in experimental autoimmune myasthenia gravis,^{2,9,10,11} antibodies to homologous muscle acetylcholine receptor (antireceptor antibodies) are directed predominantly at sites on the acetylcholine receptor other than the acetylcholine binding site. Appel, Almon, and Levy¹² recently published similar findings in human myasthenia gravis. In this report, we examine the correlation of antibody titers with the clinical parameters of myasthenia gravis and discuss the usefulness of antibody determinations as a diagnostic test for myasthenia gravis.

Methods. Subjects. In 69 of the 71 patients with myasthenia gravis included in this study, the diagnosis

From The Salk Institute for Biological Studies, San Diego (Dr. Lindstrom and Dr. Lennon); Veterans Administration Hospital and University of California, San Diego (Dr. Seybold); Walter and Eliza Hall Institute of Medical Research, Melbourne, Australia (Dr. Whittingham); and the Mayo Clinic and Medical School, Rochester, Minnesota (Dr. Duane).

This work was supported by grants from the National Institutes of Health, the Muscular Dystrophy Association of America, and the California Chapter of the Myasthenia Gravis Foundation.

Received for publication October 20, 1975.

Reprint requests should be addressed to Dr. Lindstrom, The Salk Institute for Biological Studies, PO Box 1909, San Diego, CA 92112.

was confirmed by a positive response to edrophonium or neostigmine, increased sensitivity to curare, and/or a decrementing motor action potential response to repetitive nerve stimulation. Patients who did not have such testing were included only if the case history was entirely compatible with myasthenia gravis. (All histories were reviewed by MES.)

For patients with disorders other than myasthenia gravis, the diagnoses of the referring physicians were accepted. These included 100 patients with other neurologic diseases: diabetic neuropathy (five), chronic inflammatory polyradiculoneuropathy (three), Friedreich's ataxia (five), Charcot-Marie-Tooth disease type II (six), Duchenne's muscular dystrophy (five), Duchenne's dystrophy carrier (four), facioscapulohumeral dystrophy (two), myotonic dystrophy (six), Becker's dystrophy (four), limb-girdle muscular dystrophy (six), amyotrophic lateral sclerosis (20), Werdnig-Hoffman disease (one), Kugelberg-Welander disease (one), McArdle's disease (one), multiple sclerosis (six), epilepsy (one), cerebrovascular accident (three), cerebral tumor (one), spinocerebellar degeneration (one), Eaton-Lambert syndrome (13), and Guillain-Barré syndrome (six). Also included were two patients with thymoma without myasthenia gravis, five patients with diabetes mellitus without neuropathy, and 50 patients with presumed autoimmune diseases: dermatomyositis or polymyositis (10), polymyositis with scleroderma (one), scleroderma (six), Sjögren's syndrome (13), Sjögren's syndrome and rheumatoid arthritis (eight), and systemic lupus erythematosus (12). Normal subjects included 11 males and eight females 20 to 62 years old.

Comparison of antibody titers between groups was made using median titers evaluated by chi-square and Fisher's exact tests.

Antibody assay. All sera were assayed for antibodies to human muscle acetylcholine receptor (antireceptor antibody) by immunoprecipitation using a modification of the previously described method.¹⁰ (Here, α -bungarotoxin is substituted for *Naja naja siamensis*

toxin, and benzoquinonium is used in control assays.) Full details will be published elsewhere.¹³ Human muscle acetylcholine receptor labeled with ¹²⁵I α -bungarotoxin was used as antigen. In triplicate assays, 5 μ l volumes of serum were added to 1 ml volumes of triton X-100 extracts of human muscle containing acetylcholine receptor (2×10^{-10} M). After incubation overnight at 4° C, immunoglobulins in the sera, along with any complexes of antibody-acetylcholine receptor-¹²⁵I-toxin, were precipitated by addition for 4 hours of goat antihuman immunoglobulin G, and the radioactivity of the washed pellet was determined. For each serum tested, triplicate control assays were done with benzoquinonium (10^{-4} M), added to inhibit toxin binding to receptor. This value approximated ¹²⁵I-toxin trapped nonspecifically in the pellet and was subtracted from the value in the absence of benzoquinonium. Sera with high titers ($\geq 10^{-8}$ M) of antireceptor antibody were further tested after 10-fold dilution in normal human serum. Titers of antireceptor antibody were expressed as moles of ¹²⁵I-toxin binding sites precipitated per liter of serum.

