Patient Age/Gender: Unknown Unknown Printed: 23-Sep-19 11:52:33

						Departed /
Procedure	Result	Units	Ref Interval	Accession	Collected Received	l Verified
Striated Muggle Antibodies IgC Saroon	Detected *	0112.00		19-266-900131	23-Sep-19 23-Sep-1	9 23-Sep-19
Stillated Muscle Antibodies, 196 Screen	Detected		[<1:40]		11:35:00 11:35:00) 11:44:26
Striated Muscle Antibodies IgG Titer	1:40 *		[<1:40]	19-266-900131	23-Sep-19 23-Sep-1	L9 23-Sep-19
berraeea habere mierboareb, 196 freer	1.10		[(1 10]		11:35:00 11:35:00) 11:44:32
CASPR2 Ab IgG Screen by IFA, Serum	Detected		[<1:10]	19-266-900131	23-Sep-19 23-Sep-1	L9 23-Sep-19
					11:35:00 11:35:00) 11:44:58
CASPR2 Ab IgG Titer by IFA, Serum	1:20 *		[<1:10]	19-266-900131	23-Sep-19 23-Sep-1	L9 23-Sep-19
· · · · · · · · · · · · · · · · · · ·					11:35:00 11:35:00) 11:45:07
LGI1 Ab IgG Screen by IFA, Serum	Detected		[<1:10]	19-266-900131	23-Sep-19 23-Sep-1	L9 23-Sep-19
					11:35:00 11:35:00) 11:44:58
LGI1 Ab IgG Titer by IFA, Serum	1:40 *		[<1:10]	19-266-900131	23-Sep-19 23-Sep-1	L9 23-Sep-19
					11:35:00 11:35:00) 11:45:08
Acetylcholine Binding Antibody	0.8 Н	nmol/L	[0.0-0.4]	19-266-900131	23-Sep-19 23-Sep-	19 23-Sep-19
			[0 0 C]	10 000 000101	11:35:00 11:35:00) 11:44:26
Acetylcholine Blocking Antibody	зт н	olo	[0-26]	19-200-900131	23-Sep-19 23-Sep-	11.44.26
Arctulabeline Medulating Antibedu	60 17	0.		19-266-900131	23-Sep-19 23-Sep-) 11.44.20 19 23-Sen-19
Acetylcholine Modulating Antibody	60 H	6	[<=45]	19 200 900191	11:35:00 11:35:00) 11:44:58
D/O-Time Caldium Channel Antibedu	22 0	nmol/I	[0 0_24 F]	19-266-900131	23-Sep-19 23-Sep-	19 23-Sep-19
P/Q-Type Calcium Channel Antibody	22.0	Puiot/T	[0.0-24.5]		11:35:00 11:35:0/) 11:44:26
Voltage-Gated Potassium Channel Ab, Ser	415 H	pmol/L	[0-31]	19-266-900131	23-Sep-19 23-Sep-	L9 23-Sep-19
	110 11	Pmor/H			11:35:00 11:35:00) 11:44:26
Titin Antibody	0.30	TV	[0, 00-0, 45]	19-266-900131	23-Sep-19 23-Sep-1	L9 23-Sep-19
110111 111012004/	0.00	±.,	[0.00 0.10]		11:35:00 11:35:00) 11:44:26

23-Sep-19 11:35:00 Striated Muscle Antibodies, IgG Screen

Striated Muscle Antibodies, IgG detected. Titer results to follow.

- 23-Sep-19 11:35:00 CASPR2 Ab IgG Screen by IFA, Serum CASPR2 Antibody, IgG is detected. Titer results to follow.
- 23-Sep-19 11:35:00 LGI1 Ab IgG Screen by IFA, Serum LGI1 Antibody, IgG is detected. Titer results to follow.