Inhibition of toxin binding was used to assay antibody to the acetylcholine binding site (antiacetylcholine site antibody) in sera from 16 myasthenia gravis patients and 12 normal subjects. Immunoglobulin fractions of sera were used in these experiments to minimize nonspecific blockage of toxin binding. Aliquots (20 to 100 μ l) of acetylcholine receptor extract were incubated overnight at 4° C with 200 μ l of test serum globulin or with buffer as control. Next, ¹²⁵I-*Naja naja siamensis* toxin (1×10^{-9} M) was added to the samples. Then, ¹²⁵I-toxin-labeled acetylcholine receptor was separated from free ¹²⁵I-toxin by column chromatography on Sephadex G200 and the radioactivity was measured. Results were expressed as percent of toxin binding with respect to control samples lacking immunoglobulin.

Results. Titers of antireceptor antibody for patients with myasthenia gravis and subjects without myasthenia gravis are summarized in table 1 and the figure. Only sera from myasthenia gravis patients caused precipitation of large

Figure. Distribution of antireceptor antibody titers in subjects with and without myasthenia gravis. ELS = Eaton-Lambert syndrome, ALS = amyotrophic lateral sclerosis.

Table 1. Acetylcholine receptor antibody in patients with myasthenia gravis and controls

Diagnosis	No. tested	Mean titer x 10 ⁻⁹ M	Median titer x 10 ⁻⁹ M	Range x 10 ⁻⁹ M	Percent positive*
Myasthenia gravis	71	54.3	16.3	0.844	87
Controls					
Neurologic diseases	100	0.093	0.039	0-0.415	0
Autoimmune and endocrine diseases	54	0.133	0.095	0-0.563	0
Thymoma without myasthenia gravis	2	0.0830	0.083	0-0.165	0
Healthy subjects	19	0.258	0.253	0.111-0.391	0
Total	175	0.123	0.098	0-0.563	0

*Positive ≥ 0.62 , that is, $0.123 + 4 \text{ SD}$

Table 2. Distribution of antireceptor antibody titers in patients with myasthenia gravis

	Number	Mean (x 10 ⁻⁹ M)	Median (x 10 ⁻⁹ M)	Range (x 10 ⁻⁹ M)
Sex				
Female	40	77.6	22.5	0 - 844
Male	31	24.1	16.0	0 - 82.3
Age				
20 years	15	135.0	44.8	0.389 - 844
21 - 40 years	15	46.0	31.5	0.656 - 254
41 - 60 years	18	26.4	13.0	0 - 209
61 years	23	28.8	9.60	0.182 - 137
Duration myasthenia gravis				
1 year	26	29.1	9.30	0 - 235
1 - 5 years	24	61.2	31.6	0.319 - 395
5 years	20	81.4	13.1	0.0560 - 844
Thymic status				
Thymoma	14	50.6	47.4	6.52 - 137
Thymectomy without thymoma	12	92.2	9.7	0 - 844
Nonoperated	41	48.9	9.0	0 - 395
Medication				
Steroids	9	19.8	16.3	0.520 - 59.1
No steroids	57	63.1	26.8	0 - 844
Severity*				
A	2	14.7	14.7	0.520 - 28.8
1	7	3.76	1.92	0.0560 - 16.0
2A	29	66.3	16.3	0 - 844
2B	29	60.3	34.4	0.389 - 395
3	1	53.3	---	---
4	3	24.8	24.8	2.56 - 62

*Modified Osserman classification: A = remission, 1 = ocular only, 2A = mild generalized, 2B = moderately severe generalized, 3 = acute severe, 4 = chronic severe.

amounts of ¹²⁵I-toxin-labeled acetylcholine receptor. Sera from nonmyasthenic subjects caused precipitation that differed only slightly from the amount of ¹²⁵I-toxin-labeled acetylcholine receptor nonspecifically trapped in the control (benzoquinonium) treated pellet. The upper limit of normal was defined as 0.62×10^{-9} M. This

value is equal to the mean value plus 4 standard deviations for subjects without myasthenia gravis (table 1). Because of the non-Gaussian distribution of titers, this definition of normal range is arbitrary. Similar values were found among all the subgroups without myasthenia gravis: normal, other neurologic diseases, presumed autoimmune

Table 3. Effect of immunoglobulin on 125 I-toxin binding to acetylcholine receptor (AChR)

Sera	Number	Mean percent 125 I-toxin bound* ($\pm$ SD) after adding 200 μ l immunoglobulin to:		
		1 $\times$ 10 ⁻¹⁴ mole AChR	5 $\times$ 10 ⁻¹⁴ mole AChR	25 $\times$ 10 ⁻¹⁴ mole AChR
Normal	12	86 $\pm$ 32	99 $\pm$ 17	99 $\pm$ 24
All myasthenia gravis	16	59 $\pm$ 43	78 $\pm$ 39	72 $\pm$ 31
Myasthenia gravis inhibiting binding	6	8.2 $\pm$ 5.1	31 $\pm$ 9.8	40 $\pm$ 11

*In the absence of immunoglobulin, binding of toxin to AChR was 100 percent.

or endocrine diseases, or thymoma without myasthenia gravis (table 1). The mean value for the total group without myasthenia gravis (No. = 175) was 0.123×10^{-9} M, the median 0.098×10^{-9} M, and range 0 - 0.563×10^{-9} M.

Significant titers were found in 62 of 71 (87 percent) patients with myasthenia gravis (table 1). The range of antireceptor antibody titers in patients with myasthenia gravis was very wide (0 - 844×10^{-9} M), with a mean of 54.3×10^{-9} M and median of 16.3×10^{-9} M. The difference between the groups with and without myasthenia gravis is highly significant ($p < 0.001$).

Comparisons of titers were made on the basis of sex, age, duration of symptoms, thymoma or thymectomy, modified Osserman classification of severity,¹⁴ and treatment with steroid medication (table 2). Using median values, myasthenia gravis patients with thymoma had higher titers than thymectomized patients without thymoma ($p = 0.013$), and individuals with ocular myasthenia gravis had lower titers than those with mild or moderately severe generalized disease ($p = 0.01$). All other comparisons were not statistically significant.

Sera from a woman with myasthenia gravis and from her twin newborn sons with neonatal myasthenia gravis were assayed but not included in the statistical analyses because of the lack of detailed clinical information. The mother and both infants had significant levels of antibody (31.1×10^{-9} M, 7.88×10^{-9} M, and 6.89×10^{-9} M, respectively).

Antibody to the acetylcholine binding site of the receptor molecule was assayed by examining binding of 125 I-toxin to acetylcholine receptor preincubated with serum (table 3). Fixed amounts of serum were incubated with three different concentrations of receptor. At all concentrations of receptor, normal sera caused little blockage of 125 I-toxin binding, but standard deviations were large. The average for myasthenia gravis patients indicated that some sera may have inhibited toxin binding. However, only six (38 percent) of the 16 sera from myasthenia gravis patients inhibited toxin binding to a greater extent than any normal serum under all three conditions tested. Thus, the detection of myasthenia gravis patients by immunoprecipitation assay is significantly greater than by the toxin binding method (chi-square $p = < 0.001$).

Discussion. No significant titer of antireceptor antibody was found in subjects without myasthenia gravis. This group included patients with disorders that may show decrementing motor action potential responses to repetitive nerve stimulation, such as amyotrophic lateral sclerosis,¹⁵ and patients with Eaton-Lambert syndrome,¹⁶ a disorder associated with a defect of neuromuscular transmission. Patients with systemic lupus erythematosus, a disease characterized by autoantibodies, did not have significant levels of antireceptor antibody, suggesting that this antibody is not merely indicative of an autoimmune diathesis.