23-Sep-19 11:35:00 Striated Muscle Antibodies, IgG Screen: INTERPRETIVE DATA: Striated Muscle Antibodies, IgG Screen

In the presence of acetylcholine receptor (AChR) antibody, striated muscle antibodies, which bind in a cross-striational pattern to skeletal and heart muscle tissue sections, are associated with late-onset myasthenia gravis (MG). Striated muscle antibodies recognize epitopes on three major muscle proteins, including: titin, ryanodine receptor (RyR) and Kvl.4 (an alpha subunit of voltage-gated potassium channel [VGKC]). Isolated cases of striated muscle antibodies may be seen in patients with certain autoimmune diseases, rheumatic fever, myocardial infarction, and following some cardiotomy procedures.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement A: aruplab.com/CS

23-Sep-19 11:35:00 CASPR2 Ab IgG Screen by IFA, Serum: INTERPRETIVE INFORMATION: CASPR2 Ab IgG w/Reflex to Titer, Serum Contactin-associated protein-2 (CASPR2) IgG antibody may occur as part of the voltagegated potassium channel (VGKC) complex antibodies.

The presence of CASPR2 IgG antibody is associated with a wide spectrum of clinical manifestations, including acquired neuromyotonia, limbic encephalitis, painful neuropathy and Morvan syndrome. Tumors such as thymoma, small-cell lung cancer, and other rarer tumors may occur. The full-spectrum of clinical disorders and tumors associated with the

* Abnormal, # = Corrected, C = Critical, f = Footnote, H = High, L = Low, t = Interpretive Text, @ = Reference Lab

CASPR2 IgG antibody continues to be defined. Results should be interpreted in correlation with the patient's clinical history and other laboratory findings.

This indirect fluorescent antibody assay utilizes contactin-associated protein-2 (CASPR2) transfected cell lines for the detection and semi-quantification of the CASPR2 IgG antibody.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

23-Sep-19 11:35:00 CASPR2 Ab IgG Titer by IFA, Serum: INTERPRETIVE INFORMATION: CASPR2 Ab Titer IgG by IFA, Serum Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

23-Sep-19 11:35:00 LGI1 Ab IgG Screen by IFA, Serum: INTERPRETIVE INFORMATION: LGI1 Ab IgG w/Reflex to Titer, Serum Leucine-rich, glioma-inactivated 1 protein (LGI1) IgG antibody may occur as part of the voltage-gated potassium channel (VGKC) complex antibodies.

The presence of LGI1 IgG antibody is mainly associated with limbic encephalitis, hyponatremia and myoclonic movements. LGI1 IgG antibody is rarely associated with tumors but may occur infrequently in Morvan syndrome, neuromyotonia and idiopathic epilepsy. The full-spectrum of clinical disorders associated with the LGI1 IgG antibody continues to be defined. Results should be interpreted in correlation with the patient's clinical history and other laboratory findings.

This indirect fluorescent antibody assay utilizes leucine-rich, glioma-inactivated 1 protein (LGI1) transfected cell lines for the detection and semi-quantification of the LGI1 IgG antibody.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

23-Sep-19 11:35:00 LGI1 Ab IgG Titer by IFA, Serum: INTERPRETIVE INFORMATION: LGI1 Ab Titer IgG by IFA, Serum Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

23-Sep-19 11:35:00 Acetylcholine Binding Antibody: INTERPRETIVE INFORMATION: Acetylcholine Binding Ab

Negative 0.0 - 0.4 nmol/L Positive 0.5 nmol/L or greater

Approximately 85-90 percent of patients with myasthenia gravis (MG) express antibodies to the acetylcholine receptor (AChR), which can be divided into binding, blocking, and

* Abnormal, # = Corrected, \mathbf{C} = Critical, \mathbf{f} = Footnote, \mathbf{H} = High, \mathbf{L} = Low, \mathbf{t} = Interpretive Text, @ = Reference Lab

modulating antibodies. Binding antibody can activate complement and lead to loss of AChR. Blocking antibody may impair binding of acetylcholine to the receptor, leading to poor muscle contraction. Modulating antibody causes receptor endocytosis resulting in loss of AChR expression, which correlates most closely with clinical severity of disease. Approximately 10-15 percent of individuals with confirmed myasthenia gravis have no measurable binding, blocking, or modulating antibodies.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