Eight-seven percent of patients with myasthenia gravis had antireceptor antibody in excess of that seen in any person without this disease. No correlation was found, however, with duration of the disease, age, sex, or steroid therapy. Although patients with thymoma generally had high antibody titers, this does not appear valuable diagnostically since many patients without thymoma also had high titers. As a group, patients with only ocular signs have lower antibody titers than those with generalized disease. However, within the generalized group, it is not possible to predict severity on the basis of antibody titer.

A review of the nine myasthenia gravis patients without detectable antireceptor antibody failed to reveal a consistent similarity; neither sex, age, nor duration of symptoms seemed a common factor. Two had a thymectomy prior to collection of the sera but neither had a thymoma. Only one of the nine was known to be receiving steroid medication at the time of the study, and that patient was in remission. The others were classified in severity as groups 1 (two), 2A (four), or 2B (two).

The absence of significant antibody titers in some myasthenia gravis patients is unexplained. It is possible that myasthenia gravis represents not one but two or several disorders with similar clinical and neurophysiologic appearances. One "variety" of myasthenia gravis, apparently the most common one, could be characterized by antireceptor antibody. Alternatively, our present sensitivity may not be sufficient to reliably detect very low titers of antireceptor antibody. There is some evidence that this may be the case, since 25 percent of myasthenia gravis patients had titers between 0.6 and 5.0×10^{-9} M. Another possibility is that serum antibody titers do not necessarily reflect antibody activity

in the microenvironment of the end-plate. Finally, titers may fluctuate in response to factors as yet unrecognized.

It is clear that the disease process involves more than the binding of antibody to acetylcholine receptor. There is not a close correlation between antibody titer and disease intensity, as would be expected if antibodies acted only as curarelike antagonists of acetylcholine receptor. In addition, postsynaptic membrane structure is simplified in myasthenia gravis,^{5,6} suggesting that a reduction in the number of acetylcholine receptor molecules and alteration of their arrangement account for some of the defect in transmission. Simplification of postsynaptic membrane structure probably results from the antibody response to acetylcholine receptor, since it is also observed in the immunologically induced experimental model experimental autoimmune myasthenia gravis.^{5,6} Thus, the defect in transmission in myasthenia gravis probably involves synthesis and destruction as well as antagonism of acetylcholine receptor.

Most of the antibody to receptor found in patients with myasthenia gravis was directed at determinants other than the acetylcholine binding site. Antiacetylcholine site antibody would be expected to prevent binding of toxin to acetylcholine receptor. Although 38 percent of the sera tested did reduce toxin binding to acetylcholine receptor, the effect was small and seen only when the concentration of antireceptor antibody exceeded that of acetylcholine receptor by a factor of 200 to 1,000-fold. Inhibition observed under these conditions might result from steric hindrance by extensive binding of antibodies to sites on acetylcholine receptor other than the acetylcholine site. By the criterion for significance that we applied to antireceptor titers, none of the myasthenia gravis sera differ from normal in inhibition of toxin binding.

Other investigators also have reported detection of antibodies to acetylcholine receptor in sera from patients with myasthenia gravis. Using acetylcholine receptor isolated from denervated rat muscle as antigen, antibodies were detected in 30 percent of the sera tested by inhibition of toxin binding⁸ and in 68 percent of the sera by binding to toxin-acetylcholine receptor complexes.¹² Using acetylcholine receptor isolated from *Torpedo* electric organ as antigen, antibodies were detected in 66 percent of sera by complement fixation.¹⁷ Using acetylcholine receptor isolated from human muscle as antigen, we have found antibodies in the sera of 87 percent of patients with myasthenia gravis, the average value being 440-fold that of nonmyasthenics. With the other methods for quantitating antibody, sera from patients with myasthenia gravis differed from normals by only 0.2 to 3-fold. Using sections of nonmyasthenic human muscle as substrate, inhibition of toxin binding was detected histologically in 75 percent of sera from myasthenia gravis patients tested.¹⁸ This method, while more sensitive than methods using nonhuman acetylcholine receptor, is not quantitative and is more tedious and less sensitive than the method we have described.

Defective neuromuscular transmission in myasthenia gravis probably results in part from the action of antireceptor antibodies. The number of toxin binding sites

at end-plates is reduced in both myasthenia gravis⁷ and experimental autoimmune myasthenia gravis.^{19,20} This presumably results from antibody binding to receptors in the membrane and/or from the simplification of the postsynaptic membrane. Antibodies in animals with experimental autoimmune myasthenia gravis, like those in patients with myasthenia gravis, are directed mostly at sites on the receptor other than the acetylcholine site. Experimental autoimmune myasthenia gravis sera block the electrophysiologic activity of receptors on electroplaques^{10,11} and muscle.^{19,21} These observations may also apply to myasthenia gravis. Rats immunized with eel acetylcholine receptor demonstrate rising titers of antirat muscle acetylcholine receptor antibody during the development of the late stage of experimental autoimmune myasthenia gravis, the stage that closely resembles myasthenia gravis.¹⁰ However, since serial titers in individual patients were not included in this study, comparison of fluctuations in disease with antibody titer cannot be made.

Detection of antireceptor antibodies in a mother with myasthenia gravis and her neonatally myasthenic babies suggests transplacental transfer of these antibodies. Simpson²² first suggested that such antibodies could be responsible for neonatal myasthenia and that this form of myasthenia was transient because maternal antibodies were eliminated from the newborn.

The results thus far obtained indicate that antireceptor antibody is present in the serum of most myasthenia gravis patients and is specific to myasthenia gravis. Assay of antireceptor antibody offers an additional diagnostic test for myasthenia gravis. The two most frequently used diagnostic tools for myasthenia gravis, edrophonium testing and repetitive nerve stimulation, while highly characteristic in myasthenia gravis, may yield positive results in disorders other than myasthenia gravis. The supplementation of edrophonium and electromyography by antireceptor antibody studies should increase the reliability of the usual diagnostic approach.

In summary, antireceptor antibody, similar to that found in animals with experimental autoimmune myasthenia gravis, is detectable in patients with myasthenia gravis. Antireceptor, but not antiacetylcholine site, antibody is detected in most patients with myasthenia gravis but not in persons without myasthenia gravis. Presence or level of antibody does not appear to correlate with age, sex, duration of symptoms, or steroid therapy. Correlation was observed between antibody titer and both the presence of thymoma and the restriction of the disease to the ocular muscles. Assay of antireceptor antibody may prove a useful test in the diagnosis of myasthenia gravis.

Acknowledgments

We thank Brett Einarson, Mac Campbell, and Steve Woods for expert technical assistance.

In addition to the authors, sera were obtained through the courtesy of Drs. M. Auspaugh, L. Blakey, M. Cherrington, D. Delassio, W. DeBolt, P. Dyck, P. Ebeling, R. Galbraith, E. Lambert, J. Peter, J. Rosenberg, L. Meyers, and E. Tan.

Human muscle tissue was obtained through the kind cooperation of the Departments of Surgical Pathology at the following San Diego hospitals:

REFERENCES

1. Patrick J, Lindstrom J: Autoimmune response to acetylcholine receptor. *Science* 18:871-872, 1973
2. Lennon VA, Lindstrom JM, Seybold ME: Experimental autoimmune myasthenia: A model of myasthenia gravis in rats and guinea pigs. *J Exp Med* 141:1365-1375, 1975
3. Seybold ME, Lambert EH, Lennon VA, et al: Experimental autoimmune myasthenia: Clinical, neurophysiologic, and pharmacologic aspects. *Ann NY Acad Sci* 274:285-282, 1976
4. Lambert EH, Lindstrom JM, Lennon VA: End-plate potentials in experimental autoimmune myasthenia gravis. *Ann NY Acad Sci* 274:300-318, 1976
5. Engel AG, Tsujihata M, Lindstrom JM, et al: The motor end-plate in myasthenia gravis and in experimental autoimmune myasthenia gravis. A quantitative ultrastructural study. *Ann NY Acad Sci* 274:60-79, 1976
6. Engel AG, Tsujihata M, Lambert EH, et al: Experimental autoimmune myasthenia gravis: A sequential and quantitative study of the neuromuscular junction ultrastructure and electrophysiologic correlations. *J Neuropathol Exp Neurol* (in press)
7. Fambrough D, Drachman DB, Satyamurti S: Neuromuscular junction in myasthenia gravis: Decreased acetylcholine receptors. *Science* 182:293-295, 1973
8. Almon RR, Andrew CG, Appel SN: Serum globulin in myasthenia gravis: Inhibition of α -bungarotoxin binding to acetylcholine receptors. *Science* 186:55-57, 1974
9. Lindstrom J: Immunological studies of acetylcholine receptors. *J Supramol Struct* 4:389-403, 1976
10. Lindstrom JM, Lennon VA, Seybold ME, et al: Experimental autoimmune myasthenia gravis and myasthenia gravis: Biochemical and immunochemical aspects. *Ann NY Acad Sci* 274:254-274, 1976
12. Appel S, Almon R, Levy N: Acetylcholine receptor antibodies in myasthenia gravis. *N Engl J Med* 293:760-761, 1975
11. Patrick J, Lindstrom J, Culp B, et al: Studies on purified eel acetylcholine receptor and anti-acetylcholine receptor antibody. *Proc Natl Acad Sci USA* 70:3334-3338, 1973
13. Lindstrom JM: An assay for antibodies to human acetylcholine receptor in serum from patients with myasthenia gravis. *Clin Immunol Immunopathol* 6, 1976
14. Perlo VP, Poskanzer DC, Schwab RS, et al: Myasthenia gravis: Evaluation of treatment in 1,355 patients. *Neurology (Minneap)* 16:431-439, 1966
15. Mulder DW, Lambert EH, Eaton LM: Myasthenic syndrome in patients with amyotrophic lateral sclerosis. *Neurology (Minneap)* 9:627-631, 1959
16. Lambert EH, Elmquist D: Quantal components of end-plate potentials in the myasthenic syndrome. *Ann NY Acad Sci* 183:183-199, 1971
17. Aharonov A, Tarrab-Hazdai R, Abramsky O, et al: Humoral antibodies to acetylcholine receptor in patients with myasthenia gravis. *Lancet* 2:340-342, 1975
18. Bender AN, Engel WK, Ringel SP, et al: Myasthenia gravis: A serum factor blocking acetylcholine receptors of the human neuromuscular junction. *Lancet* 1:607-609, 1975
19. Green DPL, Miledi R, Vincent A: Neuromuscular transmission after immunization against acetylcholine receptors. *Proc R Soc Lond [Biol]* 189:57-68, 1975
20. Lindstrom J, Einason BL, Lennon VA, et al: Pathological mechanisms in experimental autoimmune myasthenia gravis I. *J Exp Med* 144, 1976
21. Bevan S, Heinemann S, Lennon VA, et al: Reduced muscle acetylcholine sensitivity in rats immunized with acetylcholine receptor. *Nature* 260:438-439, 1976
22. Simpson JA: Myasthenia gravis: A new hypothesis. *Scott Med J* 5:419-436, 1960

Neurology®

Antibody to acetylcholine receptor in myasthenia gravis: Prevalence, clinical correlates, and diagnostic value

JON M. LINDSTROM, MARJORIE E. SEYBOLD, VANDA A. LENNON, et al.

Neurology 1976;26:1054

DOI 10.1212/WNL.26.11.1054

This information is current as of November 1, 1976

Updated Information & Services	including high resolution figures, can be found at: http://n.neurology.org/content/26/11/1054.full
Citations	This article has been cited by 23 HighWire-hosted articles: http://n.neurology.org/content/26/11/1054.full##otherarticles
Permissions & Licensing	Information about reproducing this article in parts (figures, tables) or in its entirety can be found online at: http://www.neurology.org/about/about_the_journal#permissions
Reprints	Information about ordering reprints can be found online: http://n.neurology.org/subscribers/advertise

Neurology® is the official journal of the American Academy of Neurology. Published continuously since 1951, it is now a weekly with 48 issues per year. Copyright © 1976 by the American Academy of Neurology. All rights reserved. Print ISSN: 0028-3878. Online ISSN: 1526-632X.