23-Sep-19 11:35:00 Acetylcholine Blocking Antibody: INTERPRETIVE INFORMATION: Acetylcholine Blocking Ab

Negative	0-26 percent	blocking
Indeterminate	27-41 percent	blocking
Positive	42 percent or	greater blocking

Approximately 85-90 percent of patients with myasthenia gravis (MG) express antibodies to the acetylcholine receptor (AChR), which can be divided into binding, blocking, and modulating antibodies. Binding antibody can activate complement and lead to loss of AChR. Blocking antibody may impair binding of acetylcholine to the receptor, leading to poor muscle contraction. Modulating antibody causes receptor endocytosis resulting in loss of AChR expression, which correlates most closely with clinical severity of disease. Approximately 10-15 percent of individuals with confirmed myasthenia gravis have no measurable binding, blocking, or modulating antibodies.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

23-Sep-19 11:35:00 Acetylcholine Modulating Antibody: INTERPRETIVE INFORMATION: Acetylcholine Modulating Ab

Negative 0-45 percent modulating Positive 46 percent or greater modulating

Approximately 85-90 percent of patients with myasthenia gravis (MG) express antibodies to the acetylcholine receptor (AChR), which can be divided into binding, blocking, and modulating antibodies. Binding antibody can activate complement and lead to loss of AChR. Blocking antibody may impair binding of acetylcholine to the receptor, leading to poor muscle contraction. Modulating antibody causes receptor endocytosis resulting in loss of AChR expression, which correlates most closely with clinical severity of disease. Approximately 10-15 percent of individuals with confirmed myasthenia gravis have no measurable binding, blocking, or modulating antibodies.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

23-Sep-19 11:35:00 P/Q-Type Calcium Channel Antibody: INTERPRETIVE INFORMATION: P/Q-Type Calcium Channel Antibody

* Abnormal, # = Corrected, **C** = Critical, **f** = Footnote, **H** = High, **L** = Low, **t** = Interpretive Text, @ = Reference Lab

Patient Age/Gender: Unknown Unknown Printed: 23-Sep-19 11:52:33

0.0 to 24.5 pmol/L Negative 24.6 to 45.6 pmol/L Indeterminate 45.7 pmol/L or greater.... Positive

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement B: aruplab.com/CS

23-Sep-19 11:35:00 Voltage-Gated Potassium Channel Ab, Ser: INTERPRETIVE INFORMATION: Voltage-Gated Potassium Channel (VGKC) Antibody, Serum

Negative 31 pmol/L or less Indeterminate... 32 - 87 pmol/L Positive 88 pmol/L or greater

Voltage-Gated Potassium Channel (VGKC) antibodies are associated with neuromuscular weakness as found in neuromyotonia (also known as Issacs syndrome) and Morvan syndrome. VGKC antibodies are also associated with paraneoplastic neurological syndromes and limbic encephalitis; however, VGKC antibody-associated limbic encephalitis may be associated with antibodies to leucine-rich, glioma-inactivated 1 protein (LGI1) or contactin-associated protein-2 (CASPR2) instead of potassium channel antigens. A substantial number of VGKC-antibody positive cases are negative for LGI1 and CASPR2 IgG autoantibodies, not all VGKC complex antigens are known. The clinical significance of this test can only be determined in conjunction with the patient's clinical history and related laboratory testing.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS

23-Sep-19 11:35:00 Titin Antibody: INTERPRETIVE INFORMATION: Titin Antibody

Negative 0.00 - 0.45 IV Indeterminate ... 0.46 - 0.71 IV Positive 0.72 IV or greater

The presence of titin antibody is associated with late onset of myasthenia gravis (MG) and a variable risk for thymoma. Titin antibody may be detected in 20-40 percent of all patients with MG; higher frequency in older population as a whole.

Test developed and characteristics determined by ARUP Laboratories. See Compliance Statement D: aruplab.com/CS